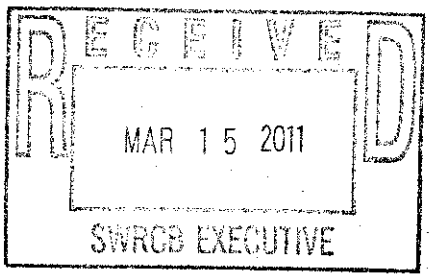

COMMENTS ON CALIFORNIA STATE WATER RESOURCES CONTROL BOARD'S

PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1, SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE

JANUARY 2011

Submitted by: Date: March 15, 2011

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Building Industry Legal Defense Foundation
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California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Southern California Contractors Association

Our organizations appreciate the opportunity to submit public comments to the California State Water Resources Control Board ("SWB"), in response to the issuance on January 28, 2011 of proposed amendments to the Water Quality Control Plan for Enclosed Bays and Estuaries – Part 1 Sediment Quality for the Protection of Fish and Wildlife ("the Bays and Estuaries Plan").[1]

Our organizations support and encourage the SWB's efforts in developing sediment quality objectives and related implementation policies in the Bays and Estuaries Plan that are protective of benthic invertebrates, fish and wildlife, and human health. However, we believe the proposed amendments to the Bays and Estuaries Plan are not consistent with SWB's stated objectives, and, instead, propose excessively low concentrations for certain compounds which have no rational basis in science and do not correspond to any identifiable decrease in risk.

Specifically, we have serious concerns with SWB's proposed amendments to the Chemical Score Index ("CSI") because these amendments are contrary to real-world, scientifically-observed conditions. The proposed amendments also increase the risk that Regional Water Quality Control Boards may misuse concentration ranges by extracting these values from the Bays and Estuaries Plan and using them in an inappropriate manner to establish cleanup levels, in contravention of the integrated, multi-step process set forth in the Bays and Estuaries Plan. Indeed, staff for one such Regional Board already has done just this, as demonstrated by the California Regional Water Quality Control Board, Los Angeles Region's ("RWB") recent draft Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters total maximum daily load for toxic pollutants ("the Draft TMDL"). Accordingly, we request that the SWB revise its proposed amendments to be consistent with good science.

I. THE PROPOSED CSI AMENDMENTS FOR DDT ARE NOT SCIENTIFICALLY BASED

A. Benthic Organisms Are Not Affected By DDT Even At Levels Hundreds Of Times Higher Than Those In The Current Or Proposed CSI.

The proposed amendments to the Bays and Estuaries Plan would lower the category score concentration ranges for the CSI for DDE, DDT, and DDD. Under these amendments, the "high disturbance" thresholds for DDE, DDT, and DDD would be lowered from 154, 89.3, and 117 ug/kg, respectively, to 45.84, 34.27, and 26.37 ug/kg respectively. In addition, the DDE, DDT,

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[1] These comments are based on SWB's report entitled, "Proposed Amendments to the Water Quality Control Plan for Enclosed Bays and Estuaries – Part 1 Sediment Quality for the Protection of Fish and Wildlife" (January 28, 2011) (hereinafter "Staff Report"), and attachments thereto. We respectfully request that the cover letter, these public comments, appendices, and attachments submitted herewith be given appropriate consideration, be placed in the administrative record for the proposed Bays and Estuaries Plan amendments and be maintained in the agency's records.

and DDD values for the moderate, low, and reference disturbance categories would also change,[2] as would the weighting factors.[3]

These proposed amendments to the DDE, DDT, and DDD thresholds in the Bays and Estuaries Plan are unnecessary because the current thresholds are far too low in the first place, and do not correspond to any level of disturbance to benthic organisms. To be consistent with relevant published thresholds regarding potential effects of DDT on benthic organisms, the current CSI thresholds would need to be increased significantly – not lowered as proposed. There is a published no-observed-effect concentration ("NOEC") for total DDTs in the Southern California Bight of 8.51 mg/kg, or parts per million ("ppm") (equivalent to 8,510 ug/kg, or parts per billion ("ppb")).[4] A NOEC is the concentration below which there is no observed harm to the organisms at issue. This NOEC level indicates that DDT does not represent a threat to the benthic community at ambient levels prevalent throughout most of the water bodies to which the Bays and Estuaries Plan applies.[5] It is based on samples collected in 1994, and, since that time, DDT in the Southern California Bight has further aged, weathered, and been subject to other processes that would tend to make it less bioavailable. Thus, the NOEC today reasonably can be expected to be significantly greater than 8,510 ppb.

The published 8.51 mg/kg value corresponds to the concentration below which there were no observed effects on organisms in 1994, yet it is *hundreds of times greater* than the values currently in the CSI "disturbance" table and those proposed for the amended "disturbance" table. There is no scientific or evidentiary basis for SWB to conclude that sediment in bays and estuaries is in any way "disturbed" by DDT at levels below 8.51 mg/kg. We respectfully request that SWB set the threshold for "low disturbance" above 8.51 mg/kg, and that the moderate and high disturbance category scores are set even higher.

B. DDT Is Not A Risk To People; Any Threshold Concentration Based On Protecting Human Health Should Be Raised, Not Lowered.

SWB's legislative mandate to promulgate the Bays and Estuaries Plan states that the Bays and Estuaries Plan should be geared towards protecting against any health risk from the exposure

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2 The upper thresholds for the moderate disturbance categories would go from (all in ug/kg) 154 to 45.84 (for DDE), 89.3 to 34.27 (DDT), and 117 to 26.37 (DDD). The upper thresholds for the low disturbance categories would go from (all in ug/kg) 4.15 to 6.01 (DDE), 1.52 to 2.79 (DDT), and 2.69 to 3.56 (DDD). Finally, the reference disturbance categories would go from (all in ug/kg) 0.50 to 1.19 (DDE), 0.50 to 0.61 (DDT), and 0.50 to 0.77 (DDD).

3 The weighting factors would change from 31 to 33 (DDE), 16 to 20 (DDT), and 46 to 45 (DDD).

4 Chapman, P.M. (1996). A Test of Sediment Effect Concentrations: DDT and PCB in the Southern California Bight. Env. Tox. and Chem., 15: 1197-1198.

5 See attached expert repot of Dr. Susan Kane Driscoll regarding bioavailability of DDT. This expert report was prepared in support of Montrose Chemical Corporation of California's ("Montrose") public comment letter on the Draft TMDL (discussed below).

of humans to pollutants through the food chain to edible fish, shell fish, or wildlife.[6] As such, the threshold values for pollutants in the Bays and Estuaries Plan should be based on values that pose a health risk to humans. As relevant here, this has not been done for DDT, as DDT does not present a risk to human health.

Numerous published studies have shown that DDT does not pose a risk to human health. In fact, discovery of DDT's insecticidal properties won Dr. Paul Mueller, the discoverer, the Nobel Prize in Physiology or Medicine in 1948.[7] These insecticidal properties have been used to improve human health throughout the world by saving lives that would otherwise be lost to malaria.[8] Multiple studies have demonstrated that there is a lack of connection between DDT and various cancers.[9]

Because DDT does not have adverse impacts on human health, any threshold value which is used in a process designed to protect human health should be raised from the current values, not lowered as contemplated by the proposed Bays and Estuaries Plan amendments to the CSI thresholds.

C. The Proposed Amendments To The Bays And Estuaries Plan Increase The Risk Of Misuse By Regional Boards.

The proposed amendments to the CSI disturbance table increase the risk of misuse by Regional Boards, as described below.

1. The Bays and Estuaries Plan is an integrated policy, where all steps are required to assess sediment quality.

It is our understanding that the Bays and Estuaries Plan is an integrated state-wide policy for the regulation of contaminated sediments. Specifically, the Bays and Estuaries Plan adopts a triad approach to assessing whether a particular sediment is impacted by toxic substances, and

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6 Cal. Water Code § 13393(b) ("[t]he state board shall base the sediment quality objectives on a health risk assessment if there is a potential for exposure of humans to pollutants through the food chain to edible fish, shellfish, or wildlife.").

7 Expert Report of Donald Roberts, PhD from Garza v. Allied Chem. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas (describing the beneficial uses of DDT and the lack of negative human health effects).

8 Expert Report of Amir Attaran, PhD, LL.B from Garza v. Allied Chem. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas (describing the lack of negative human health effects of DDT and highlighting the extensive uses which improve human health and save lives that would otherwise be lost to malaria).

9 See, e.g., Expert Report of Seymore Grufferman, M.D., Dr. P.H. from Garza v. Allied Chem. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas (examining the lack of link between DDT and cancer); Expert Report of Marion J. Fedoruk from Garza v. Allied Chem. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas (examining the lack of link between DDT and non-Hodgkin lymphoma).

then employs a step-wise approach to identify the toxic agent and set a target for cleanup.[10] The triad approach uses multiple lines of evidence ("MLOE") to conclude whether sediment is impacted. These MLOE include sediment toxicity, benthic community condition, and sediment chemistry.[11] The Bays and Estuaries Plan makes it clear that no single line of evidence ("LOE") is a sufficient tool to assess sediment quality when used alone because no "individual LOE is sufficiently reliable when used alone to assess sediment quality impacts due to toxic pollutants."[12] Additionally, the Bays and Estuaries Plan states that "the chemistry LOE . . . shall not be used for setting cleanup levels or numeric values for technical TMDLs."[13]

It is clear that the Bays and Estuaries Plan requires the use of an integrated, multi-step process, including the use of the MLOE and step-wise processes, where each step is completed and all three of the LOEs are used concurrently to assess sediment quality.[14] Unfortunately, this is not what is happening at the Regional Board level, as demonstrated by RWB's recent development of the Draft TMDL.

2. RWB impermissibly uses portions of the Bays and Estuaries Plan in isolation, and fails to complete the required multi-step process.

In December 2010, the RWB released the Draft TMDL to address alleged impairments from toxic pollutants in waterbodies in the Dominguez Channel and Greater Los Angeles and Long Beach Harbor waters (collectively, "the Harbor Waters"). As part of the Draft TMDL, the RWB proposed "sediment targets" for several pollutants, including DDT.

The Draft TMDL ignores the integrated, multi-step process set forth in the Bays and Estuaries Plan. Instead, the Draft TMDL improperly excerpts portions of the Bays and Estuaries Plan and uses these provisions in isolation as targets for the individual LOEs. For example, the Draft TMDL Staff Report states that the target for the benthic community effects LOE is either "reference" or "low" disturbance.[15] The Draft TMDL Staff Report also sets the target for the toxicity LOE as "nontoxic."[16] For the sediment chemistry LOE, the Draft TMDL improperly utilizes the "Effect Range Low" ("ERL") screening values, which are not even a part of the Bays

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10 Bays and Estuaries Plan at 9-11 (explaining the multiple lines of evidence approach and the integrated process involving each of the three lines of evidence).

11 Bays and Estuaries Plan at 3.

12 Id.

13 Id. at 20.

14 Indeed, Mr. Christopher Beegan, SWB's lead staff person and scientist for the Bays and Estuaries Plan testified to this very topic in an unrelated proceeding in October 2010. Specifically, Mr. Beegan testified in his deposition with respect to the importance of the step-wise approach in the Bays and Estuaries Plan and the risks involved in using individual LOEs apart from the integrated process. The full transcript of Mr. Beegan's deposition is being submitted with this comment letter.

15 Draft TMDL Staff Report at 47.

16 Id. at 49.

and Estuaries Plan, and uses these values in isolation to try to support sediment cleanup levels in the Harbor Waters.[17]

The Draft TMDL uses these targets and screening values to develop TMDL standards without completing the mandatory steps set forth in the Bays and Estuaries Plan, including the MLOE process, stressor identification, studies on the chemical linkage to impairment, identification of pollutant chemicals or classes of chemicals, source identification, and development of Sediment Management Guidelines ("SMGs") consistent with the course of action.[18]

The RWB's use of the ERL screening values is especially concerning because the ERL screening values were not developed through, or endorsed by, the Bays and Estuaries Plan process or any similar MLOE approach. Rather, the ERLs are screening values which are relevant only to the sediment chemistry LOE, as they do not predict sediment toxicity or benthic community effects. The study which developed the ERL screening values explicitly stated that the ERLs were not meant as indicators of toxicity or biologic effects.[19] This is especially true for mercury, nickel, total PCBs, total DDT, and DDE which were all identified in the ERL study as chemicals for which "there were relatively weak relationships between their concentrations and the incidence of effects."[20]

Two years after the ERLs first were published, government scientists from the United States Environmental Protection Agency ("EPA") and the National Oceanic and Atmospheric Administration ("NOAA") recognized the lack of utility for using the ERLs and the "effects range median" ("ERM") a higher, related screening value for purposes of setting sediment targets, stating:

> "The lack of even ER-L exceedance does mean that toxic effects are unlikely, but ER-M exceedances should only be taken to indicate that further analysis is in order. They should never be taken, by themselves, to mean that sediment is exerting a toxic

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17 Id. at 50-51.

18 See attached expert report of Drs. D. Frederick Bodishbaugh and Charles Menzie regarding the misuse of the Bays and Estuaries Plan in the Draft TMDL. This expert report was prepared as a technical comment on the Draft TMDL.

19 "The [ERL and ERM] numerical guidelines should be used as informal screening tools in environmental assessments. They are not intended to preclude the use of toxicity tests or other measures of biological effects." Long, E.R., MacDonald, D.D., Smith, S.L., and Calder, F.D. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments, Environmental Management 19(1): 81-97 at 95.

20 Id.

effect upon the environment or that there would be any benefit to decreasing its chemical content."[21]

Neither the ERLs nor the disturbance levels set out in the Bays and Estuaries Plan were intended to be directly used to develop TMDL standards or establish sediment cleanup levels, outside of the integrated process required by the Bays and Estuaries Plan. It is clear that the RWB's extraction of individual provisions of the Bays and Estuaries Plan in the Draft TMDL not only did not comply with the many requirements of the Bays and Estuaries Plan, including the MLOE and step-wise processes, but is also not related to the stated objectives of the Bays and Estuaries Plan to protect the benthic community and human health.[22]

3. Use of numeric thresholds separate from the fully integrated process risks a misallocation of resources, as demonstrated in the Draft TMDL.

Achieving compliance with the Draft TMDL would require implementation costs which RWB has estimated to exceed $900 million, including a dredging program with a cost estimated at nearly $700 million.[23] This estimate, however, is based on the low end of the sediment volume the Draft TMDL predicts will need to be dredged, and an unrealistically low value per cubic yard for the cost of remedial dredging. Applying the RWB's higher range for dredged material and a mid-range unit dredging cost, the actual cost of complying with the Draft TMDL could likely approach $7.0 billion.[24] Because the Draft TMDL has many flaws, some of which are pointed out in this comment letter, the cleanup program proposed by RWB is unwarranted, but this enormous estimated cost demonstrates the profound consequences which may occur if Regional Boards are allowed to improperly isolate numeric values out of studies and policies like the Bays and Estuaries Plan without completing the required integrated process.

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21 O'Connor, T.P., Daskalakis, K.D, Hayland, J.L., Paul, J.F., and Summers, J.K. 1998. Comparisons of sediment toxicity with predictions based on chemical guidelines. Environ. Toxico. Chem. 17:468-471 at 471 (emphasis added).

22 See attached expert report of Drs. John Slocomb and Paul Mehrle regarding the use of the ERL screening values by RWB in the Draft TMDL. This expert report was prepared as a technical comment on the Draft TMDL.

23 Draft TMDL Staff Report at 125.

24 The roughly $700 million estimate for the dredging program supplied by RWB grossly underestimates the cost per cubic yard ("cy") to be dredged, estimating it at $60.84 per cy, when $200 per cy is a more reasonable estimate for remedial dredging based on prior remedial dredging projects in California. The $700 million estimate also uses an estimated volume to be dredged which does not correspond to the sediment targets proposed in the Draft TMDL. The estimated volume which corresponds with the TMDL targets proposed by RWB is over three times larger than the estimate RWB used. When this larger volume estimate and the $200 per cy estimate are used, the cost calculated for the dredging program is $6.9 billion. See attached expert report by Dr. David Sunding regarding the economics of the Draft TMDL. This expert report was prepared as a technical comment on the Draft TMDL.

The potential cost of compliance with future TMDLs may also be increased if the numeric threshold values for DDT are lowered as proposed here. Regional Boards may take this opportunity to extract these lowered disturbance levels and use these values as cleanup standards or sediment targets in other TMDLs, which may only increase the cost of compliance with the TMDL. Any future sediment standards or targets set with these lowered DDT "disturbance" values may thus result in the potential expenditure of tremendous resources to bring sediments into compliance with a value that is not based on any available science.

4. SWB should reemphasize that the numeric values in the Bays and Estuaries Plan are not to be extracted and used for sediment targets or cleanup values.

SWB should reemphasize to the Regional Boards that numeric values, and the individual LOEs, are not to be extracted and used independently of the fully integrated Bays and Estuaries Plan process. Such statements are in the current Bays and Estuaries Plan, but as demonstrated by the RWB's Draft TMDL, Regional Boards may not appreciate the import of these statements.

To prevent further misuse of the Bays and Estuaries Plan in the future, SWB should consider adding additional clarification to the Bays and Estuaries Plan to reiterate to the Regional Boards that the Bays and Estuaries Plan is an integrated process from which individual LOEs are not to be separated and used in isolation, risking a great expenditure of resources with no commensurate environmental benefit.

Respectfully submitted,

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Paul Meyer

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American Council of Engineering Companies California

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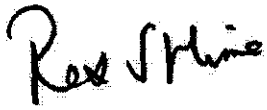

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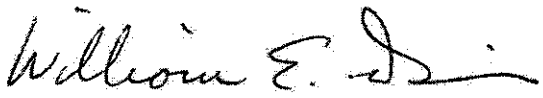

William Davis
Southern California Contractors Association

Attachments

STATE WATER RESOURCES CONTROL BOARD'S PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE - JANUARY 2011

ATTACHMENTS TO MARCH 15, 2011 LETTER SUBMITTED BY

American Council of Engineering Companies California
Building Industry Legal Defense Foundation
California Building Industry Association
California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Montrose Chemical Corporation of California
Southern California Contractors Association

Tab	Date	Description
References included in March 15, 2011 letter.		
1.	00/00/96	Chapman, P. M. (1996), A Test of sediment effects concentrations: DDT and PCB in the Southern California Bight. Environmental Toxicology and Chemistry, 15: 1197–1198
2.	02/17/11	Memorandum from Susan Kane-Driscoll, Ph.D., Exponent to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re Site-Specific Bioavailability Has Not Been Considered in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters
3.	09/30/09	Expert Report of Donald Roberts, Ph.D. from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
4.	09/00/09	Expert Report of Amir Attaran, Ph.D., LL.B from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
5.	10/00/09	Expert Report of Seymore Grufferman, M.D., Dr. P.H. from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
6.	n/a	Expert Report of Marion J. Fedoruk from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
7.	10/11/10	Digest and Transcript of Chris Beegan Deposition - In Re Tentative Cleanup and Abatement Order No. R9-2011-0001, Cal. Reg. Water Quality Control Bd., San Diego Region
8.	02/18/11	Memorandum from D. Frederick Bodishbaugh, Ph.D. and Charles Menzie, Ph.D., Exponent to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re Potential for Misuse of California Sediment Quality Objectives in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters
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11.	02/18/11	Memorandum from John Slocomb, Ph.D. and Paul Mehrle, Ph.D., Cardno Entrix to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re The Effects Range Low (ERL) Value For Numeric Target of Water Body-Pollutant Combinations in Marine Sediments of the Dominguez Channel Estuary and Greater Los Angeles and Long Beach Waters
12.	02/22/11	Letter from David Sunding, The Brattle Group to California Regional Water Quality Control Board, Los Angeles Region; United States Environmental Protection Agency, Region 9 Comments on the cost consideration of, "Dominguez Channel and Greater Los Angeles and Long Beach Harbor Water Toxic Pollutants Total Maximum Daily Loads Draft"

Tab	Date	Description
References included in attached expert reports.		
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14.	00/00/95	Alexander, M. (1995), How Toxic are Toxic Chemicals in Soil? Environ. Sci. Technol., 29: 2713–2717
15.	11/00/03	U.S. EPA (2003), Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH mixtures. EPA-600-R-02-013. Office of Research and Development, Washington, D.C.
16.	03/00/08	U.S. EPA (2008), Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Compendium of Tier 2 Values for Nonionic Organics. EPA-600-R-02-016. Office of Research and Development, Washington, D.C.
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22.	00/00/99	Middelburg, J.J., Nieuwenhuize, J. and Van Breugel, P. (1999), Black Carbon in Marine Sediments. Marine Chemical 65: 245-252
23.	00/00/07	Tomaszewski, J.E., Werner, D. and Luthy, R.G. (2007), Activated Carbon Amendment as a Treatment for Residual DDT in Sediment from a Superfund Site in San Francisco Bay, Richmond, California, USA. Environmental Toxicology & Chemistry 10: 2143-2150
24.	00/00/04	Rust, A.J., Burgess, R.M., McElroy, A.E., Cantwell, M.G. and Brownawell, B.J. (2004), Influence of Soot Carbon on the Bioaccumulation of Sediment-Bound Polycyclic Aromatic Hydrocarbons by Marine Benthic Invertebrates: An Interspecies Comparison. Environmental Toxicology & Chemistry 23: 2594–2603
25.	00/00/01	Bucheli, T.D. and Gustafsson, O. (2001), Ubiquitous Observations of Enhanced Solid Affinities for Aromatic Organochlorines in Field Situations: Are in Situ Dissolved Exposures Overestimated by Existing Partitioning Models? Environmental Toxicology & Chemistry 20: 1450-1456
26.	00/00/97	Maruya, K.A., Risebrough, R.W. and Horne, A.J. (1997), The Bioaccumulation of Polynuclear Aromatic Hydrocarbons by Benthic Invertebrates in an Intertidal Marsh. Environmental Toxicology & Chemistry 16: 1087-1097
27.	00/00/01	U.S. EPA, Office of Solid Waste and Emergency Response (2001), Eco Update. The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Risk Assessments. Publication 9345.-014. EPA 540/F-01/014
28.	00/00/10	Fuchsman P., Perruchon E., Bizzotto E., Dillard J. and Henning, M. (2010), An Evaluation of Cause-Effect Relationships Between DDT (and Metabolites) and Sediment Toxicity to Benthic Invertebrates. Presentation at the Society of Environmental Toxicology and Chemistry North America 31st Annual Meeting, Portland, OR, November 7-11, 2010

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30.	06/12/99	NOAA (1999), Sediment Quality Guidelines developed for the National Status and Trends Program
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35.	05/18/98	Giesy, J., Mehrle, P., Slocomb, J. and Suedel, B. (1998), Evaluation of Apparent Effects Threshold and Effects-Range-Median Approaches for Determining Sediment Quality Guidelines. Entrix
36.	09/00/96	Fairey, R., J. Hunt, C. Wilson, M. Stephenson, M. Pluckett and E. Long (1996), Chemistry, Toxicity and Benthic Community Conditions in Sediments of the San Diego Bay Region. Final Report, California State Water Resources Control Board
37.	04/00/97	MacDonald, D. (1997), Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments. Prepared for National Oceanic and Atmospheric Administration

Comments Submitted to:

State Water Resources Control Board
1001 I Street, 24th Floor
Sacramento, California 95814

COMMENTS ON STATE WATER RESOURCES CONTROL BOARD'S PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE - JANUARY 2011

ATTACHMENTS, VOLUME 1
(TAB 1 – TAB 7)

Submitted by:

LATHAM & WATKINS LLP

Counsel for:

Montrose Chemical Corporation of California

Also on Behalf of:

American Council of Engineering Companies California
Building Industry Legal Defense Foundation
California Building Industry Association
California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Southern California Contractors Association

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Short Communication

A TEST OF SEDIMENT EFFECTS CONCENTRATIONS: DDT AND PCB IN THE SOUTHERN CALIFORNIA BIGHT

PETER M. CHAPMAN
EVS Environment Consultants, 195 Pemberton Avenue, North Vancouver, BC V7P 2R4, Canada

(*Received* 14 *August* 1995; *Accepted* 2 *January* 1996)

Abstract—Independent efforts to determine concentrations of polychlorinated biphenyls (PCBs) and dichlorodiphenyltrichloroethanes (DDTs) along the Southern California Bight which result in adverse biological effects resulted in similar values. A correlative approach using historic data calculated the following likely sediment effects concentrations: total DDTs, 7.12 mg/dry kg (199 mg/kg organic carbon [OC]); and total PCBs, 0.592 mg/dry kg (30.4 mg/kg OC). Testing of field-collected sediments yielded the following no-observed-effect concentrations based on full life-cycle testing: total DDTs, 8.51 mg/dry kg (269 mg/kg OC); and total PCBs, 1.07 mg/dry kg (36.6 mg/kg OC).

Keywords—Sediment Toxicity tests DDT PCB

INTRODUCTION

As part of litigating damage claims for the injury or destruction of natural resources related to dichlorodiphenyltrichloroethanes (DDTs) and polychlorinated biphenyls (PCBs) deposited in the marine environment of southern California (the Southern California Bight Damage Assessment), a variety of independent studies were conducted. One of these studies involved determining the toxicity of spiked and field-collected sediments [1]. Another study involved using historic data to determine sediment effects concentrations [2]. When these studies were compared, the results of testing with field-collected sediments were determined to be consistent with predicted sediment effects values. This consistency is noteworthy and is reported herein.

METHODS

Sediment effects concentrations (SECs) for total DDTs (tDDT) and total PCBs (tPCB) in the Southern California Bight were determined to identify the concentrations of sediment-associated contaminants which are likely to cause, or to be associated with, adverse effects on sediment-dwelling organisms. The sum of *p,p′*-DDT, *o,p′*-DDT, *p,p′*-DDE (dichlorodiphenyldichloroethylene), *o,p′*-DDE, *p,p′*-DDD (dichlorodiphenyldichloroethane), and *o,p′*-DDD were defined as tDDT. All PCBs found in the Southern California Bight were defined as tPCB. Methodology involved a literature review with a weight-of-evidence approach to determine the ranges of chemical concentrations that were rarely, occasionally, or frequently associated with biological effects [3].

Full life-cycle (120-d) toxicity tests using the marine polychaete worm *Neanthes arenaceodentata* were conducted on Southern California Bight sediments contaminated with PCBs and DDTs. Full details of this testing, which is based on procedures adapted from Dillon et al. [4], will be the subject of a separate publication. Briefly, testing began with larvae and proceeded through to production of emergent juveniles. End points measured included survival, growth, fecundity, and reproduction.

Table 1. Predicted sediment effects concentration (SEC) compared to measured no-observed-effect concentration (NOEC)

Contaminant	SEC (mg/dry kg)	NOEC (mg/dry kg)	Ratio[a]	SEC (mg/kg OC)	NOEC (mg/kg OC)	Ratio[a]
tDDT	7.12	8.51	1.20x	199	269	1.35x
tPCB	0.592	1.07	1.81x	30.4	36.6	1.20x

tDDT = total dichlorodiphenyltrichloroethanes; tPCB = total polychlorinated biphenyls; OC = organic carbon.
[a] NOEC value divided by SEC value.

RESULTS AND DISCUSSION

The SECs and no-observed-effect concentrations (NOECs) for the two separate studies are provided and compared in Table 1. Effectively, though inadvertently, the measured polychaete NOECs serve to test the utility of the calculated SECs. Specifically, SECs were designed to provide a sediment value above which adverse effects would be expected to occur. The NOECs ranged from factors of 1.2 to 1.8 above the SECs, thus indicating that the SECs were appropriate.

Although neither method proves cause and effect, both are useful for determining screening level concentrations for individual contaminants. The utility of such concentrations does not include establishing rigorous sediment quality criteria but does include use as part of a burden-of-evidence approach to assessing the significance and extent of natural resource damage, a basis for identifying and delineating areas of concern, and a basis for identifying contaminants of concern.

Acknowledgement—I thank Don MacDonald for encouraging me to proceed with this publication. The manuscript was word processed by Vickie Duff.

REFERENCES

1. **EVS Consultants.** 1994. Southern California damage assessment surface water injury: Sediment. National Oceanic and Atmospheric Administration, Long Beach, CA.
2. **MacDonald, D.** 1994. Sediment injury in the Southern California Bight: Review of the toxic effects of DDTs and PCBs in sediments. National Oceanic and Atmospheric Administration, Long Beach, CA.

3. **Long, E.R., D.D. MacDonald, S.L. Smith** and **F.D. Calder.** 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. *Environ. Manage.* **19**:81–97.

4. **Dillon, T.M., D.W. Moore** and **A.B. Gibson.** 1993. Development of a chronic sublethal bioassay for evaluating contaminated sediments with the marine polychaete worm *Nereis* (*Neanthes*) *arenaceodentata*. *Environ. Toxicol. Chem.* **12**:589–605.

Exponent®

EXTERNAL MEMORANDUM

TO: California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9

FROM: Susan Kane Driscoll, Ph.D.

CC: Paul Singarella

DATE: February 17, 2011

SUBJECT: Site-Specific Bioavailability Has Not Been Considered in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters

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The TMDL report[1] presents a number of target levels for metals, PAHs, and bioaccumulative compounds such as PCBs and DDT. Bioavailability of chemicals is a critical aspect of understanding exposures and risks. However, the TMDL document does not consider this component of exposure. Instead, it presumes that chemicals are readily available. This assumption can yield target levels that are much lower than necessary for effective and wise water quality management. In this memorandum, we make the following points:

- Explicit consideration of bioavailability is a critical aspect of understanding exposures of biota to contaminants in water and sediments.
- The TMDL development is highly uncertain, because it does not factor bioavailability considerations into the development of target levels.

In this memorandum, we refer to Dominguez Channel and Greater Los Angeles and Long Beach Harbor waters as "The System."

Explicit Consideration of Bioavailability is Critical for Understanding Exposures

Since the early 1980s, when EPA began to attempt to establish sediment quality criteria, it became apparent that bioavailability of hydrophobic organic chemicals varies greatly among sediments. Whereas measured concentrations in water can generally be directly related to

[1] California Regional Water Quality Control Board, Los Angeles Region, and U.S. Environmental Protection Agency, Region 9. 2010. Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters Toxic Pollutants Total Maximum Daily Loads, Draft, December 2010.

adverse effects, measured concentrations of toxicants in sediment are often not at all directly related to expected adverse effects. This apparent difference between exposure and actual effect results from differences in bioavailability. EPA guidance acknowledges that toxicity may not be predicted accurately by simple bioaccumulation models, because properties of the sediments at the site may alter bioavailability (U.S. EPA 2003[2], 2008[3]).

Insights into the importance of considering the bioavailability of chemicals like DDT come from research on soils. For example, the effects of aging on the toxicity of DDT in soil were demonstrated in a study by Robertson and Alexander.[4] Mortality of insects exposed to soil spiked with DDT declined after aging the soil for 30 to 270 days. After 270 days, DDT-spiked soil was no longer toxic to one of three species tested, despite concentrations of DDT that were maintained at 84.7% of initial concentrations. In addition, Morrison et al. (2000[5]) documented extensive decline in bioavailability to earthworms as a result of aging DDT, DDE, DDD, in field soils. The authors of that study concluded that only 30%, 12%, 34%, and 20% of DDT, DDE, DDD, and total DDx were bioavailable in a soil treated in the field with DDT 49 years earlier. These studies highlight the importance of accounting for bioavailability when assessing exposure to residual compounds, the use of which ended decades ago. The presence of legacy compounds such as total DDT and PCBs in sediments means that they have been in the environment for a long time. This also means that the bioavailability of these chemicals is likely greatly reduced. The TMDL document makes no mention of this important time-dependent process.

The bioavailability of sediment-associated hydrophobic organic contaminants such as PCBs and DDT is a very important consideration in risk assessments and risk management. It is well established that the bioaccumulation of hydrophobic organic contaminants such as PCBs and DDT from sediment can be influenced by a variety of site-specific physical and chemical mechanisms, including adsorption, absorption, and ionic bonding, that act to reduce the concentration of toxic chemicals in sediment that is freely dissolved in sediment interstitial waters and is bioavailable (Di Toro et al. 1991[6]; U.S. EPA 2003[7], 2008[8]). Metals are also

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2 U.S. Environmental Protection Agency. 2003. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: PAH mixtures. EPA-600-R-02-013. Office of Research and Development, Washington, DC.

3 U.S. Environmental Protection Agency. 2008. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Compendium of tier 2 values for nonionic organics. EPA-600-R-02-016. Office of Research and Development. Washington, DC.

4 Alexander, M. 1995. How toxic are toxic chemicals in soil? Environ. Sci. Technol. 29:2713–2717.

5 Morrison DE, Boakaik R, Alexander M. 2000. Bioavailability to earthworms of aged DDT, DDE, DDD, and dieldrin in soil. Environ Sci Technol 34:709–713.

6 DiToro DM, Zarba CS, Hansen DJ, et al. 1991. Technical basis for establishing sediment quality criteria for nonionic organic chemicals using equilibrium partitioning. Environ Toxicol Chem 10:1541–1583.

influenced by physical and chemical mechanisms that must be considered in estimating exposure and associated risk (U.S. EPA 1999).[9] Bioavailability can also vary depending on how the chemicals are introduced into the system and in which environmental media they reside. For example, the bioavailability of chemicals that enter The System from atmospheric deposition is very different from that which enters from runoff of particulates. U.S. EPA recommends that bioavailability of hydrophobic organic contaminants be determined by estimating or measuring the freely dissolved chemical concentration in sediment interstitial water (U.S. EPA 2003).[10] This approach does not suggest that exposure to aquatic organisms is only from interstitial water, but rather that the interstitial water concentration is most closely correlated with the fraction of the total contaminant that is free to partition among all phases.

Approaches used to assess bioavailability typically assume that the contaminant is distributed into multiple phases: freely dissolved or associated with dissolved organic carbon in interstitial water, with natural sedimentary organic carbon, or with black carbon (discussed below). Recent research has made measurement of the bioavailable concentration feasible. The bioavailable concentrations can be determined in various ways: (1) estimated using a two-carbon model that takes into account the association of contaminants with black carbon, (2) extracted directly from interstitial waters, (3) estimated by passive sampling of whole sediments, and (4) estimated by passive sampling of interstitial waters (Hawthorne et al. 2007[11]; Kane Driscoll et al. 2009[12]; McArdle et al. 2010[13]). Standard methods for measuring bioavailable concentrations of certain hydrophobic organic compounds have been established (ASTM 2010[14]), and methods for other

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7 U.S. Environmental Protection Agency. 2003. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: PAH mixtures. EPA-600-R-02-013. Office of Research and Development, Washington, DC.

8 U.S. Environmental Protection Agency. 2008. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Compendium of tier 2 values for nonionic organics. EPA-600-R-02-016. Office of Research and Development. Washington, DC.

9 U.S. Environmental Protection Agency. 1999. Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Metal mixtures (cadmium, copper, lead, nickel, silver, and zinc). EPA-600-R-02-011. Office of Research and Development, Washington, DC.

10 U.S. Environmental Protection Agency. 2003. Op cit.

11 Hawthorne SB, Azzolina, NA, Neuhauser, EF, Kreitinger, JP. 2007b. Predicting bioavailablity of sediment polycyclic aromatic hydrocarbons to Hyalella azteca using equilibrium partitioning, supercritical fluid extraction, and pore water concentrations. Environ Sci Technol 41:6297–6304.

12 Kane Driscoll SB, Amos BC, McArdle ME, Menzie CA, Coleman A. 2009. Predicting sediment toxicity at former manufactured gas plants using equilibrium partitioning benchmarks for PAH mixtures. Soil Sed Contam 18(3):307–319.

13 McArdle ME, Kane Driscoll SB, Booth PN. 2010. An ecological risk-based cleanup strategy for contaminated sediments in a freshwater brook. Int J Soil Sed Water 3(2):1–24.

14 American Society for Testing and Materials (ASTM). 2007b. Standard test method for determination of parent and alkyl polycyclic aromatics in sediment pore water using solid-phase microextraction and gas. Available online at http://www.astm.org/Standards/D7363.htm.

hydrophobic organic compounds are in widespread use. Failure to incorporate approaches that are available to determine the bioavailability of sediment-associated hydrophobic organic compounds in the development of the TMDL are not consistent with current practice and contribute to the uncertainty and unreliability of the TMDL.

One parameter that is likely very important in The System is the presence of black carbon in sediments. Black carbon particles, such as soot and charcoal, are products of incomplete combustion that are ubiquitous in nearshore sediments, especially in areas of urban runoff, and can make up as much as 10%–30% of the total carbon in sediments of urban harbors (Middelburg et al. 1999).[15] This soot-like material will strongly bind DDT, PCBs, and other hydrophobic chemicals (Tomaszewski et al. 2007),[16] PCBs, and other hydrophobic chemicals, and reduces bioaccumulation of these chemicals by aquatic organisms (Rust et al. 2004).[17] Black carbon in field-collected sediments has been shown to reduce the bioavailable concentration of non-planar DDT/DDD by factors of 4 to 8, and planar DDE by factors as high as 50 to 250 (Bucheli and Gustafsson 2001). [18] Thus, black carbon reduces partitioning and bioavailability of DDT and PCBs. The reduction in bioavailability reduces the toxicity of these chemicals to benthic invertebrates and the potential for bioaccumulation into the food chain. The result is that the chemicals can be present in sediments at much higher levels without causing potentially harmful exposures.

The influence of black carbon on the bioavailability of hydrophobic chemicals has been shown to vary seasonally and spatially in an intertidal marsh in San Francisco Bay (Maruya et al. 1997).[19] The study demonstrated that bioaccumulation of hydrophobic organic chemicals by aquatic invertebrates was related to the concentration of soot particles, which increased during the local rainy season in areas affected by surface runoff. Therefore, the failure of the TMDL approach to account for seasonal and spatial variability in bioavailability of hydrophobic chemicals such as DDT and PCBs contributes greatly to the uncertainty of the method used to establish target sediment concentrations. The authors concluded that hydrophobic contaminants in the San Francisco Bay, and in general, may be approximately ten times less bioavailable than

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15 Middelburg JJ, Nieuwenhuize J, Van Breugel P. 1999. Black carbon in marine sediments. Mar Chem 65:245–252.

16 Tomaszewski JE, Werner D, Luthy RG. 2007. Activated carbon amendment as a treatment for residual DDT in sediment from a Superfund site in San Francisco Bay, Richmond, California, USA. Environ Toxicol Chem 10:2143–2150.

17 Rust AJ, Burgess RM, McElroy AE, Cantwell MG, Brownawell BJ. 2004. Influence of soot carbon on the bioaccumulation of sediment-bound polycyclic aromatic hydrocarbons by marine benthic invertebrates: An interspecies comparison. Environ Toxicol Chem 23:2594–2603.

18 Bucheli TD, Gustafsson O. 2001. Ubiquitous observations of enhanced solid affinities for aromatic organochlorines in field situations: Are in situ dissolved exposures overestimated by existing partitioning models? Environ Toxicol Chem 20:1450–1456.

19 Maruya KA, Risebrough RW, Horne AJ. 1997. The bioaccumulation of polynuclear aromatic hydrocarbons by benthic invertebrates in an intertidal marsh. Environ Toxicol Chem 16:1087–1097.

expected, and that differences in bioavailability must be accounted for to improve the reliability of theoretical predictions.

The TMDL Process is Highly Uncertain, Because it Does Not Consider Bioavailability

The TMDL process for The System is highly uncertain, because it does not take into account bioavailability. The process relies on screening levels for sediments. An example is the 1.58-µg/kg target concentration for DDT in sediments. Screening levels such as the 1.58µg/kg value are typically used for *screening*, because there is confidence that no effect occurs at that concentration. EPA guidance described the types of conclusions that can be derived from screening-level assessments (U.S. EPA 2001).[20] In general, screening levels:

- "provide a general indication of the potential for ecological risk (or lack thereof)."
- "are conservative assessments in that they provide a high level of confidence in determining a low probability of adverse risk, and they incorporate uncertainty in a precautionary manner."
- "are not designed nor intended to provide definitive estimates of actual risk, generate cleanup goals and, in general, are not based on site-specific assumptions."
- "Upon completion of a conservative screen, if no materials (contaminants) are retained by the screen, one can confidently state that there is a minimal potential for ecological risk to exist. Alternatively, if materials (contaminants) are retained by the screen, one cannot conclude that an ecological risk "actually" exists; the characteristics of the material retained by the screen are unknown, other than its size is above some specified minimum values."

Thus, the screening level does not inform the assessment regarding the concentrations at which effects might occur. Those concentrations are governed by other factors, including bioavailability. By ignoring site-specific conditions, including those involving bioavailability, the TMDL process has derived target concentrations that may be off by orders of magnitude (e.g., by a factor of 10, 100, or even 1000). For example, the influence of organic carbon on bioavailability and toxicity of DDT and metabolites was demonstrated in a recent review of

[20] U.S. EPA. 2001. Eco Update. The role of screening-level risk assessments and refining contaminants of concern in baseline ecological risk assessments. Publication 9345.-014. EPA 540/F-01/014. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.

historical data on the toxicity of sediment-associated DDT. Fuchsman et al. (2010)[21] considered multiple lines of evidence, including data from numerous laboratory and field studies, and demonstrated that an appropriate cause-effect screening value for total DDT is on the order of 50–100 mg/kg-organic carbon, which is equivalent to 2.5–10.0 mg DDT/kg (for sediment with 5% organic carbon).

The values determined by Fuchsman et al. (2010) are similar to independently derived values reported by Chapman (1996) for the Southern California Bight. Chapman (1996)[22] summarizes the results of two independent studies, one of which (EVS 1994)[23] reported a no-effect concentration (8.51 mg/kg or 269 mg/kg-organic carbon), and another (MacDonald 1994)[24] of which reported a sediment effects concentration (7.12 mg/kg or 199 mg/kg-organic carbon) for total DDT. The sediment effects concentration is intended to identify the concentration above which adverse effects to benthic-dwelling organisms would be expected to occur. The SEC is based on results of spiked sediment bioassay and field data. Field data were collected from four areas: seven sample locations in the vicinity of the Los Angeles County Sanitation District sewage outfalls on the Palos Verdes Shelf, four locations on the Palos Verdes shelf and Santa Monica Bay, seven locations in the vicinity of Palos Verdes, three locations in San Diego Harbor, and 12 locations in the vicinity of Palos Verdes Peninsula. Chapman (1996) concludes that the similarity of these independently derived threshold values supports their usefulness as screening values.

The comparison of the TMDL DDT sediment target 0.00158 mg/kg (1.58 μg/kg) to the values in the range of 2.5 to 10.0 mg/kg reported by Chapman (1996) and Fuchsman et al. (2010) indicates three orders of magnitude (1000×) uncertainty in the use of the screening level to represent a DDT direct-effects level for The System. This large uncertainty reflects the fact that bioavailability has not been considered in the TMDL process. Given this level of uncertainty and lack of any explicit consideration of uncertainty, the use of screening levels, such as that for total DDT, renders this aspect of the TMDL development process unreliable for management purposes. It is simply a screening-level analysis.

We have provided one example of the significance of not considering bioavailability. All the chemicals considered in the TMDL document are subject to the effects of bioavailability when

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[21] Fuchsman P, Perruchon E, Bizzotto E, Dillard J, Henning M. 2010. An evaluation of cause-effect relationships between DDT (and metabolites) and sediment toxicity to benthic invertebrates. Presentation at the Society of Environmental Toxicology and Chemistry North America 31st Annual Meeting, Portland, OR, November 7-11, 2010.

[22] Chapman PM. 1996. A test of sediment effects concentrations: DDT and PCB in the Southern California Bight. Environ Toxicol Chem 15:1197–1198.

[23] EVS Consultants**.** 1994. Southern California damage assessment surface water injury: Sediment. National Oceanic and Atmospheric Administration, Long Beach, CA.

[24] MacDonald, D. 1994. Sediment injury in the Southern California Bight: Review of the toxic effects of DDTs and PCBs in sediments. National Oceanic and Atmospheric Administration, Long Beach, CA.

the goal is to derive meaningful target concentrations. In addition, the bioavailability of these chemicals varies among loading sources. Therefore, the fact that the TMDL document does not consider bioavailability for any contaminant or in relation to any source indicates that the process is highly uncertain, and the resultant TMDLs are not reliable and are not wise or effective management tools for The System.

Professional Profile: Susan B. Kane Driscoll

- Ph.D., Environmental Sciences, University of Massachusetts, 1994
- B.S., Natural Resources, University of Rhode Island, 1981

Dr. Driscoll is a Managing Scientist in Exponent's EcoSciences practice. She is an aquatic toxicologist, with 22 years experience in toxicology, specializing in ecological risk assessment, environmental chemistry, sediment toxicity testing, and the toxicity and bioavailability of sediment-associated contaminants to aquatic organisms and wildlife. Dr. Driscoll has directed or participated in numerous ecological risk assessments for RCRA, Superfund, and hazardous waste sites, serving a variety of industrial, utility, and governmental clients. She has extensive experience in designing and conducting laboratory and field aquatic toxicity and environmental fate studies in accordance with rigorous quality assurance practices. She has designed and contributed to numerous environmental programs that were used to develop technically defensible solutions to environmental problems and has negotiated their acceptance with state and federal authorities. Dr. Driscoll is a specialist in the field of sediment toxicology, and her original research and publications in the areas of bioavailability and toxicity of sediment-associated contaminants are widely cited. She has extensive knowledge of sediment toxicity testing, the technical basis and predictive ability of various sediment quality benchmarks, and has served as a reviewer for the development of emerging benchmarks.

Report of

Donald R. Roberts, Ph.D.

Garza v. Allied Chemical Corp., et al.
Cause No. C-4885-99-F(10)
District Court, Hidalgo County, Texas

September 30, 2009

Table of Contents

A. Introduction

I, Donald R. Roberts, Ph.D., was retained by Montrose Chemical Corporation of California in regard to *Guadalupe Garza. v. Allied Chemical Corp., et al.*, Cause No. C-4885-99-F(10), District Court, Hidalgo County, Texas.

Much of my professional life for the past three decades has been dedicated to studying DDT and its contributions to improved health and welfare of human beings, as well as evaluating the many accusations made against DDT. Based on my thirty-plus years of professional work on DDT, my education, scientific research, literature research, and experience, I conclude and can document the following to a reasonable degree of scientific certainty:

1. The discovery of DDT's insecticidal properties revolutionized the capacity of the global public health community to control major human diseases, such as malaria, typhus, and other diseases transmitted by insects. The public health impact was so great and so immediate that Dr. Mueller was awarded the Nobel Prize in Physiology or Medicine for his discovery of DDT's insecticidal properties.

2. The United States military used DDT extensively during World War II to prevent typhus in military forces and civilian refugee populations. Millions of lives were saved.

3. In the United States, federal, state, and local governments began spraying DDT inside homes to control malaria in the mid-1940s. During the next few years, DDT was used in large scale human health campaigns in 13 states, ultimately helping to quickly eradicate malaria from the United States. The large scale public health use of DDT also helped control murine typhus and other diseases through the 1960s.

4. In the 1940s and 50s, there was extensive public health use of DDT in Texas, including Hidalgo County and Mission, Texas. Public health use of DDT in Hidalgo County included spraying inner walls of houses and dusting DDT inside and outside of commercial buildings and houses to control insect-borne diseases. Even in the 1960s, DDT was sprayed in Hidalgo County as part of the national *Aedes aegypti* Eradication Program.

5. DDT also was used internationally in major public health campaigns to control malaria and other insect-borne diseases like dengue, yellow fever, and others. DDT's ability to prevent disease transmission and, in some cases, eradicate the disease vectors led a National Academy of Sciences committee to conclude: "To only a few chemicals does man owe as great a debt as to DDT...In little more than two decades, DDT has prevented 500 million human deaths, due to malaria, that otherwise would have been inevitable."[1]

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[1] Herzog, A. An environmental failure: Restrictions on DDT. April 22, 2008. Townhall.com: http://www.townhall.com/Columnists/AshleyHerzog/2008/04/22/an_environmental_failure_restrictions_on_ddt

6. Throughout the 1950s, 1960s and early-1970s, DDT was heralded as a lifesaver and credited with saving millions of lives.

7. The period 1971-1973 marked the end of large scale DDT use in the United States. In June, 1972, the newly-formed EPA ruled against the continued use of DDT for agricultural uses. Preceding this EPA ruling, Administrative Law Judge Edmund Sweeney held 11 months of DDT hearings in 1971-72 and ruled not to de-list DDT for use in agriculture or public health. Two months later, and with no new data or evidence against DDT, EPA Administrator William Ruckelshaus overturned Judge Sweeney's ruling and de-listed the use of DDT for agricultural purposes.

8. Unsupported claims that DDT is harmful to human health have limited the effectiveness of disease control programs, allowing deadly diseases to re-emerge.

9. Even after DDT was de-listed for use in 1972, government officials and other users continued to obtain permission to employ DDT for certain public health, agriculture, forestry, and pharmaceutical uses in high-risk locations. In addition, DDT continued to be present in pesticide formulations and used widely in crops during the 1970s and 1980s, in the miticide Dicofol, also known as Kelthane.[2]

10. Today, the World Health Organization (WHO) and other U.N. bilateral and multilateral organizations have concluded that DDT is safe for humans and safely can be sprayed on house walls for disease control.

11. DDT is unequaled in its ability to control disease. It exhibits a complex of chemical actions to prevent disease transmission inside homes in three primary ways: (1) it is a powerful spatial repellent that stops mosquitoes from entering houses; (2) it is a strong contact irritant that causes mosquitoes to prematurely exit sprayed houses, often without biting; and (3) it kills mosquitoes that remain in prolonged physical contact with sprayed surfaces. DDT is the only insecticide presently recommended for spraying on house walls that actually stops a large proportion of mosquitoes from entering the house and transmitting diseases while residents are sleeping.

12. In the 1950s and 1960s, there were no adequate substitutes for major uses of DDT, such as cotton and public health. This was true even in 1972 when EPA de-listed all agricultural uses of DDT in the United States. Even today, there is no equally effective substitute for DDT in public health programs.

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2 The New York Times..March 20, 1984. Around The Nation: E.P.A. considers ban on pesticide using DDT: http://query.nytimes.com/gst/fullpage.html?res=9807E0DA1039F933A15750C0A962948260

B. Professional Background and Experience

I hold a doctoral degree in medical zoology from the University of Texas School of Public Health in Houston, Texas. For 21 years, I was an Army officer. I retired as a Lieutenant Colonel from the Army Medical Service Corps in 1987. During my career, I earned the Legion of Merit with Oak Leaf Cluster, the Meritorious Service Medal with Oak Leaf Cluster, two Army Commendation Medals, and the Army Surgeon General's "A" designation for professional achievement. I led a team of American scientists that, along with Brazilian colleagues, discovered a biting midge was responsible for transmitting an important viral disease (Oropouche virus) to humans in the Amazon Basin of Brazil. After retiring from active duty, I served as a professor of tropical public health in this nation's only Department of Defense medical school, the Uniformed Services University of the Health Sciences in Bethesda, Maryland.

I retired from academic life on June 30, 2007. The Board of Regents of the Uniformed Services University of the Health Sciences awarded me the honorary title of Professor Emeritus in May 2007.

In September 2007, I was awarded the 2007 Frank Brown Berry Prize in federal healthcare. The prize is awarded for "...medical professionals who labor hard but reap little financial gain from their achievements, as they strive to make contributions in federal medicine."

Most of my professional life has been dedicated to the study of DDT and its use in disease control. I have worked with many different formulations, worked in many different houses sprayed with DDT, worked for prolonged periods of time among populations who lived continuously in DDT-sprayed houses, and I have slept and worked in such houses. I have observed spray operators spraying houses and have had malaria control operators spray experimental houses for purposes of my field research activities. My experiences include studies on effects of DDT on bees and mosquitoes. I have published over 100 peer-reviewed publications. Until my retirement, I served as reviewer of professional papers for several scientific journals, including the American Journal of Tropical Medicine and Hygiene.

The focal point of my research and work over the last thirty years has been DDT, including DDT's (1) history, (2) chemical actions, (3) role in control of human diseases, (4) influence on wildlife and the environment, and (5) action on mosquito behavior. I published a peer-reviewed probability model on DDT as a spatial repellent, a contact irritant and an agent to kill mosquitoes.[3] The probability model was the basis for a National Institutes of Health grant to test chemicals that might be used to replace DDT in malaria control programs.[4] I was the principal investigator for that grant until I retired in 2007.

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[3] Roberts, D., W. Alecrim, et al. (2000). " A probability model of vector behavior: Effects of DDT repellency, irritancy, and toxicity in malaria control." Journal of Vector Ecology 25: 48-61.

[4] "Behavior-modifying compounds for disease vector control," NIH Partnership Grant, Roberts (PI).

I have provided testimony to Congressional committees and sub-committees on the subject of DDT's important role in public health, as follows:

- Testimony before the Senate Subcommittee on East Asian and Pacific Affairs on Neglected Diseases in East Asia: "Are Public Health Programs Working?," Washington, D.C., October 6, 2004.

- Testimony before the Senate Environment and Public Works Committee on the Misuse of Science in Environmental Policy Making: "Misrepresentations of Science During Decades of Environmental Campaigning against DDT," Washington, D.C., September 28, 2005.

- Testimony before the Senate Subcommittee on Federal Financial Management, Government Information, and International Security: "Bilateral Malaria Assistance: Progress and Prognosis," Washington, D.C., January 19, 2006.

- Testimony before the Senate Committee on Environment and Public Works: "Examining the Human Health Impacts of Global Warming," Washington, D.C., October 27, 2007.

I have been quoted and interviewed on the topic of DDT by print and broadcast news media, including Voice of America, British Broadcasting Corporation, Readers Digest, The Economist, The New York Times, The Washington Post, The Washington Times, and Public Broadcasting Service.

I am on the review board for the Integrated Vector Control Consortium. The Bill and Melinda Gates Foundation fund this program to produce new tools for disease control. The program is headquartered at the Liverpool School of Tropical Medicine and the London School of Tropical Medicine in the United Kingdom.

In December 2006 and again in Spring 2007, I served as a temporary adviser to World Health Organization ("WHO"). In that capacity, I chaired the Technical Advisory Group for Indoor Residual Spraying for Malaria Control. I also participated as a member of the Technical Research Advisory Committee of WHO. A copy of my curriculum vitae is attached.

In summary, my knowledge as to DDT comes from a lifetime of research, education, and work regarding DDT. My conclusions about DDT were reached and published long before Montrose contacted me about this litigation.

C. Opinions and Analysis

1. The discovery of DDT's insecticidal properties revolutionized the capacity of the global public health community to control major human diseases, such as malaria, typhus, and other diseases transmitted by insects. The public health impact was so great and so immediate that Dr. Mueller was awarded the Nobel Prize in Physiology or Medicine for his discovery of DDT's insecticidal properties.

DDT was first created in 1874 by a German graduate student, Othmar Zeidler. In the 1930s, Dr. Paul Mueller re-discovered DDT and also discovered its insecticidal properties.

Dr. Mueller was an employee of J. R. Geigy, a Swiss chemical company. The company was interested in finding a chemical that would control clothes moths, and Mueller worked on the clothes moth project. He found that flies died when he exposed them to DDT.[5]

The Swiss were using DDT to control beetles while others in Europe were fighting World War II. Historically, insect-borne diseases flourished during wars. Typhus is a leading example of such a disease. Body lice spread the disease and they thrive in crowded and filthy conditions of war. By 1942, the Swiss had discovered that DDT was effective against many insects. They also found that it killed fleas, mites, lice, mosquitoes and flies.[6] The Allies found that DDT was toxic to lice. Furthermore, it remained active against lice for many days after application, depending on personal hygiene. The Americans and the British conducted their own toxicity tests and confirmed the Swiss reports that DDT was safe for humans.[7]

Discovery of DDT gave the Allies a tool for preventing deadly outbreaks of typhus.[8] By early 1943, the U.S. military was working to greatly increase DDT production and was projecting even larger DDT requirements for 1944.[9] Thus, by late 1943, Geigy's subsidiary in the U.S., the Cincinnati Chemical Works, was producing DDT. By early 1944, fourteen other American chemical companies also were producing DDT, as were British corporations.[10] In a

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5 Zimmerman, O.T. and Lavine, I., "DDT Killer of Killers." Industrial Research Center, Dover, New Hampshire (1946), Pages 31–32.

6 Zimmerman, O.T. and Lavine, I., "DDT Killer of Killers." Industrial Research Center, Dover, New Hampshire (1946), Pages 31–32.

7 West. T.F. and Campbell, G.A., "DDT and Newer Persistent Insecticides," New York: Chemical Publishing Co., Inc. (1952), Page 2.

8 Spielman, and D'Antonio, "Mosquito: A Natural History of Our Most Persistent & Deadly Foe." Sequitur Books (2001), Page 143.

9 Hirschy, I.D., Memo for Director, Pre. Med. Div., "DDT Insecticide." Aug. 11, 1943.

10 Zimmerman, O.T. and Lavine, I., "DDT Killer of Killers." Industrial Research Center, Dover, New Hampshire (1946), Page 39; West. T.F. and Campbell, G.A., "DDT and Newer Persistent Insecticides," New York: Chemical Publishing Co., Inc. (1952), Page 7.

reflection of the life-saving properties of DDT as an insecticide, Mueller ultimately won the Nobel Prize in Physiology or Medicine for his discovery in 1948.

2. The United States military used DDT extensively during World War II to prevent typhus in military forces and civilian refugee populations. Millions of lives were saved.

As early as WWI, the cycle of typhus transmission was known and delousing was performed in an attempt to get rid of lice and prevent typhus transmission. However, the chemicals of that time were not adequate. Under the squalid conditions of war, the disease still struck with spectacular ferocity. Thus, typhus killed more soldiers and civilians in WWI than all the shell-fire combined.[11] Human history tells many grim stories of wars and social instabilities that allowed typhus and other diseases to sweep through and lay waste to both military and civilian populations. The advent of DDT in 1943 forever changed the role of typhus in warfare.

The discovery and use of DDT during WWII contributed to an Allied victory. Allied Forces used DDT to protect deployed fighters and war refugees from typhus and other insect-borne diseases. DDT gained fame in 1943 by successfully stopping an epidemic of typhus in Naples, Italy, an unprecedented achievement.[12] The achievement was particularly remarkable because the Swiss had only released the formula for DDT in 1942. From WWII onward, DDT was in common use as a powder for spraying or sprinkling into clothing of soldiers and refugees for typhus prevention. Almost overnight, DDT went from being unknown to being a critical military necessity.

For many years after WWII, travelers commonly were provided a small container of DDT powder for sprinkling into underclothes to control body lice, other arthropods, and disease transmission. It became a standard issue for State Department officials.[13] In spite of extensive use over many years, there were no documented cases of harm from WWII soldiers or war refugees who were doused with DDT to prevent typhus or from officials who, in later years, also used DDT in their underclothes to prevent typhus. Critical uses of DDT during and immediately after the war set the stage for DDT to become a peacetime commodity.[14]

3. In the United States, federal, state, and local governments began spraying DDT inside homes to control malaria in the mid-1940s. During the next few years, DDT was used in large scale human health campaigns in 13 states, ultimately helping to quickly eradicate malaria from the United States. The large scale public

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11 http://entomology.montana.edu/historybug/WWI/TEF.htm; http://www.epicdisasters.com/index.php/site/comments/the_worst_outbreaks_of_disease/

12 http://homepage.mac.com/msb/163x/faqs/typhus.html

13 I have a small bottle of DDT powder, dated 1959, issued by the U.S. State Department with instructions for sprinkling in underclothing.

14 War time, domestic, and public health uses of DDT internationally, nationally, and in the state of Texas have been photographically documented.

health use of DDT also helped control murine typhus and other diseases through the 1960s.

In addition to extensive use of DDT for protecting deployed war fighters and travelers abroad, DDT also became an indispensable commodity for protecting the health and life of people in the United States. At the turn of the 20th Century, the continental United States was intensely malarious. The disease was an immense cause of human death, human illness, and economic loss. In 1916, malaria was estimated to reduce economic productivity in the U.S. by $100,000,000 (in 1916).[15] The economic loss from malaria in the state of California alone was estimated to be $3,000,000, and California was not even considered a highly malarious state.[16] A conservative estimate of malaria cases for the country as a whole was 1,000,000 or more cases per year.[17]

Malaria rates declined gradually in the United States through the first half of the 20th Century, with occasional increases as a result of war and other events. Regardless, by the 1940s, infectious diseases were still major public health problems throughout the country. Malaria remained a major public health problem in southeastern states. By the early 1940s, the ability of the United States to exert effective control over malaria was still limited in spite of growing wealth and improving standards of living. In fact, control was possible only in urban settings where draining and eliminating aquatic habitats for mosquitoes and using larvicide to kill mosquito larvae was cost-effective. In contrast, the only real progress in poor rural areas was to screen houses to prevent mosquitoes from entering and transmitting disease.[18]

The office of Malaria Control in War Areas (MCWA) was created right after the bombing of Pearl Harbor in 1942 "to prevent or reduce malaria transmission around Army, Navy, and essential war industry areas [in the United States] by extending the control operations carried on by military authorities within these reservations."[19] Spraying houses with DDT quickly became established within the program as the most effective method of stopping malaria transmission in and around military installations in the U.S.

Then, beginning in 1945, the MCWA launched its Extended Program of Malaria Control. The extended program was not limited to military posts, camps, and stations, but instead was

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15 Hoffman, F.L., A Plea and a Plan for the Eradication of Malaria Throughout the Western Hemisphere. Prudential Press, Newark, N.J., 1917.

16 Hoffman, F.L., A Plea and a Plan for the Eradication of Malaria Throughout the Western Hemisphere. Prudential Press, Newark, N.J., 1917.

17 Hoffman, F.L., A Plea and a Plan for the Eradication of Malaria Throughout the Western Hemisphere. Prudential Press, Newark, N.J., 1917.

18 U.S. Public Health Service, 1944-45. Malaria Control in War Areas. Federal Security Agency. Page 14.

19 CDC Bulletin. Jul., Aug., Sept., 1946. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 1.

extended to more malarious civilian areas.[20] It consisted of spraying DDT on interior walls of homes and privies. The spraying program covered large areas of the southern U.S., including Texas. From January 1945 to September 1947, 3.2 million houses were sprayed.[21] This sum apparently did not include numbers of houses sprayed through use of local funds. As described in a 1946 report, "a number of larger cities have contributed sufficient funds to spray the cities in the 2,500-10,000 population group. The entire cost of this type of residual house spraying is paid from local funds."[22] After 1945, investigators discovered they could increase performance and get longer protection by spraying a greater concentration of DDT on house walls. Thus, as a result of a change in formulation, the number of sprayings per house varied from nearly two in 1945 to fewer than 1.5 in 1947.[23]

The MCWA was a war time organization and changes were needed when the war ended in 1945. With demobilization of military forces in 1945 and 1946, the MCWA went into a phase of rapid liquidation.[24] Liquidation of the MCWA led to creation of the Communicable Disease Center (CDC) of the United States Public Health Service on July 1, 1946.[25] The CDC was created to capture the unique resources and expertise of MCWA. The CDC was tasked to continue and expand MCWA programs in all areas of public health in the United States.[26]

The MCWA's Extended Program of Malaria Control already had been successful in reducing numbers of malaria cases in areas where houses were sprayed (Figure 1).

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20 CDC Bulletin. Jan., Feb., Mar. 1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 2-3.

21 CDC Bulletin. Oct., Nov., Dec. 1948. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 10.

22 CDC Bulletin. Oct., Nov., Dec., 1946. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 19.

23 CDC Bulletin. Oct., Nov., Dec. 1948. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 10.

24 CDC Bulletin. Jul., Aug., Sept. 1946. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 4.

25 CDC Bulletin. Oct., Nov., Dec. 1946. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 1.

26 CDC Bulletin. Oct., Nov., Dec. 1946. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 1.

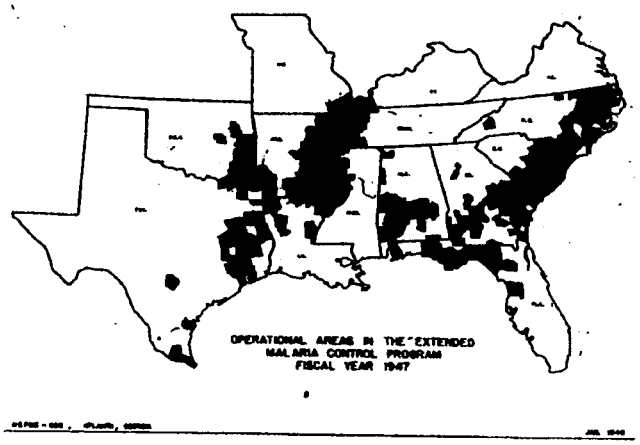

Figure 1. 1946-47 map of US counties where houses were sprayed with DDT for control of malaria.[27]

To build on early success, the CDC started a National Malaria Eradication Program, commencing operations on July 1, 1947. It was referred to as the Residual Spray Program. Spraying DDT on inner walls of rural homes in malaria-endemic counties was the key component of that program. From July 1947 to the end of 1949, the program sprayed over 4,650,000 houses. Based on surveys conducted in thirteen southeastern states, the CDC concluded that "over-all control (reduction in houses infested) was approximately 90 percent for the 5-year period [1945-1949]."[28]

Overall, beginning in 1945,[29] millions of houses were sprayed for malaria control throughout the southern U.S. DDT spraying broke the cycle of malaria transmission and, in 1949, the United States was declared free of malaria as a significant public health problem.[30]

However, spraying of houses did not end in 1949. DDT was used in many other public health endeavors:

a) Typhus - Control of murine typhus was one such endeavor. Murine typhus was an important public health problem in southern states. Control of typhus was achieved by dusting rat burrows and runways with DDT to kill fleas (oriental rat fleas) that transmit the disease (see

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27 CDC Activities, 1946-1947. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 7.

28 CDC Bulletin. Jan. 1950. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 11. Surveys were conducted in thirteen southeastern states and approximately sixty-five thousand houses were inspected. Evaluations of effectiveness were based on inspections of randomly selected sprayed and unsprayed houses for presence or absence of the malaria mosquito.

29 U.S. Public Health Service, 1944-45. Malaria Control in War Areas. Federal Security Agency. Page 18.

29 CDC. "The History of Malaria, an Ancient Disease"; http://www.cdc.gov/malaria/history/

Figures 2 & 3).[31] Over time, hundreds of thousands of premises were dusted with DDT for control of this disease.

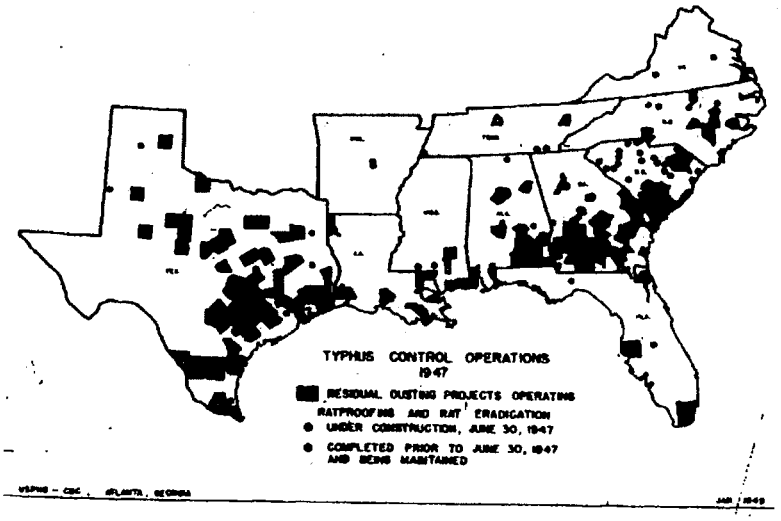

Figure 2. A map of counties with residual dusting projects that made use of DDT for control of murine typhus in 1946 and 1947.[32]

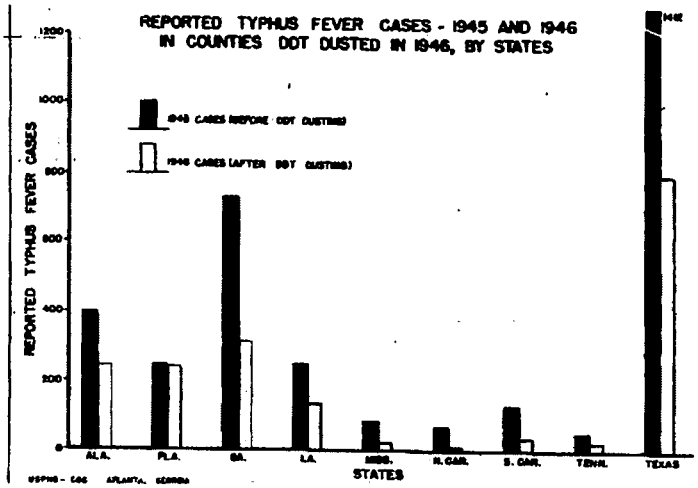

Figure 3. Comparison of numbers of typhus cases in US counties with and without dusting with DDT. The comparison data for not dusting were pulled from 1945 case data.[33]

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31 CDC Activities, 1946-1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 11.

32 CDC Activities, 1946-1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 11.

A total of 448,297 residences were dusted with DDT for typhus control in calendar year 1946.[34] Dusting of premises continued for several more years and declined only when risk of murine typhus had been greatly reduced or eliminated. Examples of extensive DDT use are 1) from September 1 to November 22, 1947, 91,083 premises were dusted;[35] and 2) from March 20 to July 2, 1949, 150,705 premises were treated.[36]

b) Housefly and dysentery control - Although houseflies eventually became resistant to DDT, it was used for a time to control them successfully. Control of houseflies also contributed to reducing problems of dysentery. Some housefly control was a byproduct of the residual spray program for eradicating malaria. As stated in a Missouri State Health Department publication (cited in a CDC report) in 1946:

> [L]ast year the U.S. Public Health Service sprayed 85,000 homes with DDT in the delta country of Mississippi. It was chiefly a malaria control project to kill mosquitoes. But so many flies died as a result of the spraying project that the infant death rate from fly-borne diseases such as dysentery, typhoid fever, diarrhea, and enteritis, was cut to less than one-third that of the year before.
>
> This means that approximately 50 children in Mississippi who otherwise would have died as a result of these diseases are alive today.[37]

c) *Aedes aegypti* control - For many years, DDT was sprayed in towns and cities for control of the mosquito that transmits dengue fever and urban yellow fever, *Aedes aegypti*. Sporadic efforts to control this important vector were converted into a national eradication program in the 1960s.

The United States, along with other countries of North, Central, and South America, first signed a resolution of the Pan American Health Organization to eradicate *Aedes aegypti* in 1947. The U.S. signed a similar resolution in 1961. Funds were appropriated to initiate a U.S. eradication program in 1963.[38] The CDC coordinated the program. This program, with rare

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33 CDC Activities, 1946-1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 10.

34 CDC Bulletin. Jul., Aug., Sept., 1946. U.S. Public Health Service, Atlanta, Georgia. Page 35; CDC Bulletin. Oct., Nov., Dec., 1946. U.S. Public Health Service, Atlanta, Georgia. Pages 38-39.

35 CDC Bulletin. Apr., May, Jun., 1948. U.S. Public Health Service, Atlanta, Georgia. Page 28.

36 CDC Bulletin. Oct., Nov., Dec., 1949. U.S. Public Health Service, Atlanta, Georgia. Page 43.

37 CDC Bulletin. Jul., Aug., Sept., 1946 U.S. Public Health Service. Atlanta, Georgia. Page 32.

38 Fontaine, R.E., Mulrennan, A., Schliessmann, D.J., "1964 Progress Report of the *Aedes aegypti* Eradication Program." Am J Trop Med Hyg., 1965.

exception, was based on spraying DDT, as a matter of official public health policy, in urban residential areas and it was continued until 1969.[39]

d) Plague control – Eventually, DDT was used even in areas of western Texas and in a broad swath of the western U.S. for dusting prairie dog colonies and ground squirrel burrows to kill fleas as a means of plague control.[40]

None of the public health programs that made use of DDT, described above, were trivial in scope. The spraying and dusting of DDT in and around homes was carried out as critical components of national disease control programs. Extensive public health use of DDT brought great improvements in health for millions of Americans.

4. In the 1940s and 50s, there was extensive public health use of DDT in Texas, including Hidalgo County and Mission, Texas. Public health use of DDT in Hidalgo County included spraying inner walls of houses and dusting DDT inside and outside of commercial buildings and houses to control insect-borne diseases. Even in the 1960s, DDT was sprayed in Hidalgo County as part of the national *Aedes aegypti* Eradication Program.

The government and people of Texas participated heavily in public health use of DDT. Malaria in San Antonio, Texas in 1904 to 1908 caused a death rate of 17.5 per 100,000. In the interval from 1909 to 1913, the rate was 13.9 per 100,000 population.[41] To put these death rates into a modern framework, the rates for malaria infections alone were 2- to 4-fold higher than the modern death rate for all cancers combined in the 0 to 19 year age group in the United States.[42] However, malaria and other infectious diseases did more than just kill people. They also reduced their economic wherewithal.

In 1909, a malaria expert characterized the malaria problem in Texas and other southern states as follows:

> Texas, Georgia and Florida are badly infected in some portions with aestivo-autumnal malaria [falciparum malaria]; this form is also common along the southern portion of the Mississippi and its

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39 CDC Operations Manual, "*Aedes aegypti* Eradication Program," Operational Letter No. 7.1 and No. 7.2, June 16, 1966. U.S. Public Health Service, Atlanta, Georgia.

40 CDC Activities, 1946-1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 151-152.

41 Hoffman, F.L., "A Plea for a National Committee on the Eradication of Malaria," Southern Med J 9(5), May 1916, Pages 413-420.

42 "Trends in Childhood Cancer Mortality: United States, 1990-2004," Morbidity & Mortality Weekly Rep. 56(48): 1257-1261, Dec. 7, 2007, http://www.cdc.gov/MMWR/preview/mmwrhtml/mm5648a1.htm#tab

> tributaries and there are regions in the delta of the Mississippi that are uninhabitable because of the prevalence of the most deadly forms of aestivo-autumnal infection.... Mexico and Central America are hot-beds of the most deadly forms of aestivo-autumnal [malaria] infection, and the low-lying coast lines of these countries are among the most dangerous of the lurking places of this form of disease.[43]

Malaria remained a formidable public health problem in Texas even in the 1940s. In 1941, the most malarious areas in Texas were "situated chiefly around the Rio Grande, Red and Sabine Rivers."[44] Public health officials believed in 1941 that malaria was "more widespread and probably more prevalent [in 1941] than in 1930."[45]

DDT was used extensively in Texas for disease control. Spraying houses with DDT to control malaria started at least by 1945. In that year, six counties, to include Hidalgo County, had county-wide residual house spraying programs.[46] During the latter half of 1945, a total of 75,850 Texas houses were sprayed with DDT.[47] Between July and October, 1946, 45,648 premises were sprayed, using 33,531 pounds of DDT, as part of the extended malaria control program.[48] This spraying included Hildago County.[49] For the period April through September, 1946 (corresponding to last half of fiscal year), a total of 46,184 houses were sprayed in Texas.[50] In addition, larviciding with DDT was carried out in nine cities in Hidalgo County, including McAllen and Mission, Texas.[51] From March 23 to June 28, 1947, a total of 455 houses were

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43 Craig, C., "Cyclical Variation in the Incidence of Malaria," A Symposium on Human Malaria, With Special Reference to North America and the Caribbean, Moulton, F.R., ed., AAAS, Washington, D.C., 1941. Pages 131-134.

44 Watson, R.B. and Hewitt, R., "Topographical and Related Factors in the Epidemiology of Malaria in North America, Central America, and the West Indies," A Symposium on Human Malaria, With Special Reference to North America and the Caribbean, Moulton, F.R., ed., AAAS, Washington, D.C., 1941. Pages 135-147.

45 Faust, E.C., "The Distribution of Malaria in North America, Mexico, Central America and the West Indies," A Symposium on Human Malaria, With Special Reference to North America and the Caribbean, Moulton, F.R., ed., AAAS, Washington, D.C., 1941. Pages 8-24.

46 Cox, G.W., Report by the State Health Officer, Texas, undated.

47 Cox, G.W., Report by the State Health Officer, Texas, undated.

48 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

49 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

50 Cox, G.W., Report by the State Health Officer, Texas, undated.

51 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

sprayed in Hidalgo County.[52] From April 1 to June 28, 1947, a total of 41,934 Texas premises were sprayed.[53] During the same time period, Hidalgo County sprayed DDT as a larvicide.[54]

The CDC malaria eradication program continued beyond 1947, and houses in Texas continued to be sprayed to prevent disease transmission. For two half-year periods, July 1 to December 31, 1947 and January 1 through June 30, 1948, a total of 231 and 3,018 houses in Hidalgo County, respectively, were sprayed with DDT.[55]

The spray program in Texas was altered in 1948 from two spray cycles per year to just one spray cycle per year. The amount of DDT applied to house walls was 200 mg/ft^2, which sums to 2.15 grams per square meter of wall surface.[56] Two grams/m^2 of wall surface eventually became the international standard for malaria control treatments. The numbers of houses sprayed in Texas were 89,600, 94,905, and 71,870 in 1947, 1948, and 1949, respectively.[57] For part of 1948 and after, house spraying became less costly and better tolerated by residents. In 1950, a total of 85,355 houses in Texas were sprayed with DDT.[58] As stated in a CDC report for July, August and September, 1952, "...a total of 145,138 spray applications have been made this season."[59] Texas was one of the states that continued spraying houses in the 1950s. For example, 23,885 and 41,273 premises were sprayed in 1952 and 1953, respectively.[60]

Spraying continued in Texas from 1950-53 in part because malaria was still endemic in South Korea during the Korean War and several United States bases were located in Texas.

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52 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947 (Table 1).

53 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

54 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

55 CDC Activities, 1947-1948. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Pages 128-130 (Tables 1 &2).

56 CDC Activities, 1947-1948. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Pages 126.

57 CDC Bulletin. Jan. 1950. U.S. Public Health Service. Atlanta, Georgia. Page 20; CDC Bulletin. Oct., Nov., Dec., 1949. U.S. Public Health Service. Atlanta, Georgia. Page 42.

58 CDC Activities, 1949-1950. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 4.

59 CDC Bulletin. Jul., Aug., Sept., 1952. U.S. Public Health Service. Atlanta, Georgia. Page 40.

60 CDC Activities, 1951-1952. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 28; Monthly Narrative Report of Insect Vector Control Activities for the Texas State Department of Health, Nov., 1953.

With the military returning from the Korean War, the United States had to maintain vigilance to prevent large re-introductions of malaria.[61]

DDT also was used in many other public health endeavors in Texas:

a) Murine Typhus - Texas started using DDT to control murine typhus in 1945.[62] A 1951 CDC report on murine typhus control in Texas stated: "In 1945, the Texas State Department of Health started an extensive typhus control program with operation policies based primarily on the incidence of murine typhus fever in humans. This program consisted of dusting rat runs and harborages with 10 percent DDT plus rat poisoning."[63] The program was effective because rats would pick up the DDT on their fur. DDT killed the rat fleas that were responsible for transmitting murine typhus to humans.

From July to December 1946, dusting of premises for control of murine typhus was performed in 47 Texas counties.[64] To illustrate the magnitude of the dusting program, from July to December, 1946, 31,502 premises were dusted.[65] A total of 3,804 pounds of DDT were used for dusting premises in Hidalgo County alone.[66] In addition, 835 premises were dusted in Hildago County during the next treatment interval from December 1946 to June 1947.[67] Given that there were potentially only about 21,000 houses for the whole county in 1940, many houses would have been dusted.[68] For all of fiscal year 1946, 90,300 premises in Texas were dusted with DDT.[69] In the first half of 1947, another 31,502 premises in 47 counties were dusted for

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61 CDC Bulletin. Jul., Aug., Sept., 1952. U.S. Public Health Service. Atlanta, Georgia. Page 30.

62 CDC Report For Fiscal Year 1946, Communicable Disease Center, Atlanta, Georgia. Feb.-Oct. 1946.

63 CDC Bulletin. Dec., 1951. U.S. Public Health Service. Atlanta, Georgia. Page 53.

64 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

65 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947. Page 4.

66 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947. Table 7.

67 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947. Table 7.

68 There were 106,059 people in Hidalgo County in 1940. Assuming five people per household Hidalgo County would have approximately 21,211 houses. See http://www.mcallen.lib.tx.us/city/population.htm.

69 Report from the Engineering Division of the Typhus Control Branch of the CDC to District, Directors, State Health Department and Others Concerned, dated August 23, 1946. Reported as tabular data, dated July 25, 1946.

murine typhus control.[70] Dusting of premises continued through the rest of the 1940s and into the 1950s.[71]

In the 1951 review of murine typhus control, public health officials concluded that "semiannual DDT dusting of rat runs and harborages in rat-infested business buildings and annual DDT dusting in the residential areas" attributed to reduction of rats and prevalence of murine typhus. All told, through those and other preventive measures, Texas reduced the incidence of murine typhus from 1,844 cases in 1945 to 222 in 1950."[72] This was an 88% reduction in disease.

b) *Aedes aegypti* - Texas and CDC initiated DDT spraying for control of *Aedes aegypti* in 1946.[73] In that year, Hidalgo County participated in a larviciding program, and DDT was one of the insecticides used.[74] The state of Texas combined the *Aedes aegypti* inspection and treatment programs with the typhus program. Activities for both programs were performed when teams visited to inspect houses. As part of *Aedes aegypti* control in 1946, 14,816 privies and dumps were sprayed in Texas, using 6,258 gallons of 5% DDT.[75] Spraying DDT for control of *Aedes aegypti* increased from that time forward and was performed by larviciding and eliminating *Aedes aegypti* larval habitats. As evidence of program evolution, in 1953, 1,704 Texas acres were sprayed for control of mosquito larvae and space spraying was performed over 54,164 acres.[76] Spraying DDT in and around the mosquito breeding sites in back yards and next to houses continued throughout the 1940s and 1950s.[77]

As stated in section 3, funds were appropriated for a national *Aedes aegypti* eradication program in the Fall of 1963.[78] The CDC-coordinated program got underway in 1964. As described in a 1964 annual report, the program had two objectives:

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70 Updated Report to George W. Cox, M.D., State Health Officer, Disease Control in Texas, undated. Page 3.

71 Monthly Narrative Report of Insect Vector Control Activities for the Texas State Department of Health, Nov., 1953.

72 CDC Bulletin. Dec., 1951. U.S. Public Health Service. Atlanta, Georgia. Page 54.

73 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

74 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

75 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947.

76 Monthly Narrative Report of Insect Vector Control Activities for the Texas State Department of Health, Nov., 1953.

77 Trotti, L.J., Report from Assistant State Director of Communicable Disease Control Activities to the State Health Director George W. Cox, M.D. for Fiscal Year 1947, Austin, Texas, Aug. 15, 1947; Monthly Narrative Report of Insect Vector Control Activities for the Texas State Department of Health, Nov., 1953.

78 Schliessmann, D.J., "Initiation of the *Aedes aegypti* Eradication Programme of the USA," Bull Wld Health Org, 1967, Vol. 36; 604-609.

> It will protect this country against outbreaks of yellow fever and dengue, and it will eliminate this reservoir of *Ae. aegypti* as a possible source of reinfestation of countries that have rid themselves of the species.[79]

When the program was initiated, a total of 16 countries within the hemisphere had used DDT to successfully eradicate *Aedes aegypti*.[80] Thus, the second of the two objectives had great significance for many countries of the Americas. Simply stated, eradicating *Aedes aegypti* from the United States would have ended the export of the mosquito back into *Aedes aegypti*-free countries.

The United States *Aedes aegypti* eradication program was based on use of DDT. Southern Texas was one of several test sites for the program. In fact, Hidalgo County was one of four pioneer counties that participated in *Aedes aegypti* eradication efforts in Texas.[81] The startup of this program was reviewed in a 1965 publication, as follows:

> The *Aedes aegypti* eradication program was activated in FY 1964, and by the end of June was operational in southern Florida, southern Texas, Puerto Rico, and the American Virgin Islands. It is planned to have full-scale operations underway in all of the nine infested states in the southeastern United States and also in the State of Hawaii by 1967. This program meets U. S. commitments to participate with other member nations of the PAHO [Pan American Health Organization] in eradicating the *Aedes aegypti* species from the Western Hemisphere as a permanent solution for the prevention of urban yellow fever.[82]

The United States' *Aedes aegypti* eradication program was stopped in 1969.

c) Dysentery – Fly-borne dysentery was a huge problem in many southern states and was a particularly grave problem in Texas. As stated in a CDC report, Hidalgo County was a highly endemic area for fly-borne dysentery.[83] A project of spraying DDT in towns was started in 1945.

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79 Annual Report, F.Y. 1964, *Aedes aegypti* Eradication Branch. U.S. Department of Health, Education, and Welfare, U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 1.

80 Schliessmann, D.J., "Initiation of the *Aedes aegypti* Eradication Programme of the USA," Bull Wld Health Org, 1967, Vol. 36; 604-609.

81 Annual Report, F.Y. 1964. "*Aedes aegypti* Eradication Branch," U.S. Department of Health, Education, and Welfare, U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 4.

82 Fontaine, R.E., Mulrennan, J.A., Schliessmann, D.J., 1964 Progress report of the *Aedes aegypti* Eradication Program. Am J Trop Med Hyg, 1946, Vol. 14, No. 6: 900-903.

83 CDC Activities, 1946-1947. U.S. Public Health Service, Communicable Disease Center, Atlanta, Georgia. Page 23.

Fly control became a front-line defense against dysentery and spraying DDT was, in the early years, an important part of dysentery control. The dysentery control project was described as:

> ...a cooperative undertaking of the Communicable Disease Center and the National Institute of Health, with headquarters at Pharr, Texas. The primary objective is to determine whether or not the control of flies by insecticides will reduce significantly the prevalence of diarrheal disease.[84]

Early CDC reports document heavy experimentation and use of DDT in Texas for fly control during the 1940s and 1950s. For example, applications of DDT for fly control resulted in 30,022 premises being sprayed, residual spraying of 41,273 premises, and space spraying 74,614 acres in Texas.[85]

d) Encephalitis – On April 16, 1954, the Governor of Texas declared a state of emergency in Hidalgo County.[86] The cause of that state of emergency was an outbreak of St. Louise encephalitis (SLE). During the period April 22-May 8, under the Governor's state of emergency and with assistance of the CDC, (1) 1,784 acres were larvicided (insecticide sprayed on surface of water to kill mosquito larvae), (2) 13,942 acres space-sprayed with insecticide to kill adult mosquitoes, and (3) 304 premises residual-sprayed.[87] DDT was used for general larviciding, and DDT was also used to control adult mosquitoes. Dusting was done with gamma isomer BHC.[88] For space spraying and dusting operations, 3310 gallons of 5 percent DDT emulsion and 21,000 pounds of 3 percent gamma isomer BHC dust were applied to 20,225 acres.[89] DDT-diesel oil larvicide in the amount of 1,312 gallons was applied on 263 acres of both actual and potential mosquito breeding area.[90] A human population of approximately 72,000 within 12 cities and towns in Hidalgo County, Texas received protection by use of DDT and BHC to stop the outbreak of SLE.[91]

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84 CDC Bulletin. Oct., Nov., Dec., 1946. U.S. Public Health Service. Atlanta, Georgia. Page 8.

85 Monthly Narrative Report of Insect Vector Control Activities for the Texas State Department of Health, Nov., 1953.

86 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:531.

87 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:531.

88 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:531.

89 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:534.

90 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:534.

91 Beadle, L.D., Menzies, G.C., Hayes, G.R., Zuben, F.J.V., Eads, R.B., "Vector Evaluation and Control," Pub Health Rep, 1957, Vol. 72, No. 6:535.

Data presented in sections a-d show the importance of public health uses of DDT to the disease prevention programs in Texas. These data also demonstrate that public health use of DDT reached down to the household level in many counties and towns in the Texas countryside. Clearly, the public health use of DDT at the household level was both extensive and intensive.

5. DDT also was used internationally in major public health campaigns to control malaria and other insect-borne diseases like dengue, yellow fever, and others. DDT's ability to prevent disease transmission and, in some cases, eradicate the disease vectors led a National Academy of Sciences committee to conclude: "To only a few chemicals does man owe as great a debt as to DDT...In little more than two decades, DDT has prevented 500 million human deaths, due to malaria, that otherwise would have been inevitable."[92]

The effectiveness of DDT for disease control stimulated various countries throughout the world to create national malaria control programs. Their successes eventually stimulated WHO to coordinate a global eradication program.

a) National Malaria Control Programs in South and Central America

In the mid-1940s, DDT was a new, effective, and exciting weapon in the battle against malaria. It was cheap, easy to apply, long-lasting once sprayed on house walls, and safe for humans. Wherever and whenever malaria control programs sprayed DDT on house walls, there were large reductions in malaria rates.

Almost all of the countries of the Americas started using DDT in their malaria control programs. For example, the experiences of countries like Venezuela and Guyana demonstrate the powerful benefit of spraying house walls with small amounts of DDT. In the mid-1940s and before, malaria rates in countries of the Americas were dangerously high. Venezuela had a national malaria program but was still reporting more than 800,000 cases per year.[93] Venezuela started spraying DDT in houses in 1946. The protection was immediate and malaria rates dropped precipitously in all sprayed areas. From the period 1941-45 (pre-spray period) to 1948,

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92 Herzog, A., "An Environmental Failure: Restrictions on DDT," Apr. 22, 2008, Townhall.com: http://www.townhall.com/Columnists/AshleyHerzog/2008/04/22/an_environmental_failure_restrictions_on_ddt

93 Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part I," Trans Roy Soc Trop Med Hyg, 1949, Vol 43, No. 2:113-132; Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part II," Trans Roy Soc Trop Med Hyg, 1949, Vol. 43, No. 2:133-164; WHO, Official Records No. 190, Executive Board Forty-Seventh Session. Geneva, Jan. 19-29, 1971. Part II, Appendix 14, Page 178.

DDT spraying dropped the number of malaria positive slides per 100,000 people from 1,083 to just 82, a 91.25% reduction in malaria.[94]

In the neighboring country of Guyana, the period from 1943 to 1945 was characterized with over 37% and 27% of rural and urban people, respectively, with malaria. Accompanying these high infection rates was a death rate of newborns of 126 per 1,000 live births. Guyana started experimental spraying DDT inside houses in 1945, expanded the program in 1946, and initiated country-wide spraying in 1947.[95] By the 1948-49 time period, as a result of house spraying alone, infant mortality declined by 39%.[96] In urban areas malaria declined by 99%. Even in the highly endemic rural areas malaria infections declined by a stunning 96%.[97]

By the early 1950s, both Venezuela and Guyana had used DDT to eliminate malaria from major geographical areas.[98] Soon malaria infections could be found only in sparsely populated forested areas of both countries. Malaria completely disappeared from heavily populated coastal regions and from much of the interior savannas.

Health workers in other countries also recognized the particular value of DDT. The head of malaria control in Brazil characterized the changes that DDT offered in the following statement:

> Until 1945-1946 [when DDT became available for malaria control], preventive methods employed against malaria in Brazil, as in the rest of the world, were generally directed against the aquatic phases of the vectors (draining, larvicides, destruction of bromeliads, etc....). These methods, however, were only applied in

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94 Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part I," Trans Roy Soc Trop Med Hyg, 1949, Vol 43, No. 2:113-132; Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part II," Trans Roy Soc Trop Med Hyg, 1949, Vol. 43, No. 2:133-164.

95 Giglioli, G., "Changes in the Pattern of Mortality Following the Eradication of Hyperdemic Malaria From a Highly Susceptible Community," Bull Wld Health Org, 1972, Vol. 46, No. 2:181-202.

96 Giglioli, G., "Changes in the Pattern of Mortality Following the Eradication of Hyperdemic Malaria From a Highly Susceptible Community," Bull Wld Health Org, 1972, Vol. 46, No. 2:181-202.

97 Giglioli, G., "Changes in the Pattern of Mortality Following the Eradication of Hyperdemic Malaria From a Highly Susceptible Community," Bull Wld Health Org, 1972,Vol. 46, No. 2:181-202; Curtis, C., ed., Demerara Doctor. An Early Success Against Malaria. The Authobiography of a Self-taught Physician George Giglioli 1897-1975. Smith-Gordon and Co Ltd. (2006).

98 Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part I," Trans Roy Soc Trop Med Hyg, 1949, Vol 43, No. 2:113-132; Gabaldon, A., "The Nation-wide Campaign Against Malaria in Venezuela, Part II," Trans Roy Soc Trop Med Hyg, 1949, Vol. 43, No. 2:133-164; Giglioli, G., "Changes in the Pattern of Mortality Following the Eradication of Hyperdemic Malaria From a Highly Susceptible Community," Bull Wld Health Org, 1972, Vol. 46, No. 2:181-202.

the principal cities of each state and the only measure available for rural populations exposed to malaria was free distribution of specific drugs.[99]

In the late-1950s, under guidance of the World Health Organization and the Pan American Health Organization, most national malaria programs were converted into malaria eradication programs.

b) National Malaria Control Programs in Africa and Asia

Historical data from many countries outside the Americas also show the huge beneficial impact of spraying houses with DDT. Annex 1 to this report contains examples of what occurred in many countries around the world. Many countries started their own malaria control programs. These countries pioneered house spray methods, and laid the groundwork for what would become a WHO-coordinated global malaria eradication program. Over a third of a billion people were freed of endemic malaria even before the WHO-led eradication effort began.[100]

c) WHO's Global Malaria Eradication Program

The 8th World Health Assembly ("WHA") in 1955 directed WHO to coordinate a global program to eradicate malaria. As revealed in statistical reports of malaria eradication in the Americas, the program was fully underway in 1959. During the next nine years, another two-thirds of a billion people were freed of endemic malaria.[101]

Nepal was just one of several countries that benefited from malaria eradication. Some have opined that "dramatic, nationwide reduction of malaria [in Nepal] was perhaps the greatest technical and logistic triumph of the 1960s."[102] That triumph over malaria was based largely on use of DDT as the exclusive preventive measure of the eradication program.

Each year in the early 1950s, there were more than two million cases of malaria in Nepal, with a mortality rate of 10%. The burden of deaths fell most heavily on children. At that time, malaria was Nepal's most serious public health problem, and it contributed to increased deaths from other diseases as well. The U.S. Agency for International Assistance started the malaria

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99 de Bustamante, F.M., Distribuição Geográfica e Periodicidade Estacional da Maaria no Brasil e Sua Relação com o Fatores Climáticos. Situação Atual do Problema. Revista Brasileira de Malariologia e Doenças Tropicais (1957) Page 187.

100 WHO, Official Records No. 190, Executive Board Forty-Seventh Session. Geneva, Jan. 19-29, 1971. Part II, Appendix 14, Table 1, Page 177; Brown, A.W.A, et al., "Malaria Eradication and Control from a Global Standpoint." J Med Entomol Vol. 13, No. 1, Pages 1-25.

101 WHO, Official Records No. 190, Executive Board Forty-Seventh Session. Geneva, Jan. 19-29, 1971. Part II, Appendix 14, Page 177.

102 Skerry, C.A., Moran, K., Calavan, K.M., Four Decades of Development. The History of U.S. Assistance to Nepal 1951-1991. USAID, Kathamandu, Nepal (1991). Page 141: http://pdf.usaid.gov/pdf_docs/PNACP500.pdf

program in Nepal in 1959. Spraying of houses in the central zone began in 1960. Spraying in the eastern zone was underway in 1964 and in the western zone in 1965.[103]

From more than two million cases a year, only 2,468 cases were found in 1968. Before the malaria control program, life expectancy in Nepal was 28 years. By 1962, life expectancy was 33 years, and by 1970 it was 42.3 years.[104] Malaria in Nepal had been an enormous problem in many areas. Malaria had been so bad that it had stopped people from occupying land and producing crops. Once malaria was brought under control, previously unoccupied areas suddenly became available for agricultural productivity. As land became available there were large movements of populations into new areas. Total cost of the multi-year program in Nepal, which oversaw vast improvements in health, dramatically reduced death and increased population longevity and, ultimately, vastly increased agricultural productivity and wealth, was $13 million.[105]

All told, from the mid-1940s to 1969, almost one billion people worldwide were freed from endemic malaria by DDT-sprayed house walls.[106] This achievement is unequaled in the history of arthropod-borne disease control. DDT was used to change the global distribution of endemic malaria. In those countries that coupled malaria eradication with economic growth and development, the disease remained banished after spraying stopped.

d) Role of DDT in Combating Other Arthropod-Borne Diseases

Beginning in 1945, many countries around the world experimented with the use of DDT in the control of a variety of other human diseases, e.g., filariasis. As a successful example of these efforts, Bolivia was the first to employ DDT to eradicate *Aedes aegypti*, the vector of dengue viruses and the urban vector of yellow fever.[107] Both diseases were a frightening burden on populations of Central and South America. *Aedes aegypti* also was the cause of major outbreaks of both diseases in cities of the United States.

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103 Skerry, C.A., Moran, K., Calavan, K.M., Four Decades of Development. The History of U.S. Assistance to Nepal 1951-1991. USAID, Kathamandu, Nepal (1991). Pages 128 and 130: http://pdf.usaid.gov/pdf_docs/PNACP500.pdf

104 Skerry, C.A., Moran, K., Calavan, K.M., Four Decades of Development. The History of U.S. Assistance to Nepal 1951-1991. USAID, Kathamandu, Nepal (1991). Page 74: http://pdf.usaid.gov/pdf_docs/PNACP500.pdf

105 Skerry, C.A., Moran, K., Calavan, K.M., Four Decades of Development. The History of U.S. Assistance to Nepal 1951-1991. USAID, Kathamandu, Nepal (1991). Pages 50-53, 141-150: http://pdf.usaid.gov/pdf_docs/PNACP500.pdf

106 WHO, Official Records No. 190, Executive Board Forty-Seventh Session. Geneva, Jan. 19-29, 1971. Part II, Appendix 14, Page 177.

107 Severo, O., "Eradication of the *Aedes aegypti* Mosquito From the Americas," Yellow Fever: A Symposium in Commemoration of Carlos Juan Finlay, The Jefferson Medical College of Philadelphia: Philadelphia, PA (1955) Pages 32 and 49.

By 1947, Bolivia had eradicated *Aedes aegypti* mosquitoes.[108] Bolivia's quick success stimulated the Pan American Health Organization to begin a program using DDT to eradicate *Aedes aegypti* from the Americas.[109] By the early 1950s, many countries of the Americas had eradicated or greatly reduced the distribution of this dangerous mosquito. Through their successes, the risks of dengue and yellow fever epidemics largely disappeared from Central and South America. The countries maintained their *Aedes aegypti* free status for many years.

In summary, DDT reduced the burden of human disease and saved lives not only in Texas and throughout the United States, but also in countries around the world.

6. Throughout the 1950s, 1960s and early-1970s, DDT was heralded as a lifesaver and credited with saving millions of lives.

DDT was held in high regard by agriculture and public health officials in the United States and the world throughout the 1950s, 1960s and early-1970s, as reflected by the following statements:

- U.S. Public Health Service, Dr. S.W. Simmons (1959): "Except for the antibiotics, it is doubtful that any material has been found which protects more people against more disease over a larger area than does DDT."[110]

- World Health Organization (1969): "DDT has been the main agent in eradicating malaria in countries whose population total 550 million people. Of having saved about 5 million lives and prevented 100 million illnesses in the first 8 years of its use, of having recently reduced the annual malaria death-rate in India from 750,000 down to 1,500, and of having saved at least 2 billion people in the world without causing the loss of a single life by poisoning from DDT alone."

  "[I]t [DDT] is so safe that no symptoms have been observed among the spray men or among the inhabitants of the spray areas, which numbered respectively 130,000 and 535 million at the peak of the campaign."[111]

- Malaria Eradication Department of the Pan American Health Organization, Dr. Guzman Garcia-Martin, Chief (1969): "To date, there is no insecticide that could

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108 Severo, O., "Eradication of the *Aedes aegypti* Mosquito From the Americas," Yellow Fever: A Symposium in Commemoration of Carlos Juan Finlay, The Jefferson Medical College of Philadelphia: Philadelphia, PA (1955) Pages 49.

109 Severo, O., "Eradication of the *Aedes aegypti* Mosquito From the Americas," Yellow Fever: A Symposium in Commemoration of Carlos Juan Finlay, The Jefferson Medical College of Philadelphia: Philadelphia, PA (1955). Page 39-58.

110 Simmons, S.W., "DDT the Insecticide Dichlorodiphenyltrichloroethane and its Significance," Human and Veterinary Medicine, Vol. II, ed. Mueller, P., Basel, Birkhauser Verlag (1959). Page 252.

111 WHO, "The Present Place of DDT in World Operations for Public Health," Symposium, Oregon State University, Corvallis, Oregon, Aug. 1969.

effectively replace DDT which would permit the continuation of the eradication program or maintain the conquests made so far. The withdrawal of DDT will therefore represent a regression to a malaria situation similar to that in 1945."[112]

In addition, leading health organizations and officials stated that DDT did not pose a threat to public health.

- National Academy of Science (1969): "Available evidence does not indicate that present levels of pesticides in man's food and environment produce an adverse effect on his health."[113]

- Environmental Protection Agency, Samuel W. Simmons (1972): "Malaria control campaigns have extended over 2 decades, and no toxic effects have been reported among the hundreds of millions of people who live in houses that have been sprayed nor among the 200,000 or more spray-men applying the material."[114]

- Communicable Disease Center, Atlanta, Georgia (1969): "Although DDT has been studied more extensively in man than any other known insecticide, no concrete evidence has been presented that it presently constitutes any health hazard to man. Its use record with regard to human safety is unparalleled in the history of insecticides."[115]

- Assistant Attorney General of the United States, William Ruckelshaus, (1970): "DDT is not endangering the public health and has an amazing and exemplary record of safe use. DDT, when properly used at recommended concentrations, does not cause a toxic response in man or other mammals and is not harmful. The carcinogenic claims regarding DDT are unproved speculation."[116]

- United States Surgeon General Steinfield (1972): "The safety, long-lasting action, and low cost of DDT make it the only known insecticide that can be used on the scale required in malaria eradication programs within the resources currently available to

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112 Letter by Dr, Guzman Garcia-Martin, Chief, Malaria Eradication Department, Pan American Health Organization, Jun. 10, 1969.

113 National Academy of Science Report, Committee on Persistent Pesticides, May, 1969.

114 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 50.

115 Beatty, R.G., The DDT Myth: Triumph of the Amateurs. John Day Co., Jun. 1973. Page 22.

116 Ruckelshaus, W.D., Brief for the Respondents, U.S. Court of Appeals for the District of Columbia, No. 23813, on Petition for Review of the Order of the Secretary of Agriculture, Aug. 31, 1970, as cited in Thomas Jukes, "DDT Stands Trial Again," Bioscience, 22 (Nov. 1972), Page 672; Aaron Wildavesky, But is it True? A Citizen's Guide to Environmental Health and Safety Issues, Harvard University Press, Cambridge, Massachusetts, 1995.

such programs or that can be reasonably expected to be available to them in the foreseeable future."[117]

- World Health Organization, (1970): "There had been no toxic effects recorded among the 200,000 spraymen employed and the 600 million population living in sprayed houses over a long period of time."[118]

Even as late as 1972, the U.S. Surgeon General stated: "We have no information on which to indict DDT either as a tumorigen or as a carcinogen for man and on the basis now available, I cannot conclude DDT represents an imminent health hazard."[119]

At the time of EPA's DDT hearings in 1972, national and international public health officials stood united against political and regulatory controls that would minimize availability, increase costs, or in any way jeopardize future uses of DDT in disease control programs. Public health professionals recognized the overwhelming benefit of using DDT for disease prevention.

7. The period 1971-1973 marked the end of large scale DDT use in the United States. In June, 1972, the newly-formed EPA ruled against the continued use of DDT for agricultural uses. Preceding this EPA ruling, Administrative Law Judge Edmund Sweeney held 11 months of DDT hearings in 1971-72 and ruled not to de-list DDT for use in agriculture or public health. Two months later, and with no new data or evidence against DDT, EPA Administrator William Ruckelshaus overturned Judge Sweeney's ruling and de-listed the use of DDT for agricultural purposes.

The anti-insecticide movement was a growing phenomenon during the 1960s. The overwhelming popularity of Rachel Carson's book, "Silent Spring," brought the movement political power over public health and agricultural programs that made use of insecticides. Claims were being trumpeted loudly that DDT was causing human health harm.

In spite of strong positions of the agriculture and public health communities in support of DDT, the stage was set for political action against DDT. The Nixon administration announced its intent to eliminate DDT and other pesticides.[120] When the Department of Agriculture and the public health service did not act precipitously against DDT, the Nixon administration removed

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117 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 50-51.

118 WHO, Executive Board Forty-Fifth Session, Geneva, Jan., 20-29, 1970, Summary Records. Mar. 1970, Page 155.

119 Surgeon General of the United States, EPA. Consolidated DDT Hearing, Mar. 1972, Tr. 1350.

120 Kramer, J.R. "Pesticide Research: Industry, USDA Pursue Different Paths." Science 166, Dec. 12, 1969, Pages 1383-1386.

authority over insecticides from the Department of Agriculture and the public health service and transferred that authority to the newly-formed Environmental Protection Agency ("EPA"). Immediately following EPA's creation on December 2, 1970, the regulatory control of DDT was one of its first action items.[121]

EPA started its review of DDT in 1971 by appointing Administrative Law Judge Edmund Sweeney, as the Hearing Examiner to hold a hearing on the benefits and claims of harm from DDT use. The consolidated DDT hearing continued for eighty-one days from August 17, 1971 to March 16, 1972. Following the testimony of 125 witnesses, 365 exhibits, and 9,312 pages of transcripts, Hearing Examiner Edmund Sweeney presented his findings of fact, conclusions of law, and opinion on April 26, 1972.[122] In reaching his conclusions, Judge Sweeney noted:

> ... [N]o Hearing Examiner will ever enjoy the privilege that I had in listening to so many leaders in the field of scientific and medical achievement; from so many areas of expertise throughout the world, really; and including among them a Nobel Peace Prize winner, and the Surgeon General of the United States.[123]

After months of testimony, the judge found in favor of continued DDT use. The Hearing Examiner's 114-page opinion reached the following conclusions:

1. "DDT is not a carcinogenic hazard to man."[124]
2. "DDT is not a mutagenic or teratogenic hazard to man."[125]
3. "The uses of DDT under the registrations involved here do not have a deleterious effect on freshwater fish, estuarine organisms, wild birds, or other wildlife."[126]
4. "There is a present need for the continued use of DDT for the essential uses defined in this case."[127]

Reportedly, EPA Administrator William Ruckelshaus did not attend the hearings or read the transcript of the hearings.[128] However, just a few weeks later, the Administrator overruled

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121 Lewis, J., "The Birth of EPA." EPA Journal, Nov. 1985; http://www.epa.gov/history/topics/epa/15c.htm

122 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 12-13.

123 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 16.

124 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 93.

125 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 93.

126 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 94.

127 EPA. Consolidated DDT Hearing, Hearing Examiner's Recommended Findings, Conclusions, and Orders (40 CFR 164.32), April 25, 1972, Page 94.

his own judge and de-listed all agricultural uses of DDT in the United States.[129] In ultimate findings of fact, Ruckelshaus found that DDT's chemical characteristics constitute a risk to the environment; an unknown, unquantifiable risk to man and lower animals; and a potential carcinogenic risk to humans.[130] His finding of basic fact that "DDT is a potential human carcinogen" was a newly formed opinion because he was the same person (William Ruckelshaus) who declared in 1970 that "[c]arcinogenic claims regarding DDT are unproven speculation." (See Ruckelshaus, Section 6, above). The Administrator's opinion was not based on any new evidence, because none was brought forward. The EPA Administrator ignored the national and international agencies that steadfastly defended DDT and opposed drastic and far-reaching actions to stop its use. Ultimately, the opinion was characterized as not being based on scientific evidence but rather on political pressure.

The Ruckelshaus decision was appealed to the United States Court of Appeals, District of Columbia Circuit.[131] On December 13, 1973, the D.C. Circuit Court affirmed the Administrator's decision to cancel DDT registrations for agricultural uses, concluding "...where questions involve a special expertise of an agency, such as in detailed scientific proceeding, the agency deserves special deference from the courts."[132] The Court noted that the hazardous nature of DDT was not "... proved beyond a reasonable doubt" and that the evidence "...might support contrary conclusions as well."[133]

To summarize this section, the 1972 decision was an amazing switch on the part of the United States government. No new scientific or medical evidence was brought forward to justify EPA's decision. There were no new insights or proofs of human health harm. In spite of official positions, as presented above, the misrepresentations of DDT science by the anti-insecticide movement brought about an abrupt change in government policy against DDT in 1972.[134]

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128 Santa Ana Register, "EPA Chief did not Read all Evidence." Jul. 23, 1972.

129 EPA. Environmental Appeals Board, I.F. & R. Docket Nos. 63, et al., Consolidated DDT Hearings, Opinion by William D. Ruckelshaus, Jun. 2, 1972.

130 EPA. Environmental Appeals Board, I.F. & R. Docket Nos. 63, et al., Consolidated DDT Hearings, Opinion by William D. Ruckelshaus, Jun. 2, 1972, Page 17-18.

131 United States Court of Appeals, District of Columbia Circuit, 489 F.2d 1247, 160 U.S.App.D.C. 123, Dec. 13, 1973.

132 United States Court of Appeals, District of Columbia Circuit, 489 F.2d 1247, 160 U.S.App.D.C. 123, Dec. 13, 1973.

133 United States Court of Appeals, District of Columbia Circuit, 489 F.2d 1247, 160 U.S.App.D.C. 123, Dec. 13, 1973.

134 Misrepresentations of science during the EPA hearings in 1972 have been reviewed systematically in: Ackerly, R.L., "DDT: A Re-evaluation. Part I," Chemical Times and Trends, Oct. 1981, Pages 47-53; Ackerly, R.L., "DDT: A Re-evaluation. Part II," Chemical Times and Trends, Jan. 1982, Pages 48-55.

8. Unsupported claims that DDT is harmful to human health have limited the effectiveness of disease control programs, allowing deadly diseases to re-emerge.

In 1972, there was no equally safe, cheap, and effective chemical for replacing DDT in disease control programs. For that reason, public health officials around the world opposed delisting DDT for agriculture use. Those officials knew that delisting DDT would create pressures to stop its use to control human diseases and its production, distribution, sale, and use would become more expensive. Today, 36 years after the EPA decision, we can document how the fears of those public health officials became reality.

DDT production declined in the U.S. and other developed countries during the 1970s. Disease-endemic countries found it increasingly difficult to obtain supplies of DDT to meet their disease control requirements. Even before the 1972 EPA decision, this concern led a WHO official (James W. Wright, Chief, Vector Biology and Control, World Health Organization, Geneva) to state in 1970: "The prospects of maintaining the gains of the malaria eradication effort and protecting the 370 million people still unprotected from malaria depended essentially on the availability of an insecticide which was effective against the vector, inexpensive, and safe to man."[135] This official also noted: "[I]t could be categorically stated than any action limiting the availability or use of DDT for the control of malaria and other vector-borne diseases in developing countries could lead to a public health disaster."[136]

House spraying controlled malaria and even eradicated it in some regions of the world. The period of spraying and intensive control of malaria lasted for about 33 years, ending in 1979. By 1979, those who opposed use of insecticides had gained political leverage over malaria control policies and strategies. During the 1970s and 1980s, through the exercise of economic and political pressures, anti-insecticide activism brought huge changes in malaria control programs. Countries began experimenting with DDT substitutes and slowly stopped using DDT. Malaria re-emerged in many of those countries even as they switched to alternative insecticides. The trend of increasing disease was strong and revealed shortfalls of substitute insecticides.

Data from countries of the Americas clearly document changes in malaria rates that coincide with changes in house spray rates (Figure 4). As illustrated in Figure 4, malaria rates increased as the number of sprayed houses declined.

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135 WHO, Executive Board, Forty-fifth Session, Summary Records, Geneva, 20-29 January 1970. WHO, Geneva, Page 155.

136 WHO, Executive Board, Forty-fifth Session, Summary Records, Geneva, 20-29 January 1970. WHO, Geneva, Page 156.

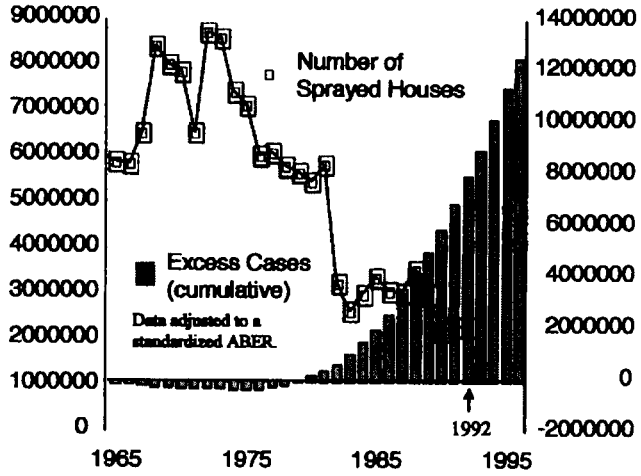

Figure 4.[137] Graph of increasing numbers of malaria cases, above baseline, with declining numbers of houses sprayed with DDT.

Similarly, before the advent of DDT, Mexico was a highly malarious country with 60% of its population living in endemic regions. During the 1940s and 1950s, malaria was responsible for as many as 24,000 deaths and 2.4 million infections each year.[138] Malaria was a huge health and economic burden.

DDT was used to bring malaria under control in Mexico until the mid-1980s. In the early 1980s, Mexico suffered a recession and reduced its house spray program. Numbers of infections increased to 133,698 cases per year. Mexico restarted its spray program and rapidly brought malaria back under control.[139]

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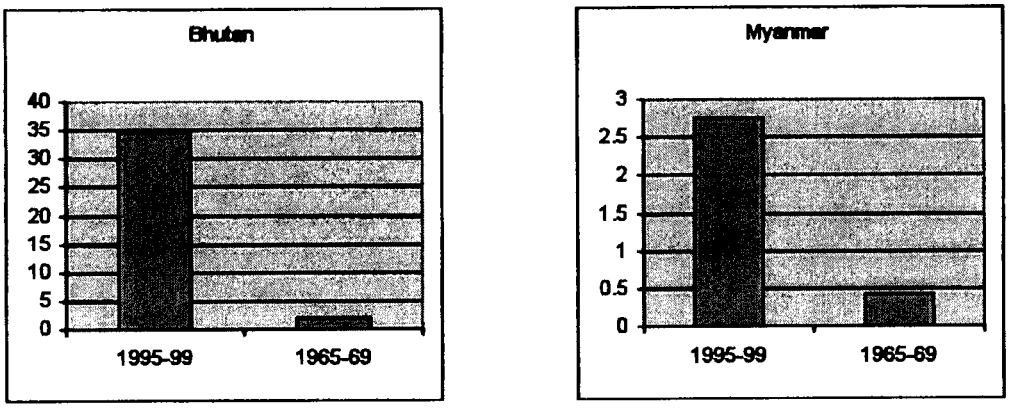
137 Impact of de-emphasizing indoor spraying of house walls in 1979 and decentralization of malaria control programs beginning in 1985. (Line graph=numbers of sprayed houses.; bar graph= cumulative numbers of excess cases over average numbers per annum from 1965-1979; left axis is sprayed houses; right axis is excess cases). Data for Brazil, Colombia, Peru, Ecuador and Venezuela. Based on data from PAHO reports (see cite 139). Numbers of cases calculated by standardizing slide positive rates per 1000 population according to a standardized annual blood examination rate. Standardized rate was calculated as average for each country during period of 1965-1979. Adjustments were made for differences in size of population across 5 countries.

138 Marquez-Escobedo, M.B., "Estado Actual de la Erradicación del Paludismo en Mexico," Boletín de la Oficina Sanitaria Panamericana, 1960:414-423.

139 Pan American Health Organization, "Status of Malaria Programs in the Americas. XL Report." Washington (DC), 1991. Page 91.

In addition, house spraying with DDT brought spectacular reductions of malaria in many countries of Asia. Years later, when countries began gradually to abandon house spray programs, the numbers of malaria cases began increasing.[140] Figures 2-5 contrast malaria rates in recent years with the years when DDT was used. The data represent annual parasite indexes (a population-based index of malaria prevalence) during the period from 1995-99 compared with identical data from 1965-69. Differences in rates for the two performance periods are stunning.

Today, out of 30 countries in Asia, Bhutan, Myanmar, and Sri Lanka are the three most malarious.[141] In Bhutan, the malaria burden has grown 17.5-fold since the period when DDT was sprayed on house walls. For the countries of Myanmar, Sri Lanka, and India, malaria rates have grown 6.7-, 6.4-, and 807-fold, respectively.



Figures 5 and 6. Annual parasite indexes (APIs) for Bhutan and Myanmar for comparison periods of 1995-99 versus 1965-69.[142] The latter (1965-69) covers a period when DDT was used to spray house walls for malaria control. The period 1995-99 represents a period when DDT was not used to spray houses. Left axes represent API values, or the number of cases per thousand population.

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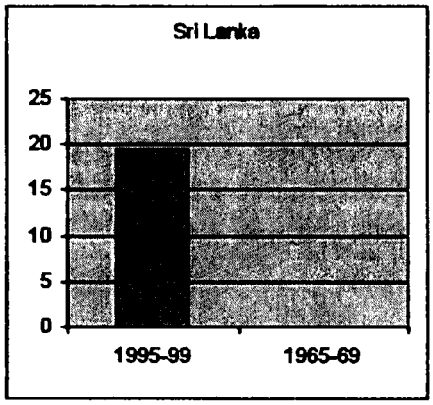
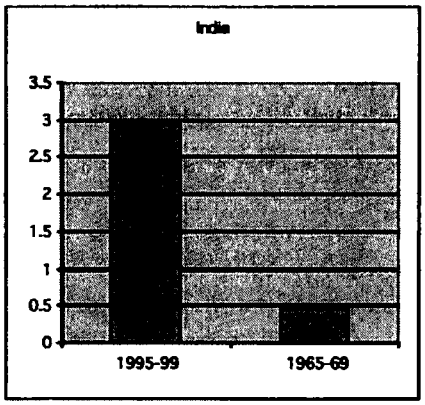
140 Roberts, D., et. al., "DDT, Global Strategies, and a Malaria Control Crisis in South America," Emerg. Inf. Dis., 1997, Page 3:297; Roberts, D., et al., "DDT House Spraying and Re-emerging Malaria," Lancet, 2000, Vol. 356:330-332. Data presented in graphs were extracted from WHO reports: "WHO, Malaria Profile," http://w3.whosea.org/malaria/pdf/ino.pdf.

141 Malaria rate by country: http://www.overpopulation.com/faq/health/infectious_diseases/malaria/asia.html.

142 Data presented in graphs were extracted from WHO reports: "WHO, Malaria Profile," http://w3.whosea.org/malaria/pdf/ino.pdf.





Figures 7 and 8. Annual parasite indexes (APIs) for Sri Lanka and India for comparison periods of 1995-99 versus 1965-69.[143] The latter (1965-69) covers a period when DDT was used to spray house walls for malaria control. The period 1995-99 represents a period when DDT was not used to spray houses (still used to a greatly reduced extent in India). Left axes represent API values, or the number of cases per thousand population.

In summation, anti-insecticide pressures caused countries to abandon use of DDT for control of malaria. The result, as illustrated by data presented above, has been a reversion to increasingly intense transmission of malaria and rapidly growing numbers of new infections each year.

The public health harm of anti-DDT advocacy was not limited to destruction of malaria control programs. The *Aedes aegypti* Eradication Program in the Americas was impacted. The use of DDT was phased out of use during the 1970s and the *Aedes aegypti* mosquito re-invaded all countries of the Americas, thereby bringing dengue and the risk of urban yellow fever back to all countries of Central and South America.

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143 Data presented in graphs were extracted from WHO reports: WHO, Malaria Profile: http://w3.whosea.org/malaria/pdf/ino.pdf

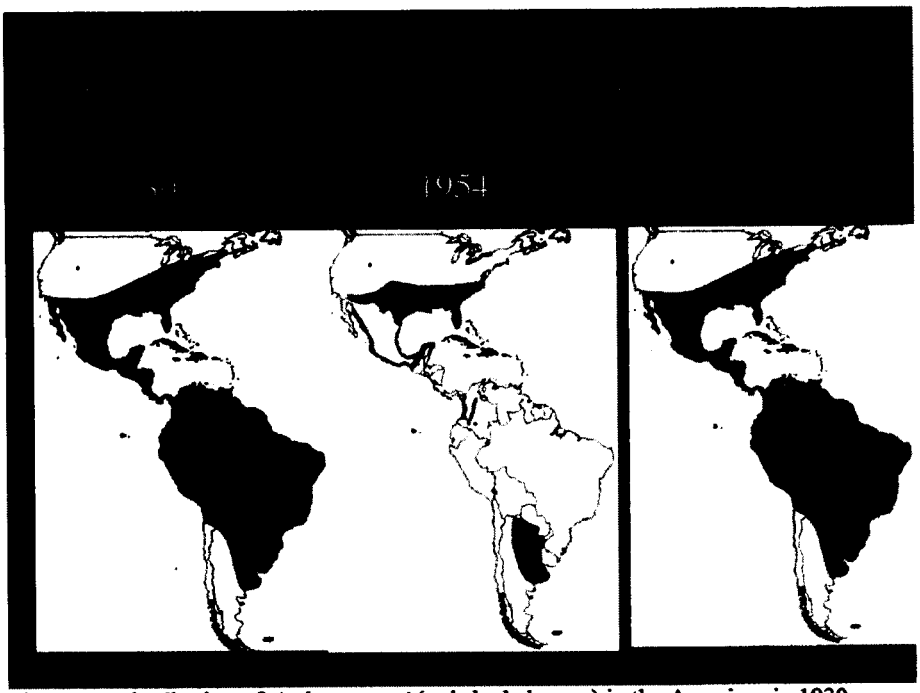

Figure 9. Distribution of *Aedes aegypti* (red shaded areas) in the Americas in 1930, during *Aedes aegypti* eradication in 1954, and in 1998 after the program had been stopped by anti-insecticide activism.[144]

The growing anti-DDT advocacy and de-listing of DDT in the United States ultimately resulted in the ramping down of eradication programs in Central and South America. The impact on other countries of stopping *Aedes aegypti* eradication in the United States was foreseen in the CDC's stated objectives for starting an eradication program in the United States:

> It will protect this country against outbreaks of yellow fever and dengue, and it will eliminate this reservoir of *Ae. aegypti* as a possible source of reinfestation of countries that have rid themselves of the species.[145]

Thus, when the United States stopped its own eradication program, Americans inadvertently harmed human health and economic growth in many other countries in the hemisphere. As programs in other countries were abandoned and *Aedes aegypti* returned to its old haunts in Central and South America, dengue fever went along.[146] Today, epidemics of dengue fever and dengue hemorrhagic fever are annual occurrences. Dimensions of the continuing catastrophe of dengue fever are growing. Each year there are millions of dengue cases and many deaths due to

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144 http://www.cdc.gov/ncidod/dvbid/dengue/map-ae-aegypti-distribution.htm.

145 CDC. Annual Report, F.Y. 1964, *Aedes aegypti* Eradication Branch, U.S. Department of Health, Education, and Welfare, Public Health Service, Communicable Disease Center, Atlanta, Georgia, Page 1.

146 CDC. Division of Vector-borne Infectious Diseases, http://www.cdc.gov/ncidod/dvbid/dengue/index.htm#current; PAHO, "Re-emergence of Dengue in the Americas," Epidemiological Bulletin, 1997, Vol. 18(2).

dengue hemorrhagic fever.[147] Given that most countries of the Americas had, for many years, eradicated the major vector of this disease, the modern burden of dengue for the people of Central and South America constitutes a colossal public health disaster. In 2008, Brazil suffered a devastating epidemic of dengue fever.[148] The 2008 epidemic may become the worst dengue outbreak in Brazil's history. Previous peaks in number of Brazilian cases for a given year occurred in 1998 with 500,000 cases, and 794,000 cases in 2002.[149] Devastating outbreaks of dengue are occurring each year in Mexico and other countries and regions of Central and South America.

Beyond the problems of a resurgent dengue fever problem, a silent but smoldering threat of urban yellow fever has accompanied the return of *Aedes aegypti* to countries of the Americas. Countries have had to redouble their yellow fever vaccination programs, at enormous costs, just to try and protect against a potential urban outbreak. Without the freedom to use DDT, the problems of dengue fever and risks of urban yellow fever are growing worse each year. Growing problems of yellow fever are illustrated by the national alerts that have been issued already in 2009. Yellow fever alerts, and in some cases multiple alerts, recently have been issued by Brazil, Argentina, Paraguay, Trinidad, and Venezuela.[150]

9. Even after DDT was de-listed for use in 1972, government officials and other users continued to obtain permission to employ DDT for certain public health, agriculture, forestry, and pharmaceutical uses in high-risk locations. In addition, DDT continued to be present in pesticide formulations and used widely in crops during the 1970s and 1980s, in the miticide Dicofol, also known as Kelthane.[151]

Even within the United States, the EPA decision to de-list agricultural uses of DDT in 1972 did not entirely close the book on issues of DDT use. Rather, EPA retained authority to make case-by-case determinations allowing use of DDT for experimentation, public health, and

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147 Gubler, D.J., "*Aedes aegypti* and *Aedes aegypti*-borne Disease Control in the 1990s: Top Down or Bottom Up," 49th Franklin Craig Lecture, American Society of Tropical Medicine & Hygiene, Washington, D.C. (Dec. 7, 1988), published Jan. 1, 1989.

148 "Thousands Hit by Brazil Outbreak of Dengue," CNN.com, Apr. 3, 2008, http://www.cnn.com/2008/HEALTH/conditions/04/03/brazil.dengue/index.html

149 Sequira, J.B., Jr., et al., "Dengue and Dengue Hemorrhagic Fever, Brazil, 1981- 2002," Emerg Inf Dis, 2005, Vol. 11(1):48-53.

150 promed@promed.isid.harvard.edu: (1) YELLOW FEVER - SOUTH AMERICA (13): VENEZUELA (ARAGUA), MONKEYS, SUSPECTED: Jan. 29, 2009, 10:01 pm; (2) YELLOW FEVER - SOUTH AMERICA (11): TRINIDAD, MONKEYS, CONFIRMED: Jan. 23, 2009, 2:09 pm; (3) YELLOW FEVER - SOUTH AMERICA (08): BRAZIL (RIO GRANDE DO SUL), MONKEY, SUSPECTED: Jan. 22, 2009, 2:40 pm; (4) YELLOW FEVER - SOUTH AMERICA (03): ARGENTINA (MISIONES) SUSPECTED: Jan. 15, 2009, 3:42 pm; (5) YELLOW FEVER - SOUTH AMERICA (04): PARAGUAY ex ARGENTINA (MISIONES): Jan. 16, 2009, 11:55 am.

151 "Around The Nation: E.P.A. Considers Ban on Pesticide Using DDT," The New York Times, Mar. 20, 1984, http://query.nytimes.com/gst/fullpage.html?res=9807E0DA1039F933A15750C0A962948260.

quarantine purposes by public officials, and even crop infestations that could not be controlled with other insecticides.[152] EPA also enacted emergency exemption regulations to permit government agencies to use DDT for a variety of purposes.[153] Exemptions were granted for, among other things, control of the pea leaf weevil, Douglas fir moth, fleas, and plague.[154]

In addition, DDT continued to be present in pesticide formulations and used widely in crops during the 1970s and 1980s. DDT was a significant component (9-15%) of the miticide Dicofol, also known as Kelthane.[155] Regulations were passed only in the mid-1980s to require that levels of DDT in Dicofol be below 0.1%. Dicofol was used widely as a miticide on cotton and used heavily in Hidalgo County, Texas. The following map, prepared by the Department of the Interior, illustrates broad usage of Dicofol on cotton in 1992 (Figure 10).

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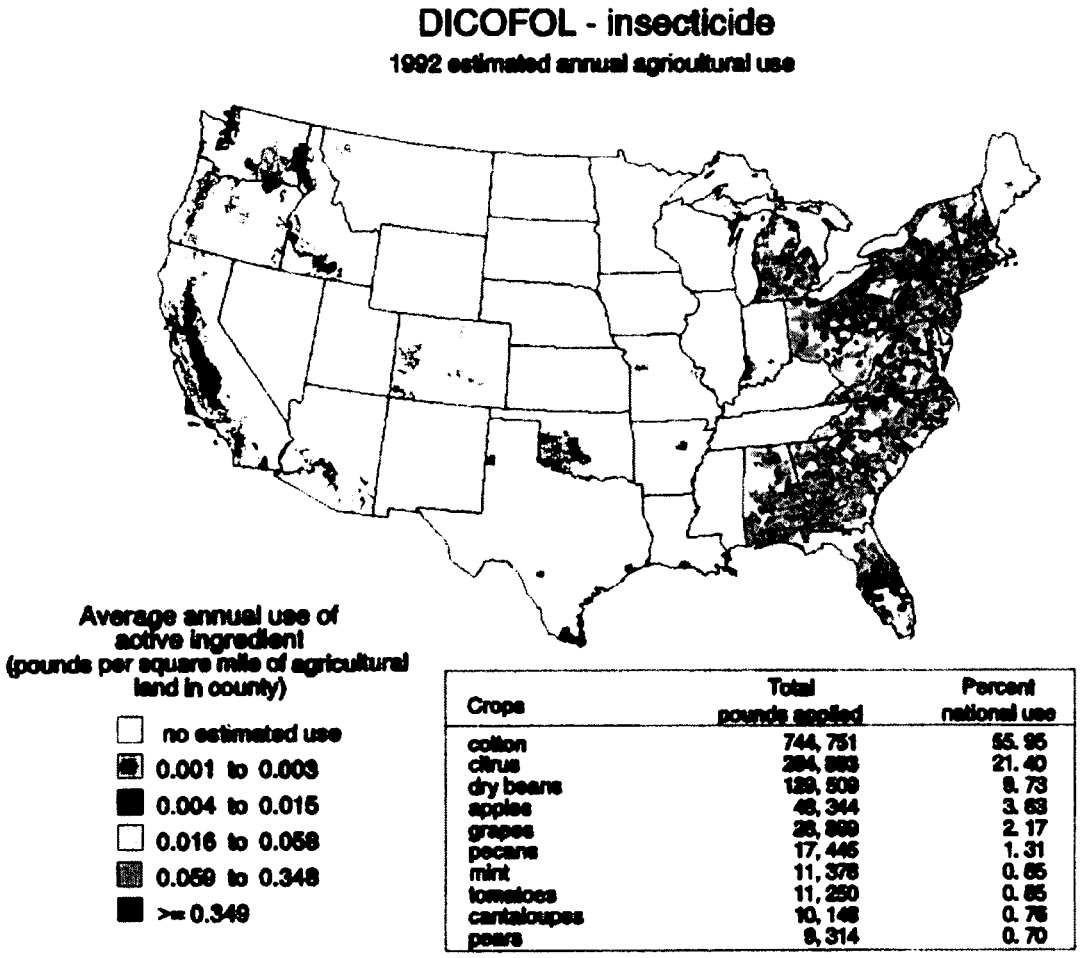
152 EPA. Environmental Appeals Board, I.F. & R. Docket Nos. 63, et al., Consolidated DDT Hearings, Opinion by William D. Ruckelshaus, Jun. 2, 1972, Page 19-20.

153 EPA. Exemption of Federal and State Agencies for Use of Pesticides Under Emergency Conditions, 40 CFR Part 166, Jan. 15, 1986. Stated in this revision of rules was notice that "Regulations implementing section 18 (40 CFR Part 166) were first promulgated in 1973..."

154 Actions for limited use registration: DDT to Control Pea Leaf Weevil, Fed Reg 39(54), Mar. 19, 1984; Request for emergency exemption: Use of DDT to Control the Douglas-fir Tussock Moth, Fed Reg 39(44), Mar. 5, 1974; Issuance of specific exemption: Use of DDT for Emergency Rabid Bat Control, Fed Reg 39(129), Jul. 3, 1974; Crisis exemption: Use of DDT to Control Flea Vectors of Plague, Fed Reg 41(111), Jun. 8, 1976.

155 "Around The Nation: E.P.A. Considers Ban on Pesticide Using DDT," The New York Times, Mar. 20, 1984, http://query.nytimes.com/gst/fullpage.html?res=9807E0DA1039F933A15750C0A962948260



Crops	Total pounds applied	Percent national use
cotton	744,751	55.95
citrus	284,883	21.40
dry beans	129,509	9.73
apples	48,344	3.63
grapes	28,899	2.17
pecans	17,445	1.31
mint	11,378	0.85
tomatoes	11,250	0.85
cantaloupes	10,148	0.76
pears	9,314	0.70

Figure 10. Map for 1992 showing distribution of Dicofol use in the United States

DDT eventually was removed as a registered public health insecticide in the United States because of failure to pay fees to re-register DDT for public health in 1989. Use of DDT for quarantine, public health, and pharmaceuticals is still possible under the exemption process as long as it is consistent with EPA's 1972 order. Moreover, DDT is still eligible for registration as a public health insecticide.

10. Today, the World Health Organization (WHO) and other U.N. bilateral and multilateral organizations have concluded that DDT is safe for humans and safely can be sprayed on house walls for disease control.

Beginning in the mid-1990s, the United Nations Environment Program started efforts to eliminate public health use of DDT through negotiations for a persistent organic pollutants ("POPs") treaty. Treaty negotiations mobilized public health workers around the world in a campaign to prevent global elimination of DDT and restore the freedom of malaria endemic countries to use DDT in disease control programs. As a result of that campaign, DDT was

placed in Annex B of the Stockholm Convention, meaning that it is still available for use in public health programs, to include malaria control.[156]

Today, the United States Agency for International Development ("USAID") promotes indoor spraying for malaria control and supports the use of DDT.[157] WHO also endorses use of indoor spraying of insecticides, to include use of DDT.[158] As official policy, DDT is still approved for public health use by the WHO, USAID, UNICEF, the Global Fund, World Bank, Roll Back Malaria, and, as stated above, by the Stockholm Convention for Persistent Organic Pollutants.

Success of the campaign to prevent global elimination of DDT reflects certain fundamental facts about DDT and its long history of both intensive and extensive use, both in agriculture and public health. The facts are:

- There is still no concrete and replicated proof that DDT causes human cancer or other diseases.
- There is not even one documented human death from chronic or environmental exposure to DDT.
- Heavily DDT-sprayed areas do not exhibit the harmful effects activists claim (higher human mortality, more instances of cancer, lower fertility, etc.)

The new freedoms that now exist which allow countries to use DDT if they choose to do so has led to large reductions in disease and death in countries that have restarted house spraying and use of DDT.[159]

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156 Stockholm Convention of Persistent Organic Pollutants, http://www.pops.int/documents/convtext/convtext_en.pdf.

157 http://www.usaid.gov/press/factsheets/2005/fs051223.html; http://www.usaid.gov/press/releases/2005/pr051215.html.

158 http://www.businessday.co.za/articles/opinion.aspx?ID=BD4A189817; http://www.who.int/mediacentre/news/releases/2006/pr50/en/index.html.

159 http://news.bbc.co.uk/2/hi/africa/4264374.stm; http://www.washtimes.com/national/20060503-122415-3878r.htm.

11. DDT is unequaled in its ability to control disease. It exhibits a complex of chemical actions to prevent disease transmission inside homes in three primary ways: (1) it is a powerful spatial repellent that stops mosquitoes from entering houses; (2) it is a strong contact irritant that causes mosquitoes to prematurely exit sprayed houses, often without biting; and (3) it kills mosquitoes that remain in prolonged physical contact with sprayed surfaces. DDT is the only insecticide presently recommended for spraying on house walls that actually stops a large proportion of mosquitoes from entering the house and transmitting diseases while residents are sleeping.

DDT is unmatched by other insecticides because of its low cost of production (no patent protections), effective duration of residual activity when sprayed on walls, and broad spectrum of chemical actions. No other insecticide can be produced and used as cheaply as DDT. DDT protects residents for as long as twelve months or more after it is sprayed on walls. No other recommended insecticide provides a comparable duration of residual activity. Other insecticides need to be sprayed in houses every two to four months and the need for frequent sprayings adds greatly to cost of the public health programs.

Although DDT is often thought of as a chemical that kills insects, it actually exerts a complex of chemical actions. It functions as a spatial repellent, a contact irritant and a contact poison. The spatial repellent and contact irritant actions function at concentrations of DDT that are far below concentrations needed for toxicity. DDT's actions of repellency, irritancy and toxicity intervene at specific points in the mosquito's sequence of malaria transmission behaviors. For instance, repellent action prevents the mosquito from entering the house altogether. If the mosquito does go indoors, then the contact irritant action causes the mosquito to exit, often before it even bites.[160] Lastly, with prolonged contact with a sprayed surface, the mosquito that enters the house in spite of repellant and irritant actions still may succumb to DDT's toxic action.[161]

In simple terms, the separate actions of DDT residues provide an interactive and cumulative benefit. For purposes of illustrating these interactions, imagine that the actions of repellency, irritancy, and toxicity each function at a level of 70 percent effectiveness. If one hundred mosquitoes enter a house not sprayed with DDT (see figure next page), then only thirty would enter if it were treated with DDT, as the other seventy would be repelled and not enter (see figure next page). Of the thirty mosquitoes that enter the house, twenty-one would be stimulated by contact irritant actions and exit, potentially without biting. This leaves nine mosquitoes that remain indoors and bite. Of these, six or seven would absorb a toxic dose of DDT and die, the other three or four would escape and survive. By summing the numbers of

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160 However, a negative aspect of the contact irritant action would be to stimulate premature exiting after the mosquito bites, but before it absorbs a toxic dose of DDT.

161 Roberts, D.R., et al., "A Probability Model of Vector Behavior: Effects of DDT Repellency, Irritancy, and Toxicity in Malaria Control," J Vect Ecol, 2000, Vol. 25, Pages 48-61.

mosquitoes impacted by the three chemical actions of DDT, we find that although all of these actions function at equal levels of activity, spatial repellent and contact irritant actions together reduce the overall risk by 91 percent, while toxicity reduces risk by a maximum of only 6 to 7 percent. Overall, risk would be reduced by 95-96 percent.

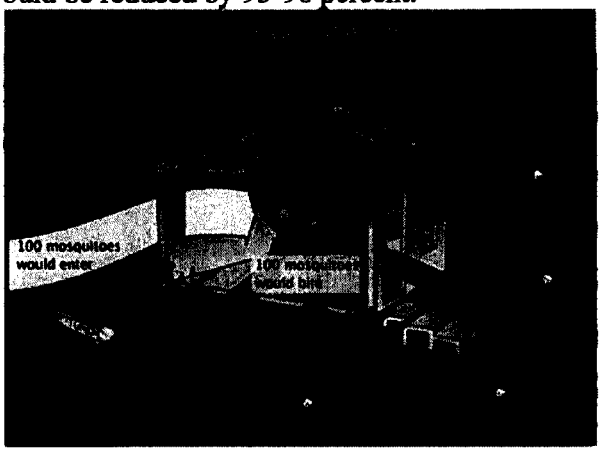

Figure 11. Behavior events for an idealized population of one hundred malaria mosquitoes that fly to a house that is not sprayed with DDT. All hundred mosquitoes would enter, bite, escape and survive if the house was not sprayed with DDT.

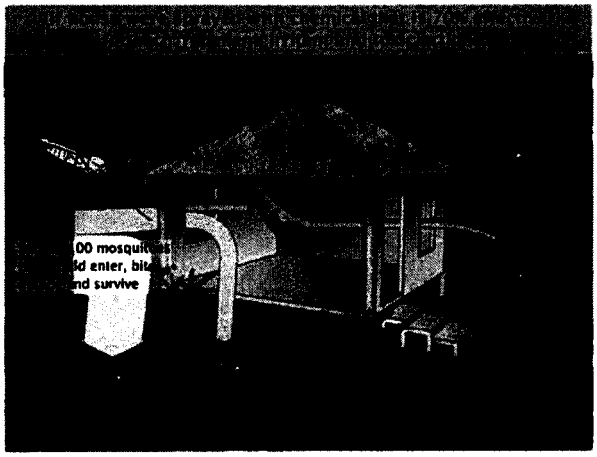

Figure 12. Behavior events for an idealized population of one hundred malaria mosquitoes that fly to a house that has been sprayed with DDT. In this model, we assume that each of the three chemical actions of DDT function at a 70% level of effectiveness. The model illustrates sequential behaviors of house entering, biting, and escaping the house.

The example above is based on repelling and irritating only 70 percent of the mosquitoes. Computer simulations of interactions between repellent, irritant and toxic actions show that an

insecticide would need to repel and irritate less than 30 percent each before toxicity would become the primary mode of chemical action against malaria vectors.[162] Yet in the real world, as demonstrated in different field studies, levels of DDT's repellent and irritant actions often function far above the 70% level of effectiveness.

Of the insecticides presently recommended for malaria control, only DDT has the complex of chemical actions described above. For this reason, no other insecticide is equally efficacious against most vector mosquitoes in most epidemiological settings. This profile of chemical actions goes far in explaining why DDT is still recommended and still needed for disease control programs in endemic countries.

12. In the 1950s and 1960s, there were no adequate substitutes for major uses of DDT, such as cotton and public health. This was true even in 1972 when EPA de-listed all agricultural uses of DDT in the United States. Even today, there is no equally effective substitute for DDT in public health programs.

The question of adequate DDT substitutes has been and continues to be polemic in the controversy over EPA's 1972 political action to stop agricultural uses of DDT in the United States. Statements presented by plaintiff's expert, Dr. Stanley Tocker, typify the controversy surrounding EPA's 1972 decision. Under the subheading of "Chlorinated hydrocarbon insecticide substitutes," Dr. Tocker states: "[L]ower persistence, efficacious technical chemicals were available as alternatives to the chlorinated hydrocarbons for virtually all insecticide markets." In the case of DDT, history and science have proven Dr. Tocker's assessment wrong – both during the 1950-1967 period and, for public health, even today.

a) Prior to 1972, prestigious study groups concluded that safer, adequate substitutes were not available for important DDT uses.

The lack of adequate substitutes for DDT was recognized even before EPA's precipitous action in 1972, as revealed in an article published in *Science* magazine in 1969:

> DDT substitutes, many experts say, are often potentially at least as dangerous as DDT, if not more so. In many cases no really adequate alternative is available.[163]

Such concerns were based on extensive deliberations of individual scientists, officials within major organizations, and important groups which had debated whether DDT was still needed. During the eight years leading up to the EPA decision to de-list DDT for agricultural uses, seven major groups met to consider whether DDT should continue to be used. The groups were:

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162 Roberts, D.R., Hshieh, P., "What is the Role of Insecticide Resistance in the Re-emergence of Major Arthropod-borne Diseases," The Resistance Phenomenon in Microbes and Infectious Disease Vectors, Institute of Medicine of the National Academies, Workshop Summary, National Academies Press, 2003, Pages 94-104.

163 Kramer, J.R., "Pesticide Research: Industry, USDA Pursue Different Paths," Science, Dec. 12, 1969, Vol. 166, Pages 1383-1386.

a. The President's Science Advisory Committee
b. The Environmental Pollution Panel of the President's Science Advisory Committee
c. The Committee on Persistent Pesticides of Biology and Agriculture, National Academy of Sciences
d. The Health, Education and Welfare Commission on Pesticides and their Relationship to Environmental Health, chaired by Dr. Emil Mrak, December, 1969
e. The Council on Occupational Health
f. The Council on Environmental and Public Health of the American Medical Association
g. The United States Congressional Committee on Agriculture, chaired by Congressman Poage, March, 1971.

The de-listing of DDT for agriculture was not recommended by a single one of these prestigious groups. In essence, each concluded that for some uses, no alternative insecticide is as cheap, effective, or as safe as DDT.[164]

The deliberations of committees affiliated with the American Medical Association have particular relevance to reports concerning whether or not DDT harms human health. In 1970, both the Council on Occupational Health and the Council on Environmental and Public Health of the American Medical Association ("AMA") made joint recommendations on continued use of DDT. The two committees, after noting that DDT accumulates in humans, reported:

> ...[T]here has been no significant increase in the storage of DDT by the general population in the United States since it was first measured in 1950. Pesticide handlers who have been studied with great care during the past 30 years have concentrations in fat as much as 50 times as high. Yet, careful research has shown no interference with their health despite long-continued exposure. Injuries to humans have been observed only in persons who accidentally received acute massive doses.[165]

The two councils jointly recommended:

> [T]he use of DDT should be continued for the control of pests on crops for which, at this time, no adequate alternative is available.[166]

Scientists and groups of scientists rather uniformly opined that DDT was safe to use, that environmental exposures had not been shown to be harmful to human health, and that precipitous

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164 Beatty, R.G., The DDT Myth: Triumph of the Amateurs, 1973, Page 22.

165 AMA. Committee on Occupational Toxicology. "Evaluation of the Present Status of DDT with Respect to Man," JAMA, May 11, 1970, Vol. 212(6):1055-1056.

166 AMA. Committee on Occupational Toxicology. "Evaluation of the Present Status of DDT with Respect to Man," JAMA, May 11, 1970, Vol. 212(6):1055-1056.

political actions against DDT would cause increased exposures of occupationally exposed workers to more highly toxic chemicals.

b) Potential DDT substitutes often have greater acute toxicities.

In his report dated February 8, 2008, Dr. Tocker incorrectly stated: "[L]ower persistence, efficacious technical chemicals were available as alternatives to the chlorinated hydrocarbons for virtually all insecticide markets." The implication is that formulators should have selected other insecticides for formulation and that the other chemicals would have safely met agriculture requirements in southern Texas. In making this statement, Dr. Tocker failed to consider how DDT-containing pesticides were being used by Hayes-Sammons' customers in southern Texas in the 1950s and 1960s.

Cotton and vegetables were important crops in southern Texas during the 1950s and 1960s. DDT was used on these crops from at least 1956 to 1964, and in 1965 and 1966, if not later.[167] In many areas, cotton producers depended on heavy use of DDT up to the time DDT was de-listed for use in agriculture in 1972. For example, as shown in an EPA document:

> Of the quantity of the pesticide [DDT] used in 1970-72, over 80 percent was applied to cotton crops, with the remainder being used predominantly on peanut and soybean crops.[168]

Given that a primary usage of DDT was on cotton, any suggestion that formulators should have (or could have) switched to safer, less persistent insecticides must take into account the options for alternative insecticides on cotton.

Dr. Tocker claims that carbamates (carbaryl), phosphonates (diazinon and malathion), soil fumigants and insect repellants were appropriate substitutes for DDT in the Hayes-Sammon's plant. None of these was as safe and cost-effective as DDT. Even the EPA formally recognizes that:

> Alternatives to DDT are not equally efficacious or economically feasible in all areas due to pest resistance and other factors.[169]

Similarly, in October 1970, the Texas Governor's Scientific Advisory Panel recognized that no "suitable alternative methods" were available to control certain agricultural pests,

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167 Stevens, L.J., et al., "Pesticides in Soil, Monitoring Pesticides in Soils From Areas of Regular, Limited, and No Pesticide Use." Pesticides Monit J 4(3), Dec., 1970: Tables 1, 3, and 4.

168 http://www.epa.gov/history/topics/ddt/02.htm.

169 EPA, DDT, A Review of Scientific and Economic Aspects of the Decision to Ban Its Use as a Pesticide. Prepared for: Committee on Appropriations, U.S. House of Representatives, U.S. Environmental Protection Agency, Washington, D.C., July, 1975: Page 167.

including cutworms, cotton flea-hopper, carrot weevil, sweet potato weevil, and grape berry moth.[170]

D. General Conclusions

Decades of research show that DDT is safe for human exposure. This is not just my opinion, but also is reflected in the official positions of WHO, USAID, UNICEF, World Bank, and the Global Fund, all of which endorse the use of DDT in malaria control programs today. Over several decades, hundreds of millions of houses have been sprayed with DDT. Generations of people have lived in those houses and still no harm has been shown to result from those DDT exposures. The same conclusion can be drawn for spray operators and chemical plant workers who were occupationally exposed, sometimes for their whole adult lives, to very high levels of DDT. There is no disputing the fact that WHO itself consistently has opined that DDT, when used according to WHO guidelines, is safe for use in malaria control programs. What this means is that the world's premier organization for public health supports spraying DDT repeatedly **inside the homes** where people live.

The high efficacy, low cost, and large margin of safety for humans exposed to DDT are important reasons why DDT-sprayed house walls are so effective in control of malaria. Anti-insecticide pressures to stop spray programs, and to stop use of DDT in particular, are the main reasons malaria has re-emerged in many countries around the world. Anti-insecticide pressures and the damage they caused to the hemisphere-wide *Aedes aegypti* eradication program are also the primary reasons for the re-invasion of the Americas by the yellow fever mosquito. This re-invasion has been accompanied by the return of dengue fever, and presence of dengue hemorrhagic fever in the Americas. The return of *Aedes aegypti* to urban areas of the Americas is also the reason for a growing threat of urban yellow fever in countries of South America. Even today, there is no chemical that equals DDT's ability to safely protect the health and welfare of poor people in poor countries from major diseases like malaria and dengue.

Signed at Clifton Forge, Virginia on 30 September 2009.

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Professor Emeritus
Department of Preventive Medicine and Biometrics
The Uniformed Services University of the Health Sciences
Bethesda, MD 20814

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170 "Use of Agricultural Pesticides in Texas," Prepared by the Governor's Scientific Advisory Panel on the Use of Agricultural Chemicals, Oct. 1970. Page 15-16.

ANNEX 1: National Malaria Control Programs in Africa and Asia

Historical data from many countries outside the Americas further evidence the huge impact of spraying houses with DDT. Selected examples are as follows:

a) South Africa

The vector control programs in southern Africa adopted DDT shortly after the end of WWII. Residual spraying with DDT brought rapid reductions in the number of malaria cases. In the Transvaal Province of South Africa, hospital admissions "fell from 1,177 cases during the 1945-46 transmission season to 601 in 1946-47 coinciding with the availability of DDT in 1946, and falling to 454 in 1948 and to a low of 61 cases in 1951."[171] Similar successes were echoed in other southern African countries where DDT was used.

b) Sri Lanka

After DDT was introduced for control of malaria in Sri Lanka (then Ceylon) in 1946, in a three-year period the spleen rate (malaria infections cause enlarged spleens) dropped from 77% to 2.7% and general infant mortality dropped by 62%.[172] By 1954, infections had declined from 413 per 1000 to 0.85 per 1000 people.[173] These statistics reveal reductions in millions of malaria cases per year.

c) Taiwan

The newly-formed Republic of China (Taiwan) adopted DDT use in malaria control shortly after the Second World War. In 1945, there were over 1 million cases of malaria on the island; however, by 1969 there were only nine cases.[174] Shortly thereafter, the disease was eradicated from the island (and remains eradicated today).[175]

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171 Mabaso, et al., "Historical Review of Malarial Control Policies in Southern Africa with Emphasis on the Use of Indoor Residual House-spraying," Trop Med Intl Health, Aug. 2004, Vol. 9, No. 8. Pages 846-856.

172 Brown, A.W.A., et al., "Malaria Eradication and Control From a Global Standpoint," J Med Entomol 1976, Vol. 13, No. 1:1-25.

173 Brown, A.W.A., et al., "Malaria Eradication and Control From a Global Standpoint," J Med Entomol 1976, Vol. 13, No. 1:1-25.

174 Malaria Eradication in Taiwan, May 1991, Department of Health, The Executive Yuan, Republic of China.

175 Malaria Eradication in Taiwan, May 1991, Department of Health, The Executive Yuan, Republic of China.

In summary, extensive uses of DDT for malaria control preceded the global eradication program. As shown by the statistics presented above, countries used DDT to combat malaria and greatly improved the health of their residents.

ANNEX 2: Properties and Functions of DDT

A) Basic Biological and Environmental Relationships

DDT is an organic compound, which means it includes atoms of carbon. It is referred to as a chlorinated hydrocarbon. Many useful chemicals are chlorinated hydrocarbons. For example, chloroquine is a drug for treating malaria infections and it is a chlorinated hydrocarbon. DDT is an aromatic compound, meaning the DDT formula includes benzene rings.[176] Chloroquine is also an aromatic compound. The chemical formula of DDT also includes five atoms of chlorine.[177] Chlorine, like fluorine and bromine, is a halogen, so DDT is characterized as a halogenated compound. As with many chlorinated hydrocarbon compounds, DDT is hardly soluble in water and has low vapor pressure.[178] Thus, very few molecules of DDT will be present as vapor in a unit of air or as a solution in a unit of water. To illustrate this point, consider that water becomes saturated with DDT at a concentration of about two parts per billion. In other words, there will be two parts DDT to one billion (1,000,000,000) parts water.

DDT vapor in air or as a solution in water can be broken down quickly. DDT binds to organic particles so it also can be present in water and air as suspensions of organic materials that contain DDT. That is, DDT has adhered to an organic particle that become suspended in water or air. DDT is soluble in oils and organic solvents and is fat-soluble (or lipophilic).[179]

DDT can degrade rapidly in many environments. The processes of breakdown include chemical, biological, and photo-degradation. Recent studies show that uptake and degradation of DDT is complex.[180] In addition to degradative processes, the ebb and flow of DDT in both living organisms and the environment works through a process of partitioning. Partitioning helps explain DDT persistence. Partitioning occurs in different ways in the environment and living organism. In a living organism, DDT is partitioned into fat. Storage of DDT in fat cells can be referred to as compartmentalization in fat, and is not something that just happens. DDT is stored

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176 The characteristic of aromaticity is related to specific chemical traits, as defined in a wikipedia, meaning "electrons are free to cycle around circular arrangements of atoms, which are alternately singly and doubly bonded to one another. (More properly, these bonds may be seen as a hybrid of a single bond and a double bond, each bond in the ring being identical to every other.)"; http://www.google.com/search?hl=en&defl=en&q=define:Aromaticity&sa=X&oi=glossary_definition&ct=title.

177 Metcalf, C.L., et al., Destructive and Useful Insects. Their Habits and Control. Fourth Edition. McGraw-Hill Co. 1962, Pages 341-342.

178 Metcalf, C.L., et al., Destructive and Useful Insects. Their Habits and Control. Fourth Edition. McGraw-Hill Co. 1962, Pages 341-342.

179 WHO, DDT and Its Derivatives. Geneva, 1979: http://www.inchem.org/documents/ehc/ehc/ehc83.htm.

180 Trevaskis, N.l., et al., "Tissue Uptake of DDT is Independent of Chylomicron Metabolism," Arch Toxicol, 2006, Vol. 80(4):196-200; Tebourbi, O., M.R. Driss, M. Sakly, K.B. Rhouma. 2006. Metabolism of DDT in different tissues of young rats J Environ Sci Health B 41(2):167-76.

in fat by very specific biological or biochemical processes in which the living organism is an active participant, and it is a rate-limited process. Most organisms will accommodate only limited quantities of DDT.[181] This has proven to be true for humans, other mammals, and birds. Quantities of DDT beyond what the organism will accept are degraded and excreted. The major excretory product of DDT in humans is a water-soluble DDT metabolite known as DDA. As an excretory product, DDA is found in urine.

Through the processes of degrading and/or excreting DDT or by controlled partitioning of DDT into and out of fatty tissues, humans deal exceedingly well with chronic exposures to DDT. Findings from medical history, physical examination, routine clinical laboratory tests, and chest x-ray film of high occupational exposures to workers in DDT production did not reveal any ill-effects.[182] These findings are consistent with results of a CDC study conducted on human volunteers in the 1950s. In that study, humans were fed 32 mg of DDT per day for two years and then underwent an additional two years of medical follow-up. The people in this study suffered no medical harm from their high chronic DDT exposures.[183]

In the environment, DDT may be degraded to a point of disappearing. DDT is photo-degraded in sunlight. DDT in a solution of seawater has a half-life of about 10 days. Some fungi can mineralize DDT, white rot fungi for example.[184] Its half-life in the presence of some plants is only two to three days.[185] Natural sunlight quickly will degrade DDT in vapor phase.

Given the multiple pathways for DDT degradation and elimination, how is it possible that the chemical can persist in the environment? Actually, as stated above, environmental persistence is due mostly to the phenomenon of partitioning. That is to say, DDT can persist for long periods when partitioned into soil and organic materials and, to varying degrees, not be available for degradation. The term sequestered describes the condition of DDT being bound tightly to organic particles (soil particles for example). It is important to note that sequestration and long-term persistence means that DDT, in its long-term persistent or partitioned form, is not readily available for biological, chemical or photo-degradation.[186] If DDT is not bio-available, it

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181 Cueto jr., C., Durham, W.F., Hayes jr, W.J.,Hayes, "The Effect of Known Repeated Oral Doses of Chlorophenothane (DDT) in Man," J Am Med Assoc 1956, Vol. 162, No. 9:890-897.

182 Laws, E.R. Jr., Curley, A., Biros, F.J., "Men With Intensive Occupational Exposure to DDT," Amer Med Assoc., 1967, Vol. 15, Pages 766-775.

183 Cueto jr., C., Durham, W.F., Hayes jr, W.J.,Hayes, "The Effect of Known Repeated Oral Doses of Chlorophenothane (DDT) in Man," J Am Med Assoc 1956, Vol. 162, No. 9:890-897.

184 Bumpus, J.A., Aust, S.D., "Biodegradation of DDT [1,1,1-trichloro-2,2-bis(4-chlorophenyl)ethane] by the White Rot Fungus *Phanerochaete chrysosporium*," Appl Environ Microbiol, 1987, Vol. 53, No. 9,: 2001-2008: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=204048

185 Garrison, A.W., Nzengung, V.A., et al.. "Phytodegradation of p,p'-DDT and the Enantiomers of o,p' DDT." Environ Sci Technol, 2000, Vol. 34, No. 9, Pages 1663-1670.

186 http://www.rosalindfranklin.edu/dnn/chicagomedicalschool/home/cms/biochem/faculty/walters/drugs.aspx As specified at this website: If a chemical is "too lipid-soluble, it may partition into fat stores and not reach the intended site of action. The balance between these two properties [solubility in octanol versus water] is measured roughly by the octanol-water partition coefficient. When octanol and water are mixed, they form

will not act against living creatures or be acted upon by living creatures (i.e., not inflict harm or be metabolized or degraded). If at some point the sequestered DDT becomes bio-available, then it will either be degraded or re-sequestered. In spite of these mechanisms for DDT persistence, once DDT is no longer applied to the environment, and as history has shown, DDT residue levels will rapidly decline. This is true for both animate and inanimate components of the global environment.

B) Routes of Exposure and Basic DDT Functions

Theoretically, DDT can be absorbed in three ways. These are (1) through the skin, (2) inhaled and absorbed through respiratory membranes, or (3) ingested and absorbed through the intestinal tract. The following general assessments relate to what might be expected from some typical environmental exposures. DDT absorption through skin is notoriously inefficient, so this is not a primary means of DDT exposure. As mentioned before, DDT has low vapor pressure and only exceedingly small concentrations of DDT vapors will be found in air. Particulate DDT can be found in air; but particulates are not absorbed through respiratory membranes. Instead, particulates actively are transported out of respiratory passages and eliminated. Thus, inhalation is not an effective means of DDT exposure. Since DDT is not very soluble in water (can be characterized as hydrophobic), imbibing DDT in water is not a primary means of DDT exposure. Basically, ingesting DDT in food is the primary method for humans to acquire DDT.[187]

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a two-phase system (like an oil-and-vinegar dressing). Octanol roughly approximates the hydrophobicity of membrane lipids. So shake your drug in a mixture of octanol and water, let the layers separate, and measure the amount of your drug in each layer. The ratio [conc. in octanol]/[conc. in water] is the partition coefficient, P. The logarithm of P (log P) should ideally be less than 5.

187 http://www.atsdr.cdc.gov/toxprofiles/tp35.html

CURRICULUM VITAE

DONALD R. ROBERTS, Ph.D
Professor Emeritus, The Uniformed Services University of the Heath Sciences in Bethesda, Maryland

Present Address: 118 First Street, Clifton Forge, VA 24422

Retirement Date: June 30, 2007

Post-retirement activities (June 2007 to present):

Member, Board of Directors, Africa Fighting Malaria (see: http://www.fightingmalaria.org)

Member, External Advisory Committee (EAC) for Sanaria, a company working to develop sporozoite-based malaria vaccines. The EAC is an advisory group for Safely Working with Plasmodium falciparum-infected mosquitoes, Address: Sanaria Inc., 9800 Medical Center Dr., Suite A209, Rockville, MD 20850

Member, Expert Scientific Advisory Committee for the Public Health Product development arm of the Innovative Vector Control Consortium (IVCC), The IVCC " is a Product Development Partnership developing vector control products and information systems. Bringing together expertise and technical resources with an initial award of $50.7 million from the Bill & Melinda Gates Foundation," (see: http://www.ivcc.com/organisation/index.htm)

I continue to write professional papers and editorials, make professional presentations at national meetings, review manuscripts for publication, and review research grant proposals.

Manuscript reviews:

American Journal of Tropical Medicine and Hygiene
Emerging Infectious Diseases
Journal of the American Mosquito Control Association
International Journal of Tropical Medicine
Acta Tropica
Environmental Entomology
Pest Management Science

Research grant reviews (2009 only):

U.S. Army Medical Research and Development Command
British Medical Research Council
Innovative Vector Control Consortium

EDUCATION:

Ph.D. - Medical Zoology, University of Texas School of Public Health, Houston, Texas. 1973.
M.S. - Entomology, University of Missouri, Columbia, MO. 1966.
B.S. - Zoology (Wildlife Conservation), University of Missouri, Columbia, MO. 1965.

ACADEMIC APPOINTMENTS:

1986-June 30, 2007: Professor, Division of Tropical Public Health, Department of Preventive Medicine and Biometrics, Uniformed Services University of the Health Sciences, Bethesda, MD 20814

1997-1999: Acting Director, Division of Tropical Public Health, Department of Preventive Medicine and Biometrics, Uniformed Services University of the Health Sciences, Bethesda, MD 20814

1984-1986: Associate Professor, Department of Preventive Medicine and Biometrics, Uniformed Services University of the Health Science, Bethesda, MD 20814

1978-1980: Visiting Professor, Nucléo De Medicina Tropical e Nutrição, Federal University of Brazil, Brasilia, Brazil and Chief, Department of Entomology, United States Army Medical Research Unit, Brasilia, Brazil.

SENATE TESTIMONY:

Roberts, DR. 2004. Senate Testimony before Senator Brownback's Subcommittee on East Asian and Pacific Affairs on Neglected Diseases in East Asia: Are Public Health Programs Working? October 6, 2004 at 2:30 p.m. in the Dirksen Building, Washington, D.C. 13 pp.

Roberts, DR. 2005. Senate Testimony before the full committee of the Environment and Public Works Committee on the misuse of science in environmental policy making. September 28, 2005. Specific topic was misrepresentations of science during decades of environmental campaigning against DDT. Presented in the Senate Building, Washington, D.C. morning of September 28, 2005.

Roberts, DR. 2006. Senate Testimony before Chairman Coburn's Subcommittee on Federal Financial Management, Government Information, and International Security. Title of hearing was "Bilateral Malaria Assistance: Progress and Prognosis." Hearing was held the afternoon of January 19, 2006. Presented views on malaria control and DDT use in public health programs.

Roberts, DR. 2007. Senate Testimony before Chairman Boxer, ranking member Inhofe and members of the Senate Committee on Environment and Public Works. Title of hearing was "Examining the Human Health Impacts of Global Warming". Hearing was held October 27, 2007. I presented views on the fear tactics used to stimulate pubic policy on

climate change-- drew parallels between fear tactics used in the anti-DDT campaign with those presently used in the global warming campaign.

LANGUAGES:
Portuguese

TEACHING:
USUHS: Courses taught:

Medical Acarology (course Director): 1988-present.
Aquatic Biology (Tutorial) (course Director): 1988-present.
Vector Biology (course Director): 1984-present.
Ecological Statistics (Tutorial) (course Director) 1998-present.

Participating instructor:
Environment and Health II: 1984-1986.
Tropical Medicine and Hygiene: 1984-1990.
Advanced Techniques in Medical Entomology: 1988-present.
Malaria Lectures in Diagnostic Parasitology: 1984-present.
Malaria Epidemiology and Control: 1989-present.

I have presented miscellaneous courses in Belize, Peru, Brazil, and Honduras. Topics include malaria, Disease control campaigns, Chagas disease, and applications of remote sensing and geographic information systems to public health.

Instructor, WRAIR Tropical Medicine Course (1982-1985)

Instructor, "Vector Biology and Control," WHO-sponsored course; International Center for Tropical Medicine Research, University of South Carolina (1986).

Instructor, National Academy of Science-sponsored "ELISA Workshop," USUHS (April 16-18, 1986)

Instructor, "Environmental Entomology and Pesticides Workshop," US Army Environmental Hygiene Agency, Aberdeen Proving Ground, Edgewood, MD (May 6-10, 1985 and April 20-24, 1987)

Instructor, "Vector Biology and Control," WHO-sponsored course taught in Portuguese at the University of South Carolina (1983).

Instructor, "Tropical Medicine for Health Care Professionals," WHO-sponsored international course conducted at the Nucléo de Medicina Tropical e Nutrição, UnB, Brasilia, Brazil (1979).

Instructor, "Extension Course in Tropical Medicine," Nucléo de Medicina Tropical e Nutricação, Universidade de Brasilia, Brasilia, Brazil (1978-1980).

Instructor, "Pest Control Operations and Equipment" Army Environmental Hygiene Agency, Edgewood Arsenal, MD (1970).

PROFESSIONAL ASSIGNMENTS:

1995-present: Director, Center for Applications of Remote Sensing and GIS in Public Health, a center within the Centers for Preventive Medicine and Public Health within the Department of Preventive Medicine/Biometrics, Uniformed Services University of the Health Sciences, Bethesda, MD.

1997-1999: Director, Division of Tropical Public Health, Department of Preventive Medicine/Biometrics, Uniformed Services University of the Health Sciences, Bethesda, MD

1980-1984: Chief, Department of Entomology, Walter Reed Army Institute of Research, Washington, D.C. 20307.

1978-1980: Deputy Commander, United States Army Medical Research Unit-Brasilia. Performed the duties of deputy commander and conducted malaria research in the Amazon Basin.

1976-1978: Commander, United States Army Medical Research Unit-Belém, Brazil. Performed the duties of unit commander and studied the mechanisms of epidemic and inter-epidemic transmission of Oropouche virus in the Amazon Basin of Brazil.

1975-1976: Deputy Commander, United States Amy Medical Research Unit-Belém, Belém, Brazil. Conducted multidisciplinary epidemiological surveillance of malaria and other diseases along the Transamazon Highway in Brazil. Conducted exploratory research on transmission of Oropouche virus.

1974-1975: Chief, Department of Entomology, United States Army Medical Research Unit-Belém, Belém, Brazil. Developed and conducted an integrated field and laboratory program of entomological surveillance along the Transamazon Highway in Brazil; investigated 3 urban epidemics of Oropouche virus; designed and supervised a malaria control program for migrant laborers near Paragominas, Para, Brazil. Also mounted epidemiological studies of malaria in the mirgrant laborers.

1969-1970: Entomologist, U.S. Army Environmental Hygiene Agency, Edgewood Arsenal, MD. Studied influence of insecticides on planktonic algae. Also conducted research on slow-release formulations of insecticides for control of mosquito larvae.

1968-1969: USARSUPTHAI Command Staff Entomologist, 712th Preventive Medicine Unit (SVC) (FLD), Korat, Thailand (Studied separate outbreaks of malaria and Japanese B encephalitis in American personnel).

1967-1968: Entomologist, First US Army Medical Laboratory, Fort Meade, MD. Studied influence of insecticides on survival and development of mosquito pupae.

EDITORIAL ACTIVITES:
I review manuscripts for:

American Journal of Tropical Medicine and Hygiene
Journal of Medical Entomology
Journal of the American Mosquito Control Association
Emerging Infectious Diseases
Bulletin of the Pan America Health Organization
Photogrammetric Engineering and Remote Sensing
Environmental Health Perspectives
The Lancet and other professonal journals.

AWARDS AND HONORS:

Delta Omega Honorary Society in Public Health.

Legion of Merit with Oak Leaf cluster for service in malaria vaccine development, Walter Reed Army Institute of Research and the Uniformed Services University of the Health Sciences.

Legion of Merit for service as Commander, United States Army Medical Research Unit-Belem, APO Miami 34030.

"A" Prefix awarded by the U.S. Army Surgeons General for the highest level of Professional Achievement.

Meritorious Service Medal (with Oak Leaf Cluster) for service as Chief, Department of Entomology, Walter Reed Army Institute of Research, Washington, D.C.

Meritorious Service Medal for service as Chief, Medical Entomology Section, United States Army Medical Research Unit-Brasilia, APO Miami 34040.

Examples of recent grants (up to time of retirement):
"Behavior-modifying compounds for disease vector control"
NIH Partnership Grant, Roberts (PI) 09/08/03-01/03/08
Goal is to develop new chemicals for vector control.

"Environmental determinants of Malaria in Belize, C.A."
NIH/NSF—Rejmankova (PI), UC-Davis 10/01/00-9/30/05
Roberts is PI for USUHS participation. Submitted in response to NIH RFA TW-00-002.

The major goal is to examine the role of anthropogenic change on the ecology of malaria and malaria vectors.

Partial Listing of Other Professional Activities:
1973-1978: Consultant in medical entomology and epidemiology to the World Health Organization, Pan American Health Organization in Brazil (consultant in Medical Entomology and Epidemiology).

1978-1980: Visiting Professor, Medical Entomology, University of Brasilia, Brasilia, D.F., Brazil.

1980-1981: Deputy Manager, Medical Entomology Study Group, USAMRDC.

1981: Chairman, Working Group on Surveillance, Attractants and Repellents. Workshop on Vector Control sponsored by the U.S. Agency for International Development,meeting site was Gainesville, FL.

1982: Consultant in arbovirology, World Health Organization, Pan Health Organization, worked in Bolivia.

1983: Member, Study Group for Vector Control, U.S. Agency for International Development, Washington, D.C.

1980-1984: Reviewer, Medical Entomology Study Group, USAMRDC

1980-1984: WRAIR Representative to the Armed Forces Pest Management Board (AFPMB)

1982-1984: Member, Medical Entomology Committee, Armed Forces Pest Management Board (AFPMB).

1984: Member, Ad Hoc committee for selection of meeting sites; American Society of Tropical Medicine and Hygiene.

1984: Chairman, Study Group for Vector Control, U.S. Agency for International Development.

1984: Consultant to the Minister of Health, Dominican Republic (consultant in malaria control).

1986: Member, Study Group of the U.S. Agency for International Development for Biological Control.

1986: Coordinator, "ELISA Workshop",sponsored by the National Academy of Sciences at USUHS, April 16-18, 1986.

1984-1988: Chairman, Local Arrangements Committee, 1988 Annual meeting of the American Society of Tropical Medicine and Hygiene. This annual meeting earned record profits, over $60,000.

1984-1988: Member, Advisory Council of the International Center for Public Health Research, University of South Carolina.

1989-1989: Member, AIBS review board for AID proposals in malaria vaccine development research

1988: Reviewer, Medical Entomology research proposals submitted to the Pan American Health Organization.

1989: Consultant, Pan American Health Organization, reviewed research program of the Center of Malaria Research, Tapachula, Mexico.

1986-1995: Chairman of "Admission Committee for the Ph.D. Program in Medical Zoology", DPMB, USUHS.

1995-1999 and 2003-present: Chairman, "Admission Committee for the Ph.D. Program in Medical Zoology," DPMB, USUHS.

1995-97: Member of Merit Review Board for the intramural grants program, USUHS.

1997: Chairman, "Ad hoc Committee to Evaluate Core MPH Curriculum," DPMB, USUHS.

1997-98. "Graduate Education Committee," DPMB, USUHS.

1997-99 "Executive Committee," DPMB, USUHS.

1999-2001: Member of the USUHS Committee for Advancement, Promotions and Tenure.

1999-present: Member of Department of Preventive Medicine's Committee for Advancement, Promotions and Tenure.

2002-2003: Member of Department of Military Medical History's Committee for Advancement, Promotions and Tenure.

2002-present: Member, Board of Directors, non-government organization "Africa Fighting Malaria."

2003-2005: Member of external committee for review of military medical research proposals (MIDRPs).

2003: Committee for review of Small Business Innovative Research proposals for the Army Medical Research Material Command.

PEER-REVIEWED PUBLICATIONS

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Roberts, et al. 1980. DDT-an attractant to male euglossine bees in Brazil. An. Acad. Bras. Cienc. 51(1):188.

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PUBLISHED ABSTRACTS AND INVITED PRESENTATIONS

Roberts, DR., J.P. Grieco, and N. Achee. Invited presentation on: Chemical actions of insecticides recommended for indoor residual spraying in malaria control programs. Invited presentation at the American Enterprise Institute before the head of WHO's Global Malaria

Program. Attended by Dr. Kochi, Kamini Mendez, and other WHO and PAHO officials. Presented at 4:20 P.M. on 28 March 2006 at the American Enterprise Institute in Washington, D.C.

Roberts, DR, J. Grieco, N. Achee, K. Chauhan, T. Chareonviriyaphap. 2006. DDT science: Why DDT is still needed for disease control. Mid-Atlantic Mosquito Control Association Meeting on 22 February 2006. York, PA

Roberts, DR. Invited presentation: DDT and Malaria. Presented at the annual meeting of the Virginia Mosquito Control Association in Williamsburg, Virginia on 8-9 February 2006.

Roberts, DR. Invited presentation: Policies to Stop/Prevent Indoor Residual Spraying for Malaria Control. Presented in a symposium at the annual meeting of the American Society of Tropical Medicine and Hygiene in Washington, D.C.. December, 2005.

Roberts, DR. 2005. Invited lecturer on the science of DDT and the continuing need for DDT in malaria control operations. International Conference on Malaria, New Delhi, India. 4-6 November 2005.

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Roberts, DR., 2005. Invited Symposium Presentation: Malaria in the New World. Presented at the Annual Meeting of the Society of Vector Ecology in Reno, Nevada. October 2005.

Roberts, DR. 2005. Invited presentation on "The Scientific Basis for use of DDT and Other Insecticides in Disease Control." Presented at the Annual Meeting of Mexico's Society of Public Health in Cuernavaca, Mexico on 2-4 March 2005. Meeting held on campus of Mexico's National Institute of Health in Cuernavaca, Mexico

Roberts, DR. 2004. Senate Testimony before Senator Brownback's Subcommittee on East Asian and Pacific Affairs on Neglected Diseases in East Asia: Are Public Health Programs Working? October 6, 2004 at 2:30 p.m. in the Dirksen Building, Washington, D.C. 13 pp. (also listed in publications because this testimony was published as part of public record of the senate hearing.)

Roberts, D.R. Invited presentation on Malaria pharmaco-suppression in Central America. Comments presented at an American Enterprise Institute for Public Policy Research conference on "The real obstacles to treating AIDS, malaria and tuberculosis in developing countries."

Conference held Wednesday, May 12, 2004, 9:00 a.m. – noon in the Wohlstetter Conference Center, Twelfth Floor, 1150 Seventeenth Street, N.W., Washington, D.C. 20036

Roberts, D.R. Invited radio talk show interview: The morning talk show "The Bob Rivers Show" on KZOK in Seattle from 10-10:15 on morning of 17 March 2004. Topic was use of DDT for malaria control. Interview also included responses to phone calls from listening audience.

Roberts, D.R. Invited presentation "The Model of Reemerging Malaria in South America." Presented in Symposium I - Malaria Resurgence and Risk Analysis (1:30 PM - 3:00 PM): Moderators:Bill Dees and Bob Wirtz. Annual Meeting of the American Mosquito Control Association in Savannah, Georgia, 24 February 2004.

Roberts, D.R. Developed background for American Broadcasting Corporation "ABC News" program 20/20 entitled "Lies, Myths & Downright Stupidity with John Stossel." My contribution was technical background on the longest segment of the program, on the need for use of DDT in malaria control. Program was aired from 10 to 11 p.m. on January 23, 2004. I worked with Deborah Colloton of ABC News for this program.

Roberts, D.R. Invited presentation "Use of insecticides for control." Presented at the Virginia Mosquito Control Association 2004 Annual Meeting at 8:15-8:45 a.m. February 5, 2004 in Williamsburg, Virginia

Roberts, D.R. Invited presentation for Johns Hopkins Bloomberg School of Public Health entitled "Complex chemical actions for Vector-borne disease control: The long road back." October 23, 2003 in Baltimore, Maryland.

Roberts, D.R., C. Shiff, R. Liroff and Williams (Organizations: USUHS, JHSPH, WWF, WHO, respectively). Debate moderated by_____: DDT for malaria control. May 2003. Sponsored by the Global Health Council. Washington, D.C.

Hshieh, P., and D.R. **Roberts**. Modeling excito-repellent actions of insecticides for malaria control. Presented at the 68th Annual Meeting of the American Mosquito control Association in Denver, Colorado, 16-21 February 2002. Abstract Number 16PS14.

Roberts, D.R. and P. Hshieh. Quantifying the impact of global malaria control strategies. Presented at the 68th Annual Meeting of the American Mosquito control Association in Denver, Colorado, 16-21 February 2002. Abstract Number 22M02.

Roberts, D., Malaria in the Americas: A model of re-emergence. Session on Emerging Pathogens.655th Annual Educational Conference and Exhibition by the National Environmental Health Association at the Hyatt Regency in Atlanta, GA on July 2, 2001.

Roberts, D.R. Foreword: DDT is still needed for malaria control. The foreword to the document "The economic costs of malaria in South Africa by Richard Tren. IEA Publications:24 pp. (internet at : http://www.iea.org.uk/env/malaria.htm)

Roberts, D.R. and P. Masuoka The influence of deforestation on vector-borne diseases. Presented August 21, 2000 in symposium on Environmental Change and Vector-Borne Disease Transmission (Symposium 3). XXI International Congress of Entomology and XVIII Brazilian Congress of Entomology. Meeting at Foz do Iguassu, Brazil, August 20-26, 2000.

Roberts, D.R. DDT for malaria control: Definition of need. Presented at the Annual Meeting of the American Association for the Advancement of Science, in Symposium "International Management of DDT: Understanding the benefits and risks in malaria control". Symposium presented at the Marriott Wardman Park at 3:00 p.m. 21 February 2000.

Roberts, D.R. DDT and Malaria: Science, policies and politics. Presented at the Tropical Medicine Association of Washington meeting at 8:00 P.M. on 13 April 2000 in Building 4 at the National Institutes of Health.

Roberts, D.R. Determinants of malaria in the Americas. Presented at the International Workshop on the Contextual Determinants of Malaria. Workshop held at Lusanne, Switzerland on May 14-18, 2000.

Roberts, D.R. Fundamentals of testing and using residual insecticides for malaria control. Plenary speaker for the British Parasitology Society's Malaria Conference at Imperial College on September 1999.

Roberts, D.R. DDT for malaria control. Invited presentation for the annual meeting of the Virginia Mosquito Control Association in Williamsburg, Virginia. February 1999.

Masuoka, Andre, Montgomery, Rejmankova, **Roberts**, Carbajal, Chamberlin, Lauglin, Ponce Garcia, Watts and Elinan. 1998. Remote sensing and GIS investigations of Bartonellosis in Peru.

Proceedings of the 1998 Geoscience and Remote Sensing Symposium, Seattle, Washington. (July).

Roberts, Lenares, Rejmankova, Alonzo, Paris, Franklin, Pope, Andre, Awerbuch, Laughlin. A conceptual basis for use of remote sensing and GIS in a malaria control program, Belize, C.A. 64th Annual Meeting of the American Mosquito Control Association, Sparks, Nevada. March, 1998.

Andre, Fernandez, Korvis, **Roberts**, Chamberlin, Laughlin, Carbajal, Watts, Ponce Garcia. Identification of bloodmeal sources of *Lutzomyia verrucarum* captured in houses of Peruvian Bartonellosis patients. 64th Annual Meeting of the American Mosquito Control Association, Sparks, Nevada. March, 1998.

Roberts. 1995 Application of RS/GIS to disease surveillance. The First Cyril Ponnamperuam International Symposium on Remote Sensing and Vector-Borne Disease Monitoring and Control, Baltimore, MD. 28-30 November 1995.

Roberts, Rejmankova, Pawley, Paris, Manguin, Polanco and Legters. 1995. Remote sensing as a tool for predicting high risk areas for malaria transmission in Belize. Meeting of the International Astronautical Federation in October 1995 in Oslo, Norway.

Roberts, Sherman and Vanzie. 1995. Abandoning DDT: A burgeoning global malaria control crisis. Annual Meeting of the Amer Soc of Trop Med Hyg, November 17-21, 1995 in San Antonio, Texas.

Roberts, D.R., Manguin, S, Rejmankova, E, Andre, R. 1994 The comparative endophagic host-seeking behaviors of *Anopheles darlingi* and *A. albimanus* mosquitoes in Belize. Soc. Trop. Med. Hyg.

Manguin, S, **Roberts**, D.R., Andre, R, Rejmankova, E, Polanco. 1994 A qualitative and quantitative characterization of *Anopheles darlingi* larval habitats in Belize, C.A. Soc. Trop. Med. Hyg.

Rejmankova, E, **Roberts**, D.R., Pawley, Manguin, S. 1994 Remote sensing as a tool in predicting villages with high or low densities of adult *Anopheles albimanus* mosquitoes in Belize. Soc. Trop. Med. Hyg.

Manguin, S., E.L. Peyton, D. R. **Roberts** & R. Fernandez Loayza. 1993. Population genetics of *Anopheles pseudopunctipennis*, vector of malaria in Central and South America. American Mosquito Control Association, (18-22.IV.93), Fort Myers, Florida.

Roberts, D.R., O. Chan, J. Pecor, E. Rejmankova, S. Manguin, J. Polanco and L. Legters. 1993. Preliminary observations on the changing roles of malaria vectors in southern Belize. Abstract and presentation at the Annual Meeting of the American Mosquito Control Association, Sheraton Harbor Place Hotel, Fort Meyers, Florida. April 18-22, 1993.

Roberts, D.R., S. Manguin, M.H. Rodriguez, E. Rejmankova, M. Spanner, L. Beck. Remote sensing technology and malaria control. Invited Presenter in the Symposium "Future Predictions and Need" of the First International Congress of Vector Ecology held in San Deigo, CA 3-8 October 1993.

Roberts, D.R., J.F. Paris, S. Manguin, R.E. Harbach, R. Woodruff, E. Rejmankova, J. Polanco, L. Legters. The use of remote sensing and landscape features to accurately predict the presence and abundance of two malaria vectors in areas of Belize. Presented at the joint annual meetings of the Am. Soc. Trop. Med. & Hyg and Am. Soc. Parasitol. held at Atlanta, GA. October 1993.

Rejmankova, E., D.R. **Roberts**, H. Savage, M. Rodriguez, M. Rejmanek. Association for tropical Biology, Puerto Rico June 1993.

Rejmankova, E., D.R. **Roberts**, R. Harbach, J. Pecor, E. L. Peyton, R. Krieg, S. Manguin. Phytoecological relationships of malaria vectors in Belize. Presented at the annual meeting of the Amer. Mosquito Control Association, Corpus Christi, TX March 1992.

Rejmankova, E., D. R. **Roberts**, R. Harbach, J. Pecor, R. Krieg, S. Manguin, L. Legters. Predictive classification of malaria vectors in Belize. Paper presented at the INTECOL International Wetland Conference, Columbus, Oh September 1992.

Roberts. 1988. Remote-sensing and malaria prediction program in Mexico. Helminthological Society of Washington, D.C. Meeting, Bethesda, MD. October 12.

Savage, Duncan, **Roberts** and Sholdt. 1988. A dipstick, dot-ELISA, assay for the rapid field identification of mosquito bloodmeal sources. Am. Mosq. Cont. Assoc., Denver, Colorado, February 21-25.

Peters and **Roberts**. 1987. Remote Sensing. Presented at the Preventive Medicine Conference in Garmisch, W. Germany, March 26, 1987.

Roberts. 1987. Remote Sensing and Malariology. Department of Entomology Seminar Series, Walter Reed Army Institute of Research, Washington, D.C.

Roberts. 1987. Applications of remote sensing to the study of Malaria. AID Vector Biology and Control (VBC) Project Seminar Series, VBC Conference Room, Arlington, VA, September 30.

Roberts. Invited speaker to the "II Assemblea General de Asociados Fundacion Mexicana para la Salud" on June 18, 1987 in the Hotel El Presidente Chapultepec, Mexico, D.F. Presentation entitled "Deteccion a distancia de criaderos de mosquitos."

Roberts. Invited speaker to the "Malaria Vector Control" symposium of the 1987 Annual Meeting of the American Mosquito Control Association in Seattle, Washington. Presentation entitled "Malaria Vector Ecology and Contol in South America"

Roberts. Invited speaker to the 27th Annual Meeting of the Louisiana Mosquito Control Association at Lake Charles, LA, 29 Oct 1984. Presentation entitled: "Progress in Developing Field Applicable Assays for Detecting Human Pathogens in Vector Populations".

Roberts. Invited speaker to the Plenary Session of the 1983 Annual Meeting of the American Mosquito Control Association and the 54th Annual Meeting of the Florida AntiMosqito Association in Lake Buena Vista, FL, 28 Feb 1983. Presentation entitled: "U.S. Army Research Programs on Mosquitoes and Mosquito Borne Diseases".

Roberts, Alecrim, Erhardt and Whitlaw. 1980. *Euplusia purpurata.* uma abelha atraida pelo DDT e que remove of insecticida das superficies borrifadas. XVI Congresso da Seciedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #312.

Roberts, Alecrim, Tavares and McNeill. Observacoes sobre o comportamento do vetor *Anopheles darlingi* Root, em uma area endemica para malaria no Amazonas. XVI Congresso da Sociedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #342.

Alecrim, **Roberts**, McNeil, Dourado and Prata. Migracoes da populacao e controle da endemia malarica na regiao endemica do Rio Ituxi. XVI Congresso da Sociedade Brasileira de Medicine Tropical. Natal, 3-8 Feb 1980. Abstract #95.

McNeil, Alecrim, Tavares and **Roberts**. Comportamento da reacao de imunofluerescencia para malaria em amostras de papel de filtro e em soro refrigerado. XVI Congresso da Sociedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #96.

Alecrim, McNeil, Tavares, **Roberts** and Olimpio. Sorologia para malaria nos habitantes do Rio Ituxi-Amazonas. XVI Congresso da Sociedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #97.

Alecrin, Alecrim, **Roberts**, Guerra, Tavares. Indice esplenico e parasitario em uma populacao vivendo no Rio Ituxi-Amazonas. XVI Congresso da Sociedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #337.

McNeill, **Roberts**, Alecrim. Manutencao de uma cepa de *Plasmodium falciparum* (Ituxi084) no sistema de sistema continuo. XVI Congresso da Sociedade Brasileira de Medicina Tropical. Natal, 3-8 Feb 1980. Abstract #343.

Expert Report of Professor Amir Attaran

In the matter of *Garza v. Allied Chemical Corporation, et al,*
Cause No. C-4885-99-F(10), 332nd Judicial District, Hidalgo County, Texas

On behalf of the defendant Montrose Chemical Corporation of California

September 2009

My Qualifications:

I am Associate Professor in the Faculty of Law (Common Law Section) and the Faculty of Medicine (Department of Epidemiology and Community Medicine) at the University of Ottawa, where I currently hold the Canada Research Chair in Law, Population Health and Global Development Policy. My training is in both areas, having received a doctoral degree in the biomedical sciences (at Oxford University) and a degree in law (at the University of British Columbia).

My research is often oriented toward the combination or intersection of the biomedical sciences and the law. Universities increasingly encourage this sort of interdisciplinary scholarship, and the Canada Research Chair that I hold is intended for it. Prior to taking my current position at the University of Ottawa, I held research or teaching positions at both the Kennedy School of Government at Harvard University, the School of Public Health at Yale University, and the Royal Institute of International Affairs (now renamed Chatham House) in London. Global public health policy was a key scholarly interest of mine while in all these positions.

I have made careful studies of the claimed relationships between DDT exposure and human health, and historical and contemporary attitudes toward public policy on DDT. As an indication of my reputation in this field, my views on DDT and public health:

- have been published in some of the leading biomedical journals (e.g., *Nature Medicine*, the *British Medical Journal*);

- have been solicited as a member or invited guest in the committee deliberations of leading science policy organizations (e.g., the World Health Organization, and the Institute of Medicine of the US National Academy of Sciences);

- were adopted by the Government of South Africa in the drafting of a treaty on persistent organic pollutants (the Stockholm Convention), and specifically, the part which deals with the public health exemption;

- have been covered by the world's top press organizations (e.g., the *New York Times*, the *Economist*);

- have contributed to a reevaluation of DDT by the United States Senate, including in Senate committee hearings held in May 2005, where DDT was favorably discussed as among the options for malaria control; and

- have been presented in invited lectures at various universities.

(1) Summary opinion

Consistent with standards of good scientific scholarship, I cite the literature containing the evidence upon which my opinion rests throughout this report.

While I am acutely aware that no concise statement can fully capture the meaning of this report, subject to that caveat, here are my summary opinions as will be further elaborated herein:

1. At the present time, leading public health agencies at the national and international levels (such as DHHS, WHO and IARC) have concluded that the evidence does not support a causal association between DDT exposure and cancer in humans.
2. In contrast, there is abundant and non-controversial evidence that the public health use of DDT has avoided human death and illness from insect-borne diseases such as malaria or typhus. The savings are in the millions of lives historically.
3. Over a course of decades, and across a range of diseases, DDT delivered ascertainable improvements to Texans' health, including in Hidalgo County.
4. Since the 1990s, the use of DDT for public health purposes is experiencing a resurgence, with the support of agencies such as USAID, WHO and the World Bank, and the agreement of leading environmental groups.
5. The EPA did not "ban" DDT, but limited its registrations, principally for political reasons. Further, EPA's reasons did not include any finding that DDT is carcinogenic in humans.

Obviously, my opinion differs from those who (incorrectly) regard DDT as extremely dangerous. My views are shaped by the reality that DDT is unquestionably a life-saving agent: one that has saved millions of people from dying of malaria and other insect-borne diseases. With that reality comes an ethical reason to prepare this report: when in the past critics used faulty scientific reasoning to cast aspersions on DDT, history teaches that DDT was eliminated from malaria control programs in Africa, Asia, Latin America and elsewhere, with the result that many, perhaps millions, of people needlessly died. By letting faulty evidence go unchallenged, DDT could be wrongly sidelined again, with fatal consequences for some of the world's poorest or most vulnerable people.

As a sign that my views are genuinely held and not matters of convenience for this litigation, please note that all my peer-reviewed papers on DDT were published before I was aware of this litigation. I first formed my views on DDT while employed by one Canada's most prominent environmental conservation charities, and I later refined those views while employed by Harvard University, Yale University and the University of Ottawa.

(2) Historical uses of DDT around the world:

The identification of DDT as an effective insecticide was the work of a Swiss chemist, Paul Müller, in the late 1930s, as the Second World War was gathering steam. In the pragmatic mindset of that war, the positive health effects of Müller's discovery were immediately recognized. Insect-transmitted diseases such as typhus and malaria were familiar scourges in earlier wars, and continued to decimate troops and civilians in Southern Europe and the Asian Pacific theatres of the War.

Controlling these diseases became part of the war effort. City-wide programs were established to dust DDT and another insecticide, MYL, in neighborhoods, in homes—and even on the person. Finding a huge typhus epidemic tearing through Naples after its liberation from Nazi occupation, the Allies dusted up to 70,000 people *each day* with DDT or MYL.[1]

In those days, it was uncommon to *limit* peoples' exposure to DDT dust. On the contrary, with a ferocious epidemic underway, the Allies and their Italian counterparts exerted themselves to the fullest to douse as many Neapolitans in DDT as possible. In this sense, Naples makes an interesting counterfactual to the Hayes-Sammons situation, for in Naples the intention was to spray citizens' bodies with DDT, and to keep spraying whenever a case of typhus flared, right up to the 1950s. The fact that the DDT was applied not very judiciously, but directly on one's head, face and body at point-blank range, is readily apparent in photos of the Naples typhus campaign reproduced on next pages.[2]

The Allies' gambit with DDT worked: Naples brought an end to its typhus epidemic, which because of continued DDT operations was not to return.[3] Over 3,000,000 people were dusted (or re-dusted) between 1943-1945, and yet, scientists at the time observed "no evidence of sensitization or intoxication due to the DDT louse powder ... during the Naples control activities."[1] In other words, millions of Italians were doused with DDT at close range and scientists at the time did not observe any harm. Nor does it appear that scientists reported any long-term or delayed harm in later years. To the best of my knowledge, and having conducted a thorough search of the biomedical literature to the present day, there has been no reported epidemic of non-Hodgkin lymphoma (NHL) in the wake of this massive DDT campaign.

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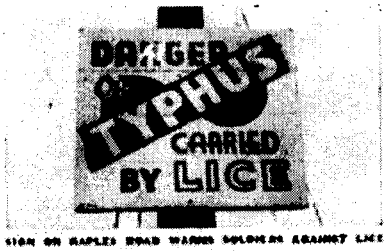
[1] Soper FL, Davis WA, Markham FS, Riehl LA. Typhus fever in Italy, 1943-1945, and its control with louse powder. **Am J Trop Med Hyg** 1947;45:305-334. See also Soper FL, "Report on the Control of Typhus in Naples, Italy, December 9, 1943 to January 2, 1944.", 21 January 1944, available at http://profiles.nlm.nih.gov/VV/B/B/H/W/_/vvbbhw.pdf (accessed 17 September 2009). Generally, MYL was used early in the campaign, and DDT later.

[2] "Typhus in Naples", **Life** (magazine) 28 February 1944: 36-37. A pagewise digital reproduction is available at http://profiles.nlm.nih.gov/VV/B/B/F/Z/ and http://profiles.nlm.nih.gov/VV/B/B/G/B/ (accessed 17 September 2009).

[3] Hill EL, Morlan HB, Utterback BC, Schubert JH. Evaluation of country-wide DDT dusting operations in murine typhus control 1946 through 1949. **Am J Public Health** 1951;41:396-401. Available at http://www.ajph.org/cgi/reprint/41/4/396 (accessed 25 January 2009).



SIGN ON NAPLES ROAD WARNS SOLDIERS AGAINST LICE

TYPHUS IN NAPLES

AMG medical officers control an epidemic with tons of delousing powder and a preventive vaccine

When the Germans evacuated from Naples they dynamited the city's water system. Without water the Neapolitans could not wash their clothes or their bodies. As they became progressively more dirty they became better and better hosts for body lice which carry typhus fever. Somehow a few of these lice got a chance to bite a person suffering from typhus and thus picked up the disease. In the crowded slums of the half-homeless city these typhus-carrying lice crawled from one person to another spreading the infection as they went. When the Americans marched in last October they found an epidemic getting under way. But

Italian baby gets dusting while family undergoes examination. Each evening Italians, like the man at the left, undress themselves into squads of dozens. Paid 80 lire per night and supervised by U.S. Army doctors, they scatter all through the city, dusting everyone they can find.

U. S. Army doctor, Major C. M. Wheeler of Berkeley, Calif., pats over a little girl who looks as if she were going to cry. Major Wheeler is head of the Contact Delousing Service which, with Italian help, has the job of getting servant and dusting people in their homes and shelters.

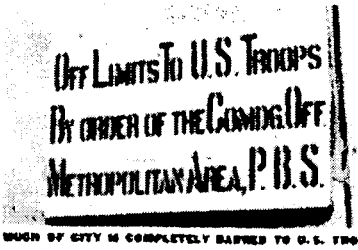
Cement mixer is used to mix the insecticide powder with Italian talc. This talc serves to prevent the powder from irritating the skin. The mixer holds half a ton of insecticide and talc at one loading and each 60-lb. bag (center) contains enough powder to delouse about 600 people.

Rockefeller Foundation worker examines the hair of a little girl. Head lice lay eggs which can be seen sticking to the individual hairs. Typhus is one of the great epidemic diseases of the world. In 1918 typhus spread through Russia, infecting 25,000,000 and killing some 3,000,000.

after four and a half months medical officers of the AMG last week seemed to be getting it under control.

Knowing that most of the population, after months of German oppression, had neither clean clothes nor clean bodies, U. S. doctors fought the epidemic by delousing 30,000 people a day with a new powder insecticide called DDT (dichlorodiphenyltrichloroethane). As a further preventive, Naples policemen and firemen were inoculated with an effective new vaccine. This same vaccine, developed by Bacteriologist Herald Rea Cox while he was at the Rocky Mountain Laboratory in Hamilton, Mont., has kept the typhus death rate of the U. S. troops stationed in the Naples area at zero.

Once a person develops typhus there is not much, aside from good nursing, that can be done for him. The incubation period lasts from eight to 12 days and the first symptoms are backache or headache and perhaps chills. By the third or fourth day the fever rises to 103° or 104°, a bumpy red rash breaks out and usually a muttering delirium sets in. Sometimes victims have terrifying, suicidal hallucinations. If infection has not become too extensive and there are no complications like bronchitis or pneumonia, fever drops abruptly after about two weeks and patient begins to recover.



MUCH OF CITY IS COMPLETELY BARRED TO U. S. TROOPS

Woman finishes as an Italian doctor gives her a blast of the powder. Most of the Italians like it, appreciating their delousing. This woman, who lives in a filthy tunnel under a road, is typical of the hundreds of people who were made homeless and miserable by the German occupation.

Squirt in the hair is added precaution. Typhus is generally carried by the body louse which never crawls above neck, but occasionally head lice become infected and transmit the disease. Body lice are the same as the cooties which tortured Americans in trenches of World War I.

Sky hits powder through the hair of his small brother. Naples has had to delouse everyone going to and coming from city to prevent spread of epidemic. Typhus, like Rocky Mountain fever, is caused by Rickettsia, a microorganism larger than a virus but smaller than bacteria.

Policemen are vaccinated by Captain Maxwell Reed of Immunization Service. Vaccine is made by infecting chick embryos and allowing Rickettsia to multiply. The infected matter is then diluted. Vaccination produces little reaction but must be repeated after about six months.

The success achieved with DDT against typhus naturally encouraged the thought that it could be useful against other insect-borne diseases. Of these, malaria was—and regrettably still is—the most significant as measured by the death or disability it causes.

In 1955, the Assembly of the World Health Organization passed a resolution committing to a global campaign of malaria eradication.[4] DDT was already the anti-mosquito insecticide of choice, and together with a highly effective malaria medicine (chloroquine), these constituted the keystones of the WHO's campaign from 1955 to (approximately) the early 1970s.

In WHO's standard technique, known as indoor residual spraying (IRS), DDT was applied once or twice yearly to the interior walls and eaves of family dwellings, at a dose of 1-2 grams per square meter.[5] The DDT so applied remains for 6-12 months as a highly effective mosquito-killing and mosquito-repelling residue. Since most species of malaria mosquitoes feed on their victims in the middle of the night as they sleep indoors, the indoor residual spraying technique could achieve a great reduction in cases of malaria.

Although the WHO campaign fell slightly short of the goal of total malaria eradication, the tactics it embodied achieved stunning results nonetheless and saved a vast number of people from the disease. After the introduction of DDT in 1945, WHO reports that transmission of malaria in Sri Lanka (then called Ceylon) was reduced from 2.8 million cases and 7,300 deaths, to 17 cases and zero deaths.[6,7] Similarly, a report from India commissioned for WHO reads that "the achievement in malaria control by DDT spraying was so spectacular that by 1965 malaria was wiped out in 373 million population ... [and] the incidence of malaria was drastically reduced from ... 75 million cases annually ... to about 0.1 million."[8] Succinctly put, in India and Sri Lanka, the malaria case rate fell by over 99%—an achievement which has not been matched by any technology since.

But a more dramatic achievement occurred in the southern United States, which totally eradicated malaria. Various social and sanitary measures such as swamp drainage or improved housing succeeded in lowering malaria cases from an estimated 600,000 cases in 1914 to 125,556 cases in 1934, but malaria remained a stubborn problem.[9] It was not until

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[4] The original resolution of the Assembly is reproduced with further explanatory text in document WHO/Mal/162 of 1 February 1956, available at http://whqlibdoc.who.int/malaria/WHO_Mal_162.pdf (accessed 17 September 2009).

[5] To visualize how much DDT residue is used, imagine approximately a sugarcube of DDT, sprayed on a wall area equal in size to the front of a typical refrigerator door. That is, roughly speaking, about 2 grams per square meter. World Health Organization. Frequently asked questions on DDT use for disease vector control. Document WHO/HTM/RBM/2004.54 rev. 1 (2005). Available at: http://www.who.int/malaria/docs/FAQonDDT.pdf (accessed 17 September 2009).

[6] World Health Organization. Document SDE/PHE/DP/02 (1999).

[7] Pinikahana J, Dixon RA. Trends in malaria morbidity and mortality in Sri Lanka. **Indian J Malariol** 1993;51-5. Full text not available, however the abstract is available on PubMed at: http://www.ncbi.nlm.nih.gov/pubmed/8405594 (accessed 17 September 2009).

[8] World Health Organization. Document SDE/PHE/DP/04 (1999). Note some spelling errors have been corrected in this passage.

[9] When one speaks of eradicating an infectious disease, that means, as a public health concept, that there is no more stable transmission of the disease. There is today no stable transmission of malaria in the USA, although there are occasional cases of imported malaria, as by traveling persons or parasite-positive mosquitoes. The

the 1950s that, thanks largely to DDT, malaria was at last totally eradicated as a native disease of the southern United States—a major public health success.

(3) The use of DDT in Texas:

Texas has been a prime user—and Texans are beneficiaries—of DDT.

Starting after World War II, and with the success of controlling epidemics abroad ringing in their ears, the US Public Health Service, Communicable Diseases Center (USPHS-CDC), established anti-malaria and anti-typhus campaigns across the American South. Those campaigns normally involved overlapping efforts of federal, state and local level agencies, whose incomplete traces are spread across various archives today. This reality makes it difficult, if not impossible, to pinpoint exactly how much DDT was used for public health purposes. What I present here is at best a highly incomplete catalogue, which nevertheless makes clear that DDT was used in substantial quantities across the South for health protection—including in Texas.

Here is a tabular summary of the extent of DDT residual spraying in Texas for malaria control, as reported in contemporary documents of the USPHS-CDC:

Interval	Houses or Applications[10]	DDT (pounds)
1946 (Feb 23 – June 30 only)[11]	46,184	26,784
FY 1947[12]	101,350	91,215
FY 1948[13]	96,056	116,281
FY 1949[14]	71,870	72,225
FY 1950[15]	85,355	75,770
FY 1951[16]	39,789	31,014
FY 1952[17]	23,885	11,914

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imported cases typically are a few dozen annually, and in the rare instance they re-establish community transmission, it is always short-lived and sputters out. *See* Zucker JR. Changing Patterns of Autochthonous Malaria Transmission in the United States: A Review of Recent Outbreaks. **Emerg Infect Dis** 1996;2:37-43. Available at ftp://ftp.cdc.gov/pub/EID/vol2no1/adobe/zucker.pdf (accessed 17 September 2009).

[10] Some of the original data reflected in the second column refer to the "number of houses sprayed," whereas other data refer to "number of house spray applications" or some similar term. There is no complete explanation found in the cited records for why the change in terminology occurred and whether it is meaningful as to a change in the statistical method. Regardless, it does not affect the conclusion for present purposes that a large number of houses in Texas were sprayed with DDT.

[11] USPHS. *Bulletin Communicable Disease Center: July, August, September 1946* (Federal Security Agency, USPHS; Atlanta), p. 13.

[12] USPHS. *CDC Activities* 1946-1947 (Federal Security Agency, USPHS; Atlanta), p. 6. Note that the calculation of pounds is subject to a footnote in the report which reads "second half of fiscal year only." In this and later footnotes in this section, note that the quantity of DDT may be approximated, as the total weight was not always reported by USPHS-CDC directly. A credible approximation can be made by multiplying the reported number of premises dusted with DDT by the reported average weight consumed per premises.

[13] USPHS. *CDC Activities 1947-1948* (Federal Security Agency, USPHS; Atlanta), p. 5.

[14] USPHS. *CDC Activities 1948-1949* (Federal Security Agency, USPHS; Atlanta), p. 76.

[15] USPHS. *CDC Activities 1949-1950* (Federal Security Agency, USPHS; Atlanta), p. 4.

[16] USPHS. *CDC Activities 1950-1951* (Federal Security Agency, USPHS; Atlanta), p. 47.

It can be seen from these data that tens of thousands of houses in Texas were sprayed with up to 58 tons of DDT annually for malaria control. Additional amounts of DDT were used for general mosquito larviciding, which while not exclusively directed to malaria mosquitoes would have had some beneficial impact: *e.g.* in FY 1948, 7,931 gallons of DDT oil emulsion were used in Texas for controlling malaria mosquito larvae.[18]

The DDT house spray operations were found in most parts of the state, including Hidalgo County. For instance, the USPHS-CDC reports that from July 1947 to June 1948, 3483 pounds of DDT were consumed to spray 3249 premises in Hidalgo County.[19] Hidalgo County is also shown in a map of USPHC-CDC malaria spray operations for the previous year (1946-1947).[20] A contemporaneous memo of the Texas State Health Officer notes that early in the history of the state's DDT use (winter 1946), "plans were formulated for presentation to the Commissioner's Court of each county calling for local participation, and agreements were signed."[21] Other memos by this same official note that DDT spraying, and also DDT larviciding, took place across Hidalgo County, including in Mission.[22]

Even larger quantities of DDT were used for typhus control in Texas. From 1941 to 1945, Texas had more cases of this potentially deadly disease than any other state.[23] When the effort to combat typhus reached full swing between FY 1948 to FY 1950, about 400,000 pounds—or 200 tons—of DDT were dusted in premises in Texas.[24] Again, contemporaneous maps published by USPHS-CDC confirm that these typhus control operations took place in Hidalgo County.[25]

Hidalgo County also resorted to DDT for other public health uses, aside from the customary malaria and typhus control uses. Starting in 1945, a scientific trial in Hidalgo County sought to reduce the highly endemic problem of dysentery (a severe, potentially life-threatening form of diarrhea) by spraying DDT to control fly populations.[26] The USPHS-CDC credits that trial with "a decreased dysentery rate" in those locales of Hidalgo County where DDT was used.[27] This trial appears to have achieved a marked reduction on the prevalence of *Shigella* infection in Hidalgo County while DDT spraying was underway.[28]

Texas also used DDT to combat mosquito-borne viral encephalitis. Such epidemics occurred from time to time in Hidalgo County and are always dangerous: e.g. Eastern equine

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[17] USPHS. *CDC Activities 1951-1952* (Federal Security Agency, USPHS; Atlanta), p. 28.
[18] USPHS. *CDC Activities 1947-1948* (Federal Security Agency, USPHS; Atlanta), p. 6.
[19] Ibid., pp. 128-30.
[20] See *CDC Activities* 1946-1947, particularly the map on p. 7, cited above at footnote 12.
[21] Undated memo of Geo. W. Cox, M.D., State Health Officer. From the context, it appears the memo is probably from 1947.
[22] Undated memo of Geo. W. Cox, M.D., State Health Officer. From the context, it appears the memo is from sometime after 1946.
[23] USPHS. *CDC Bulletin,* January 1950, p. 8.
[24] This figure is approximated from data in the reports cited above at footnotes 13, 14 and 15. As to the method of approximation used, see footnote 12 above.
[25] See *CDC Activities* 1946-1947, particularly the maps on pp. 7 and 11, cited above at footnote 12.
[26] Ibid., pp. 23-25
[27] Ibid., p. 23.
[28] See *CDC Activities* 1950-1951, particularly the graph on p. 19, cited above at footnote 16.

encephalitis kills about a third of its victims, and can brain damage survivors. A serious epidemic occurred in Hidalgo County in 1954, which led the Governor of Texas to declare a state of emergency there.[29] Both DDT spraying and larviciding were used in that epidemic.

In the 1960s, Texas also was part of a wider international program encouraged by the Pan American Health Organization (PAHO) to eradicate the *Aedes aegypti* mosquito, which is particularly dangerous because it can transmit yellow fever. While countries such as Brazil had eradicated *Aedes aegypti* in the 1950s, the mosquito remained in Texas. The USPHS-CDC strategy for *Aedes aegypti* eradication called for DDT spraying, which was successful in short order.[30] Two maps published by USPHS-CDC tell the story: the first map shows that Hidalgo County was "infested" in 1964, but just two years later, the southern tip of Texas is "uninfested or presumed uninfested."[31] The USPHS-CDC *Aedes aegypti* eradication branch records that 26,985 gallons of DDT concentrate were supplied to Texas in 1964, in anticipation of such operations.[32]

In sum, even with the often incomplete archival records available today, it is possible to draw this conclusion: over a course of decades, and across a range of diseases, DDT delivered ascertainable improvements to Texans' health, including in Hidalgo County. Of greatest significance, malaria was eradicated, and typhus is now rare. DDT brought about these health benefits, and was transformational.

(4) DDT is of overall benefit to human health:

Some critics of DDT may be tempted to view these decades-old results and brush them aside, by a sort of "that was then; this is now" reasoning. Doing so is equally unscientific as it is factually incorrect. The consensus of contemporary scientific thought is that, correctly used, DDT remains a beneficial and safe intervention against vector-borne disease.

The World Health Organization, which is the leading health agency in the United Nations system, and which counts the USA as a member, currently supports DDT as a safe and effective public health intervention. In 2005, WHO wrote:

> *"WHO recommends indoor residual spraying of DDT for malaria vector control."*[33]

In September 2006, WHO reassessed the scientific evidence on DDT, and amplified its recommendation, after finding fault with its earlier assessments that caused DDT use to decline. As WHO wrote of its 2006 reassessment:

> *"WHO actively promoted indoor residual spraying for malaria control until the early 1980s when increased health and environmental concerns surrounding DDT caused*

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[29] Beadle LD, Menzies GC, Hayes GR, Von Zuben FJ and Eads RB. Vector evaluation and Control. **Public Health Reports** 1957;72:531-5.

[30] USPHS-CDC (1964). *Aedes aegypti Eradication Program Manual of Operations, Part I*, p. 34.

[31] For the 1964 map, see Ibid., and the map therein entitled "*Distribution of Aedes aegypti.*" For the 1966 map, see USPHS-CDC (1966). *The Aedes aegypti Eradication Program*, p. 3.

[32] USPHS-CDC (undated). *Aedes aegypti Eradication Branch Annual Report, F.Y. 1964.*

[33] See WHO, Frequently asked questions on DDT use for disease vector control, cited above at footnote 5.

the organization to stop promoting its use and to focus instead on other means of prevention. Extensive research and testing has since demonstrated that well-managed indoor residual spraying programmes using DDT pose no harm to wildlife or to humans."[34]

It should be emphasized that by giving approval to DDT indoor residual spraying, WHO today sanctions the practice where DDT is intentionally and directly sprayed on the interior walls of homes, into the spaces where people live. The quantity of DDT that is used (recall it is 1-2 grams per square meter) leaves a visible crust of DDT on the walls. With time, the DDT crumbles off the walls, and some is inhaled or ingested by the homes' inhabitants. Further, this exposure pattern is repeated indefinitely, since the standard practice is to respray homes once or twice yearly. All this is consistent with WHO's current guidelines.

Other global authorities have followed WHO's lead in this regard. For example, in 2006 the World Bank states:

"...[T]here has been no scientific evidence that indoor spraying with DDT for malaria has resulted in negative health or environmental consequences. So the Bank's position is very pragmatic on its use. We will use insecticide, and that includes but is not limited to DDT. There is no room for dogma on this issue..."[35]

The United States Agency for International Development (USAID), which is the main implementing agency for the President's Malaria Initiative (under both President Bush and President Obama) currently says this about DDT:

"USAID supports indoor residual spraying (IRS) with DDT as an effective malaria prevention strategy in tropical Africa in those specific situations where it is judged to be the best insecticide for IRS both epidemiologically and entomologically and based on host-country policy..."[36]

"If used correctly for [indoor residual spraying], it poses no known risk to human health..."[37]

The world's leading foreign aid donors such as the Global Fund to Fight AIDS, TB and Malaria, the World Bank, and USAID now provide (or are preparing to provide) funding to several developing countries to use DDT for malaria control. While there appears to be no fully comprehensive list, the scientific literature discloses that Mozambique, South Africa, Swaziland, Eritrea, Ethiopia, Madagascar and India are some of the countries which have

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[34] World Health Organization. "WHO gives indoor use of DDT a clean bill of health for controlling malaria". Press release dated 15 Septmeber 2006. Available at http://www.who.int/mediacentre/news/releases/2006/pr50/en/index.html (accessed 17 September 2009).

[35] Interview with World Bank malaria spokesman, Suprotik Basu, 25 April 2006. Available at http://discuss.worldbank.org/content/interview/detail/3870/ (accessed 17 September 2009).

[36] USAID. USAID and Malaria. Available at http://www.usaid.gov/our_work/global_health/id/malaria/news/afrmal_ddt.html (accessed 17 September 2009).

[37] USAID. Indoor Residual Spraying (IRS). Available at http://www.usaid.gov/our_work/global_health/id/malaria/techareas/irs.html (accessed 17 September 2009).

used DDT in the last decade.[38] [39] The United Nations Environment Programme currently lists 15 countries using or reserving the right to use DDT for disease control.[40]

The widespread recognition by leading development or health institutions that it can be used without harm to humans invites this question: What is correct to think about the DDT exposure of the persons whose homes are sprayed in malaria control operations, relative to the DDT exposure of persons such as Ms. Garza?

In my opinion, it is very unlikely that Ms. Garza received a greater dose of DDT than the many millions of people whose homes were regularly sprayed with DDT for malaria control. Recall that in the WHO-sanctioned technique, DDT is sprayed actively and directly into one's home at frequencies sufficient to keep a constant residue on the walls—and this implies an exposure greater than what one might receive passively through the environment in the neighborhood or community of a pesticide plant. As WHO finds "no harm to ... humans" occasioned by DDT in the indoor residual spraying situation, one would not expect harm to humans in the neighborhood or community situation.

Further, and to the best of my knowledge after having conducted a thorough search of the biomedical literature to the present day, there is no reported epidemic of non-Hodgkin lymphoma (NHL) in the wake of DDT indoor residual spraying for malaria, anywhere in the world. The head of India's national malaria control program, writing for WHO, has made a similar observation:

> *"This insecticide [DDT] has been used extensively in the country for over 40 years in Public Health, especially for the control of vectors of malaria, but there has not been a single instance of its acute or chronic toxicity hazard on human being[s] or domestic pets & cattle, reported from any corner of the country."*[41]

This view—that DDT had public health benefits inuring to millions, and risks observed in precisely no one—prevailed worldwide, including in the United States. Within living memory, it was not uncommon for great leaders and persons of stature to consider DDT a good thing (see some of their quotes collected in Appendix One). Then the tide began to turn: environmental campaigners in the 1960s, notably Rachel Carson, lobbied vigorously to shift public opinion against DDT and to cancel DDT's product registration. The campaigners too often forgot the very substantial good that DDT had done for less fortunate multitudes living

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[38] Sadasivaiah S, Tozan Y, Breman JG. Dichlorodiphenyltrichloroethane (DDT) for Indoor Residual Spraying in Africa: How Can It Be Used for Malaria Control? **Am. J. Trop. Med. Hyg.** 2007;77(suppl 6):249-263, available at http://www.ajtmh.org/cgi/reprint/77/6_Suppl/249 (accessed 25 January 2009).

[39] Sharma SN, Shukla RP, Raghavendra K, Subbarao SK. Impact of DDT spraying on malaria transmission in Bareilly District, Uttar Pradesh, India. **J Vector Borne Dis.** 2005;42:54-60, available at http://www.mrcindia.org/journal/issues/422054.pdf (accessed 25 January 2009).

[40] An undated list of these countries is found at http://www.pops.int/documents/registers/ddt.htm (accessed 17 September 2009). The countries using or reserving the right to use DDT are Botswana, PR China, Ethiopia, India, Madagascar, Marshall Islands, Mauritius, Morocco, Mozambique, Myanmar, Senegal, South Africa, Swaziland, Uganda, and Yemen.

[41] World Health Organization. Document SDE/PHE/DP/04 (1999).

at risk of vector-borne diseases—including people in Texas. As the US National Academy of Sciences estimated in 1970:

> *"To only a few chemicals does man owe as great a debt as DDT. In little more than two decades DDT has prevented 500 million human deaths due to malaria that would have otherwise have been inevitable."*[42]

Of course, one may quibble with the National Academy of Sciences estimate for being only that—an estimate, which by definition lacks exact precision. But even so, I have never heard the following fact challenged or contested, even by DDT's most ardent opponents: DDT has saved a very large number—indeed, millions—of lives from typhus, malaria and other causes. Having once been a prime producer of DDT for agencies such as the US Public Health Service, WHO and UNICEF, and for countries such as Brazil and India, Montrose Chemical Corporation of California's products contributed to this humanitarian success to some extent.[43]

DDT continues to save lives in countries such as South Africa, which experienced a very serious resurgence of malaria in the 1990s when, because of environmentalists' pressure, it forsook DDT for other insecticides (the rise in malaria cases and deaths forced South Africa's return to DDT in 2000).[44] Having traveled full circle from DDT use, to non-use, and back to DDT use again, the public perception in South Africa about DDT has advanced to such an extent that the country's former president is seen publicly toting a spray can (see Figures 1 and 2).

The South African history with DDT is so compelling that even environmental campaigners have had pause to reconsider and to consider DDT's health-protecting virtues. As a World Wildlife Fund spokesman on toxins stated to the *New York Times* in 2005:

> *"South Africa was right to use DDT... If the alternatives to DDT aren't working, as they weren't in South Africa, geez, you've got to use it. In South Africa it prevented tens of thousands of malaria cases and saved lots of lives."*[45]

Environmental Defense, the organization which led the successful campaign to have DDT "banned" (a misnomer; more on that later in this report) from use in the United States, also has come to the conclusion that DDT has redeeming features. When in 2004 the US Agency for International Development was weighing the resumption of DDT use, Environmental Defense wrote:

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[42] National Academy of Sciences, Committee on Research in the Life Sciences of the Committee on Science and Public Policy. *The Life Sciences*. Washington, DC, p 432 (1970).

[43] Deposition of Samuel Rotrosen (former Montrose President), 10 February 2005, pp 25-26; 11 February 2005, pp. 260-262.

[44] Muheki C, McIntyre D, Barnes KI. Artemisinin-based combination therapy reduces expenditure on malaria treatment in KwaZulu Natal, South Africa. **Trop Med Int Health** 2004;9:959-66, available at http://www3.interscience.wiley.com/cgi-bin/fulltext/118806574/PDFSTART (accessed 17 September 2009).

[45] Nicholas D. Kristof, "It's Time to Spray DDT,", *New York Times* (8 January 2005). Available at http://www.nytimes.com/2005/01/08/opinion/8kristof.html (accessed 17 September 2009).

> *"While Environmental Defense sees absolutely no justification for re-introducing use of DDT in the US, we believe that indoor spraying of small quantities of DDT in developing countries areas [sic] where malaria is spread by indoor-dwelling mosquitoes is an important tool given the limited alternatives now available."*[46]

Elsewhere, WHO mentions the Sierra Club and Endangered Wildlife Trust as other environmental groups that have rethought the matter and that now endorse DDT for malaria control.[47]

Thus on a comprehensive view of the scientific developments and the evolution of public perception, the decision to award Paul Müller, who discovered DDT's remarkable insecticidal properties, with the Nobel Prize in medicine in 1948 can be said to have withstood the test of time.

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[46] Letter from John M. Balbus, Director, Health Program, Environmental Defense, to E. Anne Peterson, Assistant Administrator for Global Health, US Agency for International Development, May 11, 2004, available at http://www.edf.org/documents/5046_DDT-letterUSAID.pdf (accessed 19 September 2009).

[47] World Health Organization. "WHO gives indoor use of DDT a clean bill of health for controlling malaria". Press release dated 15 Septmeber 2006. Available at http://www.who.int/mediacentre/news/releases/2006/pr50/en/index.html (accessed 17 September 2009).

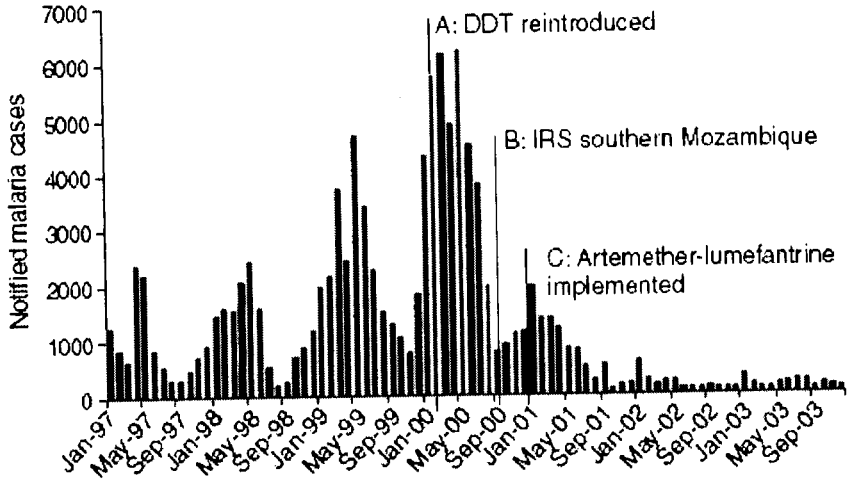

Figure 1 Number of notified malaria cases in KwaZulu-Natal by month in relation to timing of significant malaria control interventions. Source: South African National Department of Health Notification Data.

Figure 1: Time-trend observation of malaria cases in KwaZulu-Natal, the largest malaria-endemic province of South Africa. Following the failure of pyrethroid-class insecticides to quell seasonal outbreaks of malaria in the late 1990s, the authorities reverted to the use of DDT indoor residual spraying in March 2000 (point "A"). That measure, plus other insecticidal measures in neighbouring Mozambique (point "B") and the introduction of a new malaria medicine (point "C"), stopped further epidemics almost totally. As the researchers in this area write, "the reintroduction of DDT is considered to have contributed substantially to the recent decline in malaria in this province."

(Graph reproduced from Muheki C et al, cited at footnote 44.)

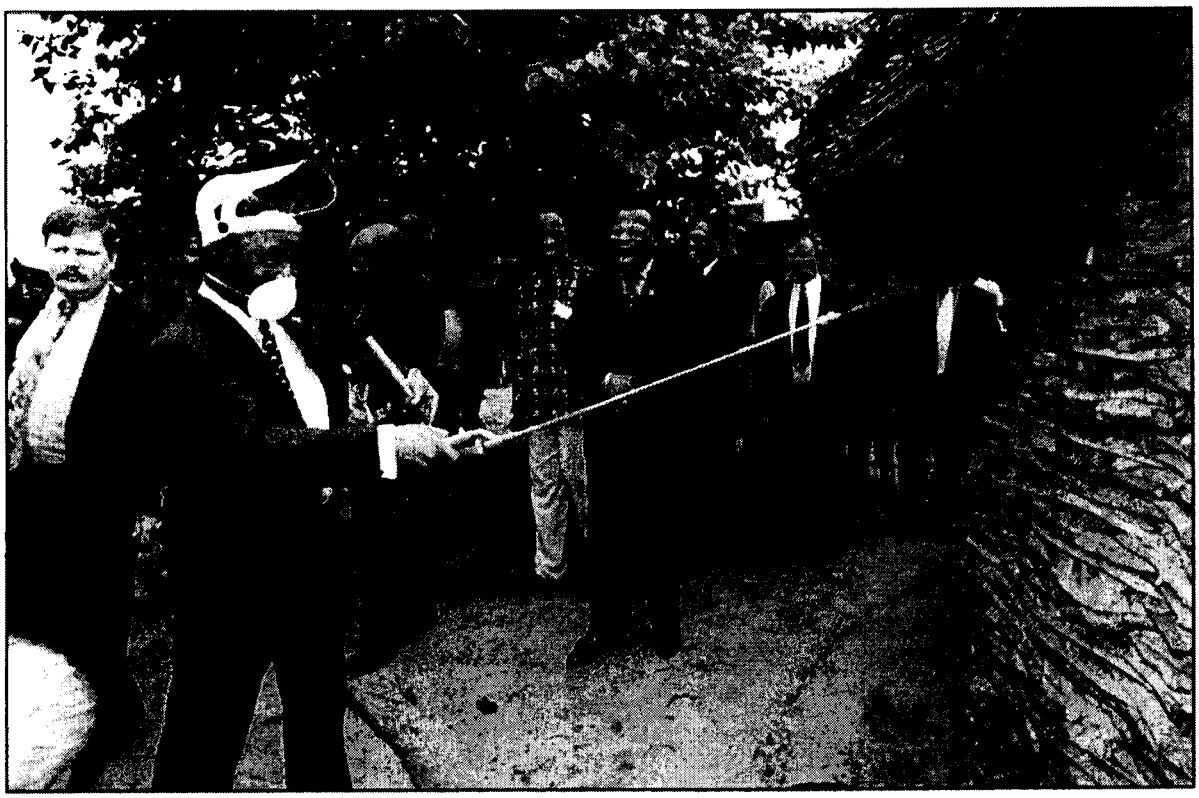

Figure 2: President Thabo Mbeki of South Africa, shown spraying a house. (But note the incorrect method, better suited to a politician's photo opportunity than the WHO technique of malaria control: he is spraying the outside, not the inside, of the house.)

(5) Leading health agencies do not link DDT to cancer in humans:

As alluded to in the previous sections, there is a misperception that DDT causes human cancers. Yet top health protection agencies in the USA and internationally have unambiguously stated that the available scientific evidence does not support the conclusion that DDT causes cancer in humans. I excerpt some of their statements in this section.

The World Health Organization wrote in 2004:

> *"[T]here is currently no direct link between DDT and any negative human health effect."*[48]

To say DDT has no direct link to "any" negative human health effect is, of course, also to mean that there is also no direct link between DDT and human cancers—including NHL. WHO's pronouncement in 2004 updates an earlier (1979), more in-depth expert consensus published by WHO and the United Nations Environment Programme, which similarly read that "there is no evidence that DDT is carcinogenic in man."[49]

The US Department of Health and Human Services (DHHS) is the leading federal government agency with responsibility for the health of Americans. DHHS basically agrees with WHO, though is more precise in its use of language. DHHS's Agency for Toxic Substances and Disease Registry carried out an evaluation of DDT in 2002, which reads:

> *"Studies have monitored human tissue and blood for DDT and its metabolites, but no correlation has been made between the levels found in these tissues and specific disease states."*[50]

In plain language, this statement means that DHHS has reviewed the available studies, and finds that people whose bodies absorbed more or less DDT from the environment are not, respectively, more or less likely to have disease. There is, as DHHS puts it, "no correlation."

The International Agency for Research on Cancer (IARC), a specialized branch of the WHO, also has done an evaluation of DDT. The IARC assessment of DDT, last updated online in 1997 (IARC has a policy of updating its assessments when needed, but apparently has found no need for DDT) states bluntly: "there is *inadequate evidence* in humans for the carcinogenicity of DDT."[51] In turn, IARC defines "inadequate evidence" to mean that:

> *"The available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer, or no data on cancer in humans are available."*[52]

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[48] See WHO, Frequently asked questions on DDT use for disease vector control, cited above at footnote 5.
[49] World Health Organization. DDT and its Derivatives. Environmental Health Criteria series volume 9. Geneva, 1979. Available at http://www.inchem.org/documents/ehc/ehc/ehc83.htm (accessed 17 September 2009).
[50] DHHS Agency for Toxic Substances and Disease Registry (2002). *2002 Toxicological Profile for DDT, DDE, and DDD*, page 206. Available at http://www.atsdr.cdc.gov/toxprofiles/tp35.pdf (accessed 17 September 2009).
[51] IARC. "DDT and Associated Compounds," available at http://www.inchem.org/documents/iarc/vol53/04-ddt.html (accessed 17 September 2009).
[52] See http://www.inchem.org/documents/iarc/monoeval/eval.html (accessed 17 September 2009).

Based on this assessment, IARC categorizes DDT as a Group 2B substance, or in plain terms, a substance that is no more than "possibly carcinogenic to humans"—a fairly low category, and well below the category of substances that are outright "carcinogenic to humans".[53] The "possibly carcinogenic" category contains numerous other industrial chemicals, prescription medicines, and foods such as coffee or pickled vegetables.[54] In IARC's assessment, all of these are possibly able to cause cancer in humans.

(6) DDT and the US Government – Regulatory History and Current Usages:

The foregoing sections invite the question of why DDT, being so effective in controlling insect-borne epidemic diseases and apparently not harming human health, had most of its registrations cancelled (sometimes called "deregistration") in the United States during or shortly after 1972.[55]

It is not accurate to state that DDT was "banned" in the United States; this is a misnomer which incorrectly implies that the US government acted to forbid all instances of DDT use. A more historically accurate statement is that US government decisions cancelled most uses of DDT, so as to narrow but not to wholly eliminate the remaining approved uses. Later, a number of approved uses were voluntarily cancelled, not as a result of US government action. Most recently, the US government (through USAID) finances the use of DDT overseas, in countries where malaria still exists.

According to the EPA's official history of events, a number of administrative actions between 1967 and 1972 resulted in canceling most household and agricultural uses in the USA.[56] Certain emergency or military uses of DDT for public health and agricultural purposes remained, and EPA approved applications for those on a case-by-case basis after 1972. Cited below are some cases documented in the *Federal Register*, where dispensation to use DDT was granted on the dates shown:

- March 19, 1974 – For the pea leaf weevil in Washington and Idaho[57]
- March 5, 1974 – For the Douglas-fir tussock moth in Oregon, Idaho and Washington.[58]
- June 8, 1976 – For fleas transmitting the plague in California.[59]

Thus DDT continued to be used in the US on an as-needed basis, for crop or forest pests or disease control, well after the 1972 decision cancelling certain DDT usages. Texas benefitted

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[53] See http://monographs.iarc.fr/ENG/Classification/crthgr02b.php (accessed 17 September 2009).
[54] Ibid.
[55] The administrative decision which cancelled most of DDT's uses was issued in 1972, but because of its terms, and subsequent appeals, it did not take effect immediately, and perhaps not until 1973. For ease of reference I adopt the convention of citing the earlier date, and those in need of a more precise timeline will find it in footnote 56, below.
[56] EPA. DDT Regulatory History: a brief survey (to 1975). Available at: http://www.epa.gov/history/topics/ddt/02.htm (accessed 17 September 2009).
[57] Page 10322.
[58] Page 8377.
[59] Page 22979.

from these uses in later years, and appears to have used DDT as late as 1980.[60] Such as-needed uses of DDT came to an end after October 1989, when all the remaining registrations of DDT were voluntarily cancelled by reason of a failure to pay maintenance fees.[61]

However, even as DDT experiences disuse in the US, it still is not banned. The Commission for Environmental Cooperation, an intergovernmental organization of the NAFTA countries (the US is a member), writes that "the United States does not have the legislative authority to prohibit production of DDT if a manufacturer wanted to initiate such production in the future"[62] The EPA Administrator retains the discretion to allow the use of DDT, either by way of a hearing to reverse DDT's cancellation, or on an emergency basis without a hearing.[63] The EPA has a duty to act expeditiously on applications claiming an emergency,[64] and can in the case of public health, stipulate an exemption of up to a year allowing DDT to be used.[65] In cases of true crisis, the option still exists in law for a federal or state branch of government to use DDT immediately on that basis, with notification to EPA later.[66] As already mentioned, Texas made use of DDT for public health purposes, even after the EPA's cancellation of general agricultural uses.

Fortunately, today there is no need for DDT use in the US, as the public health challenges that might necessitate it are limited and solvable with other techniques. But that is not true overseas, where the US government currently supports the use of DDT in malaria-affected countries. A current page on EPA's website indicates that agency is helping foreign countries to use DDT safely:

> *"DDT is one of 12 pesticides recommended by the WHO for indoor residual spray programs. It is up to countries to decide whether or not to use DDT. EPA works with other agencies and countries to advise them on how DDT programs are developed and monitored, with the goal that DDT be used only within the context of Integrated Vector Management programs, and that it be kept out of agricultural sectors."*[67]

USAID also is involved in advancing the use of DDT abroad. As the USAID website reads:

> *"USAID is currently supporting IRS with DDT in Zambia.*
>
> *This year, pending completion and satisfactory results of all necessary entomological and environmental assessments, USAID plans to support IRS with DDT in Ethiopia*

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[60] For the fact that DDT was used for rabid bats in Texas, see *Federal Register*, January 12, 1977, page 2527, and January 21, 1980, page 3970.
[61] North American Commission for Environmental Cooperation. History of DDT in North America to 1997. Available at http://www.cec.org/files/pdf/POLLUTANTS/HistoryDDTe_EN.PDF (accessed 17 September 2009).
[62] Ibid.
[63] 40 C.F.R. §164.130-33.
[64] 40 C.F.R. §166.25
[65] 40 C.F.R. §166.28
[66] 40 C.F.R. §166.40 *et seq*
[67] EPA, DDT - A Brief History and Status, available at: http://www.epa.gov/pesticides/factsheets/chemicals/ddt-brief-history-status.htm (accessed 17 September 2009).

and Mozambique (including purchase of the insecticide); and also in Madagascar (using DDT purchased by another donor)."[68]

As of 2009, the US government's efforts have grown, such that it is a leading financier of DDT for malaria control. For example in Ethiopia, the President's Malaria Initiative plans to spend $1.6 million in FY 2009 to purchase DDT and perhaps other insecticides (the exact amount of DDT that the US government will purchase in 2009 is not known at this writing, but as an indication American support is ongoing, in Ethiopia in FY2009, US government funds have been provided with the intention of purchasing 1.6 million kilograms of 75% DDT).[69]

(7) The EPA's decision-making processes, circa 1972:

The EPA arrived at its decision in 1972 to cancel the non-emergency civilian DDT registrations in two stages: (i) a decision of administrative law Judge Edmund Sweeney, before whom an extensive hearing took place on DDT's safety, and; (ii) a decision of EPA Administrator William Ruckelshaus, overturning Judge Sweeney without further hearing.

The hearing that Judge Sweeney conducted was extensive: seven months, 125 witnesses, and 365 exhibits, after which he wrote "the pros and cons of DDT have been well aired."[70] Judge Sweeney reached this terse and unqualified finding:

"DDT is not a carcinogenic hazard to man."[71]

Administrator Ruckelshaus reversed Judge Sweeney's decision three months after it was delivered. He did so not because of disagreement with Judge Sweeney's finding of fact on carcinogenicity, but rather because as Administrator Ruckelshaus wrote in his reasons:

"The possibility that DDT is a carcinogen is at present remote and unquantifiable; but if it is not a siren to panic, it is a semaphore which suggests that an identifiable public benefit is required to justify the continued use of DDT."[72]

Unable to find that DDT was carcinogenic, Administrator Ruckelshaus hinged his decision on there being a "*possibility* that DDT is a carcinogen." He then proceeded to ask if there were satisfactory alternatives to DDT. Since he also concluded that "safer alternatives exist[ed] to achieve the same benefit" for DDT's principal agricultural uses, he cancelled those uses.[73]

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[68] USAID. Indoor Residual Spraying (IRS), available at: http://www.usaid.gov/our_work/global_health/id/malaria/techareas/irs.html (accessed 17 September 2009). The reference to "this year," however, seems to be a holdover from a year prior to 2009.

[69] President's Malaria Initiative Malaria Operational Plan – Ethiopia FY 2009, available at http://www.fightingmalaria.gov/countries/mops/ethiopia_mop-fy09.pdf (accessed 17 September 2009).

[70] *In re. Stevens Industries, Inc., et al (Consolidated DDT Hearings)*, I.F.&R. Docket nos. 63 et al, 25 April 1972, per Hearing Examiner Edmund Sweeney, page 16.

[71] Ibid, page 93.

[72] *In re. Stevens Industries, Inc., et al (Consolidated DDT Hearings)*, I.F.&R. Docket nos. 63 et al., 2 June 1972, per Administrator William Ruckelshaus, page 29.

[73] Ibid., page 28.

Where there were not safer alternatives, as for public health, he declined to cancel those DDT uses.[74]

There are differing explanations of why Administrator Ruckelshaus reversed Judge Sweeney's decision. One is that Administrator Ruckelshaus acted on the basis of what later came to be known as the "precautionary principle". That is, where there were good alternatives to DDT for a particular use, it was a feasible precaution to cancel DDT in that use even absent proof that it was a human carcinogen. (A difficulty with such precautionary reasoning is that one may not always have evidence to establish that the alternatives are actually any safer.)

Another, not necessarily incompatible, explanation is that Administrator Ruckelshaus acted for political reasons. Administrator Ruckelshaus was the inaugural head of EPA, at a time when that newly-created agency was under organized pressure from groups such as Environmental Defense Fund. Some insight into the political effect of that pressure is gained by comparing Administrator Ruckelshaus's decision, to this advice he received from a young lawyer who had worked on a DDT court case:

> *"The safety record of DDT is phenomenal. Billions of pounds of DDT have been used in anti-malaria programs during the past quarter of a century, and there is no record of human illness attributable to DDT resulting directly from the normal spraying operations among either the 130,000 spraymen or the 535 million occupants of DDT treated houses... Human volunteers and employees working in DDT formulating plants have been exposed to inordinately large quantities of DDT without incurring any illness attributable to DDT... [M]edical and scientific research and investigation fail to indicate any adverse clinical effect upon human health."*[75]

The "young lawyer" who wrote this passage was actually William Ruckelshaus! He was promoted to EPA Administrator only months after penning this passage; it seems that the expectations in his new post reformed his point of view quite considerably.

In summary, EPA's reasoning in 1972 as regards carcinogenicity is fully concordant with the US government's *status quo* in 2009, and fully concordant with the WHO, DHHS and IARC statements published in intervening years—an unbroken thread connects them all.

(8) Conclusion

Based on my education, research, policy work and other career experiences, I conclude as follows:

1. At the present time, leading public health agencies at the national and international levels (such as DHHS, WHO and IARC) have concluded that the evidence does not support a causal association between DDT exposure and cancer in humans.

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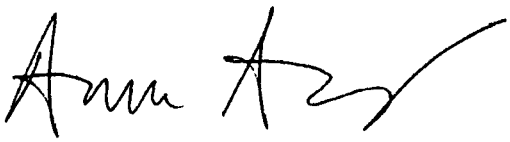
[74] Administrator Ruckelshaus emphasized that the EPA's "hearings... never involved the use of DDT by other nations in their health control programs," and so the benefits of DDT for controlling malaria and other diseases simply were not considered in EPA's decision: see Ibid., page 31.

[75] Brief for the Respondents, *Environmental Defense Fund et al v. Clifford M. Hardin, Secretary of Agriculture et al.*, file 23813, U.S.C.A. (DC Cir.). Mr. Ruckelshaus was Assistant Attorney General and his name appears as such on the Brief, dated 31 August 1970.

2. In contrast, there is abundant and non-controversial evidence that the public health use of DDT has avoided human death and illness from insect-borne diseases such as malaria or typhus. The savings are in the millions of lives historically.
3. Over a course of decades, and across a range of diseases, DDT delivered ascertainable improvements to Texans' health, including in Hidalgo County.
4. Since the 1990s, the use of DDT for public health purposes is experiencing a resurgence, with the support of agencies such as USAID, WHO and the World Bank, and the agreement of leading environmental groups.
5. The EPA did not "ban" DDT, but limited its registrations, principally for political reasons. Further, EPA's reasons did not include any finding that DDT is carcinogenic in humans.

Signed at Ottawa, Ontario, on 22 September 2009.



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Appendix One – Historical Public Attitudes Approving of DDT:

The Right Honourable Sir Winston Churchill:

"The excellent D.D.T. powder, which has been fully experimented with and found to yield astonishing results, will henceforward be used on a great scale by the British forces in Burma and by American and Australian forces in the Pacific and, indeed, in all theatres, together with other remedies consistently improving, and these will make their effect continually manifest... These remedies will be a help to all the Allies; indeed they have been a help. The eradication of lice in Naples by the strict hygienic measures taken may be held to have averted a very grievous typhus epidemic in that city and neighbourhood when we occupied it."

Winston Churchill (1945). *The Dawn of Liberation.* Boston: Little, Brown, p. 243.

Dr. Albert Schweitzer:

"We are often reminded that ours is a jungle Hospital by the natives who arrive after being wounded by wild animals. The white ants too are a constant reminder. We are continually being stirred up by them, and their presence is only discovered when they have already done considerable damage. Then everything has to be cleared out in order to find out just where they made their entrance. How much labor and waste of time these wicked insects do cause! Nothing so far has been effective in keeping them out, but a ray of hope, in the use of DDT, is now held out to us."

Albert Schweitzer (1949). *Out of My Life and Thought: an autobiography.* New York: Henry Holt, p. 261-262.

The Nobel Prize Presentation Committee (the presentation speech)

"Towards the end of the Second World War, typhus suddenly appeared anew. All over the world research workers applied their energies to trying to discover an effective delousing method. Results, however, were not very encouraging. In this situation, so critical for all of us, deliverance came. Unexpectedly, dramatically practically out of the blue, DDT appeared...

Since those days DDT has been used in large quantities in the evacuation of concentration camps, of prisoners and deportees. Without any doubt, the material has already preserved the life and health of hundreds of thousands."

Professor G. Fischer, member of the Staff of Professors of the Royal Caroline Institute, on awarding the Nobel Prize in Physiology or Medicine 1948 to Paul Hermann Müller "for his discovery of the high efficiency of DDT as a contact poison against several arthropods". Available at http://nobelprize.org/nobel_prizes/medicine/laureates/1948/press.html.

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Curriculum vitæ

HIGHER EDUCATION

Law, LL.B. 1995-98	**University of British Columbia** Degree in Canadian common law, especially the areas of constitutional and environmental law.
Biology, D.Phil. (Ph.D.) 1992-96	**Oxford University** Doctoral research in cell biology and immunology, and mechanisms of virally-mediated cell death.
Biology, M.S. 1990-91	**California Institute of Technology (Caltech)** Degree by research in cell and structural biology.
Neurobiology, B.A. 1984-88	**University of California, Berkeley** Degree in neurobiology.

EMPLOYMENT

Associate Professor 2004-present	**University of Ottawa** Professor and Chair in the Faculties of Law and Medicine and the Institute of Population Health (cross appointment)
Associate Fellow 2003-2004	**Royal Institute of International Affairs, London** International Economics Program
Lecturer 2003	**Yale University** School of Epidemiology and Public Health
Fellow / Adjunct Lecturer 2000-2003	**Harvard University** Kennedy School of Government
Staff Researcher & Lawyer 1996-2000	**Sierra Legal Defence Fund, Canada** Litigation and strategy in environmental law
Management Consultant 1996	**Corporate Value Associates, UK** Strategic process analysis of corporate R&D units

SCHOLARLY SERVICE & HONORS

Associate Editor 2008-present	**Canadian Medical Association Journal** Service on the editorial team
Editorial Board Member 2008-present	**Journal of Epidemiology & Community Health** Service on the editorial team
Board Member 2008-present	**Coalition Advancing Standards in Research Admin.** Regular member of the Board

Panelist 2006	**CMAJ Governance Panel (the Pound Panel)** Service to the Canadian Medical Association in the review of governance and editorial procedures at CMAJ
University Chair 2005-present	**Canada Research Chair** Awarded the Canada Research Chair in Law, Population Health, and Global Development Policy
Editorial Consultant 2005-present	**The Lancet** Service on the editorial team

GRANTS AND CONTRACTS

Research Grant 2008-2011	**SSHRC, Principal Applicant** Developing Accountability: Studies of the international aid enterprise and the accountability of donor institutions
Research Grant 2008-2011	**SSHRC, Principal Applicant** The Management of Drug Benefits and Access to Medicines in Canadian Health Systems; New Legal Trends and Policy Hazards
Workshop Grant 2008-2009	**SSHRC, Principal Applicant** First Decade Workshop on Crimes against Humanity and War Crimes Laws in Canada
Research Grant 2008-2011	**CIHR, Co-Applicant** Scaling Up Malaria Prevention – What Works?
Research Grant 2007-2010	**CIHR, Co-Applicant** Evaluating the need for and design of a no-fault compensation program for immunization related injuries
Consultancy 2007-Ongoing	**CMA, Editorial Board** Associate Editor, Editorials
Research Grant 2006-2009	**CIHR, Co-Applicant** Health in an Unequal World
Research Grant 2006-2009	**SSHRC, Co-Applicant** Canada and The "Brain Drain" of Health Professionals from Sub-Saharan Africa
Project Grant 2004-2004	**Gates Foundation Policy Research Network** Aids Treatment: Policy Research Network
Consultancy 2003-2004	**Novartis AG, Switzerland** Public policy advising on the non-profit distribution of a malaria medicine (Coartem) in poor countries
Project Grant 2003	**World Bank Group, USA** Developed HIV/AIDS medicine procurement toolkit
Research Grant 2002	**Africa Fighting Malaria Foundation, UK** One-year unrestricted research award

Conference Grant 2001	**World Health Organization/TDR, Switzerland** Grant for a conference on the economics of malaria
Consultancy 2000	**United Nations Development Program** Research and writing contract for the *Human Development Report 2001*
Consultancy 1999	**Médecins Sans Frontières** Public policy research, conference leadership, on aspects of trade law and access to medicines
Predoctoral Fellowship 1992-96	**Howard Hughes Medical Institute, USA** Five-year Ph.D. research and full tuition grant.
Scholarship 1992-94	**Council of Vice Chancellors & Principals, UK** Three-year scholarship for doctoral studies.
Scholarship	**National Science Foundation, USA** One-year fellowship for M.S. studies.

COURSES TAUGHT, TUTORED OR ASSISTED

Law	CML 4200 JE Law and Current Problems in Global Poverty and Public Policy
International Development	MPP Policy Development Practicum (Harvard) MPP Graduate international dev't seminar (Harvard) Undergraduate international dev't seminar (Harvard)
Public Health	MPH Health System Reform in Middle and Low Income Countries (Yale)
Biology	Cell Biology (Oxford) Immunology (Oxford) Microbial Genetics laboratory (Caltech)
Interdisciplinary	Technology and Society (Berkeley) History of American Technology (Berkeley)

SUPPLEMENTAL

UN Languages	French (good), Spanish (fair)
Media	Frequent commentator and/or contributor to the BBC, CBC, *Globe & Mail*, *New York Times*, *Wall Street Journal*, and many others.
Avocations	Bicycle touring, Backpacking, Music appreciation

PUBLICATIONS:

Books edited:

A. Attaran and B. Granville (eds) (2004) ***Delivering Essential Medicines: The Way Forward***. London: Royal Institute of International Affairs and Brookings Institution, pages 1-189

Chapters in books:

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Expert Report of Seymour Grufferman, M.D., Dr.P.H., on Behalf of Montrose Chemical Corporation of California

Guadalupe Garza v. Allied Chemical Corp., et al.

October, 2009

TABLE OF CONTENTS

Page

I.

INTRODUCTION

I have been retained by Montrose Chemical Corporation of California in connection with the *Guadalupe Garza v. Allied Chemical Co., et al.* litigation. My areas of scientific expertise include cancer epidemiology in general, the epidemiology of the hematologic malignancies and the epidemiology of non-Hodgkin lymphoma (NHL). I have reviewed the medical records, deposition testimony, and other documents related to the claim that DDT exposure was the cause or a contributing factor to Ms. Guadalupe Garza's NHL. I have reviewed the reports and claims of plaintiff's experts, Dr. Richard W. Clapp, Dr. Frank H. Gardner, Dr. William R. Sawyer and Dr. Theodore M. Farber, on NHL causation. In addition, I have reviewed (1) scientific literature on the causation of NHLs, and (2) the 40 reports in the published scientific literature that consider whether or not DDT is a cause of NHL.[1]

I will render an opinion on whether or not DDT is a scientifically established cause of NHL or a contributing factor. I also will render opinions on whether or not Ms. Garza's NHL was caused by her alleged exposure to DDT, and whether or not DDT was a contributing factor to her NHL. Finally, I will render an opinion on whether or not Drs. Clapp, Gardner, Sawyer and Farber used accepted scientific methodology in reaching their causation opinions on DDT and NHL.

[1] Unless otherwise specified, reference to "DDT" in this report also includes its metabolites.

II.

SUMMARY OF OPINIONS

Based on my years of education, training, research and peer review work on the lymphomas, together with my review of all of the materials listed above, I conclude to a reasonable degree of medical and scientific certainty that DDT is not a cause of NHL in humans. In fact, DDT has been shown to be a safe product for humans and one that has great public health value for the control of malaria. In 2006, the World Health Organization and the U.S. Agency for International Development recommended the use of DDT inside homes for malaria control and eradication in countries with endemic malaria. The epidemiologic evidence as well as human experimental evidence simply does not support the notion that DDT is a scientifically established cause of NHL. This opinion is also shared by leading scientists, not involved in this litigation, who have reviewed the scientific literature and authored review chapters on the epidemiology of NHL for leading textbooks in the field. This opinion that DDT is not a cause of NHL is accepted scientific knowledge in agreement with the scientific literature.

The published epidemiologic literature on DDT and NHL covers an extensive array of scientific research. It includes two human experiments, 36 analytic study reports (seven cohort studies, eight case-control studies employing multivariate analyses, 21 case-control studies employing univariate analysis) and two ecologic study reports. Despite searching for over two decades for evidence of a causal association between DDT and NHL, at the cost undoubtedly of millions of dollars, researchers have been unable to demonstrate any such link. In fact, the absence of any such causal link is evidenced by the overwhelming number of published studies that either have no statistically significant findings or results that center around the null value of 1.0.

At the request of Montrose, I undertook an exercise in which I aimed to identify study reports that had findings of a statistically significant doubling of the risk. The results are notable:

1. In the seven cohort study reports, *none* has a finding with a statistically significant doubling of the risk.
2. In the eight case-control study reports with multivariate analyses, *none* has a finding of a statistically significant doubling of the risk after controlling for potential chemical and other confounders.
3. In the two ecologic study reports, *neither* had a finding of a statistically significant doubling of the risk.
4. In the two DDT experiments where humans were fed extremely large amounts of DDT, *neither* evidenced an increase in cancer risk.
5. In the 21 case-control reports with only univariate analysis—reports that failed to control for confounding exposures using sophisticated, modern epidemiologic methods—just three of the 21 reports contain statistically significant DDT findings that evidenced a doubling of the risk, among numerous findings resulting from a multitude of subset analyses. Thus, in 18 of the 21 reports, there is not one finding which shows a statistically significant doubling of risk, despite the fact that these 18 studies do not control for critical confounders like other exposures. In the other three reports, each performs numerous subset analyses, increasing the likelihood of findings being due to chance and/or confounding, and/or conducts

analyses that are of questionable relevance to Ms. Garza, i.e., risk estimates for EBV EA antibodies.

Based on this evidence and my years of study relating to the causation of the lymphomas, DDT is not a cause of NHL in humans. Ms. Garza's experts have, among other things, failed to consider all 40 studies, failed to document and report their analyses of these 40 studies, and also failed to reach a conclusion through application of proper scientific methodology. Therefore, the opinions they reach are unscientific and unreliable.

I also have reviewed the epidemiologic literature regarding DDT and NHL to determine whether there is any evidence of DDT being a contributing cause for NHL. None of the papers I found in the literature on DDT and NHL provide scientific data on DDT as a contributing factor for the disease. Thus, DDT is not a contributing factor to NHL, since there is no scientific evidence to support this notion.

Lastly, to a reasonable degree of medical and scientific certainty, Ms. Garza's NHL was not caused by her claimed exposure to DDT as alleged by certain plaintiff's experts. Since the scientific evidence does not show that DDT causes NHL in humans, it could not have caused Ms. Garza's NHL. Moreover, Ms. Garza has had no history of employment that would entail occupational exposure to DDT. Occupationally exposed workers and volunteers who ingested extremely high levels of DDT had no increased risk of NHL. Whatever the level of Ms. Garza's alleged exposure to DDT, assuming she was even exposed to greater than ambient U.S. levels, and based on the scientific literature, it certainly is not the cause of her NHL. Furthermore, there is no scientific evidence in the literature to suggest that DDT was a contributing cause to Ms. Garza's NHL.

III.

EXPERT'S BACKGROUND

My education, training and experience are detailed below. I hold a B.S. degree from C.C.N.Y., where I majored in biology and chemistry. I obtained my M.D. degree from the State University of New York Upstate Medical Center in Syracuse where I received an award for the student showing the most promise for a career in medicine or surgery. I received an M.P.H. degree in 1968, an M.S. degree in 1974, and a Dr.P.H. degree in 1979, all in epidemiology, from the Harvard University School of Public Health. My doctoral dissertation at Harvard was on the possible transmissibility of Hodgkin's disease, a type of lymphoma. I was a recipient of the annual Student Prize Paper Award of the Society for Epidemiologic Research for my thesis and two papers from my thesis were published in the New England Journal of Medicine.

In addition to my medical and epidemiologic education, I also have completed clinical training in medicine. I completed a rotating internship at the University of Illinois Research and Educational Hospitals in 1965, following graduation from medical school. I then completed residency training in pediatrics at the New York Hospital – Cornell Medical Center in 1967. As part of my residency, I received training in pediatric oncology at the Memorial-Sloan Kettering Cancer Center. I am board-certified in Pediatrics, a Diplomate of the National Board of Medical Examiners, and hold active state medical licenses in North Carolina and Pennsylvania. Following completion of my clinical training and obtaining an M.P.H. degree, I served in the U.S. Air Force from 1968-71. I was Chief of Pediatrics and Military Public Health at the USAF Hospital in Tachikawa, Japan, which was a large Air Force referral and training hospital. I was also 5th Air Force Consultant in Pediatrics and provided supervisory care at bases in Japan and

Okinawa. Following this experience, I took a position as Assistant Professor and Chairman of the Department of Public Health at the Gondar Public Health College (of the then Haile Sellassie I University) in Gondar, Ethiopia. In addition to teaching epidemiology and biostatistics, I also taught organic chemistry and tropical medicine.

I returned to the United States in 1973 to resume my education in epidemiology at Harvard. Following completion of my education at Harvard, I joined the faculty at the Duke University Medical Center as Assistant Professor of Community Health Sciences and later, Pediatrics and Medicine. I was also Associate Director for Cancer Epidemiology and Prevention at the Duke Comprehensive Cancer Center. I taught epidemiology and biostatistics to third-year medical students and ran a special training program in epidemiology for medical students. While at Duke, I received a Preventive Oncology Academic award from the National Cancer Institute and a large grant in clinical epidemiology from the A.W. Mellon Foundation. These allowed me to run a large training program in epidemiology at Duke for medical students, residents, medical subspecialty fellows and faculty. While at Duke, I was awarded peer-reviewed, National Institutes of Health grants for research on rhabdomyosarcoma (a soft tissue sarcoma of children), multiple myeloma (bone marrow cancer), NHL and Hodgkin's disease. Thus, I am rendering opinions in this case on a cancer on which I personally have conducted peer-reviewed, NIH-funded, etiologic research. While at Duke, I was promoted to Associate Professor, was awarded tenure, and became Chief of the Division of Clinical Epidemiology in the Department of Pediatrics. I taught and practiced pediatrics at Duke and held admitting privileges to the Duke University Hospital.

I moved to the University of Pittsburgh in 1987 as Professor of Epidemiology in the Graduate School of Public Health and Associate Director for Cancer Epidemiology and Control of the Pittsburgh Cancer Institute. Shortly thereafter, I was appointed as Chairman of the Department of Clinical Epidemiology and Preventive Medicine in the School of Medicine. This Department evolved over the years into a Department of Family Medicine and Clinical Epidemiology. The Department did all of the medical school teaching of epidemiology and biostatistics. While I was Chairman, I served on the Executive Committee of the School of Medicine. I was elected President of the University Physicians Practice Association and performed a variety of functions related to the restructuring of the Medical Center to incorporate a network of community hospitals and physicians throughout western Pennsylvania. I stepped down as Department Chair in 1998, and retired from the University of Pittsburgh in 2004. I taught and practiced family medicine at the University of Pittsburgh and had admitting privileges to the Children's Hospital and Presbyterian-University Hospital.

I relocated to the University of New Mexico (UNM) in 2004, where I held the rank of Research Professor in the Epidemiology Division of the Department of Internal Medicine. I moved a large NIH research grant for research on Hodgkin's disease to UNM and this and rhabdomyosarcoma remain my major research activities. I retired from UNM at the end of June, 2007, and continue to hold an appointment as Adjunct Research Professor.

I have considerable experience in performing peer review work for government and non-governmental organizations. I served four-year terms on a National Cancer Institute (NCI) Review Committee that reviewed all NIH cancer center grants, a National Institute

of Allergy and Infectious Diseases committee that reviewed AIDS-related epidemiology research grants (which I chaired for four years), an NCI committee that reviewed all training and career development grants (I served two consecutive terms on this committee) and a review committee of the American Cancer Society (ACS) that reviewed research grants in the areas of cancer prevention, detection and treatment. I recently completed a term as a charter member of the Cancer Biomarkers Study Section of the NIH. This Study Section reviews all NIH grant applications that relate to biomarkers of cancer, which are mostly of a molecular biologic nature. Even though I completed my term on this Committee, I continue to serve on it as an ad hoc member. In total, I have over 20 years of service in the formal review of research grants for the NIH and ACS. In addition, I perform numerous special reviews of grants for NIH and other funding agencies in the U.S. and other countries. For example, I have served for the past five years on an annual NCI review committee to evaluate grant applications for repayment of educational loans for new investigators. I also assisted the Associazione Italiana per la Ricerca sul Cancro (Torino, Italy) in reviewing cancer research grants. I continue to perform peer review of manuscripts for leading medical and public health journals.

In summary, I have extensive experience in medical and public health practice and education, academic and medical administration, peer review work on behalf of the U.S. National Institutes of Health (NIH), American Cancer Society, and international agencies, and in the conduct of peer-reviewed NIH (and other agencies) funded research on many cancers. I have extensive experience in peer review of all aspects of cancer research, as well as of epidemiologic and biostatistical research in general, for a wide variety of governmental and private funding agencies worldwide. This equips me

particularly well for critical review of the scientific literature on the subject of DDT and NHL.

I also have extensive experience in teaching epidemiology and biostatistics. I organized and taught courses on these subjects at the Gondar Public Health College, Harvard and Duke Universities and the University of Pittsburgh. I taught in a series of International Agency for Research on Cancer (IARC) courses on Cancer Epidemiology and Biostatistics all over the world (in the former GDR and USSR, Pakistan, Thailand, Poland and Zambia). I was usually the course director for these courses that were focused on the use of epidemiology and biostatistics for cancer research. I also taught in several courses at the U.S. National Cancer Institute on cancer epidemiology and prevention and covered the topic of the hematopoietic malignancies (leukemias, lymphomas and multiple myeloma). This extensive teaching experience also equips me well for dealing with the scientific literature on DDT and NHL.

In addition, I personally have conducted peer-reviewed, NIH-funded research on NHL as well as on the possibly related diseases of Hodgkin lymphoma and multiple myeloma. I have published peer-reviewed papers on the epidemiology of NHL and have written chapters on this topic for one of the leading textbooks on hematologic malignancies. I am currently revising this chapter for the next edition of this textbook (Neoplastic Diseases of the Blood, 4th Ed., in preparation). One of my NHL papers was published in the New England Journal of Medicine and received extensive public media attention including an article in Time magazine. I am co-author of the chapter on Hodgkin lymphoma, a disease whose scientific literature overlaps with that of NHL, in the newly released third edition of the leading textbook on cancer epidemiology and

prevention. I am a Fellow of the American College of Epidemiology and an elected Fellow of the American Association for the Advancement of Science (AAAS). My election as a Fellow of the AAAS was on the basis of my contributions to research on the leukemias and lymphomas.

In summary, my expertise on the causation of NHL is not generated by this litigation. Rather, my expertise in this case derives from a lifetime of research and experience on the lymphomas (and their medical literature).

IV.

THE UNIQUE ROLE OF EPIDEMIOLOGY IN ASSESSING POTENTIALLY HARMFUL EXPOSURES

A. Scientists Presently Are Not Allowed to Conduct Experiments in Humans of Potentially Harmful Substances

In this report, I will be relying primarily on my epidemiologic expertise and my review of the epidemiologic and related medical literature. Often, the best evidence upon which scientists can rely to determine disease causation is from human experiments. Such experiments and the analogous clinical trials in humans allow for the control of many confounding factors. An experiment can be defined as a study in which the investigator allocates an "exposure" (i.e., a drug) to human subjects, typically in a random manner, and uses a comparison group of very similar, but unexposed, subjects. The exposed and unexposed groups are followed over time for occurrence of an outcome (clinical improvement in the case of a drug trial or adverse effects in a toxicology study). Rates of occurrence of the outcomes then are compared for the exposed and unexposed groups. Such trials generally provide reliable and unbiased evaluations of treatment modalities and often are considered to be the "gold standard" of clinical research.

While experiments and the experimental approach are held in high regard as generally providing definitive answers to many therapeutic and toxicologic questions, there is one major limitation. This limitation is that it is unethical and scientifically unacceptable to conduct experiments in humans of potentially toxic substances. Clinical trials can only be done ethically (and legally) to evaluate potentially beneficial exposures (usually drug treatments). There are almost no human clinical trials in the literature of potentially harmful exposures. Thus, there are usually no experimental scientific data available on the potentially adverse health effects of pesticide exposures in humans.

However, in the 1960s and 1970s, the United States government studied the possible health effects of feeding human volunteers DDT. As described below, two of these studies involved the use of prison inmates and no adverse health effects of ingesting DDT were found. By today's standards, such research would not be permissible under international and U.S. regulations, but revisions to these rules are being considered currently. Thus, there are no experimental scientific data, other than the feeding studies, on any health effects of DDT (and other pesticide) exposure in humans. While there are animal experiments on these and other similar chemical exposures, the data from such experiments cannot be extrapolated reliably to humans. For example, in addition to different susceptibilities of various animal species, the exposure doses used in most animal studies are usually quite high and typically several orders of magnitude higher than that which humans might encounter environmentally.

B. Epidemiology Allows Researchers to Approximate Experiments in Humans

It is because of this inability to obtain experimental data in humans regarding possibly harmful environmental exposures that epidemiology comes to play a central role. Methods used in epidemiology allow researchers to approximate human

experiments. Such epidemiologic approaches rely on observations of potentially toxic exposures in humans that were "allocated" to individuals by natural factors and not by a scientific investigator. In a clinical trial or experiment, the investigator determines who is given the drug or possibly harmful agent. In an epidemiologic study, natural factors or individual choices and opportunities determine who is exposed and not exposed. For example, in a study of disease occurrence in a cohort of workers in an industry or plant, the workers' exposures essentially are determined by factors such as the availability of jobs, their decision to seek employment in that industry, or their assignment to jobs in a particular plant location or process area of a company. This gets around the ethical dilemma of determining exposures, but the methods used in epidemiology cannot duplicate an experiment and provide similarly precise information. The epidemiologic approach uses a body of methods to approximate experimental results in "free-living" human populations and the approach has proven to be extremely useful and reliable. The results of epidemiologic studies have provided very valuable and important health information over the long history of the field.

V.

EPIDEMIOLOGIC APPROACHES

A. Epidemiology Studies How a Disease Occurs or is Distributed in Human Populations and Tests Disease-Related Hypotheses

Epidemiology can be defined as the scientific study of the distribution and determinants of disease occurrence in human populations (MacMahon and Pugh). The "distribution" of a disease refers to how a disease occurs or is distributed in a population. This realm of the field has often been referred to as "descriptive epidemiology" and deals with such issues as the distribution of disease occurrence in terms of time, person and

place characteristics. For example, an investigator might assess whether there is variation in occurrence of a disease by season, age or gender of those affected, or by area of residence. From such information, the investigator might derive clues as to possible causes of the disease. Such clues might lead to hypotheses (basically, educated guesses) regarding the cause of the disease.

The second part of the definition above, the "determinants" of disease occurrence, deals with the testing of such hypotheses in studies of human populations. A well-developed body of epidemiologic methods has been developed for the generation and testing of disease-related hypotheses in humans.

B. There Are at Least Two Broad Categories of Epidemiologic Studies – "Descriptive Studies" and "Analytic Studies"

For purposes of this report, I will group the commonly used epidemiologic methods into two broad categories: "descriptive studies" and "analytic studies".

1. Descriptive Studies

So-called descriptive epidemiologic studies generally are used to develop new hypotheses about the occurrence, natural history or causation of a disease or condition. They tend to focus on the first part of the MacMahon and Pugh definition of epidemiology – the distribution of diseases in human populations. Typically, an epidemiologic investigator would assess the occurrence of a disease in terms of the distribution of cases by time, place and person characteristics. For example, an investigator in the 1940s might have noticed that there was an unusually high recent occurrence of what previously had been a very rare disease, primary lung cancer. On paying closer attention to these new cases, he might have noticed that almost all of the cases were cigarette smokers. Such an observation might lead to the development of an

hypothesis that cigarette smoking is a cause of lung cancer. This type of epidemiologic investigation has proven particularly useful in gaining knowledge about the natural history and possible causes of new diseases. For example in recent years, much was learned about the possible causes of SARS (severe acute respiratory syndrome), its spread and its natural history by the simple approach of observing the occurrence of cases in small residential areas (Tsang, et al., 2003; Yu, et al., 2004). The major limitation of the descriptive epidemiologic approach is that it has not been useful for proving disease causation.

One type of descriptive study is the so-called "ecologic study." In this study method, the occurrence of a disease is related to levels of certain suspected harmful (or beneficial) exposures in a community. The notion is that if there were a true association between some disease and community levels of exposure to a putative cause, it would suggest a causal relationship. An example of this approach was the observation that people who resided in regions with high naturally occurring levels of fluoride in their water supplies had fewer decayed teeth. While this did not prove that fluoridation protected against dental cavities, it led to the more formal testing of the hypothesis via a community trial of fluoridation of water supplies (an experimental approach). Unfortunately, most such ecologic findings prove to be false leads.

The major problem with ecologic studies is that there is no information on what any individual person with disease or healthy person might have been exposed to or at what levels. This is a particular problem when ecologic studies are done of large geographic areas, such as whole states. These studies also are particularly prone to the effects of confounding (discussed below), which can severely distort results.

A second type of descriptive study is the "cross-sectional study." Subjects with a disease or health condition are studied for some current characteristic or marker. An example of this would be a survey of serum cholesterol levels in men admitted to the hospital for an acute myocardial infarction (a heart attack). While cholesterol levels in these men may be higher than the population norms, one cannot say whether the elevated cholesterol levels preceded the heart attack, let alone say that it was a cause of the heart attack. Typically, cross-sectional studies are even less clear-cut than this example and suffer from the "chicken and egg" dilemma of which came first. Such studies rarely are done nowadays because of this limitation.

These examples of descriptive epidemiologic approaches illustrate the major limitations of such studies for assessing disease causation. They are useful, however, for suggesting causes or means of prevention of disease that could be tested by more precise methods.

2. Analytic Studies

Much of modern epidemiologic research falls within the realm of analytic studies. The two main research approaches used for analytic epidemiologic studies are the case-control and cohort studies.

a. Case-Control Studies

In a case-control study, a group of people with a disease or health condition is selected for research after the disease or condition has been diagnosed. The subjects are studied for their prior history of "exposure" to potential causes of their disease, such as their occupations, their family history of the disease and a variety of other antecedent suspected "risk factors." Findings in the "cases" typically are compared to findings of antecedent events in a comparable group of people without the disease at issue –

"controls." The basic features then of case-control studies are the selection of subjects for research after they have been diagnosed with the disease being studied and the search for potentially causal events prior to their diagnosis.

By way of an example, in a typical case-control study, a researcher might identify a group of 150 persons with newly diagnosed NHL. A similar number of control subjects might be identified from cases' neighborhoods by use of random digit-telephone dialing (a method based on a case's home telephone number to call other homes in the same calling area to identify a comparable person who does not have NHL). Cases and controls then would be interviewed regarding a long list of antecedent potential "risk factors" (a suspected factor that appears to be associated with risk of a disease) such as occupational history and exposures, diet, past medical history, family history, etc. All of these factors will have occurred prior to the cases' diagnosis. Typically, in a case-control study, a very large number of risk factors are queried, often 100 or more. Comparisons then are made between the frequency of assessed risk factors in the cases and controls. In the analysis of such studies, the frequencies (odds) of exposure in the two groups are compared. Thus, the relative frequencies of exposures are determined, but case-control studies cannot provide direct measures of what the rates of disease occurrence are in exposed and unexposed individuals. Rather, the investigator obtains an "odds ratio" (the ratio of the odds of exposure in the two groups). For most diseases, the odds ratios from case-control studies prove to be comparable to the more precise relative risks (the ratio of actual risk in the exposed and not-exposed groups) from cohort studies.

Case-controls studies have several advantages over cohort studies:

1. Case-control studies have greater efficiency in the study of rare diseases. When a disease is rare, cohort studies would have to be extremely large to produce adequate numbers of cases of the rare disease to be studied. In many cohort studies of limited size, for example, there may be very few cases of NHL as an outcome since it is a relatively uncommon diagnosis. In such instances, cohort studies might lump together several of the hematologic malignancies (leukemias and the lymphomas, and occasionally multiple myeloma as well) for purposes of analysis. Such results are of limited utility for assessing risk of any specific member of the group, such as NHL. On the other hand, in case-control studies, one can assemble a sufficiently large number of cases for most analyses;
2. Another advantage of the case-control study is its ability to assess many potential causal factors, whereas in a cohort study one can assess one (or a limited number of) exposure(s), but study several possible diseases in relation to that exposure;
3. Case-control studies are generally more quickly done than cohort studies since there is no lengthy follow-up period needed between the exposure and disease outcome; and
4. A fourth advantage of case-control studies is that diagnosis of cases is generally more accurate than in cohort studies which frequently use diagnoses from death certificates rather than from pathology reports as is commonly done in case-control studies.

The disadvantages of the case-control study include:

1. No direct measure of relative risk can be obtained usually;
2. The investigator frequently relies on subjects' recall of past "exposure" history; and
3. By assessing many potential risk factors, the investigator is likely to frequently encounter chance associations. This latter disadvantage can be dealt with by use of analytic techniques for assessing the role of chance and by the application of methods for evaluating whether an observed association might be causal rather than chance (see the Hill criteria discussion below).

b. Cohort Studies

The other major research approach in this domain is the cohort study. In a cohort study, research subjects are chosen on the basis of their "exposure" status, not on the basis of whether they have the disease being studied. In most such studies, subjects with an exposure of interest, for example cigarette-smoking, are identified and followed over time for the occurrence of a suspected associated disease or group of diseases. A comparable cohort of people without the exposure of interest is identified and followed simultaneously over time to observe their disease risk. Comparison is later made between the risks of disease in the exposed and not-exposed groups. The ratio of the disease rate in the exposed group and that in the unexposed group provides a measure of risk termed the relative risk. The basic features of cohort studies are the selection of subjects for research based on their exposure status (not their disease status as in case-control studies) and their ability to prospectively determine health risks of an exposure.

An example of this type of study is the now classic cohort study of smoking in British physicians. The investigators were able to assemble large cohorts of cigarette

smokers and non-smokers among British physicians as the exposed and non-exposed groups. Physicians were studied because of the relative ease with which they could be followed reliably. These groups were then followed over time for the occurrence of lung cancer and related diseases. Rates of lung cancer in the two groups then were compared.

Unfortunately, the majority of modern cohort studies are not this well-designed. Much of cohort study research is now done in occupational settings. For example, an investigator might wish to study the risks of various diseases in an occupational group (cohort) such as farmers. A group of farmers is selected and then followed over time. For issues of feasibility, occurrence of cancer in the cohort might be assessed from death certificates rather than from medical records of affected farmers. Again for issues of feasibility, comparisons of disease occurrence in the exposed cohort will be made with occurrence of the disease(s) of interest in the general population in the region where the farmers reside. In this example, the specific exposures of the individual farmers may not be known and may have to be imputed. The use of mortality as a study endpoint is fraught with difficulty for those diseases where survival is good. Mortality endpoints would provide only a partial picture for NHL where the majority of patients survive. Factors such as access to medical care, local availability of the newest therapies, health knowledge of the groups studied, etc., could greatly skew the results of mortality studies. Another problem with cohort studies is that they frequently assess deaths (or less commonly disease incidence) from many diseases. Just on the basis of chance, many associations can be observed because of the large number of disease (mortality) endpoints assessed. This is analogous to the criticism of case-control studies in which the evaluation of multiple exposures often lead to many chance associations.

4. Temporality of the association;
5. Demonstration of a biologic gradient;
6. The plausibility of the association;
7. The coherence of the association;
8. Experimental evidence; and
9. Analogy to the association.

The first of these criteria (Hill called them "viewpoints") is an evaluation of the **strength of an association (1)**. It is good common sense that the stronger an observed association (for example, the higher the relative risk) the more likely the association is to be a causal one. Weak associations, for example, an odds ratio or relative risk of less than 2.0 are unlikely to be causal. Weak associations are often the product of bias, confounding or chance, and tend not to be reproducible in other studies. Observed statistical associations due to confounding factors generally tend to have relative risks less than 2.0, and to be highly inconsistent from study to study. Strong associations, for example, an odds ratio of 3.0-3.5 or higher, are much more likely to be valid and causal. The stronger the observed association, the more likely the finding is to be reproducible and to stand the "test of time" (i.e., to be consistent and reproducible in different studies involving different populations).

Consistency of an association (2) is a very persuasive strut for establishing causality of an association. The scientific method involves the establishment of an hypothesis and then the testing and retesting of the hypothesis in different and/or multiple settings. Only after the hypothesis has been tested and confirmed several times does it enter the realm of established science. The consistent demonstration of an association in

different studies is a strong piece of evidence that a finding is valid and likely to be causal.

The **specificity of an association (3)** is another consideration in assessing whether or not an observed association is a causal one. The more specific an association is between a given exposure and a single disease, the more likely the exposure is to be a causal one. The specific exposure of interest should be found to be associated with the specific disease of interest only. This criterion must be interpreted cautiously, since some factors have been found to be causally associated with more than one disease. For example, cigarette smoking has been found to be causal not only for lung cancer, but also laryngeal cancer and several other cancers. On the other hand, for many infectious diseases, very specific associations have been observed between an infectious agent and a single disease, and this specificity has been useful in establishing causality.

The **temporality** (4) of an association is of course essential to establishing causality. To be a causal association, the cause always should precede the disease. It is simple common sense that the cause should precede the outcome if the association is a causal one.

Demonstration of a **biologic gradient** (5) (i.e., a dose-response relationship) is suggestive of an association being valid and causal. Again, it is simple common sense that if something causes a disease, the greater the dose the greater the risk of disease should be. While the absence of a demonstrable dose-response gradient does not rule out causality, finding a strong biologic gradient is suggestive of an association being causal.

An association is more likely to be a causal one if the finding is **biologically plausible (6)**, which means it makes sense in light of what is known about the biology of the disease and the suspected cause. It is another common sense criterion.

Along these same lines of reasoning is the criterion of the **coherence of an association (7)**. What Hill meant by this is that an observed cause-effect relationship should be coherent with what is known about the biology and natural history of the disease.

One of the most persuasive of the criteria for establishing causation is the availability of supporting **experimental evidence (8)**. An example I use in teaching relates to the hypothesis that fluorides protect against dental cavities. There were many descriptive studies of the association of naturally occurring fluorides in water supplies and risk of dental disease. The causal association was proven and accepted into the mainstream of established science after a community experiment was performed that demonstrated a protective effect of adding fluorides to a city water supply.

The demonstration that **analogous associations (9)** exist is perhaps the weakest of the criteria. The demonstration of analogous causal studies is limited only by the imagination of investigators.

While there is no magic scoring formula for how well these criteria apply to a given finding, and not all of them need to be met to establish causality, they have proven highly useful to generations of epidemiologists and other scientists. In most cases, an investigator can apply these criteria and make a reasonable judgment about how well they are met and how likely the association tested is to be a valid and causal one. The only criterion that is absolutely necessary to prove causality is that of temporality - the cause

always should precede the outcome. In essence, these criteria provide common sense assessments of an association. For example, if an association is valid and causal, investigators ought to be able to demonstrate the association consistently. Most scientific papers published today will include an assessment of the consistency of their findings in the paper's discussion section. The observation of a dose-response relationship between an exposure and an outcome is usually highlighted in a paper and makes it more likely to be accepted for publication. While typically these discussions in scientific papers are not specifically labeled as applications of the Hill criteria, they are thoroughly accepted and ingrained in current generally accepted scientific methodology and are considered strongly in the peer-review process.

It should be pointed out also that the "scientific method," dating back to the times of Pasteur and Koch, involves the need to replicate observations and demonstrate consistency of such observations before they can be established as causal. Some of the "Hill criteria" antedate Hill and have been accepted scientific methodology for well over a century. Hill simply updated scientific methodology for assessing causality of observed associations.

VII.

STATUS OF CURRENT KNOWLEDGE ON CAUSATION OF THE NON-HODGKIN LYMPHOMAS

A. The NHLs are Forty or More Different Diseases with Different Characteristics, Pathology, Treatment and Prognosis

While HL and NHL are sometimes grouped together under the rubric "lymphomas," they are very different diseases with different presentations at diagnosis, different clinical courses, different treatments, different incidence and survival rates,

different risk factors and very different patterns of occurrence (i.e., descriptive epidemiology). It is likely that they will be shown to have different etiologies in the future and that the two diseases will be found to have viral etiologies.

The NHLs are actually 40 or more different diseases with different microscopic anatomical features and different immunologic characteristics. Additionally, the component lymphomas have different natural histories, prognoses, treatments and risk factors. Probably as a result of this important issue, relatively little is known about the causes of NHL and the uncertainty regarding the causes of NHL is widely recognized. Researchers in the lymphoma field increasingly are recognizing this and, as a result, therapies are being tailored individually to specific subsets of lymphomas identified by cancer biomarkers. Epidemiologists also are recommending that future studies divide the lymphomas into subgroups based on biologic features that may define homogeneous, separate component types of lymphomas. Some studies of NHL subtypes have been done utilizing relatively crude biomarkers such as chromosomal translocations. This is in contrast to recent clinical studies that use more complex and sophisticated lymphoma biomarkers for subclassification of the disease. The use of sophisticated biomarkers for lymphoma classification is a very active area of new research.

It increasingly is being recognized that the general failure to find causes of the NHLs may be due to the artificial lumping together of different diseases with different biologic features and different risk factors. As a result, associations for one subgroup may be masked by finding no associations for other different subgroups merged into the overall category of NHL. A problem with proposed studies of subgroups of NHLs is

that the individual component diseases are each uncommon to rare. This poses significant problems for conduct of case-control studies where case accrual becomes difficult and usually requires multi-center studies, and also for cohort studies where cohorts will need to be extremely large to find sufficient numbers of specific component NHL diagnoses as outcomes.

B. <u>DDT is Not One of the Established Causes of NHL</u>

The established causes of NHL include:

1. Severe immunodeficiency (congenital, iatrogenic or acquired);
2. Infectious agents:

   A. Viruses: The Epstein-Barr virus (EBV); the human T-lymphotropic virus, type one (HTLV-1); Kaposi's sarcoma herpes virus (KHSV); human immunodeficiency virus, type one (HIV-1); hepatitis C virus (HCV)

   B. Bacteria: Helicobacter pylori
3. Family history of NHL and to a lesser degree, Hodgkin lymphoma or multiple myeloma;
4. Iatrogenic factors such as chemotherapy;
5. Certain rare medical conditions (such as Sjogren's syndrome, Felty's syndrome, Crohn's disease).

No causes beyond these factors have been established to a reasonable degree of scientific certainty, and the cause of most NHLs is unknown and can be considered to be spontaneous occurrences. While the literature is replete with other risk factors for the NHLs, none of these observations has been consistent or has demonstrated strong associations, let alone demonstrating any consistent dose-response for their observations. Beyond these five broad categories of established NHL, and some reported associations of NHL with rare genetic syndromes, there are no scientifically

established or accepted known causes of NHL. Obesity and diabetes are increasingly being recognized as potential causes for NHL, and as discussed in Section XI, these factors should have been considered by the plaintiff's experts in light of the biased methodology they employed.

There is a voluminous scientific literature on associations between various occupations and specific occupational exposures and risk of NHL, but this literature is characterized by an almost total lack of consistency of results from study to study and typically weak associations. Scherr and Mueller make this point very well in a chapter on the epidemiology of NHL in a leading textbook on cancer epidemiology (Schottenfeld & Fraumeni, 2nd ed.). They present two tables that run for 6 ½ pages that summarize the literature on NHL and various occupations and occupational exposures. The most striking features of the tables include: (1) The large number of diverse occupations and exposures that have been reported as associated with NHL; (2) The lack of consistency of these results; and (3) The general lack of statistical significance and the weakness of these associations, with few relative risks or odds ratios above 2.0. (see the extensive review by Alexander, et al. 2007).

This summarizes what is currently known about the causes of NHL. For most patients with the disease, there is no established cause and their disease is considered to be of spontaneous origin. Comprehensive reviews of the literature on the epidemiology and causation of NHL have been written by leading scientists in the field (Melbye and Trichopoulos, 2002; Hartge and Wang, 2004; Grufferman, 2003; Scherr and Mueller, 1996; Hartge, et al., 2006; Alexander, et al., 2007). Importantly, none of these authoritative textbook chapters or comprehensive reviews reaches a conclusion that

DDT is a cause of NHL. Most of these reviews do not even mention DDT. Leading scientists in the field of NHL research simply do not recognize DDT as a cause of or contributing factor to NHL.

VIII.

DDT AND NHL IN HUMANS – A REVIEW OF THE SCIENTIFIC LITERATURE

Against this backdrop of what are the known causes of NHL, I will next review the scientific literature on the topic of DDT and NHL in humans. There are 40 scientific papers in total on this topic—a very large and comprehensive data set. The 40 papers can be characterized as follows:

1. Reports of Experiments/Human Feeding Studies (N = 2);
2. Reports of Analytic Studies (N = 36);
    a. Case-control study reports (N = 29)
    b. Cohort study reports (N = 7)
3. Descriptive/Ecologic Study Reports (N = 2)

The 40 papers represent all of the scientific literature that I have located to date and evaluated in connection with developing my opinions. In my efforts to identify all pertinent epidemiologic literature relating to DDT and NHL, I have searched the PubMed and other scientific literature databases and reviewed bibliographies of numerous scientific articles on related topics. Epidemiologic studies included in my analysis are restricted to those where specific information and data on DDT exposure and NHL occurrence were provided. These epidemiologic studies relating to the question of whether or not DDT is associated with NHL are identified here and discussed in detail in Attachments 1 and 2; no studies are excluded based on the magnitude of their risk

estimates. After an extensive analysis of this literature, I conclude that DDT is neither a cause nor a contributing factor to NHL, including follicular lymphoma, in humans.

All human experiments involving DDT are included. With the exception of the experimental studies, my discussion of the literature is confined to the topics of DDT and NHL—Montrose's sole product was DDT, and Ms. Garza has NHL. Studies examining occupational groups with presumed DDT exposure, or evaluating pesticides as a group, or evaluating hematopoietic malignancies as a group, without specific findings as to DDT and NHL, are excluded generally. For example, papers involving farmers as an occupational group, but providing no specific information on individual farmer's exposure to DDT, are not included. Farmers are exposed to a multitude of fuels, solvents pesticides, herbicides, viruses, etc., and it is not possible in most studies to tease apart the individual role of each exposure. The failure of such papers, as well as papers on pesticide applicators, to identify the specific and actual exposure of subjects to DDT, makes them inevaluable as to whether DDT is a cause of NHL. Similarly, papers that fail to specifically identify NHL as an outcome are inevaluable and were excluded. For example, papers that only report findings for the overall group of hematopoietic malignancies and not NHL specifically were excluded. Proper scientific evaluation of the literature requires that specific information on DDT and NHL be provided in the papers considered.

In Section A below, I first discuss the two human feeding studies that were conducted on DDT in the 1960-70s. Then, in Section B, I summarize my conclusions as to DDT and NHL after a full evaluation of the 36 analytic reports and two ecologic studies.

A. Experiments: The Feeding Studies of DDT to Humans Provide Evidence that DDT is Safe for Humans Even at High Doses

The pesticide DDT was for many years the single most widely used chemical for insect control and was particularly valuable for mosquito control in the management of malaria and typhus. It was believed to be safe for humans. DDT was presumed so widely to be harmless to humans that the U.S. Government conducted human feeding studies with the results published in leading medical journals.

The first study was conducted by the Communicable Disease Center of the U.S. Public Health Service, which was the precursor to the current U.S. Centers for Disease Control and Prevention, beginning in 1954 (Hayes, et al., JAMA, 1956). This research was conducted on U.S. prison inmates who volunteered to ingest two different types of DDT at differing doses on a daily basis for up to 18 months. Three different doses were administered ranging from 0 to 35mg per day (equivalent to 0.5 mg/kg/day, a dose estimated to be 200 times that to which the general population at the time was exposed). None of the subjects developed any symptoms or developed any sign of illness. The study concluded: "The study results indicate that a large safety factor is associated with DDT as it now occurs in the diet." Thus, the feeding of DDT on a daily basis to human volunteers over an 18-month period resulted in no apparent symptoms or illness and some of the subjects received very high doses of DDT.

A second study also was done by the Communicable Disease Center and started in 1956 (Hayes, et al., Arch Env Health, 1971). This study also used prison inmate volunteers who received the same three dosages as in the earlier study (0, 3.5 mg and 35 mg per day). This research was intended to reevaluate the findings of the first report and assess the storage and loss of DDT over a longer period of time. Twenty four subjects

ingested technical or *p,p'*-DDT at doses up to 35 mg per day for 21.5 months with no laboratory or clinical evidence of injury after lengthy follow-up (ranging from 25.5 months to five years after cessation of their DDT ingestion). The researchers concluded that "...these factors indicate a high degree of safety of DDT for the general population." The authors point out that the average dose of DDT administered was 555 times that ingested by comparably aged men in the general population.

What these two papers suggest is that DDT ingestion in humans was believed by U.S. government researchers in the 1950s to be safe, even at high doses, and their research results support this notion. Follow-up for up to five years after DDT feeding showed no adverse health effects, and there is no mention of cancer occurrence, let alone NHL specifically, in the study subjects. Here, we have one of the few scientific examples of experimental administration of DDT to humans and no harm was demonstrated. This argues strongly against DDT being a cause of lymphomas and other cancers in humans. In fact, it demonstrates that DDT, even at high doses, is safe for humans.

B. Analytic Studies: Case-Control and Cohort Studies of DDT and NHL Show That DDT Is Neither a Cause of Nor a Contributing Factor to NHL

It is important to point out that most epidemiologic studies, both case-control and cohort studies, identify broad occupational categories as suspected risk factors. However, such broad categories usually entail exposure to a very wide range of occupational exposures. Additionally, many such studies use univariate analyses (i.e., analyses that consider only a single variable or exposure at a time) to identify a single exposure as a suspected risk factor when the individuals studied may have been exposed at the same time to a multitude of other factors. As a result, a specific finding could easily be the result of confounding by other environmental exposures. To deal with such problems of

confounding, i.e., an observed association being due to another risk factor to which subjects were also exposed, biostatistical methods have been developed to tease apart the separate effects of multiple exposures in the same individual. Such methods typically involve multivariate analyses that can separate out the individual exposures for the disease being studied. These methods are particularly necessary for proper evaluation of occupational groups such as farmers with their multitude of different occupational exposures. Modern epidemiology increasingly uses multivariate analyses to deal with the important scientific issue of confounding. Thus, I have specifically identified those studies that employed multivariate analyses and have accorded them greater importance than univariate analysis studies in arriving at my causation opinions. Similarly, I have accorded substantial weight to the cohort studies on DDT and NHL, given their ability to estimate DDT exposures more precisely.

1. Case-Control Studies

a. Case-Control Studies Performing Multivariate Analyses

There are eight case-control studies on DDT and NHL that employed multivariate analyses and published results of these analyses (Baris, et al., 1998; De Roos, et al., 2003; Engel, et al., 2007; Hardell, et al., 1994; Hardell, et al., 2002; McDuffie, et al., 2001; Persson, et al., 1993; Rothman, et al., 1997). Each of these papers is summarized and discussed in detail in Attachments 1 and 2, respectively.

As shown, each report controls for other chemical exposures in their analyses. The multivariate analyses in all eight papers demonstrate no causal association between DDT exposure and NHL.

Baris, et al., 1998 demonstrated no association between DDT exposure and risk of NHL in an important multivariate analysis. My interpretation is consistent with the

authors' own concluding statement: "No strong consistent evidence was found for an association between exposure to DDT and risk of non-Hodgkin's lymphoma." De Roos, et al., 2003 similarly found no association between DDT and NHL after adjustment for all other pesticides—OR = 1.0 (95% CI = 0.7-1.3). The Baris, et al., 1998 and De Roos, et al., 2003 studies are important and compelling because of the large number of subjects and controls in their pooled analyses, which provided opportunities for proper statistical evaluation of confounding exposures.

Rothman, et al., 1997, which controlled for confounding exposures, is an excellent example of the importance of collecting pre-diagnosis blood samples and therefore avoiding potential bias and confounding. This study found no evidence of an association with DDT and NHL after controlling for PCBs (non-significant ORs ranged from 1.0-1.2). Engel, et al., 2007, which also used biologic specimens, is another large, pooled case-control study that, after performing multivariate analyses, found no significant associations between NHL risk and DDE in blood samples collected well in advance of NHL diagnosis. The four other studies employing multivariate analyses—Hardell, et al., 1994; Hardell, et al., 2002; McDuffie, et al., 2001; Persson, et al., 1993—also demonstrate no evidence of a statistically significant association between NHL and DDT following multivariate analyses. Thus, the eight studies employing sophisticated, modern epidemiologic methods produced very specific findings, all of which show no statistically significant association between DDT and NHL. Together, this is powerful scientific evidence that DDT is not a cause of NHL.

b. <u>Case-Control Studies Performing Univariate Analyses Only</u>

In addition to the eight reports above with multivariate analyses, there are 21 scientific papers considering DDT and NHL that perform univariate analyses and do not

control for other chemical exposures. Each of these papers is summarized and discussed in detail in Attachments 1 and 2, respectively. As noted in these Attachments, the failure to control for other chemical exposures is a significant limitation of the studies. Nevertheless, an evaluation of these reports further supports the conclusion that there is no valid association between exposure to DDT and risk of NHL, including follicular NHL.

Among the 21 studies are seven case-control studies that examined levels of DDT and its metabolites in biologic specimens (blood or fat samples) as indirect measures of past DDT exposure. Two Swedish studies (Hardell, et al., 1996; Hardell, et al., 2001) found no statistically significant association, while a third study (De Roos, et al., 2005) conducted by NCI showed a statistically significant protective effect in one of their analyses. Additional studies (Cocco, et al., 2008; Hardell, et al., 2009; Quintana, et al., 2004; Spinelli, et al., 2007) showed various and inconsistent results, including non-statistically significant associations. The odds ratios in all of these studies were below 2.0 for DDT and NHL, including follicular NHL. In the aggregate, the results of these seven studies provide no evidence of a causal association between DDT exposure and NHL, including specifically follicular NHL.

In addition to the seven case-control studies in the preceding paragraph, there are 12 case-control studies of DDT and NHL that do not have biologic specimen data, and instead assess exposure by history through questionnaires or interviews. This collection of studies includes several reports examining the same set of subjects, i.e., mid-western farmers and their families from a large NCI study (Cantor, et al., 1992; Cantor, et al., 1993; Lee, et al., 2004; Schroeder, et al., 2001; Zahm, et al., 1993). As pointed out in the

first study by Cantor, et al., 1992, farmers experience a multitude of occupational exposures (i.e., animal viruses, fertilizers, gasoline and diesel fuels, etc.) and multivariate analyses to control for these potentially confounding variables is critical. However, only univariate analyses were done in this group of studies.

These NCI farmer studies and additional studies employing univariate analyses (Colt, et al., 2005; Eriksson, et al., 2008; Hardell, et al., 1999; Persson, et al., 1989; Persson, et al., 1999; Woods, et al., 1987; Woods, et al., 1989) produced remarkably inconsistent results that are largely weak and not statistically significant. The weak findings generally centered around the null value of 1.0, suggesting that the weak associations between DDT and NHL were confounded by other environmental exposures. This was borne out by the multivariate analysis studies which showed uniformly that DDT was no longer associated with NHL when other exposures were controlled for in the analyses. Thus, based on a critical review and analysis of these 12 studies along with the multivariate studies, there is no basis for concluding that a causal association exists between DDT exposure and NHL risk. On the contrary, the evidence suggests otherwise.

Additionally, there are two case-control studies (Miligi, et al., 2003; Nanni, et al., 1996) that assess DDT exposure and risk of a combined category of NHL and chronic lymphocytic leukemia (CLL). Although CLL is currently considered one of the lymphoid malignancies in the new WHO classification, on clinical and epidemiologic grounds, it is a separate disease from NHL. For purposes of completeness, they are included in my review. Like the case-control studies discussed previously, these two studies do not demonstrate a causal association between DDT exposure and NHL. In

fact, Miligi, et al., 2003, found a statistically significant protective effect of DDT exposure in women (OR = 0.3, 95% CI = 0.1-0.8).

In conclusion, the results of all 29 case-control studies, particularly those studies that employed multivariate analyses, show no consistent or conclusive evidence of a causal association between DDT exposure and NHL. Rather, this collection of studies that span over two decades and were conducted in different populations by different investigators using different designs provide compelling evidence that DDT is not a cause of NHL.

2. Cohort Studies of DDT and NHL in Humans

The seven cohort reports studying DDT and NHL also present strong evidence that DDT is not a cause of NHL. See Attachments 1 and 2 for full discussion of these seven studies. As discussed previously, one notable strength of cohort studies is their ability to estimate more precisely exposure to DDT. The cohort studies each studied groups of persons who had high levels of occupational exposure to DDT. Three of the cohort studies, Laws, et al., 1967, Ditraglia, et al., 1981 and Brown, et al., 1992, are particularly important because they studied workers at plants that exclusively manufactured DDT; these workers were very heavily and specifically exposed to DDT. For example, Brown, et al., 1992, reported that the average daily intake of DDT in plant workers ranged from 438 to 450 times that of the general population. Although the sample sizes in these three reports were small, no cases of NHL were observed among DDT plant workers.

No increase in NHL was observed in studies of licensed DDT pesticide applicators or workers exposed to DDT as part of a public health malaria eradication program (Purdue, et al., 2006; Cocco, et al., 1997(a); Cocco, et al., 1997(b); Cocco, et al.,

2005). Again, both of these occupational groups were heavily and directly exposed to DDT. If a causal association between NHL and DDT existed, one would expect to see evidence of it in such heavily-exposed workers. Yet, the four reports of these groups failed to provide any support for a causal association between DDT and NHL.

Thus, the seven cohort reports—which examined the highly-exposed DDT workers and DDT pesticide applicators—each evidence no statistically significant association between DDT and NHL in these groups. There was not one exception, despite these cohorts being extremely heavily exposed to DDT. Together, this is powerful evidence for lack of any causal relationship between DDT and NHL.

C. Ecologic Studies

Finally, consistent with appropriate scientific methodology, where one must consider all relevant literature, I also evaluated the two ecologic studies regarding DDT and NHL (Cocco, et al., 2000; Pavuk, et al., 2004). See Attachments 1 and 2 for full discussion of these studies. While ecologic studies are of limited value in determining risk or causation, they nevertheless also do not support the claim that DDT causes or contributes to NHL, finding no excess of NHL with DDT exposure. In fact, one of the two studies (Cocco, et al., 2000) found a significantly protective effect between tissue DDE levels (DDE is a major metabolite of DDT) and NHL mortality in three of the four groups they studied.

D. In Summary, the Epidemiologic Literature Provides Strong and Consistent Evidence that DDT is Neither a Cause of NHL Nor a Contributing Factor

In summary, the voluminous body of scientific literature addressing DDT exposure and NHL specifically, provides strong and compelling evidence that DDT is neither a cause of nor a contributing factor for NHL. I analyzed all of the experimental

studies, analytic epidemiologic studies and ecologic studies that I was able to locate concerning DDT exposure and NHL, including follicular NHL (Attachments 1 and 2). This totaled 40 scientific papers. As an epidemiologist, I find that both the case-control studies with multivariate analyses and the cohort studies as a whole to be the most persuasive. Following the use of multivariate analyses to control for confounding exposures, not one of these eight case-control studies and none of the cohort studies showed a statistically significant association between DDT and NHL. Based on my extensive review of all relevant literature, together with my professional experience, education, training and background, I conclude to a reasonable degree of medical and scientific certainty that DDT is not a cause of NHL in humans.

My conclusion is consistent with those reached by leading governmental and public health organizations, including IARC (IARC, 1991), the U.S. Environmental Protection Agency (U.S. EPA, 1991), Agency for Toxic Substances and Disease Registry (ATSDR, 2002), and the National Toxicology Program (NTP, 2005), none of which conclude that DDT is a human carcinogen. Leading scientists in published, peer-reviewed literature (see the extensive review by Alexander, et al., 2007) also are in agreement with my conclusion.

In addition, none of the papers that I located in the literature on the subject of DDT exposure and NHL provide any evidence on DDT being a contributing factor to NHL. While plaintiff's experts may claim that there is some interaction or additive effect that occurs when a person is exposed to DDT, BHC/lindane, dieldrin and/or toxaphene in some combination, I have identified no evidence in the literature supporting such an assertion. De Roos, et al., 2003 was the only paper that considered interactions among

any of these chemicals, including the potential interactions between DDT and aldrin, chlordane, and lindane. The authors reported their findings for chlordane and DDT, which showed no significant interaction. The authors did not present data for their other DDT interaction analyses, suggesting no interaction among these pesticides. This report therefore provides no specific evidence of interaction between DDT, aldrin and lindane. Thus, it is also my opinion, to a reasonable degree of medical and scientific certainty, that DDT has not been proven to be a contributing factor to NHL in humans.

E. The DDT and NHL Epidemiologic Literature Fails to Meet the Causation Criteria

Finally, Montrose requested that I consider whether or not well-designed, properly conducted DDT and NHL studies overall report a risk estimate greater than 2.0 that is statistically significant at the 95% level such that the confidence limits do not include 1.0. I understand these specific criteria for assessing causation are based on a judicial decision, Merrell Dow Pharmaceuticals, Inc. v. Havner, Texas Supreme Court (1997). These criteria are consistent with the approach that I and the epidemiologic community typically use in the evaluation of research results.

As noted previously, the DDT and NHL literature set contains 40 different reports. In epidemiology, this constitutes a large study set from which we can draw definitive conclusions. Despite this considerable body of literature:

- There is not one cohort study report that evidences a statistically significant doubling of the risk. (0/7 reports);
- There is not one case-control study that evidences a statistically significant doubling of the risk following multivariate analysis. (0/8 reports);

- There is not one ecologic study that found a statistically significant doubling of the risk. (0/2 reports)
- The DDT experiments, where humans were fed large amounts of DDT, also found no increase in cancer risk. (0/2 reports)
- There are 21 case-control reports with only univariate analysis (i.e., they fail to control for relevant confounding exposures by using more sophisticated statistical analyses). Yet, just 3 (Cantor, et al., 1992; Eriksson, et al. 2008; K. Hardell, et al., 2009) of the 21 reports contain any statistically significant finding above 2.0 among their multitude of findings on DDT. Thus, in 18 reports, there is not one finding which evidences a statistically significant doubling of the risk, despite not controlling for critical confounders. In the three reports referenced above, each performs numerous analyses, making it likely that results reported may be due to chance and/or confounding. The authors of Cantor, et al., 1992 recognize and discuss this problem in their paper. Consequently, when Baris, et al., 1998 and De Roos, et al., 2003 evaluated the same data as Cantor, et al., 1992 using multivariate analyses, they found no significant association between DDT and NHL. Hardell, et al., 2009 and Eriksson, et al. 2008 suffer from similar deficiencies in that they perform numerous subset analyses that increase the likelihood of chance findings and/or are of unknown relevance to Ms Garza, i.e., risk estimates for EBV EA antibodies. In sum, the vast majority of findings as to DDT and NHL within these 21 reports also do not meet these criteria. (3/21 reports).

In my 42 years of professional experience as an epidemiologist, I have rarely seen a literature set as large as DDT and NHL which has such overwhelmingly negative results. There are 40 total studies—37 of which fail the "doubling of the risk" test completely, and the other three of which are not only obvious outliers, but also need to be interpreted cautiously due to their failure to control for confounders and their performance of multiple subset analyses. Thus, despite decades of scientific research, DDT has neither been proven to be a cause of NHL in humans nor a contributing factor to NHL in humans.

IX.

REPORTS OF THERAPEUTIC USE OF DDT AND/OR ITS ANALOGS

Also of interest are two additional studies (three reports) that demonstrate the safety of administration of DDT and/or its analogs to treat human patients. The first study is a case report in the Lancet from 1969 that reported the use of dicophane, which contains no less than 70% of DDT, in the successful treatment of a young man with unconjugated hyperbilirubinemia (a type of jaundice) (Thompson, et al., Lancet, 1969). DDT was considered to be extremely safe and its use was widespread in the 1960s, and even led to the use of DDT as a treatment for jaundice.

Interestingly, the use of a DDT compound for treatment of disease in humans continues to this day. A second, very recent paper in the prestigious New England Journal of Medicine (Terzola, et al., 2007) reported on the use of mitotane ("a synthetic derivative of the insecticide dichlorodiphenyltrichloroethane (DDT)") in the successful treatment of adrenocortical carcinoma. Mitotane is chemically termed *o,p'*-DDD which is important since DDT is metabolized in humans to DDD. DDD is further broken down in the body and does not persist very long, so it does not accumulate in human tissues.

The major adverse effects of mitotane treatment were gastrointestinal symptoms and neurologic symptoms in patients receiving high-dose therapy. Patients on lower dose therapy had none of these symptoms.

This is a modern-day clinical study of a DDT derivative for treatment of cancer in humans that was considered ethical to conduct. An accompanying commentary (Schteingart, N Engl J Med, 2007) states that the study "provides a compelling rationale for the use of low dose mitotane as adjuvant therapy" in adrenocortical carcinoma. Thus, it is likely that a DDT derivative will become part of the standard treatment of this rare human malignancy. Neither of these two New England Journal papers, written by oncologists, raises any concern about risk of NHL in patients receiving mitotane.

These three papers demonstrate that the administration of DDT analogs is considered safe for treatment of medical patients and has therapeutic value.

X.

REVIEW OF PLAINTIFF'S MEDICAL HISTORY

A. Ms. Garza's NHL Has Had A Typical And Uncomplicated Course And She Has Survived Ten Years Since Diagnosis

I have reviewed the available medical records, deposition testimony, replies to interrogatories, etc., that were provided to me for the plaintiff, Guadalupe Garza, who has alleged that she developed NHL as the result of environmental exposure to DDT from the Hayes-Sammons facility in Mission, Texas. I have had extensive experience in reviewing patient medical records as a practicing physician, having conducted NIH-funded, peer-reviewed epidemiologic research using medical records (on the hematologic malignancy, multiple myeloma), and having served for ten years as a member of the Medical Records Committee of the Duke University Medical Center.

Ms. Guadalupe Garza was born on February 19, 1943, and was diagnosed with NHL on December 2, 1998, at the age of 55 years. This is a common age for the diagnosis of NHL in adults.

Ms. Garza reports the following residential history: She was born in Mission, Texas and lived there for most of her life. During the period 1969-1975, she lived in Wheeling, Illinois. She returned to Mission in 1975 and she still resides there. From 1943 to 1957, she states in her interrogatories that she resided at 506 Nicholson, in Mission. From 1957 to 1967, she further indicates that she lived at 423 Canal Avenue and, from 1975 to the present, she has resided at 1015 Nicholson in Mission.

Ms. Garza reported that she assisted her husband in cleaning offices while they lived in Illinois. She reported having used ordinary household cleaning products in the work. Upon returning to Mission in 1974, she stated that she worked at Kenneth White Junior High School and kept this job until the 1990s. She was next employed by the La Joya School in La Joya, Texas. Her job duties at both schools were selling snacks to students. This appears to be the extent of her occupational history. Her husband worked for the post office in Illinois and cleaned offices as a second part-time job.

Ms. Garza's medical history reveals that she has had a typical and uncomplicated course of her NHL and has survived almost ten years since diagnosis. Moreover, she has problems with morbid obesity, diabetes, hypertension and dyslipidemia, all of which antedate her NHL. The history of her NHL is as follows: In August, 1998, she underwent mammography and a moderately enlarged left axillary lymph node was detected. A follow-up breast ultrasound examination was done one month later, and multiple hypoechoic masses were observed in the left axilla. A biopsy was

recommended. A needle biopsy was performed on November 10, 1998, at Rio Grande Regional Hospital, and the specimen was felt to be benign, but due to the small amount of biopsied material examined, an excisional biopsy was recommended.

On December 2, 1998, Ms. Garza underwent an excisional biopsy of left axillary lymph nodes, also at Rio Grande Regional Hospital. This tissue was diagnosed as a malignant lymphoma, follicular (nodular), of B-cell type, after immunophenotyping the specimen. She was referred to Dr. Suresh Ratnam, currently with the South Texas Cancer in McAllen, for further evaluation, staging and treatment. She underwent a bone marrow biopsy and was found to have marrow involvement by her lymphoma, which was confirmed to be a B-cell tumor of probable follicular center cell origin and thus a follicular lymphoma. A history of sweats and fatigue was obtained making her disease of the "b-type." Extensive staging examinations were done, and she was classified as having stage IV-b disease.

She was started on combination chemotherapy (FND) on January 6, 1999, and completed six cycles of this course of treatment on May 7, 1999. She had a "good response" to the chemotherapy, and she was next started on Rituxan therapy on May 24, 1999. Ms. Garza completed eight weekly doses of Rituxan and then was placed on maintenance alpha-interferon therapy in July, 1999. On September 22, 1999, she was found to be in complete remission from her disease. The alpha-interferon therapy was discontinued in August, 2001, and she had no evident disease at her last follow-up whole body PET scan evaluation in March, 2002. At this point, she was in complete remission, and remained in complete remission as of March, 2004.

In August, 2004, she was found to have recurrent active lymphoma and was restarted on Rituxan therapy. By September, 2005, her oncologist (Dr. Ratnam) reported no convincing evidence of active disease. In January, 2006, she was again noted to have active NHL and her oncologist switched her treatment from maintenance Rituxan to a full course of the drug. In January, 2007, she was also placed on high dose prednisone therapy. At that time her physician noted that she had mildly progressive, low volume Stage IV-B follicular lymphoma. In March, 2007, she was found to be responding to her therapy. PET and CAT scans performed in June, 2008, showed evidence of increased lymphadenopathy consistent with progressive lymphoma. A new course of Rituxan and CVP therapy as well as monthly IVIG for hypogammaglobulinemia was prescribed. As of December 18, 2008, she was responding well to her chemotherapy. She was on chemotherapy at the time of her last available medical record.

She is currently alive 10 1/2 years after her diagnosis. Ms. Garza's clinical course is typical of a well managed adult follicular NHL patient. She has had responses to therapy (remissions) and relapses of her disease with few clinical symptoms. Her relapses have responded to therapy and it is likely that she will continue to do well. She does not appear to be in imminent danger of death as a plaintiff's expert has opined. The last medical records of Ms. Garza that I reviewed were dated January 9, 2009. I reserve the right to supplement this report if and when any additional medical records are provided.

A review of her general medical history is as follows: Her past medical history and review of systems are marked by a history of extreme obesity, with body weights up to 270 pounds (on 7/2/05 and 5/11/06), with a height of 63-65 inches, even after

chemotherapy. Her last recorded body mass index (BMI) was 52.33 on 11/14/05. This was after chemotherapy and before her diagnosis of Type II diabetes. A body mass index over 40 is classified as extreme or "morbid" obesity. Her severe obesity antedated her diagnosis of NHL, with weights as high as 241 lbs. on 4/1/98. Her height at that time was 65 inches, resulting in a calculated BMI of 40, which is the lower bound of extreme obesity. She was diagnosed with Type II diabetes on 5/1/06, although she had borderline to frankly abnormal blood glucose levels for two years before diagnosis. Severe obesity, as Ms. Garza has had for many years, is a major risk factor for Type II diabetes. She additionally has a long history of treated hypertension and osteoarthritis, which are also associated with extreme obesity. Well before her NHL diagnosis she was noted to have dyslipidemia (2/20/89). The combination of obesity, particularly abdominal obesity, dyslipidemia, and hypertension has been termed the "metabolic syndrome," a condition that markedly increases the risk of atherosclerosis and its complications. At her last medical visit on 12/18/08, her weight was 258 lbs. Her morbid obesity is an important consideration in understanding the potential causes of Ms. Garza's NHL given that obesity is a potential risk factor for NHL (Alexander, et al., 2007; Hartge, et al., 2006).

Ms. Garza has no history of prior malignancies, kidney disease, hepatitis, HIV infection, infectious mononucleosis, tuberculosis, or asthma. She has had two TIAs (transient ischemic attacks), the first on 3/31/97 and the second on 6/5/08. Evaluation of her carotid arteries on 4/7/97 revealed mild plaques of both carotid arteries with no stenosis. A TIA is similar to a mild stroke of short duration (less than one hour) and is typically due to emboli from stenotic carotid or vertebral arteries. In 1985, she complained of shortness of breath, chest pain and palpitations, and underwent a cardiac

stress test and 24-hour cardiac monitoring both of which were normal. She was placed on Inderal therapy at that time and her symptoms subsided. She reports that she does not smoke or drink alcoholic beverages. She has undergone a prior hysterectomy. There is a positive family history of heart disease and her paternal grandmother died of stomach cancer. She was on atenolol, Clinoril and Premarin therapies at the time of her diagnosis.

As part of routine re-evaluation of her lymphoma status, she underwent a CT examination of her abdomen on 8/19/08. A mass was noted in her left kidney that was enlarged since first noted on 1/3/07. As a result, she underwent a biopsy on 9/23/08 that revealed renal cell adenocarcinoma, clear cell type. She underwent radioablation therapy for this tumor and the mass was stable on repeat CT examination on 1/09/09.

B. <u>DDT Did Not Cause or Contribute to Ms. Garza's NHL</u>

Because DDT is not a cause of or a contributing factor to NHL, Ms. Garza's NHL was not caused by DDT. In fact, using the occupational studies that I evaluated in this report as a reference point, it is evident that Ms. Garza's alleged exposure could not possibly have caused NHL. Specifically, Ms. Garza alleges exposure to DDT while residing in proximity to the Hayes-Sammons facility, thus she claims to have had only residential exposure. There is no evidence that Ms. Garza was a farmer, pesticide applicator, chemical plant employee or that she experienced any type of occupational exposure to DDT. Furthermore, it should be emphasized that there are no direct measurements of Ms. Garza's alleged exposure levels from the actual time of these purported exposures.

We know from the occupational cohort studies that extremely high levels of exposure to DDT, which are orders of magnitude higher than any type of residential exposure that Ms. Garza might have experienced, do not cause NHL. Therefore, even

assuming she had levels of exposure equivalent to those experienced by members of the occupational cohorts, which she did not, such exposure would not have caused her NHL. Consequently, Ms. Garza's claim that her NHL was caused by presumably much lower levels of exposure is scientifically unfounded.

Furthermore, my conclusion that DDT is not a cause of Ms. Garza's NHL is consistent with studies performed by the Texas Department of Health (TDH). Starting in 1998, TDH performed a series of surveillance studies in Mission, Texas. Specifically, in addition to various other cancers, TDH evaluated NHL incidence and mortality in the Mission, Texas zip code (78572). Based on their most recent evaluation from March 31, 2005, NHL incidence and mortality for men and women during the period 1993 - 2002 were all below the rates expected in the general Texas population (TDH Report, 2005). The standardized incidence ratios (SIR) were 0.76 in men and in women and the standardized mortality ratios (SMR) were 0.69 in men and 0.76 in women. These data clearly show that not only were there no increases in NHL incidence and mortality in the Mission, Texas zip code, but that there were fewer than expected cases and deaths. If the plaintiff's allegations regarding the Hayes-Sammons facilities were correct, we would expect to see increased cancer rates in the area around the plant. In fact, the most salient findings of the TDH surveillance studies showed statistically significant deficits of incidence and mortality for many cancers.

XI.

PLAINTIFF'S CAUSATION EXPERTS HAVE NOT USED ACCEPTED SCIENTIFIC METHODOLOGY IN REACHING THEIR OPINIONS

I have reviewed the reports and deposition testimony of plaintiff's experts, Drs. Richard Clapp, Theodore Farber, Frank Gardner, and William Sawyer. In sum, I find

that their reports and testimony completely fail to demonstrate a causal relationship between DDT exposure and the development of NHL—much less show that Ms. Garza's alleged exposure to DDT caused or contributed to her development of NHL. Plaintiff's experts have a serious flaw in common—the failure to use accepted scientific methods in reaching their opinions. More specifically, plaintiff's experts have: (1) failed to systematically search for and analyze the relevant literature; and (2) failed to systematically and critically review even the narrow subset of literature they do cite.

Not surprisingly, these limitations in methodology have resulted in seriously misleading expert reports and opinions. The four Plaintiff's experts rely on a biased and narrow selection of the relevant epidemiologic studies to support their opinions; further, they selectively cite and mischaracterize findings within this narrow range of studies. Plaintiff's experts have also cited numerous studies that are simply irrelevant. Finally, Drs. Gardner and Sawyer have ignored potential alternative risk factors that may have caused or contributed to Ms. Garza's development of NHL.

With these concerns in mind, I discuss the reports and testimony of each of these experts in turn.

A. Dr. Richard W. Clapp Has Not Used Accepted Scientific Methodology in Arriving at His Opinion on DDT and NHL

Dr. Clapp authored a curious report in 2005 to support his opinion that the organochlorine pesticides at issue are capable of causing the development of NHL. Foremost, of the 26 pages of text in Dr. Clapp's report, more than 19 pages are devoted to a lengthy philosophical discussion in which Dr. Clapp eschews traditional reliance on statistical significance and p-values and purports to follow the "Hill guidelines" for assessing causal relationships (Richard Clapp's Expert Report ("2005 Clapp Report"),

February 8, 2005, pages 1-19). Dr. Clapp's opinion regarding statistical significance is not surprising, given that the bulk of epidemiologic evidence in this case does not reach statistical significance. The overwhelming majority of the scientific community views statistical significance much differently than Dr. Clapp. Indeed, in any leading scientific journal—such as the highly-regarded New England Journal of Medicine—the authors of virtually every scientific article present p-values and/or confidence intervals. Despite his denigrating the use of p-values, Dr. Clapp himself relied on p-values in his evaluation of the scientific literature presented in his report (2005 Clapp Report, pages 24-26).

Dr. Clapp has completely failed to employ any scientific methodology. Though Dr. Clapp engages in lengthy philosophical discussion and interpretation of the Hill criteria in his report, he failed to explain how he employed the Hill criteria to reach his opinions (2005 Clapp Report at 20-26). The supplemental report Dr. Clapp filed in 2008 is similarly devoid of any explanation of his methodology (Supplemental Report of Dr. Richard Clapp ("Clapp Supplemental Report"), December 5, 2008). During deposition, Dr. Clapp claimed that he employed the Hill guidelines in excluding studies, but admitted that he did not document this process with respect to any individual pesticide or group of pesticides (Deposition of Dr. Richard W. Clapp ("Clapp Deposition"), pages 189:1-190:16).

Dr. Clapp has employed no consistent or precise methodology in his review of the literature. Dr. Clapp claimed that he has excluded "animal studies," older studies employing dated methods, and studies that have been questioned or deemed unreliable (Clapp Deposition, pages 74:4-75:1). Dr. Clapp purports to have conducted Medline searches to identify literature on NHL and pesticide exposures. Despite this

methodology, he inexplicably ignores nearly two-thirds of the studies relevant to assessing the possible relationship between DDT and NHL. Simply put, Dr. Clapp has selected epidemiologic literature that supports his conclusions. For example, I have identified 40 studies relevant to whether or not there is a causal connection between exposure to DDT and NHL development; Dr. Clapp has cited only 13 of these studies. Furthermore, regarding the 13 studies Dr. Clapp does cite, he omits the findings for DDT at least six times (2005 Clapp Report, pages 20-26; Supplemental Report of Dr. Richard Clapp, dated December 5, 2008, pages 1-2). When questioned about several of the DDT/NHL studies he ignored during deposition, Dr. Clapp admitted their relevance and—not surprisingly—had difficultly explaining his failure to consider them in reaching his opinions in this case (Clapp Deposition, pages 79:12-23; 80:24-81:3; 107:21-109:2; 81:7-18).

Dr. Clapp's biased selection of results within studies is equally problematic, especially given the narrow range of studies he cites. He seemingly recognizes that, in studies that adjusted for exposure to other potential risk factors—performing, in other words, a multivariate analysis—odds ratios regress towards the null and become statistically insignificant for DDT and NHL (for example where he discusses Hardell, et al., 1994 and Baris, et al., 1998) (2005 Clapp Report, pages 21-22). Yet, in several instances, Dr. Clapp failed to cite the results of multivariate analyses. As an example, he ignores the multivariate analysis findings in De Roos, et al., 2003 and McDuffie, et al., 2001 (2005 Clapp Report, pages 20-26). Similarly, Dr. Clapp has routinely selected only the highest odds ratios from within particular studies, ignoring many other findings. For example, with respect to De Roos, et al., 2003, he cites the findings for chlordane,

lindane, and toxaphene, but ignores the DDT findings that show no association (2005 Clapp Report).

Dr. Clapp also cites several studies that are simply not relevant to whether or not a causal association exists between a particular disease and a specific agent like DDT. For instance, he cites a study in Utah linking farming occupations to development of NHL, apparently relying on farm work as a "surrogate" for exposure to organochlorine pesticides (Schumacher, et al., 1985) (Clapp Report, page 20; Clapp Deposition, pages 147:6-148:1). Of course, farm-workers are exposed to a host of potential exposures, including animal viruses, gasoline, fertilizers, herbicides, and non-organochlorine pesticides. Thus, farm work cannot be used reliably as a surrogate for DDT exposure when trying to assess whether or not a causal relationship exists between DDT and NHL.

Finally, Dr. Clapp purportedly relies on five studies to meet the Havner criteria for demonstrating causation between DDT and NHL (Clapp Deposition, Exhibit 42). First, a proper scientific analysis and methodological evaluation should have considered all 40 studies in reaching a conclusion—not simply the five that Dr. Clapp claims support his opinion. Second, contrary to Dr. Clapp's contention, the five identified studies do not meet the criteria outlined in the Havner decision:

- Dr. Clapp cited Baris, et al., 1998 and culled one finding from dozens of non-elevated, non-significant findings; when a multivariate analysis was performed, as Dr. Clapp admits, the same finding was neither elevated nor significant—the authors themselves concluded that there was no strong consistent evidence of an association between DDT and NHL;

- Dr. Clapp cites only one of dozens of findings in Cantor, et al., 1992, though the same subjects were evaluated in two subsequent studies that used more sophisticated, multivariate analysis and found no associations (Baris, et al., 1998, and De Roos, et al., 2003);
- He cites Colt, et al., 2005 for its T-cell lymphoma findings, though (1) T-cell lymphoma is not the type of NHL Ms. Garza has, (2) the follicular lymphoma findings, which Ms. Garza has, were not significant and close to the null, and (3) the authors found no association for women;
- He cites Hardell, et al., 1994 for a single univariate finding, and ignores (1) the non-significant results of the multivariate analysis that controlled for other chemicals, and (2) the authors' conclusion that DDT was not associated with an increased risk for NHL; and
- He cites Engel, et al., 2007 for a single unadjusted DDE finding out of many analyses, and then fails to note that the significance of this one finding disappeared when adjusted for exposure to PCBs and also ignores the numerous other findings in the paper itself and its extensive supplement. Dr. Clapp's use of the Engel paper is an extreme example of highly selective use of one finding out of a multitude of findings to arrive at a causation opinion.

(Clapp Deposition, pages 149:3-150:8; 159:16-25).

In sum, Dr. Clapp has not used accepted scientific methodology to arrive at his opinions regarding DDT. He has omitted dozens of relevant studies and even ignored the conclusions in his own writings. Indeed, though Dr. Clapp has published several papers or chapters discussing the purported causes and risk factors for cancers (including NHL),

he has never claimed in his published work that DDT causes NHL. Given his incomplete, selective and distorted review of the scientific literature, Dr. Clapp's opinions are unscientific, unreliable and insufficient to demonstrate any link between DDT and NHL.

B. Dr. Theodore M. Farber Presents No Scientific Basis for His Conclusory Statements Concerning DDT and Cancer Causation

Seemingly, Dr. Farber has drafted a general causation report on DDT. On page 40 of his report, he states the following: "Long term health effects [of DDT] may include cancer, liver damage, and fertility problems" (Dr. Theodore Farber's Expert Report ("Farber Report"), February 8, 2005, page 40). However, he identified no specific cancers, liver damage, or fertility problems and provided no scientific references to support his statements. Dr. Farber goes on to list a litany of government regulations and guidelines for DDT use (Farber Report, pages 3-76). Yet, nowhere has he presented any scientific evidence that DDT is capable of causing cancer, let alone NHL. The regulations and guidelines Dr. Farber cites cannot be used to support his general causation opinion—they are government policy, not scientific evidence. Undoubtedly, regulators may elect to enact cautious policies whether justified by science or not. Dr. Farber has made no effort to review the scientific basis for such regulations or, more broadly, the literature regarding the purported relationship between DDT exposure and NHL. Thus, Dr. Farber has not even attempted to use scientific methodology to support his general causation opinions—rendering them wholly unreliable.

C. Dr. Frank H. Gardner Has Not Used Valid Scientific Methodology In Arriving at His Opinions on NHL Causation

Initially, Dr. Gardner submitted a brief five page letter comprised largely of his opinions that pesticides exert a mutagenic effect in humans (Dr. Frank Gardner's

February 14, 2005 Letter ("2005 Gardner Report")). These opinions were conjecture and largely unsupported by any scientific references. Similarly, Dr. Gardner expressed the opinion that pesticides cause or contribute to the development of NHL without providing any explanation or specific references to support his views—rather, he cited to an appendix of incomplete epidemiologic literature (2005 Gardner Report, page 5). Despite his lack of epidemiologic training or expertise, Dr. Gardner purportedly relied on this appendix without any discussion or interpretation of the studies.

On December 9, 2008, Dr. Gardner submitted an updated report, titled Affidavit of Frank H. Gardner, M.D. ("2008 Gardner Report"). In this report, Dr. Gardner provides an appendix that he claims contains studies that "indicat[e] a significant association between individual organochlorides [sic] and [NHL]." He identifies *only* nine papers relating to DDT and admits that this chart was intended to satisfy the Havner criteria (2008 Gardner Report, Appendix III; Deposition of Dr. Frank Gardner ("Gardner Deposition"), pages 242:13-17). Like Dr. Clapp, however, Dr. Gardner has failed to specify any clear, consistent parameters for defining and analyzing the relevant literature set. He then reached conclusions that were not based on any clearly articulated methodology.

During his deposition, Dr. Gardner admitted that he culled studies purporting to satisfy Havner (Gardner Deposition, pages 246:10-247:3). When pressed about his methodology, and—particularly—his exclusion of negative studies, he asserted that he eliminated studies that had insufficient numbers of cases and controls (Gardner Deposition, pages 246:4-9; 346:25-347:4). However, if he had in fact used his stated selection process, he would have included additional relevant studies. For instance, Dr.

Gardner ignored Hardell, et al., 1999 though it contained cases (66) and controls (107) with DDT exposure, outnumbering those in other studies he cited. As an example, Gardner cited a single DDT finding from Baris, et al., 1998 based upon only 11 exposed cases and 15 exposed controls (2008 Gardner Report, Appendix III). This comparison demonstrates the rule, rather than the exception. With respect to the 40 studies that I evaluated, Dr. Gardner relies on only nine of those studies for DDT; furthermore, he ignores DDT findings in at least four other studies (De Roos, et al., 2003; Lee, et al., 2004; Purdue, et al., 2006; Schroeder, et al., 2001) that he relies upon or otherwise references in his reports (2005 Gardner Report; 2008 Gardner Report, Appendix III).

Indeed, even within the studies Dr. Gardner cited, he paid little heed to case and control numbers—instead again culling individual results in an effort to satisfy Havner. For example, Gardner cited a dieldrin result from a 2001 Schroeder, et al. study with seven cases and 33 controls, ignoring two non-elevated, non-significant DDT findings based on, respectively, 13 and 22 cases, and 216 controls with a DDT exposure (2008 Gardner Report, Appendix III). As this example illustrates, he repeatedly ignored negative DDT findings in the studies he cites—instead citing only those significant findings that supported his opinion. Dr. Gardner could not explain why he cited only the statistically significant finding for DDT (Gardner Deposition, pages 263:25-270:5). Like Dr. Clapp, Dr. Gardner's approach to reviewing the literature is not accepted scientific methodology.

Finally, Dr. Gardner admits that he has not considered other potential risk factors for the development of NHL in Ms. Garza. He also has ignored other chemicals processed by Hayes-Sammons (Gardner Deposition, pages 325:2-328:16). He claims to

have briefly considered, then disregarded, potential causal associations between animal viruses and NHL, as well as organophosphate pesticides and NHL (Gardner Deposition, pages 131:5-133:1; 325:2-328:16).

Thus, Dr. Gardner has not used any systematic approach for defining the relevant literature set, assessing that literature, and then using it to reach his conclusions. By admission, Dr. Gardner has ignored "negative" studies in his attempt to only identify studies that would "come up to the Havner concepts or near them" (Gardner Deposition, pages 39:25-40:8). Even within these studies, Dr. Gardner has selected only findings favorable to his position. Moreover, Dr. Gardner admittedly has failed to consider several potential risk factors relevant to the development of Ms. Garza's NHL. For all of the above reasons, Dr. Gardner's approach and resulting opinions regarding DDT and NHL are unscientific and unreliable.

D. Dr. William R. Sawyer Presents No Scientific Basis for His Opinions Regarding NHL and DDT

Dr. Sawyer's opinions center largely on Ms. Garza's purported exposures to DDT and other organochlorines processed at the Hayes-Sammons facility. Other defense experts will comment on Dr. Sawyer's exposure opinions; here, I consider only his opinion regarding DDT and NHL. Similar to Dr. Clapp, Dr. Sawyer expounds upon the use of the Hill criteria, their application to general causation, and his supposed use of the "weight of the evidence" approach (Dr. Sawyer's "Residential, Medical History & Toxicological Causation Assessment," December 9, 2008 ("2008 Sawyer Report"), pages 10-16). A review of his report and testimony quickly shows that he failed to apply any of these methods that he admits are important in arriving at a causation opinion. Indeed, Dr. Sawyer cites to a narrow sub-set of the relevant epidemiologic literature without

specifying his selection criteria, then states his conclusions in summary fashion (2008 Sawyer Report, pages 20-30). Furthermore, Dr. Sawyer has written a lengthy discussion of Ms. Garza's medical history—which he is unqualified to do since he is not a physician.

In Dr. Sawyer's initial report, titled "Environmental Exposures and Dose Calculations" ("2005 Sawyer Report"), dated February 9, 2005, Dr. Sawyer states that "human epidemiological studies have demonstrated statistically significant increased rates of NHL associated with DDT . . . .." (2005 Sawyer Report, page 16). Dr. Sawyer did not provide a single scientific reference to support his position. In his 2008 report, Dr. Sawyer makes a similar claim, this time citing risk estimates from 15 studies that, purportedly, support this opinion (2008 Sawyer Report, pages 20-29).

When questioned about the narrow range of literature he selected to support his general causation opinions, Dr. Sawyer maintained that he had reviewed all the available literature relating to organochlorines and NHL (Deposition of Dr. William Sawyer ("Sawyer Deposition"), pages 162:24-163:4). He claimed that nearly all the "negative studies" (i.e., those studies showing no association between pesticide exposure and NHL development) "were irrelevant" (Sawyer Deposition, page 164:1-4). Yet, Dr. Sawyer had no plausible explanation for why these "negative studies," including the many studies finding no association between DDT and NHL, were irrelevant. Dr. Sawyer cites only six of the 40 studies that I rely on and then ignores the DDT findings in three of the studies he does cite (2008 Sawyer Report). Furthermore, Dr. Sawyer like the other plaintiff's causation experts, cites studies that rely on surrogate measures of exposure or

lack any clear exposure data for specific chemicals, like Merhi, et al., 2007, Orsi, et al., 2007 and Flower, et al., 2004 (2008 Sawyer Report, pages, 22-25).

Dr. Sawyer, like Drs. Clapp and Gardner, selected a narrow subset of the relevant scientific literature and then cites only findings that support his opinions, while ignoring the bulk of other results and authors' conclusions. For example, he cites findings from Quintana, et al., 2004, but omits the authors' conclusions that (1) there was "no clear association between exposure to DDT and NHL," and (2) the association between DDE and NHL was confounded by other pesticides (2008 Sawyer Report, pages 26-27). He also cites Hardell, et al., 2001 for several non-DDT findings, and ignores near null, non-significant findings for a DDT metabolite (2008 Sawyer Report, pages 23-24).

Finally, Dr. Sawyer states that his "review of [Ms. Garza's] historical medical records, direct personal interview and inspection of her 1015 Nicholson Street home failed to provide any other significant occupational or environmental exposures contributing to the onset of her NHL" (2008 Sawyer Report, page 30). Although by this statement Dr. Sawyer suggests that he examined alternative causes, there is no evidence that he in fact ruled out potential risk factors for NHL. If one used Dr. Sawyer's biased methodology for selecting and reviewing scientific literature, he would conclude that such possible risk factors as obesity, arthritis, and blood transfusions caused Ms. Garza's NHL and thus should have been considered in his analysis.

In conclusion, Dr. Sawyer has failed to systematically review the total relevant body of literature and fairly interpret the studies he cites. He did not follow the methodology he claims to use (i.e., "weight of the evidence"), and instead provides a biased and limited analysis of a highly selected subset of the scientific literature.

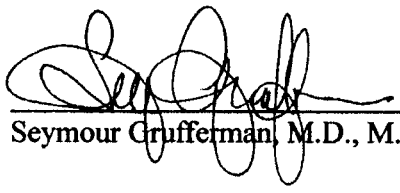
Moreover, he failed to properly consider alternative risk factors for NHL. Thus, his approach is unscientific and his resulting opinions are unreliable.

XII.
SUMMARY OF OPINIONS

In summary, based on my review of the scientific evidence in the medical and epidemiologic literature and my years of research and study of the causation of the lymphomas, I conclude:

1. To a reasonable degree of medical and scientific certainty, DDT is neither a direct nor a contributing cause of NHL in humans.
2. To a reasonable degree of medical and scientific certainty, Ms. Garza's NHL was not caused by her alleged exposure to DDT, nor did DDT contribute to the cause of her NHL. The scientific literature as a whole demonstrates very clearly and persuasively that DDT is not a cause of NHL and does not contribute to NHL causation, even at extremely high exposure levels. The vast majority of NHLs are considered to be spontaneous occurrences and Ms. Garza's NHL is highly likely to fall within this category.
3. Drs. Clapp, Gardner, Sawyer and Farber have not used accepted scientific methodology in reaching their causation opinions regarding DDT and NHL.

I reserve the right to modify and/or supplement my opinions and this report if and when new information becomes available.



Seymour Grufferman, M.D., M.P.H., S.M., Dr.P.H.

October, 2009

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Attachment 1

Non-Hodgkin Lymphoma Epidemiologic Studies Involving DDT (38 Studies)

Case–Control Studies Performing Multivariate Analyses (8 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Baris, D., et al., Agricultural Use of DDT and Risk of Non-Hodgkin's Lymphoma: Pooled Analysis of Three Case-Control Studies in the United States, Occup. Environ. Med. 55:522-7 (1998)	Pooled data from three population-based case-control studies. Exposure data from interviews.	993 cases and 2,918 controls interviewed. 161 cases and 340 controls exposed to DDT.	OR=1.2 (1.0-1.6), unadjusted. Adjusted OR = 0.9 (0.4-1.8). When analyses adjusted for other chemicals, ORs ranged from 0.3 to 1.9 (Table 5). 36 total analyses reported in Table 6.	N.S.
2.	De Roos, A., et al., Integrative Assessment of Multiple Pesticides as Risk Factors for Non-Hodgkin's Lymphoma Among Men, Occup. Environ. Med. 60:E11 (2003)	Pooled data from three population-based case-control studies. Exposure data from interviews.	870 cases and 2,569 controls interviewed. For analyses of multiple pesticides, 650 cases and 1,933 controls.	OR for DDT = 1.0 (0.7-1.3), based on 98 DDT-exposed cases and 226 DDT-exposed controls.	N.S.
3.	Engel, L., et al., Polychlorinated Biphenyl Levels in Peripheral Blood and Non-Hodgkin's Lymphoma: A Report from Three Cohorts, Cancer Res. 67:5545-5552 (2007)	Nested case-control study in 3 prospective cohorts in which blood samples previously had been collected.	Cohort 1 (Janus): 190 cases and 190 controls. Cohort 2 (CLUE 1): 74 cases and 147 controls. Cohort 3 (Nurses' Health): 30 cases and 78 controls.	Adjusted ORs for p,p'-DDE in highest quartile of exposure = 1.4 (0.7-2.9) in Janus cohort and 1.5 (0.7-3.2) in CLUE 1 cohort. Adjusted ORs for p,p'-DDE in highest quartiles = 4.3 (1.2-15.0) in Janus 2-16 years of follow-up group and 2.1 (0.7-6.3) in CLUE 1 0-12 year follow-up. Adjusted ORs for p,p'-DDE in highest quartiles = 0.8 (0.3-2.0) in Janus 17-25 year follow-up and 1.1 (0.3-3.4) in CLUE 1 13-19 year follow-up groups. In analyses adjusted for total PCBs, the ORs for p,p'-DDE in highest quartiles of exposure were 3.4 (0.9-12.7) for Janus and 0.9 (0.2-3.4) in CLUE 1 for "short" follow-ups and 0.7 (0.3-2.1) in Janus and 0.9 (0.3-3.0) in CLUE 1 for "long" follow-ups. Similar analysis for the Nurses' study in terms of the highest tertile yielded	N.S. (Analysis for only 1 of hundreds of strata was statistically significant but became N.S. when adjustment was made for both individual and all PCBs.)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
				an OR = 1.4 (0.4-4.6).	
4.	Hardell, L., et al., Exposure to Phenoxyacetic Acids, Chlorophenols, or Organic Solvents in Relation to Histopathology, Stage and Anatomical Location of Non-Hodgkin's Lymphoma, Cancer Res. 54:2386-89 (1994)	Combination of self-administered and interviewer administered, telephone questionnaires.	105 cases and 335 controls. (Cases from a single hospital and controls from population-based registries.)	Unadjusted OR = 2.4 (1.2-4.9). Adjusted for other chemicals OR = 1.5 (0.6-3.6), based on 17 exposed cases and 26 exposed controls.	N.S.
5.	Hardell, L., et al., Exposure to Pesticides as Risk Factors for Non-Hodgkin's Lymphoma and Hairy Cell Leukemia: Pooled Analysis of Two Swedish Case-Control Studies, Leuk. Lymph. 43:1043-49 (2002)	Data from two previous case-control studies. Includes data on NHL and hairy cell leukemia. Mailed, self-administered questionnaires.	515 cases and 1,141 controls.	Univariate analysis: OR = 1.27 (0.92-1.73), based on 77 cases and 138 controls with DDT exposure. "In the multivariate analysis no risk was found."	N.S.
6.	McDuffie, H., et al., Non-Hodgkin's Lymphoma and Specific Pesticide Exposures in Men: Cross-Canada Study of Pesticides and Health, Cancer Epidemiol. Biomarkers Prev. 10:1155-63 (2001)	Multi-center study. Information from mailed questionnaires followed by telephone interview.	517 cases and 1,506 community controls.	Unadjusted OR = 1.63 (1.03-2.57), based on 32 exposed cases and 59 exposed controls. DDT was "found not to contribute significantly to the risk of NHL" in multivariate analyses.	N.S.
7.	Persson, B., et al., Some Occupational Exposures as Risk Factors for Malignant Lymphomas, Cancer 72: 1773-78 (1993)	Exposure data from mailed, self-administered questionnaires.	93 cases and 204 controls. (Cases from a regional cancer registry and controls from another study.)	Crude OR = 3.0 (no CI provided) Adjusted OR = 2.0 (90% CI = 0.3-13), based on 4 exposed cases and 3 exposed controls.	N.S.
8.	Rothman, N., et al., A Nested Case-Control Study of Non-Hodgkin Lymphoma and Serum Organochlorine Residues, Lancet 350:240-44 (1997)	Nested case-control study using pre-diagnostic serum samples from a prospective cohort study.	74 cases and 147 matched-controls.	The OR for DDT in the highest quartile level vs. the lowest was 1.9 (0.8-4.5). In an analysis adjusted for PCBs, the OR for this analysis was reduced to 1.2 (0.5-3.0).	N.S.

Case–Control Studies Performing Univariate Analyses Only (21 Studies)

1. Case–Control Studies of DDT and NHL Without Biologic Specimens (12 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Cantor, K., et al., Pesticides and Other Agricultural Risk Factors for Non-Hodgkin's Lymphoma Among Men in Iowa and Minnesota, Cancer Res. 52:2447-55 (1992)	Population-based case-control study. Interviewer administered questionnaires.	622 white male cases and 1,245 matched community controls.	The study's findings regarding DDT included: • The OR for having ever handled DDT as an animal insecticide was 1.2 (95% CI = 0.9-1.7). • The OR for having handled DDT as an animal insecticide prior to 1965 was 1.3 (0.9-1.8). • The OR for having ever handled DDT as a crop insecticide was 1.7 (1.2-2.6). • The OR for having ever handled DDT as a crop insecticide prior to 1965 was 1.8 (1.1-2.7). • The OR for having ever handled DDT as an animal insecticide with protective equipment was 1.2 (0.9-1.7). • The OR for having ever handled DDT as an animal insecticide without protective equipment was 1.3 (0.9-1.8). • The OR for having ever handled DDT as a crop insecticide with protective equipment was 1.7 (1.2-2.6). • The OR for having ever handled DDT as a crop insecticide without protective equipment was 2.0 (1.3-3.1). • The OR for Iowa subjects who ever used DDT prior to 1965 as an animal insecticide was 0.9 (0.5-1.5). • The OR for Minnesota subjects who ever used DDT prior to 1965 as an animal insecticide was 1.7 (1.1-2.7). • The OR for Iowa subjects who ever used DDT prior to 1965 as a crop insecticide was 1.5 (0.9-2.6).	Some findings statistically significant; some N.S.

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
				• The OR for Minnesota subjects who ever used DDT prior to 1965 as a crop insecticide was 2.3 (1.1-4.8). • When analyses were stratified by state, the DDT results were not statistically significant for Iowa subjects and were statistically significant for Minnesota subjects. In summary, ORs ranged from 0.9 (0.5-1.5) to 2.3 (1.1-4.8) based on numerous subset analyses.	
2.	Cantor, K., et al., Letter to the Editor: Correspondence Re: K. P. Cantor, et al., Pesticides and Other Agricultural Risk Factors for Non-Hodgkin's Lymphoma Among Men in Iowa and Minnesota, Cancer Res. 53:2421 (1993)	Population-based case-control study. Interviewer administered questionnaires. Addendum to Cantor, et al., 1992.	110 white male cases and 211 matched community controls, a subset of subjects from the prior Cantor, et al., 1992 paper.	• The ORs for DDT use as a crop insecticide were 1.2 (0.5-2.8) for 1-4 days per year of use; 1.6 (0.4-5.6) for 5-9 days per year of use; and 1.7 (0.6-4.8) for 10+ days per year of use. • The ORs for DDT as an animal insecticide were 0.3 (0.1-0.8) for 1-4 days per year of use; 0.5 (0.1-1.8) for 5-9 days per year of use; and 0.9 (0.4-2.0) for 10+ days per year of use.	Statistically significant protective effect for 1 stratum. The other analyses were N.S.
3.	Colt, J., et al., Organochlorines in Carpet Dust and Non-Hodgkin's Lymphoma, Epidemiology 16:516-525 (2005)	Population-based case-control study using data from SEER Program. Exposure data from analysis of carpet dust.	603 cases and 443 frequency-matched controls who could provide vacuum cleaner dust samples.	The OR for DDE in dust samples was 1.6 (1.1-2.2) for the highest vs. lowest tertile and the p-value for trend was 0.02. The reported association was stronger in men (OR = 1.6, 1.1-2.3) than in women (OR = 1.1, 0.7-1.5). The OR for DDT in dust samples was 1.2 (0.8-1.6) for the highest vs. lowest tertile. A trend test for DDT yielded a p-value of 0.09.	N.S. for all DDT tertiles. Statistically significant result for highest DDE dust tertile only.
4.	Eriksson, M., et al., Pesticide Exposure as Risk Factor for Non-Hodgkin Lymphoma Including Histopathological Subgroup Analysis, Int. J. Cancer, 123:1657-1663 (2008)	Population-based case-control study. Self-administered mailed questionnaires supplemented by telephone interviews.	910 cases from university hospitals and 1062 controls from a national population registry.	OR for DDT = 1.46 (0.94-2.28) based on 50 exposed cases and 37 exposed controls. Subjects were subdivided into two exposure categories, ≤37 days (OR = 1.17, 0.62-2.22) and >37 days (OR = 1.76, 0.97-3.20). The authors also found an OR of 2.14 (1.05-4.40) for DDT and follicular, grade I-III NHL, based on 165 such cases.	Overall OR for DDT, N.S. Significant for only one of several subgroup analyses.
5.	Hardell, L., Eriksson, M., A Case-Control Study of Non-Hodgkin Lymphoma and Exposure to Pesticides, Cancer, 85:1353-60 (1999)	Population-based case-control study. Self-administered mailed	404 cases from a regional cancer registry and 741 controls from a national population registry.	OR for DDT = 1.1 (0.7-1.7), based on 66 exposed cases and 107 exposed controls.	N.S.

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
		questionnaires supplemented by telephone interviews.			
6.	Lee, W.J., et al., Non-Hodgkin's Lymphoma Among Asthmatics Exposed to Pesticides, Int. J. Cancer 111:298-302 (2004)	Pooled, population-based case-control study. Interviewer administered questionnaires.	872 cases and 2,381 frequency-matched controls from the Cantor, et al., 1992 study and another study by Zahm, et al., in 1990.	The ORs for DDT in non-asthmatics was 1.2 (0.9-1.5), based on 158 exposed cases and 313 exposed controls, and 1.2 (0.6-2.4) in asthmatics, based on 11 cases and 24 controls.	N.S.
7.	Persson, B., et al., Malignant Lymphomas and Occupational Exposures, Brit. J. Indus. Med. 46:516-20 (1989)	Hospital-based case-control study with population-based controls from another case-control study. Exposure levels imputed from self-administered questionnaires.	106 cases and 275 controls.	OR for DDT = 0, based on 0 exposed cases and 3 exposed controls.	--
8.	Persson, B. and Fredrikson, M., Some Risk Factors for Non-Hodgkin's Lymphoma, Int. J. Occup. Med. Environ. Health 12:135-142 (1999)	Population-based case-control study using data from 2 other studies (Persson, et al., 1989; Persson, et al., 1993).	199 cases and 479 community controls.	The OR for DDT was 1.4 (0.3-5.9), based on 4 exposed cases and 6 exposed controls.	N.S.
9.	Schroeder, J., et al., Agricultural Risk Factors for t(14;18) Subtypes of Non-Hodgkin's Lymphoma, Epidemiology 12:701-9 (2001)	Population-based case-control study using data from Cantor, et al., 1992.	182 cases studied for t(14;18) translocations and 1,245 controls.	The OR for DDT and t(14;18) positive cases vs. controls was 1.1 (0.6-1.9), based on 13 exposed positive cases and 216 exposed controls. The OR for DDT and t(14;18) negative cases vs. controls was 1.2 (0.8-1.7), based on 22 exposed negative cases and 216 exposed controls. The OR for t(14;18) positive cases vs. negative cases was 0.9 (0.4-1.9).	N.S.
10.	Woods, J., et al., Soft Tissue Sarcoma and Non-Hodgkin's Lymphoma in Relation to Phenoxyherbicide and Chlorinated Phenol Exposure in Western Washington, J. Natl. Cancer Inst. 78:899-910 (1987)	Population-based case-control study. Interviewer administered questionnaires.	576 cases from a population-based tumor registry and 694 community controls selected by random-digit telephone dialing.	OR for DDT = 1.82 (1.04-3.2), based on an overall 4.0% exposure in the study population.	Statistically significant.
11.	Woods, J., et al., Non-Hodgkin's Lymphoma Among Phenoxy Herbicide-Exposed Farm Workers in Western Washington State, Chemosphere 18:401-406 (1989)	Population-based case-control study. A subset of same subjects from Woods, et al., 1987 study who had	181 cases and 196 controls.	OR for DDT = 1.68 (0.9-3.3).	N.S.

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
		ever been farmers. Interviewer administered questionnaires.			
12.	Zahm, S., et al., The Role of Agricultural Pesticide Use in the Development of Non-Hodgkin's Lymphoma in Women, Arch. Environ. Health 48:353-58 (1993)	Population-based case-control study. Interviewer-administered telephone questionnaires.	119 female cases and 471 frequency-matched controls.	The OR for DDT was 1.7 based on 16 exposed cases and 36 controls. No confidence interval was provided, but the paper states that the results were non-significant.	N.S.

2. Case–Control Studies of DDT and NHL/Chronic Lymphocytic Leukemia (2 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Miligi, L., et al., Non-Hodgkin's Lymphoma, Leukemia, and Exposures in Agriculture: Results from the Italian Multicenter Case-Control Study, Am. J. Industrial Med. 44:627-636 (2003)	Population-based case-control study. Data collected by personal interviews.	1,145 combined NHL and CLL cases and 1,232 randomly sampled general population controls.	An OR of 0.6 (0.3-1.1) was found for DDT in men based on 21 exposed NHL and CLL cases. An OR of 0.3 (0.1-0.8) was found in women based on 5 exposed NHL and CLL cases.	Statistically significant protective effect in women. N.S. in men.
2.	Nanni, O., et al., Chronic Lymphocytic Leukaemias and Non-Hodgkin's Lymphomas by Histological Type in Farming-Animal Breeding Workers: A Population Case-Control Study Based on A Priori Exposure Matrices, Occup. Environ. Med. 53:652-57 (1996)	Population-based case-control study of CLL and NHL. Data from interviews and a priori exposure matrices.	187 combined NHL and CLL cases and 977 matched population controls.	The OR for DDT and combined NHL and CLL was 1.74 (0.93-3.27), based on subjects' recall of exposure (recall by 27 cases and 61 controls). The OR for DDT and combined NHL and CLL was 1.70 (0.91-3.17), based on job matrix estimation of exposure (28 cases and 65 controls exposed). The ORs for CLL and low grade NHL were 2.33 (0.93-5.85) in a recall analysis and 2.16 (0.86-5.43) in the job matrix analysis of subjects with exposures via farming-animal breeding. When cumulative dose was compared in CLL and low grade NHL cases and controls, an OR of 1.22 (0.95-1.57) was found.	N.S.

3. Case-Control Studies of DDT Levels in Biologic Specimens and NHL (7 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Cocco, P., et al., Plasma polychlorobiphenyl and organochlorine pesticide level and risk of major lymphoma subtypes, Occup. Environ. Med. 65:132-140 (2008)	Case-control study of plasma levels for six DDT isomers. Blood samples obtained pre-treatment for a subset of cases. Information on potentially confounding covariates based on "personal interviews."	174 NHL cases from the Epilymph study (France, Germany, and Spain) and 203 controls. Methods for selection of cases and controls are not specified.	OR for highest quartile of serum p,p'-DDE (≥ 1431.08 ppb) = 1.2 (0.7-2.4). For diffuse large B-cell lymphoma, the OR for the highest quartile of serum levels = 1.3 (0.5-3.6); for CLL/SLL the corresponding OR was 1.0 (0.4-2.5).	N.S.
2.	De Roos, A., et al., Persistent Organochlorine Chemicals in Plasma and Risk of Non-Hodgkin's Lymphoma, Cancer Res. 65:11214-226 (2005)	Population-based case-control study using data from SEER Program and from another study. Analysis of plasma samples for persistent organochlorine chemicals. Blood samples obtained after diagnosis.	100 cases and 100 controls from a large N.C.I. study of 1,321 cases and 1,057 controls.	For p,p'-DDE, the OR for the highest plasma concentration quartile was 0.85 (0.37-1.94). For the second highest quartile, the OR was 0.33 (0.14-0.80). For p,p'-DDT, the OR for the highest quartile level was 1.20 (0.39-3.70). Trend tests for both these sets of analyses were non-significant. For "extreme levels of exposure" the OR for p,p'-DDT was 3.3 (0.7-15.9).	Statistically significant protective effect for the third quartile of p,p'-DDE levels. All other values N.S.
3.	Hardell, K., et al. Concentrations of Organohalogen Compounds and Titres of Antibodies to Epstein-Barr Virus Antigens and the Risk for Non-Hodgkin Lymphoma, Oncology Reports 21:1567-1576 (2009)	Population-based case-control and cross-sectional study. Subset of cases and controls from Eriksson, et al., 2008. Analysis of blood samples from a subset of subjects obtained after cases' diagnosis	100 cases and 100 controls from a subset of subjects with available blood samples.	For all NHL, there was no significant difference between cases and controls with regard to blood concentrations of p,p'-DDE (p = 0.11). For follicular lymphoma, OR = 1.2 (0.4-3.5) for p,p'-DDE. From the numerous subgroup analyses: • For follicular NHL only (n = 20): For > the median p,p'-DDE level and EA >40, OR = 1.2 (0.3-4.3). • All NHL: For > the median p,p'-DDE level and EA >40, OR = 3.3 (1.4-7.7).	N.S. for all NHL and blood concentrations of p,p'-DDE. N.S. for follicular NHL.

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
4.	Hardell, L., et al., Higher Concentrations of Specific Polychlorinated Biphenyl Congeners in Adipose Tissue from Non-Hodgkin's Lymphoma Patients Compared with Controls Without a Malignant Disease, Int. J. Oncol. 9:603-608 (1996)	Case-control study of adipose tissue.	28 cases and 17 surgical controls.	The mean concentration of p,p'-DDE in adipose tissue was 1,420 for NHL and 1,068 for controls (p = 0.29).	N.S.
5.	Hardell, L., et al., Case-Control Study on Concentrations of Organohalogen Compounds and Titers of Antibodies to Epstein-Barr Virus Antigens in the Etiology of Non-Hodgkin Lymphoma, Leuk. Lymph. 42:619-29 (2001)	Case-control study of adipose tissue and blood samples. (Includes subjects from Hardell, et al., 1996 study.)	50 cases and 47 surgical controls for the adipose tissue portion of the study. 32 cases and 36 controls for the blood sample portion of the study.	For p,p-DDE, an OR of 1.2 (0.60-2.5) was found based on 44 cases and 41 controls. In multivariate analysis of organohalogen compounds, the OR for p,p-DDE was unchanged (OR = 1.2, 0.44-3.5). Case control analyses of p,p-DDE in relationship to EBV antibody titers yielded ORs of 2.0 to 2.9 with none of the values statistically significant.	N.S.
6.	Quintana, P., et al., Adipose Tissue Levels of Organochlorine Pesticides and Polychlorinated Biphenyls and Risk of Non-Hodgkin's Lymphoma, Environ. Health Perspect. 112:854-61 (2004)	Nested case control study using adipose samples from cadavers (> 96%) and surgical patients collected previously by the EPA.	175 cases and 481 controls selected from 2 groups: subjects with accidental injury or death and subjects with myocardial infarction.	For p,p'-DDT, the OR for the highest vs. lowest quartile was 1.39 (0.78-2.47). A trend test for these strata was statistically significant (p = 0.04). For p,p'-DDE, the OR was 1.99 (1.14-3.47) for the highest vs. lowest quartiles. A trend test for these strata was statistically significant (p = 0.002). In an analysis that used "two-pesticide models," when joint exposure to other pesticides was considered, the ORs for the highest vs. lowest quartiles of p,p'-DDE were lower and no longer statistically significant.	N.S. for DDT and DDE.
7.	Spinelli, J., et al., Organochlorines and risk of non-Hodgkin lymphoma, Int. J. Cancer 121:2767-2775 (2007)	Subset of participants in a larger NHL case-control study. Self-administered questionnaires and CATI. Blood samples obtained from cases (after diagnosis) and controls.	422 cases and 460 community controls.	For "p',p'-DDT," the OR for the quartile level > 3.24 ng/g versus no level was 0.91 (0.68-1.20). For p,p'-DDE, the OR for the highest vs. lowest quartile was 1.42 (0.92-2.19). A trend test for the quartiles of DDE levels yielded a p-value of 0.027, yet there was no statistically significant finding for any of the quartiles. For follicular lymphoma, the ORs were 0.7 (0.5-1.1) for p,p'-DDT and 1.8 (0.9-3.3) for p,p'-DDE.	N.S.

Cohort Studies of DDT and NHL (7 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Brown, D., Mortality of Workers Employed at Organochlorine Pesticide Manufacturing Plants – An Update, Scand. J. Work Environ. Health 18:155-61 (1992)	An update of the Ditraglia, et al., 1981 study, of workers at 4 organochlorine pesticide manufacturing plants.	328 exposed workers at Plant 4 with 9,797 person-years of observation.	The SMR for all malignant neoplasms among Plant 4 workers was 0.87 (0.52-1.39). There were no deaths from lymphatic/hematopoietic neoplasms in Plant 4 workers.	N.S.
2.	Cocco, P., et al., Cancer Mortality among Men Occupationally Exposed to Dichlorodiphenyltrichloroethane, Cancer Res. 65: 9588-94 (2005)	A mortality follow-up study of deaths among men who used mainly DDT in an anti-malarial campaign in Italy during the 1940s. (A follow-up of subjects from the Cocco, et al., 1997a and 1997b studies.)	4,552 male workers exposed to DDT. Of these, 2,726 died and had death certificate information available.	For the total cohort, the SMR for lymphatic cancer was 115 (87-151). For the applicator group, the lymphatic cancer SMR was 101 (65-157). For unexposed workers, the SMR for lymphatic cancers was 174 (112-271).	N.S. for applicators. Statistically significant excess in lymphatic cancer mortality for unexposed workers.
3.	Cocco, P., et al., Long-term Health Effects of Occupational Exposure to DDT; A Preliminary Report, Ann. N.Y. Acad. Sci. 837:246-56 (1997b)	A proportional mortality study of deaths among men who used mainly DDT in an anti-malarial campaign in Italy during the 1940s. (Uses the same data from the Cocco, et al., 1997a study.)	1,043 deaths between 1956 and 1992.	PMR = 81 (9-294) for NHL in the DDT exposed group, based on two NHL deaths. For all cancers of the lymphohemopoietic system, there were SMRs of 136 (36-347) for those exposed for less than 75 days and 146 (39-375) for those exposed for 150 days or more. (These results are identical to the Cocco, et al., 1997a paper.)	N.S.
4.	Cocco, P., et al., Proportional Mortality of Dichloro-Diphenyl-Trichloroethane (DDT) Workers: A Preliminary Report, Arch. Environ. Health 52:299-303 (1997a)	A proportional mortality study of deaths among men who used mainly DDT in an anti-malarial campaign in Italy during the 1940s.	1,043 deaths between 1956 and 1992.	PMR = 81 (9-294) for NHL in the DDT exposed group, based on two NHL deaths. For all cancers of the lymphohemopoietic system, there were SMRs of 136 (36-347) for those exposed for less than 75 days and 146 (39-375) for those exposed for 150 days or more.	N.S.

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
5.	Ditraglia, D., et al., Mortality Study of Workers Employed at Organochlorine Pesticide Manufacturing Plants, Scand. J. Work Environ. Health 7:140-46 (1981)	Workers at 4 organochlorine pesticide manufacturing plants. Only 1 of the plants produced DDT (Plant 4), and this plant produced only DDT.	Approximately 2,100 men who worked at manufacturing plants for at least 6 months prior to December 31, 1964.	For workers at Plant 4, mortality for all malignant neoplasms, based on 6 observed deaths, yielded an SMR of 68 (25-247). There were no observed deaths from lymphatic and hematopoietic system cancers in workers at Plant 4. In workers employed for 20 or more years at Plant 4, the SMR for deaths due to malignant neoplasms was 132 (43-309), based on 5 observed deaths.	N.S.
6.	Laws, E., et al., Men With Intensive Occupational Exposure to DDT, Arch. Environ. Health 15:766-775 (1967)	Volunteers from a DDT-manufacturing facility (a study of workers from Montrose Chemical Corporation of California by the U.S. Public Health Service).	35 men with more than 5 years of occupational exposure to DDT.	No cases of cancer or blood dyscrasia in the 35 men studied and in the 63 men at the plant with more than 5 years of DDT exposure (data obtained from medical record review).	N.S.
7.	Purdue, M., et al., Occupational Exposure to Organochlorine Insecticides and Cancer Incidence in the Agricultural Health Study, Int. J. Cancer 120:642-649 (2006)	A prospective cohort study of licensed applicators enrolled in the Agricultural Health Studies (in Iowa and North Carolina). Initial data from self-administered questionnaires.	A sample of 22,409 workers from the total of 51,011 male AHS applicators.	For NHL, a RR of 0.9 (0.6-1.5) was found for DDT based on 37 NHLs in 12,305 exposed workers.	N.S.

Ecologic Studies of DDT and NHL (2 Studies)

	Document Description	Source of Data	Number of Subjects	Findings	Statistical Significance
1.	Cocco, P., et al., Cancer Mortality and Environmental Exposure to DDE in the United States, Environ. Health Perspect. 108:1-4 (2000)	Adipose tissue DDE levels in 1968 in population samples from 22 U.S. states and mortality rates from 1975-1994. Data on DDE tissue concentrations were obtained from an EPA report in 1968.	No information is provided on the number of tissue samples or on the number of malignancies observed.	The data are difficult to interpret. The authors state that their study showed no evidence of a consistently significant increase in mortality with increasing adipose DDE levels. They further state that mortality from NHL was inversely related to DDE levels in 3 of the 4 study groups.	NA.
2.	Pavuk, M., et al., Environmental Exposure to PCBs and Cancer Incidence in Eastern Slovakia, Chemosphere 54:1509-1520 (2004)	Population-based cross-sectional study of serum levels of pesticides in 2 districts in Slovakia. An ecologic study was performed to compare cancer incidence in the same 2 districts. One of the 2 districts produced PCBs.	225 subjects in one district and 207 subjects in the second district.	In the PCB-producing district, levels of p,p'-DDT in males and females were significantly different from the second district ($p = 0.003$ for males and females). For p,p'-DDE, levels in the PCB- producing district were also significantly higher for both men and women ($p = 0.05$ for men and $p = 0.001$ for women). In the ecologic study of cancer incidence, the SIR for NHL in males was 1.12 (0.80-1.52) in the PCB-producing district and 1.11 (0.62-1.84) in the second district. The SIR for NHL in females was 1.04 (0.70-1.49) in the PCB-producing district and 0.56 (0.22-1.28) in the second non-PCB-producing district.	N. S. for NHL results.

ATTACHMENT 2

I. DETAILED ANALYSES OF CASE-CONTROL STUDIES PERFORMING MULTIVARIATE ANALYSES

(1) Baris D, Zahm SH, Cantor KP, Blair A. Agricultural use of DDT and risk of non-Hodgkin's lymphoma: pooled analysis of three case-control studies in the United States, 1998. This is a pooled study of three population-based case-control studies of NHL in four mid-western states, including the study reported by Cantor, et al., 1992 and discussed below. As stated by the authors, the "main motivation for the pooled analysis was to conduct a more detailed analysis than was possible for individual studies, and to adjust ORs for possible confounding effects from exposure to other pesticides."

The study involves subjects from four states with a relatively high proportion of farmers. The pooled subjects constituted 993 NHL cases and 2,918 controls that were interviewed to obtain information on agricultural pesticides and other risk factors. An OR of 1.2 (1.0-1.6) for any use of DDT was found, based on 161 cases and 340 controls who were exposed. Farmers who had used DDT for 15 years or more had an OR of 1.5 (1.0-2.3). Farmers who had used DDT for 5 days/year or more had an OR of 2.6 (1.1-5.9). These were the results, out of many, that the authors chose to include in the paper's abstract.

When these analyses were adjusted for use of other pesticides and herbicides, the odds ratios were all closer to 1.0 (ranging from 0.3 to 1.9) and none was statistically significant. In Table 5, the authors present the findings of analyses that adjust for the following: 1.) use of other groups of pesticides; 2.) other individual pesticides; and 3.) organophospates and 2,4-D simultaneously. The analyses were performed by duration and frequency of DDT use as well as in terms of ever/never use. Table 6 in this paper presents odds ratios for use of DDT stratified by use of 2,4-D and organophosphates. This adjustment procedure, combined with stratification by

duration and frequency of DDT use, yielded odds ratios ranging from 0.0 to 2.9. Of the 36 analyses reported, 3 were statistically significant and 3 were of borderline significance (i.e., CI lower bound was 1.0.). Interestingly, the highest observed OR (2.9) was for the stratum of farmers who used DDT for 5-9 years and never used 2,4-D or organophosphates. This is likely to be a chance observation for two reasons. First, the results in this table generally show higher risks for combined use of DDT and 2,4-D or organophosphates than for DDT alone. Second, this OR of 2.9 was found for use of DDT for 5-9 years, but the OR for use of DDT for ≥10 years was lower at 1.0. The fact that this value was an outlier was recognized by the authors; however, the authors' discussion of it suggests an apparent error in Table 6 or in the discussion. The authors state on page 526 that the finding of an OR of 2.9 was for those farmers who used DDT for ≥10 years and never used 2,4-D or organophosphates. This does not jibe with the data presented in Table 6.

To summarize, this study shows clearly that there are no associations between DDT exposure and risk of NHL when other chemical exposures can be controlled for in multivariate analyses. This study suggests that previous observations of associations between DDT and NHL likely were confounded by exposures of study subjects to other chemicals. The authors are in agreement with my assessment and state: "No strong consistent evidence was found for an association between exposure to DDT and risk of non-Hodgkin's lymphoma." It seems that the excess risk initially found may be explained by other confounding variables. Furthermore, this study is important because, as the authors state in their conclusion, "[t]he relatively large number of exposed cases and controls in this pooled analysis provided analytic opportunities which were not available in individual studies." Such analytic opportunities particularly allow for proper

statistical evaluation of confounding. As this study and the next demonstrate, the effects of confounding by other chemicals are substantial when such analyses are possible.

(2) De Roos AJ, Zahm SH, Cantor KP, et al. Integrative assessment of multiple pesticides as risk factors for non-Hodgkin's lymphoma among men, 2003. This is a pooled study of data from three NCI-conducted, case-control studies of NHL in the Midwestern U.S. done in the 1980s, which includes data from the earlier Cantor, et al., 1992 study. These are the same three studies analyzed in the previously discussed Baris, et al., 1998 paper. Using newer, highly sophisticated statistical methodology, De Roos, et al., used the data again to assess pesticide exposures in farming as NHL risk factors and shed new light on the subject.

A total of 870 cases and 2,569 controls were studied. For the analyses of multiple pesticides, 650 cases and 1,933 controls were studied. The very large sample size allowed for the analysis of 47 pesticides simultaneously, controlling for potential confounding by other pesticides. Adjustment also was made based on a pre-specified variance to make estimates more stable. After adjusting for exposures to all other pesticides, the OR for DDT exposure was 1.0 (0.7-1.3) using logistic regression. When hierarchical regression (the method based on pre-specified variances) was used, the OR for DDT was also 1.0 (0.7-1.3). These results were based on 98 DDT-exposed cases and 226 DDT-exposed controls.

This is an important study for several reasons. First, the pooled studies provided large numbers of subjects so that there was adequate statistical power for simultaneous consideration of a large number of pesticides as risk factors for NHL. Second, sophisticated biostatistical methods were employed to carefully assess confounding by multiple chemical exposures. Third, they clearly showed that the ORs for DDT and NHL risk were 1.0 when other confounding

chemical exposures were controlled in the analyses. Thus, it appears that all of the previously noted weak associations with agricultural pesticides were confounded by other exposures.

This is yet another example of a study that was able to assess associations of NHL with DDT and other agricultural chemicals such as herbicides and other pesticides in a multivariate manner. As was found in other such adequately sized multivariate analyses of herbicides and pesticides, there was little or no association between DDT and NHL when other chemical exposures were adjusted for in the analyses.

In conclusion, control of confounding exposures is essential for proper assessment of whether DDT is associated with NHL, particularly when weak associations are observed. My opinion is supported by De Roos, et al., who conclude that, "[c]onsideration of multiple exposures is important in accurately estimating specific effects and in evaluating realistic scenarios." Thus, as newer statistical methods have been developed, they have been applied to the NCI multi-site case-control study data by successive investigators. The newer statistical approaches used by De Roos, et al., effectively have controlled for potential confounders and demonstrate that DDT exposure is not associated with NHL risk.

(3) Engel L, Laden F, Anderson A, et al. Polychlorinated Biphenyl Levels in Peripheral Blood and Non-Hodgkin's Lymphoma: A Report from Three Cohorts, 2007. This is a nested case-control study in 3 prospective cohorts in which blood samples previously had been collected. The first cohort was the Janus cohort that included 87,600 Norwegian men and women who provided blood samples between 1972 and 1978. The second cohort was the CLUE 1 cohort that included 23,938 residents of Washington County, MD. Blood samples for this cohort were provided in 1974. The third cohort was the Nurses' Health Study that included 121,700 female registered nurses. Blood samples for 32,826 nurses were provided between 1989

and 1990. Cancer diagnoses for the first two cohorts were ascertained by cancer registries. Cancer diagnoses in the Nurses' cohort was by self-report on a biennial follow-up questionnaire.

Cases for the nested case-control study had a confirmed NHL diagnosis. Controls were individually matched to cases and were cohort members without cancer diagnoses. The Janus cohort provided 190 cases and 190 controls. The CLUE 1 cohort provided 74 cases and 147 controls. The Nurses' Health Study provided 30 cases and 78 controls.

The adjusted ORs for p,p'-DDE in the highest quartile of exposure were 1.4 (0.7-2.9) in the Janus cohort and 1.5 (0.7-3.2) in the CLUE 1 cohort. The adjusted ORs for p,p'-DDE in the highest quartiles were 4.3 (1.2-15.0) in Janus 2-16 years of follow-up group and 2.1 (0.7-6.3) in CLUE 1 0-12 year follow-up. The adjusted ORs for p,p'-DDE in the highest quartiles were 0.8 (0.3-2.0) in Janus 17-25 year follow-up and 1.1 (0.3-3.4) in CLUE 1 13-19 year follow-up groups.

In analyses adjusted for total PCBs, the ORs for p,p'-DDE were 1.2 (0.6-2.7) and 0.9 (0.4-2.1) for the highest quartiles in the Janus and CLUE 1 cohorts, respectively (Supp. Table 10). Similar analysis for the Nurses' study in terms of the highest tertile yielded an OR of 1.4 (0.4-4.6) (Supp. Table 10). In analyses based on time from blood draw to diagnosis and adjusted for total PCBs, the ORs for p,p'-DDE in the highest quartiles of exposure were 3.4 (0.9-12.7) and 0.9 (0.2-3.4) in Janus and CLUE 1, respectively, for "short" follow-ups and 0.7 (0.3-2.1) and 0.9 (0.3-3.0) in Janus and CLUE 1, respectively, for "long" follow-ups (Supp. Table 11). Additional values are provided in Supplementary Tables 12 and 13, but they are not adjusted for PCBs.

These data again demonstrate that initial univariate analyses of DDT metabolites and NHL typically are confounded by other exposures. The single finding of a significantly elevated

OR was attenuated and was no longer statistically significant when the confounding effect of other exposures (total PCBs) was controlled by multivariate analysis.

As the authors state: "The main *p,p'*-DDT metabolite, *p,p'*-DDE, showed a slightly increased risk of NHL across the three cohorts, which was stronger in the earlier follow-up period. However, there were no apparent exposure-response trends in most analyses, and the *p,p'*-DDE effect was attenuated more by adjustment for PCBs than vice versa. Although several epidemiologic studies have found modest increases in NHL risk related to measured or self-reported *p,p'*-DDT/*p,p'*-DDE exposure (20-24), this effect has tended to decrease substantially in those studies in which adjustment was made for other chemical exposures (21, 25)."

This conclusion by the authors is in complete agreement with my opinions regarding the need for performing multivariate analyses of data of DDT and DDE to rule out the confounding effects of other chemical exposures. In the Engel, et al., 2007 study, the only statistically significant finding from multiple analyses was no longer significant when other confounding exposures were controlled for in multivariate analyses.

In summary, this very large, pooled, nested case-controlled study showed no significant associations between NHL risk and p,p'-DDE in blood samples collected well in advance of NHL diagnosis. This study again illustrates the importance of multivariate analyses for assessing DDT and NHL.

(4) Hardell L, Eriksson M, Degerman A. Exposure to phenoxacetic acids, chlorophenols, or organic solvents in relation to histopathology, stage and anatomical localization of non-Hodgkin's lymphoma, 1994. This is a reanalysis of an earlier reported case-control study of NHL and Hodgkin lymphoma in Sweden conducted by the same group of investigators. The study is based on 105 NHL cases from a single Swedish hospital and 335

matched controls selected from population-based registries. Exposure information was obtained by a combination of self-administered questionnaires and interviewer administered questions. This paper reports an unadjusted OR for DDT and NHL of 2.4 (95% CI = 1.2-4.9). However, when multivariate analysis controlling for other chemicals was done, the OR for DDT dropped impressively to 1.5 (0.6-3.6).

This paper focused primarily on phenoxyherbicides and organic solvents. This study demonstrates well that when initial associations are observed between DDT and NHL, they usually are confounded by other chemical exposures. In this case, an OR of 2.4 that was statistically significant was reduced to 1.5 and was no longer statistically significant when other chemical exposures were adjusted for in the analyses.

In summary, this paper shows no evidence of a clear association between NHL and DDT exposure. Although the authors provide brief details of the methods used for analyses and pay little attention to DDT exposure, they did employ multivariate analyses to control for potential confounding "between exposures of interest." As in other studies that assessed the association between NHL and DDT exposure, they found that an initially elevated odds ratio that was statistically significant was lowered remarkably by controlling for confounding by other chemicals and was no longer significant. The consistency of the demonstration of confounding, when it is possible, of initially observed DDT-NHL associations by other chemical exposures is remarkable.

(5) Persson B, Fredriksson M, Olsen K, et al. Some occupational exposures as risk factors for malignant lymphomas, 1993. This is a report of a case-control study of 93 cases of NHL (and 31 cases of Hodgkin lymphoma) obtained from a regional cancer registry in Sweden and 204 community referents. The same referents were from a series of three other studies by

Flodin, et al., which makes this and the other studies overlapping and not independent. They found 4 cases and 3 controls with exposure to DDT for a crude OR of 3.0 and a logistic (adjusted) OR of 2.0 (90% CI = 0.3-13). The adjustment criteria are poorly defined only in a table footnote.

Note that the authors used a 90% CI rather than the conventional 95% CI. A 90% CI is less stringent for assessing statistical significance than a 95% CI and still the DDT results were far from significant and the interval was extremely broad. Most studies finding fewer than 5 exposed cases would not even try to estimate risk or statistical significance, let alone attempt to do meaningful multivariate analyses. Thus, the major problem with this study was that it was too small to properly assess associations with uncommon exposures such as DDT. Although the authors were less stringent than usual by employing 90% CIs rather than 95% CIs, they still did not find a statistically significant association between DDT and NHL.

(6) Hardell L, Eriksson M, Nordstrom M. Exposure to pesticides as risk factors for non-Hodgkin's lymphoma and hairy cell leukemia: Pooled analysis of two Swedish case-control studies, 2002. This is another study from Sweden that uses data from previous studies for re-analyses in an attempt to "enlarge the study size thereby allowing more precise risk estimates." Results are presented in the paper for the combined group of NHL and hairy cell leukemia cases. Subjects were 515 cases and 1141 controls, with 77 cases and 138 controls reporting DDT exposures. Exposure information was obtained via mailed, self-administered questionnaires supplemented by telephone queries if the data were "unclear." They found an OR of 1.27 (95% CI = 0.92-1.73) for DDT using univariate analysis. When latency from first exposure to diagnosis was considered, the OR for the >10-20 year latency period was higher than that for the longest (>30 year) latency period, although none of the ORs was statistically

significant. Specific data for the multivariate analysis of DDT is not provided, but the authors state that "[i]n the multivariate analysis no risk was found." The paper is unclear as to what variables were considered in the multivariate analyses.

This is another analysis of previous study data by this group of authors (data from the Hardell, et al., 1999 study were included in this study) to assess associations between NHL and pesticides and again only a slight, non-significant association with DDT was found. When multivariate analyses were done of DDT and NHL, "no risk was found." In conclusion, this is a reanalysis of previous studies that adds no new information on a relationship between DDT and NHL. Again, the study shows that weak associations between DDT and NHL tend to disappear when multivariate analyses are done, though in this case the nature of the multivariate analysis is not fully described.

(7) McDuffie HH, Pahwa P, McLaughlin JR, et al. Non-Hodgkin's lymphoma and specific pesticide exposures in men: Cross-Canada study of pesticides and health, 2001. This is a large, multi-center, case-control study of 517 NHL cases and 1,506 community controls. Cases were identified from Provincial cancer registries except in Quebec. Controls were identified via health insurance records, voter registration records and telephone listings. Exposure information was obtained by mailed questionnaires followed by a telephone interview for those reporting 10 hours/year or more of pesticide exposure, as well as for a 15% random sample of respondents. While the study was large, the number of DDT-exposed subjects was half that reported in a smaller case-control study reported by Hardell, et al., in 1999.

An unadjusted OR of 1.63 (1.03- 2.57) was found for DDT exposure based on 32 exposed cases and 59 exposed controls. The OR adjusted for medical variables and a family history of cancer was 1.73 (1.08-2.76). A dose-response relationship for DDT exposure was

reported. An OR of 1.75 (0.96-3.21) was found for the category of >0 and <2 days per year of DDT exposure compared to no exposure. An OR of 1.5 (0.77- 2.91) was found for the comparison of the >2 days per year group versus the never exposed group. No trend test result was reported. Curiously, in the paper's abstract, DDT is listed as one of the chemicals statistically significantly associated with NHL, but no odds ratio or confidence interval is provided as for the other chemicals listed as being associated with NHL.

However, when conditional logistic regression analyses were done on "individual chemical pesticides and important covariates" that were found to be statistically significant in univariate analyses ($p < 0.05$), DDT was included in the multivariate model and was **"found not to contribute significantly to the risk of NHL"** (Table 7). Thus, when the confounding effects of other pesticides and risk factors were considered in the analysis, DDT was not a significant risk factor for NHL. Interestingly, in Table 3, results are presented after adjustment for observed statistically significant medical variables and the OR for the association between DDT and NHL is slightly higher than the unadjusted OR. When adjustment was done for these same medical variables and for other chemical pesticide exposures together (Table 7), the results for DDT were no longer significant (no point estimate of the OR was presented for this analysis of DDT). This suggests that it was the other chemical pesticide exposures, per se, that were the underlying confounders in the previously observed DDT association.

In conclusion, this is a potentially misleading paper in terms of the authors' presentation of findings and conclusions. Although the authors suggest a statistically significantly increased NHL risk in DDT-exposed subjects, results of their multivariate analyses that control for confounding effects of other chemical exposures and non-chemical risk factors show that DDT

was "not found to contribute significantly to the risk of NHL." This finding is glossed over and not reported in the abstract or the paper's discussion section.

The study results for DDT are typical of what one would expect to find in an association that is confounded by other associations. In such situations, odds ratios are typically below 2.0, indicating a weak association. They also highlight another problem found in studies of DDT exposure and that is that people with DDT exposure often will have had multiple other chemical exposures. Unless one can tease apart the independent effects of DDT exposure, particularly in the face of observed weak associations, reported associations with DDT must be considered questionable and treated with skepticism. The authors performed analyses to assess DDT as an independent risk factor. When analyses were controlled for other pesticide exposures, no association with DDT remained. My interpretation of this paper and its analyses leads me to conclude that DDT is not a risk factor for NHL.

(8) Rothman N, Cantor KP, Blair A, et al. A nested case-control study of non-Hodgkin lymphoma and serum organochlorine residues, 1997. This paper reports result of a case-control study of 74 cases of NHL and 147 matched controls nested within the Washington County, MD, cohort study that has been conducted for many years by investigators at the Johns Hopkins University. In this present study, blood samples were collected when the subjects were first enrolled, an average of 12.1 years before diagnosis of cases' NHL. Serum levels of lipid-corrected concentrations of DDT, its metabolites and other organochlorines were determined for cases and controls. They found no significant differences in total lipid-corrected concentrations of DDT between cases and controls. Risk of NHL increased weakly and non-significantly with increasing DDT serum concentrations. The matched odds ratios by quartile of serum DDT levels ranged from 1.0 in the lowest group to 1.9 (95% CI = 0.8-4.5) in the highest group in univariate

analyses. When the DDT findings were adjusted for PCB levels, the ORs for DDT ranged from 1.0 to 1.2 (0.5-3.0) creating an almost flat trend with increasing levels.

This is an interesting and important study in that serum DDT levels were measured in cases and matched controls many years before the NHL cases were diagnosed. Thus, results were not confounded by disease-related weight loss and fat mobilization in cases. The Washington County cohort study is very well conducted and has yielded many important research findings that have stood the test of time. This study is persuasive and again demonstrates the important results of controlling analysis of DDT results for other confounding chemical exposures. It demonstrates clearly that blood levels of DDT found in the blood of healthy donors are not associated with the development of NHL in later years.

II. CASE-CONTROL STUDIES PERFORMING UNIVARIATE ANALYSES ONLY

A. Detailed Analyses of Case-Control Studies of DDT and NHL Without Biologic Specimens

(1) Hardell L and Eriksson M. A case-control study of non-Hodgkin lymphoma and exposure to pesticides, 1999. This is a study that obtained exposure data by self-administered mailed questionnaires and then was supplemented by telephone interview "if the information was unclear regarding specific exposures." Cases (N = 404) were from regional Swedish cancer registries and controls (N = 741) were from the Swedish National Population Registry. Specific data are provided for exposure to DDT and an unadjusted OR of 1.1 (0.7-1.7) was found based on 66 exposed cases and 107 exposed controls. This is a relatively large number of exposed subjects and provides a stable estimate of risk. No other analyses of DDT exposure were reported. While multivariate analyses are presented in Table 7, no data are presented for DDT since there were no striking or significant elevations of NHL risk associated with DDT in univariate analysis.

In conclusion, this study did not find any association between occupational exposure to DDT and NHL risk. An OR of 1.1 with a 95% confidence interval that includes the null value of 1.0 is a finding of no association and is not statistically significant. This finding is based on large numbers of DDT-exposed cases and controls and there is a very narrow confidence interval. This is a persuasive finding as a result. No multivariate analyses of DDT were reported, but were not necessary since no overall association was observed for DDT.

(2) Woods JS, Polissar L, Severson R, et al. Soft tissue sarcoma and non-Hodgkin's lymphoma in relation to phenoxyherbicide and chlorinated phenol exposure in Western Washington, 1987. This is a population-based case-control study of 576 NHL cases and 694 randomly selected community controls. Cases were identified from a population-based tumor registry in Western Washington State. Exposure data were collected by personal interviews. Four percent of the overall study population (cases and controls) was exposed to DDT. This translates into a total of 51 DDT-exposed total subjects which is far less than in other similarly sized studies.

The paper reports the results of several different analyses. An OR of 1.46 (0.8-2.8) was found among farmers who reported working with "the specific organochlorine insecticides DDT and chlordane." This provides no direct information on the NHL risk in farmers of exposure to DDT per se. Table 5 reports NHL risks "for potential chlorophenol exposure" in various wood working occupations and includes the manufacturing of chlorophenols (for which they found an OR of 1.72, 95% CI = 0.9-3.4, based on a 3.0% frequency of this exposure in the overall study population, which translates into 38 exposed subjects). "When all of these occupations involving potential exposure to chlorophenols were considered together," the risk of developing NHL was

0.99 (0.8-1.2). A pooled OR for specific DDT exposure among men with many occupational factors was 1.82 (1.04-3.2) based on 4.0 % exposure in the study population (Table 7).

The authors attempted to assess interactions between the various chemicals that were being assessed in the study using logistic regression analysis. Unfortunately, relatively crude analyses were done to assess interactions between phenoxyherbicides or chlorophenols and "organochlorine pesticides (DDT + chlordane)" and several other chemicals and risk factors. They report that "none of the interactions between chlorinated phenol or phenoxyherbicide exposure and these variables was statistically significant, with the exception of that between phenoxyherbicides and organic solvents as a risk factor for NHL." The authors state that the logistic regression analysis used "confirmed the magnitude of risks shown in Tables 6 and 7". No specific results of this analysis are presented. This analysis appears to have primarily assessed interactions rather than providing adjusted risk estimates for specific chemicals such as DDT.

So what does all this mean? This paper reports the results of a very large case-control study of NHL (576 male cases) with a very low number of cases and controls exposed to the chemical of interest, DDT. Based on their report of 4.0% of subjects having been exposed to DDT, only a total of 51 subjects – cases and controls – were exposed. Such small numbers are apt to result in unstable estimates of risk. Also, as a result, several analyses lump together DDT exposure with chlordane exposure. Results for the lumped category are non-significantly elevated for NHL in farmers. Risk of NHL was not elevated for the sum of the other occupational categories in Table 5. For the stratum of the occupational category of "manufacturer of chlorophenols" in Table 5, an OR for NHL of 1.72 (0.9-3.4) is found. This is an occupational group with presumed high exposure to these chemicals and yet the odds ratio is

low and not statistically significant. The only information on the specific NHL risk for DDT exposure is found in Table 7, an OR of 1.82 (1.04-3.2). While multivariate analysis to assess the possibly confounding effects of other chemical exposures were done, they were done for the lumped category of chlordane and DDT and only statistical significance was used to assess the interaction when exposed subject numbers were very small. Much more sophisticated multivariate analyses are done today than were done 20 years ago.

In summary, this is a study that was very limited in terms of the number of subjects with DDT exposure. Many analyses were done and only one sub-analysis found a statistically significant, weak association (OR less than 2.0) between DDT and risk of NHL. The more analyses that are done, the more likely the study is to find a chance association, particularly when the numbers of exposed subjects are small. Crude attempts to control for confounding of this association were made and no specific DDT information was provided in the text. Thus, it is quite likely that the weak results reported could be due to the confounding effects of other chemical exposures. As a result, the finding of a single variable showing a weak, but borderline statistically significant association between DDT exposure and NHL risk must be treated with scientific skepticism, as it is likely to be the result of confounding.

(3) Woods JS, Polissar L. Non-Hodgkin's lymphoma among phenoxy herbicide-exposed farm workers in western Washington State, 1989. This paper is a report of a case-control study of NHL conducted in Washington that was primarily focused on phenoxy herbicides and NHL risk. Subjects were NHL cases and their matched controls from the first study by Woods, et al. (1987). The paper is brief and is unclear, but appears to have only included cases and controls who were ever employed as farmers (N = 181 cases and 196 controls) as subjects. Thus, it is a subset analysis of a prior, larger study. An OR of 1.68 (95%

CI = 0.9-3.3) was found for DDT exposure and there is no indication of the number of exposed subjects on which the OR is based. There was no further elaboration on DDT beyond this OR presented in one of the tables. Curiously, in the first study by Woods et al. (1987), they report an OR of 1.46 (0.8-2.8) for the combined category of DDT and chlordane exposure (p. 902) in farmers. In the second study, they found ORs of 1.56 for NHL and chlordane and an OR of 1.68 for DDT. Both of these values are higher than the combined result.

While interactions between phenoxy herbicides and other agricultural chemicals were assessed, there was no attempt to assess confounding of the DDT finding by other agricultural exposures. Thus again, a weak association between DDT and NHL is observed with no attempt made to adjust for confounding by other exposures. In this paper, however, the weak elevation of the OR was not statistically significant.

This paper is an apparent second analysis of the first Washington study, and it is unclear as to how many of the analyses differ from those in the first study. However, this paper illustrates that the conduct of many analyses of the same data set will lead to disparate results simply on the basis of chance. In this sub-analysis of the same data set from the first study by Woods, et al., an elevated risk of NHL was found for farmers and exposure to DDT, but this finding was even weaker than the previously reported DDT association and was not statistically significant. No further attempts were made this time to assess the confounding effects of other chemical exposures even though farmers are a group with highly varied exposures to a multitude of different chemicals – ranging from pesticides and herbicides to motor fuels and fertilizers.

In summary, this new paper by Woods, et al., uses the same data set and many of the same analyses from their first paper (discussed previously). However, this time for a subset of the same NHL cases from the first study, they found a non-significant, weak elevation of NHL

risk in farmers exposed to DDT. No further attempt was made to deal with confounding of this finding by other chemical exposures. Thus, it adds little new information other than to illustrate the likely occurrence of chance and/or confounding in studies such as this and Woods, et al. (1987) where small numbers of subjects are exposed to the risk factor at issue.

(4) Persson B, Dahlwander A-M, Fredriksson M, et al. Malignant lymphomas and occupational exposures, 1989. This is a Swedish, hospital-based, case-control study of 106 NHL cases and 275 controls. The community controls were from two other case-control studies (Flodin, et al., 1987 and 1988). Exposure information was obtained by mailed questionnaires completed by the study subjects. The researchers imputed levels of exposure from the responses. Specific data on DDT exposure are presented with an OR of 0, based on no exposed NHL cases and 3 exposed controls.

It should be pointed out that this is the third study utilizing the same control group. All of these studies found exposure excesses in cases relative to the common set of controls, raising the possibility that the controls were a relatively underexposed group of subjects with regard to occupational exposures. Nevertheless, while the study is relatively small and can provide only limited information, more controls than cases had exposure to DDT with an OR of 0 for the association between DDT and NHL. Thus, the results of this study are completely negative for DDT.

(5) Cantor KP, Blair A, Everett G, et al. Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota, 1992. This is a report of a very large case-control study of NHL in two states with large populations of agricultural workers. The study was designed to assess agricultural risk factors for NHL and numerous analyses of suspected risk factors were done. In-person interviews were used to

collect exposure histories from 622 white men with NHL and 1,245 population-based controls in Iowa and Minnesota. Cases were ascertained from the Iowa State Health Registry and from a special surveillance of Minnesota hospital and pathology laboratory records from 1981-1983. Population-based controls were similarly interviewed.

The study's findings regarding DDT exposure were as follows:

- The OR for having ever handled DDT as an animal insecticide was 1.2 (95% CI = 0.9-1.7).
- The OR for having handled DDT as an animal insecticide prior to 1965 was 1.3 (0.9-1.8).
- The OR for having ever handled DDT as a crop insecticide was 1.7 (1.2-2.6),
- The OR for having ever handled DDT as a crop insecticide prior to 1965 was 1.8 (1.1-2.7).
- The OR for having ever handled DDT as an animal insecticide with protective equipment was 1.2 (0.9-1.7)
- The OR for having ever handled DDT as an animal insecticide without protective equipment was 1.3 (0.9-1.8).
- The OR for having ever handled DDT as a crop insecticide with protective equipment was 1.7 (1.2-2.6).
- The OR for having ever handled DDT as a crop insecticide without protective equipment was 2.0 (1.3-3.1).
- The OR for Iowa subjects who ever used DDT prior to 1965 as an animal insecticide was 0.9 (0.5-1.5).

- The OR for Minnesota subjects who ever used DDT prior to 1965 as an animal insecticide was 1.7 (1.1-2.7).
- The OR for Iowa subjects who ever used DDT prior to 1965 as a crop insecticide was 1.5 (0.9-2.6).
- The OR for Minnesota subjects who ever used DDT prior to 1965 as a crop insecticide was 2.3 (1.1-4.8)
- When analyses were stratified by state, the DDT results were not statistically significant for Iowa subjects and were statistically significant for Minnesota subjects.

This paper reports numerous analyses of DDT and other agricultural chemical exposures. While the observed ORs for DDT exposure were almost all greater than 1.0, only two reached the level of 2.0. Of the twelve DDT findings, six were statistically significant. Thus, numerous stratified analyses were done and all ORs were 2.3 or less and only half were statistically significant.

An important limitation of this study was its inability to control for other chemical exposures in farmers exposed to DDT. The authors themselves recognize this problem and state: "Interpretation of results regarding individual pesticides is fraught with difficulties, including the problems of interpreting risk of individual factors in the multiple exposure setting of modern agriculture as well as the chance occurrence of finding positive associations with multiple comparisons." I agree fully with the authors' statement. As later studies have found, even those using the same data set as this study, when multiple chemical exposures are controlled for in analyses, the associations between DDT and NHL are lessened, no longer statistically significant

and frequently are reduced to the null value of one. These later studies demonstrate the tremendous impact of confounding by other agricultural chemicals.

In summary, this is a large case-control study that performed a very large number of analyses of their data. Of the 12 results reported, all but one OR was above 1.0, with two above 2.0. Half of the reported ORs were statistically significantly elevated. There is no mention of how many more analyses were done that found no association and were not reported. Taken by itself, this paper suggests that there are weak, but consistent, associations between DDT exposure and NHL risk. However, this paper did not attempt to assess or control for the effects of confounding variables on the study results, a concern raised by the authors themselves. This concern is validated by the results of later more sophisticated statistical analyses of the same data (Baris, et al., 1998; De Roos, et al., 2003). These later studies found that when other chemical exposures were adjusted in multivariate analyses, DDT was no longer associated with risk of NHL. Thus, the Cantor, et al., findings do not hold up in later studies of the same data and confirm Canter, et al.'s stated suspicions that their findings were due to confounding or chance.

(6) Cantor KP, Blair A, Brown LM, et al. Letter to the Editor: Correspondence Re: K. P. Cantor, et al., Pesticides and Other Agricultural Risk Factors for Non-Hodgkin's Lymphoma Among Men in Iowa and Minnesota, Cancer Res. 52:2447-2455, 1992, (1993). This letter to the editor includes new data that supplements the data provided in Cantor, et al., 1992. After the initial interviews for the project reported in the Cantor, et al., 1992 paper, another study evaluated NHL risk in relation to the annual number of days of pesticide use. As a result, the authors conducted a supplemental interview of Iowa residents to obtain similar data. This portion of the study involved 107 cases and 203 controls from Iowa (or their next of kin). Data were collected by a supplemental telephone interview. Many subjects from the Cantor, et

al., 1992 study had died, and proxies were the respondents for 55% of cases and 28% of controls. These findings originally were included in the first Cantor, et al., 1992 manuscript, but were removed at the suggestion of journal peer reviewers.

The authors found an OR of 1.2 (0.5-2.8) for NHL and DDT use of 1-4 days per year as a crop insecticide. The OR for 5-9 days per year of DDT use was 1.6 (0.4-5.6) and for 10+ days of use per year, the OR was 1.7 (0.6-4.8). The ORs for DDT use as an animal insecticide were 0.3 (0.1-0.8) for 1-4 days per year of use; 0.5 (0.1-1.8) for 5-9 days per year of use; and 0.9 (0.4-2.0) for 10+ days per year of use. Because "time delay, different method of data collection, and participation of more proxies likely introduced substantial exposure misclassification that is likely to mask exposure-response gradients," the authors "consider these findings to be very weak evidence either for or against the possibility of a causal association with any single pesticide exposure."

I agree with the authors' conclusion that these data provide very little support for a causal association between DDT and NHL. There was a statistically significant protective association between NHL and DDT use as an animal insecticide for 1-4 days per year. Aside from this protective effect, none of the other DDT findings were statistically significant.

(7) Lee WJ, Cantor KP, Berzofsky JA, et al. Non-Hodgkin's lymphoma among asthmatics exposed to pesticides, 2004. This is a study based on two NCI, population-based case-control studies of NHL conducted in three mid-western states, Iowa, Minnesota and Nebraska. Exposure data were collected by interviews. Cases were derived from State Tumor registries and hospitals and pathology laboratories. Community controls were frequency matched to cases by random-digit telephone dialing and Medicare records. This study utilizes the same data as other case-control studies (Baris, et al. 1998; De Roos, et al. 2003). Using the

same merged study base, this study found an OR for DDT of 1.2 (0.9-1.5) in non-asthmatics (based on 158 exposed cases and 313 exposed controls) and an OR of 1.2 (0.6-2.4) in asthmatics (based on 11 cases and 24 controls exposed). There was no discussion of DDT and NHL risk in the paper.

The Lee, et al., study uses univariate analyses that are adjusted only for age, vital status and State. This is in contrast to the 1998 and 2003 studies that were able to adjust simultaneously for the confounding effects of other chemical exposures. Unfortunately, this study did not perform multivariate analyses adjusting for the confounding effects of exposures to other chemicals. This is probably due to the very small number of asthmatics with DDT exposure and NHL (N = 11). Furthermore, this study focuses on only a subset analysis of subjects in the farmer studies, namely asthmatics. Such multiple subset analyses of a single database increase the likelihood of finding chance associations.

In conclusion, this study found very weak, non-significant associations between DDT and NHL that did not differ between asthmatics and non-asthmatics. This lack of difference runs counter to the paper's other findings regarding pesticides, NHL and asthma. Given the weakness of the association found (OR = 1.2, a value very close to the null value of 1.0), it is quite likely that the DDT finding is due to chance or confounded by other chemical exposures, as was demonstrated in the studies by Baris, et al. (1998), and De Roos, et al. (2003) using data from the same study.

(8) Zahm S, Weisenburger DD, Saal RC, et al. The role of agricultural pesticide use in the development of non-Hodgkin's lymphoma in women, 1993. This is a report of a case-control study of 119 women with NHL and 471 controls who resided in Nebraska. This paper relies on data used in several other studies covered in this report (Baris, et al., 1998; De Roos, et

al. 2003), but only analyzes a subset of the data, i.e., data for women. Community controls were selected by random-digit dialing and Medicare records. Telephone interviews were used for exposure information.

No data for DDT and NHL are presented in the paper's tables. However, the text states that there was an OR for DDT exposure of 1.7 based on 16 exposed cases and 36 controls. No confidence interval or p-value for this finding is provided and there is no further discussion of DDT in the paper. The authors conclude: "No individual insecticide was associated with a significant risk of NHL among women." Thus, this paper provides little information regarding NHL and DDT beyond there being a weak association that was not statistically significant, and for which there was no evaluation of confounding by other exposures. Additionally, this study is yet another example of a subset analysis being performed on the same database, the NCI farmers' studies.

(9) Schroeder JC, Olshan AF, Baric R, et al. Agricultural risk factors for t(14;18) subtypes of non-Hodgkin's lymphoma, 2001. This is a re-analysis of data from a previous case-control study of NHL and farm exposures (Cantor, et al. 1992). Data are from the NCI farmers' studies that are the basis of many of the papers discussed in this review. Tumor specimens were obtained for 40% of the 622 original Iowa and Minnesota study cases and only 29% (182) were assayed successfully for the t(14;18) translocation. Of these, 68 cases were positive for the translocation and 114 cases were negative. Previously collected interview data for these cases and their controls served as the basis for the new study's analyses. In Table 5, the OR for DDT and NHL in the t(14;18)-positive NHL cases and their controls was 1.1 (95% CI = 0.6-1.9). The OR for DDT in the translocation-negative cases was 1.2 (0.8-1.7).

In the methods section, the authors discuss the problem with the fact that, "over 70% of the FARM study could not be classified as t(14:18)-positive or –negative." Because the authors were concerned that "ignoring unclassified cases may have biased estimates for case-subtypes compared with controls," they used elaborate statistical methods to include exposure data from unclassified cases. As an example, the authors "constructed pseudo-data with observations missing case-subtype data apportioned to case-subtypes according to the probabilities for their covariate substratum," and they then "modeled these data to determine new maximum likelihood probabilities, which were used to assign new pseudo-data." It is unclear from this extremely confusing text as to how these and other statistical manipulations of the data impinge on the results presented in Table 5.

This study was an interesting attempt at sub-classifying NHL on a biologic basis for analysis of risks associated with agriculture. Unfortunately, histologic materials only could be obtained for 29% of NHL cases, a proportion that could lead to significant bias, and this study's authors recognize this. The use of arcane methods to create expected numbers for missing subjects' data does not improve the quality of this study.

Based on the small proportion of cases for whom tissues were available, for DDT, there was no difference in those NHL cases with or without the t(14;18) chromosomal translocation. The study did not assess whether or not these weak, non-significant associations were the result of confounding by other exposures ("Weak associations may have resulted from confounding, since we were unable to model individual agricultural exposures simultaneously."). The weak, non-significant ORs of 1.1 and 1.2 in this study suggest little or no association and are likely the results of confounding by other exposures. Moreover, this is another example of the many sub-

set analyses performed on the NCI farmer studies data. For all of the reasons above, at best, the study could be viewed as hypothesis generating, rather than as hypothesis testing.

(10) Persson B, Fredrikson M. Some risk factors for non-Hodgkin's lymphoma, 1999. This paper merges data from two other studies discussed above (Persson, et al., 1989, 1993). The merger was done to deal with the problem of limited sample sizes: "To overcome some of the problems with limited numbers, the present study was set up as a pooled analysis of two previously published case-referent studies, focusing on some rare exposures which could not be studied before." Thus, it is not a separate and independent study. Nevertheless, the merged study was still very limited in size with regard to numbers of subjects with DDT exposure. They studied 199 NHL cases and 479 referents and found an OR for DDT and NHL of 1.4 (95% CI = 0.3-5.9) based on 4 exposed cases. Although DDT data are presented in the tables, there is not a single mention of DDT in the text. This underscores the lack of any meaningful elevation of NHL risk associated with DDT in this pooled analysis.

This study is a pooled analysis of two very small studies that adds no independent new information on DDT. The study uses cases from two other studies and compares exposures with a group of controls used in many other studies by these authors. Moreover, no attempt was made to adjust the DDT finding for exposures to other chemicals and the slight elevation in OR for DDT may well be due to confounding. In conclusion, the pooling of results brings the OR for DDT closer to 1.0. The attempt to shed new light on the authors' previous studies shows that with larger subject numbers, the OR for DDT and NHL only comes closer to the null value, i.e., a finding of no association.

(11) Colt JS, Severson RK, Lubin J, et al. Organochlorines in carpet dust and non-Hodgkin lymphoma, 2005. This is a population-based case-control study using four

Surveillance, Epidemiology, and End Results (SEER) reporting sites to identify cases. Controls were selected by random-digit telephone dialing or for older subjects, Medicare files. Organochlorine concentrations were measured in vacuum cleaner bag dust in the carpets of 603 cases and 443 controls. There were many missing values for carpet dust levels of chemicals and multiple statistical estimations were made. Only Caucasian subjects were studied and subjects had to have owned their carpets for at least five years.

No association with DDT was found, the overall OR was 0.9 (95% CI = 0.7-1.2), and an OR of 1.3 (1.0-1.7) was found for DDE. NHL risk was 1.6 (1.1-2.2) for the top tertile of DDE concentration and there was a statistically significant trend with increasing concentration (p=0.02), but the dose-response was not monotonic, i.e., there was no consistent increase in ORs with increasing DDE levels. Thus, for the middle tertile of DDE levels, the OR declined to 1.1 (0.7-1.6) from an OR of 1.3 for the lowest tertile. Additionally, the DDE results were different for men and women. For men, the OR for DDE was 1.6 (1.1-2.3) and for women, it was 1.1 (0.7-1.5). If there was a true association, it seems paradoxical since women are likely to spend more time in the home and to do more of the vacuum cleaning in the household. Importantly, the authors failed to provide similar results for DDT. The authors fully recognize the oddness of their results and state: "Our results for DDE were less consistent. An association was observed only in men, the dose-response relationship was not monotonic, and the concurrent finding of no association for DDT is difficult to explain." I agree fully with the authors' interpretation of their results.

In summary, this is a rather odd case-control study using carpet dust as a surrogate measure of past exposures to a variety of chemicals. It is like a cross-sectional study in that levels of chemicals in carpets are measured after the time of cases' diagnosis. While no one

knows whether dust levels are valid measures of past exposure, particularly remote past exposures, the authors assume in this study that levels are a valid surrogate of past household exposure levels. Further, in determining carpet dust levels of chemicals, there were many problems with missing values or measurement of specific chemicals. As a result, numerous imputations and other estimations of levels had to be computed. Thus, it is likely that results of the study were affected by imprecise estimates of specific chemical levels. Further concern about the credibility of this study is generated by some paradoxical findings regarding DDT and DDE levels in carpet dust. The study found a weak association for NHL and DDE, but not for DDT. The trend for NHL risk and DDE exposure, while statistically significant, was odd in that the OR for the middle tertile of DDE levels was 1.1. The very large sample sizes in this study make small increases in risk statistically significant. Equally puzzling is their observation of an association between DDE levels and NHL for men, but not for women.

In conclusion, no association was found between DDT in carpet dust and NHL, and the DDE findings were inconsistent with the DDT findings. The authors had no explanation for the inconsistencies observed. Nevertheless, this was an extremely large study with more than adequate statistical power to detect an association with DDT if it existed; yet they found no such association.

(12) Eriksson M, Hardell L, Carlberg M, et al. Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis, 2008. This is a report of a population-based case-control study of 995 NHL cases and 1,016 population registry controls in Sweden. Cases were identified from participating university clinics and controls from the Swedish population registry. Controls were frequency-matched in 10-year age and sex group categories. The study was designed to evaluate exposures to pesticides as risk factors for NHL.

Exposure data were collected by self-administered questionnaires supplemented by telephone interviews "if important data were lacking, incomplete or unclear." The telephone interviewers were blinded as to case/control status of the subjects.

Fifty cases and 37 controls reported exposure to DDT for an OR of 1.46 (95% CI = 0.94-2.28). These subjects were subdivided into two exposure categories, ≤37 days (OR = 1.17, 0.62-2.22) and >37 days (OR = 1.76, 0.97-3.20). The authors also found an OR of 2.14 (1.05-4.40) for DDT and follicular, grade I-III NHL, based on 165 such cases. Multivariate analyses were not performed for any DDT finding because DDT did not meet the authors' criteria for performing a multivariate analysis, which was done only for "agents with statistically significant increased OR, or with an OR > 1.50 and at least 10 exposed subjects." The authors state that "[s]ince mixed exposure to several pesticides was more a rule than an exception, and all single agents were analyzed without adjusting for other exposures, a multivariate analysis was made to elucidate the relative importance of different pesticides." The authors point out that results from multivariate analyses were "in general lower" than those from univariate analyses.

In conclusion, the study found a weak association between reported DDT exposure and NHL that was not statistically significant. It also found a moderate association between reported DDT exposure and follicular NHL that was statistically significant. However, no multivariate analyses of the DDT-NHL associations were performed despite the authors' observation that ORs were generally lower in multivariate analyses than univariate analyses and that mixed exposure to several pesticides was frequent. Based on their findings, the authors conclude as follows: "The organochlorine DDT has shown suggestive but rarely significant association with NHL in some studies. Our study showed a moderately but not significant increase OR for

exposure to DDT." No mention is made of DDT and NHL in the paper's abstract. I agree with the authors' conclusion that the study found no significant OR for exposure to DDT.

B. Detailed Analyses of Case-Control Studies of DDT and NHL /Chronic Lymphocytic Leukemia

Until recently CLL and NHL were considered to be different pathologic entities, with CLL being included in the broad category of leukemias. The new WHO classification of lymphomas includes CLL in the category of lymphomas on pathologic and immunologic bases. Most epidemiologists still consider CLL to be a disease entity separate from NHL. However, to be complete in my literature review, I am including those papers that lump CLL and NHL together in their analyses.

(1) Miligi L, Constantini AS, Bolejack V, et al. Non-Hodgkin's lymphoma, leukemia, and exposures in agriculture: Results from the Italian Muticenter Case-control Study, 2003. This is a report of a multi-site, very large, case-control study from Italy. The study was done in 11 areas, five of which had a high prevalence of agricultural activities. They accrued 1,145 NHL cases, 430 leukemia cases and 1,232 general population controls that were interviewed. For the combined groups of NHL and chronic lymphocytic leukemia (CLL), they found an OR of 0.6 in men (0.3-1.1) and 0.3 in women (0.1-0.8) for DDT exposure. These ORs were based on 21 DDT-exposed male cases and 5 exposed female cases. There is no discussion of these DDT findings anywhere in the paper and, curiously, the significantly protective effect of DDT exposure in women is ignored. The authors state: "Table V shows ORs for NHL for exposures to major classes of insecticides. No exposures were found to be significantly associated with NHL ..." Yet the ORs for women and DDT, as well as the category of diphenylethanes, were 0.3 (95% CI = 0.1-0.8) in Table V, two statistically significant values demonstrating a protective effect.

This large case-control study lumps together NHL and CLL and presents findings only for the combined category. Despite the large number of study subjects, only 21 male cases and 5 female cases were reportedly exposed to DDT in the combined disease group. The study found protective effects for DDT for both men and women, with the value for women being statistically significant, findings the authors ignore. In summary, this study combines NHL and CLL to generate a study with a very large sample size that finds that DDT protects against NHL (and CLL).

(2) Nanni O, Amadori D, Lugaresi, et al. Chronic lymphocytic leukaemias and non-Hodgkin's lymphomas by histologic type in farming-animal breeding workers: A population case-control study based on a priori exposure matrices, 1996. This was an Italian, population-based, case-control study of 187 cases of CLL and NHL (which included 162 NHL cases, 23 CLL cases and 2 mycosis fungoides cases) and 977 controls from the general population. All cases were from a single province in Northeast Italy and matched controls were selected from the general population. Exposure information was obtained by direct interview and also by use of an a priori job-exposure matrix to estimate pesticide exposures when a crop disease was reported.

For NHL and CLL combined, the study found an OR of 1.74 (0.93-3.27) based on recall of DDT exposure in 27 combined cases and 61 controls. They also found an OR of 1.70 (0.91-3.17), based on matrix-estimated DDT exposures in 28 cases and 65 controls. When the analysis was restricted to CLL and low grade NHLs (31 of the total of 162 NHL cases), they found ORs of 2.33 (0.93-5.85) for recalled exposure to DDT and 2.16 (0.86-5.43) for estimated DDT exposure. No data were presented for the other types of NHLs. Thus, the authors found weak, non-statistically significant associations between DDT exposure and NHL for all NHL cases.

When a subset analysis was done for a small portion of the NHL cases, the ORs went above 2.0, but again, the results were not statistically significant. Further, there are serious concerns about the analyses performed in this study.

The paper has several important limitations. Foremost, there was no attempt made to perform multivariate analyses to control for confounding effects of exposure to multiple chemicals despite the authors recognition that "[i]n farmer-animal breeders, the use of each pesticide was strongly correlated with the use of the others." Because there were eight main categories of pesticides and the numbers of individual exposures to each agent were relatively small, the authors state that "it was impossible to introduce in the same model more than one pesticide category to evaluate their separate effects." Given their observation of strong correlations between the other various pesticides and the weak associations observed for DDT, it is highly likely that the DDT association is confounded by other chemical exposures as other larger studies consistently have demonstrated.

An additional concern is with the artificial slicing of the data to produce a category of combined CLL and low grade lymphomas that result in non-significant ORs for DDT of 2.33 (0.93-5.85) by recall and 2.16 (0.86-5.43) by job matrix assessment of exposures. First, the combination of CLL and a subset of other lymphomas is unusual for epidemiologic studies. Second, no similar results are presented for other categories of NHL such as medium or high grade NHL. Are the ORs for these other categories less than 1.0 and demonstrative of a protective effect? Third, it is unclear as to how they selected the 31 cases with CLL or low grade lymphomas for analysis in Table 3. As described in the materials and methods section, there were 79 cases in this category – 56 with low grade NHL and 23 with CLL. Though there were 79 total cases in this category, the authors inexplicably analyzed data for only 31 cases. This

suggests they performed an analysis only for a subset of the subset. The analysis of results for this arbitrary subset is suspicious of "data dredging" to demonstrate stronger associations. Nevertheless, neither of the results for this subset analysis is statistically significant for DDT.

In conclusion, this study is fraught with methodologic problems that render interpretation of the results very difficult. Nevertheless, none of their findings for DDT are persuasive or statistically significant. The performance of multivariate analyses would have strengthened the paper and likely would have demonstrated confounding.

C. Detailed Analyses of Case-Control Studies of DDT Levels in Biologic Specimens and NHL

(1) De Roos AJ, Hartge P, Lubin JH, et al. Persistent organochlorine chemicals in plasma and risk of non-Hodgkin's lymphoma, 2005. This is a case-control study in which congener of PCBs, furans and pesticides or pesticide metabolites in plasma were measured and compared in 100 untreated NHL cases and 100 controls. Subjects were derived from a large NCI case-control study that also was used for the study by Colt, et al. 2005. The authors used the same multiple imputation procedures to fill in missing values as were used in the Colt, et al., study and conditional logistic regression methods for their analyses. For DDE and NHL, they found an OR = 0.85 (95% CI = 0.37-1.94) when comparing the highest versus the lowest quartiles of measured plasma DDE levels. Curiously, for the third quartile of DDE levels (>450.5-872.5), the OR demonstrated a highly statistically significant protective effect (OR = 0.33, 0.14-0.80), a finding not discussed by the authors. For DDT and NHL, the odds ratio for the highest versus lowest DDT categories was 1.2 (0.39-3.70). The text of the paper reports that for "extreme levels of exposure, [the authors] found elevated ORs for p,p'-DDT (OR, 3.3; 95% CI, 0.7-15.9)..." and other chemicals. Thus, they found no significant association at all between

NHL and DDT or DDE in subjects' serum, save for a protective effect of moderately high levels of DDE.

This study suffers from the concern typical to all sero-epidemiologic studies that study serum levels of fat-deposited substances after a cancer diagnosis – namely, that weight loss and cachexia associated with cancer might mobilize tissue stores of chemicals. The authors tried to minimize this effect by studying cases before initiation of treatment, but many cancer patients present with weight loss at diagnosis. Thus, any results from a study such as this must be interpreted cautiously. Additionally, this study represents another subset analysis of an NCI study which, like Lee, et al. (2004), Zahm, et al. (1993), raises concerns that multiple looks at the same data set can produce many chance findings.

An additional concern is with the authors failure to provide information on the specific levels that constitute "extreme levels" of the chemicals studied in Table 5. They also do not provide the numbers of cases and controls in this category, but the authors recognize that such numbers for DDT were small. Nevertheless, such "extreme level" subjects would be subsumed in the data in Table 5 for the top quartile of DDT levels (>9.9). The OR for this quartile (1.2) was only slightly increased over the null value of 1.0, was not statistically significant, and had a broad confidence interval as well.

Another concern is that the authors apparently were biased towards emphasizing an elevated finding for DDT (OR = 3.3) that was provided only in the text and was based on very small numbers (that are unspecified) and was not significant. This contrasts with their ignoring a weak protective effect overall for DDE, with a strong, statistically significant, protective effect for one of the levels of DDE (OR of 0.33, which is the inverse equivalent of an OR of 3.3). In

summary, this is a paper that provides little useful information regarding the association between DDT exposure and NHL.

(2) Quintana PJE, Delfino RJ, Korrick S, et al. Adipose tissue levels of organochlorine pesticides and polychlorinated biphenyls and risk of non-Hodgkin's lymphoma, 2004. This paper reports the results of a nested case-control study of pesticide levels in cadaver and surgical specimens and risk of NHL. Subjects were selected from a national repository of adipose tissue collected by the U.S. EPA. There were 175 cases (167 case specimens obtained at autopsy, 8 specimens were surgical samples) and 481 controls (173 controls from accident victims and 308 with myocardial infarction).

For DDT, there was a statistically significant trend with increasing concentrations, but there were no significant elevations of DDT residues in any of the exposure categories. For the highest level of DDT residue, the OR only reached the level of 1.39 (95% CI = 0.78-2.47). Based on these findings, the authors conclude: "Rothman, et al. (1997) also reported no significant association between total lipid-corrected serum concentrations of DDT and risk of NHL, and this finding is supported by the present study, in which we found no clear association between exposure to DDT and NHL." I agree with this conclusion and also conclude that the study found no clear association between DDT and NHL.

For DDE, they found an odds ratio of 1.99 (1.14-3.47) for the highest tissue concentration of DDE (>7.21 ppm). Oddly, for the DDE tissue residue level of 2.40-4.38 ppm, they found an OR of 0.53 (0.29-0.96), which is a statistically significant protective effect. As will be shown below, this slight elevation was lowered remarkably when statistical adjustment was made for other confounding chemical residues.

When correlations between the pesticides studied were assessed, numerous low or moderate correlations between the various chemicals were observed. As a result, they assessed "between-pesticide confounding" using conditional regression models. When both heptachlor and DDE were included in their statistical model, the odds ratio for the highest quartile of DDE residues (>7.21 ppm) was reduced by 41.2% to 1.32 and was no longer statistically significant (0.73-2.39). The ORs for the highest quartile of DDE residue levels were similarly reduced and became non-significant in models containing pairs of pesticides. Thus, the weak association observed between cadaver tissue levels of DDE and NHL in this study likely was confounded by other chemical residues. Once again, this study demonstrates that weak statistical associations between DDT and NHL risk usually prove to be the result of confounding by other chemicals when multivariate analyses are done.

However, this study also has certain problems similar to the De Roos, et al., study. The biologic materials collected for the study likely introduced a bias. Cases were decedents from NHL and death in this cancer as in most cancers is preceded by cachexia and other severe metabolic changes. Controls were more likely to have died a sudden death (from accidents or heart attacks) and thus would not have had any pre-mortem severe weight loss or metabolic changes. In fact, 308 of the 481 controls were decedents from myocardial infarction, a group that is likely to have suffered from greater overweight before death than the general population. Since it was adipose tissue that was the basis for the chemical assays, there is almost certainly a large difference in the amount, if not the quality, of adipose tissue in the case and control groups.

In summary, this study likely had significant design flaws that could introduce considerable bias. Nevertheless, the authors still did not find any significant association between NHL and DDT, nor did they find any significant association for DDE in tissues after controlling

for confounding by other chemicals. Although this study should be interpreted cautiously, it is a negative study that provides no evidence of an association between tissue levels of DDT or DDE in cadavers and risk of NHL.

(3) Hardell L, Van Bavel B, Lindstrom G, et al. Higher concentrations of specific polychlorinated biphenyl congeners in adipose tissue from non-Hodgkin's lymphoma patients compared with controls without a malignant disease, 1996. This is a very small case-control study of 28 NHL patients and 17 surgical controls that compared, among many comparisons of other chemicals, DDE levels in adipose tissues of subjects. The source of patients and controls is not stated. Samples of adipose tissue were obtained surgically from subjects' "abdominal wall." A mean DDE concentration of 1420 ng/g lipid was found in cases compared to 1068 in controls (P = 0.29), with considerable overlap in the range of levels between the two groups. Thus, cases did not differ significantly from controls in tissue levels of DDE, a major metabolite of DDT. As in the previous paper by Hardell, et al. (1994), the primary focus of this paper was on other chemicals and there is little discussion of the DDE results.

This study measures adipose tissue levels of a DDT metabolite after a diagnosis of NHL was made and is thus subject to the possible effects of weight loss or other cancer-related metabolic changes on tissue DDE levels. Nevertheless, the authors conclude: "In this investigation no association between NHL and DDE concentration in adipose tissue was found." In summary, I agree with the authors' conclusion, but believe this study is very small, is subject to bias from possible weight loss in cases, and is analyzed in a rudimentary way. As a result, this study provides little useful information.

(4) Hardell L, Eriksson M, Lindstrom G, et al. Case-control study on concentrations of organohalogen compounds and titers of antibodies to Epstein-Barr virus

antigens in the etiology of non-Hodgkin lymphoma and exposure to pesticides, 2001. This is a two-part case-control study in which adipose tissue samples were obtained from 50 NHL cases and 47 controls in the first part and blood samples from 32 NHL cases and 36 controls in the second part. Cases and controls were obtained at two Swedish hospitals. It appears that the 50 NHL cases in the first part included the 28 cases from the Hardell, et al., (1996) study of adipose tissues and pesticides. In this study, an odds ratio of 1.2 (0.60-2.5 95% CI) was found for NHL and DDE. An OR of 1.2 was found for multivariate analysis as well, but with a wider confidence interval. Interactions between DDE levels and EBV early antigen antibody titers were evaluated with no significant associations.

This study has several problems in addition to its small size. First, it must be remembered that this study is not independent from the Hardell, et al. (1996) paper on the same subject. Second, it uses both tissue and serum levels of chemicals and combines them for analyses. Tissue levels would reflect past exposure levels while serum levels would reflect recent exposure levels and/or mobilization of the chemicals from fat stores, as in a patient losing weight. Third, the EBV interactions only could be determined in the subset of subjects providing blood samples and analyses of interactions only considered EBV early antigen antibodies. Thus, the EBV results are limited and hard to evaluate. Nevertheless, no association between tissue and blood levels of DDE and NHL were found.

(5) Spinelli, J, Ng, C, Weber, J, et al. Organochlorines and risk of non-Hodgkin lymphoma, 2007. This is a population-based case-control study of NHL done in British Columbia, Canada. A subset of participants in a larger NHL case-control study, 422 cases and 460 frequency-matched, community controls, were studied. Exposure information was collected

by self-administered questionnaires and CATI. Blood samples were obtained from cases after diagnosis and from controls.

The study found for "p',p'-DDT" an OR for level > 3.24 ng/g versus no level of 0.91 (0.68-1.20). For p,p'-DDE, the OR for the highest quartile versus the lowest quartile was 1.42 (0.92-2.19). A trend test for the quartiles of DDE levels yielded a p-value of 0.027, yet there was no statistically significant finding for any of the quartiles. For follicular lymphoma, the lymphoma type of the plaintiff Ms. Garza, ORs were 0.7 (0.5-1.1) for p,p'-DDT and 1.8 (0.9-3.3) for p,p'-DDE. Multivariate analyses were not performed for DDE or DDT.

This study found no statistically significant associations between NHL and DDE or DDT. The authors performed a trend test on the results of analysis of p,p'-DDE by quartiles of blood levels and found a statistically significant trend. As a result, they state in the paper's abstract that there was a significant association with p,p'-DDE. This is an incorrect conclusion. The analysis of the associations of p,p'-DDE by quartiles found no significant associations for any of the levels. For the highest level, > 512.02 ng/g, the OR was 1.42 (0.92-2.19), and none of the other strata was significant either. The only conclusion they can reach is that there was a significant trend in non-significant data. My interpretation of these data is that there is no statistically significant association between p,p'-DDE and NHL in their study.

This study provides interesting insights into the issue of the effects of weight loss and disease in NHL patients and their effects on studies of DDT and DDE content in blood and adipose tissues. The authors attempted to deal with weight loss in NHL patients and its possible biasing effects on plasma levels of organochlorine compounds by excluding cases found to have over 10% weight loss prior to diagnosis. Nevertheless, the authors report that "organochlorine study cases had significantly lower measured plasma total lipids than controls (6.66 vs. 7.14.0

[sic] g/L,p<0.001)." They go on to state that "thirty-two cases were excluded from analyses due to weight loss or unavailable weight loss information. Organochlorine levels were generally higher in these cases compared to cases without weight loss." These findings support my concerns about the validity of results from studies of DDT and DDE levels in autopsy and other biologic specimens collected after NHL diagnosis. Both DDT and DDE are stored in body fat, and the data from the Spinelli, et al., study suggest that lipid levels differ between cases and controls and that in subjects with weight loss, results differ from those without weight loss. It must be remembered that weight loss is a relatively frequent presenting sign of NHL leading to diagnosis. Weight loss becomes more severe in terminal NHL patients. In summary, results of DDT and DDE assays of biologic specimens obtained at death or after diagnosis must be interpreted with extreme caution.

(6) Cocco P, Brennan P, Ibba S, et al. Plasma polychlorobiphenyl and organochlorine pesticide level and risk of major lymphoma subtypes, 2008. This is a case-control study of subjects from the "Epilymph study, a multicentre European case-control study of environmental exposures and lymphoid neoplasms." Cases and controls are from three countries: Spain, France, and Germany. The paper provides no further information on methods for case identification and selection or for the source of controls and what matching procedures were used. "Information on potentially confounding covariates was based on personal interviews using a structured questionnaire." Covariates used in the final regression model for analyses included age, gender, educational level, study center, and individual chemical exposure. NHL ORs were calculated using unconditional logistic regression. No further detail was provided. As a result, it is difficult to assess the methodology used in assembling subjects for this case-control study and for data collection. Additionally, analytic details are vaguely defined.

For p,p'-DDE, the OR for NHL in the highest level category ($\geq$1431.08 ppb) was 1.2 (0.7-2.4). The lowest level category ($\leq$394.99 ppb) was the reference category used. A test for trend across the categories was not statistically significant (p=0.48). The analyses also were performed separately for two subsets of NHL, diffuse large B-cell lymphoma (DLBCL) and for chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL). The OR for the highest level category was 1.3 (0.5-3.6) for DLBCL and 1.0 (0.4-2.5) for CLL/SLL. Trend tests for ORs and serum levels for both subtypes were not statistically significant, p=0.48 and 0.92 for the two subcategories respectively.

The paper attempted to deal with the possible problem of assaying serum levels after chemotherapy was initiated by performing separate analyses for the subset of Spanish patients who had blood samples drawn before treatment was begun. The results were comparable for the pre- and post-treatment cases, but no specific data were provided. Attempts were made to assess whether or not the PCB findings were confounded by p,p'-DDE levels. Although no specific data were provided in the tables or text, the authors note that PCB results did not change when controlling for DDE and "no significant interaction whatsoever was identified." No comment was made as to whether controlling for PCB levels affected DDE results.

In summary, this paper is seriously lacking in essential details for proper evaluation. There are few details regarding the study's design and methodology. For example, there is no information on who the controls were and how they were selected and/or matched. Nevertheless, no statistically significant associations (ORs of 1.0-1.3) were found between risk of NHL and its major subtypes and serum levels of p,p'-DDE.

(7) Hardell K, Carlberg M, Hardell L, et al. Concentrations of organohalogen compounds and titres of antibodies to Epstein-Barr virus antigens and the risk for non-

Hodgkin lymphoma, 2009. This study has features of both a case-control study and a cross-sectional study. The case-control study portion is based on a subset of subjects from an earlier study by the same research group (Eriksson, et al. 2008). One hundred cases and 100 controls were selected from the subset of subjects in the earlier study who had provided blood samples. The blood samples were obtained after cases were diagnosed with NHL, and thus, it is not clear whether the values obtained for cases were indicative of pre-diagnostic status or whether they were the result of cases' NHL. Data on exposure to DDT and other pesticides were obtained in the earlier, Eriksson, et al., study. Blood specimens were assayed for organohalogen compounds and EBV antigens. Both viral capsid antibody (VCA) and early antibody (EA) titers were assayed, but only data for EA titers were presented in the tables.

Overall, there was no significant difference between cases and controls with regard to blood concentrations of p,p'-DDE ($p = 0.11$). For follicular lymphoma, an OR of 1.2 (95% CI = 0.4-3.5) was found for p,p'-DDE. The median VCA antibody titer level was 1,280 in cases and 2,560 in controls ($p = 0.86$). The median EA antibody titer level was 80 in cases and 40 in controls ($p = 0.007$). Although there is only a one dilution difference in EA levels between cases and controls, there is a very significant difference between the two groups. This is likely due to at least one outlier EA value in the cases: the upper bound of the range for cases was 10,240 and only 640 in controls.

In Table IV, data are presented for both p,p'-DDE in terms of EA titers ≤ 40 and >40. For the cell > the median p,p'-DDE level and EA >40, an OR of 3.3 (1.4-7.7) was found. In Table V, similar analyses are presented for follicular NHL only ($n = 20$). For the cell > the median p,p'-DDE level and EA >40, an OR of 1.2 (0.3-4.3) was found.

It is not clear what this paper means and what its relevance to this case might be. The paper has several significant problems that limit its usefulness. First, it is based on a subset of an earlier study that found no significant association between DDT exposure and NHL risk. Second, the measurement of EBV antibody titers was based on samples obtained after cases were diagnosed. EA antibody levels are likely to be affected by the cases' NHL. EA antibody titers are typically elevated in early stages of infection by the EBV or in reactivation responses triggered by immunosuppressive events. VCA antibody titers, in contrast, occur later during EBV infection and persist for life in most persons. VCA titers are typically not elevated as part of a reactivation response as might be seen in a patient undergoing organ transplantation. Analyses of VCA antibody titers would have been more relevant for making inferences regarding possible causal roles, since these titers are less likely to be affected by development of NHL. So it is difficult to interpret the meaning of the EA findings. Third, this paper reports numerous subset analyses raising the likelihood that at least several of the findings are due to chance. Fourth, this is a small study, for example, data are available for only 20 follicular NHL cases, and there is excessive "slicing and dicing" of the data. Lastly, the relevance of these findings to Ms. Garza is unclear since we do not have any data on her EBV antibody levels nor on any blood pesticide levels. In summary, this study is small in size, difficult to interpret, and of unknown relevance with respect to Ms. Garza. Nevertheless, this study does not demonstrate an association between p,p'-DDE levels and risk of follicular NHL.

III. DETAILED ANALYSES OF COHORT STUDIES OF DDT AND NHL IN HUMANS

(1) Laws EH Jr., Curley A, Biros FJ. Men with intensive occupational exposure to DDT, 1967. This early paper reports the results of an evaluation of a group of men who were occupationally heavily exposed to DDT for more than five years. This U.S. government study

used subjects from a California manufacturing facility (Montrose Chemical Corporation of California) that solely produced DDT and had been in operation continuously from 1947. Ditraglia, et al. (1981) and Brown (1992), which are discussed below, also studied workers at this same facility. A total of 35 men with a mean of 15 years of exposure at the plant were assessed. The men gave medical histories, underwent physical examinations, had chest x-rays, and provided blood, fat biopsies and urine specimens. The heavy exposure of this group to DDT was reflected in mean DDT levels in fat specimens that were 39 to 128 times higher than levels in the general population. Their evaluations "did not reveal any ill effects attributable to exposure to DDT." They also reported that "[n]o cancer or blood dyscrasia was found in the 35 men studied."

This was a very small study of workers occupationally heavily exposed to DDT, and solely to DDT. Levels of DDT exposure were high, and this was reflected in levels in biologic specimens from the men. Neither adverse health effects nor NHL or any other cancers were found. Only about 0.1 NHL case would have been found if general population rates were applied to the number of person-years of observation of this cohort. Nevertheless, what the study does tell us is that very high DDT exposure levels do not cause demonstrable adverse health effects, even after a mean of 15 years of exposure. It also suggests that there were no excesses of cancer occurrence within this highly exposed cohort.

In summary, this is a very limited study in terms of sample size. What it does show is that there are likely no adverse heath effects of very high levels of DDT exposures in the workplace on average 15 years after exposures began.

(2) Ditraglia D, Brown DP, Namekata T, Iverson N. Mortality study of workers employed at organochlorine pesticide manufacturing plants, 1981. This paper reports the

results of a NIOSH retrospective cohort study of mortality at four organochlorine pesticide-manufacturing facilities in the U.S. This U.S. government study relied on personnel records provided by the four facilities. The study group totaled about 2,100 individuals. One of the four plants studied (Plant 4) was in California and solely produced DDT dating back to 1947. None of the other three plants produced DDT. Among Plant 4 workers, there were 6 observed deaths from all malignant neoplasms for an SMR of 68 (25-247) based on 7,601 person-years of observation. There were no deaths from "lymphatic and hematopoietic system" cancers, a category that includes NHL. Deaths from all causes at Plant 4 totaled 42 for an overall SMR of 86 (62-116).

This was a relatively small cohort study of workers at organochlorine pesticide manufacturing facilities. Specific data were available for one plant that produced only DDT dating back to 1947. No increased cancer mortality was observed among the DDT plant workers, and there were no deaths in the cancer category that includes NHL. Based on the number of person-years of follow-up, about one NHL death would have been expected, yet none was observed in this group of workers heavily exposed to DDT. The small numbers of subjects and limited numbers of deaths preclude further conclusions.

(3) Brown DP. Mortality of workers employed at organochlorine pesticide manufacturing plants – an update, 1992. This paper reports the results of continued follow-up of workers at four plants that produced organochlorine pesticides. The first study was done by Ditraglia, et al. (1981), and this government study continues the initial follow-up. One plant, Plant 4, was in California and produced only DDT. Workers were white men who had worked at the four plants for at least 6 months prior to December 31, 1964. Mortality in these workers was assessed through December 31, 1987. For all malignant neoplasms, an SMR of 0.87 (95% CI =

0.52-1.39) was determined for workers in Plant 4. This was similar to the SMR for all four plant workers combined (SMR = 0.88). No deaths from lymphatic/hematopoietic neoplasms were found for Plant 4 workers. Thus, there were also no deaths from NHL which is a component of this category. For workers at all four plants combined, an SMR of 0.98 was observed for this category (95% CI = 0.55-1.62, based on 15 deaths).

This study followed 328 workers at Plant 4, which only produced DDT, for a total of 9,797 person-years of follow-up. There were 90 deaths in this cohort and none from NHL or other hematologic neoplasms. While this cohort was small, it represents a group of industrially exposed workers who produced DDT only. If DDT were truly a cause of NHL, we would have expected to see at least two or more NHL deaths in these highly exposed individuals with very specific exposure to DDT. This study, done by NIOSH, was limited by a lack of detailed exposure information. However, for a small subset (N = 35) of workers at Plant 4, DDT was assayed in fat samples and found to range from 38 ppm to 647 ppm, compared with an average of 8 ppm for the general population. The observed worker levels were **4.75 to 80.9 times greater** than general population levels. It was estimated that the average daily intake of DDT in the 35 workers ranged from **438 to 450 times** that of the general population. Thus, workers at Plant 4 were **very heavily and specifically** exposed to DDT.

In conclusion, this study, while relatively small, provides highly specific information on subjects with well-defined, high exposure levels to DDT. No deaths from any of the category of lymphatic/hematopoietic neoplasms were observed for 9,797 person-years of follow-up. At least two NHL deaths would have been expected based on general population rates, and none was found. If risks were elevated in DDT-exposed workers, even more deaths would have been

expected. While this government-conducted study is of relatively small size, it demonstrates that workers with **very high** levels of DDT exposure did not show any increased NHL risk.

(4) Cocco P, Blair A, Congia P, et al. Proportional mortality of dichloro-diphenyl-trichloroethane (DDT) workers: A preliminary report, 1997(a). This is one of a series of three papers based on a cohort of workers exposed to DDT in Sardinia as part of a post-World War II malaria eradication program initiated by the Rockefeller Foundation. The exposures occurred from 1946-1950.

This paper reports the results of a proportional mortality study of 1,043 deaths that occurred during 1956-1992 in men who used mostly DDT in the anti-malaria campaign in Sardinia, Italy. They found PMRs of 81 for NHL (95% CI = 9-294) in exposed subjects and 53 (1-295) in unexposed subjects. Unfortunately, these PMRs are based on 1 death from NHL in the unexposed group and 2 deaths in the exposed group, which makes the results very unstable and of limited value.

Although this was a study of mostly DDT exposure (chlordane and lead arsenate also were used for a short time), it was relatively small and there were very few deaths from NHL. The major study limitation was the use of proportional mortality analyses which can produce biased results if there are disproportionate deaths from other causes. Nevertheless, no excess of NHL was observed among deaths in this cohort of heavily exposed workers. This paper reports the exact same results as those in another 1997 publication by the same authors, discussed below.

(5) Cocco P, Blair A, Congia P, et al. Long-term health effects of the occupational exposure to DDT, 1997(b). This paper reports the exact same results as the prior paper by Cocco, et al (1997a). This paper was published in a monograph and probably was not peer-reviewed. As reported above, results in this paper also are based on 1,043 deaths that occurred in

the cohort between 1956 and 1992. Analyses provide only proportional mortality ratios. In exposed decedents, there was a PMR of 81 (95% CI = 9-294), and in unexposed subjects, the PMR was 53 (1-295). However, these results are based on 2 NHL diagnoses in the exposed and 1 NHL diagnosis in the unexposed subjects.

This study is severely limited by the sparse number of cancer endpoints and by its use of proportional mortality statistics. The combination of these two problems renders the study of limited value for explicating the relationship between DDT and NHL mortality.

(6) Cocco P, Fadda D, Billai B, et al. Cancer mortality among men occupationally exposed to dichlorodiphenyltrichloroethane, 2005. This paper deals with an expansion of Cocco, et al.'s, earlier study of cancer mortality in a Sardinian cohort of men occupationally exposed to DDT as part of a malaria eradication program. They conducted a mortality follow-up study of 4,552 male workers exposed to DDT from 1946-1950. In an analysis that compared observed, cause-specific mortality with expected regional mortality rates, they observed an SMR of 101 (95% CI = 65-157) for "lymphatic cancer" in applicators (who were directly exposed). This is based on 20 deaths from lymphatic cancer in the exposed group. The SMRs for lymphatic cancer were 83 (44-153) for bystanders (those indirectly exposed) and 174 (112-271) for unexposed men. Thus, there was a statistically significant increase in lymphatic cancer mortality for **unexposed men**, while no increased mortality was found for applicators.

In another analysis in which comparisons were made between the unexposed and exposed groups, the results were similar. For applicators the relative risk was 0.7 for lymphatic cancer and for bystanders 0.6. However, in this analysis, the referent group was the unexposed group that has a statistically significantly elevated SMR for lymphatic cancers. Nevertheless, in this

enlarged study, no excess risk (mortality) of lymphomas (not further specified) was found in this well documented DDT exposure cohort.

This study is useful since it deals with a large cohort of DDT exposed men with good exposure data available for them. There was also a long follow-up period (from 1956-1999) to allow ample time for any increased cancer risk to have become manifest, and no excess lymphoma mortality was found. Unfortunately, no breakdown of NHL and Hodgkin lymphoma deaths was provided. Most of the deaths from lymphomas would be expected to be NHL, based on the relative incidence and mortality of the two groups of diseases. It is clear from the paper that leukemias were not included in this category. The reporting of data for an undefined category of lymphomas ultimately limits the usefulness of this paper.

(7) Purdue MP, Hoppin JA, Blair A, Dosemici M, Alavanja MCR. Occupational exposure to organochlorine insecticides and cancer incidence in the Agricultural Health Study, 2006. This is the most recent cohort study evaluating the incidence of cancers following occupational exposures to DDT and other organochlorine insecticides. It provides results from a large prospective cohort study of licensed pesticide applicators in Iowa and North Carolina (N = 57,311). Workers were recruited between 1993 and 1997 and were followed through 2002. The mean follow-up time was 7.3 years. Exposure assessment was via self-administered questionnaires and incident cancer diagnoses were obtained from State tumor registries. A total of 12,035 applicators were exposed to DDT. A relative risk of 0.9 (95% CI = 0.6-1.5) was found for NHL and DDT exposure. This was based on 37 NHL cases in the exposed and 63 cases in the unexposed groups. This is a large study that had relatively large numbers of NHL cases in the study groups and this is reflected in the narrow confidence interval found.

The study has a potential problem that could have biased the results in that exposure data only were available for 44% of the enrolled study subjects (i.e., those subjects who completed the take-home questionnaire). However, this potential for bias might have been mitigated in several ways. First, in an earlier analysis, the authors found that subjects completing the take-home questionnaire were older on average than non-respondents, but were otherwise comparable. Since DDT use was stopped in 1972, older subjects would be more likely to have worked with DDT. This would add to the study's statistical power by increasing the number of exposed subjects. Second, it should be recognized that a total of 12,035 subjects reported exposure to DDT before the onset of any cancer. Such a large number of exposed workers are less likely to represent a very skewed or biased subset of the study subjects.

Thus, despite such potential limitations, the study also has considerable strengths such as the large number of exposed subjects, the use of incidence data, the collection of exposure data in advance of the outcomes and the large numbers of workers who developed NHL. On balance, this study provides persuasive evidence that DDT is not a cause of NHL.

IV. DETAILED ANALYSES OF ECOLOGIC STUDIES

(1) Cocco P, Kazerouni N, Zahm SH. Cancer mortality and environmental exposure to DDE in the United States, 2000. This is an ecologic study that examined the association between (1) 1968 adipose DDE levels of population samples from 22 U.S. states and (2) age-adjusted mortality rates for these states during 1975 and 1994 for (3) several cancers including NHL. Population samples are analyzed in relation to state mortality rates with no individual DDE levels in cases or controls available. They found a statistically significant **inverse correlation** between DDE levels and NHL mortality in three of the four study groups (white men, white women and African American women). Higher tissue DDE levels were protective against NHL for three of the four population groups studied. In a multivariate

analysis, NHL mortality significantly decreased with increasing DDE levels in whites, but not in African Americans. Thus, they found that for most subjects, higher tissue levels of DDE were associated with lower NHL mortality, suggesting a protective effect.

This study is of limited value since it is an ecologic study and merely correlated area-based DDE tissue levels with cancer mortality in the same areas. As in all such ecologic studies, exposure data are not available for the individual cancer decedents. Nevertheless, the study suggests that high adipose DDE levels **protect** against NHL.

(2) Pavuk M, Cerhan JR, Lynch CF, Schecter A, Petrik J, Chovancova J, Kocan A. Environmental exposure to PCBs and cancer incidence in Slovakia, 2004. This is an ecologic study conducted in two districts in eastern Slovakia. One of the two districts chosen had "extensive environmental contamination from a former PCB production site (Michalovce) and the other matched on geography but with low (background) levels (Svidnik)." The paper was focused primarily on PCB measurements but also measured population serum levels of DDE, DDT and HCB (hexachlorobenzene). A sample of 115 males and 110 females from Michalovce and 102 males and 105 females from Svidnik provided blood samples for the chemical analyses. A large number of PCB congeners were assayed as well as DDE, DDT and HCB in the blood specimens obtained. Statistically significant differences were found in the geometric mean levels of DDT and DDE, but not HCB, between the two sampled geographic areas. However, there was considerable overlap in the range of DDE and DDT levels between the two areas.

The authors also examined NHL standardized incidence ratios (SIRs) for NHL occurrence in the two areas. The NHL SIR for males for the high exposure area, Michalovce district, was 1.12 (0.80-1.52), and for the low exposure area, Svidnik, 1.11 (0.62-1.84). The

corresponding SIRs for females were 1.04 (0.70-1.49) and 0.56 (0.22-1.28) in the two areas respectively. Thus, there was no excess in NHL incidence in the high exposure zone with the SIRs ranging from 1.04 to 1.12. Results for the low exposure area ranged from 0.56 to 1.11. The SIR of 0.56 was based on only 6 observed cases and was not statistically significant. It should be pointed out that the proper comparisons to be made are the observed numbers of cases with the expected number of cases (based on population incidence statistics for eastern Slovakia as a whole) rather than the ratio of SIRs from the two areas. Thus, the data as presented show no excess of NHL incidence in the high exposure region and similar incidence ratios in the low exposure region.

Unfortunately, this study is of limited value due to its being an ecologic study. The investigators had no information on specific chemical levels in cases and non-cases; rather they determined mean chemical levels for a very small population sample in the two regions studied. These mean levels were then related to area incidence of NHL. With regard to DDT and DDE levels, no effort was made to tease apart the possibly confounding effects of other chemical exposures. Such a study can be used for hypothesis generation at best. In summary, this study is of very limited scientific value owing to its design. Nevertheless, no increase in NHL incidence was found in the high exposure area.

October, 2009

Curriculum Vitae

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Date and Place of Birth:	November 29, 1937; New York, NY
Citizenship:	United States
Marital Status:	Married- Sue Y.S. Kimm, M.D.

Education:

B.S. - City College of New York, 1960.
M.D. - State University of New York (Syracuse), 1964.
M.P.H. - Harvard School of Public Health, 1968.
S.M. - Harvard School of Public Health, 1974.
Dr.P.H. - Harvard School of Public Health, 1979.

Medical Training:

Rotating Internship: University of Illinois Research and Educational Hospitals, Chicago, IL, 1964-65.

Pediatric Residency: New York Hospital - Cornell Medical Center, New York, NY. 1965-67.

Professional Experience:

Lecturer, Department of Biology, City College of New York, New York, NY, 1960.

Lecturer (part-time), Division of Allied Health Sciences Program in Health Care Administration, Northeastern University, Boston, MA, 1967-68.

Major, Medical Corps, U.S. Air Force; Chief of Pediatrics and Public Health, and Fifth Air Force Consultant in Pediatrics, U.S. Air Force Hospital, Tachikawa, Japan, 1968-1971.

Assistant Professor and Chairman, Department of Public Health, Gondar Public Health College, Haile Selassie I University, Gondar, Ethiopia, 1971-73.

Full-time student, candidate for Dr. P.H. degree in epidemiology, Harvard School of Public Health, Boston, MA, 1973-76. Epidemiologist, Management Sciences for Health, Cambridge, MA, 1973-74.

Teaching Fellow, Department of Epidemiology, Harvard School of Public Health, Boston, MA, 1975-76.

Volunteer Physician, The Medical Van, Bridge Over Troubled Waters (Archdiocese of Boston & Massachusetts General Hospital), Boston, MA, 1975-76.

Assistant Professor, Department of Pediatrics, Duke University Medical Center, Durham, NC, 1976-81.

Director, Epidemiology and Biostatistics Unit, Duke University Comprehensive Cancer Center, Durham, NC, 1976-87.

Attending Pediatrician, Duke University Hospital, Durham, NC, 1976-87.

Director, Epidemiology Study Program, Duke University School of Medicine, Durham, NC, 1978-87.

Associate Professor (with tenure), Department of Pediatrics, Duke University Medical Center, Durham, NC, 1981-87.

Director, Cancer Prevention and Control Program, Duke University Comprehensive Cancer Center, Durham, NC, 1981-87.

Adjunct Associate Professor, Department of Epidemiology, School of Public Health, and Member, Graduate Faculty, University of North Carolina at Chapel Hill, Chapel Hill, NC, 1982-87.

Assistant Professor (Secondary Appointment), Department of Medicine, Duke University Medical Center, Durham, NC, 1984-87.

Chief, Clinical Epidemiology Division, Department of Pediatrics, Duke University Medical Center, Durham, NC, 1984-87.

Professor, Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA, 1987-88.

Associate Director for Epidemiology and Preventive Oncology, Pittsburgh Cancer Institute, Pittsburgh, PA, 1987-89.

Chairman, Department of Family Medicine and Clinical Epidemiology, University of Pittsburgh School of Medicine, Pittsburgh, PA, 1988-98.

Professor, Department of Family Medicine and Clinical Epidemiology, University of Pittsburgh School of Medicine, Pittsburgh, PA, 1998-2004.

Director, Family Medicine Division, Children's Hospital of Pittsburgh, Pittsburgh, PA, 1993-98.

President, University Family Practice Associates, Pittsburgh, PA, 1994-98.

Senior Research Associate in Epidemiology and Biostatistics, European Institute of Oncology, Milan, Italy, 1994-2000.

Active Staff, Family Medicine Division, Children's Hospital of Pittsburgh, Pittsburgh, PA, 1994-2003.

Chief of Family Medicine Service, University of Pittsburgh Medical Center, Pittsburgh, PA, 1995-98.

Active Staff, University of Pittsburgh Medical Center, Pittsburgh, PA, 1995-2003.

President, University Physicians Practice Association, University of Pittsburgh Medical Center, Pittsburgh, PA, 1995-97.

Research Professor, Department of Pathology, School of Medicine, University of New Mexico Health Sciences Center, Albuquerque, New Mexico, 2004-2005.

Research Professor, Department of Internal Medicine, Division of Epidemiology and Biostatistics, School of Medicine, University of New Mexico Health Sciences Center, Albuquerque, New Mexico, 2005-2007.

Full Member, Cancer Research and Treatment Center, University of New Mexico Health Sciences Center, Albuquerque, New Mexico, 2004-.

Voluntary Adjunct Professor, Department of Internal Medicine, Division of Epidemiology and Biostatistics, School of Medicine, University of New Mexico Health Sciences Center, Albuquerque, New Mexico, 2007-.

Committee Memberships:

University:

Harvard School of Public Health, Admissions Committee, 1974-76.

Harvard School of Public Health, Search Committee for Director of International Health Program, 1975.

Duke University School of Medicine, Third-Year Curriculum Committee, 1976-78.

Duke University Medical Center, Medical Records Committee, 1977-87.

Duke University Medical Center, Clinical Cancer Education Program Committee, 1978-87.

Duke Comprehensive Cancer Center, Executive Committee, 1981-87.

Pittsburgh Cancer Institute, Executive Committee, 1987-89.

University of Pittsburgh School of Medicine, Executive Committee, 1988-98.

University of Pittsburgh School of Medicine, Council of Clinical Chairmen, 1988-98.

University of Pittsburgh Medical Center, Executive Planning Council, 1988-95.

University of Pittsburgh, Annual Family Practice Refresher Course, Planning Committee, 1988-98.

University Health Network Inc., Board of Directors, 1988-96; Member, Executive Committee, 1994-96.

University of Pittsburgh, Graduate School of Public Health, Occupational Medicine Residency Advisory Committee, 1989-98.

University of Pittsburgh School of Medicine, Chairman, Curriculum Revision Task Force on Ambulatory and Community Health, 1990-91.

Baby Your Baby Internship Program, Committee Member, 1990-95.

University of Pittsburgh School of Medicine, Applicant Interview Committee, 1992-93.

University of Pittsburgh School of Medicine, Chairman, Committee on Secondary Faculty Appointments, 1992-98.

University of Pittsburgh, Fibromyalgia Research Committee, 1994-95.

University of Pittsburgh Medical Center, Managed Care Committee, 1994-95.

University of Pittsburgh Medical Center, Primary Care Focus Group, 1994-96.

University of Pittsburgh Medical Center, Advisory Committee for the Center for Health Services Research, 1994-98.

Children's Health Network, Member, Manpower Committee, 1995-96.

University of Pittsburgh Medical Center, Member, Network Partnership Committee, 1995-96.

University of Pittsburgh Medical Center, Co-Chair, Committee on Satellite Clinics, 1995-96.

University of Pittsburgh Medical Center, Member, Physician Hospital Organization Planning Committee, 1995-96.

University of Pittsburgh and St. Margaret Memorial Hospital, Member, Affiliation Oversight Committee, 1995-96.

Tri-State Health System, Member, Board of Directors, 1995-97.

University of Pittsburgh Medical Center Keystone/Security Blue HMO Committee, 1995-96.

University of Pittsburgh Center for Biomedical Informatics, Advisory Committee, 1996-98.

University of Pittsburgh Medical Center Primary Care Task Force, 1996-97.

University of Pittsburgh School of Medicine, Planning and Budgeting Committee, 2000-2006.

Regional and National:

Intergroup Rhabdomyosarcoma Study Committee, 1978-95.

State of North Carolina Secretary of Human Resources' Commission on Cancer, 1979-81.

Pediatric Oncology Group, Co-chairman, Epidemiology Committee, 1983-91.

North Carolina Division, American Cancer Society, Public Education Committee, 1984-87.

National Council on Radiation Protection and Measurements, Scientific Committee 81 (Assessment of Exposures from Radiation Therapy), 1985-87.

Children's Oncology Group, Chairman, Hodgkin's Disease Epidemiology (CCG E13) and Rhabdomyosarcoma Epidemiology Committees, 1988-

SmithKline Beecham, Inc., Member, Urology Advisory Panel, 1995.

Institute of Medicine, National Academy of Sciences, Member, Committee to Review the Health Effects in Vietnam Veterans of Exposure to Herbicides, Washington, DC, 1995-96.

University of Helsinki, Faculty of Medicine, Member, Search Committee for the Professor of Public Health, 1999-2000.

Peer Review Experience:

Ad Hoc Member, Epidemiology and Disease Control Study Section, Bethesda, National Institutes of Health, 1978.

Member, Cancer Center Support Review Committee, Bethesda, National Cancer Institute, 1979-83.

Ad Hoc Grant Reviewer, American Cancer Society, 1981, March of Dimes Birth Defects Foundation, 1981, and Saudi Arabian National Center for Science and Technology, 1981.

Ad Hoc Conference Grant Reviewer, National Cancer Institute, 1981-88.

Member, Special Study Section on the Acquired Immunodeficiency Syndrome, Bethesda,

National Institutes of Health, 1982-83.

Chairman, Ad Hoc Contract Review Committee on "The Natural History of Acquired Immunodeficiency Syndrome in Homosexual Men," (The Multicenter AIDS Cohort Study) Bethesda, National Institute of Allergy and Infectious Diseases, 1983.

Ad Hoc Member, Cancer Center Support Review Committee, Bethesda, National Cancer Institute, 1985.

Member, Special Study Section to review a grant for "Screening for Neuroblastoma in Infants", Montreal, National Cancer Institute, 1987.

Chairman, Epidemiology and Technology Transfer Subcommittee, Acquired Immuno-Deficiency Syndrome Research Review Committee (AIDSRRC), Bethesda, National Institute of Allergy and Infectious Diseases, 1987-91.

Member, Scientific Advisory Committee on Clinical Investigations III - Prevention, Diagnosis and Therapy Review Committee, Atlanta, GA, American Cancer Society, 1987-91.

Member, Special Study Section-1, for review of a grant application for "Screening for Neuroblastoma in Infants". Bethesda, National Cancer Institute, 1988.

Ad Hoc Grant Reviewer, March of Dimes Birth Defects Foundation, 1989.

Chairman, Special Study Section on Biostatistics (SSS-1) (RI), Washington, National Institutes of Health, 1991.

Member, National Institutes of Health Reviewers Reserve, Bethesda, MD, 1991-95.

Member, Grant Review Committee, Aidan O'Neil Foundation for the Prevention of Childhood Cancer, Nebraska, 1993.

Member, Special Study Section to review a program project, "Cancer Prevention Research Program," Fred Hutchinson Cancer Center, National Cancer Institute, December, 1993.

Ad Hoc Member, Cancer Centers and Research Programs Grant Review Committee (Subcommittee D), National Cancer Institute, Bethesda, MD, 1994.

Ad Hoc Member, NCI Grant Review Subcommittee E, Cancer Epidemiology. Prevention and Control, Bethesda, MD, 1997.

Member, Cancer Center Support Grant Site Visit Team, Fred Hutchinson Cancer Research

Center, Seattle, WA, 1998, NCI.

Ad Hoc Member, NCI Grant Review Subcommittee E, Cancer Epidemiology, Prevention and Control, Bethesda, MD, 1998.

Member, Cancer Manpower and Training Grant Review Subcommittee (NCI-F), National Cancer Institute, Bethesda, MD, 1997-2001, 2001-2002.

Ad Hoc Member, NCI Grant Review Subcommittee E, Cancer Epidemiology, Prevention and Control, Bethesda, MD, 2001.

Member, Special Emphasis Panel and Scientific Grant Review Groups, NCI Loan Repayment Program, Washington, DC, 2003, 2004, 2005, 2006, 2007, 2008, 2009.

Ad Hoc Member, NCI Grant Review Subcommittee F, Cancer Education, Washington, DC, 2003.

Member, NCI Special Emphasis Panel, Long-Term Cancer Survivors: Research Initiative, Washington, DC, 2003.

Charter Member, NIH Centers for Scientific Review, Cancer Biomarkers Study Section, Washington, DC, 2003-4. Ad Hoc Member, 2004 –.

Mail Reviewer, Center for Scientific Review, Special Emphasis Panel on Chronic Fatigue Syndrome, Fibromyalgia Syndrome, and Temperomandibular Dysfunction. Washington, DC, 2006.

Member, Site Visit Team to review the Cancer Center Support Grant of the Fred Hutchison Cancer Research Center, Seattle, WA, 2008, NCI.

Member, Site Visit Team to review a Cancer Center Support Grant application at Emory University, Atlanta, Georgia, 2008, NCI.

Member, Site Visit Team to review a Cancer Center Support Grant application at the University of Texas, Southwestern Medical Center, Dallas, TX, 2009, NCI.

Membership on Special Review Committees (SRC):

SRC to review a program project on "Diet, Lifestyle and Cancer," Baltimore, NCI, 1982.

SRC on Acquired Immunodeficiency Syndrome, Bethesda, NIDA, 1983.

SRC to review the Center for Ulcer Research and Education, Los Angeles, NIADDKD, 1983.

SRC on Cancer Control Science Programs, Houston and Bethesda, NCI, 1984

SRC to review a program project on "Social Epidemiology of Cancer," Buffalo, NCI, 1984.

SRC to review a Cancer Center Support Grant, San Juan, NCI, 1984.

SRC to review master agreements for clinical research on the acquired immunodeficiency syndrome, Bethesda, MD, NIAID, 1985.

SRC to review the Immunodeficiency-Cancer Registry, Minneapolis, MN, NCI, 1985.

SRC for program review of studies on the natural history of HTLV- III infection, Bethesda, MD, NIAID and NCI, 1986.

SRC on Acquired Immunodeficiency Syndrome, NIDA, Rockville, MD, 1986.

SRC to review Outstanding Investigator Awards, NCI, Bethesda, MD, 1988.

SRC to review a Consortium Cancer Center Support Grant, Northern California Cancer Program, NCI, Belmont, CA, 1988.

Chair, SRC to review the Drew-Meharry-Morehouse Consortium Cancer Center Support Grant, NCI, Bethesda, MD, 1988.

SRC to review proposals for "Community Programs for Clinical Research on AIDS", NIAID, Rockville, MD, 1989.

SRC to review a Cancer Center Support Grant, University of Southern California Comprehensive Cancer Center, NCI, Los Angeles, CA, 1990.

Chair, SRC to review the Drew-Meharry-Morehouse Consortium Cancer Center Support Grant, NCI, Bethesda, MD, 1990.

SRC to review a program project on "Human Papilloma Virus: Biology Clinical Significance and Epidemiology", Fred Hutchinson Cancer Center, NCI, Seattle, WA, 1991.

Chair, SRC to review a program project on "Prevention of Primary Hepatocellular Carcinoma", Fox Chase Cancer Center, Philadelphia, PA, 1991, NCI.

Special Emphasis Panel to review a program project on "Human Papilloma Virus: Biology, Clinical Significance and Epidemiology", Fred Hutchinson Cancer Center, Reverse Site

Visit, NCI, Chevy Chase, MD, 1992.

Chair, SRC to review a program project on "Molecular Epidemiology of Primary Hepatocellular Carcinoma" Fox Chase Cancer Center, Reverse Site Visit, NCI, Bethesda, MD, 1992.

Grant Review Team to review a Program Project, "Molecular Biomarkers for Environmental Carcinogenesis", NIEHS, Research Triangle Park, NC, 1992.

SRC to review NIAID Institutional Training Awards for Clinical Research on the Acquired Immunodeficiency Syndrome, NIAID, Chevy Chase, MD, 1992.

Special Emphasis Panel to review a program project supplement on "HPV: Biology, Clinical Significance and Epidemiology", NIH telephone review, 1993.

SRC to review a program project on "Studies of Monoclonal Gammopathies", Mayo Clinic, Rochester, MN, 1993, NCI.

SRC to review a program project on "Studies of Monoclonal Gammopathies", Mayo Clinic, Rochester, MN; reverse site visit, NCI, Chevy Chase, MD, 1994.

Chair, SRC to review a Cancer Center grant application for the "Drew-Meharry-Morehouse Consortium Cancer Center", NCI, Bethesda, MD, 1994.

SRC to review a program project on "Evaluation of Carcinogenic Risks to Humans," NCI telephone review, 1995.

SRC to review grant applications for "Collaborative Cancer Prevention Research Units", NCI, Rockville, MD, 1995.

SRC to review the Small Grants Program for Cancer Epidemiology (PAR-95-077), tele-conference review, 1996, NCI.

Peer Review of Progress of the Drew-Meharry-Morehouse Consortium Cancer Center, teleconference review, 1997, NCI.

Special Emphasis Panel to review a program project on "Studies of Monoclonal Gammopathies", Mayo Clinic, Rochester, MN, 1997, NCI.

Chair, Special Emphasis Panel to review a program project on "Molecular Epidemiology of Hepatocellular Carcinoma", Fox Chase Cancer Center, Philadelphia, PA, 1997, NCI.

Special Emphasis Panel to review "Cohort and Nested Case-Control Study of AIDS-Related

Non-Hodgkin's Lymphoma, Kaposi's Sarcoma and Other Malignancies-Phase III", Rockville, MD, 1998, NCI.

Member, Special Emphasis Panel to review a program project on "Head and Neck Cancer Rehabilitation", Northwestern University, Evanston, IL, 2001, NCI.

Member, Special Emphasis Panel to review a program project on "Pre-Diagnosis Serologic Biomarkers of NHL Risk", Harvard University, Boston, MA, 2001, NCI.

Member, Special Emphasis Panel/Scientific Review Group, ZCA1-GRB-S (J1)R, Molecular Oncology – Basic, Translational and Clinical Studies, Rockville, MD, 20009, NCI.

Editorial Appointments:

Guest Editor, Reviews of Infectious Diseases, 1991.
Guest Editor, Blood, 1995.
Consulting Editor, Annals of Medical Science, 1993-2000.
Member, Editorial Board, Journal of Epidemiology and Biostatistics, 1995-2001.

Reviewer for Journals:

Cancer Epidemiology, Biomarkers and Prevention, JAMA, Clinical Cancer Research, International Journal of Cancer, Journal of Clinical Epidemiology (Journal of Chronic Diseases), American Journal of Epidemiology, Journal of the National Cancer Institute, Cancer, Lancet, New England Journal of Medicine, Cancer Causes and Control, Medical and Pediatric Oncology, Journal of Pediatric Hematology/Oncology, Epidemiology, Environmental Health Perspectives, Annals of Epidemiology, Journal of Occupational Medicine, Preventive Medicine, Cancer Research, Kidney International, American Journal of Obstetrics and Gynecology, Epidemiologic Reviews, British Journal of Cancer, Family Practice Recertification, European Journal of Cancer, Clinical Infectious Diseases and National Medical Journal of India.

Consultant Appointments:

Participant in Workshop/Conferences on Alzheimer's Disease, Senile Dementia and Related Disorders, Bethesda, MD (NINCDS, NIA, NIMH), 1977; Functional Gastrointestinal Disorders, Bethesda, MD (NIAMDD, NIMH), 1979; Epidemiology and Biostatistics in Clinical Cancer Education, Bethesda, MD (NCI), 1979; Inflammatory Bowel Disease, Princeton, NJ (NIADDKD), 1982; Neoplasia in Rheumatoid Arthritis, St. Simons Island, GA (Arthritis Foundation), 1984; Epidemiology of Multiple Myeloma, Bethesda, MD

(NCI), 1990; Cambridge Symposium on Myalgic Encephalomyelitis, Cambridge, U.K. (Nightingale Foundation), 1990; 1990 Research Conference, Chronic Fatigue Immune Dysfunction Syndrome Society, Charlotte, SC (CFIDS) NC, 1990; AIDS Epidemiology Investigators Meeting (NCI), Bethesda, MD, 1990; Workshop on Chronic Fatigue Syndrome (NIAID & NIMH), Bethesda, MD, 1990.

Research Triangle Institute, Research Triangle Park, NC, 1977-78.

International Agency for Research on Cancer (Organized and taught courses in cancer epidemiology in Zambia, Poland, Pakistan, Thailand, German Democratic Republic and U.S.S.R.), 1981-89.

World Health Organization Tropical Disease Research Programme, Geneva, 1981.

Rockefeller Foundation, New York, NY, 1982.

Panel Member, National Institutes of Health, Technology Assessment Meeting on the Feasibility and Effectiveness of a National Bone Marrow Donor Registry, Bethesda, MD, 1985.

Plough, Inc., Memphis, TN, 1985-93.

Member, National Bone Marrow Registry Evaluation Panel, National Institutes of Health, Bethesda, MD, 1988.

Member, Scientific Advisory Committee, Norris Cotton Cancer Center, Dartmouth University, Hanover, NH, 1988.

Chair, Working Group on Cancers of Childhood; The Use of Biomarkers in Field Studies of Environmentally Associated Cancers, Agency for Toxic Substances and Disease Registry, Atlanta, GA, 1994.

Member, Scientific Advisory Group on Benzene, European Institute of Oncology, Milan, Italy, 1995.

Agency for Toxic Substances and Disease Registry (ATSDR), Chair, Panel on General Methodological Challenges, Workshop on "Studying Environmental Exposures Among Children with Cancer: Current Technologies, Methodological Challenges, and Community Concerns", Atlanta, GA, 2000.

Agency for Toxic Substances and Disease Registry (ATSDR), Member, Tremolite Asbestos Registry Expert Working Group, Atlanta, GA, 2002.

Certification:

National Board of Medical Examiners (1965)
American Board of Pediatrics (1975)
American College of Epidemiology (1981)
Medical Licensure: New York (1965) (Inactive)
North Carolina (1976)
Pennsylvania (1989) (Active Retired)

Awards and Honors:

A.G. Levy Award (Chemistry) - C.C.N.Y.
J.L. Heffron Award (Medicine - Surgery) - S.U.N.Y.
N.I.H. Individual Research Fellowship - Harvard School of Public Health.
Student Prize Paper Award, Society for Epidemiologic Research, 1979.
Who's Who in the South and Southwest, 1989-90.
Who's Who in Science and Engineering, 2nd Ed., 1994-95.
Who's Who in the East, 22nd Ed., 1992; 24th Ed., 1994; 25th Ed., 1995.
Who's Who in the World, 12th Ed., 1995.
Elected Fellow, American Association for the Advancement of Science, 1995.
Who's Who in Medicine and Healthcare, 4th Ed., 2002.
Who's Who in American Education, 6th Ed., 2004-2005.
Who's Who in Science and Engineering, 9th Ed., 2006-2007, 10th Ed. 2008-2009.
Who's Who in American Education, 8th Ed., 2007-2008.
Who's Who in America, 2009.

Professional Memberships:

American Association for the Advancement of Science (Fellow)
American College of Epidemiology (Fellow)
Children's Oncology Group

Publications:

(Asterisks denote student and fellow co-authors)

Kimm SYS, Grufferman S. Teaching rural health at Haile Selassie I University, Gondar Public Health College. Harvard Public Health Alumni Bulletin 1974;31:4-6.

Grufferman S, Duong T, Cole P. Occupation and Hodgkin's disease. J Natl Cancer Inst

1976;57:1193-5.

Grufferman S, Cole P, Smith PG, Lukes RJ. Hodgkin's disease in siblings. N Engl J Med 1977;296:248-50.

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Grufferman S, Alzheimer's disease and senile dementia: one disease or two? In: Katzman R, Terry RD, Bick KL, eds. Alzheimer's Disease: senile dementia and related disorders, Aging, Vol. 7. New York: Raven Press, 1978: 35-41.

Grufferman S, Kimm SYS. An epidemiologic approach to planning: a case study from Ethiopia. In: Maddox GL, ed. Assessment and Evaluation Strategies in Aging: People, Populations, and Programs. Durham, NC: Duke University Center for the Study of Aging and Human Development 1978:77-94.

Hainline SW*, Myers LE, McLelland R, Newell J, Grufferman S, Shingleton WW. Mammographic patterns and risk of breast cancer. Am J Roentgenol 1978; 130:1157-8.

Grufferman S. Is Hodgkin's disease transmissible: An epidemiologic appraisal. [Doctoral Dissertation]. Boston, MA: Harvard University, 1979.

Grufferman S, Cole P, Levitan TR. Evidence against transmission of Hodgkin's disease in high schools. N Engl J Med 1979;300:1006-11.

Hennekens C, Lown B, Rosner B, Grufferman S, Dalen J. Ventricular premature beats and coronary risk factors. Am J Epidemiol 1980;112:93-9.

Grufferman S, Gillman ME*, Pasternak LR*, Peterson CL*, Young WG Jr. Familial carotid body tumors: case report and epidemiologic review. Cancer 1980;46: 216-22.

Wang HH, Grufferman S. Aplastic anemia and occupational pesticide exposure: A case-control study. J Occup Med 1981;23:364-6.

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Invited Talks:

Grufferman S, Taylor P. Diphtheria in a dependent child: Case report and survey of epidemiologic control methods. Third Annual Meeting, Far East Chapter, Association of Military Surgeons, Tachikawa, Japan, November 1969.

Grufferman S, Jobin R. Epidemiologic analysis of hospital admissions in Gondar, Ethiopia. Eighth Annual Meeting of the Ethiopian Medical Association, Addis Ababa, Ethiopia, May, 1972.

Grufferman S. Clustering studies in Hodgkin's disease and leukemia. National Cancer Institute Symposium on "Cancer Epidemiology and the Clinician," Boston, MA October, 1975.

Grufferman S, Cole P. Aggregation of etiologic exposures in Hodgkin's disease. 49th Annual Meeting, American Epidemiological Society, Baltimore, MD, April, 1977.

Grufferman S. Teaching cancer epidemiology at medical schools. Eleventh Annual Meeting, American Association for Cancer Education, San Francisco, CA, December, 1977.

Grufferman S. The epidemiology of carotid body tumors. Third Conference of Epi-Stat Staff of Cancer Centers, Washington, DC, March, 1978.

Grufferman S. Teaching epidemiology in medical schools. Third Conference of Epi-Stat Staff of Cancer Centers, Washington, DC, March, 1978.

Grufferman S. Epidemiology of Hodgkin's disease. St. Jude Children's Research Hospital, Memphis TN, March, 1979.

Grufferman S, Kimm SYS, Maile MC. A multilevel approach to teaching quantitative methods in the medical school setting. Meeting of the Association of Teachers of Preventive Medicine, Washington, DC, November, 1981.

Grufferman S. Preventive oncology at Duke University Medical Center. Association of Teachers of Preventive Medicine, Bethesda, MD, April, 1982.

Grufferman S. Epidemiologic approaches to the study of childhood cancers. M.D. Anderson Hospital and Tumor Institute, Houston, TX, April, 1982.

Grufferman S. In quest of Proteus: The search for new clues to the etiology of cancer. University of Colorado School of Medicine, Denver, CO, February, 1983.

Grufferman S. The epidemiology of Hodgkin's disease. University of Alabama School of Public Health, Birmingham, Alabama, GA, February, 1984.

Grufferman S. The epidemiology of Hodgkin's disease. Symposium on Hodgkin's Disease, Leukemia Society of America, Tampa, FL, February, 1984.

Grufferman S. The epidemiology of Hodgkin's disease. Oncology Seminar Series, Johns Hopkins Oncology Center, Baltimore, MD, January, 1985.

Grufferman S. Burkitt's and other non-Hodgkin's lymphomas in adults exposed to a visitor from Africa. Columbia University College of Physicians and Surgeons, New York, NY, February, 1985.

Grufferman S. The epidemiology of Hodgkin's disease. Epidemiology Colloquium, Columbia University School of Public Health, New York, NY, February, 1985.

Grufferman S. The investigation of cancer clusters. Mount Sinai School of Medicine, New York, NY, October, 1986.

Grufferman S. Cancer risk in the chronic fatigue-immune dysfunction syndrome. Pennsylvania Oncologic Society, Harrisburg, PA, November, 1987.

Grufferman S. Access to data from government-sponsored medical research. (Part of Panel Session on "Access to Government Data"), 16th Annual Meeting, American Public Health Association, Boston, MA, November, 1988.

Grufferman S. Discussant. Genetic predisposition to childhood rhabdomyosarcoma. International Symposium on Childhood Rhabdomyosarcoma, Columbus, OH, November, 1989.

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Grufferman S, Ambinder RF. A protective effect of breast-feeding on risk of Hodgkin's disease in childhood. Northeast Regional Meeting of the Society of Teachers of Family Medicine, Pittsburgh, PA, October, 1997. (awarded Best Research Paper prize)

Grufferman S. Limitations of studies of childhood cancer in assessing dietary risk factors. Ceres Forum on Nitrite as a Food Additive: State of the Science, sponsored by Georgetown University's Center for Food and Nutrition Policy, Washington, DC, December, 1997.

Grufferman S. Introduction and insights from childhood Hodgkin's disease. Workshop on Hodgkin's Disease Epidemiology and Directions for Future Research. Leukemia Research Fund Centre for Clinical Epidemiology, Leeds, U.K., January, 1999.

Grufferman S. Nutritional epidemiology. Meat Institute Research Conference, Chicago, IL October, 1999.

Gufferman, S. The hematopoietic cancers: Principles and Practice of Cancer Prevention and Control Course. National Cancer Institute, Rockville, MD, July, 2000.

Grufferman S. The epidemiology of Hodgkin's disease in childhood. Division of Pediatric Hematology/Oncology. Children's Hospital, Pittsburgh, PA, November, 2000.

Grufferman S. Limitations of epidemiologic approaches. CERES Forum on Food Safety Objectives, Georgetown University, Washington, DC, December, 2000.

Grufferman S. The enigma of Hodgkin's disease etiology. Lymphoma Rounds, Johns Hopkins Medical Institutions, Baltimore, MD, February, 2001.

Grufferman S. The enigma of Hodgkin's disease etiology. Medical Grand Rounds, University of New Mexico, Albuquerque, NM, March, 2001.

Grufferman S. The hematopoietic cancers. Principles and Practice of Cancer Prevention and Control Course. National Cancer Institute, Rockville, MD, July, 2001.

Grufferman S. New epidemiologic approaches to the study of lymphomas. Pathology Grand Rounds, University of New Mexico, Albuquerque, NM, March, 2002.

Grufferman, S. New findings in the epidemiology of childhood Hodgkin's disease. Conference on "Hematologic Malignancies: 100 Years of the Reed-Sternberg Cell", Johns Hopkins Medical Institutions, Baltimore, September, 2002.

Grufferman, S. New findings in the epidemiology of childhood Hodgkin's disease. University of Manchester, England, U.K., May 2003.

Grufferman, S. The history of the quest for an infectious cause of Hodgkin lymphoma : From Bacillus hodgkini to the Epstein-Barr virus. Harvard University School of Public Health, Boston, May, 2006.

Grants:

Grufferman S, Principal Investigator: Clustering of Etiologic Exposures in Hodgkin's Disease, F22 CA00279, N.I.H. $16,200, 06/15/74 - 06/14/75.

Grufferman S, Principal Investigator: The Epidemiology of Childhood Rhabdomyosarcoma, ROI CA21244, N.I.H, $34,572, 04/01/77 - 03/31/79.

Grufferman S, Principal Investigator: The Epidemiology of Multiple Myeloma, R01 CA22104, N.I.H., $89,213, 05/01/77 - 04/30/80.

Grufferman S, Principal Investigator: The Epidemiology of Multiple Myeloma (Competitive Renewal), ROI CA22104, N.I.H., $67,096, 05/01/80 - 04/30/83.

Grufferman S, Principal Investigator: Preventive Oncology Academic Award, K07 CA00726, N.I.H., $316,345, 09/30/80 - 08/31/85.

Grufferman S, Principal Investigator: American Family Corporation Grant for Cancer Epidemiology, $225,000, 1980 - 1985.

Grufferman S, Rosati R, Co-Principal Investigators: A.W. Mellon Foundation Award in Clinical Epidemiology, $650,000, 3/01/81 - 02/28/84.

Grufferman S, Principal Investigator: The Epidemiology of Childhood Rhabdomyosarcoma, ROI CA21244, N.I.H., $271,744, 01/01/83 - 12/31/85.

Grufferman S, Feussner J.R., Co-Principal Investigators: A.W. Mellon Foundation Award in Clinical Epidemiology, $650,000, 1/01/85 - 12/31/87.

Grufferman S, Principal Investigator: Epidemiology of Lymphomas in a Unique Exposure Group, ROI CA39163, N.I.H., $152,374, 03/01/86 - 06/30/88.

Grufferman S, Principal Investigator: The Epidemiology of Childhood Rhabdomyosarcoma (Competitive Renewal), R01 CA21244, N.I.H., $163,000, 07/01/86 -12/31/88.

Grufferman S, Principal Investigator: Screening, Detection and Prevention of Selected Cancers, State of Pennsylvania, $869,565, 9/01/87 - 08/31/89.

Grufferman S, Principal Investigator: Case Control Study of Hodgkin's Disease in Childhood, ROI CA47473, N.I.H., $410,123, 09/01/87 - 08/31/93.

Grufferman S, Principal Investigator: Epidemiologic Studies of HIV-Associated Malignancies, R01 CA48643, N.I.H., $471,305, 8/01/88 - 7/31/92.

Grufferman S, Principal Investigator: Pre-doctoral Training in Family Medicine, D15 PE83014, DHHS, $190,677, 7/l/89-6/30/92.

Grufferman S, Co-Principal Investigator: Solid Tumor Program (subcontract) Project 1 (St. Jude Children's Research Hospital), P01 CA 23099, N.I.H., $134,677, 7/1/92-4/30/95.

Grufferman S, Principal Investigator: Case-Control Study of Hodgkin's Disease in Childhood (Competitive Renewal), RO1 CA47473, N.I.H., $1,025,078, 2/l/94-9/9/99.

Expert Report of
Marion J. Fedoruk M.D., CIH, DABT, FACMT

Case No. C-4885-99-F(10)

Guadalupe Garza v. Allied Chemical Corporation, et al.
332nd Judicial District, District Court of Hidalgo County, Texas

Table of Contents

1.0 Purpose

The purpose of this report is to provide my opinions with respect to the issue of whether or not Guadalupe Garza, a 66-year-old resident of Mission, Texas developed follicular B-cell non-Hodgkin lymphoma (NHL) as a result of her alleged exposure to DDT and/or its metabolites from the Hayes-Sammons mixing facility in Mission, Texas.

The report includes an assessment concerning the issue of whether or not DDT and/or its metabolites cause or contribute to the development of NHL. The report also examines the assumptions, methods and conclusions of plaintiff's expert, Dr. Sawyer, regarding Ms. Garza's alleged exposure to DDT and her NHL. Also examined is the basis for Dr. Sawyer's general causation opinion that DDT causes NHL, as well as his specific causation opinions concerning Ms. Garza's NHL.

This report also examines the assumptions, methods and conclusions of plaintiff's other expert, risk assessor Dr. Strauss. Dr. Strauss conducted a health risk assessment which purports to provide predictions of Ms. Garza's DDT intake and overall theoretical "cancer risk" allegedly associated with the Hayes-Sammons site.

2.0 Summary of Opinions

- It is my opinion, to a reasonable degree of medical probability, that the scientific literature does not demonstrate that DDT and/or its metabolites cause or contribute to the development of NHL, including follicular lymphoma.
- DDT is considered to be one of the most investigated pesticides with respect to the occurrence of NHL.
- Cohort studies of workers with DDT exposure have not demonstrated a statistically significant increased NHL incidence and/or mortality.

- Case-control studies involving DDT, including studies that utilize DDT biomarkers, show no or very limited associations between DDT and NHL that largely disappear following adjustment for other exposure.
- Ecological data on NHL rates and DDT use in the U.S. and other countries do not support the conclusion that DDT causes NHL.
- Texas Department of Health investigations that have been conducted to evaluate potential health impacts from Hayes-Sammons show no increased cancer rates in Mission, Texas and no increased serum DDT levels in persons living and/or working in close proximity to Hayes-Sammons.
- Dr. Sawyer's claim that DDT causes, or substantially contributes to NHL is not substantiated by the epidemiological literature.
- Dr. Sawyer's reliance on the risk assessment process and Dr. Strauss's theoretical "cancer risk" estimates for the purpose of determining the cause of Ms. Garza's NHL lacks scientific validity.
- Dr. Strauss's health risk assessment is based upon incomplete environmental data and consequently her predicted theoretical "cancer risk" estimates are unreliable.
- Dr. Strauss's 3-fold increase of Ms. Garza's theoretical "cancer risk" is inconsistent with EPA guidance.
- Dr. Strauss's estimates of Ms. Garza's theoretical "cancer risk," even when increased by 300% for "506 Nicholson," are insignificant when compared to her actual background cancer risk.
- Dr. Strauss's presumed DDT intake level for Ms. Garza while residing at "506 Nicholson" is similar to the background daily intake reported for the general public, whereas Dr. Strauss's presumed DDT intake for Ms. Garza while at 1015 Nicholson is less than the amount she would have received in her diet at that time.
- Ms. Garza has other risk factors for NHL. One risk factor, obesity, is increasingly being recognized through several studies as being important. Another potential risk factor is Ms. Garza's past employment in a cleaning occupation. Although these risk factors cannot be determined as casual for her follicular lymphoma, they serve to highlight the speculative nature of the plaintiff's experts' claims that

a known cause can be specified, such as DDT. Yet, her presumed DDT intake is similar or less than that of the general public, and her theoretical "cancer risk" is minute compared to her overall background risk of developing cancer.

3.0 Qualifications

I am a licensed medical doctor, practicing in the fields of occupational and environmental medicine, medical toxicology and industrial hygiene. I received my medical degree with Distinction from the University of Alberta, Edmonton, Canada, in 1978. I performed postgraduate medical training through McGill University, Montréal and my residency in Occupational Medicine at University of California Irvine from 1981 to 1983.

I hold a primary board certification by the American Board of Preventive Medicine in Occupational Medicine. I also hold a subspecialty certification in Medical Toxicology from the American Board of Preventive Medicine. The Medical Toxicology subspecialty board is sponsored by three primary medical boards: Emergency Medicine, Pediatrics and Preventive Medicine. There are fewer than fifty physicians in the United States that hold board certification in both occupational medicine and medical toxicology. Also, I am board certified as a toxicologist by the American Board of Toxicology, and hold a board certification in industrial hygiene from the American Board of Industrial Hygiene, with a specialty focus in toxicology. The discipline of industrial hygiene deals with the assessment of workplace and environmental hazards and measures to control such hazards. As an industrial hygienist, I have expertise in the use of environmental exposure data to assess human exposure and ascertain potential doses that an individual might receive from different environments.

Currently, I am employed by the University of California, Irvine ("UCI") at the Center for Occupational and Environmental Health (COEH). The COEH was established within the University of California to train occupational health scientists and professionals, conduct research on occupational and environmental health issues, and provide services

to the public, employers and workers. I hold the title of Clinical Professor of Medicine in the Department of Medicine. I was designated as the Senior Physician at the UCI COEH occupational and environmental medicine specialty clinic, a designation awarded to a physician with outstanding expertise in their field. I am also employed as a Principal Scientist for Exponent in the Health Sciences practice. I was also elected a Fellow of the American College of Medical Toxicology (FACMT). This recognition is awarded by the American College of Medical Toxicology, the specialty society that represents the nation's medical toxicologists, to medical toxicologists who meet the highest standing in their field.

I have extensive experience in the assessment of pesticide-related hazards as well as medical assessment of persons exposed to pesticides. This includes periodic testing of pesticide exposed workers as well as treatment of persons with accidental pesticide exposure. I served as an expert member of a joint panel convened by the U.S. Department of Labor, U.S. EPA and U.S. Department of Food and Agriculture for developing a national program for training the nation's health care providers on how to recognize pesticide-related health problems. I also served on a USC/NIOSH advisory panel to develop training programs for public health and Department of Agriculture officials concerning how to deal with pesticide-contaminated waste sites.

In addition, I have conducted studies and published articles in the areas of occupational and environmental medicine, and authored chapters in several books, including the World Health Organization's Encyclopedia of Occupational Safety and Health, and the Encyclopedia of Toxicology. I am the recipient of the Merit in Authorship Award from the American College of Occupational and Environmental Medicine and the Jean Spencer Felton Award for Excellence in Scientific Writing from the Western Occupational and Environmental Medical Association.

Attached is my current curriculum vitae.

4.0 Disease at issue – Non-Hodgkin Lymphoma

Non-Hodgkin Lymphoma (NHL) is a term used to describe a heterogeneous group of malignancies that arise from lymphoid tissue. NHL occurs when a particular lymphoid cell (B-cell, T-cell, or NK cell) undergoes malignant transformation. NHL can originate in any lymphoid tissue including lymph nodes, gut-associated lymphatic tissue, and the spleen. Criteria used to classify lymphoid tumors have varied, but currently the World Health Organization (WHO) system is principally used to classify different types of lymphoma. The most common NHL types in Western countries are diffuse large B-cell (DLBC) type and follicular lymphoma.

NHL represents approximately 4 percent of cancer incidence and approximately 3 percent of all cancer-related deaths in the U.S. In 2006, about 58,870 new cases were predicted and approximately 18,840 persons were expected to die of lymphoma in the United States (Jemal, et al., 2006). The disease incidence increases with age. In the 1970s and 1980s, age adjusted incidence increased by about 3-4 % annually. In the 1990s, the increasing rate diminished. The increased incidence is attributable in part to the HIV epidemic since persons with HIV infection are at substantially greater risk of developing NHL.

The cause for most types of NHL, including follicular lymphoma, has not been established (Hartge, et al., 2006). Several risk factors have been identified for particular NHL types. Immunosuppression is considered to be a significant risk factor and persons with congenital immunodeficiency disorders are at very high risk of developing NHL. Use of immunosuppressive medications following organ or stem cell transplantation results in a substantially increased risk. Autoimmune disorders including Sjögren's syndrome and celiac disease are associated with an increased risk. Some viral infections are strongly associated with NHL risk. Infection with HTLV-1, an endemic retrovirus found in the Caribbean, Southwestern Japan, and Southeastern U.S., results in adult T-cell leukemia/lymphoma. Epstein-Barr virus infection has been identified as playing a

significant role in Burkitt's lymphoma. Obesity is being increasingly recognized as being a risk factor for NHL (Maskarinec, et al., 2008; Chiu, et al., 2007; Willet, et al., 2005; Rapp, et al., 2005; Skibola, 2007).

Numerous occupations have been identified as potential risk factors including cleaners, meat workers, farmers, animal husbandry, heavy truck drivers, metal product manufacturing, teachers, wood workers, printing industry work and textile workers (Boffeta and deVocht 2007; t'Mannetje, et al., 2007; Svec, et al., 2005; Karunanayake, et al., 2008; Alexander, et al., 2007). Numerous dietary factors have also been studied, including total fat intake, meat intake, dairy intake, fruit and vegetable consumption, and micronutrients (Cross and Lim 2006; Alexander, et al., 2007). Agricultural and residential pesticide exposures including insecticides, herbicides and fumigants have been evaluated as potential risk factors. A causal association between a specific environmental factor and NHL has not been established (Müller, et al., 2005; Alexander, et al., 2007).

Genetic factors are increasingly being recognized as underlying risk factors for NHL. Historically, familial NHL clusters have been reported and having a family member with the disease represented an increased risk factor. Proposed genetic risk factors for NHL have included polymorphisms on nitric oxide synthetase genes, which modify interactions between fruit and vegetable intake and NHL risk (Han, et al., 2009) and oxidative stress genes (Wang, et al., 2006).

Many epidemiological studies have focused on NHL in aggregate and not on the individual NHL subtype. Since NHL represents a heterogeneous group of disorders, risk factors for each subtype may vary. Several epidemiological studies are beginning to evaluate risk factors for individual subtypes. Advances in testing for genetic markers enable epidemiological studies to now begin to evaluate the role of genetic susceptibility factors.

Several studies have assessed potential risk factors for follicular lymphoma, the NHL at issue in this case. Factors reported in certain studies to potentially increase follicular lymphoma risk include use of permanent hair dyes before 1980, polymorphism in the *MGMT* gene, and the *NAT2* acetylation phenotype (Morton, et al., 2008). Variations of the *BCL2L11* gene have also been reported as a potential susceptibility factor for follicular lymphoma (Morton, et al., 2009). Obesity has also been reported to be associated with an increase in follicular lymphoma risk (Skibola, et al., 2004). Future epidemiological investigations that measure and evaluate more specific disease risk factors or potential confounders, such as genetic susceptibility, offer the potential to better understand the true cause(s) of the different types of NHL, including follicular lymphoma.

5.0 Epidemiological concepts

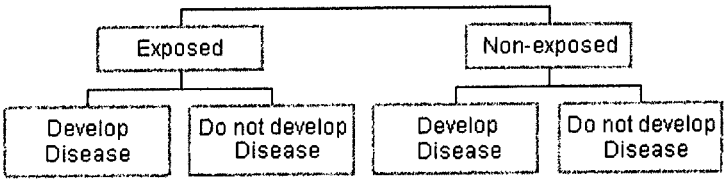
Epidemiology is the study of the cause of disease in human populations. Epidemiological studies examine the occurrence or distribution of disease in populations to help ascertain if a causal relationship exists between any exposure at issue in the population and a particular disease. Medical toxicologists routinely examine epidemiological literature as a main source of information about the effects of a chemical substance on humans. This information coupled with consideration of dose, exposure routes, and exposure duration is evaluated in aggregate to ascertain whether a substance is responsible for a specific health effect.

There are two main types of analytical epidemiological studies that are performed to evaluate whether a factor such as an environmental, occupational, or lifestyle exposure is responsible for causing a disease. The two types of epidemiological investigations are cohort and case-control studies. These are described below.

5.1 Cohort Studies

Cohort studies involve a study population that is followed over time for the occurrence of disease or death from the disease. Mortality data can be used to assess the occurrence of disease, especially when disease leads to death in the majority of cases, (e.g., lung cancer). The basis for defining the study population is the presence or absence of exposure to the factor of interest. For example, a cohort may include a population of workers exposed to coal dust. Following this population over time for development of new cases of disease (or death) provides data to define an incidence or mortality rate. A control or non-exposed population is also followed over time to serve as a comparison group for evaluating the effects of exposure in the exposed population. Cohort studies have the advantage that exposure information is collected prior to the onset of disease. A depiction of a cohort study is presented below.

Cohort Study Design



The term relative risk (RR) is used to describe the ratio of disease incidence or death in exposed individuals compared to the incidence of disease or death in unexposed individuals. Sometimes the rate of disease in a study group is compared to an external population such as a country, state, or county. The terms proportionate and standardized incidence (or mortality) ratios are used to describe such comparisons. These types of ratio measures are typically standardized by age and sex to provide a summary adjusted relative risk estimate that accounts for these factors.

If the relative risk or other risk estimate (PIR, SIR, SMR, etc.) equals 1, the incidence of disease in the exposed group equals the incidence of disease in the unexposed group and

the result is interpreted as "no association." If the RR is > 1, the incidence of disease in the exposed group exceeds the incidence of disease in the unexposed group and the result is interpreted as demonstrating an association, though not necessarily a causal association. If the RR is < 1, the incidence of disease in the exposed group is lower than the incidence of disease in the unexposed group and the result is interpreted as not demonstrating an association. If the ratio is significantly less than 1.0, it may indicate that exposure is protective in instances where the research interest concerns a vaccine, medical treatment, or some type of public health prevention program. Otherwise, without a strong biological rationale as to why exposure might be protective, it is usually interpreted simply as not associated.

5.2 Case-Control Studies

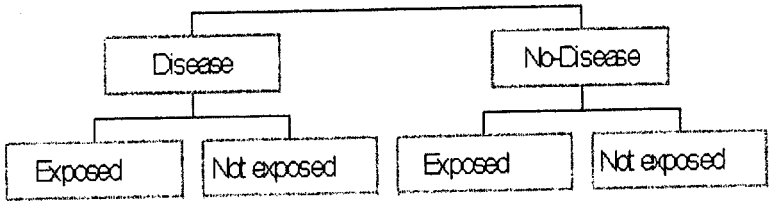
Case-control studies are used to assess factors that may cause or contribute to a disease by comparing the presence of the factor in persons with disease (cases) to persons without the disease but who are otherwise similar. Thus, the study population is defined by the presence or absence of disease. In contrast, cohort studies define study population on the basis of exposure potential. Data about exposure is obtained from persons with the disease and compared to data from control population in an attempt to ascertain whether the factor is associated with the disease.

Case-control studies are particularly useful for studying rare diseases, for which a cohort study may not be feasible because of the large population size and resources needed to conduct such a study. In a case-control study, cases are typically identified through disease registries, hospital, or medical claims data sources. Controls are then selected in a way to be representative of the population that generated the cases with respect to exposure history. In some studies the general population is selected (referred to as "population controls"), or they can also be selected from the same hospital where the cases were treated or identified. A nested case-control study is one that identifies cases and controls from within a cohort.

Case-control studies, because they involve much smaller numbers of participants, may permit more intensive collection of health, work history and other information from study participants than is typically obtained from subjects in a cohort study. Case-control studies however have several limitations including recall and selection bias. Recall bias can occur when persons with disease recollect past exposures more readily than controls. Selection bias can occur when the control population does not adequately represent cases. Because case-control studies often rely on self-reported exposure information from interviews and/or questionnaires rather than quantitative unbiased information that may be available in a cohort study (before disease has occurred), exposure misclassification can occur and bias the risk estimates.

A depiction of a case-control study is presented below.

Case-Control Study Design



To analyze an association between exposure and disease in a case-control study, the concept of odds ratio (OR) is used. An OR compares the probability that the event will occur to the probability that the event will not occur. In a case-control study, the OR represents a comparison of odds of exposure to a risk factor among cases to the odds of the same exposure among controls. Thus, the OR is a measure of relative risk in case-control studies.

Similar to the relative risk estimates, if the OR equals 1, the incidence of disease in the exposed group equals the incidence of disease in the unexposed group and the result is interpreted as "no association." If the OR is > 1, the incidence of disease in the exposed

group exceeds the incidence of disease in the unexposed group and the result is interpreted as demonstrating an association, though not necessarily a causal association. If the OR is < 1, the incidence of disease in the exposed group is lower than the incidence of disease in the unexposed group, and the result is interpreted as not demonstrating an association; although, when significantly less than one, it may indicate a protective effect.

5.3 Statistical Significance

Because the presence of an association may be due to chance, tests for statistical significance are used to evaluate the possibility of whether an observation or association occurred by chance. Two general statistical terms are used to assess the likelihood that findings occurred by chance: confidence intervals (CIs) and *p*-values. The concepts of CIs and/or *p*-values are recognized by the scientific community as valid methodologies for assessing whether observations occurred by chance.

5.3.1 Confidence intervals

Confidence intervals provide information about whether or not an association is statistically significant in addition to assessing the precision of the measure of a potential association. A confidence interval is a range of values for a parameter of interest (e.g., relative risk) constructed so that this range has a specified probability of including the true value. A 95% confidence interval indicates that the investigators are 95% confident that the true results lie within its upper and lower limit. Confidence intervals at the 95% range are routinely used in epidemiology to evaluate the possibility of whether a study finding is due to chance.

If a 95% confidence interval includes 1.0, the result is considered non-significant since the findings could be due to chance. If the 95% confidence interval does not include 1.0, the probability of an association being due to chance alone is 0.05 or lower. If the confidence interval is very wide, this indicates there is considerable uncertainty in the

value of the "true" relative risk estimate. A small number of observations can result in wide confidence intervals. Narrow confidence intervals are indicative of less uncertainty about where the "true" estimate lies.

5.3.2 *P*-Value

A *p*-value is used as a measure of the probability (likelihood) that the observed findings are due to chance alone. The lower the *p*-value, the less likely it is that the study results occurred by chance. When the probability is sufficiently low, the result is considered statistically significant. Most commonly, the cutoff for statistical significance is a *p*-value equal to, or less than, 0.05, which means that there is at most a 1 in 20 chance that the study results are due to chance.

5.4 Interpretation of Epidemiological Studies

The presence of an association, even if it is statistically significant, does not establish causation. Associations can occur as a result of chance or design factors in the study which can affect outcomes. These factors can include subject selection bias, differences in case ascertainment or definition, information bias, confounders, and misclassification of exposure. These factors should be considered in evaluating an epidemiological study.

In determining whether causation exists between specific factors and disease outcomes, the broad range of scientific literature should be examined. The Bradford Hill Criteria are guidelines commonly used to assess causation (Hill, 1965). This includes examining the scientific literature from several perspectives, including:

1) Strength of the association: Is the magnitude of change large enough to be statistically significant?

2) Consistency: Has it been repeatedly observed in different studies conducted by different researchers, in different places, circumstances and times?

3) Specificity: Is the available information sufficient to identify the specific cause and is the disease at issue specific enough to draw reasonable conclusions?

4) Time course: Is the onset of the specific health condition consistent with its natural history and the time of exposure to the chemical? i.e. have studies clearly established that exposure precedes disease and is not a consequence of the disease process?

5) Dose-response: Does the severity of incidence of the health condition increase as the magnitude or duration (or both) of exposure increase(s)?

6) Biological plausibility: Is there scientific information that offers a rational explanation for how the disease in question could be caused by the chemical?

7) Experimental association: Do changes that reduce or increase exposure also reduce or increase the occurrence or severity of the disease?

8) Analogy: Do similar exposure situations with toxicologically similar chemicals suggest an association with the disease?

9) Coherence of evidence: The cause and effect interpretation of the chemically induced disease should not seriously conflict with generally known facts of the natural history and biology of the disease. The evidence from various sources (histopathology, epidemiology, clinical studies, etc.) should be both convincing and consistent for reaching the conclusion of causation.

I understand that there is a legal standard in Texas, derived from a case called Merrell Dow Pharmaceuticals Inc. versus Havner (Havner), that is utilized by courts to interpret epidemiological studies. This standard indicates that for an epidemiological study to be considered relevant for supporting causation, the study has to demonstrate a statistically significant increased relative risk or odds ratio (OR) of more than 2.0. The approach used in Havner for evaluating epidemiological literature is consistent with the approach I used to analyze epidemiological studies dealing with DDT and NHL.

6.0 Studies of human populations do not demonstrate that DDT and/or its metabolites cause or contribute to the development of NHL

To determine whether DDT or its metabolites cause NHL, I conducted a review of the medical and scientific literature to identify studies that evaluated the potential association of DDT and/or its metabolites and NHL. I searched for peer-reviewed scientific literature using electronic data bases of the National Library of Medicine (Pub-Med and Toxline) with the search terms DDT, organochlorines, NHL, lymphoma, and follicular lymphoma. I also examined the bibliographies of published scientific review papers concerning DDT for other potential studies. I also examined textbooks dealing with occupational medicine, toxicology, and industrial hygiene for other studies that may not have been identified. Finally, I examined public health agency publications regarding DDT, including publications by the National Toxicology Program, Agency for Toxic Substances and Disease Registry, and International Agency for Research on Cancer. My objective was to identify all studies that provided information concerning both NHL and DDT–specific exposures.

I reviewed 38 published studies, including occupational cohort studies, population case-control and nested case-control studies where cases were selected from a larger cohort study. To assess the potential effects of DDT with respect to NHL, only studies that included DDT-specific exposure or biomarker data and NHL were included. Studies that

examined general risk factors such as farming or agricultural work that did not include DDT-specific exposure information were not utilized to determine the effect of DDT on NHL since they lacked the requisite specificity. Based upon a review of these 38 published studies (a very large literature set), I have concluded that studies of human populations do not demonstrate that DDT and/or its metabolites cause or contribute to the development of NHL.

The studies that I relied upon are described below.

6.1 Cohort Studies

6.1.1 Occupational cohort studies of DDT-exposed populations

Occupational or worker cohort studies provide unique insights into whether a substance is responsible for producing a particular disease since they frequently involve relatively high exposures making the occurrence of agent-specific adverse health effects more likely. Cohort studies obtain exposure information before the cohort member (person) develops the disease at issue. Consequently, differential reporting of exposures between cases and controls which can occur in case-control studies can be avoided.

Occupational cohort studies include:

- Purdue, et al., 2006: The National Cancer Institute's Agricultural Health Study is a large prospective cohort study of 57,311 private and commercial pesticide applicators in North Carolina and Iowa. The cohort was recruited between 1993-1997 and cancer incidence data were collected through December 31, 2002. Exposure was assessed by questionnaire and evaluated 28 pesticides. For organochlorine exposures, cumulative exposure was estimated for each organochlorine (including DDT) and for organochlorines that shared structural similarities (aldrin/dieldrin and chlordane/heptachlor). No association was found

between the subcohort of DDT-exposed applicators (n= 12,035) and NHL (RR = 0.9; 95% CI=0.6-1.5). Moreover, DDT use was not associated with any increased cancer risk. This study's strengths include large populations with pesticide exposure, detailed exposure information prior to diagnosis as well as controlling for a variety of potentially confounding factors, such as demographics and lifestyle.

- Cocco, et al., 1997a; Cocco, et al., 1997b: Cocco and colleagues conducted a mortality study of male Sardinian workers who had been exposed to DDT from working in an anti-malarial campaign from 1946-1950. DDT was used to eradicate malaria-carrying mosquitoes and flies. The cohort also had some exposure to chlordane and arsenic. The cohort included 2,908 workers who mainly applied DDT and inspected after DDT applications and 2,285 workers such as supervisors, administrative staff, laboratory staff, and directive staff who were considered unexposed. Applicators were reported as applying DDT both inside homes and outdoors. The DDT exposure concentrations were estimated to range from 170 – 600 mg/m^3 indoors and 24-86-mg/m^3 outdoors. Vital status was reported for 2,115 subjects and death certificates were available for 1,123 (53%) subjects. Deaths that occurred from 1956-1992 (1,043 subjects) were selected for a proportionate mortality study with the reference population consisting of Italian males. The PMR for NHL was 81 (95% CI=9-294). Thus, less than the number of expected NHL deaths was observed in this worker population.

- Cocco, et al., 2005: The Sardinian worker cohort was reexamined to include deaths through to December 31, 1999 for an additional seven years of mortality data. This cohort included 4,552 men who were subdivided into three sub cohorts: applicators (n=2,578), bystanders (n=683), and non-exposed workers (n=1,291). Among this group were 3,037 deaths, 2,726 of which had adequate death certificates. Estimates of DDT dose were assessed for this population using the European Predictive Operator Exposure Model (EUROPOEM). Daily DDT

doses were estimated to range from 54 µg to 140,400 µg and groups were divided into exposure quartiles. Mortality experience was compared to the Sardinian population to estimate a SMR. The non-exposed cohort served as an internal reference and Poisson regression was used to model specific cause of death as a function of both cumulative exposures, age of starting exposure, age at exit of exposure and ethnic origin.

The SMR for lymphatic cancer for applicators was 101 (95% CI=65-157); for bystanders 83 (95% CI=44-153); and for unexposed workers was 174 (95% CI=112-271). Cause-specific mortality risk (adjusted for age at exit for follow up, age at first exposure, ethnicity) for lymphatic cancer by DDT exposure category for applicators was 0.7 (95% CI=0.4-1.3); for bystanders 0.6 (95% CI= 0.3-1.2); and for all exposed 0.7 (95% CI= 0.4-1.1). No significant increased risk for any quartiles of daily exposure for any cancer was observed among applicators.

- Laws, et al., 1967: This study examined the health outcomes of workers at Montrose Chemical Corporation of California plant. The study included medical evaluations of 35 men who were considered to have high or medium exposure to DDT. These men had significantly elevated DDT levels compared to the general population and their total DDT adipose levels (38 to 647 ppm) were much higher than general population levels which were reported to be around 8 ppm. Medical records from 63 workers with DDT exposures over the prior five years revealed no cancers or blood dyscrasias.

- Ditraglia, et al., 1981: This study examined mortality of workers employed in the manufacture of organochlorine pesticides at four plants, including one California plant that only manufactured DDT since 1947. The study cohort included workers employed for at least six months prior to December 1964. Mortality was followed to December 31, 1976. The plant manufacturing DDT had 354

employees and included 7601 person-years of observation. Vital status was ascertained for 90% of the cohort, and death certificates were used to classify cause of death. Mortality was compared to age-adjusted mortality of U.S. white males. The SMR for all malignancies was 68 (95% CI=25-247). This decrease was attributed to healthy worker effect and possibly a lack of complete vital status ascertainment. No lymphatic or hematopoietic malignancy was reported for this DDT-exposed cohort.

- Brown 1992: This is a reassessment of the workers at the DDT plant previously evaluated by Ditraglia, et al. Mortality data was obtained through 1987. There were no deaths reported for any type of lymphatic cancer. Daily intake of DDT in heavily exposed workers at the plant formulating DDT was considered to range from 17,500-18,000 µg/day.[1]

6.2 Case-Control Studies with biomarkers

Case-control studies have been performed utilizing biomarkers which assess tissue levels of DDT and/or its metabolites as markers of exposure. The term "biomarker" is used to describe tests that can be used to measure a person's exposure to a chemical agent by measuring the substance and/or its metabolites in body tissues (e.g., blood, urine, adipose tissue, etc.). Case-control studies involving biomarkers offer a unique opportunity to evaluate the potential impact of DDT exposure on occurrence of NHL by being able to assess exposure potential through an objective measure and ascertain which subjects in the study populations were actually exposed, and depending upon the validity of the biomarker, the magnitude of such exposure.

As a medical toxicologist, I find that studies which use biomarker exposure measurements, such as blood and tissue levels of a compound, can be especially useful in examining the relationship, if any, between a chemical and a specific health outcome.

[1] Dr. Strauss estimates that Ms. Garza's daily intake during her residence at "506 Nicholson" was about 108 µg/day.

Such studies minimize ambiguity and bias that may occur when an exposure estimate is based on a person's recollection or memory, or when exposure is simply assumed to have occurred because of an individual's placement in a particular class of workers. In many case-control studies, exposure information from deceased subjects is obtained from spouses or next of kin who are even less familiar about exposure potential than persons who developed the condition.

Measurement of DDT and/or its metabolites has some limitations for determining exposure potential. One particular concern is the potential for NHL to affect tissue DDT levels. Biological changes from NHL might affect tissue DDT concentrations. Cachexia from cancer and chemotherapy can affect serum/plasma DDT levels (Baris, et al., 2000; Hoppin, et al., 2000). Weight loss in general can produce changes of DDT or its metabolites (Pelletier, et al., 2003; Backman and Kolmodin-Hedman, 1978; Chevrier, et al., 2000). Thus, when evaluating the results of case-control studies that utilized DDT biomarkers and NHL, it is essential to ascertain whether the DDT tissue samples were collected before disease onset to avoid bias that may occur from a selective effect on tissue levels in cases.

Case-control studies with biomarkers of exposure are described below:

- Cocco, et al., 2008: This case-control study examined the association between blood levels of *p,p'*-DDE and 25 other organochlorines and NHL risk. The study also evaluated association between plasma *p,p'*-DDE and two major NHL subtypes: diffuse large B-cell (DLBC) lymphoma and chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL). The study population consisted of 174 NHL cases and 203 controls (French, German and Spanish subjects) drawn from the European multicenter case-control study concerning the etiology of lymphoma (Epilymph). Blood was collected after diagnosis but before chemotherapy had started. Plasma *p,p'*-DDE levels were adjusted and categorized into quartiles. Information about potential covariates was obtained by

interview and questionnaire. Factors included in the final analysis included age, gender, education level, center, and levels of other organochlorines. There was no overall increased risk of NHL.

- Engel, et al., 2007a: This nested case-control study examined the association between serum or plasma *p,p'*-DDE levels and other organochlorines and NHL. The study population included 294 cases and 415 controls drawn from three cohorts: Janus -190 cases and 190 controls drawn from 87,600 Norwegian men and women; CLUE I -74 cases and 147 controls drawn from 23,938 Washington County, Maryland residents; and Nurses' Health Study -30 cases and 78 controls drawn from 121,700 female nurses. The 174 cases from the CLUE I cohort were previously described (Rothman, et al., 1997). Blood collection was performed years prior to NHL diagnosis. *p,p'*-DDE levels were lipid adjusted (ng/gram lipid) and categorized into exposure quartiles. Separate statistical analyses were performed for each cohort because of differences in time when blood was collected and differences in blood analyses between cohorts. Organochlorine levels were adjusted for body mass index in the Janus and Nurses' Health Study cohorts but not for CLUE I.

  The ORs and 95% CIs for NHL in relation to *p,p'*-DDE concentrations were not significant. In the Janus cohort, when examined for early (2-16 year) follow-up, the OR for the highest *p,p'*-DDE quartile (7,513 ng/gm lipid median) was 4.3 (95% CI=1.2 -15.0). This *p,p'*-DDE effect was diminished and was no longer statistically significant when controlled for PCB exposure. The OR was not significant for the late follow-up period OR 0.8 (95% CI=0.3-2.0) in the Janus cohort. In the CLUE I cohort, the ORs were not statistically significant, and there was no difference in ORs between early and late follow-up period. Importantly, the *p,p'*-DDE effect in all instances decreased and ORs were not statistically significant when controlled for PCB exposure. In contrast, *p,p'*-DDE adjustment had only a minimal effect on PCB risk estimates. The authors concluded that

there was no apparent trend or exposure response between *p,p'*-DDE and NHL in most of the analyses.

- Hardell, et al., 2009: The 99 NHL cases in this study were drawn from four county regions in Sweden with cases of newly diagnosed NHL, presumably from 2000-2002. This included 20 cases of follicular lymphoma. Controls were drawn and apparently already enrolled in a NHL study examining effects of cell phones on NHL. Blood was collected after NHL diagnosis but before treatment. No information is provided whether cases had experienced weight loss or other disease complications that could affect *p,p'*-DDE levels. Blood was analyzed for several organochlorines including PCBs, *p,p'*-DDE and PBDE. Chemical analyses of blood included internal PCB standards but apparently no standard for *p,p'*-DDE. IgG antibodies to Epstein-Barr virus (EBV) capsid antigen and to early antigen (EA) were also examined. NHL cases were divided into low and high categories based upon median *p,p'*-DDE levels and low and high EA antibody titers.

  There was no significant difference in *p,p'*-DDE between all NHL cases and controls. The OR for follicular lymphoma was 1.2 (95% CI=0.4-3.5). Of note, the BMI of cases was significantly different from controls which could be an indication that NHL disease process could be affecting *p,p'*-DDE levels in cases and influencing study findings.

- Hardell, et al., 2001: This case-control study examined the association between lipid-adjusted blood or adipose tissue *p,p'*-DDE levels and NHL. The study population included 82 cases and 83 controls. 50 NHL cases diagnosed between 1994-1997 had adipose tissue samples while 32 cases diagnosed between 1997-1999 provided blood samples. EBV antibody titers were also measured for viral capsid antigen and early antigen.

The study showed no significant association between NHL and persons with *p,p'*-DDE levels above the median value (663 ng/gm-either blood and adipose tissue) – OR 1.2 (95% = 0.60-2.5); multivariate analyses showed similar results. There was no statistically significant increased NHL risk in persons who had values above the median *p,p*-DDE level.

- Hardell, et al., 1996: *p,p'*-DDE levels in adipose tissue (collected from abdominal wall) of a group of 28 newly diagnosed patients with NHL of B-cell type were not significantly different from 17 surgical controls.

- Rothman, et al., 1997: This nested case-control study examined the association between NHL risk and serum concentration of DDT and PCBs. The study population consisted of 74 NHL cases and 147 controls drawn from persons in the Campaign against Cancer and Stroke (CLUE I) cohort from Washington County, Maryland. Controls were members of the CLUE cohort who did not have cancer and were matched for several factors including race, sex, date of birth (within 1 year) and date of blood collection (within 15 days). NHL cases and controls had prediagnostic testing for serum DDT and its metabolites and questionnaires. Serum DDT (sum of *o,p'*-DDT, *p,p'*-DDT, *o,p'*-DDE, and *p,p'*-DDE) levels were lipid corrected.

  There was no significant difference in total lipid corrected serum DDT concentrations (sum of *o,p'*-DDT, *p,p'*-DDT, *o,p'*-DDE, and *p,p'*-DDE) between NHL cases and controls. Although NHL risk was reported to increase weakly with increasing DDT concentrations, this was not considered to be significant. A non significant association was observed between NHL risk and DDT with an OR 1.9 (95% CI=0.8-4.5) for highest quartile (DDT 4140-20,500 ng/gm lipid). This risk diminished after adjustment for PCB resulting in an OR 1.2 (95% CI=0.5-3.0) for highest serum DDT quartile and OR 1.1 (95% CI=0.4-2.7) for the 2nd quartile.

The authors concluded that "total lipid-corrected serum concentrations of DDT were not associated with risk of non-Hodgkin lymphoma."

- Spinelli, et al., 2007: This case-control study examined the potential associations of plasma levels of *p,p'*-DDE and *p,p'*-DDT, and 23 other organochlorines, including other pesticides and PCBs, with NHL risk. The study examined NHL subtypes including follicular, diffuse large cell, T-cell and other B-cell lymphoma. The study population included 422 NHL cases in males and females and 460 controls. Plasma *p,p'*-DDT and *p,p'*-DDE levels were lipid-corrected and categorized into quartiles. Plasma levels were measured before starting treatment and the cases were reported to not have sustained weight loss in excess of 10% prior to NHL diagnosis. Confounders which were reported to be examined in stepwise models used for data analysis included age, sex, region, education, BMI, ethnicity, farming, and family history of NHL.

  The authors reported that six of eight pesticide analytes were significantly associated with NHL. For *p,p'*-DDE, a significant trend was reported for NHL risk. The OR for all quartile lipid adjusted plasma *p,p'*-DDE levels showed no statistically significant association. The significant *p,p'*-DDE trend diminished after adjustment for oxychlordane. A significant trend was also reported for *p,p'*-DDE and follicular lymphoma. The ORs for follicular lymphoma for the largest compared to the smallest *p,p'*-DDE quartile was 1.8 (95% CI=0.9-3.3). *p,p'*-DDT levels were not associated with NHL.

  Data identified as lacking by the authors included current body mass index (BMI) (the authors collected weight data one year prior to interview), body fat index, and lactation history, all of which could have affected measured *p,p'*-DDE levels. The authors indicated that measurement error between analytes and/or random variation may also have contributed to observed results. Another explanation for findings was that unmeasured organochlorines related to NHL and correlated with

measured organochlorines were confounding the results. The authors recommended further research into possible interactions between organochlorines and other agents such as viruses and genetic markers.

- De Roos, et al., 2005: This population based case-control study examined the potential association between NHL risk and plasma levels of *p,p'*-DDE, *p,p'*-DDT and 66 other organochlorine compounds. The study population included 100 cases and 100 controls drawn from the National Cancer Institute NHL study from four SEER regions (Iowa, Los Angeles County, Detroit, and Seattle). The cases were randomly selected from 1321 newly diagnosed NHL cases (i.e., between July 1998 and June 2000). The population controls were selected by random digit dialing and from Medicare records. Response rate was 59% cases and 44% controls. Plasma levels were lipid adjusted and categorized in quartiles. Adjustments were made for age, sex, study site, and date of blood draw, but other potential confounders were also considered. Blood was only obtained from cases that had not received treatment.

  NHL risk was not associated with *p,p'*-DDE and *p,p'*-DDT levels for any quartile plasma levels. The authors, however, conducted an ad hoc analysis of an unspecified number of persons described as representing the "extremes of our exposure distributions" for *p,p'*-DDT. This analysis yielded an OR of 3.3 with a wide confidence interval (95% CI=0.7-15.9). The number of cases was not reported and overall significance of this finding is unclear. The authors found no clear association between NHL risk and *p,p'*-DDT and *p,p'*-DDE plasma levels.

- Quintana, et al., 2004: This study utilized tissue samples that were collected from 1969 to 1983 as part of the U.S. EPA National Human Adipose Tissue Survey (NHATS). The NHATS study measured adipose concentrations of organochlorines in about 20,000 persons. Cases represented persons with NHL diagnoses that were included in the NHATS data set. Of 175 NHL cases, 167

specimens were obtained at autopsy, and 8 were taken from pathology specimens obtained during surgical procedures. 481 controls were utilized and adipose tissue was obtained from subjects who had accidental death, injury, or myocardial infarction.

The mean levels of *p,p'*-DDT and *p,p'*-DDE were not significantly different between cases and controls. *p,p'*-DDT quartiles were not significantly associated with NHL but there was a significant trend when considered as a continuous variable for log transformed data. For *p,p'*-DDE, the highest exposure quartile had an increased OR of 1.99 (95% CI=1.14-3.47) with a significant trend. The increased risk for the highest *p,p'*-DDE quartile was no longer present when the risk was adjusted for other select other organochlorines. The authors reported that there was "no clear association" between exposure to DDT and NHL, and that the association with *p,p'*-DDE was confounded by other organochlorines.

6.3 Non-biomarker case-control studies

Most case-control studies have not utilized biomarkers (i.e., levels of DDT and/or its metabolites in tissues) to assess exposure to DDT. Typically, questionnaire and/or interviews are used to collect exposure information. In some case-control studies, the exposure information collected is very limited and consists of a simple yes-or-no question as to whether the person was ever exposed, without consideration for the duration or frequency of exposure. In other studies, exposures are simply assumed to have occurred based on whether a person held a specific job (e.g., cattle farmer), although there can be significant variability in exposure between workers who hold or held the same job title. Some studies attempt to obtain more detailed exposure information by asking questions concerning items such as names of products (chemicals) used, days applied, type of application, protective equipment used, size of area applied, etc. As described above, I examined case-control studies where specific information was provided about DDT exposure.

Case-control studies examining association of DDT with NHL are discussed below:

- Eriksson, et al., 2008: This case-control study examined the relationship between NHL cases, including NHL subtypes and exposure to pesticides, organic solvents, and several other chemicals. The study population included 910 cases and 1,016 controls including both males and females diagnosed between December 1999 and April 2002. Cases were selected from four of seven regions in Sweden and diagnosed between December 1, 1999-April 30, 2002. Controls were randomly selected from same health regions and were matched for age and sex. Exposure was assessed by questionnaire. Cases had been previously interviewed about lifestyle factors for another study being conducted of the same population. Logistic regression was conducted and adjustments were made for age, sex, and year of diagnosis. The participation rate was 91% of potential cases. No statistically significant increased OR was reported for DDT use—OR 1.46 (95% CI=0.94-2.28).

- De Roos, et al., 2003: This study of three pooled case-control studies from Nebraska, Iowa and Minnesota, and Kansas was conducted to assess the potential NHL risk from individual and combined pesticide exposures that farmers encounter. The study population consisted of 650 NHL cases and 1,933 controls for multiple pesticides analyses; the overall study population was 870 cases and 2,569 controls. ORs were provided for 47 individual pesticides and combinations of these pesticides using two statistical approaches that incorporated multivariate analysis. When adjusted for other used pesticides, the OR for DDT was 1.0 (95% CI=0.7-1.3), the same for both models. There were no super additive effects reported between DDT and chlordane. The authors concluded that determination of potential pesticide NHL risks should focus on individual chemicals and not groups of pesticides within classes. This study does not show a significant

association between NHL and DDT, even among subjects who had multiple pesticide exposure.

- Colt, et al., 2005: Colt and colleagues conducted a case-control study that examined the potential association between NHL and concentrations of organochlorine compounds in carpet dust. Analyses were also conducted for NHL subtypes. Carpet dust samples were collected from the homes of 603 cases with newly diagnosed NHL (Iowa, Los Angeles County, Detroit, and Seattle) and 443 controls. *p,p'*-DDT and *p,p'*-DDE concentrations in dust were divided into tertiles.

  The authors reported that the presence of *p,p'*-DDE in carpeting was associated with increased NHL risk (OR 1.3, 95% CI=1.0-1.7) when examined by tertile. The risk was statistically significant only at the highest tertile (OR 1.6, 95% CI=1.1-2.2). A significant positive trend was reported for *p,p'*-DDE and NHL risk. The association was only significant among men versus women (OR 1.6, 95% CI=1.1-2.3 and OR 1.1, 95% CI=0.7-1.5, respectively), and the response was not monotonic. There was no significant trend or association between NHL risk and *p,p'*-DDT. Factors associated with higher levels of *p,p'*-DDE and *p,p* -DDT included homes built before 1960, oriental rugs, and education level.

- Lee, et al., 2004: This case-control study is a pooled analysis of two NHL population-based case-control studies of farmers in Iowa, Minnesota, and Nebraska (Zahm, et al., 1990; Cantor, et al., 1992). The study evaluated the potential association between pesticide use, asthma, and NHL. The population included 872 cases and 2,381 controls. Specifically for DDT, there was no significant NHL risk for either asthmatic farmers (OR 1.2, 95% CI=0.6-2.4) or non-asthmatic farmers (OR 1.2, 95% CI=0.9-1.5) who reported ever using DDT.

- Hardell, et al., 2002: This study examined the potential association between exposure to a wide array of pesticides and NHL and hairy cell leukemia (HCL), which is a rare type of NHL. Two case-control studies were pooled: one NHL and one HCL. The total population included 515 cases and 1141 controls. Associations between NHL and HCL and exposure to herbicides, insecticides (mostly DDT), fungicides, impregnating agents, and organic solvents were evaluated. Exposure was assessed by questionnaire. The OR for DDT use was 1.27 (95% CI=0.92-1.73). Time span for last exposure and NHL diagnosis did not differ between early or latter follow-up periods.

- Hardell, et al., 1999: This case-control study examined the potential association between NHL and exposure to pesticides. The study population included 404 male cases and 741 controls, diagnosed between 1987-1990. Exposures were assessed by questionnaire supplemented with telephone interviews regarding use of herbicides, insecticides (mostly DDT), fungicides, and impregnating agents. There was no statistically significant increased NHL risk associated with 66 cases reporting DDT use (OR 1.1, 95% CI=0.7-1.7).

- Hardell, et al. 1994: This case-control study involving 105 male cases and 335 controls examined the relationship between specific pesticides, solvents, asbestos, smoking and oral snuff and NHL. Using univariate analysis, NHL risk was increased for DDT use (OR 2.4, 95% CI=1.2-4.9), but this relationship became insignificant with multivariate analysis (OR 1.5, 95% CI=0.6-3.6). The authors reported no significant relationship for DDT.

- McDuffie, et al., 2001: This case-control study examined the potential relationship between specific pesticides and NHL in 517 male cases and 1506 controls from six Canadian provinces. Exposures were assessed by questionnaire, and persons reporting more than 10 hours of pesticide use per year, as well as 15% of controls, received telephone interviews. Based on 32 cases and 59

controls, DDT exposure was associated with an increased adjusted OR of 1.73 (95% CI=1.08-2.76). After controlling for other pesticides and study covariates (e.g., medical history), DDT was found "not to contribute significantly to the risk of NHL." The authors identified several study concerns including poor study response rates (67% cases, 48% controls), differential reporting of exposures among cases, and possibility of chance for statistically significant associations in studies involving multiple comparisons.

- Miligi, et al., 2003: This case-control study examined the relationship between pesticide exposure and NHL as well as solvents and leukemia. The study population consisted of 1,145 NHL cases who were diagnosed during the period of 1990-1993. The study population came from 11 different geographic areas within Italy, which included nine high or mixed-use agricultural areas. 1,232 controls were randomly selected from the general population and matched by sex and 5-year age group. Data on exposure and lifestyle factors were obtained by interview. An agriculture-specific questionnaire was utilized and compared to a pesticide crop exposure matrix developed by agronomists. Aggregate data were presented for both NHL and chronic lymphocytic leukemia (CLL). Multivariate analyses considered adjustment for factors other than age and area, including certain professions. For DDT, the OR for NHL/CLL in males was 0.6 (95% CI=0.3-1.1) and 0.3 (95% CI=0.1-0.8) in females.

- Nanni, et al., 1996: This case-control study examined occurrence of NHL and chronic lymphocytic leukemia in association with farming or animal breeding workers from Forli in Northeastern Italy. CLL was included in the study population because the authors reported a high degree of misclassification between CLL and low grade NHL. The study population included 187 newly diagnosed cases between 1987-1990. Controls (n=977) were selected from the general population and were matched by sex and five-year age group. Exposure data were obtained by interview. A pesticide crop exposure matrix was

developed based on expert input, which linked pesticide use with different crops/plant diseases. Cases were asked about use of pesticides, but also about crops, which could be cross-referenced with questionnaire response (recall) for pesticide use. Adjustments to OR calculations were made for age and sex, as well as altitude of municipality, family history of hematopoietic malignancy, and prior Herpes zoster infection.

No significant association was found for any pesticide when either recall or a pesticide matrix was used to classify exposure. For DDT, the OR for NHL/CLL was 1.74 (95% CI=0.93-3.27) based on recall, and 1.7 (95% CI=0.91-3.17) based on job matrix.

- Schroeder, et al., 2001: This case-control study examined the association of agricultural risk factors in males with NHL who were positive or negative for t(14;18) translocation. The NHL cases were drawn from a cohort of 780 newly diagnosed NHL cases from the Factors Affecting Rural Men (FARM) study (Cantor, et al., 1992). 1245 controls were also drawn from the FARM study. Archived tumor blocks were obtained from 248 (40%) of NHL cases and 182 (19%) were successfully assayed for this translocation using PCR analysis. Exposure information was obtained by in-person interviews.

  The adjusted odds ratios for DDT and NHL t(14;18)-positive cases versus controls was 1.1 (95% CI=0.6-1.9) and for t(14;18) negative NHL cases the OR was 1.2 (95% CI= 0.8-1.7). This study did not find a significant association between NHL and DDT in either t(14;18) positive or negative cases.

- Baris, et al., 1998: This study pooled results from three prior published case-control studies involving four states: Nebraska, (Zahm, et al., 1990); Iowa and Minnesota (Cantor, et al., 1992); and Kansas (Hoar, et al., 1986). The pooled study population included 933 NHL cases and 2918 controls that were reported in

published studies. The investigators stated that a principal impetus for this study was to pool data from three prior smaller studies to enable a more detailed analysis of DDT than had been previously performed and to assess the impact of confounding from other pesticide exposures. Information regarding exposure was obtained in-person (Iowa and Minnesota) or telephone interviews (Kansas and Nebraska). Factors that were examined included use of particular pesticides, use of protective equipment, use before and after 1965.

The OR for NHL cases (n=161) who reported use of DDT on animals and crops had an overall non-significant OR for NHL of 1.2 (95% CI=1.0-1.6). For farmers not using DDT, the OR was 1.1 (95% CI=0.9-1.4). Farmers from Nebraska, Iowa and Minnesota who used DDT for greater than 15 years had a non-significant OR of 1.5 (95% CI= 0.9-2.3). The risk for farmers in Nebraska who used DDT for more than 5 days a year was reported to be OR 2.1 (95% CI=0.9-4.9). All ORs adjusted for other pesticide use, including organophosphates and phenoxyacetic acid herbicides, were non-significant. ORs for specific NHL subtypes, including follicular lymphoma, were also examined. For farmers who reported using DDT, the OR for follicular lymphoma was 1.3 (95% CI= 0.8-1.9) and for farmers not using DDT, it was 1.2 (95% CI= 0.9-1.7).

Baris and colleagues concluded that the data did not show any strong evidence that DDT is associated with NHL in male farmers. They concluded that their findings were consistent with two recent investigations which showed no association between either serum DDT or adipose DDT levels and NHL (Rothman, et. al., 1997; Hardell, et.al., 1996).

- Zahm, et al., 1993: This case-control study reported results for females, including 119 cases and 471 controls who had lived or worked on a farm. Cases and controls resided on eastern Nebraska farms. The investigators reported that most of the prior studies involving agricultural risk factors and NHL had focused on

men rather than women. Telephone interviews were utilized to obtain information regarding agricultural risk factors.

The authors reported for DDT use an OR of 1.7 (based on 16 cases and 36 controls), did not provide any CIs, but did state that the OR was not significant. No significant increased risk was observed for any individual insecticide. The authors concluded that this study suggests that NHL risk from pesticides among women who live or work on farms "if real," is smaller than for men.

- Cantor, et al., 1992: This case-control study included 622 newly diagnosed NHL cases in white males drawn from an Iowa state registry and from a surveillance program involving Minnesota hospital records and 1245 population controls. Interviews were used to collect information regarding farming occupations, farming activities including work with crops and livestock. ORs were adjusted for several known and suspected risk factors, including hair dye use. ORs were also calculated for specific NHL subtypes, including follicular lymphoma.

  The authors calculated ORs by pesticide class, individual pesticides, use prior to 1965, and "*ever*" handling, mixing, or applying pesticides, use on crops, animals, and use of protective equipment. Risk for handling DDT used as an animal insecticide was OR 1.2 (95% CI= 0.9-1.7), and use on animals prior to 1965 was OR 1.3 (95% CI=0.9-1.8). Risk for handling with protective equipment for animals was 1.2 (95% CI=0.9-1.7) and without protective equipment OR 1.3 (95% CI=0.9-1.8). Risk for DDT use on crops was OR 1.7 (95% CI=1.2-2.6) and use before 1965 was OR 1.8 (95% CI=1.1-2.7). Risk for handling with protective equipment for crops was OR 1.7 (95% CI=1.2-2.6) and without protective equipment OR 2.0 (95% CI=1.3-3.1). No significant association was found for follicular lymphoma.

The authors concluded that interpretation of these study results regarding any individual pesticide was problematic because multiple exposures occur in modern agriculture and the chance occurrence of positive findings, when multiple comparisons are performed. Further epidemiological investigation was recommended.

- Cantor, et al., 1993: Cantor and coauthors wrote a letter to the editor of the Journal of Cancer Research regarding the above-referenced 1992 study. The investigators stated that their 1992 publication did not include information concerning number of days of pesticide use among cases and controls since this information was not collected in subject interviews. However, the investigators collected additional data on pesticide use on a subset of this cohort (i.e., 110 cases and 211 controls) drawn from the Iowa population several years later.

  When days of exposure (1-4 days, 5-9 days, and 10+ days) were analyzed for this subgroup, the OR for DDT and NHL was not statistically significant for use on either animals or crops. Since interviews were collected several years after the interview and by telephone, the authors reported concerns about the validity of this more recent information and felt it only provided very weak evidence in assessing possibility of causal association.

- Woods, et al., 1987: This is a population-based case-control study of 576 NHL cases and 694 randomly selected controls. The study was largely focused on evaluating the potential association between NHL and soft tissue sarcoma with occupational exposure to phenoxyacetic acid herbicides and chlorinated phenols. Exposure information was obtained by personal interviews.

  The investigators reported an increased OR for occupational exposure to DDT (OR 1.82, 95% CI=1.04-3.2). However, only 4% of the study population reported DDT as an occupational risk factor, so the OR is based upon very small numbers.

Furthermore, although detailed questions were asked regarding phenoxyacetic acid herbicides and chlorinated phenols, data collected regarding DDT appears much more limited. This is a small study with a weak increased risk.

- Woods and Polissar, 1989: This study examines a subset of subjects who were farmers from the Woods, et al., 1987 study. The weak association previously observed was no longer statistically significant—OR 1.68 (95% CI=0.9-3.3).

- Persson, et al., 1999: This pooled case-control study population consists of 199 NHL cases and 479 controls drawn from two prior case-control studies that examined the potential association between occupation and pesticide use with NHL (Persson, et al., 1989; Persson, et al., 1993). Questionnaire data were used to obtain exposure information. For DDT, the OR was 1.4 (95% CI=0.3-5.9) based upon the presence of 4 cases and 6 controls that reported DDT use.

- Persson, et al., 1993: This case-control consisted of 93 NHL cases and 31 Hodgkin's Disease (HD) cases drawn from a regional Swedish cancer registry. 204 controls were identified as being randomly selected from a population registry used for other studies. The authors investigated the association between NHL and HD with various occupations and exposures. Exposure information was obtained by a mailed questionnaire. Crude odds ratios with a 90% CI were calculated for various occupational exposures, such as solvents, pesticides, welding, etc. For DDT exposure and NHL, the OR was not statistically significant (OR 2.0, 90% CI=0.3-13), but this was based only upon 4 cases.

- Persson, et al., 1989: This case-control study included 106 NHL cases, 54 Hodgkin's Disease cases, and 275 controls drawn from a regional Swedish cancer registry. Exposure data was obtained by questionnaire. Crude odds ratios with a 90% CI were calculated for various occupational exposures, such as solvents,

pesticides, metal fumes, etc. No increased risk of NHL was reported for DDT, and no cases reported DDT use.

6.4 Ecological studies

Ecological studies examine populations to assess hypotheses regarding associations between factors in the population and health outcomes. The analysis, however, is not assessed on an individual level but rather the aggregate population, i.e. group rather than individual information are evaluated for associations.

- Cocco, et al., 2000: This study correlated the presence of state-specific adipose tissue levels collected in 1968 with age-adjusted mortality rates for 1975-1994 for NHL and other cancers. The adipose tissue samples were tested as part of the EPA-sponsored human biomonitoring program. For this analysis, adipose tissue measurements represented samples collected in 22 states. Age adjusted NHL mortality during 1975 and 1994 was not associated with DDT. In fact, among whites, NHL mortality significantly decreased with increasing adipose DDE levels.

- Pavuk, et al., 2004: This study consisted of an initial population-based cross-sectional study that measured serum levels of PCBs and organochlorine pesticides, including *p,p'*-DDT and *p,p'*-DDE, in residents of two districts in eastern Slovakia. This was followed by an ecological study that compared cancer incidence in these two districts for the period 1985-1994. One of these districts (Michalovce) had extensive environmental contamination from a former PCB production site, and the other had low background levels (Svidnik) and was used for comparison. Cancer incidence rates were compared to age-specific cancer incidence rates of the Eastern Slovakia population and standardized incidence ratios (SIR) were calculated.

The age-adjusted geometric means for the sum of 15 PCB congeners and *p,p'*-DDT levels were significantly higher in males and females of Michalovce compared to Svidnik. Levels of *p,p'*-DDE were significantly higher in females of Michalovce compared to those of Svidnik. Although levels of *p,p'*-DDE were higher in males of Michalovce as well, this difference was not statistically significant (p=0.05). Cancer incidence rates for NHL were not significantly increased in males or females of Michalovce (males: SIR 1.12 [95% CI=0.80-1.52]; females: 1.04 [95% CI=0.70-1.49]).

6.5 Overall summary of epidemiological studies

Multiple epidemiological studies have examined the potential association between DDT and/or its metabolites and NHL. DDT is considered to be one of the most investigated pesticides with respect to NHL occurrence (Engel, et al., 2007b). Based upon my review and analysis of all the relevant epidemiological literature regarding DDT and NHL, it is my opinion, to a reasonable degree of medical certainty, that DDT is not a cause of, or contributing factor to, NHL. These studies do not demonstrate a significant or strong association between DDT exposure and NHL. Most studies do not reveal a significant association, or demonstrate substantially diminished associations when adjustments are made for other chemical exposures (Engel, et al., 2007b).

Occupational cohort studies have not demonstrated increased NHL mortality and/or incidence even in heavily exposed workers. A strength of these studies is that they involve intense DDT exposure hundreds of times greater than the general population including the alleged intake for Ms. Garza, the plaintiff at issue in this case.

Case-control studies that have utilized DDT biomarkers have not demonstrated overall significant associations with NHL. Two published studies which measured DDT biomarkers before NHL diagnosis showed no overall significant association with NHL risk (Rothman, et al., 1997; Engel, et al., 2007a). A particular strength of these studies is

that DDT biomarkers were measured well before NHL diagnosis and thus the biomarker results would not be biased by the disease itself. Seven other studies involved collection of blood and/or adipose tissue samples after NHL diagnosis and thus could have been biased since NHL could preferentially affect DDT levels in cases. Nonetheless these biomarker studies overall revealed no or weak associations that in most cases were substantially diminished when adjusted for other factors, including other pesticides (De Roos, et al., 2005; Quintana, et al., 2004; Hardell, et al., 2001; Hardell, et al., 1996; Hardell, et al., 2009; Spinelli, et al., 2007; Cocco, et al., 2008). Similarly, non-biomarker case-control studies reveal no or weak findings, and demonstrate the importance of adjusting for confounding factors, including potentially confounding exposures (e.g., Baris, et al., 1998; McDuffie, et al., 2001; Rothman, et al., 1997; Cantor, et al., 1992; De Roos, et al., 2003).

Based on a search for all pertinent studies and my assessment of all epidemiological studies that have examined the potential association between DDT and NHL, and specifically, follicular lymphoma where available, it is my opinion, to a reasonable degree of medical certainty, that DDT exposure is not an established cause or a contributing factor to the occurrence of NHL.

6.6 Experimental Studies

DDT was administered to human volunteers in two studies (Hayes, et al., 1956; Hayes, et al., 1971) to ascertain whether administration of DDT at levels hundreds of times greater than background, but still considered safe, would result in clinical effects. The studies provide direct information regarding health effects of DDT in humans at levels substantially greater than the "theoretical" intake that the plaintiff's experts have assumed Ms. Garza received while residing at "506 Nicholson." The two studies are described below:

- Hayes, et al., 1956: Fifty-one male volunteers from a correctional facility received daily doses of either recrystalized *p,p'*-DDT or technical grade DDT at doses of 0 (placebo), 3,500 ug/day, or 35,000 ug/day for periods of up to 18 months. Medical examinations were performed to determine whether any participants were experiencing DDT-related adverse effects, with emphasis on neurological and liver function tests. A total of 44 volunteers completed 1 month of dosage, among which 14 also completed 12 months of dosage, and 5 completed 18 months of dosage. A total of 35 volunteers completed the final medical examination. The investigators concluded that no volunteers had symptoms or test results that showed any sign of injury related to DDT exposure.

- Hayes, et al., 1971: Twenty-four volunteers received recrystalized *p,p'*-DDT or technical grade DDT at concentrations of either 3,500 ug/day or 35,000 ug/day for 21.5 months and were observed for an additional 25. 5 months. Sixteen of the 24 were followed for 5 years. Medical examinations were conducted with emphasis on neurological tests and liver function tests. The investigators reported no symptoms or test results that showed any sign of injury related to DDT intake.

6.7 Assessment by Public Health and Government Agencies

Several public health and governmental agencies have evaluated scientific evidence concerning whether DDT has been recognized to cause cancer in humans. These agencies have determined that DDT is not a recognized cause of human cancer. These assessments include the following:

- The Agency for Toxic Substances and Disease Registry (ATSDR) of the U.S. Department of Health and Human Services. ATSDR concluded that there was insufficient evidence that DDT was a human carcinogen (ATSDR, 2002).

- The U.S. EPA determined that there was insufficient evidence that DDT was a human carcinogen (U.S. EPA, 1991). In 2002, EPA conducted a screening-level literature review of recent toxicology literature relating to its cancer assessment for DDT (U.S. EPA, 2002). EPA did not change its classification for DDT based on this most recent review.

- The National Toxicology Program of the National Cancer Institute reviewed the literature pertaining to DDT and also concluded that there was inadequate evidence of human carcinogenicity (NTP, 2005).

- The International Agency for Research on Cancer of the World Health Organization concluded that there was insufficient evidence that DDT caused cancer in humans (IARC, 1991).

The relevant public health agencies have not concluded that DDT and/or its metabolites cause human cancer.

7.0 Ecological data on NHL rates and DDT use in the U.S. and other countries do not support the conclusion that DDT causes NHL

Ecologic studies are a type of epidemiological investigation that examines the prevalence of disease in a group rather than in an individual for the purpose of exploring potential relationships between risk factors and disease outcome. Data regarding disease occurrence in a general population with a specific risk factor can be compared to other populations without the risk factor. Thus, ecological studies can provide insight into determining potential causal factors for diseases among populations. For example, it is well known that chronic infection with hepatitis B virus (HBV) is causally associated with primary liver cancer. Therefore, it is not surprising that geographic areas with a high prevalence of this virus, such as Southeast Asia, also tend to have high prevalence of liver cancer in these populations (Cancer Research UK, 2006). Ecological data must be

viewed cautiously since individual data on whether a particular person in the at-risk population who developed the disease was exposed to the risk factor are missing. However, aggregate population data can be used to assess causal hypotheses.

In a similar vein, I compared rates of NHL in countries that no longer use DDT with those of countries that permit its use to see if there is any apparent relationship between DDT use and NHL. A number of factors may affect NHL rates, but countries in which DDT was used continuously appear to have lower NHL rates than countries in which it has not been used for many years. One interesting example—Mexico, where DDT was used until 2000, has an age-standardized incidence rate (ASIR) for males of 5.7 per 100,000, while the United States, which stopped using DDT in late 1972, has an ASIR for males of 17.1 per 100,000. In Mozambique, where DDT was not used, the ASIR for males is 8.6 per 100,000, while in South Africa and Swaziland that are next to Mozambique and which still use DDT, the ASIRs for males are 5.1 per 100,000 and 3.0 per 100,000, respectively[2]. Moreover, in general, the rates of NHL have been increasing in developed societies even though use of DDT has been decreasing. The ASIR for NHL in males in more developed regions is 10.4 per 100,000 and for less developed regions is 4.3 per 100,000 (Ferlay, et al., 2004). In addition, developed countries have a higher rate of NHL than developing countries even though some developing countries still use DDT (Figure 1). Thus, this data suggests the absence of a relationship between DDT exposure and NHL.

While there are limitations to this approach, and one cannot use these data to prove or disprove whether a causal association may exist, one would expect that if there were a strong association between DDT and NHL, it would be apparent in this type of comparison, similar to the example of HBV and liver cancer. Nevertheless, alternative explanations cannot be ruled out, such as incomplete reporting of NHL by the registries. For this reason, epidemiologists and other scientists rely on "analytical" epidemiological

[2] The ASIRs for Mexico, United States, Mozambique, South Africa, and Swaziland are estimates for the year 2002, based on the most recent incidence, survival, and mortality data collected about 2-5 years earlier (Ferlay, et al., 2004).

studies such as case-control and cohort studies to evaluate causal hypotheses. However, in proper context, ecological studies can be viewed together with data from other types of studies to determine whether a coherent and consistent pattern of study results is evident. In this case, ecological, cohort and case-control study data together do not support a causal association between DDT exposure and NHL.

8.0 Texas Department of Health investigations show no increased cancer rates in Mission, Texas and no increased serum DDT levels in persons living and/or working in close proximity to Hayes-Sammons

Coupled with the epidemiological data in the scientific literature, we also have data concerning the cancer rates in this specific Mission, Texas community and data on their potential exposures analyzed from blood samples. This unique data represent specific health information about the community and therefore are useful in addressing health concerns or speculations that exist regarding the claimed impact of operations at the former Hayes-Sammons facilities.

As discussed below in further detail, the Texas Department of Health (TDH) examined whether excess cancer was present in the Mission, Texas community and evaluated serum levels of persons residing or working in close proximity to Hayes-Sammons. The TDH blood data is especially relevant because future health risks, if any, are dependent upon past exposure, and blood levels serve as a marker of exposure. Conclusions of these two analyses show both no excess cancer and no increased serum DDT levels in Mission, Texas. This is further evidence of a lack of an association between DDT exposure and health effects in this community from the Hayes-Sammons site.

8.1 TDH investigations of persons living in the Mission, Texas zip codes do not reveal excess cancer incidence or mortality

TDH conducted a series of investigations to ascertain whether or not persons residing in Mission, Texas zip codes have an increased rate of, or death from non-Hodgkin lymphoma or any other cancers. TDH examined incidence rates for the period 1995 to 2001 and mortality rates from 1993 to 2002 and concluded there was no increased incidence or mortality from non-Hodgkin lymphoma (Texas Department of Health, Cancer Registry Division 2005). Given their findings, TDH did not recommend further study.

8.2 Persons residing around the Hayes-Sammons mixing facility did not have excessive serum DDT levels

In addition to the TDH investigations of cancer experience, TDH also conducted a study to ascertain levels of certain pesticides, including DDT, in persons residing around the Hayes-Sammons mixing facility (Texas Department of Health, 1981). Blood samples were collected from both adults and children in 1980. The population categorized as "exposed" consisted of neighborhood residents, shop teachers, bus drivers, and mechanics working at the Mission Independent School District (MISD) facility adjacent to the Hayes-Sammons mixing facility. This "exposed" group included persons living across the street from the Hayes-Sammons mixing facility on the same block where Ms. Garza indicates she resided. A control population removed from the facility was selected for comparison purposes. After analyzing serum DDT and other pesticide levels from both groups, TDH concluded that "the analysis failed to show any consistent pattern of elevation of serum pesticide levels in Exposed individuals above those seen in Controls."

Therefore, although the "exposed" population had potential contact with pesticides from the Hayes-Sammons mixing facility, they did not have any demonstrable significant elevations of DDT in serum.

The finding that persons residing around the site do not have a consistent pattern of elevated DDT is not unexpected. Investigations also have not shown a correlation between soil DDT and serum DDT levels. A study in Mexico demonstrated that, in a population residing in houses with dirt floors and wooden walls, there was a poor correlation between DDT levels in outdoor soil, indoor soil, or house dust and measured blood DDT levels (Herrera-Portugal, et al., 2005). In this 2005 study, total DDT levels in outdoor soils ranged from 0.35 to 11.74 ppm, with an average level of 4.76 ppm. The indoor soil concentrations ranged from 2.08 to 68.3 ppm with an average level of 21.92 ppm. Indoor dust levels ranged from 2.66 to 108.48 ppm with average concentrations of 30.84 ppm.[3] Despite these DDT concentrations, there was no correlation between soil or house dust measurements and blood DDT concentrations in children ranging from ages 6 to 12.

In sum, based on the TDH data, the Mission, Texas community, which includes Ms. Garza, has not experienced an excess rate of any type of cancer, including non-Hodgkin lymphoma. Moreover, persons residing and/or working near the Hayes-Sammons mixing facility, where Ms. Garza reportedly lived, did not have serum DDT levels that differed substantially from a control population, suggesting that living and/or working near the facility did not result in DDT exposure sufficient to affect serum levels.

9.0 Overview of Dr. Sawyer's main opinions regarding Ms. Garza

The following represent Dr. Sawyer's main opinions concerning the cause of Ms. Garza's NHL:

- DDT is recognized to cause NHL;
- Epidemiological studies serve as a surrogate measure of Ms. Garza's DDT dose;

[3] The average soil concentration of 4.76 ppm total DDT in this 2005 study is similar to outdoor total soil DDT concentrations utilized by Dr. Strauss.

- Risk assessment is an appropriate methodology for determining causation of Ms. Garza's NHL;
- Dr. Strauss's generic cancer risk estimates for DDT can be compared with background NHL rates for the purpose of determining the cause of Ms. Garza's NHL.

10.0 Assessment of Dr. Sawyer's main opinions and methodology utilized to conclude that Ms. Garza's NHL was caused by exposure to DDT

10.1 Dr. Sawyer's claim that DDT causes NHL is not substantiated by the epidemiological literature

Dr. Sawyer has made broad, sweeping, unsubstantiated claims with respect to the findings of epidemiological studies that have examined DDT and NHL. He claimed in his initial (2005) report that DDT causes NHL and that NHL is a "common toxicological end point" for DDT and other organochlorines. At that time, he did not provide any scientific citations to support this claim. In his December 9, 2008 report, Dr. Sawyer listed fifteen epidemiological studies that he claimed were relevant to the issue of organochlorines and NHL causation. Finally, in his January 15 and February 4, 2009 deposition, he identified five epidemiological studies that he claims are sufficient to support his opinion that DDT causes NHL.

The five studies that Dr. Sawyer indicates are sufficient for demonstrating that DDT exposure causes NHL are overviewed in Section 6 of this report and are discussed in more detail below.

- Quintana, et al., 2004: Dr. Sawyer's analysis of the Quintana study is flawed and he misunderstands and/or misinterprets several critical aspects of this study, including: (1) study design, (2) potential effects of NHL and NHL treatment on

study findings, and (3) study findings, including the authors' conclusions regarding the study significance.

Dr. Sawyer appears to be confused about the study design. He provides contradictory testimony regarding a key study design parameter: the type and timing of collection of tissue samples. Dr. Sawyer testifies the study involved analysis of blood samples for organochlorines from persons who had blood historically archived (deposition p. 104). Dr. Sawyer is incorrect since this study did not involve blood testing. However, at other times, Dr. Sawyer acknowledges that adipose or fat samples were analyzed.

Dr. Sawyer also provides contradictory testimony regarding the sample collection protocol. The timing of adipose tissue collection is a critical study parameter since NHL itself may affect tissue DDT levels (Quintana, et al., 2004). If DDT levels are affected by NHL, then spurious associations could occur in a case-control study that compares adipose DDT levels in NHL cases to controls. At one point, Dr. Sawyer acknowledges that adipose tissue samples were obtained at autopsy, but then contradicts this testimony and indicates that the samples were "historically collected; that is, if a person had subsequently lost or gained weight, or breast fed, the samples were already in the freezer" (deposition pp. 241-242). Thus, in his mind, the NHL cases had tissue samples collected before their cancer had a significant impact on their body weight. In actuality, most of the NHL cases were dead at the time of sample collection since they represent autopsy specimens.

Dr. Sawyer also misunderstands the influence that NHL and NHL treatment could have on DDT adipose tissue levels and consequently fails to recognize a significant study limitation which even the authors acknowledge. Dr. Sawyer testifies that organochlorine levels would be lower rather than higher in NHL cases and thus the study results could only be more significant since the NHL

cases would have been expected to have higher organochlorine levels before the study samples were taken. (deposition p. 106) Dr. Sawyer testifies that some persons with NHL would have undergone chemotherapy which could only decrease organochlorine levels. Dr. Sawyer further testifies that no other effect would be expected and indeed "there is no pharmacologic or toxicologic mechanism plausible" for persons to have increased blood level of organochlorines from chemotherapy (deposition p. 109).

The basis for Dr. Sawyer's conclusion that organochlorine levels could only diminish as a consequence of NHL appears to be based upon a single study that measured blood organochlorine levels, including DDT, in NHL patients undergoing chemotherapy (Baris, et al., 2000). NHL cases who had undergone chemotherapy had decreased organochlorine levels. However, in the Baris, et al. study, patients had gained weight and had higher lipid content following chemotherapy, i.e., cancer patients can gain weight as their disease improves following chemotherapy. The assumption that the patients in Baris, et al., who gained weight, are comparable to the subjects in Quintana, et al., who died from the disease, is false.

In actuality, persons dying from NHL following unsuccessful chemotherapy would be expected to have weight loss or substantial wasting associated with terminal cancer. The term cachexia is used to describe wasting that occurs in persons with terminal cancer. Bioconcentration of DDT and other organochlorines occurs in cachexic patients (Hoppin, et al., 2000). Weight loss even in persons with no cancer is known to increase adipose tissue concentrations of organochlorines (Pelletier, et al., 2003; Backman, et al., 1978; Chevrier, et al., 2000). Thus, Quintana and colleagues indicate that NHL-associated cachexia could affect adipose organochlorine levels and even though they tried to exclude such cases, they could not ascertain whether such efforts were effective. The authors also acknowledge that clinical information from the NHL cases was

lacking, including data on weight loss or the presence of cachexia. Thus, Dr. Sawyer fails to understand the generally recognized limitations of the study.

Dr. Sawyer was unaware that the adipose tissues used for the Quintana, et al. study were collected as part of the U.S. EPA National Human Adipose Tissue Survey (NHATS) program, which was severely criticized by the National Research Council (NRC), one of the nation's most respected scientific organizations (NRC, 1991). The NRC raised many concerns about the program, including whether the collected samples were representative of the U.S. population. Significant concerns were raised about the integrity of data concerning donors, i.e., persons from whom samples were collected, and whether they actually had the conditions reported, i.e., some samples may not have come from persons with accidental deaths. The integrity of stored tissue samples also was severely questioned, including the possibility of contamination during storage and inadequate storage temperatures. The NRC concluded that the stored adipose tissue samples would have little or no value.

Dr. Sawyer also reaches conclusions regarding the study's findings, especially with respect to establishing causation, that are not supported by the investigators. Although Dr. Sawyer testified that Quintana, et al. was one of five studies that were sufficient to establish general causation between DDT and NHL, Quintana and colleagues concluded as follows: "We found no clear association between exposure to DDT and NHL. We report associations with *p,p'*-DDE that were confounded by heptachlor epoxide." Based on this conclusion, this study does not provide evidence of a causal association between DDT or its metabolites and NHL.

Dr. Sawyer's failure to understand (1) the potential impact of NHL on adipose organochlorine levels, (2) the many weaknesses of the Quintana, et al. study, and (3) the findings of the study generally undermines his adoption of this study as

one of the support pillars for his general causation conclusion that DDT causes NHL.

- Colt, et al., 2005: Dr. Sawyer indicates that the Colt, et al. study is important for proving causation since it demonstrates a significant dose-response trend (deposition p. 77). Dr. Sawyer also testifies that this study can be used to assess dose. Although Dr. Sawyer considers this study as being very significant to proving causation, the authors did not reach such a sweeping conclusion. The authors only conclude that the data provide "some" evidence that carpet dust *p,p'*-DDE can contribute to NHL risk, but that further research is needed to evaluate the significance of carpet dust DDE levels. In contrast to Dr. Sawyer's assertion that the study is useful to determine the dose, the authors indicate that carpet dust levels are only a "crude" indicator of historic organochlorine exposure (i.e., not dose) and cannot be used to determine the DDT source or when such compounds entered the carpet. It also is unknown if DDT was added to the carpet when it was manufactured. The limitations of using carpet contaminant levels to determine exposure potential and health risk are well recognized (Lioy, et al., 2002). The authors further acknowledge that the main source of DDT in NHL cases of this study would have originated from the diet rather than carpet dust. Thus, the Colt, et al. study does not determine a DDT dose nor any level of DDT intake for the NHL cases and thus cannot serve to provide a meaningful comparison to any DDT studies where dose is measured (e.g., feeding studies) or to theoretical dose estimates predicted by Dr. Strauss.

  The limitations of using *p,p'*-DDE carpet dust for assessing NHL risk is highlighted by the finding from De Roos, et al., 2005. This study measured blood *p,p'*-DDE levels in NHL cases also included in the Colt, et al. study and found no correlation between *p,p'*-DDE or *p,p'*-DDT and NHL.

- Baris, et al., 1998: Dr. Sawyer indicates that this is a key study for demonstrating causation between DDT and NHL. However, based on their findings, the authors concluded that there was no strong evidence that DDT was associated with NHL. Moreover, the authors reported that their study findings were consistent with two recent studies which found no association between DDT exposure and NHL risk (Rothman, et al., 1997 and Hardell, et al., 1996). Dr. Sawyer did not cite these two studies in any of his reports as being relevant for his analysis.

- Cantor, et al., 1992: Dr. Sawyer indicates that this study was also key in demonstrating causation between DDT and NHL. This study involved making multiple comparisons between numerous pesticides and NHL, which led the authors to caution readers that drawing conclusions about any single pesticide was "fraught with difficulties" because of the likelihood of a chance occurrence of a positive finding as a result of conducting multiple comparisons. Thus, the authors concluded that this study lacks specificity with respect to making causal determinations between individual pesticides such as DDT and NHL. In contrast, Dr. Sawyer utilizes this data to make a causation conclusion about a specific agent, DDT.

  Dr. Sawyer also identified the Cantor, et al. study as being useful for demonstrating a significant dose response between DDT and NHL (deposition p. 77). This study, however, did not determine a dose or demonstrate a dose response relationship. The study did not present any dose data or even data concerning the years or days of pesticide use which might theoretically be used to formulate a theoretical dose estimate. With respect to duration of exposure, a crucial element of any dose response determination, the authors only reported pesticide use into two groups: before and after 1965. Thus, the study does not evaluate any dose response relationships. Moreover, Dr. Sawyer was not aware that subsequently Cantor and colleagues published data on a subset of these NHL cases where they reported data concerning days of pesticide use and that a non-

significant relationship was found between days of DDT use and NHL (Cantor, et al., 1993).

- Spinelli, et al., 2007: Dr. Sawyer identifies this as a key study for demonstrating a causal association between DDT and NHL. This study evaluated the potential association between plasma levels of *p,p'*-DDE or *p,p'*-DDT and NHL. Dr. Sawyer failed to note that although the study demonstrated a significant trend between plasma *p,p'*-DDE and NHL risk, but the trend diminished substantially after adjustment for oxychlordane, and the authors found no statistically significant association between quartiles of DDT/DDE exposure and NHL or follicular lymphoma. Thus, the findings do not support Dr. Sawyer's conclusions.

Dr. Sawyer's claim that the epidemiological literature shows that DDT causes NHL is not substantiated. Dr. Sawyer misinterprets and/or utilizes isolated study segments to support the claim of a causal association, even when the scientists conducting the study recognize their findings have limitations with respect to determining causation or provide no evidence of an association. Dr. Sawyer did not conduct a comprehensive analysis of the scientific literature between DDT exposure and NHL and mainly cited a few studies that he claims support his position. Dr. Sawyer was not even aware that the Cantor, et al., 1992 authors published additional data that was not consistent with such a relationship (Cantor, et al., 1993). Although Dr. Sawyer indicated that he conducted a weight of the evidence analysis of the scientific literature and used the Bradford-Hill criteria, his report and testimony do not show this type of analysis or assessment.

10.2 Dr. Sawyer's use of the human health risk assessment process for making individual disease determinations lacks scientific validity

Dr. Sawyer utilizes the human health risk assessment process to opine that Ms. Garza's potential exposure to DDT was sufficient to produce or significantly contribute to the

production of her non-Hodgkin lymphoma. He utilizes theoretical "cancer risk" data derived by Dr. Strauss for this assessment.

Human health risk assessment was defined by the National Academy of Sciences/National Research Council (NAS/NRC) in the milestone document entitled "Risk Assessment in the Federal Government: Managing the Process," commonly referred to as the "Red Book" (National Research Council, 1983). The risk assessment process is aimed at the protection of public health before the occurrence of a disease. It is an analytical tool used by health and regulatory agencies to establish standards or guidelines to protect public health and establish cleanup levels. The risk assessment process incorporates many conservative assumptions and errs on the side of health protection. It includes the calculation of theoretical cancer risks that are used to prioritize and allocate public health resources. Given the purpose of the risk assessment process and specifically the reasons outlined below, risk assessment is not a tool that is used for making individual disease determinations.

10.2.1 Dr. Sawyer's use of risk assessment is inconsistent with U.S. EPA guidance

As indicated above, Dr. Sawyer relies upon Dr. Strauss's theoretical cancer risk estimates derived from the risk assessment process. In turn, this process utilizes a U.S. EPA cancer slope factor for DDT developed by the Integrated Risk Information System (IRIS), to calculate this theoretical "cancer risk" for Ms. Garza. However, the EPA cautions against using the slope factor for making specific disease determinations such as those made by Dr. Sawyer for Ms. Garza.

Specifically, U.S. EPA states as follows:

> "In general IRIS values (e.g., cancer slope factors) cannot be validly used to accurately predict the incidence of disease or the types of effects that chemical

> exposures have on humans. This is due to the numerous uncertainties involved in risk assessment, including those associated with extrapolation from animal data to humans, and from high experimental doses to lower environmental exposure. The organs affected and type of adverse effect resulting from chemical exposure may differ between the study animals and humans. In addition, many factors besides exposures to a chemical influence the occurrence and extent of human disease." (U.S. EPA IRIS, 2009)

Despite this U.S. EPA guidance, Dr. Sawyer uses "cancer risk" estimates developed by Dr. Strauss to determine that Ms. Garza developed NHL. His methodology violates the accepted use of cancer "slope factor," and as a result, his use of the risk assessment process, which is based upon the "slope factor," is not scientifically valid.

10.2.2 Theoretical "cancer risks" are extrapolated from animal data, and therefore do not predict the cause of NHL in an individual

Dr. Sawyer uses Dr. Strauss's DDT "cancer risk" as being equivalent to Ms. Garza's NHL risk for purposes of determining the cause of her NHL. However, Dr. Strauss's theoretical "cancer risk" is not based on human data but on data derived from rodent feeding studies. Extrapolating the effects from such studies to lower exposure levels in humans is a process that involves considerable uncertainty (U.S. EPA, 2005a). Differences in uptake rate, species metabolic variability, biologic half-life and potential metabolites, target tissue susceptibility, and species differences in ability to repair DNA can contribute to differences in severity or type of effects across species.

Because many regulated chemicals lack sufficient study in human populations, in the regulatory context, the use of high dose animal cancer studies is assumed to predict upper bound human cancer risks at much lower environmental doses. However, the common use of these assumptions and calculations in regulatory risk assessments does not correlate to cause and effect in populations or individuals. Thus, although EPA may

predict theoretical cancer risks for populations based upon carcinogenic responses in animals, this does not establish that cancer in humans would be expected to occur from such exposures.

10.2.3 The U.S. EPA DDT cancer slope factor is based on mouse liver tumors and thus lacks specificity for NHL

The cancer risk estimates used by Dr. Sawyer for Ms. Garza from her alleged DDT exposure are not based upon NHL, but are based upon the occurrence of liver tumors in mice fed high doses of DDT (U.S. EPA IRIS, 2009). There is no scientific evidence that demonstrates that occurrence of liver tumors in mice is related to the risk of NHL in humans. Dr. Sawyer's transference of a risk based on rodent liver tumors to human NHL risk lacks scientific validity since there is no data to support that rodent liver tumors are related to human NHL. To reach a conclusion about NHL risk and/or cause, a person should examine data concerning the specific cancer at issue rather than the most sensitive tumor endpoint identified in rodents.

10.2.4 Human health risk assessments do not focus on a specific disease

The risk assessment process is aimed at the protection of public health before the occurrence of a disease. It predicts a theoretical lifetime cancer risk typically based on cancer occurrence in animals. In reality there is no "generic cancer disease," but rather separate types of cancer, each with individual risk factors and susceptibilities. For example, basal cell skin cancer is related to excessive sunlight exposure and persons with fair skin are at increased risk, while others, such as cancer involving female reproductive organs have other separate risk factors. Therefore, it is inappropriate to apply a theoretical generic cancer risk estimate to a specific cancer like NHL.

10.2.5 The risk assessment process involves a conservative approach employed by regulatory agencies to protect public health and as a result, are expected to overestimate health risk

Risk assessment is an analytical tool that is used by regulatory agencies to establish standards or guidelines to protect public health and often to establish site cleanup levels. The process incorporates many highly conservative assumptions to err on the side of health protection and provide a margin of safety. For example, the process incorporates a default linear extrapolation model that is used for predicting responses from low level exposures when the mode of action of an agent has not been ascertained (U.S. EPA, 2005a). This hypothesizes that there is a "no threshold" assumption for cancer risk, i.e., that some positive cancer risk occurs at any dose above zero, even though there may be no scientific proof for such a premise and the human body has defense mechanisms for protection of carcinogenicity. In addition, the cancer slope factor likely overestimates the risk of a substance in producing cancer, in part because it is based upon the occurrence of cancer in the most sensitive animal species tested for the most sensitive tumor end point. The cancer slope factor also is based upon a 95% upper confidence limit. The use of this 95% upper confidence interval for cancer potency makes it very unlikely that the risk could be higher, and is much more likely to be lower than predicted.

The U.S. EPA recognizes the inherent conservatism incorporated in risk assessment and the slope factor and has indicated the following:

> "Because the slope factor is often an upper 95th percentile confidence limit of the probability of a response based on experimental animal data used in the multistage model, the carcinogenic risk estimate will generally be an upper-bound estimate. This means that EPA is reasonably confident that the "true risk" will not exceed the risk estimate derived through use of this model and is likely to be less than that predicted." (U.S. EPA, 1989, p. 8-6)

Furthermore, the U.S. EPA concludes as follows:

> "It should be emphasized that the linearized multistage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of risk. The true value of risk is unknown, and may be as low as zero." (U.S. EPA, 1986)

Consequently, Dr. Sawyer's use of generic cancer risk data from risk assessment to determine the cause of Ms. Garza's NHL is inappropriate and lacks scientific validity.

10.3 Dr. Sawyer misunderstands the Department of Energy's (DOE) risk assessment guidelines and misapplies the guidelines

Dr. Sawyer takes the position that risk assessment is a valid methodology for determining disease causation and relies upon the DOE guidelines as an example of how risk assessment can be used for making disease determinations (deposition p. 198). The DOE risk assessment methodology for determining individual cancer risk, however, is a fundamentally different process than Dr. Strauss's risk assessment, which Dr. Sawyer relies upon to reach a medical causation determination.

The DOE approach does not validate Dr. Sawyer's use of Dr. Strauss's theoretical "cancer risk" estimates to make a causation determination for Ms. Garza's NHL. Instead, the DOE guidelines describe a risk assessment process for determining if a cancer is related to radiation in workers who manufactured nuclear weapons. The DOE cancer risks are not based upon animal data, but on actual human health cancer data, e.g., Japanese atomic bomb blast survivors. The DOE guidelines also incorporate the use of real exposure data in estimating a worker's cancer risk. As a result, the DOE calculates cancer risks for different organs based upon actual experience of populations exposed to different types of radiation, and thus provides a real world measure of cancer risk. In

contrast, Dr. Strauss's risk estimates are based upon mice liver tumor data, incorporate no actual human cancer risk data and are based on a hypothetical dose or intake, not actual exposure measurements. Moreover, because the DOE guidelines incorporate person, agent (radiation), and organ specific information to determine a specific person's risk, Dr. Sawyer's testimony that the DOE methodology does not calculate risk for individual tumors is false (deposition p. 129).

10.4 Dr. Sawyer's comparison of Dr. Strauss's theoretical "cancer risk" with Ms. Garza's background risk of NHL for causation determination is scientifically inappropriate

To assess whether Ms. Garza's NHL is attributable to DDT, Dr. Sawyer compares Dr. Strauss's generic, theoretical "cancer risk" to Ms. Garza's background NHL risk from SEER data. He testifies that once Dr. Strauss's theoretical cancer risk exceeds the background NHL risk for a woman like Ms. Garza, i.e., 2.3 in 10,000 (deposition p. 86), it is established that these agents caused Ms. Garza's NHL. This premise is false. First, Dr. Sawyer does not have any data on Ms. Garza's actual risk, if any, of developing NHL from DDT. Thus, he cannot conduct a "person to person comparison" (i.e., compare risk of NHL from DDT to background risk). Second, he is comparing theoretical risk of mice developing liver tumors to background risk of NHL in human populations without even demonstrating that the two species (mice and humans) or conditions (mouse liver tumor and human NHL) are similar. Even Dr. Strauss admits that her generic theoretical "cancer risk" estimate does not predict the risk of developing NHL (deposition pp.105-106). For these reasons, there is no scientific basis for Dr. Sawyer's use of this comparison to reach a causation determination of Ms. Garza's NHL.

11.0 Increased "cancer risks" estimated for DDT by Dr. Strauss and used by Dr. Sawyer are insignificant when compared to background cancer risk levels

Assuming Dr. Strauss's theoretical "cancer risk" estimates are valid and relevant to Ms. Garza, which they are not for the reasons discussed previously, they are insignificant when compared to her actual background cancer risk.

To provide a benchmark for interpreting the quantitative significance of Ms. Garza's theoretical DDT-related "cancer risks," they can be compared to the actual cancer risks of a woman of similar age and race in Texas. The actual cancer risk (based on historical occurrence in human populations) of a 55-year-old Hispanic woman (Ms. Garza was age 55 at time of diagnosis) can be obtained from cancer incidence data reported for the State of Texas. Table 1 provides a listing of age-specific probability of cancer risk for Hispanic females derived from the State of Texas cancer registry. At the time of her diagnosis, Ms. Garza's accumulated risk of developing any cancer was 6.5% or 0.065. This background risk is about 650 times greater than Dr. Strauss's theoretical "cancer risk" for DDT based upon mouse liver tumor data (i.e., 0.15% of her background cancer risk). Even with the application of a 3-fold increase in cancer potency for early life exposures, Ms. Garza's actual accumulated background cancer risk was about 216 times greater than Dr. Strauss's theoretical cancer risk (i.e., 0.5% of background cancer risk). Thus, the hypothetical contribution that DDT had with respect to causing Ms. Garza's NHL is insignificant given the magnitude of difference between her actual background and Dr. Strauss's theoretical "cancer risk," i.e., hundreds of times less.

12.0 Predicted DDT intake is less than WHO Acceptable Daily intake (ADI)

Several organizations have developed acceptable daily intake levels of DDT. The World Health Organization has also suggested an Acceptable Daily Intake (ADI) for DDT and its metabolites of 0.01 mg/kg/day. The ADI represents daily intake that is considered to

be safe for a lifetime. Dr. Strauss's predicted daily intakes of DDT/DDE are all less than the WHO acceptable intake, i.e., 0.003 mg/kg during Ms. Garza's period of occupancy in the initial Nicholson residence (assumes 108 µg day intake for her combined DDT, DDE and DDD). Ms. Garza's combined DDT, DDE, and DDD intake during her residence at 423 Canal would be substantially less. The Permissible Exposure Level (PEL) established by the Occupational Safety and Health Administration (OSHA) for DDT is 1 mg/m^3 for an eight-hour work day (29 CFR, 1910.120). Based on the assumption that 10 cubic meters of air is inhaled over an eight-hour work day (EPA 1997), a worker would receive about 1,000 micrograms a day for a lifetime of work. Ms. Garza's combined DDT intakes, assuming they are accurate, are well below these levels. In sum, Dr. Strauss's predicted DDT intakes for Ms. Garza are below WHO and OSHA thresholds.

13.0 Overview of Dr. Strauss's risk assessment and opinions regarding Ms. Garza

- Dr. Strauss's health risk assessment is based upon incomplete environmental data and consequently the predicted theoretical cancer risk estimates are unreliable.
- Dr. Strauss has no objective evidence that chickens would have fed at the MISD location across the lateral irrigation canal.
- The presumed DDT intake, calculated by Dr. Strauss, is similar to daily background intake.
- Dr. Strauss's 3-fold increase of Ms. Garza's "cancer risk" is inconsistent with EPA guidance.

Assuming Dr. Strauss's theoretical "cancer risk" estimates have any relevance for Ms. Garza, which they do not, as previously discussed, Dr. Strauss's risk assessment suffers from several shortcomings.

14.0 Dr. Strauss's health risk assessment is based upon incomplete environmental data and consequently the predicted theoretical cancer risk estimates are unreliable

As discussed in detail in the report of Austin Cooley, P.E., in Guadalupe Garza vs. Allied Chemical Corp., et al., Dr. Strauss, in her initial report, used only five soil samples to estimate Ms. Garza's theoretical "cancer risk." In Dr. Strauss's supplemental report, she expands the number of soil samples she relies upon to only 13, ignoring thousands of soil data points available in or around the Hayes-Sammons mixing facility. See above-referenced Cooley report. Furthermore, based upon Mr. Cooley's report, I understand that the DDT soil concentrations used by Dr. Strauss to determine Ms. Garza's theoretical DDT intake ignore data on or adjoining Ms. Garza's former residence and represent a limited subset of available data relating to Ms. Garza's residence. Consequently, Dr. Strauss's theoretical cancer risk estimates for Ms. Garza are unreliable for the reasons noted below.

One of the key requirements in performing a health risk assessment is to determine the actual concentrations that were present at the site for the relevant time period (U.S. EPA). To that end, a risk assessor must analyze all available environmental data to develop accurate exposure estimates, which are necessary to calculate reliable cancer risk estimates. Reliance on environmental measurements that are not representative of actual historical concentrations can lead to misleading risk estimates. Dr. Strauss's risk assessment relies on a very limited data set that was obtained decades after Ms. Garza's alleged exposure on the 500 block of Nicholson and therefore is based upon non-validated extrapolated data. Consequently, Dr. Strauss's risk assessment does not reliably predict Ms. Garza's DDT intake. Indeed, the majority of all of Ms. Garza's theoretical DDT intake and theoretical DDT "cancer risk" is based upon a single soil sample. As a toxicologist, I find it highly suspect that the majority of Dr. Strauss's calculated chemical intake for Ms. Garza is based on a single soil sample when a multitude of other samples are available yet not included in her analysis. See Mr. Cooley's report. Thus, Dr. Strauss's risk assessment cannot be considered to be valid

since it is based upon limited environmental data and does not incorporate many environmental measurements which characterize actual DDT levels near or around Ms. Garza's prior Nicholson residence.

14.1 Dr. Strauss has no objective evidence that chickens would have fed at the MISD location across the lateral irrigation canal

According to Dr. Strauss, Ms. Garza ate eggs on a daily basis for seven years during her childhood. The eggs were reported to be produced by free-range chickens living near her "506 Nicholson" residence. Dr. Strauss assumes that (1) these chickens ingested soil containing DDT, (2) the chickens ingested soil from around the home and the area across the irrigation canal, and (3) the DDT was transferred from the soil to the chicken and then transferred to the eggs consumed by Ms. Garza. Based on Dr. Strauss's predicted DDT intakes, Ms. Garza's egg consumption represented about 98% of her daily DDT intake for about seven years. Critically, the majority of the DDT received by the chickens and entering the eggs originates from soil the chickens supposedly ingested from the area across the irrigation canal.

Dr. Strauss provides no reliable evidence concerning the anticipated range of chickens or whether these chickens were able to or even likely to cross an irrigation canal. For example, chickens are considered to be animals that would not be expected to swim across an irrigation canal. Chickens lack natural adaptations associated with swimming, including having non-water proof feathers and absence of webbed feet. Lack of webbed feet would be expected to make paddling difficult in a chicken compared to ducks, or other species of birds with webbed feet. In addition, some birds have a fat layer beneath the skin to keep them buoyant in the water, such as ducks and geese, but chickens lack this feature (USDA, 2006). Dr. Strauss lacks objective data that these free-range chickens crossed the irrigation canal near Ms. Garza's residence, and as a result, Dr. Strauss's assumption that chickens would regularly cross the irrigation canal is unsubstantiated.

14.2 The presumed DDT intake, calculated by Dr. Strauss, is similar to daily background intake

Ms. Garza's presumed intake of DDT, DDE, and DDD according to Dr. Strauss, from multiple exposure pathways is similar to background DDT intake levels. Table 2 summarizes Dr. Strauss's initial predicted daily DDT intake (DDT and metabolites) for Ms. Garza while she resided at "506 Nicholson" for several childhood years. Her presumed intake at this location was 108 µg. For her residence at 1015 Nicholson, from 1975 to 1999, Ms. Garza's presumed DDT intake was less than 1 microgram a day.

The predicted total DDT intake that Ms. Garza presumably received is within the range of reported DDT intakes for the general population during the same general time period.[4] An assessment of DDT intake published in 1959 reported that an average man consuming about 2,974 calories daily would have an intake of about 184 µg of total DDT (Hayes, 1959). The Food and Drug Administration began to collect data on DDT and other pesticides beginning in the mid-1960s. The estimated daily total DDT intake from 1964-1967 for a 34 kg person was about 31 µg/day (Duggan 1968). For the time period from 1968 to 1970, a 34 kg person would be expected to consume about 18 µg/day (Duggan and Corneliussen, 1972). Thus, the range of Ms. Garza's presumed DDT intake was similar to or less than intake from background consumption of food during the 1950s, 1960s and 1970s.

14.3 Dr. Strauss's 3-fold increase of Ms. Garza's "cancer risk" is inconsistent with EPA guidance

Dr. Strauss increased the theoretical "cancer risk" for Ms. Garza by 300% for her purported exposure at "506 Nicholson." She claims this 3-fold increase is consistent with EPA guidance for evaluating risks for early life exposures (deposition p. 82; Original

[4] Throughout this time period, the key source of DDT intake for the general population was diet, including dairy products, meat, vegetables, fruits, and grains.

Report, page 18). Dr. Strauss's application of a 3-fold increase is erroneous for the following reasons:

- EPA has established guidance for conducting risk assessments that involve childhood exposures because of the concern that children may be more susceptible to select agents (U.S. EPA, 2005b). Based on this guidance, EPA recommends for risk assessment purposes that a default 3-fold increase be applied to cancer risks only for carcinogens that act through a mutagenic mode of action (U.S. EPA, 2005b). In this same guidance, EPA uses DDT as an example of a non-mutagenic compound. Because DDT does not have a mutagenic mode of action, according to this guidance, the application of a 3-fold default factor by Dr. Strauss to DDT contradicts EPA guidance.

- Despite clear guidance by EPA, Dr. Strauss attempts to justify her application of a 3-fold increase by relying on juvenile-adult ratios derived from a single study of male mice. Specifically, Dr. Strauss testified that a study in the EPA guidance demonstrated a 2.5 increased ratio, supporting the 3-fold increase of risk for Ms. Garza's "506 Nicholson" exposures (deposition pp. 99-100). Regardless of what this single study may or may not show, the guidance is clear that the 3-fold increased risk should only apply to carcinogens that act through a mutagenic mode of action.

In summary, Dr. Strauss's risk assessment and resulting theoretical "cancer risk" estimates for Ms. Garza are unreliable. The estimates are based on incomplete soil data, and unsupported assumptions, including the notion that free-range chickens regularly crossed an irrigation canal, consumed certain soil with DDT, and Ms. Garza daily consumed DDT through those eggs. Dr. Strauss also misapplies EPA guidance in estimating Ms. Garza's theoretical "cancer risks." Moreover, as Dr. Strauss admits, the theoretical "cancer risks" are unrelated to human NHL risks and have no significance for determining the cause of Ms. Garza's NHL.

15.0 Summary of opinions

- It is my opinion, to a reasonable degree of medical probability that the scientific literature does not demonstrate that DDT or its metabolites cause or contribute to the development of NHL, including follicular lymphoma.
- DDT is considered to be one of the most investigated pesticides with respect to the occurrence of NHL.
- Cohort studies of workers with DDT exposure have not demonstrated a statistically significant increased NHL incidence and/or mortality.
- Case-control studies involving DDT including studies that utilize DDT biomarkers show no or very limited associations between DDT and NHL that largely disappear following adjustment for other exposure factors.
- Ecological data on NHL rates and DDT use in the U.S. and other countries do not support the conclusion that DDT causes NHL.
- Texas Department of Health investigations that have been conducted to evaluate potential health impacts from Hayes-Sammons show no increased cancer rates in Mission, Texas and no increased serum DDT levels in persons living and/or working in close proximity to Hayes-Sammons.
- Dr. Sawyer's claim that DDT causes, or substantially contributes to NHL is not substantiated by the epidemiological literature.
- Dr. Sawyer's reliance on the risk assessment process and Dr. Strauss's theoretical "cancer risk" estimates for the purpose of determining the cause of Ms. Garza's NHL lacks scientific validity.
- Dr. Strauss's health risk assessment is based upon incomplete and extrapolated environmental data; consequently, her predicted theoretical "cancer risk" estimates are unreliable and misapplied to show causation of NHL.
- Dr. Strauss's 3-fold increase of Ms. Garza's theoretical "cancer risk" is inconsistent with EPA guidance.
- Dr. Strauss's estimated Ms. Garza's theoretical "cancer risk," even when increased by 300% for "506 Nicholson," are insignificant when compared to her

actual background cancer risk based upon Texas and national data for a woman her age.

- Dr. Strauss's presumed DDT intake levels for Ms. Garza while residing at "506 Nicholson" is similar to background daily intake reported for the general public at that time, whereas Dr. Strauss's presumed DDT intake for Ms. Garza while at 1015 Nicholson is less than the amount she would have received in her diet.
- Ms. Garza has other risk factors for NHL. One risk factor, obesity, is increasingly being recognized through several studies as being important. Another risk factor is Ms. Garza's past employment in a cleaning occupation. Although these risk factors cannot be determined as causal for her follicular lymphoma, they serve to highlight the speculative nature of the plaintiff's expert claims that a known cause can be specified, such as DDT. Yet, her presumed DDT intake is similar or less than that of the general public, and her theoretical increased "cancer risk" is minute compared to her overall background risk of developing cancer.

I reserve the right to modify and/or supplement this report and my opinions if and when additional information becomes available.

Respectfully submitted September 30, 2009,

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Appendix

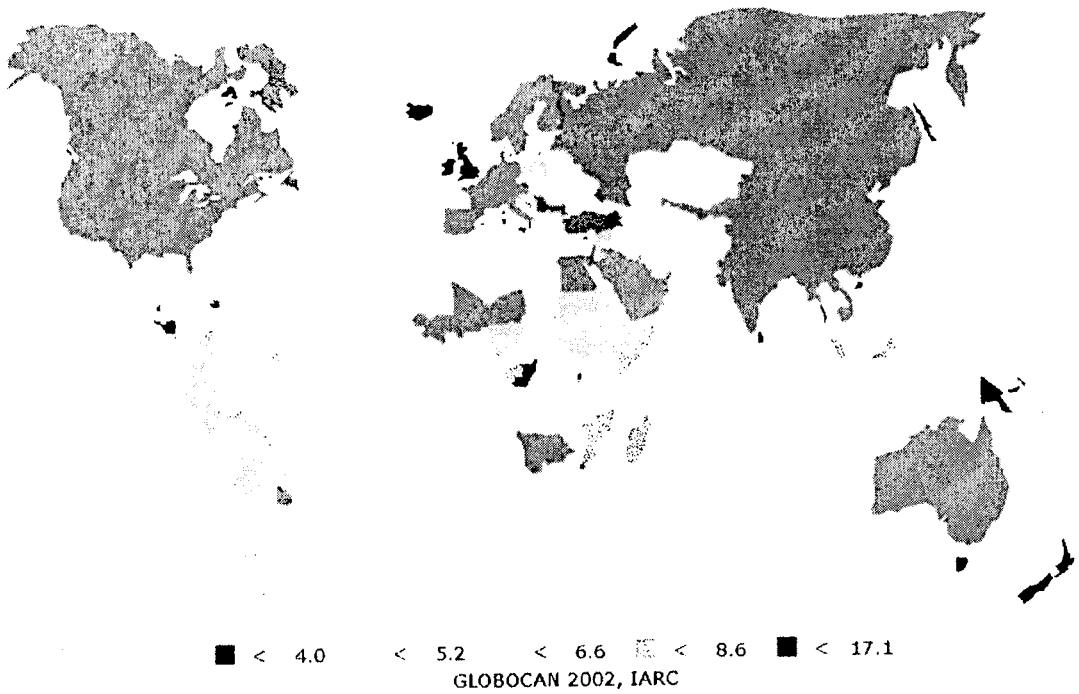

Figure 1. Non-Hodgkin Lymphoma, Males. Age-standardized incidence rate per 100,000 from GLOBOCAN 2002 (Ferlay, et al., 2004).

Table 1. All cancer cumulative risk rates by age (5-year increments), among Hispanic females (Texas Cancer Registry (1998-2002), Department of State Health Services).

Age	Age Group	All Cancer Rate (per 100,000)	Cumulative Risk by age
25	15-34	39.900	0.00655741
30	15-34	39.900	0.00853735
35	35-44	165.200	0.01175239
40	35-44	165.200	0.01988170
45	45-54	344.100	0.02968159
50	45-54	344.100	0.04623312
55[1]	55-64	625.600	0.06513767
60	55-64	625.600	0.09392754
65	65-74	1004.200	0.12514927
70	65-74	1004.200	0.16799098
75	75-84	1330.000	0.21130847
80	75-84	1330.000	0.26205058
85	85+	1459.800	0.31042375
90	85+	1459.800	0.35896293
95	85+	1459.800	0.40408545

1. Age of Ms. Garza at diagnosis.

Table 2. Dr. Strauss's theoretical DDT, DDE, and DDD intakes for Ms. Garza at "506 Nicholson" (Assumes average body weight of 34 kilograms and original Exposure Point Concentration).

	DDT (mg/day)	DDE (mg/day)	DDD (mg/day)	Total (mg/day)	Total (µg/day)
Soil Ingestion[1]	51.68 E-5	160.48 E-5	11.76 E-5	224.0 E-5	2.2
Dermal Contact[1]	11.83 E-5	36.72 E-5	2.69 E-5	51.2 E-5	0.51
Inhalation[2]				9.0 E-5	0.09
Ingestion of Eggs[3]	3,130 E-5	7,290 E-5	0.882 E-5	10,500 E-5	105.0
			Total	**0.108 mg/day**	**108.0 µg/day**

1. Soil EPCs assumed for soil ingestion and dermal contact from "506 Nicholson."
   DDT (o,p' & p,p') 1.292 mg/kg
   DDE (o,p' & p,p') 4.013 mg/kg
   DDD 0.294 mg/kg
2. Inhalation based upon assumption of 0.009 µg/m^3 DDT (total) and inhalation rate of 10 m^3/day.
3. Soil EPCs for ingestion of eggs assuming average soil concentration for "506 Nicholson" and MISD side of canal.

	"506 Nicholson"	MISD Side	EPC for Hens
DDT (o,p' & p,p')	1.292 mg/kg	9.14 mg/kg	5.22 mg/kg
DDE (o,p' & p,p')	4.013 mg/kg	20.30 mg/kg	12.16 mg/kg
DDD	0.294 mg/kg	(assumed 0)	0.15 mg/kg

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Professional Profile

Dr. Marion Joseph Fedoruk is a Principal Scientist in Exponent's Health Sciences and is an advisor to all the Health Sciences Centers. Dr. Fedoruk holds subspecialty board certification in Medical Toxicology and primary board certification in Occupational Medicine from the American Board of Preventive Medicine. There are fewer than 40 physicians in the U.S. who hold both these certifications. He is also a Diplomate of the American Board of Toxicology (DABT) and is also certified by the American Board of Industrial Hygiene as a Certified Industrial Hygienist (CIH).

Dr. Fedoruk maintains an active clinical practice and is engaged in teaching and research at the University of California, Irvine, where he is Clinical Professor of Health Sciences, and Senior Physician at the UCI Center for Occupational and Environmental Health Clinic.

Dr. Fedoruk has extensive experience in the assessment of exposure and related health effects for a broad range of chemical and biological hazards. His 25-year experience involves evaluating health significance from environmental, occupational, and product related exposures. His assessments have involved mercury, lead, arsenic, cadmium, nickel, polychlorinated biphenyl (PCBs), benzene, volatile organic hydrocarbons, isocyanates, pesticides, combustion products, talc, silica, and other dusts. He has designed, developed, and implemented medical monitoring programs for workers in several industries including hazardous waste, energy, asbestos, and electronics, and directed a course on medical surveillance. He performed an assessment of health needs for military and civilian personal involved in chemical weapons destruction in Johnson Atoll, in Micronesia. His work has involved assessment of air quality problems in homes, offices, schools, and work environments including assessments for microbial hazards.

Dr. Fedoruk has served as advisor occupational and environmental health matters to both government and industry. He served as a member of an expert panel convened by EPA and the U.S. Department of Labor to develop national priorities for educating health care providers about the adverse health effects of pesticides. Dr. Fedoruk has served as advisor to NFPA for developing medical guidelines for the Nation's firefighters. He has assisted health and fire departments in evaluating public health impacts associated with accidental chemical releases. He has also been involved in developing occupational medical programs and guidelines for several jurisdictions, including the City of New York.

Dr. Fedoruk has conducted studies and published articles in the areas of occupational and environmental medicine. He authored chapters in several books, including the World Health Organization's Encyclopedia of Occupational Safety and Health, the Encyclopedia of Toxicology, and textbooks in occupational lung diseases and thoracic oncology. He most

recently designated as a Fellow of the American College of Medical Toxicology, the society for board certified medical subspecialists.

Academic Credentials and Professional Honors

M.D., University of Alberta, Edmonton, Canada (with distinction), 1978
Biology Studies, University of Alberta, Edmonton, Canada, 1974
Premed, Selkirk College, British Columbia, Canada (honors standing), 1971

Medical Toxicology, Subspecialty, Certification in 1998 (certified by the American Board of Preventive Medicine)
Occupational Medicine, Specialty, Certification in 1984 (certified by the American Board of Preventive Medicine)
Certified Industrial Hygienist, Toxicology Aspects Certification December 1985 (certified by the American Board of Industrial Hygiene
Diplomate, American Board of Toxicology (DABT), Certification in 1987

Residency: Occupational Medicine, Department of Community and Environmental Medicine, University of California, Irvine, California, 1981–1983; Internship: Montreal General Hospital, Montreal, Canada (McGill University), mixed internship, 1978–1979

1995 Recipient of Merit in Authorship Award from the American College of Occupational and Environmental Medicine for publication, "Personal Risk Assessment Under the Americans with Disability Act: A Decision Analysis Approach," Journal of Occupational Medicine 1993, 35:1000–10

2001 Recipient of The Jean Spencer Felton Award for Excellence in Scientific Writing awarded by The Western Occupational and Environmental Medical Association

Fellow American College of Medical Toxicology, 2007

Adolph G. Kammer Merit in Authorship Award, American College of Occupational and Environmental Medicine, 1995

Publications/Abstracts

Gujral J, Proctor D, Su S, Fedoruk MJ. Water adherence factors for human skin. Poster Presentation, Society of Toxicology Annual Meeting, Baltimore, MD, March 2009.

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Richter RO, Kerger BD, Hoyt S, Fedoruk MJ. Influence of hydrocarbon composition on benzene vapor emissions in mixed products. Abstract selected for platform presentation at 17th International Society for Exposure Analysis Annual Meeting, Proceedings, Durham RTP, NC, October 21, 2007.

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Jones P, Greaney P, Fedoruk M. A model medical surveillance program. Presented at National Institute of Occupational Health and Safety (NIOSH) symposium Best Practices in Workplace Surveillance: Identification and Tracking of Workplace Injury, Illness, Exposures, and Hazards, Cincinnati, OH, November 7–9, 2001.

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Fedoruk J, Miller JD. Health effects of bioaerosols. In: Field Guide for the Determination of Biological Contaminants in Environmental Samples, 2nd edition. Hung LL, Miller JD, Dillon HK (eds), American Industrial Hygiene Association, Fairfax, VA, 2005.

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Harber P, Fedoruk M. Job demand, preplacement evaluation, and accommodation. pp. 857–867. In: Occupational and Environmental Respiratory Disease. Harber P, Schenker M, Balmes J (eds), Mosby, 1996.

Guidotti, Fedoruk MJ. The private alternative in providing occupational health care: Options, objectives, and object lessons. pp. 94–103. In: Occupational Health Services. In Canada through the Year 2000 (Proc. Conference Organized by the Canadian Occupational Health Association, Calgary, November 1986). Gibbs GW, Markham JW (eds), University Press of Canada, North York, Ontario, 1988.

Medical and Scientific Presentations

Fedoruk M. The toxicology of mold—Review of the literature. Presented at UCLA/UCI MOLD: State of the Medical and Environmental Science conference, February 27, 2008.

Fedoruk M. Health effects of bioaerosols. Presented at annual AIHce (American Industrial Hygiene Association) Professional Development Conference, Philadelphia, PA, May 5, 2007.

Fedoruk M. Building associated health problems 2007: What are the issues? Presented at Grand Rounds, Center for Occupational and Environmental Health, UCI, Irvine, CA, August 22, 2006.

Fedoruk M. Building-related health problems: The 2006 perspective. Presented at UCLA Occupational-Environmental Preventive Medicine Conference Los Angeles, CA, May 30, 2006.

Fedoruk M. Exposure assessment. Presented at the California Industrial Hygiene Council, 14th Annual Conference, Redondo Beach, CA, December 2, 2004.

Fedoruk M. Industrial toxicology. Presented at Internal Medicine Rounds, UCI Medical Center, Orange, CA, December 2004 and Veterans Administration Hospital, Long Beach, CA, December 2004.

Fedoruk M. National strategies for health care providers: Pesticide initiative. Presented at American Occupational Health Conference, Kansas City, MO, May 4, 2004.

Fedoruk M. Fungal-related health issues for the integrated waste industry. Integrated Waste Services Association (IWSA) Health and Safety Conference, Charleston, SC, April 20 2004.

Fedoruk M. Molds and mycotoxic effects. Presented at Pulmonary Medical Round, Newport Beach CA, February 2004.

Fedoruk M. Industrial hygiene and exposure assessment issues. Presented at Internal Medicine rounds, UCI Medical Center, Orange, CA, December 2003 and Veterans Administration Hospital, Long Beach, CA, December 2003.

Fedoruk M. Mold remediation: medical toxicological considerations. Presented at the American Conference of Industrial Hygienist (ACGIH) symposium entitled, "Mold Remediation: The National Quest for Uniformity Symposium," Orlando, FL, November, 2003.

EX

Fedoruk M. Indoor health effects associated with residential mold exposure: Science versus public perception. Presented at the UCI Medical Center, Department of Medicine Grand Rounds, Orange, CA, October 29, 2002.

Fedoruk M. Home assessments: How are they done? Presented at the 60th Annual Meeting of the American College of Allergy, Asthma and Immunology, San Antonio, TX, November 16, 2002.

Fedoruk M. Fungi: Measurement and medicine. Presented at American Occupational Health Conference, American College of Occupational and Environmental Medicine, April 2001.

Fedoruk M. Fungi and mycotoxic effects. Presented at Allergy Medical Round, UCI Medical Center, Orange CA, March 2001.

Fedoruk M. Fungi: Exposure and health effects update. Presented at the 10th Annual Conference of the California Industrial Hygiene Council, Redondo Beach, CA, December 4, 2000.

Fedoruk M. Industrial hygiene and exposure assessment issues for internal medicine residents. Presented at UCI Medical Center, December 2000, and Veterans Administration Hospital, Long Beach, CA, December 2000.

Fedoruk M. Acute exposure and health issues related to Hazmat incidents. Presented at the ACOEM State of the Art Conference (SOTAC), Nashville, TN, October 28, 2000.

Fedoruk M. Industrial hygiene and environmental exposure assessment issues. Presented for the ACOEM Basic Curriculum in Occupational Medicine, Segment 3 at the State of the Art Conference (SOTAC), Nashville, TN, October 25–26, 2000.

Fedoruk M. Industrial hygiene and environmental exposure assessment issues. Presented for the ACOEM Basic Curriculum in Occupational Medicine, Segment 3, San Diego, CA, June 8–9, 2000.

Fedoruk M. Industrial toxicology. Presented to Residents in Internal Medicine at weekly rounds at UC Irvine Medical Center and Long Beach Veterans Administration Hospital, CA, January 1999.

Fedoruk M. Tuberculosis in workplace. Presented at UMA Pacific Section, American Industrial Hygiene Association, San Diego, CA, January 27, 1997.

Fedoruk M. Bioaerosol update. Presented at the University of California, Irvine, Center for Occupational and Environmental Health, January 1997.

Fedoruk M. Occupational lead toxicity: ethical aspects. Presented at the 39th Western Occupational Health Conference, Monterey CA, April 15, 1995.

EX

Fedoruk M. Medical surveillance for reproductive hazards. Presented at American Occupational Health Conference, American College of Occupational and Environmental Medicine, Chicago, IL, May 7, 1994.

Fedoruk M. Biological monitoring methods for industrial chemical exposure. Presented at Orange County Section of the Industrial Hygiene Association Annual Conference, Norwalk CA, October 12, 1993.

Fedoruk M. Sick building syndrome: An update. 36th Annual Western Occupational Health Conference, San Diego, IL, October 25, 1992.

Fedoruk M. Environmental assessment. 58th Annual Scientific Assembly, American College of Chest Physicians, Chicago, IL, October 25, 1992.

Fedoruk M. Hazardous waste workers. American Occupational Health Conference, American College of Occupational and Environmental Medicine, Washington, DC, May 7, 1992.

Fedoruk M. Building-related illnesses. Presented at Occupational Medicine Rounds, University of California, Irvine, November, 1991.

Fedoruk M. The fire department medical program. Presented at the Symposium on the Occupational Health and Hazards of the Fire Service, International Association of Firefighters, 20th John P. Redmond Foundation Conference, Las Vegas, NV, September 20, 1991.

Fedoruk M. Environmental medicine: Consulting. Presented at the American Occupational Health Conference at the American College of Occupational Medicine, San Francisco, CA, May 3, 1991.

Fedoruk M. Sick building syndrome: Health effects. Presented at University of California, San Diego, California, School of Medicine, Pulmonary Rounds, California, March 17, 1991.

Fedoruk M. Sick building syndrome perceived risks and case studies. Presented at UCLA Occupational Medicine Rounds, Los Angeles, CA, February 27, 1991.

Fedoruk M, Levine S. Hazardous waste workers. Presented at the American College of Occupational Medicine Annual State of the Art Conference on Fitness for Work, Pittsburgh, PA, October 8, 1990.

Fedoruk M. Occupational/environmental concerns associated with use of Ribaviron and Pentamidine. Presented at the American Lung Association of Orange County meeting entitled Issues In Neonatal And Pediatric Pulmonary Care, Orange, CA, May 5, 1990.

Fedoruk M. Man-made mineral fibers. Presented at the 21st Annual Technical Symposium, American Industrial Hygiene Association-Southern California Section, Long Beach, CA, November 16, 1989.

EX

Fedoruk M. Indoor air pollution. Presented at Occupational Medicine Symposium, AMI Tarzana Regional Medical Center, Tarzana, CA, June 6, 1989.

Fedoruk M. Occupational medicine in the hazardous waste industry: Regulatory aspects. Presented at American Occupational Health Conference, Boston, MA, May 1989.

Fedoruk M. Toxicologic risk assessment, a mechanism for establishing environmental standards. Presented at 32nd Annual Western Occupational Health Conference, Irvine, CA, October 1988.

Fedoruk M, Thorne D, and Yang M. Toxicology and health effects of asbestos fibre exposure. Proc. Third Annual Hazardous Material Management Conference—West, Long Beach, CA, December 1987.

Yang M, Thorne D, Fedoruk M, and Turl E. Assessment of exposure and public health risks during the excavation of a refinery waste contaminated site. Presented at the Air Pollution Control Association meeting, New York, NY, June 1987.

Fedoruk M. Asbestos—Perceived risks and interactive effects. Presented at the 6thUniversity of California, Irvine, Symposium on Environmental Psychology, May 14–15, 1987.

Fedoruk M. Walk-through assessment. Rohm and Haas, Corporate Occupational Health Conference, Philadelphia, PA, April, 1987.

Fedoruk M. Medical surveillance for hazardous waste workers. Presented at Occupational Medicine, Grand Rounds, School of Medicine, University of California, Irvine, March, 1987.

Fedoruk M. Lead and mercury—hazardous exposures. Occupational Health and Preventive Medicine Seminar, Naval Medical Clinic, San Diego, CA, April 6, 1986.

Fedoruk M. Health hazards and toxicology. Presented at 9th Annual Pacific Southwest Safety and Health Workshop/Seminar, San Diego, CA, April 16, 1986.

Fedoruk M. Worker "Right to Know Laws" health hazard training and industrial hygiene practice. Presented at the Occupational Medicine and Health Symposium, American Lung Association, San Diego, CA, March, 1986.

Fedoruk M. Heavy metal toxicology. Presented at the course: Industrial Toxicology Workers Right to Know, University of California, Irvine, CA, August 15, 1985.

Fedoruk M. Chemical exposures in the work place. Presented at St. Joseph's Medical Center, Burbank, CA, August 6, 1985.

Fedoruk M. Reproductive issues in the hospital setting. Presented at the Annual Conference Meeting of the Association of Hospital Employee Health Professional, San Diego, CA, October 17, 1984.

EX

Fedoruk M. Asbestos diseases: An epidemic? Mercy Hospital and Medical Center, Medical Grand Rounds, San Diego, CA, August 9, 1983.

Congressional Testimony

Hearing on San Bernardino, California Pipeline Rupture, Subcommittee on Investigations and Oversight Committee on Public Works and Transportation, U.S. House of Representatives, July 7, 1989.

Academic Appointments

- Clinical Professor of Medicine, Department of Medicine, University of California, Irvine, School of Medicine, 2002–current
- Associate Clinical Professor of Medicine, Department of Medicine, University of California, Irvine, School of Medicine, 1998–2002
- Assistant Clinical Professor of Medicine, Department of Community and Environmental Medicine, University of California, Irvine, School of Medicine, 1985–1991; Department of Medicine 1991–1997
- Assistant Clinical Professor of Medicine, Pulmonary Division, Department of Medicine, University of California, San Diego, School of Medicine, July 1991–January 2000
- Lecturer, Graduate School of Public Health, Division of Public Health, Division of Occupational and Environmental Health, San Diego State University, 1983–1988
- Clinical Instructor of Medicine, Department of Medicine, University of California, San Diego, School of Medicine, 1985–1986

Teaching Experience

Graduate of Public Health, Division of Occupational and Environmental Health, San Diego State University. Primary instructor for two courses:

- "Occupational Medicine"—Core 3-unit course for MPH students specializing in occupational health, 1984–1987
- "Occupational Health Management"—3-unit course for MPH students specializing in occupational health, 1985

University of Southern California, Institute of Safety Systems and Management, Los Angeles, California:

- Instructor for several USC-sponsored extension programs and lecturer on several subjects including industrial hygiene, toxicology, occupational disease and carcinogens.
- Developed and presented a one and one half day course regarding the development and management of medical surveillance programs. This course was given in September 1985, and has been presented biannually through 1990.

EX

Department of Health Services Division of Toxics, State of California, 1987–1988:

- Instructed portions of a course entitled "Introduction to Toxicology" for State of California, D.H.S. employees engaged in Hazardous Waste Operations. (Los Angeles, Berkeley) Sacramento.

University of California, Irvine. Instructor for courses entitled:

- "Air Pollutants and Toxic Chemicals," Social Ecology; 498.3; 1988–1991
- "Managing Indoor Air Quality Episodes," Social Ecology; February 12, 1991

University of California, Berkeley/Northern Occupational Health Center:

- Instructor for a course entitled "Pesticide Contaminated Hazardous Waste Sites" and taught section on pesticide toxicology and medical surveillance, 1989

Committees

American Lung Association, Committee on Environmental and Occupational Health, San Diego, California, 1983–1987.

County of San Diego, Subcommittee Advisory Panel, Hazardous Materials Management Team. Designated representative of County of San Diego Medical Society. The subcommittee advises County Government about the applicability, enforcement, and technical aspects of County Ordinances dealing with hazardous materials management, 1984–1987.

Agricultural/Urban Pesticide Issues Task Force. Task force member of a committee appointed by the County Board of Supervisors to review pesticide usage and determine how it impacts on both urban and agricultural concerns, January 1986–November 1986.

NIOSH/Northern and Southern California Educational Resource Centers. Served on the Advisory Committee on pesticide related hazardous waste training for health and safety professionals, 1989–1990.

National Fire Protection Association. Subcommittee member on the 1001 and 1500 committees concerning medical and physical fitness criteria for structural firefighters, 1989–present.

American College of Occupational and Environmental Medicine. Planning committee member for 1990 State of the Art Conference on Fitness for Work. Pittsburgh, Pennsylvania, October 8–12, 1990.

American College of Occupational and Environmental Medicine. Member of Occupational and Clinical Toxicology committee, 1991–2000, appointed chairman, May 1992.

American College of Occupational and Environmental Medicine. Member of Committee on Scientific Affairs, May, 1992–2000.

EX

American College of Occupational and Environmental Medicine. Member of Committee on Medical Surveillance May, 1993–2000.

Toxicology Excellence for Risk Assessments, Cincinnati, Ohio. Member of Peer Review Committee sponsored by EPA, Health Canada and Metal Finishing Association of Southern California, concerning a risk assessment on the carcinogenicity and toxicity of soluble nickel salts.

U.S. Environmental Protection Agency. Member of expert panel concerning the development of a strategy for educating the nation's health care providers on pesticide-related health matters. Proceedings published in Pesticides and National Strategy for Health Care Providers, July 1998 (EPA 735-R-98-001).

Special Projects

American College of Occupational Medicine, Chicago, IL. Developed questions for the national Medical Self-Assessment Program 2 for physicians to test their knowledge base in occupational medicine.

American Lung Association. Prepared "Solvents in the Workplace" fact sheet for ALA publication Lung Hazards at Work, a program developed to inform employers and employees on the control of harmful occupational exposures that can lead to lung disease.

City of San Diego Fire Department. Developed and presented a risk communication program concerning the fetal health risks from physical and chemical exposures to female firefighters. The program covered the areas of health effects of heat stress, carbon monoxide and other agents on the fetus. The program was presented to all female firefighters with the City of San Diego in 1988 and 1990.

EX

DIGEST OF DEPOSITION OF MR. CHRISTOPHER BEEGAN, STATE WATER WATER BOARD STAFF, PROJECT MANAGER FOR DEVELOPMENT OF THE SEDIMENT QUALITY OBJECTIVES

All page and line references to: Transcript of Beegan Dep. October 11, 2010, In Re Tentative Cleanup and Abatement Order No. R9-2011-0001, Cal. Reg. Water Quality Control Bd., San Diego Region

Participants:
Deponent – Mr. Christopher Beegan – State Water Board Staff, Project Manager for development of the Sediment Quality Objectives
Deposing counsel – Mr. Paul Singarella, Latham & Watkins, LLP
Defending counsel – Mr. Dan Fuchs, Deputy Attorney General, California Attorney General's Office

Other deposing counsel – Mr. Bill Brown, Brown & Winters

Pages(s) : Line(s)	Topic	Text of Deposition
11:9-13	Scope of deposition	By Mr. Fuchs: "Mr. Beegan's testimony will be limited to the following subject: How Mr. Beegan would expect the Phase I Sediment Quality Objectives, DSQOs [sic], to be applied in the abstract without reference to any particular location."
38:20-21, 24-25 39:6-12	Use of stressor identification	By Mr. Singarella: "Q. Turning back to page 13 of Part 1 VI, Human Health . . . Did I understand you to testify that VI contains Lines of Evidence?" By Mr. Beegan: "A. . . . Sections V and VI still describe how you would determine whether sediments – umm – meet or exceed both the human health and the benthic community sediment quality objectives. If you've determined that you've had an exceedance under certain conditions, you move on to stressor identification."
40:16-25 41:1-10	Steps in stressor identification	By Mr. Singarella: "Q. Turning back to stressor identification on page 17, please, there are certain components of stressor identification identified in Section F; correct?" By Mr. Beegan: "A. Yes." "Q. And one step is a confirmation and characterization process; correct?"

		* Edited by Mr. Beegan after the deposition. Original said, "total logical."
47:11-21	Confounding factors	By Mr. Singarella: "Q. . . . I was asking about confounding factors and how are physical disturbance and nonpollutant constituents – how are they confounding factors." By Mr. Beegan: "A. They can – confounding factors – um [affect*] – the response of the indicator in a way that – um – masks the signal you're looking for, and that is the primary reason why we are using multiple Lines of Evidence to assess sediment quality." "Q. And what signal are you looking for?" "A. We're looking for a toxic pollutant-related signal." *Edited by Mr. Beegan after the deposition. Original said, "a fact."
48:21-25 49:1-25	Confounding factors	By Mr. Singarella: "Q. There's a confirmation step under stressor identification; correct?" By Mr. Beegan: "A. Correct." "Q. And one of the things being confirmed is the absence of confounding factors; right?" "A. Correct." "Q. And confounding factors are those nontoxic constituent factors that could result in the signal; right?" "A. Correct." "Q. And the signal that you're looking for is impacts to sediment from toxic constituents; correct?" "A. Correct." "Q. Because you've got a sequential process here; is that right?"

		"Q. In other words, the confounding factors can produce a condition that does not appear protective; correct?" "A. Correct." "Q. Even though that condition is not caused by toxic pollutants; correct?" "A. Correct."
67:10-25 68:1-18	Pollutant identification	By Mr. Singarella: "Q. Turning to page 18 of Part 1, pollutant identification, what is the purpose of pollutant identification?" "A. When you follow Part 1, you apply the three Lines of Evidence, you end up with simply a station categoric – categorization for each station. I've described those in the past. Shall I describe them again?" "Q. No, that's fine." "A. Okay. Umm – in order to proceed further, you need to determine what is causing the problem – umm – so likely impacted, clearly impacted, possibly impacted, it doesn't – the – it only is used in the assessment. After that to determine what the stressor is you need to determine – you need to go down this stressor identification process. As we've gone over in the past, you rule out other nonpollutant-related factors like dredging, prop wash. The next step would be, okay, what pollutant is causing the problem? Once you've identified what pollutant is causing the problem, you can – umm – manage it, or restore, or cleanup, or control. Does that help?" "Q. Yes. Thank you." "A. Okay." "Q. And so is pollutant identification a mandatory component of stressor identification?"

		We clearly stayed [sic, probably stated] up front, don't use these chemistry values to identify which chemical is causing the problem, because that's not a fruitful or accurate approach. Those sediment quality guidelines basically – I believe they have similar limitations. The empirical ones have similar limitations. In some cases where your – umm – you have one pollutant that's screaming, maybe multiples of the sediment quality guideline it – they perform better. But in general their use was intended for assessment, not stressor identification." "Q. Uh-huh. When you say 'screaming' you mean exceedingly high concentrations?" "A. Yeah, like two, three, five times the – um – the guideline." "Q. And probably where there aren't other pollutants around?" "A. Correct. There's – we could spend a day talking about the limitations of empirical and mechanistic guidelines."
76:12-17	Steps in stressor identification	By Mr. Singarella: "Q. So with regard to the structure of stressor identification, it has three principal components; correct?" By Mr. Beegan: "A. Yes." "Q. Are they all mandatory?" "A. Yes."
76:24-25 77:1-22	Management actions	By Mr. Singarella: "Q. Do you see the reference to 'management actions?'" By Mr. Beegan: "A. Yes."

		By Mr. Beegan: "A. No."
87:19-25 88:1-25 89:1-13	Pollutant-specific concentration-based approaches	By Mr. Singarella: "Q. Now turning back to page 19 of Part 1, Sub-H refers to the Site-Specific Sediment Management Guidelines; correct?" By. Mr. Beegan: "A. Yes." "Q. Would that be a pollutant-specific concentration-based approach?" "A. Oh, yes. Yes." "Q. So there was a shifting away from pollutant-specific concentration-based approaches; right?" "A. Yes." "Q. Help me understand why it came back in in the last section of Part 1, Section H." "A. Okay. The – umm – there – umm – I'm trying to figure out how to state this in a way that makes sense, but – The assessment approach was the portion that I was referring to. Many people want to chemic – sediment chemistry based numeric values that could be applied broadly to all bays and all estuaries. The level of science isn't there to create reliable numeric sediment chemistry values. So that – umm – so we use the multiple Line of Evidence approach, the three LOE. The chemistry is – has confounding factors, toxicity, benthic community. They all have confounding factors. So what I was referring to in the shifting away was – umm – to state up front that we can't develop numeric sediment chemistry-based objectives, however, once you identify the stressor and it's a pollutant-related stressor, the – I guess the practical way in which you manage that is to develop some sort of concentration-based approach.

		"A. Yes." "Q. There's no indication that a pollutant-specific guideline may be designated prior to the establishment of that relationship; right?" "A. Right."
94:8-25 95:1-18	Site-specific cleanup levels	By Mr. Singarella: "Q. You mean the numbers on page 8 are used to set site-specific cleanup levels?" By Mr. Beegan: "A. Oh, absolutely not." "Q. I'm sorry. Then I may have misunderstood." "A. Oh, okay. Okay. So these are not – these- these values are not intended to drive cleanup, or a site-specific cleanup, or even be water body or –umm – segment-specific sediment targets. These are only for use as a – one of the LOE, Line of Evidence, and the assessment framework. Once you've moved through – once you've applied the assessment framework –." "Q. Sir, may I hold you up there? When you said 'These values are not used as cleanup levels,' were you referring to the values in Table 6 and 7 on page 8 of Part 1?" "A. Yes. Yes." "Q. Thank you for that clarification. Please proceed with your answer." "A. So, again, those values that we discussed in those tables are only for the assessment purpose. The next step is stressor identification, and then once you define a – that it's a pollutant-related stressor you look at – um – relationships between the stressor identified at your site

		"Q. And ERL is empirical based?" "A. Yes." "Q. You did not use either of these in the sediment chemistry prong of the SQO; right?" "A. Right."
107:23-25 108:1-8	Purpose of SQOs	By Mr. Singarella: "Q. And is it – is it your understanding that the SQOs are a means to differentiate sediment?" By Mr. Beegan: "A. Yes." "Q. Basically between the impacted and those that are not impacted; right?" "A. Correct." "Q. By bioavailable toxic pollutants; correct?" "A. Correct." "Q. So it's a tool to distinguish the good sediment from the bad sediment; right?" "A. Essentially, yes."
119:5-22 120:1-19	Use of chemistry LOE	By Mr. Singarella: "Q. And it absolutely says that the chemistry LOE shall not be used for setting cleanup levels; correct?" By Mr. Beegan: "A. Correct." "Q. What does that mean? "A. It means that those values in – umm – in those tables – umm – are – are inappropriate for use as establishing cleanup levels. I – I - ."

	evidence and causality	correct?" By Mr. Beegan: "A. Absolutely correct."
122:1-16	Use of Lines of Evidence	By Mr. Singarella: "Q. And, in fact, the Lines of Evidence are not sufficiently reliable to be used independent in isolation of the other Lines of Evidence; right?" By Mr. Beegan: "A. Correct." "Q. Because they can underestimate the risk in some circumstances; right?" "A. Correct." "Q. Or overestimate risks in other circumstances; correct?" "A. Correct." "Q. And this imprecision can be significant; correct?" "A. Correct."
126:1-11	SQO process	By Mr. Singarella: "Q. And so the way this works is you go through the MLOE; right?" By Mr. Beegan: "A. Yes." "Q. You do stressor identification; correct?" "A. Yes." "Q. And then, if you identify a particular constituent associated with an unacceptable impact from a particular source, at that point the Regional Board requires the discharger to take management actions; correct?" "A. Correct."
191:7-25	Rejection of	By Mr. Singarella: Referencing a page of the SQO staff report: "Q. Turning to page 5-

		"A. Umm – correct." "Q. And McDonald et al, 2000; right?" "A. Uh-huh." "Q. Yes?" "A. Yes."
206:8-25 207:1-25 208:1	Relationship between SQOs and TMDLs	By Mr. Brown: "Q. Okay. And then just one last generalization question. I'm going to be off very quickly. I'm still trying to separate in my mind, Mr. Beegan, the difference between the TMDL track and the SQO track. Did they run parallel or do they intersect at some point? By Mr. Fuchs: "Objection. Vague and ambiguous. Also calls for a legal conclusion. If you understand the question, you can take a stab at answering it." By Mr. Beegan: "A. Well – I'm sorry. I must not have – umm – answered the question – umm – clearly before, because -." By Mr. Brown: "I wouldn't presume that, but I'm trying to take in a lot of information all at once. You may have answered it very clearly." By Mr. Beegan: "No. No. So – umm – the Sediment Quality Objectives are used to assess sediment. Once you've assessed sediment – umm – and you have a sediment quality related listing – umm – your – In order to restore it you would go down the TMDL path. You've demonstrated that your sediment quality isn't meeting the narrative. From there stressor identification, target – umm – some sort of sediment target or load, waste load allocation would be – umm – appropriate. I – A TMDL is developed based on a degradation or an impairment of a water body. A

		"Q. And that paragraph – I'm sorry. Did I cut you off?" "A. No. The – the regulatory decisions that are referred to are referring to the exceedance of a sediment quality guideline and biological effects, and it's attempting to explain that these are not good – indicators of cause, they merely establish occurrence or co-occurrences, or, you know, an association." "Q. So they would build more or less a circumstantial case against a particular compound?" "A. Right." "Q. When there might be a host of other potential causative agents at play; right?" "A. Correct."
255:10-23	TMDLs and SQOs	By Mr. Singarella: "Q. . . . And then third sentence refers to enormous resources applied instead of focusing on the causes; correct?" By Mr. Beegan: "A. Right." "Q. What does that mean?" "A. It means that – umm – that TMDLs – to develop – TMDLs develop for stressors – and I use – I shouldn't use the term 'stressors.' TMDLs develop for pollutants based on a – an exceedance of a sediment quality guideline, with associated biological effects – may not result in an improvement of sediment quality."
260:11-25 261:1-	Limitations of the SQOs	By Mr. Singarella: "Q So the multiple Line of Evidence approach just doesn't go to the issue of cause; correct?" By Mr. Beegan: "A. It does not identify pollutants that are causing impacts."

		By Mr. Beegan: "Okay." By Mr. Singarella: "Q. If I wanted to find the level of constituent that's protective of the critters in the – in here, can you point me to it?" By Mr. Beegan: "A. No." "Q. If it were in here, you'd be able to point me to it; right?" "A. Correct."

CALIFORNIA REGIONAL WATER QUALITY CONTROL BOARD

SAN DIEGO REGION

IN THE MATTER OF:	)
TENTATIVE CLEANUP AND ABATEMENT ORDER NO. R9-2011-0001	)

VIDEOTAPED DEPOSITION OF DAVID CHRISTOPHER BEEGAN

MONDAY, OCTOBER 11, 2010

SACRAMENTO, CALIFORNIA

Reported By: CAROL NYGARD DROBNY, CSR No. 4018, RPR

State Water Resources Control Board

Linda S. Adams
Secretary for
Environmental Protection

Office of Chief Counsel
1001 I Street, 22nd Floor, Sacramento, California 95814
P.O. Box 100, Sacramento, California 95812-0100
(916) 341-5161 ♦ FAX (916) 341-5199 ♦ http://www.waterboards.ca.gov

Arnold Schwarzenegger
Governor

December 1, 2010

Ryan Waterman
Latham & Watkins LLP
600 West Broadway, Suite 1800
San Diego, CA 92101-3375

Dear Mr. Waterman:

Re: TRANSCRIPT OF VIDEOTAPED DEPOSITION OF DAVID CHRISTOPHER BEEGAN, OCTOBER 11, 2010; CORRECTIONS AND SIGNATURE PAGE

Enclosed please find a copy of the edits and signature of Mr. Beegan following his review of the above-referenced deposition transcript. I am informed that I am to retain the original copy of the transcript. Please contact me if you need anything further in reference to this matter.

I can be reached at (916) 341-5169.

Sincerely,

Marleigh Wood,
Senior Staff Counsel

Enclosures

Signed under penalty of perjury:

DAVID CHRISTOPHER BEEGAN

11-14-10

DATE

CERTIFICATE

I, the undersigned, do hereby certify that I have read the foregoing deposition and that, to the best of my knowledge, said deposition is true and accurate (with the exception of the following changes listed below).

PAGE No.	LINE No.	
16	8	Art + Lawson should read "Harding Lawson"
	13	same as above
43	7	"tautological" in place of total logical
47	15	"a fact" should read "affect"
79	16	"WIS" should read "NPDES"
82	16	Delete "this"
124	4	"permanent" should be "permit"
128	3	"CEQUA" should read "CEQA"
139	17	"sufficient" should read "sovereign"
200	23	Change "No" to "YES"
258	2	"[illegible]" should read "[illegible]"
253	18	insert "not"

Please turn to back of transcript and

Summary of edits to October 11, 2010 Deposition on SQOs

Page No/Line No.	Description
16/8	"Art Lawson" should read "Harding Lawson"
16/13	Same as above
43/7	"total logical" should read "tautological"
47/15	"a fact" should read "affect"
79/16	"M13 permitting" should probably read "NPDES permitting"
82/16	Delete "this"
124/9	"Permanent limit" should read "permit limit"
128/3	"CEQUA" should read "CEQA"
139/17	"sufficient" should read "surficial"
209/23	Changed 'no" to "yes"
258/2	"by variability" should read "bioavailability"
258/18	Added "not"

LAWYER'S NOTES

PAGE NO.	LINE NO.

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INDEX

EXAMINATION BY COUNSEL

EXHIBITS

Exhibit 708	Evaluation of Methods for Measuring Sediment Toxicity in California Bays and Estuaries, Southern California Coastal Water Research Project		268

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CALIFORNIA REGIONAL WATER QUALITY CONTROL BOARD

SAN DIEGO REGION

IN THE MATTER OF:

TENTATIVE CLEANUP AND ABATEMENT ORDER NO. R9-2011-0001

BE IT REMEMBERED, that on Monday, October 11, 2010, commencing at the hour of 8:09 a.m. thereof, at the offices of Carol Nygard & Associates, 2295 Gateway Oaks Drive, Suite 170, Sacramento, California, before me, Carol Nygard Drobny, a Certified Shorthand Reporter of the State of California, there personally appeared

DAVID CHRISTOPHER BEEGAN,

called as a witness by the Designated Party, who, being by me first duly sworn, was thereupon examined and interrogated as hereinafter set forth.

VIDEOGRAPHER: My name is Sean McAleer. 08:06:36

I will be videotaping this proceeding on behalf of Peterson Reporting & Video Services at 530 B Street, Suite 350 in San Diego, California. 08:06:40 08:06:44 08:06:51

The date is October 11th, 2010. The time on 08:06:56

the video monitor is 8:06 a.m. 08:06:59

Our location is 2295 Gateway Oaks Drive, Suite 170 in Sacramento, California. 08:07:03 08:07:10

We are here in the matter of Tentative Cleanup and Abatement Order Number R9-2011-0001. 08:07:13

This is the deposition of Chris Beegan. 08:07:25

The noticing attorney is Paul Singarella. 08:07:29

The Court Reporter is Carol Nygard of Peterson Reporting. 08:07:32 08:07:36

This is a single-track recording. Overlapping voices cannot be separated. Private discussions while on the record will also be recorded. 08:07:37 08:07:41 08:07:44

Will counsel please identify yourselves, your firms, and those you represent. 08:07:46 08:07:48

MR. SINGARELLA: Good morning. 08:07:50

Paul Singarella from Latham & Watkins representing National State and Shipbuilding Company, also referred to as NASSCO, N-A-S-S-C-O. 08:07:52 08:08:01

MR. RICHARDSON: Kelly Richardson with Latham & Watkins also for NASSCO. 08:08:04 08:08:08

MR. FUCHS: Dan Fuchs, California Attorney General's Office, Deputy Attorney General, representing the State Water Resources Control Board and here for Mr. Beegan today. 08:08:09 08:08:13 08:08:15 08:08:18

(Thereupon the oath was administered to the

Witness by the Court Reporter.)

MR. SINGARELLA: Good morning, Mr. Beegan. 08:08:32

THE WITNESS: Good morning.

MR. SINGARELLA: Could you state your full 08:08:33
name and -- and place of residence. 08:08:34

MR. FUCHS: Before you do that I just have a 08:08:39
preliminary statement I wanted to read in to the record. 08:08:42

MR. SINGARELLA: I was going to turn it right 08:08:42
over to you after. 08:08:44

MR. FUCHS: Okay. I figured now that he's 08:08:45
been sworn before any responses are given. 08:08:46

MR. SINGARELLA: Sure. 08:08:48

MR. FUCHS: This deposition is being taken 08:08:48
under protest. 08:08:49

The State Water Resources Control Board does 08:08:51
not concede that the deponent, Chris Beegan, has any 08:08:53
nonprivileged information relative to Tentative Cleanup 08:08:57
and Abatement Order R9-2011-0001 or the Draft Technical 08:09:05
Report accompanying the same. 08:09:07

Mr. Beegan specifically has no information 08:09:09
regarding the Regional Board's rationale for selecting 08:09:11
its methodology and application thereof. 08:09:16

The State Water Resources Control Board 08:09:17
continues to view this deposition as an impermissible 08:09:19
inquiry in to the mental processes of its 08:09:22

decision-makers and an impermissible and unethical attempt to engage in discovery regarding the Superior Court action entitled Cal Chamber versus State Water Resources Control Board, Sacramento County Superior Court Case Number 34-2008-00006509, which case has been stayed by stipulation. 08:09:26 08:09:28 08:09:30 08:09:35 08:09:38 08:09:48

As described in the conference call overseen by Discovery Referee Tim Gallagher on September 27th, 2010, Mr. Beegan's testimony will be limited to the following subject: How Mr. Beegan would expect the Phase I Sediment Quality Objectives, DSQOs, to be applied in the abstract without reference to any particular location. 08:09:49 08:09:52 08:09:56 08:09:59 08:10:02 08:10:07 08:10:10

Accordingly, the following subjects, for example, are beyond the scope of this deposition and Mr. Beegan will be instructed not to respond to any questions regarding them: Any questions regarding how and by whom the Phase 1 SQOs were developed and the bases of the Phase 1 SQOs and any questions regarding use or application of the Phase 1 SQOs, if any, by the San Diego Regional Board or its Cleanup Team or by anyone else. 08:10:11 08:10:14 08:10:17 08:10:20 08:10:23 08:10:28 08:10:33 08:10:38 08:10:41

This is a nonexclusive list of subjects outside the scope of this deposition, and I will instruct Mr. Beegan not to respond to any questions 08:10:41 08:10:44 08:10:47

outside the very limited scope described above, even if not included in the foregoing examples. 08:10:50 08:10:53

Mr. Beegan is appearing as a percipient and not an expert witness. He has not been retained, noticed or paid as an expert. 08:10:55 08:11:00

Further, pursuant to numbered paragraph 3 of the September 27, 2010 correspondence from Kelly Richardson to Tim Gallagher by taking Mr. Beegan's deposition today NASSCO and its counsel are stipulating that Mr. Beegan's provision of deposition testimony today will not affect his ability to advise the State Water Board in any future proceeding regarding the Phase 1 SQOs; that insofar as Mr. Beegan is a member of State Water Board staff, his deposition testimony will not prevent the State Water Board from independently evaluating the CAO/DTR or the validity of the Phase 1 SQO themselves if it is ultimately called upon to do so, and that Mr. Beegan's deposition testimony will in no way constitute a predetermination of issues by the State Water Board. 08:11:02 08:11:05 08:11:08 08:11:12 08:11:14 08:11:17 08:11:21 08:11:25 08:11:29 08:11:32 08:11:36 08:11:42 08:11:46 08:11:51 08:11:52

By proceeding NASSCO and its counsel further stipulate that they will not use nor will they permit or suggest that anyone else use the testimony given today or the documents received pursuant to the subpoena duces tecum served in this matter in the aforementioned 08:11:53 08:11:57 08:11:59 08:12:02 08:12:08

Superior Court case in any form or manner. 08:12:09

MR. SINGARELLA: Mr. Fuchs, if you had wanted a stipulation, you could have approached this over the last week, so let's table that for right now. 08:12:15 08:12:17 08:12:19

I will take your objection as including a request for a stipulation. We will be glad to discuss said stipulation with you when you properly join the subject with us. 08:12:23 08:12:27 08:12:31 08:12:34

You have not properly joined the subject by coming in here and starting off with a unilateral statement about what we're stipulating to or not. 08:12:36 08:12:38 08:12:42

That's not the way lawyers enter in to stipulations, and you know it. So we'll discuss that later. 08:12:45 08:12:48 08:12:52

In terms of the substance of your statement, the suggestion that this is unethical is taken personally and is just way out of line. 08:12:53 08:12:58 08:13:02

I really don't think I have any other burden at this point to respond. 08:13:06 08:13:09

I thought that your characterization of where you're going to instruct Mr. Beegan to not answer is incredibly crad and unlimited in light of where we were in front of the Special Master last week, but I think we ought to proceed and -- and see where we get, and if we reach an impasse, as you know, Mr. Gallagher is 08:13:10 08:13:14 08:13:22 08:13:25 08:13:29 08:13:32

available and we can take it up with -- with him. 08:13:35

EXAMINATION 08:13:40

BY MR. SINGARELLA: 08:13:40

Q. Good morning, Mr. Beegan. 08:13:41

Could you state your full name for the record 08:13:42

and your place of residence. 08:13:44

A. My name is David Christopher Beegan. 08:13:45

I reside at 3828 Arden Way, Sacramento, 08:13:49

California. 08:13:55

Q. And we understand that you are employed by the 08:13:55

State Water Resources Control Board? 08:13:57

A. Yes, that is true. 08:13:59

Q. And what is your title at the State Water 08:14:01

Board? 08:14:03

A. Engineering Geologist. 08:14:03

Q. And this morning I'll refer to your employer 08:14:06

as "The State Board" or "The State Water Board." 08:14:11

Is that acceptable to you? 08:14:13

A. Yes. 08:14:14

Q. And how long have you held that position at 08:14:14

The State Water Board? 08:14:17

A. Umm -- going on 11 years -- umm -- in 08:14:18

November. 08:14:31

Q. Did you start work at The State Water Board in 08:14:31

about 1999? 08:14:36

A. Yes. 08:14:37

Q. And so you've held that position since -- since you started working there? 08:14:37 08:14:40

A. Yes. 08:14:41

Q. And do you have a particular relationship or responsibility with respect to the Sediment Quality Objectives Initiative of the State Board? 08:14:41 08:14:48 08:14:53

A. Yes. 08:14:55

Q. And what is your relation to the Sediment Quality Objectives Initiative? 08:14:56 08:14:59

A. I guess the business description would be as a Project or Program Manager. 08:15:01 08:15:07

Q. And when I refer to "Sediment Quality Objectives," you'll understand that I'm referring to the statutory program being implemented by The State Board to develop Sediment Quality Objectives for bays and estuaries in the State of California? 08:15:08 08:15:15 08:15:20 08:15:23 08:15:28

A. Yes. 08:15:29

Q. And I may refer to "Sediment Quality Objectives," or just "The Objectives," or "SQOs." 08:15:29 08:15:35

Is that agreeable to you? 08:15:39

A. Yes. 08:15:40

Q. And you'll understand what I mean when I refer to "SQOs" or "The Objectives"? 08:15:40 08:15:43

A. Yes. 08:15:46

Q. Thank you. 08:15:46

What was your prior employment before coming 08:15:47
to the State Water Board? 08:15:49

A. I worked for a -- engineering environmental 08:15:50
consulting firm here in Sacramento. I worked as a 08:16:02
geologist. 08:16:03

Q. And the name of that firm was? 08:16:04

A. Art & Lawson Associates, which -- I guess 08:16:07
became MacTech, either right before I left or right 08:16:10
after. 08:16:18

I'm not sure. 08:16:19

Q. Did you work on issues of sediment quality 08:16:20
while at Art & Lawson? 08:16:23

A. Not that I recall. 08:16:25

Q. And when did you first start working on issues 08:16:27
of sediment quality? 08:16:30

A. Probably some time around 2002. 08:16:32

Q. As part of your work for the State Water 08:16:40
Board? 08:16:43

A. Yes. 08:16:43

Q. And could you give me your educational 08:16:43
background, just college and any grad studies. 08:16:48

A. Umm -- Bachelors Degree in -- Bachelors of 08:16:51
Science in geology from Cal State Long Beach. 08:16:56

Q. And while at Cal State Long Beach did you have 08:17:03

occasion to learn about or study sediment quality? 08:17:05

A. Umm -- sediment quality -- no. 08:17:10

Q. And you understand that you're under oath here today, Mr. Beegan? 08:17:25 08:17:30

A. Right. Yes. 08:17:31

Q. And you understand that it's as if you were testifying in a Court of Law with reference to potential penalties of perjury? 08:17:32 08:17:35 08:17:39

A. Correct. 08:17:40

Q. Have you had your deposition taken before, Mr. Beegan? 08:17:40 08:17:44

A. No. 08:17:44

Q. Well, the way a deposition works is I'll be asking you a series of questions. 08:17:45 08:17:52

You understand that? 08:17:54

A. (Nodding head) 08:17:55

Q. And it's -- it's important that you answer each question orally, not with -- nodding of the head -- 08:17:56 08:17:59

A. Yes. 08:18:05

Q. -- and so forth; correct? 08:18:05

And the best way for this to work is -- is for you to give me the opportunity to get my question fully out and -- and on the record, and the technical nature of -- of this subject matter is such that I may struggle in doing so, so please be patient with me. 08:18:09 08:18:12 08:18:15 08:18:19 08:18:22

Once I get my question out you'll be given a full and fair opportunity to respond. 08:18:26 08:18:28

Is that agreeable to you? 08:18:30

A. Yes, it is. 08:18:31

Q. And, if you don't understand my question or any part of it, you have no burden to respond, but I would ask you to please tell me, "Counselor, I can't answer that question. I don't understand it. Could you please rephrase it." 08:18:32 08:18:36 08:18:39 08:18:45 08:18:48

Is that acceptable to you? 08:18:49

A. Yes. 08:18:49

Q. And if you do go ahead and answer the question, I'll assume that you did understand the question. 08:18:50 08:18:53 08:18:56

Is that agreeable to you? 08:18:57

A. Yes. 08:18:59

Q. The Court Reporter is creating a transcript of the proceedings. 08:18:59 08:19:06

You will be given an opportunity to review that transcript a month or so after the deposition ends, and you'll also be given the opportunity to make any changes or edits to the transcript that you wish to make. 08:19:07 08:19:09 08:19:14 08:19:17 08:19:20

Do you understand that? 08:19:20

A. Yes. 08:19:21

Q. If you do make changes, however, at any hearing on the matter or in any trial I will have the opportunity to comment on those changes including issues of -- of credibility and things of that nature. 08:19:21 08:19:25 08:19:30 08:19:35

Is that -- is that understood? 08:19:38

A. Yes. 08:19:40

Q. Now, you're represented by Mr. Fuchs here today, who's already made an objection on the record. 08:19:40 08:19:50

He actually may make additional objections on the record today. 08:19:53 08:19:55

If he does make an objection on the record, it does not mean that -- that you should not answer the question. In fact, let him make his objection, and then please answer my question. 08:19:56 08:19:59 08:20:01 08:20:05

Is that agreeable to you? 08:20:06

A. Umm -- I guess I'll -- I'll -- umm -- I'll answer -- umm -- if -- umm -- Mr. Fuchs says that that is okay. 08:20:07 08:20:16 08:20:26

Q. Well, Mr. Fuchs in addition to making an objection may actually instruct you to not answer. 08:20:27 08:20:33

A. Oh, right. 08:20:36

Q. That's an instruction. 08:20:36

A. Right. Okay. 08:20:38

Q. That's not an objection. 08:20:38

A. Yes. Okay. 08:20:40

Q. As a matter of fact, he has suggested that he may be making such instructions today, but that would be different than an objection. 08:20:41 08:20:43 08:20:46

If he's just saying "Objection to the form of the question" or "Objection. Asked and answered," something like that, let him make his objection and -- and -- and please go ahead and answer the question. 08:20:47 08:20:49 08:20:51 08:20:54

I think that's acceptable to your attorney. I want to make sure it's acceptable to you. 08:20:57 08:21:01

A. Yes. Yes. 08:21:02

Q. Thank you. 08:21:03

And is there any reason today that you can't answer my questions once they're understood -- at least fully and truthfully? 08:21:04 08:21:08 08:21:12

A. No. There's -- no reason. 08:21:13

Q. Is there any reason that the deposition should not proceed today? 08:21:19 08:21:22

A. No. 08:21:23

Q. Are you of clear mind today and ready to proceed? 08:21:24 08:21:27

A. For a Monday, yes. 08:21:28

Q. As clear as any Monday? 08:21:29

A. Yes. 08:21:31

Q. Excellent. Excellent. 08:21:31

Okay. As we proceed, people are drinking 08:21:33

coffee and -- and such, and we'll probably break at some point. 08:21:36 08:21:41

It's at your -- at your convenience, please. 08:21:42

Let me know if you'd like to take a break. 08:21:45

If you do want to take a break, I would most likely ask you to answer any question pending at the time before we break. 08:21:48 08:21:50 08:21:54

Is that acceptable to you? 08:21:55

A. Yes. 08:21:57

Q. Okay. Let me just say a little bit about the exhibits that will be placed in front of you, and I -- I simply want to refer to the numbering scheme so that you have an understanding of it before we proceed. 08:21:57 08:22:03 08:22:06 08:22:09

In this matter there have been a number of depositions, and I think there will be some -- some further depositions of different people, and so we have created a master set of common exhibits, and we've numbered those 1 through whatever. 08:22:12 08:22:14 08:22:17 08:22:20 08:22:25

I'm going to be presenting you, for example, with the SQOs themselves, and that's Master Exhibit Number 6. 08:22:28 08:22:31 08:22:37

I don't want you to get thrown off by not having Exhibits 1 through 5. 08:22:37 08:22:40

A. Okay. 08:22:42

Q. And -- and then when I put in front of you an 08:22:42

exhibit that's unique to your deposition, I'll start that with a new hundred series. 08:22:46 08:22:49

I'm not sure what hundred series we're using today, it might be 200 or 300, but when we get to Beegan exhibits that are unique to this depo it will start with a 200 series. 08:22:51 08:22:54 08:22:58 08:23:02

Do you understand that? 08:23:04

A. Yes. 08:23:04

Q. I just don't want you to get thrown off -- 08:23:05

A. Okay. 08:23:08

Q. -- by -- by the numbering system. 08:23:09

A. Okay.

Q. Thank you. 08:23:12

Do we have in front of the witness Master Exhibit Number 6? 08:23:12 08:23:18

Yes? 08:23:25

A. Yes. 08:23:25

Q. We've caused to be placed in front of you Master Exhibit Number 6. 08:23:25 08:23:28

Do you see that, Mr. Beegan? 08:23:30

A. Yes. 08:23:31

MR. SINGARELLA: Let me just hand out our copies of this around the room. 08:23:39 08:23:41

MR. FUCHS: Thank you. 08:23:44

BY MR. SINGARELLA: 08:23:45

Q. Okay. Turning to Master Exhibit Number 6, do you recognize this document, Mr. Beegan? 08:23:55 08:23:59

A. Yes. 08:24:00

Q. And do you understand it to be the Part 1 SQOs? 08:24:01 08:24:05

A. Yes. 08:24:06

Q. And could you just take a quick look at it for me and verify that it is, in fact, the Part 1 SQOs? 08:24:07 08:24:10

A. Yes, it appears to be Part 1 SQOs. 08:24:39

Q. Thank you. 08:24:43

Could you turn to the Resolution and Acting Part 1. 08:24:43 08:24:46

And you see Resolution Whereas Number 6 referring to the Narrative SQOs? 08:24:49 08:24:53

A. Yes. 08:24:57

Q. And by -- by "Narrative Objectives" is that to be differentiated from "Numeric Objectives"? 08:24:59 08:25:05

A. Yes. 08:25:09

Q. And so the Part 1 SQOs are Narrative Objectives; is that right? 08:25:15 08:25:19

A. Correct. 08:25:20

Q. And they consist of words; is that right? 08:25:20

A. Yes. 08:25:25

Q. And -- and formula; is that right? 08:25:26

A. I don't understand what you mean by "formula." 08:25:29

Q. Do you sometimes use the word "formula" with regard to the work that you do? 08:25:35 08:25:38

A. No. 08:25:40

Q. The -- help me out then. 08:25:41

In the Lines of Evidence there are certain calculations that are made? 08:25:46 08:25:49

A. Oh. Correct. Correct. 08:25:50

Q. So would you be more comfortable referring to those parts of the SQO as "calculations"? 08:25:52 08:25:55

A. Sure. 08:25:58

Q. And there are also numbers in the Narrative SQOs; is that right? 08:26:04 08:26:09

A. Yes. 08:26:11

Q. So you've got words, and calculations, and numbers, and that is a Narrative to be distinguished from a Numeric Objective; correct? 08:26:12 08:26:17 08:26:21

A. I think of it as -- I think of it a little differently. 08:26:23 08:26:36

I think of the Narrative Objective -- umm -- as a statement of what sediment -- the quality the sediment needs to be at to be healthy or -- unimpacted, and then I think of the -- what you call "the calculations," the three Lines of Evidence, as being used to interpret the Narrative Sediment Quality Objective. 08:26:38 08:26:44 08:26:52 08:27:00 08:27:05 08:27:11 08:27:22

Q. Okay. Thank you. 08:27:24

Thank you. 08:27:25

And did you write Part 1? 08:27:26

A. Yes. Umm -- I was not alone in the preparation or the -- 08:27:31 08:27:39

Q. And by "Part 1" you'll understand that I'm referring to Master Exhibit Number 6? 08:27:43 08:27:46

A. Umm -- yes. 08:27:50

Q. And why is it called "Part 1" versus any other Part? 08:27:51 08:27:55

A. That's -- the -- Water Quality Control Plan for Enclosed Bays and Estuaries -- there are potentially other issues that could be included in that Water Quality Control Plan. 08:27:55 08:28:04 08:28:15 08:28:19

Umm -- I think -- umm -- the Division of Water Quality State Water Board is working on various other -- umm -- Water Quality Objectives for -- umm -- Enclosed Bays and Estuaries, and so those would become potentially Part 2, Part 3, Part 4. 08:28:21 08:28:28 08:28:34 08:28:39 08:28:43

It was a means to -- umm -- if those additions were made, to keep things -- umm -- kind of clean in terms of separating out different portions. 08:28:48 08:28:52 08:28:58

That's the only reason. 08:29:02

Does that make sense? 08:29:04

Q. Yes, it does. 08:29:05

Thank you. 08:29:06

Well, let's turn to one of the Lines of 08:29:07
Evidence, and when I refer to "Lines of Evidence" or 08:29:13
"LOE," you'll understand that I'm referring to the three 08:29:16
Lines of Evidence in Part 1? 08:29:19

A. Yes. Yes. 08:29:22

I apologize. 08:29:28

Q. I wanted to give you time to remind yourself 08:29:29
of -- 08:29:32

A. Yes. 08:29:33

Q. -- actually giving the -- the verbal answer. 08:29:33

Thank you very much. 08:29:36

So, turning to Master Exhibit Number 6 at page 08:29:36
6, I want to ask you some questions to help us 08:29:40
understand how this methodology is intended to work; is 08:29:46
that okay? 08:29:52

A. Yes. 08:29:52

Q. Okay. And specifically with reference to 08:29:53
sediment toxicity I'm actually looking at the last 08:29:58
paragraph on the top of page 6 dealing with the 08:30:01
averaging process. 08:30:06

Are you with me? 08:30:21

A. Yes. 08:30:22

Q. And you see there's a statement about taking 08:30:22
an average of all test categories. 08:30:26

Do you see that? 08:30:29

A. Yes. 08:30:30

Q. And by "test categories" could you describe to me perhaps with reference from the prior page what is meant by that? 08:30:32 08:30:36 08:30:39

A. Yes. 08:30:40

So in Part 1 you generally -- you're -- you're required -- umm -- to perform both the survival and sublethal toxicity test and the -- umm -- your response. 08:30:44 08:30:51 08:31:00

The -- the test response will fit in to one of these categories on Table 4. 08:31:12 08:31:15

Do you see that? 08:31:18

Q. I do. 08:31:20

A. Okay. Umm -- so -- umm -- if -- if you get a response, it will fit in to either nontoxic, low toxicity, moderate toxicity, or high toxicity. 08:31:21 08:31:29 08:31:33

If you have two -- if you have -- what that -- umm -- what number 4 is trying to explain is -- if you -- when you're trying to -- categorize your overall toxicity, you would -- umm -- you would take the average of the two. 08:31:39 08:31:40 08:31:45 08:31:52 08:31:58

Umm -- so you're -- 08:32:01

I'm sorry. I'm not explaining this very well. 08:32:02

But if your survival is nontoxic and your Mytilus is low toxicity -- 08:32:06 08:32:13

Do you see those? 08:32:16

Q. So let's -- let's walk through this. 08:32:18

So let's say survival is nontoxic and it's for the first critter. 08:32:20 08:32:26

A. Yes. 08:32:27

Q. How do you pronounce that? 08:32:28

A. Eohaustorius. 08:32:29

Q. Eohaustorius? 08:32:30

I'll spell it. It's E-o-h-a-u-s-t-o-r-i-u-s. 08:32:32

So if Eohaustorius is nontoxic, let's assume there's a hundred percent survival. 08:32:39 08:32:43

A. Yes. 08:32:46

Q. Okay. 08:32:46

A. And then Mytilus, M-y-t-i-l-u-s. 08:32:47

Q. And the example you gave for Mytilus would be low toxicity? 08:32:54 08:32:57

A. Yes. 08:32:58

Q. Let's assume it's 79 percent. 08:32:58

A. Umm -- right. 08:33:06

And so -- 08:33:06

Wait. No. 08:33:07

Mytilus, let's assume it's -- umm -- 08:33:08

Oh, yeah. 08:33:12

Umm -- let's assume -- 08:33:14

Umm -- 08:33:29

Umm -- yeah, okay. 08:33:30

So it's significantly, getting in to more 08:33:31
details -- umm -- Mytilus if you're significantly 08:33:34
different from controls, and you're at 79 percent, you 08:33:39
would become -- umm -- you would be -- you would fall in 08:33:42
to the category of low toxicity. 08:33:46

For the Eohaustorius we're at -- we're in the 08:33:48
nontoxic category. 08:33:52

So in this case what number 4 says is that you 08:33:53
would -- you would take the result -- umm -- you would 08:33:58
round up -- umm -- so -- umm -- you would use -- umm -- 08:34:01
low toxicity. 08:34:08

Q. Then how do you actually take the average? 08:34:12

What numbers would you use to come up with the 08:34:16
average for those two tests that we just ran through? 08:34:20

Would it just be a function of taking 100 08:34:35
percent survival for the Eohaustorius and 79 percent for 08:34:38
Mytilus, adding those two and dividing by two? 08:34:43

A. No. 08:34:47

Q. Okay. Then please explain. 08:34:47

A. Umm -- I think the term "average" was used in 08:34:54
there -- umm -- because there is an option to run more 08:35:01
than two tests, and -- umm -- 08:35:07

Q. More than two tests? 08:35:21

A. Toxicity tests. 08:35:24

Q. So let's assume then that you've got more than two tests for sediment toxicity with lethality being the end point; okay? 08:35:26 08:35:30 08:35:38

A. (Nodding head) 08:35:40

Q. Yes? 08:35:40

A. Oh. Yes. 08:35:41

Q. This is a very particularized kind of proceeding, Mr. Beegan, so I know you haven't been deposed before, so we'll -- we'll get you there in terms of avoiding the -- the nods of the head. 08:35:42 08:35:48 08:35:53 08:35:56

Can -- can you -- 08:35:58

Let's assume for purposes of -- of this example then that there's an Eohaustorius test and a Rhepoxynius test. 08:36:02 08:36:06 08:36:14

Those are both for the lethality end point; correct? 08:36:14 08:36:18

A. Yes. 08:36:19

Q. And Rhepoxynius is spelled R-h-e-p-o-x-y-n-i-u-s. 08:36:20

So if you had two results or two tests for lethality -- 08:36:29

Lethality is l-e-t-h-a-l-i-t-y. 08:36:39

How would you apply paragraph 4 on page 6 to determine the appropriate result? 08:36:42 08:36:47

A. Umm -- you would take the average -- well -- 08:36:48

umm -- you would take the average of the -- the three results -- umm -- so in the case of -- well -- umm -- nontoxic -- if you had two nontoxic and you had a low toxicity -- umm -- let's assume that Rhepoxynius was nontoxic. 08:37:02 08:37:12 08:37:27 08:37:32 08:37:38

Your overall response for that LOE would be nontoxic. 08:37:39 08:37:43

Q. So the "averaging" refers to the categories then? 08:37:44 08:37:49

A. Yes. 08:37:49

Q. As opposed to the specific numeric values? 08:37:50

A. Yes. But -- 08:37:54

Umm -- yes. Yes. 08:37:58

Q. And so that's what's meant by the "average of all test response categories" on the top of page 6? 08:38:01 08:38:09

A. Yes. 08:38:12

Q. The average of the categories? 08:38:13

A. Yes. Yes. 08:38:17

Q. The average of the categories determines the final category; correct? 08:38:18 08:38:21

A. Yes. 08:38:23

Q. What happens if you have more than one test for any particular test species -- if you know? 08:38:23 08:38:33

A. I don't know. 08:38:45

We -- I don't think -- 08:39:05

Umm -- in terms of a recent -- umm -- I guess you could look at it in terms of the most recent data would be applied. 08:39:08 08:39:19 08:39:26

I guess when you have multiple tests for a single station of the same organism -- umm -- I guess there are a variety of ways you could apply it. 08:39:29 08:39:34 08:39:42

If you're looking at temporal changes over time, you would use them separately -- umm -- but I don't think we -- thought of taking a -- a single grab for a particular -- from a particular sampling event and running multiple -- umm -- independent tests on that sample -- or that sediment. 08:39:45 08:39:47 08:39:51 08:40:05 08:40:11 08:40:21

Q. When you say you did not contemplate, you mean you did not comment on it in Part 1? 08:40:26 08:40:29

A. Umm -- yes. 08:40:31

Q. Part 1 requires a minimum of one test for lethality; correct? 08:40:34 08:40:46

A. Uh-huh, yes. 08:40:46

Q. And a minimum of one test for sublethal; correct? 08:40:47 08:40:56

A. Yes. 08:40:56

Q. And, in fact, on pages 4 and 5 you see that under Section F "sediment toxicity" refers to those minimums. 08:40:57 08:40:59 08:41:04

Both in F paragraph 1 and F paragraph 2, it 08:41:05

says "a minimum of 1"? 08:41:09

Do you see that, sir? 08:41:11

A. Umm -- yes. 08:41:11

Q. And that's -- when you indicated that there's an option to run more than two tests, you're referencing the fact that it's allowable to exceed the minimum of one; correct? 08:41:18 08:41:23 08:41:29 08:41:33

A. Yes -- umm -- one each. 08:41:34

Q. Right. 08:41:43

"One each" meaning? 08:41:43

A. One sublethal test and one acute test. 08:41:44

So that's the minimum. You can go beyond the minimum. 08:41:47 08:41:50

Q. Is there any maximum that's specified in the Part 1? 08:41:51 08:41:56

A. No. 08:41:56

Q. Is there some data cutoff where you've got too much data? 08:42:02 08:42:08

A. No. 08:42:10

Q. And on page 4 of Part 1 under paragraph E, "Laboratory Testing" -- are you with me? 08:42:15 08:42:26

A. Yes. 08:42:27

Q. It says "All samples will be tested in accordance with -- with certain methods." 08:42:28 08:42:32

Do you see that? 08:42:34

A. Yes. 08:42:35

Q. And so the SQO requires the -- the actual analysis of all samples; is that right, sir? 08:42:35 08:42:42

A. Could -- could you rephrase that last part. 08:42:46

Q. Sure. 08:42:56

A. It requires the analysis of all samples? 08:42:56

Umm -- I guess I don't understand what you mean by "all samples." 08:42:59 08:43:02

Q. What -- what is meant by the reference to "all samples" in paragraph -- 08:43:04 08:43:07

A. Oh, okay. Okay. 08:43:10

So basically what Paragraph E is saying is that -- umm -- for -- umm -- for the -- for the samples that you are applying or collecting for the purposes of assessing sediment quality in relationship to this direct effects SQO, the community protection SQO, all samples will be tested in accordance with these particular methods where they exist. 08:43:13 08:43:18 08:43:27 08:43:32 08:43:37 08:43:42 08:43:45

Q. Okay. Thank you. 08:43:50

Now, going back to Table 4 on page 5, you need a minimum of two samples to apply Table 4; correct? 08:43:57 08:44:09

A. Umm -- two samples? 08:44:15

Q. Two tests? 08:44:20

A. Two tests, -- 08:44:22

Q. Thank you. 08:44:23

A. -- two samples. 08:44:24

Q. How would you distinguish those two? 08:44:25

A. Umm -- you can take a -- 08:44:28

MR. SINGARELLA: Bless you. 08:44:37

MR. BROWN: Thank you.

THE WITNESS: -- a benthic grab. 08:44:40

There's -- you -- a single grab could 08:44:43
potentially use -- be used for multiple tests or 08:44:50
multiple analyses. 08:44:54

So -- usually a single grab sample, regardless 08:44:58
of how you split up the -- apportion the sediment in 08:45:09
that grab for different tests, it's considered same 08:45:15
sample, or the same station -- yeah, same station -- umm 08:45:20
-- sample -- or you may need to take multiple grabs at 08:45:27
the same time. 08:45:35

Those would all be considered the same -- the 08:45:37
same sample or the same -- 08:45:44

Yeah, the same sample. 08:45:46

BY MR. SINGARELLA: 08:45:48

Q. So if you had a grab sample with multiple test 08:45:49
results, would you use those test results to apply Table 08:45:58
4? 08:46:02

A. Yes. 08:46:03

Q. And, referring to page 4 of Part 1 under C, 08:46:04
"Water Bodies," C(2) -- 08:46:30

Are you with me, Mr. Beegan? 08:46:33

A. Yes, I am. 08:46:38

Q. Do you see the reference to situations where LOE, referring to Lines of Evidence, measurement tools are unavailable? 08:46:43 08:46:47 08:46:53

Do you see that, sir? 08:46:54

A. Yes. 08:46:55

Q. So in those situations there's an alternative procedure described in Part 1; correct? 08:46:55 08:47:00

A. Yes. 08:47:04

Q. And that's described in Section V.J; right? 08:47:04

A. Yes. 08:47:12

Q. I don't want to go to section V.J yet. 08:47:13

I simply want to distinguish situations where the tools are unavailable from situations where they are available. 08:47:20 08:47:23 08:47:25

Are you with me? 08:47:26

A. Yes. 08:47:27

Q. So in situations where the tools are available, you use them; right? 08:47:27 08:47:30

A. Yes. 08:47:31

Q. And in situations where the measurement tools are unavailable there's an alternative approach provided in Part 1; correct? 08:47:32 08:47:35 08:47:40

A. Yes. 08:47:42

Q. Okay. Let's turn to page 17, please, of Part 1. 08:47:42 08:47:51

Now, help me understand what Part 1 consists of. This section, F, "Stressor Identification," is it -- is it correct that this is part of a program of implementation? 08:47:52 08:48:08 08:48:14 08:48:21

A. Yes. 08:48:22

Q. Is it to be -- is stressor identification to be distinguished from the SQOs themselves? 08:48:22 08:48:27

MR. FUCHS: Objection. Calls for a legal conclusion. 08:48:29 08:48:32

You can answer. 08:48:32

THE WITNESS: Yes. 08:48:34

BY MR. SINGARELLA: 08:48:35

Q. And in the first sentence of Section F it refers to sediments that failed to meet the narrative SQOs in accordance with Sections V and VI. 08:48:39 08:48:44 08:48:49

Do you see that? 08:48:54

A. Yes. 08:48:55

Q. Is that meant to indicate that the narrative SQOs are contained in Sections V and VI? 08:48:55 08:49:01

MR. FUCHS: Objection. Vague and ambiguous. 08:49:07

You can answer if you can answer. 08:49:19

THE WITNESS: Umm -- can you repeat that, Mr. Singarella? 08:49:21 08:49:50

MR. SINGARELLA: Absolutely. 08:49:50

Could you read it back for Mr. Beegan. 08:49:51

MR. SINGARELLA: She can repeat it. 08:49:55

THE WITNESS: Okay. 08:49:57

(Record Read) 08:49:57

THE WITNESS: No. What -- what it -- umm -- 08:50:13

what that sentence was intended to mean was if you've 08:50:21

failed the narrative sediment quality objective that 08:50:34

protects benthic communities as determined within 08:50:41

Sections V and VII, so you're -- your interpretation 08:50:49

tools are in Section V; right? 08:50:52

Am I -- the LO -- the LOE MLOE are in V and 08:50:54

VII. 08:51:00

If -- if through those analyses you've 08:51:02

demonstrated a failure to meet that narrative SQO, then 08:51:08

you move on to stressor identification under certain 08:51:14

conditions, which come in in the next couple of 08:51:23

sentences in Section F. 08:51:28

BY MR. SINGARELLA: 08:51:30

Q. Okay. Turning back to page 13 of Part 1 VI, 08:51:31

Human Health -- 08:51:37

Are you with me? 08:51:54

A. Yes. 08:51:55

Q. Did I understand you to testify that VI 08:51:55

contains Lines of Evidence? 08:51:59

A. That was my mistake. 08:52:02

I thought -- umm -- 08:52:19

It's more generalized than what -- after 08:52:35

reading it, it's more general than how I had stated my 08:52:39

previous response. 08:52:46

So -- umm -- those Sections V and VI still 08:52:47

describe how you would determine whether sediments -- 08:52:53

umm -- meet or exceed both the human health and the 08:53:01

benthic community sediment quality objective. 08:53:09

If you've determined that you've had an 08:53:14

exceedance under certain conditions, you move on to 08:53:20

stressor identification. 08:53:23

That's -- 08:53:24

Q. Well, I notice that -- that Part 1 is called a 08:53:30

"Water Quality Control Plan;" correct? 08:53:35

A. Yes. 08:53:36

Q. And the title actually of Part 1 does not 08:53:36

refer to Sediment Quality Objectives. 08:53:40

Do you see that? 08:53:43

A. Yes. 08:53:44

Q. So my questioning here is really to try to get 08:53:45

at the structure of this to understand where the 08:53:49

Sediment Quality Objectives are versus a program of 08:53:51

implementation. 08:53:55

Do you -- do you understand? 08:53:56

A. Yes. Yes. 08:53:57

Q. Do you have something that can help us on that? 08:53:59 08:54:02

A. Umm -- 08:54:03

MR. FUCHS: Objection. Vague and ambiguous. 08:54:04

THE WITNESS: The -- umm -- the narrative objectives that -- the Sediment Quality Objectives are solely contained in -- Section IV. 08:54:08 08:54:16 08:54:23

BY MR. SINGARELLA: 08:54:41

Q. IV? 08:54:42

A. Yes. 08:54:42

Q. And -- 08:54:43

A. Everything else would be Program of Implementation. 08:54:48 08:54:56

Q. Thank you. 08:54:56

Turning back to stressor identification on page 17, please, there are certain components of stressor identification identified in Section F; correct? 08:55:04 08:55:10 08:55:26 08:55:30

A. Yes. 08:55:31

Q. And one is a confirmation and characterization process; correct? 08:55:32 08:55:36

A. Yes. 08:55:38

Q. And the second is a pollutant identification process or step; correct? 08:55:38 08:55:42

A. Yes. 08:55:44

Q. And then the third is a source identification step; correct? 08:55:49 08:55:53

A. Yes. 08:55:53

Q. And to do a complete stressor identification would it be important to complete those three steps? 08:55:58 08:56:07

A. Not necessarily all three. 08:56:10

Umm -- I believe the third one is -- umm -- not absolutely necessary to determine which pollutant is causing a problem. 08:56:36 08:56:45 08:56:57

Q. Okay. And in the first paragraph of -- of this section it indicates that "The Water Boards shall direct the regional monitoring coalitions or Permittees to conduct stressor identification." 08:56:58 08:57:13 08:57:22 08:57:26

Do you see that? 08:57:28

A. Yes. 08:57:28

Q. So with reference to that first paragraph stressor identification is mandatory? 08:57:29 08:57:32

A. No. 08:57:41

Q. Well, it says "shall direct." 08:57:42

In what way is that nonmandatory? 08:57:43

A. Well, there's -- umm -- there's certain criteria, and that's what I was getting at. 08:57:46 08:57:52

If -- umm -- if your sediments are characterized as possibly impacted, but there are no 08:57:57 08:58:05

clearly likely impacted sites, shall -- umm -- conduct confirmation monitoring. 08:58:13 08:58:21

Q. But not necessarily stressor identification? 08:58:23

A. Correct. 08:58:27

Q. And with respect to the confirmation and characterization step -- 08:58:27 08:58:37

Are you with me? 08:58:40

A. Yes. 08:58:42

Q. That is to confirm pollutant-related impacts; correct? 08:58:42 08:59:01

MR. FUCHS: Objection. Vague and ambiguous. 08:59:01

THE WITNESS: It's to confirm -- 08:59:13

In general, yes, I would -- I would say yes. 08:59:18

BY MR. SINGARELLA: 08:59:22

Q. Well, I really don't want this to be either vague or ambiguous. So -- 08:59:22 08:59:24

A. Okay. Well, there's -- 08:59:27

MR. FUCHS: Wait for a question. 08:59:29

BY MR. SINGARELLA: 08:59:30

Q. So in the fourth paragraph there's an indication that step 1 "consists of confirmation and characterization of pollutant related impacts;" correct? 08:59:32 08:59:37 08:59:41

A. Yes. 08:59:46

Q. You wouldn't need a step to confirm pollutant-related impacts unless they required 08:59:46 08:59:51

confirmation; correct? 08:59:56

A. Say that once more, please. 08:59:59

Restate. 09:00:04

Q. You wouldn't need a -- a step to confirm pollutant-related impacts unless the impacts needed to be confirmed? 09:00:05 09:00:08 09:00:13

MR. FUCHS: Objection. Total logical. 09:00:15

MR. SINGARELLA: But important. 09:00:22

And we're only here because of prior objections. 09:00:27 09:00:29

THE WITNESS: The -- the -- the direct effects assessment described in -- umm -- Section V, it was designed to develop -- developed and intended to categorize sediment quality in a -- a robust manner using multiple Lines of Evidence. 09:00:33 09:00:46 09:00:58 09:01:09 09:01:24

By using those three Lines of Evidence, by applying them, the framework -- 09:01:32 09:01:38

By using three Lines of Evidence the -- the -- the station classification is stronger than relying on one or two Lines of Evidence. 09:01:47 09:01:52 09:01:57

The end result is that you have an integrated scheme that provides a series of categories for each station. Those stations are, you know -- categories are unimpacted, likely unimpacted, possibly impacted, likely impacted, clearly impacted. 09:02:01 09:02:09 09:02:16 09:02:23 09:02:28

That's it. They don't identify what the problem is. They don't identify what the specific pollutant is. 09:02:31 09:02:35 09:02:38

And, as a result, if there is some degradation to proceed further you would need to perform stressor identification to get at the cause. 09:02:43 09:02:52 09:03:00

So -- I'm trying to -- assess whether I'm getting at your question or not. 09:03:07 09:03:15

BY MR. SINGARELLA: 09:03:18

Q. Thank you. 09:03:19

Yes. That's really helpful. 09:03:19

Okay. Let's approach it a slightly different way. There's a reference to confounding factors -- 09:03:25 09:03:28

A. Yes. 09:03:30

Q. -- under stressor identification. 09:03:30

Correct? 09:03:32

A. Yes. 09:03:32

Q. For example. Physical disturbance; right? 09:03:33

A. Correct. 09:03:36

Q. Or nontoxic constituents? 09:03:36

It says "nonpollutant constituents." 09:03:41

Do you see that? 09:03:45

A. What page? 09:03:46

Q. I'm sorry. 09:03:47

That sentence was compound. 09:03:47

I'm on page 17. I'm referring to confounding factors on F(1). 09:03:49 09:03:53

Are you with me? 09:03:54

A. Yes. 09:03:55

Q. And there's a reference "nonpollutant constituents." 09:03:56 09:03:58

Do you see that? 09:04:00

A. Yes. 09:04:00

Q. As opposed to "toxic constituents;" correct? 09:04:01

A. Yes. 09:04:05

Q. What are "nonpollutant constituents"? 09:04:05

MR. FUCHS: Do you mean examples or a definition? 09:04:14 09:04:15

MR. SINGARELLA: I'm going to ask for both, but let's start, how would you define "nonpollutant constituents"? 09:04:19 09:04:20 09:04:25

THE WITNESS: I guess temperature, turbidity -- umm -- debris -- umm -- 09:04:27 09:04:50

BY MR. SINGARELLA: 09:05:12

Q. Anything else, Mr. Beegan? 09:05:12

A. I'm -- I'm sure there are others, -- 09:05:13

Q. Okay. 09:05:15

A. -- but I can't think of any at the moment. 09:05:16

Q. And then the reference to "physical disturbance," to what does that refer? 09:05:18 09:05:20

A. That could refer to anything that would disturb the surficial sediments, prop wash, dredging, scour from currents or tidal action. 09:05:24 09:05:30 09:05:46

In shallow waters can be wave action. 09:05:50

I'm sure there's others. 09:06:02

I -- that's it off the top of my head. 09:06:04

Q. Thank you. 09:06:07

And how are these confounding factors? 09:06:07

VOICE ON TELEPHONE: Hello? 09:06:24

MR. SINGARELLA: Good morning. 09:06:26

We're in the middle of a deposition here. 09:06:27

Are you intending to sit in on the deposition of Mr. Beegan? 09:06:29 09:06:32

VOICE ON TELEPHONE: Yes, I am. 09:06:33

MR. SINGARELLA: Well, then welcome. 09:06:35

VOICE ON TELEPHONE: Thank you.

MR. SINGARELLA: And who are you? 09:06:36

VOICE ON TELEPHONE: This is Kristin Reyna for the City of San Diego. 09:06:37 09:06:40

MR. SINGARELLA: Welcome, Kristin. 09:06:40

This is Paul Singarella. 09:06:41

And while we're at it, just so Mr. Beegan knows who's in the room and who's on the phone, a couple of gentlemen have come in since we started, so let's have them introduce themselves on the record. 09:06:43 09:06:46 09:06:48 09:06:51

MR. BROWN: Hi. I'm Bill Brown. I'm appearing for the Port of San Diego. 09:06:53 09:06:55

MR. TRACY: I'm Mike Tracy. I represent BAE Shipyards. 09:06:57 09:07:01

MR. SINGARELLA: Thank you. 09:07:01

Always nice to know who's in the room -- as a courtesy to you. 09:07:05 09:07:08

BY MR. SINGARELLA: 09:07:09

Q. Okay. So we were in the middle of a question, but let me -- let me tee it back up for you. 09:07:12 09:07:15

I was asking about confounding factors and how are physical disturbance and nonpollutant constituents -- how are they confounding factors. 09:07:18 09:07:21 09:07:25

A. They can -- confounding factors -- umm -- a fact -- the response of the indicator in a way that -- umm -- masks the signal you're looking for, and that is the primary reason why we are using multiple Lines of Evidence, to assess sediment quality. 09:07:28 09:07:39 09:07:49 09:08:03 09:08:11

Q. And what signal are you looking for? 09:08:18

A. We're looking for a toxic pollutant-related signal. 09:08:20 09:08:25

Q. And by "toxic pollutant" to what are you referring? 09:08:25 09:08:29

A. Priority pollutants, anything -- umm -- current use pesticides, priority pollutants, that sort 09:08:29 09:08:40

of thing, as opposed to -- umm -- nutrients, which will have a -- could have an effect, but -- for the purpose of this plan we didn't consider nutrients as toxic pollutants. 09:08:45 09:08:52 09:08:59 09:09:06

Q. So nutrients also could be a confounding factor? 09:09:08 09:09:12

A. Umm -- yes, there are many things that could be -- that could -- umm -- be considered potentially confounding. 09:09:13 09:09:22 09:09:28

Q. And the idea under step one of stressor identification, at least in part, is to confirm the absence of such confounding factors? 09:09:29 09:09:33 09:09:36

A. Yes. 09:09:39

Q. And that is because the SQOs themselves do not rule out confounding factors? 09:09:45 09:09:55

MR. FUCHS: Vague and ambiguous. 09:09:59

Calls for a legal conclusion. 09:10:01

MR. SINGARELLA: I'm asking for the technical here, Mr. Beegan. 09:10:03 09:10:05

BY MR. SINGARELLA: 09:10:07

Q. There's a confirmation step under stressor identification; correct? 09:10:07 09:10:10

A. Correct. 09:10:11

Q. And one of the things being confirmed is the absence of confounding factors; right? 09:10:12 09:10:14

A. Correct. 09:10:17

Q. And confounding factors are those nontoxic constituent factors that could result in the signal; right? 09:10:18 09:10:24 09:10:27

A. Correct. 09:10:28

Q. And the signal that you're looking for is impacts to sediment from toxic constituents; correct? 09:10:28 09:10:33

A. Yes. 09:10:38

Q. And so step 1 of stressor identification is to rule out those confounding factors as the cause of the signal; right? 09:10:38 09:10:47 09:10:52

A. Correct. 09:10:54

Q. Because you've got a sequential process here; is that right? 09:10:54 09:10:59

A. Yes. 09:11:00

Q. And at this step in the sequence it's important to take that confirmation step; right? 09:11:00 09:11:04

A. Yes. 09:11:08

Q. And it's also important to do characterization of the impact; correct? 09:11:09 09:11:14

A. Correct. 09:11:16

Q. And so step 1 of stressor I.D. is not only to confirm the absence of confounding factors, but also to characterize the pollutant-related impacts; correct? 09:11:20 09:11:25 09:11:28

A. Correct. 09:11:32

Q. And under the last paragraph of page 17 under "Physical Alteration" do you see the reference to "prop wash from passing ships"? 09:11:33 09:11:40 09:11:44

A. Yes. 09:11:47

Q. And how can that mask the signal? 09:11:54

A. It can -- umm -- disturb or -- umm -- it could disturb the benthic community, but that's just one of the Lines of Evidence. 09:11:58 09:12:13 09:12:25

Q. And prop wash from passing ships is another form of a stressor; correct? 09:12:26 09:12:31

A. I don't know if I would define that as a "stressor." 09:12:33 09:12:49

Only -- umm -- that's -- I'm having a hard time with that label associated with prop wash. 09:12:53 09:13:06

Let's proceed. 09:13:15

Q. Well, can -- can prop wash from passing ships produce a nonreference condition? 09:13:18 09:13:22

A. Absolutely. 09:13:29

Q. And on page 17, last paragraph, does that paragraph basically tie the concept of prop wash to the concept of stressors? 09:13:29 09:13:37 09:13:45

A. Oh, okay. 09:13:47

Yes. Yes. Yes. 09:13:53

Q. There's -- the first sentence refers to prop wash from passing ships; correct? 09:13:54 09:13:57

A. Yes. 09:13:59

No, I agree with you now. 09:13:59

Q. Yeah. 09:14:01

Thank you. 09:14:01

And then just to -- just to be clear here, though, then the second sentence refers to "these types of stressors"? 09:14:02 09:14:04 09:14:07

A. Right. Right. 09:14:08

Q. Referring back to, among other things, prop wash from passing ships; right? 09:14:09 09:14:12

A. Yes. 09:14:14

Q. And stressors for purpose of the deposition is spelled s-t-r-e-s-s-o-r-s. 09:14:14 09:14:21

And the second sentence refers back to very fine or coarse grain size as well; correct? 09:14:21 09:14:25

A. Correct. 09:14:28

Q. Which is another stressor; right? 09:14:28

A. Yes. 09:14:31

Q. That can also produce a nonreference condition; right? 09:14:32 09:14:35

A. Yes. 09:14:36

Q. And what is that a reference to, grain size? 09:14:36

Help me understand that. 09:14:43

A. Umm -- grain size can affect the communities. 09:14:47

It's a habitat factor. 09:14:58

Umm -- some organisms, many organisms, prefer fine grain sediments. If you get to the point where the sediments are too coarse, you'll see a change in the community, you'll also see potential effects with your toxicity test response over -- umm -- a range of grain sizes. 09:15:03 09:15:10 09:15:15 09:15:22 09:15:35 09:15:44

But most toxicity tests have been evaluated as well as the -- umm -- the benthic community have been evaluated with regard to how they respond to those grain sizes. 09:15:46 09:15:56 09:16:01 09:16:08

Q. So, turning back to page 3, V, "Benthic community protection," the Lines of Evidence -- are you with me? 09:16:10 09:16:17 09:16:20

A. Page 3. 09:16:21

Q. You know that this section, V, does not refer explicitly to confounding factors; correct? 09:16:31 09:16:36

A. I guess I would have to read it all the way through. 09:16:41 09:16:55

Umm -- should I? 09:16:55

Q. Well, -- 09:17:02

A. Umm -- 09:17:09

Q. -- not right now. I really -- 09:17:12

Sorry. I thought you'd know offhand. 09:17:15

A. Okay. Well, I mean -- 09:17:17

Q. Is there anything in V that deals explicitly 09:17:19

with confounding factors? 09:17:27

MR. FUCHS: If you can answer without reading it, -- 09:17:38 09:17:40

THE WITNESS: Oh. 09:17:40

MR. FUCHS: -- because he just told you not to read it. 09:17:41 09:17:43

THE WITNESS: Oh, right. Okay. 09:17:44

I think "Limitations" states that there are certain -- umm -- factors -- 09:17:45 09:17:49

I don't know if it describes them as confounding factors. 09:17:56 09:17:58

BY MR. SINGARELLA: 09:18:13

Q. Yes. 09:18:14

And on the top of page 4, referring to the Lines of Evidence applied to assess biological effects, it says that the lines "can respond to stresses associated with natural or physical factors;" right? 09:18:14 09:18:21 09:18:27 09:18:32

A. Correct. 09:18:35

Q. "Such as sediment grain size;" right? 09:18:37

A. Correct. 09:18:40

Q. Which we just identified as a confounding factor; correct? 09:18:41 09:18:44

A. Correct. 09:18:45

Q. And physical disturbance, another confounding factor; right? 09:18:46 09:18:49

A. Correct. 09:18:50

Q. How about organic enrichment? 09:18:51

Is that also a potential confounding factor? 09:18:54

A. Yes. 09:18:56

Q. And to what does that refer? 09:18:56

A. Umm -- organic matter that settles on the bottom, could be plant matter, could be -- 09:18:58 09:19:20

Q. So plant matter could -- 09:19:36

A. Animal matter -- umm -- that's it. 09:19:38

Q. So plant -- plant matter -- 09:19:59

A. Umm -- I guess organic matter from a discharge. 09:20:07 09:20:11

Umm -- I can't think of anything else. 09:20:15

Q. I'm sorry I interrupted you. I didn't realize you were in mid-thought there. 09:20:21 09:20:26

So plant matter is a confounding factor; is that right? 09:20:29 09:20:34

A. An enrichment of organic matter composed the -- umm -- dead and decaying aquatic plant matter -- umm -- anything -- any enrichment in organic detritus that settles on the bottom could alter the community, and it could -- umm -- affect your toxicity test, but there are measurements that you would take, total organic carbon and ammonia, that could allow you to factor in -- umm -- those issues. 09:20:35 09:20:51 09:21:03 09:21:12 09:21:27 09:21:40 09:21:50 09:22:00

Q. And with specific reference to this first full sentence on page 4 -- 09:22:02 09:22:09

Are you with me? 09:22:11

A. Umm -- 09:22:12

Q. I do want you to read that sentence, the one that begins "The LOEs apply." 09:22:16 09:22:20

A. "The LOEs applied to assess biological effects can respond to stresses associated with natural or physical factors, such as sediment grain size, physical disturbances, or organic enrichment." 09:22:25 09:22:29 09:22:33 09:22:36

Q. Yes. 09:22:39

Is that -- is that sentence intended to inform the reader that the LOEs in this section may mistake an impact caused by these other stressors for an impact caused by toxic pollutants? 09:22:41 09:22:47 09:22:58 09:23:02

A. Yes. 09:23:04

Q. It -- that sentence is not meant to inform the reader that these confounding factors are actually taken in to account in this section, V? 09:23:11 09:23:16 09:23:20

MR. FUCHS: Vague and ambiguous as to what "taken in to account" means. 09:23:25 09:23:27

THE WITNESS: Well, let me think about that for a minute; okay? 09:23:37 09:23:46

MR. SINGARELLA: Sure. 09:23:47

THE WITNESS: The -- the -- 09:23:48

As I said before, the multiple Lines of Evidence are used to minimize the potential for confounding factors to -- to drive the -- overall assessment. 09:24:09 09:24:13 09:24:22 09:24:37

Umm -- the -- the way the categories were set up, the station categories, the -- at the high end we are very confident or most confident that issues are pollutant driven. 09:24:42 09:24:49 09:24:59 09:25:07

That's what I mean by the categories of likely or clearly impacted, where you have responses from at least two indicators that are relatively strong. 09:25:10 09:25:13 09:25:22

We have a category of possibly impacted where the potential for confounding factors to -- umm -- affect the station category are greater, and that is why -- umm -- we include it, a lot of this -- a lot of these -- umm -- these -- umm -- texts about, you know, things to be concerned about. 09:25:32 09:25:41 09:25:50 09:25:58 09:26:10 09:26:17

The whole idea with this framework is to use multiple Lines of Evidence so you're confident in your assessment, acknowledging that the tools are imperfect. 09:26:20 09:26:25 09:26:30

Once you've completed your assessment there's still a chance that things are driven by nonpollutant-related stressors. 09:26:39 09:26:42 09:26:51

At the high end likely clearly that's unlikely the way the indicators were developed, but there's a 09:26:54 09:27:04

chance, so you want to rule those out first. 09:27:08

The other thing to do is to make sure that -- 09:27:19

umm -- the endusers have a thorough understanding of 09:27:25

their data, they've reviewed it, they've looked at 09:27:32

controls, they've looked at ammonia measurements, 09:27:37

compared with, you know, what the guidance or the EPA 09:27:42

manual says is within range, because those things are 09:27:50

important even if confounding factors -- umm -- aren't a 09:27:54

problem. 09:28:02

Q. Referring to data and controls, would 09:28:05

replicate help rule out the potential for confounding 09:28:18

factors causing the signal? 09:28:20

A. Potentially. 09:28:25

Q. And, Mr. Beegan, at one point with respect to 09:28:29

the possible impacting category did you recommend that 09:28:34

possibly impacted be a category that actually satisfied 09:28:42

the SQO protective measure? 09:28:47

MR. FUCHS: That -- that would, I think, fall 09:28:53

outside the scope of the inquiry permitted here insofar 09:28:59

as it invades the mental processes of the 09:29:03

decision-makers and involves extra record evidence 09:29:06

unless you're asking whether the SQOs themselves contain 09:29:12

that question. 09:29:20

If you're saying in the development of the 09:29:21

SQOs did he consider that, then that's outside the 09:29:23

scope. 09:29:28

BY MR. SINGARELLA: 09:29:30

Q. During the -- during the development of the methodology there was considerable discussion about where the cut-off between protective and nonprotective is; right, Mr. Beegan? 09:29:33 09:29:35 09:29:38 09:29:41

MR. FUCHS: That -- 09:29:45

I'm sorry. That's outside the scope. 09:29:46

Are we talking about -- 09:29:48

MR. SINGARELLA: Are you telling him not to answer? 09:29:48 09:29:50

These are question about the methodology and where the line should be drawn. 09:29:51 09:29:53

This is completely relevant to how the line should be drawn in this case. 09:29:54 09:29:57

I'm not talking about San Diego Bay. I'm purposefully trying to stay away from San Diego Bay, but that cut-off is essential to our ability to defend ourselves against the use of this method in San Diego Bay. 09:29:58 09:30:01 09:30:05 09:30:11 09:30:17

I don't understand. 09:30:17

I can't believe you're going to tell him not to answer this, Dan. 09:30:18 09:30:20

MR. FUCHS: I understand where -- what you're saying, but the SQOs as they are are taken as given, and 09:30:21 09:30:24

the decisions that went in to development of them are outside the scope. 09:30:31 09:30:36

So you can ask him where the line is, but not how that line was drawn. 09:30:38 09:30:43

BY MR. SINGARELLA: 09:30:48

Q. Do you recall the discussion of where the cut-off should -- should be drawn, Mr. Beegan, during the SQO proceedings? 09:30:48 09:30:51 09:30:55

MR. FUCHS: You can answer that question. 09:30:58

THE WITNESS: Vaguely. 09:31:00

BY MR. SINGARELLA: 09:31:00

Q. And -- and do you recall that there was some debate as to whether possibly impacted should be in or outside of the protective condition? 09:31:03 09:31:07 09:31:11

MR. FUCHS: I think when we get to that question that goes with beyond the scope, and I'm going to instruct you not to answer. 09:31:14 09:31:16 09:31:19

MR. SINGARELLA: Okay. Well, we'll have to go in front of Mr. Gallagher at some point, but maybe we'll just add this up and meet with Mr. Gallagher later today, but I definitely want an answer to that question, and I think it's fundamental to the use and application of the methodology. 09:31:20 09:31:21 09:31:30 09:31:32 09:31:35

THE WITNESS: Can I take a break? 09:31:39

MR. SINGARELLA: Absolutely. 09:31:41

THE WITNESS: Okay. 09:31:43
MR. SINGARELLA: Good place to take a break. 09:31:43
THE WITNESS: Okay. 09:31:45
MR. SINGARELLA: Thank you, Mr. Beegan. 09:31:46
VIDEOGRAPHER: We're going off the record at 09:31:47
9:31 a.m. 09:31:49
(Thereupon a recess was taken at 9:31 a.m. 09:31:50
and the deposition resumed at 9:39 a.m.) 09:39:35
(Exhibit 700 was marked for Identification.) 09:39:35
VIDEOGRAPHER: We are back on the record at 09:40:35
9:40 am. 09:40:42
This is the beginning of disk number two. 09:40:43
BY MR. SINGARELLA: 09:40:45
Q. Mr. Beegan, welcome back. 09:40:46
A. Thank you. 09:40:48
Q. Mr. Beegan, I'm placing in front of you a 09:40:48
device that you brought in this morning. 09:40:52
Do you see that? 09:40:54
A. Yes. 09:40:55
Q. Is that a flash drive? 09:40:55
A. Yes, it is. 09:40:57
Q. Okay. And is it -- 09:40:58
Can you tell me what's on this flash drive? 09:41:00
A. Umm -- what's on that flash drive is -- umm -- 09:41:03
I think there's three or four folders in there. 09:41:08

Last week I had provided the State Board attorney with a list of links to State Board SQO-related documents and technical reports. 09:41:16 09:41:18 09:41:27

Umm -- this flash drive, I tried to assemble all those technical documents, Water Board documents, in to a couple of folders, and I also included -- umm -- a few peer reviewed journal articles which are not readily available on the web without a subscription or something. 09:41:37 09:41:41 09:41:44 09:41:48 09:41:56 09:42:01

And so I put all those files on that USB flash drive, so that you would be able to -- grab them for your own purposes. 09:42:03 09:42:10 09:42:16

I'm sure you have a bulk of the material, but -- just trying to make sure you had it all. 09:42:18 09:42:23

Q. We'll do our level best to try to download the information today. If we can't accomplish it today, could we get this flash drive back to you later in the week? 09:42:25 09:42:30 09:42:34 09:42:37

A. Oh, sure. 09:42:37

No problem. 09:42:38

Q. Okay. Thank you. 09:42:39

If you don't mind handing it back to me. 09:42:40

A. Oh. 09:42:43

Q. Thank you. 09:42:44

And the documents on this flash drive, how far 09:42:44

back in time do they go approximately? 09:42:49

A. Umm -- maybe 2004. 09:42:51

I'm guessing -- or -- yeah. 09:43:05

Q. And the development of the Part 1 SQOs, does that process go back to 2004? 09:43:08 09:43:15

A. Yes. 09:43:17

Q. Was it a long-term proceeding with interim work product, so-to-speak, technical reports, and the like? 09:43:18 09:43:30 09:43:35

A. Yes. 09:43:36

Q. And these were, in essence, building blocks to eventually the production of the Part 1 SQO? 09:43:36 09:43:39

A. Yes. 09:43:43

Q. I'm placing in front of you what has been marked as Exhibit 700 to your deposition, which doesn't mean that we've been here for weeks. 09:43:43 09:43:55 09:44:00

It simply means that we've taken some other depos, and we're starting each one with a unique hundred series, and you're the lucky recipient of the 700 series. 09:44:02 09:44:06 09:44:11 09:44:17

Do you see that, sir? 09:44:17

A. Yes. 09:44:18

Q. And Exhibit 700 consists of the first page of Appendix E, the comments and responses related to the adoption of the Part 1 SQOs. 09:44:19 09:44:30 09:44:34

Do you see that, Mr. Beegan? 09:44:38

A. Yes. 09:44:39

Q. And then three pages providing comments and responses to particular comments. 09:44:39 09:44:46

Do you see that, sir? 09:44:48

A. Yes. 09:44:49

Actually, four pages. 09:44:57

Q. Three pages after the first page? 09:44:59

A. Oh. Yes. Sorry. 09:45:01

Q. And did you write the responses to comments? 09:45:02

MR. FUCHS: Objection. Vague and ambiguous as to whether he wrote them alone, or with others, or how. 09:45:12 09:45:16

MR. SINGARELLA: He's objecting to the form of the question. So -- 09:45:23 09:45:25

MR. FUCHS: Oh, you can go ahead and answer if you can answer. 09:45:26 09:45:29

THE WITNESS: Oh. I -- 09:45:29

Yes, with help from others. 09:45:38

BY MR. SINGARELLA: 09:45:42

Q. And so on the second page of Exhibit 700 you see the response to comment number 60? 09:45:43 09:45:49

Do you see that? 09:45:53

A. Uh-huh. 09:45:55

MR. FUCHS: Is that a "yes"? 09:45:59

THE WITNESS: Yes. Sorry. 09:46:00

MR. SINGARELLA: Thank you, Mr. Fuchs. 09:46:02

BY MR. SINGARELLA: 09:46:04

Q. And at the end of that response there's a description of reference conditions. 09:46:04 09:46:07

Do you see that? 09:46:09

A. Yes. 09:46:10

Q. And it refers to a condition as defined by benthic indices; correct? 09:46:17 09:46:25

A. Yes. 09:46:28

Q. And to what does the phrase "benthic indices" refer? 09:46:48 09:46:54

A. Well, in reference to -- umm -- the Water Quality Control Plan Part 1 SQOs it would refer to the BRI, IBI, RBI, and the RIVPACs. 09:46:55 09:47:06 09:47:11

THE REPORTER: I'm sorry. "And the --"

THE WITNESS: RIVPACs, R-I-V-P-A-C-s. 09:47:23

BY MR. SINGARELLA: 09:47:27

Q. And those are acronyms all capitalized? 09:47:27

A. Yes. Good point. 09:47:30

Which are benthic community metrics. 09:47:33

Q. And the "reference condition" is defined by benthic indices as one in which stressors have not detectably altered the assemblage of species expected for the habitat. 09:47:40 09:47:43 09:47:48 09:47:53

Do you see that, sir? 09:47:54

A. Yes. 09:47:56

Q. And do you agree with that? 09:47:56

MR. FUCHS: That is outside the scope -- so please don't answer that question. 09:48:01 09:48:05

BY MR. SINGARELLA: 09:48:05

Q. To your knowledge is that a correct statement of the reference condition? 09:48:06 09:48:08

MR. FUCHS: That's vague and ambiguous as to what is meant by "correct." 09:48:17 09:48:19

THE WITNESS: Can I -- 09:48:23

MR. FUCHS: If you understand the question, you can -- you can answer it. 09:48:25 09:48:26

THE WITNESS: Yes. 09:48:28

BY MR. SINGARELLA: 09:48:28

Q. And the last sentence indicates that "This provides a standard to assure that sensitive species within the assemblage are protected;" correct? 09:48:30 09:48:35 09:48:42

A. Correct. 09:48:45

Q. And, tying those two sentences together, does that mean that the reference condition is considered protective? 09:48:45 09:48:50 09:48:55

A. Correct. 09:48:57

Q. Okay. And turning back to page 17 of Part 1, bottom paragraph, middle of the paragraph, there's a reference to how the confounding factors may produce a 09:48:58 09:49:32 09:49:37

nonreference condition; correct? 09:49:41

A. Yes. 09:49:43

Q. In other words, the confounding factors can produce a condition that does not appear protective; correct? 09:49:45 09:49:49 09:49:53

A. Correct. 09:49:53

Q. Even though that condition is not caused by toxic pollutants; correct? 09:49:59 09:50:04

A. Correct. 09:50:06

Q. And it is the impacts of toxic pollutants that is the subject of the SQO Part 1; correct? 09:50:06 09:50:12

A. Correct. 09:50:17

Q. And so then, turning back to page 5 of Part 1, Table 4, the categories -- are you with me? 09:50:18 09:50:29

A. Table 4, page 5? 09:50:35

Yes. 09:50:38

Q. With regard to low toxicity, can confounding factors produce low toxicity? 09:50:38 09:50:41

A. Yes. 09:50:48

Q. And with respect to moderate toxicity, can confounding factors produce moderate toxicity? 09:50:55 09:51:04

A. Potentially. 09:51:13

Q. And also high toxicity? 09:51:28

A. Potentially. 09:51:30

Q. So one of the goals of stressor identification 09:51:40

is to confirm that low, moderate, or high toxicity is not caused by confounding factors; correct? 09:51:48 09:51:57

A. I disagree with that. 09:52:01

Q. In -- in what respect do you disagree? 09:52:18

A. Stressor identification is intended to -- identify the cause of the overall integrated response of the three Lines of Evidence, which -- umm -- again, is intended to minimize the influence of confounding factors on the conclusion or the overall assessment. 09:52:22 09:52:39 09:52:52 09:53:01 09:53:05

Q. Turning to page 18 of Part 1, pollutant identification, what is the purpose of pollutant identification? 09:53:19 09:53:26 09:53:41

A. When you follow Part 1, you apply the three Lines of Evidence, you end up with simply a station categoric -- categorization for each station. 09:53:42 09:54:02 09:54:14

I've described those in the past. 09:54:17

Shall I describe them again? 09:54:20

Q. No, that's fine. 09:54:21

A. Okay. Umm -- in order to proceed further, you need to determine what is causing the problem -- umm -- so likely impacted, clearly impacted, possibly impacted, it doesn't -- the -- it only is used in the assessment. 09:54:24 09:54:31 09:54:41 09:54:46

After that to determine what the stressor is you need to determine -- you need to go down this stressor identification process. 09:54:53 09:54:55 09:54:59

As we've gone over in the past, you rule out other nonpollutant-related factors like dredging, prop wash. 09:55:02 09:55:08 09:55:15

The next step would be, okay, what pollutant is causing the problem? 09:55:15 09:55:19

Once you've identified what pollutant is causing the problem, you can -- umm -- manage it, or restore, or cleanup, or control. 09:55:21 09:55:26 09:55:33

Does that help? 09:55:52

Q. Yes. Thank you. 09:55:54

A. Okay. 09:55:55

Q. And so is pollutant identification a mandatory component of stressor identification? 09:55:56 09:56:02

A. Yes. 09:56:04

Q. And up to the point where the pollutant is identified under stressor identification has it been previously identified? 09:56:04 09:56:06 09:56:10

A. No. No. No. No. 09:56:11

Q. Now, with regard to pollutant identification, one of the recommended approaches is this TIE approach, TIE, do you see that? 09:56:19 09:56:28 09:56:32

A. Yes. 09:56:33

Q. And what is a "TIE" method? 09:56:34

A. It's -- umm -- nobody calls it "TIE." 09:56:39

Everybody refers to it as "T-I-E." 09:56:45

Just -- 09:56:47

Q. Thank you. I'm not -- 09:56:48

Shows that I'm out of the loop. I'm just not 09:56:50
in the know. 09:56:52

A. Umm -- but -- basically it's -- umm -- it's -- 09:56:53
umm -- using sediment toxicity tests to -- umm -- to 09:56:59
isolate the fraction of -- pollutants that are causing 09:57:12
the toxicity. 09:57:26

It's analogous with EPA's TIE protocols used 09:57:32
on waste water effluence, if you're familiar with that 09:57:44
process. 09:57:50

Q. Are there other methods identified in Part 09:57:51
1 -- 09:57:57

A. Yes. 09:57:58

Q. -- that will identify the fraction of 09:57:58
pollutants? 09:58:01

A. Umm -- there are other tools that you can use. 09:58:01

It's an iterative approach. 09:58:19

It -- like the assessment process itself, 09:58:23
typically requires multiple Lines of Evidence or -- umm 09:58:26
-- a series of tests, or a series of tools. 09:58:39

It's -- there's no one test that will -- umm 09:58:48
-- give you a result. It's -- those tools are not 09:59:01
available to us right now. 09:59:05

Q. When you refer to the use of multiple Lines of 09:59:07

Evidence under pollutant identification, were you referring to the MLOE approach in V? 09:59:14 09:59:16

A. No. No. 09:59:20

That's -- I was referring to the -- a multiple Line of Evidence approach in general, so that in V is a multiple Line of Evidence approach applied to the assessment process because the indicators are not as reliable when used alone. 09:59:21 09:59:30 09:59:35 09:59:38 09:59:47

So that's an example. 09:59:49

In this case it's another situation where you would use -- you would perform different analyses or tests. 09:59:51 09:59:57 10:00:03

Q. Does the TIE itself identify the specific pollutant causing the impact to the sediment? 10:00:05 10:00:08

A. Yes, but you would want to look at your data set or confirm that -- you would look for secondary confirmation -- umm -- some other way. 10:00:12 10:00:30 10:00:37

Q. So the identification would be as part of the fraction to which you referred? 10:00:41 10:00:46

A. Yes. 10:00:49

Q. So you'd narrow it down to this fraction of pollutants under the TIE; right? 10:00:50 10:00:55

A. Yes. 10:01:00

Q. Then you'd use other Lines of Evidence as part of stressor identification to identify the actual 10:01:00 10:01:04

stressor? 10:01:07

A. Yes. 10:01:08

Q. And at that point, if all goes well, you know what particular toxic agent is causing the particular impact? 10:01:08 10:01:11 10:01:16

A. Correct. 10:01:18

Q. But you wouldn't know that before that point in time? 10:01:18 10:01:21

A. No. 10:01:22

Q. With regard to page 18 under "Confirmation and Characterization" there's a reference to "mechanistic benchmarks." 10:01:32 10:01:39 10:01:42

Do you see that? 10:01:43

A. Umm -- 10:01:45

Q. Under C, little C? 10:01:45

A. Oh, yes. 10:01:49

Q. These are benchmarks that are used as part of the confirmation and characterization; correct? 10:01:49 10:01:54

A. Yes. 10:01:56

Q. And they're a specific kind of benchmark; right? 10:01:56 10:02:02

A. Yes. 10:02:02

Q. Mechanistic benchmarks; right? 10:02:03

A. Correct. 10:02:06

Q. There's no recommendation here to use 10:02:06

empirical benchmarks; correct? 10:02:09

A. Correct. 10:02:11

Q. Or consensus benchmarks; correct? 10:02:13

A. Correct. 10:02:15

Q. What are "mechanistic benchmarks"? 10:02:16

A. They are -- umm -- equilibrium-partitioning coefficients. 10:02:18 10:02:32

They're based on a relationship between -- umm -- pore water chemistry -- umm -- and -- 10:02:33 10:02:39

Well, they are -- they establish a relationship between water quality criteria and the -- the concentrations of specific chemicals in pore water. 10:02:48 10:02:50 10:02:59

MR. FUCHS: For the Reporter "pore" here is p-o-r-e as opposed to p-o-o-r. 10:03:08 10:03:13

THE WITNESS: Oh. Thank you for the -- yes.

MR. SINGARELLA: "TIE" is without periods, just all caps, T-I-E. 10:03:17 10:03:19

I told Carol this would be a challenge. 10:03:23

THE WITNESS: Do we need to -- 10:03:26

I think -- toxicity identification evaluation for -- 10:03:27 10:03:30

BY MR. SINGARELLA: 10:03:34

Q. You wouldn't use empirical benchmarks for this confirmation step? 10:03:37 10:03:41

A. No. 10:03:43

Q. Why not? 10:03:43

A. They are -- 10:03:44

MR. FUCHS: Let me interrupt during this 10:04:03

pause. 10:04:05

Why were they not adopted in the Phase 1 SQOs? 10:04:06

If that's the question, then that's outside 10:04:10

the scope. 10:04:13

MR. SINGARELLA: I don't think that was the 10:04:13

pending question. 10:04:15

MR. FUCHS: Then I'll let this one go. 10:04:16

MR. SINGARELLA: I'm trying to understand the 10:04:18

boundaries of the methodology here, what's in, what's 10:04:20

out. 10:04:23

THE WITNESS: The -- umm -- the empirical 10:04:23

guidelines don't -- umm -- they -- they -- they can 10:04:28

classify a sediment as to the potential for toxicity or 10:04:46

biological effects, but they don't perform well when 10:04:54

used to identify which particular chemical is causing 10:05:01

the problem, and -- umm -- you'll actually see that in 10:05:08

-- umm -- some of the papers written by the empirical 10:05:16

guideline developers. 10:05:20

They are -- in general the intent of those 10:05:25

empirical approaches was just to help assess, as in the 10:05:30

use of our -- umm -- chemistry Line of Evidence in the 10:05:36

MLOE framework. 10:05:41

We clearly stayed up front, don't use these chemistry values to identify which chemical is causing the problem, because that's not a fruitful or accurate approach. 10:05:43 10:05:48 10:05:53 10:06:01

Those sediment quality guidelines basically -- I believe they have similar limitations. The empirical ones have similar limitations. 10:06:02 10:06:08 10:06:15

In some cases where your -- umm -- you have one pollutant that's screaming, maybe multiples of the sediment quality guideline it -- they perform better. 10:06:19 10:06:23 10:06:34

But in general their use was intended for assessment, not for stressor identification. 10:06:44 10:06:49

Q. Uh-huh. 10:06:54

When you say "screaming," you mean exceedingly high concentrations? 10:06:55 10:06:59

A. Yeah, like two, three, five times the -- umm -- the guideline. 10:07:00 10:07:06

Q. And probably where there aren't other pollutants around? 10:07:09 10:07:12

A. Correct. There's -- we could spend a day talking about the limitations of empirical and mechanistic guidelines. 10:07:14 10:07:16 10:07:21

Q. And you probably know that I would enjoy that. It says something about me. 10:07:23 10:07:26

And I don't think Mr. Fuchs is going to allow 10:07:29

us to go down that road. 10:07:32

Okay. Okay. I won't -- not the whole day. 10:07:36

And -- nor are consensus benchmarks referred to in stressor identification; correct? 10:07:40 10:07:46

A. Correct. 10:07:48

Q. Because they wouldn't be useful for stressor identification? 10:07:48 10:07:52

A. Correct. 10:07:54

Q. And why is that? 10:07:54

A. Umm -- the consensus benchmarks are all based on these empirical guidelines, and -- umm -- so they have -- umm -- since they're based on those empirical guidelines they're still -- they still suffer from the same limitations. 10:07:55 10:08:04 10:08:16 10:08:23 10:08:26

Q. Okay. Turning to page 19 of Part 1, the third step of stressor identification which is "Sources Identification and Management Actions," do you see that? 10:08:27 10:08:38 10:08:41

A. Yes. 10:08:43

Q. And you -- you mentioned that this is a nonmandatory step in stressor identification; correct? 10:08:45 10:08:52

A. Is that -- 10:08:56

I think you're miscategorizing what I had said previously. 10:09:12 10:09:15

You -- you asked if 3 was a necessary step to determine what pollutant was causing the problem -- or 10:09:16 10:09:25

at least that's how I interpreted it. 10:09:31

Q. I see. 10:09:34

And thank you, -- 10:09:35

A. And so -- 10:09:36

Q. -- because I actually did not mean that. 10:09:37

A. Okay. 10:09:39

Q. I was referring to the -- let me -- let me explain. 10:09:39 10:09:43

I was referring to the structure of Section F, Stressor Identification. 10:09:43 10:09:47

A. Yes. Yes. 10:09:47

Q. So with regard to the structure of stressor identification, it has three principal components; correct? 10:09:48 10:09:50 10:09:53

A. Yes. 10:09:53

Q. Are they all mandatory? 10:09:54

A. Yes. 10:09:55

Q. Thank you. 10:09:56

Is it with regard to this last step of stressor identification that you reached the issue of management actions? 10:09:56 10:10:06 10:10:10

MR. FUCHS: Objection. Vague. Ambiguous. 10:10:20

BY MR. SINGARELLA: 10:10:22

Q. Do you see the reference to "management actions"? 10:10:22 10:10:25

A. Yes. 10:10:25

Q. To what does that refer? 10:10:26

A. That would refer to -- umm -- some sort of final action, -- umm -- or a cleanup would be a management action. 10:10:28 10:10:42 10:10:53

A -- reopening a permit would be a management action. 10:10:55 10:10:59

Umm -- 10:11:02

BY MR. SINGARELLA: 10:11:13

Q. Could monitoring be a management action? 10:11:14

A. Yes. 10:11:20

Q. Could monitoring natural attenuation be a management action? 10:11:21 10:11:25

A. Yes. 10:11:26

Q. Might there be no management action if it was previously determined that confounding factors masked the signal? 10:11:26 10:11:33 10:11:37

A. Yes. 10:11:37

Q. And by "previously determined" I'm referring to in steps 1 and 2 of stressor identification. 10:11:41 10:11:45

Do you understand that? 10:11:49

A. Yes. 10:11:50

Q. Did you have occasion to read the SQOs Part 1 before you came to the deposition today? 10:11:52 10:12:02

A. Oh. Oh. I've -- I've read it. 10:12:04

But did I review it for purposes of today's deposition? 10:12:08 10:12:13

No, I did not. 10:12:14

Q. Okay. Do you understand that -- that this step 3 of stressor identification is where Part 1 addresses management actions? 10:12:15 10:12:19 10:12:25

A. No. I mean, I don't understand -- or I -- I'm not -- umm -- 10:12:29 10:12:41

There are -- umm -- other sections that influence management actions. 10:12:53 10:12:59

So to say that -- umm -- that that number 3 is it is -- umm -- is -- it's kind of misleading. 10:13:03 10:13:10

Q. Okay. Thank you. 10:13:15

I certainly don't mean to mislead. 10:13:16

A. Well, I -- 10:13:18

MR. FUCHS: It's okay. 10:13:20

MR. SINGARELLA: Just no intent. 10:13:22

THE WITNESS: Yeah. 10:13:23

MR. SINGARELLA: Just getting the questions phrased properly. 10:13:24 10:13:26

THE WITNESS: No. I didn't mean to imply. 10:13:27

MR. SINGARELLA: No. We're good, Mr. Beegan. 10:13:30

BY MR. SINGARELLA: 10:13:32

Q. So -- going back to V, are management actions addressed in V? 10:13:33 10:13:44

MR. FUCHS: Objection. Vague. Ambiguous. Calls for a legal conclusion. 10:13:52 10:13:55

THE WITNESS: Umm -- I don't think so. 10:13:57

Umm -- actually -- umm -- I think I was wrong. 10:14:24

Umm -- 10:14:31

BY MR. SINGARELLA: 10:14:56

Q. In what regard? 10:14:56

A. Well, "Program Section --" VII, "Program of Implementation," -- 10:14:58 10:15:07

Q. Yes? 10:15:07

A. -- there's -- umm -- I think, various references to what I would consider management actions, even if they're not specifically called out as management actions. 10:15:08 10:15:16 10:15:25 10:15:28

Much of it or some of it is -- umm -- related to M13 permitting. 10:15:33 10:15:38

Q. You're referring to number VII, Program of Implementation? 10:15:46 10:15:50

A. Yes. 10:15:50

Q. My question was whether management actions are -- are a subject of V, the MLOE approach. 10:15:51 10:15:54

A. Oh. Umm -- 10:15:59

MR. FUCHS: Same objection. 10:16:03

THE WITNESS: I'm looking. 10:16:04

MR. SINGARELLA: Sure. Take your time. 10:16:18

THE WITNESS: Based on my quick read -- umm -- no. 10:16:21 10:17:17

BY MR. SINGARELLA: 10:17:18

Q. And so is it safe to say that after application of the V, MLOE, there are additional steps before management action? 10:17:23 10:17:30 10:17:35

A. Correct. 10:17:38

Q. Including those identified under stressor identification on page 17 through 19; correct? 10:17:41 10:17:45

A. Correct. 10:17:49

Q. Thank you. 10:17:50

Now, on page 19 of Part 1, paragraph after E refers to a situation where stressor identification or stressor I.D. yields inconclusive results. 10:17:57 10:18:06 10:18:10

Do you see that? 10:18:19

A. Yes. 10:18:20

Q. What happens in that situation? 10:18:20

A. Umm -- in that situation "The Water Board shall require --" and I'm reading -- "-- the Permitee, or regional monitoring coalition to perform a one-time augmentation to that study, or, alternatively, the Water Board may suspend further stressor identification studies pending the results of future routine sediment quality objectives on it." 10:18:21 10:18:45 10:18:50 10:18:56 10:18:58 10:19:01 10:19:03

Q. Thank you. 10:19:07

So let's try to unpackage that a little bit -- 10:19:08
A. Sure. 10:19:10
Q. -- because it's not totally apparent to me. 10:19:11
This is with respect to stations classified as 10:19:14
possibly impacted; correct? 10:19:18
A. Yes. 10:19:19
Q. Stations or sites; right? 10:19:20
A. Yes. 10:19:22
Q. And do I -- do I read this right, you -- 10:19:22
Do you -- 10:19:27
I'm sorry. 10:19:27
Strike that. 10:19:28
Where you have a possibly impacted station and 10:19:29
inconclusive results do you -- proceed to Step 3 of 10:19:34
stressor identification or do you do something else? 10:19:41
A. That depends. 10:19:44
Q. Okay. Okay. 10:19:46
A. So earlier in Part 1, I don't know the 10:19:52
specific reference, but it describes situations where 10:19:56
you only have stations that are possibly impacted. You 10:20:01
do not have stations on your site, or segment, or area 10:20:09
of concern that are likely or clearly impacted. 10:20:13
In those sites -- in those segments where you 10:20:18
just have possibly impacted, you -- can -- rather than 10:20:23
jumping on to stressor identification you reconfirm your 10:20:32

results. 10:20:36

If you still come up with possibly impacted, 10:20:38
remember what "possibly impacted" means, you have low -- 10:20:41
or the -- the level of responses are not huge, they're 10:20:46
in the area where they're -- umm -- it looks like it's 10:20:51
-- could be impacted or caused by pollutants, or 10:20:56
concentrations could be high enough, but there's a lack 10:21:02
of confidence at that -- at that level of response, 10:21:09
which is why it's called "possibly impacted." 10:21:16

But you would reconfirm it by going back, 10:21:19
resampling. 10:21:23

If you get the same results, you would do 10:21:24
stressor identification in those cases. 10:21:31

If you have indicators that are responding at 10:21:39
a low level, it may be difficult to identify what the 10:21:46
particular cause is, and this Part 1 takes that in to 10:21:50
account. 10:21:57

Q. Thank you. 10:21:57

As part of the reconfirmation step that you 10:22:01
just mentioned, if you had replicate data that hadn't 10:22:09
been yet even run through the SQO, could you use it as 10:22:16
part of reconfirmation? 10:22:21

A. Yes, assuming it -- umm -- meets, you know -- 10:22:23
it's the tests -- or the -- the -- contains the data 10:22:40
that you need to apply the multiple Line of Evidence 10:22:46

approach. 10:22:51

Q. The data to apply V of Part 1? 10:22:51

A. Yes. Yes. 10:23:00

Q. If you had such replicate date, you wouldn't have to actually resample; right? 10:23:00 10:23:04

A. Assuming it's recent, I guess, there would be a temporal concern, yeah. 10:23:06 10:23:12

Q. In other words, the results -- 10:23:25

A. Recent. Recent. 10:23:26

Q. The results would only be good for the point of time where the replicates were collected? 10:23:28 10:23:31

A. Yes. 10:23:35

Q. Is that perhaps too limiting? 10:23:37

A. It may be. 10:23:43

Umm -- having no frame of reference in terms of 10 years -- 10-year-old data, 20-year-old data, 5-year-old data, the -- the -- the way Part 1 was written -- umm -- you were -- we were trying to address recent data, data from, you know, the time that Part 1 became effective forward. 10:23:45 10:23:53 10:23:57 10:24:04 10:24:12 10:24:20

So were we necessarily thinking of grabbing 10-year-old data? No. 10:24:22 10:24:27

And -- so we're moving -- this moves forward from, you know, August 25th, I guess, of 2009. 10:24:32 10:24:36

Q. Does stressor identification produce a 10:24:41

specific pollutant concentration to be achieved? 10:25:05

A. No. 10:25:11

Q. Would Section H, Development Of Site Specific 10:25:13

Sediment Management Guidelines on page 19 of Part 1, 10:25:19

produce that specific concentration value? 10:25:23

A. That is the intent of that section. 10:25:28

MR. SINGARELLA: 701. 10:25:47

(Exhibit 701 was marked for Identification.) 10:25:50

BY MR. SINGARELLA: 10:26:01

Q. I'm placing in front of you what has been 10:26:03

marked as Exhibit 701 to your deposition. 10:26:05

Do you see that, Mr. Beegan? 10:26:08

A. Uh-huh. 10:26:09

Q. And do you recognize this document? 10:26:12

A. Umm -- yes. 10:26:14

Q. Does this look like a PowerPoint that you 10:26:22

prepared for presentation to the Regional Board in San 10:26:25

Diego? 10:26:30

A. Yes. 10:26:30

Q. At a workshop on the SQOs back in August of 10:26:30

2005? 10:26:37

A. Yes. 10:26:37

Q. Could you turn to page 2 of the exhibit, 10:26:39

please. 10:26:45

You identified three challenges associated 10:26:48

with the development of SQOs; is that right? 10:26:56

A. Yes. 10:26:59

Q. And the first bullet refers to a "shifting away from a pollutant-specific concentration-based approach." 10:27:00 10:27:07 10:27:11

Do you see that? 10:27:11

A. Yes. 10:27:12

Q. And does Part 1 do that? 10:27:12

Does it shift away from a pollutant-specific concentration-based approach? 10:27:15 10:27:19

MR. FUCHS: Let me just object to this line of questioning regarding this PowerPoint. 10:27:22 10:27:25

I'm concerned that we may be beyond the scope. 10:27:33

It's not as clear as in the previous instances where I've made this objection. 10:27:37 10:27:40

Maybe you can explain why you think this is relevant to -- umm -- application in the abstract. 10:27:42 10:27:44

MR. SINGARELLA: Sure. 10:27:50

I'll tie it together with the actual methodology. That's my intent, is to understand the boundaries of the methodology and what the methodology is. 10:27:52 10:27:54 10:27:57 10:28:00

MR. FUCHS: I'll let it go for a couple more questions, but if it doesn't look like we're in the scope, I'm going to -- 10:28:02 10:28:04 10:28:07

MR. SINGARELLA: Sure. 10:28:08

I don't understand that objection, but -- 10:28:09

BY MR. SINGARELLA: 10:28:11

Q. So the first bullet refers to a shifting away 10:28:11

from a pollutant-specific concentration-based approach; 10:28:15

right? 10:28:20

Is part one a pollutant-specific 10:28:20

concentration-based approach? 10:28:23

A. No. 10:28:25

Q. What is a "pollutant-specific 10:28:25

concentration-based approach"? 10:28:32

A. Umm -- a -- I guess what I would consider that 10:28:33

would be a -- umm -- pollutant-specific -- 10:28:46

Or I'm sorry. 10:28:49

A con -- a pollutant -- 10:28:51

Yeah, pollutant-specific concentration-based 10:28:55

value. Classic example would be a numeric water quality 10:28:59

criteria, California Toxic Rule, those types of numeric 10:29:05

values that are -- umm -- single stand-alone criteria, 10:29:12

single Line of Evidence numeric-based 10:29:20

concentration-based. 10:29:24

Q. And the third bullet refers to the "need to 10:29:24

minimize BPJ;" correct? 10:29:37

A. Yes. 10:29:39

Q. And what is "BPJ"? 10:29:40

A. Best professional judgment. 10:29:42

Q. And what does "BPJ" mean? 10:29:44

A. Umm -- it's -- umm -- the use of judgment in assessing a site or assessing -- umm -- results from various tests, making a decision as to whether a particular result is -- umm -- great enough to be of concern or not. 10:29:47 10:30:01 10:30:08 10:30:18 10:30:27

There's -- best professional judgment is -- could be applied to -- umm -- just about any aspect of sediment quality assessment. 10:30:29 10:30:39 10:30:49

I -- I don't know if I'm explaining things very well, but it's a nonprescriptive approach, where you use experience, professional experience, knowledge, understanding, to make some sort of determination. 10:30:53 10:30:56 10:31:02 10:31:08

It's not prescribed in a guidance document, or a plan, or a policy. 10:31:12 10:31:18

Q. And does the Part 1 SQO minimize BPJ? 10:31:19

A. Yes. 10:31:26

Q. Now, turning back to page 19 of Part 1, Sub-H refers to the Site-Specific Sediment Management Guidelines; correct? 10:31:34 10:31:42 10:31:45

A. Yes. 10:31:46

Q. Would that be a pollutant-specific concentration-based approach? 10:31:47 10:31:53

A. Oh, yes. Yes. 10:31:56

Q. So there was a shifting away from pollutant-specific concentration-based approaches; right? 10:32:04 10:32:14 10:32:17

A. Yes. 10:32:17

Q. Help me understand why it came back in in the last section of Part 1, Section H. 10:32:17 10:32:27

A. Okay. The -- umm -- there -- 10:32:32

Umm -- I'm trying to figure out how to state this in a way that makes sense, but -- 10:32:49 10:32:56

The assessment approach was the portion that I was referring to. 10:33:01 10:33:11

Many people want to chemic -- sediment chemistry based numeric values that could be applied broadly to all bays and all estuaries. 10:33:14 10:33:20 10:33:27

The level of science isn't there to create reliable numeric sediment chemistry values. 10:33:38 10:33:50

So that -- umm -- so we use the multiple Line of Evidence approach, the three LOE. 10:33:59 10:34:08

The chemistry is -- has confounding factors, toxicity, benthic community. They all have confounding factors. 10:34:11 10:34:16 10:34:21

So what I was referring to in the shifting away was -- umm -- to state up front that we can't develop numeric sediment chemistry-based objectives, however, once you identify the stressor and it's a 10:34:21 10:34:24 10:34:31 10:34:41

pollutant-related stressor, the -- I guess the practical way in which you manage that is to develop some sort of concentration-based approach. 10:34:51 10:34:56 10:35:01

And on a site-specific level that is the -- umm -- standard practice. 10:35:05 10:35:11

But what concentration is protective at one site may be different at another site, because of various properties of the sediment. 10:35:13 10:35:18 10:35:25

So the assessment process is based on a multiple Line of Evidence approach. 10:35:33 10:35:36

Once you've assessed, you come back to or you -- you progress to a pollutant-specific concentration-based approach that's site specific. 10:35:39 10:35:46 10:35:51

Does that make sense? 10:35:58

Q. Yes. 10:35:59

When you indicated that the level of a constituent that can cause stress at different sites can be different, can it be substantially different? 10:36:01 10:36:03 10:36:09

MR. FUCHS: Vague and ambiguous. 10:36:14

THE WITNESS: Yes. 10:36:16

BY MR. SINGARELLA: 10:36:22

Q. Can it be different by a factor of two to your knowledge? 10:36:23 10:36:26

A. Yes. 10:36:26

Q. Factor of five? 10:36:26

A. Umm -- don't know. 10:36:28

Q. Is Section H on pages 19 and 20 -- is -- is that the step needed to tie a sediment impact to a specific concentration of a toxic pollutant? 10:36:34 10:36:47 10:36:52

A. Yes. 10:36:57

Q. And, as you indicated, that can be done -- you believe that can be done on a site-specific basis; right? 10:37:03 10:37:07 10:37:10

A. Yes. 10:37:10

Q. And then the product of that work in Subsection H, pages 19 and 20, would be to define a concentration that is protective; correct? 10:37:10 10:37:18 10:37:22

A. Correct. 10:37:28

Q. And for purposes of Part 1 it would be protective of the benthic community; correct? 10:37:29 10:37:32

A. Yes. 10:37:35

Q. Meaning the animals that live in the sediment? 10:37:35

A. Yes. 10:37:38

Q. And it would specify concentration if exceeded the animals would be at risk; correct? 10:37:38 10:37:42

A. Yes. 10:37:44

Q. And, if not exceeded, you would conclude that there is a protected -- or protective state; right? 10:37:45 10:37:50

A. Correct. 10:37:55

Q. So it creates more of a bright line rule; 10:37:55

right? 10:37:59

A. Correct. 10:37:59

Q. And on page 20, Mr. Beegan, first full paragraph refers to the goal of Section H as to establish a relationship; correct? 10:38:33 10:38:37 10:38:44

A. Umm -- page 20? 10:38:47

I'm sorry. 10:38:50

Section what? 10:38:51

Q. First full paragraph, second sentence, the goal of this Section H site-specific guidelines is to establish this relationship; correct? 10:38:52 10:39:00 10:39:04

A. Oh. Yes. Yes. Yes. Yes. Yes. Yes. 10:39:06

Q. Between the exposure of the animals in the sediment and the biological effect; correct? 10:39:10 10:39:13

A. Yes. 10:39:16

Q. The "exposure" is referring to a chemical concentration; correct? 10:39:16 10:39:19

A. Yes. 10:39:20

Q. And then it says, next sentence, "Once this relationship is established;" correct? 10:39:20 10:39:23

A. Yes. 10:39:24

Q. It says once that is established then the guideline may be designated; correct? 10:39:26 10:39:30

A. Yes. 10:39:33

Q. And by "designated" do you mean specified what 10:39:33

the concentration is? 10:39:37

A. Yes. 10:39:37

Q. There's no indication that a 10:39:38

pollutant-specific guideline may be designated prior to 10:39:45

the establishment of that relationship; right? 10:39:48

A. Right. 10:39:50

Q. Turning back to page 8 of Part 1, Table 6 -- 10:39:53

Are you with me? 10:40:13

Page 8, Table 6? 10:40:17

A. Yes, I'm with you. 10:40:20

Q. Does Table 6 identify the chemicals that are 10:40:21

part of the Part 1 SQOs? 10:40:25

A. It identifies which chemicals are used in the 10:40:28

chemistry Line of Evidence for assessing sediment 10:40:51

quality. 10:40:59

That's all. It doesn't do any more. 10:41:01

Q. And is the chemistry Line of Evidence in Part 10:41:06

5 -- is it based on knowledge of the relationship 10:41:10

between these chemicals listed in Table 6 and the 10:41:14

organism's exposure to those chemicals and the 10:41:19

subsequent biological effect? 10:41:24

A. Yes. 10:41:25

Q. Is it based on a numeric relationship between 10:41:28

the organism's exposure and the biological effect? 10:41:34

A. Yes. 10:41:38

Q. Is it based on site-specific sediment management guidelines defining those relationships? 10:41:38 10:41:43

A. No. 10:41:46

Q. What is the difference between what's in Section 5 and what is -- Section 5 -- Section 5(h), Sediment Chemistry, and what is described in Section H on site-specific guidelines? 10:41:46 10:41:50 10:42:07 10:42:10

A. Umm -- Section 5 was -- those values were developed through -- through empirical relationships with -- umm -- between biological effects and chemical concentrations in -- in all bays for which we had data in California. 10:42:13 10:42:27 10:42:38 10:42:44 10:42:55

So those represent -- umm -- a broader set of conditions and types of sediment and are intended to solely assess potential -- the potential for effects. 10:42:57 10:43:07 10:43:23

There is no -- there is no intended use of those chemical values for anything else other than assessing sediment quality with the other two Lines of Evidence. 10:43:33 10:43:38 10:43:43 10:43:50

The site specific -- umm -- section -- site development and site specific targets, is a very site-specific -- umm -- study that would provide more representative, more accurate, more -- a more precise concentration-based value. 10:43:51 10:43:57 10:44:01 10:44:09 10:44:17

And I think -- umm -- those empirical 10:44:21

relationships are strongest on a site specific basis, because of all the different -- umm -- you know, variations in organic carbon, grain size, percent fine grain sediment clays, silts. 10:44:27 10:44:36 10:44:42 10:44:47

On a site-specific basis those empirical relationships are typically used to establish cleanup levels, and I believe that's the standard practice 10:44:51 10:44:55 10:45:04

Q. You mean the numbers on page 8 are used to set site-specific cleanup levels? 10:45:06 10:45:10

A. Oh, absolutely not. 10:45:11

Q. I'm sorry. 10:45:13

Then I may have misunderstood. 10:45:14

A. Oh, okay. Okay. 10:45:16

So these are not -- these -- these values are not intended to drive cleanup, or a site-specific cleanup, or even be water body or -- umm -- segment-specific sediment targets. 10:45:17 10:45:24 10:45:30 10:45:36

These are only for use as a -- one of the LOE, Line of Evidence, and the assessment framework. 10:45:41 10:45:47

Once you've moved through -- once you've applied the assessment framework -- 10:45:53 10:45:54

Q. Sir, may I hold you up there? 10:45:56

When you said "These values are not used as cleanup levels," were you referring to the values in Table 6 and 7 on page 8 of Part 1? 10:45:59 10:46:01 10:46:05

A. Yes. Yes. 10:46:08

Q. Thank you for that clarification. 10:46:09

Please proceed with your answer. 10:46:11

A. So, again, those values that we discussed in those tables are only for the assessment purpose. 10:46:16 10:46:20

The next step is stressor identification, and then once you define a -- that it's a pollutant-related stressor you look at -- umm -- relationships between the stressor identified at your site and the biological responses at your site and use that data to develop whatever -- umm -- targets are appropriate. 10:46:25 10:46:30 10:46:37 10:46:43 10:46:50 10:46:57

It's all site specific. 10:47:05

Q. And with regard to the target development that you just mentioned, do you mean pursuant to a process similar to that spelled out on pages 19 and 20 -- 10:47:09 10:47:12 10:47:16

A. Correct. 10:47:19

Q. -- of Part 1? 10:47:20

A. Yes, correct. 10:47:22

Q. Thank you. 10:47:23

So, to be clear, on page 8 of Part 1, Tables 6 and 7, do those values reflect knowledge of the dose response relationship between any of these chemicals and sediment qualify effects? 10:47:24 10:47:30 10:47:33 10:47:38

A. Yes. 10:47:42

Q. How so? 10:47:48

A. They -- umm -- they represent the relationship between chemistry and biological effects for -- umm -- all bays. 10:47:49 10:47:56 10:48:07

So there's -- 10:48:14

Do you see the difference? 10:48:14

Developing empirical relationships for -- umm -- that are based on a data set from all bays versus doing the same thing for a very small specific site. 10:48:18 10:48:23 10:48:32

Q. Let's try to get there. 10:48:35

A. Okay. 10:48:38

MR. SINGARELLA: Would you mark that as 702. 10:48:40

(Exhibit 702 was marked for Identification.) 10:48:42

BY MR. SINGARELLA: 10:49:05

Q. I'm causing to be placed in front of you what has been marked as Exhibit 702 to your deposition. 10:49:10 10:49:14

Do you see that? 10:49:17

A. Yes. 10:49:18

Q. And is this a presentation that you made as part of the SQO Program to the Regional Board in San Diego? 10:49:19 10:49:23 10:49:28

A. Yes. 10:49:28

Q. On November 14th, 2007; is that right? 10:49:29

A. Uh-huh. 10:49:32

Q. And it's -- the pages aren't numbered, Mr. Beegan, but six or seven pages in there's a -- 10:49:34 10:49:38

A. Mine are numbered. 10:49:44

MR. SINGARELLA: Yours are numbered? 10:49:45

MR. FUCHS: I think you're looking at a different exhibit. 10:49:46 10:49:48

MR. SINGARELLA: Oh, great. Thank you. 10:49:49

Yes, slide 8. 10:49:52

BY MR. SINGARELLA: 10:49:53

Q. Dose response relationship in water. 10:49:55

Now, this is a relationship between copper and a biological endpoint in the water column; correct? 10:50:07 10:50:12

A. Yes. 10:50:17

Q. To be distinguished from a relationship between copper and some biological effect in the underlying bottom sediments; right? 10:50:17 10:50:21 10:50:25

A. Correct. 10:50:29

Q. And what was -- what was your intent, if you recall, in -- in showing this kind of dose response relationship? 10:50:29 10:50:33 10:50:37

MR. FUCHS: I'm going to object. 10:50:37

I just don't see how that is within the scope of this deposition. 10:50:39 10:50:41

MR. SINGARELLA: I'll tie it -- tie it in closely. 10:50:42 10:50:45

MR. FUCHS: Maybe you can explain how you're going to tie it in now. 10:50:46 10:50:47

MR. SINGARELLA: Well, Mr. Fuchs, I -- you know, you're really asking me a lot of questions about my deposition strategy, and it's wholly outside of Mr. Gallagher's limiting instruction to deal with the methodology of the SQO. 10:50:49 10:50:52 10:50:54 10:50:58

So, if you'll allow me, I'm sure I can tie it up to your satisfaction. 10:51:02 10:51:06

MR. FUCHS: I'll give you a little rope. 10:51:08

MR. SINGARELLA: Okay. 10:51:10

MR. FUCHS: Once he asks the question. 10:51:12

MR. SINGARELLA: Yeah. 10:51:15

BY MR. SINGARELLA: 10:51:15

Q. You show a series of slides showing dose response between copper and biology in water and then in sediment; right? 10:51:15 10:51:18 10:51:24

A. Right. 10:51:25

Q. Did you intend to draw a contrast? 10:51:25

A. Yes. 10:51:28

Q. To help -- to help inform people as to what the SQO methodology is and isn't; right? 10:51:29 10:51:33

A. Absolutely. 10:51:37

Q. Yes. 10:51:38

Okay. So on slide 8 you're showing a characteristic dose response relationship for copper in the water column; correct? 10:51:39 10:51:42 10:51:46

A. Yes. 10:51:47

Q. And then you have some notes. 10:51:48

Are these your notes underneath the slide? 10:51:49

Do you recall these being your notes? 10:51:51

A. Yeah. I -- yeah. 10:51:53

I mean, I don't -- I don't recall these notes, 10:51:59

but they're on my PowerPoint, so I'll assume for all 10:52:02

intents and purposes they are my notes. 10:52:05

Q. And there's an indication that there are two 10:52:07

reasons this works, referring to the dose relationship 10:52:10

in the water column; right? 10:52:14

A. Uh-huh. 10:52:15

Q. And the first is the linkage to the pipe or 10:52:16

plant; correct? 10:52:19

A. Yes. 10:52:20

Q. And the second is that the dose response in 10:52:20

water is known; correct? 10:52:22

A. Yes. 10:52:24

Q. And you know what will happen to organisms 10:52:24

exposed to at or above the specific levels; right? 10:52:29

A. Uh-huh. 10:52:32

Q. Is that a "yes"? 10:52:33

A. Yes. 10:52:33

I'm sorry. 10:52:34

Q. And then there's a statement "with sediments 10:52:35

we have neither;" correct? 10:52:38

A. Yes. 10:52:42

Q. And then there's a -- additional statement at the bottom that says "The dose response relationships are not predictable in the sediment;" correct? 10:52:43 10:52:49 10:52:54

A. Correct. 10:52:57

Q. And it says "does," but -- you understand that to be mean "dose;" correct? 10:52:57 10:53:00

A. Yes. 10:53:03

Q. Okay. So when it says here "With sediments we have neither," one of the implications is -- is with respect to the characteristic clean dose response in the water column; right? 10:53:04 10:53:09 10:53:15 10:53:18

A. Correct. 10:53:20

Q. And so then, turning to slide 9, we see an attempt to find a dose response relationship in the sediment; right? 10:53:20 10:53:29 10:53:33

A. Correct. 10:53:34

Q. And to do that you plot copper versus some biological endpoint; right? 10:53:34 10:53:38

A. Correct. 10:53:40

Q. And, in fact, were these data used, if you know, to develop the SQOs? 10:53:40 10:53:43

MR. FUCHS: That's -- that is beyond the scope. 10:53:45 10:53:49

How the SQOs were developed is beyond the scope of this deposition. How they are applied in the abstract is not. How they were developed is. 10:53:51 10:53:54 10:53:56

So please don't answer that question. 10:53:59

MR. SINGARELLA: Let's keep track of this because, obviously, to understand how the methodology works I think we're entitled to know what -- what data were used to build it. 10:54:02 10:54:04 10:54:07 10:54:10

BY MR. SINGARELLA: 10:54:12

Q. So do you recall where you obtained these data in Slide 9? 10:54:15 10:54:18

A. I don't recall. 10:54:20

I probably got it from -- umm -- Steve Bay. 10:54:27

Q. The keeper of the SQO database; right? 10:54:35

A. Umm -- yeah, and SCCWRP -- 10:54:41

Well, Steve Bay was, as you know, the principal scientist responsible for developing the various Lines of Evidence, and I would assume I got that particular slide from him. 10:54:44 10:54:46 10:54:54 10:55:02

Q. Does Slide 9 reflect a clean dose response relationship between copper in the sediment and a biological endpoint? 10:55:03 10:55:08 10:55:11

A. No. 10:55:12

Q. That's a little messy; isn't it? 10:55:13

A. Yes. 10:55:20

Q. And so, for example, just taking a -- 10:55:21
concentration of a hundred milligrams per kilograms or 10:55:28
there about -- 10:55:34
Are you with me? 10:55:35
A. Yep. Yes. 10:55:36
Q. You can have close to a hundred percent 10:55:37
survival of the test critters; right? 10:55:40
A. Yes. 10:55:48
Q. Or a hundred percent die off of the test 10:55:49
critters; right? 10:55:55
A. Yes. 10:55:57
Q. And that -- those two statements could be true 10:55:57
for a range of these copper concentrations; right? 10:56:01
A. Yes. 10:56:03
Q. And you can't develop a clean dose response 10:56:04
relationship when the critters might die or survive for 10:56:09
the same concentration; right? 10:56:13
A. Right. 10:56:14
Q. And is this -- to your knowledge is this 10:56:17
characteristic of the kinds of -- of data for different 10:56:22
chemical constituents? 10:56:27
A. It's characteristic of the kinds of data that 10:56:29
you observe in mixtures. So this is probably from -- 10:56:35
umm -- a variety of samples that contain a variety of 10:56:48
pollutants. 10:56:53

There could be other pollutants that are causing the biological effects in that plot. 10:56:58 10:57:02

Does that make sense? 10:57:10

Q. Yes, it does. 10:57:12

A. The relationship between these two slides is not absolutely -- umm -- the same. 10:57:14 10:57:20

The slide before was a -- probably -- probably, I'm surmising -- was probably a -- a laboratory test with spiked copper concentrations where copper was the only contaminant introduced. 10:57:26 10:57:31 10:57:37 10:57:42

This slide is probably a slide applied -- ambient sediment samples. 10:57:46 10:57:56

So there could be other pollutants in there causing that biological response. 10:58:00 10:58:04

Q. Or there could be confounding factors? 10:58:07

A. Absolutely. 10:58:09

Q. Or physical disturbance? 10:58:10

A. Well -- yes. Yes. Yes. 10:58:13

Q. And -- 10:58:15

A. It's probably the sum of those. 10:58:18

Q. With respect to "ERL," to what does that refer? 10:58:20 10:58:23

A. Umm -- it's a sediment quality guideline developed by -- I believe it was Ed Long. 10:58:23 10:58:34

It -- I think it stands for Effects Range Low. 10:58:39

I don't remember the exact -- umm -- mathematical relationship to the effects, but they're at the low end where you expect not to see biological effects in sediment. 10:58:45 10:58:54 10:59:01 10:59:08

Q. And this -- this figure shows that even above the ERL there's a tremendous amount of data showing no effects as well; correct? 10:59:10 10:59:15 10:59:23

A. Correct. 10:59:24

Q. And the ERM, is that another sediment-quality guideline? 10:59:25 10:59:31

A. Yes. 10:59:31

Q. Is it empirical based? 10:59:32

A. Yes. 10:59:34

Q. And ERL is empirical based? 10:59:34

A. Yes. 10:59:37

Q. You did not use either of these in the sediment chemistry prong of the SQO; right? 10:59:38 10:59:40

A. Right. 10:59:44

Q. Are CSI and CALRM -- are those empirical based? 10:59:46 10:59:52

A. Correct. 10:59:52

Q. If you know, is there a statistically significant relationship between copper and the biological endpoint shown in Slide 9? 10:59:53 11:00:04 11:00:08

A. I don't know. 11:00:10

Q. You wouldn't expect that, though, looking at the data; would you? 11:00:18 11:00:21

A. Umm -- a significant -- a statistically significant between concentrations and -- are talking about -- 11:00:22 11:00:32 11:00:34

Q. Survival. 11:00:35

MR. FUCHS: I'll object that lacks foundation and calls for speculation, but you can go ahead and answer if you can answer. 11:00:38 11:00:40 11:00:42

THE WITNESS: There may be. 11:00:44

Yeah, it would be -- umm -- 11:00:45

BY MR. SINGARELLA: 11:00:54

Q. So, going back to Slide 8, you understand when -- when people speak to statistically significant relationships, they're talking about fitting a curve; right? 11:00:54 11:01:01 11:01:05 11:01:08

A. Right. 11:01:08

Q. Drawing a line through data; right? 11:01:08

A. Right. 11:01:10

Q. You would agree that in Slide 8 you could draw a line through those data that would pretty much capture the data; right? 11:01:11 11:01:14 11:01:18

MR. FUCHS: That lacks foundation and calls for speculation, but you can go ahead and answer if you can answer. 11:01:21 11:01:23 11:01:28

THE WITNESS: The -- umm -- the -- umm -- 11:01:29

You could draw a curve to that, but I don't 11:01:42

know whether those top three points are significantly 11:01:45

different. 11:01:51

BY MR. SINGARELLA: 11:01:52

Q. Could you draw a line through it for me? 11:01:53

A. You would like me to? 11:01:56

Q. Please. 11:01:58

Let the record show that Mr. Beegan has drawn 11:02:22

a slight curve through the data on Slide 8 of Exhibit 11:02:25

702. 11:02:33

Is that correct, Mr. Beegan? 11:02:36

A. Yeah. 11:02:38

Q. Turning to Slide 9 -- could you draw a line 11:02:39

through those data. 11:02:48

A. Umm -- no. 11:02:50

MR. FUCHS: Let the record reflect that the -- 11:02:54

there are one, two, three, four, five, six dots on the 11:02:57

chart on page 8, and I wouldn't dare count how many 11:03:02

dozens on the chart on page 9. 11:03:07

BY MR. SINGARELLA: 11:03:14

Q. And, turning to Slide 10 -- I have trouble 11:03:19

with this line understanding the difference between the 11:03:33

black circles and the open circles. 11:03:36

Do you know what the distinction is? 11:03:39

A. Umm -- I don't recall. 11:03:43
Q. Would you be able to draw a fit to these data? 11:03:45
A. Umm -- 11:04:01
MR. FUCHS: Lacks foundation. Calls for 11:04:04
speculation. 11:04:05
THE WITNESS: The relationship looks better, 11:04:06
but I don't think I could -- umm -- draw a line to it. 11:04:16
MR. FUCHS: Again, let the record reflect that 11:04:23
the image on page 10 contains dozens of points. 11:04:26
Are we done with this exhibit? 11:04:37
MR. SINGARELLA: No. 11:04:40
What made you think that? 11:04:41
BY MR. SINGARELLA: 11:04:45
Q. Could we turn to Slide 5, -- 11:04:45
A. Sure. 11:04:48
Q. -- please, of Exhibit 702. 11:04:49
So this description of -- of SQOs in -- in the 11:04:53
first bullet, do you see that, sir? 11:05:07
A. Yes. 11:05:09
Q. And did you -- did you prepare this -- this 11:05:10
slide? 11:05:15
A. Yes. 11:05:16
Q. And is it -- is it your understanding that the 11:05:18
SQOs are a means to differentiate sediment? 11:05:22
A. Yes. 11:05:27

Q. Basically between the impacted and those that are not impacted; right? 11:05:29 11:05:34

A. Correct. 11:05:35

Q. By bioavailable toxic pollutants; correct? 11:05:35

A. Correct. 11:05:40

Q. So it's a tool to distinguish the good sediment from the bad sediment; right? 11:05:41 11:05:47

A. Essentially, yes. 11:05:49

Q. And when you say that the SQOs are a standard for sediment quality, what does that mean to you? 11:05:51 11:06:03

A. Umm -- 11:06:07

MR. FUCHS: You know, I'm going to put an end to this line of questioning. 11:06:19 11:06:22

I'm sorry. 11:06:23

This -- I just fail to see how any of the questions have been asked in the last 10 minutes regarding Exhibit 702 relate to the scope of this deposition as set forth by Mr. Gallagher in the conversation of September 27th, which is how in the abstract the Statewide Sediment Quality Objectives should be or could be applied. 11:06:24 11:06:26 11:06:28 11:06:34 11:06:37 11:06:40 11:06:44

MR. SINGARELLA: Well, Mr. Fuchs, I have to tell you I'm stunned by your failure to see that. 11:06:49 11:06:51

Are you telling him to stop here? 11:06:53

MR. FUCHS: Yeah. 11:06:56

MR. SINGARELLA: And so you don't think that we're entitled to find out whether the Sediment Quality Objectives are a tool to distinguish the good from the bad sediment versus a tool that would actually mandate cleanup? 11:06:57 11:06:58 11:07:01 11:07:05 11:07:09

This is what this is all about, Mr. Fuchs. That's what I've been talking about all morning. 11:07:10 11:07:12

We need to understand the boundaries of this policy. It doesn't speak for itself. 11:07:14 11:07:17

There's a lot of -- of detail and unpackaging that has to go in to this. 11:07:19 11:07:23

And I'm sorry. 11:07:25

And, you know, Mr. Beegan put a lot in to these materials, and they don't just come in and speak to us, and this is our opportunity to understand the boundaries of this policy. 11:07:26 11:07:30 11:07:32 11:07:35

This is extremely relevant to the use of the SQOs in San Diego Bay. 11:07:38 11:07:41

We don't want Donald McDonald or the board members to misunderstand what these are and what they aren't. 11:07:44 11:07:47 11:07:50

I'm very comfortable with what they are, but no one is going to listen to me. They need to hear from Mr. Beegan. 11:07:51 11:07:54 11:07:57

That's why we're taking this deposition. 11:07:58

That's why we're here. 11:08:00

I'm not on some wild goose chase here. I'm 11:08:01

trying to defend my client. 11:08:06

So take it under advisement, please, because 11:08:07

you're constantly interrupting me, and I don't think 11:08:11

it's fair. 11:08:14

This is about methodology. 11:08:15

MR. FUCHS: You asked him, and he answered as 11:08:18

to the -- this interesting question as to whether it's 11:08:21

to distinguish good from bad sediment, which I think is 11:08:24

kind of legally and scientifically a meaningless 11:08:31

statement, but I let it go. 11:08:34

Your argument can be made to Mr. Gallagher. 11:08:36

Please don't answer any more questions 11:08:40

regarding this exhibit. 11:08:42

I've let it go on quite a while. 11:08:43

MR. SINGARELLA: Well, fortunately, I'm 11:08:54

finished with this exhibit. 11:08:59

MR. FUCHS: A happy coincidence. 11:09:01

MR. SINGARELLA: Yes, I would agree. 11:09:04

BY MR. SINGARELLA: 11:09:05

Q. So, turning back to page 8 of Part 1, are -- 11:09:15

Strike that. 11:09:36

With regard to Tables 6 and 7, can you show me 11:09:37

where the dose response threshold is for copper? 11:09:43

A. The -- umm -- the -- there's two indicators that we use. 11:09:49 11:10:13

One is logistic direction. That would be the one identified in Table 7. 11:10:14 11:10:19

And the other one is the -- umm -- the CSI. 11:10:21

The CSI was derived from looking at relationships between chemistry and benthic community, and then the -- umm -- the LRM was developed looking at relationships between chemistry -- sediment chemistry and, you know, broad water bodies and toxicity. 11:10:29 11:10:35 11:10:42 11:10:49 11:10:57

To -- we developed the thresholds based first on a definition of categories, and so we had categories defined as, you know, reference, and we have the narrative -- umm -- we have the narrative description of those four categories, and I think they're defined in one of those technical documents, perhaps even the Staff Report. 11:11:05 11:11:12 11:11:19 11:11:25 11:11:30 11:11:33 11:11:37

I did not develop those or perform the statistics myself. 11:11:38 11:11:44

There is an empirical relationship -- umm -- and I -- I guess -- I don't know how to answer that question other than to say that both tables were developed based on a relationship between chemistry and biological effects, and the specific thresholds fall along a similar -- umm -- philosophy of what is 11:11:47 11:11:56 11:12:03 11:12:08 11:12:15 11:12:24

reference, what is considered low, what is considered moderate, and what is considered high. 11:12:33 11:12:37

And those categories and -- and the description of those categories was similar for each of the Lines of Evidence, and that's kind of what -- umm -- kind of the -- the overall framework for establishing the -- the thresholds based on a dose response relationship. 11:12:40 11:12:44 11:12:49 11:12:54 11:12:57 11:13:06

But those thresholds are only developed based on mixtures, so -- umm -- it's messy data, very messy data. 11:13:08 11:13:16 11:13:23

When you -- when you're looking at data from even one water body or multiple water bodies, it's very messy. 11:13:24 11:13:29 11:13:34

That probably didn't do a very good job, but -- umm -- it's difficult trying to be accurate yet -- umm -- describe the -- describe things in a way that's concrete. 11:13:36 11:13:39 11:13:45 11:13:55

There's a methodology. There are documents that describe that methodology that I've provided. 11:13:57 11:14:02

So I -- I guess I don't know how to better answer that question. 11:14:10 11:14:15

Q. Well, if we were dealing with a water quality objective for copper in the water column, we could go find the dose response relationship; correct? 11:14:16 11:14:21 11:14:25

A. Oh, yes. Yes. 11:14:28

Q. How would we do that here? 11:14:29

A. Umm -- you could -- 11:14:31

MR. FUCHS: Let me object just for a second. 11:14:38
That's vague and ambiguous. 11:14:40

You mean where in these tables does the dose 11:14:42
relationship appear? 11:14:45

Is that the question? 11:14:47

MR. SINGARELLA: If they're not here, where 11:14:48
are they, so we can go and look at it and confirm it and 11:14:49
understand it. 11:14:55

MR. FUCHS: If you understand the question, 11:14:57
you can answer. 11:14:58

THE WITNESS: Umm -- I think the background 11:15:00
for these indicators would be in one of those technical 11:15:16
documents by the Technical Team. 11:15:23

There may be some description of that in the 11:15:28
staff report. 11:15:32

BY MR. SINGARELLA: 11:15:35

Q. Is the dose response relationship actually in 11:15:41
Part 1? 11:15:43

A. It serves as the foundation for these values. 11:15:44

I -- I don't -- 11:15:53

The dose response relationship. 11:15:54

The empirical relationship between 11:15:57

concentration and biological effects was the basis for developing these values. 11:16:01 11:16:05

Q. And do you know if those empirical relationships are statistically significant? 11:16:16 11:16:18

A. I would assume they are. 11:16:22

I'm not -- umm -- I'm -- 11:16:24

I don't recall, but I would assume they are. 11:16:35

This goes back -- umm -- this is -- you know, five years ago. 11:16:38 11:16:46

To me it seems like 20 years ago. 11:16:47

I -- 11:16:51

Q. Where would we find these statistics demonstrating statistical significance between dose and response? 11:16:54 11:16:56 11:17:00

A. Umm -- one of the technical reports? 11:17:01

MR. SINGARELLA: Would you mark that, please. 11:17:35

(Exhibit 703 was marked for Identification.) 11:17:36

THE REPORTER: 703. 11:17:38

BY MR. SINGARELLA: 11:17:51

Q. I've placed in front of you what has been marked as Exhibit 703 to your deposition. 11:17:51 11:17:54

Do you see that, Mr. Beegan? 11:17:56

A. Uh-huh. 11:17:58

Q. Yes? 11:17:59

A. Yes, I do. 11:18:00

Q. Is this a document that you prepared for this deposition? 11:18:00 11:18:03

A. Yes. 11:18:03

Q. On October 5, 2010? 11:18:07

A. Yes. 11:18:09

Q. And is it an index to some of the technical documents that you were just referring to? 11:18:09 11:18:15

A. Yes. 11:18:17

Q. Do you have -- 11:18:17

Strike that. 11:18:28

Can you tell us where we would find the dose response relationship supporting this sediment chemistry Line of Evidence -- if you know? 11:18:28 11:18:30 11:18:35

MR. FUCHS: I'm going to object that the -- 11:18:48

Well, it's my standard objection. 11:18:50

We're beyond the scope. 11:18:52

How the -- Table 6 and Table 7 were developed is not relevant to how they are applied. 11:18:53 11:18:58

It's a different question. 11:19:01

I understand that they're related, but it's a different question. 11:19:03 11:19:06

MR. SINGARELLA: It's a fine read, indeed. 11:19:07

You can go ahead and answer, please. 11:19:10

THE WITNESS: Under the heading "Sediment Chemistry" there are a couple of SCCWRP reports. 11:19:13 11:19:19

They may also be -- umm -- included in similar articles provided on the -- that USB flash drive. 11:19:27 11:19:38

BY MR. SINGARELLA: 11:19:46

Q. And are these sediment chemistry references on the second page of Exhibit 703 -- are they helpful to make sure that the SQO is properly applied? 11:19:48 11:19:51 11:19:56

A. Yes. 11:20:04

Q. They relate to the SQO methodology; correct? 11:20:08

A. The methodology -- umm -- to assess sediment quality in relationship to direct effects, yes. 11:20:15 11:20:23

Q. And this is an extremely complicated subject area; right? 11:20:25 11:20:28

A. Yes. 11:20:30

Q. With a series of technical reports related to the Part 1 SQOs; correct? 11:20:30 11:20:37

A. Correct. 11:20:40

Q. Most of which aren't just raw data reports; right? 11:20:40 11:20:44

A. Correct. 11:20:44

Q. They are reports that inform the user of Part 1 how to you use it; correct? 11:20:44 11:20:50

A. Umm -- yes, and -- 11:20:51

Yes. 11:21:03

Q. Now, turning back to the sediment chemistry Line of Evidence, there are two empirical relationships 11:21:03 11:21:20

used in that Line of Evidence; right? 11:21:26

A. Yes. 11:21:28

Q. One is the CSI; right? 11:21:29

A. Yes. 11:21:31

Q. And the other is the California LRM; correct? 11:21:31

A. Yes. 11:21:34

Q. What does "CSI" stand for? 11:21:35

A. Umm -- I don't recall. 11:21:37

Q. What does "California LRM" stand for? 11:21:42

A. It's Logistic Regression -- umm -- 11:21:47

I don't remember what the "M" is. 11:21:51

Q. Can you turn to page 7, please, of Part 1. 11:21:53

Does this refresh your recollection that "CSI" 11:22:07

refers to Chemical Score Index? 11:22:11

A. Yeah. 11:22:13

Q. And that "California LRM" refers to California 11:22:13

Logistic Regression Model? 11:22:17

A. Yes. 11:22:20

Q. And to use the Part 1 SQO you need to use both 11:22:21

of those guidelines; right? 11:22:28

A. Correct. 11:22:30

Q. You can't just decide to use one or the other; 11:22:31

right? 11:22:36

A. Right. Correct. 11:22:37

Q. And, if you just used one, you might be using 11:22:37

a Line of Evidence, but you would not be using the multiple Lines of Evidence in Part 1; correct? 11:22:49 11:22:52

A. Correct. 11:22:54

Q. And with regard to the benthic indices, on pages 6 and 7 of Part 1 -- 11:22:58 11:23:11

Are you with me? 11:23:15

A. Yes. 11:23:18

Q. There are four of those; correct? 11:23:23

A. Yes. 11:23:26

Q. Written in to Part 1; right? 11:23:26

A. Correct. 11:23:28

Q. All four of them are required to be used; right? 11:23:29 11:23:33

A. Correct. 11:23:33

Q. To have an accurate and proper application of Part 1; right? 11:23:34 11:23:36

A. Correct. 11:23:37

Q. And, if you were to use fewer than four of these benthic indices, you might have a Line of Evidence, but you wouldn't have Part 1? 11:23:38 11:23:43 11:23:47

A. Correct. 11:23:50

Q. And, turning back to page 20 of Part 1, just before the glossary there's a reference to the chemistry LOE, Line of Evidence. 11:23:52 11:24:14 11:24:18

Do you see that? 11:24:20

A. Yes. 11:24:21

Number 5? 11:24:24

Q. Yes. 11:24:26

A. Absolutely. 11:24:26

Q. Yeah. 11:24:27

And it absolutely says that the chemistry LOE 11:24:28
shall not be used for setting cleanup levels; correct? 11:24:31

A. Correct. 11:24:34

Q. What does that mean? 11:24:35

A. It means that those values in -- umm -- in 11:24:36
those tables -- umm -- are -- are inappropriate for use 11:24:48
as establishing cleanup levels. 11:25:00

I -- I -- 11:25:03

Q. To you it's pretty plain? 11:25:07

A. It is. 11:25:09

Q. It's black-and-white, you just don't use the 11:25:09
chemistry Line of Evidence to set a cleanup level? 11:25:13

A. Right. 11:25:17

Q. Nor would you use the chemistry Line of 11:25:17
Evidence to set a numeric value for a technical TMDL; 11:25:24
correct? 11:25:28

A. Well, correct, yes. 11:25:29

But there is -- umm -- there's a note in this 11:25:35
Water Quality Control Plan that says something like -- 11:25:45
umm -- nothing in this Water Quality Control Plan or 11:25:52

nothing in this section shall limit a Regional Board's authority to develop TMDLs. 11:25:57 11:26:02

Are you familiar with that language? 11:26:06

I -- 11:26:07

Q. Yes. 11:26:08

I don't know where it is precisely. 11:26:09

A. Neither do I. I'm just trying to respond to your question accurately. 11:26:11 11:26:15

The intent was to -- for all sites, whether it's a TMDL cleanup, or whatever, other sediment-related problem, is to assess if problems go through stressor identification, and then develop some biologically-relevant target based on the site conditions, or the segment conditions, or the water body conditions. 11:26:16 11:26:25 11:26:30 11:26:39 11:26:47 11:26:51 11:26:54

Q. The sediment chemistry Line of Evidence does not provide the biologically-relevant target; correct? 11:26:56 11:27:05

A. For target for a site? 11:27:08

Correct. 11:27:23

Q. And then, turning back to page 19 of Part 1 -- I think I've covered this, Mr. Beegan. 11:27:24 11:27:55

Is -- is stressor identification relevant to defining appropriate remediation activity? 11:27:59 11:28:23

A. Yes. 11:28:26

Q. And -- and so, too, is the development of the 11:28:36

site specific guidelines in Section H? 11:28:44

A. Yes. 11:28:49

Q. Those are the parts of -- of this plan where you tie knowledge of sediment quality through the multiple Lines of Evidence with management action and cleanup; correct? 11:28:50 11:29:01 11:29:05 11:29:09

A. Correct. 11:29:10

Q. And the chemistry Line of Evidence does not establish causality; correct? 11:29:11 11:29:24

A. Absolutely correct. 11:29:27

Q. And what does that mean? 11:29:28

A. It means that -- umm -- the empirical guidelines, the CSI -- the empirical chemistry indicators, the CSI and the LRM, are -- empirical relationships based solely on association. 11:29:29 11:29:39 11:29:44 11:29:51

There is no -- umm -- 11:29:57

There's no -- 11:30:01

They don't assume cause. 11:30:05

They don't assume that -- that particular chemical at that concentration is causing the biological effects. 11:30:07 11:30:11 11:30:16

That is why we included stressor identification. Stressor identification is -- 11:30:18 11:30:23

The intent of stressor identification is to determine the cause. 11:30:27 11:30:30

Q. And, in fact, the Lines of Evidence are not sufficiently reliable to be used independent in isolation from the other Lines of Evidence; right? 11:30:31 11:30:42 11:30:46

A. Correct. 11:30:49

Q. Because they can underestimate risk in some circumstances; right? 11:30:52 11:31:06

A. Correct. 11:31:07

Q. Or overestimate risks in other circumstances; correct? 11:31:08 11:31:11

A. Correct. 11:31:11

Q. And this is a risk to the benthic community; correct? 11:31:11 11:31:15

A. Correct. 11:31:15

Q. And this imprecision can be significant; correct? 11:31:15 11:31:19

A. Correct. 11:31:19

Q. So let me just give you an example so I'm sure I understand this. 11:31:19 11:31:39

You're applying -- or you're attempting to apply the MLOE, but you used three of the benthic indices and not the fourth, and you used only lethal test results and not sublethal. 11:31:40 11:31:47 11:31:52 11:31:56

Are you with me? 11:31:59

A. Uh-huh. 11:32:00

Q. Yes? 11:32:00

A. Yes. Yes. 11:32:02

Q. You -- you would not actually be applying this MLOE, referring to Section 5 of Part 1; right? 11:32:03 11:32:07

A. Correct. 11:32:11

Q. To apply Section 5 of Part 1 you need sublethal test results; correct? 11:32:11 11:32:21

A. Correct. 11:32:23

Q. In addition to lethal test results; right? 11:32:23

A. Correct. 11:32:26

Q. You need to have information on the benthic community; correct? 11:32:26 11:32:31

A. Correct. 11:32:32

Q. Numbers of critters, fila, things of that nature; correct? 11:32:32 11:32:37

A. Correct. 11:32:37

Q. Sufficient to support the calculation of all four benthic entities; correct? 11:32:37 11:32:43

A. Correct. 11:32:44

Q. And then you need to use the results from all four of those calculations as part of the benthic community Line of Evidence; correct? 11:32:45 11:32:47 11:32:50

A. Correct. 11:32:50

Q. Turning to page 14, "Receiving Water Limitations," for a minute. 11:32:51 11:33:01

On page 14 Section C "Exceedance of receiving 11:33:05

water limit" describes two conditions that must be satisfied for there to be an exceedance of a Receiving Water Limit. 11:33:17 11:33:21 11:33:24

Do you see that? 11:33:26

A. Yes. 11:33:27

Well, -- yeah. Yeah. 11:33:29

Q. And what is a "receiving water limit" as used in this context? 11:33:30 11:33:33

A. Umm -- it's a -- umm -- it's a permanent limit. It's -- 11:33:34 11:33:41

Or it's a -- 11:33:43

Yeah, it's a permit limit. It's located in the receiving water rather than what most people think of as permit limits, which are effluent-based associated with the direct measure of effluent. 11:33:44 11:33:51 11:33:58 11:34:04

Q. And before there is an exceedance of such a limit you have to do -- you have to know two things; right? 11:34:07 11:34:13 11:34:18

A. Yes. 11:34:20

Q. And what is the first one? 11:34:20

A. Umm -- you have to have -- umm -- the total number of stations designated as not meeting the protective condition. 11:34:22 11:34:29 11:34:33

I think that's -- umm -- would characterize that as possibly likely or clearly impacted. 11:34:36 11:34:41

Supports the rejection of the null hypothesis as presented in Table 15. 11:34:44 11:34:48

Basically you need multiple stations to -- in order to be -- umm -- considered a potential exceedance, and the number of exceedance is relative to the total number of stations is identified in Table 15. 11:34:50 11:34:56 11:35:02 11:35:09

Q. And then there's a second element of determining the presence of an exceedance; correct? 11:35:12 11:35:17

A. Yes. 11:35:21

Q. And what is that, Mr. Beegan? 11:35:21

A. Umm -- it is demonstrated that the discharge is causing or contributing to SQO exceedance following the completion of the stressor identified studies. 11:35:23 11:35:27 11:35:31

Q. And that's a reference to the three-step process described in stressor identification in Part 1; correct? 11:35:35 11:35:39 11:35:41

A. Correct. 11:35:41

Q. And, in fact, when you get to Step 3, management actions, on page 19, there's a reference to requirements for dischargers; correct? 11:35:42 11:35:45 11:35:50

A. Yes. 11:35:52

Hold on. 11:35:56

19? 11:35:56

Q. Page 19, yeah. 11:35:57

A. Yes. 11:36:03

Q. And so the way this works is you go through the MLOE; right? 11:36:03 11:36:09

A. Yes. 11:36:15

Q. You do stressor identification; correct? 11:36:15

A. Yes. 11:36:18

Q. And then, if you identify a particular constituent associated with an unacceptable impact from a particular source, at that point the Regional Board requires the discharger to take management actions; correct? 11:36:18 11:36:26 11:36:32 11:36:37 11:36:41

A. Correct. 11:36:41

MR. SINGARELLA: Could we get Master Exhibit Number 7 out? 11:36:51 11:36:54

And we're almost out of tape according to Sean. 11:36:54 11:36:59

So why don't we take a five-minute break. 11:36:59

MR. FUCHS: All right. 11:37:03

VIDEOGRAPHER: We are going off the record at 11:36 a.m. 11:37:04 11:37:05

This is the end of disk number two. 11:37:07

(Thereupon a recess was taken at 11:36 a.m. and the deposition resumed at 11:54 a.m.) 11:37:10 11:54:08

(Exhibit No. 704 was marked for Identification.) 11:54:08 11:54:37

VIDEOGRAPHER: We are back on the record at 11:54:37

11:54 a.m. 11:54:46

This is the beginning of disk number three. 11:54:48

BY MR. SINGARELLA: 11:54:50

Q. Welcome back, Mr. Beegan. 11:54:51

A. Thank you. 11:54:52

Q. I've placed in front of you what has been marked as Exhibit 704 to your deposition. 11:54:53 11:54:56

Do you see that? 11:54:59

A. Yes. 11:55:00

MR. SINGARELLA: And this is just housekeeping, Mr. Beegan, but for the record and for Mr. Fuchs, this is actually -- I believe it's -- it's also Master Exhibit Number 7, but the original Master Exhibit does not seem to have made its way up here, so just out of an abundance of caution I'll reenter it as Beegan's 704. 11:55:01 11:55:03 11:55:07 11:55:10 11:55:13 11:55:16 11:55:21

BY MR. SINGARELLA: 11:55:22

Q. Mr. Beegan, do you recognize this document? 11:55:22

A. Yes. 11:55:25

Q. And what is it? 11:55:26

A. It's the Staff Report for the Water Quality Control Plan For Enclosed Bays and Estuaries, Part 1, Sediment Quality. 11:55:27 11:55:31 11:55:37

Q. And could you turn to page 2-1, please, sir. 11:55:37

Now, just by way of background, what is a 11:55:51

"Staff Report"? 11:55:54

A. Umm -- a "Staff Report" is our document that 11:55:55

acts as a functionally equivalent CEQUA document. 11:56:06

It also is -- provides the supporting or the 11:56:11

background information for the Water Quality Control 11:56:16

Plan. 11:56:19

It describes -- umm -- many factors that the 11:56:20

Water Board must consider when developing a Water 11:56:26

Quality Control Plan and/or objectives. 11:56:30

It's been called -- in the past we used to 11:56:38

call it a Functional Equivalent Document or FED. 11:56:42

Sometimes it's been referred to as an SED. 11:56:46

I think at this point we're calling them just 11:56:50

"staff reports." 11:56:54

Q. So this is staff's report used during the 11:56:56

proceedings to adopt the Part 1 SQO? 11:57:00

A. Yes. 11:57:04

Q. And it contains information relevant to the 11:57:05

SQO methodology; correct? 11:57:09

A. Yes. 11:57:11

Q. And, in fact, explains the agency's rationale 11:57:11

basis for Part 1? 11:57:18

A. Yes. 11:57:19

Q. And so on page 2-1 of the Staff Report, just 11:57:19

above the figure there's a reference to "diversity of 11:57:29

sources." 11:57:35

The last sentence, do you see that? 11:57:35

A. Yes. 11:57:37

Q. What -- what do you mean? 11:57:38

What sources are being referred to there? 11:57:42

A. Umm -- I assume -- 11:57:45

Well, it's referring to the sentences earlier 11:57:54
in that paragraph. 11:57:58

Umm -- so there's -- umm -- sources of 11:58:02
contaminants within the water body, and there's sources 11:58:15
from rivers, creeks, drainage channels, storm water, dry 11:58:22
weather run-off, industrial activities, municipal 11:58:27
activities, atmospheric deposition, contaminated 11:58:34
groundwater. 11:58:40

I think it's referring to all these sources 11:58:41
that are described. 11:58:43

Q. Are rivers, creeks, and channels a known 11:58:43
source of toxic pollutants to California bays? 11:58:47

A. They can be. 11:58:51

Q. Have they been identified as such? 11:58:52

A. Umm -- yes. 11:58:55

Q. Then there's a reference to "various physical 11:59:00
mixing processes." 11:59:04

Do you see that? 11:59:05

A. Yes. 11:59:06

Q. And in this context why are the physical mixing processes important? 11:59:10 11:59:15

A. The mixing processes are important because physical mixing can -- umm -- mix pollutants -- umm -- they -- this is kind of a generic sentence that talks about the combination of pollutants or pollutants mixing, settling out, being pushed around by various processes, so that it may not be obvious what the source is or what the problem actually is. 11:59:16 11:59:42 11:59:51 11:59:59 12:00:04 12:00:08 12:00:17

Q. So currents can obscure the source and nature of the problem? 12:00:20 12:00:26

A. Potentially, yes. 12:00:27

Q. Referring to impacted bottom sediments; right? 12:00:29

A. Yes. 12:00:34

Q. And how do currents potentially obscure the source and nature of the problem? 12:00:34 12:00:39

A. Well, I guess -- umm -- suspended sediments could be -- deposited in different areas, not necessarily located right at the source. 12:00:40 12:00:47 12:00:55

A pollutant from one discharge could be mixed with a pollutant from another discharge through the same currents. They all would eventually settle out in to fine grain areas. 12:01:01 12:01:05 12:01:08 12:01:13

In some places -- I think we talked about scour before, the physical processes that could 12:01:15 12:01:21

resuspend and move contaminants or contaminant bound sediments from one area and redeposit in another area. 12:01:26 12:01:31

Q. And can rivers and creeks produce such currents? 12:01:36 12:01:45

A. Yes. 12:01:46

Q. Can ships? 12:01:46

A. Umm -- produce currents? 12:01:48

Q. Vessel traffic? 12:01:50

A. Umm -- yes, I guess so on a very -- very small -- umm -- over a very small area, yeah, sure. 12:01:52 12:02:07

Q. How does ship traffic produce physical mixing processes? 12:02:10 12:02:13

A. Well, I guess the -- the prop wash, which we talked about before, the wave action generated by the bow wake. 12:02:14 12:02:17 12:02:24

Q. What is "prop wash"? 12:02:30

Is that a reference to a propeller? 12:02:32

A. Yes. 12:02:36

Q. And what is "wash from a propeller"? 12:02:36

A. "Wash" is the -- it's the pressure generated by the propeller that would be great enough to potentially stir up the -- surface layer sediments. 12:02:41 12:02:47 12:02:52

Q. And what is "bow wake" that you just referred to? 12:02:57 12:03:02

A. Umm -- when a surface vessel moves, it 12:03:03

generates a bow wake. Those waves can -- if they're large enough, they could stir up -- umm -- shallow sediments. 12:03:12 12:03:20 12:03:26

Q. Sorry to be asking you about concepts that may be obvious to you. 12:03:27 12:03:33

A. No. I just -- umm -- 12:03:34

Q. We're lawyers. Assume no pre-existing knowledge. 12:03:37 12:03:42

Well, thank you. 12:03:43

Can -- can anchors also produce physical disturbance? 12:03:47 12:03:49

A. Sure. Yes. 12:03:52

Q. And as part of -- of staff's preparation for adoption, did staff review the different bays around the state? 12:03:52 12:04:04 12:04:09

A. Yes. 12:04:12

Q. You wanted to look at the actual conditions where these SQOs would be implemented; correct? 12:04:13 12:04:18

A. Yes. 12:04:22

Q. And would apply; correct? 12:04:23

A. Yes. 12:04:24

Q. Could you turn to -- Chapter 3 of the Staff Report at page 21, 3-21? 12:04:25 12:04:33

First paragraph refers to San Diego Bay. 12:04:55

Do you see that -- on page 3-21? 12:04:59

A. Yes. 12:05:04

Q. And it's merely descriptive, it refers to the vessels moored in San Diego Bay; correct? 12:05:04 12:05:08

A. Yes. 12:05:12

Q. Up to 9,000; right? 12:05:16

A. Uh-huh. 12:05:20

Q. Yes? 12:05:21

A. Yes. 12:05:21

Q. And it refers to four major U.S. Navy bases; correct? 12:05:21 12:05:26

A. Yes. 12:05:26

Q. With approximately 80 surface ships and submarines; right? 12:05:26 12:05:30

A. Correct. 12:05:31

Q. And this was included in the Staff Report because of the potential for -- for, I imagine, contamination from these potential sources; right? 12:05:32 12:05:34 12:05:42

A. Umm -- I think it was broader. 12:05:45

Q. In what sense? 12:06:00

A. Umm -- to describe the overall use of the bay or what kind of -- bay, but -- 12:06:02 12:06:08

Yeah, I mean, the purpose of this is to get at -- umm -- potential problems. 12:06:13 12:06:19

Q. Would potential problems include potential confounding factors? 12:06:25 12:06:30

A. Sure. 12:06:31

Q. And vessel traffic can produce these phenomena 12:06:32
that you've described; right? 12:06:39

A. Under specific conditions, right. 12:06:41

Q. Prop wash; right? 12:06:45

A. Right. 12:06:47

Q. Bow wake; right? 12:06:47

A. Right.

Q. Physical disturbance of the bottom sediment; 12:06:49
right? 12:06:53

A. Right. 12:06:53

Q. Relative to other bays does San Diego have a 12:06:54
high level of vessel traffic? 12:07:01

MR. FUCHS: Lacks foundation, calls for 12:07:03
speculation and outside the scope. 12:07:05

I guess it's not particularly material, so you 12:07:14
might as well answer -- if you know. 12:07:18

THE WITNESS: Relative to what? 12:07:20

BY MR. SINGARELLA: 12:07:24

Q. Well, to the other bays that are discussed in 12:07:25
the Staff Report. 12:07:29

A. Umm -- I guess it's in the top three of 12:07:31
California. 12:07:53

Q. Okay. 12:07:53

A. I mean, -- 12:07:54

Q. Sure. 12:07:54

A. -- I think that's true to the best of my knowledge. 12:07:55 12:07:57

Q. Uh-huh. 12:07:57

I just didn't see any other description that had quite an impressive number of boats that were referred to. 12:08:03 12:08:06 12:08:10

A. To -- 12:08:11

Well -- okay. 12:08:12

Q. Seemed that that was relevant to staff, they included this information; correct? 12:08:19 12:08:26

A. I don't remember why I included that information, to be honest with you, or where -- what the source was. 12:08:31 12:08:33 12:08:36

Q. Fair enough. 12:08:38

Let's turn to Staff Report 5-6. 12:08:45

Now, the bottom of that page in the Staff Report just before the bullets refers to the "optimal sediment receptor." 12:08:57 12:09:06 12:09:10

Do you see that? 12:09:12

A. Can you repeat that once more, 5-6? 12:09:13

Q. Sure, 5-6 just above the bullets. 12:09:17

A. Oh, okay. 12:09:21

Q. "Benthic communities are recognized;" correct? 12:09:22

A. Yes. 12:09:29

Q. "As the optimal sediment receptor;" right? 12:09:30
A. Yes. 12:09:33
Q. What does that mean? 12:09:33
A. They exhibit characteristics that make them -- 12:09:37
umm -- an ideal indicator. 12:09:56
Umm -- they are ecologically important. 12:10:02
They are relatively sessile or exhibit for the 12:10:11
most part little movement. 12:10:21
Umm -- they live for long periods of time. 12:10:28
Umm -- they've been demonstrated to be 12:10:36
sensitive. 12:10:45
Umm -- and they are -- umm -- measurable in 12:10:45
terms of community composition and community health. 12:10:57
Q. And what does it mean that they're optimal? 12:11:02
A. Well, I guess what it means -- umm -- is that 12:11:07
there are -- umm -- there are a whole slough of 12:11:12
receptors out there that one could choose, but for 12:11:21
various reasons -- umm -- they're not considered as 12:11:25
reliable. 12:11:34
I guess the -- umm -- the example given is 12:11:37
aquatic plants. 12:11:46
Q. Aquatic plants? 12:11:47
A. Yeah. 12:11:51
If you read the -- the paragraph above, that's 12:11:53
an example of -- another receptor that you could apply, 12:11:58

but for various factors it wouldn't be optimal. 12:12:05

Q. And by "receptor," what does that term refer to? 12:12:13 12:12:16

A. "Receptor" is -- umm -- an organism that you've identified that's potentially at risk of exposure for the problem or the release that you're trying to address. 12:12:16 12:12:26 12:12:35 12:12:42

So I guess for sediment quality an improbable receptor would be a bear or a beaver. 12:12:44 12:12:52

Other receptors could be fish, could be humans that consume fish, could be wetland birds, marine -- or marine mammals. 12:13:00 12:13:04 12:13:12

Those are all receptors that are potentially exposed. 12:13:15 12:13:18

Q. And in which line of evidence is information about the benthic community explicitly used? 12:13:19 12:13:23

A. The -- the benthic community Line of Evidence. 12:13:27

Q. With the four indices? 12:13:40

A. Yes. 12:13:42

Q. So those -- those four indices relate to the optimal sediment receptor; correct? 12:13:43 12:13:54

A. Yes. 12:13:57

Q. Let me ask you a question on page 5-4 related to the "biologically active layer." 12:13:58 12:14:06

Are you familiar with that term? 12:14:09

A. Yes. 12:14:11

But you referred to a page? 12:14:12

Q. Yes, page 5-4, two pages prior. 12:14:14

A. Okay. 12:14:18

Q. Under Alternative 2, which was the staff recommendation, it refers to the "biologically active layer." 12:14:18 12:14:23 12:14:26

Do you see that? 12:14:27

A. Oh. 12:14:28

MR. FUCHS: Alternative 2 up at the top, not at the bottom. 12:14:32 12:14:35

THE WITNESS: Oh, oh, oh. 12:14:36

MR. SINGARELLA: Thank you, Dan. 12:14:38

THE WITNESS: I've got to get rid of this. 12:14:44

Yes, I see that. 12:14:49

BY MR. SINGARELLA: 12:14:50

Q. And it's referring to the tools; right? 12:14:51

A. Correct. 12:15:00

Q. That have been developed; right? 12:15:01

A. Correct. 12:15:03

Q. And is that a reference to Part 1? 12:15:03

A. Umm -- I'd have to read the previous page. 12:15:08

Q. Okay. Please -- please do. 12:15:15

A. Yes, you're correct. 12:15:54

Q. Thank you. 12:15:57

And I appreciate your patience working with me, Mr. Beegan, to get clarity on -- on these questions. 12:15:58 12:16:01

So Alternative 2 says, in essence, that -- that Part 1 has been developed solely to assess this biologically active layer; correct? 12:16:05 12:16:11 12:16:17

A. Correct. 12:16:19

Q. None other than that particular layer; correct? 12:16:20 12:16:23

A. Correct. 12:16:23

Q. And please tell me how the biologically active layer is defined spatially. 12:16:23 12:16:28

A. The biologically active layer is the active layer where most of the -- the benthic community resides in. 12:16:36 12:16:39 12:16:51

Q. Is there a depth of it that's relevant to Part 1? 12:16:51 12:16:57

A. Well, I think we've defined sufficient sediments -- in Part 1. 12:16:57 12:17:02

Q. As being what? 12:17:06

A. Oh. It may have even been in the Staff Report, but I think -- 12:17:07 12:17:15

Q. Page 22 -- of Exhibit 6? 12:17:25

A. Yes. Umm -- page 22 of Part 1, those sediments represent the most recent depositional materials and contain the majority of benthic 12:17:34 12:17:45 12:17:50

invertebrate community. 12:17:53

Q. Is there an understanding as to the 12:17:53

characteristic depth of that layer? 12:17:55

A. Umm -- for the purposes of applying the 12:17:59

multiple Lines of Evidence, there are criteria for -- 12:18:01

umm -- collecting samples, and I believe that is 12:18:07

contained in another section within Part 1. 12:18:11

Q. Let's see if we can -- if you can find it, 12:18:18

Mr. Beegan. 12:18:25

A. Field procedures? 12:18:26

Umm -- Part 5(d) -- umm -- Part 5(d) -- umm -- 12:18:29

4 -- or 3. 12:18:44

Yeah, upper five centimeters of sediment. 12:18:49

Q. About two inches; is that right? 12:18:55

A. Yes. 12:18:58

Q. And the -- the Part 1 doesn't assess the need 12:18:58

to do any cleanup below the top two inches; right? 12:19:13

A. Part 1 doesn't -- doesn't -- umm -- doesn't 12:19:20

require the assessment of deeper samples off the bat. 12:19:39

Q. After application of the MLOE in Section 5, do 12:19:49

you have information relevant to the quality of the 12:20:04

sediment below that top five centimeters? 12:20:09

A. No. 12:20:12

Q. When it comes to management actions, are you 12:20:13

aware of -- of management actions that can actually 12:20:25

address just the top two inches of sediment? 12:20:30

A. You're -- it's hard to understand. 12:20:33

Where the -- 12:21:03

Are you saying where contamination only or the -- the stressor only extends two-and-a-half inches? 12:21:04 12:21:07

Is that what you're asking, would that happen? 12:21:12

I -- 12:21:17

Q. No, no. 12:21:17

A. Does it limit? 12:21:18

Q. Are their techniques to just get at the two inches from a cleanup perspective -- if you know? 12:21:20 12:21:24

A. I don't know. I don't know. 12:21:34

Q. Okay. All right. 12:21:36

Can you turn to page 5-7, please. 12:21:37

A. 5-7. 12:21:42

Q. Second bullet back to benthic communities, there's a reference to benthic communities being an in-situ measure of actual conditions and biological effects that are or have occurred." 12:21:45 12:21:49 12:21:54 12:21:57

Do you see that? 12:22:00

A. Yeah. 12:22:00

Q. What does that mean, "in-situ measure," i-n hyphen s-i-t-u? 12:22:01 12:22:09

A. It's a -- umm -- in place -- umm -- measure of sediment quality, unlike other measures. 12:22:09 12:22:19

A laboratory method, that would not be in-situ. In-situ is in place -- umm -- undisturbed -- umm -- 12:22:26 12:22:34 12:22:41

Is that the part that you were having a question on? 12:22:46 12:22:49

Q. Yes. 12:22:49

A. Okay. 12:22:50

Q. And these other measures are, at best, surrogate measures; is that right? 12:22:50 12:22:58

A. Umm -- yes. 12:23:00

Q. What is meant by the word "surrogate" in that context? 12:23:02 12:23:06

A. Umm -- a -- umm -- an indicator that is applied to assess a particular in this case exposure where a more representative tool -- umm -- can't be developed or -- umm -- it was -- umm -- 12:23:07 12:23:26 12:23:36 12:23:49

A "surrogate tool" is a tool that you can interpret, but it only provides a secondary -- umm -- information -- or it's not considered as precise or accurate. 12:23:56 12:23:59 12:24:07 12:24:11

Q. Can you give me an example of a surrogate measure? 12:24:13 12:24:16

A. Umm -- off the top of my head, no. 12:24:17

There's plenty -- 12:24:28

Oh, how about temperature? 12:24:30

Q. How about it? 12:24:35
A. Okay. 12:24:36
Q. Why is that a surrogate? 12:24:36
A. Well, I mean -- umm -- if you have a -- if you're running -- if the human body is running hot, it means you're sick, but that's about all it tells you, yet there is probably -- there's probably -- it -- it helps, but the doctor would need more information -- umm -- to -- umm -- to determine what the true problem is, or to decide whether you're really sick or -- 12:24:39 12:24:46 12:24:51 12:24:54 12:24:58 12:25:04 12:25:07
I think that's a surrogate. 12:25:11
Q. But the benthic communities are a measure of the actual condition of what's actually happening in the sediment; right? 12:25:12 12:25:22 12:25:25
A. Yes. Yes. Yes. 12:25:26
Q. And it's desirable because of that? 12:25:30
A. It's desirable for several reasons. 12:25:33
That's one of them. 12:25:36
Q. And its an in place measure of the actual biological effects that are happening; right? 12:25:37 12:25:44
A. Yes. 12:25:46
Q. And in the next bullet there's a reference to "sublethal toxic effects." 12:25:46 12:25:53
Do you see that? 12:25:56
A. Uh-huh. 12:25:56

Q. And there's a reference to how those types of effects can cause subtle changes in community structure. 12:25:57 12:26:03

Do you see that? 12:26:07

A. Uh-huh. 12:26:09

Q. Is that one of the reasons that Part 1 includes a sublethal toxicity test? 12:26:10 12:26:15

A. Umm -- no. 12:26:19

I mean -- umm -- that's not the -- umm -- main reason. 12:26:32 12:26:39

The sublethal toxicity tests were included with the acute to -- because they represent different organisms, different exposures, and were thought to potentially respond to -- umm -- exposures that may not be picked up with the benthic community indices. 12:26:47 12:26:53 12:26:57 12:27:03 12:27:12

Q. So is the converse true, that a good community structure would suggest the absence of sublethal toxic effects? 12:27:16 12:27:21 12:27:25

A. Not necessarily. 12:27:25

I guess the -- umm -- the individual Lines of Evidence may -- umm -- have various strengths and weaknesses, which is going back to the reason why we're using multiple Lines of Evidence. 12:27:33 12:27:40 12:27:52 12:27:57

If one of these indicators was clearly better than the others, we would have gone with that one -- or we would have gone with one. 12:28:01 12:28:07 12:28:15

Q. You agree that the benthic index is -- 12:28:18

Strike that. 12:28:26

You agree that -- that the Line of Evidence 12:28:27

based on benthic community structure is the ultimate 12:28:30

endpoint; right? 12:28:34

A. It's the ultimate endpoint in relationship to 12:28:35

the Narrative Objective that protects the communities, 12:28:40

but that doesn't necessarily mean it's -- can't be 12:28:47

confounded or -- is necessarily the most precise. 12:28:51

Q. It's -- 12:29:00

Because it's the ultimate endpoint it's given 12:29:04

greater weight in the assessment; right? 12:29:08

A. It is given -- umm -- yes. 12:29:10

Yes. 12:29:15

MR. SINGARELLA: Turn -- 12:29:26

Well -- I'm sorry. 12:29:27

Did the food show up already? 12:29:29

MR. RICHARDSON: Yes.

MR. SINGARELLA: Well, know what? 12:29:32

12:30, you know, say you break at 12:00 or 12:29:33

12:30, shall we go off the record now, Mr. Beegan, or -- 12:29:38

THE WITNESS: I mean, that's up to you guys. 12:29:41

I can keep going if you want. 12:29:42

MR. SINGARELLA: Okay. You want to go until 12:29:44

-- we'll try another 15 -- 12:29:46

THE WITNESS: Sure. 12:29:47

MR. SINGARELLA: -- to 30 minutes? 12:29:48

THE WITNESS: Sure. 12:29:50

MR. SINGARELLA: I don't think the food is going to rot. 12:29:51 12:29:53

BY MR. SINGARELLA: 12:29:54

Q. Okay. Turning to 5-9, please -- third full paragraph refers to the "limited utility of sediment -- as a result of the factors described above"? 12:30:00 12:30:12 12:30:18

A. Okay. 12:30:21

Q. And it refers to "the limited utility of sediment quality indicators based on concentrations in sediment." 12:30:21 12:30:26 12:30:31

Do you see that? 12:30:31

A. Yes. 12:30:32

Q. And that's referring to constituent concentrations of chemicals in sediment; right? 12:30:32 12:30:34

A. Correct. 12:30:37

Q. And to what extent do those chemical concentrations have limited utility? 12:30:38 12:30:43

A. Umm -- I think we went over a lot of that earlier this morning. 12:30:46 12:30:54

We talked about the different factors affecting the empirical -- empirical guidelines. 12:30:55 12:30:57

Umm -- they're -- they're unreliable as 12:31:03

stand-alone indicators. 12:31:15

Q. And to what extent should their use be limited? 12:31:18 12:31:23

A. Umm -- the empirical guidelines uses should be limited to the -- umm -- assessment of chemical mixtures, to evaluate the potential for -- umm -- pollutant-associated effects. 12:31:23 12:31:30 12:31:39 12:31:48

I wouldn't go much further than that. 12:31:55

Q. Uh-huh. 12:31:57

And then in the next paragraph there's a reference to "the potential flaws of the other Lines of Evidence." 12:31:59 12:32:03 12:32:08

Do you see that? 12:32:08

A. Uh-huh. 12:32:09

Q. And you agree that the other Lines of Evidence have potential flaws? 12:32:12 12:32:16

A. Yes. 12:32:19

Q. And then further down in that paragraph there's a reference to "the hierarchal response scheme." 12:32:21 12:32:26

Do you see that? 12:32:35

A. Is that still on 5-9? 12:32:36

Q. 5-9, yeah. 12:32:38

A. Yes. Okay. 12:32:41

Q. What is the "hierarchal response scheme" referred to in that paragraph? 12:32:42 12:32:45

A. Umm -- it -- umm -- it's a scheme that basically breaks down this concept where effects will occur first at the cellular level within an organism and then they'll affect the whole organism, and then they'll affect the community. 12:32:47 12:32:54 12:33:04 12:33:11 12:33:18

So -- okay. 12:33:25

Basically it describes it in there. 12:33:37

Q. And this paragraph refers to that scheme as a "paradigm;" correct? 12:33:39 12:33:46

A. Umm -- right. 12:33:47

I guess that paragraph was included because some had made an argument that valued the toxicity test more so than benthic community indicator, and I guess ideally following that paradigm you would think it would be true, but it's not, and -- umm -- this sediment quality assessment -- because there's just too much -- umm -- uncertainty within our -- our variability than these individual lines. 12:33:53 12:33:57 12:34:07 12:34:16 12:34:19 12:34:26 12:34:31 12:34:38

Q. And is this the paradigm that was the understanding that led to development of water quality standards for the water column? 12:34:39 12:34:49 12:34:54

A. Umm -- 12:35:01

MR. FUCHS: Lacks foundation. Calls for speculation. 12:35:03 12:35:04

BY MR. SINGARELLA: 12:35:05

Q. Well, let's look at the paragraph. 12:35:06

It indicates that that paradigm formed the 12:35:08

basis for water quality control. 12:35:12

Do you see that, Mr. Beegan? 12:35:14

A. Should I go ahead? 12:35:16

MR. FUCHS: He's referring to a particular 12:35:18

sentence. 12:35:19

MR. SINGARELLA: Yeah. I'll just try it right 12:35:20

in to this sentence here to address Mr. Fuchs' 12:35:22

objection. 12:35:25

I'm only trying to understand this paragraph. 12:35:25

THE WITNESS: Okay. 12:35:28

BY MR. SINGARELLA: 12:35:28

Q. So this paragraph refers to water quality 12:35:29

control. 12:35:32

I was trying to get to the bottom of it, and I 12:35:33

thought that what you were referring to here is the use 12:35:36

of this paradigm to form, you know, water quality 12:35:39

standards. 12:35:44

A. Yes. 12:35:47

Q. And was your copper example from the other 12:35:48

exhibit -- was that the type of analysis you do in this 12:35:55

paradigm, compare concentrations versus some biological 12:36:00

endpoint -- 12:36:04

A. Yes. 12:36:05

Q. -- and relationship? 12:36:06

A. Yes. 12:36:08

Q. Here it says that that particular paradigm has never been proved in sediments. 12:36:08 12:36:14

Do you see that? 12:36:16

A. Right. 12:36:17

Q. And that's a reference to Griffith, et al., 2008; right? 12:36:17 12:36:22

A. Yes. 12:36:23

Q. An obscuring of the differences between methods. 12:36:24 12:36:33

Do you see that? 12:36:34

A. Yes. 12:36:34

Q. What does that refer to? 12:36:34

A. I -- umm -- I think what it means is that with the -- these many confounding factors, or variability between tests, or -- umm -- variability within results we can't tease out the -- or we -- we -- we can't come to a conclusion that the -- umm -- the toxicity test, the organism test, is more sensitive than the community tests. 12:36:36 12:36:42 12:36:48 12:36:55 12:37:03 12:37:09 12:37:14

Q. Yet in the water quality control situation the application of this paradigm leads to knowledge of dose versus response; correct? 12:37:15 12:37:23 12:37:26

A. Yes, the toxicity-spiked water column 12:37:28

bioassays have been used -- have been relied on for the development of water quality criteria. 12:37:38 12:37:44

Q. In the specification of the dose response relationship; correct? 12:37:47 12:37:49

A. Yes. 12:37:50

Q. And here at the end of this paragraph it refers to a number of factors weaken this relationship. 12:37:51 12:37:55

Do you see that? 12:37:59

A. Yes. 12:38:00

Q. And that's a reference to the sediment context; correct? 12:38:01 12:38:08

A. I believe so, yes. 12:38:08

Q. And so the first bullet under "Weaknesses" refers to different sensitivities to different contaminants. 12:38:10 12:38:18 12:38:23

Do you see that? 12:38:24

A. Yes. 12:38:26

Q. And what is -- what does that mean? 12:38:27

Relevant to test species. 12:38:31

A. Umm -- different organisms will respond differently to -- umm -- contaminants. 12:38:44 12:38:48

Q. And how is that -- how is that a weakness? 12:38:54

A. Well, I guess -- umm -- it's a weakness because you -- there's no such thing as an all-encompassing sensitive organism, that is, most 12:39:04 12:39:33 12:39:37

sensitive that you could apply to be kind of your -- your primary sentinel organism for -- establishing a level that would necessarily be protective of all other benthic communities. 12:39:50 12:39:53 12:40:04 12:40:11

Q. None of the animals used in the sediment toxicity Line of Evidence are -- are such all-encompassing organisms? 12:40:19 12:40:22 12:40:25

A. No. 12:40:27

Q. Those are traditional test species; right? 12:40:30

A. They're traditional test species that were selected because of various criteria, one of which was sensitivity, but, as I said before -- umm -- none of those species is going to represent all potential -- umm -- contaminant-type exposures in the sediment. 12:40:36 12:40:40 12:40:46 12:40:51 12:41:01

Umm -- feeding strategies -- umm -- metabolism -- umm -- all those factors which affect an organism's specific response. 12:41:07 12:41:14 12:41:19

So -- 12:41:20

Q. Do they come from the site? 12:41:22

A. No. 12:41:24

Q. Where do they come from? 12:41:25

A. Umm -- suppliers. 12:41:26

Are you talking about the laboratory test organisms? 12:41:34 12:41:38

Q. Yes. 12:41:39

A. Yeah. 12:41:40

Some -- I believe most of them come from 12:41:40

suppliers. Some may be -- 12:41:43

Yeah, they all -- I think all of them come 12:41:46

from suppliers. 12:41:49

Q. What do you mean by a "supplier"? 12:41:52

A. I think there are -- there are -- umm -- 12:41:54

companies out there that grow, collect test organisms 12:42:02

for the purposes of -- umm -- performing bioassays, 12:42:10

sediment bioassays. 12:42:19

Q. So the test species are not taken from the 12:42:22

in-situ benthic community? 12:42:27

A. Right. 12:42:28

Q. In that sense are they a surrogate? 12:42:29

A. Yes. 12:42:34

Q. Is sediment toxicity a surrogate measure for 12:42:35

the benthic community? 12:42:43

A. Umm -- at one time I believe it was thought to 12:42:44

be. 12:42:54

Now it's thought to be a -- umm -- a test that 12:42:54

-- umm -- isn't -- it's not intended to be a surrogate. 12:43:08

It's intended to measure -- umm -- 12:43:15

invertebrate response under a specific set of controlled 12:43:20

conditions, and that information is -- umm -- different 12:43:26

than what you get when you collect a benthic community 12:43:35

grab. 12:43:40

Q. If the test species is harmed in the lab test, does that mean that the benthic community at the site is harmed? 12:43:41 12:43:44 12:43:48

A. Not necessarily. 12:43:48

Q. Those test species may not even be present at the site; right? 12:43:49 12:43:55

A. May not be. 12:43:55

Q. On page 5-10, next page, Mr. Beegan, there's three more bullets. 12:43:56 12:44:01

I'd like to turn your attention to the last bullet, the "presence of natural factors." 12:44:02 12:44:05

Do you see that? 12:44:07

A. I'm sorry? 12:44:09

What -- "presence of --" 12:44:11

Yeah, sure. 12:44:13

Q. And that refers to "spurious results." 12:44:13

Do you see that? 12:44:19

A. Yes. 12:44:19

Q. How can the presence of natural factors produce spurious results? 12:44:20 12:44:25

A. I think we discussed that earlier under -- when we were talking about the water quality control plan. 12:44:27 12:44:34 12:44:36

We discussed confounding factors at length. 12:44:37

It's basically discussing -- it's the same thing, same concept. 12:44:43 12:44:46

Q. I think you had mentioned nutrients as a potential confounding factor; right? 12:44:47 12:44:51

A. Yes. 12:44:54

Q. Is ammonia one of the nutrients you had in mind? 12:44:54 12:44:58

A. Yes. 12:44:58

Q. Is hydrogen sulfide a confounding factor? 12:44:58

A. Potentially. 12:45:05

Q. What does "physical abrasion" mean? 12:45:06

A. Ooh. That's a good question. 12:45:09

Q. I'm glad I asked. 12:45:11

A. Yeah. 12:45:15

Scour? 12:45:18

I -- that's -- that's -- seems -- I -- would assume that that is referring to scour. 12:45:18 12:45:25

Q. And in what sense would the results be spurious? 12:45:30 12:45:36

A. Umm -- they would be -- umm -- the results would -- umm -- could potentially mislead you. 12:45:37 12:45:44

The results could be misinterpreted. 12:45:53

Q. And this is a weakness of the sediment toxicity line? 12:45:55 12:46:00

A. Umm -- yes. 12:46:00

Q. And why the hierarchal response scheme and a paradigm don't -- don't fit well in the sediment toxicity context? 12:46:09 12:46:16 12:46:22

A. Umm -- it doesn't fit well when you're trying to rank -- umm -- indicators and their importance in an assessment framework -- so subtle difference there. 12:46:23 12:46:30 12:46:39

Q. The bullet just above that "Toxicity tests do not mimic the sediment structure" and other things, do you see that? 12:46:46 12:46:52 12:46:56

A. Yes. 12:46:56

Q. What does that mean? 12:46:56

A. The -- umm -- toxicity tests are performed on disturbed samples. 12:46:58 12:47:04

By "disturbance" I mean when you take your benthic grab, structure the -- the way the sediments are layered, all that is disturbed when you take the grab. 12:47:08 12:47:11 12:47:20

Umm -- all the conditions that the benthic community are exposed to are based on that naturally occurring structure. 12:47:35 12:47:38 12:47:43

So the point in this bullet is that it's a disturbed sample, therefore, conditions in your toxicity test do not mimic those found in the -- umm -- natural surficial sediments. 12:47:43 12:47:46 12:47:52 12:48:01

Q. How can one ensure that the lab test is representative of the field condition? 12:48:02 12:48:05

A. Umm -- that's pretty difficult to accomplish in the lab. 12:48:07 12:48:19

Q. So is this weakness that disturbed sediment tested in the lab may not be representative of the field condition? 12:48:20 12:48:26 12:48:29

A. Yes. 12:48:30

Q. And, in fact, that's proven to be the case; right? 12:48:30 12:48:39

A. Yes. 12:48:39

MR. SINGARELLA: What I would like to do, Mr. Beegan, is when we get back from our sandwich break I've prepared a grossly over-simplified example of the MLOE, and I don't think we need to spend a lot of time on it, but I just want to make sure through this simple example that we understand how it works. 12:48:52 12:48:54 12:49:01 12:49:05 12:49:09 12:49:12

So I'm going to have it marked, and I'll put it in now, and we can -- we can discuss it after our break. 12:49:15 12:49:17 12:49:21

(Exhibit 705 was marked for Identification.) 12:49:22

MR. SINGARELLA: 705? 12:49:29

THE REPORTER: Correct. 12:49:30

MR. SINGARELLA: This is an attempt to honor the time honored principle, keep it simple. 12:49:53 12:49:55

THE WITNESS: Okay. 12:50:00

BY MR. SINGARELLA: 12:50:00

Q. I've placed in front of you what has been marked as Exhibit Number 705 to your deposition. 12:50:01 12:50:03

Do you see that? 12:50:07

A. Yes. 12:50:08

Q. This is a document that we prepared, Mr. Beegan. 12:50:08 12:50:11

Do you understand that? 12:50:13

A. Yes. 12:50:14

Q. And I simply want to ask you a few questions about it after our lunch break; is that okay? 12:50:15 12:50:17

A. Okay. 12:50:20

MR. SINGARELLA: Okay. I suggest we go off the record now, if that's okay with everyone. 12:50:24 12:50:26

VIDEOGRAPHER: We're going off the record at 12:50 p.m. 12:50:29 12:50:32

(Thereupon a recess was taken at 12:50 p.m. and the deposition resumed at 1:31 p.m.) 12:50:33 13:31:19

VIDEOGRAPHER: We are back on the record at 1:31 p.m. 13:31:19 13:31:22

BY MR. SINGARELLA: 13:31:23

Q. Mr. Beegan, good afternoon. 13:31:24

Before lunch I placed in front of you what has been marked as Exhibit Number 75 -- excuse me -- Exhibit Number 705 to your deposition. 13:31:26 13:31:29 13:31:34

Do you see that? 13:31:36

A. Yes. 13:31:36

Q. And I'm wondering if you can indulge me just 13:31:37
for a few minutes to make sure we understand how to work 13:31:41
through the MLOE. 13:31:44

Is that okay? 13:31:46

A. Okay. 13:31:46

Q. And you'll see that by coincidence we ended up 13:31:46
with a sediment toxicity example that is very similar to 13:31:50
the one we discussed this morning, using the 13:31:54
Eohaustorius and Mytilus. 13:32:01

Do you see that? 13:32:01

A. Uh-huh. 13:32:02

Q. Could you confirm that if you use values of 92 13:32:03
percent for survival and 80 percent for the sublethal 13:32:08
effect you end up with a result of nontoxic for the 13:32:14
sediment LOE? 13:32:18

A. Okay. Yes. I confirm. 13:32:19

Q. Thank you. 13:33:00

And how did you get to your conclusion? 13:33:01

A. Well, I made -- I looked at -- umm -- Table 4. 13:33:03

I looked at the -- Eohaustorius survival. 13:33:11

I made an assumption that there was 13:33:20
statistical difference -- statistical significance. 13:33:29

I went to the -- the row that was 90 to a 13:33:34
hundred, and I did the same for -- umm -- Mytilus 13:33:40

Normal. 13:33:53

Q. Okay. Thank you. 13:33:53

Now, moving to the benthic line, and with 13:33:54

reference to Exhibit 705, and Table 5 and Part 1, which 13:34:02

is on page 7, do you agree that the four values in 13:34:11

Exhibit 705 for BIR, IBI, RBI, and RIVPACs produce a 13:34:20

result using Table 5 of low disturbance? 13:34:29

A. Correct. 13:34:33

Q. And how did you reach that conclusion, sir? 13:34:33

A. I prepared an index value to the numbers 13:34:36

listed in the rows, and all four of the values were -- 13:34:40

values for each index fall in to the range -- umm -- 13:34:50

characterized as low disturbance in the Table 5 benthic 13:34:56

index categorization values. 13:35:00

Q. As you can see from Exhibit 705, the next step 13:35:03

in the illustration which we furnished you is to turn to 13:35:07

Table 9. 13:35:11

Do you see that? 13:35:12

Do you see that on Exhibit 705? 13:35:21

A. Yes. Yes. 13:35:23

Q. Is that an appropriate next step? 13:35:24

A. Let's see. 13:35:26

Yes. 13:35:30

Q. And did we use Table 9 correctly, taking the 13:35:44

value of sediment toxicity or the category of nontoxic 13:35:50

and the benthic category of low disturbance to determine 13:35:55
based on Table 9 that the severity of biological effects 13:35:59
is unaffected? 13:36:04
A. Yes. 13:36:05
Q. And how did you reach that conclusion? 13:36:06
A. Umm -- the Table 9 has -- umm -- toxicity 13:36:11
categories horizontally, going across the top, and the 13:36:23
benthic community condition categories vertically on the 13:36:32
right. 13:36:39
So you align your nontoxic -- or you identify 13:36:40
the nontoxic category for toxicity where it meets the 13:36:48
low disturbance category for Benthos. 13:36:54
There's an easier way in the back, but -- this 13:37:01
is -- follows the conceptual approach. 13:37:04
Q. Just for purposes of being complete, what is 13:37:09
the easier way in the back, sir? 13:37:12
A. It -- it -- it uses Attachment B where you 13:37:14
basically start with bullet -- 13:37:19
Umm -- it has each -- 13:37:25
It's not necessarily simpler. It results in 13:37:34
the same thing. Some people find Attachment B simpler 13:37:38
to use. 13:37:42
In this case there -- one LOE is held constant 13:37:44
while you run through all the other potential categories 13:37:52
for the other Lines of Evidence. 13:37:56

It's just a different way of resulting -- coming up with the same result. 13:38:00 13:38:05

Again, it was provided because some people thought that that would be easier. 13:38:07 13:38:10

Q. Thank you. 13:38:12

In our example, Exhibit 705, we next proceeded to Table 10. 13:38:12 13:38:18

Is that an appropriate next step? 13:38:20

A. I'm sorry. 13:38:23

Table 10? 13:38:26

Proceeding to Table 10? 13:38:27

Q. Yes. 13:38:29

A. Let me get there. 13:38:30

Yes. 13:38:31

Table 10 is the next one. 13:38:38

Q. And there we combined our assumption of high exposure for sediment chemistry with our conclusion of low disturbance for the benthic, which we -- we think results in a moderate potential. 13:38:40 13:38:45 13:38:48 13:38:56

Do you agree with that? 13:38:58

A. Let's see. 13:39:03

Exposure on the chemistry side? 13:39:04

And low disturbance on the -- 13:39:09

Yes, I agree. 13:39:43

You're -- you're -- I think used the term 13:39:46

"benthos" in there, and that was confusing. 13:39:50

Q. Is there a better term? 13:39:52

A. Well, I think we're at -- we're on Table 10, which is chemistry and toxicity, so I think you were confused with the previous table that was toxin benthos. 13:39:54 13:39:57 13:40:01

Q. Yes, Table 10. 13:40:07

A. Sediment toxicity plus chemistry. 13:40:12

Q. Yes. Thank you. 13:40:19

So is our formula -- 13:40:21

A. Yeah, so far it seems fine. 13:40:28

Q. But under "chemically mediated" we're combining -- 13:40:30 13:40:33

A. Sediment toxicity and chemistry. 13:40:34

Q. And the chemistry is high exposure but the sediment toxicity is nontoxic; right? 13:40:37 13:40:44

A. It's low disturbance; right? 13:40:47

How did we -- 13:40:50

I think there's an error in there. 13:40:51

Q. There may be. I want to explore that with you. 13:40:52 13:40:55

A. Okay. Unless -- 13:40:55

Q. This is why I wanted to go through an example. 13:40:56

This is a surprise to me if there is? 13:40:59

A. Wait. 13:41:01

Chemistry -- nontoxic -- 13:41:03

So that would be nontoxic and -- high exposure -- 13:41:06

Oh, oh okay. So maybe they -- umm -- used the wrong -- umm -- that could be a typo. 13:41:09 13:41:13 13:41:17

Q. Referring to? 13:41:21

A. "Low disturbance." 13:41:23

Q. Yes. That should say "nontoxic." 13:41:25

Do you agree? 13:41:28

A. Yes. 13:41:29

Q. Could you -- could you make that correction? 13:41:29

A. Oh, yeah. I am. I am. 13:41:31

Q. Thank you. 13:41:32

A. I am. 13:41:33

Q. Thank you. 13:41:34

I think you were doing that in your head for us, but if -- 13:41:40 13:41:43

Do you agree that with -- with your correction -- 13:41:44 13:41:46

A. High exposure and nontoxic is -- umm -- 13:41:49

High exposure, nontoxic is moderate potential. 13:41:59

MR. SINGARELLA: Yes. 13:42:05

Thank you. 13:42:06

Let the record show that Mr. Beegan corrected our Exhibit 705 by changing the phrase "nondisturbance" to "nontoxic." 13:42:07 13:42:10 13:42:15

MR. FUCHS: "Low disturbance." 13:42:18

MR. SINGARELLA: By changing the phrase "low disturbance" to "nontoxic." 13:42:20 13:42:22

Thank you. 13:42:24

BY MR. SINGARELLA:

Q. And do you agree that the next appropriate step is to move on to Table 11? 13:42:25 13:42:29

A. Yes. 13:42:31

Q. And did we appropriately use Table 11 in light of the unaffected bioeffects and the moderate potential for chemically mediating effects? 13:42:32 13:42:40 13:42:44

A. Umm -- yes. 13:42:48

And you got -- umm -- likely unimpacted, which is what I get. 13:42:53 13:42:57

Q. Okay. Thank you. 13:42:59

Turning back to the benthic community condition on page 6 of Exhibit 6, Part 1 -- 13:43:01 13:43:10

So we're on page 6 of Part 1. 13:43:15

You were looking at Exhibit 705. 13:43:26

Is there any follow-up on it? 13:43:30

A. A -- no. 13:43:32

I was just -- umm -- no. 13:43:33

Q. Okay. But you agree that with your one correction, and thank you, that applying -- 13:43:35 13:43:40

A. Well, -- 13:43:45

Q. -- the MLOE properly? 13:43:46

A. I would probably -- umm -- 13:43:48

We -- umm -- 13:43:54

Yeah. Yeah, you are. 13:43:59

Yeah. Yeah. Yeah. Yeah. 13:44:00

Q. Thank you. 13:44:01

Okay. Under "Benthic Community Condition" on page 6 -- 13:44:02 13:44:08

A. Umm -- "Benthic Community Condition." Page 6. 13:44:08

Q. -- I didn't see anything in here that was similar to the averaging approach taken for sediment toxicity. 13:44:13 13:44:17 13:44:20

Am I missing something? 13:44:23

A. Oh. 13:44:25

Q. Is it -- is it this median approach in the bottom paragraph on page 6, "The median of all benthic index response categories shall determine"? 13:44:38 13:44:44 13:44:49

A. Yes. 13:44:51

Q. So in your own words how do you combine -- 13:44:52

A. Umm -- 13:44:58

Q. Sorry. 13:45:00

A. Oh, no. 13:45:01

I guess if there was more, please finish -- the question. 13:45:01 13:45:05

Q. In your own words how would you combine 13:45:06

multiple benthic condition results from the same station? 13:45:08 13:45:13

A. In -- umm -- in this case what -- umm -- you would assign a category to each -- each indicator, and you would take the median category and use that in the -- umm -- in the assessment. 13:45:14 13:45:30 13:45:41 13:45:49

Does that make sense? 13:45:54

Q. It does. 13:45:55

So let's take an example. 13:45:56

For -- let's just say RIVPACs. 13:45:59

If you had four replicates for a station, one turned out to be reference, one turned out to be low disturbance, one turned out to be moderate disturbance, and one turned out to be high disturbance. 13:46:02 13:46:06 13:46:09 13:46:14

Are you with me? 13:46:16

A. Yeah, that -- I'm with you. 13:46:17

But we were thinking of -- umm -- a sample where RIVPACs gives you low disturbance, RBI gives you low disturbance, IBI gives you the next highest one, and maybe -- umm -- BRI gives you low disturbance. 13:46:22 13:46:33 13:46:45 13:46:51

And the -- 13:46:57

Q. But before -- give me your example again, please. 13:47:00 13:47:03

A. Okay. 13:47:04

Q. So you would give me a situation where you 13:47:04

have four tests from one station? 13:47:10

A. You -- so at each -- 13:47:13

For each station you apply all four metrics; 13:47:17

right? 13:47:20

Q. Right. 13:47:21

A. Not all four -- 13:47:22

Unlike your example, the -- one -- 13:47:24

The indexes may provide you with a slightly 13:47:33

different -- or a different category. 13:47:37

So you could have a BRI saying its reference, 13:47:39

an IBI saying it's -- what's the next one up? Low 13:47:43

disturbance, the RBI saying it's reference, and RIVPAC 13:47:48

saying it's reference. 13:47:53

You would apply the median to those 13:47:54

categories. 13:47:59

Does that make sense? 13:47:59

Q. Yes. 13:48:01

And what would you get in that scenario? 13:48:01

A. In that scenario you would get a reference. 13:48:04

Q. Because you have three reference and one low 13:48:06

disturbance? 13:48:09

A. Yes. 13:48:10

That was why -- umm -- that was the intent of 13:48:13

G benthic community condition 4, to do -- umm -- to 13:48:22

integrate when you have -- or to -- to come up with your 13:48:30

final benthic community category when you have individual indexes that don't all match or don't all give you the same categorical response 13:48:37 13:48:42 13:48:47

Q. Can we return to my example? 13:48:49

A. Yeah. 13:48:55

Q. If you actually had the same result, three reference and one low disturbance, but it was for one of the benthic indices because you were doing replicates at a site, would you apply the same logic? 13:48:56 13:49:01 13:49:06 13:49:10

A. Umm -- not necessarily. 13:49:12

I'd have to think about that. 13:49:21

Again, -- 13:49:23

Q. Okay. 13:49:24

A. -- that was not the -- the -- the basis for that integration of benthic categories. 13:49:24 13:49:29

Integration of benthic categories, again, was -- was to be applied to a given sediment that you had collected benthic community information on, and then you applied these four -- umm -- tools, and in the case where you didn't just get low disturbance all the way down the line, you say, okay, take the median value. 13:49:33 13:49:36 13:49:42 13:49:46 13:49:54 13:49:57

Q. Thank you. 13:50:01

Question on page 7 regarding the California LRM: Is that limited to amphipods toxicity results and the correlation of those results to sediment chemistry? 13:50:02 13:50:07 13:50:20

A. I don't recall specifically which test organisms it relates to, but it does relate to toxicity test organism response to -- umm -- sediment chemistry. 13:50:26 13:50:37 13:50:43

Q. Do you recall what "test organisms CSI" relates to? 13:50:49 13:50:54

A. It doesn't relate to test organisms necessarily. It relates to benthic community health. 13:50:55 13:50:59

Q. Do you recall that the LRM approach does relate to sediment toxicity results? 13:51:04 13:51:10

A. Oh, yes. 13:51:14

I think -- umm -- yeah. 13:51:15

Q. But the CSI is actually linked to benthic community directly? 13:51:17 13:51:23

A. Yes. 13:51:24

Q. And so the CSI involves the available information on benthic community structure at a site; right? 13:51:24 13:51:33 13:51:37

A. The CSI related statewide sediment chemistry to -- benthic community response on -- umm -- on a regional or a statewide basis. 13:51:37 13:51:50 13:52:00

So -- relating it to a given site, that's not entirely accurate. 13:52:02 13:52:09

Q. I'm just trying to get behind the biological nexus on the CSI versus the LRM. 13:52:10 13:52:22

One seems to tie to benthic community 13:52:27

structure, the actual critters in-situ at CSI; right? 13:52:29

A. Right. 13:52:34

Q. And the LRM is not tied directly to the in-situ information; right? 13:52:35 13:52:39

A. Correct. 13:52:40

Q. It's tied to those laboratory tests that we talked about this -- earlier today? 13:52:41 13:52:46

A. Correct. 13:52:48

Q. And so it's tied to the response of test species to disturbed sediment in a laboratory condition? 13:52:48 13:52:54

A. Correct. 13:52:59

Q. What you're not sure of is which test species are used to build the LRM? 13:53:00 13:53:05

A. Right. I mean -- 13:53:08

Q. We can find it? 13:53:09

A. -- I'm sure it's in there -- somewhere. 13:53:11

Q. Yeah. 13:53:13

Let's turn back to this Staff Report, Mr. Beegan. 13:53:17 13:53:24

We were on page 5-10. 13:53:25

This is Exhibit 704. 13:53:31

We were at the top there after those bullets. 13:53:38

And first full paragraph, the "however" sentence -- 13:53:49 13:53:55

A. I'm sorry. 13:53:57

5-10? 13:53:59

Q. 5-10, the first full paragraph there after the bullets? 13:54:00 13:54:05

A. Oh. 13:54:05

Q. I want to talk about that paragraph. 13:54:06

A. Okay. 13:54:08

Q. And there's a "however" sentence refers to "their use alone is problematic"? 13:54:08 13:54:12

A. Right. 13:54:14

Q. What does "their use" refer to? 13:54:15

A. Oh, benthic community, -- 13:54:17

Q. Okay: 13:54:18

A. -- "their use," 13:54:19

Q. Okay. And it indicates that the benthic community can be potentially affected by a large number of factors other than chemical contamination; correct? 13:54:27 13:54:30 13:54:33

A. Uh-huh. 13:54:37

Q. And then the next sentence -- 13:54:37

A. Excuse me. 13:54:42

Q. Sure. 13:54:43

The next sentence indicates that "Without the other lines it's difficult to distinguish --" 13:54:44 13:54:47

Right? 13:54:50

A. Correct. 13:54:52

Q. "Whether degraded benthic communities resulted 13:54:53

from chemicals or something else;" right? 13:54:57

A. Uh-huh. Yes. 13:55:00

Q. So this seems to be a rational basis for using chemistry and toxicity data when the benthic community is degraded; correct? 13:55:01 13:55:10 13:55:16

A. Umm -- 13:55:17

Sure. 13:55:39

Q. I mean, there's a reference to "degraded benthic communities" right in this sentence; right? 13:55:40 13:55:43

A. Yes. 13:55:46

Q. And the notion is be careful not to assume that the degradation resulted from chemical exposure; right? 13:55:46 13:55:50 13:55:55

A. Correct. 13:55:55

Q. Because it might also result from all these other confounding factors; right? 13:55:55 13:55:58

A. Right. 13:56:00

Q. So that suggests not to use benthic community structure in isolation when the community structure suggests a problem; right? 13:56:01 13:56:07 13:56:11

A. Umm -- yes. 13:56:13

It's an example of a -- umm -- a weakness within the Line of Evidence. 13:56:18 13:56:24

Q. That weakness does not pertain to the situation where the benthic community is actually 13:56:25 13:56:31

healthy; right? 13:56:34

A. Umm -- that is not -- one hundred percent 13:56:35
accurate, and I guess what I'm going on is -- umm -- my 13:56:47
memory from various discussions we had in developing the 13:56:54
Lines of Evidence. 13:57:07

Umm -- we don't know with one hundred percent 13:57:10
certainty that -- umm -- our benthic community measures 13:57:18
are sensitive or measuring the right properties. 13:57:27

So there is some uncertainty in our model of 13:57:38
benthic community health, and -- umm -- therefore, the 13:57:47
-- the -- I think it was the Scientific Steering 13:58:00
Committee and -- was adamant that we not just use 13:58:04
benthic community by itself no matter what the situation 13:58:12
was. 13:58:17

Q. Okay. Fair enough. 13:58:17

But I'm speaking to this sentence itself. 13:58:23

A. Oh. Yeah. Yeah. Yeah. 13:58:26

Okay. Yeah. Yes. 13:58:27

Q. This sentence itself -- 13:58:28

A. Sure. 13:58:30

Q. Excuse me. 13:58:31

This sentence itself does not provide a 13:58:32
rational basis to use chemistry or toxicity data coupled 13:58:34
with benthic community data when the benthic community 13:58:39
is in a reference or healthy condition? 13:58:43

A. Yes, I would agree with that. 13:58:45

Q. Thank you. 13:58:47

And at the bottom of the page, which carries over to page 5-11, there's a discussion of the BPJ we discussed before. 13:58:48 13:58:59 13:59:04

Do you see that? 13:59:05

A. Uh-huh. Yes. 13:59:06

Q. And the carryover sentence says "BPJ will be ineffective for use in SQOs." 13:59:07 13:59:12

Do you see that? 13:59:15

A. Uh-huh. 13:59:16

Q. And why is BPJ ineffective for use in SQOs? 13:59:17

A. Umm -- one expert would make an assumption about which categories to apply to -- umm -- an individual Line of Evidence or even the selection of the Lines of Evidence or -- umm -- experts will do slightly different things or -- umm -- assess sediment differently. 13:59:25 13:59:48 13:59:55 13:59:59 14:00:11 14:00:21

They'll have different ideas. 14:00:23

They may interpret the data slightly differently, which could result in differences in, you know, how you would rank a site or prioritize a site -- umm -- in -- 14:00:27 14:00:31 14:00:40 14:00:48

That could happen between water bodies. It could happen between regions. 14:00:53 14:00:56

We are -- 14:00:58

Q. And the particular MLOE approach required by Part 1 is in part designed to deal with the ineffective use of BPJ; right? 14:01:15 14:01:26 14:01:35

A. Yes. It gets rid of some of the BPJ -- umm -- and it probably -- quite a bit of the BPJ in the assessment process, but there's -- in terms of sediment quality and management there's a whole slough of places where it doesn't address. 14:01:38 14:01:46 14:01:51 14:01:56 14:02:02

Q. And this Part 1, by the way, it's not some menu where sediment quality specialists can pick and choose that which they want to do; right? 14:02:06 14:02:10 14:02:15

A. Right. Correct. 14:02:17

Q. It is an attempt to prescribe a method; right? 14:02:18

A. Yeah, it prescribes a method. 14:02:22

It -- yeah. 14:02:26

It's -- 14:02:29

Yes. Yes. 14:02:31

I would agree with that. 14:02:32

Q. And so, if one were to take a different approach and pick and choose that which they wanted to do in your MLOE, would it introduce the subjectivity associated with the BPJ? 14:02:33 14:02:39 14:02:42 14:02:47

MR. FUCHS: Calls for speculation. 14:02:50

Lacks foundation. 14:02:52

You can go ahead and answer if you -- if you can provide an answer 14:02:56 14:02:58

THE WITNESS: Umm -- yes. 14:02:59

BY MR. SINGARELLA: 14:03:04

Q. Okay. On page 5-12 there's a discussion of these different alternatives for the form of the SQO. 14:03:15 14:03:19

Do you see that? 14:03:25

A. Uh-huh. 14:03:26

Q. And the staff recommendation is Alternative 3; right? 14:03:26 14:03:29

A. Yes. 14:03:29

Q. And that is the narrative approach; correct? 14:03:32

A. Yes. 14:03:34

Q. An Alternative 4, the numeric approach, was rejected; correct? 14:03:36 14:03:39

A. Correct. 14:03:40

Q. And one of the reasons that it was rejected is that there isn't enough data collected; right? 14:03:40 14:03:45

A. Umm -- yeah. 14:03:49

I mean, there's a far simpler reason. 14:04:05

Q. Which is? 14:04:09

A. Umm -- we don't know -- 14:04:09

If you were to assign numerical values to those categories, unimpacted, like unimpacted, possibly impacted, likely impacted, clearly impacted, say 1 14:04:15 14:04:19 14:04:32

through 5, you have no way of evaluating those relationships, and what I mean by there's -- there's -- there would be an assumed mathematical relationship that possibly impacted is three times worse than unimpacted, and -- umm -- we don't have any information to be able to state one way or another whether that's true or not. 14:04:36 14:04:42 14:04:47 14:04:51 14:04:59 14:05:08

But those values, once they start being used for statistical purposes, would make some assumptions about those relationships, and they're simply not -- there's no rationale or no basis. 14:05:12 14:05:16 14:05:20 14:05:24

It's an arbitrary scale. 14:05:30

Q. Did you use the three times as an example -- 14:05:32

A. Yes. 14:05:35

Q. -- to illustrate your point? 14:05:35

A. Oh, yes. Absolutely. 14:05:36

It was -- yeah. Yeah. 14:05:39

Q. Whether it's three times worse or 30 times worse, there's just no rational basis to get at that; right? 14:05:40 14:05:46 14:05:50

A. Correct. 14:05:50

Q. Or it could be just 1.2 times worse; right? 14:05:51

A. Correct. 14:05:56

Q. We just don't know? 14:05:56

A. Right. 14:05:58

Q. You can't -- you can't take the relative 14:05:58

rankings by category and convert it in to some numeric expression? 14:06:04 14:06:08

A. Correct. 14:06:09

Q. Here in Alternative 4, which was rejected, there's a reference to a numeric scale. 14:06:09 14:06:17

Do you see that? 14:06:21

A. Yes. 14:06:23

Q. And is this what you were describing, this numeric scale? 14:06:23 14:06:26

A. Yes. 14:06:27

Umm -- yes. 14:06:29

Q. And it is concluded here that a scientifically defensible numeric cannot be developed. 14:06:30 14:06:35

Do you see that? 14:06:38

A. Yes. Yes. 14:06:38

Q. What does -- what does the "numeric" mean there? 14:06:39 14:06:43

A. I don't know. There's probably a missing word in there. 14:06:43 14:06:45

Q. Ahh. 14:06:46

A. Like -- between the various lines of evidence to create a valid numeric scale -- 14:06:47 14:06:50

Well -- umm -- 14:06:54

So to me -- geez. 14:06:55

Umm -- obviously, that could be better edited. 14:07:14

But -- umm -- what that sentence means to me is we don't have the data to develop a -- a -- these mathematical relationships between the categories. 14:07:20 14:07:26 14:07:34

At some point, if we collect enough data, perhaps we'll have enough information to develop some way to establish a numerical categoric goal ranking system. 14:07:37 14:07:41 14:07:47 14:07:55

Q. Does that mean that the more data you have the better chance you have of locating a particular station or site in the right category? 14:07:55 14:08:05 14:08:09

A. No. No. 14:08:11

That's not at all what I was trying to get at. 14:08:14

Q. What were you trying to get at? 14:08:18

A. Umm -- I was trying to -- umm -- describe what this sentence is attempting to address in terms of converting a narrative ranking, a station category system, in to a numeric category system -- umm -- those five categories, assign a value to them. 14:08:19 14:08:27 14:08:34 14:08:42 14:08:51

Q. Okay. At the bottom of page 5-12, three lines up there's a reference to sediment toxicity tests, and it says that they cannot reliably predict effects to the benthic communities. 14:08:54 14:09:05 14:09:09 14:09:14

Do you see that? 14:09:16

MR. FUCHS: Counsel, is that the sentence that begins "While this concept is logical"? 14:09:32 14:09:35

MR. SINGARELLA: That's right. 14:09:37

THE WITNESS: Umm -- okay. I see it. 14:09:38

BY MR. SINGARELLA: 14:09:45

Q. And what is it about sediment toxicity tests that make them an unreliable predictor of these effects? 14:09:48 14:09:56

MR. FUCHS: Calls for speculation. 14:10:01

MR. SINGARELLA: If you know. 14:10:03

THE WITNESS: Many of the factors we've talked about this morning. 14:10:04 14:10:16

BY MR. SINGARELLA: 14:10:17

Q. Okay. Such as the confounding factors? 14:10:17

A. Yes. 14:10:20

Q. Anything else? 14:10:21

A. Umm -- it's -- umm -- most of these factors -- umm -- I think I said this this morning -- affect the results at -- umm -- low -- low responses. 14:10:22 14:10:36 14:10:42

So the higher the magnitude of response the more the different Lines of Evidence start to respond in the same way. 14:10:52 14:10:55 14:11:04

Q. But this sentence here just refers to sediment toxicity tests in general; right? 14:11:05 14:11:10

A. Yes. 14:11:13

Q. And it appears to make the blanket statement that they're not reliable as the predictor to the effects to benthic communities; right? 14:11:21 14:11:24 14:11:27

A. Umm -- right. 14:11:30

Q. And then on to page 5-15, please. 14:11:33

I'd like to ask you a couple of questions about the alternatives considered for the sediment toxicity tests; okay? 14:11:49 14:11:51 14:11:57

A. Okay. 14:11:59

Q. And it turns out that Alternative 4 was recommended by staff; right? 14:12:01 14:12:04

A. I'm sorry. Go back to -- 14:12:06

What page are we on? 14:12:08

Q. I'm sorry. 14:12:09

Page 5-15 of the Staff Report. 14:12:11

A. Yes, Alternative 4. 14:12:17

Q. And that included the combination of acute and sublethal toxicity methods; right? 14:12:19 14:12:22

A. Yes. 14:12:26

Q. And staff rejected Alternative 2; correct? 14:12:26

A. Umm -- yes. 14:12:33

Q. And that would have specified only acute toxicity methods; right? 14:12:36 14:12:42

A. Correct. 14:12:44

Q. And two paragraphs up, the paragraph that says "Two sublethal test methods were recommended," do you see that? 14:12:44 14:12:53 14:12:56

A. Yes. 14:12:56

Q. And further down in that paragraph it indicates that the sublethal tests were recommended to complement the ability of acute tests to detect; right? 14:12:56 14:13:00 14:13:05

A. Correct. 14:13:11

Q. To detect toxicity; right? 14:13:12

A. Yes. 14:13:14

Q. It was important to complement the acute toxicity results; right? 14:13:14 14:13:21

A. Yes. 14:13:22

Q. And that's why you recommended Alternative 4, including the sublethal test methods; right? 14:13:22 14:13:25

A. Uh-huh. 14:13:28

Q. Yes? 14:13:29

A. Yes. 14:13:30

Q. And rejected Alternative 2, which would have been acute only? 14:13:30 14:13:33

A. Yes. 14:13:35

Q. Let's turn to page 5-22, chemistry line. 14:13:35

Now, this first paragraph refers to these complicating factors. 14:13:53 14:14:02

Do you see that? 14:14:04

A little bit more than midway down through this first paragraph in Section 5.4.4.1. 14:14:08 14:14:10

A. Oh. Right. Right. 14:14:14

Q. What is that referring to? 14:14:15

A. That's probably referring to -- 14:14:17
I'm sorry. 14:14:19
Q. Please proceed. 14:14:20
A. Okay. 14:14:23
Confounding factors. 14:14:24
Q. Well, it looks a little broader than that, Mr. Beegan. 14:14:24 14:14:30
Let's just walk through them so we make sure we understand what the complicating factors are. 14:14:31 14:14:33
The first one is a lack of guidance. 14:14:36
Do you see that? 14:14:39
A. Oh, yes. Yes. 14:14:39
Q. What does that refer to? 14:14:41
A. Umm -- there's not a whole lot of material out there that describes how sediment quality guidelines are to be used. 14:14:43 14:15:06 14:15:14
Actually, probably in the past. 14:15:15
And -- umm -- or the limitations have been -- umm -- not well described by those documents. 14:15:19 14:15:27
I'm making an assumption here that -- I don't remember specifically -- umm -- this so I'm basically reading it and trying to remember -- umm -- 14:15:36 14:15:43 14:15:51
Q. Is it fair to ask you to help us given that you were a principal author of this Staff Report? 14:15:59 14:16:03
A. No. Yeah. 14:16:06

Q. Thank you. 14:16:06

A. Again, this is a long time ago. 14:16:07

Q. I understand. 14:16:10

A. That's -- umm -- I haven't moved -- I haven't -- I haven't put much effort in to direct effects over the last year or so. Everything is focused on bioaccumulation. 14:16:11 14:16:16 14:16:23 14:16:29

But I acknowledge or at least I've read the fact that these sediment quality guidelines have been misused for purposes other than those intended by the developing author. 14:16:30 14:16:35 14:16:38 14:16:44

Q. And so these complicating factors are with reference to these sediment quality guidelines; right? 14:16:49 14:16:52

A. Yeah. 14:16:56

I just -- "Sediment quality guidelines, tools that, SQGs, tools that relate contaminant concentrations to the potential for adverse effects on sediment-dwelling organisms, are often used to help interpret sediment chemistry data. SQGs have been used for over 30 years to assess sediment contamination, yet there are many factors that make their use a complex and challenging task. These complicating factors include a lack of guidance on how to evaluate the many types of SQGs in order to select the approach best suited for a particular application." 14:16:57 14:17:00

Okay.

So -- "Uncertainty regarding how to asses complex mixtures of contaminants, and uncertainty in how to establish thresholds for SQG interpretation that define acceptable and unacceptable sediment quality." 14:17:20 14:17:22 14:17:36

Q. And so the complicating factors with regard to sediment quality guidelines include the lack of guidance; right? 14:17:36 14:17:43 14:17:47

A. Yep. 14:17:47

Q. The uncertainty regarding how to assess the complex mixtures; right? 14:17:48 14:17:51

A. Correct. 14:17:53

Q. The inability to reliably predict the contaminant bioavailability; right? 14:17:53 14:18:00

A. Yes. 14:18:01

Q. And also the uncertainty how to establish thresholds for SQG interpretation; right? 14:18:01 14:18:05

A. Correct. 14:18:07

Q. And with respect to those thresholds the uncertainty is with respect to distinguishing between acceptable and unacceptable sediment quality; right? 14:18:08 14:18:10 14:18:16

A. Yes. 14:18:18

Q. And those are all complications; right? 14:18:19

A. Correct. 14:18:25

Q. And challenges; right? 14:18:27

A. Correct. 14:18:28

Q. With regard to sediment quality guidelines; right? 14:18:29 14:18:33

A. Correct. 14:18:34

Q. Is this with regard to empirical SQGs? 14:18:34

A. Umm -- yes. 14:18:45

This is specific to -- 14:19:04

Umm -- empirical. 14:19:06

It doesn't look that way. 14:19:10

Q. It's -- could be broader than that? 14:19:11

A. It looks like. 14:19:13

Q. It's a broad critique of SQGs; correct? 14:19:14

A. Yes. 14:19:19

Q. And then the second paragraph refers to "particular challenges when there are intermediate levels of contamination." 14:19:19 14:19:22 14:19:27

Do you see that? 14:19:28

A. Umm -- yes. 14:19:29

Q. That's where SQGs can produce the highest error rates; correct? 14:19:33 14:19:38

A. Yes. 14:19:39

Q. In other words, where there are intermediate levels of contamination reliance on an SQG can produce an error; right? 14:19:40 14:19:44 14:19:50

A. Correct. 14:19:50

Q. And in that circumstance the rate of error is at its highest? 14:19:50 14:19:54

A. Correct. 14:19:55

Q. So those types of predictions might be erroneous; right? 14:19:56 14:20:02

A. Correct. 14:20:03

Q. And then in the next paragraph there's a reference to the "misuse of SQGs." 14:20:04 14:20:08

Do you see that? 14:20:12

A. Uh-huh. Yes. 14:20:13

Q. And you understand that there's considerable concern over that misuse; correct? 14:20:15 14:20:18

A. Uh-huh. Yes. 14:20:20

Q. And that's with regard to the implementation of narrative water quality objectives in Basin Plans; correct? 14:20:21 14:20:31 14:20:37

A. Yes. 14:20:38

Q. And then the next sentence of that paragraph refers to the uncertainty of using SQGs; right? 14:20:39 14:20:49

A. Yes. 14:20:55

Q. And it indicates that that uncertainty is substantial; right? 14:21:00 14:21:04

A. Yes. 14:21:05

Q. And it also refers to the controversy over the use of chemical SQGs. 14:21:13 14:21:17

Do you see that? 14:21:20

A. Yes. 14:21:21

Q. And is that controversy in the scientific community? 14:21:21 14:21:24

A. Yes. 14:21:24

Q. And that controversy is substantial as well; correct? 14:21:25 14:21:29

A. Yes. 14:21:29

Q. And this all runs from the fact that no single SQG is able to account for all of the factors; right? 14:21:29 14:21:33

A. Correct. 14:21:37

Q. All of the factors that influence contaminant effects; correct? 14:21:37 14:21:41

A. Correct. 14:21:42

Q. Let's turn to the next page, please. 14:21:42

Now, is sediment chemistry another surrogated measure of exposure? 14:21:53 14:22:10

A. Yes. 14:22:14

Well, yeah, it -- yes. 14:22:16

It's an estimate -- I guess. 14:22:19

It's a surrogate. 14:22:24

Yeah, sure. 14:22:26

Q. Now, in the carryover paragraph, first full sentence there refers to "the lack of plans or policies in California." 14:22:27 14:22:35 14:22:39

Do you see that? 14:22:40

A. Umm -- where are we? 14:22:41

I'm sorry. 14:22:49

Q. Oh. The carryover paragraph, Mr. Beegan? 14:22:49

A. Oh. "In California there are no current --" 14:22:53

okay "-- plans or policies that define what guidelines 14:22:55

shall be used, how the guidelines should be applied --" 14:22:59

Q. Do you see that? 14:23:05

A. Yeah. 14:23:10

Q. Was the Part 1 an effort to fill this void? 14:23:10

A. Umm -- I never thought of it like that. 14:23:21

Umm -- 14:23:39

Q. Why was it relevant to indicate that there are 14:23:42

no current plan or policies in California that define 14:23:44

what guidelines shall be used? 14:23:48

A. Umm -- I do not know. 14:23:51

Umm -- I would probably have to read this 14:24:02

paragraph a little more closely to understand where it 14:24:08

was getting at, because -- umm -- it doesn't make sense. 14:24:14

Q. Why doesn't it make sense? 14:24:23

A. Because there are guidelines used in -- umm -- 14:24:24

the 303(d) listing policy, and -- umm -- 14:24:32

Yeah, so I -- 14:24:44

Like I said, I know that the 303(d) listing 14:24:45

policy was adopted while we were in development, and 14:24:52

maybe that was something that's left over from a very early document, but, again, without reading this paragraph, it -- I may be taking something out of context. 14:25:01 14:25:05 14:25:08 14:25:12

Q. Don't want you to do that, so we'll move on. 14:25:13

A. Okay. 14:25:16

Q. Turning to page 5-27, this refers to the alternatives considered for sediment chemistry; right? 14:25:16 14:25:36

A. Umm -- top of the page? 14:25:42

Q. Yeah. 14:25:44

A. Yes. 14:25:45

Q. And Alternative 2 refers to the use of existing national empirical SQGs; right? 14:25:45 14:25:50

A. Yes. 14:25:54

Q. Without consideration of actual predictive ability when applied to California; right? 14:25:57 14:26:00

A. Correct. 14:26:02

Q. And that was rejected; right? 14:26:03

A. Yes. Yes. 14:26:05

Q. In favor of new empirical SQGs? 14:26:05

A. Yes. 14:26:09

Q. And as applicable to Part 1 those would be the CSI and the CALRM; right? 14:26:09 14:26:14

A. Yes. Yes. 14:26:17

Q. And a number of SQGs were -- were considered 14:26:18

in this Staff Report; right? 14:26:27

A. Yes. 14:26:29

Q. Including those by McDonald, et al, 1996; 14:26:29

right, going back to 5-23? 14:26:37

A. Umm -- correct. 14:26:40

Q. And McDonald et al, 2000; right? 14:26:41

A. Uh-huh. 14:26:47

Q. Yes? 14:26:48

A. Yes. 14:26:48

Q. And those were rejected in favor of 14:26:48

Alternative 3; correct? 14:26:52

A. Umm -- yes. 14:26:53

MR. SINGARELLA: Mr. Beegan, would you mind if 14:27:04

we took a quick break? 14:27:06

THE WITNESS: No. 14:27:08

MR. SINGARELLA: Okay. Thank you. 14:27:08

VIDEOGRAPHER: We are going off the record at 14:27:09

2:26 p.m. 14:27:11

This is the end of disk three. 14:27:12

(Thereupon a recess was taken at 2:27 p.m.

and the deposition resumed at 2:35 p.m.) 14:35:31

VIDEOGRAPHER: We are back on the record at 14:35:31

2:35 p.m. 14:35:35

This is the beginning of disk number four. 14:35:36

EXAMINATION 14:35:38

Q. Hi, Mr. Beegan. 14:35:39

My name is Bill Brown. 14:35:41

I represent the Port of San Diego. 14:35:42

I'm going to ask you a few questions. I think it's going to be relatively short. 14:35:44 14:35:47

My guess is you're going to tell me you don't know the answers to a lot of these specifics, and I'm going to be asking you if you can direct me to the person who would know. 14:35:48 14:35:51 14:35:53 14:35:54

The reason I'm going to ask you these questions is because I noticed that you're listed as a point of contact information on a website for the Bay Protection and Toxic Cleanup Program. 14:35:56 14:35:57 14:36:01 14:36:04

Are you aware of that? 14:36:06

A. Uh-huh. 14:36:07

Q. One of the areas that it lists is an economic analysis, and that's one of the documents that's listed. 14:36:08 14:36:14

Are you aware of that? 14:36:18

A. Umm -- there are a couple Bay Protection-related websites. 14:36:19 14:36:24

If you give me a time frame -- 14:36:26

Q. Are you aware of a report that was done by SAIC in 19 -- I mean in 2008? 14:36:29 14:36:32

A. Oh, yeah. Yes. 14:36:35

Umm -- as -- the -- economic analysis for -- 14:36:37

umm -- support of SQG development? 14:36:43

Is that the same one? 14:36:47

Q. Yes.

And it's also referred to in the Staff Report. 14:36:49

A. Yeah, yeah, yeah. 14:36:51

Q. And it's named as 704? 14:36:52

A. Right. 14:36:54

Q. The document doesn't list an author, but just 14:36:55
that rather large corporation. 14:36:58

If I wanted to know some of the specifics 14:37:00
about the analysis that they did in that report, who 14:37:03
would I be talking to? 14:37:06

A. Umm -- 14:37:07

MR. FUCHS: If you know. 14:37:12

MR. BROWN: If you know. 14:37:14

THE WITNESS: There -- there are two people at 14:37:18
SAIC that were principal authors. 14:37:21

One of them I know is no longer there. She 14:37:26
works for another consulting company. 14:37:28

But -- umm -- I can't -- remember her name. 14:37:31

It's been a couple years. 14:37:38

Q. Okay. 14:37:41

A. But I can provide that to you. 14:37:41

Q. Right. 14:37:43

And that would be one of my follow-up 14:37:43

questions is asking you if I can. 14:37:46

A. Yeah.

Q. Because we're in some administrative 14:37:49
proceedings and everything, and I want to make sure we 14:37:51
do it in a way that doesn't disturb you or the counsel 14:37:54
that's representing you. 14:37:57

I'm asking questions that appear to be 14:37:57
available through a State website, but because of the 14:38:00
predicament we're in I'm just being cautious. 14:38:03

Do you remember who the other person is at 14:38:06
SAIC who may have contributed to that report? 14:38:08

A. I just remember the main point of contact. 14:38:10

Like I said, I don't know her name off the top 14:38:13
of my head, but she was the primary author, Project 14:38:15
Manager. 14:38:20

If it helps you, she was the same person who 14:38:20
performed the economic analysis for the State 14:38:26
Implementation Policy, the SIP, back in '98, '99. 14:38:30

Q. Okay. 14:38:37

A. Sorry. 14:38:39

Q. All right. 14:38:39

But, in any event, if I E-mail you and include 14:38:40
your counsel on the E-mail, you could get back to me 14:38:42
with the names of these people? 14:38:45

A. Oh, I'll do it. 14:38:46

I could potentially do it tonight if I get home at a decent hour. 14:38:47 14:38:51

Q. If that's okay with your counsel, that would be fine with me. 14:38:53 14:38:56

A. But I can dig it up. 14:38:56

It's -- 14:38:58

Q. Okay. And these are some general questions that you may or may not know the answer to. 14:38:59 14:39:02

Is it a correct statement that in preparing SQOs that the State does have to take in to account economic impacts? 14:39:04 14:39:09 14:39:13

A. Umm -- broadly, yes. 14:39:14

Yes. 14:39:19

Q. Are you involved in that in any way? 14:39:19

A. Umm -- yes, to a general extent. 14:39:22

Umm -- I -- it was -- umm -- it's myself, the former attorney at the State Water Board, Sheila Vassey, who had a lot of experience on that, and -- 14:39:33 14:39:46 14:39:50

Eloise Castille. That's the author. 14:39:57

Q. At the SAIC? 14:40:00

A. Yes. 14:40:03

But, again, she's not there anymore. 14:40:03

Q. Okay. What was your involvement -- and I'm not trying to get in to your thought process or anything. 14:40:05 14:40:09 14:40:11

I'm just trying to figure out where you come in to the picture. 14:40:11 14:40:14

What was your involvement with the economic analysis? 14:40:15 14:40:17

A. Umm -- putting together a -- a scope -- umm -- of work. 14:40:18 14:40:27

Again, as the Project Manager, Program Manager -- umm -- with the economic analysis I relied quite a bit on -- umm -- the -- the Water Board attorney, Sheila Vassey, to help me with that -- 14:40:28 14:40:35 14:40:41 14:40:53

Umm -- so that was -- that was -- I -- I kind of -- umm -- pushed it through, made sure -- made sure it made sense, sent the scope out, made sure that the -- umm -- econ analysis fulfilled the scope of work requirements. 14:40:56 14:41:05 14:41:09 14:41:16 14:41:24

That was about it. 14:41:24

Q. And was that scope of work ultimately performed by SAIC? 14:41:25 14:41:28

A. Umm -- yes. Yes. 14:41:31

Q. Okay. Did you use any of their materials in coming up with any SQOs? 14:41:37 14:41:45

A. Oh, no, absolutely not. 14:41:47

Q. Okay. 14:41:49

A. Their -- their -- their role only related to -- umm -- economic analysis. It was limited. This was 14:41:52 14:41:58

nothing else. 14:42:06

They -- they did not have -- they were not 14:42:08

part of the technical team, per se. 14:42:12

So -- umm -- if you read all those technical 14:42:14

reports -- umm -- SAIC or Eloise had no -- umm -- no -- 14:42:19

she had no participation in the development of any of 14:42:29

that stuff. 14:42:33

Q. And that's where I'm kind of confused, and I'm 14:42:33

not trying to suggest anything. 14:42:37

I'm just trying to get the general frame -- 14:42:39

framework. 14:42:41

If economic analysis is considered in 14:42:42

conforming -- in preparing SQOs -- I'm sorry I 14:42:46

misspoke -- where does it come in at? 14:42:51

MR. FUCHS: Hang on a second. 14:42:53

I'm going to object that calls for a legal 14:42:55

conclusion. 14:42:57

MR. BROWN: If you know. 14:42:58

MR. FUCHS: If you can answer it, go ahead. 14:42:59

THE WITNESS: The -- umm -- the economic 14:43:02

analysis is performed on whatever is being proposed in 14:43:24

relation to baseline. 14:43:32

BY MR. BROWN: 14:43:37

Q. Let me give you a few different examples, and 14:43:37

you can tell me when I'm getting warm, if this works for 14:43:40

you. 14:43:44

Does it come in -- I don't think it comes in, 14:43:44

but does it come in during the MLOE phase? 14:43:47

A. Umm -- I -- for me I have a hard time 14:43:52

understanding -- 14:44:01

Are you talking about in the development of 14:44:04

the MLOE? 14:44:06

Q. Yes. 14:44:07

A. Okay. Umm -- yes. 14:44:08

Q. At what juncture? 14:44:17

A. Umm -- I don't recall specifically. 14:44:18

Q. Who would be the person who would know the 14:44:22

most about this subject at the Water Board? 14:44:25

A. Umm -- it probably should be me -- umm -- in 14:44:29

terms of when particular things happened, and -- umm -- 14:44:37

I just don't -- umm -- I -- 14:44:43

Like I had indicated to Mr. Singarella, there 14:44:52

are a lot of hoops that I jumped through or had to jump 14:45:02

through, or a lot of information that I had to put 14:45:06

together, assess, evaluate, five, six years ago. 14:45:10

Since that time there's a lot of new 14:45:19

information that I'm trying to deal with. 14:45:22

So asking me something like that -- umm -- you 14:45:26

know, at one time I probably had a good understanding of 14:45:32

the econ or better understanding, had a better 14:45:36

understanding of the timeline, what took place when. 14:45:39

Right now I -- umm -- I can't remember that 14:45:44

type of detail. 14:45:52

It would be guesswork or -- it would be 14:45:53

guessing. 14:45:55

Q. Let me ask it another way. 14:45:56

Do you come up with a scientific basis for a 14:45:58

SQO and then adjust it for economic considerations? 14:46:03

MR. FUCHS: Hang on. 14:46:08

Let me -- let me just object that this is 14:46:08

getting pretty far afield from the scope of this 14:46:11

deposition, which is how in the abstract in general the 14:46:15

SQOs get applied. 14:46:18

This sounds an awful lot like how they were 14:46:20

formed, how they were developed, at what point the 14:46:24

economic analysis came in. 14:46:27

So I guess I would suggest that, if you really 14:46:29

want to pursue this line of questioning, you send your 14:46:31

own deposition notice, and we can talk to Mr. Gallagher 14:46:34

about whether that gets quashed. 14:46:39

MR. BROWN: Okay. 14:46:42

BY MR. BROWN:

Q. Okay. Let's put it another way. 14:46:43

How does the economic analysis get applied? 14:46:45

And, more importantly, when? 14:46:49

I'm not so -- 14:46:51

I don't want to get in to the thought patterns 14:46:53
of how you do it so much as -- I'm trying to figure out 14:46:55
do you come up with an analysis first based on science 14:46:59
and then factor in economics, or is it all done at the 14:47:02
same time? 14:47:06

A. Umm -- I think -- 14:47:06

I believe it was developed by framework, 14:47:12
scientific-based framework, and then considered the -- 14:47:18
or performed the economic analysis. 14:47:27

Q. Okay. And who performed -- is that done by 14:47:30
you or is it done by somebody else? 14:47:35

A. The -- the economic analysis? 14:47:39

That was performed by -- this -- umm -- I just 14:47:44
mentioned her name -- Eloise at SAIC. 14:47:51

Q. And after the economic analysis is done how is 14:47:54
it applied to come up with the end product of SQOs? 14:47:57

MR. FUCHS: I'm sorry. 14:48:02

We're still not talking about applying the 14:48:03
SQOs, so -- I'm going to have to instruct Mr. Beegan not 14:48:04
to respond to any more questions regarding how the 14:48:12
economic analysis went in to developing the SQOs at what 14:48:16
point chronologically or logically. 14:48:23

MR. BROWN: Okay. Let me jump to another 14:48:26
subject, although they're interrelated, and then I 14:48:28

promise I will be done relatively shortly. 14:48:31

BY MR. BROWN:

Q. I'm curious about the interplay of TMDLs and SQOs and how they are applied on a statewide basis. 14:48:33 14:48:37

Are they -- 14:48:42

Are they developed jointly or does one inform the other? 14:48:43 14:48:47

A. The SQOs are applied -- as would any water quality objective to a water body or watershed situation. 14:48:49 14:49:06 14:49:12

And I'll attempt to explain. 14:49:13

Q. Okay. If you could explain, that would be great. 14:49:15 14:49:17

A. The -- the SQOs establish this means to differentiate good sediment from bad sediment. That's all they do. 14:49:18 14:49:26 14:49:30

So you're -- by assessing sediment quality in a segment, if you have enough impacted stations, you could list that segment for impairment of the direct effects, quote, "Sediment Quality Objective." 14:49:31 14:49:41 14:49:49 14:49:59

Once that listing got approved and went through -- jumped through the hoops, the State Water Board, it would be put on a list as what you need to do. 14:50:02 14:50:07 14:50:11

If it's on the TMDL list, the next step would be essentially going through the stressor identification 14:50:15 14:50:21

process and the target cleanup process. 14:50:26

That's how Part 1 is intended in the big 14:50:34

picture. 14:50:40

But -- umm -- there is, as I mentioned 14:50:41

earlier, when you -- I don't know if you were here, 14:50:45

there's a section in there that says that there's 14:50:48

nothing in that plan or in the specific section that 14:50:52

shall impinge upon the authority of the Regional Board 14:50:58

to set TMDL targets. 14:51:01

Something -- the language is something like 14:51:06

that. I don't remember it specifically, but I do know 14:51:08

that it's there. 14:51:11

So -- how did they relate? 14:51:12

It's a stepwise process. Use the SQOs to 14:51:17

assess and use the TMDLs in a kind of watershed water 14:51:22

body wide approach to restore sediment quality. 14:51:30

Q. In applying the TMDLs and the SQOs it appeared 14:51:36

in the Staff Report that they compared them for economic 14:51:45

analysis. 14:51:48

Are you aware of that? 14:51:49

A. Umm -- they -- umm -- they used -- umm -- 14:51:51

I was under the impression we used 303(d) 14:52:02

listings to make some comparisons and the TMDLs to -- 14:52:07

umm -- put estimates together as far as what restoration 14:52:18

would be required. 14:52:25

Q. Did you try to determine for economic analysis purposes whether SQOs would be more or less expensive than the SQO process? 14:52:25 14:52:30 14:52:34

A. Could you rephrase that? 14:52:36

Q. Did you compare the costs of TMDM -- TMDL implementation to the cost of SQO implementation for the economic analysis? 14:52:41 14:52:46 14:52:51

A. Umm -- I would have to reread the Staff Report. 14:52:53 14:53:00

Q. And I read the Staff Report and I saw the underlying document earlier in the day. 14:53:00 14:53:04

If I wanted to talk to the person who may know this subject most in depth, would it be the people you hired from SAIC or would it be you after you reviewed those documents? 14:53:06 14:53:09 14:53:14 14:53:18

A. It would probably be a combination of myself, Steve Bay at SCCWRP, Eloise Castille at SAIC. 14:53:19 14:53:29

Yeah. I mean, that's -- it's -- one of those three, but -- we considered a lot of things -- 14:53:32 14:53:36

Umm -- basically we looked at baseline conditions and moved forward from there. 14:53:45 14:53:48

Q. Okay. I promised everybody I wouldn't take long on this, so this is going to be very short, but I just am going to have you look at this because I think it's in here in a very summary fashion. 14:53:50 14:53:55 14:53:58 14:54:01

Could you look at Exhibit 704. 14:54:04

A. I think that's what I have. 14:54:12

MR. FUCHS: Yes, that's the Staff Report. 14:54:14

BY MR. BROWN:

Q. Or the one that's called "Staff Report," and then section is called Section 7.4. 14:54:16 14:54:18

MR. FUCHS: It's on page 7-2. 14:54:33

MR. BROWN: Okay. 14:54:37

BY MR. BROWN: 14:54:43

Q. I'm looking at the last -- second to the last paragraph on page 7-3. 14:54:43 14:55:16

And on the second to last sentence in that paragraph it mentions the 303(d) listings. 14:55:23 14:55:30

Do you see where I'm referring to? 14:55:34

A. Uh-huh. 14:55:35

Q. Does that refresh your recollection that in looking at the economic impact of SQOs that you compared it to listings with 303(d)? 14:55:35 14:55:39 14:55:45

A. Umm -- yes. 14:55:48

Q. And who did that work? 14:55:51

A. Umm -- well -- I developed a list -- listings of 303(d) as to water bodies, and then I believe -- umm -- the -- Eloise looked at -- 14:55:58 14:56:18 14:56:29

Actually, well -- it was probably -- it was probably a combination of people, I think. 14:56:46 14:56:52

I put together an initial list of what was -- 14:56:55
umm -- what was -- what the -- where -- where the 14:56:59
existing listings were. 14:57:06
SCCWRP put together a list or a document 14:57:11
describing sediment quality in -- in bays of California 14:57:17
and then -- umm -- Eloise probably looked at the 14:57:30
differences or the changes. 14:57:37
Q. Okay. And then just one last generalization 14:57:39
question. I'm going to be off very quickly. 14:57:45
I'm still trying to separate in my mind, 14:57:47
Mr. Beegan, the difference between the TMDL track and 14:57:50
the SQO track. 14:57:53
Did they run parallel or do they intersect at 14:57:56
some point? 14:57:59
MR. FUCHS: Objection. Vague and ambiguous. 14:58:00
Also calls for a legal conclusion. 14:58:04
If you understand the question, you can take a 14:58:07
stab at answering it. 14:58:10
THE WITNESS: Well -- I'm sorry. 14:58:12
I must not have -- umm -- answered the 14:58:15
question -- umm -- clearly before, because -- 14:58:18
MR. BROWN: I wouldn't presume that, but I'm 14:58:27
trying to take in a lot of information all at once. 14:58:29
You may have answered it very clearly. 14:58:32
THE WITNESS: No. No. 14:58:34

So -- umm -- the -- Sediment Quality Objectives are used to assess sediment. 14:58:35 14:58:45

Once you've assessed sediment -- umm -- and you have a sediment quality related listing -- umm -- your -- 14:58:51 14:58:59 14:59:09

In order to restore it you would go down the TMDL path. 14:59:15 14:59:19

You've demonstrated that your sediment quality isn't meeting the narrative. 14:59:25 14:59:28

From there stressor identification, target -- umm -- some sort of sediment target or load, waste load allocation would be -- umm -- appropriate. 14:59:30 14:59:36 14:59:42

I -- 14:59:48

A TMDL is developed based on a degradation or an impairment of a water body. 14:59:51 15:00:00

A TMDL usually must include and identify -- must identify what beneficial use or what criteria you're basing this all on. 15:00:02 15:00:07 15:00:12

So there's a relationship there. 15:00:14

If your sediment quality is impaired, or you've demonstrated that it's impaired due to a specific narrative objective, that becomes part of the TMDL, and it kind of forms the basis of where you go. 15:00:17 15:00:20 15:00:24 15:00:31

I -- I -- I'm trying to think of what -- what relationship beyond that exists, and just off the top of 15:00:36 15:00:44

my head I can't. 15:00:48

I wish I could help you more. I -- 15:00:50

Q. Oh, no. 15:00:53

I think you're probably being very helpful. 15:00:54

Let me put it to you another way. 15:00:57

To some extent in the economic analysis we just read it does appear that they're contrasting the 303(d) listing and the SQO process for purposes of economic analysis, and to some extent that implies that they're two different processes, but to -- based on what you just said it appears that one feeds in to the other. 15:01:00 15:01:04 15:01:08 15:01:13 15:01:17 15:01:20

Is that correct? 15:01:23

A. Umm -- well, I think your -- you're comparing apples and oranges. 15:01:24 15:01:28

So the TMDL process is not the same as a 303(d) listing process. 15:01:30 15:01:34

Q. Okay. 15:01:38

A. We were comparing -- the -- existing 303(d) list, or the existing 303(d) policy, or whatever baseline policies were out there, and listings with what could potentially occur through the adoption of SQOs. 15:01:38 15:01:49 15:02:01 15:02:07

Q. Do you know if they did a similar process for comparing TMDLs to SQOs, or was it only 303(d)s to SQOs? 15:02:18 15:02:20

A. Umm -- I think -- I -- umm -- 15:02:27

They may have been projections on forthcoming 15:02:42

TMDLs, but, again, the SQO is not a TMDL. 15:02:46

So -- 15:02:53

I -- I'm having a hard time answering that 15:03:05

question. 15:03:09

Umm -- I know that listings were -- form the 15:03:09

basis. 15:03:14

I know that costs for TMDLs were -- umm -- 15:03:15

were used in some cases. 15:03:21

I don't recall the specifics, but I think 15:03:24

there were some in there. 15:03:27

Umm -- how those were used, they would have to 15:03:30

be used in some sort of -- umm -- comparison of, you 15:03:33

know, cost to restore a water body. 15:03:38

Q. And in preparing the Staff Report did they try 15:03:42

to -- compare the incremental costs of SQOs above and 15:03:45

beyond what would be required by 303(d) or TMDL? 15:03:54

A. What do you mean by "incremental costs"? 15:04:00

Q. I'll try to define it for you. 15:04:02

If to implement a TMDL for a particular site 15:04:04

would cost X, and to implement the SQO process would 15:04:08

cost Y, did they try to determine what the difference 15:04:13

was between X and Y? 15:04:15

A. Umm -- off the top of my head I would say no. 15:04:18

MR. BROWN: Okay. Well, without making it any 15:04:27

further murkier I think they may have done that, -- 15:04:29

THE WITNESS: Okay. 15:04:33

MR. BROWN: -- but I don't think you're the 15:04:33
person that I should bugging about this, so I'm going to 15:04:34
turn the questioning back to Mr. Singarella. 15:04:37

THE WITNESS: Okay. 15:04:40

MR. BROWN: All right. Thank you. 15:04:41

FURTHER EXAMINATION 15:04:43

BY MR. SINGARELLA: 15:04:44

Q. Okay. Mr. Beegan, back to the Staff Report. 15:05:12

Can you turn to page 5-31, please. 15:05:16

And let's talk a little bit about best 15:05:25
professional judgments. 15:05:29

And you see the bullets on page 5-31? 15:05:31

A. Yes. 15:05:37

Q. And the lead in to the bullets indicates that 15:05:37
there are many reasons why the utility of BPJ is 15:05:43
limited. 15:05:49

Do you see that? 15:05:49

A. Yes. 15:05:50

Q. And those four bullets list some of those many 15:05:50
reasons; right? 15:05:54

A. Umm -- yes. 15:05:55

Q. And the first reason is that BPJ may result in 15:05:57
inconsistent decisions; right? 15:06:05

A. Yes. 15:06:08

Q. Within a single region itself; right? 15:06:09
A. Yes. 15:06:13
Q. Or in between different regions; right? 15:06:13
A. Correct. 15:06:16
Q. And inconsistent decisions with regard to what, Mr. Beegan? 15:06:17 15:06:22
A. Umm -- whether -- sediments are impacted or not impacted, I would assume without reading the rest of the section. 15:06:23 15:06:35 15:06:38
Q. You think that's a safe assumption? 15:06:39
A. Yeah. Yeah. Yeah. Yeah. 15:06:43
Q. And BPJ's also limited because it can be time consuming and resource intensive; right? 15:06:44 15:06:49
A. Absolutely. 15:06:51
Q. And BPJ is limited because it may not always lead to transparent results; right? 15:06:53 15:06:57
A. Right. 15:06:59
Q. Or unbiassed decisions; right? 15:06:59
A. Right. 15:07:02
Q. What does that mean, "transparent and unbiased decisions"? 15:07:02 15:07:05
MR. FUCHS: I'm going to start objecting here because we've been going through this Staff Report for several hours, and I've let it go on because I think to a certain extent the Staff Report can be used to aid 15:07:06 15:07:09 15:07:11 15:07:17

folks who may want to apply the SQOs, but we're really delving in to how the SQOs were developed here, which is clearly outside the scope of what has been delineated for this deposition. 15:07:22 15:07:27 15:07:33 15:07:38

Whether BPJs are appropriate or not has little to do with what one does to apply the SQOs. 15:07:40 15:07:48

So, you know, as I said, I've let this go on for quite a while, but I think it's time to bring it to an end. 15:07:52 15:07:55 15:07:59

Q. Is BPJ relevant to Part 1? 15:07:59

MR. FUCHS: That's vague and ambiguous. 15:08:05

You can answer that question -- if you can answer it 15:08:09 15:08:13

THE WITNESS: It's relevant to certain portions of Part 1. Other portions it's almost irrelevant. 15:08:14 15:08:24 15:08:29

BY MR. SINGARELLA: 15:08:30

Q. Is Part 1 an attempt to limit the problems with regard to BPJ? 15:08:32 15:08:38

A. Yes. 15:08:40

Q. And is it important to understand those problems in order to appreciate how to avoid them -- within the context of applying the SQOs? 15:08:41 15:08:45 15:09:01

A. Not -- necessarily. 15:09:08

Q. Is BPJ a methodology itself? 15:09:14

A. Umm -- yeah, I would say yes. 15:09:18

Q. And is there any BPJ whatsoever in Part 1? 15:09:33

A. Umm -- like I said, there are specific 15:09:36

portions of Part 1 that rely on -- umm -- BPJ -- umm -- 15:09:41

there's stressor identification requires -- umm -- 15:09:53

significant amount of best professional judgment in 15:09:59

terms of how to -- umm -- how to most effectively 15:10:05

determine which -- what stressor is causing the problem. 15:10:11

Study design would require best professional 15:10:18

judgment. Part 1 is other than it says to develop the 15:10:22

work plan, it's silent on those types of issues. 15:10:29

So -- there -- there's significant room in 15:10:32

Part 1 for BPJ, but within the application of the Lines 15:10:38

of Evidence for bays, marine bays, it's very limited, 15:10:46

but in the delta application, estuary applications, 15:10:56

there's a little more. 15:11:02

So, I mean, -- you could -- umm -- there's 15:11:03

portions, sections, where it's significant and sections 15:11:08

where it's less so, a lot less so. 15:11:14

Q. Why did the Staff Report spend so much time 15:11:18

discussing BPJ? 15:11:23

A. Umm -- because sediment quality assessments 15:11:25

are typically based on best professional judgment, and 15:11:34

in a state where there's multiple regions that act -- 15:11:42

can act somewhat independently, you could have very 15:11:51

different approaches. 15:11:59

None of those approaches are -- I think are 15:12:03
described in the basin plans or any other policies. 15:12:07

Q. Does the Staff Report help inform the use of 15:12:11
BPJ in the Part 1 SQO? 15:12:19

A. Does it help inform the use of BPJ -- in Part 15:12:23
1. 15:12:33

I guess it could. 15:12:33

That wasn't the intent. 15:12:44

Q. And what was the intent? 15:12:46

A. The intent is to describe -- umm -- the basis 15:12:47
for Part 1, not necessarily provide a -- an enduser 15:12:57
reference. 15:13:07

Q. And so one purpose of Part 1 is to prevent the 15:13:17
misuse of BPJ; right? 15:13:21

A. It's to -- 15:13:24

Well, yes. 15:13:30

Q. So to prevent the misuse of BPJ one needs to 15:13:30
understand how it can be misused; correct? 15:13:36

A. Umm -- the way I think of BPJ is -- umm -- 15:13:39
someone coming up with a method to assess sediment 15:13:58
quality, and Part 1 has a method to assess sediment 15:14:08
quality. 15:14:15

You don't necessarily need to know the 15:14:17
rationale for other approaches to follow Part 1. 15:14:21

Q. Well, for example, in Table 5 of Part 1, the benthic index categorization values -- is there a judgment that goes in to these different categories in the numeric ranges specified for each? 15:14:27 15:14:47 15:14:57 15:15:02

A. Umm -- 15:15:04

MR. FUCHS: Objection. Vague and ambiguous. 15:15:09

You mean in coming up with these numbers or in applying them? 15:15:14 15:15:16

MR. SINGARELLA: In coming up with them. 15:15:17

MR. FUCHS: Well, then that is outside the scope and invades the mental processes. 15:15:21 15:15:24

MR. SINGARELLA: I'm not asking him about the judgment itself. 15:15:29

I'm wondering if there is any judgment that goes in to this. 15:15:32 15:15:33

BY MR. SINGARELLA: 15:15:34

Q. Let me ask you a different way. 15:15:34

Do these ranges fall out of some mathematical equation that is known to be firm and precise in nature, for example, force equals mass times acceleration? 15:15:36 15:15:40 15:15:45

A. No. 15:15:49

Q. You're not just applying some basic principle of -- of physics or science to come up with these ranges; right? 15:15:50 15:15:52 15:15:57

A. Correct. 15:15:57

Q. You're not using a clean dose response relationship to come up with these ranges; correct? 15:15:58 15:16:00

MR. FUCHS: Once again, we're getting in to how things were developed, not how they're applied. 15:16:07 15:16:10

MR. SINGARELLA: We're asking what they are, actually. 15:16:12 15:16:14

MR. FUCHS: I mean, literally how they came up -- how he came up with them. 15:16:14 15:16:17

MR. SINGARELLA: No, what they are. 15:16:20

MR. FUCHS: Mr. Beegan, don't answer that question. 15:16:20 15:16:22

MR. SINGARELLA: So you're not going to let him tell us what's behind these numbers, what these numbers represent, are they -- are they based on physical principles or judgment? 15:16:23 15:16:24 15:16:27 15:16:30

MR. FUCHS: He's already answered that. 15:16:31

MR. SINGARELLA: What did he say? 15:16:33

MR. FUCHS: He said they're -- 15:16:34

You can read the record yourself. I'm not going to answer for him. 15:16:36 15:16:39

BY MR. SINGARELLA: 15:16:40

Q. So is there -- is there a judgment that went in to specifying the ranges? 15:16:40 15:16:44

MR. FUCHS: Asked and answered. 15:16:46

BY MR. SINGARELLA: 15:16:47

Q. Did you say that they weren't a product of a clean dose response relationship? 15:16:48 15:16:51

MR. FUCHS: He didn't answer that question because I told him not to. 15:16:54 15:16:56

We're done with figuring out how these numbers were derived. 15:16:57 15:17:02

MR. SINGARELLA: Okay. Let's call Gallagher. 15:17:02

This is silly. We'll be here until tomorrow the way this is going. 15:17:05 15:17:07

You've been interrupting me all day, and you're half the record, so let's call Gallagher -- if we can get him. 15:17:08 15:17:10 15:17:16

MR. RICHARDSON: Yeah. I'll probably call him and ask him to dial in to the main number here. 15:17:20 15:17:22

MR. SINGARELLA: Yeah.

MR. FUCHS: Go for it.

MR. RICHARDSON: Do you want to go off the record for just a moment? 15:17:25 15:17:26

MR. FUCHS: Yes. 15:17:27

VIDEOGRAPHER: We are going off the record at 3:17 p.m. 15:17:29 15:17:30

(Discussion off the record) 15:17:34

MR. SINGARELLA: Mr. Gallagher? 15:21:37

Mr. Gallagher, it's Paul Singarella. 15:21:38

Thanks for calling in. 15:21:40

MR. GALLAGHER: Hi. 15:21:41

Who do we have here? 15:21:42

MR. SINGARELLA: Well, we should probably go around the table and make appearances. 15:21:43 15:21:45

I was asking Kelly to see if we could ring you up. 15:21:47 15:21:49

Kelly is to my right. 15:21:50

VIDEOGRAPHER: Excuse me, sir. 15:21:52

Did you want this on the video? 15:21:53

MR. SINGARELLA: No. 15:21:55

VIDEOGRAPHRE: Okay. 15:21:56

MR. GALLAGHER: If we did, I couldn't see your faces. 15:21:58

Again, it's Paul, Kelly. 15:22:00

Who else? 15:22:00

MR. TRACY: Mike Tracy is here. 15:22:03

MR. GALLAGHER: Okay. 15:22:05

MR. FUCHS: This is Dan Fuchs. 15:22:08

MR. GALLAGHER: Hi, Dan. 15:22:09

Okay. 15:22:10

THE WITNESS: Chris Beegan, State Water Board. 15:22:10

MR. SINGARELLA: And we've got a few people on the line phoning in as well, Mr. Gallagher. 15:22:13 15:22:15

MR. GALLAGHER: Who is that next to Dan? 15:22:18

THE WITNESS: Chris Beegan. 15:22:21

MR. GALLAGHER: Oh, Chris. Okay. 15:22:22

MR. FUCHS: Our deponent. 15:22:24

MR. SINGARELLA: Mr. Gallagher, we -- 15:22:25

MS. REYNA: The witness. 15:22:25

MR. GALLAHGER: Who is this, I should say?

MS. TRACY: This is Jill. I'm on the phone. 15:22:33

MS. REYNA: And Kristin. I'm on the phone, 15:22:36
too.

MR. CONDER: Hi. This is Jason Conder.

How are you? 15:22:41

MR. GALLAGHER: Hi, Jason. 15:22:42

MR. SINGAREALL: And Bill Brown may be joining 15:22:44
us momentarily. He stepped out for a moment. 15:22:46

MR. GALLAGHER: Okay.

MR. SINGARELLA: Mr. Gallagher, Mr. Fuchs and 15:22:49
I have -- have reached an impasse over a few lines of 15:22:50
questioning. 15:22:53

I'm trying to -- to move this along so that we 15:22:53
can hopefully wrap this up today. 15:22:57

I must admit that -- umm -- the level of 15:22:59
speaking objections and statements on the record is 15:23:03
troubling me and has been preventing me from getting 15:23:07
through in an order early fashion. 15:23:12

Let me just give you a sense of where we are, 15:23:14
and then Mr. Fuchs, I'm sure, will have a contrary view 15:23:16

of things, but -- 15:23:21

I -- I have spent the vast majority of the day 15:23:23
on two exhibits that -- each of which requires very 15:23:26
material unpackaging to understand this rule, the 15:23:33
Sediment Quality Objectives, and those two exhibits 15:23:37
include the Sediment Quality Objective itself and as 15:23:43
adopted by the State Board, and then the second exhibit 15:23:45
is a very, very detailed and long Staff Report that the 15:23:48
State Water Board staff presented to the State Water 15:23:54
Board pursuant to the adoption hearing. 15:23:59

I'm not getting in to, you know, E-mails, and 15:24:01
ancient history, and personalities. 15:24:04

I'm spending my entire day trying to get at 15:24:07
the SQOs as those are understood in the SQO document 15:24:11
itself and -- 15:24:19

MR. GALLAGHER: So, counsel, the document is
the SQO document itself and the other is a Staff Report 15:24:25
presented to -- and sorry to interrupt -- just trying 15:24:25
not to get the exhibits wrong. 15:24:29

The other is the Staff Report presented to the 15:24:30
State Water Board at the adoption hearing. 15:24:31

MR. SINGARELLA: Exactly. 15:24:35

And Mr. Fuchs has objected on several grounds. 15:24:36

The first ground relates to this all important 15:24:40
category called possibly impacted, and I was trying to 15:24:44

get at the issue of whether that should be in the protective condition or not, meaning -- meaning if you're in possibly impacted are you flunking the SQO or are you passing it. 15:24:50 15:24:57 15:25:00 15:25:06

That was a very, very close call at the State Water Board. I'm not speaking from the deposition today, but just from my knowledge of it. 15:25:08 15:25:10 15:25:12

And I thought it was very, very important to -- an understanding of the methodology and explanation. 15:25:14 15:25:19

MR. GALLAGHER: So the first line of questioning, and you both can interrupt me at any time. 15:25:23 15:25:24

Possibly impacted -- is that a protective condition or not, did it pass or notpass. 15:25:28 15:25:33

Go on. 15:25:38

MR. SINGARELLA: Exactly. Exactly. 15:25:39

And how was -- how was -- how was that decision made at the State Water Board, how did they decide to put it on one side of the line or another. 15:25:41 15:25:44 15:25:48

It's critical because we have some of these possibly impacted stations. 15:25:51 15:25:53

The second issue deals with all these numbers inside the SQO itself. 15:25:55 15:26:00

There are a tremendous number of numbers, ranges and the like, and it's my belief that those ranges do not result from, you know, known scientific 15:26:03 15:26:06 15:26:10

formula, or principles, or equations, you know, like E=MC squared, but, rather, there is a good deal of judgment going in to these ranges, and I'm simply trying to ask questions to get an understanding as to where these numbers came from. 15:26:15 15:26:19 15:26:24 15:26:28 15:26:31

And to understand the SQO methodology we've got to know a little bit about these numbers. 15:26:32 15:26:36

It's not a rote application without any context, and I'm simply seeking that context, and I'm seeking a record that would clarify where these numbers came from. 15:26:39 15:26:42 15:26:46 15:26:49

And then we even had some trouble agreeing on whether I would be allowed to ask Mr. Beegan where certain data came from. 15:26:49 15:26:53 15:26:57

Here my belief is that the data that -- that Mr. Beegan has shown to the San Diego Regional Board on a couple of occasions is the data from the SQO, and it looks like a Rorschach test as opposed to something you draw a straight line through. 15:26:59 15:27:03 15:27:08 15:27:12 15:27:16

I'd like to know where those data come from and to what extent the SQO relies upon them. 15:27:19 15:27:21

So, you know, basic -- 15:27:25

MR. GALLAGHER: Let me -- the data that you're referring to is not data specific to the San Diego marine sediment. 15:27:26 15:27:31 15:27:35

It's other -- what -- what data are we talking about? 15:27:38 15:27:42

MR. SINGARELLA: It's statewide. 15:27:42

It appears to be everything that they looked at, for example copper. 15:27:44 15:27:46

There's -- there's a plot that -- that I believe is the data used to develop the -- the sediment chemistry prong of the SQO for copper, and I'm simply trying to link, you know, the data that I'm presenting to Mr. Beegan to SQO itself -- not for San Diego Bay. 15:27:49 15:27:52 15:27:58 15:28:03 15:28:08

It's not -- it probably has some San Diego Bay data in it. I have no idea. 15:28:11 15:28:14

This is some broad stroke stuff that we're trying to get our arms around here. 15:28:15 15:28:18

And so -- 15:28:20

MR. GALLAGHER: On the numbers inside the SQO not only are you, you know, trying -- 15:28:22 15:28:25

Just so I understand, not only are you trying to understand the judgment put in to the ranges, but when you -- but whether or not there's -- 15:28:29 15:28:32 15:28:36

In other words, A, you're trying to understand the numbers put in the -- with the judgment put in to the ranges, but then, B, you're trying to understand, you know, once you have some understanding of that judgment you're trying to understand where the data came 15:28:42 15:28:43 15:28:47 15:28:50 15:28:54

from for the judgment to put in the SQO numbers? 15:28:57

MR. SINGARELLA: Correct. 15:29:03

And I must be missing something, because 15:29:05
Mr. Fuchs has been repeatedly objecting and has issued a 15:29:07
series of limiting instructions limiting -- instructing 15:29:11
Mr. Beegan to not answer this or that. 15:29:15

MR. GALLAGHER: So those are two lines of 15:29:21
questioning. Those are the two exhibits. 15:29:23

Dan, do you want to clarify anything as far 15:29:27
as -- 15:29:34

MR. FUCHS: Oh, yeah, yeah, naturally. 15:29:34

I think it's very significant that Mr. 15:29:37
Singarella said that he wanted to, as he put it, get at 15:29:45
the SQOs. 15:29:48

My distinct recollection from our September 27 15:29:50
conference was that you had limited the questioning at 15:29:54
this deposition to how in the abstract the SQOs would be 15:29:59
or should be applied and not how they were arrived at, 15:30:06
because that -- would impermissibly invade the mental 15:30:14
processes of the decision-makers and would create an 15:30:23
opportunity for extra record evidence in the Cal Chamber 15:30:29
case which has been stayed and which is a record case 15:30:33
insofar as it is a -- a writ case. 15:30:37

MR. GALLAGHER: Right. 15:30:40

MR. FUCHS: So the -- Mr. Singarella is, of 15:30:41

course correct, that the majority of the questioning has been based on the SQO document itself and the Staff Report. There have been some others. 15:30:47 15:30:49 15:30:53

He didn't spend very much time on those, but those were certainly not the only two. 15:30:56 15:30:58

And -- and I've -- you know, it's obviously -- it's after 3:00 so -- we began at 8:00. 15:30:59 15:31:05

I've, obviously, let the questioning go on quite long today. 15:31:07 15:31:09

I've let Mr. Singarella ask a great many questions, many of which I think actually I probably should not have let him ask because they were outside the scope, as I've just described it. 15:31:13 15:31:17 15:31:21 15:31:24

So, for example, what he just -- the two examples he just gave, I think, are perfect examples of lines of questioning that should not be permitted, and I think that you did not permit, and those are, for example, how did the State Board decide how to put the possibly impacted category in to one side or the other? 15:31:27 15:31:30 15:31:35 15:31:39 15:31:42 15:31:46

That they did is what is relevant here, and one should do with a possibly -- 15:31:52 15:31:57

MR. GALLAGHER: Dan, say that last sentence again. 15:31:59 15:32:02

MR. FUCHS: Yeah. 15:32:02

What's relevant here is that they did put 15:32:03

possibly impacted in to one side or the other, and going on from there what one should do if one is applying the SQOs and one has a possibly impacted station. 15:32:06 15:32:10 15:32:15

How the State Board decided -- 15:32:21

MR. GALLAGHER: Say that second sentence, Dan. When you got you said they didn't, and the second sentence you said was? 15:32:24 15:32:26 15:32:30

MR. FUCHS: Right, what one should do when applying the SQOs -- 15:32:31 15:32:36

MR. GALLAGHER: Right. 15:32:41

MR. FUCHS: -- and -- and has a station that is possibly impacted. 15:32:41 15:32:45

That is a relevant line of questioning. 15:32:47

But how the State Board decided how to characterize or categorize, possibly impacted stations is not within the scope as you described it on the 27th and -- 15:32:50 15:32:56 15:33:03 15:33:10

MR. GALLAGHER: That's right. 15:33:11

MR. FUCHS: -- and is not a permissible avenue insofar as it invades the privileges and creates extra record evidence. 15:33:12 15:33:16 15:33:22

MR. GALLAGHER: Right. 15:33:25

MR. FUCHS: And the -- the second example that Mr. Singarella gave, I think, is also an example of an impermissible avenue. 15:33:25 15:33:29 15:33:36

For example, he said where the numbers came from, and the numbers here are the ranges of outcomes when various tests are performed. 15:33:39 15:33:41 15:33:48

So, for example, the health of the benthos, -- the benthic organisms, whether they're low disturbance or mid-disturbance, what I understood you to have said is that Mr. Singarella could ask how one who is applying the SQOs would decide whether something is low, or moderate, or heavily disturbed, but not how the State Board decided what range of values should qualify for each of those categories, because, again, to the extent that Mr. Singarella wants to explore the State Water Board's decision-making, that evidence is in the administrative record. 15:33:52 15:34:00 15:34:05 15:34:10 15:34:19 15:34:25 15:34:33 15:34:38 15:34:44 15:34:49 15:34:54

MR. SINGARELLA: Mr. Gallagher, I am not phrasing my questions based on, you know, deliberative process. 15:34:59 15:35:01 15:35:07

I'm not coming anywhere -- anywhere near deliberative process. 15:35:08 15:35:10

I am not asking Mr. Beegan, "How did the State Board decide this?" or "How did the State Board decide that?" 15:35:12 15:35:14 15:35:17

I'm simply looking at the words in these documents and asking questions about them. 15:35:17 15:35:20

For example, there are pages in the Staff 15:35:22

Report about best professional judgment and its limitations, and the idea, as I understand the SQO, is to overcome those limitations but, Mr. Beegan has testified there is some judgment that goes in to the SQOs, and I have been exploring that with him. 15:35:25 15:35:29 15:35:34 15:35:37 15:35:40

Where is it, show me where it is, not what did the State Board decide about best professional judgment. 15:35:43 15:35:46

And with respect to these -- this possible impacted category, you can't know how to use this tool correctly unless you understand how these categories were generated. 15:35:50 15:35:53 15:35:57 15:36:01

And whether something is on one side of the line or the other is the key issue here. 15:36:02 15:36:05

I'm simply trying to create a record of that so that no one relies on this thing as if it's handed down from Mount Sinai on tablets. 15:36:08 15:36:12 15:36:15

It's not Einstein's equation -- no -- no offense to the authors of the SQO here. 15:36:19 15:36:21

MR. FUCHS: Well, the -- 15:36:25

Again, I think it's -- what Mr. Singarella says is very significant. 15:36:27 15:36:30

He wants to -- reduce the credibility of the document. He just -- he just told you that. 15:36:33 15:36:40

And one of the ways he's doing that is by showing -- and this is the example we're using, because 15:36:43 15:36:46

it's the one that just -- we finished on, so it's the one freshest in our minds, I think. 15:36:50 15:36:54

The best professional judgment, there is some paragraphs in the Staff Report that suggest that best professional judgment may lead to inconsistent results, for example. 15:36:55 15:36:59 15:37:01 15:37:05

So then Mr. Singarella turns to the SQO document and says, "Well, essentially weren't these numbers derived using best professional judgment?" 15:37:06 15:37:09 15:37:13

That's not relevant to how one applies best professional judgment when using the SQOs. 15:37:17 15:37:23

He's exploring how best professional judgment was employed in deriving the SQOs. 15:37:28 15:37:32

It's very different. 15:37:35

MR. GALLAGHER: So -- so when you look at the Staff Report and you talk about best professional judgment, is it -- Mr. Beegan testified that it could lead to inconsistent results? 15:37:39 15:37:42 15:37:46 15:37:50

MR. SINGARELLA: Correct. 15:37:52

MR. FUCHS: Yes. 15:37:52

MR. SINGARELLA: The Staff Report says so, and I was -- 15:37:52 15:37:55

MR. GALLAGHER: And you -- and then now you, Paul, you want to understand that better? 15:37:56 15:37:59

MR. SINGARELLA: Absolutely. 15:38:01

I -- I spent the whole day basically trying to unpackage the sentences that are not self-explanatory and that led to a discussion of these concepts that are laced throughout these two exhibits. 15:38:02 15:38:06 15:38:10 15:38:17

MR. GALLAGHER: Then you may cross over to the deliberative process. 15:38:21 15:38:23

MR. SINGARELLA: Well, I think you cross over in to deliberative process if you say, "Mr. Beegan, you know, how did staff get together and decide, you know, how to develop these ranges?" 15:38:23 15:38:24 15:38:28 15:38:33

What I'm asking is are these ranges like E=MC squared or are they something else, the product of your judgment that you referred in to the Staff Report. 15:38:36 15:38:43 15:38:47

That's the nature of my question. 15:38:49

I'm not getting in to the -- the history of the proceedings. 15:38:51 15:38:53

There is a fine line here, Mr. Gallagher. I grant you that. 15:38:54 15:38:57

And I've been very solicitous of it, and I've been very mindful not to, you know, go too far. 15:38:57 15:39:01

There -- just like the SQOs, there is a bit of a line drawing going on here. 15:39:05 15:39:08

And -- and Mr. Fuchs characterized my questioning as "How did the State Board decide this? How did --" 15:39:09 15:39:13

That's not the nature of my questioning. 15:39:15

I am -- my questions are springing from these 15:39:17
materials in front of us here today very specifically, 15:39:20
and if you saw the transcript you'll see that I'm taking 15:39:24
specific sentences and asking four or five questions 15:39:27
about the sentences, because they don't explain 15:39:30
themselves. 15:39:32

There's -- there's a list of four reasons why 15:39:33
best professional judgment is dangerous and can lead to 15:39:36
spurious results. I think we're entitled to understand 15:39:40
that. 15:39:43

MR. GALLAGHER: In the -- in the Staff Report? 15:39:44

MR. SINGARELLA: In the Staff Report. 15:39:45

MR. GALLAGHER: And those four reasons are? 15:39:47

MR. SINGARELLA: Well, BPJ -- 15:39:50

Here we are on page 5-31 of the Staff Report. 15:39:53

BPJ, number one, may result in inconsistent 15:39:58
decisions. That can happen even within a single region. 15:40:00

BPJ number two, can be time-consuming and 15:40:04
resource intensive, and the utility of BPJ is limited 15:40:10
for a third reason, and that is it may not always lead 15:40:11
to transparency or unbiased decisions, and then number 15:40:14
4, BPJ is limited because it may not allow Regional 15:40:18
Board staff or interested parties to assess the outcome 15:40:23
independently. 15:40:27

And -- and what I understand is that staff were very aware of test problems with BPJ and did their best to try to tease those problems out of the SQO, but they couldn't do it fully, and I'm trying to figure out where that remaining judgment lies. 15:40:28 15:40:31 15:40:36 15:40:42 15:40:45

They were not -- 15:40:48

MR. GALLAGHER: So each of these issues 1 through 4 -- is a decision to -- to assess independence of it. 15:40:49 15:40:53 15:40:59

You're just trying to take each one of these and then you -- understand how -- how each one of these -- these conclusions -- 15:40:59 15:41:02 15:41:10

You're just trying to understand the discussion that took place in regards to these four BPJ points? 15:41:11 15:41:13 15:41:18

MR. SINGARELLA: Yes. 15:41:18

That was the last line of questioning before we called you. 15:41:19 15:41:21

MR. GALLAGHER: Okay. And then the legal documents on the possibly impacted, you know, there obviously -- the possibly impacted, you know, protected condition or not, pass or no pass -- umm -- trying to make you understand how that decision was based, but they're -- they're obviously -- you know, just like BPJ, there's a series of factors, you know, obviously taken 15:41:22 15:41:26 15:41:28 15:41:34 15:41:39 15:41:42 15:41:46

in to consideration in determining the station or whatever it is that's possibly impacted. 15:41:49 15:41:57

So what -- how did that line of questioning go? 15:42:06 15:42:09

I mean, where did you -- what in the Staff Report or in the SQO document itself -- where did you take off? 15:42:09 15:42:12 15:42:16

How did that line of questioning proceed? 15:42:16

MR. SINGARELLA: Well, and I -- I will admit that that one I did not launch as well as I could have from the document itself. And -- 15:42:19 15:42:23 15:42:27

MR. GALLAGHER: Okay. 15:42:30

MR. SINGARELLA; -- let me tell you where I'd like to launch from so we can discuss it, and -- and then maybe we can resolve this issue. 15:42:31 15:42:33 15:42:36

But I have some more questions on possibly impacted, and the reason is the Staff Report says that if you've got a site that's possibly impacted, the biological effect may be nominal or transient, and then it also says that for possibly impacted sites the ability to differentiate, you know, natural stresses, something just naturally happening in the environment or random noise or variability from the actual impact may be difficult. 15:42:39 15:42:42 15:42:45 15:42:49 15:42:55 15:42:58 15:43:02 15:43:06 15:43:10

Now, that's right in the Staff Report. 15:43:10

And so the State Board was -- and staff here, Mr. Beegan, they were very aware that this possible impact, that it's kind of on the cusp, and I was trying to get at that much more directly, and I think perhaps, you know, the way I did it led to the objection. 15:43:12 15:43:15 15:43:18 15:43:23 15:43:25

And the way I did it directly is I asked Mr. Beegan if at one point possibly impacted was considered by staff to be part of the protective condition, and then it switched over, and -- and really I can get at this point simply by discussing page 5-46 of the Staff Report, because it's right here. 15:43:29 15:43:32 15:43:36 15:43:37 15:43:41 15:43:48

I mean, they're saying -- I think we're entitled to understand, well, what do you mean by the affecting nominal or transient? 15:43:50 15:43:52 15:43:54

What do you mean by gee, I can't tell the difference between a real effect and random variability of possible impacted stations? 15:43:57 15:43:59 15:44:04

Those are the kinds of questions I would like to ask and haven't had the opportunity to. 15:44:05 15:44:08

MR. FUCHS: Well, those are the kind of questions that -- 15:44:10

Are you done, Paul? 15:44:10

I don't want to interrupt you. 15:44:11

MR. SINGARELLA: Yes, Dan. 15:44:13

MR. FUCHS: Okay. Those are the kind of 15:44:14

questions that do not address how someone who wants to use the SQOs would do so. 15:44:15 15:44:21

I want to just call your attention back to the rationale given originally in correspondence from Mr. Singarella's office as to why they wanted to take Mr. Beegan's deposition. 15:44:25 15:44:28 15:44:38 15:44:41

They said to address the Regional Board's rationale for selecting its methodology and application thereof. 15:44:42 15:44:45 15:44:49

So it's a very important to remember that this is all -- 15:44:50 15:44:53

MR. GALLAGHWER: Now, Dan, read that again.

Dan, read that again. 15:44:54

MR. FUCHS: Pardon me? Oh. 15:44:56

MR. GALLAGHER: That sentence again. 15:44:59

MR. FUCHS: Right. 15:45:01

The -- it was the Regional Board's rationale for selecting its methodology and application thereof. 15:45:02 15:45:06

And I don't have the -- the whole sentence here. I could find it. 15:45:11 15:45:14

But that's the -- the reason given for taking Mr. Beegan's deposition, the reason given in a letter to me from Mr. Richardson. 15:45:15 15:45:18 15:45:22

MR. GALLAGHER: All right. And you feel that, you know -- that you've allowed to identify rationale in 15:45:24 15:45:26

the application, but -- the rationale -- 15:45:33

Let's -- let's take -- take one of the 15:45:39

examples. 15:45:44

Take an example of how you believe he -- 15:45:45

you've allowed the deponent to discuss the rationale of 15:45:48

the application, just, I mean, BPJ, or possibly 15:45:52

impacted, just one of those or something else, whatever 15:45:58

you feel comfortable with. 15:46:01

You gave him rationale, you gave him 15:46:03

application, application. 15:46:05

Can you give me something -- 15:46:10

MR. FUCHS: Well, it -- the -- 15:46:13

What we're here for are the -- the case we're 15:46:17

here in is the San Diego Regional Board's Tentative 15:46:21

Cleanup And Abatement Order and -- 15:46:25

MR. GALLAGHER: Which I wasn't going allow you 15:46:28

to dig in to at all. 15:46:32

MR. FUCHS: Well, right. 15:46:34

So what -- we're not here to explore the -- 15:46:36

the background of the SQOs except that I've allowed, 15:46:42

frankly, a lot of questioning on the background because 15:46:48

Mr. Singarella made the case that someone who wants to 15:46:52

go to apply the SQOs might be interested or might be 15:46:56

aided by knowing some of the background. 15:47:01

So I've allowed an enormous amount of 15:47:04

questioning on the background. 15:47:07

But there comes a point when -- when we've 15:47:08
done enough on that, and when we're really invading the 15:47:11
mental processes, not -- not just the deliberative 15:47:15
process, but the broad category of mental processes of 15:47:18
folks who are involved. 15:47:22

And so, again, you know, I think Mr. 15:47:24
Singarella and I are in great agreement as to what the 15:47:28
questioning is relevant and what his basis is, and my 15:47:32
point is that the precise arguments he's made for why he 15:47:35
should be allowed to continue the questioning are why he 15:47:41
should not be allowed to continue the questioning. 15:47:46

MR. SINGARELLA: Which I find rather ironic 15:47:49
because -- 15:47:51

Mr. Gallagher, may I be heard? 15:47:53

MR. GALLAGHER: Yeah, go ahead. Please. 15:47:55

MR. SINGAREALLA: Yeah.

I mean, my client is facing a, you know, 15:47:57
potential action here with this cleanup order that, as 15:48:00
you know, is -- is very, very substantial, and we are 15:48:05
getting at the methodology used by the Regional Board, 15:48:12
and it's not by directly -- asking about the CAO today. 15:48:18

You put that off limits, and I understand 15:48:23
that. 15:48:25

But what is at issue is this SQO and that 15:48:25

Staff Report and whether this agency is going to try to wrap itself in the cloak of those documents and say, "Well, you know, the SQO didn't apply as a matter of law in San Diego Bay, but we used methods that are similar or the same to what was laid out in those materials, and that gives us the cloak of -- of, you know, an appropriate approach and an approach blessed by the State Board." 15:48:30 15:48:33 15:48:37 15:48:42 15:48:46 15:48:52 15:48:56

And I can imagine when we get to briefing here that they're going to be using these sentences from these materials, and they'll have their viewpoint on what they mean, and they will be using that to support a very substantial cleanup order. 15:48:59 15:49:02 15:49:06 15:49:10 15:49:15

And what I'm trying to do is create a record in this evidentiary proceeding so that these statements don't get misconstrued to prejudice my client. 15:49:17 15:49:20 15:49:23

So am I trying to get at the SQOs? 15:49:28

Of course, I am. 15:49:32

Am I trying to get at the deliberative process of the State Board? 15:49:33 15:49:37

Of course not. 15:49:37

Those are two separate things, and I think I've been at this long enough to understand how to judge that line. 15:49:39 15:49:41 15:49:44

MR. GALLAGHER: On the BPJ line of 15:49:46

questioning, and I know you said 5-31, but -- 15:49:49

Dan, has -- there's been discussion of how -- 15:49:58

how BPJ is employed? 15:50:03

MR. FUCHS: There's been a discussion as to 15:50:06

whether BPJ -- 15:50:11

Well, I stopped the questioning when Mr. 15:50:14

Singarella started asking whether Mr. Beegan had applied 15:50:18

BPJ in developing the -- Benthic index. 15:50:21

MR. GALLAGHER: Okay. 15:50:31

MR. FUCHS: Sorry. 15:50:34

Go ahead. 15:50:35

MR. GALLAGHER: The Staff Report -- the Staff 15:50:35

Report says BPJ is applied when? 15:50:38

What is the sentence on page 5-31? 15:50:44

I understand this can be time-consuming 15:50:46

inconsistent decisions, maybe not a lot of transparency, 15:50:50

you know, and -- may -- may not allow the Regional Board 15:50:55

to assess the outcome independently. 15:50:59

So I understand that, and I probably would 15:51:03

have stopped the line of questioning. 15:51:06

I wouldn't allow a line of questioning 15:51:07

understand how the decisions made, blah, blah, blah. 15:51:11

But have -- there's -- 15:51:12

Has there been some discussion how BPJ is 15:51:16

applied in the context of what you just said, Dan? 15:51:20

MR. FUCHS: How BPJ would be -- 15:51:22

No. 15:51:24

I would allow Mr. Singarella to ask how 15:51:24

someone using the SQOs would apply BPJ, and, in fact, I 15:51:29

did allow such questioning without any complaint or 15:51:35

objection. 15:51:38

It's how BPJ was employed in generating or 15:51:39

deriving the SQOs that -- where I put my foot down. 15:51:44

MR. GALLAGHER: But the line of questioning 15:51:50

that you did allow -- 15:51:51

Go on, Dan. 15:51:55

Fill that in for me. 15:51:56

MR. FUCHS: And -- and would continue to allow 15:51:57

is how someone, anyone, who is going to use the SQOs 15:51:59

should or could use their own best professional 15:52:10

judgment. 15:52:16

MR. GALLAGHER: Okay. 15:52:19

MR. FUCHS: It all comes back to the -- the 15:52:21

parameters that we all agreed on two weeks ago. 15:52:26

MR. GALLAGHER: Yeah. All right. 15:52:30

And then -- umm -- 15:52:31

Let's see. 15:52:39

MR. FUCHS: But, you know, I don't want you to 15:52:42

be mistaken. 15:52:44

Mr. Sangarella gave you a couple of examples 15:52:45

of instances where I have -- I've refused to let Mr. Beegan answer, and it's not limited to that. 15:52:47 15:52:51

I've -- I've told him not to answer about other things as well. 15:52:54 15:52:56

MR. GALLAGHER: Right. 15:52:58

MR. FUCHS: But generally I think, you know -- 15:52:58

So he's actually portraying me as more reasonable than I've been. 15:53:01 15:53:04

MR. GALLAGHER: Okay. 15:53:06

MR. FUCHS: So I think I've been reasonable, but -- 15:53:07 15:53:09

MR. SINGARELLA: Well, I'd like to just get through this, Mr. Gallagher. 15:53:12 15:53:14

You know, Dan's right, he has been interrupting, quite a bit, and I've got more stuff in this Staff Report that springs right from the language of the Staff Report that is the document used to adopt these SQOs and help people understand them, and I'd simply like to get through it. 15:53:16 15:53:18 15:53:22 15:53:26 15:53:31 15:53:37

MR. FUCHS: And I absolutely share -- 15:53:39

MR. GALLAGHER: Go ahead, Dan. 15:53:41

MR. FUCHS: I absolutely share the desire to just get through this. 15:53:43 15:53:46

I thought we would have been done in the morning, and I thought we would have been done if Mr. 15:53:46 15:53:48

Singarella had limited his questioning to the -- to be within the parameters that were set forth on September 27th. 15:53:49 15:53:54 15:53:57

MR. GALLAGHER: All right. Possibly impacted, page 5-46 saying it's nominal or transient -- can be nominal or transient -- 15:53:58 15:54:01 15:54:11

What was the next step with that? 15:54:15

What was the -- the -- questioning, Paul? 15:54:18

MR. SINGARELLA: Sorry to interrupt, sir. 15:54:24

Yeah. 15:54:26

My line of questioning was to understand what "possibly impacted" means with regard to these statements by the staff about, you know, nominal and transient biological effects and the fact that you can have these other things, natural influences, and just noise, statistical noise as -- 15:54:26 15:54:31 15:54:35 15:54:41 15:54:44 15:54:47

MR. GALLAGHER: Right. 15:54:50

MR. SINGARELLA: -- misleading you and thinking there's a pollutant-related stress there. 15:54:51 15:54:53

And, you know, I wish these words just spoke for themselves, but I think this possibly impacted category needs some understanding lest it be, you know, misconstrued. 15:54:55 15:54:58 15:55:02 15:55:08

MR. GALLAGHER: Well, how do you want to do this? 15:55:09 15:55:11

How do you want to go forward? 15:55:12

MR. SINGARELLA: Well, I -- 15:55:13

You know, if I'm limiting my questions to the 15:55:14
Staff Report, then in my mind I just -- I'm having a lot 15:55:17
of trouble finding this -- this distinction. 15:55:22

This is methodology. 15:55:25

I certainly have no intent to cross over any 15:55:27
line that you set, Mr. Gallagher, and I thought I was 15:55:30
vigilantly respecting it by limiting my questions 15:55:37
principally to these two seminal documents. 15:55:39

And I -- I've got probably another hour max in 15:55:42
-- in the Staff Report, and then I have some additional 15:55:47
questions about executive summaries and conclusions and 15:55:50
the documents that Mr. Beegan pointed us to last week, 15:55:55
and I'm done. 15:56:00

I don't know if I can finish today given where 15:56:01
we are, but I've done my level best. That's for sure. 15:56:03

MR. GALLAGHER: Okay. So let me just give you 15:56:07
a -- I lean a bit in favor of what Dan's saying in terms 15:56:10
of BPJ and page 541 and getting in to inconsistent 15:56:26
decisions and getting in to each one of those four 15:56:31
categories. 15:56:33

I'm not so sure where do you cross the line 15:56:34
between methodology and the mental process and -- you 15:56:38
know, jumping to possibly impacted, and, you know, 15:56:44

nominal or transient, and understand what that means or at least -- identifying factors such as statistical noise. I'm not exactly sure where you'll go -- 15:56:50 15:56:54 15:57:00

I'm happy -- I won't say I'm happy, but I'm more than pleasantly willing to -- or unpleasantly willing to be on the phone to, you know, deal with these questions to help you get through it for the next hour if that's what you want to do. 15:57:08 15:57:10 15:57:17 15:57:22 15:57:26

And, obviously, you know, the objective, and it doesn't mean what I decide, if it's methodology or mental process it's hundred percent right, leaning one direction or another, but because it does get a little difficult. 15:57:28 15:57:34 15:57:39 15:57:44 15:57:48

MR. SINGARELLA: We certainly don't want to impose, or I don't want to impose, but I do think that perhaps if you are available, perhaps taking advantage of -- of your kind offer to sit in on this for the next hour might not be a bad idea. 15:58:00 15:58:01 15:58:06 15:58:12 15:58:16

MR. GALLAGHER: I think so, too. 15:58:19

I'm in Court doing my Special Master functions on another matter, so I'm not available, you know, tomorrow morning, so why don't we just hop to it now. 15:58:21 15:58:24 15:58:28

MR. SINGARELLA: Okay. 15:58:32

MR. FUCHS: All right. Mr. Beegan stepped out briefly. So he should -- he should be back in a minute 15:58:32 15:58:34

or two. 15:58:40

MR. GALLAGHER: Okay. He ran somewhere else? 15:58:40

MR. FUCHS: He ran off to the restroom, I think. 15:58:43 15:58:45

MR. GALLAGHER: Yeah. And please don't hesitate to interrupt me, and say "I don't think you have it right" and explain, and I take no offense, and so, you know, we've talked it out a little, but I want to move as expeditiously as possible. 15:58:45 15:58:48 15:58:53 15:58:57 15:59:02

MR. SINGARELLA: I appreciate that, sir. 15:59:06

So we'll just wait for Mr. Beegan to return. 15:59:12

MR. FUCHS: Yeah. 15:59:15

MR. GALLAGHER: Okay. 15:59:15

(Discussion off the record) 16:01:24

VIDEOGRAPHER: We are back on the record at 4:01 p.m. 16:01:25 16:01:28

BY MR. SINGARELLA: 16:01:29

Q. Mr. Beegan, could you turn to Part 1, Attachment B, pages 26 and 27. 16:01:29 16:01:38

A. Okay. I'm there. 16:01:49

Q. And on page 27 there's a Line of Evidence category combination number 52. 16:01:50 16:01:55

Do you see that? 16:01:59

A. 52. 16:02:00

Umm -- yes. 16:02:02

Q. Now, what are these category combinations? 16:02:04

A. These -- category combinations are -- the sum total of the individual line of evidence categories integrated in to a station assessment category. 16:02:15 16:02:23 16:02:33

Q. And the station assessment category is whether the station passes or fails the SQO? 16:02:39 16:02:45

A. Well -- umm -- yes. 16:02:49

Q. And likely impacted as a station assessment means failure of the SQO; right? 16:02:55 16:02:59

A. Umm -- not necessarily. 16:03:01

Q. Sure. 16:03:06

Please explain. 16:03:09

A. So you don't make a pass/fail decision on the SQO based on a single station. 16:03:10 16:03:16

So there's multiple stations involved, and so I guess you can say that -- umm -- there's a line drawn at some point in these categories between possibly impacted and -- likely unimpacted. 16:03:19 16:03:27 16:03:31 16:03:34

That doesn't necessarily mean you failed the SQO. It's not conducted at a station-by-station basis. 16:03:41 16:03:43

Okay? 16:03:50

Q. If you had a series of stations that you formally fell in to the category of likely impacted and nothing else, would you fail the SQO? 16:03:50 16:03:53 16:03:58

A. Umm -- yes, that would be defined as an 16:04:00

exceedance, yeah. 16:04:04

Q. That would not be a protective condition; 16:04:05

correct? 16:04:08

A. No. No. 16:04:09

Q. And in category combination 52 -- 16:04:09

Are you with me? 16:04:14

A. 52? 16:04:14

Yes. 16:04:16

Q. You see how reference benthic community 16:04:16

condition can produce an SQO result of likely impacted? 16:04:22

A. Yes. 16:04:26

Q. And turning to page 26, can I turn your 16:04:26

attention to categories -- category combination 20 and 16:04:42

category combination 36? 16:04:46

Do you see those two? 16:04:48

A. 20 and 36? 16:04:50

Q. Yes. 16:04:51

A. Yes. 16:04:57

Q. And do you see how benthic community condition 16:04:57

of reference can result in a station assessment of 16:05:01

possibly impacted for those two combinations? 16:05:04

A. Yes. 16:05:07

Q. And if all you had were possibly impacted 16:05:08

stations, you would conclude that there is an exceedance 16:05:10

of the SQO for that site; right? 16:05:15

A. Yes. 16:05:18

MR. SINGARELLA: Okay. 16:05:25

MR. GALLAGHER: May I interrupt, folks, for a minute? 16:05:29 16:05:32

MR. SINGARELLA: Please. 16:05:32

MR. GALLAGHER: This is Tim Gallagher speaking, Discovery Referee. 16:05:33 16:05:35

This line of questioning, Dan, you can correct me, but really goes to methodology, so at least up to this point, so I didn't have a degree of difficulty. 16:05:38 16:05:41 16:05:45

Obviously, Dan didn't either, but, you know, clearly methodology. 16:05:48 16:05:51

MR. FUCHS: Agreed. 16:05:52

This is Dan. Thank you. 16:05:53

MR. SINGARELLA: Thank you. 16:05:54

MR. GALLAGHER: I don't need to say that, and I apologize, but I -- you know. 16:05:55 16:05:58

Within that framework I don't have a problem. 16:06:01

MR. SINGARELLA: Thank you. 16:06:04

No, that's helpful. 16:06:04

BY MR. SINGARELLA: 16:06:05

Q. So let's turn back to the Staff Report, page 5-46. 16:06:06 16:06:11

This is on the section regarding receiving water limits. 16:06:18 16:06:23

Do you see that? 16:06:24

A. Yes. Yes. 16:06:25

Q. And the third paragraph discusses performing 16:06:30
stressor identification; right? 16:06:34

A. Yes. 16:06:36

Q. And it indicates that stressor identification 16:06:38
can be particularly useful for those sites classified as 16:06:45
possibly impacted; right? 16:06:51

A. Yes. 16:06:52

Q. And one of the reasons that stressor 16:07:02
identification at possibly impacted sites is helpful is 16:07:06
that biological effects at such sites might be nominal; 16:07:14
right? 16:07:20

A. Correct. 16:07:20

Q. Or transient; right? 16:07:21

A. Correct. 16:07:23

Q. What does it mean when a biological effect is 16:07:24
not or transient? 16:07:29

A. It means it is -- 16:07:30

Maybe it's -- 16:07:35

That's -- 16:07:38

It occurs on a temporal basis or over time. 16:07:39
It -- it comes and goes, or it's measurable, and then 16:07:44
it's not. 16:07:48

It's at the low end of the -- umm -- 16:07:50

detectible response where -- noise that is regular -- or regular -- umm -- variability in organism response may affect the result, various factors such as that. 16:07:56 16:08:11 16:08:19

Q. And all those things you just described could be happening at possibly impacted sites; right? 16:08:27 16:08:31

A. They could be happening -- umm -- at -- Yes. Yes. 16:08:34 16:08:41

Q. Now, the next sentence refers to natural stressors; right? 16:08:42 16:08:46

A. Yes. 16:08:48

Q. And random variability; correct? 16:08:50

A. Right. 16:08:52

Q. And those are distinguished, on the one hand, from pollutant-related stressors on the other; correct? 16:08:53 16:08:56

A. Yes. 16:09:00

Q. And this sentence indicates that it might be difficult to distinguish the natural stressors and the random variability from pollutant-related stress at possibly impacted sites; correct? 16:09:01 16:09:08 16:09:13 16:09:17

A. Correct. 16:09:19

Q. What is meant by the reference to "natural stressors"? 16:09:19 16:09:24

A. Umm -- scour, currents, salinity, habitat factors, grain size, where those factors aren't caused by anthropogenic inputs or -- 16:09:25 16:09:39 16:09:49

Q. Are these natural stressors the same as those confounding factors that we spoke about earlier? 16:09:58 16:10:04

A. They could -- they -- confounding factors could include natural factors. 16:10:07 16:10:16

Q. And "random variability," what does that refer to, Mr. Beegan? 16:10:19 16:10:25

A. Umm -- 16:10:26

Q. Let me help out. 16:10:30

Random variability in what? 16:10:33

A. Right. 16:10:35

I'm going to read the paragraph. 16:10:36

Q. Oh, please do. 16:10:37

MR. GALLAGHER: Can somebody put a speaker to the phone if you could? 16:10:45 16:10:47

THE WITNESS: Random variability may have to do with -- umm -- your toxicity test organism may exhibit some variability. 16:10:53 16:10:55 16:11:03

The benthic community could exhibit some variability. 16:11:05 16:11:10

Umm -- basically what this sentence is describing is -- umm -- being able to differentiate a pollutant-related stressor from the background noise within the system for -- all three Lines of Evidence, chemistry, sediment toxicity, and benthic community. 16:11:14 16:11:22 16:11:30 16:11:36 16:11:45

BY MR. SINGARELLA: 16:11:50

Q. And that's a particular challenge for possibly impacted sites? 16:11:50 16:11:53

A. It's more of a challenge for possibly -- 16:11:54

Yes, I would agree. 16:11:57

Q. And the point of this paragraph is that's why you go through stressor identification; correct? 16:11:59 16:12:05

A. Umm -- 16:12:07

Q. Let me rephrase. 16:12:34

The first sentence of that paragraph refers to using stressor identification to address confidence; correct? 16:12:35 16:12:39 16:12:42

A. Yes. 16:12:42

Q. And does that word "confidence" refer to confidence that a possibly impacted station is truly affected by toxic pollutants? 16:12:44 16:12:48 16:12:53

A. Yes. 16:12:56

Q. And the idea isto avoid mistaking toxic pollutants as the causative agent when, in fact, it might be these other things; right? 16:12:57 16:13:12 16:13:16

A. Correct. 16:13:18

Q. And stressor identification helps you get to the requisite level of confidence in that determination; right? 16:13:18 16:13:28 16:13:31

A. Yes. 16:13:31

Q. Turning to page 5-47, under "Potential 16:13:32

Response Actions for Exceedances," do you see that? 16:13:40

A. Yes. 16:13:42

Q. This is a new term here that we haven't talked about today. 16:13:44 16:13:48

It refers to this "simple co-occurrence of pollutants." 16:13:49 16:13:52

Do you see that in the first sentence? 16:13:53

A. Umm -- yes. 16:13:55

Q. And it says that sediment management actions typically are based on the simple co-occurrence guidelines; right? 16:13:56 16:14:01 16:14:05

A. Yes. 16:14:07

Q. What is "simple co-occurrence" as used in this context? 16:14:07 16:14:12

A. Going back to the development of empirical sediment guidelines that we talked about -- umm -- we discussed the fact that those are based on mixtures and -- umm -- so they're not causal -- they're not -- there's no establishment of cause. 16:14:12 16:14:22 16:14:26 16:14:33 16:14:41

Umm -- and the reason that is is because these are, again, mixtures. 16:14:48 16:14:52

There are typically multiple chemicals at any one station that's used in the development of these empirical guidelines. 16:14:55 16:15:01 16:15:04

So oftentimes you can -- you may see a -- some 16:15:06

sort of relationship between a chemical and biological effects, but it doesn't necessarily relate to cause. 16:15:13 16:15:26

That's probably -- and I guess what I'm getting at is it's a demonstration of the weakness associated with these empirical quality sediment guidelines. 16:15:29 16:15:35 16:15:39 16:15:44

Q. And that paragraph -- 16:15:44

I'm sorry. 16:15:46

Did I cut you off? 16:15:47

A. No. 16:15:49

The -- the regulatory decisions that are referred to are referring to the exceedance of a sediment quality guideline and biological effects, and it's attempting to explain that these are not good -- indicators of cause, they merely establish occurrence or co-occurrence, or, you know, an association. 16:15:50 16:15:53 16:15:59 16:16:04 16:16:13 16:16:22

Q. So they would build more or less a circumstantial case against a particular compound? 16:16:27 16:16:30

A. Right. 16:16:35

Q. When there might be a host of other potential causative agents at play; right? 16:16:36 16:16:44

A. Correct. 16:16:48

Q. So going down through that paragraph, the paragraph says that "Those relationships," referring to these sediment quality guidelines, "don't demonstrate 16:16:48 16:16:55 16:16:58

causality;" right? 16:17:02

Second sentence. 16:17:08

A. I'm -- 16:17:15

Q. The second sentence says "This relationship does not demonstrate causality;" correct? 16:17:16 16:17:20

A. Yes. 16:17:23

Q. What does "this relationship" refer to? 16:17:24

A. Umm -- a co-occurrence of biological effects with sediment quality guideline. 16:17:26 16:17:35

Q. Okay. Thank you. 16:17:38

And then third sentence refers to enormous resources applied instead of focusing on the causes; correct? 16:17:39 16:17:45 16:17:51

A. Right. 16:17:51

Q. What does that mean? 16:17:52

A. It means that -- umm -- that TMDLs -- to develop -- 16:17:54 16:18:09

TMDLs develop for stressors -- and I use -- 16:18:10

I shouldn't use the term "stressors." 16:18:19

TMDLs develop for pollutants based on a -- an exceedance of a sediment quality guideline, with associated biological effects -- may not result in an improvement of sediment quality. 16:18:21 16:18:28 16:18:37 16:18:45

Q. And then further on in paragraph -- there's a reference to the performance of stressor identification; 16:18:55 16:19:03

correct? 16:19:05

A. Umm -- what is that sentence? 16:19:06

Q. It's the sentence that begins -- six lines up, 16:19:14

"If stressor identification is performed and a stressor 16:19:20

is identified." 16:19:22

Do you see that? 16:19:25

A. Oh, yes. Yes. Yes. 16:19:26

Q. And that refers to the stressor identification 16:19:28

section in Part 1; correct? 16:19:30

A. It refers to the stressor identification 16:19:32

section and the -- umm -- the section -- the two 16:19:38

sections later, I think, that we talked about, 16:19:45

management guidelines. 16:19:48

I think it refers to both of those. 16:19:50

Q. It's the next section. 16:19:53

A. Oh, okay. Okay. 16:19:55

Q. It follows right after. 16:19:56

A. Okay. 16:19:58

Q. The site specific guideline specification? 16:19:59

A. Right. 16:20:03

There was the cleanup and abatement section. 16:20:03

Q. I'm sorry. I should have known better. 16:20:06

Yes, Section F is stressor identification. 16:20:09

Section G is cleanup and abatement, and 16:20:13

Section H is development of site specific sediment 16:20:17

management guidelines; right? 16:20:19

A. Correct. 16:20:21

Q. Sorry. I misspoke, Mr. Beegan. 16:20:21

My apologies. 16:20:25

And so this paragraph on page 5-47 refers to 16:20:28
stressor identification and also the development of site 16:20:33
specific sediment management guidelines; correct? 16:20:36

A. Yes, correct. 16:20:38

Q. And those can lead to remediation goals; 16:20:39
correct? 16:20:47

A. They can develop -- lead to TMDL development 16:20:47
or remediation goals. 16:20:54

Q. And then finally at the end of the paragraph 16:20:58
there's a statement that the SQGs would not fulfill this 16:21:01
role; correct? 16:21:08

MR. FUCHS: I object. 16:21:14

That actually misstates the sentence. 16:21:15

BY MR. SINGARELLA: 16:21:17

Q. And then what does the last sentence mean, 16:21:18
Mr. Beegan? 16:21:21

Rather than leave it that way and have me 16:21:26
botch it. 16:21:29

A. Okay. So this is not absolutely one hundred 16:21:32
percent clear. 16:21:44

The second to the last sentence, headline 16:21:45

development, would account for site receptor specific factors that control by variability refers to Section H, development of site specific sediment management guidelines. 16:21:49 16:21:52 16:21:59 16:22:02

And then the next sentence is -- saying that the adoption of Sediment Quality Guidelines to fill this role does not account for factors -- those guidelines are the national guidelines, or statewide guidelines, or nonsite specific. 16:22:04 16:22:11 16:22:16 16:22:20 16:22:24

So I can see where that would be confusing. 16:22:26

But I believe that's my intent. 16:22:31

Q. In the last sentence your intent was to refer to the national guidelines that were discussed in the earlier part of the Staff Report? 16:22:34 16:22:37 16:22:43

A. Either the national guidelines -- 16:22:44

Yeah, it was probably the national guidelines. 16:22:47

But you could -- the chemistry guidelines used in the MLOE approach also -- you also would want to use those chemical values for the same reasons, because they don't account for those factors. 16:22:49 16:22:55 16:23:04 16:23:09

Q. Referring to the CSI and California LRM? 16:23:11

A. Yes. 16:23:15

Q. And so on page 5-48, staff recommended Alternative 2, which incorporated in to Part 1 the stressor identification steps of Section F; right? 16:23:16 16:23:20 16:23:23

A. Yes. 16:23:28

Q. And the biologically relevant guidelines in Section H; correct? 16:23:30 16:23:35

A. Correct. 16:23:36

Q. And those were ultimately adopted in to the SQO? 16:23:36 16:23:40

A. Correct. 16:23:41

Q. Okay. Let's turn to the next chapter, Chapter 6, 6-4. 16:23:41 16:23:48

6-4, first paragraph, the last sentence indicates the expectation that the Regional Boards would likely control -- control strategies and remediation only for highly impacted localized sites. 16:23:57 16:24:04 16:24:09 16:24:17

Do you see that? 16:24:20

A. Umm -- yes. 16:24:22

Q. And what is meant by the phrase in reference to "highly impacted localized sites"? 16:24:35 16:24:42

A. I guess -- what was meant by that was not -- umm -- 16:24:45 16:24:58

We were -- the Board was unlikely to require remediation for whole water bodies or large areas within a water body. 16:25:05 16:25:13 16:25:27

Q. Okay. Turning to page 6-6, this first paragraph refers to the primary limitation of Part 1; is that right? 16:25:29 16:25:43 16:25:47

A. I'm sorry. 16:25:47

Correct. 16:25:50

Q. And the primary limitation of the Part 1 Line of Evidence approach is that it doesn't provide any direct information on potential cause of exceedance; correct? 16:26:00 16:26:07 16:26:09 16:26:14

A. Right. 16:26:14

Q. And when it says "an exceedance," it's referring to exceedance of the SQO? 16:26:15 16:26:22

A. Yes. Correct. 16:26:25

Q. So the multiple Line of Evidence approach just doesn't go to the issue of cause; correct? 16:26:26 16:26:31

A. It does not identify pollutants that are causing impacts. 16:26:34 16:26:44

Q. Does the next sentence of this paragraph identify an additional limitation? 16:26:48 16:26:52

A. Yes. 16:26:55

Q. And what is that limitation? 16:27:06

A. It does not provide nor does the proposed SQO provide pollutant specific concentrations that would be protective of aquatic life in sediment. 16:27:08 16:27:14 16:27:17

Q. So the proposed SQO doesn't give you that level of a constituent that would be protective; right? 16:27:21 16:27:25

A. Not -- not on a single pollutant-by-pollutant basis. 16:27:29 16:27:42

Q. Could you develop that level of constituent using Section H of Part 1; right? 16:27:43 16:27:47

A. Umm -- correct. 16:27:50

Actually, you know, if you were to apply those pollutant-specific concentrations singularly to sediments where you knew the concentrations and the makeup of all the pollutants, if you applied the reference concentrations, you would likely have a low probability for biological effects. 16:28:17 16:28:25 16:28:35 16:28:42 16:28:48 16:29:00

Is that protective? 16:29:05

I don't know. 16:29:10

Q. What -- 16:29:11

I'm sorry. 16:29:14

What don't you know to be protective? 16:29:14

A. Well, I was trying to tell you that -- umm -- 16:29:16

I'll go back to just agreeing with you as far as what that sentence says. 16:29:54 16:29:59

Q. Okay. Okay. 16:30:02

Mr. Beegan, you steered us in the direction of a number of technical reports, and I'd like to ask you some questions about a few of them. 16:30:10 16:30:13 16:30:19

MR. GALLAGHER: Paul, can you speak up just a little? 16:30:28 16:30:30

MR. SINGARELLA: Yes. Yes. 16:30:30

Moving on to a -- new exhibit. 16:30:31

(Exhibit 706 was marked for Identification.) 16:30:45

BY MR. SINGARELLA: 16:30:45

Q. Mr. Beegan, I've placed in front of you what has been marked Exhibit Number 706 to your deposition. 16:30:45 16:30:48

Do you see that? 16:30:51

A. Yeah. 16:30:52

Q. And this document is entitled "Sediment Quality in California Bays and Estuaries." 16:30:58 16:31:02

Do you see that? 16:31:05

A. Yes. 16:31:05

Q. By Arthur Barnett, et al.; correct? 16:31:06

A. Correct. Yeah. Right. 16:31:08

Q. And this was work funded by the State Board; is that right? 16:31:10 16:31:15

A. Correct. 16:31:15

Q. As part of the SQO? 16:31:16

A. As part of the SQO and SCCWRP. 16:31:20

Q. Are you familiar with this document? 16:31:27

A. Yes. 16:31:28

Q. What did the authors do as -- as part of this exercise reflected in Exhibit 706? 16:31:29 16:31:38

A. They -- umm -- they compiled recent data that met certain criteria, generally regional monitoring data that had chemistry, toxicity, and benthic community measures, and as -- as best as possible, they applied 16:31:43 16:31:55 16:32:07 16:32:16

the SQO framework to that data set to get a -- an estimate of how the framework would apply. 16:32:26 16:32:39

Q. Do you have any understanding how the investigators would have used replicate samples and information from -- in this particular study? 16:32:46 16:32:49 16:32:54

A. No. 16:33:05

Q. Could you turn to page 22 of Exhibit 706, "Conclusions and Recommendations," last paragraph, please. 16:33:05 16:33:10 16:33:13

This last paragraph, once again, addresses these possibly impacted stations, and in particular the second half of that last paragraph on page 22. 16:33:18 16:33:23 16:33:26

Are you with me? 16:33:30

A. Umm -- what's it begin with? 16:33:31

Q. "Future statewide and regional assessments can be improved." 16:33:34 16:33:38

A. Oh, okay. 16:33:39

Yes. I'm there. 16:33:39

Q. You see that? 16:33:40

A. Yes. 16:33:41

Q. And then halfway through it discusses the environmental significance of sediments classified as possibly impacted. 16:33:41 16:33:48 16:33:52

Do you see that? 16:33:53

A. Uh-huh. 16:33:53

Q. And it characterizes the environmental significance of possibly impacted sediments as uncertain. 16:33:54 16:33:58 16:34:03

Do you see that? 16:34:03

A. Yes. 16:34:05

Q. And that's consistent with what was said in the Staff Report; right? 16:34:05 16:34:09

A. That's consistent with what was said in the Staff Report and the process defined in Part 1. 16:34:10 16:34:13

Q. And this paragraph goes on to refer to the minor level of contaminant effect that may be present at a possibly impacted site; right? 16:34:19 16:34:23 16:34:27

A. Correct. 16:34:30

Q. It also refers to the fact that there may be substantial disagreement among the Lines of Evidence at possibly impacted sites; right? 16:34:30 16:34:35 16:34:37

A. Correct. 16:34:39

Q. In fact, we saw that, didn't we, in Attachment B, that you can use -- you can have reference benthic community and generate a category assessment of possibly impacted; right? 16:34:43 16:34:49 16:34:52 16:35:00

A. Right. 16:35:02

Q. And, then, finally, the last sentence here refers to those stressor identification studies that are needed at possibly impacted sites; right? 16:35:03 16:35:10 16:35:12

A. Correct. 16:35:16

Q. And they're needed to determine whether 16:35:18

sediment quality at these sites is adversely impacted by 16:35:21

contaminants; correct? 16:35:26

A. Correct. 16:35:28

Q. So the stressor identification process itself 16:35:28

is where the determination of adverse impact at possibly 16:35:41

impacted sites is -- is made; right? 16:35:46

A. Correct. 16:35:48

Q. Is it just plain feasible to develop a numeric 16:35:50

objective for sediment quality? 16:36:09

MR. FUCHS: I have to object there. 16:36:12

MR. GALLAGHER: I agree with Dan. 16:36:17

MR. SINGARELLA: Well, I have a document to 16:36:21

prove this up, Mr. Gallagher. 16:36:24

MR. GALLAGHER: Okay. 16:36:26

MR. SINGARELLA: I don't think this is part of 16:36:28

deliberative process because it actually wasn't 16:36:34

exchanged between one agency and another. 16:36:38

MR. GALLAGHER: Let's see the document. 16:36:41

Okay. 16:36:43

(Exhibit 707 was marked for Identification.) 16:36:54

BY MR. SINGARELLA: 16:36:55

Q. Mr. Beegan I've caused to be placed in front 16:36:56

of you Exhibit 707 to your deposition. 16:36:58

Do you see that, sir? 16:37:01

A. Yes. 16:37:02

Q. And do you recognize this as an E-mail between you and a member of another California agency, the Office of Administrative Law? 16:37:11 16:37:14 16:37:20

A. Yes. 16:37:22

Q. And this is actually an E-mail from Mr. Mentink? 16:37:23 16:37:28

Did I say his name right? 16:37:30

A. Umm -- I -- that sounds right. 16:37:31

Q. M-e-n-t-i-n-k, dated April 10th, 2008; is that right? 16:37:35 16:37:40

Is that right, sir? 16:37:47

A. Yes. Yes. 16:37:47

Q. And in that last full paragraph he indicates that you mentioned to him that when numeric criteria are unfeasible the Water Code in the Clean Water Act authorizes the use of narratives; correct? 16:37:48 16:37:51 16:37:57 16:38:02

A. Hold on. 16:38:07

Last paragraph? 16:38:09

Q. Yes. 16:38:10

A. Wait. 16:38:18

Which -- 16:38:18

MR. FUCHS: Counsel, where are you on this? 16:38:25

MR. SINGARELLA: Physically on the page? 16:38:30

MR. FUCHS: Yeah. 16:38:31

MR. SINGARELLA: Oh. I'm on the bottom of page 1. 16:38:32 16:38:33

MR. FUCHS: Okay. 16:38:34

THE WITNESS: Oh, okay. 16:38:36

Umm -- 16:38:57

MR. FUCHS: Hang on. 16:39:05

Was there a question pending? 16:39:07

MR. SINGARELLA: No. 16:39:10

BY MR. SINGARELLA: 16:39:14

Q. Do you recall mentioning to Mr. Mentink that you can proceed with narrative objectives when numeric criteria are unfeasible? 16:39:15 16:39:20 16:39:24

MR. FUCHS: I still think the objection I made before you handed out the document stands. 16:39:26 16:39:28

MR. SINGARELLA: Which is? 16:39:31

MR. FUCHS: Which is that this is outside the scope of applying the SQOs. 16:39:32 16:39:34

MR. GALLAGHER: I agree with Dan. 16:39:37

MR. SINGARELLA: Okay. 16:39:40

MR. GALLAGHER: Maybe you want to ask a question later when -- we're already done with this. 16:39:41 16:39:43

MR. SINGARELLA: Sure. 16:39:49

I will attempt that. 16:39:51

MR. GALLAGHER: Okay. Sorry, Paul. 16:39:52

MR. SINGARELLA: I understand. 16:39:55

MR. GALLAGHER: Let's cut to the chase here. 16:39:56

MR. SINGARELLA: Sure. 16:39:58

I'm doing my level best here. 16:39:58

BY MR. SINGARELLA: 16:40:00

Q. Would you agree that it was not feasible to develop a numeric sediment quality objective? 16:40:00 16:40:02

MR. FUCHS: Same objection. 16:40:07

BY MR. SINGARELLA: 16:40:09

Q. Was it feasible to develop a numeric quality objective? 16:40:10 16:40:13

MR. FUCHS: Same objection. 16:40:16

MR. SINGARELLA: Are you instructing him not to answer? 16:40:26 16:40:28

MR. FUCHS: Oh, yeah. 16:40:29

THE WITNESS: Yes what? 16:40:29

MR. FUCHS: I'm instructing you not to answer. 16:40:31

THE WITNESS: Okay. 16:40:33

(Exhibit 708 was marked for Identification.) 16:40:33

MR. SINGARELLA: I'll move on. 16:40:41

MR. FUCHS: Thank you. 16:40:43

BY MR. SINGARELLA: 16:40:43

Q. I'm placing in front of you what has been marked as Exhibit 708. 16:40:44 16:40:47

Do you see that, sir? 16:40:51

A. Yes. 16:40:53

Q. Exhibit 708 is another one of these technical reports, this one called "Evaluation of Methods for Measuring Sediment Toxicity in California Bays and Estuaries;" right? 16:40:55 16:41:00 16:41:07 16:41:12

A. Correct. 16:41:12

Q. Could you turn to the Executive Summary, please. 16:41:12 16:41:15

Q. First page of the Executive Summary iii, three paragraphs down? 16:41:15 16:41:26

A. iii on the Executive -- 16:41:29

MR. FUCHS: Page iii. 16:41:34

THE WITNESS: Okay. 16:41:36

BY MR. SINGARELLA: 16:41:36

Q. Third full paragraph, last sentence, there's a -- reference to sublethal tests and acute tests; right? 16:41:37 16:41:41

A. Yes. 16:41:47

Q. And those were found to be complementary rather than redundant; correct? 16:41:51 16:41:54

A. Correct. 16:41:56

Q. Indicating that they're both important as part of your Part 1 SQO; right? 16:42:00 16:42:03

A. Correct. 16:42:06

Q. And then on the second page, iv, that paragraph below the top table, that starts "The use of 16:42:06 16:42:19

multiple toxicity tests," do you see that? 16:42:26

A. Yes. 16:42:29

Q. And then there's a second sentence "The use of a diversity of test methods provides two key advantages." 16:42:29 16:42:33 16:42:38

Do you see that? 16:42:38

A. Yes. 16:42:39

Q. And first it says that "The use of two test methods reduces the influence of spurious results from a test." 16:42:41 16:42:46 16:42:50

Do you see that? 16:42:51

A. Yes. 16:42:51

Q. And the two test methods referred to here are a test for lethality and a test for a sublethal effect; right? 16:42:52 16:42:58 16:43:05

A. Correct. 16:43:05

Q. And so, on the other hand, proceeding with one of the other would increase the potential influence of the spurious result; correct? 16:43:06 16:43:15 16:43:19

A. Correct. 16:43:22

Q. If you would turn to page 17, please -- the paragraph underneath the table. 16:43:23 16:43:39

And that first sentence refers to the important features of the two sublethal tests that were incorporated in to Part 1; correct? 16:43:50 16:43:52 16:43:56

A. Yes. 16:43:58

Q. I'm sorry? 16:44:01

A. Yes. 16:44:02

Q. And those important features were not present in the suite of amphipod acute tests; correct? 16:44:02 16:44:04

A. Correct. 16:44:09

Q. Including the amphipod acute tests incorporated in to Part 1; correct? 16:44:13 16:44:16

A. Correct. 16:44:19

Q. Okay. Next page, please. 16:44:22

A. Okay. 16:44:24

Q. Page 18. 16:44:25

Bottom paragraph, first sentence, "The use of multiple toxicity tests," do you see that? 16:44:28 16:44:35

A. Correct. Yes, I do. 16:44:39

Q. And that says that "Multiple toxicity tests is needed for a complete confident evaluation;" correct? 16:44:41 16:44:45

A. Correct. 16:44:49

Q. Suggesting that the absence of the multiple toxicity tests would not be as complete or confident; right? 16:44:49 16:44:53 16:44:57

A. Correct. 16:44:57

Q. Thank you. 16:44:58

Could you go back to Exhibit 703, please, the October 5 document that you prepared for us last week. 16:45:08 16:45:21

A. Okay. 16:45:28

MR. FUCHS: You can just use my copy. 16:45:28

BY MR. SINGARELLA: 16:45:30

Q. On page 2, Mr. Beegan, you referred us to the three sediment chemistry documents here; right? 16:45:30 16:45:34

A. Correct. 16:45:38

Q. You thought that perhaps we could find the dose response relationship in these documents; is that right? 16:45:40 16:45:42 16:45:46

A. Umm -- yes, or a description of the relationship that was being evaluated. 16:45:47 16:45:58

I mean -- when you describe the dose response relationship, I don't know what exactly you're looking for. 16:46:00 16:46:05 16:46:10

Q. Okay. 16:46:10

A. What does that mean to you? 16:46:13

Q. Well, is it a -- 16:46:14

What does it mean to you? 16:46:18

Do you have a working definition of "dose response relationship"? 16:46:22 16:46:31

A. The -- dose -- "dose response relationship" implies cause. 16:46:32 16:46:37

These are -- umm -- developed based on associations, not -- not necessarily causal, and these relationships are looking just at the concentrations of 16:46:40 16:46:47 16:46:57

a chemical within a mixture as they relate to biological effects, over, you know, the spectrum of concentration ranges that exist within the bays. 16:47:05 16:47:16 16:47:20

And I think the way those relationships were evaluated is described in those documents. 16:47:29 16:47:38

Q. And -- 16:47:50

Thank you. 16:47:56

Just turning back to the SQO, Part 1 -- 16:47:57

MR. GALLAGHER: Yeah.

I just want to make note it's 10 to 5:00. 16:48:02

MR. SINGARELLA: Thank you, Mr. Gallagher. 16:48:06

MR. GALLAGHER: Thank you, Paul. 16:48:13

MR. SINGARELLA: I think we're -- I'm very close to winding up here. 16:48:15 16:48:17

THE WITNESS: Part 1? 16:48:21

MR. SINGARELLA: Yes. 16:48:22

THE WITNESS: Okay. 16:48:24

BY MR. SINGARELLA: 16:48:24

Q. If I wanted to find the level of constituent that's protective of the critters in the -- in here, can you point me to it? 16:48:24 16:48:28 16:48:34

A. No. 16:48:35

Q. If it were in here, you'd be able to point me to it; right? 16:49:00 16:49:12

A. Correct. 16:49:13

MR. SINGARELLA: I have no further questions for Mr. Beegan. 16:49:30 16:49:31

Thank you, Mr. Beegan, for your time and patience today. 16:49:33 16:49:34

Thank you, Mr. Gallagher, for listening in. 16:49:35

MR. GALLAGHER: Thank you, folks. 16:49:43

Mr. Beegan, thank you. 16:49:46

MR. FUCHS: Thank you. 16:49:46

MR. RICHARDSON: Thank you. 16:49:48

MR. GALLAGHER: All right. Go in peace. 16:49:51

VIDEOGRAPHER: Can we go off the record? 16:49:53

MR. FUCHS: Yes. 16:49:55

Off the record? 16:49:56

MR. SINGARELLA: Yes. 16:49:57

VIDEOGRAPHER: We are going off the record at 4:49 p.m. 16:49:58 16:50:00

This is end of disk number four and the end of the deposition. 16:50:01 16:50:04

(Thereupon the deposition was adjourned at 4:49 p.m.)

--o0o--

Signed under penalty of perjury:

_____

DAVID CHRISTOPHER BEEGAN

_____

DATE

I, CAROL NYGARD DROBNY, a Certified Shorthand Reporter of the State of California, duly authorized to administer oaths, do hereby certify:

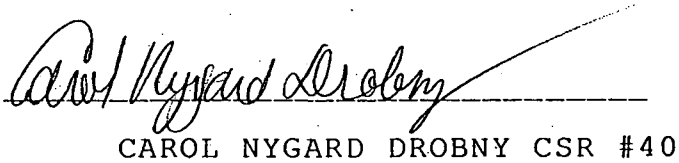
That the foregoing proceedings were taken before me at the time and place herein set forth; that any witnesses in the foregoing proceedings, prior to testifying, were duly sworn; that a record of the proceedings was made by me using machine shorthand which was thereafter transcribed under my direction; that the foregoing transcript is a true record of the testimony given.

Further, that if the foregoing pertains to the original transcript of a deposition in a Federal Case, before completion of the proceedings review of the transcript [ ] was [ ] was not requested.

I further certify I am neither financially interested in the action nor a relative or employee of any attorney or party to this action.

IN WITNESS WHEREOF, I have this date subscribed my name:

Dated:



CAROL NYGARD DROBNY CSR #4018

C

E

		APPENDIX E	COMMENTS AND RESPONSES	
No.	Subject	Comment	Response	Author
		VERBAL COMMENTS (02 05 08)		
V1		SQO is a complex policy	Staff agree. Incorporating multiple lines of evidence into a draft Water Quality Control Plan requires a unique and complex approach	WSPA, CASQA
V2		Appreciate the clarifications, and figures in the January 2006 draft Part 1	Comment noted.	WSPA, CASQA
V3		There are inconsistencies between some items and we will talk to staff about those issues, however we support the Draft Part 1	Comment noted.	WSPA, CASQA
V4		We are disapointed with the loophole associated with the Possibly Impacted response actions between Section V.I and Section VII.F. Concerned about option to postpone stressor ID pending further monitoring. We disagree with the this text.	Comment noted. As with any attempt to be protective, the draft Part 1 must realistically address those areas of uncertainty. An unsuccesful TIE is a real possibility and continuing to spend time and money on a study that results in a inconclusive results is not an appropriate use of resources. Staff believe the proposed course of action is prudent and responsible.	SFBK
V5		Implementation langauge still vague, lacks clarity, Staff need to clarify the implementation langauge to make the document stronger	Comment noted. See comment V2. Staff believe that the nessecary clarifications have been made.	SFBK
V6		Permitees should not be allowed to delay categorizing sites for cleaning up pollutions; document still lacks a scheme to prioritize sites for cleanup	The Draft Part 1 requires stressor identification because only after the stressor is identified can beneficial uses be effectively restored. If the stressor is no longer being discharged then some sort of remedial action may be appropriate. However if the stressor is being discharged, then any remedial action would only result in a short term benefit. In this situation, the ongoing discharges would continue to contribute the causative pollutants to the water body. Stressor identification reduces this risk and provides the Regional Boards with a better means to focus both cleanup actions and TMDLs on the pollutants causing problems.	SFBK
V7		The draft Part 1 should ensure that sites are prioritized for cleanup actions not just stressor ID	See response to comment V7	SFBK

No.	Subject	Comment	Response	Author
60 co			These changes are the result of the response of individual species to the presence of stressors with those most sensitive showing the greatest and earliest response. The benthic indices developed and validated for use as the metric for the benthic line of evidence are designed to track this response. Along a stress gradient within a given habitat type, the progressive reduction or loss of members of the benthic community, beginning with those species most sensitive, drives the index values, allowing that change to be quantified and rated. A site that is within the reference condition as defined by the benthic indices is one in which stressors have not detectably altered the assemblage of species expected for the habitat. This provides a standard to assure that the sensitive species within the assemblage are protected.	RB5

No.	Subject	Comment	Response	Author
1003	5.3.2	If narrative objectives are to be used to protect all benthic species, they need to clearly state the level of protection for all benthic species that simply provide for protection at the community level. As Dr. Schaffner stated in her peer review "protecting at the community level and protecting all species are different." To try to rectify the differences, state board staff have greatly exaggerated the sensitivity of the benthic metrics making unsubstantiated claims that are misleading at best.	The diversity and abundance of the benthic invertebrates that compose the benthic community varies from location to location based upon habitat factors such as dissolved oxygen, depth, salinity, grain size, hydrodynamics and available food and anthropogenic factors such as the toxic pollutants and nutrients. The presence and absence of individual species will vary as the natural conditions change from location to location. As a result, the utility of individual organisms as an indicator or as the receptor of concern is limited. However, a healthy benthic community will still exhibit distinct characteristics that differentiate it from a community that has been stressed in most cases regardless of the habitat. These characteristics include functionality, balance, presence of sensitive species, and limited abundance of highly tolerant species. As a result, staff have focused on the protection of benthic communities.	HBK
1003			In regards to exaggerated claims, it is difficult to respond to the commenter with out knowing what statements the commenter feels are in question. Staff have based the technical portions of the draft Staff Report and draft Part 1 on information and conclusions drawn from external scientific articles or documents prepared by state or federal agencies, or from studies and reports prepared by the science team during the course of the SQO development process. Staff do not feel that exaggerating or misleading claims were made in the staff report or draft Part 1.	HBK

No.	Subject	Comment	Response	Author
61	5.3.2	The Delta could have a lower level of protection for benthic organisms from toxicity than the rest of the water bodies in the Central Valley Region. A higher level of impact would be allowed if the level of protection were set at the community level as opposed to the organism or population level. Showing "toxicity to benthic communities" would be much more difficult than showing detrimental effects to sediment-associated aquatic life. Toxic effects could occur to organisms and species before such impacts were manifested at the community level. We recommend that the proposed aquatic life SQO be amended to replace the term "benthic communities" with "aquatic organisms". The relationship of the proposed narrative SQO to existing narrative objectives in the Central Valley Water Board's Basin Plans should be specified and changes in the level of protection resulting from the SQO Plan should be analyzed.	Staff disagree. The regulatory baseline information provided by Central Valley Region staff for preparation of Section 4 of the staff report does not support the commenters assertion. Furthermore, staff requested by email that the Regional Board provide some evidence for the record that would support this claim. To date, no response has been provided.	RB5
1105	5.4.1	Section 5.4.1: Support Staff Alternative 3 (Page 79)	Comment noted	TJ
1106	5.4.2	Section 5.4.2: Support New Alternative 5--combination of Alternative 3 and 4 (Page 80)	Comment noted	TJ
155	5.4 (5.4.2)	Numeric objectives would create a bright line test that would eliminate the confusion caused by the vague narrative objectives and muddled integration of multiple lines of evidence. Specific numeric objectives would create consistency among regional boards and consistency over time because inherently numeric objectives are clear, transparent, cautious and easy to use regardless of the approach. Moreover, numeric objectives eliminate the need to use MLOE that introduce more variability and less transparency	Staff agree that numeric objectives would be easier to implement; however, there is no single tool or measure that can be used at this time to assess sediment quality reliably and confidently. Without a single robust tool, numeric objectives are not possible.	SFBK
156	5.4 (5.4.2)	We have grave concerns with the use of narrative objectives. Coupled with the multiple lines of evidence (MLOE) assessment approach, they are an ineffective way to determine if sediments are contaminated and impaired	Staff disagree. A narrative objective coupled with indicators to interpret the narrative objectives represents a logical means to assess sediment quality.	SFBK

State Water Board's Program to Develop Sediment Quality Objectives

San Diego Regional Water Board
Workshop
August 10, 2005

Chris Beegan
cbeegan@waterboards.ca.gov
916 341 5577

Challenges

- Shifting away from a pollutant-specific concentration-based approach
- Integration with other programs that address sediment quality but have very different goals or objectives (dredged materials)
- Need to minimize BPJ

Program Goals

- Develop scientifically defensible SQOs that are protective of specific beneficial uses.
- Provide consistent sediment quality assessment tools and ecologically relevant thresholds throughout the state.
- Include numeric targets or thresholds that would be applied to each indicator
- Develop approach to integrate the multiple responses into a station quality value or quality category.
- Develop approach for assessing multiple stations.

Schedule

- By June 30 2003 adopt Workplan
- By August 5, 2005 circulate draft objectives and policy*
- By February 28, 2007 submit SQO policy to Office of Administrative Law.

Boundaries

- California Water Code – Bay Protection Chapter
  - Bays and Estuaries
- The program approach could be applied to all these waterbodies.
- However the MLOE indicators have only been developed for marine embayments
  - Enough existing data present to support development

Numeric versus Narrative SQOs

- Numeric Objectives
- Narrative Objectives

How Narrative SQOs Could Be Implemented

- **Example** of Narrative Objective for Direct Effects

Sediment quality shall be maintained at a level that protects benthic communities from degradation or toxicity do to exposure to bio-available pollutants in bottom sediments... This narrative shall be implemented using the multiple lines of evidence described in....

Programs

- Dredging - Effects of disposal versus in-place effects
- Non dredge-related programs
  - Waterbody Assessments
  - Conventional NPDES permits
  - Stormwater NPDES Permits
  - Nonpoint Source Programs
  - TMDLS
  - Cleanup Efforts

Sequential Approach

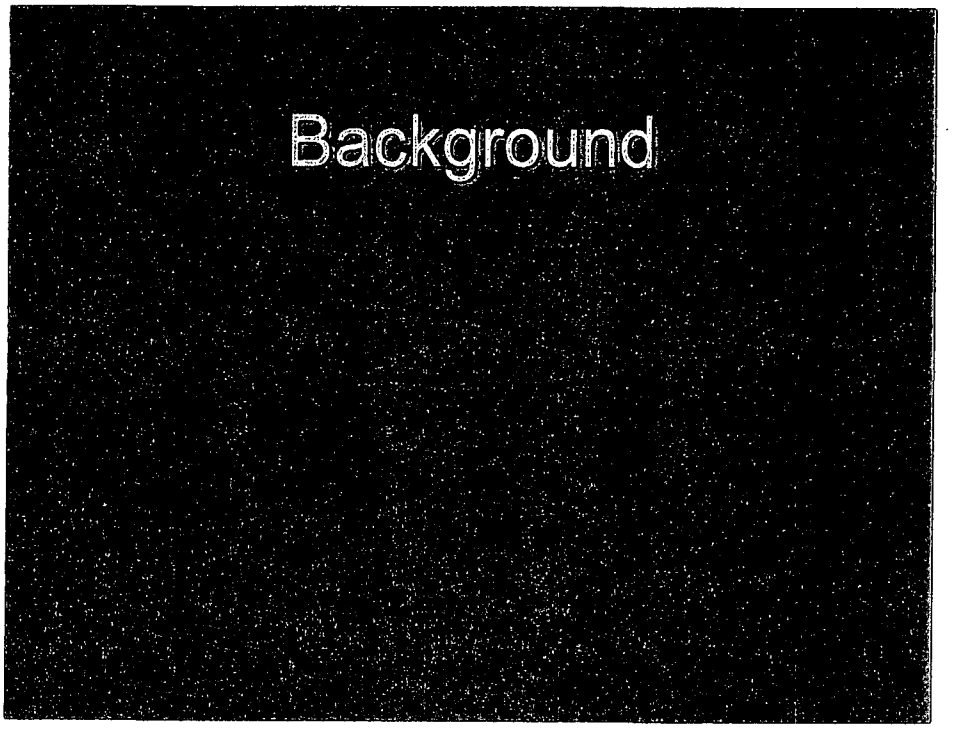
The general approach would consist of four steps

- Assessment - multiple sites
- Confirmation – What's the Stressor
  - Chemical Pollutant?
  - Which pollutant
- Source Identification and Loading
  - Source Identification
  - Load Evaluation,
  - Load Allocation
- Permits and Regulatory Tools
  - Options

Development of Sediment Quality Objectives for Enclosed Bays and Estuaries

San Diego Regional Board
November 14, 2007

Chris Beegan
State Water Resources Control Board
cbeegan@waterboards.ca.gov
Office 916 341 5577
Cell 916 662 2247



BPJ Best Professional Judgment

Standard:.

Legally: Water Code uses much of the same language for WQO as SQOs

Development: no established process for developing SQOs

Implementation: very different WQ regulatory programs focuses on control of effluents.....

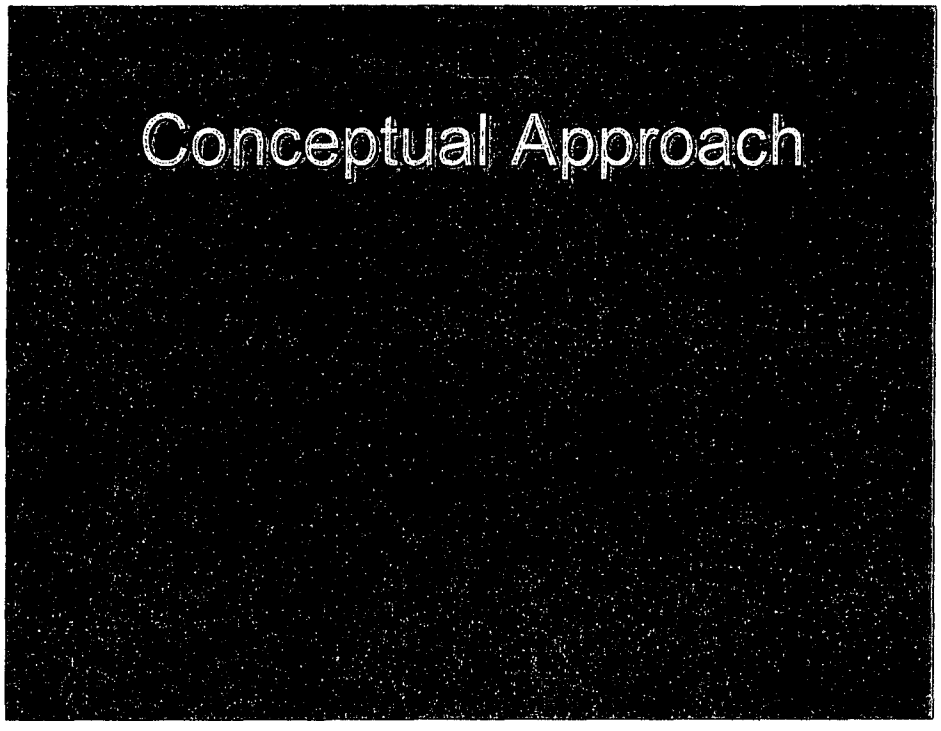
Two reasons this works:

1. Linkage to the pipe or plant.....and
2. dose response in water we know what will happen to organisms exposed to at or above specific levels

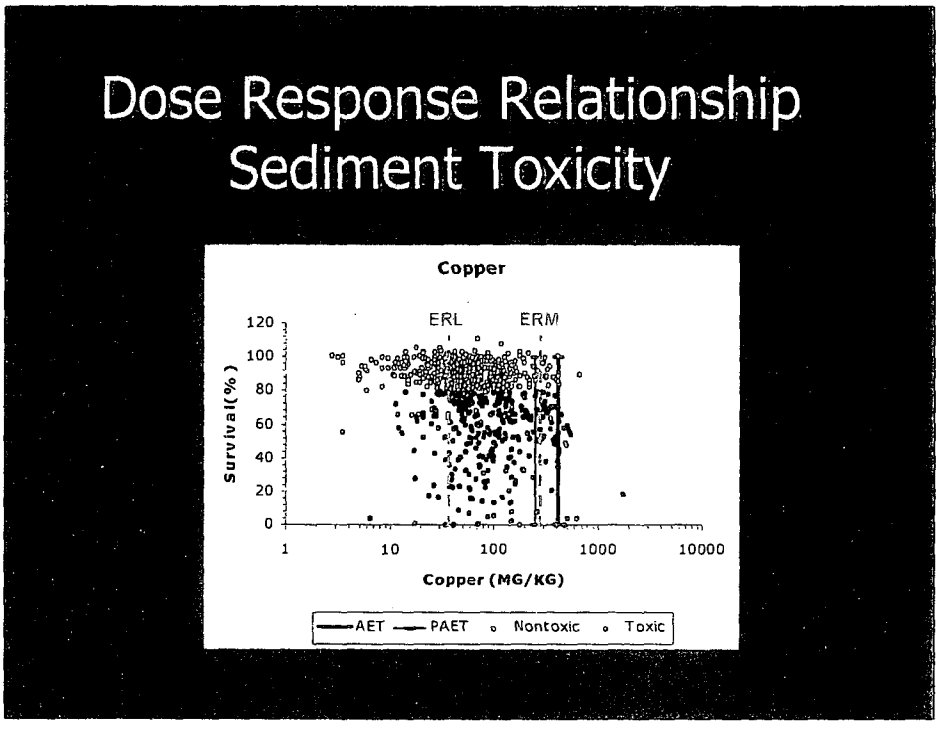
With Sediments we have neither.....

Sediments are in the receiving water.

Does response relationships not predictable because the bioavailability of pollutants in sediments is highly variable.....



BPJ Best Professional Judgment



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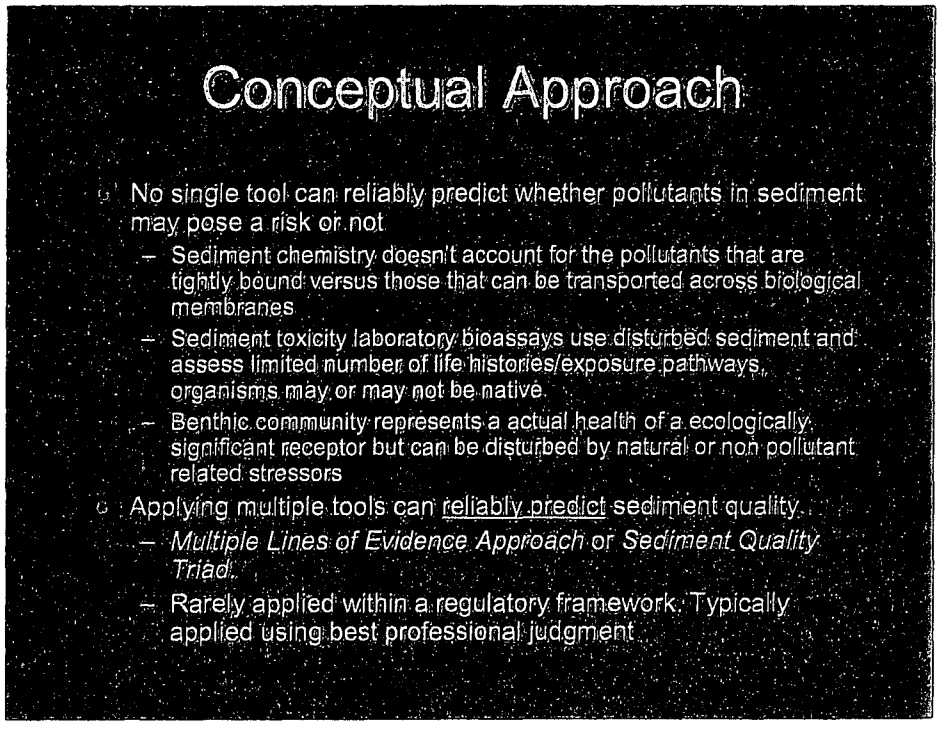


BPJ Best Professional Judgment

Draft Plan

Overview of Plan

- Narrative SQOs
- Interpreted using specific indicators and thresholds
- Implementation language describing;
  - Application of SQOs within specific programs
    - NPDES Permits
    - 303(d) listings (waterbody impairment)
    - dredging
  - Exceedence of SQOs
  - Response Actions
    - Stressor Identification
    - Biological based pollutant targets

Draft Plan

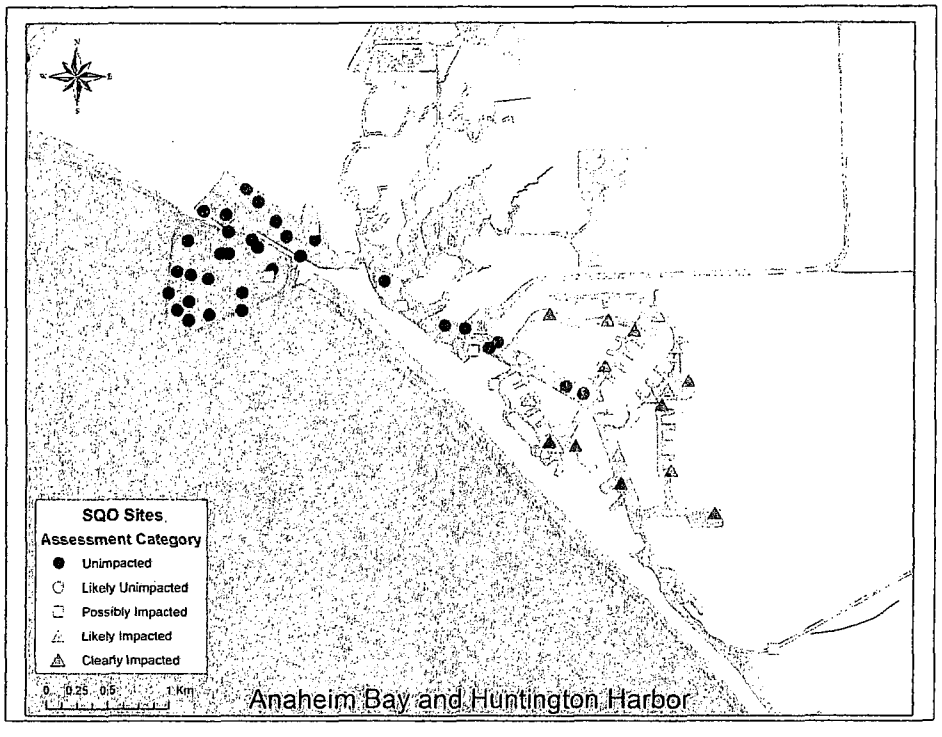
To protect benthic communities in bays and estuaries of California, the proposed plan describes

- MLOE Approach
  - Sediment Toxicity
  - Sediment Chemistry
  - Benthic Community Analysis
- Approach to integrate the MLOE into a station level assessment
- Appendix C Example Problem in draft Staff Report

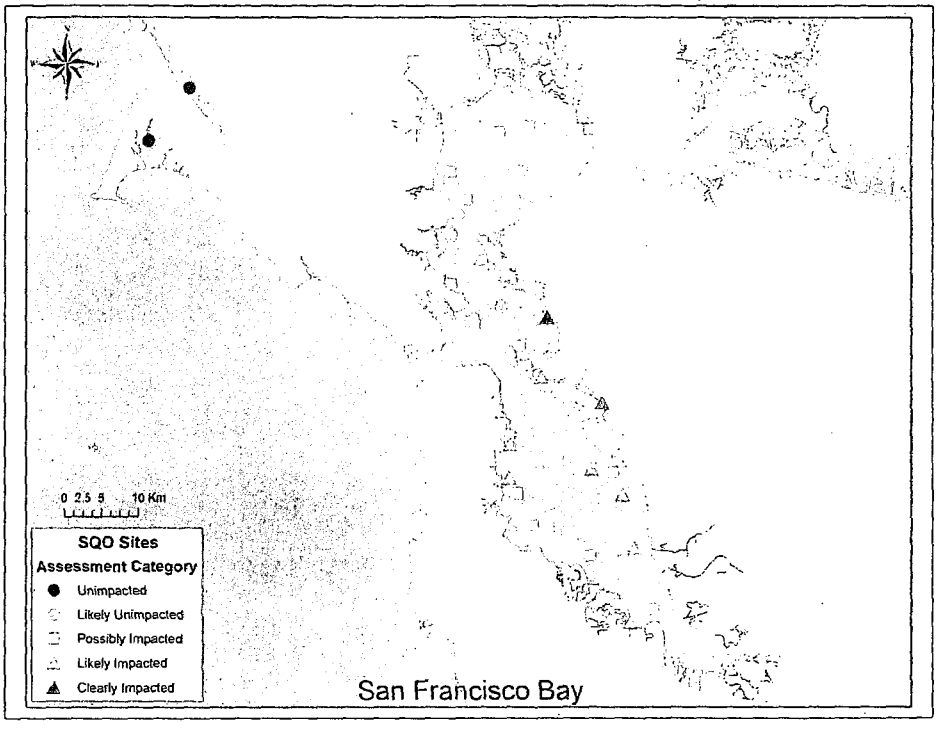
Draft Plan

Station Assessment categories

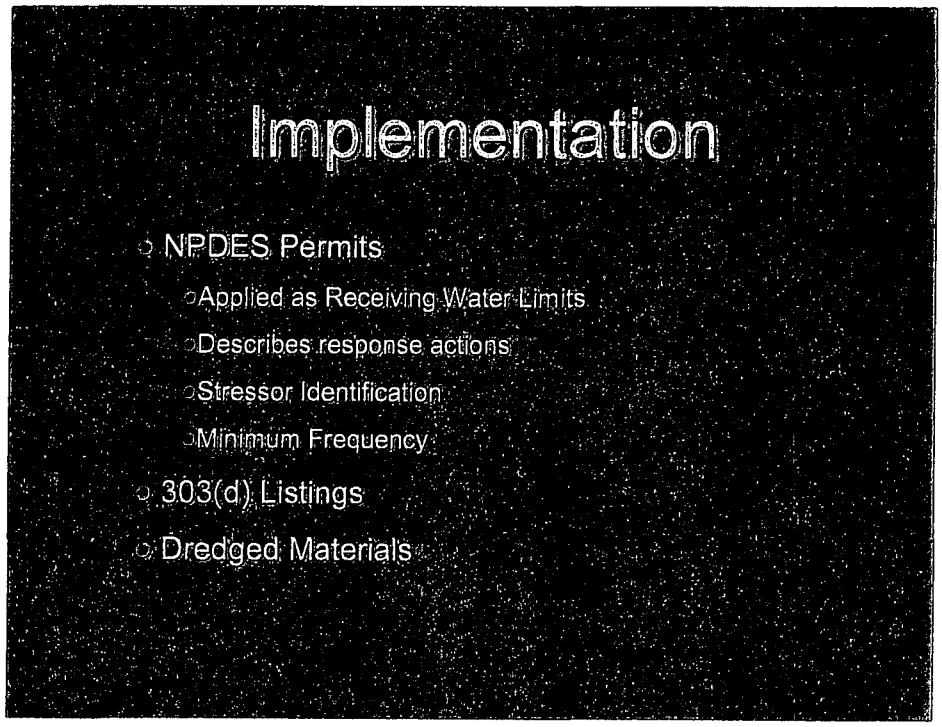
- Unimpacted
- Likely Unimpacted
- Possibly Impacted
- Likely Impacted
- Clearly Impacted
- Inconclusive



Data for Anaheim Bay and Huntington Harbor (Southern California) shows a gradient of greater response at inner harbor locations that is relatively consistent.



Most stations are possibly impacted, although greater impacts indicated near port/commercial areas.



BPJ Best Professional Judgment

More Information

- Web page
  - http://www.waterboards.ca.gov/bptcp/sediment.html
- Email/Phone
  - Chris Beegan cbeegan@waterboards.ca.gov 916 341 5577
  - Steve Bay Steveb@sccwrp.org, 714 372 9204

Files Describing Development of the State Water Boards Direct Effects SQO Multiple Lines of Evidence
October 5, 2010
Prepared by Chris Beegan

Documents presentations and meeting summaries describing the State Water Boards effort to develop sediment quality objectives are posted at:
http://www.waterboards.ca.gov/water_issues/programs/bptcp/sediment.shtml

State Board and Technical Team document inks are presented below: The documents prepared by the Technical Team provided the technical foundation for the State Water Boards approach described in the staff report.

State Board Documents

Staff Report – Water Quality Control Plan for Enclosed Bays and Estuaries Part 1 Sediment Quality
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/final_staffreport091808.pdf

Appendix E Comments and Responses
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/appndx_e091508_comments.pdf

Technical Team Documents

Sediment Toxicity

Technical Team Chronic Toxicity Methods Comparison: Preliminary Summary of Results. May 1, 2004.
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sqoindicator/chronictoxicitymethods.pdf

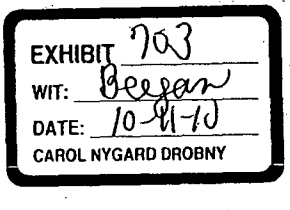
Technical Report 503 - Evaluation of Methods for Measuring Sediment Toxicity in California Bays and Estuaries, SCCWRP
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/503_toxicity_indicator_methods.pdf

Benthic Community

Technical Team Workplan for: Development of Benthic Community Condition Indicators. October 19, 2004.
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sqoindicator/benthicindicators.pdf

SCCWRP Technical Report 523 - Level of Agreement Among Experts Applying Best Professional Judgment to Asses the Condition of Benthic Infaunal Communities, SCCWRP
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/523_goldstandard_tr2.pdf

SCCWRP Technical Report 524 - Evaluation of Five Benthic Indicators of Benthic Community Condition in Two California Bay and Estuary habitats, SCCWRP



http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/524_eval_benthic_community_indicators3.pdf

Sediment Chemistry
Technical Team Workplan for: Development of Chemistry Indicators October 24, 2004.
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sqoindicator/chemistryindicators.pdf

Comparison of National and Regional Sediment Quality Guidelines for Predicting Sediment Toxicity in California, SCCWRP Final Report
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/comparison_guide_sedtoxicity.pdf

Development and Evaluation of Sediment Quality Guidelines Based on Benthic Macrofauna Response, SCCWRP Final Report
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/deveval_guide_benthicmacrofauna.pdf

Multiple Lines of Evidence
Technical Team Work Plan for: Incorporating Multiple Lines of Evidence into Sediment Quality Objectives
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sqoindicator/mloeworkplan.pdf

Evaluating the Consistency of Best Professional Judgment in the Application of a Multiple Line of Evidence Sediment Quality Triad
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/sccwrp_tech_report.pdf

Framework for Interpreting Sediment Quality Triad Data, SCCWRP Final Report
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/framework4interpreting_sedqual.pdf

Technical Report 522 Sediment Quality in Bays and Estuaries of California
http://www.waterboards.ca.gov/water_issues/programs/bptcp/docs/sediment/sedimentqual_baysestuaries.pdf

Scientific Steering Committee Meeting Presentations
Presentations to the SQO Scientific Steering Committee describing progress on the development of the Sediment Toxicity, Sediment Chemistry, Benthic Community and Multiple Line of Evidence Approach are posted at:
http://www.waterboards.ca.gov/water_issues/programs/bptcp/sqoscientific.shtml

SQO Advisory Committee Presentations
http://www.waterboards.ca.gov/water_issues/programs/bptcp/sqoac.shtml

SQO Database
Link to the SQO Database compiled and used to develop sediment toxicity, sediment chemistry, benthic community indicators:

http://www.sccwrp.org/ResearchAreas/Contaminants/SedimentQualityAssessment/DirectEffectsInBays.aspx

STAFF REPORT

WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY

September 16, 2008

State Water Resources Control Board

California Environmental Protection Agency

State of California
Arnold Schwarzenegger, Governor

California Environmental Protection Agency
Linda S. Adams, Secretary

State Water Resources Control Board
http://www.waterboards.ca.gov

Charles R. Hoppin, Chair

Francis Spivy-Weber, Vice Chair

Tam M. Doduc, Member

Arthur G. Baggett, Jr., Member

Dorothy Rice, Executive Director

Jonathan Bishop, Chief Deputy Director

Thomas Howard, Chief Deputy Director

History of the Water Quality Control Plan for Enclosed Bays and Esutaries – Part 1 Sediment Quality
Adopted by the State Water Resources Control Board on September 16, 2008 (Resolution 2008-0070)
Approved by the Office of Administrative Law on January 5, 2009
Approved by the U. S. Environmental Protection Agency on August 25, 2009

Prepared by
Chris Beegan, Ocean Unit, Division of Water Quality

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

TABLE OF CONTENTS (Continued)

APPENDICES

LIST OF TABLES

LIST OF TABLES (Continued)

LIST OF FIGURES

LIST OF ACRONYMS AND ABBREVIATIONS

AET	Apparent Effects Threshold
AVS	Acid Volatile Sulfides
BAT	best available technology economically achievable
BCT	best conventional pollutant control technology
BLM	U.S. Bureau of Land Management
BSAF	Biota-sediment bioaccumulation factor
BPTCP	Bay Protection and Toxic Cleanup Program
BRI	Benthic Response Index
CAA	Cleanup and Abatement Account
CAC	County Agricultural Commissioners
CAP	Corrective Action Plan
Cal/EPA	California Environmental Protection Agency
CCC	California Coastal Commission
CDF	California Department of Forestry
CEQA	California Environmental Quality Act
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CTR	California Toxics Rule
CWA	Federal Clean Water Act
CWC	California Water Code
DPR	Department of Pesticide Regulation (Cal/EPA)
DOC	Department of Conservation
DTSC	Department of Toxic Substances Control (Cal/EPA
EqP	Equilibrium Partitioning
ESA	Endangered Species Act
ESG	Equilibrium-Partitioning Sediment Guideline
EMAP	Environmental Monitoring and Assessment Program
ERL	Effects Range Low
ERM	Effects Range Median
FED	Functional Equivalent Document
GHG	Greenhouse Gas
ITM	Inland Testing Manual
LA CSTF	Los Angeles Contaminated Sediments Task Force
MEP	Maximum Extent Practical
MMs	Management Measures
NMFS	National Marine Fisheries Service
NOAA	National Oceanic and Atmospheric Administration
NOA	Notice of Applicability
NOI	Notice of Intent
NPDES	National Pollutant Discharge Elimination System
NPS	Nonpoint Source
NSI	National Sediment Inventory
OPA	Federal Oil Pollution Act
OTM	Ocean Testing Manual
PAHs	Polyaromatic Hydrocarbons
PCBs	Polychlorinated Biphenyls
PEC	Probable effect concentration (consensus-based)
PEC-Q	Probable effect concentration quotient
PEL	Probable effect level
POTW	Publicly Owned Treatment Works

LIST OF ACRONYMS AND ABBREVIATIONS (Continued)

RCRA	Resource Conservation and Recovery Act
RWQCBs	Regional Water Quality Control Boards (CalEPA)
RMP	Regional Monitoring Program
SARA	Superfund Amendments and Reauthorization Act
SCCWRP	Southern California Coastal Water Research Project
SEM	Simultaneously Extracted Metals
SIP	Policy for the Implementation of Toxic Standards for Inland Surface Waters
SMARA	Surface Mining and Reclamation Act
SQG	Sediment Quality Guidelines
SQO	Sediment Quality Objective
SSC	Scientific Steering Committee
SWPPP	Storm Water Pollution Prevention Plan
SWRCB	State Water Resources Control Board (Cal/EPA)
THP	Timber Harvest Plan
TIE	Toxicity Identification Evaluation
TMDL	Total Maximum Daily Load
TMDL	Total Maximum Daily Loads
TOC	Total Organic Carbon
TRA	Tissue Residue Approach
TRG	Tissue residue guideline
TRV	Toxicity reference values
TVS	Total Volatile Sulfides
USACE	U.S. Army Corps of Engineers
U.S. EPA	U.S. Environmental Protection Agency
USFS	U.S. Forestry Service
USF&WS	U.S. Fish and Wildlife Service
USGS	United States Geological Survey
WDR	Water Discharge Requirements

1. INTRODUCTION

1.1 PURPOSE

This report represents the State Water Resources Control Board (State Water Board) formal water quality planning and substitute environmental document for the adoption of sediment quality objectives (SQOs) and program of implementation that would apply to enclosed bays and estuaries of California. The title of the proposed plan where the SQOs and policy of implementation would reside is *Water Quality Control Plan for Enclosed Bays and Estuaries of California, Part 1 Sediment Quality* (Part 1). SQOs would provide the State and Regional Water Quality Control Boards stakeholders and interested parties with a technically robust mechanism to differentiate sediments impacted by toxic pollutants from those that are not consistently through out the coastal regions. The proposed SQOs developed through this program do not address excessive sediment loading (siltation or sedimentation) related impairment or degradation.

Sediments in enclosed bays and estuaries are, with few exceptions, the most highly polluted sediments in the State. Historically, areas adjacent to bays and estuaries were the first heavily industrialized regions in the State; and, as a result, wastes have been discharged into bays either directly as point sources, indirectly as runoff, or accidentally through releases and spills for many years. Sediment carried down rivers and creeks also contributes to the contaminant loading into bays and estuaries. Many contaminants, such as metals and pesticides, readily attach to the sediments. Through this mechanism, contaminants from inland sources can be transported long distances. Poor flushing and low current speeds allow the sediments and contaminants to settle out in the bays and estuaries before reaching the open ocean. Few states have attempted to develop SQOs due to the lack of ecologically relevant tools, difficulties interpreting and integrating the results, and an inability to establish causality. In 2003, the State Water Board initiated a program to protect these water bodies through the development of SQOs for enclosed bays and estuaries.

1.2 MANDATE TO DEVELOP SQOS

In 1989, the Legislature added chapter 5.6 to Division 7 of the California Water Code. The legislation required the State Water Board to develop sediment quality objectives as part of a comprehensive program to protect beneficial uses in enclosed bays and estuaries. The objectives are required "for toxic pollutants" that were identified in toxic hot spots or that were identified as pollutants of concern by the State Water Board or the Regional Water Quality Control Boards (Regional Water Boards).[1] The waters targeted for protection are enclosed bays and estuaries.

The Legislature defined a "sediment quality objective" (SQO) as "that level of a constituent in sediment which is established with an adequate margin of safety, for the reasonable protection of the beneficial uses of water or the prevention of nuisance."[2] The SQOs have to "be based on scientific information, including, but not limited to, chemical monitoring, bioassays, or established modeling procedures."[3] They must "provide adequate protection for the most

[1] See Wat. Code sec. 13392.6.
[2] *Id.* sec. 13391.5.
[3] *Id.* sec. 13393.

sensitive aquatic organisms."[4] The State Water Board is not precluded from adopting SQOs for a pollutant even though additional research may be needed.[5]

In addition, if there is a potential for human exposure to pollutants through the food chain, the State Water Board must base SQOs on a health risk assessment.[6] A health risk assessment is an analysis that evaluates and quantifies the potential human exposure to a pollutant that bioaccumulates in edible fish, shellfish, or wildlife.[7] A health risk assessment "includes an analysis of both individual and population wide health risks associated with anticipated levels of human exposure, including potential synergistic effects of toxic pollutants and impacts on sensitive populations."[8]

The Legislature required the State Water Board to develop a workplan by July 1991 for the adoption of SQOs and to adopt the SQOs pursuant to the workplan.[9] In 1991, the State Water Board developed a seven year conceptual approach, which is described in the Workplan for the Development of Sediment Quality Objectives for Enclosed Bays and Estuaries of California (91-14 WQ) (1991 Workplan).

This 1991 Workplan included a schedule and specific tasks to develop direct effects tools that would protect benthic communities and an element to assess the human and ecological risk in bays and estuaries from pollutants in sediments. This conceptual approach embodied in the workplan was never implemented because available resources were primarily focused on identifying toxic hot spots using multiple lines of evidence.

In 1999, a lawsuit was filed against the State Water Board for failing, among other things, to adopt SQOs. The Court sided with the petitioners and ordered the State Water Board to develop SQOs and implementation measures. The Court also required the State Water Board to prepare a revised workplan. The draft revised workplan was circulated for public comment and adopted by the State Water Board on May 21, 2003. The targeted receptors, proposed objectives and indicators described in this staff report are based upon the technical elements described in that workplan.

1.3 Scientific Peer Review

In 1997, Section 57004 was added to the California Health and Safety Code. Section 57004 requires external scientific peer review of the scientific basis for any rule proposed by any board, office, or department within California Environmental Protection Agency (Cal/EPA). Scientific peer review ensures that public resources are managed effectively. Scientific peer review was requested through a contract with the University of California at Berkeley in November 2008. The following scientists agreed to review the technical issues associated with the staff report and Part 1:

- Dr. Dominic Di Toro, Edward C. Davis Professor of Civil and Environmental Engineering Department of Civil and Environmental Engineering, University of Delaware
- Dr. John P. Knezovich, PhD, Director, Center for Accelerator Mass Spectrometry, L-397 Lawrence Livermore National Laboratory

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[4] *Ibid.*
[5] See *id.* sec. 13392.6.
[6] *Id.* sec. 13393.
[7] *Id.* sec. 13391.5(c).
[8] *Ibid.*
[9] *Id.* secs. 13392.6, 13393.

- Dr. Linda C. Schaffner, Professor Department of Biological Sciences, School of Marine Science Virginia Institute of Marine Science The College of William and Mary
- Dr. David L. Sedlak, Professor, Environmental Engineering Program Department of Civil and Environmental Engineering, University of California at Berkeley

Peer reviews are posted at http://www.waterboards.ca.gov/bptcp/sediment.html. Responses to peer review comments are presented as Appendix XXX

1.4 Advisory and Scientific Committees

Advisory Committees

The 1989 amendments to the Water Code required the State Water Board to form an advisory committee to assist in the implementation of chapter 5.6. State Water Board staff invited stakeholders and interested parties to participate in this committee, which was intended to focus on SQOs development and implementation within bays. The organizational meeting for this committee was held on July 29, 2003. A second advisory committee was formed on April 13, 2006 to advise the State Water Board on issues associated with the development and implementation of SQOs within the Sacramento-San Joaquin Delta and other estuarine waters in the State. Dr. Brock Bernstein serves as Chairperson and facilitator on both committees.

Scientific Steering Committee

The Scientific Steering Committee (SSC) was formed for the purpose of independently assessing the soundness and adequacy of the technical approach and ensuring that all findings and conclusions are well supported. The SSC provided the State Water Board's technical team with a very high level of expertise and experience from around the nation. The members on this committee are:

- Dr. Peter Landrum, Committee Chair: .Research Chemist NOAA/Great Lakes Environmental Research Laboratory Ann Arbor, MI
- Ed Long, Former NOAA Scientist and developer of empirically derived sediment quality guidelines for NOAA's Status and Trends Program.
- Tom Gries, Environmental Scientist Washington Dept. of Ecology, Sediment Management Section, Olympia, WA
- Dr. Todd Bridges, Research Biologist and Director of the Center for Contaminated Sediments, Waterways Experiment Station (WES) U.S. Army Corps of Engineers, ERDC, Vicksburg, MS
- Dr. Robert F. Van Dolah, Benthic Ecologist and Director of the South Carolina Marine Resources Research Institute.
- Dr. Robert Burgess, Research Scientist, EPA's Office of Research and Development (Atlantic Ecology Division-Narragansett)

Agency Coordination Committee

The Agency Coordination Committee is an informal committee composed of staff from agencies that assess, regulate or manage contaminated sediments. Participants include staff from the coastal Regional Water Boards, Department of Toxic Substances Control, Department of Fish and Game, U.S EPA, and U.S Fish and Wildlife Service. The role of this committee was

to assist Water Board staff in the integration of other programs and policies related to sediment quality and identify potential areas of conflict.

1.5 CEQA Analysis and Impact of the Proposed Policy

When developing water quality objectives and water quality control plans, the State Water Board must comply with the California Environmental Quality Act (CEQA), Public Resources Code §21000 et seq. The objectives of CEQA are to: 1) inform the decision makers and public about the potential significant environmental effects of a proposed project, 2) identify ways that environmental damage may be mitigated, 3) prevent significant, avoidable damage to the environment by requiring changes in projects, through the use of alternatives or mitigation measures when feasible, and 4) disclose to the public why an agency approved a project if significant effects are involved. (Cal. Code Regs., tit. 14, § 15002(a).)

Although state agencies are subject to the environmental impact assessment requirements of CEQA, CEQA authorizes the Secretary of the Resources Agency to exempt specific state regulatory programs from the requirements to prepare Environmental Impact Reports (EIRs), Negative Declarations, and Initial Studies, if certain conditions are met (Public Resources Code, §21080.5). The water quality control (basin)/208 planning program of the State Water Board has been certified by the Secretary for Resources as meeting the requirements for exemption (California Code of Regulations (CCR), title 14, §15251(g)). Agencies qualifying for this exemption must comply with CEQA's goals and policies; evaluate environmental impacts; consider cumulative impacts; consult with other agencies with jurisdiction; provide public notice and allow public review; respond to comments on the environmental document; adopt CEQA findings; and provide for monitoring of mitigation measures. State Water Board regulations (CCR, tit. 23, §3777) require that a document prepared under its certified regulatory programs include:

- A brief description of the proposed project;
- Reasonable alternatives to the proposed project; and
- Mitigation measures to minimize any significant adverse environmental impacts of the proposed activity.

Accordingly, the State Water Board prepares programmatic substitute environmental documents (SEDs) in lieu of EIRs or other environmental documents when proposing statewide water quality objectives and a program of implementation. This Staff Report fulfills these requirements of a substitute environmental document. Until recently, the State Water Board referred to these formal planning documents as functional equivalent documents. There is no substantive difference between these documents.

Responses to comments and consequent revisions to the information in the Draft Staff Report are subsequently presented in a draft Final Staff Report for consideration by the State Water Board. After the State Water Board has certified the document as adequate, the title of the document becomes the Final Staff Report.

1.6 Compliance with CWC Sections 13241 and 13242

Chapter 5.6 requires that the State Water Board adopt sediment quality objectives in accordance with the procedures proscribed in the Water Code for adopting and amending water quality control plans. The procedures include notice and a public hearing prior to plan adoption. In addition, Section 13241 of the Water Code requires that the Water Boards consider specified

factors when they establish water quality objectives to ensure the reasonable protection of beneficial uses. These factors include:

(a) Past, present, and probable future beneficial uses of water.

(b) Environmental characteristics of the hydrographic unit under consideration.

(c) Water quality conditions that could reasonably be achieved through control of all factors affecting water quality.

(d) Economic considerations.

(e) The need for developing housing within the region.

(f) The need to develop and use recycled water.

Water Code section 13242 requires that the Water Boards formulate a program of implementation for the water quality objective under consideration by the Board. The program of implementation for achieving water quality objectives must include, at least:

(a) A description of the nature of actions that is necessary to achieve the objectives, including recommendations for appropriate action by any entity, public or private.

(b) A time schedule for the actions to be taken.

(c) A description of surveillance to be undertaken to determine compliance with objectives

1.7 Authors and Contributors

Mr. Chris Beegan from the Division of Water Quality – Ocean Unit prepared this staff report and Part 1. Principal Scientist Mr. Steve Bay, Mr. Ana Ranasinghe, Dr. Kerry Ritter, Dr. Art Barnett and Dr. Steve Weisberg with the Southern California Coastal Water Research Project provided the technical analysis and studies in support of the proposed SQP. Drs. Mike Connor and Bruce Thompson and Mr. Ben Greenfield at San Francisco Estuary Institute also contributed technical analysis and studies for this program. Mr. Dominic Gregorio and Mr. Craig J. Wilson from the Division of Water Quality and Ms. Sheila Vassey from the Office of Chief Counsel provided valuable input during the preparation of this document. Ms. Eloise Castillo and Ms Lauren Praesel from Science Application International Corporation (SAIC) prepared the economic analysis of the Part 1.

1.8 Proposed Project and Description

The State Water Board is proposing the following project: the adoption of a Water Quality Control Plan for Enclosed Bays and Estuaries of California, Part I Sediment Quality (Part 1)." Part 1 contains narrative SQOs indicators and threshold used to interpret the narrative objectives and a program of implementation. Part 1 if adopted would be applicable to all enclosed bays and estuaries of California.

Enclosed bays are defined in Water Code section 13391.5 as:

> *indentations along the coast which enclose an area of oceanic water within distinct headlands or harbor works. Enclosed bays include all bays where the narrowest distance between headlands or outermost harbor works is less than 75 percent of the greatest dimension of the enclosed portion of the bay. This definition includes, but is not limited to: Humboldt Bay, Bodega Harbor, Tomales Bay, Drakes Estero, San Francisco Bay, Morro Bay, Los Angeles Harbor, Upper and Lower Newport Bay, Mission Bay, and San Diego Bay.*

This section defines estuaries as:

> *waters at the mouths of streams that serve as mixing zones for fresh and ocean waters during a major portion of the year. Mouths of streams that are temporarily separated from the ocean by sandbars shall be considered as estuaries. Estuarine waters will generally be considered to extend from a bay or the open ocean to the upstream limit of tidal action but may be considered to extend seaward if significant mixing of fresh and salt water occurs in the open coastal waters. The waters described by this definition include, but are not limited to, the Sacramento-San Joaquin Delta as defined by Section 12220 of CWC, Suisun Bay, Carquinez Strait downstream to Carquinez Bridge, and appropriate areas of the Smith, Klamath, Mad, Eel, Noyo, and Russian Rivers.*

If adopted, the regulatory provisions of Part 1 would be enforced by the State Water Board and coastal Regional Water Boards, consisting of the North Coast, San Francisco Bay, Central Coast, Los Angeles, Central Valley, Santa Ana and San Diego Regional Water Boards.

Those regulated under Part 1 would include individual or organization that discharges toxic pollutants to enclosed bays and estuaries of California or rivers or streams draining into enclosed bays and estuaries.

1.9 Statement of Goals

The Water Code defines a sediment quality objective as that level of a constituent in sediment established with an adequate margin of safety for the reasonable protection of beneficial uses or prevention of nuisances. The Water Code does not define the term "reasonable"; however, the American Heritage Dictionary defines the term as governed by or in accordance with reason or sound thinking, within the bounds of common sense, not excessive or extreme; fair moderate (American Heritage Dictionary of English Language, New College Edition 1976).

The objective of this program since 2002 has been to develop SQOs and robust indicators in conjunction with a program of implementation that protects two beneficial uses, aquatic life and human health. The goals of this program are to:

- Establish narrative receptor-specific SQOs.
- Establish a condition that is considered protective for each targeted receptor.
- Identify appropriate lines of evidence for each receptor that when integrated can support a confident interpretation of the narrative objective.
- Develop and/or refine and validate specific indicators for each line of evidence so that the condition of each station can be measured relative to the protected condition.
- Build a program of implementation based upon these tools and the current level of scientific understanding to promote the protection of sediment quality related beneficial uses.
- Define a process that will result in better management and more effective restoration of polluted sediments

Staff believes the approach developed to assess aquatic life via benthic communities for Southern California's enclosed bays and marine lagoons and polyhaline San Francisco Bay has met these goals. For other bays on the central and north coast such as Morro Bay, Humboldt Bay, Tomales Bay, and all estuaries including the Sacramento-San Joaquin Delta, the lack of

available data prevented the staff and technical team from achieving these goals. In response, State Water Board staff have proposed a less robust means to determine if sediment quality is meeting the narrative aquatic life – benthic community SQO in these waters. However, State Water Board staff believe that work conducted in the next phase will provide superior indicators, which could replace these tools if adopted and be comparable to those developed for Southern California Bay and polyhaline San Francisco Bay in Phase II of the SQO program.

Although extensive progress was also made on developing an approach to interpret the human health-based narrative objective, Staff are proposing in this first phase to use existing site-specific human health risk methodology to interpret the narrative. As State Water Board staff stated in the May 2003 Workplan, developing sediment quality objectives that protect human health from consumption of contaminated fish is extremely complex for several reasons.

- The fate and transport of pollutants from sediment to tissue and the water column pollutants is highly site specific.
- Indirect exposure to pollutants from sediments transported up the food web is difficult to relate directly to specific sites or stations of area of a waterbody.
- The home range, habitat, feeding strategies, and lipid content of each fish species may vary seasonally and as the fish matures, all of which affects the rate of contaminant accumulation in the tissue.
- The type and size of prey-fish targeted by sport-fisherman and subsistence fisherman also varies considerably as do the methods of preparation, types of tissue consumed and consumption rates.

A more detailed approach to support the human health based SQOs will require greater time and effort. Staff expects this effort to be completed in the next phase, which would trigger a new proposed methodology for State Water Board consideration.

1.10 Document Organization

This document is organized as follows. A conceptual model describing the fate and transport of pollutants in sediments, potentially affected receptors and exposure mechanisms is described in Section 2. Section 3 describes the environmental setting of the coastal and estuarine Regional Water Board basins. The regulatory baseline is described in Section 4. Issues and Alternatives evaluated during the formulation of Part 1 are discussed in Section 5. Section 6 describes the CEQA analysis and Water Code section 13241 factors. Part 1 is presented in Appendix A. The CEQA Checklist is included in Appendix B. Appendix C presents the application of a data set assessed by applying the indicators and appropriate thresholds included in Part 1. Summary Maps of Toxic Hot Spots are presented by Region in Appendix D.

Comments on the staff report received by the State Water Board and staff's responses are presented in Appendix E.

2. CONCEPTUAL MODEL FOR SEDIMENT QUALITY

Sediment is a complex and dynamic environment, which can influence the fate and effects of the contaminants it contains. Sediment particles can vary from coarse sand with a diameter of about 1 millimeter (mm) to fine silts and clays with diameters less than 0.01 mm. Variations in the size and composition of these particles have an effect on the binding of contaminants to them, with the finer particles generally containing higher contaminant concentrations due to a much greater surface area and greater number of chemical sorption sites.

The assessment of sediment quality in bays and estuaries relies on information regarding the sources, fates and effects of contaminants of concern. The types of sources determine the overall magnitude, and spatial and temporal patterns of contaminant input to these nearshore environments. Contaminants in the receiving water environment are influenced by many processes that ultimately determine the type and amount of contaminant exposure to organisms. There are many gaps in our knowledge of contaminant sources and fate. Consequently, measurement of biological effects is often needed to determine the ecological significance of chemical measurements.

Multiple sources contribute to sediment contamination in embayments (Figure 2.1). Runoff and discharge from rivers, creeks, and drainage channels that carry storm water and dry weather runoff from the upland watershed are major nonpoint contaminant sources. Contaminants may also come from point source discharges, such as municipal wastewater and industrial discharges that are located within embayments, as well as spills. Additional nonpoint contaminant sources include atmospheric deposition and groundwater. A large portion of the contaminants from most of these sources may be associated with particles, either as suspended particles in the discharge or receiving water body. However, each of these discharges influences water and sediment quality on different spatial and temporal scales. This diversity of sources, combined with various physical mixing processes such as currents, tidal exchange, and ship traffic, can produce complex and widespread patterns of sediment contamination.

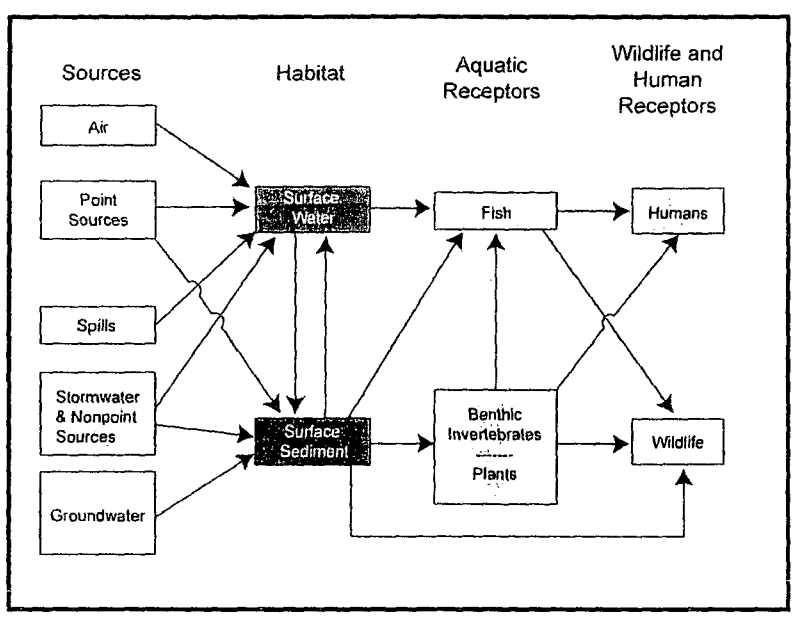


Figure 2.1. Principal Sources, Fates, and Effects of Sediment Contaminants in Enclosed Bays and Estuaries (Adapted from Brides et al. 2005)

There are a number of processes occurring in embayments that affect the fate and distribution of sediment contaminants (Figure 2.1). Upon introduction into the water body, dissolved contaminants in the source may bind to suspended particles in the water column or particle associated contaminants may desorb back into the water column. In brackish embayments in particular, flocculation and aggregation of small-suspended particles into large agglomerates that then settle out of the water column is a primary mechanism for introduction of contaminants to surface sediments. Where river or tidal currents are present, some contaminants will be transported (advected) out of the system. The fraction that remains and eventually settles forms the sediment's surface, a layer (5-20 centimeters (cm)) of high physical, chemical, and biological activity. Most of the benthic infauna resides in this surface layer. The layer of sediment below is less active and contaminants that are contained in this layer generally exert little influence on organisms. However, contaminants in the deep sediment layer can affect habitat quality if they are transported to the surface by deep burrowing organisms, transformed into different chemical species under anaerobic conditions, or resuspended by physical processes such as sediment erosion or dredging.

Sediment contaminants in the surface layer are not static, their concentration, distribution, and chemical form are being continually modified. For example, particle bound contaminants can move into the water column by diffusion (desorption from particles), resuspension, or from the burrowing and feeding activities of many benthic organisms (bioturbation).

The form and biological availability of contaminants is influenced by many factors in the sediment. The sediment particles contain variable amounts and types of organic carbon, including natural plant or animal detritus, microbial films, and anthropogenic materials such as ash, soot, wood chips, oils, and tars. The partitioning of many contaminants between sediment particles, water, and biota is strongly influenced by the nature of sediment organic carbon (Figure 2.2). The predominant forms for metals (or speciation) are largely governed by the reduction-oxidation (redox) potential (or E_h) and the co-occurrence of binding constituents such as sulfides, organic material, metal oxides, and clay minerals. Although the general mechanisms affecting partitioning and speciation of contaminants are known, it is often difficult to predict such changes from chemical measurements with sufficient accuracy to determine their bioavailability, which in turn is key for assessing biological effects.

Microbial activities also influence the characteristics of sediment contaminants. The microbial degradation of sediment organic matter can alter the pH and oxygen content of sediments, which may in turn affect the rates of metal desorption/precipitation. Bacterial metabolism or chemical processes can also transform or degrade some contaminants to other forms. In some cases, the transformation product may have greater biological availability or toxicity, such as methyl mercury. In other cases, such as for some pesticides, degradation may alter the contaminant so that it is no longer toxic.

California's bays and estuaries are home to a tremendous diversity of life. As such, there are multiple routes by which these organisms can be exposed to and affected by sediment contaminants. There are two general types of contaminant exposure: direct and indirect. Most of the direct exposure results from the contact of organisms with the sediment and sediment ingestion. Organisms living in the sediment are exposed through the uptake of contaminants from the pore water, which is the water associated with the sediment particles. This process is analogous to the exposure of water column organisms from dissolved contaminants. Organisms that ingest sediments may accumulate contaminants that are desorbed by digestive processes in the gut. Indirect contaminant exposure results from the consumption of contaminated prey. Examples include fish feeding on benthic invertebrates, birds feeding on benthic invertebrates or fish, and humans consuming fish (Figure 2.1).

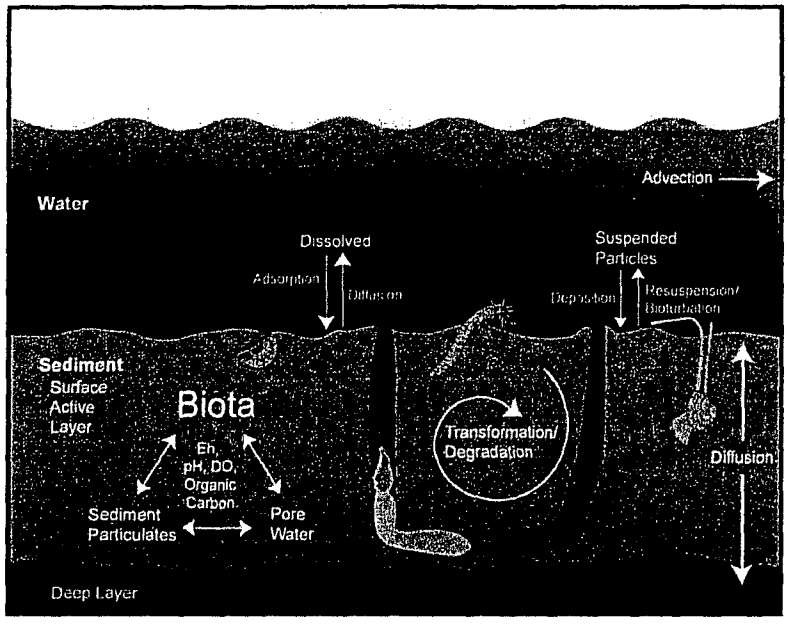


Figure 2.2. Sediment Processes Affecting the Distribution and Form of Contaminants

Benthic organisms are generally at greatest risk for adverse effects from direct sediment contaminant exposure, because these organisms often live in continual direct contact with sediment/pore water, and many species ingest significant quantities of sediment as a source of nutrition. The relative importance of sediment ingestion vs. sediment contact for contaminant exposure varies depending upon the life history of the species. In addition, there are species-specific differences in contaminant uptake rates and metabolism that affect the amount of contaminant (or dose) accumulated by benthic organisms. As a result, benthic species vary in their sensitivity to sediment contamination. This in turn produces a gradation of benthic community composition change that corresponds to the magnitude of contaminant exposure.

A variety of biological methods are needed to assess the direct effects of sediment contamination. Measurement of changes in the benthic community, such as abundance and species composition, are a sensitive measure of the direct effects of sediment contamination because these organisms live in the surface sediment layer. However, variations in sediment composition complicate this assessment because benthic organisms often have specific preferences or tolerances for variations in sediment grain size and organic content, in addition to other environmental factors such as water depth, salinity, and temperature. Consequently, the benthic community present at a site may be altered by a variety of environmental factors in addition to adverse effects from contaminants. It is necessary to understand how these environmental factors affect benthic communities before the effects of contaminants can be discerned.

Laboratory toxicity tests are also useful for assessing the direct effects of sediment. These tests measure the lethal or sublethal response of a test species exposed to the sediment under controlled conditions. Toxicity tests provide a measure of the bioavailability and toxicity of sediment contaminants from direct exposure and are not affected by many of the environmental factors that confound benthic community analyses or other measurements of effect in the field.

The magnitude of indirect contaminant exposure is affected by several key factors: biomagnification potential, feeding rate, and trophic level. Some contaminants, such as PCBs and methyl mercury, have an affinity for tissue lipids and tend to be retained and biomagnified in organisms. The tissue concentration of such contaminants often increases at higher trophic levels, such as fish-eating birds and mammals. The indirect exposure to some contaminants, such as inorganic forms of metals, may be relatively more significant for species that feed directly on benthic organisms, where the tissue concentrations are higher.

Feeding rate and movement also affect the amount of indirect exposure to contaminants. Unlike benthic organisms, fish, birds and mammals are often highly mobile and may spend a substantial portion of their lives away from the area of sediment contamination. Consequently, it is often difficult to determine the amount of contaminant exposure in these organisms that is due to feeding in the area of interest. Assessing the amount of indirect exposure resulting from sediment contamination is much more difficult than for direct exposure, as all of the complexities associated with the effects of sediment processes on contaminant exposure are compounded by additional variations in feeding and life history.

3. ENVIRONMENTAL SETTING

California encompasses a variety of environmental conditions ranging from the Sierra Nevada to deserts (with a huge variation in between these two extremes) to the Pacific Ocean. Specific geographical features that form basins, the availability of natural resources coupled with climate and topography have created a very broad range of land use patterns and population densities throughout California. Because of these unique differences around the State, the Legislature in the Porter-Cologne Water Quality Control Act, Water Code section 13000 et seq. (Porter-Cologne) divided the State into nine different hydrologic regions or basins. These regions consist of the North Coast , San Francisco Bay, Central Coast, Los Angeles, Central Valley, Lahontan, Colorado River, Santa Ana and San Diego Regions. Though many regions share some common environmental problems, each of the regions has a unique suite of factors, such as types of discharges, pollutants, potential risks to beneficial uses and receptors that are specific to that region.

The following section provides a brief description of the regions and waters within the regions. For each region, the section includes a summary of bays and estuaries within the region that have been listed on the State Water Board's 2006 Clean Water Act section 303(d) list for impairments related to sediment quality. The listings described below include water column, tissue and sediment quality impacts associated with toxic pollutants identified on the 2006 Section 303(d) list. Tissue listings are discussed because the food web exposure pathway frequently begins in the sediment. Water column listings are also included because the toxic pollutants eventually settle out and are deposited in the surface sediments. Many of these sediment- and tissue-related listings were designated previously by the State Water Board as Toxic Hot Spots and proposed for cleanup. Toxic Hotspots are identified in Table 4.2. Maps of hot spots are presented by Region in Appendix D.

The Lahontan and Colorado River Regions do not include enclosed bays[10] and estuaries[11] and are not considered further in this document. Descriptions of the regions were obtained from the individual water quality control plans (basin plans).

3.1 NORTH COAST REGION

The North Coast Region comprises all regional basins, including Lower Klamath Lake and Lost River Basins, draining into the Pacific Ocean from the California-Oregon state line southern

[10] ENCLOSED BAYS are indentations along the coast which enclose an area of oceanic water within distinct headlands or harbor works. Enclosed bays include all bays where the narrowest distance between headlands or outermost harbor works is less than 75 percent of the greatest dimension of the enclosed portion of the bay. This definition includes but is not limited to: Humboldt Bay, Bodega Harbor, Tomales Bay, Drakes Estero, San Francisco Bay, Morro Bay, Los Angeles Harbor, Upper and Lower Newport Bay, Mission Bay, and San Diego Bay.

[11] ESTUARIES AND COASTAL LAGOONS are waters at the mouths of streams that serve as mixing zones for fresh and ocean waters during a major portion of the year. Mouths of streams that are temporarily separated from the ocean by sandbars shall be considered as estuaries. Estuarine waters will generally be considered to extend from a bay or the open ocean to the upstream limit of tidal action but may be considered to extend seaward if significant mixing of fresh and salt water occurs in the open coastal waters. The waters described by this definition include but are not limited to the Sacramento-San Joaquin Delta as defined by Section 12220 of the California Water Code, Suisun Bay, Carquinez Strait downstream to Carquinez Bridge, and appropriate areas of the Smith, Klamath, Mad, Eel, Noyo, and Russian Rivers.

boundary and includes the watershed of the Estero de San Antonio and Stemple Creek in Marin and Sonoma Counties (Figure 3.1). Two natural drainage basins, the Klamath River Basin and the North Coastal Basin, divide the Region. The Region covers all of Del Norte, Humboldt, Trinity, and Mendocino Counties, major portions of Siskiyou and Sonoma Counties, and small portions of Glenn, Lake, and Marin Counties. It encompasses a total area of approximately 19,390 square miles, including 340 miles of coastline and remote wilderness areas, as well as urbanized and agricultural areas.

Beginning at the Smith River in northern Del Norte County and heading south to the Estero de San Antonio in northern Marin County, the Region encompasses a large number of major river estuaries. Other North Coast streams and rivers with significant estuaries include the Klamath River, Redwood Creek, Little River, Mad River, Eel River, Noyo River, Navarro River, Elk Creek, Gualala River, Russian River, and Salmon Creek (this creek mouth also forms a lagoon). Northern Humboldt County coastal lagoons include Big Lagoon and Stone Lagoon. The largest enclosed bay in the North Coast Region is Humboldt Bay in Humboldt County. Another enclosed bay, Bodega Bay, is located in Sonoma County near the southern border of the Region.

Distinct temperature zones characterize the North Coast Region. Along the coast, the climate is moderate and foggy with limited temperature variation. Inland, however, seasonal temperature ranges in excess of 100°F (Fahrenheit) have been recorded. Precipitation is greater than for any other part of California, and damaging floods are a fairly frequent hazard. Particularly devastating floods occurred in the North Coast area in December 1955, December 1964, and February 1986. Ample precipitation in combination with the mild climate found over most of the North Coast Region has provided a wealth of fish, wildlife, and scenic resources. The mountainous nature of the Region, with its dense coniferous forests interspersed with grassy or chaparral covered slopes, provides shelter and food for deer, elk, bear, mountain lion, fur bearers, and many upland bird and mammal species. The numerous streams and rivers of the Region contain anadromous fish, and the reservoirs, although few in number support both cold water and warm water fish.

Tidelands and marshes are extremely important to many species of waterfowl and shore birds, both for feeding and nesting. Cultivated land and pasturelands also provide supplemental food for many birds, including small pheasant populations. Tideland areas along the north coast provide important habitat for marine invertebrates and nursery areas for forage fish, game fish, and crustaceans. Offshore coastal rocks are used by many species of seabirds as nesting areas.

Major components of the economy are tourism and recreation, logging and timber milling, aggregate mining, commercial and sport fisheries, sheep, beef and dairy production, and vineyards and wineries. In all, the North Coast Region offers a beautiful natural environment with opportunities for scientific study and research, recreation, sport, and commerce.

Approximately two percent of California's total population resides in the North Coast Region. The largest urban centers are Eureka in Humboldt County and Santa Rosa in Sonoma County. The most common factors affecting beneficial uses in the North Coast Region are temperature, nutrients and sedimentation in creeks and rivers that drain the region. Few toxic pollutants have been identified at levels causing degradation of beneficial uses in the bays and estuaries of the North Coast Region. Humboldt Bay was added to the 2006 303(d) List by the State Water Board due to dioxin compounds reported in fish tissue caught from that bay. Although some lakes are impaired do to mercury, there are no other toxic pollutant-related listings in bays and estuaries in this Region.

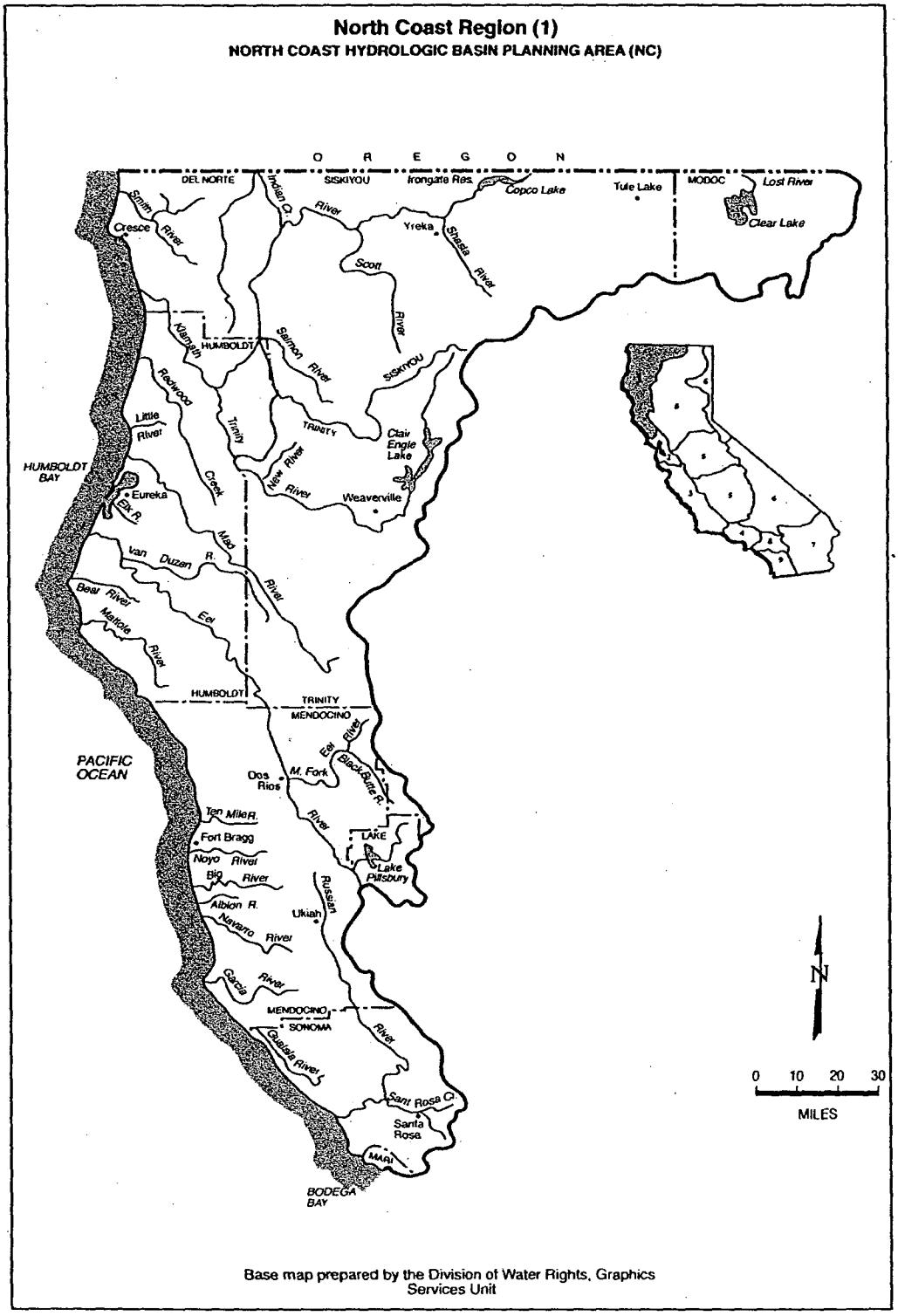


Figure 3.1. North Coast Region

3.2 SAN FRANCISCO BAY REGION

The San Francisco Bay Region comprises San Francisco Bay, Suisun Bay beginning at the Sacramento River, and San Joaquin River westerly, from a line which passes between Collinsville and Montezuma Island (Figure 3.2). The Region's boundary follows the borders common to Sacramento and Solano Counties and Sacramento and Contra Costa Counties west of the Markely Canyon watershed in Contra Costa County. All basins west of the boundary, described above, and all basins draining into the Pacific Ocean between the southern boundary of the North Coast Region and the southern boundary of the watershed of Pescadero Creek in San Mateo and Santa Cruz Counties are included in the Region.

The Region comprises most of the San Francisco Estuary to the mouth of the Sacramento-San Joaquin Delta. The San Francisco Estuary conveys the waters of the Sacramento and San Joaquin Rivers to the Pacific Ocean. Located on the central coast of California, the Bay system functions as the only drainage outlet for waters of the Central Valley. It also marks a natural topographic separation between the northern and southern coastal mountain ranges. The Region's waterways, wetlands, and bays form the centerpiece of the fourth largest metropolitan area in the United States, including all or major portions of Alameda, Contra Costa, Marin, Napa, San Francisco, San Mateo, Santa Clara, Solano, and Sonoma Counties.

The San Francisco Bay Regional Water Board has jurisdiction over the part of the San Francisco Estuary, which includes all of the San Francisco Bay segments extending east to the Delta (Winter Island near Pittsburg). The San Francisco Estuary sustains a highly dynamic and complex environment. Within each section of the Bay system lie deepwater areas that are adjacent to large expanses of very shallow water. Salinity levels range from hypersaline to fresh water and water temperature varies widely.

The Bay system's deepwater channels, tidelands, marshlands, fresh water streams and rivers provide a wide variety of habitats within the Region. Coastal embayments including Tomales Bay and Bolinas Lagoon are also located in this Region. The Central Valley Regional Water Board has jurisdiction over the Delta and rivers extending further eastward.

The San Francisco Estuary is made up of many different types of aquatic habitats that support a great diversity of organisms. Suisun Marsh in Suisun Bay is the largest brackish-water marsh in the United States. San Pablo Bay is a shallow embayment strongly influenced by runoff from the Sacramento and San Joaquin Rivers.

The Central Bay is the portion of the Bay most influenced by oceanic conditions. The South Bay, with less freshwater inflow than the other portions of the Bay, acts more like a tidal lagoon. Together these areas sustain rich communities of aquatic life and serve as important wintering sites for migrating waterfowl and spawning areas for anadromous fish.

Sediment quality-related impairments are summarized in Table 3.1. Tissue listings potentially related to pollutants in sediment are summarized in Table 3.2. Water column listings are presented in Table 3.3.

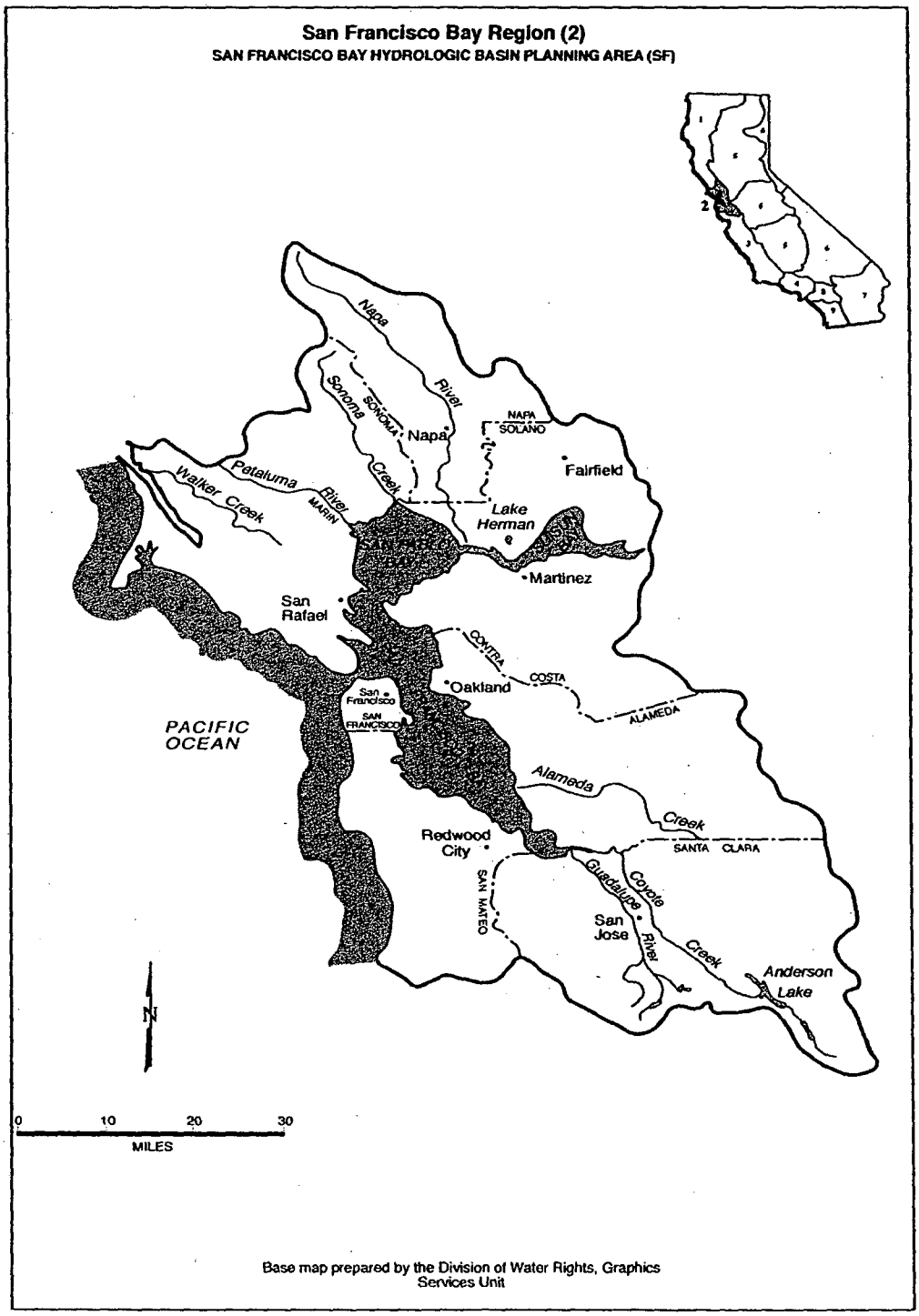


Figure 3.2. San Francisco Bay Region

Table 3.1. Summary of sediment Quality Related 303(d) Listing of Bays and Estuaries in the San Francisco Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Stege Marsh	Estuary	Chlordane, Copper, Dacthal, Dieldrin, Mercury, PCBs[2], Zinc, Sediment Toxicity, Benthic Community Impacts
Islais Creek	Estuary	Chlordane Dieldrin, PAH[3], Sediment Toxicity, Benthic Community Impacts
Mission Creek	Estuary	Chlordane, Dieldrin Lead, Mercury, PAHs[3], PCBs[2], Silver, Zinc, Lead, Mercury, Sediment Toxicity, Benthic Community Impacts
Petaluma River (tidal portion),	Estuary	Nickel
Oakland Inner Harbor (Fruitvale Site)	Bay	Chlordane, PCBs[2], Sediment Toxicity
Oakland Inner Harbor (Pacific Dry-dock Yard)	Bay	Chlordane, Copper, Dieldrin, Lead, Mercury, PCBs[2], Zinc, Sediment Toxicity
Castro Cove, Richmond	Bay	Dieldrin, Mercury, PAHs[3], Selenium
Central Basin, San Francisco Bay	Bay	Dieldrin, Mercury, PAHs[3], Selenium, Sediment Toxicity
San Leandro Bay	Bay	Lead, Mercury, PAHs[3], Chlordane, Dieldrin, Zinc, Sediment Toxicity, Benthic Community Impacts
San Pablo Bay	Bay	

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.2. 303(d) Tissue Listings in Bays and Estuaries of the San Francisco Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Carquinez Strait	Bay	Mercury, PCBs[2], Selenium
Central Basin, San Francisco Bay	Bay	Mercury, PCBs[2], Selenium
Oakland Inner Harbor (Fruitvale Site)	Bay	Mercury, PCBs[2], Selenium
Oakland Inner Harbor (Pacific Dry-dock Yard)	Bay	Mercury, PCBs[2], Selenium
Suisun Bay	Estuary	Mercury, PCBs[2], Selenium
Tomales Bay	Bay	Mercury
San Pablo Bay	Bay	Mercury, PCBs[2], Selenium

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.3. 303(d) Water Quality Listings in Bays and Estuaries of the San Francisco Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
San Francisco Bay, Richardson Bay	Bay	Chlordane, Dieldrin, DDT
San Francisco Bay, San Pablo Bay	Bay	Chlordane, Dieldrin, DDT
San Francisco Bay, Central Basin	Bay	Chlordane, Dieldrin, DDT
San Francisco Bay, Oakland Inner Harbors	Bay	Chlordane, Dieldrin, DDT
San Francisco Bay, San Leandro Bay	Bay	Chlordane, Dieldrin
San Francisco Bay, Lower Basin	Bay	Mercury, Chlordane, Dieldrin, DDT
San Francisco Bay, South Basin	Bay	Mercury, Chlordane, Dieldrin, DDT

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)

3.3 CENTRAL COAST REGION

The Central Coast Region comprises all basins (including Carrizo Plain in San Luis Obispo and Kern Counties) draining into the Pacific Ocean from the southern boundary of the Pescadero Creek watershed in San Mateo and Santa Cruz Counties; to the southeastern boundary of the Rincon Creek watershed, located in western Ventura County (Figure 3.3). The Region extends over a 300-mile long by 40-mile wide section of the State's central coast. Its geographic area encompasses all of Santa Cruz, San Benito, Monterey, San Luis Obispo, and Santa Barbara Counties as well as the southern one-third of Santa Clara County, and small portions of San Mateo, Kern, and Ventura Counties. Included in the region are urban areas such as the Monterey Peninsula and the Santa Barbara coastal plain; prime agricultural lands such as the Salinas, Santa Maria, and Lompoc Valleys; National Forest lands; extremely wet areas such as the Santa Cruz Mountains; and arid areas such as the Carrizo Plain. Water bodies in the Central Coast Region are varied. Enclosed bays and harbors in the Region include Morro Bay, Elkhorn Slough, Tembladero Slough, Santa Cruz Harbor, Moss Landing Harbor, San Luis Harbor, and Santa Barbara Harbor. Several small estuaries also characterize the Region, including the Santa Maria River Estuary, San Lorenzo River Estuary, Big Sur River Estuary, and many others. Major rivers, streams, and lakes include San Lorenzo River, Santa Cruz River, San Benito River, Pajaro River, Salinas River, Santa Maria River, Cuyama River, Estrella River and Santa Ynez River, San Antonio Reservoir, Nacimiento Reservoir, Twitchel Reservoir, and Cuchuma Reservoir. The economic and cultural activities in the basin have been primarily agrarian. Livestock grazing persists, but has been combined with hay cultivation in the valleys. Irrigation, with pumped local groundwater, is very significant in intermountain valleys throughout the basin. Mild winters result in long growing seasons and continuous cultivation of many vegetable crops in parts of the basin.

While agriculture and related food processing activities are major industries in the Region, oil production, tourism, and manufacturing contribute heavily to its economy. The northern part of the Region has experienced a significant influx of electronic manufacturing; while offshore oil exploration and production have heavily influenced the southern part. Total population of the Region is estimated at 1.22 million people.

Water quality problems frequently encountered in the Central Coastal Region include excessive salinity or hardness of local groundwaters. An increase in nitrate concentrations is a growing problem in a number of areas, in both groundwater and surface water. Surface waters suffer from bacterial contamination, nutrient enrichment, and siltation in a number of watersheds. Pesticides are a concern in agricultural areas and associated downstream water bodies. Sediment quality-related impairments and water column listings associated with toxic pollutants are summarized in Tables 3.4 and 3.5 respectively.

3.4 LOS ANGELES REGION

The Los Angeles Region comprises all basins draining into the Pacific Ocean between the southeastern boundary of the watershed of Rincon Creek, located in western Ventura County, and a line which coincides with the southeastern boundary of Los Angeles County, from the Pacific Ocean to San Antonio Peak, and follows the divide, between the San Gabriel River and Lytle Creek drainages to the divide between Sheep Creek and San Gabriel River drainages (Figure 3.4).

The Region encompasses all coastal drainages flowing into the Pacific Ocean between Rincon Point (on the coast of western Ventura County) and the eastern Los Angeles County line, as well as the drainages of five coastal islands (Anacapa, San Nicolas, Santa Barbara, Santa Catalina and San Clemente). In addition, the Region includes all coastal waters within three miles of the continental and island coastlines.

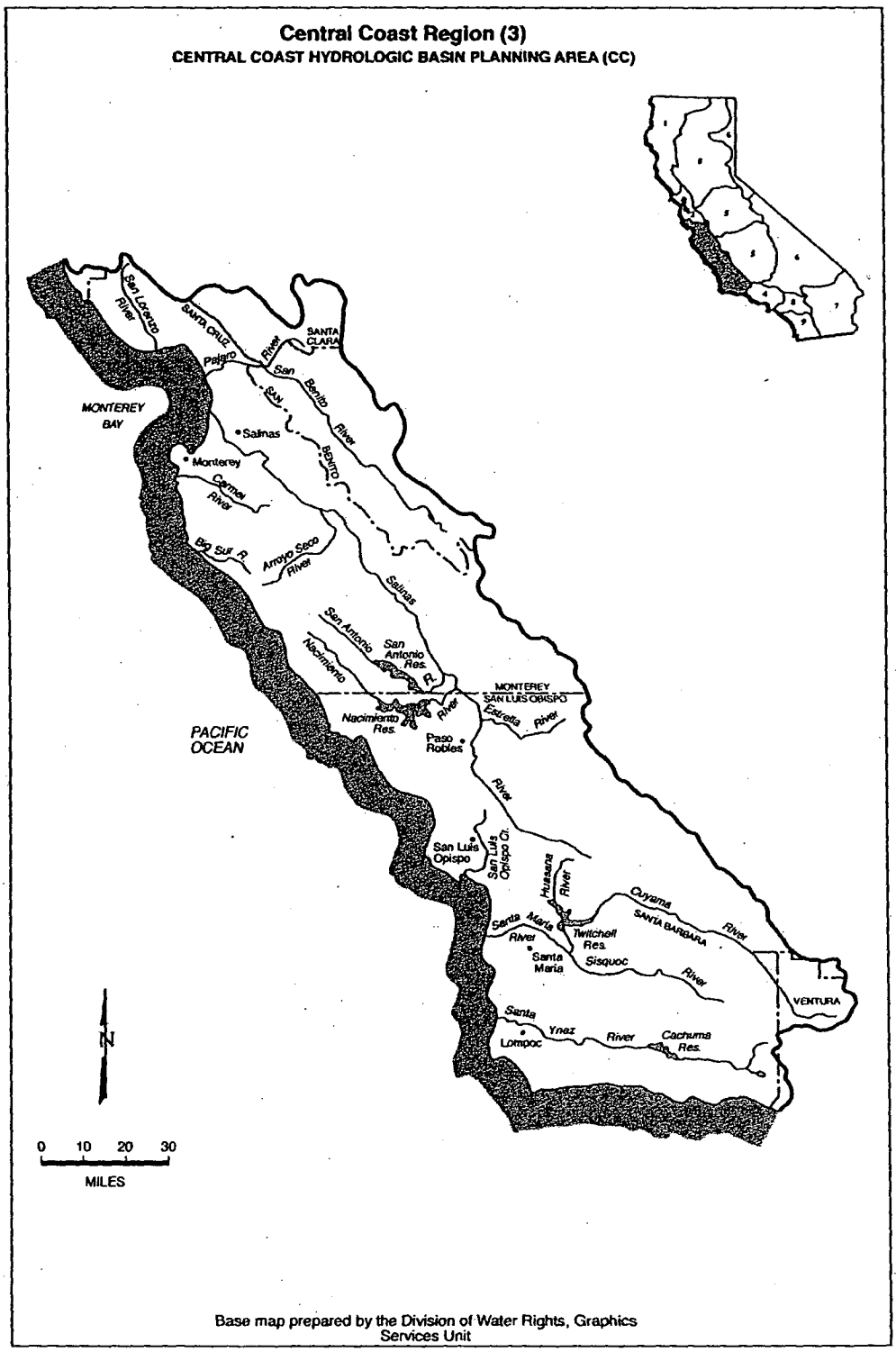


Figure 3.3 Central Coast Region

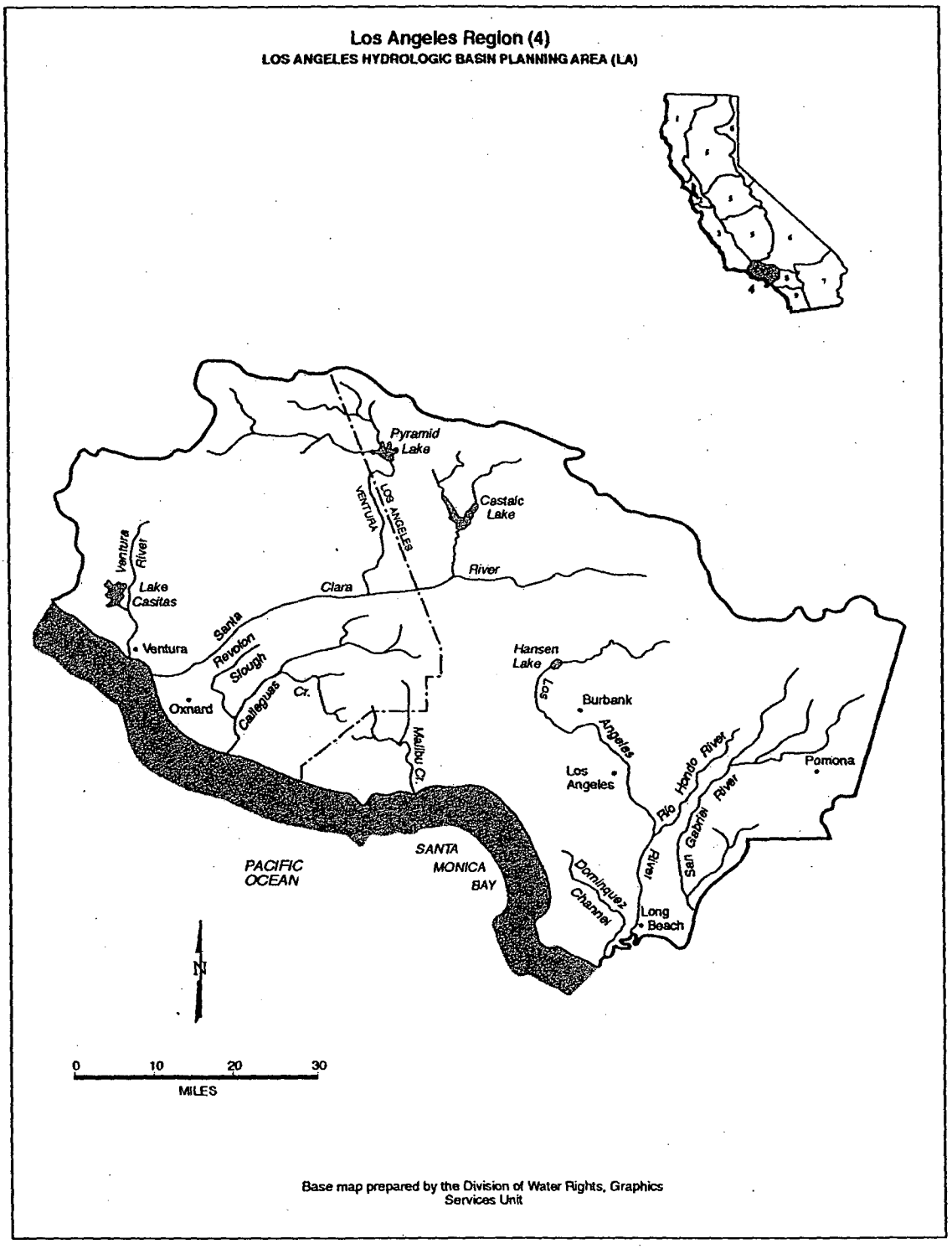


Figure 3.4. Los Angeles Region

Table 3.4. 303(d) Listings Related to Sediment Quality in Bays and Estuaries of the Central Coast Region (SWRCB, 2006).

Water Body	Type[1]	Basis For Impairment
Carpenteria Marsh (El Estero Marsh)	Estuary	Priority Organics
Elkhorn Slough	Estuary	Pesticides
Monterey Harbor	Bay	Metals, Toxicity
Moss Landing Harbor	Bay	Pesticides
Moro Cojo Slough	Estuary	Pesticides
Old Salinas River Estuary	Estuary	Pesticides
Salinas River Lagoon (North	Bay	Pesticides

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.5. 303(d) Listings Related to Water Quality in Bays and Estuaries of the Central Coast Region (SWRCB, 2006).

Water Body	Type[1]	Basis For Impairment
Monterey Harbor	Bay	Metals, Toxicity
Moss Landing Harbor	Bay	Pesticides

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)

Two large deepwater harbors (Los Angeles and Long Beach Harbors) and one smaller deepwater harbor (Port Hueneme) are contained in the Region. There are small craft marinas within the harbors, as well as tank farms, naval facilities, fish processing plants, boatyards, and container terminals.

Several small-craft marinas also exist along the coast (Marina del Rey, King Harbor, Ventura Harbor); these contain boatyards, other small businesses and dense residential development.

Several large, primarily concrete-lined rivers (Los Angeles River, San Gabriel River) lead to unlined tidal prisms, which are influenced by marine waters. Salinity may be greatly reduced following rains since these rivers drain large urban areas composed of mostly impermeable surfaces. Some of these tidal prisms receive a considerable amount of freshwater throughout the year from publicly owned treatment works discharging tertiary-treated effluent. Lagoons are located at the mouths of other rivers draining relatively undeveloped areas (Mugu Lagoon, Malibu Lagoon, Ventura River Estuary, and Santa Clara River Estuary). There are also a few isolated coastal brackish water bodies receiving runoff from agricultural or residential areas.

Santa Monica Bay, which includes the Palos Verdes Shelf, dominates a large portion of the open coastal water bodies in the Region. The Region's coastal water bodies also include the areas along the shoreline of Ventura County and the waters surrounding the five offshore islands in the region.

Sediment quality, tissue and water quality listings for toxic pollutants are summarized in Tables 3.6, 3.7 and 3.8 respectively.

Table 3.6. Summary of Sediment Quality Related 303(d) Listing of Bays and Estuaries in the Los Angeles Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Ballona Creek Estuary	Estuary	Chlordane, DDT, Lead, PCBs[2], PAHs[3], Zinc, Sediment Toxicity, Benthic Community Impacts
Calleguas Creek Reach 1 (Mugu Lagoon)	Estuary	DDT, Sediment Toxicity
Channel Islands Harbor	Bay	Lead, Zinc
Dominguez Channel	Estuary	DDT, Zinc, Sediment Toxicity, Benthic Community Impacts
Los Angeles Harbor – Fish Harbor	Bay	Benzo[a]anthracene Dibenz[a,h]anthracene, Chlordane, Chrysene (C1-C4) Copper, Lead, Mercury, Phenanthrene, Pyrene, Zinc, Sediment toxicity
Los Angeles River Estuary (Queensway Bay)	Estuary	Chlordane, DDT, Lead, PCBs[2], Sediment Toxicity
Los Angeles Harbor – Inner Cabrillo Beach	Bay	Copper
Los Angeles Harbor – Consolidated Slip	Bay	Cadmium, Chlordane, Chromium, Copper, DDT, Lead, Mercury, PCBs[2], Zinc, Sediment Toxicity Benthic Community Impacts
Los Angeles/Long Beach Inner Harbor	Bay	Benthic Community Impacts, Sediment Toxicity
Los Cerritos Channel	Estuary	Chlordane
Malibu Lagoon	Estuary	Benthic Community Impacts
Marina del Rey Harbor – Back Basins	Bay	Chlordane, Copper, DDT, Lead, PCBs[2], Zinc, Sediment Toxicity
McGrath Lake	Estuary	Dieldrin, PCBs, Sediment Toxicity
San Pedro Bay Near/Off Shore Zones	Bay	Chlordane, Copper, Chromium, DDT, PAHs[3], Zinc, Benthic Community Impacts, Sediment Toxicity

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.7. Summary of 303(d) Tissue listings in Bays and Estuaries of the Los Angeles Region Included (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Ballona Creek Estuary	Estuary	Chlordane, PCBs
Dominguez Channel	Estuary	Chlordane, DDT, Dieldrin, Lead
Los Angeles Harbor – Fish Harbor	Bay	DDT, PCBs
Los Angeles River Estuary (Queensway Bay)	Estuary	DDT, PCBs
Los Angeles Harbor – Consolidated Slip	Bay	Dieldrin
Los Angeles/Long Beach Inner Harbor	Bay	Chlordane, DDT, PCBs
Los Angeles/Long Beach Outer Harbor (inside breakwater)	Bay	Chlordane, DDT

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.8. Summary of 303(d) Water Quality Listings in Bays and Estuaries of the Los Angeles Region Included (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Calleguas Creek Reach 1 (Mugu Lagoon)	Estuary	Copper, Mercury, Nickel
Dominguez Channel	Estuary	PAHs
Los Angeles Harbor – Fish Harbor	Bay	PAHs, DDT, PCBs[2], Copper, Lead, Mercury, Zinc
Los Angeles Harbor – Consolidated Slip	Bay	Chlordane, DDT, PCBs[2], Toxaphene
Los Angeles/Long Beach Inner Harbor	Bay	DDT, PCBs[2]
Los Angeles Harbor – Inner Cabrillo Beach Area	Bay	Copper, DDT, PCBs[2]
Los Angeles/Long Beach Outer Harbor (inside breakwater)	Bay	DDT, PCBs[2]
Marina del Rey Harbor – Back Basins	Bay	Chlordane, DDT, Dieldrin, PCBs[2]
San Pedro Bay Near/Off Shore Zones	Bay	Chlordane, PCBs[2]
Santa Clara River Estuary	Estuary	Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls

3.5 CENTRAL VALLEY REGION

The Central Valley Region includes approximately 40 percent of the land in California stretching from the Oregon border to the Kern County/ Los Angeles county line. The Region is divided into three basins. For planning purposes, the Sacramento River Basin and the San Joaquin River basin are covered under one Basin Plan and the Tulare Lake Basin is covered under a separate distinct one (Figures 3.5, 3.6 and 3.7).

The Sacramento River Basin covers 27,210 square miles and includes the entire area drained by the Sacramento River. The principal streams are the Sacramento River and its larger tributaries: the Pitt, Feather, Yuba, Bear, and American Rivers to the East; and Cottonwood, Stony, Cache, and Putah Creek to the west. Major reservoirs and lakes include Shasta, Oroville, Folsom, Clear Lake, and Lake Berryessa.

The San Joaquin River Basin covers 15,880 square miles and includes the entire area drained by the San Joaquin River. Principal streams in the basin are the San Joaquin River and its larger tributaries: the Consumnes, Mokelumne, Calaveras, Stanislaus, Tuolumne, Merced, Chowchilla, and Fresno Rivers. Major reservoirs and lakes include Pardee, New Hogan, Millerton, McClure, Don Pedro, and New Melones.

The Tulare Lake Basin covers approximately 16,406 square miles and comprises the drainage area of the San Joaquin Valley south of the San Joaquin River. The planning boundary between the San Joaquin River Basin and the Tulare Lake Basin is defined by the northern boundary of Little Pinoche Creek basin eastward along the channel of the San Joaquin River to Millerton Lake in the Sierra Nevada foothills, and then along the southern boundary of the San Joaquin River drainage basin. Main rivers within the basin include the King, Kaweah, Tule, and Kern Rivers, which drains the west face of the Sierra Nevada Mountains. Imported surface water supplies enter the basin through the San Luis Drain- California Aqueduct System, Friant-Kern Channel and the Delta Mendota Canal.

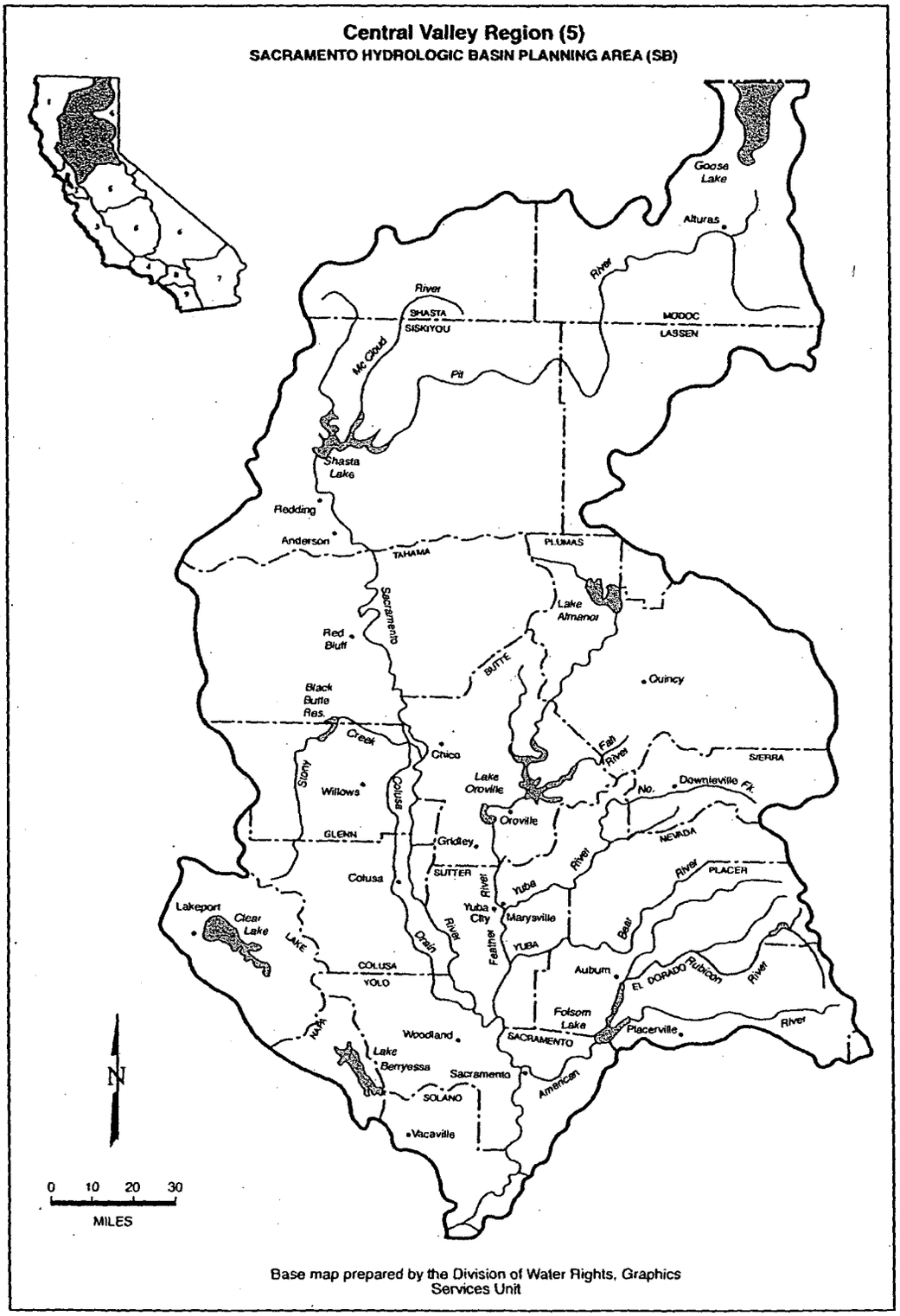
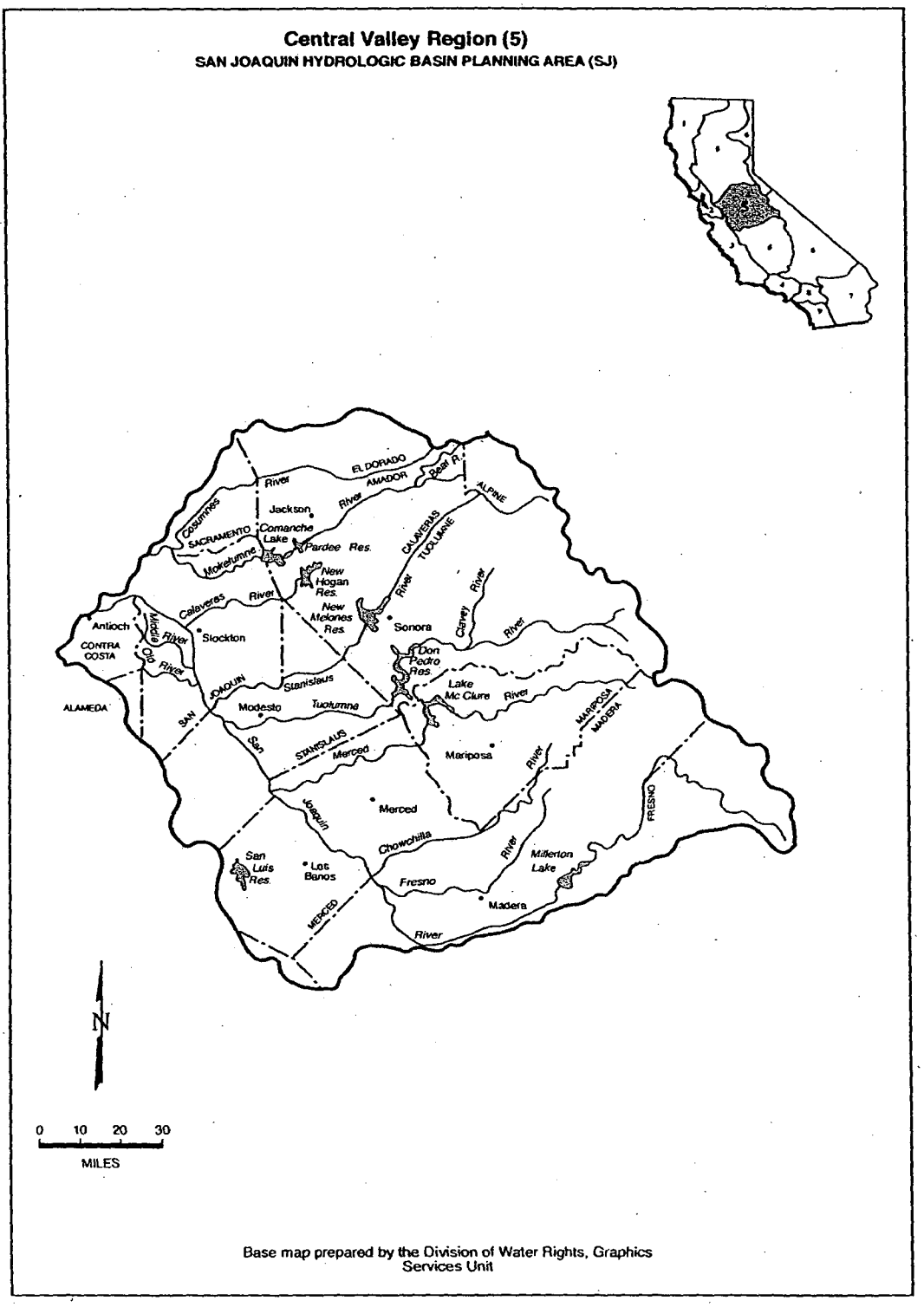


Figure 3.5. Central Valley Region Sacramento Hydrologic Basin



Base map prepared by the Division of Water Rights, Graphics Services Unit

Figure 3.6. Central Valley Region San Joaquin Hydrologic Basin

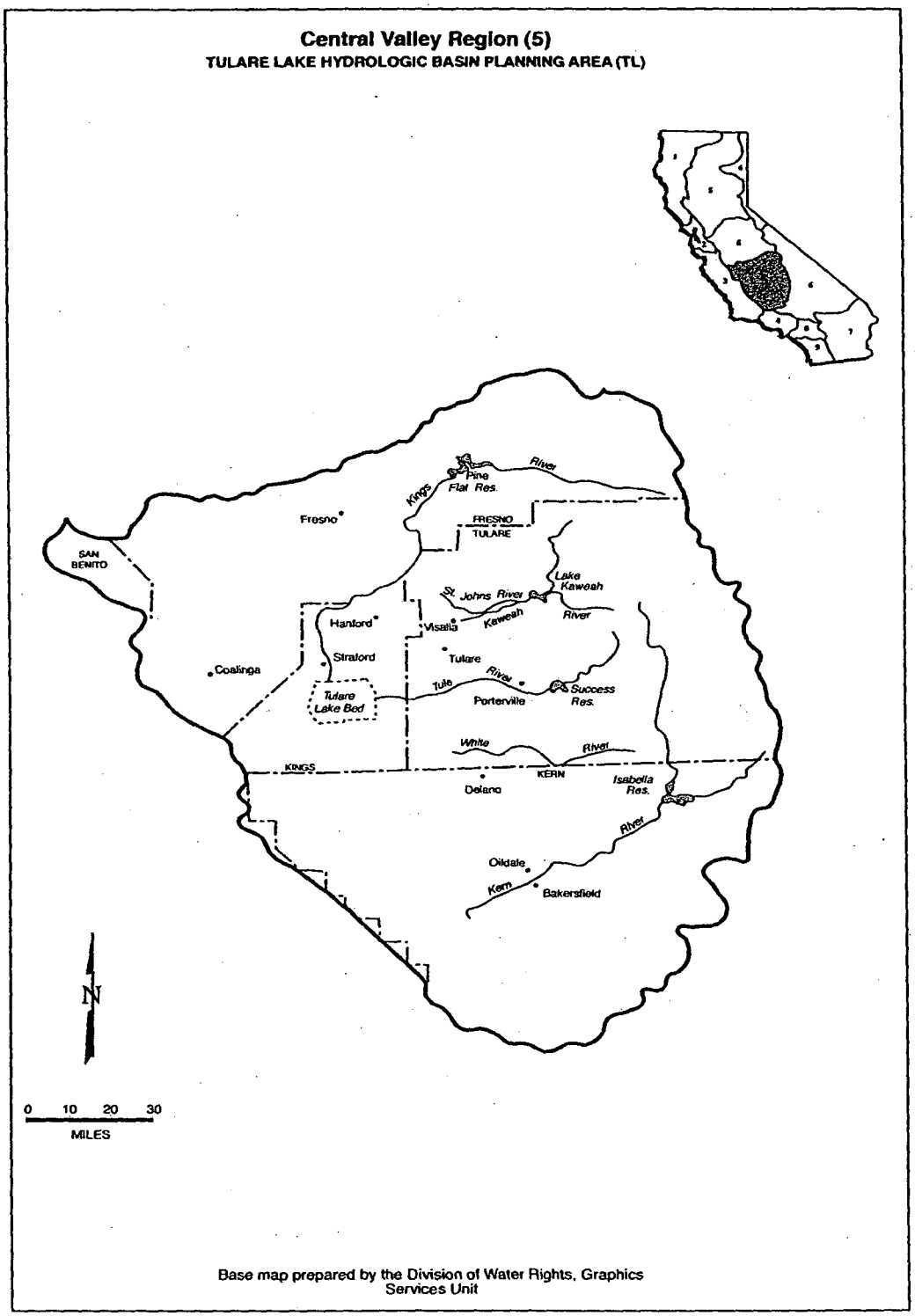


Figure 3.7. Central Valley Region Tulare lake Hydrologic Basin

The two northern most basins are bound by the crests of the Sierra Nevada on the east and the Coast Range and Klamath Mountains on the west. They extend about 400 miles from the California-Oregon border southward to the headwaters of the San Joaquin River. These two river basins cover about one fourth of the total area of the State and over 30 percent of the State's irrigable land. The Sacramento and San Joaquin Rivers furnish roughly 50 percent of the State's water supply. Surface water from the two drainage basins meet and form the Delta, which ultimately drains into the San Francisco Bay. The Delta is a maze of river channels and diked islands covering roughly 1,150 square miles, including 78 square miles of water area. Two major water projects located in the South Delta, the Federal Central Valley Project and the State Water Project, deliver water from the Delta to Southern California, the San Joaquin Valley, Tulare Lake Basin, the San Francisco Bay Area, as well as within the Delta boundaries. The legal boundary of the Delta is described in Water Code section 12220.

Tissue and water quality listings for toxic pollutants are summarized in Tables 3.9 and 3.10. The major pollutants affecting estuarine waters in the Central Valley include nutrients, metals, pathogens, and pesticides among others (SWRCB, 2003a).

Table 3.9. Summary of 303(d) Tissue Listings in Estuaries of the Central Valley Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Delta Waterways Northern Portion	Estuary	DDT, PCBs[2], Mercury
Delta Waterways Southern Portion	Estuary	DDT, Mercury
Delta Waterways Central Portion	Estuary	DDT, PCBs[2], Mercury
Delta Waterways Eastern Portion	Estuary	DDT, Mercury
Delta Waterways Western Portion	Estuary	DDT, Mercury
Delta Waterways Stockton Ship Channel	Estuary	DDT, Dioxins, Mercury, PCBs[2]

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.10. Summary of 303(d) Water Quality Listings in Estuaries of the Central Valley Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Delta Waterways Northern Portion	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene
Delta Waterways Southern Portion	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene
Delta Waterways Central Portion	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene
Delta Waterways Eastern Portion	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene
Delta Waterways Western Portion	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene
Delta Waterways Stockton Ship Channel	Estuary	Chlorpyrifos, DDT, Diazinon, Mercury, Aldrin, Dieldrin, Chlordane, Endrin, Heptachlor, Heptachlor Epoxide, Hexachlorocyclohexane (including Lindane), Endosulfan, and Toxaphene

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)

There are also a number of sediment quality-related 303(d) listings for waters upstream of affected bays and estuaries (see SWRCB, 2006). Impaired sediments can be carried downstream and settle into bays and estuaries, contributing to existing impairments or causing new impairments.

3.6 SANTA ANA REGION

The Santa Ana Region comprises all basins draining into the Pacific Ocean between the southern boundary of the Los Angeles Region and the drainage divide between Muddy and Moro Canyons, from the ocean to the summit of San Joaquin Hills; along the divide between lands draining into Newport Bay and Laguna Canyon to Niguel Road; along Niguel Road and Los Aliso Avenue to the divide between Newport Bay and Aliso Creek drainages; and along the divide and the southeastern boundary of the Santa Ana River drainage to the divide between Baldwin Lake and Mojave Desert drainages; to the divide between the Pacific Ocean and Mojave Desert drainages (Figure 3.8). The Santa Ana Region is the smallest of the nine regions in the state (2,800 square miles) and is located in southern California, roughly between Los Angeles and San Diego.

Although small geographically, the region's four-plus million residents (1993 estimate) make it one of the most densely populated regions. The climate of the Santa Ana Region is classified as Mediterranean: generally dry in the summer with mild, wet winters. The average annual rainfall in the region is about fifteen inches, most of it occurring between November and March.

The enclosed bays in the Region include Newport Bay, Bolsa Bay (including Bolsa Chica Marsh), and Anaheim Bay. Principal Rivers include Santa Ana, San Jacinto and San Diego. Lakes and reservoirs include Big Bear, Hemet, Mathews, Canyon Lake, Lake Elsinore, Santiago Reservoir, and Perris Reservoir.

The section 2002 303(d) list for the Santa Ana Region included nine water bodies affecting an estimated 7,886 acres (bays, estuaries, lakes, and wetlands) and 24 water bodies affecting 191 miles of rivers and shoreline. The major pollutants affecting these water bodies included nutrients, metals, pathogens, pesticides, and sediments among others (SWRCB 2003a). Sediment quality-related impairments are summarized in Table 3.11. Tissue listings potentially related to pollutants in sediment are summarized in Table 3.12.

Table 3.11. Summary of Sediment Quality Related 303(d) Listing of Bays and Estuaries in the Santa Ana Region (SWRCB, 2006)

Water Body	Type[1]	Basis for Impairment
Anaheim Bay	Bay	Sediment Toxicity
Huntington Harbour	Bay	Chlordane, Lead, Sediment Toxicity
Newport Bay – Lower	Bay	Chlordane, Copper, DDT, PCBs, Sediment Toxicity
Newport Bay – Upper (Ecological Reserve)	Bay	Chlordane, DDT, PCBs, Metals, Benthic Community Degradation, Sediment Toxicity
Rhine Channel	Bay	Sediment Toxicity

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

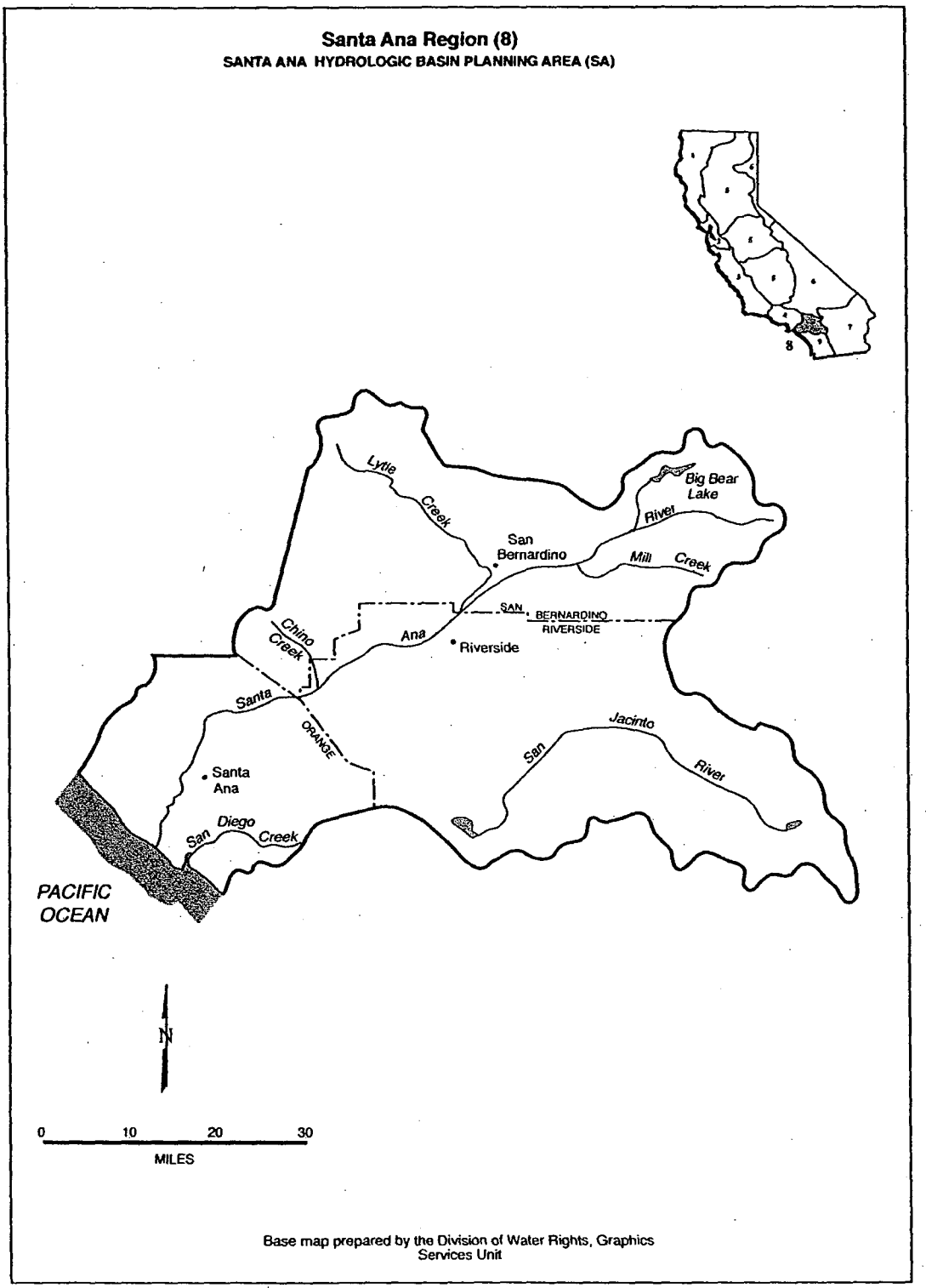


Figure 3.8. Santa Ana Region

Table 3.12. Summary of 303(d) Tissue Listing of Bays and Estuaries in the Santa Ana Region (SWRCB, 2006)

Water Body	Type[1]	Basis for Impairment
Anaheim Bay	Bay	Chlordane, Dieldrin, PCBs[2]
Huntington Harbour	Bay	PCBs[2]

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.13. Summary of 303(d) Water Quality Listings for Toxic Pollutants in Bays and Estuaries of the Santa Ana Region (SWRCB, 2006)

Water Body	Type[1]	Basis for Impairment
Huntington Harbour	Bay	Copper
Bolsa Bay	Bay	Copper
Upper Newport Bay	Bay	Copper, PCBs[2], Chlordane, DDT, Metals
Lower Newport Bay	Bay	Copper, PCBs[2], Chlordane, DDT
Rhine Channel	Bay	Copper, Lead, Mercury, Zinc, PCB[2]

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

3.7 San Diego Region

The San Diego Region comprises all basins draining into the Pacific Ocean between the southern boundary of the Santa Ana Region and the California-Mexico boundary (Figure 3.9). The San Diego Region is located along the coast of the Pacific Ocean from the Mexican border to north of Laguna Beach. The Region is rectangular in shape and extends approximately 80 miles along the coastline and 40 miles east to the crest of the mountains. The Region includes portions of San Diego, Orange, and Riverside Counties.

The population of the Region is heavily concentrated along the coastal strip. Six deepwater sewage outfalls and one across the beach discharge from the new border plant at the Tijuana River empty into the ocean. Two harbors, Mission Bay and San Diego Bay, support major recreational and commercial boat traffic. Coastal lagoons are found along the San Diego County coast at the mouths of creeks and rivers.

The 2002 section 303(d) list for the San Diego Region included 26 water bodies affecting an estimated 6,907 acres (bays, estuaries, lakes, and wetlands) and 40 water bodies affecting 148 miles of rivers and shoreline. The major pollutants affecting these water bodies included nutrients, metals, pathogens, pesticides, and sediments among others (SWRCB, 2003a).

Weather patterns are Mediterranean in nature with an average rainfall of approximately ten inches per year occurring along the coast. Almost all the rainfall occurs during wet cool winters. The Pacific Ocean generally has cool water temperatures due to upwelling. This nutrient-rich water supports coastal beds of giant kelp. The cities of San Diego, National City, Chula Vista, Coronado, and Imperial Beach surround San Diego Bay in the southern portion of the Region.

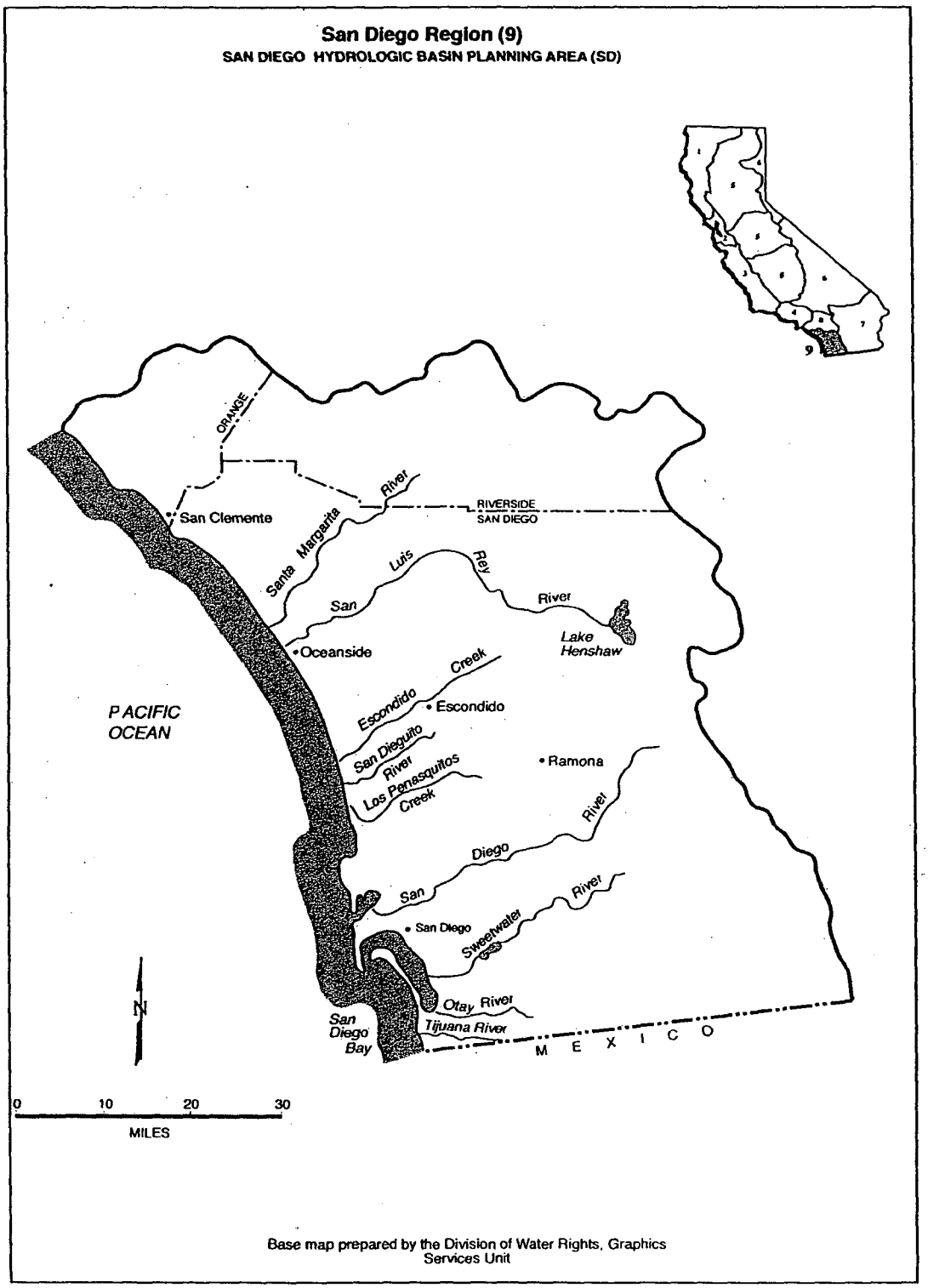


Figure 3.9. San Diego Region

San Diego Bay is long and narrow, 15 miles in length and approximately one mile across. A deep-water harbor, San Diego Bay has experienced waste discharge from former sewage outfalls, industries, and urban runoff. Up to 9,000 vessels may be moored there. San Diego Bay also hosts four major U.S. Navy bases with approximately 80 surface ships and submarines. Coastal waters include bays, harbors, estuaries, beaches, and open ocean. Sediment quality-related impairments are summarized in Table 3.14. Tissue listings potentially related to pollutants in sediment are summarized in Table 3.15.

Table 3.14. Summary of Sediment Quality Related 303(d) Listing of Bays and Estuaries in the San Diego Region (SWRCB, 2006)

Water Body	Type[1]	Basis for Impairment
San Diego Bay Shoreline, 32nd St San Diego Naval Station	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, Downtown Anchorage	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, near Chollas Creek	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, near Coronado Bridge	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, 9 B near sub base	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, near Switzer Creek	Bay	Chlordane, Lindane/Hexachlorocyclohexane (HCH), PAHs
San Diego Bay Shoreline, North of 24th Street Marine Terminal	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, Seventh Street Channel	Bay	Benthic Community Effects, Sediment Toxicity
San Diego Bay Shoreline, Vicinity of B St and Broadway Piers	Bay	Benthic Community Effects, Sediment Toxicity

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.15. Summary of Sediment Quality Related 303(d) Tissue Listing of Bays and Estuaries in the San Diego Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
San Diego Bay	Bay	PCBs

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

Table 3.16. Summary of Water Column Related 303(d) Listing for toxic Pollutants in Bays and Estuaries of the San Diego Region (SWRCB, 2006)

Water Body	Type[1]	Basis For Impairment
Mission Bay	Bay	Lead
San Diego Bay Shoreline, near Switzer Creek	Bay	Chlordane, PAHs
San Diego Bay Shoreline at Coronado Cays	Bay	Copper
San Diego Bay, Shoreline at Glorietta Bay	Bay	Copper
San Diego Bay, Shoreline at Harbor Island (East Basin)	Bay	Copper
San Diego Bay, Shoreline at Harbor Island (West Basin)	Bay	Copper
San Diego Bay, Shoreline at Marriott Marina	Bay	Copper
San Diego Bay, Shoreline between Sampson and 28th St.	Bay	Copper
San Diego Bay, Shoreline Chula Vista Marina	Bay	Copper

1. Based upon beneficial uses provided in fact sheets (SWRCB, 2006)
2. Polychlorinated biphenyls
3. Polyaromatic hydrocarbons

4. REGULATORY BASELINE

This section describes current state and federal laws, programs, and practices that govern sediment quality in bays and estuaries. These laws, programs and practices represent the regulatory baseline for measuring incremental impacts of Part 1. As explained in greater detail in the following discussion, the basin plans for the coastal Regional Water Boards all contain narrative water quality objectives that apply to sediment quality in bays and estuaries. These narrative objectives provide the basis for sediment cleanup activities under current state and federal law.

The section begins with a brief overview of Porter-Cologne and the Clean Water Act, 33 U.S.C. section 1251 et seq. A more detailed discussion of relevant laws, programs, and practices follows. Porter-Cologne is the primary water quality control law for California. It addresses two key functions – planning and waste discharge regulation. The State Water Board adopts state policy for water quality control, which is binding on the Regional Water Boards. (Wat. Code §13140 et seq.) The State Water Board is also authorized to adopt water quality control plans for waters that require water quality standards under the Clean Water Act and must adopt plans for ocean waters and for enclosed bays and estuaries. (Wat. Code §§13170, 131702., 13391.) The Regional Water Boards are required to adopt water quality control plans, or basin plans, for waters within their respective regions. Water quality control plans designate beneficial uses of water, establish water quality objectives to protect those uses, and contain a program to implement the objectives. (*Id.*§13050(j).) The beneficial use designations and water quality objectives (together with an antidegradation policy) constitute water quality standards for purposes of the Clean Water Act. (See Clean Water Act §303(c)(2)(A); 40 C.F.R. §§131.3(i), 131.6.)

The Water Boards have designated for protection a variety of beneficial uses for bay and estuarine waters, including, among others, the preservation and enhancement of fish, wildlife, and other aquatic resources and habitats; commercial and sport fishing; and shellfish harvesting. They have also adopted water quality objectives to protect the uses, which can be either numeric or narrative. All regional basin plans include narrative toxicity objectives.

Porter-Cologne establishes a program to regulate waste discharges that could affect water quality through waste discharge requirements, conditional waivers, or prohibitions. (See Wat. Code §§13243, 13263, 13269.) This program is the principal way in which water quality control policies and plans are implemented. The term "waste" is broadly defined in Porter-Cologne and includes toxic pollutants, as well as other waste substances. (*Id.* §13050(d).) The term "waters of the state" is similarly broadly defined to include all surface waters, including bays and estuaries, and groundwater within state boundaries. (*Id.* §13050(e).)

Porter-Cologne also authorizes the Water Boards to investigate water quality and to require waste dischargers to submit monitoring and technical reports. (*Id.* §13267, 13383.) In addition, Porter-Cologne gives the Water Boards extensive enforcement authority to respond to unauthorized discharges, discharges in violation of applicable requirements, discharges that cause pollution or nuisance, and other matters. The enforcement options include, among others, cleanup and abatement orders, cease and desist orders, and administrative civil liability orders. (*Id.* §13301, 13304, 13323.)

In 1989, Porter-Cologne was amended to specifically address the threat posed to bays and estuaries from toxic pollutants. The legislation, which added chapter 5.6 to Division 7 of the Water Code, mandated that the State Water Board develop a consolidated toxic hot spot cleanup plan and adopt sediment quality objectives for bays and estuaries. The State Water

Board established the Bay Protection and Toxic Cleanup Program to implement the requirements of chapter 5.6.

The Water Boards also implement the federal Clean Water Act. As required under section 303(c) of the Act, the Water Boards adopt water quality standards for waters of the United States. In addition, the Water Boards issue National Pollutant Discharge Elimination System (NPDES) permits pursuant to section 402 of the Clean Water Act. Section 402 of the Clean Water Act requires that all point source discharges of pollutants to waters of the United States be regulated under a permit. The State Water Board is the state water pollution control agency for all purposes stated in the Clean Water Act. (*Id.* §13160.) As such, the State Water Board is authorized to issue water quality certifications under Clean Water Act §401 . The Water Boards also implement the total maximum daily load (TMDL) program, which is required under section 303(d) of the Clean Water Act.

4.1 Existing Water Quality Standards Related to Sediment Quality

As explained above, water quality standards consist of beneficial uses, criteria (which are the federal equivalent of water quality objectives) and an antidegradation policy. All basin plans for the coastal regions contain water quality objectives or prohibitions that apply to sediment quality. None of the Regional Water Boards has adopted numeric water quality objectives for sediments. Rather, the Regional Water Boards typically rely on narrative toxicity objectives to protect and manage ambient sediment quality. The current narratives and prohibitions used to regulate sediment quality are listed below in Section 4.1.1. These narratives (and associated beneficial uses) provide the bases for permit requirements, cleanup actions, Clean Water Act §303(d) listings, and other regulatory activities. Section 4.1.2 explains how the Regional Water Boards currently assess sediment quality to ascertain compliance with water quality standards. Section 4.1.3 describes state policies and federal regulations for toxic pollutant standards applicable to bay and estuarine waters.

4.1.1 Applicable Basin Plan Narrative Objectives or Prohibitions

Water Quality Control Plan for the North Coast Region

Regional Water Quality Control Board 5550 Skylane Blvd., Suite A Santa Rosa, CA 95403 (http://www.waterboards.ca.gov/northcoast/programs/basinplan/bpdocs.html).

- *All waters shall be maintained free of toxic substances in concentrations that are toxic to, or that produce detrimental physiological responses in human, plant, animal, or aquatic life. Compliance with this objective will be determined by use of indicator organisms, analyses of species diversity, population density, growth anomalies, bioassays of appropriate duration, or other appropriate methods as specified by the Regional Water Board.*
- *No individual pesticide or combination of pesticides shall be present in concentrations that adversely affect beneficial uses. There shall be no bioaccumulation of pesticide concentrations found in bottom sediments or aquatic life.*

Water Quality Control Plan (Basin Plan) for the San Francisco Bay Basin

San Francisco Regional Water Quality Control Board, 1515 Clay St. Suite 1400, Oakland, CA 94612 (http://www.waterboards.ca.gov/sanfranciscobay/basinplan.htm).

- Many pollutants can accumulate on particles, in sediment, or bioaccumulate in fish and other aquatic organisms. Controllable water quality factors shall not cause a detrimental increase in concentrations of toxic substances found in bottom sediments or aquatic life. Effects on aquatic organisms, wildlife, and human health will be considered.
- *Controllable water quality factors shall not cause a detrimental increase in the concentrations of toxic pollutants in sediments or aquatic life.*
- *All waters shall be maintained free of toxic substances in concentrations that are lethal to or that produce other detrimental responses in aquatic organisms. Detrimental responses include, but are not limited to, decreased growth rate and decreased reproductive success of resident or indicator species.*
- *There shall be no chronic toxicity in ambient waters. Chronic toxicity is a detrimental biological effect on growth rate, reproduction, fertilization success, larval development, population abundance, community composition, or any other relevant measure of the health of an organism, population, or community. Chronic toxicity generally results from exposures to pollutants exceeding 96 hours. However, chronic toxicity may also be detected through short-term exposure of critical life stages of organisms.*
- *The health and life history characteristics of aquatic organisms in waters affected by controllable water quality factors shall not differ significantly from those for the same waters in areas unaffected by controllable water quality factors.*
- *Bottom deposits or aquatic growths to the extent that such deposits or growths cause nuisance or adversely affect beneficial uses*
- *Toxic or other deleterious substances to be present in concentrations or quantities, which will cause deleterious effects on wildlife, waterfowl, or other aquatic biota, or which render any of these unfit for human consumption, either at levels created in the receiving waters or as a result of biological concentration.*

Water Quality Control Plan for the Central Coastal Basin

Central Coast Regional Water Quality Control Board
895 Aerovista Place, Suite 101
San Luis Obispo, CA 93401
http://www.swrcb.ca.gov/rwqcb3/BasinPlan/Index.htm.

- *All waters shall be maintained free of toxic substances in concentrations which are toxic to, or which produce detrimental physiological responses in, human, plant, animal, or aquatic life. Compliance with this objective will be determined by use of indicator organisms, analyses of species diversity, population density, growth anomalies, toxicity bioassays of appropriate duration, or other appropriate methods as specified by the Regional Board.*
- *No individual pesticide or combination of pesticides shall reach concentrations that adversely affect beneficial uses. There shall be no increase in pesticide concentrations found in bottom sediments or aquatic life.*

Water Quality Control Plan Los Angeles Region

Los Angeles Regional Water Quality Control Board
320 W. 4th St., Suite 200
Los Angeles, CA 90013

http://www.waterboards.ca.gov/losangeles/html/meetings/tmdl/Basin_plan/basin_plan_doc.html

- *No individual pesticide or combination of pesticides shall be present in concentrations that adversely affect beneficial uses. There shall be no increase in pesticide concentrations found in bottom sediments or aquatic life*
- *Toxic pollutants shall not be present at levels that will bioaccumulate in aquatic life to levels which are harmful to aquatic life or human health*

Water Quality Control Plan for the Sacramento and San Joaquin River Basins

Central Valley Regional Water Quality Control Board
Sacramento Main Office 11020 Sun Center Drive, Suite 200
Rancho Cordova, CA 95670-6114
Fresno Branch Office 1685 E Street Fresno, CA 93706-2007
Redding Branch Office 415 Knollcrest Drive, Suite 100 Redding, CA 96002
http://www.waterboards.ca.gov/centralvalley/available_documents/index.html#anchor616381

- *All waters shall be maintained free of toxic substances in concentrations that produce detrimental physiological responses in human, plant, animal or aquatic life.*
- *Compliance with this narrative objective will be determined by analyses of indicator organisms, species diversity, growth anomalies, and biotoxicity tests of appropriate duration or other methods as specified by the Regional Water Board.*
- *The Regional Water Board will also consider all material and relevant information submitted by the discharger and other interested parties and numerical criteria and guidelines for toxic substances developed by the State Water Board, the California Office of Environmental Health Hazard Assessment, the California Department of Health Services, the US Food and Drug Administration, the National Academy of Sciences, the US Environmental Protection Agency, and other organizations to evaluate compliance with this objective.*
- *No individual pesticide or combination of pesticides shall be present in concentrations that adversely affect beneficial uses. Discharges shall not result in pesticide concentrations in bottom sediments or aquatic life that adversely affect beneficial uses*
- *Where compliance with these narrative objectives is required (i.e., where the objectives are applicable to protect specified beneficial uses), the Regional Water Board will, on a case-by-case basis, adopt numerical limitations in orders which will implement the narrative objectives. To evaluate compliance with the narrative water quality objectives, the Regional Water Board considers, on a case-by-case basis, direct evidence of beneficial use impacts, all material and relevant information submitted by the discharger and other interested parties, and relevant numerical criteria and guidelines developed and/or published by other agencies and organizations.*
- *In considering such criteria, the Board evaluates whether the specific numerical criteria, which are available through these sources and through other information supplied to the Board, are relevant and appropriate to the situation at hand and, therefore, should be used in determining compliance with the narrative objective.*

Water Quality Control Plan Santa Ana River Basin

Santa Ana Regional Water Quality Control Board
3737 Main St., Suite 500
Riverside, CA 92501
http://www.waterboards.ca.gov/santaana/html/basin_plan.html

- Toxic substances shall not be discharged at levels that will bioaccumulate in aquatic resources to levels which are harmful to human health.
- The concentrations of toxic substances in the water column, sediments or biota shall not adversely affect beneficial uses

Water Quality Control Plan for the San Diego Basin

San Diego Regional Water Quality Control Board
9174 Sky Park Court, Suite 100
San Diego, CA 92123
http://www.waterboards.ca.gov/sandiego/programs/basinplan.html

- All waters shall be maintained free of toxic substances in concentrations that are toxic to, or that produce detrimental physiological responses in human, plant, animal, or aquatic life. Compliance with this objective will be determined by use of indicator organisms, analyses of species diversity, population density, growth anomalies, bioassays of appropriate duration, or other appropriate methods as specified by the Regional Board
- The survival of aquatic life in surface waters subjected to a waste discharge or other controllable water quality factors, shall not be less than that for the same water body in areas unaffected by the waste discharge or, when necessary, for other control water that is consistent with requirements specified in US EPA, State Water Resources Control Board or other protocol authorized by the Regional Board. As a minimum, compliance with this objective as stated in the previous sentence shall be evaluated with a 96-hour acute bioassay
- In addition, effluent limits based upon acute bioassays of effluents will be prescribed where appropriate, additional numerical receiving water objectives for specific toxicants will be established as sufficient data become available, and source control of toxic substances will be encouraged

4.1.2 Current Regional Water Board Approaches for Assessing Whether Sediment Quality Complies with Applicable Standards

Indicators and Interpretive Tools

The type of monitoring and testing currently required by the Regional Water Boards to assess sediment quality varies by region. Each Regional Water Board has the discretion to determine how much information is enough to initiate an enforcement action. To assess direct exposure within the regions, one, two or three lines of evidence, such as sediment chemistry, sediment toxicity and benthic community analysis may be used to initiate an action. In the Central Valley Region, one line of evidence is adequate justification for an action. The lack of assessment tools has limited the use of bioassessment data in regulatory programs within the Central Valley Region (Bruns et al. 2007).

The San Diego Regional Water Board has devoted extensive resources to the assessment of sediment quality in San Diego Bay. Staff typically utilize sediment chemistry, sediment toxicity testing and benthic community analysis to assess direct effects to aquatic life. The selection of interpretative tools and thresholds are site specific and typically involve input from other organizations such as California Department of Fish and Game (DFG), U.S. Fish and Wildlife Service (U.S. FWS), California Department of Toxics Substance Control (DTSC), and National Oceanic and Atmospheric Administration (NOAA).

In the San Diego Region, sediment quality guidelines used recently to classify chemical concentrations in sediment are ERMs developed for metals (Long et al., 1998), Consensus midrange effects concentration developed for PAHs and PCBs (Swartz, 1999; MacDonald et al., 2000), and Sediment Quality Guideline Quotient (SQGQ) for chemical mixtures (Fairey et al., 2001). When attempting to distinguish localized impacts from regional or waterbody wide disturbances, these data are also compared with reference sites. The statistical procedure used by the San Diego Regional Water Board to identify stations where conditions are significantly different from the Reference Condition consists of identifying station sample values outside boundary established by the 95% prediction limit (PL) reference pool of data for each contaminant of concern. The sediment toxicity tests applied consisted of a 10-day amphipod survival test, a 48-hour bivalve larva development test exposed to the sediment-water interface, and 40-minute echinoderm egg fertilization test exposed to sediment pore water. The results of these toxicity tests are compared statistically to their respective negative controls using a one-tailed Student t-test ($\alpha = 0.05$). Toxicity results were ranked as low, moderate, and high toxicity based upon the magnitude of the response and type and significance of response and exposure (acute versus sublethal, whole sediment versus porewater). Benthic Community was classified as low, moderate, and high potential for benthic community degradation classifications. In this example, the benthic community structure indices at each station were compared to thresholds developed for the Bight'98 Benthic Response Index for Embayments (BRI-E) (Ranasinghe et al., 2003) and to the Reference Condition sample stations.

For the other Regional Water Boards, sediment chemistry is frequently interpreted by comparison with ambient levels or sediment quality guidelines. Sediment toxicity is characterized by a significant difference in mean survival between a sample and the control and if the magnitude of this difference was biologically significant or comparison to a waterbody specific reference envelope or more recent approaches developed to more effectively integrate the response with other lines of evidence. , Where benthic community tools have been developed, those applied include the Relative Benthic Index also developed for the BPTCP, the Index of Biotic Integrity (Thompson and Lows, 2004) and the Benthic Response Index (Smith *et al.*, 2003) utilized by Regional Boards, the regulated community, SCCWRP and others to monitor the southern California Bight.

Monitoring

The Regional Water Boards have varying approaches to sediment monitoring. Resolution 92-043 adopted by the San Francisco Bay Regional Water Board on April 15, 1992 officially established the Regional Monitoring Program (RMP) in San Francisco Bay. Resolution 92-043 authorized Regional Board staff to suspend some site-specific monitoring requirements for permittees, if the permittees would contribute to the development and support of a regional monitoring program. The Regional Board recognizing the advantages of a regional program cited the cost effectiveness and the greater ability to assess both the effectiveness of controls and overall waterbody health in comparison to data only collected from specific discharges. A component of includes sediment quality monitoring

Within the Los Angels Region, the City of Los Angeles' Terminal Island Treatment Plant, which discharges into the Los Angeles Long Beach Harbor, is required to perform both routine sediment quality monitoring and to participate in Regional Monitoring Studies. The routine monitoring studies are curtailed while regional monitoring studies are ongoing. Both of these efforts utilize sediment chemistry, sediment toxicity testing and benthic community analysis in addition to other indicators (trawls, tissue residue analysis) (For more information visit http://63.199.216.5/webdata/data/docs/2171_R4-2005-0024_MRP.pdf). Recently the Los Angeles Region has required five permittees to perform a joint sediment characterization study in Marina Del Rey in support of TMDL development. This monitoring program will be used to determine if the controls such as BMPS are effective alone or if sediment remediation will be required in addition to the controls to restore beneficial uses.

4.1.3 Toxic Pollutant Standards

Regulation of toxic pollutant discharges to bay and estuarine waters is important because these discharges can adversely affect sediment quality. In addition to the narrative objectives listed above, state water quality standards include numeric water quality objectives for toxic and other pollutants in water quality control plans and federally-promulgated criteria for toxic pollutants, which are contained in the California Toxics Rule. (See 40 C.F.R. §131.38.) The California Toxics Rule (CTR) criteria apply to inland surface waters, enclosed bays, and estuaries in the state. The numeric criteria and objectives establish permissible water column concentrations for the affected pollutants.

The State Water Board may also consider adopting a policy establishing a water quality objective for methylmercury in fish tissue in the future. In 2001, U.S. EPA issued a recommended fish tissue criterion for methylmercury. The State Water Board's proposed policy would modify the recommended criterion to reflect California-specific information on fish consumption. Elements of the proposed policy may include a methylmercury fish tissue objective, a total mercury water quality objective, a methylmercury water quality objective, or some combination of these objectives. The proposed policy may also include implementation procedures related to the NPDES permitting process.

In 2000, the State Water Board adopted state policy for water quality control to implement toxic standards in bays, estuaries and inland surface waters. The policy, entitled "Policy for Implementation of Toxic Standards for Inland Surface Waters, Enclosed Bays, and Estuaries of California," (SIP) provides a standardized approach for permitting discharges of toxic pollutants to non-ocean surface waters in a manner that promotes statewide consistency. The SIP describes: (1) applicable priority pollutant criteria and objectives; (2) data requirements and adjustments; (3) the identification of priority toxic pollutants requiring water quality-based effluent limitations; (4) the calculation of effluent limitations; (5) appropriate translators for metals and selenium;(6) factors to consider in the designation of mixing zones and dilution credits (7) ambient background concentrations and (8) intake water credits.

The SIP is not applicable to stormwater discharges nor does the SIP address sediment quality specifically. However, Section 1.4.2.1 does prohibit mixing zones from causing "objectionable bottom deposits" (SWRCB, 2000). This term is defined as "an accumulation of materials ... on or near the bottom of a water body which creates conditions that adversely impact aquatic life, human health, beneficial uses, or aesthetics. These conditions include, but are not limited to, the accumulation of pollutants in the sediment."

Additionally, the State Water Board's "Water Quality Control Policy for the Enclosed Bays and Estuaries of California" prescribes requirements pertaining to toxic pollutant discharges to enclosed bays and estuaries:

- Persistent or cumulative toxic substances shall be removed from the waste to the maximum extent practical through source control or treatment prior to discharge.
- New discharges of municipal wastewaters and industrial process waters (excluding cooling water) to enclosed bays and estuaries (excluding the San Francisco Bay Delta) are prohibited unless the effluent is discharged in a manner that enhances the quality of the receiving water.

4.2 CURRENT SEDIMENT CLEANUP AND REMEDIATION ACTIVITIES

Under current law, sediment cleanup activities may be undertaken in response to a Clean Water Act §303(d) listing, an enforcement order issued pursuant to Porter-Cologne, or the Bay Protection and Toxic Cleanup Program. In addition, cleanup of hazardous wastes may be driven by the California Health and Safety Code well as federal Laws such as Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) Superfund Amendments and Reauthorization Act (SARA).

4.2.1 Section 303(d) Activities

State Water Board Listing Policy

Clean Water Act section 303(d) requires that the states list waters that do not meet applicable water quality standards with technology-based controls alone. In 2004, the State Water Board adopted a Water Quality Control Policy for Developing California's Section 303(d) List. For sediments, the policy provides that a water segment will be listed as impaired if the sediments exhibit statistically significant toxicity based on a binomial distribution of the sampling data and exceedances. When applying this methodology, if the number of measured toxicity exceedances supports rejection of the null hypothesis, the water segment is considered impaired. The policy indicates that a segment should be listed if the observed toxicity is associated with a toxicant or toxicants, or for toxicity alone. If the toxicant causing or contributing to the toxicity is identified, the pollutant should be added to the 303(d) list as well.

Appropriate reference and control measures must be included in the toxicity testing. Reference conditions may include a response less than 90% of the minimum significant difference for each specific test organism. Acceptable methods include, but are not limited to, those listed in water quality control plans, the methods used by Surface Water Ambient Monitoring Program, the Southern California Bight Projects of the Southern California Coastal Water Research Project, American Society for Testing and Materials, EPA, the Regional Monitoring Program of the San Francisco Estuary Institute, and the Bay Protection and Toxic Cleanup Program (BPTCP) (SWRCB, 2004b).

Association of pollutant concentrations with toxic or other biological effects should be determined by one of the following (SWRCB, 2004b):

- Sediment quality guidelines are exceeded using the binomial distribution; in addition, using rank correlation, the observed effects are correlated with measurements of chemical concentration in sediments
- An evaluation of equilibrium partitioning or other type of toxicological response that identifies the pollutant that may cause the observed impact; comparison to reference conditions within a watershed or ecoregion may be used to establish sediment impacts
- Development of an evaluation (such as a TIE) that identifies the pollutant that contributes to or caused the observed impact.

TMDLs

Clean Water Act section 303(d) mandates that the state develop TMDLs for its listed waters. A TMDL, in general, identifies the maximum amount of a pollutant that a waterbody can assimilate while still meeting water quality standards. The TMDL identifies pollutant sources and includes an implementation plan that describes the actions necessary to achieve standards, including a schedule and monitoring and surveillance activities to determine compliance. Exhibit 3-10 of the report entitled "Economic Considerations of Sediment Quality Plan for Enclosed and Estuaries in California" (2008) summarizes sediment-related toxic pollutant TMDLs that have already been completed for enclosed bays and estuaries in California. Section 3 of this report identifies bays and estuaries in the coastal regions that are currently on the State Water Board's 2006 Section 303(d) list for water column, tissue and sediment quality impacts associated with toxic pollutants for which TMDLs must be developed.

TMDLs developed by the San Francisco Bay and Los Angeles Regional Water Boards illustrate application of the TMDL program to address sediment quality. The San Francisco Bay Regional Water Board recently adopted a TMDL to address bay-wide exceedances of the narrative bioaccumulation objective caused by excessive methyl-mercury levels. High mercury levels in sediments are due, in large part, to legacy gold mining operations and have resulted in bay-wide fish consumption advisories. The San Francisco Bay Regional Water Board has also listed bay waters for failure to achieve the bioaccumulation narrative objective due to PCBs, another legacy contaminant found in sediments, which was used in many high voltage applications as a dielectric fluid. For both pollutants, the mechanism to restore beneficial uses is through the development of TMDLs where all sources of loading regardless of media are evaluated and controlled to the extent practical. The mercury targets were derived based upon the estimated reduction in mercury mass in tissue that would be needed to be protective of human health and wildlife (California Regional Water Quality Control Board San Francisco Bay Region 2006). Unlike mercury, the movement of PCBs and other hydrophobic organochlorine compounds up through the food web can be predicted with food web models. Once a model has been validated by agreement with actual data, the model can also be used to predict the sediment concentrations that will lower prey tissue to levels that protect the target receptors (California Regional Water Quality Control Board San Francisco Bay Region 2007).

The Los Angeles Regional Water Board adopted the Marina del Rey TMDL in 2005 to address toxic pollutants in sediments and fish tissue. The TMDL established sediment chemistry targets for Marina del Rey, which address both sediment quality and fish tissue. The toxic pollutants include copper, lead, and zinc and chlordane and total PCBs. Numeric targets for these pollutants in sediments are based on ERLs developed by Long *et al.* (1995). In addition to sediment chemistry, the monitoring plan includes both acute and chronic toxicity tests as well as fish tissue testing to monitor progress (Technical Committee County of Los Angeles, Chair, 2007). Toxicity tests utilize three marine organisms; 28-day chronic and a10-day acute amphipod mortality test; pore water testing utilizing the sea urchin fertilization test; and the testing of overlying water using the red abalone larval development test. Toxic sediment will be identified by an average amphipod survival of 70% of less. During accelerated testing, if the response average of two tests is less than 90% survival, stressor identification is required.

4.2.2 Cleanup and Abatement Actions

Resolution No. 92-49

In 1992, the State Water Board adopted Resolution No. 92-49, "Policies and Procedures for Investigation and Cleanup and Abatement of Discharges Under Water Code Section 13304," The resolution describes the policies and procedures that apply to the cleanup and abatement of all types of discharges subject to Water Code section 13304. These include discharges, or threatened discharges, to surface and groundwater. The Resolution requires dischargers to clean up and abate the effects of discharges in a manner that promotes attainment of either background water quality or the best water quality that is reasonable if background levels of water quality cannot be restored, considering economic and other factors. In approving any alternative cleanup levels less stringent than background, Regional Boards must apply section 2550.4 of Title 23 of the California Code of Regulations.[12] Section 2550.4 provides that a Regional Water Board can only approve cleanup levels less stringent than background if the Regional Water Board finds that it is technologically or economically infeasible to achieve background. Resolution No. 92-49 further requires that any alternative cleanup level shall: (1) be consistent with maximum benefit to the people of the state; (2) not unreasonably affect present and anticipated beneficial uses of such water; and (3) not result in water quality less than that prescribed in the water quality control plans and policies adopted by the State and Regional Water Boards.

A Regional Water Board must apply Resolution No. 92-49 when setting cleanup levels for contaminated sediment if such sediment threatens beneficial uses of the waters of the state, and the contamination or pollution is the result of a discharge of waste. Contaminated sediment must be cleaned up to background sediment quality unless it would be technologically or economically infeasible to do so.

Examples of Cleanup Actions Related to Sediment Quality

The Regional Water Boards have issued enforcement orders, primarily cleanup and abatement orders, to address violations of narrative water quality objectives related to sediment quality. For example, the San Diego Regional Water Board issued a cleanup and abatement order to Paco Terminals, Inc. in 1985 to require cleanup of copper-contaminated sediments in San Diego Bay. In 1992, the State Water Board revised the order to impose more stringent cleanup levels. (State Water Board Order No. WQ 92-09.) The State Water Board determined that revised cleanup levels were necessary to ensure that sediments did not contain copper levels that would result in exceedance of either numeric water column objectives or narrative objectives for the protection of aquatic life and to comply with Resolution No. 92-49.

Similarly, in 2005, the San Diego Regional Water Board issued a tentative cleanup and abatement order to address discharges of metals and other pollutant wastes to San Diego Bay marine sediments and waters. The tentative order is based, in part, on alleged exceedances of the basin plan's narrative toxicity objectives. Proceedings to consider adoption of the order are ongoing.

Additional examples of ongoing or completed sediment quality related cleanup actions include Castro Cove, Stege Marsh, Moffet Field, Hamilton Air Base Salt Marsh, Peyton Slough in San Francisco Bay and Convair Lagoon, Bay City Marine, Kettenburg and America's Cup Harbor in southern California.

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[12] Resolution No. 92-49, Section III.G.

4.2.3 Bay Protection and Toxic Cleanup Program

As stated above, chapter 5.6 mandated that the State Water Board fulfill two key tasks – adopt a consolidated hot spots cleanup plan and develop sediment quality objectives for enclosed bays and estuaries. The State Water Board focused initially on the former task.

4.2.3.1 Consolidated Hotspots Cleanup Plan

Chapter 5.6 Requirements

To address toxic hot spots, Water Code section 13392.5 required the Regional Water Boards to develop a consolidated data base that identified all known and potential toxic hot spot spots. In consultation with the State Water Board, the Regional Water Boards were directed to develop an ongoing monitoring and surveillance program that included suggested guidelines to promote standardized analytical methodologies and consistency in data reporting and identification of additional monitoring and analyses needed to complete the toxic hot spot assessment for each enclosed bay and estuary.

In addition, by January 1, 1998, the Regional Water Boards were required to complete and submit to the State Water Board a toxic hot spot cleanup plan for affected waters within their respective regions. (Wat. Code §13394.) Toxic hot spots are defined in Water Code section 13391.5 (e) "as *locations where hazardous substances have accumulated in the water or sediment to levels which (1) may pose a substantial present or potential hazard to aquatic life, wildlife, fisheries, or human health, or (2) may adversely affect the beneficial uses of the bay, estuary, or ocean waters as defined in water quality control plans, or (3) exceeds adopted water quality or sediment quality objectives.*

Each regional toxic hot spots cleanup plan had to include:

(a) A priority ranking of all hot spots, including the state board's recommendations for remedial action at each toxic hot spot site.

(b) A description of each hot spot site including a characterization of the pollutants present at the site.

(c) An estimate of the total costs to implement the plan.

(d) An assessment of the most likely source or sources of pollutants.

(e) An estimate of the costs that may be recoverable from parties responsible for the discharge of pollutants that have accumulated in sediment.

(f) A preliminary assessment of the actions required to remedy or restore a toxic hot spot.

(g) A two-year expenditure schedule identifying state funds needed to implement the plan.

(h) A summary of actions that have been initiated by the regional board to reduce the accumulation of pollutants at existing hot spot sites and to prevent the creation of new hot spots.

The State Water Board was mandated to submit a consolidated statewide toxic hot spot cleanup plan to the Legislature by June 30, 1999. The statewide plan had to include findings and recommendations on the need for establishing a toxic hot spots cleanup program.

Chapter 5.6 further required the Regional Water Boards to revise waste discharge requirements for dischargers that discharged all or part of the pollutants that caused the toxic

hot spot "*to ensure compliance with water quality control plans and water quality control plan amendments, including requirements to prevent the creation of new toxic hot spots and the maintenance or further pollution of existing toxic hot spots.*" (Wat. Code §13395.) A Regional Water Board could determine that it was unnecessary to revise waste discharge requirements only if the Regional Water Board determined that the discharger's contribution was insignificant or that the discharger no longer conducted the practices that led to creation of the toxic hot spot. Water Code section 13396 also prohibits any person from dredging or disturbing a toxic hot spot site without first obtaining a water quality certification under Clean Water Act section 401 or waste discharge requirements.

Program Goals and Actions

The BPTCP was driven by four major goals (SWRCB 2004a): (1) protect existing and future beneficial uses of bay and estuarine waters; (2) identify and characterize toxic hot spots; (3) plan for the prevention and control of further pollution at toxic hot spots; and (4) develop plans for remedial actions of existing toxic hot spots and prevent the creation of new toxic hot spots.

The BPTCP identified benthic organisms and human health as the key targets for protection (SWRCB, 1991) and used both exposure and effects-based measurements of the sediment quality triad (sediment toxicity, benthic community structure and measures of chemical concentrations in sediments) and other measures such as biomarkers and tissue residue to identify toxic hot spots. The sediment quality triad coupled with additional lines of evidence formed the basis for making hot spots determinations. The need for multiple lines of evidence was based upon the uncertainty and technical limitations associated with the tools (Stephenson, et al. 1994).

Sediment samples were taken only in summer months at a depth of 2-cm below the sediment surface. Evaluation of cause or stressor identification was not included in this program. As a result, biological effects at a site were determined to be associated with toxic chemicals if chemical analysis demonstrated significantly higher levels compared to the reference sites. The Bay Protection and Toxic Cleanup Program Quality Assurance Project Plan (Stephenson, et al. 1994) stated that, *because a strict determination of cause-and-effect will not have been achieved, we anticipate that responsible parties will have the opportunity to conduct Toxicity Identification Evaluations as an initial step in site remediation.* The technical team clearly understood the value of stressor identification **preceding** site remediation or restoration, however the difficulty associated with these studies was at the time considered far to expensive to be a requirement (Stephenson, et al. 1994).

Consolidated Hotspots Cleanup Plan

The Consolidated Toxic Hot Spots Cleanup Plan (Consolidated Plan) identified and ranked known toxic hot spots, and presented descriptions of toxic hot spots, actions necessary to remediate sites, the benefits of remediation, and a range of remediation costs. The plan is applicable to any point and nonpoint source discharges that the Regional Water Boards reasonably determine contribute to or cause the pollution at toxic hot spots. The Consolidated Plan required Regional Water Boards to implement the remediation action to the extent that responsible parties can be identified, and funds are available and allocated for this purpose. When the Regional Water Boards cannot identify a responsible party, the Consolidated Plan indicated that they are to seek funding from available sources to remediate the site. The Regional Water Boards determined the ranking of each known toxic hot spot based on the five general criteria specified in the Consolidated Plan as shown in Table 4.1. Table 4.2 describes the rank and reason for listing each hotspot identified in the Consolidated Plan.

Table 4.1. Toxic Hot Spot Ranking Criteria

Criteria Category	High	Moderate	Low
Human Health Impacts	Human health advisory for consumption of nonmigratory aquatic life from the site	Tissue residues in aquatic organisms exceed FDA/DHS action level or U.S. EPA screening levels	None
Aquatic Life Impacts[1]	Hits in any two biological measures if associated with high chemistry	Hit in one of the measures associated with high chemistry	High sediment or water chemistry
Water Quality Objectives	Objectives exceeded regularly	Objectives occasionally exceeded	Objectives infrequently exceeded
Areal Extent of Hot Spot	More than 10 acres	1 to 10 acres	Less than 1 acre
Natural Remediation Potential	Unlikely to improve without intervention	May or may not improve without intervention	Likely to improve without intervention

Source: SWRCB (1999).

1. Site rankings are based on an analysis of the sediment chemistry, sediment toxicity, biological field assessments (including benthic community analysis), water toxicity, TIEs, and bioaccumulation.

Table 4.2. Enclosed Bays Listed as Known Toxic Hot Spots

Rank	Site Identification	Reason for Listing	
		Definition trigger	Pollutants
High	Delta Estuary, Cache Creek watershed including Clear lake	Human health impacts	Mercury
High	Delta Estuary	Aquatic life impacts	Diazinon
High	Delta Estuary – Morrison Creek, Mosher Slough, 5 Mile Slough, Mormon Slough & Calaveras River	Aquatic life impacts	Diazinon & Chlorpyrifos
High	Delta Estuary – Ulatis Creek, Paradise Cut, French Camp & Duck Slough	Aquatic life impacts	Chlorpyrifos
High	Humboldt Bay Eureka Waterfront H Street	Bioassay toxicity	Lead, Silver, Antimony, Zinc, Methoxychlor, PAHs
High	Los Angeles Inner Harbor Dominguez Channel, Consolidated Slip	Human health, aquatic life impacts	DDT, PCBs, PAH, Cadmium, Copper, Lead, Mercury, Zinc, Dieldrin, Chlordane
High	Los Angeles Outer Harbor Cabrillo Pier	Human health, aquatic life impacts	DDT, PCBs, Copper
High	Lower Newport Bay Rhine Channel	Sediment toxicity, exceeds objectives	Arsenic, Copper, Lead, Mercury, Zinc, DDE, PCB, TBT
High	Moss Landing Harbor and Tributaries	Sediment chemistry, toxicity, bioaccumulation, and exceedances of NAS and FDA guidelines	Pesticides, PCBs, Nickel, Chromium, TBT
High	Mugu Lagoon/ Calleguas Creek tidal prism, Eastern Arm, Main Lagoon, Western Arm	Aquatic life impacts	DDT, PCBs, metals, Chlordane, Chlorpyrifos
High	San Diego Bay Seventh St. Channel Paleta Creek, Naval Station	Sediment toxicity and benthic community impacts	Chlordane, DDT, PAHs and Total Chemistry[2]
High	San Francisco Bay Castro Cove	Aquatic life impacts	Mercury, Selenium, PAHs, Dieldrin

Table 4.2. Enclosed Bays Listed as Known Toxic Hot Spots (Continued)

Rank	Site Identification	Reason for Listing	
		Definition trigger	Pollutants
High	San Francisco Bay Entire Bay	Human health impacts	Mercury, PCBs, Dieldrin, Chlordane, DDT, Dioxin Site listing was based on Mercury and PCB health advisory
High	San Francisco Bay Islais Creek	Aquatic life impacts	PCBs, chlordane, dieldrin, endosulfan sulfate, PAHs, anthropogenically enriched H_2S and NH_3
High	San Francisco Bay Mission Creek	Aquatic life impacts	Silver, Chromium, Copper Mercury, Lead, Zinc, Chlordane, Chlorpyrifos, Dieldrin, Mirex, PCBs, PAHs, anthropogenically enriched H_2S and NH_3
High	San Francisco Bay Peyton Slough	Aquatic life impacts	Silver, Cadmium, Copper, Selenium, Zinc, PCBs, Chlordane, ppDDE, Pyrene
High	San Francisco Bay Point Potrero/ Richmond Harbor	Human health	Mercury, PCBs, Copper, Lead, Zinc
High	San Francisco Bay Stege Marsh	Aquatic life impacts	Arsenic, Copper, Mercury, Selenium, Zinc, chlordane, dieldrin, ppDDE, dacthal, endosulfan, endosulfan sulfate, dichlorobenzophenone, heptachlor epoxide, hexachlorobenzene, mirex, oxidiazon, toxaphene and PCBs
Moderate	Anaheim Bay, Naval Reserve	Sediment toxicity	Chlordane, DDE
Moderate	Ballona Creek Entrance Channel	Sediment toxicity	DDT, zinc, lead, Chlordane, dieldrin, chlorpyrifos
Moderate	Bodega Bay-10006 Mason's Marina	Bioassay toxicity	Cadmium, Copper, TBT, PAH
Moderate	Bodega Bay-10028 Porto Bodega Marina	Bioassay toxicity	Copper, lead, Mercury, Zinc, TBT, DDT, PCB, PAH
Moderate	Delta Estuary Delta	Aquatic life impacts	Chlordane, Dieldrin, Lindane, Heptachlor, Total PCBs, PAH & DDT
Moderate	Delta Estuary Delta	Human health impacts	Chlordane, Dieldrin, Total DDT, PCBs, Endosulfan, Toxaphene
Moderate	Los Angeles River Estuary	Sediment toxicity	DDT, PAH, Chlordane
Moderate	Upper Newport Bay Narrows	Sediment toxicity, exceeds water quality objectives	Chlordane, Zinc, DDE
Moderate	Lower Newport Bay Newport Island	Exceeds water quality objectives	Copper, Lead, Mercury, Zinc, Chlordane, DDE, PCB, TBT
Moderate	Marina del Rey	Sediment toxicity	DDT, PCB, Copper, Mercury, Nickel, Lead, Zinc, Chlordane
Moderate	Monterey Harbor	Aquatic life impacts, sediment toxicity	PAHs, Cu, Zn, Toxaphene, PCBs, Tributyltin
Moderate	San Diego Bay Between "B" Street & Broadway Piers	Benthic community impacts	PAHs, Total Chemistry
Moderate	San Diego Bay Central Bay Switzer Creek	Sediment toxicity	Chlordane, Lindane, DDT, Total Chemistry
Moderate	San Diego Bay Chollas Creek	Benthic community impacts	Chlordane, Total Chemistry

Table 4.2. Enclosed Bays Listed as Known Toxic Hot Spots (Continued)

Rank	Site Identification	Reason for Listing	
		Definition trigger	Pollutants
Moderate	San Diego Bay Foot of Evans & Sampson Streets	Benthic Community Impacts	PCBs, Antimony, Copper, Total Chemistry
Moderate	San Francisco Bay Central Basin, San Francisco Bay	Aquatic life impacts	Mercury, PAHs
Moderate	San Francisco Bay Fruitvale (area in front of storm drain)	Aquatic life impacts	Chlordane, PCBs
Moderate	San Francisco Bay Oakland Estuary. Pacific Drydock #1 (in front of storm drain)	Aquatic life impacts	Copper, Lead, Mercury, Zinc, TBT, ppDDE, PCBs, PAHs, Chlorpyrifos, Chlordane, Dieldrin, Mirex
Moderate	San Francisco Bay, San Leandro Bay	Aquatic life impacts	Mercury, Lead, Selenium, Zinc, PCBs, PAHs, DDT, pesticides
Low	Huntington Harbor Upper Reach	Sediment toxicity	Chlordane, DDE, Chlorpyrifos

Source: SWRCB (1999).

As described in Table 4.2 a significant number of hotspots were identified in bays and estuaries. Although the program focused on specific sites, some hotspots encompass large portions of waterbodies and support many of the 303(d) listings described in the previous section. Under the Bay Protection program, all designated hotspots regardless of priority require corrective action, management action or delisting. Appendix D provides additional information on the enclosed bays listed as known toxic hot spots in the Consolidated Plan, including ranking and reason for listing. Appendix D also provides a summary of the remedial actions and estimated costs for the high priority toxic hot spots. Note that several of the remedial actions identified by the State and Regional Boards only characterize the problem at a hot spot. Thus, the costs identified for those actions do not include all actions necessary to fully remediate the toxic hot spot. Additional funds would be required for remediation after characterization studies are complete.

Depending on the source and areal extent of the known toxic hot spot, the actions to remediate the sites include: (1) Institutional controls/education, (2) Better characterization of the sites and problem, (3) Dredging, (4) Capping, (5) A combination of dredging and capping, (6) Source control, (7) Watershed management, and (8) Implementation of a no-action alternative (natural attenuation).

The estimated total cost to implement the Consolidated Plan ranges from $72 million to $812 million. According to the plan, much of this amount is considered recoverable from responsible dischargers. The un-funded portion of the cost to implement the Consolidated Plan ranges from approximately $40 million to $529 million. Although much of the Consolidated Plan can be implemented through existing Water Code authorities, no funding was obtained to fully implement the Consolidated Plan.

4.2.3.2 SQO Development

In addition to requiring the remediation of toxic hot spots, chapter 5.6 mandated that the State Water Board develop SQOs. The objectives were required for toxic pollutants that had been identified in know or suspected toxic hot spots and for toxic pollutants that the Water Boards had identified as pollutants of concern. The objectives had to be established with an adequate safety margin to reasonably protect beneficial uses and to prevent nuisance. (Wat.

Code §13391.5(d).) Further, the objectives had to ensure adequate protection for the most sensitive aquatic organisms. (Wat. Code §13393.) If humans could be exposed to pollutants through the food chain, the objectives had to based on a health risk assessment. (*Ibid.*)

After January 1, 1993, Water Code section 13396 prevents the Water Boards from approving a dredging project that involves the removal or disturbance of sediment which contains pollutants at or above the sediment quality objectives established pursuant to Section 13393 unless the board determines all of the following:

(a) *The polluted sediment will be removed in a manner that prevents or minimizes water quality degradation.*

(b) *Polluted dredge spoils will not be deposited in a location that may cause significant adverse effects to aquatic life, fish, shellfish, or wildlife or may harm the beneficial uses of the receiving waters, or does not create maximum benefit to the people of the state.*

(c) *The project or activity will not cause significant adverse impacts upon a federal sanctuary, recreational area, or other waters of significant national importance.*

Funding for the program was provided under former Water Code section 13396.5, which authorized the Water Boards to collect fees from point and nonpoint dischargers that discharged into enclosed bays, estuaries, or adjacent waters to fund the program. The fee period was limited under section 13396.5(h) to January 1, 1998. After that date. the program was no longer fee-funded.

4.2.4 Hazardous Waste Site Cleanups

U.S. EPA, Regional Water Boards and DTSC share responsibility for providing regulatory oversight for the cleanup of hazardous waste sites. The extent of site cleanup actions are based upon the desired goals and end uses established for the site, the evaluation of risks to human health and the environment at the site, and the selection of appropriate management alternatives that will reduce the risks to acceptable levels that are consistent with the desired goals and end uses. In order to evaluate existing risks and potential future risks, conceptual models are prepared that identify receptors potentially at risk and the probable exposure pathways. This conceptual model serves as the basis for formulating the human health and ecological risk assessment. At sites where polluted sediments are the primary concern, receptors commonly evaluated include:

- benthic communities exposed directly to pollutants in sediment,
- fish exposed directly to pollutants in sediment or indirectly through consumption of pollutants in prey tissue or
- birds, marine mammals and humans also exposed indirectly through consumption of pollutants in prey tissue.

For many receptors, risk is estimated by comparing pollutant concentrations in sediments and prey tissues to calculated risk thresholds developed specifically for those receptors. For other receptors, such as benthic invertebrates, direct measurements such as benthic community metrics, sediment toxicity and chemistry may be applied instead. Typically, those most sensitive receptors identified will become the focus of the remedial effort. Water quality objectives may be utilized to assess where the objective is based upon the receptor of concern and reflects the appropriate exposure pathway. However many aquatic life and human health based water quality objectives were not derived to protect these receptors from the exposure

pathways that exist at the site such as trophic transfer and bioaccumulation (U.S EPA 1985). Although risk assessments may guide the development of appropriate cleanup targets, the targets must comply with State Water Board Resolution No. 92-49.

4.3 Maintenance and Navigation Dredging

Dredging to maintain ports and waterways generates approximately 300 million cubic yards of material annually that requires characterization and disposal (U.S. EPA 1998). Maintenance dredging differs from sediment quality assessments described above because the goal of the programs is to maintain safe navigation. For dredging projects, the assessment is performed in order to identify appropriate disposal sites and controls that may be required to minimize environmental impacts associated with the disposal. Dredge materials are also characterized differently than ambient surface sediments. When assessing dredge materials, often only a small percentage of the material slated for disposal is present as surficial sediment. As a result, dredged materials characterization requires samples collected from multiple depths to adequately characterize the material.

4.3.1 Clean Water Act Section 404/MPRSA

There are three principal acts for the federal regulation of dredging and disposal operations in the United States. These are the Clean Water Act , the Marine Protection Research and Sanctuaries Act (MPRSA) and the Rivers and Harbors Act (RHA). Only the Clean Water Act and MPRSA prescribe the need to assess the quality of the sediment for disposal purposes.

The discharge of dredged or fill materials into "waters of the United States" is regulated under Section 404 of the Clean Water Act. Under section 404, applicants are required to seek permits from the U.S. Army Corp of Engineers (USACE) for proposed discharges of dredged material into waters of the U.S. with concurrence by U.S. U.S. EPA. Under Section 404, U.S. EPA and USACE have jointly developed an effect-based testing program to assess the suitability of dredged materials for inland waters in the USACE/U.S. EPA. Document titled "Evaluation of Dredged Material Proposed for Discharge in Waters of the U.S. – Inland Testing Manual (1998) commonly referred to as the Inland Testing Manual or ITM. USACE/U.S. EPA. 1998. The ITM utilizes a tiered, effects-based evaluation scheme to determine the suitability of dredged material for aquatic placement or disposal. Unlike other programs that only assess surficial sediments, dredge materials characterization requires that the sediment be evaluated to the anticipated maximum depth of the proposed activity. Therefore, none of the tools adopted in this program are depth dependent.

The ITM recognizes three distinct exposure pathways for a suitability determination

1. Water column toxicity
2. Benthic toxicity
3. Benthic bioaccumulation.

Suitability determinations for aquatic discharge of dredged material take into account not only the technical sediment test results from the ITM, but also the characteristics of the individual disposal sites and the practicability of alternatives to aquatic disposal (including beneficial reuse alternatives).

Tier I of the suitability determination consists of gathering all available chemical, biological, and physical data and information on the source area or waterbody. The information is assessed relative to the characteristics of the disposal site. If enough information is available, a

suitability determination can be made within Tier 1 without the need for additional testing. If insufficient information is available, the suitability determination would proceed to Tier 2.

The ITM requires Tier II to evaluate the potential for the disposal to cause an exceedance of water quality standards and the potential for the disposal to impact benthic organisms. To assess the potential exceedance of water quality standards outside the mixing zone, either a numerical mixing model or the chemical analysis of the sediment or elutriate are utilized. The Theoretical Bioaccumulation Potential (TBP) is used to screen potential impacts to benthic organisms. The TBP is a product of the chemical concentration in the sediment normalized to total organic carbon, the biota sediment accumulation factor, and the lipid content of the test organism. This result is compared to the results from a reference site.

The focus of Tier III is on toxicity and bioaccumulation tests. Water column toxicity is evaluated by exposing a sensitive test organism to the elutriate. To make a suitability determination, the LC50 or EC 50 concentrations are assessed after allowing for dilution/mixing to determine if there is potential for water column toxicity. Toxicity of the sediment is evaluated by exposing a benthic organism to the bulk sediment. Sediment toxicity suitability is based on comparison to a reference site. Results from the 28-day bioaccumulation are compared with accepted human health benchmarks such as those published by the Food and Drug Administration.

Tier IV is a more rigorous and site-specific evaluation of toxicity and bioaccumulation. This could include using tests of longer duration, or using other sensitive species and endpoints. Although Tier IV provides the greatest flexibility, the staff from USACE, U.S. EPA and the State must approve the proposed approach, test methods, and corresponding analysis before this study can be initiated.

None of the methods or analyses described in the ITM are intended to assess the quality of bedded undisturbed surface sediments, rather the methodology was developed solely to assess the risk associated with disposal.

Ocean disposal is not regulated under the Clean Water Act, these actions fall under the MPRSA. Section 103 regulates transportation of dredged material for the purpose of ocean disposal (i.e., outside the three mile baseline). Under the MPRSA, the U.S. EPA has the lead in the designation of suitable disposal sites and the USACE in consultation with U.S. EPA issues the permit. Since ocean disposal by definition falls outside state jurisdiction, the state generally has limited regulatory authority for permitting disposal under MPRSA. Like the ITM, the Ocean Testing Manual or OTM is also based upon a tiered, effects-based evaluation scheme to determine the suitability of dredged material for aquatic placement or disposal. The tiered scheme follows the same general approach and methodology utilized for the ITM. The OTM is also not intended for uses to assess the quality of bedded surface sediments.

Under the Clean Water Act there is an allowance for greater flexibility with the level of information required differing for different regions of the country. Differences in the regional implementation of the 404 requirements exist between Northern and Southern California as to the extent and nature of information required. In Northern California for example, suitability determinations for in-bay disposal in San Francisco Bay generally require solid and suspended phase toxicity data but rarely require information on bioaccumulation. In both Northern and Southern California, if an area proposed for dredging has been tested within the past 3 years, then there is an allowance for a "Tier I exclusion with confirmatory sediment chemistry" which means the material is exempted from any effects-based testing so long as the sediment chemistry is similar to what previously has been deemed suitable based upon results of earlier testing.

In Southern California, there are fewer options for a Clean Water Act section 404 disposal (i.e., most material is either ocean disposed under MPRSA, used beneficially for beach replenishment, or managed upland). Material being placed beneficially for beach nourishment generally does not require bioassay testing because only clean materials with grain size compatible with the proposed receiver site are eligible for beach replenishment. The clean sands typically required for stability in high energy environments have little or no ability to bind with pollutants because of the low organic carbon content and limited binding capacity of the minerals that make up most sand size particles.

When there are opportunities for confined or unconfined in-water placement at areas other than approved ocean disposal sites, the Corps' and U.S. EPA regulations allow for materials to be excluded from testing if acceptable engineering controls are available to contain potentially contaminated materials, or if the material is of such a large grain size that contaminants should not be present. When material is placed as a nearshore or upland fill and there is a return flow or exchange with water of the U.S., then typically sediment chemistry and possibly elutriate chemistry may be required. In those instances where there is little or no recent information and/or there is a reason to believe that sediment-associated contaminants are present, then a full suite of chemical and sediment toxicity and bioaccumulation testing may be required.

4.3.2 Water Quality Certifications

Clean Water Act section 401 allows states to deny or grant water quality certification for any activity which may result in a discharge to waters of the United States and which requires a federal permit or license. Certification requires a finding by the State that the activities permitted will comply with all water quality standards individually or cumulatively over the term of the permit. Certification must be consistent with the requirements of the Clean Water Act, CEQA, the California Endangered Species Act (CESA), and the State Water Board's mandate to protect beneficial uses of waters of the State.

The State Water Board considers issuance of water quality certifications for the discharge of dredged and fill materials. Clean Water Act section 401 allows the State to grant or deny water quality certification for any activity which may result in a discharge to navigable waters and which requires a federal permit. State Water Board regulations (Cal. Code Regs., tit. 23, §3830 et seq.) provide the regulatory framework under which the State Water Board issues water quality certifications. The Corps may not issue a Section 404 permit if the State denies water quality certification.

In order to certify a project, the State Water Board must certify that the proposed discharge will comply with all of the applicable requirements of Clean Water Act sections 301, 302, 303, 306, and 307 (42 U.S.C. §§ 1311, 1312, 1313, 1316, and 1317). Essentially, the State Water Board must find that there is reasonable assurance that the certified activity will not violate water quality standards. Clean Water Act section 401 requires the water quality certification process to comply with the Clean Water Act section 404(b)(1) Guidelines.

In California, wetlands are also regulated through under Clean Water Act section 401. Seasonally and permanently flooded wetlands are sites for methylmercury production due to the presence of sulfate-reducing bacteria in wetland environments (CVRWQCB, 2005a). Wetlands can be significant sources of methylmercury production; for example, the Central Valley Water Board (2005c) estimated that 21,000 acres of wetland in the Sacramento-San Joaquin River Delta produce about 16% of the annual methylmercury load to the watershed. A complicating issue is that wetland restoration efforts are ongoing because wetlands provide important services for ecosystems and human communities.

Management practices to reduce methylmercury discharge could include aeration, changing the stream channel, revegetation, sediment removal, and levees. Some of these practices may be applied upstream to reduce inorganic mercury in water flowing into the wetland, thus reducing methylmercury formation. Other practices may reduce the downstream transport of methylmercury formed in the wetland (CVRWQCB, 2005b).

In March of 2007, the State Water Board circulated a CEQA scoping document announcing the States intent to develop and propose for adoption a Wetland and Riparian Area Protection Policy.

4.4 Point Sources Regulated under Clean Water Act §402

As explained previously, the Water Boards issue and administer NPDES permits in California. Under the Clean Water Act, all point source discharges of pollutants to waters of the United States must be regulated under a permit. Thus, all point source discharges of toxic pollutants to enclosed bays and estuaries must be regulated under an NPDES permit.

Under the NPDES permit program, discharges are regulated under permits that contain both technology-based and water quality-based effluent limits. Water quality-based effluent limits are developed to implement applicable water quality standards As discussed in section 4.1 above, applicable water quality standards for toxic pollutants include narrative and numeric objectives and CTR criteria. The State Water Board's SIP addresses the implementation of numeric toxic pollutant criteria and objectives for bay, estuarine, and inland surface waters.

Typical discharges that are regulated under NPDES permits include discharges from publicly-owned treatment works and industrial facilities. In addition, storm water discharges are regulated under the permit program. The following subsection explains the State Water Board's storm water permit program.

4.4.1 Storm Water

The State Water Board has three distinct storm water programs – municipal, industrial, construction- and a fourth that encompasses parts of the other three because of the number, diversity and geographic extent of the discharges. This fourth program, referred to as Caltrans, describes the stormwater permits associated with the California Department of Transportation

Municipal Discharges

The municipal program regulates storm water discharges from municipal separate storm sewer systems (MS4s). Large (Phase I) and small (Phase II) MS4s implement best management practices (BMPs) to comply under the program. BMPs include both source controls and treatment measures. The Clean Water Act and implementing federal regulations require MS4s subject to NPDES permits to reduce pollutants in storm water to the maximum extent practicable (MEP). The regulations require implementation of BMPs to meet the MEP discharge standard. In California, MS4 permits also require permittees to reduce the discharge of pollutants so that water quality standards are met. This is usually accomplished under a storm water management plan (SWMP).

Industrial Discharges

Under the industrial program, the State Water Board issues a general NPDES permit that regulates discharges associated with ten broad categories of industrial activities. This general permit requires the implementation of management measures that will achieve the performance

standard of best available technology economically achievable (BAT) and best conventional pollutant control technology (BCT) and achieve the water quality standards. The permit also requires that dischargers develop a Storm Water Pollution Prevention Plan (SWPPP) and a monitoring plan. Through the SWPPP, dischargers are required to identify sources of pollutants, and describe the means to manage the sources to reduce storm water pollution. For the monitoring plan, facility operators may participate in group monitoring programs to reduce costs and resources.

Construction

The construction program requires dischargers whose projects disturb one or more acres of soil or whose projects disturb less than one acre but are part of a larger common plan of development that in total disturbs one or more acres to obtain coverage under the general permit for discharges of storm water associated with construction activity. The construction general permit requires the development and implementation of a SWPPP that lists BMPs the discharger will use to control storm water runoff and the placement of those BMPs. Additionally, the SWPPP must contain a visual monitoring program; a chemical monitoring program for non-visible pollutants to be implemented if there is a failure of BMPs; and a sediment monitoring plan if the site discharges directly to a water body impaired for sediment.

Caltrans

In 1996, Caltrans requested that the State Water Board consider adopting a single NPDES permit for storm water discharges from all Caltrans properties, facilities, and activities, which would encompass both the MS4 requirements and the statewide construction general permit requirements. The State Water Board issued the Caltrans general permit in 1999, requiring Caltrans to control pollutant discharges to the MEP for the MS4s and to the standard of BAT/BCT for construction activities through BMPs. The State Water Board also required Caltrans to implement more stringent controls, if necessary, to meet water quality standards.

4.5 Nonpoint Source Control

Under Porter-Cologne, all waste discharges that could affect water quality must be regulated, including nonpoint source discharges of pollution. Nonpoint source (NPS) pollution, unlike point source pollution from industrial and sewage treatment plants, comes from many diffuse sources. Some types of NPS pollution are caused by rainfall or snowmelt moving over and through the ground. As the runoff moves, it picks up and carries away natural and human-made pollutants, depositing them into lakes, rivers, wetlands, coastal waters, and groundwater. NPS pollution may originate from several sources, including agricultural runoff, forestry operations, urban runoff, boating and marinas, active and historical mining operations, atmospheric deposition, and wetlands.

Nonpoint sources in California must be regulated under waste discharge requirements (WDRs), conditional waivers of WDRs, or basin plan prohibitions. However, WDRs need not necessarily contain numeric effluent limits. The state's Policy for Implementation and Enforcement of the Nonpoint Source Pollution Control Program (NPS Policy) provides guidance regarding the prevention and control of nonpoint source pollutant discharges and enforcement of nonpoint source regulations (e.g., WDRs). In practice, the Regional Water Boards do not usually impose numeric effluent limits on nonpoint pollution sources; rather they primarily rely on implementation of BMPs to reduce pollution.

In 1998, California began implementing its Fifteen-Year Program Strategy for the Nonpoint Source Pollution Control Program, as delineated in the Plan for California's Nonpoint Source

Pollution Control Program (NPS Program Plan). The legal foundation for the NPS Plan is the Clean Water Act and the Coastal Zone Act Reauthorization Amendments of 1990 (CZARA) (SWRCB, 2000), and state law. The agencies primarily responsible for the development and implementation of the NPS Program Plan are the State Water Board, the nine Regional Water Boards, and the California Coastal Commission (CCC). Various other federal, state, and local agencies have significant roles in the implementation of the NPS Plan.

The NPS Program Plan addresses six categories of nonpoint sources including agriculture, forestry, urban areas, marinas and recreational boating, hydromodification, and wetlands/riparian areas/vegetated treatment systems. For each category, the NPS Program Plan specifies management measures (MMs) and the corresponding management practices or BMPs. The NPS Program Plan provides five general goals:

- Track, monitor, assess, and report NPS Program activities
- Target NPS Program activities
- Coordinate with public and private partners in all aspects of the NPS Program
- Provide financial and technical assistance and education
- Implement MMs and associated BMPs

The following sections discuss the objectives and policies relevant to sediment quality for specific NPS sources.

4.5.1 Agriculture

Impacts from agricultural activities that may affect sediment quality include sedimentation and the runoff of pesticides. These impacts can be caused by:

- Farming activities that cause excessive erosion, resulting in sediment entering receiving waters
- Improper use and over-application of pesticides
- Over-application of irrigation water resulting in runoff of sediments and pesticides (SWRCB, 2006b).

Although wastewater discharges from irrigated land, including stormwater runoff, irrigation tail-water, and tile drainage are subject to regulation under Porter-Cologne, the Regional Water Boards have historically regulated these discharges under waivers as authorized by Water Code section 13269. This section allows the Regional Water Boards to waive the requirement to have waste discharge requirements if it is in the public interest and the waiver is consistent with any applicable water quality control plans. Although waivers are always conditional, the historic waivers had few conditions. In general, they required that discharges not cause violations of water quality objectives, but did not require water quality monitoring.

In 1999, Senate Bill 390 was enacted into law. The law amended section 13269 and required Regional Water Boards to review and renew their waivers, or replace them with waste discharge requirements. Waivers not reissued automatically expired on January 1, 2003.

To comply with SB 390, as well as to control and assess the effects of these discharges, the Los Angeles, Central Coast, Central Valley, and San Diego Water Boards have adopted comprehensive conditional waivers. An estimated 80,000 growers, who cultivate over 9 million acres, are subject to conditional waivers in the Central Coast, Los Angeles, and Central Valley regions. These Regional Water Boards have made significant strides to implement their waiver programs and are committed to continue their efforts to work with the agricultural community to protect and improve water quality. The number of acres and agricultural operations will

increase as other Regional Water Boards adopt conditional waivers for discharges from irrigated agricultural land. The North Coast, San Francisco Bay and Lahontan Water Boards have no immediate plans to adopt waivers for agricultural discharges, but may do so eventually to implement TMDLs. The Santa Ana Water Board is in the process of developing a conditional waiver for discharges from irrigated agricultural lands.

In conjunction with the conditional waivers, Regional Water Boards regulate agricultural discharges from cropland under NPS programs that rely on BMPs to protect water quality. For example, the State Water Board and the CCC oversee agricultural control programs, with assistance from the Department of Pesticide Regulation (DPR) for pesticide pollution and the Department of Water Resources for irrigation water management (SWRCB, 2006b).

The pesticide management measure (MM 1D) is likely to have the greatest impact on sediment toxicity. This MM reduces contamination of surface water and ground water from pesticides through:

- Development and adoption of reduced risk pest management strategies (including reductions in pesticide use)
- Evaluation of pest, crop, and field factors
- Use of Integrated Pest Management (IPM)
- Consideration of environmental impacts when choosing pesticides for use
- Calibration of equipment
- Use of antibackflow devices (SWRCB, 2006b).

IPM is a key component of pest control. IPM strategies include evaluating pest problems in relation to cropping history and previous pest control measures, and applying pesticides only when an economic benefit will be achieved. Pesticides should be selected based on their effectiveness to control target pests and their potential environmental impacts, such as persistence, toxicity, and leaching potential (SWRCB, 2006b).

There are many planned, on-going, and completed activities related to management of pesticides. However, as reported in the most recent NPS Program Plan progress report (SWRCB, 2004a), efforts to improve water quality impaired by agriculture activities are highly challenging because of the different perspectives that exist between the regulatory community and the agricultural community.

As of 2003, the SWRCB (2004a) reports the following progress:

- 16 watershed working groups are actively developing farm water quality plans, with 19 new groups being formed
- Of the over 90 farmers that attended a farm water quality course, half have developed comprehensive water quality plans for more than 10,700 acres of irrigated crops
- Over 750 farmers have attended 35 workshops designed to train farmers in specific conservation practices.

To address local issues, the Regional Water Boards adopted conditional waivers that use different regulatory models, as follow:

- **Central Coast Region:**
  - Requires the submittal of a Notice of Intent (NOI) for each grower;

- Several waiver conditions were based on recommendations developed by an advisory panel of agricultural and environmental representatives, including individual enrollment, education, farm plan development and a checklist of management practices.
- For group and individual waivers, the focus of monitoring is primarily nutrients and toxicity. A region-wide Monitoring and Reporting Program, adopted by the board, includes provision for follow-up monitoring when water quality objectives are exceeded or toxicity is detected.
- Requires 15 hours of training in farm water quality management. The training is funded through grants in some cases, in others education is provided by cooperators throughout region.
- Requires development of farm water quality management plans that address, at a minimum, irrigation management, nutrient management, pesticide management, and erosion control; and implementation of management practices identified in their plans (CCRWQCB, 2006a).

- **Los Angeles Region:**
  - Provision for individual growers to participate in a group. Groups will submit one NOI for all documented participants in the group. NOI to discharge for all dischargers includes individual grower description of location, crop type, and management practices. A Monitoring Plan is submitted with NOI;
  - Requires the submittal of NOI's for each individual grower that does not participate in an approved group;
  - Monitoring can be performed after the Regional Water Board issues a Notice of Applicability (NOA) to participate. NOA is provided within 6 months of NOI submittal;
  - Monitoring is conducted twice in wet weather and twice in dry weather for physical parameters, nutrients, and pesticides. Individual dischargers monitor surface water at the end of property. Group dischargers monitor surface water and watershed-wide receiving water;
  - A Corrective Action Plan (CAP) with time-specific management modifications is required when routine monitoring shows the basin plan, CTR, or TMDL limits are not attained;
  - Requires 8 hours of training in farm water quality management. Annual monitoring plan requires evidence of education.
- **Central Valley Region:**
  - Group participation emphasized;
  - NOI required of each grower that chooses to acquire an Individual Waiver. For a Group, the coalition submits one NOI on behalf of the participating growers.
  - Coalitions required to submit participant lists and update annually.
  - Two step communication report and then Management Plan request (via Executive Officer) to correct problems.
  - Monitoring plan submitted in second year after group receives approval to participate.
  - Timeline for compliance with water quality objectives is no later than 10 years.
  - The Central Valley does not require education or training.

- **San Diego Region:**
  - Conditional Waiver adopted by the R9 Water Board on October 10, 2007 that includes the following requirements.
  - Operators must perform a self assessment to identify the pollutants present on the site and assess the potential for runoff and/or infiltration to adversely affect the quality or beneficial uses of the waters of the state .
  - Agricultural and nursery operators must complete at least 2 hours of water quality management related training annually.
  - Agricultural and nursery operations must implement MMs/BMPs to minimize or eliminate the discharge of pollutants that may adversely impact the quality or beneficial uses of waters of the state.
  - Agricultural and nursery operators must maintain records pertaining to the water quality management efforts for the operation.
  - No later than December 31, 2010, agricultural and nursery operations must form or join a monitoring group.
  - No later than January 1, 2011, owners/operators of agricultural and nursery operations must file a Notice of Intent, as either an individual operation or as part of a monitoring group, with the San Diego Water Board.
  - Currently the County Farm Bureau is working with operators to form a region-wide monitoring group with the intent to submit a NOI to the Regional Water Board by December 31, 2010.

4.5.2 Forestry

Timber harvesting and associated activities can result in the discharge of chemical pollutants and petroleum products, in addition to other conventional pollutants. Chemical pollutants and metals can be discharged through runoff and drift. Potential sources of chemical runoff include roads that have been treated with oils or other dust suppressing materials and herbicide applications.

Forest chemical management focuses on reducing pesticides that are occasionally used for pest management to reduce mortality of desired tree species, and improve forest production. Pesticide use on state or private forestry land is regulated by DPR. However, a large proportion of California's forested lands are owned or regulated by the federal government (SWQCB, 2004a), and the U.S. Forest Service (USFS) Region 5 controls pesticide use.

In addition to the NPS Program MMs, forestry activities are also controlled through WDR and conditional waivers. Recently, Regional Water Boards have adopted conditional waivers for timber harvesting activities, which require compliance with applicable requirements contained in each region's basin plan.

DPR regulates the sale and use of pesticides and, through county agricultural commissioners (CACs), enforces laws pertaining to pesticide use. CACs inspect pesticide applications to forests and ensure that applications do not violate pesticide laws and regulations. Landowners must also submit timber harvest plans (THPs) to the California Department of Forestry (CDF) outlining what timber will be harvested, how it will be harvested, and the steps that will be taken to prevent damage to the environment. CDF will only approve those THPs that comply with all applicable federal and state laws.

4.5.3 Urban Runoff

Pollutants found in runoff from urban areas include, among others, sediments, heavy metals, petroleum hydrocarbons, and plastics. As population densities increase, pollutant loadings generated from human activities also increase. Most urban runoff enters surface waters without undergoing treatment.

Urban runoff is addressed primarily through the NPDES program, although the State Water Board's NPS Program applies where runoff is not regulated as a permitted point source. The NPDES program supersedes the Water Boards' NPS Program in the areas where there is overlap. As mentioned in Section 4.4.1, NPDES storm water permits typically require implementation of BMPs, which may or may not be similar to the MMs in the NPS Program.

The control of urban NPS pollution requires the use of two primary strategies: preventing pollutant loadings from entering waters and reducing the impact of unavoidable loadings. The major opportunities to control NPS loadings occur during the following three stages of development: (1) the siting and design phase, (2) the construction phase, and (3) the post-development phase. Before development occurs, land in a watershed is available for a number of pollution prevention and treatment options, such as setbacks, buffers, or open space requirements, as well as wet ponds or constructed urban runoff wetlands that can provide treatment of the inevitable runoff and associated pollutants. In addition, siting requirements and restrictions and other land use ordinances, which can be highly effective, are more easily implemented during this period. After development occurs, these options may no longer be practicable or cost-effective.

In 1976, the State Legislature enacted the California Coastal Act to provide for the conservation and planned development of the State's coastline. The Coastal Act directs each of the 73 coastal cities and counties to prepare, for review and certification by the CCC, a local coastal plan (LCP) consisting of land use plans, zoning ordinances, zoning district maps, and, other implementation actions. The CCC also works with local governments to incorporate urban MMs and MPs into their respective LCPs. Certified LCPs are important tools for implementing urban runoff MMs and MPs that prevent, reduce or treat polluted runoff from proposed developments. Storm water programs can become more effective because of local planning and permitting decisions throughout the State.

4.5.4 Marina and Recreational Boating

Poorly planned or managed boating and related activities (e.g., marinas and boat maintenance areas) may threaten the health of aquatic systems and pose other environmental hazards. Sources include poorly flushed waterways, pollutants discharged from boats (recreational boats and commercial boats), and pollutants generated from boat maintenance activities on land and in the water (SWRCB, 2006b). For example, as mentioned in Section 2.1.1, copper is often found in marina sediments due to the leaching of antifoulant paints.

There are many planned, on-going, and completed activities related to NPS pollution in marinas. The primary focus of these activities is to prevent discharges of waste oil, sewage, petroleum, solid waste, and hazardous substances from surface runoff, improper boat cleaning/maintenance activities, lack of disposal facilities, or improper maintenance of facilities at marinas. The state relies on education and outreach efforts aimed at marina owners and operators, and the boating public, to provide information on pollution problems and management practices that can be implemented to prevent or control improper disposal of pollutants into surface waters (SWRCB, 2006b).

The Federal Oil Pollution Act (OPA) is a comprehensive prevention, response, liability, and compensation regime for dealing with vessel- and facility-generated discharges of oil or hazardous substances. Under the OPA, any hazardous waste spill from a vessel must be reported by the owner of the vessel, and vessel owners are responsible for any costs of a resulting environmental cleanup and any damage claims that might result from the spill. Marinas are responsible for any oil contamination resulting from their facilities, including dumping or spilling of oil or oil-based paint and the use of chemically treated agents. The Department of Fish and Game's Office of Spill Prevention and Response enforces the laws designed to prevent spills, dispatches units to respond to spills, and investigates spills.

Note that commercial and military ports are subject to storm water NPDES permits regulating industrial and construction activities. Commercial ports are also required to submit a port master plan to the CCC. The master plan must include an estimate of the effect of development on habitat areas and the marine environment, a review of existing water quality, habitat areas, and quantitative and qualitative biological inventories, and proposals to minimize and mitigate any substantial adverse impact. In addition, the state has the opportunity to ensure that appropriate pollution prevention and control measures are in place at all military ports.

Obstacles facing the implementation of BMPs related to MMs for marinas can be primarily attributed to the insufficiency of the number of regulatory or inspection authorities relative to the number of registered boats and marinas, as well as other budgetary constraints that affect marina programs and activities. There are nearly 1 million registered boats and approximately 650 marinas in California. Marinas and boaters fall under the jurisdiction of multiple State and local agencies. In many cases, marina facilities are not being regulated and are rarely inspected. NPS pollution in marinas is often seen as a low priority for many regulatory agencies, and boating enforcement actions have primarily been in the area of boater safety (SWRCB, 2004a).

4.5.5 Abandoned and Active Mines

The State Water Board and Regional Water Boards have identified approximately 40 mines that cause serious water quality problems resulting from acid mine drainage and acute mercury loading (SWRCB, 2000). Although all mines may not be significant polluters individually, cumulatively mines may contribute to chronic toxicity due to increased metals loadings. Additionally, drainage structures and sluices associated with abandoned hydraulic gold mines are a potential source of mercury to surface waters. Mercury from abandoned mines poses a serious potential threat to coastal waters because mercury transported from these sites may bioaccumulate in fish.

The NPS Program Plan does not contain management measures for abandoned mines, and there is no specific, comprehensive program at either the state or federal level for cleaning up abandoned and inactive mines other than coal. Rather, abandoned and inactive mine cleanup is carried out under a variety of state, federal, and local programs. Regional Water Boards may issue WDRs to the most serious sites. The federal Superfund Program addresses only the most extreme pollution sites, such as Iron Mountain Mine. Federal land management agencies have specific, marginally funded programs for cleaning up abandoned mines on federal land, but most projects address safety hazards rather than water quality. California's Title 27 Program regulates discharges of wastes to land, and can be used to pursue mine cleanups.

Enforcement actions, however, are costly and have not been effective because responsible parties are difficult to locate, and current property owners either do not have, or will not spend money, to clean up their sites. The main barrier to a comprehensive program for

abandoned mines is liability. Under the federal Clean Water Act, a third party can sue an agency or private party that performs abatement actions at an abandoned mine if the discharge from the mine continues to violate the Clean Water Act.

In June 2000, the Department of Conservation (DOC) inventoried the number of abandoned mine sites in California. DOC estimates that of the 47,084 historic and inactive mine sites in the state, approximately 11% (5,200) present an environmental hazard. The most common hazards include heavy metals from acid rock drainage and methylmercury from mercury contaminated sediments. DOC (2000) indicates that some bays have been or could be impacted by acid rock drainage and mercury from abandoned mines.

As a land-managing agency, the USFS also has an abandoned mine reclamation program. The program includes an inventory of abandoned mines and locations, environmental and/or resource problems present, rehabilitation measures required, and potential sources of funding. The USFS has worked with various Regional Water Boards on numerous occasions in the rehabilitation of mine sites. Restoration funding comes from USFS funds, CERCLA, and RCRA sources. All lands disturbed by mineral activities must be reclaimed to a condition consistent with resource management plans, including air and water quality requirements (SWRCB, 2000; SWRCB, 2003). In addition, the Bureau of Land Management (BLM) has an extensive abandoned mine land program.

All active mining projects must comply with the Surface Mining and Reclamation Act (SMARA). The goal of SMARA is to have mined lands reclaimed to a beneficial end use. Local Enforcement Agencies (LEAs), usually counties, implement SMARA. The DOC's Office of Mine Reclamation provides technical support to LEAs but has limited enforcement authority.

Mining projects that could impair water quality or beneficial uses may also be subject to NPDES permits or conditions under the Clean Water Act section 401 Water Quality Certification Program.

4.5.6 Atmospheric Deposition

Atmospheric deposition may be a potential NPS to bays through either direct or indirect deposition. Indirect deposition reflects the process by which metals and other pollutants such as PAHs deposited on the land surface are washed off during storm events and enter surface water through storm water runoff (LARWQCB, 2005a). For example, Sabin (2005) concluded that atmospheric deposition potentially accounts for as much as 57–100% of the total trace metal loads in storm water within Los Angeles. In the Los Angeles Region (LARWQCB 2005a, 2005b), loadings associated with indirect atmospheric deposition are included in the storm water waste load allocations. Therefore, NPS pollution from atmospheric deposition is not directly addressed, but indirectly addressed through storm water management. Typically, direct deposition accounts for a very small fraction of NPS pollution (for example, see LARWQCB, 2005a and LARWQCB, 2005b).

Currently, there are no policies in California to directly address potential NPS pollution from atmospheric deposition. Atmospheric deposition is also not directly addressed in the NPS Program Plan, and only MM 2G (Fire Management) would address possible pollution of PAHs from forest fires.

5. ISSUES AND ALTERNATIVES

This section describes the major policy related issues identified and alternatives that have been considered by staff during the development of Part 1. Each issue analysis contains the following sections:

Issue—The subject matter or brief question framing the issue followed by an explanation or description of the issue and concerns.

Issue Description—A description of the issue or topic and (if appropriate) any additional background information, list of limitations and assumptions, descriptions of related programs or other information.

Baseline—A description of how the State and Regional Water Quality Control Boards (Regional Water Boards) currently act on the issue.

Alternatives—For each issue or topic, at least two alternatives are provided for consideration. Each alternative is evaluated with respect to the program needs and the appropriate sections within Division 7 of the California Water Code (CWC). For those issues that address scientific questions, the SQO Scientific Steering Committee's position is also stated.

Staff Recommendation—In this section, a recommended alternative (or combination of alternatives) is identified and proposed for adoption by the State Water Board.

Example Language—Following each recommendation, the reader is directed to proposed language in Part 1 presented in Appendix A where applicable. Presented in Appendix C is the analysis of a data set as applied using the indicators and thresholds described in Part 1.

5.1 PROJECT ALTERNATIVES

5.1.1 No Project Alternative

CEQA requires that the State Water Board consider the "No-Project" alternative. As explained in Section 4 above, the basin plans for all coastal regions have narrative water quality objectives or prohibitions that apply to sediment quality. These objectives currently provide the basis for Clean Water Act section 303(d) listings, cleanup orders, and other regulatory actions. If this project is not adopted, the assessment of sediment quality does and would continue to occur; however, the lines of evidence, test organisms, community indices and data reduction and analysis would continue to differ significantly by Region. These factors not only limit consistency amongst the regions, but also lower the confidence in, and technical basis for, these assessments. The No-Project" alternative does not comply with the mandate in chapter 5.6 or the judgment against the State Water Board for failure to comply with the mandate. The "No Project" alternative would not achieve the objectives of the proposed action. Additional discussion of this alternative is presented in Section 6.

Alternative 1—Adopt the no project alternative. As state above the "No Project" alternative would not achieve the objectives of the proposed action.

Alternative 2—Do not adopt the no project alternative.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A.

5.1.2 What Issues Should Part 1 Address?

At a minimum, the State Water Board is required to comply with the procedures in CWC §§13240 through 13247 in adopting SQOs. Section 13241 lists the factors that the State Water Board must consider when adopting water quality objectives, and section 13242 specifies the elements that must be included in a program to implement the objectives. State Water Board staff believes that sediment quality protection is significantly different from the tools and methods commonly applied to develop water column-based objectives. Therefore, additional information and implementation guidance should be provided to foster greater understanding and consistency when the SQOs are applied within the various regions.

Baseline—Not applicable.

Alternative 1—Include only the SQOs and tools and thresholds needed to implement the objectives.

Alternative 2—Include the narrative objectives and tools and thresholds needed to implement the objectives, and provide a framework that will better support the restoration of sediment quality and beneficial uses.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Section I.B.

5.2 Applicable Waters and Sediment

5.2.1 Applicable Waters

Chapter 5.6 requires the State Water Board to develop SQOs for bays and estuaries. Since 2003, State Water Board staff and the technical team have been developing SQOs and associated tools and thresholds for specific embayments in California. This focus on San Francisco Bay and enclosed bays south of Point Conception was based upon the large volume of data and an understanding of aquatic communities in these waterbodies. Sediment quality within these bays has been relatively well studied since the late 1980's when the State Water Board initiated the Bay Protection and Toxic Hotspots Cleanup Program. Through this program and others such as U.S. EPA's Environmental Monitoring and Assessment Program (EMAP), the San Francisco Estuary Institute Regional Monitoring Program (RMP), and the Southern California Coastal Water Research Project (SCCWRP) Bight 94, 98, and 03, and various site cleanup and dredging projects, a large volume of coupled biological effects and chemistry data existed for the major embayments in California. The technical team was able to rely on this data to evaluate potential indicators for use in this program and was able to refine each indicator and develop California specific thresholds to assess response. The database created for this program included over 150 studies and approximately 5,000 data points. In comparison, very few coupled data sets are available for all the estuaries, including the Sacramento San Joaquin Delta, San Pablo Bay (an estuary) and enclosed bays such as Morro and Humboldt Bays located on the central and north coast respectively.

The indicators and thresholds developed for bays cannot be applied to estuarine water bodies without undergoing rigorous assessment for a variety of reasons. Chapman et al. (2001) provides a detailed explanation of the fundamental physical and chemical differences between the two types of water bodies. The bioavailability of both hydrophobic organic and inorganic pollutants can be influenced by salinity. Chemical equilibrium may not exist within the highly dynamic environments of estuaries. While many of the organisms present in bays are also

found in estuaries, their tolerance to external stressors may vary greatly (Chapman et al. 2001). Within bays, even during wet years, the denser salt water can provide protection from osmotic shock to marine benthic organisms while estuarine organisms could be exposed to wide variations in salinity through tidal fluctuations. As result the indicators proposed for use within San Francisco Bay and enclosed bays south of Point Conception cannot be considered as reliable when applied to other water bodies until additional analyses are conducted.

Within estuaries, a different approach could be applied to interpret the narrative objective. This approach would utilize the same indicators as proposed for embayments, but would rely on a reference envelope approach to aid the assessment of sediment quality. The reference envelope approach has been applied most notably in San Francisco Bay (Hunt et al. 1998a).

This approach could be proposed for use within north and central coast bays as well. An approach for these bays could also be developed that relies on a combination of indicators developed for use in San Francisco Bay and enclosed bays south of Point Conception with the reference approach.

Baseline—Not applicable.

Alternative 1—Develop SQOs for both bays and estuaries as mandated under chapter 5.6 that utilize the same conceptual approach for all bays and estuaries, but relies on less robust interim tools in some water bodies as described above. These tools would be replaced under Phase II of the SQO program by more robust indicators and thresholds. This alternative is consistent with the Water Boards' negotiated settlement with the litigants associated with the original lawsuit described in Section 1.2.

Alternative 2—Develop SQOs and an implementation policy for bays first, followed by estuaries in a phased approach. This alternative would not fully comply with the negotiated settlement agreement approved by the Court.

Staff Recommendation—Alternative 1.

Proposed Language—See Appendix A, Sections II.B and V.C.

5.2.2 Applicable Sediments

Sediment quality programs are designed for specific needs. For example, dredged materials are frequently evaluated by collecting samples from multiple depths. This is performed because the properties of the sediment differ at depth, and characterization of the entire volume proposed for dredging is required before an appropriate disposal site can be selected (USACE/U.S. EPA. 1998). For dredged materials characterization, the USACE in coordination with U.S. EPA has designed a series of methods and tools that can be applied to deep sediments to assess risk associated with these materials relative to the disposal sites.

The State Water Board is most concerned with those pollutants that have the greatest potential to harm beneficial uses. Within contaminated sediments, the most direct exposure pathway for pollutants is through surficial sediments or the biologically active layer. In these surficial sediments, the presence of pollutants has the greatest potential to affect valuable and sensitive receptors either through direct exposure or indirectly as the pollutants in surface sediments are transferred up the food chain to piscivorous fish and birds and finally humans. This pathway was evaluated under the Bay Protection and Toxic Cleanup Program where only the upper two centimeters of sediment were sampled (Stephenson et al., 1994) and is also consistent with the conceptual approach used by Washington Department of Ecology in the regulation of polluted sediments in Puget Sound (WDOE, 1995).

Baseline—Previous assessment conducted through the Bay Protection and Toxic Hotspots Cleanup Program focused on the surficial sediments within the biological active layer. As stated in the Bay Protection and Toxic Cleanup Program Quality Assurance Project Plan, the target depth was designated as the upper two centimeters of sediment (Stephenson et al., 1994).

Alternative 1—Do not identify specific sediments applicable within the proposed Part 1. This alternative would severely limit the effectiveness of the program through inconsistent application of the indicators.

Alternative 2—Surficial sediments only. The tools that have been developed are intended solely to assess the biologically active layer.

Alternative 3—Specify a range of depths. As discussed above, the greatest risk from pollutants is with surficial sediments. Developing additional indicators and thresholds for deeper sediments would not provide enough additional value to offset the additional effort and costs to collect and evaluate this data.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Sections II.C.

5.3 Beneficial Uses and Receptors

5.3.1 Beneficial Uses Potentially Addressed in Part 1

Chapter 5.6 requires the State Water Board to develop SQOs for the reasonable protection of beneficial uses. The Water Boards are required to protect all beneficial uses designated within each water body. Beneficial uses established for bays and estuaries are identified in Table 5.1. Within the context of this program, State Water Board staff considered those beneficial uses that met the following criteria.

- Relationship between the beneficial uses and pollutants in sediment. Some beneficial uses are unaffected by pollutants in sediments. Other beneficial uses are clearly affected by pollutants in sediment but are also highly influenced by natural and anthropogenic water quality factors. Other beneficial uses are linked to pollutants in sediments that have not been considered within the context of this program such as indicator bacteria.
- Ability to utilize robust indicators to measure the potential risk to each beneficial use.
- Ability to consistently assess the risk to the beneficial use within the context of a sediment quality regulatory program.

The beneficial uses that best meet these criteria consist of Marine and Estuarine Habitat, Commercial and Sport Fishing, and Rare and Endangered Species. All of these beneficial uses can be severely affected by pollutants in sediment and assessed using the indicators described in the following Section.

Baseline—Not applicable.

Alternative 1—Attempt to develop SQOs indicators and thresholds to assess the health of all beneficial uses including Municipal, Industrial, Water Contact Recreation Non-contact Water Recreation and spawning reproduction and or early development.

Alternative 2—Beneficial uses linked to specific receptors (Examples—Marine and Estuarine Habitat, and Commercial and Sport Fishing).

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Sections III.

Table 5.1. Beneficial Uses for Enclosed Bays and Estuaries

Beneficial Uses	Description
Industrial Service Supply	Uses of water for industrial activities that do not depend primarily upon water quality including, but not limited to, mining, cooling water supply, hydraulic conveyance, gravel washing, fire protection and oil well repressurization.
Navigation	Uses of water for shipping, travel, or other transportation by private military or commercial vessels.
Water Contact Recreation (1):	Uses of water for recreational activities involving body contact with water where ingestion of water is reasonably possible. These uses include, but are not limited to, swimming, wading, waterskiing, skin and scuba diving, surfing, whitewater activities, and fishing, and uses of natural hot springs.
Non-contact Water Recreation (2):	Uses of water for recreational activities involving proximity to water but not normally involving contact with water where water ingestion is reasonably possible. These uses include, but are not limited to, picnicking, camping, boating, tide pool and marine life study, hunting, and sightseeing, or aesthetic enjoyment in conjunction with the above activities.
Ocean Commercial and Sport Fishing	Uses of water for commercial or recreational collection of fish, shellfish, or other organisms in oceans, bays, and estuaries including, but not limited to, uses involving organism intended for human consumption.
Aquaculture	Uses of water for aquaculture or mariculture operations including, but not limited to, propagation, cultivation, and maintenance or harvesting of aquatic plants and animals for human consumption or bait purposes.
Estuarine Habitat	Uses of water that support estuarine ecosystems including, but not limited to, preservation and enhancement of estuarine habitats, vegetation, shellfish or wildlife (e.g., estuarine mammals, waterfowl, shorebirds), and the propagation sustenance and migration of estuarine organism.
Marine Habitat	Uses of water that support marine ecosystems including, but not limited to, preservation or enhancement of marine habitats vegetation such as kelp, fish, shellfish, or wildlife habitats (e.g., marine mammals, shorebirds).
Preservation of Biological Habitats of Special Significance	Includes uses of water that support designated areas or habitats such as established refuges, parks, sanctuaries, ecological reserves, or Areas of Special Biological Significance where the preservation or enhancement of natural resources requires special protection.
Rare Threatened or Endangered Species	Uses of water that support habitats necessary for the survival and successful maintenance of plant or animal species established under State/or federal law as rare, threatened, or endangered.
Migration of Aquatic Organism	Uses of water that support habitats necessary for the migration, acclimatization between freshwater and salt water, and the protection of aquatic organism that are temporary inhabitants of waters within the region.
Spawning, Reproduction and/or Early Development	Uses of water that support high quality aquatic habitats suitable for the reproduction and early development of fish.
Shellfish Harvesting	Uses of water that support habitats suitable for the collection of crustaceans and filter-feeding shellfish (e.g., clams, oysters, and mussels) for human consumption and commercial or sport purposes

5.3.2 Choice of Receptors

Selection of appropriate receptors is a critical element of every standards development proposal. U.S. EPA's program to develop sediment quality criteria focused on the protection of

benthic organisms (EPA 2003 A and B). Other potential sediment-related receptors include demersal fish, aquatic macrophytes, marine birds, and mammals. Each of these receptors is essential to support a healthy ecosystem. Humans are also potentially affected through the consumption of fish tissue containing contaminant residues. Selecting a receptor as a primary indicator of beneficial use protection is relatively straightforward. For example, human health is an obvious receptor to assess Commercial and Sportfishing. Endangered species such as the least tern could be an appropriate receptor to assess Rare and Endangered Species Beneficial Uses protection. Selection of appropriate receptors to assess risk to other beneficial uses is more difficult because of the broad nature of these beneficial uses (See Table 5.1). For beneficial uses such as Estuarine Habitat and Marine Habitat, many different receptors could be applied. Within the context of this program, receptors were considered based upon the following criteria:

- Ecological Importance.
- Potential for direct or significant exposure.
- Strong link to pollutants in sediment.
- Understanding of response to pollutant exposure understood.
- Availability of tools that can reliably measure response.
- Successfully application in sediment monitoring programs within other sediment monitoring programs in the country.

Fish are an important receptor that can be affected by pollutants in sediments and pollutants that bioaccumulate up the food chain. Fish are ecologically and economically important and provide a source of food to many people. Fish are relatively long lived and exhibit a variety of responses to stress. In terms of a sediment specific receptor, fish exhibit many characteristics that limit their utility in a regulatory framework. Many fish are highly mobile, and, as a result, they can avoid highly impacted areas (Gibson et al. 2000). Their mobility also limits the ability to qualitatively assess exposure without detailed long-term studies. Mobility within unconfined water bodies such as bays and estuaries also makes it difficult to utilize community attributes as a measure of fish health. Fish populations also respond rapidly to environmental disturbance or habitat changes. External anomalies such as fin erosion, lesions, and external parasites can be more sensitive indicators of contaminant effects than community integrity and have been utilized within monitoring programs by coastal publicly owned treatment works (POTWs) or regional monitoring programs in the Southern California Bight (Schiff et al. 2001). However, these effects cannot be directly linked to pollutants in specific sediments without significant and detailed site-specific studies.

Aquatic macrophytes are the most important primary producers and provide stability to the substrate as well as critical habitat for fish and invertebrates. Aquatic macrophytes can respond to pollutants in sediments; however, water quality factors may play a more significant role (Gibson, 2000).

Benthic communities are recognized as the optimal sediment receptor for several reasons. They play a critical role in aquatic ecosystem health because they:

- Digest a significant portion of the organic detritus that settles out in bays and estuaries.
- Significantly enhance sediment mixing and oxygenate deeper sediments that stimulate bacteria-driven biogeochemical processes.
- Create habitat that enhances recruitment for other organisms.

- Provide food for most fish species that utilize bays and estuaries. Waterfowl and wetlands birds also rely on benthic invertebrates as a primary food source.

As an aquatic life indicator of sediment quality, benthic communities also exhibit the following characteristics (Jackson et al. 2000, Gibson et al. 2000):

- Benthic communities are an in-situ measure of actual conditions and biological effects that *are or have* occurred within surface sediments. Other tools commonly applied such as laboratory toxicity tests are at best surrogate measures that may or may not be reflective of actual conditions.
- Benthic invertebrates typically spend at least one or all life stages in direct contact with bottom sediments and characteristically exhibit limited range or mobility. This long-term exposure scenario allows for sublethal toxic effects to cause subtle changes in community structure. Other receptors such as fish and birds are more difficult to utilize because of their mobility and migratory life histories.
- The great variety of taxa within a healthy benthic community represents many different feeding and reproductive strategies that create a great range in sensitivity or tolerance to pollutants and other stressors. These tolerances can be used collectively to identify relatively subtle community responses above reference conditions creating a very robust tool.
- A variety of tools have been used to support the assessment of benthic community health in addition to community measures. These tools include sediment toxicity tests and empirical sediment quality guidelines (SQGs).
- Benthic communities are used by many State and federal agencies to evaluate the effects associated with impaired sediments, and to assess the effectiveness of mitigation actions. Existing data and assessment tools have been developed for many water bodies throughout the nation. While variability is always a factor when evaluating biological communities, compared to other indicators, the analysis of benthic community data does not rely on complex food web fate and transport studies and models to link a pollutant or stressor to a specific region or trophic level.

The State Water Board is required to protect all receptors associated with a specific beneficial use. However, many receptors are not understood well enough to develop tools and define appropriate thresholds for measuring the health of the receptor, or the linkage to pollutants in sediments is easily overshadowed by other factors. For these situations, ecological risk assessments are an appropriate means to assess the risk to other receptors.

Baseline—Selection of appropriate receptors for the assessment of sediment quality is site or water body specific with the final decision approved by the Regional Water Board.

Alternative 1—Consider all potential receptors including aquatic plants, plankton, and bacteria. In order to protect all receptors, detailed ecological risk assessments would be required for each water body of concern.

Alternative 2—Consider a variety of important and ecologically relevant receptors. The process could focus on only the most sensitive organisms; however, sensitivity is specific only to types or groups of pollutants. As with Alternative 1, the application of different indicators would require extensive use of best professional judgment and is counter to the argument for statewide consistency of assessment tools.

Alternative 3—Consider important, relevant, and understood receptors (benthic invertebrates, and human health) exposed either directly or indirectly to pollutants in sediments.

This alternative focuses on those sensitive and ecologically relevant receptors that have been evaluated and applied as sentinel organisms in sediment quality programs throughout the nation. This alternative would utilize the following sediment-related exposure receptor relationships:

1. Benthic communities exposed directly to pollutants in sediment.
2. Human health exposed indirectly through fish and shellfish tissue.

The receptors and corresponding exposures must be clearly described in the policy. The selection of these receptors is not intended to trivialize the importance of other receptors. Receptors such as fish and wildlife are assessed often during the assessment of contaminated sediments through ecological risk assessment. These detailed site-specific studies are the appropriate mechanism to evaluate risk to those receptors not considered within the proposed Part 1. Additional receptors can be evaluated in later phases of the program.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Sections III. and IV.

5.4 Benthic Communities Exposed Directly to Pollutants Within Enclosed Bays

5.4.1 Lines of Evidence

Water quality is routinely assessed based on a single line of evidence (LOE), chemical-specific concentration-based thresholds developed from toxicological studies. A single LOE is appropriate in the water column because the binding effects of other water column constituents are well understood, and the performance of these chemical-specific criteria is reproducible under a variety of conditions (U.S. EPA, 1985, 1991). Moreover, there is a single predominant means for chemical exposure in the water column, transport across the gills. As a result, scientists have been able to integrate this information to describe site-specific bioavailability of chemical contaminants using tools such as the Biotic Ligand Model (Paquin et al., 2002).

Sediment, however, is a more complex matrix that makes establishment of an objective based on chemical concentration alone problematic. There are two primary factors that create this complexity: variations in the bioavailability of sediment-associated contaminants, and multiple pathways of exposure resulting in both direct effects (from contact with the sediment) and indirect effects (as a result of bioaccumulation and transfer to higher trophic levels). Bulk measures of chemical concentration fail to differentiate between the fraction that is tightly bound to sediment and that which is found in interstitial waters and more available for transport across the gill. Further complicating interpretation of chemical data is that transport of chemicals in interstitial waters across the gill is not the only mechanism for exposure, as many benthic organisms ingest the sediment and can uptake chemicals sorbed onto particles. Thus, even chemical measurement approaches that attempt to differentiate interstitial chemical concentrations, such as using equilibrium partitioning models or direct measurement of pore water chemistry, do not fully describe chemical bioavailability in the sediment. Only the bioavailable fraction of pollutant has the potential to alter basic functional processes such as oxygen transfer or reproduction.

Factors that affect bioavailability of contaminants in sediment include the proportion of organic matter, grain size, hydrogen ion activity (pH), and aerobic state, salinity, chemical form of the pollutants, and the composition and mineralogy of the sediment itself (Chapman et al. 2001, U.S. EPA 2000A). These factors can create large spatial and temporal differences in

pollutant bioavailability within a given region or water body (Chapman et al., 2001, U.S. EPA 2001A).

Assessing the indirect effects of sediment contamination presents additional challenges besides those identified for direct effects. As predators consume many prey throughout their lifespan, bioaccumulative pollutants with an affinity for fatty tissue, such as DDT, polychlorinated biphenyls (PCBs), and methyl mercury can build up to levels many times greater then those observed in lower trophic levels or in the sediment (biomagnification). Numerous studies have demonstrated that the biomagnification of sediment-associated compounds can cause deleterious effects in fish and in wildlife or human consumers of seafood (Beyer et al. 1996). The presence of multiple trophic levels and different types of receptors for effects creates additional complexity and uncertainty in the interpretation of sediment contamination data.

A thorough understanding of fish communities, trophic structure and uptake, and the pollutant contribution from all sources must be assessed in order to quantifiably link sediment and fish tissue contaminant levels. Fish are highly mobile; at a given site, a portion of an organism's contaminant body burden may result from uptake from other locations, or from other sources such as the overlying water column. Although specific case studies indicate that certain contaminants are accumulated from the sediments (Gobas et al., 2002), this could vary on a site-by-site basis. Variation in home range can affect the relative impact of contamination at a specific site as a result of the heterogeneous distribution of chemicals in the sediment. Variations in food web structure among locations can also cause differences in contaminant bioaccumulation (Gobas et al., 2002).

As a result of the factors described above, sediment quality indicators based on pollutant concentrations in sediment have only limited utility when used by sediment managers unless bolstered by effects data such as toxicity and benthic community disturbance (Chapman 1990, Ingersoll et al. 2002c, Wenning et al. 2002). This limitation is acknowledged in the ecological risk assessment process, where measures of both chemical exposure and effects are required in order to evaluate the potential for adverse impacts due to either the direct or indirect effects of contaminants.

Other LOE applied to sediments also have potential flaws that make them inappropriate for establishment of SQOs when used alone. Toxicity tests improve in some ways on chemical measurements because they integrate the effects of multiple contaminants- even those chemicals that are not routinely measured. These tests measure individual organism responses relative to endpoints such as growth reproduction and mortality. In the hierarchal response scheme toxicity associated with these organism level endpoints should equate to some affect in community assuming that the indigenous and test species are similarly sensitive and similarly exposed. This paradigm formed the basis for water quality control by relying upon sensitive species and bioassays to establish water quality criteria that are protective of more tolerant organisms. Unfortunately the paradigm has never been proven in sediments. As Griffith (et al. 2008) states *organism-level effects are predictive to some extent of effects at the community level. However, this relationship is obscured by differences between these methods other than the hierarchical differences in the level of biological organization between their measurement endpoints.* This conclusion is supported by other authors including Chapman (et al. 2001, 2002) Ferraro (2002), Griffith (2008) Luoma (1996) and others. A number of factors weaken this relationship including.

- Toxicity test species and species that compose the benthic community have different sensitivities to different contaminants.

- Toxicity tests typically rely on short-term exposures using relatively few species and end points, making it difficult to interpret ecological significance of the results when used alone.
- Toxicity tests do not mimic the sediment structure, the bio-geochemical processes that influence bioavailability and the exposures that occurs in-situ.
- Presence of natural factors such as ammonia, hydrogen sulfide, or physical abrasion can lead to spurious results.

Benthic community condition is a good indicator because the benthos are directly exposed to sediment contamination and are one of the target biological resources the SQOs are intended to protect. However, their use alone is problematic because they are potentially affected by a large number of factors other than chemical contamination. Without chemistry or toxicity data for confirmation, it is difficult to distinguish whether degraded benthic communities resulted from chemical exposure or from physical disturbance, such as an anchor or prop-wash.

Bioaccumulation is also a useful measure, but sediments classified based on only a tissue uptake/bioaccumulation LOE would not account for toxicants that tend not to bioaccumulate in tissues of biota. Most trace metals and polynuclear aromatic hydrocarbons (PAHs) do not bioaccumulate in tissues, so their presence and toxicity would not be accounted for in such an approach. In addition, impacts from readily biotransformed pollutants would not be addressed by this LOE. The measurement of fish or shellfish tissue contamination provides an important measure of potential effects to wildlife or human consumers, but the mobility and varied life histories of the species makes it difficult to associate the effects with sediment contamination in specific locations.

For these reasons, multiple lines of evidence (MLOE) that represent both contaminant exposure and effects are frequently used in sediment assessments. The State Water Board's Bay Protection and Toxic Hotspots Cleanup Program relied primarily on MLOE to make critical decisions regarding management of sediment in bays and estuaries throughout the State (Anderson et, al 1997, 1998, Fairey, R, 1998, Hunt et al., 1998).

Virtually all of the estuarine ambient monitoring programs in this country rely on some form of the sediment quality triad, where chemistry and multiple measures of biological effect are used together to assess sediment quality (Crane, J.L., et al. 2000, Ingersoll, C. et al. 2002, MacDonald et al., 2003, U.S. EPA, 1998, 2004). These include the two largest nationwide estuarine monitoring programs, U.S. EPA's EMAP and the National Oceanic and Atmospheric Administration's (NOAAs) National Status and Trends Program, as well numerous regional monitoring programs, including those for the Great Lakes, Puget Sound, San Francisco Bay, Chesapeake Bay, Southern California Bight, Tampa Bay, and New York/New Jersey harbors.

The triad concept has been used and published in the United States, Canada, Australia, United Kingdom, France, the Netherlands, and Brazil, among others. Most regulatory programs, including those that control open water disposal of dredged material, require tests of sediment chemistry, toxicity, and bioaccumulation. Comprehensive ecological risk assessments invariably use a weight of evidence approach from multiple kinds of assays and tests to estimate and manage risks at waste sites. Even the national chemical benchmarks issued by U.S. EPA that rely on one LOE encourage users to apply them in concert with other sediment assessment tools in making management decisions.

While various MLOE approaches have been used to describe and classify sediment quality, they have typically been applied for site-specific or regional assessments. Moreover, MLOE applications are often based on use of BPJ for combining the individual LOE. BPJ will be

ineffective for use in SQOs because the expertise of the individuals applying them will vary considerably across the State, and there is a need for statewide consistency in their application. While there is no direct precedent for translation of MLOE into criteria, standards, or objectives, there are some applications that move in that direction from which lessons can be learned. The State of Washington's SQSs have provisions to use chemical, toxicological, and benthic composition data to classify sediments for multiple purposes, including disposal of dredged material. The Tampa Bay Estuary Program has adopted a triad of measures of sediment quality for management purposes there. The States of Minnesota and Illinois, in partnership with the U.S. EPA Assessment and Remediation of Contaminated Sediment (ARCS) Program of the Great Lakes National Program Office, use the triad of measures to assess sediment quality for management in the Great Lakes.

Baseline—Sediment quality assessment programs throughout the nation rely on MLOE to assess impacts to beneficial or designated uses.

Alternative 1—Do not specify LOE.

Alternative 2—Base policy on application of a single LOE. This alternative would base the policy on a single LOE, such as sediment toxicity, chemistry, or benthic community. Such an approach would be very simple to implement; however, any single LOE is affected by confounding factors, measurement errors, and variability and would contradict the approach recommended by U.S. EPA.

Alternative 3—Base policy on application of MLOE. The suite of tools and LOE would be specific to each receptor.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Sections I.A, V.A and B.

5.4.2 Form of Sediment Quality Objectives

The State Water Board has the option of establishing narrative or numeric objectives, or some combination of the two. In order to implement an approach based upon MLOE, as described above consideration must be given to the importance of each tool. Sediment quality is assessed with a combination of tools and results, in contrast to a numeric water quality objective for which a single specific measurement may be used. Within this approach, a narrative objective can be proposed that can be implemented with a high degree of confidence using a robust suite of tools; the MLOE approach. This approach would also minimize potential conflicts associated with discordant results. In addition, as better tools are developed to support the narrative objectives, these tools could be added as amendments to the plan while maintaining a consistent narrative objective.

Baseline—As described in Section 4 above, basin plans include narrative objectives that apply to sediment quality, as Implementation of the narrative objectives varies from region to region because the Regional Water Boards typically rely on best professional judgment (BPJ) applied on a case-by-case basis. There are no applicable numeric objectives in California that apply specifically to sediment quality.

Alternative 1—Do not adopt SQOs. This alternative would conflict with chapter 5.6, which requires the State Water Board to adopt SQOs.

Alternative 2—Numeric objectives could be developed and proposed for each LOE. However, each numeric objective would need to be integrated into a weight of evidence approach. The numeric objective would be meaningless without the other LOE.

Alternative 3—Narrative objectives could be proposed that would be implemented using MLOE and corresponding thresholds coupled to a logic based data integration process.

Alternative 4—Numeric objective based upon the integration of data from the three LOE. This alternative would provide greater utility for statistical analysis however until enough data is collected to evaluate the response relationships between the various LOE to create a valid numeric scale, a scientifically defensible numeric cannot be developed.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Section IV.

5.4.3 Sediment Toxicity

5.4.3.1 Sediment Toxicity to Support the Direct Effects of SQO

Sediment toxicity tests are considered an important component of sediment quality assessments (U.S. EPA 2001a, 2004a, 2004b, 2005, Wenning et al. 2005). Recent California assessment programs, such as the Bay Protection and Toxic Cleanup Program, and current programs, such as RMP and the Southern California Bight Regional Monitoring Program, use sediment toxicity as one of multiple measures of sediment quality. Much of the testing has employed acute amphipod survival methods using protocols established by U.S. EPA (U.S. EPA 1994). Many of the projects have also included a measure of sublethal toxic effects in sediments using a wide variety of test methods, including long-term growth tests, elutriate toxicity tests, porewater toxicity tests, and tests of toxicity at the sediment-water interface. The Environmental Monitoring and Assessment Program of U.S. EPA has used amphipod acute testing in conjunction with a variety of sublethal methods in different parts of the country (Ringwood *et al.* 1996, Bay *et al.* 1998). The State of Washington has a program for monitoring and assessing sediments that has been in place for nearly two decades using a combination of acute amphipod tests, polychaete growth tests, and modified elutriate testing with invertebrate larvae (Puget Sound Water Quality Authority 1995).

Laboratory toxicity tests consist of exposing test organisms to sediments within a controlled environment. The toxicity test response provides a direct measure of the combined effects of all chemicals present in the sample and can thus indicate the presence of toxic quantities of chemicals that were not detected or analyzed for in a chemical analysis. Because toxicity tests are conducted using sediments from the environment, the results incorporate the effects of sediment characteristics such as organic carbon that can alter the biological availability of the contaminant. The laboratory environment of the toxicity test allows for the control of confounding factors such as salinity, temperature, or dissolved oxygen that may vary in the field, thus permitting a distinction between toxic effects and effects due to natural habitat variability. For these reasons, some have argued that toxicity tests are the only line of evidence that is required to adequately asses sediment quality. Supporting this argument is the concept that a response causing mortality or reduced growth and reproduction in test organisms should translate to affects within resident community, such as decreased diversity and abundance (Griffith (2008). While this concept is logical and studies have demonstrated correlations between toxicity observed in laboratory organisms and community impacts, sediment toxicity tests cannot reliably predict effects to benthic communities (Chapman et al. 2001, 2002) (Ferraro (2002), Griffith (2008) Luoma (1996). Factors affecting this relationship are described

in Section 5.4.1. The toxicity test result may overestimate or underestimate effects occurring in the field due to variations in the sensitivity of the test organism or to changes in chemical exposure caused by sediment handling in the laboratory.

Baseline—The State and Regional Water Boards have relied upon sediment toxicity tests.

Alternative 1—Do not consider sediment toxicity tests for measuring direct effects.

Alternative 2—Propose sediment toxicity tests for inclusion in the implementation of direct effects narrative SQOs.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Section V.A.

5.4.3.2 Choice of Toxicity Tests Should Be used

The only means by which the State Water Board can maintain a high level of consistency and data quality is by limiting the tests that can be used in this program to those that meet specific criteria. Various methods for measuring sediment toxicity are available. Key differences between tests include: species, life history stage, duration, endpoint, and mode of exposure. Different species vary in their sensitivity to contaminants as a result of physiological differences, body type, and degree of exposure to the sediment. Crustaceans, bivalves, or polychaete worms are commonly used in toxicity tests, and there is no single species that is consistently the most sensitive to all contaminants of interest (Long *et al.* 1990, Burton *et al.* 1996, Anderson *et al.* 1998b, Bay *et al.* 2007a).

Various life history stages, including embryos, juveniles, and adults, are used in toxicity tests (Lamberson *et al.* 1992). Embryos and juveniles are generally more sensitive to contaminants than adults, but adult test organisms may be less sensitive to confounding factors that complicate test interpretation. There are a variety of endpoints that are specific to each test. The simplest endpoint is survival or lethality which is the endpoint associated with acute tests. Sublethal test endpoints include growth, reproduction, egg fertilization, embryo development, and biochemical responses such as DNA damage or cellular stress.

Test duration varies widely among toxicity test methods; tests generally range from 48 hours to 28 days in length. Longer duration tests may be more sensitive to the effects of chemicals that require bioaccumulation before toxicity is caused, but they also are more difficult and expensive to conduct. The method of exposure can also affect the sensitivity of the toxicity test or the data interpretation. Many tests expose the organism directly to whole sediment, which provides potential chemical exposure from direct particle contact, the pore water, and from sediment ingestion. Other test methods expose the organism to pore water extracted from the sediment, an elutriate, overlying water, or a solvent extract of the sediment (Anderson *et al.* 1996, Carr and Nipper 2003). These variations in exposure method are used to facilitate tests with organisms that cannot tolerate sediment contact (e.g., embryos) or to investigate specific mechanisms of exposure.

Because toxicity test responses are governed by so many different factors, a suite of standard test methods is often used to measure sediment toxicity in various assessment or regulatory programs. By requiring the use of specific test methods, (1) consistency is established throughout the State, (2) statewide thresholds can be developed that minimize subjective decision making, and (3) inappropriate tests will not be performed.

The process of selecting the recommended toxicity methods for the SQO program is described in Bay *et al.* (2007a). A review of the scientific literature and consultation with other scientists was used to identify a set of candidate sediment toxicity protocols that had the following characteristics: adopted or approved by U.S. EPA, USACE, American Society for Testing and Material Standards (ASTM), or other states; tolerance of expected sediment physical characteristics; diversity of taxonomic groups; association between response and sediment exposure; sensitivity to individual contaminants; and representative of benthic community species. The selection process resulted in a candidate test method list consisting of acute methods with the four commonly used amphipod species (*Ampelisca abdita, Eohaustorius estuarius, Rhepoxynius abronius,* and *Leptocheirus plumulosus*) plus six sublethal methods using amphipods (*Leptocheirus plumulosus),* polychaete worms *(Neanthes arenaceodentata),* sea urchins *(Strongylocentrotus purpuratus),* bivalves (*Mytilus galloprovincialis, Mercenaria mercenaria, Crassostrea virginica*), and copepods (*Amphiascus tenuiremis*). The tests are summarized in Table 5.2 from Bay *et al.* (2007a).

Table 5.2. List of Candidate Sediment Toxicity Tests, the Citations Containing Testing Protocols and Whether Quality Assurance and Test Acceptability Criteria Have Been Established

Species	Taxonomic Group	Duration (days)	Matrix	Endpoint(s)	Citations	State or National Program Use[1]
Ampelisca abdita/ Eohaustorius estuarius Rhepoxynius abronius Leptocheirus plumulosus	Amphipod	10	Whole sediment	Survival	(U.S. EPA 1994, ASTM 1996)	EMAP NOAA USACE WA, RMP
Leptocheirus plumulosus	Amphipod	28	Whole sediment	Growth, reproduction, survival	(U.S. EPA 2001)	
Neanthes arenaceodentata	Polychaete	28	Whole sediment	Growth, survival	(ASTM 2002b) modified	USACE[2] WA
Strongylocentrotus purpuratus	Sea urchin	3	Sediment-water Interface	Embryo development	(Anderson *et al.* 1996)	
Mytilus galloprovincialis	Mussel	2	Sediment-water Interface	Embryo development	(Anderson *et al.* 1996)	RMP
Amphiascus tenuiremis	Copepod	14	Whole sediment	Reproduction, survival	(Chandler and Green 1996)	NOAA
Mercenaria mercenaria	Clam	7	Whole sediment	Growth, survival	(Ringwood and Keppler 1998, Keppler and Ringwood 2002)	
Crassostrea virginica	Oyster	4	Whole sediment	lysosomal stability	(Ringwood *et al.* 1998, Ringwood *et al.* 2003)	

[1] EMAP: Environmental Monitoring and Assessment Program; NOAA: NOAA National Status and Trends Program; USACE (U.S. Army Corps of Engineers: dredged material evaluation for disposal under USACE or U.S. EPA guidance; WA: dredged material evaluation for disposal under Washington State guidance; RMP: San Francisco Bay Regional Monitoring Program

[2] The same species and endpoint is used in dredged material evaluations, but the duration and aspects of the test method differ

Toxicity tests on sediment pore water or elutriate samples were not considered for evaluation because of technical limitations in the methods. Pore water tests are widely used for testing sediment toxicity (Carr and Nipper 2003), but it is difficult to collect enough sample for testing. Other characteristics of pore water toxicity tests make these methods less suited for use in the SQO program, including potential changes in metal toxicity due to oxidation, change in sample pH, sorption of contaminants to test chambers, confounding effects of ammonia toxicity, and elimination of sediment ingestion as a route of uptake (Ho *et al.* 2002). Elutriate tests were also not included in the list of candidate methods. These tests, where sediments are added to water with agitation, allowed to settle, and then the water is removed for testing, are often used for testing the effects of sediment resuspension during dredged material disposal. The elutriate sample is subject to many of the confounding factors associated with pore water, and the relationship of the results to direct sediment exposure is not known. The decision to exclude pore water and elutriate tests was endorsed by the SQO Scientific Steering Committee.

Each of the candidate methods was ranked relative to the following characteristics: organism availability, method documentation, technical difficulty, sensitivity, precision, and cost. Results of these are shown on Tables 5.3 and 5.4. Survival tests using the amphipods *E. estuarius, R. abronius,* and *L. plumulosus* were recommended as the best choices for acute testing in California. *E. estuarius* and *R. abronius* have a substantial history of successful use in California for both monitoring and assessment studies. The *L. plumulosus* 10-day test has been conducted in California on a much more limited basis. However, it has long been used in other parts of the country, especially on the Gulf Coast for monitoring and assessment studies. *Leptocheirus* is also easily cultured in the laboratory and available year round from commercial suppliers.

Two sublethal test methods were recommended for use in the SQO program: a 28-day growth test using the polychaete worm *Neanthes arenaceodentata* and a 2-day development test using embryos of the mussel *Mytilus galloprovincialis* exposed at the sediment-water interface. These tests had the best combination of characteristics related to test feasibility, method documentation, and sensitivity. The recommended sublethal tests complement the ability of the acute tests to detect toxicity by providing diversity in test species, length of exposure, and mode of exposure. The other candidate sublethal tests were not recommended for a variety of reasons, including incomplete documentation of the method, high cost, and relatively low sensitivity to contaminated sediments

Baseline—The State and Regional Water Boards have used different amphipod species for acute tests within different programs, though *A. abdita* and *E. estuarius* are the species most commonly required. Sublethal sediment toxicity tests are not typically required by State and Regional Water Boards in NPDES programs.

Alternative 1—Do not specify toxicity methods.

Alternative 2—Specify only acute toxicity methods.

Alternative 3—Specify only sublethal toxicity methods.

Alternative 4—Specify a combination of acute and sublethal toxicity methods.

Staff Recommendation—Alternative 4.

Proposed Language—See Appendix A, Section V.F.

Table 5.3. Characteristics of Candidate Sediment Toxicity Test Methods from Bay et al. (2007a)

	Organism Availability[1]	Method Description[2]	Technical Difficulty[3]	Concordance at Clearly Clean or Impacted Sites[4]	More Sensitive than *Eohaustorius*[5]	Reproducibility Among Laboratories[6]	Reproducibility Within Laboratories[6]	Relative Precision of Response[7]	Documentation of Confounding Factors[8]	Cost of Method[9]
Amphipod Acute										
Eohaustorius	12 (+)	Standard	Low	NA	NA	Good	Good	NA	Good	Low
Rhepoxynius	12 (+)	Standard	Low	NA	Sometimes	Good	Good	NA	Good	Low
Leptocheirus	12 (+)	Standard	Low	NA	Often	Fair	Poor	NA	Fair	Low
Ampelisca	8 (+)	Standard	Moderate	NA	Rarely	Poor	Good	NA	Fair	Low
Sublethal Methods										
Mercenaria growth	8(+)	Published	Low	Fair	Sometimes	Fair	Fair	Similar	Good	Low
Neanthes growth and survival	12(1)	Published	Moderate	Fair	Sometimes	Good	Good	Low	Good	High
Sediment-Water Interface										
Mussel development	12(++)	Published	Low	Fair	Sometimes	Fair	Good	Low	Fair	Low
Sea urchin development	5(++)	Published	Low	Fair	Rarely	None	Good	Low	Good	Low
Leptocheirus chronic	12(+)	Standard	Moderate	Fair	Sometimes	Fair	Good	Low	Good	High
Copepod life cycle	12(1)	Published	High	Good	Often	None	Good	High	Fair	Very High
Oyster lysosomal stability	8(++)	Report	Moderate	Poor	Rarely	None	None	Low	Poor	Moderate

NA = not applicable for test

[1] Number of months (relative number of available suppliers, + +for many, + for few, 1 for one)

[2] Standard = Established method by government agency; Published = Peer reviewed publication of method; Report = In gray literature

[3] Low = Similar skills and equipment needed as for acute amphipod test; Moderate = More difficult to obtain acceptable controls, special techniques or more complex exposure system; High = Combination of special skills and more complex exposure system needed

[4] Concordance with acute amphipod test: Good = >75%; Fair = <75%>50%; Poor<50%.

[5] Of the stations found to be toxic by at least one endpoint: Often = >50% of stations; Sometimes = <50%>20, Rarely <20%; Never = 0

[6] Good = CV<50%; Fair = CV >50%<75%; Poor = CV>75% (CV = coefficient of variation; mean/standard deviation)

[7] Categories based on the range of median acute amphipod standard deviations. High = below range; Similar = within range; Low = above range

[8] Data available for confounding factors: Good = Four or more factors; Fair = 2 or 3 factors; Poor = Less than 2 factors

[9] Low = 150% or less the cost of acute amphipod; Moderate = 150% to 200% of amphipod; High = 200% to 300% of amphipod; Very High = >300% of amphipod.

Table 5.4. Ratings of Acute and Sublethal Sediment Toxicity Methods from Bay et al. (2007a)

	Feasibility				Performance and Cost							
	Organisms Availability	Method Description	Technical Difficulty	Overall Feasibility	Concordance with Amphipods at Clearly Clean or Impacted Sites	More Sensitive than Acute *Eohaustorius* Test	Reproducibility Among Laboratories	Reproducibility Within Laboratories	Relative Precision of Response	Documentation of Confounding Factors	Relative per Station Cost	Total Score
				Factor:	**2**	**4**	**2**	**2**	**1**	**2**	**2**	
Amphipod Acute												
Eohaustorius	+	+	+	Yes	NA	8	6	6	2	6	6	34
Rhepoxynius	+	+	+	Yes	NA	8	6	6	2	6	6	34
Leptocheirus	+	+	+	Yes	NA	12	4	2	2	4	6	30
Ampelisca	+	+	+	Yes	NA	4	2	6	2	4	6	24
Sublethal Methods												
Mercenaria growth	+	–	+	No	4	8	4	4	2	6	6	34
Neanthes survival and growth	+	+	+	Yes	4	8	6	6	1	6	2	33
Sediment-Water Interface												
Mytilus galloprovincialis	+	+	+	Yes	4	8	4	6	1	4	6	33
Strongylocentrotus purpuratus	+	+	+	Yes	4	4	0	6	1	6	6	27
Leptocheirus - 28 Day	+	+	+	Yes	4	8	4	6	1	6	2	31
Amphiascus Lifecycle	–	+	–	No	6	12	0	6	3	4	0	31
Crassostrea lysosomal stability	+	–	–	No	2	4	0	0	1	2	4	13

Note: Total score is sum of ratings

5.4.3.3 *Evaluation of Toxicity Test Responses*

To provide consistent interpretation and assessment of sediment toxicity, Part 1 should describe how the responses to the tests recommended above are assessed. If Part 1 did not include this information, the interpretation of sediment toxicity would have to be decided by individual staff at the Regional Water Boards using best professional judgment, which would create a greater risk of inconsistent assessment both within and across the regions.

Interpretation of sediment toxicity is commonly assessed using a binary approach (nontoxic/toxic) or by using three or more categories to distinguish different levels of response and confidence. The advantage of multiple categories versus the binary approach is that it provides greater information about the toxicity response and thus provides greater potential resolution when combining the toxicity data with other lines of evidence in a sediment quality triad approach. This is especially important when the end user must be able to distinguish not only the highly impacted stations from the unimpacted stations, but also those stations that exhibit low levels of impact as well. For this reason, members of the SSC strongly supported the development of multiple categories for all LOE.

In response to this need, the SQO technical team developed a multi-category system adapted from the three-category system commonly used to classify sediment toxicity (Long et al., 2000). In the three-category system, the test response is classified as nontoxic, marginal, or toxic. Within the SQO program, the technical team developed a system based upon four categories. Each of the four categories was based on a narrative description of condition that incorporated both the degree of confidence that a toxic effect was present and the magnitude of response (Bay et al., 2007).

Nontoxic—Response not substantially different from that expected in sediments that are uncontaminated and have optimum characteristics for the test species

Low Toxicity—A response that is of relatively low magnitude; the response may not be greater than test variability

Moderate Toxicity—High confidence that a statistically significant effect is present

High Toxicity—Highest confidence that a toxic effect is present and the magnitude of response is among the strongest effects observed for the test

The nontoxic and marginal categories used in previous studies such as the Water Boards' Bay Protection Program correspond to the nontoxic and low toxicity categories of the scoring system proposed for here. The category designated as toxic in past studies typically represented a reliably statistically significant response that encompassed a wide range of effects (*e.g.*, 0 – 80% survival) and as a result provided little discrimination among the majority of the toxic samples. The proposed approach described here divides this broad response category into two categories defined as moderate and high, in order to provide the ability to distinguish severe effects from more moderate responses.

Figure 5.1 illustrates the relationship between these four categories, the numeric thresholds and statistical criteria. In order to assess toxicity response within a given sample, the end user would simply compare test results (e.g., % survival) to Low, Moderate, and High thresholds and statistical significance criteria.

Basis for Thresholds

The thresholds were developed using test-specific characteristics, such as test variability (minimum significant difference (MSD)) and distribution of the toxicity response data. A statistical criterion was also used in the classification scheme (Figure 5.1). Samples qualifying for the Low or Moderate categories based on test response magnitude were classified into the next lower category if the response was not significantly different relative to the control (t test, $p \leq 0.05$). A statistical significance criterion was not applied to the highest toxicity category because the derivation of the high toxicity threshold already incorporated a high degree of statistical confidence.

The basis for establishing each of the sediment toxicity thresholds that bound each category is summarized below. The analyses used to derive the thresholds are described in Bay et al. (2007a). This report can be downloaded directly from www.sccwrp.org.

Low Toxicity Threshold

The threshold separating the nontoxic and low categories was defined as the lowest acceptable control response value for the given test, as established in the test protocols. The response value is defined as the mean value for the endpoint for a given test method (i.e. survival, growth). This threshold was based on the rationale that any response that fell within the range expected of animals exposed to optimum sediment conditions (i.e., controls) should indicate a nontoxic condition in the test sample. The control acceptability criteria were obtained from the appropriate protocol for each test method. Any test sample having a response value that is greater than or equal to the low threshold will be classified as nontoxic, regardless of whether a statistical difference from the control is present. A test response that is less than the low threshold will be classified as Low, Moderate, or High, depending on the magnitude of response and statistical significance (Figure 5.1).

Moderate Toxicity Threshold

The intent of the Moderate Threshold is to distinguish between samples producing a small response of uncertain significance and larger responses representing a reliably significant difference relative to the control. This threshold was based on the Minimum Significant Difference (MSD), which was specific to each test method. The MSD represents the minimum difference between the control and sample response that is necessary to be statistically different at $p \leq 0.05$ level. The moderate threshold was equal to the 90^{th} percentile of the MSD for a given toxicity test method. This approach for calculating a toxicity threshold has been used by other researchers (Phillips *et al.* 2001). Use of the 90^{th} percentile results in a threshold with a high degree of confidence that the sample is different from the nontoxic condition.

The MSD values were calculated using the replicate control and sample data from many toxicity tests. Details of this calculation can be found in Phillips *et al.* (2001). For each combination of a control and a sample, the variance of the replicates, number of replicates, and the t-critical value for the pair were used to calculate a single MSD value. All of the MSD values in the dataset for each toxicity test method were then sorted in rank order. The 90^{th} percentile value of this set of data was then calculated (MSD_{90}). The MSD_{90} values were calculated using all available data for each toxicity test method. Finally, the moderate threshold value was calculated by subtracting the MSD_{90} from 100% in order to produce a value that could be compared to the control-adjusted test response value.

Sample response values (i.e. survival or growth) between the low and moderate thresholds are classified as Low Toxicity if they are significantly different from the control

response (Figure 5.1). Sample response values that are less than the moderate threshold and are significantly different from the control are categorized as moderately toxic.

High Toxicity Threshold

The intent of the High Threshold is to identify samples producing a severe and highly significant effect from those samples producing lesser effects. No precedent for this threshold was available from the literature, so this threshold was based on a combination of test variability and response distribution that corresponded to the category definition. This approach was recommended by the SQO Scientific Steering Committee.

The 99th percentile MSD value was used to link the High threshold to test variability. A sample having a response that falls below this limit would be expected to be significantly different from the control 99% of the time. This value therefore represents a response that is associated with a very high level of confidence of statistical significance. The 99th percentile MSD for the high threshold was calculated using the same data and methodology described for the calculation of the MSD_{90} for the moderate threshold.

The response distribution component of the high threshold was based on the distribution of toxic samples from California. For purposes of this calculation, toxic samples were defined as samples having a mean response that was significantly different from the control response. The toxic samples were ranked in descending order based on the control-adjusted mean survival. The response magnitude component of the high threshold corresponded to the 75th percentile of the data. The value obtained from this calculation represents the response associated with the most strongly affected 25% of the toxic samples found in California. It was required that data for this calculation be from stations within California in order to obtain a response value that was relevant to the characteristics of sediments in California.

Both the variability and data distribution response values represented important, but partial, aspects of the High Threshold. Therefore, the mean of the two values was used as the High Threshold. Response values (i.e. survival or growth) below the high threshold are classified as high toxicity regardless of whether they are significantly different from the control response or not (Figure 5.1).

Sediment Toxicity Thresholds

The toxicity test thresholds developed for the SQO program are summarized in Table 5.5. These thresholds are similar to comparable thresholds utilized within the California Bay Protection and Toxic Cleanup Program and the Southern California Bight Regional Monitoring Programs.

Table 5.5. Proposed Toxicity Threshold Values for the Sediment Toxicity Test Methods

Species	Low (%)	Moderate (% Control)	High (%Control)
Eohaustorius	90	82	59
Rhepoxynius	90	83	70
Leptocheirus	90	78	56
Neanthes	90[1]	68	46
Mytilus	80	77	42

[1] % of control growth.

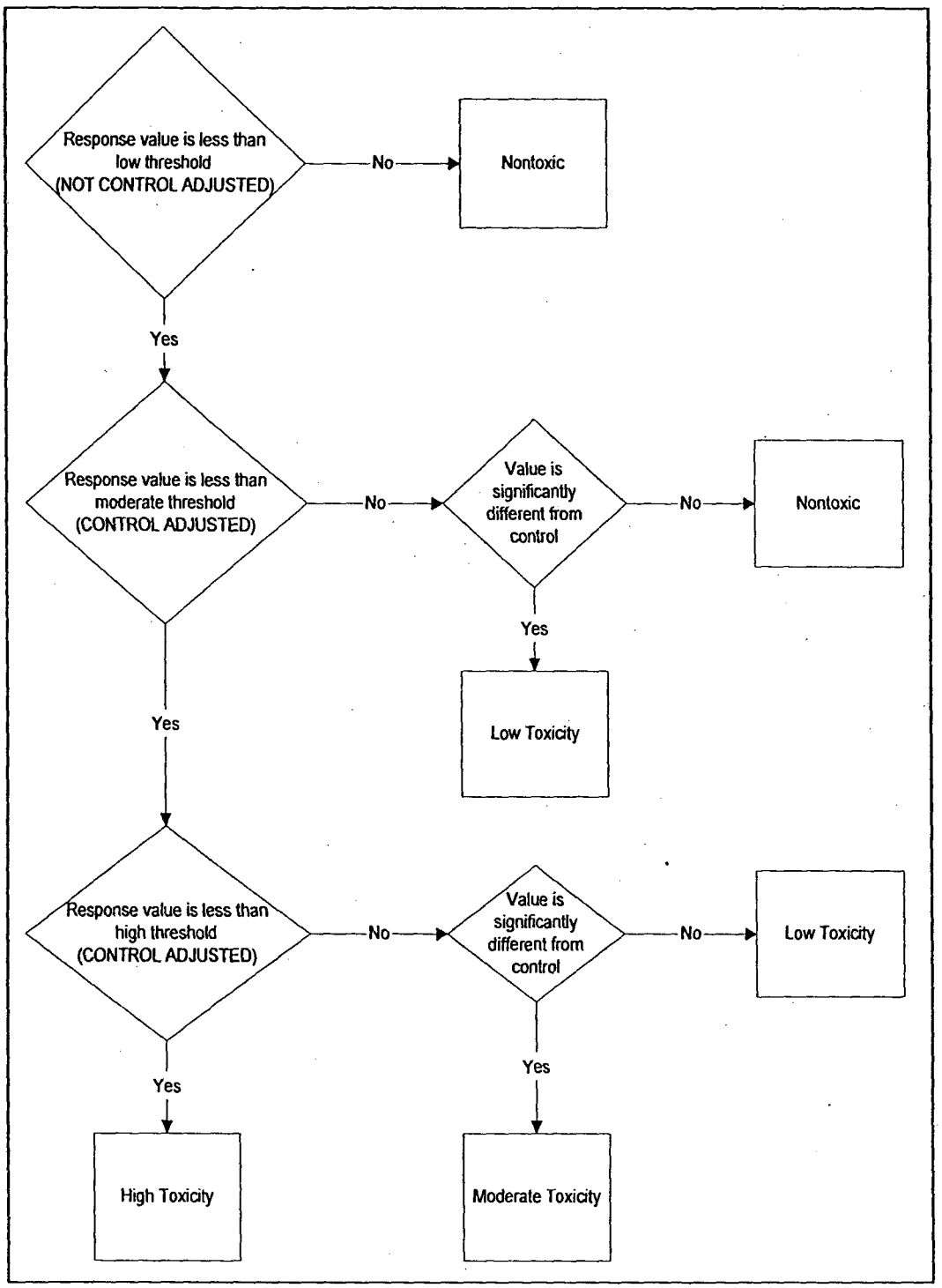

Figure 5.1. Conceptual Approach and Process for Assigning the Category of Toxicity from Laboratory Test Results

Baseline—Existing programs typically categorize response as either toxic or nontoxic where the toxic response is defined as a reliably statistically significant response that encompasses a wide range of effects (*e.g.*, 0 – 80% survival).

Alternative 1—Categorize toxicity response as toxic or nontoxic

Alternative 2—Categorize toxicity response by the toxicity thresholds identified in Table 5.5.

Staff Recommendation—Alternative 2

Proposed Language—See Appendix A, Section V.F. Presented in Appendix C is an example problem and solution based upon the proposed language.

5.4.4 Chemical Analysis

5.4.4.1 Chemical concentrations used to support the Direct Effects of SQOs

Many monitoring and assessment programs evaluate the effects of chemical contamination on sediment quality. Sediment quality guidelines (SQGs), tools that relate contaminant concentrations to the potential for adverse effects on sediment-dwelling organisms, are often used to help interpret sediment chemistry data. SQGs have been used for over 30 years to assess sediment contamination (Engler *et al.* 2005), yet there are many factors that make their use a complex and challenging task. These complicating factors include a lack of guidance on how to evaluate the many types of SQGs in order to select the approach best suited for a particular application, uncertainty regarding how to assess complex mixtures of contaminants, the inability to reliably predict contaminant bioavailability, and uncertainty in how to establish thresholds for SQG interpretation that define acceptable and unacceptable sediment quality (Wenning *et al.* 2005).

Numerous studies have shown that each type of SQG has predictive ability with respect to biological effects (Wenning *et al.* 2005). The predictive ability is often greatest in instances of high/low contaminant concentrations. Predictions of the biological effect based on SQGs have the highest error rates when applied to samples containing intermediate levels of contamination (Long *et al.* 1998, Fairey *et al.* 2001). The predictive ability of SQGs has also been shown to vary among datasets from different regions (Fairey *et al.* 2001, Crane *et al.* 2002), which complicates the selection of the most reliable approach and thresholds for a given application.

There is considerable concern over the misuse of sediment chemistry guidelines to implement narrative water quality objectives in Basin Plans. The use of chemical SQGs is often accompanied by substantial uncertainty and controversy, as no single SQG approach is able to account for all of the factors that influence contaminant effects. In sediments, if pollutant concentrations are very low or not detected but significant effects are observed, two possible scenarios exist: (1) a non-pollutant-related stressor, such as physical disturbance or habitat alteration, is the cause of impairment; or (2) a pollutant is present that was not identified by the suite of analytical methods selected (Chapman 1990, Ingersoll *et al.* 2001). Both scenarios assume that the effects data and the chemistry data accurately reflect the conditions at the station. Conversely, if pollutant concentrations are elevated but effects are not observed, the pollutant may not be bioavailable. Simple effective approaches to quantify the bioavailable fraction of a pollutant in sediment are not currently available and are not likely to be developed in the near future (U.S. EPA 2005).

Baseline—Sediment chemistry is frequently used as an indicator to assess potential impacts. In this role, sediment concentrations are compared to various SQGs (ERLs, ERMs, PELs, AETs) either independently or in conjunction with other LOEs to determine if the

pollutants in sediment pose a risk. In California, there are no current plans or policies that define what guidelines shall be used, how the guidelines should be applied, or what conclusions can appropriately be drawn based solely on chemistry.

Alternative 1—Do not consider sediment chemistry as a direct-effects implementation tool. As described previously, sediment chemistry is not a measure of the bioavailable fraction of pollutants in sediment. As a result, this tool would have little or no utility within a state sediment quality program.

Alternative 2—Propose specific sediment chemistry indicators for inclusion in the implementation of direct effects narrative SQOs. Within the policy, sediment chemistry would be proposed as a surrogate measure of exposure and used only with other LOEs.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Section V.A.

5.4.4.2 Choice of Chemistry Indicators

There are three principal types of SQGs, based on the approach used in their derivation: empirical, mechanistic, and consensus. Empirical SQGs are the most widely used type; these guidelines are derived from the statistical analysis of large databases of matched sediment chemistry and biological effects data. Examples of empirical SQGs for the marine environment include the effects range-median (ERM) probable effects level (PEL), apparent effects level (AET), and logistic regression models (LRM) (Long *et al.* 1995, MacDonald *et al.* 1996, Barrick *et al.* 1988, Field *et al.* 2002). Mechanistic SQGs take into account chemical and biological processes that affect contaminant bioavailability and toxicity. Current mechanistic SQGs are based on equilibrium partitioning theory and apply to selected classes of contaminants, primarily divalent metals and some types of nonionic organics (U.S. EPA 2004c, 2004d). Consensus guidelines are derived from the aggregation of several types of SQGs having a similar narrative intent (e.g., median effect). Marine consensus SQGs have been developed for a relatively small number of constituents, including metals, PCBs, and PAHs (MacDonald *et al.* 2000, Swartz 1999, Vidal and Bay 2005).

There are two potential applications of chemical SQGs in a SQO policy setting: overall assessment of the presence of impacts due to chemical pollutants, and determination of the cause of the impacts. The different types of SQGs vary in their effectiveness for these applications. Empirical and consensus SQGs provide an estimate of the probability of effects due to chemical contamination level and are thus well suited for overall assessment of impacts. Mechanistic SQGs use partitioning models to determine cause and effect and are thus well suited for applications where determination of cause is needed. The different SQG approaches are complementary in their uses and limitations and both have applications in the assessment and management of contaminated sediment (Di Toro *et. al.* 2005).

The utility and performance of SQGs based on mechanistic, empirical and consensus approaches were evaluated. The approaches included EqP models for nonpolar organics and metals, existing national empirical and consensus guidelines, regional guidelines calibrated to California data, and newly developed guidelines. The evaluation consisted of two phases: preliminary and final. The preliminary evaluation examined a wide range of SQG approaches and assessed the predictive ability (e.g., correlation with respect to sediment toxicity) and feasibility of each approach. Mechanistic SQGs based on EqP models were found to have no significant correlation with California sediment toxicity data in the preliminary analyses and insufficient data (e.g., sediment acid volatile sulfides and simultaneously extracted metals) were

available to enable further evaluation of EqP SQGs for metals. These results were consistent with previous analyses using southern California data that showed poor predictive ability of mechanistic SQGs (Vidal and Bay 2005).

The final evaluation of SQG performance examined several empirical and consensus approaches that were identified in the preliminary analyses as best meeting the needs of an SQO assessment framework. The results for the individual chemical components of each SQG were summarized for evaluation as either a mean quotient or maximum probability (Bay *et al.* 2007b, Ritter *et al.* 2007). These summary statistics integrate the effects of the mixture of chemicals present in each sample and have been shown to improve the predictive ability of empirical SQGs (Field *et al.* 2002, Long *et al.* 2006). The SQG approaches evaluated include:

National SQGs

Effects Range Median (National ERM)

The Effects Range Median (ERM; Long *et al.* 1995) represents the concentration above which adverse effects are frequently observed. This value corresponds to the 50th percentile (median value) of the distribution of chemical concentrations associated with adverse biological effects. The subset of National ERM values used in this study was the same as that used in other studies of ERM performance (Long *et al.* 2000). The mean ERM quotient was calculated for a sample by dividing each chemical concentration by its respective ERM and subsequently averaging the individual quotients.

Mean Sediment Quality Guideline Quotient 1 (SQGQ1)

The mean sediment quality guideline quotient 1 (SQGQ1) is based on a set of metal SQGs selected from ERM or PEL and consensus SQGs for PAHs and PCBs (Fairey *et al.* 2001). The suite of chemicals included in the SQGQ1 was selected by Fairey *et al.* to obtain high predictive ability with respect to the incidence of toxicity. The SQGQ1 quotient was calculated for a sample by dividing each chemical concentration by its respective SQG and subsequently averaging the individual quotients.

Consensus Midpoint Effect Concentration (Consensus)

The Consensus SQG approach is based on the integration of different SQG types. Consensus MEC values are the geometric mean of three or more SQGs that correspond to the same biological effect level. This study evaluated Consensus SQG values representing the midpoint effect concentration (MEC), an intermediate level of effect. Consensus values for PAHs and PCBs were obtained from Swartz (1999) and MacDonald et al. (2000), respectively. Values for DDTs, dieldrin, arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc were obtained from Vidal and Bay (2005). The mean Consensus quotient was calculated for a sample by dividing each chemical concentration by its respective SQG and subsequently averaging the individual quotients.

Logistic Regression Model (National LRM)

The Logistic Regression Model (LRM) approach was based on the statistical analysis of paired chemistry and amphipod toxicity data from studies throughout the U.S. (Field et al. 1999, 2002). A logistic regression model is developed for each chemical to estimate the probability of toxicity at a given concentration. LRM models for 18 chemicals having low rates of false positives were selected for use in this study. The LRM method does not establish specific concentration values for each chemical, but rather describes the relationship between

contaminant concentrations and the probability of toxicity. The maximum probability of effects obtained from the individual chemical models (P_{max}) was selected to represent the chemical mixture present in a sample (Field et al. 2002).

Regional SQGs

Regional chemical indicators were developed based on two national SQG approaches: ERM and LRM. Three versions of each regional indicator were developed: a statewide version that was calibrated to data from throughout California (CA ERM or CA LRM), and two region-specific versions. The region-specific versions were calibrated separately for northern California (NorCA ERM or NorCA LRM) and southern California (SoCA ERM or SoCA LRM) data sets.

CA ERM, SoCA ERM, NorCA ERM

Individual chemical values analogous to national ERMs were calculated using California data. The data were screened to select toxic samples (>20% mortality) with chemical concentrations >2x median concentration of nontoxic samples. After screening, the data were sorted in ascending order and the median concentration of each chemical was selected as the region-specific ERM value. CA ERM and So CA ERM values were calculated for 27 chemicals, and NorCA ERMs were calculated for 25 chemicals.

CA LRM, SoCA LRM, NorCA LRM

LRM models for individual chemicals were developed for the statewide and regional California data sets. The specific LRM models included in the CA LRM, SoCA LRM, and NorCA LRM approaches were selected from a library of candidate models that included national models as well as models derived using the California data sets. The selected models were chosen based on the goodness of fit with the observed probability of toxicity.

Mean Chemical Score Indicator (CSI)

The mean CSI is a new SQG developed for the SQO program that is based on the association between chemicals and the magnitude of benthic community disturbance (Ritter *et al.* 2007). Two types of data are combined to calculate the mean CSI: a set of predicted benthic community effects categories based on the individual chemical concentrations and a set of weighting factors reflecting the strength of association between the chemical and benthos response. The chemical values determining the benthic community effect categories were determined for each chemical by a statistical process that identified the chemical ranges producing the best agreement with the biological response categories. Each constituent's predicted effect level is then multiplied by its respective weighting factor to produce a CSI score. Individual CSI scores were combined as a weighted mean to represent chemical mixture effects.

The results of the SQG performance evaluations are described in Bay *et al.* (2007b) and Ritter *et al.* (2007) and summarized in Tables 5.6, 5.7, and 5.8. A regional SQG approach, the CA LRM, had the best ability to predict the toxicity of California sediments. Among the statewide-calibrated SQGs, the CA LRM ranked highest in all three performance measures (correlation, weighted kappa, % agreement). Some of the other statewide-calibrated SQG approaches performed similar to the CA LRM in some respects, but their performance was less consistent (Table 5.6). This study identified regional differences in SQG performance and found that the use of regional data to develop and calibrate SQGs produced a small, but inconsistent, improvement in performance (Table 5.7).

Table 5.6. Nonparametric Spearman Correlation (r) and Classification Accuracy of Statewide SQG Approaches for Amphipod Mortality

Region	Approach	Weighted Kappa	% Agreement	r
State	CA LRM	[illegible]	[illegible]	[illegible]
State	National ERM	0.17	32	0.25
State	Consensus	0.17	31	0.25
State	National LRM	0.15	[illegible]	0.22
State	CA ERM	0.17	[illegible]	0.20
State	SQGQ1	0.12	32	0.16

Note:
Values in the shaded cells are within the 90th percentile of the highest median value for the bootstrapped analyses. Analyses were conducted on the combined data for the north and south validation data sets and used thresholds calibrated statewide. (Table from Bay *et al.* 2007b)

Table 5.7. Classification Accuracy and Spearman Correlation of Regional SQG Approaches for Amphipod Mortality

Approach	Northern California			Southern California		
	Weighted Kappa	% Agreement	r	Weighted Kappa	% Agreement	r
Regional Calibration						
CA LRM	[illegible]	27	[illegible]	[illegible]	[illegible]	[illegible]
National ERM	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	0.28
Consensus	[illegible]	[illegible]	0.23	[illegible]	[illegible]	[illegible]
National LRM	[illegible]	[illegible]	0.15	[illegible]	[illegible]	[illegible]
CA ERM	[illegible]	[illegible]	0.22	0.13	33	0.18
SQGQ1	[illegible]	[illegible]	0.25	0.18	33	0.26
Nor/SoCA LRM	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Nor/SoCA ERM	[illegible]	[illegible]	[illegible]	0.18	[illegible]	0.18

Note:
Values in the shaded cells are within the 90th percentile of the highest median value of the bootstrapped analyses. Analyses were conducted using thresholds for each region separately. (Table from Bay et al. 2007b)

Table 5.8. Classification Accuracy of CSI and Toxicity-based SQG Approaches for Benthic Community Condition

Region	Approach	Weighted Kappa	% Agreement
SoCA	CSI	0.44	52
SoCA	CA LRM	0.31	31
SoCA	National ERM	0.26	43

Note:
Analyses were conducted using thresholds and data for southern California. (Table from Ritter et al. 2007)

Different correlations with chemistry were obtained for toxicity and benthic condition, suggesting that these two indicators of biological effect are responding differently to contamination or other sediment characteristics. The new benthos-based CSI SQG had greater accuracy for predicting benthic community condition than did SQGs based on toxicity (Table 5.8). The results indicated that the accuracy and ecological relevance of chemical SQGs could be improved by incorporating benthic response data into SQG development.

Baseline—Sediment chemistry is typically evaluated by comparison to one or more national empirical SQGs, with little consistency in approach among regions.

Alternate 1: Establish narrative guidance.

Alternate 2: Use existing national empirical SQGs without consideration of actual predictive ability when applied to California data.

Alternate 3: Use either existing, regional, or new empirical SQGs derived from California data. Methodologies and thresholds for applications would be selected based upon how the approach performs within the SQO framework.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Section V.H. Presented in Appendix C is an example problem and solution based upon the proposed language.

5.4.5 Benthic Community

Benthic communities are found almost universally in aquatic soft sediments and are indicators of choice for monitoring and assessing anthropogenic effects for two main reasons. First, they possess many attributes considered desirable in indicator organisms, including limited mobility, diversity of organism types, life histories that are short enough to reflect recent changes in stressors, and direct exposure to sediment contamination. Second, they are important components of aquatic food webs, transferring carbon and nutrients from suspended particulates in the water column to the sediments by filter feeding and serving as forage for bottom-feeding fishes.

Despite these appealing characteristics, benthic infaunal monitoring data are maximally useful in a regulatory context only when they can be interpreted in relation to scientifically valid criteria or thresholds that distinguish "healthy" from "unhealthy" benthic communities. While reducing complex biological data to index values has disadvantages, the resulting indices remove much of the subjectivity associated with data interpretation. Such indices also provide a simple means of communicating complex information to managers, tracking trends over time, and correlating benthic responses with stressor data (Dauer *et al.* 2000, Hale *et al.* 2004).

During the past decade, several scientifically valid measures of marine and estuarine benthic community condition, often called benthic indices, have been developed for regulatory use. Benthic indices are increasingly accepted by regulators and incorporated into regulatory processes. The U.S. EPA's guidance for biocriteria development (Gibson *et al.* 2000) recognizes three types of benthic indices, and the agency included benthic assessments in a recent report on nationwide coastal condition to Congress (U.S. EPA 2004). In Maryland and Virginia, the Index of Biotic Integrity is one of the measures used to report on the condition of Chesapeake Bay waters under sections 305(b) and 303(d) of CWA. In California, the Relative Benthic Index (RBI) (Hunt *et al.* 2001) was one of the indicators used by the State Water Board to designate toxic hotspots (SWRCB 2004a) and the Benthic Response Index (BRI)(Smith *et al.* 2001, 2003 and Ranasinghe 2004 was applied by the San Diego Regional Water Board to

assess clean-up for three toxic hot-spots in San Diego Bay (Exponent 2002, SCCWRP and Space and Naval Warfare Systems Center San Diego 2004). Due to the presence of benthic communities in good condition as measured by the BRI and other reasons, Santa Monica Bay, which previously was listed as impaired under section 303(d) of the CWA due to sediment concentrations of six metals, was removed from the list in 2003. The BRI has also been used in southern California to assess the extent of bottom area supporting unhealthy benthic communities since 1994 (Bergen *et al.* 1998, Bergen *et al.* 2000, Ranasinghe *et al.* 2003).

5.4.5.1 Choice of Metrics Used to Support the Direct Effects SQO

There are several impediments to applying benthic indices statewide in California's bays and estuaries. First, several different habitats and benthic assemblages are present in California embayments, each of which requires metric development and calibration. Second, different benthic indices have been used in California at different times and different places, and results cannot be compared across regions because the various indices have not yet been rigorously compared and intercalibrated. Third, initial development of each existing benthic index was constrained by data limitations, and they would all benefit from refinement with additional data as well as independent validation. In addition, there is a lack of knowledge of the effects of differences in: (1) sampling procedures traditional in different regions, (2) habitat factors such as seasonality and sediment type, and (3) accuracy of identification of benthic organisms on performance of California benthic indices. As a result, significant work is required to develop benthic tools for all bays and estuarine habitats.

In order to select the appropriate benthic indices for this program, the technical team compared a number of indexes and combinations of indexes to a California data set validated by nine highly regarded benthic ecologists. This study is described in Ranasinghe *et al.* (2007) and consisted of the following tasks:

- Data for sampling sites in each of the two habitats were identified, acquired, and adjusted to create consistency across sampling programs.
- Five benthic indices were calibrated using a common set of data for all indices.
- Threshold values were selected for each index to assess benthic condition on a four-category scale.
- Performance of the indices and all possible combinations was evaluated by applying the calibrated indices to independent data and comparing the index condition assessments with benthic condition assessments of nine benthic experts.

The benthic indices evaluated in the study include:

Benthic Response Index (BRI), which was originally developed for the southern California mainland shelf and extended into California's bays and estuaries (Smith *et al.* 2001, 2003). The BRI is the abundance-weighted average pollution tolerance score of organisms occurring in a sample.

Index of Benthic Biotic Integrity (IBI), which was developed for freshwater streams and adapted for California's bays and estuaries (Thompson and Lowe 2004). The IBI identifies community measures that have values outside a reference range.

Relative Benthic Index (RBI), which was originally developed for California's Bay Protection and Toxic Cleanup Program (Hunt *et al.* 2001). The RBI is the weighted sum of: (a) several community metrics, (b) the abundances of three positive indicator species, and (c) the presence of two negative indicator species.

River Invertebrate Prediction and Classification System (RIVPACS), which was originally developed for British freshwater streams (Wright *et al.* 1993, Van Sickle *et al.* 2006) and adapted for California's bays and estuaries. The RIVPACS index calculates the number of reference taxa present in the test sample and compares it to the number expected to be present in a reference sample from the same habitat.

Benthic Quality Index (BQI)

The BQI was originally developed for the west coast of Sweden by Rosenberg *et al.* (2004) and applied in the United States for the first time in this project. The BQI is the product of the logarithm ($base_{10}$) of the total number of species and the abundance-weighted average tolerance of organisms occurring in a sample. Species tolerance scores are calculated differently than for the BRI; instead, they are based on relationships of the abundance distributions to Hurlbert's (1971) expected number of species.

Summary of Findings

Index performance was evaluated by comparing index assessments of 34 sites to the best professional judgment of nine benthic experts (Table 5.9). None of the individual indices performed as well as the average expert in ranking sample condition or evaluating whether benthic assemblages exhibited evidence of disturbance. However, several index combinations outperformed the average expert. When results from both habitats were combined, two four-index combinations and a three-index combination performed best.

Baseline—No methods have been approved or adopted by the Water Boards for the habitats under consideration. However, several tools have been applied by the Water Boards for the purposes of hot spot identification, water body assessment and site assessments. Those tools used most frequently in California are the BRI applied currently to embayments and nearshore waters south of Point Conception, (Ranasinghe et al. 2007a, 2007b), RBI used within the Bay Protection Program (Hunt et al. 1998, Hunt et al. 2001, Fairey et al. 1996) and IBI used in pilot studies in the San Francisco Bay Regional Monitoring Program (Davis, et al. 2006).

Alternative 1—Do not specify the methods.

Alternative 2—Select a single benthic index for all applicable water bodies.

Alternative 3—Select a combination of benthic indices for applicable water bodies.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A Section .V.G. Presented in Appendix C is an example problem and solution based upon the proposed language.

5.4.6 Integration of Direct Effects LOE Within Embayments

Sediment quality is frequently assessed using a triad of chemical concentration, sediment toxicity, and benthic infaunal community condition (Long and Chapman 1985). These are used in combination because sediments are a complex matrix and chemical concentration data alone fails to differentiate between the fraction that is tightly bound to sediment and that which is biologically available. Multiple approaches for integrating these multiple lines of evidence (MLOE) data have been developed (Chapman *et al.* 2002). These integration approaches mostly rely on a similar suite of indicators for each LOE, but differ in how the LOEs are combined into a single assessment. Some are based on combinations of binary responses for each LOE, while others use a more complex statistical summarization. Additionally, some

approaches weight the three LOEs equally, while others place differing weight among them. Even within an integration framework, thresholds need to be determined for each LOE. Consensus thresholds for these LOEs don't yet exist and these threshold decisions are particularly important when the integration is based on a binary decision for each LOE.

Table 5.9. Classification Accuracy and Bias for Indices and Index Combinations

No. of Indices	#	Measure	Southern California Bays (n=24)			Polyhaline San Francisco Bay (n=10)		
			Category Accuracy (%)	Category Bias	Status Accuracy (%)	Category Accuracy (%)	Category Bias	Status Accuracy (%)
One	1	BQI	62.5	8	79.2	90.0	-1	100.0
	2	BRI	58.3	-3	87.5	70.0	-1	100.0
	3	IBI	50.0	-8	70.8	75.0	-1	100.0
	4	RBI	50.0	10	70.8	70.0	3	100.0
	5	RIV	66.7	3	87.5	80.0	0	100.0
Two	6	BQI, BRI	54.2	7	79.2	90.0	1	100.0
	7	BQI, IBI	58.3	6	79.2	90.0	-1	100.0
	8	BQI, RBI	45.8	13	75.0	70.0	3	100.0
	9	BQI, RIV	62.5	11	75.0	80.0	0	100.0
	10	BRI, IBI	66.7	0	83.3	70.0	-1	100.0
	11	BRI, RBI	58.3	9	83.3	70.0	3	100.0
	12	BRI, RIV	62.5	6	83.3	90.0	1	100.0
	13	IBI, RBI	45.8	8	70.8	70.0	3	100.0
	14	IBI, RIV	66.7	3	87.5	80.0	0	100.0
	15	RBI, RIV	45.8	13	75.0	70.0	3	100.0
Three	16	BRI IBI RBI	70.8	-1	87.5	80.0	2	100.0
	17	BQI BRI IBI	66.7	0	87.5	80.0	0	100.0
	18	BQI BRI RBI	70.8	5	83.3	90.0	1	100.0
	19	BQI BRI RIV	70.8	3	91.7	80.0	0	100.0
	20	BQI IBI RBI	66.7	6	83.3	70.0	1	100.0
	21	BQI IBI RIV	75.0	2	91.7	80.0	0	100.0
	22	BQI RBI RIV	66.7	6	83.3	80.0	0	100.0
	23	BRI IBI RIV	62.5	-3	87.5	80.0	0	100.0
	24	BRI RBI RIV	75.0	2	91.7	90.0	1	100.0
	25	IBI RBI RIV	75.0	2	91.7	70.0	1	100.0
Four	26	BRI IBI RBI RIV	75.0	4	91.7	90.0	1	100.0
	27	BQI IBI RBI RIV	66.7	6	83.3	80.0	0	100.0
	28	BQI BRI RBI RIV	70.8	7	83.3	90.0	1	100.0
	29	BQI BRI IBI RIV	79.2	5	91.7	80.0	0	100.0
	30	BQI BRI IBI RBI	70.8	7	83.3	90.0	1	100.0
Five	31	All	75.0	4	91.7	80.0	0	100.0
Individual Experts vs Consensus		Minimum	62.5	+1, -1	83.3	60.0	0	90.0
		Average	80.1	-0.2	91.2	84.4	0.56	94.4
		Maximum	87.5	+4, -3	100.0	100.0	+4, -2	100.0

Note:
Classification accuracy is presented for "undisturbed" vs. "disturbed" status and four condition categories. Each of 34 evaluation samples was assessed into one of four numeric categories by the index or index combination and compared with consensus categories from an independent assessment by nine benthic experts. Bias is the sum of differences between index combination and consensus categories; positive values indicate a tendency to score samples as more disturbed than the expert consensus, while negative values indicate a tendency to score samples as less disturbed. The categories were 1: Reference; 2: Marginal; 3: Affected; 4: Severely Affected. Categories 1 and 2 were considered "undisturbed" and 3 and 4 as "disturbed." Index results were combined as the median of the numeric categories; if the median fell between categories, it was rounded to the higher effect category. Means, minima and maxima for concordance between individual experts and the expert consensus are presented below to provide context for the index results. (Table from Ranasinghe et al. 2007a)

At present, no single, universally accepted method for interpreting triad data and classification of sediments based on an MLOE approach exists (Chapman *et al.* 2002; Wenning *et al.* 2005). Each regulatory or monitoring program uses an approach developed through their unique experience. As a result, most triad applications rely on some degree of best professional judgment (BPJ) (Burton *et al.* 2002, Chapman and Anderson 2005). Despite the many decisions inherent in integration of LOEs, BPJ has been found to be reasonably repeatable for interpretation of triad data (Bay *et al.* 2007c). Thus, BPJ can be an acceptable means of integration for site-specific assessments, but it is not easily applicable to large-scale assessments where many sites are involved. As discussed in Section 2, these approaches are rarely if ever applied within the context of a water quality control program.

Within a large and densely populated state like California, the utility of BPJ is limited for many reasons. Its use:

- May result in inconsistent decisions within a single region and from region to region.
- Can be time consuming and resource intensive.
- May not always lead to transparent and unbiased decisions.
- May not allow Regional Water Board staff, permittees, or interested parties to assess the outcome independently.

Logic systems are frequently used to integrate MLOE data; the sediment quality triad was one of the first examples of the use of a logic system to evaluate sediment quality data. Tabular decision matrices that provide an interpretation of various MLOE scenarios are used to apply a logic system. These logic systems are based on a transparent set of criteria used to infer the likelihood of causality for contaminant-related impacts and the system can accommodate various types of scoring systems within each LOE. The rules applied in a logic system can also be modified to reflect specific policy objectives or scientific assumptions, such as giving greater weight to benthic community disturbance relative to toxicity.

The State Water Board's technical team developed a logic-based framework for integrating MLOE to make a station level determination of the likelihood of biological effects due to sediment contamination (Bay and Weisberg 2007). This system was developed in consultation with a stakeholder advisory committee and an independent scientific steering committee. The framework for integrating the three lines of evidence (LOE) to create a station assessment involves a three-step process (Figure 5.2). First, the response for each LOE is assigned into one of four response categories: 1) no difference from background conditions, 2) a small response that might not be statistically distinguishable from background conditions, 3) a response that is clearly distinguishable from background, and 4) a large response indicative of extreme conditions.

Second, the individual LOEs are combined to address two key elements of a risk assessment paradigm: 1) Is there biological degradation at the site and 2) Is chemical exposure at the site high enough to potentially result in a biological response? To answer the first question, the benthos and toxicity LOE are integrated to assess the severity of effect (Table 5.10). Benthos is given greater weight in this assessment, as it is the ultimate endpoint of interest (Chapman 2007). The second question arises because the biological response may be attributable to factors other than chemical contaminants. The potential that effects are chemically mediated is assessed using the sediment chemistry and toxicity LOEs (Table 5.11). Chemistry is the more direct measure, but toxicity is also included in this step because of the potential that unmeasured chemicals are present and because of uncertainties in thresholds used to interpret chemical data (Ingersoll *et al.* 2005).

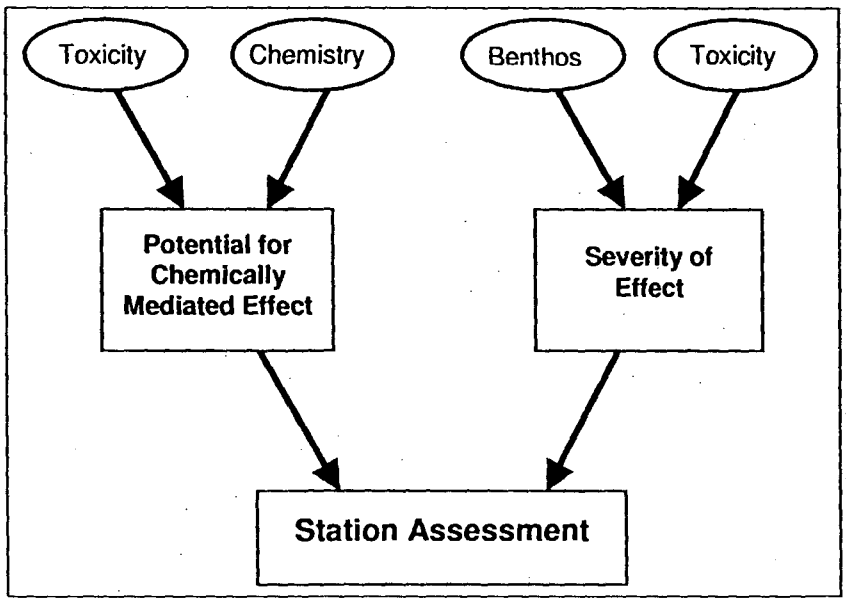

Figure 5.2. Schematic of Multiple Lines of Evidence (MLOE) Integration Framework

Table 5.10. Severity of Effect Classifications, Derived from Benthos and Toxicity LOE

		Toxicity			
		Nontoxic	Low Toxicity	Moderate Toxicity	High Toxicity
Benthos	Reference	Unaffected	Unaffected	Unaffected	Low effect
	Low disturbance	Unaffected	Low effect	Low effect	Low effect
	Moderate disturbance	Moderate effect	Moderate effect	Moderate effect	Moderate effect
	High disturbance	Moderate effect	High Effect	High Effect	High Effect

Table 5.11. Potential that Effects Are Chemically-Mediated Categories, Derived from Chemistry and Toxicity LOE

		Toxicity			
		Nontoxic	Low Toxicity	Moderate Toxicity	High Toxicity
Chemistry	Minimal exposure	Minimal potential	Minimal potential	Low potential	Moderate potential
	Low exposure	Minimal potential	Low potential	Moderate potential	Moderate potential
	Moderate exposure	Low potential	Moderate potential	Moderate potential	Moderate potential
	High exposure	Moderate potential	Moderate potential	High potential	High potential

The final data integration step combines the severity of effect and potential for chemically mediated effects to assign a site into one of six impact categories:

- ***Unimpacted***—Confident that sediment contamination is not causing significant adverse impacts to aquatic life living in the sediment at the site.
- ***Likely Unimpacted***—Sediment contamination at the site is not expected to cause adverse impacts to aquatic life, but some disagreement among the LOE reduces certainty in classifying the site as unimpacted.
- ***Possibly Impacted***—Sediment contamination at the site may be causing adverse impacts to aquatic life, but these impacts are either small or uncertain because of disagreement among LOE.
- ***Likely Impacted***—Evidence for a contaminant-related impact to aquatic life at the site is persuasive, even if there is some disagreement among LOE.
- ***Clearly Impacted***—Sediment contamination at the site is causing clear and severe adverse impacts to aquatic life.
- ***Inconclusive***—Disagreement among the LOE suggests that either the data are suspect or that additional information is needed before a classification can be made.

The decision process for determining the station assessment category is based on a foundation that there must be some evidence of biological effect in order to classify a station as impacted (Table 5.12). Additionally, there must be some evidence of elevated chemical exposure in order to classify a station as chemically impacted.

Table 5.12 Multiple lines of evidence station classifications.

		Severity of Effect			
		Unaffected	Low Effect	Moderate Effect	High Effect
Potential that effects are chemically-mediated	Minimal potential	Unimpacted	Likely unimpacted	Likely unimpacted	Inconclusive
	Low potential	Unimpacted	Likely unimpacted	Possibly impacted	Possibly impacted
	Moderate potential	Likely unimpacted	Possibly impacted or Inconclusive*	Likely impacted	Likely impacted
	High potential	Inconclusive	Likely impacted	Clearly impacted	Clearly impacted

* Inconclusive category when chemistry = minimal exposure, benthos = reference, and toxicity= high.

The efficacy of the framework was assessed by applying it to data from 25 sites and comparing the site classifications to that of six experts that were provided the same data. The framework produced an answer that better matched the median classification of the experts than did five of the six experts (Table 5.13). Moreover, there was little bias in response, as the errors were relatively evenly divided between sites classified as more impacted or less impacted than the median expert classification. The framework was also applied and found to distinguish well sites from known degraded and reference areas within California.

Table 5.13. Summary of Categorical Assessments for Each Expert

	Expert 1	Expert 2	Expert 3	Expert 4	Expert 5	Expert 6	Framework
# Sites	25	22	25	19	25	22	25
Disagreement	6	16	13	10	14	5	6
Bias	4	-14	12	7	-14	-1	2

Note:
Differences in the number of sites are due to the exclusion of sites classified as inconclusive. Disagreement values represent the total number of category differences between the expert's assessment and the median of all other experts' assessments. Bias values reflect the net of positive or negative assessment differences, with positive numbers indicating a bias toward rating the site as more impacted.

Baseline—MLOE is integrated based upon BPJ on a case-by-case basis.

Alternative 1—Support an approach based upon BPJ. As described above, using BPJ does provide some consistency when highly experienced sediment quality scientists are making the assessment, however discrepancies still occur. Water Board staff do not currently have the same level of expertise. A lack of qualified staff would limit the ability to implement this alternative

Alternative 2—Select an integration method that is based upon a transparent logic-based framework that has been evaluated for accuracy relative to experts and is supported by independent scientific peer review.

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Section VII. I. Presented in Appendix C is an example problem and solution based upon the proposed language.

5.5 Indicators Applicable in Estuarine Habitats

5.5.1 Potential Interim Tools and Methods for the Delta and Other Estuaries

The State Water Board initiated development of SQOs in 2003 in order to comply with Water Code section 13393 and a court-ordered compliance schedule (See Section 1.2). The schedule required the State Water Board to circulate draft objectives and an implementation policy by August 2006 and to approve and submit the package to the Office of Administrative Law by February 2008.

Section 13393 requires the State Water Board to develop SQOs for bays and estuaries of California. As described in Section 2.2, the State Water Board's Phase I effort focused on those water bodies where chemical and biological data were available to develop indicators and tools to assess sediment quality. Only within southern California bays and most of San Francisco Bay were enough data available to evaluate exposure and effects relationships. Most estuaries including the Sacramento-San Joaquin Delta have not been monitored routinely to assess the impact of toxic pollutants to sediment dwelling organisms; therefore, very little combined effects and exposure data exist within these water bodies. Where data is available, it often consists of only one to three data points. Clearly, the robust data sets required to assess the relationship between exposure and biological effects to benthic communities are far too sparse for the development of assessment tools.

Generally, the type of data required would consist of sediment chemistry-sediment toxicity and benthic community data that encompasses the range of pollutant impacts expected within

these water bodies. With such a data set, effects measures such as toxicity and community degradation can be assessed relative to pollutant loading and other disturbances. This is the general approach that has been applied to develop SQOs within California's embayments and is supported by the SQO Scientific Steering Committee. Although the State Water Board recognizes the need to collect additional data and provide funding to achieve this goal, the technical team will not have the data necessary to complete the appropriate analyses until 2008. As a result, there is a need to consider other interim options in order to comply with the court's decision.

Single LOE Chemistry or Toxicity

The State Water Board could propose the use of Sediment Chemistry Guidelines (SQGs) such as the ERMs (See Section 5.5.3.2) or apparent effects thresholds as a single LOE indicator of sediment quality in estuaries. SQGs are existing chemical thresholds that have been applied to assist managers when making decisions about sediment quality. Some of these approaches were developed in part from estuarine data. This approach would require little or no resources to prepare as existing sediment thresholds could be proposed and could be applied to determine whether sediment exceeds the narrative objective. As stated previously, there are significant problems when this LOE is used without the benefit of the other LOE.

Sediment toxicity could be proposed as a stand-alone tool for the assessment of sediment quality. There are two species within the proposed embayments suite of toxicity test methods that tolerate the salinity range of some estuarine waters. However, additional test methods need to be selected and calibrated in order to apply the recommended combination of acute and sublethal toxicity tests at most sites. As described above, this approach could be applied to determine whether sediment exceeds the narrative objective described in Section 2.11, or a toxicity-specific narrative objective could be proposed. Sediment toxicity has been applied within many different water bodies; however, similar limitations persist with this tool as well. The use of toxicity tests without other LOE would increase the likelihood of underestimating sediment that is due to seasonal events or contaminants that require chronic exposure to produce an adverse effect. Confounding factors and uncertainty also limit the ability to use this single LOE to assess sediment quality.

Combination of Sediment Chemistry and Toxicity

Sediment chemistry and toxicity could be integrated into a two-line of evidence approach. This approach would provide greater confidence in the assessment compared to a single LOE approach. However, the selection of appropriate thresholds would be difficult. Thresholds could be adopted from those proposed for sediment chemistry and toxicity in embayments. However, there may be little or no correlation between organism response in embayments and that in estuaries. The toxicity and chemistry lines of evidence could be interpreted relative to site-specific reference sites, providing only two possible outcomes for each LOE: good or bad. However, determination of reference sites is often contentious and typically requires a large amount of data to support the hypothesis. This approach gives more flexibility and responsibility to local agencies, and may be inconsistently applied.

The State Water Board would need to establish some thresholds to implement the two LOE approach in order to reduce the use of BPJ, which does not promote statewide consistency and promotes adversarial science. While it may not be possible to develop multiple thresholds that provide the same level of discrimination as those being developed for embayments, the

State Water Board could provide thresholds that would enable a manager to respond quickly to relatively high level of effects.

This approach would be developed based on the following considerations.

- Develop an integration approach that accounts for greater uncertainty associated with application in estuaries.
- Utilize fewer categories of effect or exposure to reflect present lack of knowledge.
- May require a greater number of inconclusive categories for situations where LOE are not in agreement, additional data collection (e.g., benthos) or analysis is needed before an assessment can be made. Current embayment chemical indicators and thresholds have not been validated for use in estuaries, and as a result may not be accurate or effective.
- Additional toxicity test methods that are compatible with freshwater (e.g., *Hyallella azteca* survival test and *Chironomus dilutus* growth test) may be needed, depending on salinities at time of collection.

Three LOE: Chemistry, Toxicity and Benthic Community

A more rigorous approach would be to use the sediment quality triad as it has been applied traditionally in areas where little prior sediment quality information was available. In this case, two independent data sets of chemistry, sediment toxicity and benthic community measures are required. The first data set would define the baseline conditions or reference envelope for the area of interest. A second data set would contain the sediment quality measures in the area of interest. Data from each line of evidence would be compared to the baseline data if adequate thresholds for data interpretation were not available. Statistically significant differences relative to the reference envelope among two of the three lines of evidence would trigger an impacted designation for the study site. This approach is consistent with the overall conceptual approach and underlying philosophy of the embayments approach and has been applied throughout the country.

Table 5.14. Potential Measures for LOE Evaluation in Estuaries

LOE	Measures	Comparison Value
Chemistry	Existing analyte list plus other chemicals of concern	Reference envelope or SQGs
Sediment Toxicity	Survival – *Hyalella azteca* Growth – *Chironomus dilutus*	Reference envelope or numeric threshold from similar programs
Benthic Community	Benthic macrofauna identification and abundance	Reference envelope

The sediment quality triad is commonly applied to assess sediment quality in habitats when little is known about the biological and toxicological characteristics of the study area. This approach requires an even greater use of BPJ compared to the two LOE approach. BPJ would be required to decide which measures to use, what thresholds or reference envelope to compare the results against, and how to integrate the LOE. The need to collect additional data in order to establish a reference envelope may also increase the cost and complexity of monitoring programs.

Baseline—Not applicable.

Alternative 1—Do not propose any tools for implementing the narrative SQOs until data is collected in Phase II, and the technical team has the time to develop appropriate tools.

Alternative 2—Propose the use of a single LOE for delta waters.

Alternative 3—Propose using sediment toxicity and chemistry to implement the narrative objective. The Scientific Steering Committee was critical of this approach.

Alternative 4—Propose using the sediment quality triad (chemistry, toxicity, benthic community condition) to implement the narrative objective. Additional development and evaluation will be required before a detailed approach is proposed.

Staff Recommendation—Alternative 4.

Proposed Language—See Appendix A, Section .V.J.

5.5.2 Sunset Date for Interim Tools

Some stakeholders have expressed concern that the State Water Board could adopt interim tools for the Delta and other estuaries without providing any guarantee that these tools will not be replaced by more fully developed implementation measures scheduled for development under Phase II. Although the State Water Board provided additional funding to develop Phase II tools, there is always some uncertainty associated with future planning efforts.

Baseline—Not applicable.

Alternative 1—Do not provide sunsetting language in Part 1 for the water bodies with less robust tools.

Alternative 2—Provide language that sunsets interim implementation tools if the State Water Board has not developed more robust tools by a specific date.

Alternative 3—Provide language in the resolution adopting Phase I that the State Board will revisit the interim implementation tools in Phase II

Staff Recommendation—Alternative 3.

5.6 Protective Condition

While proposing six categories to describe the condition of sediments provides for greater understanding of the sediment quality in a water body, the proposed Part 1 must define what categories are considered protective or degraded in order to fit the binary (pass/fail) model applied within all current regulatory programs. Section 13391.5(d) of Porter Cologne provides some guidance stating that the SQOs must be established with an adequate margin of safety for the reasonable protection of the beneficial uses of water. Defining what is protective versus what is considered the unprotective or degraded condition must meet this requirement.

As described previously, the six categories are:

- ***Unimpacted***—Confident that sediment contamination is not causing significant adverse impacts to aquatic life living in the sediment at the site.
- ***Likely Unimpacted***—Sediment contamination at the site is not expected to cause adverse impacts to aquatic life, but some disagreement among the LOE reduces certainty in classifying the site as unimpacted.

- ***Possibly Impacted***—Sediment contamination at the site may be causing adverse impacts to aquatic life, but these impacts are either small or uncertain because of disagreement among LOE.
- ***Likely Impacted***—Evidence for a contaminant-related impact to aquatic life at the site is persuasive, even if there is some disagreement among LOE.
- ***Clearly Impacted***—Sediment contamination at the site is causing clear and severe adverse impacts to aquatic life.
- ***Inconclusive***—Disagreement among the LOE suggests that either the data are suspect or that additional information is needed before a classification can be made.

Most would agree that from the definitions, *Unimpacted* would describe a protected condition while *"Clearly Impacted"* would represent a highly degraded condition. These two cases are the easiest to classify confidently as a result of strong concordance amongst all three LOE. The next two cases; *"Likely Unimpacted"* and *"Likely Impacted"* represent the protective and degraded condition albeit with a lower level of confidence as a result of some disagreement among the LOE, however within these categories, two of the LOE are compelling. The middle category designated *"Possibly Impacted"* represents the greatest uncertainty and disagreement amongst the LOE of the categories. Stations within this category may be either unimpacted or impacted.

There are five possible options that could be applied to provide a binary determination: Three of these options are considered below.

1. Protected sediments could be defined as those sediments within the *"Unimpacted"* Category only. All other categories would be considered as not representing the protective condition. This would represent a very conservative approach but does provide for an adequate margin of safety.
2. Protected sediments could be defined by the categories *"Unimpacted"* and *"Likely Unimpacted"*. All other categories would be considered as not representing the protective condition. This option also provides for a margin of safety as the next category *"Possibly Impacted"* indicates that there would be more sites in this category that are unimpacted then actually impacted.
3. Protected sediments could be defined by the categories *Unimpacted, Likely Unimpacted and Possibly Impacted.* All other categories would be considered as not representing the protective condition. While the *Possibly Impacted* category only suggests the possibility of the station being impacted, there is lower confidence that sediment quality at this site is protective relative to the proposed narrative objective.

Baseline—MLOE assessments applied sediment quality are typically decided by best professional judgment.

Alternative 1—Protected sediments could be defined as those sediments within the *"Unimpacted"* Category only.

Alternative 2—Protected sediments could be defined by the categories *"Unimpacted"* and *"Likely Unimpacted"*.

Alternative 3—Protected sediments could be defined by the categories *Unimpacted, Likely Unimpacted and Possibly Impacted*

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A Section .V.I and J.

5.7 Application of Proposed within Specific Programs

As explained in Section 4, the Basin Plans for all of the coastal Regional Water Boards contain water quality standards, including narrative water quality objectives, that apply to sediment quality in bays and estuaries. Under existing law, these standards are implemented through several regulatory programs. The standards are implemented in NPDES permits regulating the point source discharges and in waste discharge requirements, conditional waivers or prohibitions for nonpoint source discharges to bay and estuarine waters. The standards also provide the basis for enforcement actions, including cleanup and abatement activities, and for water quality certifications under Clean Water Act section 401. Bay and estuarine waters that do not meet the standards must be listed under Clean Water Act section 303(d), and appropriate TMDLs must be developed to attain the standards.

The proposed SQOs will add an objective that specifically addresses sediment quality in the coastal regions. The narrative SQOs and implementation tools were developed to assess whether pollutants in sediments pose a risk or are causing or contributing to the degradation of ecologically important and sensitive sediment dwelling organisms directly exposed to the pollutants in sediment. As a result, the SQO and tools will provide a robust measure of ambient sediment quality that directly relates to beneficial use protection.

The proposed SQOs will be implemented under the existing regulatory programs described in Section 4. This Section describes how the proposed SQOs could be implemented within these programs.

5.7.1 Applicability to Sediment Cleanup Actions

Part 1 could be applied to support site cleanup actions if the receptors addressed in Part 1 are consistent with those at risk. . Receptors that may be exposed include benthic invertebrates, fish, birds, marine mammals and humans through consumption of fish tissue. As a result human health and ecological risk assessments are used to both assess risk and assist in the derivation of receptor specific cleanup goals. The SQOs and supporting tools could be applied to determine what sediments within a specific area are protected or degraded for benthic communities. Stressor identification and development of site-specific management guidelines could also be applied to address potential cleanup actions focused on benthic communities.

As discussed in Section 4.2.2, Resolution *No. 92-49, "Policies and Procedures for Investigation and Cleanup and Abatement of Discharges Under Water Code Section 13304,* could be incorporated into Part 1 which encompasses both the investigation and development of cleanup goals. Under 92-49, cleanup levels range from background to the best water quality that is reasonable, but not to exceed applicable water quality standards. Development of biology based site-specific sediment management guidelines would assist Regional Boards in complying with this policy.

Baseline—Regional Water Boards require human health and/or ecological risk assessments to assess the exposure to all receptors. The relative risks posed to each receptor are calculated to determine which receptors are most sensitive to the pollutants of concern.

Alternative 1—Apply 92-49 to cleanups of sites not meeting the SQOs. Under 92-49, cleanup levels range from background to the best water quality that is reasonable, but not to

exceed applicable water quality standards. Stressor identification and development of site-specific sediment management guidelines could support this effort.

Alternative 2—Prepare language describing how and when the SQOs could be applied to cleanup actions. This policy could be applied to assist in characterizing risk at cleanup action sites when the receptors of interest, the exposure type, and scale of effort are identical or similar to those protected by this policy. The exposure receptor scenarios not protected by this policy would need to be evaluated using ecological and human health risk assessment guidance such as that prepared by the Department of Toxic Substances Control (DTSC), the Office of Environmental Health Hazard Assessment (OEHHA), and U.S. EPA.

Staff Recommendation—Alternative 1.

Proposed Language—See Appendix A, Section VII.G.

5.7.2 Applicability to dredged materials management

Water Code section 13396 states that the State and Regional Water Boards shall not grant approval for a dredging project that involves the removal or disturbance of sediment that contains pollutants at or above the (SQOs) established pursuant to Section 13393 unless the Water Boards determine all of the following:

(a) the polluted sediment will be removed in a manner that prevents or minimizes water quality degradation.

(b) polluted dredge spoils will not be deposited in a location that may cause significant adverse effects to aquatic life, fish, shellfish, or wildlife or may harm the beneficial uses of the receiving waters, or does not create maximum benefit to the people of the State.

(c) the project or activity will not cause significant adverse impacts upon a federal sanctuary, recreational area, or other waters of significant national importance.

California SQOs for enclosed bays and estuaries are being developed to protect sensitive aquatic organisms and other beneficial uses from the adverse effects of exposure to pollutants present in in-place surficial sediments. Section 13396 makes it clear that SQOs apply to dredged material. However, Section 13396 also allows dredged material that exceeds SQOs to be approved for discharge into waters of the State of California when conditions (a)-(c) are met. One difficulty is that some of the procedures used by California to determine the SQOs are not technically applicable to sediments below the biologically active layer (e.g., benthic community analysis). Dredged material, however, is typically composed *primarily* of sediments from below the biologically active layer. In addition, some of the test species used to determine the California SQOs are not necessarily appropriate to use for dredged material testing in all cases. The federal evaluation procedures discussed below were specifically developed to characterize the full spectrum of dredged material (not just surface sediments) in order to determine suitability for aquatic discharge in a variety of disposal or placement scenarios. Furthermore, the federal procedures emphasize conducting these dredged material evaluations in a nationally consistent manner.

Under the authority of the CWA and the Marine Protection, Research, and Sanctuaries Act (MPRSA), and their implementing regulations, the USACE and U.S. EPA jointly developed national testing guidance manuals for dredged material (the Inland Testing Manual or ITM for non-ocean waters, USACE and U.S. EPA 1998; and the Ocean Testing Manual or OTM for ocean waters, USACE and U.S. EPA 1991). These manuals utilize a tiered, effects-based evaluation scheme to determine the suitability of dredged material for aquatic placement or disposal. Each

of these national sediment-testing manuals is implemented under a national Technical Framework for Dredged Material Management ("Framework") also jointly published by the USACE and U.S. EPA. (1992). The purpose of the Framework is to facilitate consistency in how the sediment evaluation procedures are applied within and between various areas of the United States. In addition, the Framework describes the broader regulatory context within which sediment evaluations conducted under the ITM or OTM are carried out so as to meet the overall goals of the CWA and MPRSA. In particular, under the Framework, suitability determinations for aquatic discharge of dredged material take into account not only the technical sediment test results from the ITM or OTM, but also the characteristics of the individual disposal sites and the practicability of alternatives to aquatic disposal (including beneficial reuse alternatives).

Certain other federal programs that otherwise address contaminated sediments generally defer to this Framework when it comes to management of dredged material. For example, in U.S. EPA Region 9, U.S. EPA regularly allows navigation dredging to continue within the boundaries of sediment remediation study areas for projects in the Remedial Investigation/Feasibility Study (RI/FS) stage under the Comprehensive Environmental Recovery, Cleanup, and Liability Act (CERCLA), provided that the dredged material is first specifically evaluated under the Framework, and its discharge is managed under a CWA Section 404 or MPRSA Section 103 permit. Similarly, at the national level, U.S. EPA excluded dredged material from the definition of hazardous waste under Subtitle C of the Resource Conservation and Recovery Act (RCRA), when it is subject to a CWA Section 404 or MPRSA Section 103 permit. As U.S. EPA noted in the Hazardous Remediation Waste Management Requirements (HWIR-Media) Final Rule (U.S. EPA 1998A):

> "Dredged material that is subject to the requirements of a permit that has been issued under 404 of the Federal Water Pollution Control Act (33 U.S.C.1344) or section 103 of the Marine Protection, Research, and Sanctuaries Act of 1972 (33 U.S.C. 1413) is not a hazardous waste."
>
> "Testing procedures under the CWA and MPRSA ... are better suited to the chemical and biological evaluation of dredged material disposed of in the aquatic environment. These tests are specifically designed to evaluate effects such as the potential contaminant-related impacts associated with the discharge of dredged material into oceans and waterways of the United States. The Agency believes that the CWA and MPRSA permit programs protect human health and the environment from the consequences of dredged material disposal to an extent that is at least as protective as the RCRA Subtitle C program. These programs incorporate appropriate biological and chemical assessments to evaluate potential impacts on water column and benthic organisms, and the potential for human health impacts caused by food chain transfer of contaminants. As improved assessment methods are developed, they can be incorporated into these procedures. The programs also make available appropriate control measures (for example, 40 CFR 230.72) for addressing contamination in each of the relevant pathways."

Under the federal Framework (USACE and U.S. EPA, 1992) the ITM and OTM provide for application of relevant chemical sediment quality criteria (SQC) or Sediment Quality Standards (SQS) issued by U.S. EPA or by a state, respectively, as screening step in "Tier I" or "Tier II" of their evaluation procedures. Exceedance of SQC or SQS indicates the need for direct effects-based testing at a higher tier. Any numeric chemical SQOs that California promulgates could be applied in this manner. Section 13396 provides that even when California SQOs are exceeded, dredging and discharge may still be allowed when conditions (a)-(c) are met. As described

below, the higher-tier evaluation procedures of the ITM or OTM, and other considerations of the CWA and MPRSA as described in the Framework, provide an appropriate and consistent basis for the State to determine whether conditions (a)-(c) have in fact been met.

Condition (a) requires that the polluted sediment will be removed in a manner that prevents or minimizes water quality degradation. This condition focuses on the dredging (or removal) site itself, as opposed to the dredged material disposal site. It is addressed by any Best Management Practices (BMPs) or special conditions, incorporated in the dredging permit(s) or other authorizations, that federal or State agencies (including the State and Regional Water Boards) determine to be necessary for the protection of water quality and beneficial uses. These may include monitoring; constraints on dredging equipment type; operation; and timing, control technologies such as silt curtains, etc. The federal evaluation Framework generates specific information relevant to making determinations about the need for any controls at the dredging site, via physical-chemical characterization and via the water column (suspended-liquid phase) bioassays conducted on dredged material samples.

Condition (b) focuses on the discharge of dredged material at the disposal or placement site. The evaluation procedures in the ITM and OTM were specifically designed to address each of the relevant pollutant exposure pathways that may be associated with dredged material discharges at aquatic disposal sites. These procedures provide for the comprehensive physical, chemical, and biological evaluation of the specific sediments to be dredged and discharged. Biological testing includes both liquid-suspended phase and solid phase sediment testing using appropriately sensitive indicator organisms that cover a range of functional feeding types. There is flexibility to use appropriate species for different dredged material types and situations. When necessary, information from the bioaccumulation tests can be readily used to assess the environmental risk of food web transfer of pollutants to different trophic levels. The national testing manuals also provide for updating the specific tests used; for example, to include regionally important species or as more sensitive tests (possibly including chronic/sublethal assays) are developed sufficiently for reliable regulatory use nationwide.

Another important consideration is that dredged material that may pose a risk at a particular disposal site or when managed in a particular manner, may not pose such a risk at a different disposal site or if managed in a different manner. The overall federal Framework incorporates CWA and MPRSA provisions that ensure suitable determinations take into account all relevant sediment-specific and disposal site-specific factors, and any management actions necessary to minimize adverse impacts. SQOs as stand-alone factors cannot do this.

Condition (c) is consistent with already existing requirements of the CWA and MPRSA programs. In particular, the USACE generally may not authorize the discharge of dredged (or fill) material into waters of the United States that would cause the kinds of impacts listed in 40 C.F.R. §230.10, including significant impacts to designated marine sanctuaries, whether such impacts are caused by pollutants associated with the sediments or simply by the physical discharge of the sediments. In addition, the CWA program focuses on identifying and, to the maximum extent possible, avoiding impacts to "aquatic resources of national importance."

Baseline—USACE, under the authority of the federal CWA and MPRSA and in coordination with U.S. EPA, prepared the ITM (USACE and U.S. EPA 1998) and the OTM (USACE and U.S. EPA 1992) to address the suitability of dredged material for disposal. These manuals are not intended to assess in-place sediments; rather, these methodologies were designed to assess potential effects that may occur during or after disposal of the dredged materials. At the regional level, USACE, U.S. EPA, State Water Board staff, and staff from other State agencies have also prepared water body specific guidance and formed dredged materials management teams to streamline the onerous multi-jurisdictional regulatory process (USACE et al., 2001).

Alternative 1—SQOs should be applicable to dredged material. The proposed SQOs could be applied to dredged materials; however, collection of this information would not eliminate the need to perform the suitability tests described in the ITM or the OTM in accordance with the federal CWA or MPRSA.

Alternative 2—SQOs should not be applicable to dredged materials. These SQOs and supporting tools were intended to evaluate beneficial uses protection and, as a result, only focus on the in-place biologically active layer. The Dredged Materials program was designed to measure average bulk properties of sediment to determine both the appropriate method of disposal or reuse and assess potential effects caused by the dredging and disposal action. While some tools are similar, the application and implementation of the tools differs significantly.

Alternative 3—SQOs would only apply under specific conditions specified in section 13396.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Section VII.A.

5.7.3 Applicability to 303(d) Listings

As explained in Section 4.2.1 of this report, the State Water Board's Section 303(d) Listing Policy currently provides that a water segment shall be placed on the list if the segment exhibits statistically significant sediment toxicity based on a binomial distribution. The segment must be listed if the observed sediment toxicity is associated with a pollutant. The segment may also be listed for toxicity alone. The Section 303(d) Listing Policy predates Part 1; consequently, the policy does not specifically address listings based on the proposed SQOs.

A multi-station assessment tool will integrate the results of many single station assessments into a single watershed-based or water body assessment. This tool will help determine whether the water body is consistent with the narrative SQOs. The proposed MLOE approach uses evidence from chemistry, toxicity, and the benthic community structure to make a single station assessment. At each station, sediment quality will be categorized into one of five ordered categories: "unimpacted" "likely unimpacted" "possibly impacted" "likely impacted" "clearly impacted." This type of ordinal data is interpretable in terms of its arrangement in a given order, e.g., from lowest to highest.

Results measured on an ordinal scale, however, may limit the types of appropriate statistical methods that can be applied during a multi-station assessment. Nonparametric methods are usually used with ordinal data, while parametric methods are usually used with interval or ratio data (Stevens 1946). Some researchers, however, have concluded that treating ordinal data as if they were interval data is unlikely to lead to improper conclusions (Gardner 1975). The following is a list of preliminary ideas for statistical tests that could be used to assess multiple station sediment data:

- ***Tests of Exceedance***—Convert each single station assessment into binary yes-or-no type data value. A water body would then be characterized by a count of the number of exceedances and the number of non-exceedances. A binomial test can then be used to determine if the proportion of exceedances is significantly excessive. This is the approach taken in the State's current 303(d) Listing Policy (SWRCB 2004) for listings based on exceedances of numeric criteria or objectives for toxic pollutants. This approach does not consider the magnitude of the exceedance. For this alternative, it is important to understand that the application of SQOs cannot supersede all sediment listing criteria for several reasons
  - There are many waterbodies where SQOs do not apply, such as rivers, lakes, and ocean waters.
  - The SQOs were not developed to assess exposure associated with "non" priority pollutants
  - The SQOs were not developed to explicitly protect receptors such as fish, birds, marine mammals and the bioaccumulation from sediment up the aquatic food chain
- ***Goodness of Fit Tests***—The observed frequencies in each assessment category are compared to frequencies expected in each category under a specified null distribution. Sufficiently large deviations from the expected frequencies will support the conclusion that the data did not come from the hypothesized distribution. Chi-squared and Kolmogorov-Smirnov one-sample goodness-of-fit tests are examples. This option does not fully utilize the ordinal scale of the data.
- ***Tests of Location***—These tests work by subjectively assigning numeric integer values to ordinal data. For example, a value of 1 is assigned to stations classified as "unimpacted," a value of 2 is assigned to stations classified as "likely unimpacted," and so on. A one-sample parametric *t*-test can be used to test for a significant difference between the observed mean and the hypothesized mean. Similarly, a one-sample non-parametric Wilcoxon signed rank test can be used to test for a significant difference between the observed median and the hypothesized median. These tests of location account for magnitude.

Alternative 1—Do not consider the SQOs for listing purposes.

Alternative 2—Utilize an approach that is consistent with the approach for listing waters based on exceedances of numeric criteria or objectives for toxic pollutants, which is described in 303(d) listing policy (SWRCB 2004) and under a. above.

Alternative 3—Evaluate a variety of approaches such as b and c described above for applying SQOs to the listing process.

Staff Recommendation—Alternative 2. Under this alternative, the Water Boards will continue to list water segments for sediment toxicity under the current Section 303(d) Listing Policy, unless the listing is due to exceedance of the aquatic life SQOs in Part 1 in bays or estuaries. In the latter case, listings will follow the approach described in a. above. The State Water Board may reconsider the Section 303(d) Listing Policy, if appropriate, in the future to further address listings for sediment toxicity.

Proposed Language—See Appendix A Section VII.E.8.

5.7.4 Applicability to NPDES Permits

In general, under the Clean Water Act and implementing regulations, water quality objectives are typically translated into effluent limits when the discharge of specific pollutants has the "reasonable potential" to cause or contribute to water quality standards exceedances. In assessing reasonable potential, the permitting authority can consider a variety of factors and information. The State Water Board's SIP contains specific requirements for determining the need for numeric effluent limitations regulating the discharge of priority toxic pollutants. Additional guidance on determining reasonable potential is found in U.S. EPA's Technical Support Document for Water Quality-based Toxics Control (1991).

During the late 1980's, the State Water Board assessed the relationship between sediment deposition, pollutant loading, and effluent quality (Hendricks 1990) in an attempt to develop a process for deriving sediment-based effluent limits. The Washington Department of Ecology developed similar tools to calculate effluent limits based upon chemical concentrations in sediments within Puget Sound (Bailey 2005). Application of these tools to derive effluent limits has been limited for several reasons.

- Chemical concentrations in sediment do not represent the bioavailable fraction.
- Chemical thresholds are not based upon causal association.
- Pollutants discharged undergo chemical processes that vary depending upon the chemistry and physical properties of the effluent and receiving water.
- Sediment fate and transport must be well characterized.

In appropriate cases, water quality objectives can also be implemented in NPDES permits as receiving water limits. Receiving water limits are typically used when the water quality objective cannot be directly translated to effluent limits or when there is a clear need to monitor compliance within the receiving water. Examples include biological narratives and bacteria receiving water limits described in the California Ocean Plan (SWRCB, 2005).

Because it is not feasible at the present time to directly translate the SQOs into numeric effluent limits, the SQOs and implementation tools can be implemented as receiving water limits in NPDES permits. Receiving water limits should be included in permits if sediment quality in the vicinity of a permitted discharge to a bay or estuary is potentially at risk due to toxic pollutants in the discharge. In determining the need for receiving water limits, the Water Boards will have to use BPJ and consider all available and relevant information. This could include the location and characteristics of the discharge and the receiving waters.

Baseline—Not applicable.

Alternative 1—Do not address implementation of SQOs in NPDES permits.

Alternative 2—Develop translator tools that would enable the calculation of effluent limits from chemistry-based sediment thresholds.

Alternative 3—Propose that the narrative SQOs be implemented in NPDES permits as receiving water limits.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Section VII.B.

5.7.4.1 Defining Receiving Water Limit Exceedances

In general, demonstrating an exceedance of a numeric effluent limitation is fairly straightforward. Typically, there is an exceedance if the pollutant concentration in a monitoring sample is greater than the effluent limitation. Determining an exceedance of a receiving water limit implementing the proposed SQOs poses a greater challenge.

The proposed aquatic life SQO addresses pollutants in sediments that, alone or in combination, are toxic to benthic communities. The protected condition defined in Section 5.6 can be applied to individual stations. However integrating data from multiple stations is also necessary to ensure the evaluation of receiving water limits takes into consideration all available data. The protected condition defined in Section 5.6 could be coupled with the binomial statistic used by the Water Boards for 303(d) Listings to assess exceedances of receiving water limits using multiple stations designated in the permit. However coupling the MLOE based protected condition with the binomial statistic does not lead to the identity of a specific toxic pollutant stressor. In order to demonstrate an exceedance of the proposed SQO, a toxic pollutant or pollutants must be identified. Additional studies would be required to identify the specific cause. This effort requires stressor identification studies similar to the Toxicity Identification Evaluation process developed and utilized by U.S. EPA for the Whole Effluent Toxicity (WET) program (U.S. EPA 1999) and the process described in U.S. EPA's aquatic stressor identification guidance document (U.S. EPA 2002).

Performing stressor identification can also be tailored to address the confidence and magnitude of the assessment. For example sites classified as possibly impacted indicate that toxic pollutants may be causing adverse impacts to aquatic life (described in Section 5.6). In this case the exposure and biological effect maybe nominal or transient. In this example the ability to differentiate natural stressors and random variability from pollutant related stress might be difficult. However sites classified as likely or clearly impacted should clearly be prioritized for several reasons. First, the confidence in these assessment categories supports the need for priority response. Second, as the magnitude of the exposure and the biological effects increases, a greater number of tools could be applied to stressor identification, which increases the probability of establishing cause. Finally, resolving some of problems associated with likely and clearly impacted stations may help in resolving some of the problems associated with possibly impacted station clusters in the vicinity.

Baseline—Not applicable.

Alternative 1—Provide no guidance beyond the MLOE based protected condition as described in Section 5.6 to assess exceedances of receiving water limit.

Alternative 2—Provide guidance in Part 1 that would consist of a multi station assessment followed by stressor identification to determine the cause based upon the station categories. This language would also describe situations where findings support a conclusion that the narrative objective is met.

Staff Recommendation—Alternative 2

Proposed Language—See Appendix A, Section VII.C.

5.7.4.2 Monitoring Frequency in NPDES Permits

Sediment toxicity studies in southern California bays are indicate that there may be variable rates of temporal changes in sediment quality. Sediment toxicity in some bays has changed little over five years whereas conditions in other bays may change more frequently

(Bay et al. 2005). In San Francisco, sediment toxicity varies seasonally, yet the year-to-year sediment toxicity appears in many places to be relatively consistent (Anderson et al., 2007). The model monitoring program developed by SCCWRP for the southern California Bight recommends that the sediment monitoring frequency be based on the management objectives of the program, within a range of one to five years (Schiff et al., 2001). In order to monitor the impact of discharges the model-monitoring program suggests that the appropriate frequency be established after time series data has been collected for major dischargers. A frequency-limiting factor is the benthic community. The benthic indices proposed for use were developed using data collected in summer periods and as a result should only be applied during this season. Considering the above information the maximum frequency of once a year and a minimum frequency of once every five years (one permit cycle) would be appropriate.

Baseline—Not applicable.

Alternative 1—Do not specify a monitoring frequency. This alternative does not provide consistency throughout the coastal Regional Water Boards.

Alternative 2—Require permittees to collect time series data to determine appropriate frequency. This alternative may require the collection of a great deal of data before a monitoring frequency can be established.

Alternative 3—Require Phase I Stormwater Discharges and Major Discharges to monitor less frequently than twice per permit cycle. Require Phase II Stormwater and Minor Discharges to monitor more often then twice per permit cycle or less then once per permit cycle.

Staff Recommendation—Alternative 3.

Proposed Language—See Appendix A, Section VII.D.

5.7.4.3 Potential response actions for exceedances

Regulatory decisions or management actions are typically based upon the simple co-occurrence of pollutants that exceed a sediment quality guideline and biological effects measured at the same station or another station within the waterbody segment. Although this relationship does not demonstrate causality, TMDLs for each of these pollutants that exceed a sediment quality guideline are frequently required. As a result enormous resources are applied to develop control strategies for a large number of pollutants instead of focusing on the specific causes. There are also situations where routine chemical analysis does not include the identification of the pollutants that are responsible for the observed biological effects. In such situations, the true stressor is not considered in the development of control strategies. If stressor identification is performed and a stressor is identified, a logical application would be the development of biologically relevant guidelines that could be applied to support TMDL development or remediation goals. Guideline development would account for site and receptor specific factors that control bioavailability. Adopting sediment quality guidelines to fulfill this role does not account for these factors

Baseline—Not applicable.

Alternative 1—Do not provide guidance in Part 1 to support stressor identification and the development of additional biologically relevant guidelines in support of TMDLs or remediation goals

Alternative 2—Provide guidance in Part 1 to support stressor identification and the development of additional biologically relevant guidelines in support of TMDLs or remediation goals

Staff Recommendation—Alternative 2.

Proposed Language—See Appendix A, Section 3.VII.F and H.

5.7.4.4 Process Diagram for Application of the Direct Effects Narrative Objective

The Biological monitoring requires Sections 5.8.1 through 5.8.4 describe the potential means by which the direct effects narrative SQO and supporting tools could be applied within different water quality protection and control programs. However, members of the Advisory Committee have expressed a desire to include process figures to better communicate the basic approach and to further support consistent implementation and use across regions. The process figures would describe relevant response actions when an exceedance occurs and also describe situations where findings support a conclusion that the narrative objective is met.

Baseline—Not applicable.

Alternative 1—Do not include process figures in Part 1.

Alternative 2—Include process figures in Part 1

Proposed Language—See Appendix A, Figures 1 and 2.

6. ENVIRONMENTAL EFFECTS OF PART 1

6.1 REGULATORY REQUIREMENTS

This section presents the regulatory requirements for assessing environmental impacts under CEQA for the proposed *Water Quality Control Plan for Enclosed Bays and Estuaries of California Part 1 Sediment Quality* (Part I). Part I (Appendix A) is evaluated at a program level of detail under a certified regulatory program. As described in Section 1.5, state agencies are subject to the environmental impact assessment requirements of CEQA . However, CEQA authorizes the Secretary of the Resources Agency to exempt specific State regulatory programs from the requirements to prepare Environmental Impact Reports (EIRs), Negative Declarations, and Initial Studies, if certain conditions are met (Public Resources Code, §21080.5). While the "certified regulatory programs" of the State and Regional Water Boards are exempt from certain CEQA requirements, they are subject to the substantive requirements of California Code of Regulations, title 23, section 3777(a). This section requires a written report that includes a description of the proposed activity, an analysis of reasonable alternatives, and an identification of mitigation measures to minimize any significant adverse environmental impacts based on information developed before, during, and after the CEQA scoping process that is specified in California Public Resources Code section 21083.9.

Public scoping meetings were held in San Diego, Oakland and Rancho Cordova in the fall of 2006 to obtain input on the scope of this analysis. Comments received are posted on the Water Boards website at http://www.waterboards.ca.gov/bptcp/comments_sqo.html.

Section 3777(a) also requires the State Water Board to complete an environmental checklist as part of its substitute environmental documents. This checklist is provided in Appendix B of this document.

In addition, the State Water Board must fulfill substantive obligations when adopting performance standards, including water or sediment quality objectives. Public Resources Code section 21159 provides that an agency shall perform, at the time of the adoption of a rule or regulation requiring the installation of pollution control equipment, or a performance standard or treatment requirement, an environmental analysis of the reasonably foreseeable methods of compliance. The statute further requires that the environmental analysis, at a minimum, include all of the following:

- An analysis of the reasonably foreseeable environmental impacts of the methods of compliance.
- An analysis of reasonably foreseeable feasible mitigation measures to lessen the adverse environmental impacts.
- An analysis of reasonably foreseeable alternative means of compliance with the rule or regulation that would have less significant adverse impacts. (Pub. Resources Code, § 21159(a).)

6.2 DESCRIPTION OF ANALYSIS

Public Resources Code §21159(d) specifically states that the public agency is not required to conduct a "project level analysis." Rather, the project level analysis must be done by the lead agency that is required to comply with or implement the performance standard. Neither the State Water Board nor the Regional Water Boards can specify the manner of compliance with their regulations under Water Code §13360. Rather, the lead agency charged with complying

with or implementing the standard must conduct a project-level environmental review based on the particular compliance strategy.

Instead, this CEQA document represents a program level environmental analysis of thet Part 1 proposal. The document analyzes the reasonably foreseeable environmental impacts of the reasonably foreseeable methods of compliance within Part 1l. In conducting the program-level analysis, the State Water Board is not required to engage in speculation or conjecture. Reasonably foreseeable methods of compliance within Part 1 may include additional controls, remediation or the development of TMDLs to restore sediment quality. The corrective actions that require additional controls and or remediation will require a project level CEQA analysis (Pub. Res. Code § 21159.2.).

This analysis is based on the description of the environmental setting and existing conditions in Section 3, the regulatory baseline described in Section 4, the incremental changes that could result from the adoption of Part 1, the reasonably foreseeable environmental impacts associated with the reasonably foreseeable methods of compliance within Part 1, and reasonably foreseeable mitigation measures and alternatives.

As explained previously, the State Water Board's proposed program consists of the adoption of SQOs that address direct effects on benthic communities and indirect effects on human health of toxic pollutants in bays and estuaries. The primary outcome of this program will be the adoption of scientifically-defensible and environmentally-protective SQOs that can be consistently implemented throughout the state. As discussed in Section 4, the all coastal Regional Water Board basin plans currently contain narrative water quality objectives for toxicity or toxic substances, pesticides, bioaccumulation, or a combination of these that apply to sediment quality. In addition, existing basin plan prohibitions and numeric objectives and criteria for toxic pollutants, for example, the CTR criteria, affect sediment quality. Sediment cleanup and remediation programs are underway or planned in many regions because the sediments do not achieve the applicable objectives or other applicable requirements. These regulatory controls and activities would continue in the absence of this program. The extent to which additional controls on pollutant sources or additional remediation would be required under the proposed program, over the current baseline, is very difficult to determine. This analysis, nevertheless, assumes that adoption of Part I could potentially result in incremental controls or remediation activities over the current baseline.

If Part I is adopted, significant adverse environmental impacts are unlikely to occur from the Part 1 requirements for sampling, testing, sediment quality assessment, or stressor identification. If, however permittees or responsible parties are required to institute additional controls or corrective actions to comply with the proposed aquatic life SQOs for bays, over baseline conditions, these actions could result in potentially significant environmental impacts.

No potential significant adverse environmental impacts, over baseline conditions, are reasonably foreseeable if the proposed human health objective is adopted. Currently, waters are listed under CWA §303(d) as impaired if fish tissue advisory levels or other criteria are exceeded, and the levels or criteria are based on human health risk assessments. The proposed policy continues to use this approach.

Under Part I, compliance with the proposed aquatic life SQO for estuaries would be based on comparing coupled biological effects and chemistry data to reference site conditions. Due to a lack of existing coupled data and known reference sites, staff is unable to determine whether adoption of the proposed objective could result in potentially significant adverse environmental impacts. As noted above, the State Water Board is not required to engage in speculation. Nevertheless, the additional controls or corrective actions, if any, over baseline conditions,

stemming from adoption of the proposed objective for estuaries would likely be the same controls and actions required to comply with the proposed aquatic life objective for bays.

This report analyzes the reasonably foreseeable methods of compliance with Part I. This analysis takes into account the knowledge and understanding of baseline conditions and current Regional Water Board actions to restore beneficial uses. For example, it is not reasonably foreseeable that a project proponent would propose or that the Regional Water Board would approve, dredging and disposal of sediment from an entire water body as a result of sediment in the waterbody failing to meet a SQO. Dredging of this magnitude would be environmentally and economically infeasible. In the existing TMDL program, even legacy pollutants, those that are no longer in regular use or production, such as DDT, PCBs and mercury, are being controlled through means other than waterbody-wide dredging. Nor would staff anticipate a need for new wastewater treatment plants. All POTWs are required by the CWA to meet secondary treatment standards and many inland dischargers have or are in the process of upgrading to tertiary treatment. In addition, POTWs that discharge to bays and estuaries must comply with stringent CTR toxic pollutant criteria, which are implemented under the State Water Board's SIP, and must meet U.S. EPA's existing pretreatment program requirements. It is, therefore, unlikely that major modifications to existing POTWs or new POTWs would have to be constructed to meet the SQOs.

6.3 Summary of Baseline Conditions

Section 4 described the authority and means by which the State and Regional Boards initiate action to restore and protect beneficial uses through the control of existing discharges causing or contributing to the impact and/or the remediation of the impacted media itself by responsible parties. Currently, the risk to beneficial uses is evaluated based upon water, sediment and tissue data, which is compared to water quality criteria and objectives for priority pollutants in the CTR and basin plans, other numeric and narrative water quality objectives and prohibitions contained in basin plans, and other water quality control plans and policies, such as the 303(d) Listing Policy.

Section 3 described the beneficial uses designated for enclosed bays and estuaries that are impaired based upon the State Water Boards 303(d) List and/or designated as a Toxic Hotpots. Over one hundred segments are listed in bays and estuaries as a result of water-column, sediment or fish tissue-based impairments (Tables 3.1 through 3.16). There are also a number of sediment quality-related 303(d) listings for waters upstream of affected bays and estuaries. Impaired sediments can be carried downstream and settle into bays and estuaries, contributing to existing impairments or causing new ones. Unless de-listing occurs, all of these segments will require development of a TMDL to restore the beneficial use. The types of actions taken by permittees to comply with permit limits or wasteload allocations include additional pollution prevention education and awareness, modifications to pretreatment programs, construction or implementation of new BMPs or modification to existing BMPs, or process optimization or construction of additional treatment works.

Many Toxic Hotpots have been designated as 303(d) listed segments, however if existing sources are not contributing to the impairment, the extent of the impact is relatively localized and the listed segment or hotspot is significantly impacting beneficial uses, Regional Water Boards may require the area to be remediated. The types of action currently implemented by responsible parties to comply with cleanup and abatement orders include removal actions, capping and sequestering, in-situ remediation, natural attenuation or by other means described in the Consolidated Toxic Hotspots Cleanup Plan Amended Final Functional Equivalent Document (SWRCB 2004a).

6.4 INCREMENTAL IMPACTS ABOVE BASELINE CONDITIONS

If waters are identified as impaired because they fail to comply with the proposed SQOs, remediation activities or source control, or both, will be required to bring them into compliance. Many bays and estuaries are currently listed for sediment impairments and require controls under baseline conditions. Incremental sediment remediation, over baseline conditions, would be required under the proposed Part 1 only if monitoring data revealed biological impacts in areas that would not be designated for clean up under existing objectives. However, it is likely that most sites with sediment conditions that would require cleanup and remediation under Part 1 would also exceed current objectives. To the extent that results differ, it is possible that the additional assessment activities under Part 1 could lead to cleanup strategies that are more cost effective compared to baseline activities. In addition, based on the implementation plans for existing TMDLs, Regional Water Boards are likely to pursue source controls for ongoing sources and only require remediation activities for highly impacted localized sites affected by historical pollutants with no known, ongoing sources.

A review of available data and existing listings indicates that there is insufficient data to assess compliance with the SQOs for several enclosed bays and all estuaries. For enclosed bays with sufficient data, the review indicates that there are potentially eight bay segments that are not currently on the state's 303(d) list for sediment toxicity-related impacts for which the MLOE data indicates impairment under Part 1. Under baseline conditions, it is possible that the Regional Water Boards could identify these segments as impaired based upon existing narrative objectives even in the absence of Part 1. It should be noted that the Regional Water Boards identified the need for sediment cleanup and remediation for three of the eight segments under BPTCP. Assuming, however, that stressor identification and TMDL development are required for these segments under Part 1 and that these activities would not be pursued under baseline conditions, sediment remediation or other cleanup activities would be necessary.

In addition to the eight segments discussed above, the review indicated that three segments, which are currently listed on the 303(d) list for sediment-related degradation under the baseline, would not be impaired under Part 1. Adoption of Part 1 would result in cost savings for these sites.

Additional pollution control activities for on-going discharges under the proposed Part 1 could be required if the concentration of pollutants in discharges had to meet levels more stringent than required to achieve compliance with existing water quality objectives. Moreover, additional controls might be required to address previously unidentified chemical stressors. Without being able to identify the particular pollutants causing biological effects, however, or to determine the discharge concentrations necessary to achieve the proposed SQOs, it is difficult to determine whether, and to what extent, additional remediation or control activities will be necessary.

Assuming that additional controls on pollutant sources are necessary, the controls will likely focus on storm water sources, marinas, and wetlands. The degree to which incremental controls on these sources, over baseline conditions, would be required is uncertain. In any event, the reasonably foreseeable methods of compliance for storm water sources include increased or additional nonstructural and structural BMPs. For marinas and boating activities, reasonably foreseeable methods of compliance include the use of less toxic paint on boats and the use of containment or recovery equipment during hull maintenance activities. Wetlands controls may include aeration, channelization, revegetation, sediment removal, levees, or a combination of these practices.

6.5 PROGRAM ALTERNATIVES

Section 5 identified a series of issues and alternatives considered in the development of SQOs and Part 1. Of those Issues Staff have considered the following in this Tier I Programmatic Analysis:

1. No project alternative as described in Section 5.1.1
2. Selection of receptors as described in Section 5.3.2
3. The number of LOE as described in Section 5.5.1
4. Selecting alternative designation for the protected condition described in Section 5.6.1
5. The staff proposed Part 1 that protects specific receptors and utilizes MLOE to interpret the narrative objectives. The rationale and information supporting this approach that forms the foundation for Part 1 is described in Section 5.

No Project Alternative

Section 5.1.1 described the legal mandate that the State Water Board adopt SQOs. The State Water Board is bound by chapter 5.6 and the amended Settlement Agreement to develop and adopt SQOs. For this reason, the no project alternative is not feasible and is not considered further in this analysis.

Section of Alternate Receptors

The strengths and limitations of various receptors are examined in detail in Section 5.3.2. Although all receptors are important, the selection of receptors was based upon the type and magnitude of exposure based upon the life history of the organism, the ecological significance, sensitivity and response and the ability to evaluate the health of the receptor relative to pollutants in sediment. Selection of inappropriate receptors can have a significant impact on the environment. For instance, the selection of transient receptors may not respond to pollutants in sediment because the duration of the exposure is limited or the receptor may be exposed in other waterbodies and thus not represent an exposure at the area of concern. The selection of benthic communities and human health are both sensitive, relevant receptors and appropriate for Part 1

The Number of LOE to Assess Benthic Community Narrative Objective

The State Water Board could propose fewer LOE to support the narrative SQO, however the use of fewer LOE was not supported by the Scientific Steering Committee as an appropriate measure of sediment quality. As explained in Section 5.5.1, each LOE has strengths and weaknesses that must be considered in the application of the LOE as a measure of sediment quality. Through the application of three LOE, the weight of evidence can provide a more confident assessment that minimizes the weaknesses or limitations associated with the individual LOE used alone.

Alternative Designation for the Protected Condition

The selection of the protected condition clearly has significant potential to impact the environment. Staff has recommended that stations designated as Possibly Impacted, Likely Impacted and Clearly Impacted be considered as degraded. As discussed in Section 5.6.1, establishing this classification is consistent with chapter 5.7 , because the Possibly Impacted category represents the lowest level of impact. As described in Section 1.9, the purpose of the SQOs is to provide reasonable protection of beneficial uses, with an adequate margin of safety.

6.6 Reasonably Foreseeable Methods of Compliance

The primary limitation of the proposed SQOs is that the application of the indicators and thresholds to existing MLOE data from bays and estuaries does not provide any direct information on potential cause of an exceedance. Nor does the proposed SQO provide a pollutant-specific concentration that would be protective of aquatic life in sediment. As a result, evaluating reasonable means of compliance is difficult. It is also very difficult to determine whether there will be any reasonably foreseeable adverse environmental impacts stemming from the reasonably foreseeable methods of compliance over the current baseline.

There are an unlimited number of reasonable and foreseeable actions that could be implemented by permittees or responsible parties to comply with Part I. These actions can be categorized by controls that are applicable to the quality of water being discharged and remedial actions that are applied to reduce the risk associated with the pollutants already in the sediment. Controls may include the following:

Non-Structural Controls

- ***Public Education***—Education to promote pollution awareness on the proper use and proper disposal of products containing toxic pollutants, pollution prevention and minimization, and environmental stewardship
- ***Training***—Training programs can be used to support effective use of BMPs
- ***Water Conservation***—Water conservation reduces dry weather runoff that may carry sediment and pollutants directly into enclosed bays and estuaries or rivers draining into these waterbodies.
- ***Street cleaning (includes sweeping or washing)***—Frequent or more effective street sweeping or washing can reduce both sediment and pollutant runoff.

Structural Controls

Detention Basins/Retention Ponds—These ponds and basins can reduce the volume of suspended sediment and pollutants in stormwater by allowing suspended solids to settle out and reduce hydraulic load on the conveyance system.

Stormwater Diversions—Stormwater diversions have been constructed to divert dry season flows to wastewater treatment plants.

Vegetated Swales/Buffer Strips—Well maintained buffer strips constructed along roadsides and in medians can reduce the volume of sediment carried to storm drains.

Removal and Disposal of Polluted Soils—Soil containing toxic pollutant residuals may be removed from sewer lines and excavated out of stormwater channels or conveyances or public rights-of-way.

Treatment process optimization—Measures wastewater treatment plants can implement to modify or adjust the operating efficiency of the existing wastewater treatment process.

Pretreatment Program Assessment—Wastewater treatment plants can evaluate the effectiveness of the pretreatment programs and require upstream sources to reduce pollutant loading into the plant influent.

Treatment Plant Upgrades. Treatment plants may be upgraded to reduce pollutant concentrations in effluent.

Outfall Modifications—Treatment plants may relocate or redesign an outfall to reduce the potential impacts associated with the discharge of effluent. Redesign may include construction of a multi-port diffuser to increase dilution or relocation of the discharge into a location close to the ocean.

Remedial Actions

Remedial Actions are applied to restore the beneficial uses by reducing the risk of exposure to pollutants in sediment. The types of remedial action, potential environmental impacts and mitigation and relative costs are described in the Consolidated Toxic Hotspots Cleanup Plan Amended Final Functional Equivalent Document (SWRCB 2004a). Potential actions include:

- Capping/Sequestering of Polluted Sediments—If the polluted sediments are not limiting navigation and risk minimization is the objective, a well-engineered cap can reduce the mass of pollutants available for uptake or exposure.
- Dredging—Polluted sediments may be dredged from the water body for offsite disposal or remediation.
- In-situ Remediation
- Natural Attenuation

6.7 Potential Adverse Environmental Effects

For waterbodies identified as hotspots or placed on the CWA §303(d) list due to impaired sediment quality, the Regional Water Boards currently have the authority to issue and revise waste discharge requirements for ongoing pollutant sources, issue and implement enforcement actions to require remediation of these sites and/or develop TMDLs wasteload and load allocations to restore beneficial uses. Adoption of Part 1 will not alter this authority nor does adoption of Part 1 change the physical way in which the sites or waterbodies could be remediated or protected. Adoption of Part 1 could, however, result in incremental remediation activities or controls, or both, that could have reasonably foreseeable adverse environmental impacts.

Actions taken by the Regional Water Boards in response to sediment exceeding the proposed SQOs could result in degraded or adversely impacted biological resources, at least temporarily, during the construction of controls, treatment works, BMPs, or cleanup and mitigation efforts if these actions are not carefully planned and executed. Other impacts related to air quality, aesthetics, noise, hazardous materials, vehicle or vessel traffic could occur as well. Staff has determined that all of these potential impacts can be mitigated to less then significant levels with mitigation at the project level. When the SQOs are implemented on a project-specific basis, the agencies responsible for the project can and should incorporate the alternatives and mitigation measures into the project or project approvals.

Finally, it should be noted that Part 1 and management actions that occur as a result of adoption of Part 1 are intended to protect and restore the beneficial uses within bays and estuaries of California.

6.8 GROWTH-INDUCING IMPACTS

CEQA defines the expected discussion of growth-inducing impacts and indirect impacts associated with growth in section 15126(g) of the CEQA guidelines. That section states:

> "...Discuss the ways in which the proposed project could foster economic or population growth, or the construction of additional housing, either directly or indirectly, in the surrounding environment. Included in this are projects that would remove obstacles to population growth (a major expansion of a wastewater treatment plant might, for example, allow for more construction in service areas). Increase in the population may further tax existing community service facilities so consideration must be given to this impact. Also discuss the characteristics of some projects which may encourage and facilitate other activities that could significantly affect the environment, either individually or cumulatively. It must not be assumed that growth in any area is necessarily beneficial, detrimental, or of little significance to the environment."

Part 1 provides consistent approach to assess sediment quality relative to the narrative SQOs. The analysis of environmental impacts concludes that Part 1 will not have a significant effect on the environment. Part 1 is not expected to foster or inhibit economic or human population growth, or the construction of additional housing.

6.9 CUMULATIVE AND LONG-TERM IMPACTS

No cumulative adverse environmental impacts are expected at the program level from the adoption of Part I. Neither the State nor the Regional Water Boards have previously adopted SQOs. The State Water Board anticipates adopting refined SQOs for direct effects in estuaries and indirect effects in bays and estuaries in Phase II. The cumulative environmental impacts from the adoption of Phase I and Phase II are expected to be beneficial. The adoption of scientifically defensible and protective SQOs will ensure that aquatic life and human health beneficial uses are maintained and protected in coastal bays and estuaries. At the project level, the lead agency will have to analyze whether a compliance project could have environmentally cumulative effects. This analysis will depend on whether other related or unrelated projects are occurring in the same general time and space as the compliance project. Whether or not any potential significant adverse cumulative impacts could occur at the project level will depend on site-specific information related to the location, timing, and nature of the compliance action.

When considering cumulative and long-term impacts, Staff also considered the Part 1 potential contribution to global climate change. The State of California adopted Assembly Bill 32, the Global Warming Solutions Act of 2006. The Act requires the State to reduce its global warming emissions to 2000 levels by 2010 (11% below business as usual), to 1990 levels by 2020 (25% below business as usual), and 80% below 1990 levels by 2050. To that end, this CEQA analysis considers the potential of the proposed sediment quality objectives to impede efforts to achieve the mandated reductions.

Adoption of the proposed sediment quality objectives will not directly contribute to greenhouse gas (GHG) emissions, but consequent implementation of monitoring, clean-up and remediation activities could require the operation of equipment and vehicles that will generate emissions potentially contributing to GHG levels. Emissions from such operations are unknown but are unlikely to be significant when considered in the context of the state emissions inventory. In any event, due to the lack of data on potential emissions and their relative significance on global climate change, the potential cumulative impacts are far too speculative to analyze. At the programmatic level, it is not possible to estimate the number of monitoring

and remediation efforts that could be initiated, the equipment or vehicles that might be required, or the locations throughout the state where such actions might be undertaken. Efforts to assess the level of benefits or adverse impacts of such projects would be speculative at this time. Individual projects will be subject to the appropriate level of environmental review at the time they are proposed, and mitigation would be identified as warranted prior to approval.

6.10 Potential Environmental Impacts and Mitigation

In this section, Staff presents the rationale for the ratings of environmental impacts listed in the CEQA checklist presented in Appendix B and potential means to mitigate the impacts. As used in this analysis and as defined by CEQA (Article 20, Section 15370), mitigation can be divided into four types:

1. Avoiding the impact altogether by not taking a certain action or part of an action.
2. Minimizing impacts by limiting the degree or magnitude of the action and its implementation.
3. Rectifying or eliminating the impact over time by preservation and maintenance operations during the life of the action.
4. Compensating for the impact by replacing or providing substitute resources or environments.

It is likely that all of these mitigation strategies will be used alone or in a variety of combinations to address specific impacts associated with individual projects developed to restore or protect beneficial uses related to sediment quality.

It should be noted that Part 1 does not mandate any actions or projects that would lead to significant, permanent, negative impacts on the environment. However, this analysis also considers the reasonably foreseeable potential adverse environmental impacts stemming from the reasonably foreseeable methods of compliance with Part I, including additional controls or remediation or the development of TMDLs. Staff anticipate that all reasonably foreseeable potential environmental impacts will be mitigated to less-than-significant levels through a project-specific CEQA analysis, the Water Board's regulatory and permitting process or through other agencies with jurisdiction in relevant areas, such as U.S. EPA, USFWS, NMFS, OSHA, USACE, CDFG, DTSC, California Coastal Commission and San Francisco Bay Conservation and Development Commission (BCDC).

Aesthetics

Failure to meet the objectives could potentially result in construction activities for additional treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities. Thus, reasonably foreseeable short term impacts could occur during construction related activities. No long term impacts are anticipated that would result in substantial physical changes to the environment, including light or glare that would affect aesthetics. Construction activities could be limited to spring, fall, and winter week-days to avoid disrupting recreational, pleasure boating or site-seeing activities associated with the summer tourist season.

Agricultural Resources

Significant impacts would occur if a project substantially affected agricultural lands or production processes. There are no known or reasonably foreseeable impacts to agricultural resources due to the proposed adoption of Part 1. Furthermore, Part 1 relies on the Regional

Water Boards' Irrigated Lands Programs to determine how the SQOs will be implemented for those specific agricultural discharges that drain into bays and estuaries.

Air Quality

Failure to meet the proposed objectives could potentially result in construction activities for treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities. Emissions from this equipment vehicles and vessels have the potential for temporary adverse effects to air quality. The primary pollutants of concern in these emissions are NOx or nitrogen oxides. NOx are precursors to ozone formation, and many of the major embayments and the Sacramento San Joaquin Delta are located in areas designated as nonattainment areas for ozone. Other emissions of concern could be carbon monoxide and PM_{10} (particulate matter < 10 microns). Potential air quality impacts can be mitigated by operating equipment under permit, use of electric dredging equipment, planning the project for the time of year or day when emissions would be least likely to cause an exceedance of air quality standards, optimizing the mode of transportation, favoring disposal sites closer to dredge sites, and minimizing the number of trips necessary to transport dredged material to the disposal site or rehandling facility. Mitigation of air quality impacts will be considered under CEQA for each specific project.

Biological Resources

Failure to meet the proposed objectives could potentially result in construction activities for treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities. On land, there are no reasonably foreseeable impacts to biological resources from adoption of Part 1. The removal of soil could occur as part of land-based corrective action and control activities; however, many toxic pollutants found in sediments are typically found in highly urbanized, industrial areas where the presence of sensitive native species and habitats are improbable. Measures designed to intercept, divert, treat, and convey urban runoff to municipal wastewater treatment systems are only likely to occur at strategic locations in highly urbanized areas where the runoff requires additional controls.

In water, dredging, disposal, and capping all have the potential to cause adverse effects to biological resources in several ways: short-term habitat destruction and displacement of sensitive species, possibly during critical periods such as nesting, disturbance of sensitive spawning or migrating fish species due to turbidity, and "take" of endangered species.

Specific mitigation measures include adherence to established work windows to time dredging activities to avoid key seasonal activity of anadromous fish and bird species that inhabit nearshore areas either seasonally or year-round; use of electric dredge equipment; use of environmental (closed) clamshell buckets on dredges; and noise dampening material on equipment. Identification and mitigation of impacts to biological resources would be determined under CEQA for each specific project in consultation with the DFG and the USFWS.

Cultural Resources

Staff is not aware of any cultural resources present beneath subtidal sediments in bays and estuaries that could potentially be impacted through the adoption of Part 1. However, our lack of awareness does not preclude the possibility of previously unmapped cultural resources in near-shore locations that could be impacted by activities in response to exceedance of the narrative SQOs. As a result, any future actions that could impact cultural resources would be subject to CEQA on an individual case-by-case basis, and evaluated at that time.

Geology and Soils

Significant impacts to geology and soils would occur if a project exposed people or structures to potential, substantial adverse effects related to rupture of a known earthquake fault, other seismic events, or landslides. Significant impacts would also occur if a project caused substantial erosion or was located in areas with unsuitable soils or landslide-prone conditions. Although Part 1 does not mandate any specific remediation or corrective action, failure to meet the proposed objectives could potentially result in construction activities for treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging, excavation or construction activities. Dredging activities have the potential to destabilize channel slopes and undermine pilings. Standard engineering practices such as installation of sheet pile walls at the toe of the shore slope would reduce or avoid this impact.

Hazards and Hazardous Materials

This category refers to chemicals that have been discharged to the environment that may adversely impact the environment or human health and safety. Soil and groundwater impacted by such chemicals are also included. Significant impacts would occur if a project led to increased hazards to the public or environment from transport, handling, or emissions of such materials. Also included are projects located near airports and listed hazardous materials sites.

Failure to meet the proposed objectives could potentially result in construction activities for treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities. For these situations, potential impacts related to hazardous materials can be mitigated to less than significant levels with appropriate mitigation measures. In any action involving toxic pollutants, there is a potential for release of pollutants due to an accident or upset condition. The potential for such releases can be greatly reduced by proper planning. Measures to prevent releases of toxic pollutants include such things as pollution prevention technology (e.g., automatic sensors and shut-off valves, pressure and vacuum relief valves, secondary containment, air pollution control devices, double walled tanks and piping), access restrictions, fire controls, emergency power supplies, contingency planning for potential spills and releases, pollution prevention training and other types of mitigation appropriate to the cleanup plan.

Trucking hazardous wastes through neighborhoods has the potential to result in the possibility of fire or explosion; exclusion of hazardous waste from certain neighborhoods; inability to get bridge-crossing permits in a timely manner may limit the feasibility of remedial measures. It may be necessary to select a remediation measure such as capping to avoid such hazards. Fuels, lubricating oils, and other petroleum products will be used during cleanup activity. Well-established techniques for controlling spills, leaks, and drips will be incorporated in the work plans to assure the control of petroleum products and any other chemicals used during the cleanup activity.

Project workers and supervisors are required to comply with applicable Occupational of Health and Safety Administration (OSHA) training requirements for site clean-up personnel. In addition, site-specific health and safety plans would be prepared in accordance with California Code of Regulations, tit. 8, §5L92 and 29 C.F.R. § 1910.120, which govern site clean-up.

Potential management and remedial actions could include handling and transport of equipment, debris, scrap materials, soil and sediment containing potentially hazardous material. To protect people and the environment from potential impacts, the hazardous material must be handled, transported, and stored in accordance with applicable laws and regulations.

Hydrology and Water Quality

Significant impacts to hydrology and water quality would occur if a project substantially alters existing drainage patterns, alters the course of a river or stream, violates water quality standards, or creates or contributes to runoff that would exceed the capacity of local stormwater drainage systems. Significant impacts would also occur if a project placed housing or other structures within the 100-year flood plain, or exposed people or structures to significant risks from flooding, seiches, or tsunamis.

Failure to meet the proposed objectives could potentially result in construction activities for treatment works, BMPs, and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities.

Dredging equipment can cause turbulence in the water body, and, thus, the dredging process can cause short-term adverse impacts to water quality from turbidity or from stirring up pollutants in the sediment. These impacts can be regulated through WDRs and can be reduced by requiring use of dredging equipment or operations that minimize the discharge of chemical pollutants during dredging (e.g., use of clam shell dredger, etc.), use of settling tanks to reduce excessive turbidity in discharge, use of silt curtains to reduce dispersal of the turbidity plume beyond the dredge site, coffer dams in small channels, and accurate positioning of disposal equipment during dredging. DFG also has dredging regulations to protect against adverse biological impacts.

Some control or remedial actions could occur on the shoreline. Depending on the cleanup method selected for the shoreline activity, minor changes in absorption rates, drainage patterns, and the rate of surface runoff may change. On land, excavation can be mitigated by performing all work during the dry season and using BMPs for the control of erosion.

In addition, runoff from construction of BMPs, treatment works, excavation activities, or disposal of dredged materials above sea level can adversely affect surface water quality. Impacts from these activities can be reduced by doing work during the dry season or by implementing BMPs to reduce erosion. Most local governments also have erosion control ordinances and grading ordinances.

Stormwater diversions intended to improve water and sediment quality are not expected to degrade receiving water quality, rather these actions would improve water and sediment quality by means of additional treatment.

Changes in bottom contours brought by dredging or capping would probably have minimal effects on water circulation if properly managed. Relatively small areas are under consideration for modification at most of the sites. At larger sites, removal and placement will attempt to retain regional bottom depth and contour, except where bathymetry is planned for environmental improvement.

Land Use and Planning

Significant impacts to land use and planning would occur if a project physically divided a community, conflicted with a land use plan, policy or regulation, or caused conflict with a habitat conservation plan. General plans and zoning delineate those areas that will be developed, and the type and density of development to be allowed. There is nothing in Part 1 that requires the properties to be used in any way.

Mineral Resources

Significant impacts to mineral resources would occur if a project resulted in the loss of a mineral resource of value locally, regionally, or statewide. There is no evidence that the adoption of Part 1 would result in the loss of a known mineral resource or availability of the mineral resources.

Noise

Significant impacts from noise would occur if a project exposed people to noise or groundborne vibration in excess of established standards in a local general plan or noise ordinance or resulted in a substantial permanent increase to ambient noise levels. Significant impacts can also occur if a project causes substantial temporary or periodic increases in noise or if a project is located in the vicinity of an airport and would expose people residing or working in the project area to excessive noise levels.

Although Part 1 does not mandate any specific remediation or corrective action, failure to meet the objectives could potentially result in short-term noise related to construction activities and use of land or vessel-based heavy equipment for all projects involving dredging or construction activities. Mitigation would consists of compliance with local noise ordinances (typical standards include blackouts prohibiting use of heavy equipment on Sundays, early morning hours and evenings all week, and on holidays), use of noise dampening material or barriers around equipment, locating equipment as far as practical from noise-sensitive areas and selecting haul routes that affect the lowest number of people. These alternatives would be considered under CEQA for each specific project.

Population and Housing

Significant impacts to population and housing would occur if a project substantially encouraged population growth, displacing substantial numbers of people from existing housing and thereby necessitating construction of replacement housing elsewhere. Adoption of Part 1 will not result in the need for more housing or displace residents in existing communities. See discussion of growth-inducing impacts in Section 6 and Section 13241 factors in Section 7.

Public Services

Significant impacts to public services would occur if a project resulted in substantial physical impacts as a result of requirements for increased public services such as police, fire protection, schools, or other public facilities. Adoption of Part 1 will not result in the need for new government services for fire or police protection, education, or maintenance of public services.

Recreation

Significant impacts to recreation would occur if a project increased the use of existing park facilities such that physical impacts occurred if a project included construction or expansion of park facilities leading to physical impacts. Adoption of Part 1 would not create additional demand for parks or recreational facilities, but would have a positive impact on existing recreational opportunities such as fishing and swimming.

Transportation / Circulation

Significant impacts to transportation and traffic would occur if a project caused a substantial increase in traffic in relation to existing traffic load/capacity of the existing street

system, exceeded established level of service standards, resulted in change in air traffic patterns, lead to increases in road-related hazards, resulted in inadequate emergency access or parking. Adoption of Part 1 would not create additional vehicle or air traffic, or alter traffic patterns. Remediation of contaminated sediments may temporarily alter vessel traffic that would require approval from port authorities, Harbor Master and U.S. Coast Guard. However these impacts would be mitigated under CEQA specifically for each project.

Utilities and Service Systems

Significant impacts to utilities and service systems would occur if a project exceeded wastewater treatment standards, required construction of new water or wastewater treatment facilities or new or expanded storm water drainage facilities, or a project's water needs exceeded existing resources or entitlements. Significant impacts would also occur if a project was not served by a landfill with sufficient capacity or the project failed to comply with federal, state, or local regulations for solid waste. Although Part 1 does not mandate the construction of wastewater treatment facilities, failure to meet the objectives could potentially result in additional controls and treatment to reduce the discharge of pollutants into waterbodies. As stated previously, it is unlikely that treatment plants that comply with the CWA, the Water Code , the toxic pollutant criteria in the CTR, the implementation provisions in the SIP, and basin plans will cause exceedances of the proposed SQOs and Part 1.

Discharge reductions can be accomplished through (1) treatment process optimization (measures facilities can implement to modify or adjust the operating efficiency of the existing wastewater treatment process – such measures usually involve engineering analysis of the existing treatment process to identify adjustments to enhance pollutant removal or reduce chemical additional); (2) waste minimization/pollution prevention costs (conducting a facility waste minimization or pollution prevention study); (3) pretreatment (conducting study of sources and reducing inflow from indirect discharges); or (4) new or additional treatment systems. For stormwater, implementation of BMPs can also be applied to *reduce* pollutants, rather than treatment of storm water to *remove* pollutants. Because of the nature of storm water discharges, the Water Boards have not typically established numeric effluent limitations for toxic pollutants in storm water permits. The limitations contained in storm water permits are typically narrative and include the requirement to implement the appropriate control practices and/or BMPs. BMPs can range from good housekeeping to structural controls.

6.11 Mandatory Findings of Significance

The results of this analysis demonstrate that Part 1 if adopted could potentially result in reasonably foreseeable adverse environmental impacts.. There are reasonably foreseeable mitigation measures identified above, and those required by federal, state, and local laws and regulations, that the lead agency responsible for the project level environmental review can and should adopt. These mitigation measures should mitigate any potential adverse impacts at the project level to less than-significant levels.

7. CWC SECTION 13241 AND ANTIDEGRADATION

The State Water Board must analyze the factors described in section 13241 of the Water Code when establishing water quality objectives. Chapter 5.6 requires that the State Water Board adopt SQOs "pursuant to the procedures established by [Division 7] for adopting or amending water quality control plans." (Wat. Code §13393(b).) While the State Water Board is not statutorily required to comply with the substantive requirements for adoption of water quality objectives, when adopting SQOs, the State Water Board has, nevertheless, considered the section 13241 factors. In addition, the State Water Board must ensure that its actions are consistent with Resolution No. 68-16, the state's antidegradation policy.

7.1 Past, Present, and Probable Future Beneficial Uses of Water

The proposed SQOs address:

1. Benthic communities exposed directly to pollutants in sediment.
2. Human health exposed indirectly through fish and shellfish tissue.

As a result these objectives will protect sediment quality for all the beneficial uses that focus on these specific receptors and the associated exposure pathways. The proposed SQOs and interpretive tools will compliment and support the Water Boards' existing water quality control plans and policies, and provide a better means to ensure that beneficial uses are protected.

7.2 Environmental Characteristics of the Hydrographic Unit Under Consideration, Including the Quality of Water Thereto

The indicators proposed to interpret the narrative objective protecting benthic communities were developed based upon the specific physical, environmental biological characteristics of these waters. Unlike many of the numeric criteria in the CTR or used in the development of national sediment quality guidelines, very little data collected from outside the state was used in the development and validation of each indicator. For this reason, all the indicators proposed in Part 1 exhibit better performance in general than indicators developed from national studies, and, as a result, better protect the beneficial uses in waters of the State.

The implementation language proposed in Part 1 provides direction on how the SQOs shall be implemented within the regions, however within Part 1 each Regional Water Board retains the authority and flexibility to apply the SQOs in the appropriate regulatory program. Part 1 does not describe how a particular site should be corrected or remediated. Selection of corrective action can be addressed only after many site-specific factors are considered such as:

- The hydrodynamics and flow regime in the area of concern
- The specific pollutant that is causing the degradation or impairment
- The receptors at risk due to the presence of the pollutants at the levels observed within the area of concern.
- The aerial extent
- Presence of existing sources or legacy releases
- Types of controls in place and feasibility of additional controls.

7.3 Water Quality Conditions that Could Reasonably Be Achieved Through the Coordinated Control of All the Factors Which Affect Water Quality in the Area

As described in Section 1, wastes have been discharged into bays and estuaries either directly as point sources, indirectly as runoff, or accidentally through releases and spills for many years. In addition, many contaminants readily attach to the sediments and are carried down rivers and creeks contributing to the contaminant loading. Once these sediments reach the bays and estuaries, poor flushing and low current speeds allow the sediments and contaminants to settle before reaching the open ocean.

The State and Regional Water Boards are required to ensure that all discharges, regardless of type, comply with all water quality control plans and policies. If the SQOs are adopted into a permit as receiving water limits, the discharge must meet the limits or, if the limits are not being met due to the discharge of toxic pollutants, determine the causative pollutant. If the discharger is contributing to the accumulation of the pollutant causing the degradation, the permittee would be required under existing authority to control the pollutant to the extent practical through BMPs or additional treatment. The same approach would occur if multiple discharges are contributing to the accumulation of the pollutant. For additional control measures see Controls under economic considerations

7.4 Economic Considerations.

The Water Boards must consider economic factors in establishing water quality objectives. Generally, this analysis entails consideration of whether the objectives and alternatives are currently being attained, the methods available to achieve compliance, and the costs of those methods. In addition, the Water Boards must consider economic factors under Public Resources Code §21159 when adopting rules that establish performance standards or treatment requirements.

For the proposed SQOs, the available compliance methods and costs depend on the types of sources that may be affected by the SQOs, which could include a variety of point and nonpoint sources. In order to assess the economic impacts of the proposed objectives and Part 1, DWQ staff consulted with Scientific Applications International Corporation (SAIC). More details of the economic considerations given here may be found in the report "*Economic Considerations of Sediment Quality Plan for Enclosed Bays in California*" (SAIC 2007).

Incremental Impact of Part 1

The incremental economic impacts of Part 1 include the cost of activities above and beyond those that would be necessary in the absence of Part 1 under baseline conditions, as well as the cost savings associated with actions that will no longer need to occur. Baseline conditions include current objectives and policies regulating activities and pollutant discharges that affect sediment quality (e.g., narrative Basin Plan objectives, CTR criteria, and other policies), existing monitoring programs, ongoing cleanup and remediation activities, and planned or anticipated cleanup and remediation actions that have not yet been completed [e.g., TMDL development and implementation schedules].

Under Part 1, Regional Water Boards would list sediment as exceeding the SQOs if multiple lines of evidence (with sufficient data) indicate impairment. This requirement for additional evidence of impairment could potentially reduce the number of water bodies that would be incorrectly listed as impaired for toxic substances. Potential costs or cost savings

associated with implementing the SQOs depend on the relative stringency of the objectives. Table 7.1 indicates the different incremental impacts that could occur under Part 1.

Table 7.1. Incremental Impacts Associated with Part 1

Assessment Under Existing Objective	Assessment Under Proposed SQOs	
	No Sediment Impairment	Sediment Impairment
No Sediment Impairment	• No change in sediment quality. • Potential incremental assessment costs.	• Sediment quality improvement. • Potential incremental assessment and control costs.
Sediment Impairment	• Sediment quality remains the same as now, which may be lower than under implementation of baseline narrative objective. • Potential incremental assessment costs, but will avoid unnecessary control costs.	• Change in sediment quality if better information leads to a change in control strategies. • Potential incremental assessment costs; potential incremental costs or cost-savings depending on differences between control strategies.

Under Part 1, compliance with the proposed aquatic life objective for estuaries would be based on comparing coupled biological effects and chemistry data to reference site conditions. Due to a lack of existing coupled data and known reference sites, an analysis of potential incremental impacts is not possible at this time. The State Water Board will adopt a final direct effects objective for estuaries under Phase II. Thus, it is likely that any control actions identified for compliance with the interim objective would not be implemented until it could be shown that those actions would also be required for compliance with final objective.

Compliance with the proposed human health objective under Part 1 would be based on a human health risk assessment that utilizes OEHHA policies for fish consumption as well as other fish tissue threshold values. In the absence of Part 1, waters will continue to be listed as impaired based on exceedances of fish tissue advisory levels or criteria. Because these same levels and criteria will be used under Part 1 to determine compliance with the objective there would be no incremental impacts associated with the interim human health SQO.

For the proposed aquatic life objective, the Southern California Coastal Water Research Program (SCCWRP) used the assessment matrices in Part 1 to determine compliance at sites for which available sediment monitoring data includes all three of the required sample types (toxicity, chemical exposure, and benthos community). To estimate incremental impacts of Part 1, these results can be compared to existing assessments [i.e., 303(d) listings] for the pollutants of concern in sediment, fish tissue, or the water column. This data is insufficient to determine compliance for all bays. However, for those for which data is available, the results indicate both potential incremental impairments and reduced listings, depending on the water body.

Monitoring and Assessment

The comparison of available assessment data and existing impairments indicates that there is insufficient data to assess compliance with the proposed SQOs for a number of bays, as well as estuaries. In addition, for those waters with sediments that exceed the proposed SQOs, Part 1 indicates that further investigation into stressor identification is necessary (SWRCB, 2006). Thus, the incremental impacts of Part 1 include monitoring and stressor identification costs. Although data for some parameters may not be needed at each sampling site or for each bay, potential per sample costs may range from $3,940 to $5,810 as shown in Table 7-2.

Table 7.2. Potential Sampling Costs under the Plan

Parameter	Cost per sample
Metals suite	$175 – $225
Total Mercury	$65 – $135
PAH suite	$400
Chlorinated pesticides	$200 – $575[a]
PCB congeners (not coplanar)	$200 – $575[a]
Sediment toxicity (acute lethal)	$800
Sediment toxicity (sublethal)	$800 – $1,400
Benthic survey	$800 – $1,200[b]
Sediment collection on boat	$500[c]
Total cost per sample	$3,940 – $5,810

Source: Chemistry cost estimates obtained from price lists used for southern California and San Francisco Bay regional monitoring programs; sediment toxicity and benthic survey costs obtained from southern California regional monitoring program and development of the Plan; sediment collection estimate from SCCWRP (2007).

a. High estimate represents low detection limit analyses.

b. High estimate represents difficult to sort samples, such as 0.5 mm mesh screen samples in San Francisco Bay.

c. Includes the cost of the boat, crew, and any activities associated with preparing the samples for transport to the analysis laboratory (e.g., compositing and subsampling and screening of benthic samples to remove excess sediment).

The number of stations needed to assess bay sediment quality will vary based on site-specific factors. Based on between 5 and 30 samples per bay, depending on area, statewide monitoring costs to assess those bays for which existing data are insufficient (a total of 131 samples representing 20,000 acres) may range from $516,000 to $762,000. . These estimated costs by water body are presented in Table 7.3. Costs associated with confirmatory monitoring for segments with only possibly impacted sites (no clearly or likely impacted sites) would be $8,000 to $12,000. A more detailed description of the assumptions and basis used to develop these costs are described in the report by SAIC (2007).

There are potentially eight bay segments not currently on the 303(d) list for sediment toxicity related impairments for which MLOE data indicate impairment under the Plan. If stressor identification and possible TMDL development activities are needed for those segments and would not be pursued in the future under existing objectives (for three of these segments, MLOE indicate sediment toxicity, and the Regional Board identified sediment cleanup and remediation necessary under the BPTCP), incremental cost could be approximately $8 million. There are also three segments listed for sediment related impairments under the baseline for which MLOE data indicate no impairment under the Plan. Assuming that no stressor identification or TMDL development would be needed for these segments under Part 1, there could be a potential cost savings of $3 million. Thus, the net incremental cost associated with assessment activities could be approximately $5 million (or lower if such costs would be incurred in the absence of the Plan for any of the 3 sites that exhibit sediment toxicity and for which cleanup and remediation actions are necessary).

For estuaries, the State Water Board is collecting data as part of the Phase II effort to develop appropriate tools and thresholds for implementing the SQO. These data can also be used to assess compliance with the final SQO. Thus, additional monitoring may be necessary for those waters not currently being sampled as part of this effort. However, costs of these monitoring efforts cannot be estimated until the data collection effort is complete.

Table 7.3. Potential Incremental Sediment Quality Monitoring Costs

Water Body	Size (Acres)	Number of Samples	Total Monitoring Costs (Low)[1]	Total Monitoring Costs (High)[2]
Region 1				
Crescent City Harbor	374	5	$19,700	$29,100
Bodega Bay	822	12	$47,300	$67,700
Region 2				
Drakes Estero Bay	12,780	30	$118,200	$174,300
San Francisco Bay, Richardson Bay	2,439	12	$47,300	$67,700
Half Moon Bay	355	5	$19,700	$29,100
Region 3				
Moss Landing Harbor	79	5	$19,700	$29,100
Monterey Harbor	76	5	$19,700	$29,100
Santa Barbara Harbor	266	5	$19,700	$29,100
Region 4				
Ventura Harbor	179	5	$19,700	$29,100
Port Hueneme Harbor	65	5	$19,700	$29,100
King Harbor	105	5	$19,700	$29,100
Los Angeles Harbor Consolidated Slip	36	5	$19,700	$29,100
Los Angeles Harbor – Cabrillo Beach	156	5	$19,700	$29,100
Region 8				
Bolsa Bay	116	5	$19,700	$29,100
Region 9				
Mission Bay	2,032	12	$47,300	$67,700
San Diego Bay, Shoreline, at Marriott Marina	32	5	$19,700	$29,100
San Diego Bay, Shoreline, Chula Vista Marina	49	5	$19,700	$29,100
Total	19,961	131	$516,200	$761,700

Detail may not add to total due to rounding.
1. Equals the number of samples times the low estimate of cost per sample ($3,940).
2. Equals the number of samples times the high estimate of cost per sample ($5,810).

Controls

For waters that Regional Water Boards identify as being impaired under the proposed Part 1, remediation actions and/or source controls will be needed to bring them into compliance. Many bays and estuaries are already listed for sediment impairments and, therefore, would require controls under baseline conditions. When the baseline controls are identical to the ones that would be implemented under Part 1, there is no incremental cost or cost savings associated with Part 1. When the baseline controls differ, there is potential for either incremental costs or cost-savings associated with the Plan.

Because strategies to meet current narrative objectives at many impaired sites are still in the planning stages and the overall effects of implementation strategies are unknown, estimates of incremental costs would be highly speculative. For incremental sediment remediation and/or cleanup activities to be required under Part 1, monitoring data would have to indicate biological impacts under the proposed SQOs in areas that would not be designated for clean up under

existing objectives. However, it is likely that most sites with sediment conditions that would require cleanup and remediation under Part 1 would also exceed current objectives. To the extent that results differ, it is possible that the additional assessment activities under Part 1 could lead to cleanup strategies that are more cost effective compared to baseline activities. In addition, based on the implementation plans for existing TMDLs, Regional Water Boards are likely to pursue source controls for ongoing sources and only require remediation activities for historical pollutants with no known, ongoing sources.

For an increased source control cost associated with additional pollution controls under the proposed Part 1, the concentration of toxic pollutants in discharges would have to meet levels that are more stringent than what is needed to achieve compliance with existing objectives (e.g., since they could have to control based on the narrative sediment objectives or the CTR). Incremental costs for controls may also result from the identification of additional chemical stressors that are not included in the CTR or Basin Plans. Since many practices that may be employed under existing TMDLs are applicable for controlling the mobilization of pollutants in general, this situation is also difficult to estimate. For example, the TMDL for pesticides and PCBs in the Calleguas Creek watershed indicates that the BMPs needed to achieve the nutrient and toxicity TMDLs for the watershed would likely reduce pesticides and PCBs to necessary levels as well (LARWQCB, 2005).

Thus, without being able to identify the particular pollutants causing biological effects, and the development of discharge concentrations needed to achieve the proposed objectives, the needed cleanups and/or controls to achieve those concentrations are difficult to estimate. Review of existing impairments and TMDL actions for the various bays suggests that incremental impacts may be unlikely. If there are incremental impacts as a result of the Part 1, controls are likely to focus on storm water sources, marinas, and wetlands. However, some level of control for these sources would occur under the implementation plans for existing TMDLs.

For any situation in which these sources are specifically required to control toxic pollutants to levels that are lower than what would be necessary in the absence of Part 1, potential means of compliance for storm water sources include increased or additional nonstructural BMPs (e.g., institutional, education, or pollution prevention practices designed to limit generation of runoff or reduce the pollutants load of runoff); and structural controls (e.g., engineered and constructed systems designed to provide water quantity or quality control). For marinas and boating activities, potential means of compliance may include use of less toxic paint on boats; performing all boat maintenance activities above the waterline or in a lined channel to prevent debris from entering the water; removing boats from the water and clean in a specified location equipped to trap debris and collect wastewater; prohibiting hull scraping or any process that removes paint from the boat hull from being conducted in the water; and developing a collection system for toxic materials at harbors. Wetlands controls may include aeration, channelization, revegetation, sediment removal, levees, or a combination of these practices.

For estuaries, Regional Water Boards need additional data to identify the sources that may need an incremental level of control.

7.5 The Need for Developing Housing Within the Region

The adoption of Part 1 is not expected to increase the need for housing in the areas surrounding enclosed bays and estuaries of California. Part 1 applies to the protection of subtidal sediments in surface waters.

7.6 THE NEED TO DEVELOP AND USE RECYCLED WATER

The adoption of Part 1 is not expected to increase the need to develop and use recycled water.

7.7 ANTIDEGRADATION

In 1986, the State Water Board adopted Resolution No. 68-16, entitled "Statement of Policy with Respect to Maintaining High Quality of Waters in California." The policy expresses the State Water Board's intent that the quality of existing high quality waters be maintained to the maximum extent possible. Lowering of water quality is allowed only if the lowering is consistent with the maximum benefit to the people of the state, will not unreasonably affect present and anticipated beneficial uses of waters, and will not result in water quality less than that prescribed in applicable policies. Resolution No. 68-16 has been interpreted to incorporate the provisions of the federal antidegradation policy as well, where the federal policy applies.

The federal policy, in 40 C.F.R. §131.12, establishes three tiers of water quality protection and, like Resolution No. 68-16, allows a lowering of water quality for high quality waters only if certain conditions are met. The state and federal antidegradation policies must be considered for a variety of actions, including water quality standards actions.

The State Water Board does not anticipate any lowering of water quality as a result of the adoption of Part I. For the first time, the state will have scientifically-defensible sediment quality objectives for bays and estuaries. These objectives can be consistently applied across the state to assess sediment quality, regulate waste discharges that can impact sediment quality and provide the basis for appropriate remediation activities where sediments are impaired. Adoption of the SQOs, rather than lowering water quality, should result in water quality improvements.

Currently, Regional Water Boards implement a variety of narrative objectives to address sediment quality. The objectives, in general, do not explicitly address sediment quality. The proposed SQOs, on the other hand, are specific to sediments, were developed with data from California waters, have undergone rigorous scientific review, and are intended to protect sediment quality. The proposed SQOs are likely to be more protective, vis-à-vis sediment quality, than current standards.

8. GLOSSARY

ACUTE TOXICITY: Short-term lethal response of an organism to a pollutant.

BEST MANAGEMENT PRACTICES (BMPs): Methods, measures, or practices designed and selected to reduce or eliminate the discharge of pollutants to surface waters from point and nonpoint source discharges including storm water.

BMPs include structural and non-structural controls, and operation and maintenance procedures, which can be applied before, during, and/or after pollution producing activities.

BENTHIC: Living on or in bottom of the ocean, bays, and estuaries, or in the streambed.

BINOMDIST: An Excel® function that can be used to calculate the cumulative binomial distribution.

BINOMIAL DISTRIBUTION: Mathematical distribution that describes the probabilities associated with the possible number of times particular outcomes will occur in series of observations (i.e., samples). Each observation may have only one of two possible results (e.g., standard exceeded or standard not exceeded).

BIOACCUMULATION: A process in which an organism's body burden of a contaminant exceeds that in its surrounding environment as a result of chemical uptake through all routes of chemical exposure; dietary and dermal absorption and transport across the respiratory surface.

BIOACCUMULATION FACTOR (BAF): The ratio of contaminant concentration in biota to contaminant concentration in some other matrix. In this report, unless specified otherwise, the term "bioaccumulation factor" refers to wet weight concentration in fish or invertebrate tissue divided by dry weight concentration in sediment.

BIOAVAILABILITY: The fraction of a chemical pollutant or contaminant that can be absorbed by an organism through gills or other membranes, potentially causing an adverse physiological or toxicological response. Bioavailability is dependent on the chemical form of the pollutant in the media, the physical and biogeochemical processes within the media, the route and duration of exposure, and the organism's age, metabolism, size and sensitivity.

BIOTA-SEDIMENT ACCUMULATION FACTOR (BSAF): This is the bioaccumulation factor for tissue vs. sediment, normalized for lipid and organic carbon. BSAF = (tissue contaminant concentration in wet wt. * sediment % organic carbon) / (sediment contaminant concentration in dry wt. * tissue % lipid).

BIOASSESSMENT: Assessment of biological community information along with measures of the physical/habitat quality to determine, in the case of water quality, the integrity of a water body of interest.

BTAG: Biological Technical Assistance Group, a multi-agency group of State and federal ecological and human health risk assessors supported by U.S. EPA responsible for providing technical assistance for Site remediation and mitigation.

CHEMICALS OF CONCERN (COCS): Pollutants that occur in environmental media at levels that pose a risk to ecological receptors or human health.

CONTAMINATION: An impairment of the quality of the waters of the State by waste to a degree that creates a hazard to the public health through poisoning or through the spread of disease. "Contamination" includes any equivalent effect resulting from the disposal of waste whether or not waters of the State are affected (CWC section 13050(k)).

CHRONIC TOXICITY: Sublethal response of an organism to repeated, long-term exposure to a chemical substance. Typical observed endpoints include growth expressed as length and weight.

CALIFORNIA TOXICS RULE (CTR): Numerical water quality criteria established by U.S. EPA for priority toxic pollutants for California's inland surface waters, enclosed bays, and estuaries.

DEGRADATION OF SEDIMENT QUALITY: Sediment toxicity and changes in benthic community attributes as a result of exposure to toxic pollutants in bedded surficial sediments. Unacceptable risk to human health and wildlife as a result of bioaccumulation from pollutants in bedded surficial sediments that are transported up the aquatic food chain.

DEMERSAL: Organisms that prefer to spend the majority of their time on or near the bottom of a water body.

DIEL: Measurements pertain to measurements taken over a 24-hour period of time.

DREDGED MATERIAL: Any material excavated or dredged from the navigable waters of the United States, including material otherwise referred to as "spoil."

EFFECTS RANGE-MEDIAN (ERM)/EFFECTS RANGE-LOW (ERL): Sediment quality guidelines based on a biological effects empirical approach. These values represent chemical concentration ranges that are rarely (i.e., below the ERL), sometimes (i.e., between ERL and ERM), and usually (i.e., above the ERM) associated with toxicity for marine and estuarine sediments. Ranges are defined by the tenth percentile and fiftieth percentile of the distribution of contaminant concentrations associated with adverse biological effects.

EFFECT SIZE: Maximum magnitude of exceedance frequency that is tolerated.

ENCLOSED BAYS: Indentations along the coast that enclose an area of oceanic water within distinct headlands or harbor works. Enclosed bays include all bays where the narrowest distance between headlands or outermost harbor works is less than 75 percent of the greatest dimension of the enclosed portion of the bay. This definition includes, but is not limited to: Humboldt Bay, Bodega Harbor, Tomales Bay, Drakes Estero, San Francisco Bay, Morro Bay, Los Angeles Harbor, Upper and Lower Newport Bay, Mission Bay, and San Diego Bay.

ENDPOINT: A measured response of a receptor to a stressor. An endpoint can be measured in a toxicity test or in a field survey.

EQUILIBRIUM PARTITIONING APPROACH: Approach used to relate the dry-weight sediment concentration of a particular chemical that causes an adverse biological effect to the equivalent free chemical concentration in pore water and to that concentration sorbed to sediment organic carbon or bound to sulfide. Based on the theory that the partitioning of a nonionic organic chemical between organic carbon and pore water and the partitioning of a divalent metal between the solid and solution phases are at equilibrium.

EQUILIBRIUM PARTITIONING SEDIMENT GUIDELINES: Sediment quality guidelines derived using the EqP approach. When used in conjunction with appropriately protective water only exposure concentration, a resulting guideline represents the sediment contaminant concentration that protects benthic organisms from the effects of that contaminant.

ESTUARIES AND COASTAL LAGOONS: Waters at the mouths of streams that serve as mixing zones for fresh and ocean waters during a major portion of the year. Mouths of streams that are temporarily separated from the ocean by sandbars shall be considered as estuaries. Estuarine waters will generally be considered to extend from a bay or the open ocean to the

upstream limit of tidal action but may be considered to extend seaward if significant mixing of fresh and salt water occurs in the open coastal waters. The waters described by this definition include, but are not limited to, the Sacramento-San Joaquin Delta as defined by Section 12220 of the California Water Code, Suisun Bay, Carquinez Strait downstream to Carquinez Bridge, and appropriate areas of the Smith, Klamath, Mad, Eel, Noyo, and Russian Rivers.

EUHALINE: Waters ranging in salinity from 25–32 practical salinity units (psu).

INDIRECT EFFECTS: Adverse effects to humans and wildlife as a result of consuming prey items exposed to polluted sediments.

INFAUNA: Organisms that live within sediment or substrate.

INLAND SURFACE WATERS: All surface waters of the State that do not include the ocean, enclosed bays, or estuaries.

LOAD ALLOCATION (LA): The portion of a receiving water's total maximum daily load that is allocated to one of its nonpoint sources of pollution or to natural background sources.

MIXING ZONE: Limited zone within a receiving water that is allocated for mixing with a wastewater discharge where water quality criteria can be exceeded without causing adverse effects to the overall water body.

MAXIMUM CONTAMINANT LEVEL (MCL): The maximum permissible level of a contaminant in water delivered to any user of a public water system.

MAXIMUM TISSUE RESIDUE LEVEL (MTRL): Tissue values developed from human health water quality objectives in the 1997 California Ocean Plan and from the California Toxic Rule as established in the Policy for Implementation of Toxics Standards for Inland Surface Waters, Enclosed Bays, and Estuaries of California. MTRLs are used as alert levels or guidelines indicating water bodies with potential human health concerns and are an assessment tool and not compliance or enforcement criteria. The MTRLs are calculated by multiplying human health water quality objectives by the bioconcentration factor for each substance.

MESOHALINE: Waters ranging in salinity from 5 to 18 psu.

NATIONAL ACADEMY OF SCIENCE TISSUE GUIDELINES: Guidelines established for the protection of predators. Values are suggested for residues in whole fish (wet weight) for DDT (including DDD and DDE), aldrin, dieldrin, endrin, heptachlor (including heptachlor epoxide), chlordane, lindane, benzene hexachloride, toxaphene, and endosulfan either singularly or in combination.

NATIONAL TOXICS RULE: Numerical water quality criteria established by U.S. EPA for priority toxic pollutants for 12 states and two Territories who failed to comply with the section 303(c)(2)(B) of the Clean Water Act.

NONPOINT SOURCE POLLUTION: Sources are diffused and do not have a single point of origin or are not introduced into a receiving stream from a specific outlet. The commonly used categories for nonpoint sources are agriculture, forestry, mining, construction, land disposal, and salt intrusion.

NULL HYPOTHESIS: Statement used in statistical testing that has been put forward either because it is believed to be true or because it is to be used as a basis for argument, but has not been proved.

OBJECTIONABLE BOTTOM DEPOSITS: An accumulation of materials or substances on or near the bottom of a water body which creates conditions that adversely impact aquatic life, human health, beneficial uses, or aesthetics. These conditions include, but are not limited to, the accumulation of pollutants in the sediments and other conditions that result in harm to benthic organisms, production of food chain organisms, or fish egg development. The presence of such deposits shall be determined by Regional Water Board(s) on a case-by-case basis.

OCEAN WATERS: Territorial marine waters of the State as defined by California law to the extent these waters are outside of enclosed bays, estuaries, and coastal lagoons. Discharges to ocean waters are regulated in accordance with the State Water Board's California Ocean Plan.

PELAGIC: Organisms living in the water column.

PERSISTENT POLLUTANTS: Substances for which degradation or decomposition in the environment is nonexistent or very slow.

POLLUTANT: Defined in section 502(6) of the CLEAN WATER ACT as "dredged spoil, solid waste, incinerator residue, filter backwash, sewage, garbage, sewage sludge, munitions, chemical wastes, biological materials, radioactive materials, heat, wrecked or discarded equipment, rock, sand, cellar dirt and industrial, municipal, and agricultural waste discharged into water."

POLLUTANT MINIMIZATION: Waste minimization and pollution prevention actions that include, but are not limited to, product substitution, waste stream recycling, alternative waste management methods, and education of the public and businesses.

POLLUTION: Defined in section 502(19) of the CLEAN WATER ACT as the "the man-made or man-induced alteration of the chemical, physical, biological, and radiological integrity of water." *Pollution* is also defined in CWC section 13050(1) as an alternation of the quality of the waters of the State by waste to a degree that unreasonably affects either the waters for beneficial uses or the facilities that serve these beneficial uses.

POLLUTION PREVENTION: Any action that causes a net reduction in the use or generation of a hazardous substance or other pollutant that is discharged into water and includes, but is not limited to, input change, operational improvement, production process change, and product reformulation (as defined in Water Code Section 13263.3). Pollution prevention does not include actions that merely shift a pollutant in wastewater from one environmental medium to another environmental medium, unless clear environmental benefits of such an approach are identified to the satisfaction of the State Water Board or the Regional Water Boards.

POLYHALINE: Waters ranging in salinity from 18–25 psu.

PROBABLE EFFECT CONCENTRATION (PEC): Empirically derived freshwater sediment quality guidelines (SQG) that rely on the correlation between the chemical concentration in field collected sediments and observed biological effects. PECs are based on geometric means of various SQG approaches (with matching chemical and toxicity field data) to predict toxicity for freshwater sediment on a regional and national basis.

PROBABLE EFFECTS LEVEL (PELS)/THRESHOLD EFFECTS LEVELS (TEL): Empirically derived sediment quality guidelines based on a biological effects empirical approach similar to ERMs/ERLs. A generalized approach used to develop effects-based guidelines for the state of Florida and others. The lower of the two guidelines for each chemical (i.e., the TEL) is assumed to represent the concentration below which toxic effects rarely occur. In the range of

concentrations between the two guidelines, effects occasionally occur. Toxic effects usually or frequently occurs at concentrations above the upper guideline value (i.e., the PEL). Ranges are defined by specific percentiles of both the distribution of contaminant concentrations associated with adverse biological effects and the "no effects" distribution.

RANK CORRELATION: The association between paired values of two variables that have been replaced by their ranks within their respective samples (e.g., chemical measurements and response in a toxicity test).

REFERENCE CONDITION: The characteristics of water body segments least impaired by human activities. As such, reference conditions can be used to describe attainable biological or habitat conditions for water body segments with common watershed/catchment characteristics within defined geographical regions.

SIMULTANEOUSLY EXTRACTED METALS (SEM): Metal concentrations that are extracted during the same analysis in which the acid-volatile sulfide (AVS) content of the sediment is determined.

STATISTICAL SIGNIFICANCE: When it can be demonstrated that the probability of obtaining a difference by chance only is relatively low.

TOXIC POLLUTANT: As used in this staff report toxic pollutants refers to priority pollutants AS USED

TOXICITY IDENTIFICATION EVALUATION (TIE): Techniques used to identify the unexplained cause(s) of toxic events. TIE involves selectively removing classes of chemicals through a series of sample manipulations, effectively reducing complex mixtures of chemicals in natural waters to simple components for analysis. Following each manipulation the toxicity of the sample is assessed to see whether the toxicant class removed was responsible for the toxicity.

TOXICITY REDUCTION EVALUATION (TRE): Study conducted in a step-wise process designed to identify the causative agents of effluent or ambient toxicity, isolate the sources of toxicity, evaluate the effectiveness of toxicity control options, and then confirm the reduction in toxicity. The first steps of the TRE consist of the collection of data relevant to the toxicity, including additional toxicity testing, and an evaluation of facility operations and maintenance practices, and best management practices. A Toxicity Identification Evaluation (TIE) may be required as part of the TRE, if appropriate. (A TIE is a set of procedures to identify the specific chemical(s) responsible for toxicity. These procedures are performed in three phases [characterization, identification, and confirmation] using aquatic organism toxicity tests.)

WASTE: As used in this document, waste includes a discharger's total discharge, of whatever origin, i.e., gross, not net, discharge.

WATER QUALITY-LIMITED SEGMENT: Any segment of a water body where it is known that water quality does not meet applicable water quality standards, and/or is not expected to meet applicable water quality standards, even after application of technology-based effluent limitations required by CLEAN WATER ACT sections 301(d) or 306.

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APPENDICES

CALIFORNIA

Water Boards

STATE WATER RESOURCES CONTROL BOARD

Office of Public Affairs: (916) 341-5254
Office of Legislative Affairs: (916) 341-52
Office of the Ombudsman (916) 341-5254

P.O. Box 100, Sacramento, CA 95812-010
www.waterboards.ca.gov

Water Quality information: (916) 341-5455
Water Rights information: (916) 341-5300
Financial Assistance information: (916) 341-5700

CALIFORNIA REGIONAL WATER QUALITY CONTROL BOARDS

NORTH COAST REGION (1)
www.waterboards.ca.gov/northcoast
5550 Skylane Blvd., Suite A
Santa Rosa, CA 95403
E-mail: info1@waterboards.ca.gov
(707) 576-2220 TEL • (707) 523-0135 FAX

SAN FRANCISCO BAY REGION (2)
www.waterboards.ca.gov/sanfranciscobay
1515 Clay Street, Suite 1400
Oakland, CA 94612
E-mail: info2@waterboards.ca.gov
(510) 622-2300 TEL • (510) 622-2460 FAX

CENTRAL COAST REGION (3)
www.waterboards.ca.gov/centralcoast
895 Aerovista Place, Suite 101
San Luis Obispo, CA 93401
E-mail: info3@waterboards.ca.gov
(805) 549-3147 TEL • (805) 543-0397 FAX

LOS ANGELES REGION (4)
www.waterboards.ca.gov/losangeles
320 W. 4th Street, Suite 200
Los Angeles, CA 90013
E-mail: info4@waterboards.ca.gov
(213) 576-6600 TEL • (213) 576-6640 FAX

CENTRAL VALLEY REGION (5)
www.waterboards.ca.gov/centralvalley
11020 Sun Center Drive, Suite 200
Rancho Cordova, CA 95670
E-mail: info5@waterboards.ca.gov
(916) 464-3291 TEL • (916) 464-4645 FAX

Fresno branch office
1685 E Street, Suite 200
Fresno, CA 93706
(559) 445-5116 TEL • (559) 445-5910 FAX

Redding branch office
415 Knollcrest Drive
Redding, CA 96002
(530) 224-4845 TEL • (530) 224-4857 FAX

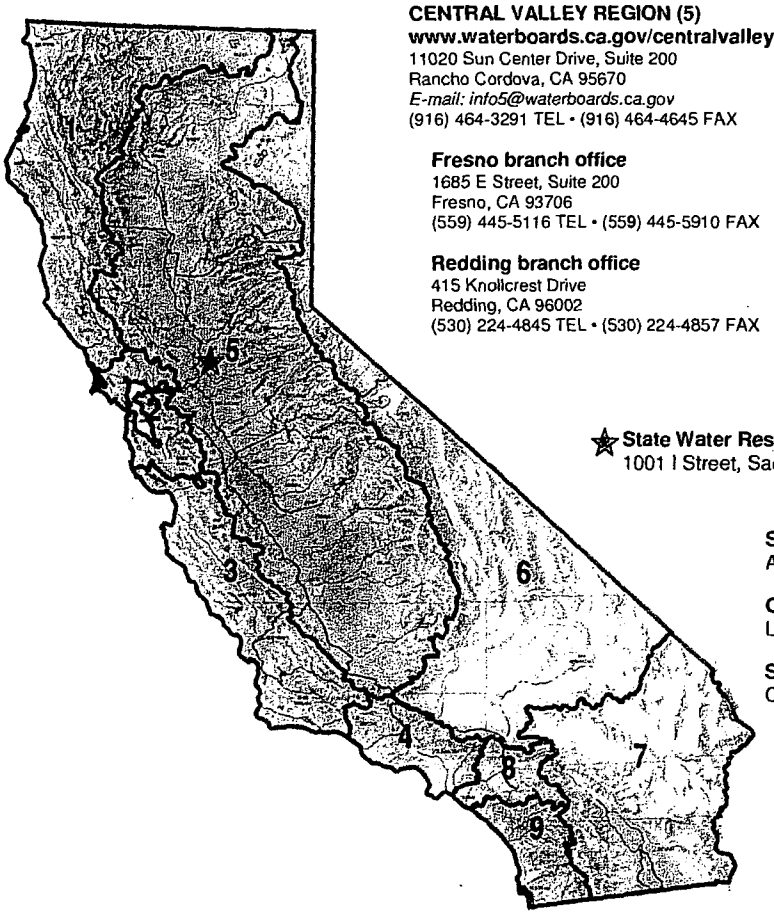
LAHONTAN REGION (6)
www.waterboards.ca.gov/lahontan
2501 Lake Tahoe Blvd.
South Lake Tahoe, CA 96150
E-mail: info6@waterboards.ca.gov
(530) 542-5400 TEL • (530) 544-2271 FAX

Victorville branch office
14440 Civic Drive, Suite 200
Victorville, CA 92392
(760) 241-6583 TEL • (760) 241-7308 FAX

COLORADO RIVER BASIN REGION (7)
www.waterboards.ca.gov/coloradoriver
73-720 Fred Waring Dr., Suite 100
Palm Desert, CA 92260
E-mail: info7@waterboards.ca.gov
(760) 346-7491 TEL • (760) 341-6820 FAX

SANTA ANA REGION (8)
www.waterboards.ca.gov/santaana
3737 Main Street, Suite 500
Riverside, CA 92501-3339
E-mail: info8@waterboards.ca.gov
(951) 782-4130 TEL • (951) 781-6288 FAX

SAN DIEGO REGION (9)
www.waterboards.ca.gov/sandiego
9174 Sky Park Court, Suite 100
San Diego, CA 92123
E-mail: info9@waterboards.ca.gov
(858) 467-2952 TEL • (858) 571-6972 FAX



★ **State Water Resources Control Board** (Headquarters)
1001 I Street, Sacramento, CA 95814

State of California
Arnold Schwarzenegger, Governor

California Environmental Protection Agency
Linda S. Adams, Secretary

State Water Resources Control Board
Charles R. Hoppin, Chair

Beegan Depo. Ex.

Sediment Chemistry: for simplicity, assume "high exposure"

Sediment Toxicity [see SQOs, Table 4, p. 5]

Eohaustorius Survival	92%
Mytilus Normal	80%
Result:	**Nontoxic**

Benthos [see SQOs, Table 5, p. 7]

BRI	46.2
IBI	1
RBI	0.25
RIVPACs	0.87
Result:	**Low Disturbance**

Biological Effects Matrix (Sediment Toxicity + Benthos): [see SQOs, Table 9, p. 9]

Nontoxic + Low Disturbance = "Unaffected"

Chemically Mediated Effect Matrix (Sediment Toxicity + Chemistry): [see SQOs, Table 10, p. 10]

High Exposure + ~~Low Disturbance~~ Non toxic = "Moderate Potential"

Station Assessment Matrix: [see SQOs, Table 11, p. 10]

Unaffected + Moderate Potential = "Likely Unimpacted"

EXHIBIT 205
WIT: Beegan
DATE: 10-12-10
CAROL NYGARD DROBNY

SEDIMENT QUALITY IN CALIFORNIA BAYS AND ESTUARIES

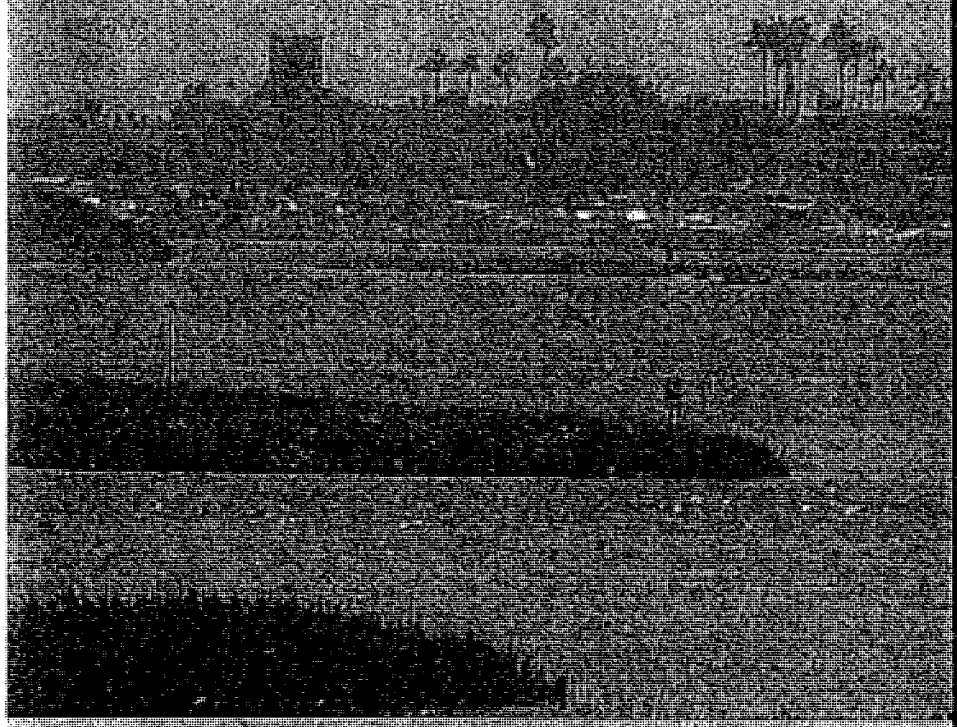
Arthur M. Barnett

Steven M. Bay

Kerry J. Ritter

Shelly L. Moore

Stephen B. Weisberg



SCCWRP

Established 1969

SWAMP

Surface Water Ambient Monitoring Program

Southern California Coastal Water Research Project

Technical Report 522 - January 2008

Sediment Quality in California Bays and Estuaries

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January 2008

Technical Report 522

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EXECUTIVE SUMMARY

Sediment quality in California bays and estuaries was evaluated using a multiple lines of evidence (MLOE) assessment framework. This framework has been proposed for adoption as part of the sediment quality objectives (SQOs) portion of California's water quality control plan for bays and estuaries. Chemistry, toxicity, and benthic community data, each representing an independent line of evidence (LOE) regarding sediment quality, from six surveys conducted over eight years were analyzed. The analysis consisted of three parts: 1) determining sediment condition at each sampling station (site) using the assessment framework; 2) establishing a single integrated data set with known spatial attributes from the combined data of each survey; and 3) analyzing the integrated data set using spatial statistics to determine the percentage of area corresponding to each sediment condition category.

The assessment framework was used to classify 381 sites into one of the following six condition categories:

- **Unimpacted.** Confident that contamination is not causing significantly adverse impacts to aquatic life in the sediment.
- **Likely Unimpacted.** Contamination is not expected to cause adverse impacts to aquatic life in the sediment, but some disagreement among LOEs reduces certainty that the site is unimpacted.
- **Possibly Impacted.** Contamination at the site may be causing adverse impacts to aquatic life in the sediment, but the level of impact is either small or is uncertain because of disagreement among LOEs.
- **Likely Impacted.** Evidence of contaminant-related impacts to aquatic life in the sediment is persuasive, in spite of some disagreement among LOEs.
- **Clearly Impacted.** Sediment contamination at the site is causing clear and severe adverse impacts to aquatic life in the sediment.
- **Inconclusive.** Disagreement among LOEs suggests that either data are suspect or additional information is needed for classification.

Two levels of assessment were conducted. The first level used a combined data set from all surveys and evaluated statewide conditions. At the second level, spatial assessments were conducted independently for three regions within the state in order to investigate patterns related to differences in size of embayments, land use, and hydrological characteristics. The regions were: North, consisting of multiple small coastal embayments north of Point Conception to the Oregon border; South, which included multiple small coastal embayments south of Point Conception to the US-Mexico border; and the San Francisco Bay and its contiguous marine embayment areas (SFB).

Approximately 83% of the 1295 km^2 of California marine embayments included in the analysis were classified as having some degree of impact related to sediment contamination. Most of the area was classified as Possibly Impacted and less than 1% of the area was classified as Clearly

Impacted (Figure 4; Table 3). The statewide analysis results were dominated by the conditions present in SFB, which represented nearly 80% of the embayment area.

Large variations in sediment condition were present among the three geographic regions. The North region had the best sediment conditions, with 58% of the area classified as Unimpacted and no sites classified as Clearly Impacted (Figure 5; Table 4). Somewhat poorer sediment quality was observed in the South, with 43% of the area classified as Unimpacted and 2% classified as Clearly Impacted. A different distribution of sediment condition categories was present in San Francisco Bay; no sites were classified as Unimpacted and the proportion of area classified as Possibly Impacted (77%) was more than three times greater than that measured in the other regions.

The regional differences in sediment quality identified through the assessment framework were evaluated by analysis of the underlying LOEs (Chemistry, Toxicity, and Benthic Community) to examine various levels of response within each site's sediment. Sediment chemistry was least impacted in the North and most impacted in the South. The incidence of biological effects (toxicity or benthic community disturbance) was greatest in SFB and appeared to account for the comparatively high percent area classified as Possibly Impacted or Likely Impacted.

The large percentage of Possibly Impacted area within SFB suggests that sediment contaminants are more widespread and less concentrated in this region, possibly due to contaminant dilution and redistribution as a result of greater rainfall, high runoff inputs from urban and agricultural sources, and tidal mixing. There is also evidence that the relationship between sediment contamination and toxicity in SFB differs from that observed in other regions. As the causes of toxicity in California embayments have not been identified, the reason for this apparent difference in toxicity response cannot be determined. Unmeasured contaminants, such as current use pesticides, may be influencing these relationships. It is also possible that contaminant bioavailability differs between regions or that different contaminants are causing toxicity in each area.

The results of this study's integrated analysis using the assessment framework are consistent with previous studies of sediment quality in California bays and estuaries. However, use of the framework and combined survey data produced a more comprehensive and robust assessment of statewide sediment quality than has been achieved previously. Moreover, this study's assessment of sediment conditions on both statewide and regional scales can be used as a benchmark for future studies.

TABLE OF CONTENTS

INTRODUCTION

Sediment quality has an important influence on the overall condition of a water body. Sediments act as a reservoir for contaminants that can be transferred to the water column through physical disturbance, diffusion, and biological activities. Also, sediments are a primary source of contaminant exposure for sediment-dwelling organisms and animals that feed on the bottom, such as crabs and flatfishes. This exposure can produce adverse impacts on benthic communities and can also lead to indirect effects on wildlife and human health due to the accumulation of contaminants from the food chain.

Historically, sediment quality assessment has been an important feature of many California monitoring programs. It was a major focus in the Bay Protection and Toxic Cleanup Program (BPTCP; Anderson *et al.* 1997), the California Environmental Mapping and Assessment Program (EMAP; USEPA 2005), the San Francisco Regional Monitoring Program (SFEI 2005), and the Southern California Bight Regional Monitoring Program (SCCWRP 2003, 2007). Although numerous sediment quality surveys have recently been conducted, like the ones cited above, these studies focused on areas and used methods for data interpretation different from those used in this study, thereby preventing the integration of such data for analysis and inclusion in a statewide assessment of sediment conditions in California's embayments (bays and estuaries). Comprehensive sediment quality information is needed for California's 305(b) and 303(d) programs to establish priorities for water quality programs at the State and Regional Boards. The present study, under the auspices of the State Water Ambient Monitoring Program (SWAMP), is intended to provide this assessment.

Sediment is a complex matrix of components and forms. Consequently, evaluating contaminant impacts on beneficial uses based on a single line of evidence is problematic. For example, bulk measures of chemical concentration fail to differentiate between the fraction of a contaminant that is tightly bound to sediment and that which is biologically available. Multiple mechanisms of contaminant exposure, including uptake of chemicals from interstitial water, sediment ingestion, and bioaccumulation through the food web further complicate interpretation of sediment chemistry data.

For these reasons, sediment quality assessment often involves simultaneously evaluating multiple lines of evidence (MLOE) that measure both contaminant exposure and effects on organisms: an approach commonly known as the sediment quality triad (Long and Chapman 1985). Lines of evidence (LOEs), such as sediment chemistry, toxicity, and benthic community condition are often used. Virtually all of the ambient sediment quality monitoring programs in this country rely on more than one line of evidence (USEPA 1998, Crane *et al.* 2000, MacDonald and Ingersoll 2002, USEPA 2004). Such programs include the two largest nationwide estuarine monitoring programs: the United States Environmental Protection Agency (USEPA) EMAP and the National Oceanic and Atmospheric Administration (NOAA) National Status and Trends Program, as well numerous regional monitoring programs. The California State Water Board BPTCP also relied primarily on MLOE to assess sediment quality in bays and estuaries throughout the state (Anderson *et al.* 1997, Fairey *et al.* 1998, Phillips *et al.* 1998, Anderson *et al.* 2001, Hunt *et al.* 2001).

Staff at the State Water Resources Control Board (SWRCB) has proposed draft sediment quality objectives (SQOs) that use an assessment framework based on an MLOE approach to evaluate sediment quality in embayments (SWRCB 2007). If adopted, these SQOs will become the regulatory standard against which ambient sediment quality is measured, influence management and regulatory decisions, and serve as the basis for evaluating water body impairment (e.g., 303(d) listings) with regard to sediment quality.

Previous statewide assessments of sediment condition in California have been limited in terms of data integration and interpretation. Results from a 1999 EMAP survey were used to describe the statewide extent of sediment contamination, toxicity, and benthic community characteristics, but these separate LOEs were not integrated to assess overall sediment condition (USEPA 2005). Recent 305(b) reports of California sediment quality have included data from multiple studies, but again the condition assessment was limited by a lack of integration of LOEs and the use of variable data interpretation approaches among studies (SWRCB 2006, USEPA 2004).

This report represents the first application of the proposed assessment framework on a statewide basis to evaluate sediment quality in California's marine and estuarine embayments. The focus of this analysis is on the direct effects of contamination on aquatic life due to sediment contact or ingestion, rather than effects on humans or wildlife due to indirect exposure through the consumption of fish and shellfish. For this assessment, data from recent EMAP, SWAMP, and southern California Bight surveys were combined and evaluated using a common set of assessment indicators within an assessment framework.

Two levels of assessment were conducted (Figure 1). The first level evaluated statewide conditions. The purpose of this level was to determine the percentages of the State's embayments with various levels of impact from sediment contamination. At the second level, spatial assessments were conducted independently for three regions within the state in order to investigate patterns related to differences in size of embayments, land use, and hydrological characteristics. The northern region (North) included multiple small coastal embayments north of Point Conception to the Oregon border. The North embayments were characterized by low population density, where agricultural use is important and freshwater inputs are relatively high. The southern region (South) included multiple small coastal embayments south of Point Conception to the US-Mexico border. These southern embayments were often surrounded by high population density, extensive commercial/industrial use, and low freshwater inputs. The third assessment region was the San Francisco Bay and its contiguous marine embayment areas (SFB). The hydrology of the SFB is different from the North and South in that runoff into SFB is nearly continuous, tidal mixing is strong, and agricultural and industrial uses are relatively high.

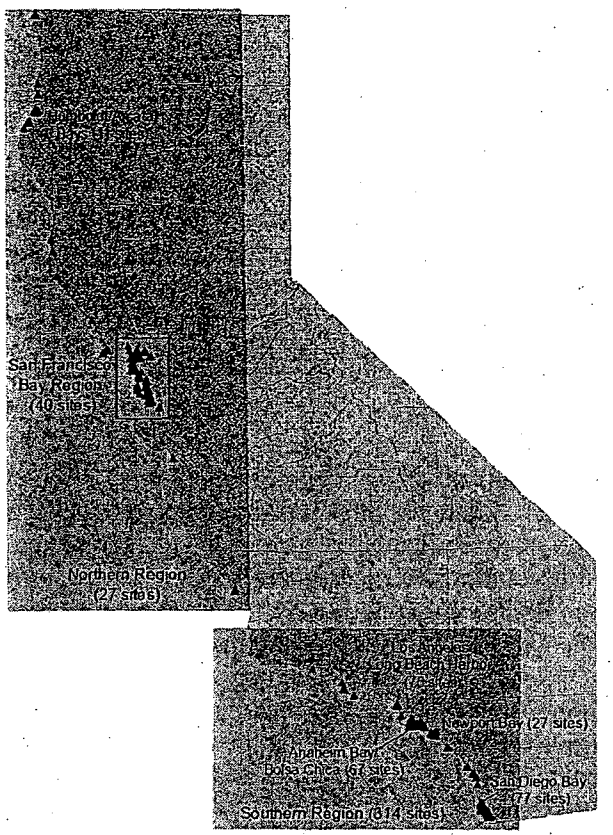


Figure 1. Distribution of sampling sites for the statewide assessment. The shaded boxes indicate the three regional assessment areas.

METHODS

The proposed assessment framework for California's SQOs (SWRCB 2007) was applied to data from multiple random stratified surveys conducted throughout the state to evaluate the sediment quality of marine embayments. The analysis consisted of three parts: 1) determining sediment condition at each sampling station (site) using MLOE response classifications or attributes; 2) establishing a single statewide data set with known spatial attributes based on the integrated data for all stations within each survey; and 3) analyzing the integrated data set using spatial statistics to determine the percentage of area corresponding to each sediment condition category. Spatial analyses were conducted for the state as a whole and regionally for northern California (North), the San Francisco Bay (SFB), and southern California (South).

Data

The statewide and regional estimates of sediment condition were based on data collected from six stratified random surveys with probability-based designs, conducted over eight years (Table 1). Probability-based designs were selected because the area represented by each site was known, allowing sampling results to be expressed as the percent area affected. In addition, each survey met the following criteria: (i) samples were collected within 10 years of the current analysis, (ii) site locations were subtidal areas within bays and estuaries, (iii) corresponding data for sediment chemistry, toxicity, and benthic macrofauna were available, and (iv) sampling and analysis methods were comparable to those specified in the proposed SQO assessment framework. Several recent regional surveys did not meet these criteria and were not used in this study for reasons that included lack of a probability-based design (e.g., San Francisco Bay Regional Monitoring Program) and lack of sediment toxicity data or comparable toxicity/benthic macrofauna measurement methods (e.g., selected Western EMAP (WEMAP) surveys).

Sample collection for each survey was conducted in the summer and used comparable methods; however, the surveys encompassed different years and geographic regions. Two WEMAP surveys examined embayments along the entire California coast in 1999 and 2005, while one survey was limited to San Francisco Bay (2000). Three surveys included only southern California embayments: two examined multiple embayments along the entire southern coast (1998, 2003), while the third was an intensive study of only Huntington Harbor and Anaheim Bay (2001). These surveys followed the USEPA's Generalized Random Tessellation Stratified (GRTS) design with the intent of balancing samples spatially while allowing for intensification in certain areas of interest (http://www.epa.gov/nheerl/arm/designpages/design&analysis.htm).

Table 1. Probability-based surveys and number of sites per region for each survey.

Survey	Year	Area (km^2)	Number of Sites		
			North	SFB	South
Southern California Bight Regional Monitoring Program	1998	122	0	0	113
	2003	135	0	0	102
WEMAP	1999	139	19	0	24
	2000	1020	0	40	0
	2005	139	8	0	15
Huntington Harbor and Anaheim Bay Survey	2001	1.4	0	0	60
Total			27	40	314

Determination of Sediment Condition

Three lines of evidence: sediment chemistry, toxicity, and benthic macrofaunal community condition (benthos) were evaluated at each site. The indices and thresholds described in the draft SQO policy for California were then used to assign sediment assessments to one of four response-level categories relevant to respective LOEs. Details of the specific measures used for each LOE are provided in SWRCB (2007). The LOE responses were then integrated using the assessment framework to determine the level of impact, if any, with respect to sediment contamination for each site. A summary of each LOE and the integration process is provided below.

Lines of Evidence

Chemistry. A combination of two sediment chemistry indices was used to determine the magnitude of chemical exposure at each site: the California Logistic Regression Model (CA LRM) and the Chemical Score Indicator (CSI). The CA LRM was developed using a logistic regression modeling approach that estimates the probability of acute toxicity in sediments based on the chemical concentration (Field *et al.* 2002, USEPA 2005) calibrated using California data (Bay *et al.* 2007a). The CSI was developed using California data and is based on the association of chemical concentration with benthic community disturbance (Ritter *et al.* 2007). Calculation of the CSI differed from Ritter *et al.* (2007) by not including data for cadmium in order to maintain consistency with the SWRCB draft policy. Index-specific thresholds were then applied and resulting CA LRM and CSI exposure categories were averaged to determine an overall response for the chemistry LOE. The response-level categories used to define chemical exposure assessments were:

- **Minimal Exposure** - Sediment-associated contamination may be present, but exposure is unlikely to result in effects.
- **Low Exposure** - Small increase in contaminant exposure that may be associated with increased effects, but magnitude or frequency of occurrence of biological impacts is low.
- **Moderate Exposure** - Clear evidence of sediment contaminant exposure at concentrations that are likely to result in biological effects.
- **High Exposure** - Contaminant exposure is highly likely to result in substantial biological effects.

Toxicity. The 10-day amphipod survival test using *Eohaustorius estuarius* was used to determine the magnitude of sediment toxicity at each site (USEPA 1994). Thresholds based on percentage survival and statistical significance were applied to assign test results to one of the following response-level categories used to define toxicity assessments (Bay *et al.* 2007b):

- **Nontoxic** - Response not substantially different from that in uncontaminated control sediments.
- **Low Toxicity** - A low magnitude response that differs from control survival, but is within the variability typical for that test and thus may not be a reproducible effect.
- **Moderate Toxicity** - High confidence that a statistically significant toxic effect is present.
- **High Toxicity** - High confidence that a toxic effect is present and the magnitude of response includes the strongest effects observed for the test.

Benthos. A combination of up to four benthic community condition indices was used to determine the magnitude of disturbance to the benthos at each site. The indices include approaches based on community metrics and abundance of individual species. The benthic indices used include:

Benthic Response Index (BRI), which was originally developed for the southern California mainland shelf and extended into California's bays and estuaries (Smith *et al.* 2001, 2003). The BRI is the abundance-weighted average pollution tolerance score of organisms occurring in a sample.

Index of Benthic Biotic Integrity (IBI), which was developed for freshwater streams and adapted for California's bays and estuaries (Thompson and Lowe 2004). The IBI identifies community measures that have values outside a reference range.

Relative Benthic Index (RBI), which was originally developed for California's Bay Protection and Toxic Cleanup Program (Hunt *et al.* 2001). The RBI is the weighted sum of: (i) several community metrics, (ii) the abundances of three positive indicator species, and (iii) the presence of two negative indicator species.

River Invertebrate Prediction and Classification System (RIVPACS), which was originally developed for British freshwater streams (Wright *et al.* 1993, Van Sickle *et al.* 2006) and

adapted for California's bays and estuaries. The RIVPACS index calculates the number of reference taxa present in the test sample and compares it to the number expected to be present in a reference sample from the same habitat.

Not all indices were used in each region, due to the lack of calibration for some habitats. All four indices were used for most stations in the South (except that RIVPACS data were not available for the Huntington Harbor and Anaheim Bay survey) and portions of SFB. The RBI and IBI were used to evaluate the remainder of the SFB sites. The RBI was used to evaluate all of the North sites.

Thresholds specific to regional assemblages were applied to the results in order to classify each index result according to the level of disturbance. The resulting disturbance categories were then combined to provide an overall benthos LOE category. The four response-level categories used to define benthic condition assessments were:

- **Reference** - A community composition equivalent to a "least affected" or "unaffected" site.
- **Low Disturbance** - A community that shows some indication of stress, but could be within measurement error of unaffected condition.
- **Moderate Disturbance** - Confident that the community shows evidence of physical, chemical, natural, or anthropogenic stress.
- **High Disturbance** - Changes in the benthos are substantial enough to limit community function.

Integration of LOE Response Levels

The response-level categories within each of the three LOEs resulted in 64 possible combinations of outcomes (Appendix A). Each combination was associated with one of six final site condition classes. This was accomplished in a two-step process (Figure 2). Individual LOEs were first combined to form two intermediate classifications describing (i) the severity of biological effects and (ii) the potential for chemically mediated biological effects. These intermediate classifications were then integrated to determine the final MLOE assessment of site condition.

The benthos and toxicity LOEs were integrated to determine the severity of biological effects category for the site: Unaffected, Low Effect, Moderate Effect, or High Effect. The benthos LOE was given greater weight for determining this classification, as the benthic community is the resource to be protected. Moreover, the severity of effects classification reflects disturbance to the benthic community due to a variety of causes and is not intended to differentiate between effects that are due to chemical contaminants, physical disturbance of the habitat, or organic enrichment.

The potential for chemically mediated effects was determined using the toxicity and chemistry LOE categories data. These data were integrated to assign samples into one of four classifications describing the potential that the observed biological effects were caused by chemical contaminants: Minimal Potential, Low Potential, Moderate Potential, or High

Potential. The chemistry LOE was given slightly greater weight in determining this classification. The toxicity LOE was included in this classification because sediment toxicity is a measure of the bioavailability of sediment contaminants and also indicates whether unmeasured chemicals are present at levels of potential biological concern. The relationship of each LOE category to the intermediate response classifications is shown in Appendix A.

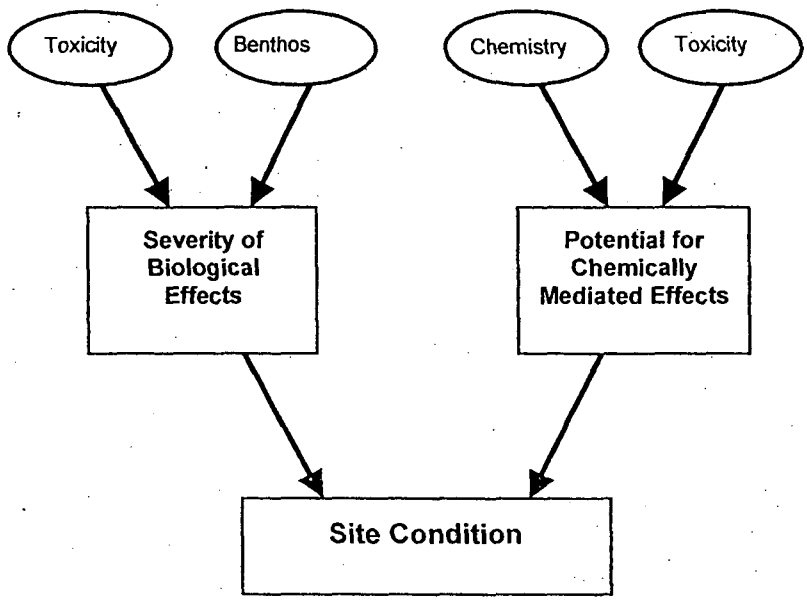


Figure 2. MLOE integration for site assessment.

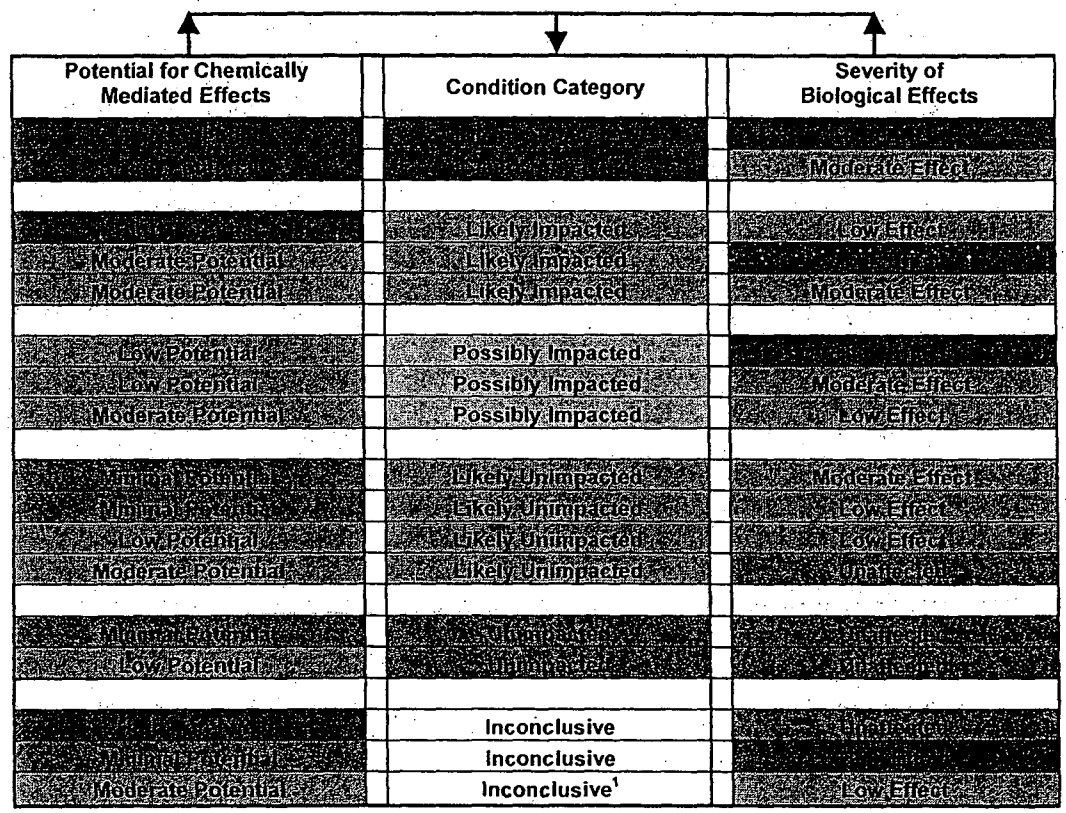
The final MLOE site condition categories (Table 2; Appendix A) were based on the severity level of biological effects and the potential for chemically mediated effects. Six assessment classes were developed to describe the contaminant impact in terms of level of certainty and magnitude:

- **Unimpacted.** Confident that chemical contamination is not causing significantly adverse impacts to aquatic life in the sediment.
- **Likely Unimpacted.** Chemical contamination is not expected to cause adverse impacts to aquatic life in the sediment, but some disagreement among the LOEs reduces certainty that the site is Unimpacted.
- **Possibly Impacted.** Chemical contamination at the site may be causing adverse impacts to aquatic life in the sediment, but the level of impacts is uncertain because of disagreement between LOEs.
- **Likely Impacted.** Evidence of contaminant-related impacts to aquatic life in the site sediment is persuasive, in spite of possible disagreement among LOEs.
- **Clearly Impacted.** Sediment chemical contamination at the site is causing clear and significantly adverse impacts to aquatic life in the sediment.

- **Inconclusive.** Disagreement among the LOEs suggests that either data are suspect or additional information is needed for classification.

Two central concepts were incorporated in the determination of the impact categories: (i) both exposure and effect must be present in order to classify a site as impacted and (ii) a greater magnitude of effect or exposure results in a more severe impact assessment category.

Table 2. Relationship of intermediate LOE classifications to final MLOE site condition categories. Arrows indicate the sequence of classification. The site condition assessment resulting from each possible LOE combination is shown in Appendix A.



[1] Inconclusive category results when High toxicity, Minimal chemical exposure, and a Reference benthic community are present.

Determination of Percent Area of California Embayments for Each Site Condition Category

The random stratified sampling design for each of the six surveys considered in this study consisted of three main components used for tesselation: a sampling frame, stratification, and polygons. The sampling frame represented the boundaries of the survey. Some surveys included strata (e.g., ports, marinas), while no stratification was used in others. Different polygons (subregions within a stratum) were used to constrain sample point distribution or control sample density. Consequently, the area weights (proportional to the number of sites within a stratum) of individual sample points varied greatly between surveys.

In order to conduct a statewide assessment that was spatially representative, the survey designs were combined to produce a common sampling frame and level of stratification. Three strata (regions) were established: North, SFB, and South. Within each region, the polygons representing survey-specific sampling frames and different sample densities were compared for each survey and a single set of polygons were drawn that included all of the combined area sampled. New area weights were calculated for the sites within each region by dividing the area of each final polygon by the number of sites within the area. Figure 3 provides an example of combining survey data points and sampling polygons for Newport Bay in the South region.

Two years of survey data were combined for the North: WEMAP 1999 and WEMAP 2005. No stratification was used for the data from the 2005 survey. As a result, we used the polygons from the 1999 survey and recalculated area weights based on the number of samples from both surveys falling within these polygons. For San Francisco Bay, there was only one survey, WEMAP 2000, therefore no adjustment of polygons or area weights were needed.

In the South, combining the data and calculating new area weights were more complex, as five surveys were integrated. Polygons that overlapped among surveys were split into subpolygons that reflected disjoint areas. New area weights were calculated by dividing the area of each subpolygon by the number of samples that fell into that subpolygon, regardless of survey.

Estimates of the percent area representing various sediment condition classifications were calculated using the new area weights. The proportion of each region representing each MLOE condition category was calculated as the sum of the area weights of the samples that fell into that category divided by the sum of the area weights for all samples within the region. This proportion was then converted to a percentage. The area (km^2) represented by this percentage was calculated by multiplying the proportion by the total area of the region. Confidence intervals for these estimates were computed using the local variance estimator option in the EPA analysis tools (Stevens and Olsen 2003, http://www.epa.gov/nheerl/arm/designpages/design&analysis.htm).

Statewide estimates of condition were calculated in the same manner used for the regional estimates. The area weights of sites having the same MLOE sediment condition classification in all regions were summed and then divided by the sum of the area weights in all regions. This calculation was repeated for each MLOE site condition category. The statewide area corresponding to each an MLOE condition category was calculated by multiplying the proportion by the total area of the three regions.

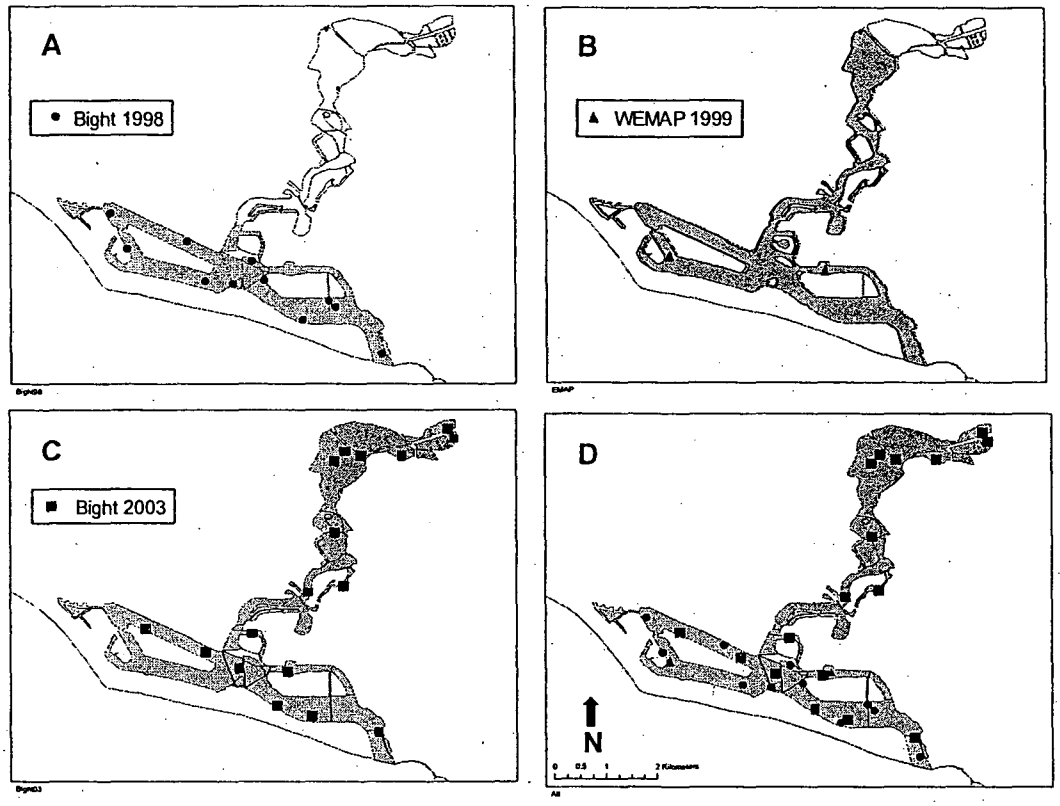


Figure 3. Combination of data from multiple surveys (illustrated for Newport Bay). Sampling polygon and data points for Southern California Bight Regional Monitoring Program 1998 survey restricted to lower bay (A). Sampling polygon and data points for WEMAP 1999 survey (B). Southern California Bight Regional Monitoring Program 2003 sample points associated with two separate polygons representing different sampling intensities (C). Combined data from all surveys associated with two polygons representing entire area sampled (D).

RESULTS

Statewide Assessment of Sediment Quality

Approximately 83% of the 1295 km^2 of California marine embayments included in the analysis was classified as having some degree of impact related to sediment contamination. Most of the area was classified as Possibly Impacted, the most uncertain classification, and less than 1% of the area was classified as Clearly Impacted, the most severe impact category (Figure 4; Table 3). The statewide analysis results were dominated by the conditions present in SFB, which represented nearly 80% of the embayment area.

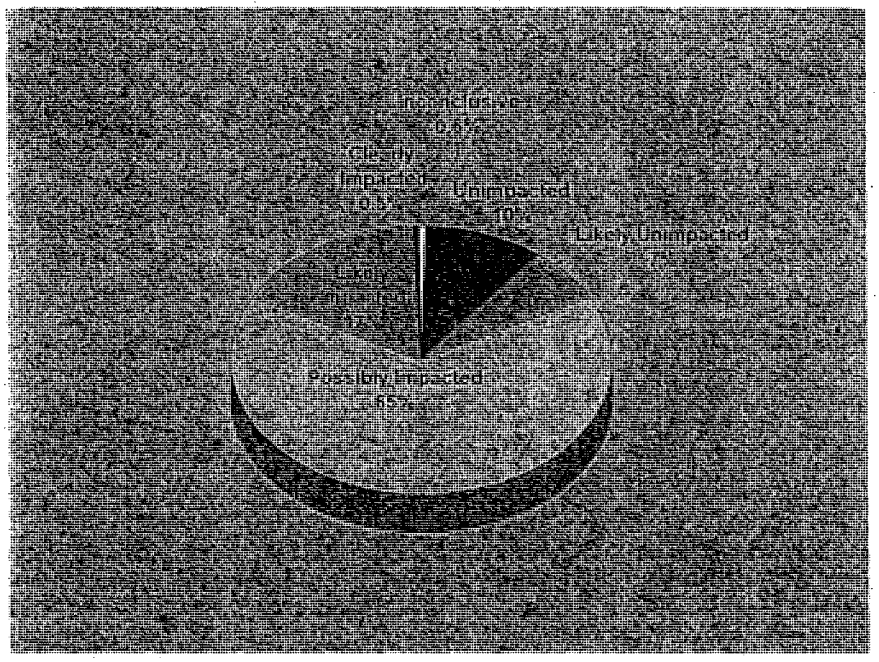


Figure 4. Percent area of California embayments for each sediment condition category as classified by the MLOE assessment framework.

Table 3. Statewide embayment sediment quality condition based on MLOE assessment. Further details on confidence limits and areas represented by each condition classification can be found in Appendix B.

Condition Category	Number of Sites	Percent Area	0.95 Confidence Limits
Unimpacted	131	10%	8 – 12%
Likely Unimpacted	57	7%	2 – 12%
Possibly Impacted	111	65%	55 – 76%
Likely Impacted	51	17%	7 – 26%
Clearly Impacted	25	0.5%	0 – 1%
Inconclusive	6	0.6%	0 – 1%
Total	381	100%	

Regional Assessment of Sediment Quality

Large variations in sediment condition were present among the three geographic regions. The North region had the best sediment condition, with 58% of the area classified as Unimpacted and no sites in the Clearly Impacted category (Figure 5; Table 4). Somewhat poorer sediment quality was observed in the South, with 43% of the area classified as Unimpacted and 2% classified as Clearly Impacted. A different distribution of sediment condition categories was present in San Francisco Bay; no stations were classified as Unimpacted and the proportion of area assigned to the Possibly Impacted category (77%) was more than three times greater than that measured in the other regions. The uncertainty in condition estimates varied among regions as a function of sample size. The estimates were most precise for the South, with 95th percentile confidence intervals of about 10%; confidence intervals for SFB and the North were usually two to three times greater (Table 4).

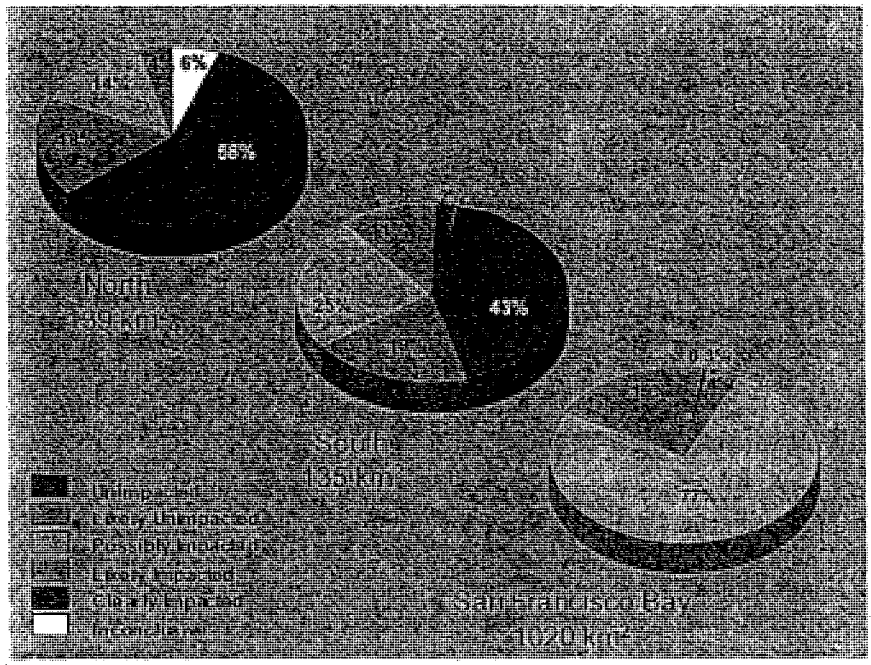


Figure 5. Percent area of sediment quality classification for regional MLOE assessments.

Table 4. Regional embayment sediment quality condition based on MLOE assessment. Further details on confidence limits and areas represented by each condition classification can be found in Appendix B.

Condition Category	Number of Sites	Percent Area	0.95 Confidence Limits
North			
Unimpacted	9	58%	37 – 80%
Likely Unimpacted	9	18%	1 – 34%
Possibly Impacted	4	14%	0 – 29%
Likely Impacted	2	4%	0 – 9%
Clearly Impacted	0	0%	-
Inconclusive	3	6%	0 – 12%
Total	27	100%	
SFB			
Unimpacted	0	0%	-
Likely Unimpacted	2	4%	0 – 10%
Possibly Impacted	28	77%	64 – 89%
Likely Impacted	9	19%	7 – 31%
Clearly Impacted	1	0.3%	0 – 1%
Inconclusive	0	0%	-
Total	40	100%	
South			
Unimpacted	122	43%	36 – 49%
Likely Unimpacted	46	19%	13 – 25%
Possibly Impacted	79	25%	19 – 30%
Likely Impacted	40	11%	7 – 15%
Clearly Impacted	24	2%	1 – 3%
Inconclusive	3	0.3%	0 – 0.6%
Total	314	100%	

Sediment Condition in Individual Embayments

A total of 381 sites were assessed in this study. Eight embayments contained 84% of the data and had sufficient numbers of sites to examine spatial patterns of condition within them (Figure 6; LOE combinations that resulted in the designated impact condition at each site are presented in Appendix C). Patterns of sediment condition could not be described for many of the small embayments because only one or two sites were located within them.

Two major spatial patterns of site condition were evident among the selected embayments. First, there was a greater proportion of Likely Impacted and Clearly Impacted sites in inner harbor and marina areas (e.g., Los Angeles and Huntington Harbors). Second, the more impacted sites tended to be located near the perimeters of the embayments where ports or commercial areas are situated (e.g., San Francisco and San Diego Bays).

Locations having the greatest severity of impacts (Clearly and Likely Impacted) were Huntington Harbor (a marina) and inner Los Angeles Harbor including Dominguez Channel. Sediment conditions were better at the deeper locations in Outer Los Angeles and Long Beach Harbors and outer Anaheim Bay, presumably due to increased distance from sources and better circulation. Similar trends were observed in the deeper waters of mid- and northern San Diego Bay, and Alamitos Bay. Typical of the North, most of the sites in Humboldt Bay were classified as Unimpacted or Likely Unimpacted (Figure 6).

Sites having Likely Impacted and Possibly Impacted sediment quality were most prevalent in Newport Bay and San Francisco Bay (Figure 6). Each of these embayments had over 80% of sites classified as either Likely Impacted or Possibly Impacted.

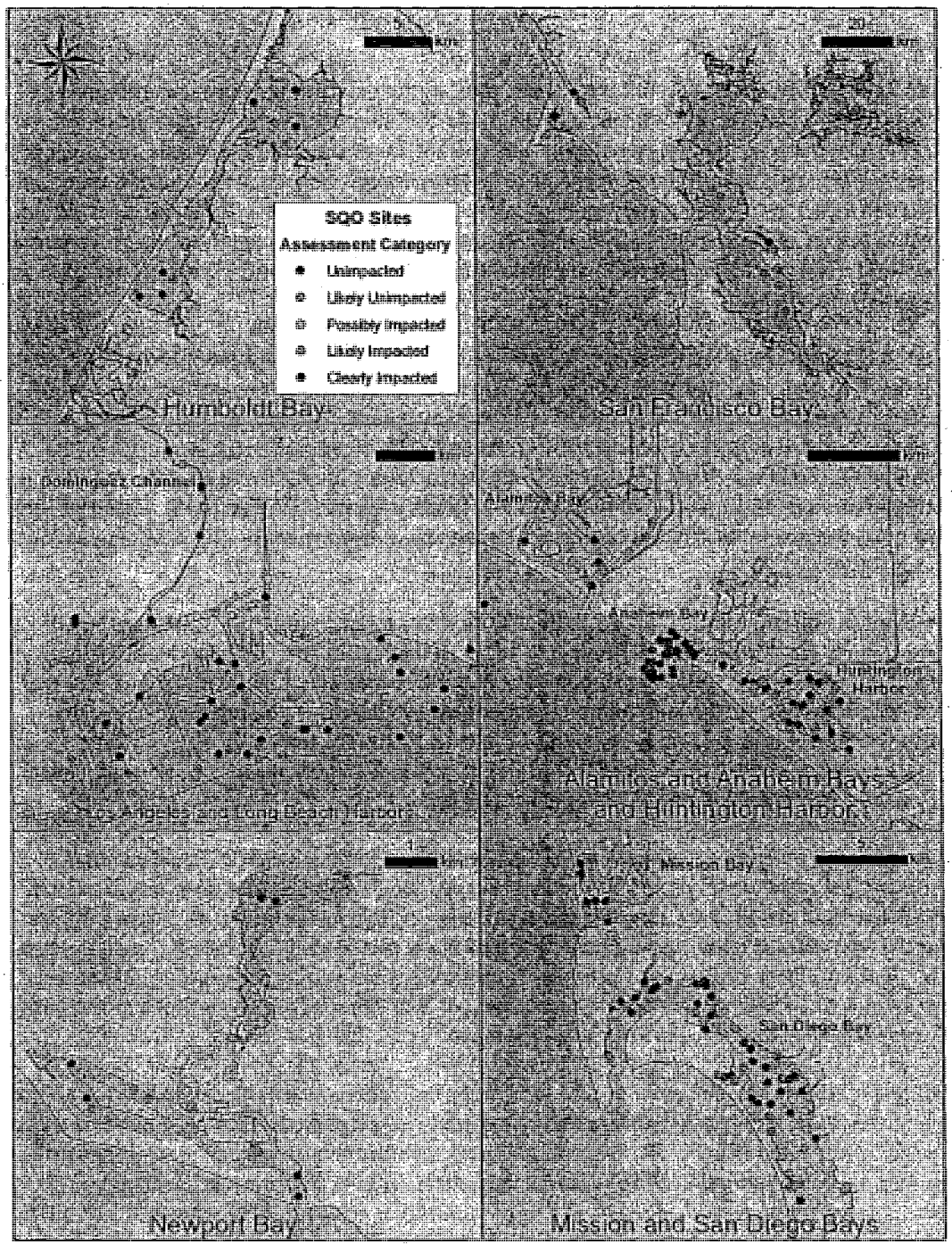


Figure 6. Sediment quality in selected California embayments. Further details for each site in these embayments are shown in Appendix C.

DISCUSSION

Sediment quality was found to be highly variable among California's marine and estuarine embayments. The SFB region had seven and one-half times the area of either the North or the South regions. As a result, the statewide assessment of condition in California embayments was dominated by the condition of SFB. A more representative view of the status of California's bays and estuaries was obtained from the regional analyses (Figure 5; Table 4), which found northern embayments to be the least impacted, and southern embayments to be less impacted than SFB. However, in contrast to the North and South, most of the SFB region was assessed as Possibly Impacted.

This study used an integrated analysis based on a novel MLOE assessment framework, resulting in a more spatially and temporally comprehensive and standardized analysis than previous studies of sediment quality in California bays and estuaries. Nevertheless, the results of this study are consistent with prior analyses. An assessment of coastal condition in 1999 (that included the WEMAP 1999 data used in the present study) found low levels of metal and organic contamination in embayments in the North and South regions (USEPA 2005). The 1999 survey also measured a similar extent of sediment toxicity to *E. estuarius* (19 - 24% of the area) as the present assessment.

The high prevalence in the South of sediment with Possibly, Likely, or Clearly Impacted conditions is consistent with previous studies by BPTCP. The BPTCP surveys also found a high frequency of sediment toxicity to amphipods, benthic community degradation, and elevated contaminant concentrations in multiple Southern embayments, including San Diego Bay, Newport Bay, Huntington Harbor, and Los Angeles Harbor (Fairey *et al.* 1998, Phillips *et al.* 1998, Anderson *et al.* 2001). The BPTCP program had different objectives, however, and focused on identifying the most highly impacted sites.

The widespread toxicity reported for SFB has been documented in BPTCP and regional monitoring studies since the 1980s (Anderson *et al.* 2007). While the spatial extent of toxicity calculated from the SFB data analyzed in this study appears to be somewhat larger than that found in the other studies, certain locations in SFB are consistently toxic to *E. estuarius* and other species. Prior studies have also observed benthic community degradation and reduced populations of localized clam species in portions of San Francisco Bay, with the greatest impacts associated with shallow water locations (Thompson *et al.* 2007).

SFB had a greater percentage of area in the Likely Impacted and Possibly Impacted condition than in the South where a greater portion of the area was classified in the most extreme category of Clearly Impacted (Figure 5). Whereas southern California is an area of greater industrial, commercial, and population concentration, the pattern in SFB suggests that sediment contaminants are more widespread and less concentrated, possibly due to contaminant dilution and redistribution as a result of greater rainfall, runoff, and tidal mixing.

Relationships Among LOEs

Because SFB sediment quality was so different from that in the North or South, further analyses and regional comparisons were conducted to investigate the results. The regional differences in sediment quality identified by the MLOE assessment were evaluated by analysis of the underlying lines of evidence (Chemistry, Toxicity, and Benthic Community). The percentage of area classified as having Moderate or High effects (i.e., affected) for each LOE were calculated for each region (Table 5). Sediment chemistry showed the lowest level of response in the North (1%) and greater impacts in the South and SFB. The North also had a low percentage of area with elevated toxicity (Table 5); however, a moderately high percentage of the area was classified as having affected benthos. This combination of results suggests that the benthos in the North might be affected by physical disturbance or noncontaminant stressors or that our indices are less well calibrated in this region.

The greater proportion of area with Possibly Impacted or Likely Impacted designations in SFB (Figure 5) was reflective of large percentages of this region's total area having either affected benthos or toxicity (Table 5; Appendix C). With lower percentages of areas of the South in the affected categories for benthos and toxicity (relative to SFB) and a moderate percentage affected for chemistry, the MLOE assessment framework seemed consistent in classifying most of the South as Unimpacted or Likely Unimpacted, although to a lesser degree than in the North (Figure 5). Thus, the patterns of individual LOE responses found in each region were consistent with the regional percentage area results.

Table 5. Percent of area affected for each LOE. Area 'Affected' = sum of percent area classified as moderate and high response categories.

	Percent Area Affected Per LOE		
Region	Benthos	Toxicity	Chemistry
North	27	17	1
SFB	34	85	20
South	23	28	40

There appeared to be a different relationship between chemistry and toxicity for the South and SFB. San Francisco Bay had high incidences of affected benthos and toxicity relative to the South, yet the extent of chemical contamination was lower in general (Table 5). The difference in this relationship is evident when the magnitude of toxicity (percent mortality) in a sample is compared to the magnitude of contamination between the regions (Figure 7). San Francisco Bay sediments tend to produce a greater toxic response than southern California sediments at similar levels of contamination (as represented by the CA LRM Pmax value). As the causes of toxicity in the South and San Francisco Bay were not identified in this study, the reason for this apparent difference in toxicity response cannot be determined. Unmeasured contaminants, such as current use pesticides, may be influencing these relationships. Prior studies in San Francisco Bay have

shown a correlation between biological impacts and sediment contamination in general, but a specific chemical cause for the majority of the effects has yet to be identified (Thompson *et al.* 2007). It is also possible that regional differences in contaminant bioavailability or contamination patterns are affecting the relationship between chemistry and toxicity.

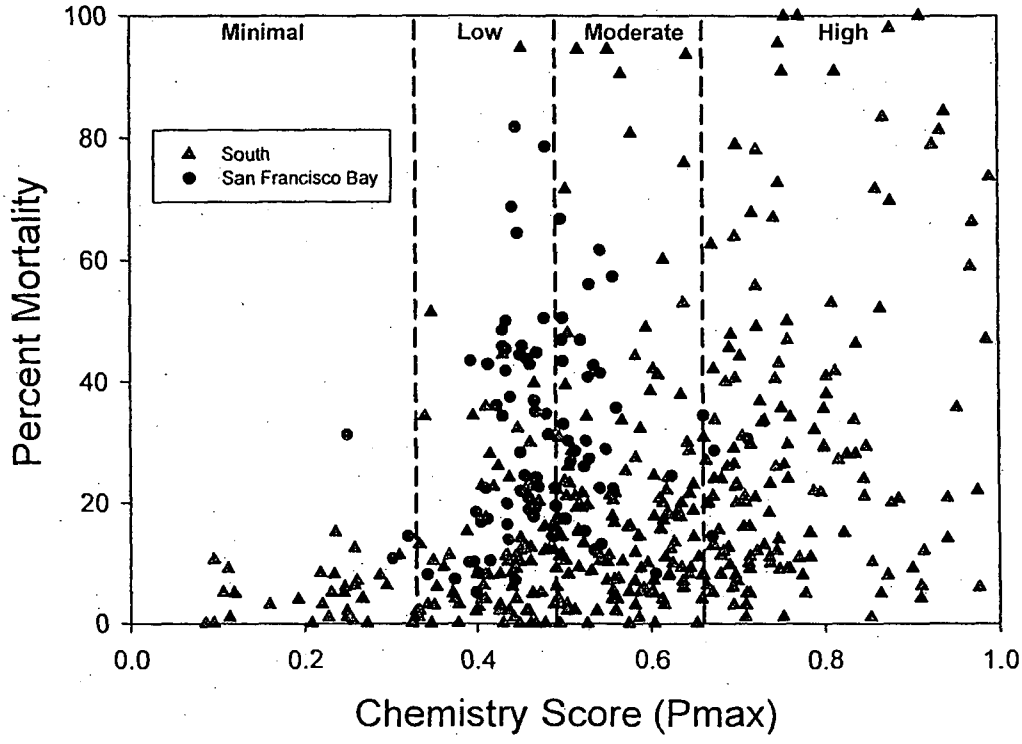


Figure 7. Toxicity:chemistry relationships in the South and SFB regions. The chemistry score is the CA LRM maximum probability of toxicity; dashed lines indicate the probability thresholds for the four response-level categories of chemical exposure.

Sources of Uncertainty

This assessment utilized a new approach and there are several sources of uncertainty in the results. First, the indices used to classify benthic community condition varied among regions due to a lack of habitat-specific calibration data for some of the indices. Four indices were used in the South, whereas only one index (RBI) was available for the North. Four benthic indices were also used in central SFB, but only the RBI and IBI were available for use in interpreting data from San Pablo Bay and the south Bay. All available indices were used wherever possible, as analyses have shown that the use of multiple indices gives a more accurate assessment of benthic community condition (Ranasinghe *et al.* 2007). To test the effect of using various combinations of benthic indices on the classifications, the analyses were repeated using only the RBI to classify benthic community condition in each region. While the percent of area classified as having affected benthos was increased when only the RBI was used, the effect on the overall sediment condition assessment was minor (Table 6).

The high abundance of nonindigenous species in SFB is another source of uncertainty in the benthic community evaluation. The effect of nonindigenous species on the assessments is expected to be small, since these species were included in the calibration of SFB benthic indices and prior analyses of southern California data indicate they do not confound the benthic index results. However, a detailed study to investigate the influence of nonindigenous species on the performance of the SFB benthic indices has not been conducted.

Table 6. Variability among regional area estimates based on benthic indices applied.

Region	Benthic Indices Applied	Benthos (% Moderate or High Disturbance)	MLOE (% Possibly, Likely, or Clearly Impacted)
North	All	27	18
North	RBI only	27	18
SFB	All	34	96
SFB	RBI only	85	100
South	All	23	38
South	RBI only	36	40

Another source of uncertainty is the limited number of sites available to characterize sediment quality in the North and SFB. In the present study, only 40 sites from a single survey were used in the SFB assessment, and only 27 sites were available to represent the North. Consequently, individual sites in the North and SFB had much greater area weights and a greater influence on the results than did individual sites in the South. This resulted in larger confidence intervals for the North and SFB area assessments (Table 4; Appendix B). However, even with these large intervals, statistically significant differences were observed between regions for some sediment condition categories.

A final source of uncertainty is related to the toxicity assessment. The results are based on only a single test of sediment toxicity: the 10-day amphipod survival test. While this is a widely used measure of sediment quality, the use of multiple tests is recommended for sediment quality assessment (Burton, Jr. *et al.* 1996, Greenstein *et al.* In press); the SQO assessment framework is intended to be used with at least two tests. The impact of using a single test in this assessment is unknown, but a greater proportion of the samples might have been identified as toxic if additional tests, especially those that measure sublethal effects, had been used.

Conclusions and Recommendations

The integration of multiple surveys and use of a standardized assessment framework provided a more comprehensive and robust assessment of California embayment sediment quality than has been achieved previously. This assessment yielded results that were consistent with expectations based on earlier studies, thus increasing confidence in the overall accuracy of the sediment condition assessments.

The SQO assessment approach used in this study provides a highly comparable and reproducible measure of sediment condition throughout the State. This approach identified regional differences is sediment condition and potentially different relationships between chemistry and toxicity that can only be detected by a statewide survey. Consequently, this evaluation of sediment condition at both statewide and regional scales can be used as a guide for prioritizing further research and management actions, as well as establishing a benchmark for future assessments.

Future statewide and regional assessments can be improved in several ways. The precision and confidence in the assessment can be improved by sampling more sites in SFB and North using methods that are compatible with the MLOE assessment framework. Future studies should also include multiple toxicity tests and benthic indices in order to provide greater confidence in the measurement of these lines of evidence. The environmental significance of sediments classified as Possibly Impacted is uncertain, as this category may indicate a minor level of contaminant effect, or substantial disagreement among the LOEs. Stressor identification studies, such as toxicity identification evaluations, are needed at Possibly Impacted sites to determine whether sediment quality at these sites is adversely impacted by contaminants.

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APPENDIX A. RELATION OF LOE CATEGORIES TO SQO MLOE ASSESSMENTS

Table A.1. Relationship of LOE response-level categories to intermediate classifications and final MLOE assessment site condition categories. Arrows indicate the sequence of classification.

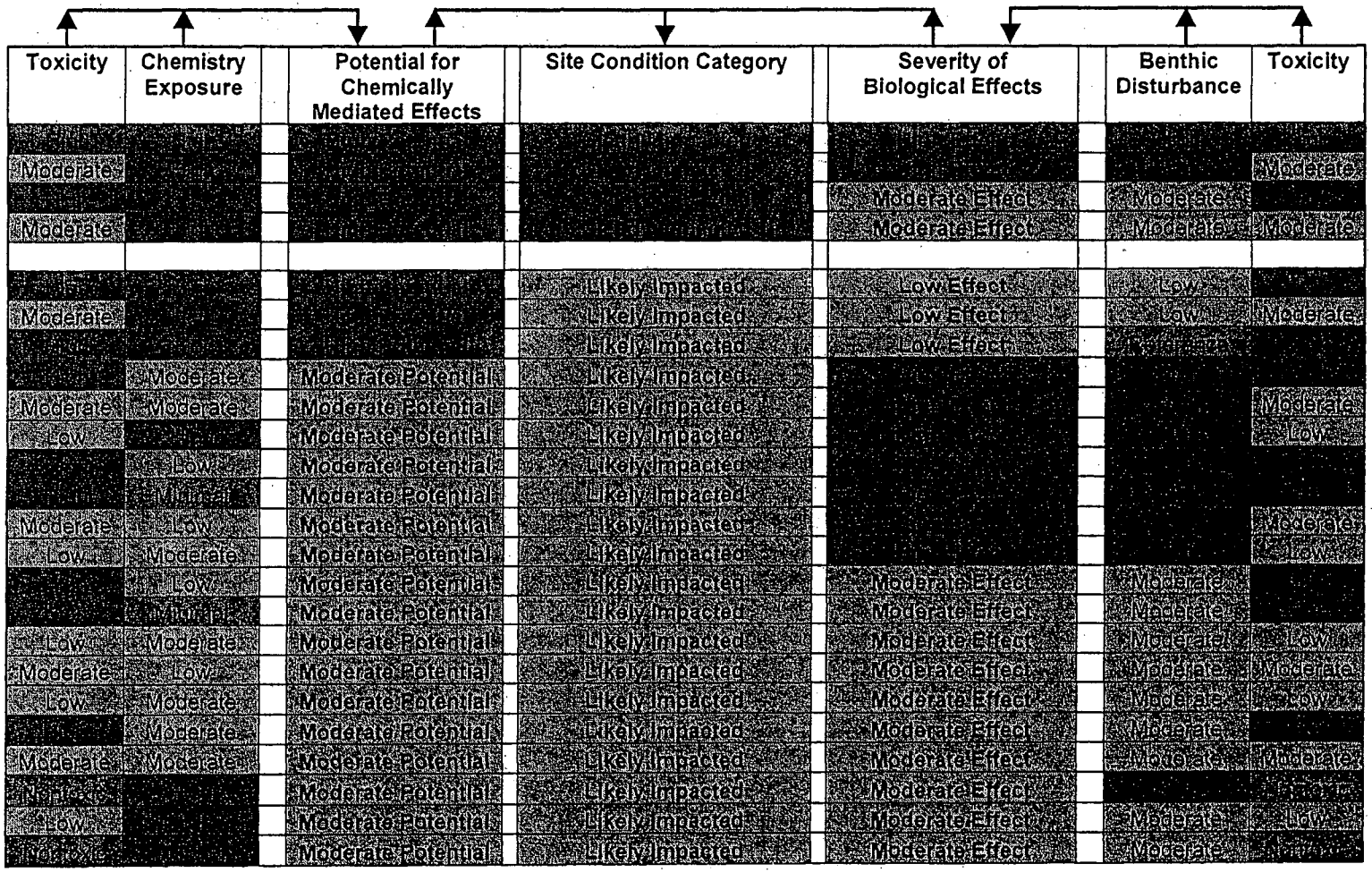
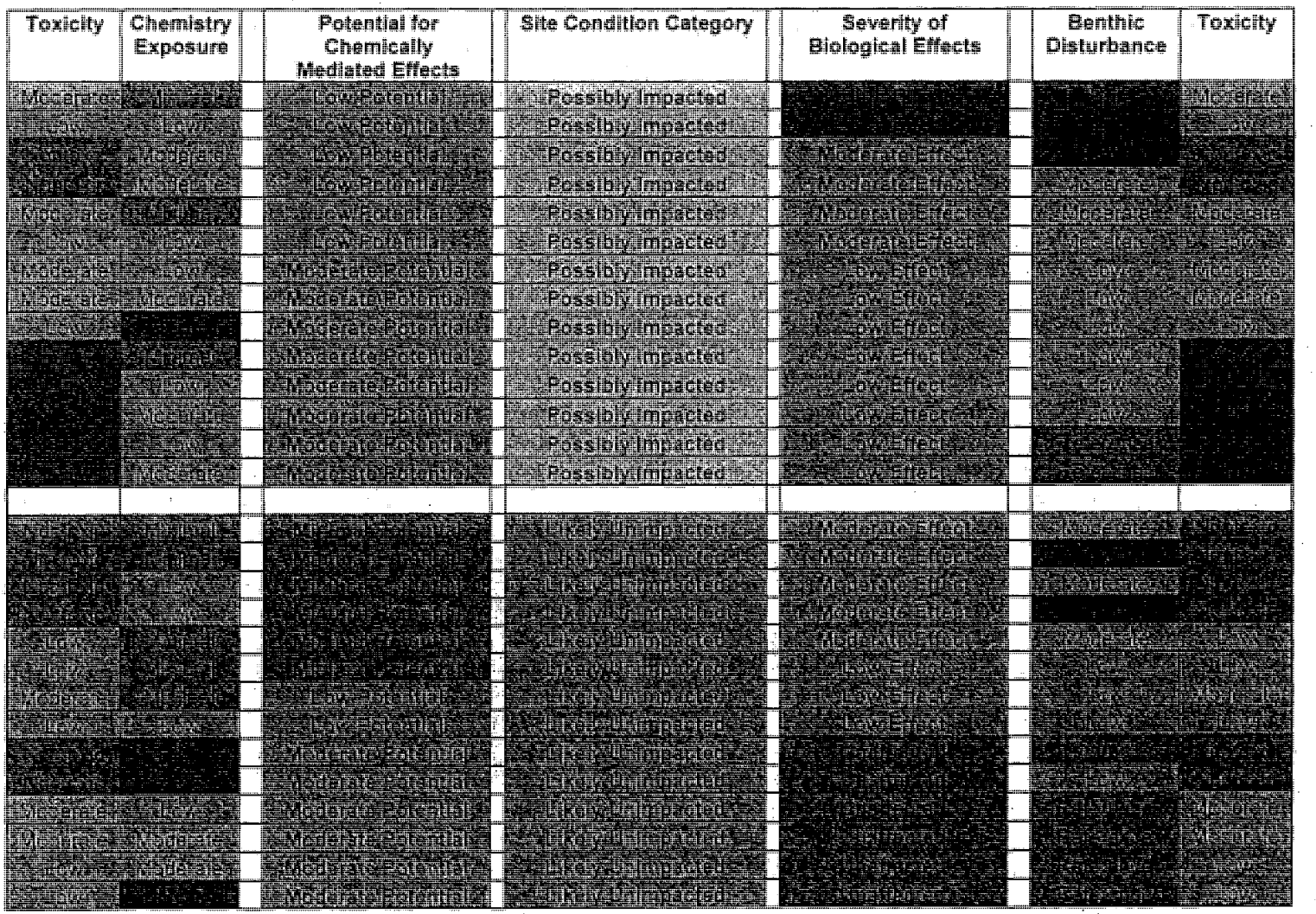


Table A.1 Continued



Toxicity	Chemistry Exposure	Potential for Chemically Mediated Effects	Site Condition Category	Severity of Biological Effects	Benthic Disturbance	Toxicity
Moderate	Low	Low Potential	Possibly Impacted			Moderate
Low	Low	Low Potential	Possibly Impacted			Low
Low	Moderate	Low Potential	Possibly Impacted	Moderate Effect		Low
Low	Moderate	Low Potential	Possibly Impacted	Moderate Effect	Moderate	Low
Moderate	Moderate	Low Potential	Possibly Impacted	Moderate Effect	Moderate	Moderate
Low	Low	Low Potential	Possibly Impacted	Moderate Effect	Low	Low
Moderate	Low	Moderate Potential	Possibly Impacted	Low Effect	Low	Moderate
Moderate	Moderate	Moderate Potential	Possibly Impacted	Low Effect	Low	Moderate
High	High	Moderate Potential	Possibly Impacted	Low Effect	Low	High
High	Moderate	Moderate Potential	Possibly Impacted	Low Effect	Low	High
High	Low	Moderate Potential	Possibly Impacted	Low Effect	Low	High
High	Moderate	Moderate Potential	Possibly Impacted	Low Effect	Low	High
High	Low	Moderate Potential	Possibly Impacted	Low Effect	Low	High
High	Moderate	Moderate Potential	Possibly Impacted	Low Effect	Low	High
Moderate	High	Moderate Potential	Likely Impacted	Moderate Effect	Moderate	Moderate
Moderate	High	Moderate Potential	Likely Impacted	Moderate Effect	High	Moderate
Moderate	High	Moderate Potential	Likely Impacted	Moderate Effect	Moderate	Moderate
Moderate	High	Moderate Potential	Likely Impacted	Moderate Effect	High	Moderate
Low	High	Low Potential	Likely Impacted	Moderate Effect	Moderate	Low
Moderate	High	Low Potential	Likely Impacted	Low Effect	Moderate	Moderate
Low	High	Low Potential	Likely Impacted	Low Effect	Moderate	Low
High	High	Moderate Potential	Likely Impacted	Moderate Effect	High	High
High	High	Moderate Potential	Likely Impacted	Moderate Effect	High	High
Moderate	Moderate	Moderate Potential	Likely Impacted	Moderate Effect	Moderate	Moderate
Low	Moderate	Moderate Potential	Likely Impacted	Moderate Effect	Moderate	Low
Low	High	Moderate Potential	Likely Impacted	Moderate Effect	Moderate	Low

Table A.1 Continued

Toxicity	Chemistry Exposure	Potential for Chemically Mediated Effects	Site Condition Category	Severity of Biological Effects	Benthic Disturbance	Toxicity
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Low	[illegible]
[illegible]	Low	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	Low	[illegible]	[illegible]	[illegible]	Low	[illegible]
Low	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	Low
Moderate	[illegible]	Low Potential	[illegible]	[illegible]	[illegible]	Moderate
[illegible]	Moderate	Low Potential	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	Moderate	Low Potential	[illegible]	[illegible]	Low	[illegible]
Low	Low	Low Potential	[illegible]	[illegible]	[illegible]	Low
Moderate	[illegible]	[illegible]	Inconclusive	[illegible]	[illegible]	Moderate
Low	[illegible]	[illegible]	Inconclusive	[illegible]	[illegible]	Low
[illegible]	[illegible]	Moderate Potential	Inconclusive	Low Effect	[illegible]	[illegible]

APPENDIX B. CALIFORNIA SQO ASSESSMENT RESULTS

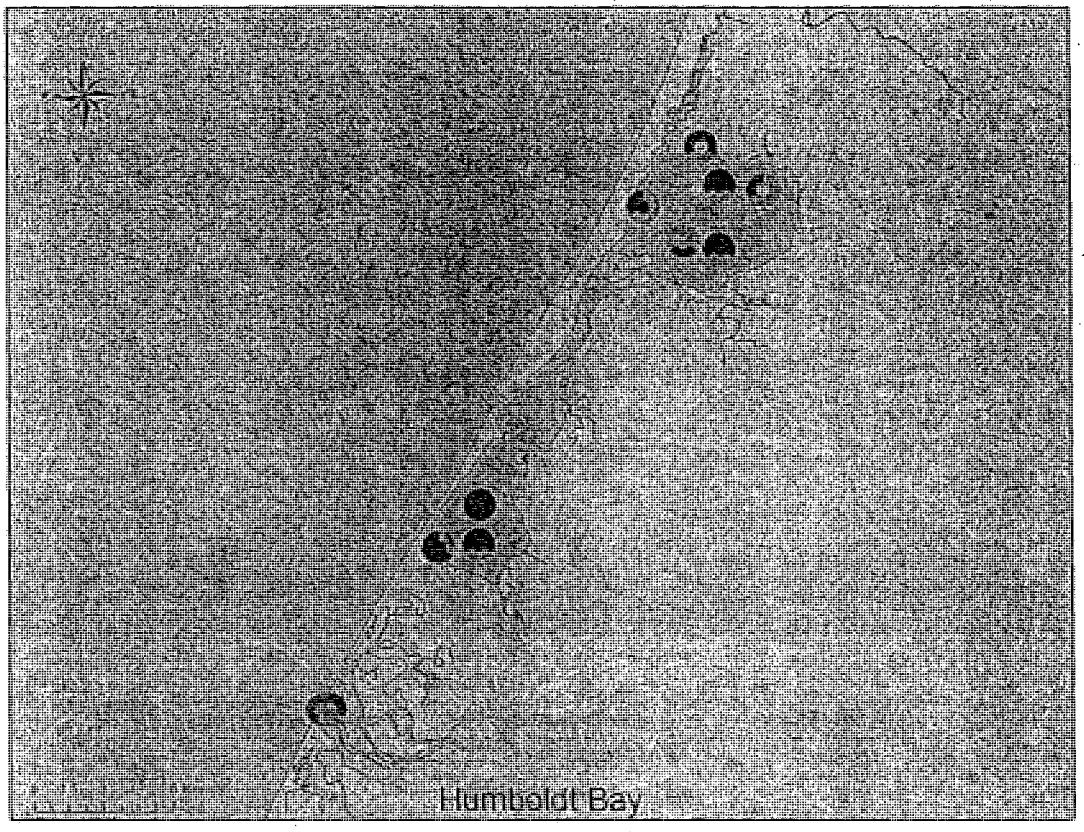
Table B.1. Statewide embayment sediment quality condition with confidence limits of the percent area and estimated area for each MLOE classification.

Area	Condition Category	No. of Sites	Estimated Portion (%)	0.95 LCB (%)	0.95 UCB (%)	Estimated Area (km^2)	0.95 LCB (km^2)	0.95 UCB (km^2)
Statewide	Unimpacted	131	10.0	7.9	12.2	129.5	101.7	157.4
Statewide	Likely Unimpacted	57	6.7	1.7	11.8	87.3	21.4	153.1
Statewide	Possibly Impacted	111	65.4	55.3	75.5	847.1	716.2	978.1
Statewide	Likely Impacted	51	16.8	7.1	26.4	217.4	92.3	342.5
Statewide	Clearly Impacted	25	0.5	0.0	1.0	6.3	0.2	12.5
Statewide	Inconclusive	6	0.6	0.0	1.2	7.4	0.0	15.5
Total		381	100			1295.1		

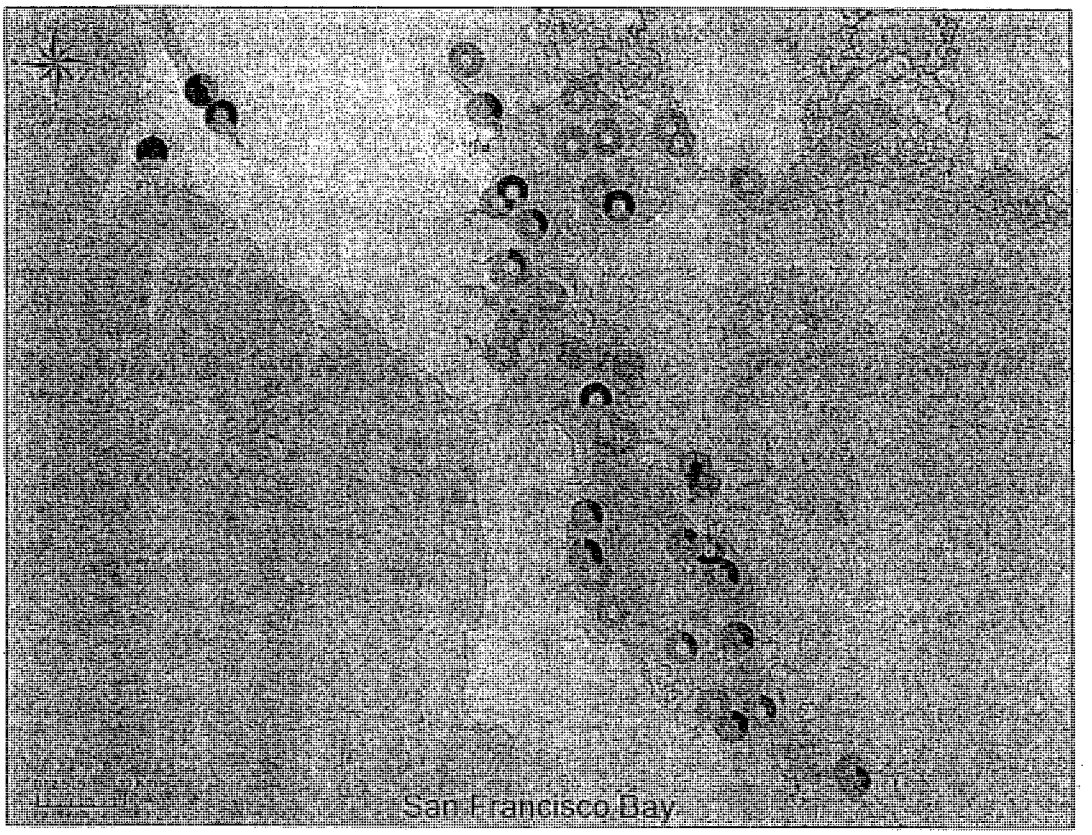
Table B.2. Regional embayment sediment quality condition with confidence limits of the percent area and estimated area for each MLOE classification.

Regional Area	Condition Category	No. of Sites	Estimated Portion (%)	0.95 LCB (%)	0.95 UCB (%)	Estimated Area (km^2)	0.95 LCB (km^2)	0.95 UCB (km^2)
North	Unimpacted	9	58.5	37.0	79.9	81.4	51.5	111.3
North	Likely Unimpacted	9	17.6	1.3	34.0	24.5	1.8	47.3
North	Possibly Impacted	4	14.4	0.0	29.2	20.1	0.0	40.6
North	Likely Impacted	2	3.8	0.0	8.7	5.2	0.0	12.1
North	Clearly Impacted	0	0.0	0.0	0.0	0.0	0.0	0.0
North	Inconclusive	3	5.8	0.0	12.3	8.0	0.0	17.1
Total		27	100.0			139.3		
SFB	Unimpacted	0	0.0	0.0	0.0	0.0	0.0	0.0
SFB	Likely Unimpacted	2	3.9	0.0	9.9	39.6	0.0	100.8
SFB	Possibly Impacted	28	76.7	64.3	89.2	783.1	655.7	910.3
SFB	Likely Impacted	9	19.1	7.0	31.1	194.4	71.7	317.2
SFB	Clearly Impacted	1	0.3	0.0	0.9	3.4	3.4	9.3
SFB	Inconclusive	0	0.0	0.0	0.0	0.0	0.0	0.0
Total		40	100.0			1020.5		
South	Unimpacted	122	42.9	36.5	49.3	58.1	49.4	66.8
South	Likely Unimpacted	46	18.9	13.2	24.6	25.5	17.8	33.2
South	Possibly Impacted	79	24.6	19.2	30.1	33.3	25.9	40.7
South	Likely Impacted	40	11.2	7.0	15.5	15.2	9.5	20.9
South	Clearly Impacted	24	2.1	1.0	3.2	2.8	1.4	4.4
South	Inconclusive	3	0.3	0.0	0.6	0.4	0.0	0.8
Total		314	100.0			135.3		

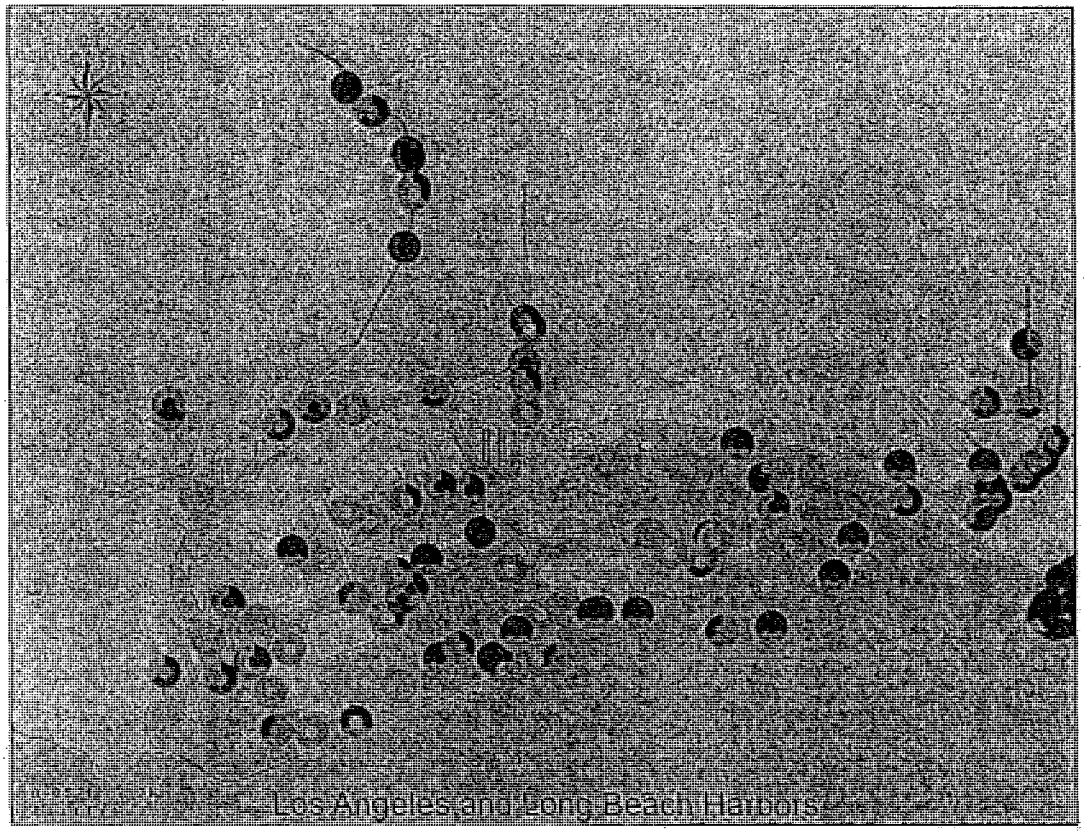
APPENDIX C. ASSESSED SEDIMENT CONDITION AND LOE CATEGORIES AT INDIVIDUAL STATIONS IN SELECTED CALIFORNIA EMBAYMENTS



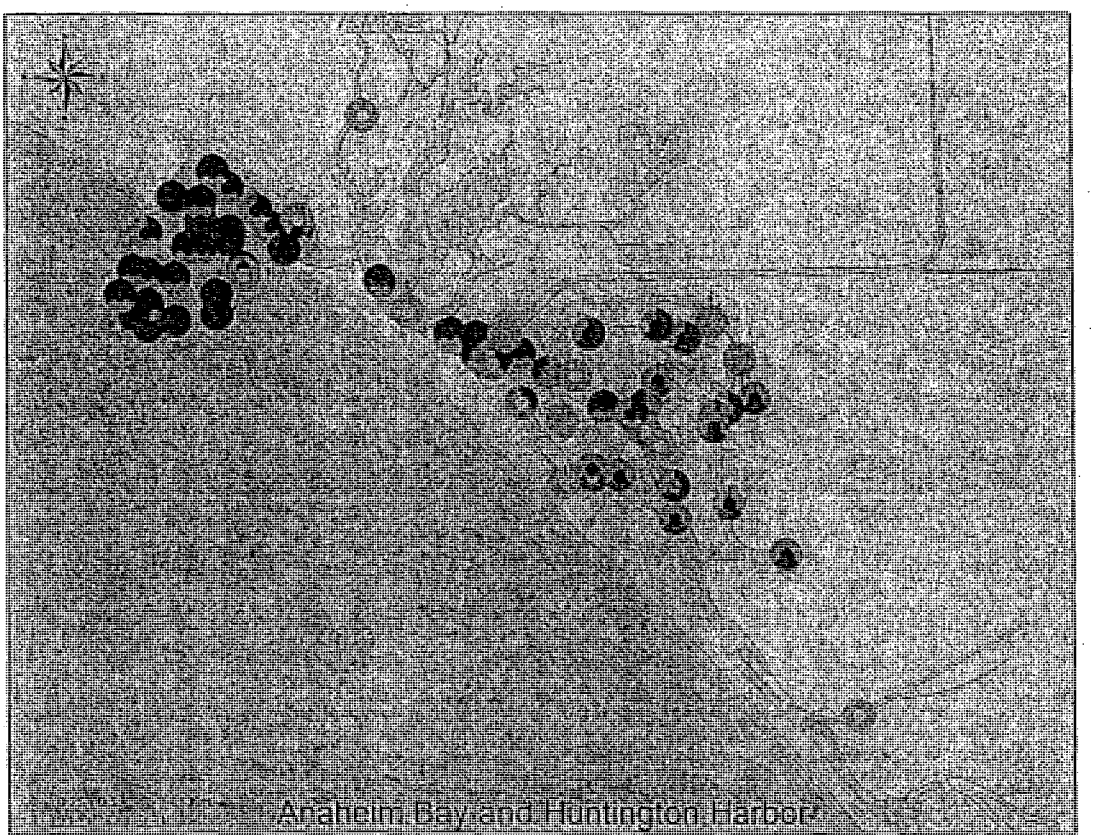
LOE Categories	MLOE Assessment	LOE Assessment			
	Unimpacted Likely Unimpacted				
B=Benthic Disturbance	Possibly Impacted	Reference	Low	Moderate	High
T=Toxicity	Likely Impacted	Nontoxic	Low	Moderate	High
C=Chemistry Exposure	Clearly Impacted	Minimal	Low	Moderate	High
M=MLOE Assessment					



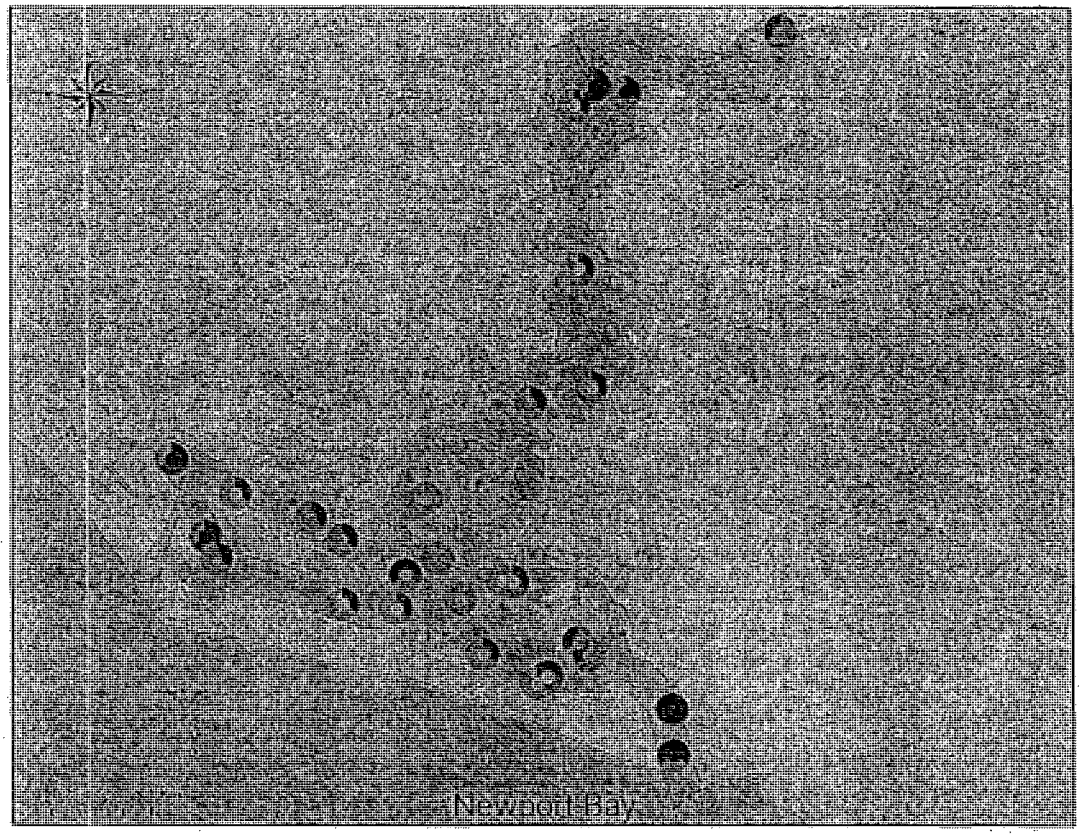
LOE Categories	MLOE Assessment	LOE Assessment			
	Unimpacted Likely Unimpacted				
B=Benthic Disturbance T=Toxicity C=Chemistry Exposure M=MLOE Assessment	Possibly Impacted Likely Impacted Clearly Impacted	Reference Nontoxic Minimal	Low Low Low	Moderate Moderate Moderate	High High High



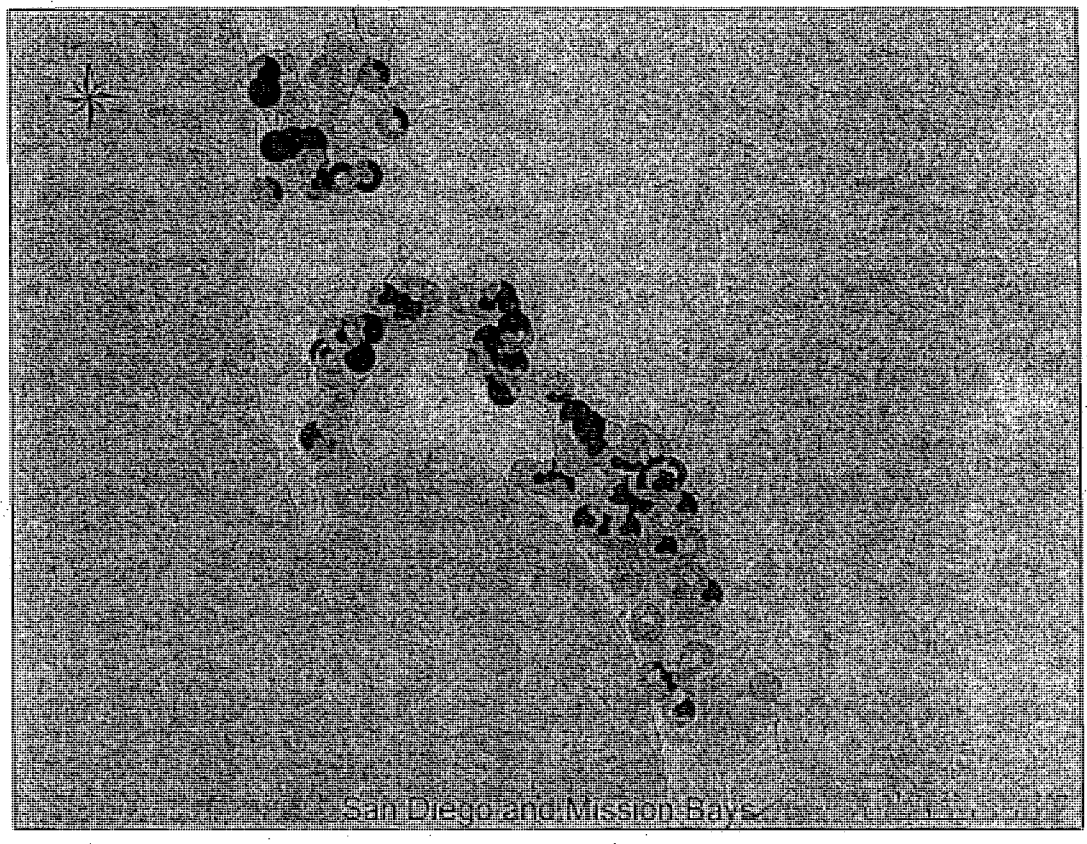
LOE Categories	MLOE Assessment	LOE Assessment			
	Unimpacted Likely Unimpacted Possibly Impacted Likely Impacted Clearly Impacted				
B=Benthic Disturbance T=Toxicity C=Chemistry Exposure M=MLOE Assessment		Reference Nontoxic Minimal	Low Low Low	Moderate Moderate Moderate	High High High



LOE Categories	MLOE Assessment	LOE Assessment			
B=Benthic Disturbance T=Toxicity C=Chemistry Exposure M=MLOE Assessment	Unimpacted Likely Unimpacted Possibly Impacted Likely Impacted Clearly Impacted	Reference Nontoxic Minimal	Low Low Low	Moderate Moderate Moderate	High High High



LOE Categories	MLOE Assessment	LOE Assessment			
B=Benthic Disturbance T=Toxicity C=Chemistry Exposure M=MLOE Assessment	Unimpacted Likely Unimpacted Possibly Impacted Likely Impacted Clearly Impacted	Reference Nontoxic Minimal	Low Low Low	Moderate Moderate Moderate	High High High



LOE Categories	MLOE Assessment	LOE Assessment			
	Unimpacted Likely Unimpacted				
B=Benthic Disturbance	Possibly Impacted	Reference	Low	Moderate	High
T=Toxicity	Likely Impacted	Nontoxic	Low	Moderate	High
C=Chemistry Exposure	Clearly Impacted	Minimal	Low	Moderate	High
M=MLOE Assessment					

Mentink, Dale@OAL

From:	Dale P. Mentink
Sent:	Thursday, April 10, 2008 4:58 PM
To:	'Chris Beegan'
Cc:	Dale P. Mentink
Subject:	OAL file no. SWRCB_2008-0229-07 sediment quality

Chris, I left a message for you this morning as to how things are going, but maybe you're out today.

A couple of other questions came up from our rulemaking file review committee. Two were just questions on the plan and one was a request for some additional information in response to one of the comments as long as I had asked about those other 14.

In terms of the plan, on page 08602 (page 8 of the plan), the committee wondered what the standard will be for approval of the "other methods" approved for use by the boards? In other words, if there are some standards already in mind, those should be stated in the plan, or the plan should simply explain why that approval process needs to be case by case.

On page 08614 (page 20 of the plan), what does the phrase "or other waters of significant national importance" refer to?

The other response to comment (and I'll find the page in the record in the morning) was just the one in which the use of the narrative as opposed to numeric object was challenged and being responded to and you mentioned that when numeric criteria are infeasible, the Water Code and Clean Water Act authorize the use of narratives. Could you provide the Water Code and Clean Water Act cites for that?

Thank you.

Please let me know how things are going. We have a due date of 4/14.
Dale Mentink
OAL, 323-6817

EXHIBIT 707
WIT: Beegan
DATE: 10/9/10
CAROL NYGARD DROBNY

In terms of responses, I identified 12 where the response was only that Staff Disagrees, but the comment wasn't lacking in specifics or merely rhetorical. Could you flesh out the responses to those 12 somewhat please? They are on the following pages: 08717 (30), 08718 (228), 08719 (231), 08719 (557), 08727 (23), 08737 (257), 08739 (262), 08747 (20), 08777 (298), 08800 (366), 08805 (381), and 08805 (384).

On page 08725 (241), the comment contains a specific question [Does this mean that significant differences for any two lines of evidence could drive an impairment designation?] and a specific recommendation [We recommend that benthic community data must be one of the two lines of evidence suggesting adverse effects before an impairment designation is assigned.]. The response is not really responsive. Could you flesh out that response?

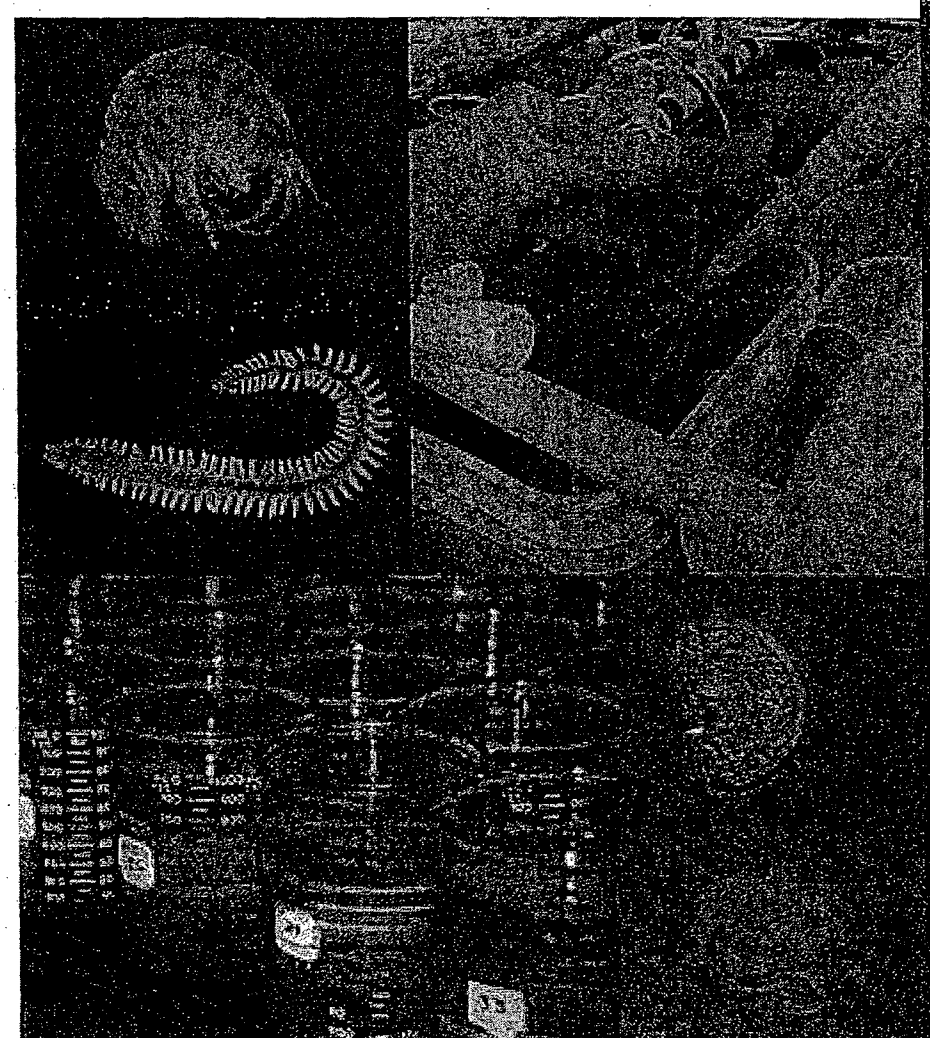
On page 08803 (375), the comment is that monitoring may be as infrequent as once in five years and that such infrequent monitoring will allow degradation. The response is that the language describing maximum frequency has been deleted. There is nothing in the response explaining/justifying the minimum frequency monitoring which the commenter raised. Could you flesh out that response?

Please call or reply if you have any questions.

Dale Mentink, Office of Administrative Law, 323-6817.

EVALUATION OF METHODS FOR MEASURING SEDIMENT TOXICITY IN CALIFORNIA BAYS AND ESTUARIES

Technical Report 503
March 2007



Steven Bay
Darrin Greenstein
Diana Young

Southern California Coastal Water Research Project

Evaluation of Methods for Measuring Sediment Toxicity in California Bays and Estuaries

Steven Bay, Darrin Greenstein and Diana Young

Southern California Coastal Water Research Project
3535 Harbor Blvd., Suite 110
Costa Mesa, CA 92626

www.sccwrp.org

March 2007

Technical Report 503

FOREWARD

The State Water Resources Control Board (SWRCB) initiated a program in 2003 to develop sediment quality objectives (SQOs) for chemical contaminants in California bays and estuaries. The SQOs will include narrative descriptions of the condition to be protected and the associated analytical methods needed to determine whether the condition has been attained. The Southern California Coastal Water Research Project, in partnership with other state and federal agencies, conducted a series of technical studies in order to provide a sound scientific foundation for the selection of methods and the development of a data interpretation framework for use in the SQO program. This report presents the results of an evaluation of sediment toxicity test methods for use in the assessment of the direct effects of sediment contamination. Other reports will describe studies related to the assessment of benthic macrofaunal community condition, sediment contamination, assessment of indirect effects from consumption of contaminated seafood by humans and wildlife, and the integration of all of these data to assess overall sediment quality. Copies of this and related reports are available for download at www.sccwrp.org and www.waterboards.ca.gov.

This study was funded in part by agreement 01-274-250-0 with the State Water Resources Control Board.

ACKNOWLEDGMENTS

The authors would like to thank Art Barnett and Stephen Weisberg for their assistance in the preparation of this document. We would like to thank J. Daniel Farrar, David Moore, Bryn Phillips, and Michele Redmond for providing data that was used in the evaluation and calculation of thresholds.

Many thanks are due to the members of the Scientific Steering Committee (Todd Bridges, Rob Burgess, Tom Gries, Peter Landrum, Ed Long, and Bob Van Dolah) for their advice during all phases of this project. Finally, the authors wish to thank members of the Sediment Quality Advisory Committee and Agency Coordination Committee for their input during this study.

EXECUTIVE SUMMARY

Toxicity tests have been widely used to assess sediment quality in a variety of research, monitoring, and regulatory programs. While many programs use a combination of test methods and follow standardized protocols, there is variation between programs in the selection of test methods or in the way that the data are interpreted. This is problematic when incorporating sediment toxicity into a regulatory program with broad applicability, such as the sediment quality objectives program under development in California. Multiple factors such as test feasibility, relevance to program/policy objectives, data comparability, cost, and sensitivity must be considered, yet this information is frequently not available. In addition, a consistent method of toxicity data interpretation is needed so that station assessments conducted in one region are comparable to the results from other locations or times.

The current study had two objectives: to evaluate a variety of acute and sublethal toxicity tests in order to identify methods that were best suited for use in a statewide regulatory program, and to develop a system to classify the toxicity test results into a series of categories of effect. A list of candidate test methods was developed based on a literature review and consultation with other scientists. The candidate test methods list included acute test methods with four amphipod species. Six sublethal methods were also evaluated: a copepod life cycle test, amphipod growth, polychaete growth, clam growth, oyster cell stress, and mussel or sea urchin embryo development.

Data on the feasibility, sensitivity, variability, and cost of each candidate method were compiled from the literature and from two sets of laboratory experiments. The first set of experiments compared the relative sensitivity of each of the candidate test methods for detecting toxicity in a set of 15 sediment samples from various California embayments. A wide range of responsiveness to the samples was observed. The copepod life cycle and polychaete growth tests showed the greatest responses to the sediment samples. Some of the sublethal tests identified a smaller total number of stations as toxic than the standard amphipod survival test(s), yet each of the sublethal tests detected toxicity in some samples that were classified as nontoxic by the amphipod survival test. This suggests that sublethal tests and acute tests are complementary rather than redundant and can provide different sensitivity responses.

Experiments were also conducted to evaluate the interlaboratory variability of the clam growth and embryo development test methods when applied to both field and laboratory-spiked sediments. The interlaboratory variability of these tests was greater than reported for some amphipod survival tests, but was within the range of variability for other sublethal test methods.

The data were compiled into a matrix of test characteristics and scored based on relative performance of each test. The acute and sublethal methods were evaluated separately.

The following five tests were identified as best suited for use in a California statewide sediment quality assessment program.

Species	Taxonomic Group	Matrix	Duration (days)	Endpoint(s)
Acute				
Eohaustorius estuarius *Leptocheirus plumulosus* *Rhepoxynius abronius*	Amphipod	Whole sediment	10	Survival
Sublethal				
Neanthes arenaceodentata	Polychaete	Whole sediment	28	Growth
Mytilus galloprovincialis	Bivalve	Sediment-water interface	2	Embryo development

The use of multiple toxicity tests to assess sediment quality is suggested, as none of the test methods ranked consistently highest with respect to sensitivity or reliability. The use of a diversity of test methods provides two key advantages: it reduces the influence of spurious results from a test and it also increases the overall sensitivity of the testing program by using species with different patterns of contaminant sensitivity.

A data analysis framework was developed for the highest rated test methods. This framework was based on an ordinal scoring system consisting of four categories of effect.

- **Nontoxic:** Response not substantially different from that expected in sediments that are uncontaminated and have optimum characteristics for the test species
- **Low toxicity:** A response that is of relatively low magnitude; the response may not be greater than test variability
- **Moderate toxicity:** High confidence that a statistically significant effect is present
- **High toxicity:** Highest confidence that a toxic effect is present and the magnitude of response is among the strongest effects observed for the test

Three response thresholds (low, moderate, and high) were developed for use in assigning one of the above response categories to each test result.

Species	Low (%)	Moderate (% Control)	High (% Control)
Eohaustorius estuarius	90	82	59
Rhepoxynius abronius	90	83	70
Leptocheirus plumulosus	90	78	56
Neanthes arenaceodentata	90[1]	68	46
Mytilus galloprovincialis	80	77	42

[1] % of control growth.

Several data limitations were encountered in the course of this study that either reduced the ability of a test method to meet the minimum evaluation criteria or complicated the calculation of the classification thresholds. Research is needed to improve the feasibility

of some of the candidate test methods. Additional data are also needed to refine the thresholds for the *Leptocheirus plumulosus* and *Neanthes arenaceodentata* tests.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

Toxicity tests are an integral part of the sediment quality triad used in many monitoring and assessment programs (Long and Chapman 1985). These tests provide information on the potential for adverse biological effects from contaminants and are recognized as a key component of the ecological risk assessment process (USEPA 1998) and programs to evaluate the suitability of dredged material for ocean disposal (USEPA 1991, PSWQA 1995). Sediment toxicity tests have also been widely used in monitoring and assessment programs to evaluate sediment quality within coastal bays and estuaries (Fairey *et al.* 1998) and at regional and national scales (Long 2000, USEPA 2004).

A wide variety of methods have been used to measure sediment toxicity (Lamberson *et al.* 1992). Many studies use a suite of tests that includes both acute (short-term survival) and sublethal methods. Much of the acute testing has employed amphipod survival methods using standard protocols established by the U.S. Environmental Protection Agency (USEPA 1994). The use of these standard protocols provides a measure of biological effects that can be compared among regions statewide and nationwide; such comparisons are not always possible using other measures of biological effects. The types of sublethal toxicity tests used in assessment studies is more variable, with methods including growth and reproduction tests of whole sediment, pore water, water or solvent extracts of the sediment (Ringwood *et al.* 1996, Bay *et al.* 1998, Long *et al.* 1999, Long *et al.* 2005). There is little consistency among programs in the types of the sublethal tests used; selection is often performed on a site-specific basis and is based on factors such as availability of test organisms, expected sensitivity, cost, local interests, and availability of collaborators. Consequently, only a few sublethal methods have been used commonly; they include the amphipod *Leptocheirus plumulosus* 28-day growth and reproduction test (USEPA 2001), a 20-day polychaete growth test using *Neanthes arenaceodentata* (PSWQA 1995), pore water or elutriate tests using echinoderm or bivalve gametes or embryos (PSWQA 1995, ASTM 2002a, Carr and Nipper 2003), and a sediment-water interface (SWI) test using sea urchin or mussel embryos (Anderson *et al.* 1996).

Information on the comparative sensitivity of sediment toxicity tests is an important factor to consider in test selection, yet only limited data are available. Most comparative studies include just a few species and sometimes provide conflicting results (Table 1). The differences in species, test methods, sample type, and relative sensitivity of the test methods complicate the integration of the results of these studies for use in selecting methods for use in other studies. Additional comparative studies that use a consistent study design applied to each test are needed to help evaluate the relative sensitivity of the toxicity tests of interest.

The selection of sediment toxicity test methods requires a consideration of many factors in addition to sensitivity, depending upon the study's objectives and design. Much variability in method selection is found among research studies conducted on a small scale, as the emphasis is often on selecting methods to address site-specific scientific questions, method development, or building upon previous work by an investigator. Additional factors must be considered when selecting test methods for use in large-scale monitoring or regulatory programs. For example, the methods must be feasible for use by many different laboratories and at different times of the year, and have a wide tolerance of habitat variables such as sediment grain size and salinity.

Toxicity test method selection for these types of programs must consider factors such as test feasibility, relevance to program/policy objectives, data comparability, and cost, in addition to sensitivity. The sediment quality objectives (SQO) program under development by the State of California provides an example of the many factors to be considered when sediment toxicity tests are used in a regulatory context. The California SQO program is based on the sediment quality triad and will be applied to bays and estuaries throughout the state (SWRCB 2006). The selection of toxicity test methods for a statewide regulatory program must be sensitive to environmental contamination at levels that are ecologically relevant, standardized to ensure consistent application, and feasible for application in a variety of situations. The test methods should also be ecologically relevant, meaning that the choice of species and test conditions results in a test that responds to environmental contamination on a scale that is useful for describing potential impacts on California species. In addition, a consistent and relatively simple method of toxicity data interpretation is needed so that station assessments conducted in one region are comparable to the results from other locations or times. Past comparisons of sediment toxicity test methods have not addressed many of these issues or were limited to a small subset of test methods that do not fully address the needs of a statewide regulatory program.

The current study had two principal objectives. The first objective was to evaluate a variety of acute and sublethal toxicity tests in order to identify methods that were best suited for use in a statewide regulatory program. To address this objective, a candidate list of potential tests was identified and evaluated with respect to feasibility, performance, and cost. The second objective was to develop a consistent and comparable system to classify the toxicity test results into a series of categories of effect. The approach to address this second objective included developing a conceptual data analysis framework and identifying a series of test-specific response thresholds that incorporated the magnitude and uncertainty in the test response.

Table 1. Summary of studies comparing the sensitivity of acute survival (A) and sublethal (S) toxicity tests.

Species and Methods	Sample Type	Relative Sensitivity	Reference
Ampelisca abdita (A) *Eohaustorius estuarius* (A) *Leptocheirus plumulosus* (A)	Field Sediment and Cadmium	Sediment: *A. abdita*>*L. plumulosus*>*E. estuarius* Cd: *A. abdita* =*L. plumulosus*>*E. estuarius*	(Schlekat *et al.* 1995)
A. abdita (A) *Ampelisca verrilli* (A) *Mercenaria mercenaria* (A) *Palaemonetes pugio* (A) *Brachionus plicatilis* (A) *Amphiascus tenuiremis* (A) Microtox (S)	DDT, Fluoranthene, Cadmium	DDT: *P. pugio* most sensitive Fluoranthene and Cd: *M. mercenaria* most sensitive	(Fulton *et al.* 1999)
Polydora cornuta (S) *Boccardia proboscidea* (S) *Neanthes arenaceodentata* (S) *L. plumulosus* (S) *Schizopera knabeni* (S)	Copper	*S. knabeni* most sensitive *L. plumulosus* and *B. proboscidea* least sensitive	(Farrar *et al.* 1998)
L. plumulosus (S) *E. estuarius* (A) *N. arenaceodentata* (S)	Field Sediment	*E. estuarius*>*N. arenaceodentata*>*L. plumulosus*	(Pinza *et al.* 2002)
L. plumulosus (S) *A. abdita* (A) *N. arenaceodentata* (S)	Field Sediment	*L. plumulosus*>*A. abdita*>*N. arenaceodentata*	(Kennedy *et al.* 2004)
L. plumulosus (A) *L. plumulosus* (S) *N. arenaceodentata* (S)	Field Sediment	*L. plumulosus*> *N. arenaceodentata*	(Moore *et al.* 2003)
A. abdita (A) *Rhepoxynius abronius* (A) *Mytilus galloprovincialis* (S) *Strongylocentrotus purpuratus* (S) *Dinophilus gyrociliatus* (S)	Field Sediment	*M. galloprovincialis* and *R. abronius* most sensitive *A. abdita* least sensitive	(Long *et al.* 1990)

EVALUATION OF ACUTE AND SUBLETHAL TESTS

Approach

A set of candidate acute and sublethal test methods was selected for evaluation. Methods were selected that had a direct sediment exposure, appeared to be technically feasible and had data available that indicated sensitivity to contaminated sediments. The test methods and species included those that have been recommended for use in other regulatory programs in California (USEPA and Engineers 1998) or were documented in standard procedures developed by government or scientific agencies (e.g., EPA or ASTM). Priority was given to methods using species resident in California and species representative of important infaunal groups. In order to increase the diversity of life histories and biological endpoints evaluated, additional candidate methods were selected based on a review of the scientific literature and from recommendations by other scientists familiar with sediment toxicity testing. This process led to the identification of six candidate sublethal methods for evaluation (Table 2). Four amphipod species recommended by the USEPA for testing acute sediment toxicity were also included in the list (USEPA 2001).

Each test was evaluated based on a set of characteristics relating to test feasibility, performance and cost. The list of characteristics was established to include parameters used in previous test comparisons (Long *et al.* 1990, Lamberson *et al.* 1992) and was refined using input from an external scientific review committee. The following characteristics were evaluated:

- **Organism availability.** This category relates to both abundance of suppliers of the animals and any seasonal aspect of either their availability or sensitivity. Ideally, test organisms should be available from multiple suppliers on a year-round basis with no seasonal variation in test sensitivity. Information for this parameter came from contacting suppliers or from experience in using the organisms.

- **Method description.** This category describes whether a standardized protocol for a given test has been established. Methods that are termed as "standard" have a protocol that has received the rigorous testing necessary to be published as an EPA or ASTM method and is the preferred level of method description. These methods have control acceptability criteria and quality assurance standards for parameters such as water quality associated with them.

- **Technical difficulty.** An important consideration is the ease for laboratories to successfully conduct the test. If a method is difficult to perform, laboratories may have to perform multiple tests just to obtain acceptable results. The difficulty was rated based on ability to obtain acceptable controls (i.e., relative number of test failures), the necessity of special techniques or equipment, and complexity of the exposure system. The information for this parameter was based on a combination of personal experience of the authors and comments from others who routinely perform the tests.

- **Concordance of results.** For evaluating the degree of concordance, the effects on the sublethal methods were compared to those of the acute methods tested simultaneously. For the sublethal methods, there was an expectation that if a site were strongly, acutely

toxic to a test organism, then an effect would also be seen for the sublethal test. Conversely, if a site were considered to be in "reference condition" then there would be an expectation that no toxicity would be found for any of the test methods. The information for this parameter was taken from published reports in which both an amphipod species and at least one of the sublethal tests had been applied on the same samples. To evaluate concordance, the acute amphipod test was used as the ground truth, so no acute amphipod data appear in Table 3.

- **Relative sensitivity.** This category describes the relative response of the acute and sublethal tests by observing the relative frequency that the test identifies a sample as being toxic, compared to a benchmark test. Sensitivity in the context of this study refers to the range in response obtained using a specific test method, not the inherent sensitivity of a species to individual chemicals. Many factors related to the specifics of the test, such as duration, temperature, and life stage can affect the response and apparent sensitivity of a toxicity test. Test sensitivity was evaluated relative to the acute amphipod test species most commonly used in California, *Eohaustorius estuarius*. This species has a substantial history of use in California for both monitoring and assessment studies. The logic behind this assessment was that if a test method was usually less sensitive than the most commonly used test, then its value in providing additional information would be limited. Information for this characteristic was gathered from published reports where the benchmark test was conducted alongside at least one of the sublethal methods. For many of the methods, no data was available, so a study was conducted to help fill this information gap (Appendix A).

- **Reproducibility among laboratories.** This category describes the relative amount of variability in the results that is observed when multiple laboratories test the same sample. The information was mostly obtained from literature reports on round-robin tests. In the case of the *Mercenaria mercenaria* growth test and the SWI test using mussel embryos, round-robin testing was conducted to add information that was missing from the literature (Appendix B).

- **Reproducibility within laboratories.** This category describes the relative amount of variability in the results when an individual lab tests the same sample multiple times. The information was obtained mostly from reference toxicant exposures.

- **Precision.** The relative precision of response describes the between-replicate variability of the methods. Information for this parameter was obtained from published reports and journal articles.

- **Documentation of confounding factors.** Most toxicity tests are sensitive to some type of non-contaminant effect (e.g., grain size) that can have a confounding effect on test results. Knowledge of which factors can affect a test and the range where effects occur is needed for study design and data interpretation. Information for this parameter was gathered from test protocols or from values published in the literature.

- **Cost.** Cost is a limiting factor in many sediment assessment studies. The use of sensitive tests that are also relatively inexpensive will enable a larger number of stations to be evaluated, thus improving spatial resolution and overall confidence in the results. The unit cost of each test was evaluated relative to the standard 10-day amphipod survival test. The first source of information for this parameter was from the costs associated with the tests that were commissioned as part of this study. Secondarily, biological consulting firms in California provided costs for tests that they currently perform. For the tests that were new to California, the firms were asked to estimate what they would charge to conduct them.

The characteristics were summarized into narrative categories that reflected the relative level of attainment for each of the candidate tests (e.g., poor, fair, good). The acute and sublethal test methods were treated separately during this process due to differences in the characteristics evaluated.

A scoring system was then applied to integrate the category level information in order to produce an overall evaluation and ranking of each test. Test selection was based on consideration of both test feasibility and relative performance/cost. The three feasibility characteristics (organism availability, method description, and technical difficulty) were evaluated using a binary (yes/no) scoring system. These characteristics were deemed to be so important that the test was classified as not feasible if minimum criteria were not met. For organism availability, at least one commercial source of animals must currently be available to purchase animals ready to use for testing. For method description, there must be a published document available that has a complete description of the method, including test acceptability criteria. The technical difficulty criterion was that there was a reasonable expectation that a laboratory experienced in performing other toxicity tests could follow the protocol and successfully conduct the method without receiving additional outside training. For each of these characteristics, the method was assigned a "+" if the criterion was met and a "-" if it was not.

The remaining performance and cost characteristics were evaluated using a weighted scoring system based on the narrative categories. A weighting factor was established for each category based on our assessment of the relative importance of each category. The comparative sensitivity category was assigned the highest weight: a factor of 4. The high weight given to this category was based on the assumption that high sensitivity to contaminants was the most desirable trait for a sediment toxicity test method. The "relative precision of response" category was deemed to be the least important and was assigned a weighting factor of 1. All of the remaining categories were considered to be of intermediate importance and were assigned a weighting factor of 2.

A numeric value was assigned for each of the performance and cost characteristics. The values for each category ranged from 0 to 3 and corresponded to the narrative categories assigned based on the data review. A value of zero was assigned when no data were available for a characteristic. Each individual value was multiplied by its respective weighting factor to produce a score for the characteristic. The scores were then summed to obtain final score for each candidate test method.

Table 2. List of candidate sediment toxicity tests, the citations containing testing protocols and whether quality assurance and test acceptability criteria have been established.

Species	Taxonomic Group	Duration (days)	Matrix	Endpoint(s)	Literature Level	Citations	QA Criteria[1]	State/National Program Use[2]
Ampelisca abdita *Eohaustorius estuarius* *Rhepoxynius abronius* *Leptocheirus plumulosus*	Amphipod	10	Whole sediment	Survival	Well established	(USEPA 1994, ASTM 1996)	Yes	EMAP NOAA USACE WA, RMP
L. plumulosus	Amphipod	28	Whole sediment	Growth, reproduction, survival	Well established	(USEPA 2001)	Yes	USACE
Neanthes arenaceodentata	Polychaete	28	Whole sediment	Growth, survival	Exposure method under revision	(ASTM 2002b) modified	Yes	USACE[3] WA
Strongylocentrotus purpuratus	Sea urchin	3	Sediment-water interface	Embryo development	Published	(Anderson *et al.* 1996)	Yes	
Mytilus galloprovincialis	Mussel	2	Sediment-water interface	Embryo development	Published	(Anderson *et al.* 1996)	Yes	RMP
Amphiascus tenuiremis	Copepod	14	Whole sediment	Reproduction, survival	Published	(Chandler and Green 1996)	No	NOAA
Mercenaria mercenaria	Clam	7	Whole sediment	Growth, survival	Journal	(Ringwood and Keppler 1998, Keppler and Ringwood 2002)	No	EMAP
Crassostrea virginica	Oyster	4	Whole sediment	Lysosomal stability	Exposure method not published	(Ringwood *et al.* 1998, Ringwood *et al.* 2003)	No	

[1]Information on acceptable water quality ranges, reference toxicants, guidelines, acceptable control parameters, and within test variability are available

[2]EMAP: Environmental Monitoring and Assessment Program; NOAA: NOAA National Status and Trends Program; USACE (U.S. Army Corps of Engineers: dredged material evaluation for disposal under USACE or USEPA guidance; WA: dredged material evaluation for disposal under Washington State guidance; RMP: San Francisco Bay Regional Monitoring Program

[3]The same species and endpoint is used in dredged material evaluations, but the duration and aspects of the test method differ

Results

Acute Test Method Evaluation

The four acute amphipod test species were similar in regards to the test feasibility characteristics of organism availability, method description, and technical difficulty (Table 3). Each of the species is available from commercial suppliers, test methods have been standardized, and the level of difficulty is generally low. All of the amphipod species were scored as having met the feasibility criteria (Table 4).

E. estuarius received the highest overall score for the performance and cost characteristics (Table 4). *E. estuarius* has an extensive history of use in toxicity testing studies on California sediments (Anderson *et al.* 1997, Bay *et al.* 2000, Bay and Brown 2003, Bay *et al.* 2005). The method has been shown to have good reproducibility between laboratories (Bay *et al.* 2003).

A slightly lower total score was obtained for *L. plumulosus* (Table 4), which was due to lower reproducibility within and among laboratories. *L. plumulosus* received a lower rating compared to *E. estuarius* and *Rhepoxynius abronius* regarding documentation of confounding factors due to a lack of information on sensitivity to hydrogen sulfide, which was available for *E. estuarius* and *R. abronius*. The high ranking for relative sensitivity compared to *E. estuarius* was based on limited data from a single study and may not represent overall trends. The *L. plumulosus* 10-day test has been conducted in California on a very limited basis. However, it has long been used in other parts of the country, especially on the Gulf coast for monitoring and assessment studies. In studies using diluted, contaminated field sediments or spiked sediments, it has been shown that *L. plumulosus* has a sensitivity similar to the other species (Schlekat *et al.* 1995, Boese *et al.* 1997, DeWitt *et al.* 1997). One of the most attractive attributes of *L. plumulosus* is that it is easily cultured in the laboratory and available year round from commercial suppliers who have them in culture.

The *R. abronius* 10-day test was ranked similarly to the other acute methods, except for a low score for relative sensitivity compared to *E. estuarius*. The relative sensitivity score was based on limited data for split samples from a single study and may not represent overall trends. *R. abronius* has been previously used in California sediment toxicity programs (Long *et al.* 1990, Anderson *et al.* 1998, Anderson *et al.* 2001). These studies found the *R. abronius* method to have equal or better sensitivity to contaminated sediments as compared to other methods tested simultaneously. An interlaboratory comparison exercise using this method found good agreement amongst the testing laboratories (Mearns *et al.* 1986). However, test organism availability has recently been a problem with *R. abronius*. Laboratories have had recent difficulty in locating a supplier of *R. abronius*. The only available source of animals is in Washington, which requires an export permit prior to receipt of the animals. These factors may interfere with the ability to conduct this amphipod test in a timely manner. Sediments with a silt-clay content of ≥80% have also been shown to be an adverse confounding factor for *R. abronius* (DeWitt *et al.* 1988). Care should be taken when planning a survey that sediment grain size will not be an issue and that an animal source is readily available.

The Ampelisca abdita 10-day test was assigned the lowest total score among the four acute test species. The low score was driven by a lack of sensitivity compared to *E. estuarius* and a lower reproducibility among laboratories (Table 3). Specifically, in tests of California sediments where *A. abdita* has been tested simultaneously with *E. estuarius* or *R. abronius* it has consistently been found to be less sensitive (Figures 1 and 2). Very few data are available to make direct comparisons between *E. estuarius* and *R. abronius*, but toxicity in southern California sediments has been detected at a similar frequency using either of these species. The lower apparent sensitivity of *A. abdita* may be due to the fact that this species does not burrow in sediment, but lives in a tube-like structure and does not ingest sediment.

A. abdita also received a lower rating regarding documentation of confounding factors due to a lack of information on sensitivity to hydrogen sulfide. In addition, it is difficult to obtain *A. abdita* during the winter months and if they are available, they are of a size that is smaller than desired for use in testing (Table 3). The *A. abdita* test was also rated as being more difficult to conduct than other 10-day amphipod survival tests, based on the experiences of several California laboratories in having a higher test failure rate when using *A. abdita*, compared other amphipod species (Table 3). These difficulties are not due to intrinsic problems with the test organism, but are likely due to problems in obtaining *A. abdita* from suppliers within California. *A. abdita* is widely used as an indicator of sediment toxicity in many monitoring programs and the data have been used to characterize sediment quality on a national scale (Long 2000, USEPA 2004). Laboratories outside of California have had a high rate of success in conducting tests with *A. abdita* and technical difficulties reported in California do not preclude the use of the test in other regions.

Table 3. Characteristics of candidate sediment toxicity test methods. Not applicable for test (NA).

	California Samples Tested	Organism Availability[1]	Method Description[2]	Technical Difficulty[3]	Concordance at Clearly Clean or Impacted Sites[4]	More Sensitive Than *Eohaustorius estuarius* (number of comparisons)[5]	Reproducibility Among Laboratories[6]	Reproducibility Within Lababoratoriess[6]	Relative Precision of Response[7]	Documentation of Confounding Factors[8]	Cost of Method[9]
Amphipod Acute											
Eohaustorius estuarius	1697	12 (+)	Standard	Low	NA	NA	Good	Good	NA	Good	Low
R. abronius	1026	12 (1)	Standard	Low	NA	Never (9)	Good	Good	NA	Good	Low
Leptocheirus plumulosus	15	12 (+)	Standard	Low	NA	Often (15)	Fair	Poor	NA	Fair	Low
A. abdita	710	8 (+)	Standard	Moderate	NA	Rarely (228)	Poor	Good	NA	Fair	Low
Sublethal Methods											
Mercenaria mercenaria	15	8(+)	Published	Low	Fair	Sometimes (15)	Fair	Fair	Similar	Good	Low
Neanthes arenaceodentata	15	12(1)	Published	Moderate	Fair	Sometimes (15)	Good	Good	Low	Good	High
Sediment-water Interface											
Mytilus galloprovincialis	117	12(++)	Published	Low	Fair	Rarely (117)	Fair	Good	Low	Fair	Low
Strongylocentrotus purpuratus	195	5(++)	Published	Low	Fair	Rarely (184)	None	Good	Low	Good	Low
L. plumulosus	15	12(+)	Standard	Moderate	Fair	Sometimes (15)	Fair	Good	Low	Good	High
A. tenuiremus	10	12(1)	Published	High	Good	Often (10)	None	Good	High	Fair	Very High
C. virginica	15	8(++)	Report	Moderate	Poor	Sometimes (15)	None	None	Low	Poor	Moderate

[1]Number of months (relative number of available suppliers, + +for many, + for few, 1 for one)
[2]Standard=Established method by government agency; Published = Peer reviewed publication of method; Report = In gray literature
[3]Low = Similar skills and equipment needed as for acute amphipod test; Moderate = More difficult to obtain acceptable controls, special techniques or more complex exposure system; High=Combination of special skills and more complex exposure system needed
[4]Concordance with acute amphipod test: Good = >75%; Fair = <75% and >50%; Poor <50%
[5]Of the stations found to be toxic by at least one endpoint: Often = >50% of stations; Sometimes = <50% and >20%, Rarely <20%; Never = 0%
[6] Good = CV <50%; Fair = CV >50% and <75%; Poor = CV>75% (CV = coefficient of variation; mean/standard deviation x 100)
[7]Categories based on the range of median acute amphipod standard deviations. High = below range; Similar = within range; Low = above range
[8]Data available for confounding factors: Good=Four or more factors; Fair= 2 or 3 factors; Poor= Less than 2 factors
[9]Low=150% or less the cost of acute amphipod; Moderate = 150% to 200% of amphipod; High = 200% to 300% of amphipod; Very High = >300% of amphipod.

Table 4. Numerically based rating matrix of acute and sublethal sediment toxicity methods. Final score is sum of ratings.

	Feasibility					Performance and Cost							
	Organisms Availability	Method Description	Technical Difficulty	Overall Feasibility		Concordance with Amphipods at Clearly Clean or Impacted Sites	More Sensitive Than Acute *Eohaustorius estuarius* Test	Reproducibility Among Laboratories	Reproducibility Within Laboratories	Relative Precision of Response	Documentation of Confounding Factors	Relative per Station Cost	Total Score
					Factor	2	4	2	2	1	2	2	
Amphipod Acute													
Eohaustorius estuarius	+	+	+	Yes		NA	8	6	6	2	6	6	34
Rhepoxynius abronius	+	+	+	Yes		NA	0	6	6	2	6	6	26
Leptocheirus plumulosus	+	+	+	Yes		NA	12	4	2	2	4	6	30
Ampelisca abdita	+	+	+	Yes		NA	4	2	6	2	4	6	24
Sublethal Methods													
Mercenaria mercenaria growth	+	-	+	No		4	8	4	4	2	6	6	34
Neanthes arenaceodentata survival and growth	+	+	+	Yes		4	8	6	6	1	6	2	33
Sediment-water Interface													
Mytilus galloprovincialis	+	+	+	Yes		4	4	4	6	1	4	6	29
Strongylocentrotus purpuratus	+	+	+	Yes		4	4	0	6	1	6	6	27
L. plumulosus –28-day	+	+	+	Yes		4	8	4	6	1	6	2	31
Amphiascus tenuiremus Life Cycle	-	+	-	No		6	12	0	6	3	4	0	31
Crassostrea virginica lysosomal stability	+	-	-	No		2	8	0	0	1	2	4	17

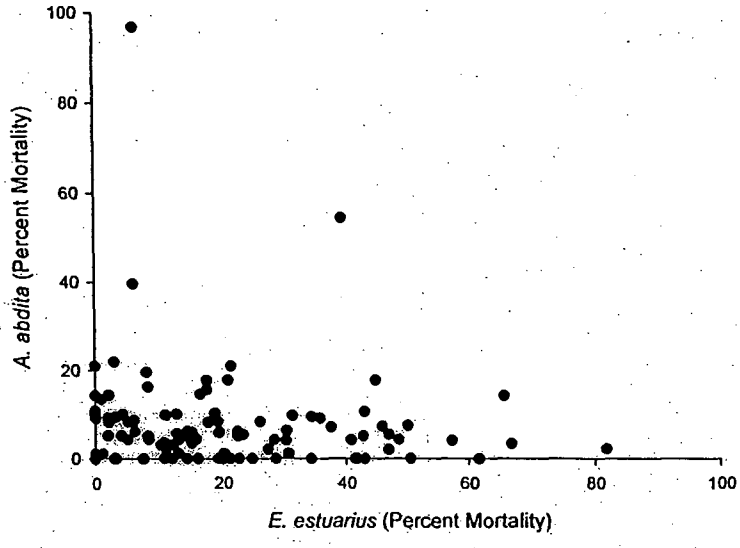


Figure 1. Comparison of mortality data between *Ampelisca abdita* and *Eohaustorius estuarius* on split samples. Data were obtained from multiple regional assessment studies in California.

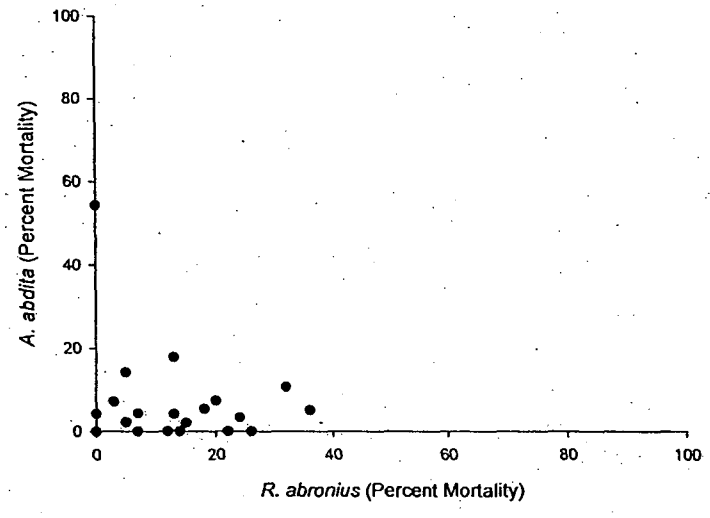


Figure 2. Comparison of mortality data between *Ampelisca abdita* and *Rhepoxynius abronius* on split samples. Data were obtained from multiple regional assessment studies in California.

Sublethal Test Method Evaluation

The candidate sublethal tests were more variable in regards to feasibility, performance, and cost than the acute methods (Table 3). Three of the sublethal test methods had substantial limitations in regard to method documentation, organism availability, or technical difficulty that resulted in an overall rating of not feasible for use in a statewide assessment program at this time (Table 4). These methods were the bivalve *M. mercenaria* growth test, the copepod *Amphiascus tenuiremus* life cycle test, and the lysosomal destabilization test using the oyster *Crassostrea virginica*.

The *M. mercenaria* growth test received the highest total performance and cost score of any of the methods, based on average to slightly above ratings in all of the categories (Tables 2 and 3). However, there is not a single, cohesive document that completely details the protocol and there are no published test acceptability criteria. The test is economical (Table 5) and is not technically difficult to perform. The method exhibited fair reproducibility between laboratories in a round-robin study (Appendix B). In a previous study in the EMAP Carolinian Province, the clam test found no toxicity in reference areas, but did well at identifying areas that were clearly degraded as being toxic; it did better in both these regards than did the *A. abdita* 10-day test (Hyland *et al.* 1998). However, in testing on California sediments the clam test proved to be less sensitive than the *E. estuarius* 10-day test and was one of the least sensitive tests overall (Appendix A). In the Carolinian Province it was found that the *M. mercenaria* test was the best of the toxicity tests conducted at predicting expected bioeffects (Van Dolah *et al.* 1999).

The life cycle test with the copepod *Amphiascus* was by far the most sensitive of the sublethal methods compared to amphipod acute tests (Table 4, Appendix A). This method was also shown to very sensitive compared to an amphipod acute test in a previous study in Florida (Long *et al.* 1999). Nevertheless, the *Amphiascus* test did not pass two of the feasibility criteria. There is no established commercial supplier of the test animals. Only one laboratory in the country maintains a culture of the animals that can be used by other laboratories to start their own cultures. The necessity to culture the animals in individual laboratories leads to the second feasibility limitation, which is technical difficulty. In order to conduct this toxicity test, a laboratory must maintain a copepod culture, and cultures of three algal species used to feed the copepods. In addition, the protocol requires specialized exposure containers and a finely-controlled seawater flow through system. The *Amphiascus* life cycle test is also approximately three times more expensive than other tests (Table 5) and has received no interlaboratory testing to document reproducibility.

Table 5. Per sample cost of performing sediment toxicity tests. Prices are based on quotes from a minimum of three laboratories.

Test	Low Quote ($)	High Quote ($)
Amphipod Acute		
Ampelisca abdita	600	800
Eohaustorius estuarius	600	800
Leptocheirus plumulosus	600	800
Rhepoxynius abronius	600	800
L. plumulosus 28-day Growth and Reproduction	1,375	1,800
Neanthes arenaceodentata 28-day Growth	800	1,400
Sediment-water Interface Embryo Development		
Strongylocentrotus purpuratus	550	1,100
Mytilus galloprovincialis	700	1,200
Mercenaria mercenaria Survival and Growth	600	750
Crassostrea virginica Lysosomal Stability	400	1,500
Amphiascus tenuiremus Life Cycle	2,200	2,800

The oyster lysosomal destabilization test had the lowest total score of any of the test methods (Table 4). Besides the low ranking, this test method does not have a complete protocol that is published (Table 2). In preliminary tests of the procedure, we also found the endpoint determination to be very difficult to discern without significant training from someone very experienced in the procedure, leading to the acceptability failure for technical difficulty. Further, this method has had very limited testing with individual chemicals and until this project, had not been used in field studies along side other test methods. In the testing conducted to date, the oyster lysosomal destabilization test has not been demonstrated to be particularly sensitive compared to acute amphipod tests (Appendix A).

The remaining three sublethal test methods, the *N. arenaceodentata* 28-day growth test, the SWI test with either *M. galloprovincialis* or *Strongylocentrotus purpuratus*, and the *L. plumulosus* 28-day growth and reproduction test, met all of the feasibility criteria. The ranking scores of these tests covered a fairly narrow range of 27 to 33 (Table 4).

The *N. arenaceodentata* growth test received the highest ranking of the remaining sublethal tests (Table 4). It is fairly well established with an ASTM method, although the method documentation is currently under revision to reflect some changes in the procedure. It has been used in multiple field studies and individual chemical exposures to spiked sediments (Dillon *et al.* 1993, Green *et al.* 1999, Lotufo *et al.* 2000, Lotufo *et al.* 2001b, Moore *et al.* 2003, Kennedy *et al.* 2004). The *N. arenaceodentata* 28-day test has also been the subject of considerable refinement efforts considering animal age, test duration and food ration (Bridges and Farrar 1997, Bridges *et al.* 1997). For the methods comparison study using California sediments, the *N. arenaceodentata* test was the second most sensitive test (Appendix A). In that study, the *N. arenaceodentata* test either agreed with the *E. estuarius* test or identified stations as toxic that the *E. estuarius* did not; there were no stations that were found to be toxic by *E. estuarius*, but not *N. arenaceodentata*. When compared to the *L. plumulosus* 10-day results, the *N. arenaceodentata* test was about equal in its ability to detect toxicity, and was second only to the copepod test in sensitivity. While the *N. arenaceodentata* test is one of the more expensive to conduct (Table 5) it has relatively high sensitivity, reliability, and technical feasibility.

The SWI test using mussel embryos received a lower total score than the *N. arenaceodentata* test (Table 4). The SWI test using either developing sea urchin or mussel embryos is an established test method that has been used by multiple laboratories to assess California sediments. The exposure protocol for this procedure is published in a well respected compendium of toxicity test methods (Table 2) and the embryo testing methods are based on standard EPA procedures (USEPA 1995). The protocol has previously been successfully employed in multiple studies within California (Hunt *et al.* 2001, Bay *et al.* 2004, Brown and Bay 2005). The cost of conducting the test is relatively low and the mussels are available in spawning condition year round from multiple suppliers. The test protocol also addresses an important pathway of toxicant effects: exposure of water column organisms to chemicals released from contaminated sediments. The relative sensitivity of this protocol compared to amphipod acute tests is uncertain since the results of side-by-side testing have been mixed. The SWI tests were classified as having relatively low precision (Table 3). This low score reflects increased variability among replicates due to the SWI test design, where the replicates often represent discrete sediment core samples as opposed to replicates of a homogenized sample. Between replicate precision of mussel or sea urchin embryo tests in water only tests is much higher than the SWI results.

The SWI test has been used in the past with both sea urchin and mussel embryos; however, review of the data available for the sea urchin method led to a lower score than for the mussels (Table 4). This low score was due to low interlaboratory reproducibility. Greater technical difficulty is associated with conducting the SWI test with sea urchins. One issue is that sea urchins have a short spawning season in the field and it is cumbersome to extend the spawning season by maintaining the animals in the laboratory. Second, laboratories have reported greater difficulty in recovering the sea urchin embryos at the end of the exposure period. This may be due to a more delicate structure of the sea urchin embryos, which may cause them to stick to the exposure chamber. The reduced embryo recovery success may produce higher between replicate variability for the sea urchins, which may account for the lower sensitivity and reproducibility scores. Compared to sea urchins, *M. galloprovincialis* embryos provide advantages of being available year round in spawning condition and having an endpoint that is easier to measure with precision.

The *L. plumulosus* 28-day test received a relatively high total score that was only two points below the *N. arenaceodentata* test method. This test is both well established and documented (USEPA 2001). The method has been used in multiple field studies and individual chemical exposures to spiked sediments (DeWitt *et al.* 1997, McGee *et al.* 1999, Lotufo *et al.* 2001a, McGee *et al.* 2004). The *L. plumulosus* 28-day test was the third most sensitive of the sublethal methods tested using California sediments (Appendix A). However, there were several California stations where the acute amphipod tests detected toxicity and *L. plumulosus* 28-day did not. Also during this testing, the *L. plumulosus* 28-test experienced a test failure and there were questions regarding the reliability of the reproduction data (Appendix A). Inconsistent reliability of the *L. plumulosus* 28-day test reproductive endpoint has also been reported in another study (Kennedy *et al.* 2004). In a study of sediments in Chesapeake Bay, it was found that the 28-day test did not provide more information regarding toxicity than the 10-day test with the same species and that the 10-day test data had a better correlation with changes in the benthic

community (McGee *et al.* 2004). The *L. plumulosus* 28-day test is the second most expensive test to perform (Table 5).

Discussion

The evaluation of the candidate acute and sublethal tests identified five methods that had the best overall combination of technical feasibility and relatively high performance. These methods include three acute amphipod and two sublethal test methods (Table 6). Each of these methods is well suited for use in a California statewide sediment quality assessment program where feasibility, sensitivity, reliability, and cost are all important factors.

Table 6. Sediment toxicity test methods with the highest overall ranking with respect to the evaluation characteristics.

Species	Taxonomic Group	Matrix	Duration (days)	Endpoint(s)
Acute				
Eohaustorius estuarius *Leptocheirus plumulosus* *Rhepoxynius abronius*	Amphipod	Whole sediment	10	Survival
Sublethal				
Neanthes arenaceodentata	Polychaete	Whole sediment	28	Growth, survival
Mytilus galloprovincialis	Bivalve	Sediment-water interface	2	Embryo development

The two sublethal tests in Table 6 provide important features not present in the suite of amphipod acute tests that are most commonly used to assess sediment quality. The use of a polychaete worm in the *N. arenaceodentata* test provides greater taxonomic diversity among the test organisms and is representative of one the most abundant taxonomic groups comprising the benthic community. The SWI test also represents a different taxon that is also a dominant member of most benthic macrofaunal communities, and the use of an early life-stage may provide enhanced sensitivity to different contaminants. The incorporation a SWI exposure in the *M. galloprovincialis* test also provides a means to evaluate the significance of sediment contaminant impacts on organisms residing in the water column, and thus increases the chance that the testing program will detect toxicity that is present under a diversity of conditions.

Only one of several sediment toxicity methods using the polychaete *N. arenaceodentata* was evaluated in this study. The two methods that are the most established are a 20-day growth test used in the Pacific Northwest (PSWQA 1995), California and many other regions for dredged material characterization, and a 28-day test (ASTM 2002b) that has been optimized to achieve a more sensitive growth endpoint (Bridges *et al.* 1997). The 20-day method has been successfully used in the state of Washington for over 15 years. However, some researchers have found it to be less sensitive than amphipod survival tests (Anderson *et al.* 1998, Pinza *et al.* 2002). In side-by-side testing, one study found the 28-day test to be more sensitive than the 20-day method (Gardiner and Niewolny 1998). Based on the results of these studies, it was decided to focus the evaluation on the 28-day method.

The *L. plumulosus* 28-day test is also a feasible test that had a relatively high total score and could be used in a statewide assessment program. This method was judged to have lower overall suitability because the test is fairly costly to perform, provides no increase in taxonomic diversity, and an uncertain increase in sensitivity relative to the acute amphipod methods already in widespread use.

This study was restricted to toxicity tests where whole sediment samples were included in the exposure. Tests on sediment pore water or elutriate samples were not considered for evaluation because of technical limitations in the methods and a greater uncertainty in the relationship between the test exposure and sediment contaminant concentrations. Pore water tests are a widely used method for testing sediment toxicity (Carr and Nipper 2003), but it is often difficult to collect enough sample for testing. There are other issues associated with pore water toxicity tests that make these methods problematic for use as an initial test of sediment toxicity, including potential changes in metal toxicity due to oxidation, change in sample pH, sorption of contaminants to test chambers, confounding effects of ammonia toxicity, and elimination of sediment ingestion as a route of uptake (Chapman *et al.* 2002, Ho *et al.* 2002). While many of these issues may also be associated with whole sediment tests, they are magnified with the use of pore water.

While elutriate tests are used in several assessment programs, the relationship of the results to direct sediment exposure is not clear. Elutriate tests were developed for testing the effects of the resususpension of the dredged sediment on water column toxicity, not the toxicity of bedded sediment. The proportions of sediment and water and the method of agitation used to prepare the elutriate are operationally defined and the relationship of the resulting exposure experienced by a test organism to that from a whole sediment exposure is unknown. The State of Washington uses a modified elutriate toxicity test method that includes the whole sediment after mixing with the water and tests bivalve or echinoderm larvae (PSWQA 1995, ASTM 2002a). These methods have been used successfully in Washington for over a decade. The Puget Sound method was not included in the present study because of concerns that the organism's response to the whole sediment in the test chamber would be confounded by the presence of the elutriate.

The *A. abdita, M. mercenaria,* and *A. tenuiremus*, tests showed good potential as tests that might be feasible for statewide application in the future. For now, more work needs to be performed on issues regarding animal availability, method development, relative sensitivity, and interlaboratory variation to make these protocols viable choices. Although the oyster lysosome test scored poorly in our ratings, the endpoint represents an important indicator of cellular stress that is responsive to toxicant exposure. The applicability of this method to assess sediment toxicity would be improved through the use of an organism with a greater direct exposure to the sediment, such as a crustacean, polychaete or deposit-feeding bivalve.

The use of multiple toxicity tests is needed to provide a complete and confident evaluation of sediment toxicity. None of the methods identified in Table 6 has been shown to be consistently the most sensitive or reliable test. This situation is to be expected, since there are species-specific variations in contaminant sensitivity and mode of exposure among the test organisms, and many different combinations of chemical type and magnitude may produce sediment toxicity. The use of multiple tests provides two key advantages. First, this approach provides a more reliable assessment of toxicity by reducing the chance that a spurious result in any one test will determine the toxicity classification. The influence of potentially confounding factors such as sediment grain size and organic carbon content are still not entirely known for many tests. Confidence in the results is increased when the results of multiple toxicity tests are similar. Second, the use of multiple test methods increases the sensitivity of the testing program by using

a variety of species, response endpoints, and exposure methods. This combination reduces the chance of a false negative (failure to detect sediment toxicity) due to species-specific variations in contaminant sensitivity or mode of exposure. Multiple toxicity tests were used in NOAA's National Status and Trends Program (Long *et al.* 1996) and are currently used in Washington's Puget Sound Ambient Monitoring Program (Long *et al.* 2005).

TOXICITY RESPONSE THRESHOLDS

Approach

An ordinal scoring system consisting of four categories of response was developed for each of the toxicity tests listed in Table 6. The use of multiple categories, as opposed to a simple binary approach (nontoxic/toxic) retains more information about the toxicity response and thus provides greater potential resolution when combining the toxicity data with other lines of evidence in a sediment quality triad approach. Each category was based on a narrative description of condition that incorporated both the degree of confidence that a toxic effect was present and the magnitude of mean response to the sample.

- **Nontoxic:** Response not substantially different from that expected in sediments that are uncontaminated and have optimum characteristics for the test species
- **Low Toxicity:** A response that is of relatively low magnitude; the response may not be greater than test variability
- **Moderate Toxicity:** High confidence that a statistically significant effect is present
- **High Toxicity:** Highest confidence that a toxic effect is present and the magnitude of response is among the strongest effects observed for the test

This four-category system is an adaptation of the three-category system that is often used to classify sediment toxicity (Long *et al.* 2000), where the test response is classified as nontoxic, marginal, or toxic. The nontoxic and marginal categories correspond to the nontoxic and low toxicity categories of the scoring system used here. The toxic category used in many studies usually represents a reliably statistically significant response that encompasses a wide range of effect (e.g., 20 to 100% mortality) and thus provides little discrimination among the majority of the toxic samples. Two categories of response, moderate and high, were established to represent these toxic samples in order to provide the ability to distinguish severe effects from more moderate responses.

A conceptual approach was developed to relate each of the above categories to a series of numeric thresholds and statistical criteria (Figure 3). This approach relies on the comparison of the test result (e.g., % survival) to Low, Moderate, and High thresholds, corresponding to the upper bound of the response range for the Low Toxicity, Moderate Toxicity, and High Toxicity categories. The thresholds were developed using test-specific characteristics, such as test variability (minimum significant difference (MSD)) and distribution of the toxicity response data. A statistical criterion was also used in the classification scheme (Figure 3). Samples qualifying for the Low or Moderate categories based on test response magnitude were classified into the next lower category if the response was not significantly difference relative to the control (t test, $p \leq 0.05$). A statistical significance criterion was not applied to the highest toxicity category because the derivation of the high toxicity threshold already incorporated a high degree of statistical confidence.

The methodology used to derive the numeric thresholds is described in the following sections.

Low Threshold

The threshold separating the Nontoxic and Low categories was defined as the lowest acceptable control response value for the given test, as established in the test protocols. The response value is defined as the mean value for the endpoint for a given test method (i.e., survival, growth). Any test sample having a response value that is greater (e.g., greater survival) than or equal to the low threshold will be classified as nontoxic, regardless of whether a statistical difference from the control is present. A test response that is less (e.g., lower survival) than the low threshold will be classified as low, moderate, or high, depending on the magnitude of response and statistical significance (Figure 3).

This threshold was based on the rationale that any response that fell within the range expected of animals exposed to optimum sediment conditions (i.e., controls) should indicate a nontoxic condition in the test sample. The control acceptability criteria were obtained from the appropriate protocol for each test method.

Moderate Threshold

The intent of the Moderate Threshold is to distinguish between samples producing a small response of uncertain significance and larger responses representing a reliably significant difference relative to the control. This threshold was based on the Minimum Significant Difference (MSD), which was specific to each test method. The MSD represents the minimum difference between the control and sample mean response that is necessary to be statistically different at $p \leq 0.05$ level. The moderate threshold was equal to the 90th percentile of the MSDs for a given toxicity test method. This approach for calculating a toxicity threshold has been used by other researchers (Phillips *et al.* 2001). Use of the 90th percentile results in a threshold with a high degree of confidence that the sample is different from the nontoxic condition.

The MSD values were calculated using a dataset of replicate control and sample data that were compiled from the SQO database and from laboratories outside of California. Details of this calculation can be found in Phillips *et al.* (2001). An MSD was calculated for each combination of a control and a sample using the following equation:

$$MSD = t_{critical} (s_1^2/n_1 + s_2^2/n_2)^{-½}$$

where $t_{critical}$ = t value from the standard statistical table ($\alpha = 0.05$); s_1^2, s_2^2 = variances for control and field sample; and n_1, n_2 = numbers replicates. All of the MSD values in the dataset for each toxicity test method were then sorted in rank order. The 90th percentile value of this set of data was then calculated (MSD_{90}). The MSD_{90} values were calculated using all available data for each toxicity test method. Finally, the moderate threshold value was calculated by subtracting the MSD_{90} from 100% in order to produce a value that could be compared to the control-adjusted test response value.

Sample response values (i.e., survival or growth) between the low and moderate thresholds are classified as Low Toxicity if they are significantly different from the control response (Figure 3). Sample response values that are less than the moderate threshold and are significantly different from the control are categorized as moderately toxic.

High Threshold

The narrative intent of the High Threshold is to identify samples producing a severe and highly significant effect from those samples producing lesser effects. No precedent for this threshold was available from the literature, so this threshold was based on a combination of test variability and response distribution that corresponded to the category definition.

The 99th percentile MSD value was used to link the high threshold to test variability. A sample having a response that falls below this limit (e.g., lower survival) would be expected to be significantly different from the control 99% of the time. This value therefore represents a response that is associated with a very high level of confidence of statistical significance. The 99th percentile MSD for the high threshold was calculated using the same data and methodology described for the calculation of the MSD_{90} for the moderate threshold.

The response distribution component of the high threshold was based on the distribution of toxic samples from California. For purposes of this calculation, toxic samples were defined as samples having a mean response that was significantly different from the control response. The toxic samples were ranked in descending order based on the control-adjusted mean survival. The response magnitude component of the high threshold corresponded to the 75th percentile of the data. The value obtained from this calculation represents the response associated with the most strongly affected 25% of the toxic samples found in California. It was required that data for this calculation be from stations within California in order to obtain a response value that was relevant to the characteristics of sediments in California.

Both the variability and data distribution response values represented important, but partial, aspects of the High Threshold. Therefore, the mean of the two values was used as the High Threshold. Response values (i.e., survival or growth) below the high threshold are classified as high toxicity regardless of whether they are significantly different from the control response or not (Figure 3).

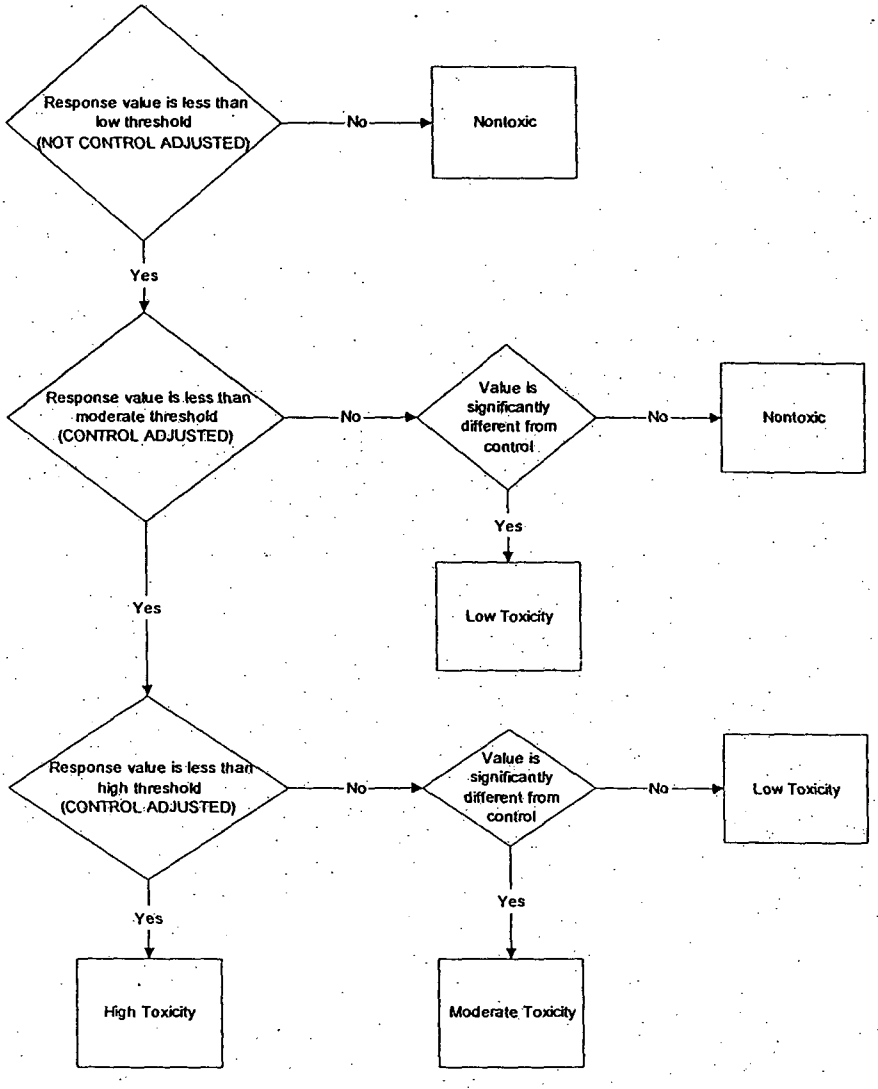


Figure 3. Conceptual approach for assigning the category of toxic effect from exposure response data. The test response value is expressed as survival, embryo development or growth.

Results

Low Threshold

For the amphipod acute survival tests the threshold is 90% survival (USEPA 1994). For the *N. arenaceodentata* growth endpoint, the threshold is 90% of the mean control growth, according to the revised ASTM protocol that is in preparation (J. D. Farrar, personal communication). For the SWI test using *M. galloprovincialis* embryos, the low threshold is 80% normal-alive (not control adjusted). The control criterion for the *M. galloprovincialis* test was established by the Marine Pollution Studies Laboratory, Granite Canyon (B. Phillips, personal communication).

Moderate Threshold

The moderate threshold for the *E. estuarius* 10-day survival test was calculated using data from the California Sediment Quality Objectives database, which included 876 MSD values. The 90th percentile of the MSD values was 18%, which corresponds to a control adjusted survival of 82% (Figure 4).

The *R. abronius* 10-day acute test threshold was also calculated using data from the California database. The dataset included 264 data points (Figure 5). The calculated control adjusted survival threshold for *R. abronius* was 83%, very similar to the *E. estuarius* value.

The threshold for the *L. plumulosus* 10-day survival test was calculated using data from tests on sediment from throughout the U.S. The data were provided by multiple laboratories. Few of the 199 samples in the data set were from stations located in California. The calculated control adjusted survival threshold for the *L. plumulosus* acute test was 78% (Figure 6).

Like the *L. plumulosus* 10-day value, the threshold of the *N. arenaceodentata* growth test was calculated from tests of samples from throughout the United States, with few California stations included. There were less data available for this test method; the calculation was based on 92 data points. The threshold value for the *N. arenaceodentata* growth endpoint was 68% of the mean weight of the control animals (Figure 7).

The threshold for the SWI test with *M. galloprovincialis* embryos was calculated using data from the statewide SQO database. The threshold value of 77% was calculated from 118 MSD values (Figure 8).

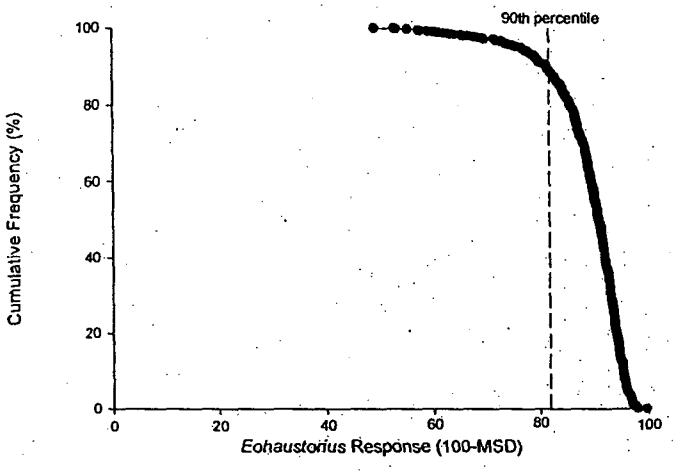


Figure 4. Cumulative frequency of *Eohaustorius estuarius* response (100-MSD) values expressed as a percentage of control survival. The 90th percentile value is the moderate response threshold. Sample size = 876.

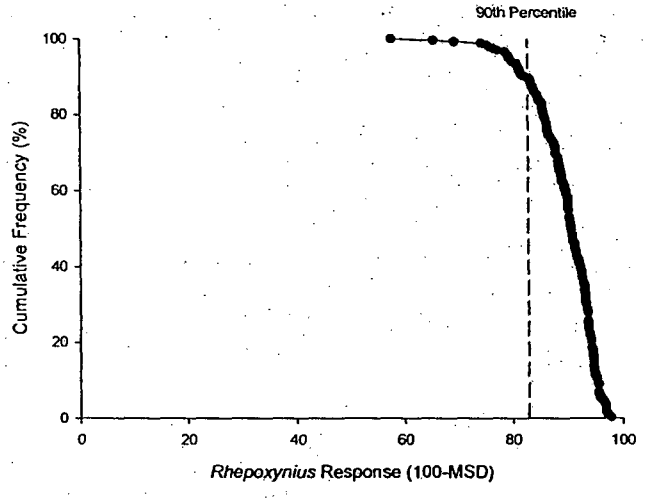


Figure 5. Cumulative frequency of *Rhepoxynius abronius* response (100-MSD) values expressed as a percentage of control survival. The 90th percentile value is the moderate response threshold. Sample size = 264.

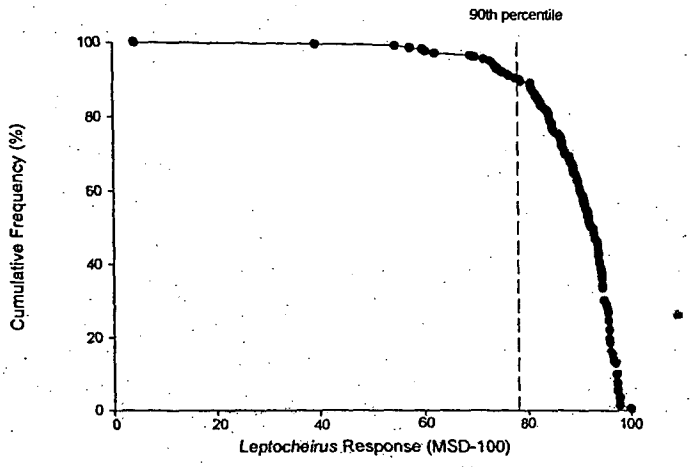


Figure 6. Cumulative frequency of *Leptocheirus plumulosus* response (100-MSD) values expressed as a percentage of control survival. The 90th percentile value is the moderate response threshold. Sample size = 199.

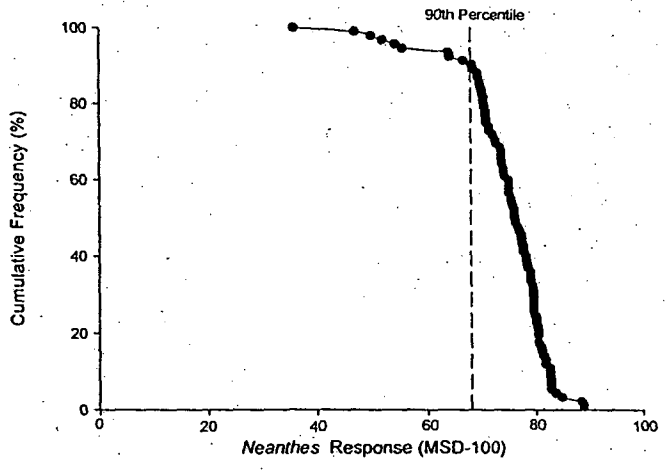


Figure 7. Cumulative frequency of *Neanthes arenaceodentata* growth response (100-MSD) values expressed as a percentage of control growth. The 90th percentile value is the moderate response threshold. Sample size = 92.

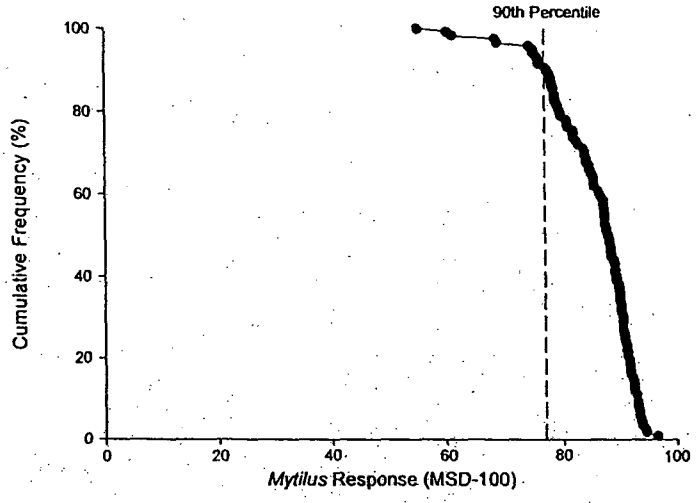


Figure 8. Cumulative frequency of *Mytilus galloprovincialis* sediment-water interface normal-alive response (100-MSD) values expressed as a percentage of response. The 90th percentile value is the moderate response threshold. Sample size = 118.

High Threshold

The species-specific MSD_{99} values were calculated using the same data described for the moderate threshold (Figures 4 through 8). The MSD_{99} values (expressed as the control normalized response) ranged from 46% for *N. arenaceodentata* to 73% for *R. abronius* (Table 7).

The 75th percentile of the toxic *E. estuarius* samples corresponded to a control-adjusted survival of 57% (Figure 9). The 75th percentile value for *R. abronius* was 66% (Figure 10). The data distribution of the toxic *M. galloprovincialis* samples from California produced the lowest 75th percentile value: 24%. This relatively low value may have been related to the small number of toxic samples available for analysis (Figure 11). The toxic data distribution approach could not be used for the *L. plumulosus* and *N. arenaceodentata* tests since most of the samples in the dataset were from outside of California. For *L. plumulosus*, the 75th percentile value of 57% from the *E. estuarius* dataset was substituted for the threshold calculation.

Calculation of the mean of the MSD_{99} and 75th percentile values produced high threshold values ranging from 42% for *Mytilus* to 70% for *R. abronius* (Table 7). This threshold was more variable than the Moderate or Low thresholds, which had ranges of 14% and 10% respectively.

The calculated toxicity test thresholds are summarized in Table 8. For application of the moderate and high thresholds, the data from each exposure must first be normalized to the control response ((sample÷control) x 100). The low threshold is evaluated using the raw data (not normalized), except for the *N. arenaceodentata* 28-day growth endpoint. Normalized data are used for the low, moderate, and *N. arenaceodentata* thresholds because these thresholds are defined relative to the control response, which can vary among tests.

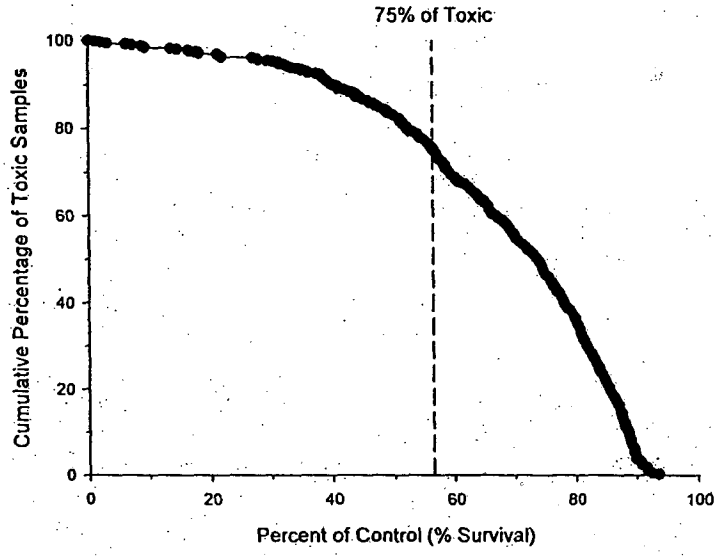


Figure 9. Cumulative frequency distribution plot of *Eohaustorius estuarius* survival data used for 75th percentile of toxic stations calculations. Sample size = 333.

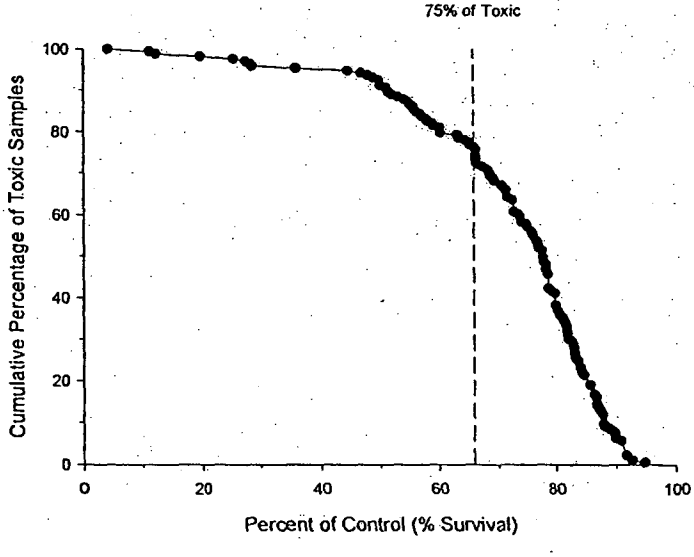


Figure 10. Cumulative frequency distribution plot of *Rhepoxynius abronius* survival data used for 75th percentile of toxic stations calculations. Sample size = 114.

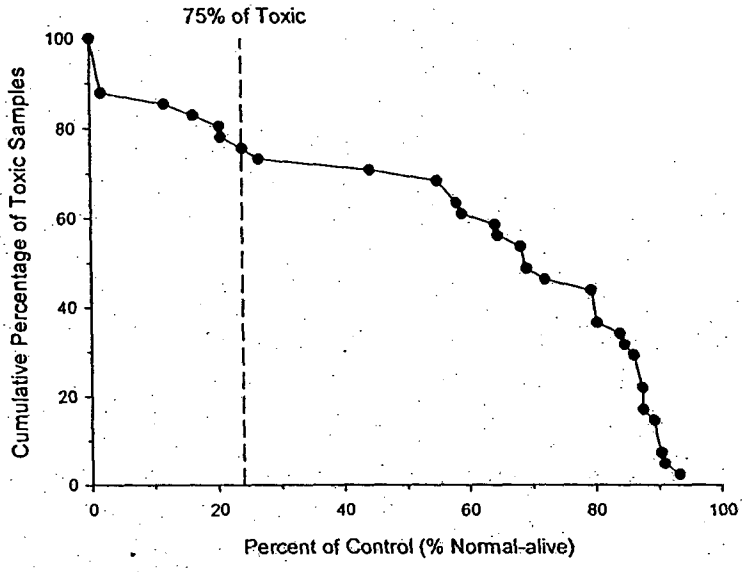


Figure 11. Cumulative frequency distribution plot of sediment-water interface method *Mytilus galloprovincialis* embryo percent normal-alive data used for threshold calculations. Sample size = 28.

Table 7. Data used in calculation of high threshold values for acute and sublethal sediment toxicity test methods. The high threshold is the mean of the two response values shown in the table.

Species	99th MSD	75th of Toxic	High Threshold
Eohaustorius estuarius	61	57	59
Rhepoxynius abronius	73	66	70
Leptocheirus plumulosus	54	57[1]	56
Neanthes arenaceodentata	46	-[2]	46
Mytilus galloprovincialis	60	24	42

[1] No California data available, so *E. estuarius* data was used for this calculation
[2]No California data available

Table 8. Toxicity threshold values for the proposed sediment toxicity test methods.

Species	Low (% Control)	Moderate (% Control)	High (% Control)
Eohaustorius estuarius	90	82	59
Rhepoxynius abronius	90	83	70
L. plumulosus	90	78	56
Neanthes arenaceodentata	90[1]	68	46
Mytilus galloprovincialis	80	77	42

[1] % of control growth.

Discussion

The thresholds derived in this study represent a unique combination of established and new approaches to achieve the goal of being able to classify sediment toxicity into multiple clearly delineated categories. By incorporating both magnitude of response and statistical uncertainty, these categories represent the two factors that are essential to describing a toxicity test response.

Thresholds based on minimum significant difference (MSD_{90}) values have been used by others to establish a threshold representing a test response associated with moderate to strong toxicity (Phillips *et al.* 2001, Field *et al.* 2002). Control acceptability criteria are also frequently used to characterize test responses. This study represents the first known application of the MSD_{99} and 75th percentile of toxic samples for classifying samples in a high toxicity category.

The thresholds developed for this study are similar to comparable thresholds calculated by others. The calculated value of 82% for the *E. estuarius* test is within the range of thresholds of 83% calculated for the Bight'03 regional monitoring project in southern California (Bay *et al.* 2005) and 75% for data from the California Bay Protection and Toxic Cleanup Program (Phillips *et al.* 2001). The moderate threshold of 77% for the SWI test with *M. galloprovincialis* is similar to the MSD value of 80% reported by Phillips *et al.* (2001) for a larger dataset for *M. galloprovincialis* that included pore water and water column data.

The *M. galloprovincialis* SWI test low and moderate thresholds appear to represent a very narrow range of response (Table 8). This response window is not as small as it first seems because the low and moderate thresholds are expressed differently. The low threshold value is not control adjusted while the moderate threshold is adjusted. The average control value for *M. galloprovincialis* SWI tests in the statewide database is 85% normal-alive. Therefore, the control-adjusted value of 77% for the moderate threshold represents a noncontrol-adjusted value of 65% (77% x 85% = 65%), representing a response window of about 15% for the low toxicity category.

Little data from California stations was available to calculate the MSD for the *L. plumulosus* and *N. arenaceodentata* test methods. This is of little concern since the MSD is a measurement of the inherent variability of the test method and should not be affected to a great extent by sample source. However, as more data becomes available the MSD should be recalculated to provide a more confident value. The thresholds for the SWI test with *M. galloprovincialis* should also be recalculated when more data become available, since the number of data points was limited in comparison to the *E. estuarius* and *R. abronius* datasets.

The greatest amount of uncertainty is associated with the high threshold values. The approach used to calculate these values is new so there is no basis of comparison to help identify spurious values. In addition, this threshold is based on the analysis of extreme portions of data distributions (99th and 75th percentiles), which are more sensitive to data quantity and may be more variable. Confidence in the high threshold values would be improved by the availability of more data collected on samples from within California. For the calculation of the 75th percentile of toxic stations, it is vital that the data is generated using California samples so that future comparison of samples from within the State will be evaluated in the correct context.

Currently, there is a very limited amount of California data for the *L. plumulosus* 10-day and *N. arenaceodentata* growth tests.

RESEARCH NEEDS

The analyses described in this report were used to select a suite of test methods for use in sediment toxicity testing. These represent a minimum suite of test methods that had the best available combination of feasibility and performance. Several data limitations were encountered in the course of this study that either restricted the suite of suitable test methods or complicated the calculation of the classification thresholds. The following research activities are needed to improve the use of toxicity tests for evaluating sediment quality:

- **Refine thresholds for the *L. plumulosus* and *N. arenaceodentata* tests as new data become available.** Limited data were available to calculate the toxicity thresholds for these species. More toxicity data from California samples are needed to refine calculation of the 75th percentile of toxic stations, which would improve confidence in the calculation of the high toxicity threshold values.

- **Evaluate additional sublethal test methods for inclusion in the suite of recommended test methods.** A wider variety of sublethal test methods that are feasible and sensitive should be available. Use of a wider variety of toxicity tests would help ensure that the toxicity information addresses variations in routes of exposure and sensitivity to sediment contaminants among the sediment-dwelling organisms. Some of the methods evaluated in the current study showed promise for future use, but were lacking in protocol development, had little field testing, and had not been compared in sensitivity to more established methods. Research is needed to fully document these tests and develop quality assurance criteria, such as required pH, salinity and temperature ranges. Research should be conducted to field test any additional methods side by side with the methods already evaluated in this document in order to evaluate relative sensitivity and produce the data needed for threshold development.

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APPENDIX A

Comparison of Methods for Evaluating Acute and Chronic Toxicity in Marine Sediments

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ABSTRACT

Sublethal test methods are being used with increasing frequency to measure sediment toxicity, but little is known about the relative sensitivity of these tests compared to the more commonly used acute tests. A study was conducted to compare the sensitivity of several acute and sublethal toxicity methods, and investigate their correlations with sediment chemistry and benthic community condition. Six sublethal methods (amphipod, *Leptocheirus plumulosus* 28-day survival, growth and reproduction; polychaete, *Neanthes arenaceodentata* 28-day survival and growth; benthic copepod, *Amphiascus tenuiremis*, 14-day life cycle; seed clam, *Mercenaria mercenaria* 7-day growth; oyster, *Crassostrea virginica* lysosome destabilization; and sediment-water interface (SWI) testing with embryos of the mussel *Mytilus galloprovincialis*) and two acute methods (10-day amphipod survival with *Eohaustorius estuarius* and *Leptocheirus plumulosus*) were used to test split samples of sediment from stations in southern California and San Francisco Bay. The most sensitive sublethal test, and most sensitive overall, was the life cycle test with the copepod, *Amphiascus*. The *L. plumulosus* 10-day survival test was the most sensitive of the acute tests. The sublethal tests were not, in general, more sensitive to the sediments than the acute tests. Of the sublethal tests only the *A. tenuiremus* endpoints and polychaete growth correlated with sediment chemistry. There was poor correspondence between the toxicity endpoints and indicators of benthic community condition. Differences in test characteristics such as mode of exposure, species-specific contaminant sensitivity, changes in contaminant bioavailability, and the influence of noncontaminant stressors on the benthos may have been responsible for the variations in response among the tests and low correspondence with benthic community condition. The influence of these factors cannot be easily predicted and underscores the need to use multiple toxicity methods in combination with other lines of evidence to provide an accurate and confident assessment of sediment toxicity.

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INTRODUCTION

Acute sediment toxicity testing has been routinely conducted as part of monitoring and assessment programs, such as the USEPA's Environmental Monitoring and Assessment Program (Strobel *et al.* 1995). The toxicity tests are usually conducted on whole sediments using amphipod 10-day survival tests in accordance with standard protocols (USEPA 1994). Sublethal testing has been conducted on a much more limited basis, but there is increased interest in using sublethal methods due to the assumption that they are more sensitive to contaminated sediments than the acute methods (Adams *et al.* 2005). Sublethal methods include embryo development tests and other tests with various life stages of animals having endpoints such as growth and reproduction in addition to survival. A wide variety of sublethal methods have been described (Lamberson *et al.* 1992), but only a few such methods have been used commonly; they include the amphipod *Leptocheirus plumulosus* 28-day growth and reproduction test (USEPA 2001), a 20-day polychaete growth test using *Neanthes arenaceodentata* (PSWQA 1995), pore water testing using echinoderm gametes or embryos (Carr and Nipper 2003) and a SWI test using sea urchin or mussel embryos (Anderson *et al.* 1996). Additional promising sublethal tests that have been developed recently and include the measurement of copepod reproduction (Chandler and Green 1996), juvenile clam growth (Ringwood and Keppler 1998), and oyster biomarker responses (Ringwood *et al.* 1998).

Because sublethal toxicity methods have been used less commonly, there are questions regarding whether these test methods are practical, reproducible, and more sensitive than the acute methods already in use (Anderson *et al.* 1998, Pinza *et al.* 2002). Few studies have been conducted that were designed specifically to compare the relative attributes of various sublethal tests. Studies conducted to date have only compared two or three methods together (DeWitt *et al.* 1997, Anderson *et al.* 1998, Green *et al.* 1999), or have focused more on sublethal elutriate or pore water tests rather than whole sediment tests (Long *et al.* 1990). Important factors to consider in the selection and interpretation of toxicity tests include the degree of exposure to whole sediment, the relative sensitivity to sediment contaminants, and the level of concordance with benthic community impacts. Information on these factors is extremely limited for many sublethal tests.

This study was designed to investigate relative performance of several acute and sublethal test methods with whole sediments. Three specific points were examined. First, the relative sensitivity of the toxicity test methods was compared. Sensitivity was defined as the relative ability of a test method to detect toxicity in a sample. Sensitivity comparisons were made both between acute and sublethal methods and among the sublethal methods. Secondly, the relationship between sediment chemical concentrations and toxicity of each method was examined. Finally, this study investigated the relationship between changes in benthic community condition and toxicity.

METHODS

Six candidate whole sediment sublethal methods were selected (Table 1). These methods appeared to be technically feasible and had data available that indicated some level of sensitivity to contaminated sediments. Methods were first selected that had established, published methods by a government or scientific agency (e.g., USEPA methods, ASTM methods). Additional methods were selected from the scientific literature and from recommendations by toxicologists with experience in sediment quality assessment. Acute amphipod testing was also conducted for comparison with sublethal methods using two species, *E. estuarius* and *L. plumulosus*.

Table 1. Characteristics of the sublethal sediment toxicity methods included in the comparison study. Duration given in days.

Species	Taxon	Test endpoint(s)	Duration
Mytilus galloprovincialis	mussel	sediment-water interface, embryo development	2
Mercenaria mercenaria	clam	growth	7
Crassostrea virginica	oyster	lysosomal destabilization	4
Leptocheirus plumulosus	amphipod	growth, reproduction, survival*	28
Neanthes arenaceodentata	polychaete	growth, survival*	28
Amphiascus tenuiremis	benthic copepod	reproduction, survival*	14

* Secondary endpoint

The sediment samples that were tested were collected as part of two regional monitoring surveys, Southern California Bight 2003 Regional Monitoring Program (Figure 1) and the San Francisco Estuary Institute Regional Monitoring Program (RMP; Figure 2). The stations represented a wide range of expected contamination levels and habitat types with the aim being to target stations expected to have a low to moderate level of acute toxicity. Stations expected to have a high degree of acute toxicity were not included in the study because they would be less effective in eliciting different sublethal responses among the tests. The stations from southern California were selected to include a range of geographical location, proximity to sources of contamination, and expected sediment grain size. The RMP sites have been monitored for about 10 years and were chosen based on their wide geographic distribution and a range of acute toxicity to amphipods.

Tests on split samples were conducted by laboratories with extensive experience using the various tests. The *L. plumulosus* and *N. arenaceodentata* testing was conducted at the Army Corps of Engineers, Research and Development Center, Environmental Laboratory in Vicksburg, MS. The *A. tenuiremis* assays were performed at the University of South Carolina in Columbia, SC. The *M. mercenaria* growth test and *C. virginica* lysosomal destabilization procedures were done at the South Carolina Department of Natural Resources, Marine Resources Research Institute in Charleston, SC. The SWI testing was conducted at the University of California, Davis, Marine Pollution Studies Laboratory in Carmel, CA. Ten-day *E. estuarius* acute survival tests were performed on sediment from each station. These acute tests were performed by multiple laboratories, as part of the regional monitoring efforts. The laboratories that performed the *E. estuarius* tests on southern California stations participated in intercalibration procedures, which showed

reasonable agreement between laboratories (Bay *et al.* 2005). The laboratory testing the San Francisco Bay stations did not participate in this intercalibration. A summary of the characteristics of all of these test methods can be found in Bay *et al.* (2007). Samples were also analyzed for organic and metals chemistry, total organic carbon (TOC), grain size and benthic infauna.

Sediments were collected in July through August 2003. A Van Veen grab was used to collect whole sediment from the surface (top 2 cm) and subcores. Surface sediment was obtained from multiple grabs at each site, composited, transferred to plastic containers, and stored at 5°C. Sediment-water interface subcores were also collected from the Van Veen grab by inserting a polycarbonate core tube into the sediment to a depth of 5 cm and capping the bottom and top of the tube. All sediment samples were transported to Southern California Coastal Water Research Project (SCCWRP) within 24 hours of collection. The core samples were then transported with ice packs to the testing laboratory within 24 hours. Core samples from the San Francisco Bay stations were transported directly to the testing laboratory.

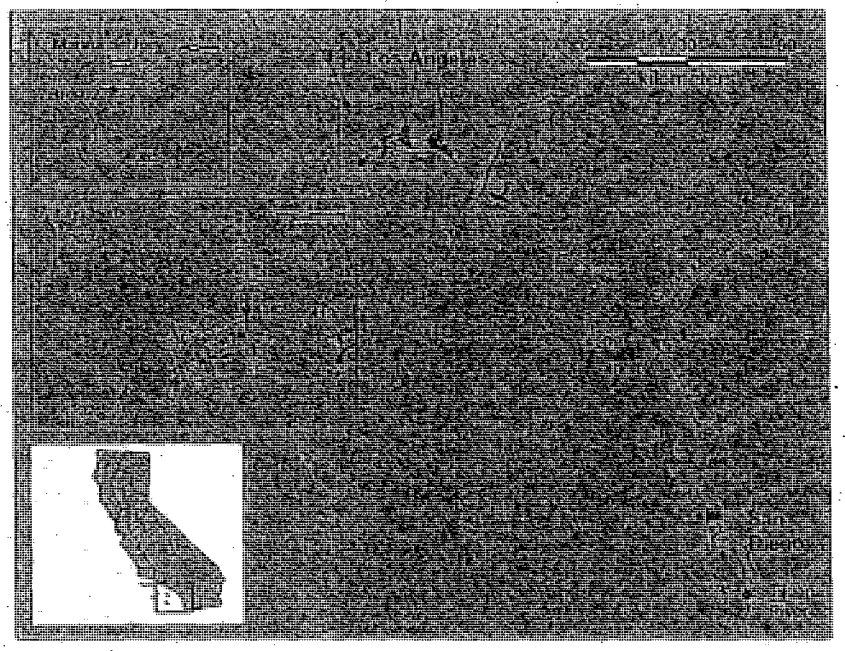

Figure 1. Location of southern California stations used for the sediment toxicity methods comparison study.

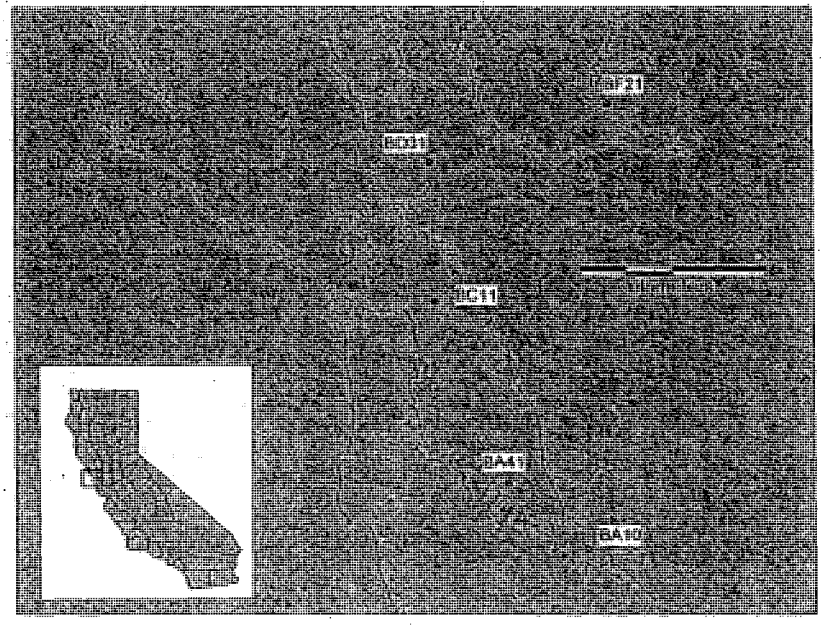

Figure 2. Location of San Francisco Bay stations used for the sediment toxicity methods comparison study.

The subcores were shipped to the testing laboratory within 48 hours of collection and the SWI tests were initiated within 10 days of collection (Table 2). The whole sediment samples were shipped to the testing labs in two batches, one with six of the southern California stations, the other with the remaining four southern California and all five San Francisco stations. Before shipment of each batch, all of the sediment from each station was placed in a large polycarbonate bowl and homogenized with a polycarbonate spoon. Samples for each laboratory were then aliquoted into polyethylene containers and shipped overnight with sufficient quantities of ice packs to maintain temperature at 5°C. Holding time between collection and testing of the composites varied from 6 to 116 days (Table 2).

Table 2. Holding times (number of days) for sediment samples tested with acute and sublethal toxicity methods. *Eohaustorius estuarius* (*Eohaustorius*), *Leptocheirus plumulosus* (*Leptocheirus*), Sediment-water Interface (SWI), *Merceneria mercenaria* (*Mercenaria*), *Crassostrea virginica* (Lysosome), *Neanthes arenaceodentata* (*Neanthes*), and *Amphiascus tenuiremus* (*Amphiascus*).

		Leptocheirus						
Station	*Eohaustorius*	10-Day	28-Day	SWI	*Mercenaria*	Lysosome	*Neanthes*	*Amphiascus*
Batch 1								
4066	27	26	116	6	13	26	32	-
4130	26	26	116	6	13	26	32	12
4142	27	26	116	6	13	29	32	-
4008	11	22	112	10	9	25	28	8
4209	11	22	112	10	9	22	28	8
4695	10	21	111	9	8	24	27	7
Batch 2								
4202	13	41	90	6	21	37	58	19
4262	12	40	89	5	20	36	57	18
BRI-02	14	28	77	1	8	24	45	6
4085	7	28	77	1	8	24	45	6
BA10	8	36	85	1	16	32	53	-
BA41	11	39	88	4	19	35	56	17
BC11	13	41	90	6	21	34	58	19
BD31	13	41	90	6	21	34	58	-
BF21	15	43	92	8	23	36	60	-

Toxicity Testing

Eohaustorius estuarius 10-day survival

Ten day survival tests with *E. estuarius* were conducted using standard USEPA testing procedures (1994). Sediment samples were pre-sieved through a 2-mm mesh screen and homogenized in the laboratory before testing. Sediment was placed in 1-L glass jars to a depth of 2 cm. The samples were aerated and allowed to equilibrate overnight before addition of 20 adult amphipods to each of five replicates. All of the laboratories obtained the amphipods from Northwestern Aquatic Sciences (Yaquina Bay, OR). The exposures took place at 15°C, at a salinity of 20 g/kg with constant lighting. The animals were not fed and the water was not renewed during the exposures. At the end of the exposure, the sediment from each jar was sieved and the surviving animals were counted and recorded. Water quality measurements (dissolved oxygen, pH, salinity and overlying water ammonia) were determined at day 0 and prior to test termination.

Leptocheirus plumulosus 10-day survival

The experimental design followed guidelines set forth by the USEPA (1994). Sediment was added to each of 5 replicate 1-L beakers to obtain a 2 cm depth. Sediment was then overlain with 20 g/kg synthetic seawater. Temperature was maintained at 25°C with constant illumination and the beakers were aerated during the exposure. At day 0, 20 *L. plumulosus* (500- to 750-µm sieve size class) obtained from in-house cultures were gently transferred to each replicate beaker. The animals were not fed and the water was not renewed during the exposures. Water quality measurements (dissolved oxygen, pH, salinity and overlying water ammonia) were determined at day 0 and prior to test termination. On day 10, the sediment in each beaker was sieved and the surviving animals recovered. The number of surviving organisms was counted and recorded.

Leptocheirus plumulosus 28-day survival, growth and reproduction

The *L. plumulosus* 28-day experiments were conducted following the guidelines provided by the USEPA (2001). Due to conflicts in the laboratory schedule and a test failure, the samples for this test method were held for a much longer period than the other test methods (Table 2). Sediment was added to 5 replicate 1-L beakers to obtain the required depth of 2 cm. Sediment was then overlain with 20 g/kg synthetic seawater and gently aerated. Temperature was maintained at 25°C and the light cycle was set at 16:8 h light:dark. At day 0, *L. plumulosus* (250- to 600-µm sieve size class) were obtained from in-house cultures. Twenty animals were transferred to each replicate beaker. Water quality measurements (dissolved oxygen, pH, salinity and overlying water ammonia) were determined at day 0, prior to test termination and in one replicate per sediment three times per week. Water was changed in each beaker after water quality parameters were measured. Each beaker was provided with 20 mg of Tetramin® three times per week for the first two weeks and 40 mg per beaker the final two weeks of testing. On day 28, the sediment in each beaker was sieved and surviving animals were recovered. Surviving adults and neonates were counted and recorded. The surviving adults from each replicate were placed on a tared pan and dried at 60°C for 24 hours. The pans were then removed, allowed to cool and weighed to obtain total dry-weight for each replicate. The reproductive endpoint had an acceptability criteria failure in batch and an abnormal response in another. It was therefore decided that the reproductive data would not be used for analysis.

Neanthes arenaceodentata 28-day survival and growth

The 28-d *N. arenaceodentata* experiments were conducted following guidelines developed by the US Army ERDC (Bridges and Farrar 1997, Bridges *et al.* 1997). Sediment was added to 10 replicate 300 ml tall-form beakers to obtain the required depth of 2 cm. Sediment was then overlain with 30 g/kg synthetic seawater and gently aerated. Temperature was maintained at 20°C and light cycle was set at 12:12 hour light:dark. Organisms were obtained from Dr. Don Reish (California State University, Long Beach, CA). On day 0, one *N. arenaceodentata* (≤7 days old) was gently transferred to each replicate beaker. Water quality measurements (dissolved oxygen, pH, salinity and overlying water ammonia) were determined at day 0, prior to test termination, and in three replicates per sample weekly. Water was changed in each beaker once per week after water quality parameters were measured. Each beaker was provided 2 mg of

Tetramarin® once per week and 2 mg of Tetramarin® plus 2 mg of Alfalfa once per week. On day 28, the sediment contained in each beaker was sieved and surviving worms were recovered, counted and recorded. Surviving animals from each replicate were put on a pre-weighed pan and placed in a drying oven at 60°C for 24 hours. The pans were then removed, allowed to cool, and weighed to obtain the individual dry weight for each replicate/animal.

Amphiascus tenuiremis 14-day life cycle

Testing of the copepods followed the methods of Chandler and Green (1996). A sediment reference sample was collected from Oyster Landing at North Inlet, SC. Stations BA41, BC11, BRI2, 4085, 4202, 4262, were press-sieved through a 125 µm sieve in order to facilitate recovery of the animals at the conclusion of the exposure. A larger sieve size was used for some of the larger grained stations in order to obtain a sufficient volume of sediment for testing. Sediment samples 4008 and 4695 were screened with a 250 µm sieve while 4209 and 4130 were sieved through a 212 and 180 µm sieve, respectively. Sediment samples 4066 and 4142 were too sandy to pass a 250 µm sieve, and could not be tested with the copepod method. A total of ten stations were tested with *Amphiascus*. Teflon 50-ml Erlenmeyer flasks with mesh-covered outflow holes were filled with 0.45-µm filtered, aerated seawater. Press-sieved sediment samples were then packed into Teflon syringes and slowly extruded onto the bottoms of their respective chambers (4 replicates per sediment sample). Adult non-gravid female and adult male copepods (*Amphiascus tenuiremis*) were then counted into each quadruplicate test chamber (25/sex). Chambers were placed in an incubator at 20°C under continuous dripping flow for 14 days with a 12:12 hour light:dark cycle. Chambers were fed every third day a mixture of frozen algal stock (10^7 cells of 1:1:1 *Isochrysis galbana, Phaeodactylum tricornutum* and *Dunaliella tertiolecta*). Water quality parameters (dissolved oxygen, pH, and salinity) were measured every third day. Overlying water ammonia was measured once, at the end of each exposure period. At the end of 14 days of exposure, copepods were collected on a 63-µm sieve. Samples were checked/counted for dead bodies. Copepods were stained with Rose Bengal and preserved in 5% borate-buffered formalin. Non-gravid adult females, gravid adult females, adult males, copepodites, nauplii, and clutch sizes were enumerated under a Nikon SMZ-U stereo dissection microscope. Two endpoints of the *A. tenuiremus* test were calculated: the number of copepodites produced and the realized offspring production (output of new animals normalized to the number of females surviving at the end of the test).

Mercenaria mercenaria 7-day growth

The clam tests measured growth during a 7-day exposure to whole sediment (Ringwood and Keppler 1998, Keppler and Ringwood 2002). Sediment samples were pressed through a 500-µm sieve, homogenized, and 50-ml aliquots were placed into four replicate 250-ml beakers. The sediment was then overlain with clean 25 g/kg seawater. The replicates were gently aerated for the duration of the experiment, and the assays were conducted at room temperature (22 to 25°C) for 7 days with a 16:8 light cycle. Juvenile clams (*M. mercenaria*) used for all experiments were obtained from Atlantic Littleneck Clam Farm, Charleston, SC. Clams were sieved through two mesh sizes (1.0 mm and 1.2 mm) to ensure that the clams were of a similar size range. Twenty-five clams were used

for each replicate. Pre-assay wet weights of each clam group were taken for growth rate estimates, and to ensure that all replicate groups had similar initial weights. Replicate subsets of clams were also counted, wet weighed, dried overnight and reweighed to verify the wet:dry weight ratio used to estimate initial dry weights. Each replicate was fed on the first, third and sixth days of the assay (50:50 mix of *I. galbana* and *Chaetoceros gracilis*: 20 x 10^6 cells / replicate). The overlying water was not renewed during the exposure. At the end of the exposures, clams were sieved from the sediments and placed in fresh 25 g/kg seawater for approximately 2 hours to depurate. Dead clams were counted and removed, and percent mortalities were calculated. The surviving clams were counted and rinsed with distilled water to remove excess salts. Post-assay wet weights were determined, and clams were then dried for 48 hours (at 70°C). Each clam replicate was recounted and final dry weight per clam was determined. Initial dry weights were subtracted from the final dry weights, and the results were expressed as growth rates (µg/clam/day). Sediment pore water chemistry parameters (salinity, pH, and total ammonia – nitrogen [TAN]) were measured for each sediment sample prior to use in any assay. Overlying water quality was also measured.

Crassostrea virginica 4-day lysosomal destabilization

The lysosomal destabilization assay was conducted following the methods described in Ringwood et al. (1998). Sediment samples were homogenized and 100-ml aliquots were placed into three replicate 1L beakers. The sediment was topped with clean 25 g/kg seawater. The beakers were allowed to settle for 2 hours, and then 3 clean-scrubbed oysters were gently added to each replicate. Oysters (5.3 $\pm$0.7 cm) used for laboratory sediment exposures were collected from control sites and acclimated to laboratory conditions for at least 24 hours prior to the start of the experiment. The replicates were gently aerated for the duration of the experiment, and the assays were conducted at room temperature (22 to 25°C) for 4 days with a 16:8 light cycle. Each replicate was fed on the first and third days of the assay (algal paste mixed into filtered sea water, 70 x 10^6 cells / replicate). The overlying water was not renewed during the exposure. Water quality parameters for both the pore and overlying waters were measured in the same way as for the *M. mercenaria* testing. Digestive gland tissue from the exposed oysters was diced and treated with trypsin to produce a cell suspension. A cell suspension aliquot was mixed with an equal aliquot of neutral red (NR) solution, placed on a microscope slide and examined under a light microscope to evaluate NR retention by digestive gland cells containing lysosomes. At least 50 cells were scored as stable (NR retention in the lysosomes) or destabilized (NR leaking into the cytoplasm), and the data were expressed as the percentage of cells with destabilized lysosomes per oyster.

Mytilus galloprovincialis 2-day embryo development at the sediment-water interface

Exposure procedures followed those detailed by Anderson *et al.* (1996). One day prior to the start of the test, 300 ml of clean seawater (1-µm filtered, approximately 34 g/kg) was added over the sediment to each of five replicate core tubes. Samples were then aerated overnight to equilibrate. On test day 0, water quality samples were collected from the core tubes and tubes containing a 25-µm screen were placed on the sediment surface. The screen was approximately 1 cm above the sediment. Mussel embryos were unavailable to test stations 4008, 4209, and 4695, so sea urchin embryos were used

instead. Embryos were prepared following USEPA protocols (USEPA 1995) and added to the screen tubes. Mussels were exposed for 48 hours and sea urchins for 96 hours. Exposures were carried out at 15°C with gentle aeration. Water quality parameters of dissolved oxygen, total ammonia, pH, and salinity were measured at the beginning and end of the exposure period. Temperature was measured continuously. The exposures were terminated by removing the screen tube, rinsing the embryos into a vial, and adding formalin to fix and preserve embryos. The samples were then examined microscopically for normal embryo development. Data were expressed as percentage normal-alive. This endpoint is calculated by dividing the number of normal embryos by initial number of embryos inoculated into the chambers.

Chemical Analysis

Sediment samples were analyzed for a suite of parameters that included metals, organics, grain size and TOC. Analyses were conducted by a variety of laboratories participating in the regional monitoring programs and used standardized EPA recommended methods (Bight'03 Coastal Ecology Committee 2003, SFEI 2005). The laboratories had achieved acceptable comparability during pre-project intercalibration exercises and the data were subjected to rigorous post survey review. Quality assurance samples were included in each sample batch and included method blanks, duplicates, matrix spikes, and a certified reference material. Sediment particle size was measured by light-scattering technology using either a Coulter LS230 or a Horiba LA900 instrument. The sediment samples analyzed for all metal analytes except mercury were digested in strong acid according to the procedures described in EPA Method 3050B. Metals were quantified using either inductively coupled plasma mass spectrometry, inductively coupled plasma emission spectroscopy, flame atomic absorption, or graphite furnace atomic absorption. Mercury was analyzed using cold vapor atomic absorption spectroscopy. Samples for organic chemistry analysis were solvent extracted using accelerated solvent extraction, sohxlet, or roller table. The extracts obtained were subjected to each laboratory's own clean-up procedures and were analyzed by gas chromatographic method (e.g., dual-column GC-ECD or GC-MS in the selected ion monitoring mode).

Benthic Community Analysis

A separate grab sample was taken for benthic community analysis at all the stations. The contents of the grab were washed through a 1.0-mm screen and all of the retained animals identified to species or the lowest possible taxon. Different benthic indices were used to assess community status for the San Francisco Bay and southern California stations because of habitat differences between the two regions that affected species composition. The benthic community condition of the southern California stations was assessed using the Benthic Response Index (BRI; Ranasinghe *et al.* 2003). The BRI is the abundance-weighted average pollution tolerance score of organisms occurring in a sample. The Index of Biotic Integrity (IBI) was used to determine benthic community condition for the San Francisco Bay stations (Thompson and Lowe 2004). The IBI uses a multimetric index to discriminate between impacted and reference areas.

Data Analysis

Toxicity data were control normalized ((station value/control) x 100) to facilitate comparisons among the test methods. Statistical significance was tested using Student's t-test ($p \leq 0.05$) assuming unequal variance (Zar 1999). For sublethal methods having more than one endpoint, if either or both endpoints were significantly different from control, the station was designated as toxic.

The mean ERM quotient (ERMq; Long *et al.* 1998) was calculated for each station to integrate a subset of the analyzed chemicals into a value that is predictive of toxic effects. The ERM for DDT was not used in calculations because it has been found to be unreliable (Long *et al.* 1995). Relationships between sediment chemistry parameters or benthic community condition and toxicity response were analyzed using a non-parametric Spearman's rank correlation.

RESULTS

The experimental batches for all toxicity data that is presented passed test control acceptability criteria, except for one SWI batch with *M. galloprovincialis*. That batch contained the only sample with a significant toxic response for the SWI test and had a low control normal-alive percentage. Because the difference between the control and sample response was very large, we have chosen to include the data.

There were two quality assurance issues with the *L. plumulosus* 28-day test. First, there was a test failure based on insufficient reproduction in the controls. When the test was repeated, the controls reproduced sufficiently, but all of the other samples had greatly less reproduction than the controls. This situation had never been encountered by the testing laboratory and led to our decision to not use the reproduction data for analysis. The second issue with this test was the very long holding time of the sediments before testing began compared to the other methods (Table 2). The effects of this prolonged holding time are unknown. The data are presented for the purposes of comparison, but may have differed had the holding times been identical between methods.

Water quality measurements made during testing indicated that the values were within acceptable range for the majority of sample/test combinations. For the *M. mercenaria* test, station 4130 exhibited elevated pore water ammonia (37.5 mg/L total ammonia-nitrogen). While the tolerance of *M. mercenaria* to ammonia is not known, there is correlative evidence that the ammonia level in the sample may have been the cause of toxicity. For the SWI test, station BC11 had an overlying water ammonia concentration of 0.145 mg/L un-ionized ammonia, which is very near the EC50 (approximately 0.17 mg/L, unpublished data).

Comparisons Among Sublethal Tests

There was a wide range in the percentage of stations that each of the sublethal methods identified as toxic (Figure 3). The highest percentage was for the copepod, *Amphiascus* that found 9 out of the 10 stations tested to be toxic, followed by *N. arenaceodentata* with 8 out of 15 stations. The proportion of stations identified as toxic was much lower for the remaining test methods, with the lowest percentage for the SWI testing which identified 1 out of 15 stations as toxic.

Comparisons Between Acute Tests

The *E. estuarius* method was the less sensitive of the two amphipod acute protocols, identifying 4 out of 15 stations as toxic (Figure 4). Overall the *E. estuarius* method was near the mid-point in sensitivity relative to the sublethal tests. The *L. plumulosus* 10-day method identified 9 out of the 15 sites as toxic and was more sensitive than all but one of the sublethal methods.

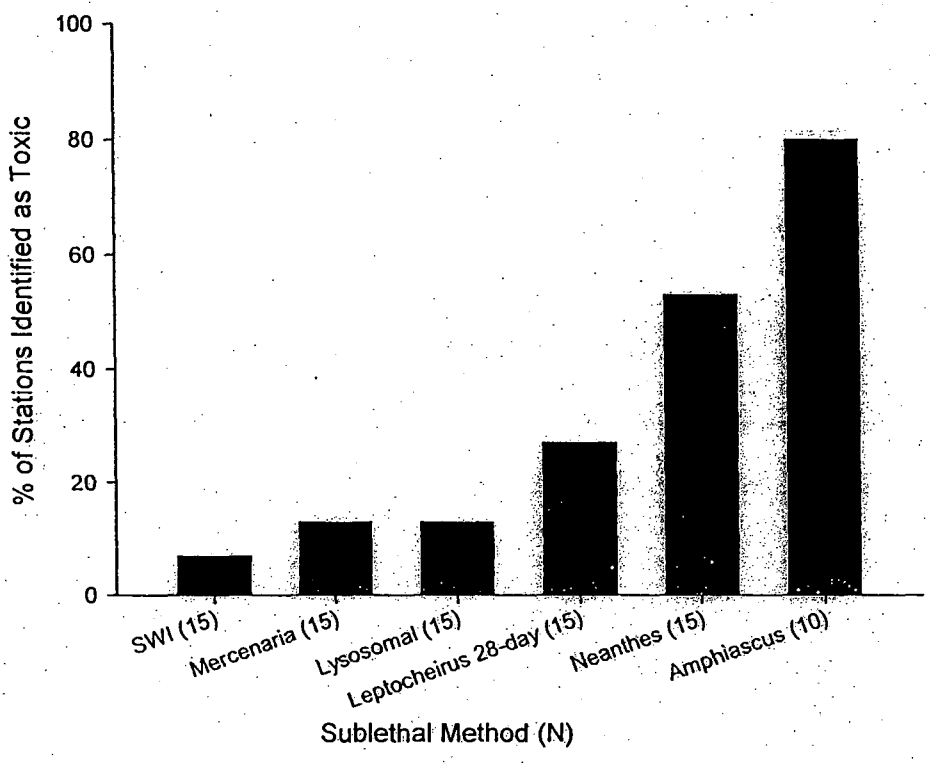

Figure 3. Percentage of stations that each sublethal method identified as being toxic. Number of samples tested is in parentheses. *Leptocheirus plumulosus* (*Leptocheirus*), Sediment-water Interface (SWI), *Mercenaria mercenaria* (*Mercenaria*), *Crassostrea virginica* (Lysosomal), *Neanthes arenaceodentata* (*Neanthes*), and *Amphiascus tenuiremus* (*Amphiascus*).

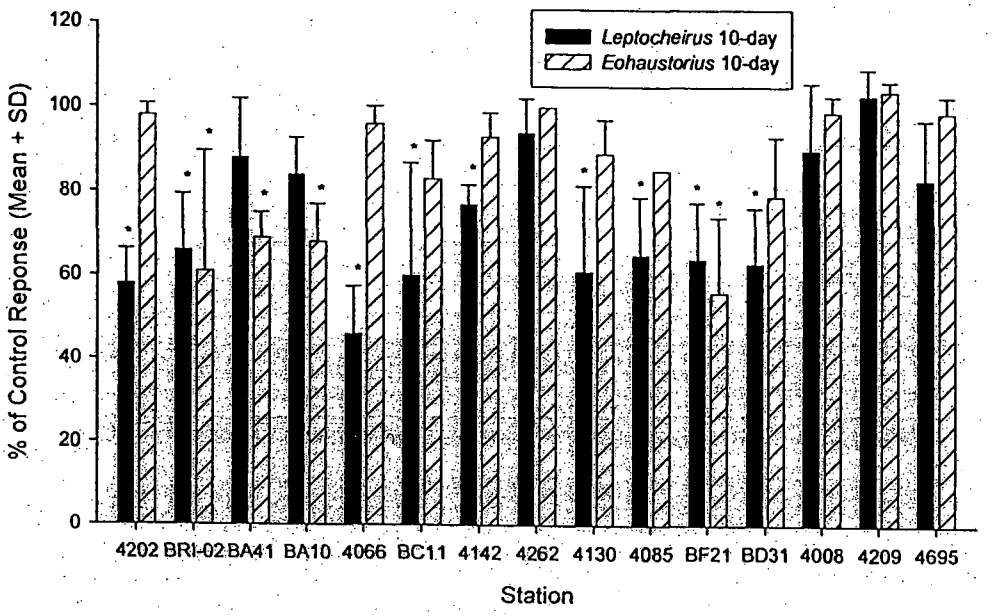

Figure 4. Results of *Eohaustorius estuarius* (*Eohaustorius*) 10-dayand *Leptocheirus plumulosus* (*Leptocheirus*) 10-day survival tests conducted as part of the Bight'03 and RMP regional monitoring programs. Stations marked with * are significantly different from control values (p <0.05) and less than 80% of the control response.

Comparisons Between Sublethal and Acute Tests

The *N. arenaceodentata* and *Amphiascus* tests detected toxicity at stations where *E. estuarius* did not at 27% and 70% of the stations, respectively; while in no cases did *E. estuarius* demonstrate toxicity where either of these two tests did not (Table 3). Alternatively, the *E. estuarius* test identified a higher percentage of stations as toxic than did the SWI, *M. mercenaria* and *C. virginica* tests. The *E. estuarius* test identified toxicity in 27% of the samples that the other tests classified as nontoxic.

Table 3. Comparative ability of acute and sublethal sediment toxicity test methods to detect toxicity in stations from southern California and San Francisco Bay. Numeric values are expressed as percentage of stations tested. The station order is based on a combined ranking of chemical contamination and benthic community health, with the most contaminated/impacted stations listed first. *Eohaustorius estuarius* (*Eohaustorius*), *Leptocheirus plumulosus* (*Lepto*), Sediment-water Interface (SWI), *Merceneria mercenaria* (*Mercenaria*), *Crassostrea virginica* (Lysosome), *Neanthes arenaceodentata* (*Neanthes*), and *Amphiascus tenuiremus* (*Amphiascus*).

	Acute Methods		Sublethal methods					
Station	*Eohaustorius*	*Lepto* 10-day	SWI	*Mercenaria*	Lysosome	*Lepto*28 -day	*Neanthes*	*Amphiascus*
4202	N	Y	N	N	N	Y	N	Y
BRI-2	Y	Y	N	N	N	Y	Y	Y
BA41	Y	N	N	N	N	N	Y	Y
BA10	Y	N	N	N	N	N	Y	–
4066	N	Y	N	N	Y	Y	N	–
BC11	N	Y	Y	N	Y	N	Y	Y
4142	N	Y	N	N	N	N	N	–
4262	N	N	N	N	N	N	Y	Y
4130	N	Y	N	Y	N	N	Y	Y
4085	N	Y	N	N	N	N	N	Y
BF21	Y	Y	N	N	N	N	Y	–
BD31	N	Y	N	N	N	N	N	–
4008	N	N	N	Y	N	N	Y	Y
4209	N	N	N	N	N	N	N	Y
4695	N	N	N	N	N	Y	N	N
% Sublethal Toxic, *Eohaustorius* Not Toxic	–	–	7	13	13	20	27	70
% *Eohaustorius* Toxic, Sublethal Not Toxic	–	–	27	27	27	20	0	0
% Agree Toxic	–	–	0	0	0	7	27	20
%Agree Not Toxic	–	–	67	60	60	53	47	10
	–	–						
% Sublethal Toxic, *Leptocheirus* Not Toxic	–	–	0	7	0	7	27	40
% *Leptocheirus* Toxic, Sublethal Not Toxic	–	–	53	53	47	40	33	0
% Agree Toxic	–	–	7	7	13	20	27	50
%Agree Not Toxic	–	–	40	33	40	33	13	10

Y = Station identified as toxic
N = Station not identified as toxic
– = Station or comparison not tested

The *L. plumulosus* 10-day test found a higher percentage of toxic stations than all of the sublethal methods except for the *Amphiascus* test (Table 3). The *Amphiascus* test found four stations (40%) to be toxic that were not identified by the *L. plumulosus* acute test. There was concordance between the *L. plumulosus* 10-day test and the *Amphiascus* test for the remaining stations with both finding five stations to be toxic and one not. The *N. arenaceodentata* test found four stations to be toxic that were not identified by the *L. plumulosus* acute test. However, there were five stations that were toxic in the *L. plumulosus* acute test, but were not toxic in the *N. arenaceodentata* test. For the *M. mercenaria, C. virginica, L. plumulosus* 28-day and SWI tests there was a high percentage of stations (40% or more) that the *L. plumulosus* acute test found to be toxic that the sublethal methods did not.

Combining the data from either a lethal and sublethal test or two lethal tests provided more information regarding toxicity than conducting just one test of either kind. The greatest sensitivities (most toxic stations detected) were found with the combinations of *L. plumulosus* 10-day and *N. arenaceodentata* or *Amphiascus* methods (Table 3). Nearly as sensitive was the combination of the two acute tests (see Figure 4 where 11 out of fifteen stations were identified as toxic by one or both tests).

Chemistry

Sediment physical parameters were very wide ranging with grain sizes that were nearly 100% fines (silt + clay) to 100% sand (Table 4). TOC values ranged from 0.02% to 2.9%.

Sediment contaminant concentrations also were variable among stations (Table 4). Three stations had elevated chemistry compared to the other stations. Station 4202, on the Palos Verdes shelf, had a very high concentration of total DDTs. Station BRI-02, in Marina Del Rey, had low concentrations of organic contaminants, but substantial concentrations of copper, lead and zinc. Station 4085 contained intermediate concentrations of several metals and organics. Based on the mean ERMq calculations, all of the stations tested fell into what would be considered the low to moderate range of contaminant concentrations with all mean quotients less than 0.7 (Table 4). Five samples had mean ERMq values below 0.1, a level not expected to be toxic. The mean quotients for the remaining stations fell between 0.11 and 1.0, a range that has been found to be toxic in about half of the cases (Long *et al.* 1998).

Eohaustorius estuarius survival, both *Amphiascus* endpoints and *N. arenaceodentata* growth had significant Spearman correlations with sediment chemistry (Table 5). Correlations with various metals were present, but none with organics. All of the significant correlations were negative, indicating that as the concentration increased the endpoint decreased (e.g., decreased survival or growth). All the toxicity test methods that correlated with chemistry also had significant correlations with sediment grain size. The chemical constituents that correlated with toxicity also correlated with the grain size parameters.

Table 4. Selected chemistry data from southern California and San Francisco Bay sediment samples on which toxicity tests were performed.

Station	Arsenic (mg/kg)	Cadmium (mg/kg)	Chromium (mg/kg)	Copper (mg/kg)	Lead (mg/kg)	Mercury (mg/kg)	Nickel (mg/kg)	Silver (mg/kg)	Tin (mg/kg)	Zinc (mg/kg)
4202	8.5	6.6	136	5	30.0	0.46	29.0	1.9	NA	180
BRI-02	13.0	0.3	94	362	113.0	0.98	41.6	2.0	6.3	382
BA41	4.5	0.2	NA	30	17.4	0.34	58.2	0.1	NA	90
BA10	4.1	0.1	NA	24	11.3	0.24	46.9	0.2	NA	70
4066	1.0	0.1	7	7	4.7	0.10	4.0	0.6	0.4	22
BC11	4.0	0.3	NA	39	29.7	0.23	65.9	0.1	NA	108
4142	1.0	0.2	5	6	4.3	0.06	4.1	0.3	0.5	49
4262	4.0	0.6	46	3	38.9	0.23	21.2	0.7	NA	92
4130	7.0	0.8	49	87	61.6	0.40	25.8	0.8	3.8	248
4085	11.6	1.7	78	101	130.0	0.41	33.1	2.9	6.3	315
BF21	8.5	0.2	NA	53	16.4	0.27	88.6	0.2	NA	126
BD31	7.5	0.2	NA	51	17.8	0.24	87.8	ND	NA	126
4008	2.5	0.1	34	14	4.7	0.08	10.2	0. 7	1.6	48
4209	1.5	ND	10	3	1.4	0.02	3.0	0.2	0.5	14
4695	1.1	ND	5	1	1.2	0.02	0.9	0.2	0.3	6

Table 4. (continued)

Station	TOC %	Sand %	Silt %	Clay %	ΣPAHs µg/kg	ΣDDTs µg/kg	ΣPCBs µg/kg	Mean ERMq*	ERMq Ranking
4202	2.06	39	50	11	678	2301.3	193.9	0.68	1
BRI-02	1.99	8	74	18	76	2.2	ND	0.26	4
BA41	1.09	20	22	49	1923	0.2	2.5	0.14	7.5
BA10	2.34	44	15	36	724	0.6	2.3	0.10	10
4066	0.02	100	0	0	52	1.0	ND	0.02	12
BC11	1.80	22	22	48	740	0.6	111.3	0.34	2
4142	0.27	62	NA	NA	73	ND	ND	0.02	13
4262	1.50	56	36	8	625	49.8	66.0	0.29	3
4130	2.04	44	46	10	1206	9.9	15.7	0.17	6
4085	2.93	30	57	13	578	14.6	22.6	0.24	5
BF21	1.37	1	39	60	582	0.8	0.8	0.14	7.5
BD31	1.33	9	32	59	450	1.4	0.8	0.14	9
4008	0.67	54	40	6	12	1.3	ND	0.04	11
4209	0.04	98	2	ND	ND	ND	ND	0.01	14
4695	ND	100	ND	ND	ND	ND	ND	0.01	15

* DDT concentrations not included in ERMq calculation.

Table 5. Spearman rank correlations on selected sediment parameters and toxicity endpoints. Boxed values are significant ($p \leq 0.05$). *Eohaustorius estuarius* (*Eohaus*), *Leptocheirus plumulosus* (*Lepto*), Sediment-water Interface (SWI), *Crassostrea virginica* (Lysosome), ERMq (effects range mean quotient).

r	Eohaus Survival	Lepto 10 Survival	SWI Mussel	Clam Growth	Lysosome	Lepto 28 Survival	Lepto 28 Growth	Worm Survival	Worm Growth	Number of Copepodites	Realized Offspring
Arsenic	-0.604	-0.239	0.274	-0.145	-0.080	-0.0502	-0.422	0.136	-0.542	-0.585	-0.806
Cadmium	-0.155	-0.401	0.264	-0.295	-0.099	-0.307	-0.295	0.132	-0.264	-0.206	-0.488
Copper	-0.786	-0.375	0.196	-0.354	-0.059	0.039	-0.293	-0.051	-0.565	-0.829	-0.952
Lead	-0.366	-0.350	0.337	-0.306	-0.025	-0.251	-0.247	0.233	-0.390	-0.482	-0.842
Mercury	-0.596	-0.406	0.476	-0.143	-0.093	-0.196	-0.351	0.059	-0.514	-0.572	-0.742
Nickel	-0.836	-0.289	-0.386	-0.382	0.136	0.222	-0.111	-0.022	-0.594	-0.866	-0.709
Silver	0.220	-0.089	0.533	0.012	-0.225	-0.373	-0.209	0.188	-0.080	-0.043	-0.455
Zinc	-0.549	-0.434	0.250	-0.301	-0.085	-0.196	-0.443	0.138	-0.476	-0.567	-0.842
TOC (%)	-0.440	-0.250	0.119	-0.268	0.070	-0.043	-0.181	-0.012	-0.424	-0.390	-0.661
Sand (%)	0.820	0.237	0.091	0.349	0.081	-0.120	0.288	-0.069	0.653	0.933	0.794
Clay (%)	-0.823	-0.229	-0.320	-0.326	0.139	0.228	-0.116	-0.032	-0.596	-0.881	-0.717
ΣPAHs	-0.491	-0.259	0.032	-0.354	0.222	0.104	0.181	-0.314	-0.490	-0.520	-0.486
ΣDDTs	-0.013	-0.333	0.123	-0.264	0.014	-0.201	-0.100	0.382	-0.320	-0.086	-0.365
ΣPCBs	-0.124	-0.295	-0.078	-0.192	0.339	-0.052	0.043	0.062	-0.211	-0.066	-0.125
Mean ERMq	-0.288	-0.268	0.018	-0.402	0.124	-0.221	-0.150	0.306	-0.449	-0.329	-0.370

Benthic Community

A range of benthic community condition was present among the stations. Most stations were classified as being in reference condition (8/15) or having an intermediate level of disturbance (5/15 stations at Level 2 or 3). Two stations (4066 and 4142) had Level 4 designations (Table 6), which indicated severe effects to the benthic community. The variations in benthic community condition did not correspond with the sediment contamination gradient. The average mean ERMq of all stations in each benthic condition category was lowest for the Level 4 stations and highest for the Level 2 stations (Table 6).

There was little correspondence between changes in benthic community condition and toxicity for most of the test methods. *L. plumulosus* 10-day survival was the only test that consistently detected toxicity at the Level 4 stations (Table 6). Most of the stations that did show toxicity were in the Reference or Level 2 categories for benthic community condition. Four of the test methods (*E. estuarius*, *L. plumulosus* 10-day and 28-day and *Amphiascus*) showed an increased incidence of toxicity among all impacted stations (Levels 2 through 4 combined) compared to stations classified as having a reference benthic condition. Correlations of BRI values for the southern California stations showed that only the *L. plumulosus* 10-day test method had a significant correlation with benthic community condition (Table 6). The correlation coefficients were negative for all but the *C. virginica* lysosome method, indicating that as the BRI value increased the toxicity endpoint value decreased (i.e., survival or growth decreased).

Table 6. Incidence of toxicity within benthic index categories and Spearman's Rank Correlation values for toxicity test endpoints. Boxed values are statistically significant ($p \leq 0.05$).

	Benthic Index Category					
Test	Ref[1]	Level 2[2]	Level 3[3]	Level 4[4]	Levels 2-4	r^5
Number of Stations	8	4	1	2	7	
Benthic Station Rank	11.5	5.5	3.0	1.5		
Mean ERMq	0.15	0.31	0.10	0.02	0.20	
	Incidence of Toxicity (%)					
Eohaustorius estuarius 10-day Survival	12	50	100	0	42	-0.52
Leptocheirus plumulosus 10-day Survival	50	75	0	100	71	**-0.64**
Mytilus galloprovincialis Sediment-water Interface	12	0	0	0	0	-0.27
Mercenaria mercenaria Growth	12	25	0	0	14	-0.20
Crassostrea virginica Lysosome	12	0	0	50	14	0.04
L. plumulosus 28-day Growth	12	50	0	50	50	-0.25
Neanthes arenaceodentata Growth	50	75	100	0	57	-0.12
Amphiascus No. Copepodites	83	100	na	na	100	-0.44

[1] Reference stations: BC11, 4262, 4085, BF21, BD31, 4008, 4209, 4695
[2] Level 2 (Loss of biodiversity): 4202, BRI-02, BA41, 4130
[3] Level 3 (Loss of community function): BA10
[4] Level 4 (Defaunation): 4066, 4142
[5] Correlation calculated using southern California data only

Ranking of Stations to Reflect Sediment Condition

Since most of the stations in this study had not been previously sampled, there was not a known gradient of expected sediment condition. To put the data into this context, the stations were ranked by a combination of chemical contamination and benthic community health. To achieve this the stations were ranked by their mean ERMq values (Table 4). The stations were also ranked similarly by the benthic community analysis results (Table 6). These two rankings were then summed and the stations re-ranked to get the combined effect. The data presented in Figure 5 have the stations with the lowest rankings (highest chemistry and most degraded benthos) on the left and highest rankings on the right. Station 4202 had the highest concentrations of the most chemical constituents and showed a toxic response to two of the sublethal test endpoints. It ranked as having the worst sediment condition of all the stations even without the high value of DDT taken into consideration. Station BRI-2 with high concentrations of three metals and with a Level 2 benthic designation ranked as the second worst. Station 4085 with moderate levels of several chemicals ranked in the middle. Although stations 4066 and 4142 had Level 4 benthic designations, they fell in the middle of the ranks because their chemical concentrations were lower.

DISCUSSION

The sensitivity of the toxicity methods were variable within the two broad categories of tests evaluated, indicating that general classifications of tests as either acute or sublethal do not reliably indicate their relative sensitivity. For example, the most sensitive test in this study was the sublethal *Amphiascus* life cycle method, but the acute *L. plumulosus* survival test was more sensitive that any of the other sublethal tests compared. This variation in sensitivity between acute and sublethal tests is consistent with other studies, suggesting that the relative sensitivity of acute and sublethal tests to whole sediment samples varies according to the combination of tests and sample types evaluated. Comparative studies using the *L. plumulosus* 28-day test have shown that the sublethal endpoints from this test are not consistently more sensitive than acute amphipod tests to field and spiked sediments (DeWitt *et al.* 1997). Another study found that the acute *A. abdita* test was more sensitive than the *L. plumulosus* 28-day test, which was more sensitive than the *N. arenaceodentata* 28-day test (Kennedy *et al.* 2004). In contrast to the results of the present study, the *M. mercenaria* test was found to be more sensitive than the acute *A. abdita* survival test when sediment samples from the Carolinian Province were tested (Ringwood *et al.* 1996).

Our finding that *Amphiascus* was the most sensitive method overall is consistent with other studies indicating the high sensitivity of this life cycle test. Tests using sediments from Biscayne Bay in Florida by Long *et al.* (Long *et al.* 1999) found a greater incidence of toxicity with the *Amphiascus* life cycle method (73%) than with the *A. abdita* 10-day survival test (7%). The high sensitivity, chronic exposure and multiple endpoints that are characteristic of this test are desirable qualities, however, more investigation is needed to determine whether the high level of response of the test to southern California samples having low contaminant concentrations and reference benthic community condition reflect chemical toxicity or the effects of potentially confounding factors such as ammonia or organic carbon.

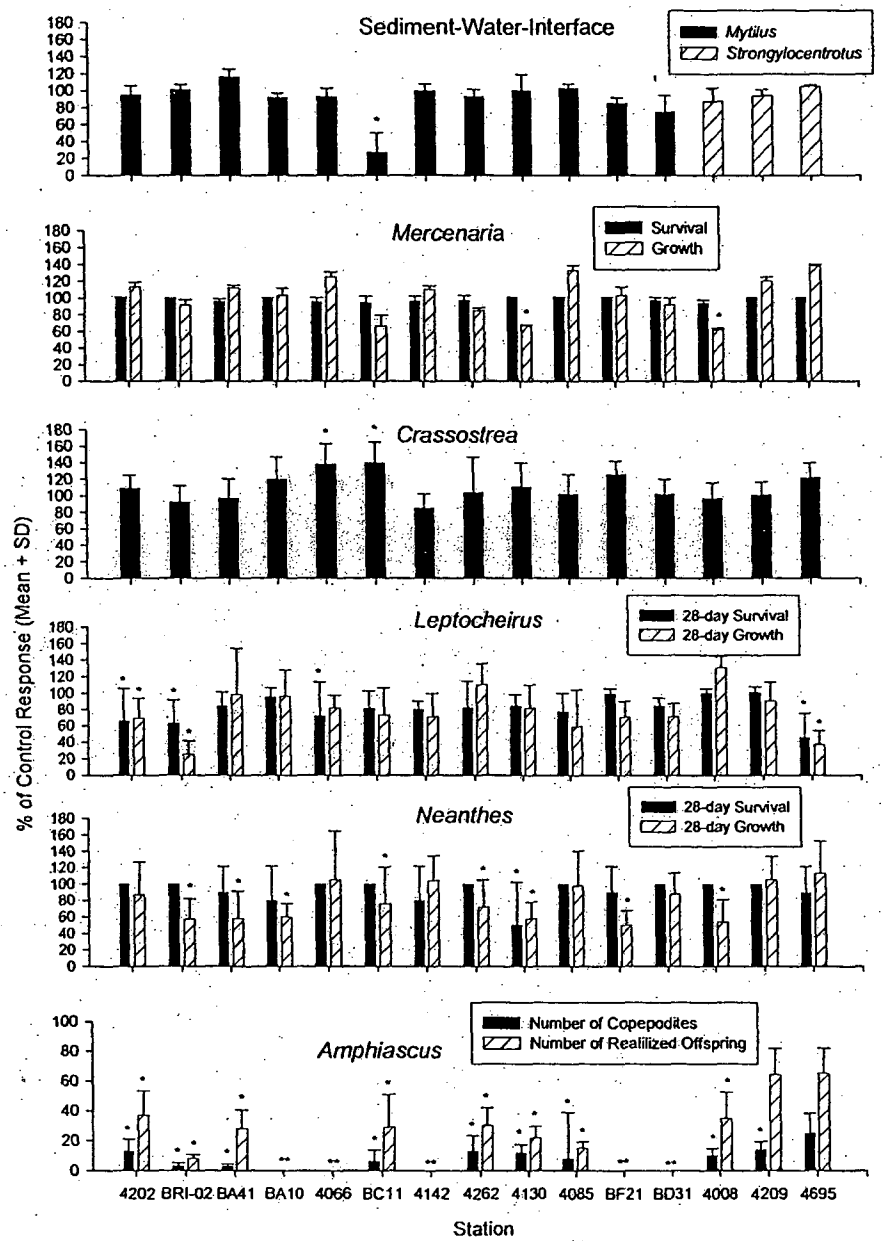

Figure 5. Results of sublethal test methods conducted on samples from southern California and San Francisco Bay. Stations marked with * are significantly different from control values ($p < 0.05$). Stations with ** indicate that the station was not tested using that method. *Eohaustorius estuarius* (*Eohaustorius*), *Leptocheirus plumulosus* (*Leptocheirus*), *Merceneria mercenaria* (*Mercenaria*), *Crassostrea virginica* (Crassostrea), *Neanthes arenaceodentata* (*Neanthes*), *Mytilus galloprovincialis* (*Mytilus*), *Strongylocentrotus pupuratus* (*Strongylocentrotus*) and *Amphiascus tenuiremus* (*Amphiascus*).

Several factors may have accounted for the variation in sensitivity among methods observed in this study, including: mode of exposure, species-specific sensitivity to contaminants, and the influence of confounding factors. The mode of exposure varied greatly among tests and those tests with the longest exposure duration and most direct contact with the sediment (i.e., *Amphiascus* and *N. arenaceodentata*) tended to be most sensitive. For the SWI method, which was least sensitive, the organisms are in the water column directly above the sediment, and are exposed for a relatively short period of time to only those contaminants diffusing into the overlying water. These differences in exposure method and sample response can be used to advantage to investigate the mode of contaminant exposure or identify the cause of toxicity.

Differences in contaminant sensitivity among test methods have been documented for some of the test species and may have influenced the results of this study. Several studies have been conducted that compared the *L. plumulosus* 10-day and 28-day tests and the *N. arenaceodentata* 28-day test to various chemicals and found varying patterns of response. The *N. arenaceodentata* test was more sensitive than *L. plumulosus* to sediments contaminated with metals or the explosive TNT, both of these sublethal tests were more sensitive than the acute *L. plumulosus* test to PCBs, yet the *L. plumulosus* acute method was more sensitive to PAH contaminated sediments than *N. arenaceodentata* (Farrar *et al.* 2005, Green *et al.* 1999). Comparisons among acute tests using *A. abdita*, *E. estuarius* and *R. abronius* showed that *E. estuarius* was the most sensitive to DDT, while *A. abdita* and *R. abronius* were more sensitive to cadmium (Weston 1996). Sediment contaminant mixtures varied among the stations in the present study, with differences of up to two orders of magnitude in metals, PCB, and PAH concentrations, and up to three orders of magnitude in DDT. These differences may have contributed to the variation in response among the test methods.

Variations in holding time or sediment handling that occurred among the laboratories are potential confounding factors that may have altered the toxicity of the samples through changes in bioavailability or chemical composition. The nature and magnitude of such effects was not determined in this study, but an analysis of the data indicates that the patterns of relative sensitivity observed among the test methods were independent of holding time. For example, holding times were shortest and similar for the SWI and *Amphiascus* methods, yet these two tests had very different patterns of response to the samples (Table 7). The patterns of relative response among the tests were also similar for the two batches of whole sediment tested (e.g., *Amphiascus* most sensitive, *M. mercenaria* and *C. virginica* usually least sensitive), indicating that variations in holding time or sediment handling among the tests and batches were not major confounding factors.

The most responsive of the acute and sublethal toxicity tests showed a general correspondence with the gradient of sediment condition described by a combination of the chemistry and benthic community data. The *Amphiascus* and *N. arenaceodentata* tests reflected the expected pattern of decreasing toxicity with improving sediment condition (Figure 5), as did both of the acute tests (Figure 4). These relationships were inconsistent for stations having intermediate rankings of sediment conditions, indicating

substantial uncertainty in the relationships among the different indicators of sediment quality. In addition to the sources of variability mentioned previously for the toxicity tests, measures of sediment chemistry and benthic community condition also have inherent uncertainty and sources of error that may have accounted for the inconsistent relationships.

Significant correlations with chemistry concentrations were found in the present study for the *E. estuarius* survival, *Amphiascus* reproduction and *N. arenaceodentata* growth tests. Similar relationships have also been documented in many other studies for a variety of test organisms and form the basis for empirical sediment quality guidelines (Long *et al.* 1995, Fairey *et al.* 2001). There were also significant correlations with grain size for each test. The chemistry values also correlated with grain size and many of the chemical constituents also correlated with one another. These intercorrelations make determining whether toxicity is associated with chemistry or the confounding factor of grain size a difficult matter. Grain size is not known to be a confounding factor for *E. estuarius* (USEPA 1994). Grain size should not have been an issue for *Amphiascus* since all samples were sieved to remove large particles and optimize the sediments for the animals. *Neanthes arenaceodentata* have been tested in grain sizes ranging from 5 to 100% sand with no effects on either survival or growth (Dillon *et al.* 1993). These factors indicate that there is a likelihood of an association between sediment contamination and toxicity for these three methods in the current study, rather than a grain size effect.

The lack of correlations with sediment chemistry for some of the test methods may have several causes. There was little observed toxicity for many of the tests making the detection of correlations difficult. In addition, no measure of bioavailability of chemical constituents was made for the sediments, adding uncertainty regarding the actual chemical dose received by the test animals. Sediment chemistry analyses do not quantify all possible toxicants, so it is possible that unmeasured chemical constituents or interactions between compounds may have caused the observed toxicity. Another potential source of uncertainty is toxicity from confounding factors such as ammonia or sulfides. While the sensitivity of some of the test methods to these factors is poorly known, water quality data from the tests showed that dissolved ammonia concentrations were low and below concentrations of concern for most of the samples, indicating that these factors did not have a significant influence on the results.

A strong relationship between the toxicity results and benthic community condition was not found in this study, suggesting that these indicators were responding to different aspects of sediment quality. Other studies have reported similar results. Analyses of Chesapeake Bay sediment toxicity using the *L. plumulosus* 10-day and 28-day tests found a similar lack of correspondence with benthic community response (McGee *et al.* 2004). A statistically significant correlation between *E. estuarius* mortality and benthic community impact was found for southern California embayment sediments, but the relationship accounted for only 10% of the variation in community condition (Ranasinghe *et al.* 2003). Toxicity tests differ from the *in situ* benthic environment in many aspects, such as the exposure duration, species type, and laboratory handling of the

sediment. These factors can affect contaminant bioavailability or the sensitivity of the response and may have accounted for the relatively high frequency of toxicity detected in samples containing an unimpacted benthic community. It is not possible for toxicity tests to perfectly replicate environmental exposure conditions and provide a substitute for assessment of biological effects on resident organisms; these tests are intended to provide a measure of potential contaminant effects that is complementary to chemical and biological measures.

The effects of noncontaminant factors on the benthic community analyses may have also influenced the correlation analyses with toxicity. Changes in benthic community condition did not correspond with increasing contamination levels, as represented by the mean ERMq (Table 6). This finding contrasts with studies in other regions of the United States that have shown an increase in the incidence of degraded benthos within the mean ERMq range present among the southern California samples (Hyland *et al.* 2003). It is possible that variations in noncontaminant factors related to the diversity of habitats and sediment types included in this study may have influenced the benthic community results and confounded the ability of to discern impacts due to toxicity.

This study and others have shown marked differences in sensitivity among toxicity tests that cannot be easily predicted on the basis of biological endpoint and mode of exposure. This diversity presents both a challenge and opportunity for sediment toxicity evaluation. The challenge lies in selecting the most appropriate tests for use in a particular study. Variations in relative sensitivity related to contaminant type and uncertainties in the interpretation of chemistry and benthic community data suggest that the use of just a single test method, selected on the basis of high sensitivity to a subset of samples, is unlikely to provide a complete or confident assessment of toxicity. Data from multiple toxicity tests that represent a diversity of species, endpoints, and exposure modes, in addition to sediment chemistry and benthic community analyses, are needed to assess sediment quality to the level of confidence needed to support management decisions (Chapman and Anderson 2005). The use of a diverse suite of toxicity tests also provides an opportunity to improve our understanding of the causes of sediment toxicity, as differences in the patterns or symptoms of response between tests can be used to help identify the case of toxicity (USEPA 1993).

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APPENDIX B

Interlaboratory Comparison of Sublethal Sediment Toxicity Test Methods Using *Mercenaria mercenaria* and *Mytilus galloprovincialis*

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INTRODUCTION

Many sediment quality monitoring and assessment programs use a combination of acute amphipod survival and sublethal sediment toxicity test methods. The acute amphipod methods are usually conducted using standard protocols for a small number of species (USEPA 1994) and several studies have been conducted that document important aspects of the tests such as relative sensitivity and interlaboratory variability. A greater diversity of sublethal sediment toxicity test methods have been applied in various studies (Lamberson *et al.* 1992), yet few studies have been conducted that compare the relative performance of these methods.

A significant data gap for some sublethal toxicity tests is information on interlaboratory variability. An understanding of the amount of variation associated with conducting the test in different laboratories is needed to assist in decisions regarding the selection of test methods for use in a study and for determining the significance of various ranges in the organism's response to the test samples. Interlaboratory variability data are not available for two sublethal methods that are promising candidates for use in regional monitoring programs: the seven-day growth test using the seed clam, *Mercenaria mercenaria* (Ringwood and Keppler 1998), and the two-day sediment-water interface (SWI) test using embryos of the mussel *Mytilus galloprovincialis* (Anderson *et al.*, 1996). Interlaboratory variability for these two test methods is needed to support the evaluation of these methods for use in sediment testing programs.

The objective of this study was to measure the interlaboratory variability associated with the seed clam and mussel embryo sediment toxicity tests. Interlaboratory comparison tests were conducted with both test methods using field and spiked sediments.

METHODS

Concurrent sediment toxicity tests were conducted by two laboratories for the mussel embryo test and by three laboratories for the seed clam test. Two types of samples were tested in each set of experiments: dilutions a contaminated field sediment and several concentrations of sediment spiked with nonylphenol. In both cases, one of the participating laboratories was the originator of the test method. For both test methods, each laboratory also conducted reference toxicant exposures to demonstrate laboratory comparability. Additionally, range finding tests were conducted to determine the proper concentrations of the spiked and diluted samples.

Field

The sediment used for spiking with nonylphenol and for dilution of contaminated field sediment was collected by Orange County Sanitation District near their reference site 18. This station is located offshore and has low levels of chemical contamination and a moderate grain size (~50% sand). The contaminated field sediment was from Consolidated Slip (CS) in Los Angeles Harbor and had been in storage since collection in October 2002. Consolidated Slip has a long history of contamination from industrial sources with very high levels of PAHs, DDT and metals and very fine grain size. Both the sediment from CS and Orange County (OC) were stored in plastic containers at 5 °C.

Test Sediment Preparation

Stock solutions of 4-n-nonylphenol (Alfa Aesar) in acetone were placed into 2 L glass jars and the carrier was allowed to volatilize on a Wheaton roller apparatus (Distworth *et al.*, 1990). After volatilization, OC sediment was added to the containers in amounts in amounts corresponding to nominal nonylphenol concentrations of 0.1-1000 mg/kg and rolled for the first 24 hours of the seven-day equilibration time. Sediment was stored at 5°C in amber glass jars for the remainder of the equilibration period. Chemical verification of the final sediment concentrations was not preformed.

The CS dilutions were made as 10, 25, and 50 percent wet weight:wet weight CS sediment diluted with OC sediment. Mixing was accomplished with a polycarbonate spoon in a large polycarbonate bowl. A control sample consisting of 100% OC sediment was also tested. Aliquots of the mixtures were placed into separate containers for each laboratory. The samples were then stored in plastic containers at 4°C and allowed to equilibrate for seven days, before being used in the interlaboratory experiments.

Mussel Embryo Development Test

The University of California Davis Marine Pollution Studies Laboratory (MPSL) and Southern California Coastal Water Research Project (SCCWRP) conducted the laboratory intercalibration for the sediment-water interface (SWI) mussel embryo development test. The mussels (*M. galloprovincialis*), obtained from Carlsbad Aquafarms in Carlsbad, CA, were acclimated in 32 g/kg seawater at 15°C overnight. The procedure for the mussel development test and the exposure procedures followed methods described in Appendix A. To simulate a core sample, the core tubes were filled with 5 cm of the sediment samples, with five replicate tubes per treatment. Seawater was

added over the sediments, aeration was added and the system was allowed to equilibrate overnight.

Both laboratories also performed a 48-hour water only reference toxicant experiment with copper. A stock solution of $CuCl_2$ was provided by SCCWRP. Each laboratory prepared dilutions of the stock to achieve concentrations of 4.5, 6.5, 9.5, 13.9, 20.4, and 30.0 µg/L copper plus a water only seawater control. Four replicates of each concentration were tested.

At the end of the experiment all normal and abnormal embryos were counted. The %Normal-Alive endpoint was calculated by dividing the number of normal embryos in each vial by the mean initial embryo count and then multiplying by 100.

Juvenile Clam Growth Test

Three laboratories participated in the seed clam (*M. mercenaria*) interlaboratory calibration experiment: South Carolina Marine Resources Research Institute (MRRI), SCCWRP, and Weston Solutions (Carlsbad, CA). Exposure methods followed those described in Appendix A. The clams were fed the algae *Isochrysis galbana* during all exposures. For the interlaboratory experiment all laboratories used live *I. galbana* cultures. However, during the range finding tests, a concentrated *I. galbana* solution obtained from Reed Mariculture was used for feeding after proper dilution.

All laboratories performed a water only 7-day reference toxicant test exposure to copper with the same feeding regime as for the sediment experiment. The reference toxicant experiment used a 10,000 µg/L stock solution of $CuCl_2$ provided by SCCWRP. Dilutions were prepared at each of the laboratories to achieve concentrations of 6.25, 12.5, 25, 50, and 100 µg/L copper.

Although OC sediment was used as a control, it had never been previously tested using the juvenile clams. Therefore, a second control was included that has historically been used as a reference for this clam test, to ensure reasonable control response. MRRI provided this reference sediment (coded LTH), which was sandy sediment from a clean site in South Carolina.

Data analysis

Data for all tests were adjusted to control response within each laboratory. For the SWI test, the data was adjusted to the water only control value from the reference toxicant test. For the *M. mercenaria* test, the data was adjusted to the response in the LTH sample. Significant differences between controls and treatments were calculated by t-tests assuming unequal variance ($p \leq 0.05$). Differences between laboratories were calculated with either t-tests (SWI) assuming unequal variance or ANOVAs (*M. mercenaria*) followed by Tukey's multiple comparison test. EC50s for reference toxicant exposure for the mussel embryos were calculated using probit analysis. For the clam reference toxicant exposure, the IC50 (the inhibition concentration where a 50% reduction in growth is predicted to occur) was calculated using the EPA ICP program.

RESULTS

Mussel Embryo Development

Range Finding

For the SWI mussel embryo development test, range finding experiments using nonylphenol and CS samples were completed at SCCWRP. An initial selected series of 10, 100, and 1000 mg/L nonylphenol produced a dose response with 87% of control normal-alive embryos in the 10mg/L nonylphenol, 80% in the 100 mg/L nonylphenol, and 21% in the 1000 mg/L nonylphenol sample. Because this was a suitable dose-response, these concentrations were selected for use in the intercalibration exercise.

The CS dilutions were tested at 5, 10, and 25% of CS sediment. The percent of control normal-alive embryos at 5 and 10% CS was 99%, and at 25% CS was 79%. In order to increase the range of response, the percentage of CS in the samples was increased to 10, 25 and 50% for the intercalibration exercise.

Interlaboratory Calibration

MPSL results showed a significant difference between all three concentrations of the nonylphenol spiked sediments and the non-spiked OC control station. MPSL obtained a good dose response, with each concentration showing substantially more toxicity than the previous one and severe toxicity at 1000 mg/L nonylphenol, with no normally developed embryos (Figure 1). SCCWRP found only the 1000 mg/L nonylphenol sample significantly different from the control with 0% of the embryos developed normally (Figure 1). SCCWRP found development in the other two nonylphenol concentrations was similar to the OC sediment.

MPSL found the highest two concentrations of the CS sediment to be significantly different from the OC station. However, the toxicity in the dilution series of 10, 25, and 50% CS was of moderate degree with 77, 70, and 57% normal-alive relative to the control, respectively (Figure 2). SCCWRP did not find a dose response for CS dilution sediments and did not find any of the dilutions to be significantly different from the OC station. The two higher concentrations of CS had normal development only slight less than that found in the water only controls.

There was little agreement between the two laboratories' results. Of the seven samples tested only two, OC and 10% CS, were not significantly different between the laboratories. The five other samples were significantly different from each other, and in all cases the MPSL %normal-alive results were lower than those of SCCWRP.

Reference Toxicant

The EC50s for the two laboratories were comparable with MPSL being 6.8 µg/L copper (upper and lower 95% confidence limits were 6.5 and 6.9) and SCCWRP 7.6 µg/L copper (lower and upper 95% confidence limits were 7.2 and 8.0). The dose-response plots of the copper exposure were remarkably similar between the two laboratories (Figure 3).

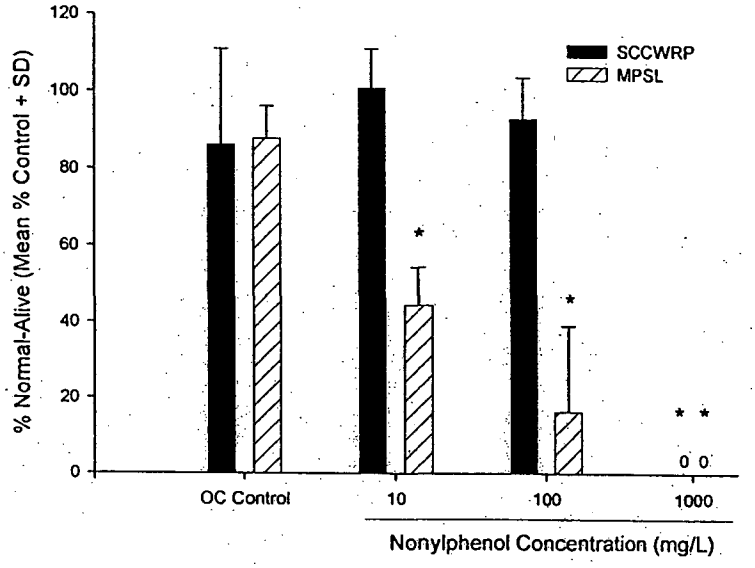

Figure 1. Water only control adjusted sediment-water interface mussel embryo development responses to nonylphenol from Marine Pollution Studies Laboratory (MPSL) and Southern California Coastal Water Research Project (SCCWRP). Results marked with * are significantly different from OC.

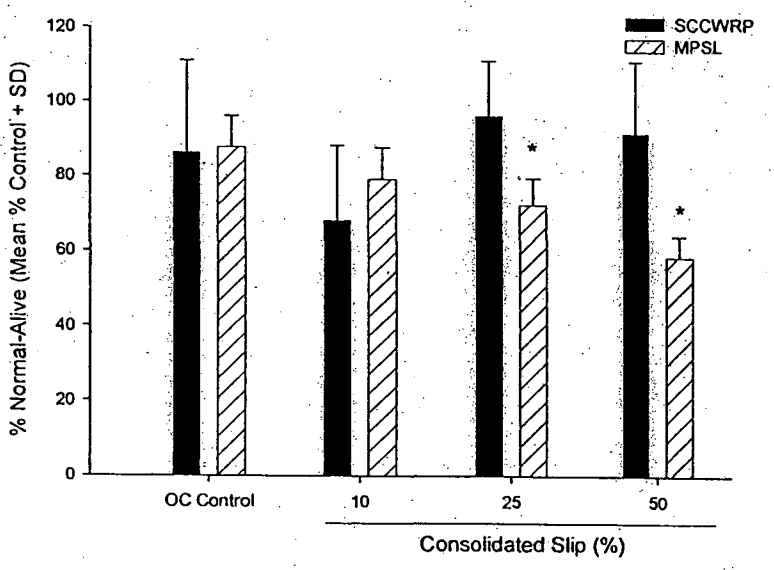

Figure 2. Water only control adjusted sediment-water interface embryo development responses to consolidated slip dilutions from Marine Pollution Studies Laboratory (MPSL) and Southern California Coastal Water Research Project (SCCWRP). Results marked with * are significantly different from OC.

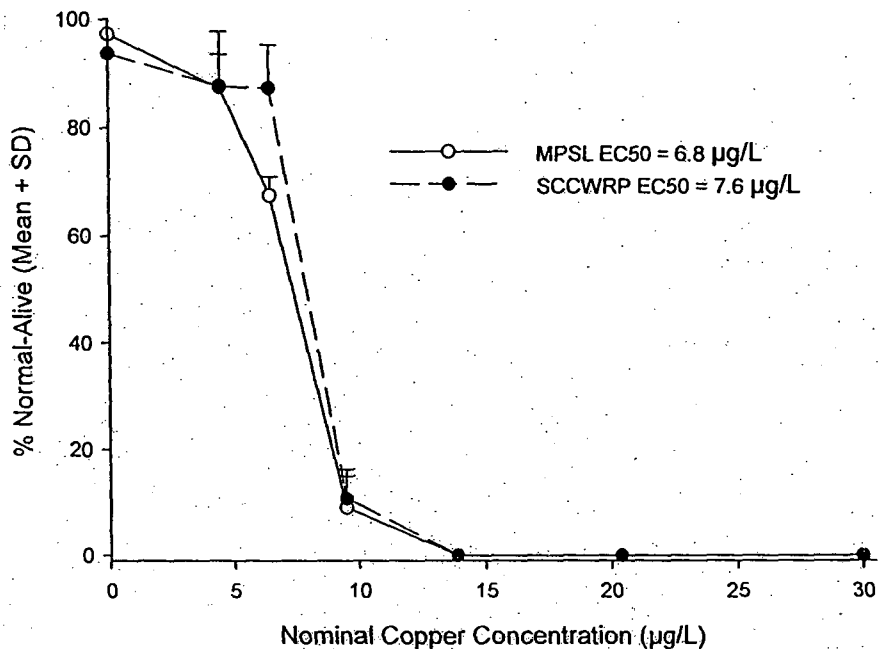

Figure 3. *Mytilus galloprovincialis* dose-response plot of copper reference toxicant exposures to copper for Marine Pollution Studies Laboratory (MPSL) and Southern California Coastal Water Research Project (SCCWRP).

Juvenile Clam Growth

Range Finding

Range finding tests were conducted at SCCWRP to select the concentrations for nonylphenol and the CS dilution samples in the interlaboratory exposures. Two experiments were needed to find concentrations of nonylphenol and CS that showed a dose response. In the first range finding experiment, the three nonylphenol concentrations (10, 100, and 1000) mg/L showed a very similar strong response, each producing growth between 25 to 30 % of control response. Both the CS dilutions tested, 0.5 and 2.0%, had no effect, with growth very similar to the control.

Adjustments were made for the second range finding experiment, decreasing the concentration of nonylphenol by a factor of ten and increasing the concentration of CS in the samples to 5, 10, and 25%. For the second nonylphenol experiment, there was a range of response from 10 to 40% of control growth among the three treatments and these concentrations were selected for use in the intercalibration experiment. In the CS dilution series a strong response in the 25% CS was still not present, therefore concentrations of 10, 25, and 50% CS were tested in the interlaboratory comparison.

Interlaboratory Calibration

None of the laboratories found a significant difference between any of the nonylphenol concentrations and the OC sediment. However, two of the laboratories found that the OC sediment had significantly less growth than the LTH sediment (Figure 4). Therefore, further comparisons were made between all nonylphenol treatments and the LTH sediment (Figure 4). MRRI found a significant difference between all the nonylphenol concentrations and the LTH sample. SCCWRP found a significant difference between the 0.1 and 1.0 mg/kg concentrations and the LTH sample. Weston found no significant difference between any of the nonylphenol samples and the LTH sample.

SCCWRP and Weston found no significant difference in clam growth between LTH and any of the three CS (10, 25, and 50%) dilutions. MRRI found only the 10% CS dilution to be significantly different with 74% of control growth (Figure 5). For all of the laboratories, the growth in the highest two concentrations was similar to or greater than what was observed in the LTH sediment.

The above comparisons detailed whether samples were significantly different from control values, which was deemed a reflection of whether a sample was toxic or not. Another method of comparison is to examine the differences in the growth values themselves between laboratories. For this analysis, the control adjusted means were compared using ANOVAs. There was only one sample (1.0 mg/L nonylphenol) where there was not statistical agreement between the laboratories for clam growth. However, the statistical agreement may be more due to between replicate variability rather than close agreement of the mean growth data from each laboratory. MRRI, SCCWRP and Weston had mean coefficients of variation of 26.6, 35.4, and 42.9, respectively (Table 1). While the mean coefficients of variation were not very different, the differences within individual samples were quite high in many cases. The variation is a little higher than for

the SWI tests where SCCWRP and MPSL had coefficients of variation of about 17 and 36 respectively.

Reference Toxicant

There was a large range of IC50s between MRRI, SCCWRP, and Weston, with 50.2 (95% CI = 43.1 and 58.3), 29.9 (95% CI = 11.8 and 37.5), and 13.5 (95% CI = 10.7 and 19.0) µg/L copper, respectively. All of the laboratories showed decreasing growth with increasing copper concentration (Figure 6). The values above were compared to previous data from Keppler and Ringwood (2002), of the MRRI laboratory. They published an IC50 for copper of 37.6 µg/L from five separate exposures. The IC50 data from MRRI, SCCWRP, and Weston were within one standard deviation of the mean of the five values from the published exposures. Therefore it was concluded that the three laboratories did not differ in reference toxicant outcomes.

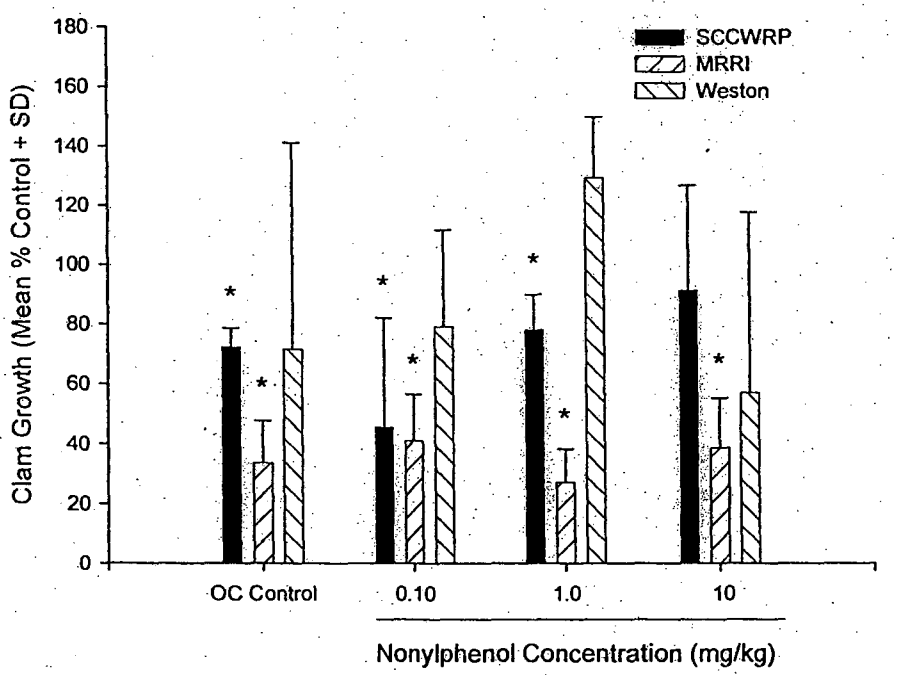

Figure 4. LTH sediment control adjusted juvenile clam 7-day growth test responses to nonylphenol from MRRI, SCCWRP, and Weston. * indicates values significantly different from LTH sediment. Marine Pollution Studies Laboratory (MPSL), Marine Resources Research Institute (MRRI), Weston Solutions (Weston), and Southern California Coastal Water Research Project (SCCWRP).

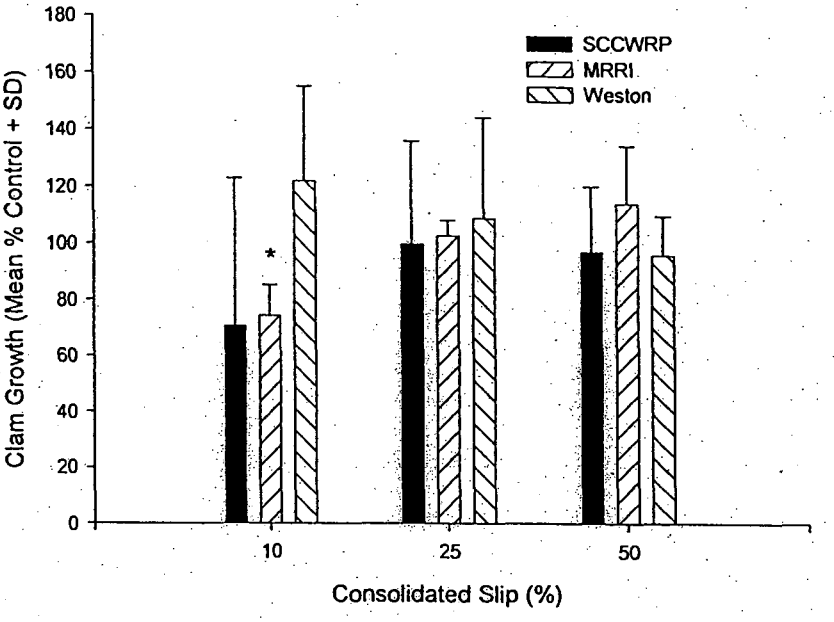

Figure 5. LTH sediment control adjusted juvenile clam 7-day growth test responses to CS dilutions from Marine Resources Research Institute (MRRI), Weston Solutions (Weston), and Southern California Coastal Water Research Project (SCCWRP).. *indicates values significantly different from LTH.

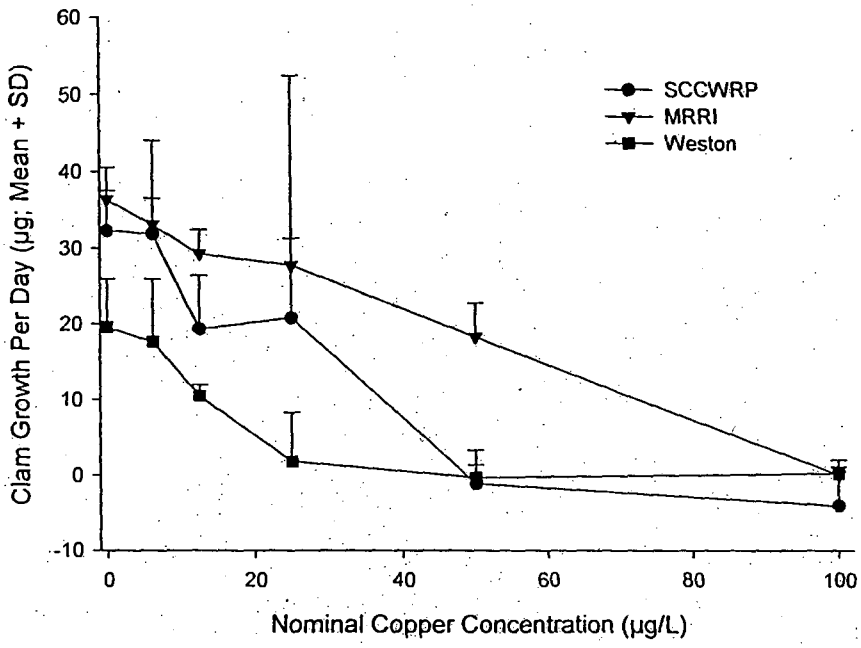

Figure 6. ***Mercenaria mercenaria*** **growth dose-response plot of copper reference toxicant exposures to copper for Marine Resources Research Institute (MRRI), Weston Solutions (Weston), and Southern California Coastal Water Research Project (SCCWRP).**

Table 1. Coefficients of variation for Marine Resources Research Institute (MRRI), Weston Solutions (Weston), and Southern California Coastal Water Research Project (SCCWRP) results for the *Mercenaria mercenaria* 7-day growth endpoint.

	Laboratory		
Sample	MRRI	SCCWRP	Weston
LTH	12.8	6.7	13.9
OC	41.8	8.7	97.2
0.1 mg/L nonylphenol	37.8	80.2	41.3
1.0 mg/L nonylphenol	40.1	15.0	15.8
10.0 mg/L nonylphenol	42.8	38.9	106.5
10% CS	14.5	73.8	27.2
25% CS	5.2	36.1	32.5
50% CS	17.9	23.5	8.6
Mean	26.6	35.4	42.9

DISCUSSION

An important attribute of any toxicity test is that the results are comparable between laboratories using the method. There must be confidence that similar results can be achieved when any given test is used by a reputable laboratory. In the current study comparisons were made for the SWI test using mussel embryos and the *M. mercenaria* test. For each intercalibration, the results of a laboratory highly experienced in the use of the method was compared to laboratories with much less experience.

The SWI test has been used previously by a number of laboratories for field studies. However, no previous intercalibration testing has been conducted. For this study, only two laboratories performed the intercalibration. All but two of the samples tested had a significant difference between the laboratories. In all of the cases where there was a difference, the more experienced laboratory had more sensitive results. No other clear explanation for the differences between the laboratories is apparent. Possible explanations are differences in toxic exposure due to differences in sample handling, differences in interpretation of the microscopic endpoint and differences in animal sensitivity. Given the simplicity of the endpoint determination and the similarity in the EC50 values of the reference toxicant between the laboratories, the last two reasons seem unlikely. While there was no previous interlaboratory testing for the SWI test, there is interlaboratory data for the *M. galloprovincialis* embryo test in aqueous solutions. In that testing, it was found that coefficient of variation between five laboratories was 23.6% for cadmium and 14.4% for lyophilized pulp mill effluent (U.S. EPA 1995). The coefficient of variation from the copper reference toxicant exposure in the current study was 7.9%, which compares favorably with the previous study.

For the *M. mercenaria* test, there was no significant difference in growth among the laboratories for most of the sediment samples. However, the less experienced laboratories encountered a higher degree of between replicate variability than the experienced laboratory. This variability may in part explain the lack of a significant difference among the laboratories. With more familiarity with the procedures, the between replicate variability should decrease, as should the degree of difference in mean growth.

Examining various aspects of the results can help to make an overall assessment of the degree of variability between laboratories in this study. For the SWI testing, the agreement between the laboratories for the nonylphenol spikes was judged to be fair, with one laboratory finding significant toxic response for all three concentrations, while the other found only one. However, both laboratories agreed that there was complete mortality at the highest concentration. There was poor agreement for the CS dilutions with one laboratory finding toxicity in two dilutions and the other finding no toxicity in any. Finally, both laboratories had very good agreement on the reference toxic exposure. Given this mixture of results the overall assessment is that the interlaboratory agreement was assessed as fair.

The *M. mercenaria* results can be judged for interlaboratory agreement using the same method. For the nonylphenol spiked sediments, there was good agreement between two of the laboratories, but poor agreement with the third. For the CS dilutions, there was decent agreement among all three laboratories, however there was very little toxicity associated with the samples. While there was a fairly wide spread in the IC50 data for the reference toxic, data fell within range of variability observed during previous testing. As for the SWI test, it was judged that the overall degree of interlaboratory variability for the *M. mercenaria* test was a rating of fair.

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Comments Submitted to:

State Water Resources Control Board
1001 I Street, 24th Floor
Sacramento, California 95814

COMMENTS ON STATE WATER RESOURCES CONTROL BOARD'S PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE - JANUARY 2011

ATTACHMENTS, VOLUME 2
(TAB 8 – TAB 35)

Submitted by:

LATHAM & WATKINS LLP

Counsel for:

Montrose Chemical Corporation of California

Also on Behalf of:

American Council of Engineering Companies California
Building Industry Legal Defense Foundation
California Building Industry Association
California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Southern California Contractors Association

Submittal Date:

March 15, 2011

Exponent®

EXTERNAL MEMORANDUM

TO:	California Regional Water Quality Control Board, Los Angeles Region, U.S. Environmental Protection Agency, Region 9
FROM:	D. Frederick Bodishbaugh, Ph.D., and Charles Menzie, Ph.D.
CC:	Paul Singarella
DATE:	February 18, 2011
SUBJECT:	Potential for Misuse of California Sediment Quality Objectives in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters

This memorandum addresses the potential use of Sediment Quality Objectives (SQO Part 1) as part of the TMDL process. We refer to these as the *direct effects SQOs* when they apply to benthic invertebrates living within the sediments and *indirect effects SQOs* when they refer to sediment concentrations associated with specific fish tissue levels. We argue here and elsewhere that working backwards from sediments to derive TMDLs introduces substantial uncertainties into the process. We also argue that the SQOs themselves do not translate into reliable TMDLs even presuming sediment should be the driver for TMDL development. Nevertheless, the SQO process represents the approach that the state has developed for such assessments. The TMDL development has not relied on this process and instead defaults to the type of screening-level approach that the California presumably recognized was unreliable for management purposes. The SQO approach was meant to be the more reliable scientific policy alternative to the potential for misuse of screening levels. We refer to Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters as "The System." With respect to this aspect of the TMDL process we make the following points:

- The State of California has developed a step-by-step SQO plan for assessment of sediments. Although discussed with respect to the TMDL process, direct effects SQOs have not been developed and therefore have not been applied to The System.
- Direct effects SQOs are designed to yield information on the degree to which contaminants in sediments impact benthic invertebrates and the degree of confidence regarding contaminant-related impact. Direct effects SQOs do not provide numeric standards, and they cannot be used to establish single chemical effects on benthic organisms. Therefore direct effects SQOs are to

be used as "narrative" standards, in addition to the numeric standards derived through other means. For systems such as ports and harbors that have many different types of contaminants in sediments, the direct effects SQOs in of themselves do not provide a basis for developing contaminant-specific TMDLs.

- The proposed use of direct effect SQOs to develop TMDL standards is not consistent with the SQO assessment procedure, as described by State Board guidance. The proposal oversimplifies interpretation of the SQO station scores, and fails to consider many of the complexities inherent in the use of multiple line of evidence (MLOE) methods to assess potential impacts of sediment chemicals on benthic communities. Most importantly, the proposed use of SQOs makes no provision for establishment of causality between any observed biological effects and specific chemicals or stressors. State Board guidance on implementation of SQOs to support development of response actions is ignored.

- While the indirect effects SQOs process has not been completed for California, sediment target levels are developed for The System for indirect (bioaccumulation) effects intended to be protective of human health. These values are derived using a BAF-based approach that incorrectly infers causation between particular sediment levels and fish tissue levels.

- The uncertainties associated with deriving direct and indirect effects sediment concentrations are not considered in the development of TMDLs. Thus, risk managers are given a false impression about relationships between effects, target levels, and loadings. There is considerable uncertainty throughout the TMDL development process. As a result, the environmental and health benefits of actions designed to meet TMDLs are very uncertain. Explicit consideration of uncertainties would provide risk managers and affected parties with critical information now absent, regarding potential benefits and environmental/health costs of alternative TMDLs and actions.

- A risk zone approach for sediments would provide a means of incorporating uncertainty into the TMDL process and of evaluating benefits and costs of alternative TMDLs and associated actions.

Use Of SQOs In The TMDL Process

The State of California has developed a step-by-step SQO plan for assessment of sediments. This approach involved a stakeholder process and considerable scientific review. This approach is designed to provide a more scientifically reliable basis for assessing impairment that the historical screening-level approach that involves comparisons of sediment concentrations to benchmarks such as the Effects-Range Low (ER-L). The considerable effort invested in developing an alternative approach to screening-levels, underscores the uncertainty and unreliability associated with relying on screening levels for management purposes. But, despite

the recognition of this uncertainty and the availability of an approach that California developed as a more reliable alternative, the TMDL process for The System remains a screening-level assessment inasmuch as it uses ER-Ls as the targets for TMDLs (see Table 3-7 of the TMDL report and associated rationale given in the report). The Staff Report in support of the TMDL[1] makes reference to the use of Sediment Quality Objectives (SQOs) as part of the TMDL process. Specifically, the report discusses the potential application of The California Water Quality Control Board has set a State policy, *The State Water Quality Control Plan for Enclosed Bays and Estuaries – Part 1 Sediment Quality* (SQO Part 1)[2]. SQO Part 1 relates to direct toxic effects on benthic invertebrates, the animals that reside within or on the sediments. While the Staff Report makes reference to the Sediment Quality Plan – Part 2 – Indirect Effects (pp 96-97 of report), the report notes that these SQOs are still in development and, therefore, are not utilized to develop TMDLs at this time. However, the report does make use of a Biota Sediment Accumulation Factor (BAF) to derive sediment target levels for bioaccumulative compounds such as PCBs and DDT that are associated with a health-based tissue target level in fish (Table 3-8 on p 52 of report). We discuss this BAF-based indirect effects approach later in this memorandum.

Limitations and Uncertainties on the Use of Direct Effects SQOs for TMDLs

The draft TMDL approach describes the use of Part 1 SQOs in Sections 3.2.1 and 6.4 of the staff report. The major limitation on their use is that the direct effects SQOs do not provide numeric standards, and they cannot be used to establish single chemical effects on benthic organisms for systems that have many different types of contaminants and differential exposures for these contaminants. Therefore SQOs are intended to be "narrative" standards.

The narrative standards are applied on a station by station basis and include sixteen exposure effects combinations and six resultant station classifications that reflect degree of effects and degrees of confidence that the effect is associated with the presence of contaminants (Table 1).

[1] California Regional Water Quality Control Board, Los Angeles region and U.S. Environmental protection Agency, Region 9. 2010. Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters Toxic Pollutants Total Maximum Daily Loads, Draft December 2010.

[2] State Water Resources Control Board California Environmental Protection Agency. 2009. Water Quality Control Plan for Enclosed Bays and Estuaries – Part 1 Sediment Quality. Effective August 25, 2009.

Table 1. Station classification system for California Part 1 direct effects SQOs (Table 11 from SQO Part 1).

		Severity of Effect			
		Unaffected	Low Effect	Moderate Effect	High Effect
Potential For Chemically-Mediated Effects	Minimal Potential	Unimpacted	Likely Unimpacted	Likely Unimpacted	Inconclusive
	Low Potential	Unimpacted	Likely Unimpacted	Possibly Impacted	Possibly Impacted
	Moderate Potential	Likely Unimpacted	Possibly Impacted or Inconclusive[1]	Likely Impacted	Likely Impacted
	High Potential	Inconclusive	Likely Impacted	Clearly Impacted	Clearly Impacted

[1] Inconclusive category when chemistry is classified as minimal exposure, benthic response is classified as reference, and toxicity response is classified as high.

Source: Available at http://www.swrcb.ca.gov/water_issues/programs/bptcp/docs/sediment/sed_qlty_part1.pdf

Consistent with the Part 1 SQO document, the TMDL report for The System identifies protective conditions that meet the narrative objective for direct effects to include two of the six categories: "Unimpacted" and "Likely Unimpacted" (p. 20 of the report) and these are presented as goals for the TMDL (p. 46 of report). These are the two lowest of the 6 possible Multiple Line Of Evidence (MLOE) station score categories. However, The TMDL report does not include a discussion of a third category that also may be deemed protective according to the Part 1 SQO document[3]:

> *The Water Board shall designate the category **Possibly Impacted** as meeting the protective condition if the studies identified in Section VII.F demonstrate that the combination of effects and exposure measures are not responding to toxic pollutants in sediments and that other factors are causing these responses within a specific reach segment or water body. In this situation, the Water Board will consider only the Categories **Likely Impacted** and **Clearly Impacted** as degraded when making a determination on receiving water limits and impaired water bodies described in Section VII.*

This caveat indicates that there is uncertainty regarding the classification of contaminant-related effects on sediment dwelling organisms. And, as we describe later, the degree of effect and confidence regarding their attribution to contaminants (i.e., unimpacted to clearly impacted) provides one basis for defining risk zones and the appropriateness of various degrees of intervention or controls to attain goals.

The System is an urban harbor that is regularly disturbed by a variety of physical and chemical stressors. Navigational dredging is carried out in areas that have silted in and these areas would

[3] Part 1 SQO document p. 11.

be favored depositional areas (sediment traps). The U.S. Environmental Protection Agency recognizes the importance of considering the nature of a water body when attempting to achieve sediment management goals such as those contemplated in the TMDL report. These considerations are described in the Agency's Sediment Management Guidance[4]. This guidance is germane to the TMDL because dredging of sediments is being contemplated as an action to meet goals; that type of action is essentially a sediment remediation. With respect to sediment management, Section 2.5 of the guidance discusses watershed considerations. The essential point of this section is to understand the nature of the water body and current and future uses. The guidance recognizes that there are differences among water bodies and these differences are important to consider when evaluating the appropriateness of various remedial alternatives. The specific introductory language in this section of the Agency guidance is:

> *A unique aspect of contaminated sediment sites is their relationship within the overall watershed, or drainage area, in which they are located. Within the watershed there often is a spectrum of issues that the project manager may need to consider. Foremost among them at many sites is to work with the state to ensure that fish consumption advisories are in place and well publicized. In addition, project managers should understand the role of the contaminated water body in the watershed, including the habitat or flood control functions it may serve, the presence of non-site-related contaminant sources in the watershed, and current and reasonably anticipated or desired future uses of the water body and surrounding land.*

A large and operational urban port is a very different type of environment for benthic invertebrates than is an undisturbed coastal embayment. This difference is a factor that should be considered for assessment and risk management. In light of this difference, the TMDL report is very restrictive with respect to evidence for judging effects on benthic invertebrates. This is reflected in the target individual lines of evidence station scores specified for the biological Lines of Evidence (LOEs). The report identifies that the benthic community should resemble either "reference" or "low disturbance" (p. 47). These are the lowest two of four possible benthic LOE categories. The target toxicity LOE score is "nontoxic" (p. 49). This is the lowest of four possible toxicity LOE categories. Considering the uncertainty associated with factors influencing benthic invertebrates in a large operating urban harbor, it may be more appropriate to consider a range of biological states and/or degrees of toxicity. This would allow for a valid consideration of a range of goals and associated range of interventions. We discuss this later with respect to establishing risk zones. We also observe that the SQO chemistry LOE does not appear to factor into the proposed TMDL standards, though it must be calculated in order to assess the MLOE station score.

The stated objectives may not be realistic or attainable for a harbor system and if those objectives are not met the TMDL target defaults to a numeric ER-L, a very restrictive value for

[4] U.S. EPA. 2005. Contaminated sediment remediation guidance for hazardous waste sites. EPA-540-R-05-012. U.S. Environmental Protection Agency, Washington, DC.

which the limitations are discussed elsewhere. The TMDL report states that the proposed numeric TMDL objectives are expected to attain the narrative objectives (p. 88):

> *Attainment of the narrative sediment quality objective may occur either through demonstrating the water body has achieved the desired qualitative condition [clearly unimpacted or likely unimpacted] or the quantitative condition; i.e., if the ambient sediment chemistry levels within a waterbody are equal to or below the sediment quality values.* (p.90).

> *Compliance with the TMDL shall be determined through water, sediment, and fish tissue monitoring and comparison with the TMDL waste load and load allocations and numeric targets. Compliance with the sediment TMDL for metals and PAH compounds shall be based on achieving the loads and waste load allocations or, alternatively, demonstrating attainment of the SQO Part 1 through the triad/multiple lines of evidence approach outlined therein.* (p.116).

Inconsistencies Between the Use of SQOs as TMDL Standards and SQO Guidance

Direct effect SQOs are not in any way analogous to sediment quality values or concentration benchmarks that are associated, even theoretically, with a chemical adverse effect threshold. Rather, the SQO method establishes a framework for assessing potential impacts of sediment chemicals on benthic communities, through the use of Triad data (synoptic measurement of sediment chemistry, toxicity, and benthic community condition). In and of themselves, the MLOE station scores generated by the SQO assessment paradigm do not establish causality between observed biological effects and any given sediment chemical. They cannot therefore be used to identify "safe" chemical levels, or to establish cleanup triggers or target levels for contaminants. Part 1 SQOs were never intended to be used as a standalone cleanup indicator, but rather as a tool to identify sediments for which a sediment management strategy may be needed. With respect to appropriate use of Part 1 SQOs to establish a management strategy for specific pollutants, the method document states the following: [5]

> *In order to demonstrate an exceedance of the proposed SQO, a toxic pollutant or pollutants must be identified. Additional studies would be required to identify the specific cause.*

The guidance goes on to describe and discuss toxicity identification evaluation methods. This process requires additional evaluation of site-specific information and significant application of professional judgment. Steps required include an evaluation of confidence in the SQO assessment, consideration of the magnitude of exceedances, and stressor identification. None of these essential steps has been incorporated into the proposed TMDL narrative standards based on Part 1 SQOs. The Part 1 SQO method document further states that bioavailability of

[5] Part 1 SQO document p. 119.

measured contaminants must be considered in the development of potential response actions, including TMDL development:[6]

> *If stressor identification is performed and a stressor is identified, a logical application would be the development of biologically relevant guidelines that could be applied to support TMDL development or remediation goals. Guideline development would account for site and receptor specific factors that control bioavailability. Adopting sediment quality guidelines to fulfill this role does not account for these factors.*

In summary, the TMDL's proposed use of Part 1 SQO MLOE station scores as strict indicators of a need for dredging fails to incorporate any of the required implementation considerations, and is inconsistent with the intended use of the SQOs, as promulgated by The Board and described in State guidance.

Uncertainty Associated with TMDL Target Sediment Concentration for Indirect Effects

Sediment target concentrations are developed in the TMDL process for bioaccumulative compounds such as PCBs and DDT. We use the TMDL process for DDT to illustrate the uncertainties in this approach. The TMDL document derives a DDT sediment target level of 1.9 µg/g for protection of human health based on a fish tissue concentration of 21 µg/g. There are two areas of uncertainty with regard to this derivation. First, there is an implication of proportional causation between the sediment DDT levels and fish tissues. In other words, the TMDL document presumes that a sediment concentration of 1.9 µg/g will cause a fish tissue level of 21 µg/g. This reflects a presumed Biota to Sediment Accumulation Factor (BAF) of 11 (21 divided by 1.9). Second, the TMDL presumes that a fish tissue concentration of 21 µg/g is the only value that should be considered by risk managers for TMDL development.

The presumed causal relationship between fish tissues and sediments is attributed to a report by SFEI (2007)[7]. This report considers two case studies – Newport Bay and San Francisco Bay. The TMDL document appears to rely mainly on the case study for Newport Bay but both case studies are important to consider with respect to judging the uncertainty in the process. The SFEI report does a good job at identifying the uncertainties associated with the derivation process. However, none of these uncertainties are discussed or carried into the TMDL draft. Examples of uncertainties highlighted in the SFEI report that are especially relevant for the development of TMDLs include:

- Because of limited data for Newport Bay, it is not possible to develop meaningful spatially explicit statistical models of tissue vs. sediment

[6] Part 1 SQO document p. 121

[7] Greenfield, B. K., A. R. Melwani, J. J. Oram, and S. M. Bay. 2007. Indicator development and framework for assessing indirect effects of sediment contaminants. SFEI Contribution #524. San Francisco Estuary Institute, Oakland, CA

chemistry relationships. Several fish species have relatively wide average estimates for DDT. In particular, standard errors of the mean are wide for diamond turbot, spotted sand bass, California corbina, arrow goby, and pacific staghorn sculpin. Generally, the standard error of the mean estimates for these species is larger than the standard error taking the combined average of all fillet samples, all whole body samples, or all samples.

- Newport Bay differs from many estuaries in having a significant proportion of primary production due to benthic algae. Simulation results indicated consistently higher predicted contaminant concentrations for benthic algae, than for phytoplankton. Although substantial efforts were made to develop model parameters appropriate for benthic algae, local data are lacking.

- For Newport Bay, there was uncertainty associated with several technical policy decision and technical parts of the analyses. These were associated with various scenarios that incorporated variable assumptions. (These scenarios were not described in the TMDL document.) The following aspects introduce moderate to high uncertainty to the analyses: risk assessment assumptions and averaging method (arithmetic vs. geometric) substantially influenced sediment thresholds. Changing to geometric averaging methods (Scenario G), increasing allowable cancer risk tenfold (Scenario E), and switching to subsistence fisher consumption rates (Scenario D) each caused an order of magnitude or greater difference in sediment thresholds for DDTs. These changes often resulted in a different categorization of the majority of sediments. For example, when allowable increased cancer risk was switched from one-in-one hundred thousand to one-in-ten thousand (Scenario E), the percent of sediment samples exceeding the high human health threshold for DDTs changed from 63% to 0%.

- For humans, Scenarios D and E caused the greatest effects on outcome. These scenarios changed standard risk assessment parameters. Scenario D changed assumptions regarding the definition of the target population to protect and Scenario E changed the acceptable level of increased carcinogenic risk. Both of these parameters are strongly influenced by policy decisions, which often vary among water bodies based on agency judgments. These findings support the need for substantial efforts to engage stakeholders in framework application, to adequately resolve these policy issues.

The TMDL document indicates in Table 3-8 that the BAF for DDT in The System is ~11. However, this particular value is not identified in either of the case studies included in SFEI (2007) and the TMDL document does not describe how it selected a BAF of ~11. The BAF values associated with the case studies are variable but the “average” BAF for both case studies is less than 11. For Newport Bay, a sediment concentration of 1.9 µg/g is associated with a “low tissue threshold” of 9.8 µg/g in fish; this is an apparent BAF of 5 (see Table 4-19 in SFEI, 2007). For human resident prey only, the apparent BAF for Newport Harbor is ~1.8 (see Table

4-19 in SFEI, 2007). The apparent BAFs for San Francisco Bay (around 5 on average) are similarly lower than the one used in the TMDL document. Therefore, even presuming that the sediments were the cause of DDT in fish tissues, the selection of the BAF for The System inflates the sediment contribution relative to that for the case studies on which the TMDL process relies.

The TMDL document also fails to communicate the uncertainty described in SFEI (2007) concerning the contribution of sediments to tissue levels. Although the TMDL document assumes a proportional relationship between sediment concentrations and fish tissue levels, the modeling results included in SFEI (2007) would argue that there is not such a one-to-one correspondence. Instead the SFEI (2007) report states on p. 145 that:

> *The case studies also revealed a number of technical uncertainties and data limitations. Mechanistic model simulations indicated that a significant portion of biota exposure to DDTs stemmed from dissolved and particulate compounds in the water column. In general, modeled BAFs were highly sensitive to water column concentrations, highlighting the potential uncertainty regarding the ultimate source of pollutants. Although it is likely that a large portion of the water column concentrations were linked to direct sediment resuspension, direct loading from the watershed and upstream rivers should also be considered.*

The larger contribution of water column concentrations to fish tissue levels for DDT-related compounds is shown in the following figure for Newport Bay (Figure 1). This indicates that the assumption of one-to-one causal relationship between fish tissues and sediments is incorrect and that use of a BAF as a tool for back-calculation of sediment target levels is flawed. As the figure shows, water exposures are the most important source of tissue levels. These would include dissolved and particular DDT-related compounds in the water column. The figure shows that sediments vary in contribution from 0% to 10% for the Jack Smelt up to ~25% for white croaker. While sediments may be a contributor to the water column, the modeling results indicate that it is incorrect to presume a proportional relationship between fish tissue concentrations and sediment concentrations for DDT-related compounds. Furthermore, applying an apparent BAF that presumes a 100% contribution of sediments will result in target levels that likely is low by a factor of four or more. The TMDL development process for The System is not based on the nature of the system or on what is currently known about the relationships among exposures and fish tissue levels. Those exposures vary spatially (exposure within The System v external to the system) and with respect to media and the nature of loads (water v sediment; in-place contaminants v external loads). Thus, the calculations are not based on reality. As such, they are unreliable for management pourposes.

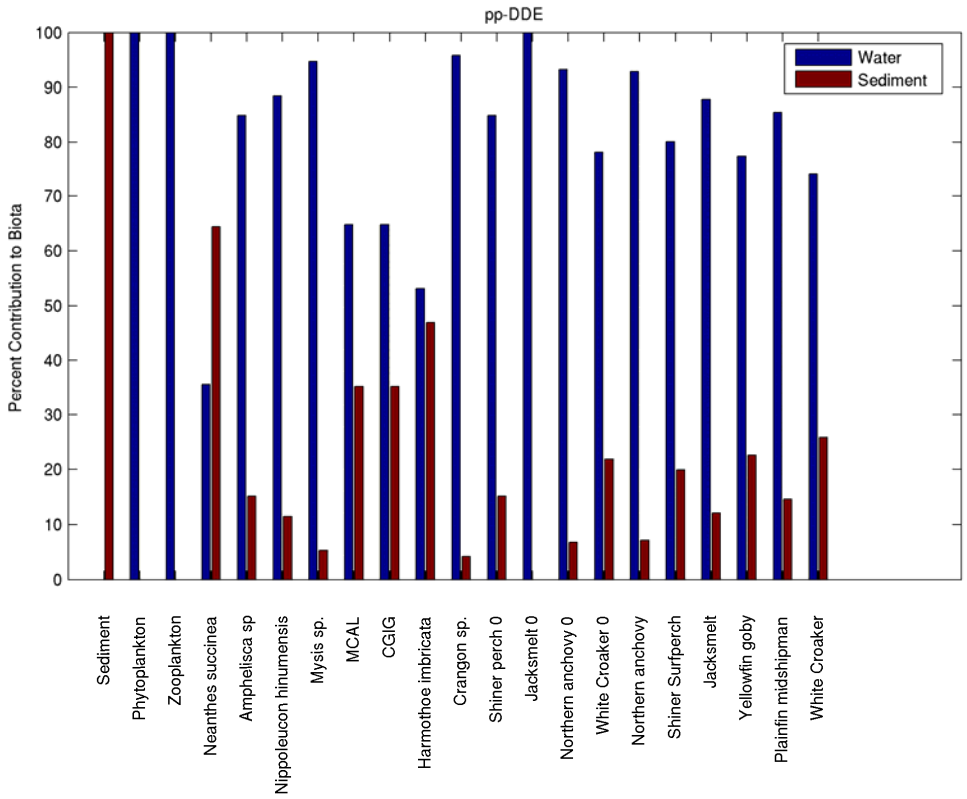

Figure 1. Relative contribution of sediments and water to fish tissue levels in Newport Bay (presentation by Ben Greenfield of SFEI to the Santa Ana Water Control Board).

The second source of uncertainty involves selecting and presenting single values to represent protective levels for human health. The TMDL document utilizes a fish tissue level of 21 µg/g wet weight for DDT. This value is associated with an upper bound estimate for a one-in-one million risk of cancer. However, risk managers in California and elsewhere have relied upon other values to make informed management decisions. Typically, a risk manager will weigh relative risks and benefits of actions. But when one value is put forward as the only target, as it has in the case of the TMDL document, the opportunity to make informed management decisions is greatly limited. A few examples illustrate the arbitrary aspects of putting forward one particular value – 21 µg/g. The most obvious contrast is the OEHHA screening value for DDT in fish tissues, 100 µg/g, which is ~ five times higher than the value used to derive TMDL target levels. This screening value is used for the TMDL development for Newport Bay. And, there are many other values that could have been selected as described in the SFEI (2007) report. Rather than limit the risk manager to a single value for a single risk level, it is more common to discuss a range of values. For example, the one-in-one hundred thousand lifetime incremental risk tissue concentration would be 10 times higher, i.e., 210 µg/g. A range in target

levels with associated risk information would provide the risk manager with insight and opportunity to identify meaningful interventions and controls for protecting human health.

Uncertainties in Deriving Target Levels and TMDLs Have Not Been Considered

The TMDL document does not consider uncertainties in the various components of the assessment or in the derivation of TMDLs. To a large extent the TMDL document deals with uncertainty by selecting bounding values such as ER-Ls for direct effects and the presumption that contaminants in sediments are the only contributor to contaminants in fish for indirect effects. While such an approach may be useful as a screening analysis, it suffers greatly when it is used as a basis for making major engineering judgments. The actions associated with these judgments carry with them potentially significant health, safety, environmental, and economic costs that the TMDL document does not acknowledge. Because the TMDL document does not share information on uncertainty and is essentially constructed as a screening-level analysis, it places the risk manager in a box. There is no reasonable range of risks and associated confidence upon which to compare the efficacy and appropriateness of interventions. Instead, TMDLs are presented with high precision (e.g., multiple decimal places) implying that there is an in-depth understanding on the part of the technical analysts that prepared the staff report. That in-depth understanding is missing from the report.

Information is available for conveying uncertainty to the risk manager. For example, instead of selecting bounding estimates, the TMDL document could discuss the range of possible target levels with attendant degrees of confidence. Some of these target levels may already be met or could be met in a short period of time while others might require some additional control. However, because the TMDL document is silent on uncertainty, the risk manager has not been provided with an opportunity to review and consider such information. Instead, the TMDL document leads only to engineering solutions that are substantially invasive and may carry unaddressed health and environmental costs.

"Risk Zones" for Sediments Provide a Means for Incorporating Uncertainty

The concept of risk zones is evolving as a way of considering alternative remedial strategies that take into account that there are variable degrees of risk and also confidence about risks. Therefore, risk zones provide a way to incorporate uncertainties in an explicit way. These can be established for direct and indirect (bioaccumulative) effects or risks. For example, each of the targets for DDT has an associated range that captures degree of effects or risks and attendant uncertainty. Therefore, rather than selecting only the bounding (typically lowest) value, the target could be expressed as a range or as several values. With respect to the Part 1 direct effects SQOs, these too could include the full range. This approach avoids the binary (risk v no risk) approach associated with the selection of a single TMDL target concentration.

The ranges in target levels can then be used to develop isopatch maps of sediments that reflect the regions associated with the intervals of target concentrations for contaminants or direct effects levels. That provides the risk manager with a visual representation of surface areas of

The System for which a variety of management approaches can be contemplated. Such an approach does not nullify or undermine the TMDL development process and is entirely consistent with the history of water management in California. This is certainly evidenced by the six classification categories currently used for the Part 1 direct effects SQOs and with the water, fish, and sediment target levels associated with various human health risk levels.

Professional Profiles

D. Frederick Bodishbaugh

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Dr. Rick Bodishbaugh is a Managing Ecotoxicologist in Exponent's EcoSciences practice. He has 19 years of diverse experience in aquatic toxicology research, chemical and site assessment, ecological risk assessment (ERA) in aquatic and terrestrial systems, and natural resource damage assessment (NRDA). His specific areas of technical expertise include fish and wildlife toxicity assessment, resource/habitat equivalency analysis (REA/HEA), bioavailability of chemical contaminants in aquatic and terrestrial ecosystems, and chemical structure-activity relationships. Dr. Bodishbaugh is experienced in evaluating the effects of contaminated soil, groundwater, surface water, and sediments on ecological receptors. He has conducted assessments of chemical risk at dozens of sites for energy, petrochemical, pulp and paper, manufacturing, and mining industry clients. He is intimately familiar with federal, regional, and various state guidance and standards or practice for ERA under common regulatory frameworks. He is also experienced in evaluating and interpreting field bioaccumulation and laboratory toxicity bioassay data for use in assessing ecological risk. He is well versed in the environmental toxicology and assessment of metals and persistent organic pollutants, especially PCBs and PAHs.

Charles A. Menzie

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Dr. Menzie is a Principal Scientist and Director of Exponent's EcoSciences practice. His primary area of expertise is the environmental fate and effects of physical, biological, and chemical stressors. He has investigated a broad range of contaminants, including solvents (TEC, PCE, TCA, and others), persistent chlorinated compounds such as DDT and PCBs, hydrocarbons, cyanides, and metals. Dr. Menzie is recognized as one of the leaders in the field of risk assessment and was awarded the Risk Practitioner Award by the Society for Risk Analysis. He has served on the Councils of SRA and the Society of Environmental Toxicology and Chemistry. Dr. Menzie has participated in numerous national and regional scientific panels and advisory groups and has led many scientific peer reviews for industry and for government.

He has taken the lead in developing guidance documents for industry and government and has focused on methods that are workable and acceptable to a broad range of parties. He has developed and applied a formal causal-analysis methodology for assessing causation in cases of environmental impairment and contributions of chemical contamination.

Incidence of Adverse Biological Effects Within Ranges of Chemical Concentrations in Marine and Estuarine Sediments[1]

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ABSTRACT / Matching biological and chemical data were compiled from numerous modeling, laboratory, and field studies performed in marine and estuarine sediments. Using these data, two guideline values (an effects range-low and an effects range-median) were determined for nine trace metals, total PCBs, two pesticides, 13 polynuclear aromatic hydrocarbons (PAHs), and three classes of PAHs. The two values defined concentration ranges that were: (1) rarely, (2) occasionally, or (3) frequently associated with adverse effects. The values generally agreed within a factor of 3 or less with those developed with the same methods applied to other data and to those developed with other effects-based methods. The incidence of adverse effects was quantified within each of the three concentration ranges as the number of cases in which effects were observed divided by the total number of observations. The incidence of effects increased markedly with increasing concentrations of all of the individual PAHs, the three classes of PAHs, and most of the trace metals. Relatively poor relationships were observed between the incidence of effects and the concentrations of mercury, nickel, total PCB, total DDT and p,p'-DDE. Based upon this evaluation, the approach provided reliable guidelines for use in sediment quality assessments. This method is being used as a basis for developing National sediment quality guidelines for Canada and informal, sediment quality guidelines for Florida.

Chemical analyses indicate that coastal sediments in some areas of North America are contaminated (Bolton and others 1985, O'Connor 1991, US NOAA 1991, Wells and Rolston 1991, Goyette and Boyd 1989). However, data on the mixtures and concentrations of contaminants in sediments, alone, do not provide an effective basis for estimating the potential for adverse effects to living resources. Moreover, interpretive tools are needed to relate ambient sediment chemistry data to the potential for adverse biological effects. A variety of biological measures (including toxicity and/or bioaccumulation tests) can be performed to determine the biological significance of sediment-associated contaminants (Burton 1992). Furthermore, numerical, effects-based, sediment quality guidelines can be used as screening tools to evaluate sediment chemistry data and to identify and prioritize potential problem areas (Di Toro and others 1991, Persaud 1992, MacDonald 1993, Long and Morgan 1990, Smith and MacDonald 1992, US EPA 1989a, 1992a). In this respect, effects-based guidelines can be used to help identify those areas in which the potential for biological effects is greatest.

KEY WORDS: Sediment quality guidelines; Ecological risk assessment; Contaminants; Biological effects; Marine; Estuarine

[1]The methods and guidelines presented in this report do not necessarily represent the policy of the National Oceanic and Atmospheric Administration, Environment Canada, or Florida Department of Environmental Protection.

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A variety of biological effects-based approaches to the development of sediment quality guidelines have been reviewed by many investigators (US EPA 1989a, 1992a, Adams and others 1992, Chapman 1989, MacDonald and others 1992). These approaches can be grouped into three categories: equilibrium-partitioning modeling, laboratory bioassays, and field studies. Each approach has particular strengths and weaknesses and each defines guidelines in different ways. Thus far, there is no general agreement as to which approach will provide the most reliable, flexible, and credible guidelines for evaluating sediment quality. However, sediment quality guidelines derived from the combination of the results of multiple methods have been recommended for a broad range of applications (Adams and others 1992, US EPA 1989b, Lorenzato and others 1991).

Using data available from all the major approaches to the development of effects-based criteria, Long and Morgan (1990) prepared informal guidelines for use by the National Oceanic and Atmospheric Administration (NOAA). Subsequently, the data base with which these values were prepared was updated and expanded and the approach was refined (MacDonald 1993, Smith and MacDonald 1992). In both the NOAA (Long and Morgan 1990) and Florida (MacDonald 1993) studies, two guideline values were developed for each chemical. These values defined three ranges in chemical concentrations that were anticipated to be: (1) rarely, (2) occasionally, or (3) frequently associated with effects. The identification of ranges in chemical concentrations has been recommended in the development of sediment quality criteria (US EPA 1992b).

The objectives of the present study are: (1) to present updated guideline values based upon the expanded data base, (2) to quantify the percent incidence of adverse biological effects associated with the guidelines, and (3) to compare the guidelines with those developed with other data or methods. In this paper we determined the percent incidence of effects as a measure of the "accuracy" of the guidelines.

Methods

The methods used in this study have been described in detail (Long and Morgan 1990, MacDonald 1993, Smith and MacDonald 1992, Long 1992) and will be only summarized here. Sediment chemistry and biological effects data from numerous reports were assembled to support the derivation of the guidelines. The data base used by Long and Morgan (1990) was refined by excluding data from freshwater studies and including data from additional sites, biological test end points, and contaminants (MacDonald 1993, Smith and MacDonald 1992). Briefly, the approach involved three steps: (1) assemble, evaluate, and collate all available information in which measures of adverse biological effects and chemical concentrations in sediments were reported; (2) identify the ranges in chemical concentrations that were rarely, occasionally, or frequently associated with effects; and (3) determine the incidence of biological effects within each of the ranges in concentrations for each chemical as an estimate of guideline accuracy.

Development of a Biological Effects Database for Sediments

A biological effects database for sediments (BEDS) was developed to compile and integrate chemical and biological data from numerous studies conducted throughout North America. Nearly 350 publications were reviewed and screened for possible inclusion in the BEDS. Data from equilibrium-partitioning modeling, laboratory spiked-sediment bioassays, and field studies of sediment toxicity and benthic community composition were critically evaluated. Only matching, synoptically collected biological and chemical data from marine and estuarine studies were included in the database. Data were excluded if the methods were not clearly described. Data were excluded if sediments were frozen before toxicity tests were initiated or if toxicity of controls was higher than commonly acceptable. If there was less than a tenfold difference in the concentrations of all contaminants among sampling stations, all data from that particular field study were excluded. The tenfold criterion was selected to ensure that data were included in the BEDS only from studies in which significant contaminant gradients were reported. Furthermore, data were excluded if the chemical analytical procedures were inappropriate for determining total concentrations in bulk sediments; for example, trace metals data were excluded if strong acid digestions were not used. The majority of the data sets that were excluded were those in which either no biological data or no chemical data were reported. A total of 89 reports met all the screening criteria and were included in the BEDS. The screening criteria and their use were described previously (MacDonald 1993, Smith and MacDonald 1992). The potential limitations of using data "encountered" from many different studies have been described (Long 1992).

The data entered into the BEDS were expressed on a dry weight basis. Only a minority of the reports included measures of factors that are thought to influ-

ence bioavailability (e.g., grain size, total organic carbon, acid-volatile sulfides). Sediment quality guidelines derived from the equilibrium-partitioning approach (US EPA 1988) were converted from units of organic carbon to units of dry weight, assuming a total organic carbon (TOC) concentration of 1.0%. These conversions were based upon a TOC concentration of 1.0% since the overall mean TOC concentration in the BEDS was 1.2%. Data from spiked-sediment bioassays were incorporated directly into the BEDS.

Guideline values derived using the apparent effects threshold (AET approach, Barrick and others 1988) and national screening level concentration (SLC approach, Neff and others 1986) were entered into the BEDS as reported. AET and SLC values represent large amounts of data compiled from multiple surveys. Therefore, extremely high and extremely low concentrations in some parts of study areas used to produce these values may be ameliorated by highs and lows in other regions, resulting in intermediate concentrations. Raw data from other individual field surveys that passed the initial screening steps were evaluated in "co-occurrence analyses" with either of two methods (Long 1992). If the statistical significance of the data was reported, then the mean chemical concentrations in the statistical groups (i.e., toxic and nontoxic) were compared. If no such statistical evaluations were reported, the frequency distributions of the biological data were examined, and mean concentrations in subjectively determined groups of samples were compared (e.g., most toxic versus least toxic). The extreme high and low concentrations reported in individual studies, generally performed over relatively small spatial scales, were not masked by merging data from other studies.

To maximize the broad applicability of the guidelines, a wide variety of measures of adverse biological effects was included in the BEDS. The kinds of adverse effects included: (1) measures of altered benthic communities (depressed species richness or total abundance), significantly or relatively elevated sediment toxicity, or histopathological disorders in demersal fish observed in field studies; (2) EC_{50} or LC_{50} concentrations determined in laboratory bioassays of sediments spiked with single compounds or elements; and (3) toxicity predicted by equilibrium-partitioning models. All of the measures of effects were treated as if equivalent. However, by screening prospective data sets and including only those biological data that were in concordance with chemical gradients, the prevalence of data from relatively insensitive measures of effects was minimized.

Each entry was assigned an "effects/no-effects" descriptor. An entry was assigned an "effects" descriptor (identified with an asterisk in the data tables) if: (1) an adverse biological effect, such as acute toxicity, was reported; and (2) concordance was apparent between the observed biological response and the measured chemical concentration.

The documentation supporting each BEDS record included the citation, the type of test or biological effect observed or predicted, the approach that was used, the study area, the test duration (if applicable and reported), the species tested or the benthic community considered, the total organic carbon (TOC) and acid-volatile sulfide (AVS) concentrations (if reported), and the chemical concentration.

In our co-occurrence analyses of field-collected data entered into BEDS, an effects descriptor was assigned to data entries in which adverse biological effects were observed in association with at least a two-fold elevation in the chemical concentration above reference concentrations. Either "no gradient," "small gradient," or "no concordance" descriptors were assigned when no differences between stations were reported in the concentration of the chemical of concern, when mean chemical concentrations differed by less than a factor of two between the groups of samples, or when there was no concordance between the severity of the effect and the chemical concentration, respectively. In these cases, we assumed that other factors (whether measured or not) were more important in the etiology of the observed effect than the concentration of the contaminant considered. Finally, a "no effects" descriptor was applied to biological data from background, reference, or control conditions.

Collectively, the effects data sets from the modeling, laboratory, and field studies were assigned an asterisk in the ascending tables and used to derive the guidelines. All of the effects data were given equal weight in the guidelines derivation. Collectively, data assigned no gradient, small gradient, no concordance, and no effects descriptors were regarded as the no-effects data set.

Derivation of Sediment Quality Guidelines

For each chemical, the data from BEDS were retrieved and arranged in ascending order of concentration in a tabular format. These ascending data tables, as reported by Long and Morgan (1990) and updated by MacDonald (1993) and Smith and MacDonald (1992), summarized the available information for each chemical or chemical group that was considered.

Table 1. Summary of available data on effects of sediment-associated acenaphthene (ppb) in coastal sediments

Concentration (±SD)	Area	Analysis type[a]	Test duration[b]	End point measured[c]
1	Puget Sound, WA	COA		Low prevalence of hepatic cellular alterations (0%)
1	Puget Sound, WA	COA		Low prevalence of hepatic lesions (0%)
1	Puget Sound, WA	COA		Low prevalence of hepatic idiopathic lesions (32.5%)
<3	Halifax Harbour, NS	COA	10 d	Significantly toxic (61.7 ± 12.5% mortality)
<3.5 ± 1	Halifax Harbour, NS	COA	10 d	Not significantly toxic (5.2 ± 3.5% mortality)
<3.5 ± 1	Halifax Harbour, NS	COA	20 d	Not significantly toxic (1 ± 2% mortality)
3.92 ± 1.59	Southern California	COA	10 d	Significantly toxic (51.7% mortality)
<5	Halifax Harbour, NS	COA	10 d	Not significantly toxic (3% mortality)
<5	Sidney Tar Pond, NS	COA	10 d	Not significantly toxic (4% mortality)
<5	Sidney Tar Pond, NS	COA	10 d	Not significantly toxic (3% mortality)
6.92 ± 11.8	Southern California	COA	10 d	Not significantly toxic (23.2% mortality)
<8.8 ± 5.3	Sidney Tar Pond, NS	COA	20 d	Not significantly toxic (8 ± 5.66% mortality)
9	San Francisco Bay, CA	AETA	48 h	San Francisco Bay AET
<12.5	Sidney Tar Pond, NS	COA	10 d	Significantly toxic (100% mortality)
<12.5	Sidney Tar Pond, NS	COA	10 d	Significantly toxic (100% mortality)
16				**ER L (10th percentile)**
16	California	AETA	48 h	California AET
16	California	AETA		California AET
16	Northern California	AETA		Northern California AET
<23.5	Sidney Tar Pond NS	COA	20 d	Significantly toxic (52% mortality)
<30.8 ± 25.6	Halifax Harbour, NS	COA	10 d	Not significantly toxic (6.8 ± 7.31% mortality)
<30.8 ± 25.6	Halifax Harbour, NS	COA	10 d	Not significantly toxic (8.5 ± 6.06% mortality)
<30.8 ± 25.6	Halifax Harbour, NS	COA	20 d	Not significantly toxic (0.7 ± 1.63% mortality)
50	Burrard Inlet, BC	SQO		Sediment quality objectives
56	Northern California	AETA	10 d	Northern California AET
56	California	AETA	10 d	California AET
56	San Francisco Bay, CA	AETA	10 d	San Francisco Bay AET
56.7 ± 70	Commencement Bay, WA	COA	48 h	Least toxic (15.1 ± 3.1% abnormality)
63	Puget Sound, WA	AETA		PSDDA screening level concentration
85.9 ± 97	Commencement Bay, WA	COA	10 d	Least toxic (12.5 ± 4.5% mortality)
119 ± 105	Commencement Bay, WA	COA	48 h	Moderately toxic (23 ± 2.3% abnormality)
127 ± 117	Commencement Bay, WA	COA	10 d	Moderately toxic (26 ± 5.2% mortality)
150	Eagle Harbor, WA	COA	4 d	LC_{50}
160	Puget Sound, WA	SQG		Chemical criteria
247 ± 147	Burrard Inlet, BC	COA	10 d	Not toxic (4.5 ± 3.02% emergence)
247 ± 147	Burrard Inlet, BC	COA	10 d	Not toxic (5.21 ± 3.61% emergence)
283 ± 140	Burrard Inlet, BC	COA	10 d	Not toxic (97.2 ± 2.84% reburial)
283 ± 140	Burrard Inlet, BC	COA	10 d	Not toxic (8.9 ± 2.99% mortality)
293 ± 73.8	Elizabeth River, VA	COA	96 h	No significant change in respiration rate
306 ± 604	Commencement Bay, WA	COA	48 h	Highly toxic (44.5 ± 19% abnormality)

The distributions of the effects data were determined using percentiles (Byrkit 1975). Two values were derived for each chemical or chemical group. The lower 10th percentile of the effects data for each chemical was identified and referred to as the effects range-low (ERL). The median, or 50th percentile, of the effects data was identified and referred to as the effects range-median (ERM). Percentiles of aquatic toxicity data were used by Klapow and Lewis (1979) to calculate marine water quality standards; the authors noted that this approach tended to minimize the influence of single (potentially outlier) data points on the development of guidelines. Environment Canada and Florida Department of Environmental Protection used a slight modification to this method, the rationale for which has been documented (MacDonald 1993, Smith and MacDonald 1992).

Determination of Percent Incidence of Adverse Biological Effects

The two guideline values, ERL and ERM, delineate three concentration ranges for a particular chemical. The concentrations below the ERL value represent a minimal-effects range; a range intended to estimate conditions in which effects would be rarely observed. Concentrations equal to and above the ERL, but be-

Species	Life stage[d]	Effects/no effects[e]	TOC (%)[f]	Reference[g]
Parophrys vetulus (English sole)	ADT	NE		1
Parophrys vetulus (English sole)	ADT	NE		1
Parophrys vetulus (English sole)	ADT	NE		1
Rhepoxynius abronius (amphipod)	ADT	NC		2
Corophium volutator (amphipod)	ADT	NE		2
Neanthes sp. (polychaete)	JUV	NE		2
Grandidierella japonica (amphipod)	JUV	NC		3
Rhepoxynius abronius (amphipod)	ADT	NE		2
Corophium volutator (amphipod)	ADT	NE		2
Rhepoxynius abronius (amphipod)	ADT	NE		2
Grandidierella japonica (amphipod)	JUV	NE		3
Neanthes sp. (polychaete)	JUV	NE		2
Oyster, mussel	LAR	*		4
Corophium volutator (amphipod)	ADT	*		2
Rhepoxynius abronius (amphipod)	ADT	*		2
Mytilus edulis (bivalve)	LAR	*		5
Benthic species		*		5
Benthic species		*		5
Neanthes sp. (polychaete)	JUV	*		2
Rhepoxynius abronius (amphipod)	ADT	NE		2
Corophium volutator (amphipod)	ADT	NE		2
Neanthes sp. (polychaete)	JUV	NE		2
Aquatic biota		NE		6
Rhepoxynius abronius (amphipod)	ADT	*		5
Rhepoxynius abronius (amphipod)	ADT	*		5
Rhepoxynius abronius (amphipod)	ADT	*		4
Oyster	LAR	NE		7
Aquatic biota		NE		8
Rhepoxynius abronius (amphipod)	ADT	NE		7
Oyster	LAR	*		7
Rhepoxynius abronius (amphipod)	ADT	SG		7
Rhepoxynius abronius (amphipod)	ADT	*		9
Benthic community		*	1	10
Rhepoxynius abronius (amphipod)	ADT	NE	2.66 ± 2.15	11
Corophium volutator (amphipod)	ADT	NE	3.18 ± 2.1	11
Rhepoxynius abronius (amphipod)	ADT	NE	2.8 ± 1.96	11
Corophium volutator (amphipod)	ADT	NE	2.8 ± 1.96	11
Palaemonetes pugio (grass shrimp)	ADT	NE		12
Oyster	LAR	*		7

(Continued)

low the ERM, represent a possible-effects range within which effects would occasionally occur. Finally, the concentrations equivalent to and above the ERM value represent a probable-effects range within which effects would frequently occur. The incidence of adverse effects within each range was quantified by dividing the number of effects entries by the total number of entries and expressed as a percent. The ERL and ERM values were derived with only the effects data set, whereas the calculations of the percent incidence of effects within each concentration range were based upon both the effects and no-effects data sets.

An evaluation of the reliability of any proposed guidelines is essential to determine their applicability in sediment quality assessments. In this study, the reliability of the guidelines for each chemical was considered to be relatively high when: (1) they agreed closely (within factors of 3.0 or less) with those developed with other methods and/or with guidelines developed with the same methods applied to different data; (2) the incidence of effects was low (<25%) in the minimal-effects ranges; (3) the incidence of effects increased consistently and markedly in concordance with increasing chemical concentrations; and

Table 1. (*Continued*)

Concentration (±SD)	Area	Analysis type[a]	Test duration[b]	End point measured[c]
350 ± 45.8	Burrard Inlet, BC	COA	10 d	Not toxic (7.9 ± 5.12% mortality)
390	Burrard Inlet, BC	COA	10 d	Highly toxic (30.5% emergence)
390	Burrard Inlet, BC	COA	10 d	Highly toxic (23% emergence)
<403	Charleston Harbor, SC	COA		High species richness (14.9 ± 2.04) SRUs
<403	Charleston Harbor, SC	COA		Moderate species richness (9.05 ± 1.33) SRUs
<403	Charleston Harbor, SC	COA		Low species richness (5.16) SRUs
<403	Charleston Harbor, SC	COA		High species diversity (4.15 ± 0.59) SDUs
<403	Charleston Harbor, SC	COA		Moderate species diversity (2.3 ± 0.2) SDUs
<403	Charleston Harbor, SC	COA		Low species diversity (1.16) SDUs
486 ± 714	Elizabeth River, VA	COA	96 h	Not significantly toxic (4.5 ± 3.24% mortality)
500	Puget Sound, WA	AETA	15 m	1986 Puget Sound AET
500	Puget Sound, WA	AETA	48 h	1986 Puget Sound AET
500				**ER M (50th percentile)**
500	Puget Sound, WA	AETA	15 m	1988 Puget Sound AET
500	Puget Sound, WA	AETA	48 h	1988 Puget Sound AET
500	Puget Sound, WA	AETA		1986 Puget Sound AET
630	Puget Sound, WA	AETA	10 d	1986 Puget Sound AET
630	Puget Sound, WA	AETA		PSDDA maximum level criteria
654 ± 1049	Commencement Bay, WA	COA	10 d	Highly toxic (78.5 ± 19.5% mortality)
679 ± 469	Elizabeth River, VA	COA	96 h	Significantly toxic (50.7 ± 39% mortality)
680 ± 814	Elizabeth River, VA	COA	96 h	Significant decrease in respiration rates
730	Puget Sound, WA	AETA		1988 Puget Sound AET
2000	Puget Sound, WA	AETA	10 d	1988 Puget Sound AET
3031 ± 4271	Puget Sound, WA	COA	10 d	High prevalence of hepatic lesions (26.7 ± 6.4%)
3031 ± 4271	Puget Sound, WA	COA		High prevalence of hepatic idiopathic lesions (88.0 ± 3.7%)
3031 ± 4271	Puget Sound, WA	COA		High prevalence of hepatic cellular alterations (44.1 ± 8.5%)
5599 ± 24,392	Eagle Harbor, WA	COA	10 d	Least toxic (13 ± 7% mortality)
6522 ± 8915	Eagle Harbor, WA	COA	10 d	Moderately toxic (41 ± 9% mortality)
16,500	United States	EqPA		Chronic marine EqP threshold
39,557 ± 48,678	Eagle Harbor, WA	COA	10 d	Highly toxic (95.5 ± 8.5 mortality)

[a]Analysis type: COA = co-occurrence analysis; AETA = apparent effects threshold approach; EqPA = equilibrium partitioning approach; SQO = sediment quality objective; SQG = sediment quality guideline; SSBA = spiked sediment bioassay approach; SLCA = screening level criteria approach.

[b]Test duration: d = day; h = hour; m = minute.

[c]End point measured: AET = apparent effects threshold; PSDDA = Puget Sound dredge disposal analysis; LC_{50} = lethal concentration to 50% of the tested organisms; SRUs = species richness units; SDUs = species diversity units.

[d]Life stage: ADT = adult; LAR = larval; JUV = juvenile.

[e]Effects/No effects: NE = no effect; NC = no concordance; SG = small gradient; NG = no gradient; * = effects data used to calculate ERL and ERM values.

[f]1, Malins and others, 1985; 2, Tay and others, 1990; 3, Anderson and others, 1988; 4, Long and Morgan, 1990; 5, Becker and others, 1990; 6, Swain and Nijman, 1991; 7, Tetra-Tech, 1985; 8, US Army Corps of Engineers, 1988; 9, Swartz, and others, 1989; 10, Washington Department of Ecology, 1989; 11, McLeay and others, 1991; 12, Alden and Butt, 1987; 13, Winn and others, 1989; 14, Beller and others, 1986; 15, PTI, Inc., 1988; 16, CH2M-Hill, Inc., 1989; 17, Bolton, 1985.

(4) the incidence of effects was very high (>75%) in the probable-effects ranges. The reliability of the guidelines that failed to meet these evaluation criteria was considered to be lower.

Results

ERL and ERM values were derived for 28 substances: nine trace metals, total PCBs, 13 individual polynuclear aromatic hydrocarbons (PAHs), three classes of PAHs (total low molecular weight, total high molecular weight, and total PAH), and two pesticides (p,p'-DDE and total DDT). The data available for acenaphthene and phenanthrene are shown in Tables 1 and 2, respectively, to illustrate the format and content of the ascending tables with which the guidelines were derived. Space limitations preclude inclusion of equivalent tables for all of the substances.

Species	Life stage[d]	Effects/no effects[e]	TOC (%)[f]	Reference[g]
Rhepoxynius abronius (amphipod)	ADT	NE	2.64 ± 2.14	11
Rhepoxynius abronius (amphipod)	ADT	SG	3.5	11
Corophium volutator (amphipod)	ADT	SG	3.5	11
Benthic species		NE		13
Benthic species		NG		13
Benthic species		NG		13
Benthic species		NE		13
Benthic species		NG		13
Benthic species		NG		13
Palaemonetes pugio (grass shrimp)	ADT	NE		12
Microtox		*		14
Crassostrea gigas (oyster)	LAR	*		14
Microtox		*		15
Crassostrea gigas (oyster)	LAR	*		15
Benthic species		*		14
Rhepoxynius abronius (amphipod)		*		14
Aquatic biota		*		8
Rhepoxynius abronius (amphipod)	ADT	*		7
Palaemonetes pugio (grass shrimp)	ADT	SG		12
Palaemonetes pugio (grass shrimp)	ADT	*		12
Benthic community		*		15
Rhepoxynius abronius (amphipod)	ADT	*		15
Parophrys vetulus (English sole)	ADT	*		1
Parophrys vetulus (English sole)	ADT	*		1
Parophrys vetulus (English sole)	ADT	*		1
Rhepoxynius abronius (amphipod)	ADT	NE		16
Rhepoxynius abronius (amphipod)	ADT	SG		16
Aquatic biota		*	1	17
Rhepoxynius abronius (amphipod)	ADT	*		16

Adverse effects measured in association with acenaphthene included high amphipod mortality in sediment toxicity tests, low species richness in benthic communities, high prevalence of liver lesions in demersal fish, and chronic toxicity predicted by an equilibrium-partitioning model (Table 1). No data from spiked-sediment bioassays were available. As an example of the kinds of data analyses that were performed for entry into the BEDS, matching sediment chemistry and amphipod mortality data from Commencement Bay (Washington) were evaluated in a co-occurrence analysis. The average concentration of acenaphthene was 85.9 ppb in the samples that were the least toxic to amphipods (12.5 ± 4.5% mortality). This data entry was assigned a no-effects (ne) descriptor. In samples that were moderately toxic (26 ± 5.2% mortality), the average concentration of acenaphthene was 127 ppb. The ratio of 127 ppb to 85.9 ppb was less than 2.0, therefore, the moderately toxic data entry was assigned a small-gradient descriptor. The average acenaphthene concentration associated with highly toxic samples (78.5 ± 19.5% mortality) was 654 ppb, a factor 7.6-fold higher than the average concentration in the least toxic samples. It was assigned an asterisk and used in the calculation of the ERL and ERM values. A total of 30 data entries for acenaphthene were assigned effects designators. No biological effects were reported over the range of 1–8.8 ppb acenaphthene. The lower 10th percentile value of the effects data (the ERL) was 16 ppb and the median value (ERM) was 500 ppb. The percent incidence of adverse effects within the minimal-effects, possible-effects, and probable-effects ranges were 20%, 32%, and 84%, respectively.

Phenanthrene data were available from equilibrium-partitioning studies, spiked sediment bioassays, and numerous field surveys performed in many different areas (Table 2). A total of 51 data entries were assigned effects designators in the phenanthrene database. Adverse effects were not observed in asso-

Table 2. Summary of available data on effects of sediment-associated phenanthrene (ppb) in coastal sediments

Concentration (±SD)	Area	Analysis type[a]	Test duration[b]	End point measured[c]
4.6 ± 1.6	Laboratory	SSBA	~4 mo	No significant change in liver somatic indices
<5	Halifax Harbour, NS	COA	10 d	Not significantly toxic (3% mortality)
<5	Sidney Tar Pond, NS	COA	10 d	Not significantly toxic (3% mortality)
15	Burrard Inlet, BC	SQO		Sediment quality objectives
<20	Sidney Tar Pond, NS	COA	10 d	Not significantly toxic (4% mortality)
39.4 ± 47.6	Laboratory	SSBA	~4 mo	No signifcant change in kidney MFO induction
64.6	San Francisco Bay, CA	COA	48 h	Least toxic (23.3 ± 7.3% abnormal)
66.2 ± 57.5	Laboratory	SSBA	~4 mo	No significant change in spleen condition indices
88	San Francisco Bay, CA	AETA	48 h	San Francisco Bay AET
110	United States	EqPA		99% chronic marine criteria
119	Southern California	COA	10 d	Not significantly toxic (23.2% mortality)
150	Puget Sound, WA	COA		Low occurrence of hepatic cellular alterations (0%)
150	Puget Sound, WA	COA		Low prevalence of hepatic lesions (0%)
150	Puget Sound, WA	COA		Low prevalence of hepatic idiopathic lesions (32.5%)
159	San Francisco Bay, CA	COA	48 h	Not significantly toxic (31.9 ± 15.5% abnormal)
170	California	AETA	48 h	California AET
170	Northern California	AETA		Northern California AET
180 ± 325	Narragansett Bay, RI	COA	10 d	Not significantly toxic (5.28 ± 3.04% mortality)
188	San Francisco Bay, CA	COA	10 d	Least toxic (18 ± 6.6% mortality)
199	San Francisco Bay, CA	COA	10 d	Not significantly toxic (18.4 ± 6.8% mortality)
220	San Francisco Bay, CA	COA	10 d	Significantly toxic (42.9 ± 19.2% mortality)
222 ± 136	Southern California	COA	10 d	Significantly toxic (51.7% mortality)
223 ± 169	Burrard Inlet, BC	COA	10 d	Not toxic (4.5 ± 3.02% emergence)
223 ± 169	Burrard Inlet, BC	COA	10 d	Not toxic (5.21 ± 3.61% emergence)
224	San Francisco Bay, CA	COA	48 h	Moderately toxic (59.4 ± 11.3% abnormal)
228	San Francisco Bay, CA	COA	10 d	Moderately toxic (33.8 ± 4.7 mortality)
233	San Francisco Bay, CA	COA	48 h	Significantly toxic (55.7 ± 22.7% abnormal)
240	United States	EqPA		95% chronic marine criteria
240				**ER L (10th percentile)**
242	San Francisco Bay, CA	COA	10 d	Highly toxic (67 ± 11.8% mortality)
259	United States	SLCA		NSLC-marine
270	California	AETA		California AET values
270	Southern California	AETA		Southern California AET values
>290	Southern California	AETA	10 d	Southern California AET values
297	Commencement Bay, WA	COA	48 h	Least toxic (15.1 ± 3.1% abnormality)
316 ± 582	Elizabeth River, VA	COA	96 h	No significant change in respiration rate
320	Puget Sound, WA	AETA		PSSDA screening level concentration
368	United States	SLCA		NSLC-marine
374 ± 461	Elizabeth River, VA	COA	96 h	Not significantly toxic (4.5 ± 3.24% mortality)
383 ± 332	Laboratory	SSBA	~4 mo	Significant change in liver somatic indices
<403	Charleston Harbor, SC	COA		High species richness (14.9 ± 2.04) SRUs
<403	Charleston Harbor, SC	COA		Moderate species richness (9.05 ± 1.33) SRUs
<403	Charleston Harbor, SC	COA		Low species richness (5.16) SRUs
<403	Charleston Harbor, SC	COA		High species diversity (4.15 ± 0.59) SDUs
<403	Charleston Harbor, SC	COA		Moderate species diversity (2.3 ± 0.2) SDUs
<403	Charleston Harbor, SC	COA		Low species diversity (1.16) SDUs
<408 ± 501	Halifax Harbour, NS	COA	10 d	Not significantly toxic (6.8 ± 7.31% mortality)
<408 ± 501	Halifax Harbour, NS	COA	20 d	Not significantly toxic (0.7 ± 1.63% mortality)
<410 ± 498	Halifax Harbour, NS	COA	10 d	Not significantly toxic (8.5 ± 6.06% mortality)
475	San Francisco Bay, CA	COA	48 h	Highly toxic (92.4 ± 4.5% abnormal)
478	Commencement Bay, WA	COA	10 d	Least toxic (12.5 ± 4.5% mortality)
487 ± 318	Laboratory	SSBA	~4 mo	Significant increase in kidney MFO induction
510	Northern California	AETA	10 d	Northern California AET
510	California	AETA	10 d	California AET
510	San Francisco Bay, CA	AETA	10 d	San Francisco Bay AET
593	Commencement Bay, WA	COA	48 h	Moderately toxic (23 ± 2.3% abnormality)

Species	Life stage[d]	Effects/no effects[e]	TOC (%)	Reference[f]
Pseudopleuronectes americanus (flounder)	ADT	NE		18
Rhepoxynius abronius (amphipod)	ADT	NE		2
Rhepoxynius abronius (amphipod)	ADT	NE		2
Aquatic biota		NE		6
Corophium volutator (amphipod)	ADT	NE		2
Pseudopleuronectes americanus (flounder)	ADT	NE		18
Bivalve	LAR	NE		4
Pseudopleuronectes americanus (flounder)	ADT	NE		18
Oyster, mussel	LAR	*		4
Aquatic organisms		*	1	19
Grandidierella japonica (amphipod)	JUV	NE		3
Parophyrs vetulus (English sole)	ADT	NE		1
Parophyrs vetulus (English sole)	ADT	NE		1
Parophrys vetulus (English sole)	ADT	NE		1
Bivalve	LAR	NE		4
Mytilus edulis (bivalve)	LAR	*		5
Benthic species		*		5
Ampelisca abdita (amphipod)	ADT	NE		20
Rhepoxynius abronius (amphipod)	ADT	NE		4
Rhepoxynius abronius (amphipod)	ADT	NE		4
Rhepoxynius abronius (amphipod)	ADT	SG		4
Grandidierella japonica (amphipod)	JUV	SG		3
Rhepoxynius abronius (amphipod)	ADT	NE	2.68 ± 2.15	11
Corophium volutator (amphipod)	ADT	NE	3.18 ± 2.1	11
Bivalve	LAR	*		4
Rhepoxynius abronius (amphipod)	ADT	SG		4
Bivalve	LAR	SG		4
Aquatic organisms		*	1	19
Rhepoxynius abronius (amphipod)	ADT	SG		4
Benthic species		*	1	21
Benthic species		*		5
Benthic species		*		5
Rhepoxynius abronius (amphipod)	ADT	—		5
Oyster	LAR	NE		7
Palaemonetes puglo (grass shrimp)	ADT	NE		12
Aquatic biota		NE		8
Benthic species		*	1	21
Palaemonetes puglo (grass shrimp)	ADT	NE		12
Pseudopleuronectes americanus (flounder)	ADT	*		18
Benthic species		NE		13
Benthic species		NG		13
Benthic species		NG		13
Benthic species		NE		13
Benthic species		NG		13
Benthic species		NG		13
Rhepoxynius abronius (amphipod)	ADT	NE		2
Neanthes species (polychaete)	JUV	NE		2
Corophium volutator (amphipod)	ADT	NE		2
Bivalve	LAR	*		4
Rhepoxynius abronius (amphipod)	ADT	NE		7
Pseudopleuronectes americanus (flounder)	ADT	*		18
Rhepoxynius abronius (amphipod)	ADT	*		5
Rhepoxynius abronius (amphipod)	ADT	*		5
Rhepoxynius abronius (amphipod)	ADT	*		4
Oyster	LAR	*		7

(Continued)

Table 2. (*Continued*)

Concentration (±SD)	Area	Analysis type[a]	Test duration[b]	End point measured[c]
597	Commencement Bay, WA	COA	10 d	Moderately toxic (26 ± 5.2% mortality)
670	Laboratory	SSBA	~4 mo	Significant change in spleen condition indices
918 ± 1395	Burrard Inlet, BC	COA	10 d	Not toxic (97.2 ± 2.84% reburial)
918 ± 1395	Burrard Inlet, BC	COA	10 d	Not toxic (8.9 ± 2.99% mortality)
950	Eagle Harbor, WA	COA	4 d	LC_{50}
987 ± 1654	Elizabeth River, VA	COA	96 h	Significant decrease in respiration rates
1000	Puget Sound, WA	SQG		Chemical criteria
1020	United States	EqPA		Interim marine sediment quality criteria (FCV)
1213 ± 1547	Burrard Inlet, BC	COA	10 d	Not toxic (7.9 ± 5.12% mortality)
<1267 ± 2528	Halifax Harbour, NS	COA	20 d	Not significantly toxic (1 ± 2% mortality)
<1271 ± 2526	Halifax Harbour, NS	COA	10 d	Not significantly toxic (5.2 ± 3.5% mortality)
1379 ± 2545	Commencement Bay, WA	COA	48 h	High toxic (44.5 ± 19% abnormality)
1500	Puget Sound, WA	AETA	15 m	1986 Puget Sound AET
1500	Puget Sound, WA	AETA	48 h	1986 Puget Sound AET
1500	Puget Sound, WA	AETA	15 m	1988 Puget Sound AET
1500				**ER M (50th percentile)**
1500	Puget Sound, WA	AETA	48 h	1988 Puget Sound AET
<1688 ± 2920	Halifax Harbour, NS	COA	96 h	Significantly toxic (61.7 ± 12.5% mortality)
1913 ± 2693	Elizabeth River, VA	COA	10 d	Significantly toxic (50.7% ± 39% mortality)
2142	Eagle Harbor, WA	COA	10 d	Moderately toxic (41 ± 9% mortality)
2600	Eagle Harbor, WA	COA	10 d	Least toxic (13 ± 7% mortality)
2838	Commencement Bay, WA	COA	10 d	Highly toxic (78.5 ± 19.5% mortality)
3000	Burrard Inlet, BC	COA	10 d	Highly toxic (30.5% emergence)
3000	Burrard Inlet, BC	COA	10 d	Highly toxic (23% emergence)
3200	Puget Sound, WA	AETA		PSDDA maximum level criteria
3200	Puget Sound, WA	AETA		1988 Puget Sound AET
3680	Eagle Harbor, WA	COA	4 d	LC_{50}
5400	Puget Sound, WA	AETA	10 d	1986 Puget Sound AET
5400	Puget Sound, WA	AETA		1988 Puget Sound AET
6900	Puget Sound, WA	AETA	10 d	1988 Puget Sound AET
10,000	Laboratory	SSBA	10 d	Significant toxicity
11,656 ± 14,472	Puget Sound, WA	COA		High prevalence of hepatic lesions (26.7 ± 6.4%)
11,656 ± 14,472	Puget Sound, WA	COA		High prevalence of hepatic idiopathic lesions (88.0 ± 3.7%)
11,656 ± 14,472	Puget Sound, WA	COA		High prevalence of hepatic cellular alterations (44.2 ± 8.5%)
14,000	United States	EqPA		Chronic marine EqP threshold
14,000	United States	EqPA		EPA acute marine EqP threshold
>30,000	Laboratory	SSBA	14 d	LC_{50}
>30,000	Laboratory	SSBA	14 d	LC_{50}
33,603	Eagle Harbor, WA	COA	10 d	Highly toxic (95.5 ± 8.5% mortality)
<45,903 ± 64,909	Sidney Tar Pond, NS	COA	20 d	Not significantly toxic (8 ± 5.66% mortality)
91,800	Sidney Tar Pond, NS	COA	10 d	Significantly toxic (100% mortality)
91,800	Sidney Tar Pond, NS	COA	10 d	Significantly toxic (100% mortality)
105,500	Elizabeth River, VA	COA	28 d	LC_{50}
484,000	Sidney Tar Pond, NS	COA	20 d	Significantly toxic (52% mortality)
2,363,200	Elizabeth River, VA	COA	24 h	LC_{50}
4,220,000	Elizabeth River, VA	COA	2 h	Highly toxic (100% mortality)

[a]Analysis type: COA ± co-ocurrence analysis; AETA = apparent effects threshold approach; EqPA = equilibrium partitioning approach; SQO = sediment quality objective; SQG = sediment quality guideline; SSBA = spike sediment bioassay approach; SLCA = screening level criteria approach.

[b]Test duration: d = day; h = hour; min = minute; mo = month.

[c]End point measured: ER L = effects range low; ER M = effects range-median; AET = apparent effects threshold; PSDDA = Puget Sound dredge disposal analysis; organisms; SRUs = species richness units; SDUs = species diversity units; MFO = mixed-function oxidase; FCV = final chronic value; LC_{50} = lethal concentration to 50% of the tested organisms; EPA = Environmental Protection Agency.

Species	Life stage[d]	Effects/no effects[e]	TOC (%)	Reference[f]
Rhepoxynius abronius (amphipod)	ADT	*		7
Pseudopleuronectes americanus (flounder)	ADT	*		18
Rhepoxynius abronius (amphipod)	ADT	NE	2.8 ± 1.96	11
Corophium volutator (amphipod)	ADT	NE	2.8 ± 1.96	11
Rhepoxynius abronius (amphipod)	JUV/ADT	*		9
Palaemonetes puglo (grass shrimp)	ADT	*		12
Benthic community		*	1	10
Benthic community		NE	1	22
Rhepoxynius abronius (amphipod)	ADT	NE	2.64 ± 2.14	11
Neanthes species (polychaete)	JUV	NE		2
Corophium volutator (amphipod)	ADT	NE		2
Oyster	LAR	*		7
Microtox		*		14
Crassostrea gigas (oyster)	LAR	*		15
Microtox		*		
Crassostrea gigas (oyster)	LAR	*		15
Rhepoxynius abronius (amphipod)	ADT	*		2
Palaemonetes pugio (grass shrimp)	ADT	*		12
Rhepoxynius abronius (amphipod)	ADT	NC		16
Rhepoxynius abronius (amphipod)	ADT	NE		16
Rhepoxynius abronius (amphipod)	ADT	*		7
Rhepoxynius abronius (amphipod)	ADT	*	3.5	11
Corophium volutator (amphipod)	ADT	*	3.5	11
Aquatic biota		*		8
Benthic species		*		14
Rhepoxynius abronius (amphipod)	JUV/ADT	*		9
Rhepoxynius abronius (amphipod)	ADT	*		14
Benthic community	ADT	*		15
Rhepoxynius abronius (amphipod)	ADT	*		15
Rhepoxynius abronius (amphipod)	ADT	*	0.9	23
Parophrys vetulus (English sole)	ADT	*		1
Parophrys vetulus (English sole)	ADT	*		1
Parophrys vetulus (English sole)	ADT	*		1
Aquatic biota		*	1	17
Aquatic biota		*	1	24
Grandidierella japonica (amphipod)	ADT	—	0.1	25
Grandidierella japonica (amphipod)	ADT	—	1	25
Rhepoxynius abronius (amphipod)	ADT	*		16
Neanthes species (polychaete)	JUV	NE		2
Corophium volutator (amphipod)	ADT	*		2
Rhepoxynius abronius (amphipod)	ADT	*		2
Leiostomus xanthurus (spot)	JUV	*		26
Neanthes species (polychaete)	JUV	*		2
Leiostomus xanthurus (spot)	JUV	*		26
Leiostomus xanthurus (spot)	JUV	*		26

[d]Life stage: ADT = adult; LAR = larval; JUV = juvenile.

[e]Effects/no effects: NE = no effect; NC = no concordance; SG = small gradient; NG = no gradient; * = effects data used to calculate ERL and ERM values.

[f]1, Malins and others, 1985; 2, Tay and others, 1990; 3, Anderson and others, 1988; 4, Long and Morgan, 1990; 5, Becker and others, 1990; 6, Swain and Nijman, 1991; 7, Tetra-Tech, 1985; 8, US Army Corps of Engineers, 1988; 9, Swartz and others, 1989; 10, Washington Department of Ecology, 1989; 11, McLeay and others, 1991; 12, Alden and Butt, 1987; 13, Winn and others, 1989; 14, Bellar and others, 1986; 15, PTI, Inc., 1988; 16, CH2M-Hill, Inc., 1989; 17, Bolton, 1985; 18, Payne and others, 1988; 19, Pavlou and others, 1987; 21, Neff and others, 1986; 22, US EPA, 1988; 23, Plesha and others, 1988; 24, Lyman and others, 1987; 25, SCCWRP, 1989; 26, Roberts and others, 1989.

Table 3. ERL and ERM guideline values for trace metals (ppm, dry wt) and percent incidence of biological effects in concentration ranges defined by the two values

Chemical	Guidelines		Percent (ratios) incidence of effects[a]		
	ERL	ERM	<ERL	ERL-ERM	>ERM
Arsenic	8.2	70	5.0 (2/40)	11.1 (8/73)	63.0 (17/27)
Cadmium	1.2	9.6	6.6 (7/106)	36.6 (32/87)	65.7 (44/67)
Chromium	81	370	2.9 (3/102)	21.1 (15/71)	95.0 (19/20)
Copper	34	270	9.4 (6/64)	29.1 (32/110)	83.7 (36/43)
Lead	46.7	218	8.0 (7/87)	35.8 (29/81)	90.2 (37/41)
Mercury	0.15	0.71	8.3 (4/48)	23.5 (16/68)	42.3 (22/52)
Nickel	20.9	51.6	1.9 (1/54)	16.7 (8/48)	16.9 (10/59)
Silver	1.0	3.7	2.6 (1/39)	32.3 (11/34)	92.8 (13/14)
Zinc	150	410	6.1 (6/99)	47.0 (31/66)	69.8 (37/53)

[a]Number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range.

ciation with phenanthrene concentrations of <5 ppb to 66 ppb. The ERL value for phenanthrene was 240 ppb and the ERM value was 1500 ppb. The percent incidence of adverse effects within the minimal-effects, possible-effects, and probable-effects ranges were 18%, 46%, and 90%, respectively.

The incidence of adverse effects increased with increasing concentrations of all trace metals, except nickel (Table 3). The incidence of effects was 10% or less in the minimal-effects ranges and 11%–47% in the possible-effects ranges from all of the trace metals. The incidence of adverse effects exceeded 75% in the probable-effects ranges for chromium, copper, lead, and silver but was only 42.3% and 16.9% for mercury and nickel, respectively. However, the incidence of effects in the probable-effects range for chromium was greatly influenced and exaggerated by data from multiple tests conducted in only two field surveys.

The incidence of adverse effects consistently and markedly increased with increasing concentrations of all organic compounds, except p,p'-DDE and total DDT (Table 4). The incidence of effects ranged from 5.0% to 27.3% in the minimal-effects ranges for organic compounds and was 25% or less for all but one of the compounds—fluorene. Within the possible-effects ranges, the incidence of effects ranged from 18% to 75%. The incidence of effects ranged from 50% to 100% in the probable-effects ranges and equaled or exceeded 75% for all but four compounds. The incidence of effects in the probable-effects range for total PCBs was relatively low (51%).

Discussion

Guidelines Accuracy

Among the trace metals, the most accurate guidelines appeared to be those for copper, lead, and silver; the incidence of effects were very low (<10%) in the minimal-effects ranges, increased steadily through the possible-effects and probable-effects ranges, and were very high (>83%) in the probable-effects ranges. Among the organic compounds, the guidelines appeared to be highly accurate for all of the classes of PAHs and most of the individual PAHs. Except for fluorene, the incidence of effects was 25% or less at concentrations below the respective ERL values. Except for dibenzo(a,h)anthracene, p,p'-DDE, total DDT, and total PCBs, the incidence of effects was 75% or greater at concentrations that exceeded the respective ERMs. At concentrations in the probable-effects ranges, the incidence of adverse effects was 100% for acenaphthylene, 2-methyl naphthalene, and low-molecular-weight PAHs and 90% or greater for chromium, lead, silver, benz(a)anthracene, and fluoranthene.

The accuracy of the guidelines for some substances appeared to be relatively low. For example, the incidences of effects associated with nickel were 1.9%, 16.7%, and 16.9%, respectively, in the three concentration ranges. The incidence of effects did not increase appreciably with increasing concentrations of nickel and were very low in all three ranges. The incidence of effects in the probable-effects ranges for mercury and total PCBs were relatively low (42.3% and 51.0%, respectively). Furthermore, the incidence of effects did not increase consistently and markedly with increasing concentrations of p,p'-DDE, and total DDT. The p,p'-DDE and total DDT databases may have been unduly influenced by relatively low equilibrium-partitioning values, which were based upon chronic marine water quality criteria intended to protect against bioaccumulation in marine fish and birds, not toxicity to benthic organisms. The incidence of effects in the probable-effects range for chromium

Table 4. ERL and ERM guideline values for organic compounds (ppb, dry wt) and percent incidence of biological effects in concentration ranges defined by the two values

Chemical	Guidelines		Percent (ratios) incidence of effects[a]		
	ERL	ERM	<ERL	ERL-ERM	>ERM
Acenaphthene	16	500	20.0 (3/15)	32.4 (11/34)	84.2 (16/19)
Acenaphthylene	44	640	14.3 (1/7)	17.9 (5/28)	100 (9/9)
Anthracene	85.3	1100	25.0 (4/16)	44.2 (19/43)	85.2 (23/27)
Fluorene	19	540	27.3 (3/11)	36.5 (19/52)	86.7 (26/30)
2-Methyl naphthalene	70	670	12.5 (2/16)	73.3 (11/15)	100 (15/15)
Naphthalene	160	2100	16.0 (4/25)	41.0 (16/39)	88.9 (24/27)
Phenanthrene	240	1500	18.5 (5/27)	46.2 (18/39)	90.3 (28/31)
Low-molecular weight PAH	552	3160	13.0 (3/23)	48.1 (13/27)	100 (16/16)
Benz(a)anthracene	261	1600	21.1 (4/19)	43.8 (14/32)	92.6 (25/27)
Benzo(a)pyrene	430	1600	10.3 (3/29)	63.0 (17/27)	80.0 (24/30)
Chrysene	384	2800	19.0 (4/21)	45.0 (18/40)	88.5 (23/26)
Dibenzo(a,h)anthracene	63.4	260	11.5 (3/26)	54.5 (12/22)	66.7 (16/24)
Fluoranthene	600	5100	20.6 (7/34)	63.6 (28/44)	92.3 (36/39)
Pyrene	665	2600	17.2 (5/29)	53.1 (17/32)	87.5 (28/32)
High molecular weight PAH	1700	9600	10.5 (2/19)	40.0 (10/.25)	81.2 (13/16)
Total PAH	4022	44792	14.3 (3/21)	36.1 (13/36)	85.0 (17/20)
p,p'-DDE	2.2	27	5.0 (1/20)	50.0 (10/20)	50.0 (12/24)
Total DDT	1.58	46.1	20.0 (2/10)	75.0 (12/16)	53.6 (15/28)
Total PCBs	22.7	180	18.5 (5/27)	40.8 (20/49)	51.0 (25/49)

[a]Number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range.

ostensibly appeared to be very high but was unduly exaggerated by data from multiple tests performed in only two studies.

Comparisons with Other Guidelines

Agreement within a factor of 3 or less among guidelines developed with different methods has been recommended by a panel of experts as an indication of good precision (Lorenzato and others 1991). In the following discussion, the comparisons of guidelines were conducted by determining the ratios between them, i.e., the larger of the two values was divided by the smaller value.

The ERL and ERM values reported in Tables 3 and 4 were based upon a considerable expansion and revision of the database used by Long and Morgan (1990). The quantities of data used to derive the present values exceeded those used previously by factors of 1.4 to 2.6. About 30%–50% of the data used in the present analysis came from the database used previously. Furthermore, the considerable amounts of freshwater data in the previous database were deleted in the present analysis. Of the 25 ERL values derived in the two analyses, seven remained unchanged, nine decreased, and nine increased. The ratios between the two sets of ERL values ranged from 1.0 to 9.4 (average of 1.88, N = 25). The ERL values for only two substances changed by factors greater than 3.0×: arsenic (decreased by 4.2×); and acenaphthene (decreased by 9.4×). The ratios between the two sets of ERM values ranged from 1.0 to 7.6 (average of 1.63, N = 25). The average ratios between the two sets of ERM values was 1.2 for the individual PAHs and 1.5 for the trace metals; seven remained unchanged, seven decreased, and eight increased. Only one ERM value changed by a factor greater than 3.0: total DDT (decreased by 7.6×). The ERL and ERM values for p,p'-DDE increased by factors of 1.1 and 1.8, respectively. The ERL value for total PAHs remained unchanged and the ERM value increased by a factor of 1.3. The results of these comparisons indicate that the guidelines are relatively insensitive to changes in the database, once the minimum data requirements have been satisfied.

The national sediment quality criteria proposed by the US Environmental Protection Agency for fluoranthene, acenaphthene, and phenanthrene in salt water are based upon equilibrium-partitioning models (US EPA 1993a–c). The proposed mean criterion for fluoranthene is 300 μg/g organic carbon (with 95% confidence limits of 140 and 640 μg/goc). For acenaphthene the mean criterion is 240 μg/goc (with 95% confidence limits of 110 and 500 μg/goc). For phenanthrene the mean criterion is 240 μg/goc (with 95% confidence limits of 110 and 510 μg/goc). Assuming a TOC concentration of 1%, these criteria

values are equivalent to 3000 (1400–6400) ppb dry weight for fluoranthene; 2400 (1100–5000) ppb dry weight for acenaphthene; and 2400 (1100–5100) ppb dry weight for phenanthrene. The mean criteria exceeded the ERM values of 500 ppb for acenaphthene and 1500 ppb for phenanthrene by factors of 4.8, and 1.6, respectively. The criterion for fluoranthene was lower than the ERM by a factor of 1.7. The criteria expressed in units of dry weight would increase with increasing TOC concentrations.

The ERL and ERM values generally agreed within factors of two to three with freshwater effects-based criteria issued by Ontario (Persaud and others 1992). Lowest effect levels and severe effect levels were reported, based upon a screening level concentration (SLC) approach applied to matching benthic community and sediment chemistry data. The ratios between the present ERL values and the lowest effect levels for Ontario ranged from 1.25 to 3.1 (average of 1.7) for eight trace metals (As, Cd, Cr, Cu, Pb, Hg, Ni, Zn). The ratios between the present ERM values and the severe effect levels for Ontario ranged from 1.0 to 3.4 (average of 2.0) for the same eight trace metals. Of the 16 comparisons, the ERL/ERM values were lower than the respective values for Ontario in six cases and higher in ten cases.

Among all of these comparisons, most of the guidelines agreed within the recommended factor of 3.0 or less. In the worse case, two values (previous and present ERL values for acenaphthene) differed by a factor of 9.4.

Merits of the Approach

This approach attempts to identify the concentrations of toxicants that are rarely associated with adverse biological effects and those usually associated with effects, based upon data from many studies. The advantages of this approach are that guidelines can be developed quickly with existing information and that they are based upon data gathered from many different studies. An underlying assumption of the approach is that, if enough data are accumulated, a pattern of increasing incidence of biological effects should emerge with increasing contaminant concentrations.

Data from all available sources were considered in this study, including those from equilibrium-partitioning models, spiked sediment bioassays, and numerous field surveys. The modeling and bioassay methods differ considerably from those used in the field studies, since they generally are performed with single chemicals as if they were acting alone. The field studies invariably involve complex mixtures of contaminants, acting synergistically, additively, or antagonistically. Whereas the modeling studies and spiked sediment bioassays can be used to establish cause–effect relationships for single chemicals, the data from field studies cannot establish such relationships. However, the data from field studies of complex mixtures reflect real-world, natural conditions in ambient sediments. We believe that the most meaningful assessment tools are those that are based upon evidence from and agreement among all three of these methods. If data compiled from different study areas with different pollution histories and physical–chemical properties converge upon ranges of contaminant concentrations that are usually associated with effects, then guidelines derived from those studies should be broadly applicable to many other areas and situations. Therefore, in this report, the data from numerous studies were used to identify the concentrations of individual chemicals that were rarely, occasionally, and usually associated with effects.

The biological data compiled for derivation of the guidelines included a variety of different taxonomic groups and toxicological end points. The sensitivities of the taxa to toxicants may have differed considerably, and, therefore, contributed to variability in the data base. However, we believe that the inclusion of data from multiple taxa ensures the broad applicability of the guidelines and the protection of a diversity of organisms.

The bioavailability of sediment-associated contaminants is controlled to a large degree by certain physical–chemical properties of the sediments. For example, high acid-volatile sulfide (AVS) concentrations appear to reduce the bioavailability of cadmium, and, possibly, other trace metals in sediments (Di Toro and others 1990). Similarly, the influence of increasing TOC concentrations in reducing the bioavailability of many nonionic organic compounds has been demonstrated in modeling and laboratory studies (Di Toro and others 1991, Swartz and others 1990, Pavlou and others 1987). Significant differences in toxicity can occur at similar toxicant concentrations over relatively small ranges in TOC and/or AVS concentrations (Adams and others 1992). It has been argued that sediment quality criteria are indefensible if they do not account for factors that control bioavailability (Di Toro and others 1991). The data evaluated in the present analysis were not normalized to either TOC or AVS concentrations, since only a small minority of the reports that were encountered included results for these parameters. Nevertheless, the present evaluation indicates that the guidelines derived using the approach reported herein are accurate for most

chemicals and agree reasonably well with other guidelines. Therefore, they are likely to be reliable tools in sediment quality assessments.

While factors that are thought to control bioavailability were not considered explicity, surely they were operative in the tests of field-collected sediments and influenced the bioavailability of all of the potential toxicants. However, the data that were encountered indicated that TOC concentrations usually ranged from 1% to 3% in most study areas. In contrast, the concentrations of some chemicals differed by several orders of magnitude among the same samples. These observations suggest that, over these large concentration gradients, the relatively small differences in TOC and/or AVS concentrations may have been relatively unimportant in controlling toxicity or, otherwise, were masked in the data analyses.

Since the data bases used to develop the present guidelines included data from many field studies, the guidelines may tend to be more protective than those based upon only single-chemical approaches. The cumulative (e.g., synergistic) effects of mixtures of toxicants in ambient sediments, including those not quantified may tend to drive the apparent effective concentrations of individual toxicants downward (i.e., toward lower concentrations).

Conclusions

Based upon an evaluation of existing data, three ranges in chemical concentrations were determined for 28 chemicals or chemical classes. These ranges were defined by two guideline values: the lower 10th percentile (ERL) and the 50th percentile (ERM) of the effects data distribution. The incidence of biological effects was quantified for each of these ranges as an estimate of the accuracy of the guidelines. The incidence of effects usually was less than 25% at concentrations below the ERL values. For most chemicals, the incidence of effects increased markedly as the concentrations increased. Furthermore, the incidences of effects often were greater than 75% (occasionally 100%) at concentrations that exceeded the ERM values. However, for a few chemicals (especially mercury, nickel, total PCBs, total DDT, and p,p'-DDE) there were relatively weak relationships between their concentrations and the incidence of effects. The guideline values reported herein generally agreed within factors of 3× or less with guidelines derived earlier using the same methods applied to a different data base and with guidelines developed with other methods. The numerical guidelines should be used as informal screening tools in environmental assessments. They are not intended to preclude the use of toxicity tests or other measures of biological effects. The guidelines should be accompanied by the information on the incidence of effects. The percent incidence data may prove useful in estimating the probability of observing similar adverse effects within the defined concentration ranges of particular contaminants.

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COMPARISONS OF SEDIMENT TOXICITY WITH PREDICTIONS BASED ON CHEMICAL GUIDELINES

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Abstract—The U.S. Environmental Protection Agency (EPA) Environmental Monitoring and Assessment Program–Estuaries and the National Oceanic and Atmospheric Administration Bioeffects Surveys provide large data sets with which to test proposed relationships between sediment chemistry and toxicity. We conclude that guidelines based on bulk chemistry can provide useful triggers for further analysis but should not be used alone as indicators of toxicity. The sediment quality criteria for nonionic organic compounds proposed by the EPA are exceeded in so few samples that they may be of limited practical value. Toxicity was present in many cases when acid-volatile sulfide (AVS) concentrations exceeded the sum of concentrations of sulfide-insoluble metals. However, there is no way to test whether that toxicity was due to those trace elements. The AVS criterion is much more sensitive to AVS concentration than to trace metal contamination.

Keywords—Sediment Equilibrium partitioning Acid-volatile sulfide Cooccurrence Measured toxicity

INTRODUCTION

Adams et al. [1] reviewed the three main approaches used in the United States to estimate biological effects of contaminated sediment based on chemical data alone. The cooccurrence [2] method is representative of those that use bulk chemical concentrations. The equilibrium partitioning method for neutral organics [3] assumes that pore water is in equilibrium with sediment and that, to be nontoxic, pore water must meet water quality standards. The method based on acid-volatile sulfide (AVS) concentration [4] applies to metals with insoluble sulfides and assumes that as long as AVS is in excess, those metals are unavailable and not toxic. A recent U.S. Environmental Protection Agency draft report [5] describes application of all of these chemical-based methods to categorize sediments into tiers on the basis of their potential for causing adverse biological effects.

Theoretical reasons exist for doubting that any of these chemical-based guidelines reflect sediment toxicity. For example, the equilibrium assumption at the foundation of equilibrium partitioning may not be valid, the presence of AVS should mean a lack of oxygen and obvious toxic conditions, and the cooccurrence methods assume that bulk concentrations represent biological availability of chemicals. Such considerations aside, however, we have a large data set with which to empirically test the applicability of these methods. The EPA Environmental Monitoring and Assessment Program—Estuaries (EMAP–E) [6–9] and the National Oceanic and Atmospheric Administration (NOAA) National Status and Trends Bioeffects Surveys [10–13] provide a 1,508-sample data set containing measurements of both chemical concentrations and toxicity.

The EMAP–E program was designed to randomly sample estuaries within entire biogeographical provinces of the United States. For example, a random grid of hexagonal cells was overlain on a map of the United States, and samples were taken from the cells. We have annual data for 1990–1993 from sampling in the Virginian Province (Chesapeake Bay, VA, to Cape Cod, MA), for 1991–1994 in the Louisianian Province (Gulf of Mexico except south of Tampa Bay, FL), and for 1993 in the Carolinian Province (Cape Henry, VA, to St. Lucie Inlet, FL). Because the samples were selected with a probability-based method, there was no tendency or bias for them to be from particularly contaminated areas near harbors, industrial waterways, or urban centers. Sediment samples from the NOAA Bioeffects Surveys, on the other hand, are from intensive sampling in urban waterways. In general, therefore, they have higher concentrations of contaminants than the EMAP–E samples. Taken together, these data allow us to evaluate how well the three chemical-based methods predict sediment toxicity.

METHODS

All data and methods have been previously reported [6–14]. Chemical analyses for trace elements were based on hydrofluoric acid extractions and are total metal analyses [6–8,14]. Acid-volatile sulfide was measured by the method of Di Toro et al. [4]. Amphipod (*Ampelisca abdita*) survival bioassays [6–13] were all 10-d exposures to whole sediment. The other bioassays used different endpoints and different components of sediment. Microtox® bioassays of sediments from Boston Harbor, Long Island Sound, the Hudson–Raritan Es-

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Table 1. Effects range–low and effects range–median values from Long et al. [2]

Chemical	ER–L[a]	ER–M[b]
Ag	1 ppm	3.7 ppm
As	8.2	70
Cd	1.2	9.6
Cr	81	370
Cu	34	270
Hg	0.15	0.71
Ni	21	52
Pb	47	220
Zn	150	410
p,p′-DDE[c]	2.2 ppb	27 ppb
Total DDT[c]	1.6	46
Total PCBs	23	180
LMW PAHs[c]	550	3,160
HWW PAHs[c]	1,700	9,600
Total PAHs[c]	4,000	45,000
Acenapthene	16	500
Acenaphthylene	44	640
Anthracene	85	1,100
Fluorine	19	540
2-Methylnaphthalene	70	670
Naphthalene	160	2,100
Phenanthrene	240	1,500
Benzo[*a*]pyrene	261	1,600
Chrysene	384	2,800
Dibenzo[*a,h*]anthracene	63	260
Fluoranthene	600	5,100
Pyrene	670	2,600

[a] Effects range–low (ER–L) values are bulk sediment concentrations (dry weight) below which sediment is unlikely to be toxic.
[b] Effects range–median (ER–M) values are concentrations above which toxicity is probable.
[c] Dichlorodiphenyldichloroethylene (*p,p′*-DDE) is part of the total dichlorodiphenyltrichloroethane (DDT) and individual polycyclic aromatic hydrocarbon (PAH) compounds are part of the total PAH, low-molecular-weight (LMW) PAH (2- and 3-ring compounds), and high-molecular-weight (HMW) PAH (more than 3-ring compounds). However, double and triple counting is of no consequence when we are interested in knowing only whether there is at least one exceedance of ER–L or ER–M. PCB = polychlorinated biphenyl.

tuary, and Tampa Bay [10–13] all began with dichloromethane extracts of homogenized sediment. Subsequently, the solvent was replaced with ethanol and bioluminescence of the bacterium *Photobacterium phosphoreum* measured in the standard Microtox diluent. Sea urchin fertilization and embryo development assays of sediments from Boston Harbor [13] and Tampa Bay [11] were conducted with gametes of *Arbacia punctulata* exposed to pore water that was pressure-extracted from whole sediment. Bivalve survival and development tests with sediments from Long Island Sound [10] and the Hudson–Raritan Estuary [12] began with embryos of the clam *Mulinia lateralis* exposed to elutriates (i.e., seawater extracts) of homogenized sediment.

Our procedure for this report was simply to categorize the data according to the different criteria and guidelines and to observe how closely predicted responses conformed to measured toxicity. The amphipod survival toxicity test is common to all data sets and serves as our primary measure of toxicity.

RESULTS

Cooccurrence

It has been suggested [2] that sediment toxicity is unlikely when bulk concentrations in sediment of all chemicals listed in Table 1 are below the effects range–low (ER–L) value. Conversely, toxicity is probable when any chemical concentration exceeds an effects range–median (ER–M) value. We have chemical data on 1,508 samples along with the results of 10-d exposures of amphipods (*A. abdita*) to whole sediment. Sediments are considered toxic if fewer than 80% of the amphipods, relative to controls, survive the exposure [6–8,15]. Table 2 lists numbers of samples in various categories relative to ER–L and ER–M exceedances and the numbers that were toxic. Of the 481 samples without an ER–L exceedance, only 5% were toxic. This is in agreement with the prediction. On the other hand, of the 239 samples that had at least one concentration greater than an ER–M, only 38% were actually toxic. While the cooccurrence of ER–M exceedances with amphipod toxicity is less than ''probable,'' exceedances may identify samples for further examination.

Equilibrium partitioning

Five sediment quality criteria (SQC) for nonionic organic compounds based on assuming equilibrium between sediment

Table 2. Total numbers of samples per data set, number of effects range–low (ER–L) nonexceedances, number of ER–L nonexceedances that were toxic to amphipods, number with at least one effects range–median (ER–M) exceedance, number with at least one ER–M exceedance that were toxic, and total number that were toxic (<80% survival after 10 d)

Data set[a]	Total samples	Total samples with all <ER–L	Toxic samples with all <ER–L	Total samples with at least 1 >ER–M	Toxic samples with at least 1 >ER–M	Total toxic samples (%)
EMAPV	537	211	12	82	25	59 [11]
EMAPL	642	195	9	11	3	68 [11]
EMAPC	93	66	2	6	0	2 [2]
LIS	63	2	2	21	13	32 [51]
HRE	38	2	0	34	18	20 [53]
BOS	30	0	0	22	4	6 [20]
LA[b]	44	0	0	38	18	21 [48]
TB[b]	61	5	1	25	9	10 [6]
Total	1,508	481	26	239	90	218 [14]

[a] EMAPV = Environmental Monitoring and Assessment Program (EMAP), Virginia Province (1990–1993 data); EMAPL = Louisiana Province (1991–1994 data); EMAPC = Carolina Province (1994 data); LIS = Bioeffects Survey, Long Island Sound; HRE = Bioeffects Survey, Hudson–Raritan Estuary; BOS = Bioeffects Survey, Boston Harbor; LA = Bioeffects Survey, Los Angeles Harbor; and TB = Bioeffects Survey, Tampa Bay.
[b] The data sets for Los Angeles and Tampa Bay contain toxicity results for 63 and 80 additional samples, respectively, but not full suites of chemical data. In those cases not all ERL and ERM exceedances can be known, so the data were not used.

Table 3. Results of all toxicity tests in the Bioeffects Surveys[a]

Area[c]	Total samples	No. of samples toxic[b] by each test					
		Amphipod survival	Microtox®	Bivalve survival	Bivalve development	Urchin fertilization	Urchin development
BOS	30	6	17	—	—	1	30
LIS	63	32	40	16	0	—	—
HRE	38	20	18	16	11	—	—
TB	61	10	—	—	—	52	—

[a] The various tests (see text) in addition to that for amphipod survival are based on Microtox® (bacterial luminescence), bivalve (*Mulinia lateralis*) embryo survival and development, and sea urchin (*Arbacia punctulata*) gamete fertilization and embryo development.
[b] Sample deemed toxic if response is statistically different and <80% of control.
[c] See footnote a in Table 2 for definitions of abbreviations.

and interstitial water and requiring that interstitial water meet surface water quality criteria have been proposed [3]. The chemicals endrin, dieldrin, acenaphthalene, fluoranthene, and phenanthrene exceed criteria when their concentrations (on a per total organic carbon [TOC] basis) exceed 0.76, 20, 230, 300, and 240 μg/g TOC, respectively. Among the 1,179 EMAP–E samples, only nine exceeded at least one of those criteria, and only two of the nine were toxic. Among the 236 samples from the Bioeffects Surveys, three from the Hudson–Raritan Estuary exceeded at least one of those criteria, and all were toxic.

Swartz et al. [16] introduced a variation on this approach for polycyclic aromatic hydrocarbons (PAHs). Their method first assumes equilibrium to calculate the interstitial water concentration of each PAH compound, that concentration is converted to a toxic unit by dividing by the 10-d median lethal concentration (LC50) for the compound, then the individual toxic units are summed to a total that indicates a high probability of toxicity if it exceeds 1.0. Using the equilibrium coefficients and LC50 concentrations for the 13 PAH compounds in the report by Swartz et al. [16], we calculated toxicity units for the data in hand. The results were about the same as for the original application of equilibrium partitioning, that is, fewer than 10 samples had 1 or more total toxic units, and about half of those were not toxic to amphipods.

Acid-volatile sulfide

Di Toro et al. [4] proposed that if the simultaneously extracted metals (SEM)/AVS ratio is less than 1, there will be no toxic effect from Cd, Cu, Hg, Ni, Pb, or Zn. The SEM and AVS values are the concentrations of those metals and sulfide, respectively, extracted from sediment with 1 N hydrochloric acid. Toxicity may still be present but would be attributed to other elements, to organic chemicals, or to other stressors. Conversely, nothing about toxicity is to be implied when the SEM/AVS ratio is greater than 1.

Among the Virginian and Louisianian EMAP–E samples are 978 with measured AVS concentrations. Only 544 of those also had measured SEM concentrations, but we can substitute the summed concentration of the six metals extracted for total elemental concentrations. This sum, called TotM, exceeds SEM; so, wherever TotM/AVS was less than 1, SEM/AVS must also have been less than 1. In 355 cases TotM/AVS was greater than 1. Nothing is predicted in these cases. In the 623 cases where TotM/AVS was less than 1, 48 (8%) were toxic. However, the existence of toxicity does not conflict with prediction. The method only predicts that such toxicity could not have been due to any of the six sulfide-insoluble metals. We have no way of knowing whether or not it was.

Among the 978 samples, the median concentrations for AVS and TotM were 2.47 and 1.62 μM/g (dry weight), respectively. Among the 355 where the ratio exceeded 1, the corresponding medians were 0.45 and 1.70 μM/g. The fact that median concentrations of metals are almost the same in both cases illustrates that the method is sensitive to AVS but not to metal contamination.

Among the Bioeffects Survey data sets, there were 272 samples with AVS and SEM data (letting SEM equal TotM for Tampa Bay). The AVS criterion applies in the 252 cases where SEM/AVS was less than 1, yet toxicity was observed in 72 (29%) of them. Again, the toxicity may have been due to characteristics other than concentrations of sulfide-insoluble metals.

Other biological tests

If other biological tests were used to assess toxicity, there would be more cases where ER–M exceedances coincide with a toxic response. The Bioeffects Surveys included other tests, and it is evident that more instances of toxicity were found when other tests were conducted in conjunction with the amphipod test (Table 3). If a toxic response in any test is sufficient to categorize a sample as toxic, it is more likely that an ER–M exceedance will cooccur with toxicity. As seen in Table 4, 91% of the samples with an ER–M exceedance were toxic by at least one biological test. However, of the 183 samples with only an ER–L exceedance, 156 (85%) were toxic. Moreover, five of the nine (55%) samples without even an ER–L exceedance were toxic. What has happened by including all biological tests is that even a random sample now has an 84% chance of being toxic. In effect, any connection between bulk chemistry or any sediment characteristic and toxicity has been overwhelmed.

DISCUSSION AND CONCLUSIONS

An SQC based on equilibrium partitioning was exceeded in only 12 cases among the 1,508 samples. Only six of those were toxic, but the paucity of extremely contaminated samples probably disallowed a rigorous test of the SQC. As a practical matter, the SQCs would be exceeded so rarely that they would have limited application.

When the SEM/AVS ratio is less than 1 there should be no toxicity due to sulfide-insoluble metal concentrations. In the data we examined, toxicity was observed in 120 cases where the SEM/AVS ratio was less than 1, but that may have been due to sediment characteristics other than sulfide-insoluble metals. The AVS criterion did not apply in the 30% of the samples where AVS was exceeded by the metal concentrations. These were usually not cases where metal concentrations were

Table 4. Same as Table 2 but with toxicity from any test deemed sufficient to put sample into the toxic category

Data set[a]	Total samples	Total samples with all <ER–L[b]	Toxic samples with all <ER–L	Total samples with at least 1 >ER–M[c]	Toxic samples with at least 1 >ER–M	Total toxic samples (%)
BOS	30	0	0	22	22	30 [100]
LIS	63	2	2	21	20	51 [81]
HRE	38	2	0	34	26	28 [74]
TB	61	5	3	25	25	52 [85]
Total	192	9	5	102	93	161 [84]

[a] See footnote a in Table 2 for definitions of abbreviations.
[b] ER–L = effects range–low.
[c] ER–M = effects range–median.

high but where AVS was low. In effect, the SEM/AVS criterion is sensitive to AVS and not to sediment contamination with trace metals.

The cooccurrence method is, by definition, sensitive only to concentrations of trace elements and organic compounds. This advantage is also a failing because it does not acknowledge that noncontaminant characteristics of sediment can mitigate toxicity. Thus, while there were many samples with ER–M exceedances, fewer than 40% of those were toxic to amphipods.

Among the three methods, the cooccurrence method is empirically the most useful because it applies to both organic and inorganic contaminants, it predicts toxicity (not its absence), and it can be applied within concentration ranges that occur in less than extremely contaminated areas. The lack of even an ER–L exceedance does mean that toxic effects are unlikely, but ER–M exceedances should only be taken to indicate that further analysis is in order. They should never be taken, by themselves, to mean that sediment is exerting a toxic effect upon the environment or that there would be any benefit to decreasing its chemical content.

A call to further analysis based on chemical data means that bioassays should be done, but as more types of tests are done, the likelihood of a toxic response increases. So, one toxic response accompanied by no response in other tests should not signal a need for action with regard to mitigating sediment. As pointed out by Adams et al. [1], one needs to consider the spatial scale of the sediment in question and, when that is large, also examine the community living in and on the sediment. Even these considerations can be insufficient because indigenous community alteration can be due to excess organic matter, not chemical contaminants, and because considerations of scale are somewhat subjective.

In the end, many aspects of the sediment come into consideration. Chemical data are important, as are bioassay data, but decisions on sediment management cannot be prescribed. They need to be based on judgement.

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Date: February 18, 2011

To: California Regional Water Quality Control Board, Los Angeles Region; United States Environmental Protection Agency, Region 9

cc: Paul Singarella

From: John Slocomb, Ph.D.
Paul Mehrle, Ph.D.

RE: **The Effects Range-Low (ERL) Value for Numeric Target of Water Body-Pollutant Combinations in Marine Sediments of the Dominguez Channel Estuary and Greater Los Angeles and Long Beach Waters**

SUMMARY

The Regional Board's intended use of the effects range-low (ERL) sediment quality guideline (SQG) as the basis for total daily maximum loads in the Los Angeles and Long Beach Harbor marine and estuarine waters is not scientifically defensible. The limitations noted by the original authors of the ERL and others concluded that:

- ERLs should be used only as an informal screening tool and should not preclude gathering additional information such as adverse sediment effects and toxicity testing;

- The relationship between concentrations of nickel, mercury, total PCBs, and total DDT and adverse effects is at most, weak and therefore, the Regional Board's use of the ERL will not result in expected gains in sediment quality; and,

- The presence of unmeasured or unknown contaminants will lead to large uncertainties in sediment toxicity, thereby substantially limiting the usefulness of the ERL as a sediment target.

The principal conclusions of this report are as follows:

- Use of the ERLs to set sediment targets for the Harbor Waters TMDL is inappropriate because data used to develop the ERLs has not been made available by the ERL authors, so it is presumed that the Regional Board does not possess those data and has not reviewed them. Also, it is not known whether the ERLs are based on data relevant to the nine water bodies that are the subject of the draft TMDL, or whether the author's underlying database (which is not publicly available) contains any data from these water bodies. Under these circumstances, it would be inappropriate to base a regulatory decision on the ERLs, and neither I nor anyone else who is interested in this TMDL can independently verify or reproduce the ERLs.

- The ERLs are not intended or designed for the use to which the draft TMDL intends. The authors of the ERLs and federal agencies caution against the use of the ERLs for anything other than an informal screening tool to help rank and prioritize different sediment sites. The ELRs are not recommended by the authors to be used for making remedial decisions with regard to contaminated sediments, as is being done in the draft TMDL. In fact, over many years of experience at various contaminated sediment sites around the country, I have never once seen the ERLs used to establish sediment remedial or cleanup levels as is the Regional Board has proposed. This unprecedented use of ERLs is not warranted and invalid.

- An independent statistical evaluation of other sediment screening values prepared by McDonald, one of the authors of the ERLs, has demonstrated a very weak, and almost random, relationship between DDT on the one hand, and impacts to the benthic ecosystem, on the other. In fact, the authors of the ERLs acknowledged that their ERLs provide a poor relationship between the concentration of DDT in contaminated sediment and any toxic effects to benthic biota that might be from the DDT. In short, the ERLs are not a reliable or useful measure of the potential for DDT to adversely impact the benthic ecosystem, and no reasonable scientist would rely on them for this purpose.

- What is known about the ERLs is that they ascribe toxicity to DDT and other compounds based on pulling data from published studies where the authors of those underlying studies do not ascribe the reported toxicity to DDT or these other compounds. In some cases, the authors of the underlying studies specifically state that DDT is not the cause of the observed toxicity. In other instances, the authors of the underlying studies ascribe the toxicity to the presence of compounds or conditions other than DDT. In this regard, the use by the ERLs of data from such studies to build a threshold for DDT is inappropriate and not technically justified.

INTRODUCTION

The California Regional Water Quality Board, Los Angeles Region (Regional Board) and U.S. Environmental Protection Agency (USEPA) have developed total daily maximum loads (TMDLs) for water body-pollutant combinations to help achieve water quality standards in the Dominguez Channel Estuary and the Greater Los Angeles and Long Beach Harbor waters (Regional Board and USEPA 2010). These areas addressed by the TMDLs are associated with the greater Los Angeles and Long Beach Harbor marine and estuarine waters. In its problem statement, the Regional Board stated that one or more environmental media (i.e., water, sediments or tissues) were contaminated by one or more of the following constituents: cadmium, copper, mercury, lead, zinc, chlordane, dieldrin, DDT, polychlorinated biphenyls (PCBs), and polycyclic aromatic hydrocarbons (PAHs). This report primarily uses DDT as an example. For sediments in these areas, the Regional Board identified a total of 8 metals and organic chemical pollutant impairments for the Dominquez Channel and Greater Los Angeles and Long Beach Harbor waters, and 15 for the Consolidated Slip; DDT is included in both areas. It is noteworthy that the freshwater portion of the Dominquez Channel also contains pollutant impairments but does not include DDT; therefore, this area is not considered further in this report.

The Regional Board established numeric sediment targets for these metals and organics based on effects range-low (ERL) sediment quality guidelines developed by Long *et al.* (1995). The purpose of this report is to assess whether these ERL values for the identified water body-pollutants are scientifically defensible based on the limitations identified by numerous authors who have reported on their use and on how values are to be interpreted.

SEDIMENT QUALITY GUIDELINES (SQGS)

Numerous federal and state agencies have developed or adopted sediment quality guidelines (SQGs) to provide informal, non-regulatory chemical benchmarks for evaluating risks to sediment dwelling organisms (Long *et al.* 1995; Long and MacDonald 1998; National Oceanic and Atmospheric Administration [NOAA] 1999; among others). In one set of guidelines, the ERL and the effects range-median (ERM) were developed for helping interpret sediment sample data obtained under the National Status and Trends Program of NOAA (Long and Morgan 1990; Long *et al.* 1995). A biological effects database for sediments (BEDS) was developed by these investigators from numerous studies that were conducted throughout North America. However, these data did not include information for the Los Angeles or Long Beach Harbor marine and estuarine waters. These data included synoptically collected biological effects and chemical data from sediments; other conditions to which the data were subjected are described by Long *et al*. (1995). The BEDS database is not publically available and therefore, the derived ERL and ERM values cannot be independently reproduced and are not transparent. Another set of marine SQGs called the threshold effects level (TEL) and probable effects level (PEL) were developed

for the State of Florida (MacDonald *et al*. 1996). Values of the two sets of SQGs (ERLs and ERMs, and TELs and PELs) are different, having been developed using different rules.

The ERL and ERM values were developed using paired chemical concentrations associated with only adverse biological effects, which included measures of altered benthic communities, sediment toxicity, histopathological disorders in dimersal fish, EC_{50} or LC_{50} concentrations from laboratory bioassays, and toxicity predicted by equilibrium partitioning models. All of these adverse effects were considered equivalent. The ERL and ERM values for 9 trace metals, 13 individual PAHs, 3 classes of PAHs, and 3 classes of chlorinated organic hydrocarbons are shown in Tables 1 and 2. The values are statistically based in that the 10^{th} percentile of the effects data was termed the ERL and the 50^{th} percentile (median) was termed the ERM. Very generally, the ERL value reflects sediment concentrations below which adverse effects were rarely observed. Alternatively, the ERM reflects sediment concentrations above which adverse effects frequently occurred. The TEL and PEL values were developed using both effects and no observed effects but have generally the same interpretations as the ERL and ERM values. The TEL and PEL values are not shown in this report and are mentioned only because they appear later in a procedure used for classifying sites.

Measures of reliability, recommended uses and limitations of the empirically derived in ERL and ERM values are presented in Long and MacDonald (1998) and NOAA (1999). The reliability and uses are reflected in Table 3, which also includes TEL and PEL values. This table represents how sites can be prioritized based on 1) mean values of ERM or PEL quotients, 2) the number of ERMs or PELs exceeded and 3) whether any ERLs or TELs are exceeded. Associated with each priority categorization are the probabilities of amphipod toxicity as reported by Long *et al*. (1998). Mean ERM quotients are determined using the data in Tables 1 and 2. For example, for *each* constituent listed in the tables, the concentration found in a sediment sample is divided by its associated ERM value. The quotients are then averaged for the sample. If multiple samples are collected for a site, then the grand average is determined. Based on the mean ERM quotient, the site can be classified into one of four categories that reflect increasing probabilities of toxicity. From Table 3, there are four priority categories from highest to lowest. The number of individual ERMs exceeded is also determined, which can lead to different toxicity probabilities in each of the four priority categories. The only use of the ERL values is to define the lowest priority category, which generally indicates that no further action is needed. Recall that if no ERLs are exceeded then the likelihood of toxicity was rare. The methods for determining mean PEL quotients are equivalent but use different PEL values.

LIMITATIONS OF SQGs

The ERM and ERL sediment quality guidelines were intended to provide informal and interpretive tools to aid in assessing contamination and the potential for toxicity. They are intended to be used to rank or prioritize chemicals of potential concern and to assess whether further investigation or management action is needed. These further investigations can include

but are not limited to planning of monitoring activities, designing whole-sediment or spiked sediment bioassays. Long *et al.* (1995) stated that "The numerical [ERL and ERM] guidelines should be used as informal screening tools in environmental assessments. They are not intended to preclude the use of toxicity tests or other measures of biological effects. The guidelines should be accompanied by the information on the incidences of effects." Long and MacDonald (1998) stated that "Data from investigations of hazardous waste sites can be compared to the SQGs to aid in ranking and prioritizing sites and to determine the need for further information to support management decisions."

However, there are several serious limitations to ERL values associated with mercury, nickel, total PCBs, total DDT, and ρ,ρ'-DDE. There is a very low correlation between toxic effects and the ERL values for these constituents such that the ecological thresholds for these compounds should be used cautiously. Long *et al.* (1995) stated that ". . . for a few chemicals (especially mercury, nickel, total PCBs, total DDT, and ρ,ρ'-DDE) there were relatively weak relationships between their concentrations and the incidence of effects." Hence, it is unlikely that the Regional Board's use of the ERL for total DDT would yield defensible results in terms of sediment quality objectives. Data reported by Fairy *et al.* (1996) and assessed by Geisy *et al.* (1998) were used to assess the relationship between total DDT and amphipod mortality in sediments from San Diego, CA harbor. This relationship is shown in Figure 1. The Pearson correlation between the plotted variables is -0.304 meaning that amphipod mortality (a measure of toxicity) *decreases* with *increasing* concentrations of total DDT. This relationship does not correspond at all with a dose-response that would be typical if total DDT were causing toxicity. It is noteworthy that the highest toxicity was found at concentrations of total DDT that were below the ERL value. This analysis provides further evidence that the Regional Boards intended use of the ERL as a sediment target is not defensible.

The low correlation means that even if the ERL for total DDT is exceeded, there is no reasonable confidence that the sample would in fact be toxic and therefore, no reason to use this value as a sediment quality target. The same limitations apply to the ERM values for mercury, nickel, total PCBs and total DDT. The likelihood that these ERMs accurately predict adverse (toxic) effects are much lower than for the other constituents shown in Tables 1 and 2. Regarding exceeding an ERM value, O'Conner *et al.* (1998) stated that "ERM exceedance should only be taken to indicate that further analysis is in order. They should never be taken, by themselves, to mean that sediment is exerting a toxic effect upon the environment or that there would be any benefit to decreasing its chemical content."

Chemicals in sediments largely occur as mixtures and, while the ERM or ERL value for a single constituent may be exceeded, the exceedance does not provide confidence that it is toxic to sediment organisms. In addition, there are no SQGs available for many toxic constituents. The ability of SQGs to predict toxicity of co-occurring constituents for which there are no SQG values is unknown. Di Toro (2008) further added that ERMs, ERLs, and other SQGs cannot ". . .

predict with more than a modest degree of certainty the outcome of a toxicity test on sediment from the field that is contaminated with many, and possibly unknown and unmeasured constituents." This situation is true in the Los Angeles and Long Beach Harbor marine and estuarine waters and reflects the co-occurrence of toxic chemicals in sediments. Di Toro's statement means that even if, for example, DDT exceeds the ERL value, it is not the cause of sediment toxicity (see Figure 1). Toxicity results from numerous, co-occurring chemicals many of which are not measured but which can act independently to produce adverse effects to sediment organisms. Given that only a modest degree of certainty is achievable with respect to toxicity, it is clear that exceeding an ERL cannot lead to the conclusion that DDT (or any other single chemical) is responsible for toxicity.

NOAA (1999) stated that "SQGs should be used with caution and common sense. There are no SQGs available for many substances that can be highly toxic in sediments. The abilities of the SQGs to correctly predict toxicity of co-varying substances for which there are no SQGs are unknown. The SQGs were derived in units of dry weight sediments; therefore, they do not account for the potential effects of geochemical factors in sediments that may influence contaminant bioavailability. The SQGs were not intended for use in predicting effects in wildlife or humans through bioaccumulation pathways. The SQGs were neither calculated nor intended as toxicological thresholds; therefore, there is no certainty that they will always correctly predict either non-toxicity or toxicity."

Furthermore, NOAA (1999) stated that "The SQGs are best applied when accompanied by measures of effect such as laboratory toxicity tests and/or benthic community analyses and/or bioaccumulation tests, which lead to the preparation of a weight of evidence. Furthermore, they are best applied in a comprehensive assessment framework involving the establishment of clear study objectives, a priori methods for data analyses, and well-understood decision points regarding the uses of the data."

USE OF ERL AS NUMERIC TARGET

The Regional Board's intended use of the ERL as a conservative numeric target of mercury, nickel, total PCBs and total DDT in marine sediments of the Dominquez Channel Estuary and the Greater Los Angeles and Long Beach Harbor waters is not defensible scientifically. Given the limitations stated above, it is quite likely that these ERLs do not accurately forecast sediment toxicity. Even exceeding the higher ERM value, which the Regional Board does not use, does not meet this accuracy criteria. Sediments in these two areas likely contain a mixture of toxic constituents that are not listed in the SQG tables and therefore, cannot be understood toxicologically.

The ERL, and in fact all SQGs, were originally developed as tools or benchmarks to aid in further understanding of adverse effects in sediments. It was never meant to be used in a regulatory manner as the Regional Board is intending as the basis for TMDL allocations. To

date, the ERMs or ERLs have not been promulgated and implemented for standards or criteria in the U.S. If used as the Regional Board intends, it likely will lead to unreasonable allocations and sediment remedial actions that will not have the effect of restoring sediment quality. Finally, the ERL (and the SQGs in general) do not provide a direct means of determining which constituents are causing toxicity in sediments.

The Regional Board has minimal data upon which to assign numeric targets for mercury, nickel, total PCBs and total DDT in sediments. At a minimum, these data should lead to further investigations of adverse effects including sediment toxicity that include:

- Field validation of the ERL for total DDT, PCBs and other chemicals
- Comprehensive and statistically designed whole-sediment toxicity testing
- Spiked bioassays using DDT, PCBs and other chemicals to assess toxicity in single chemical situations
- Laboratory-based bioaccumulation testing
- Initiation of Phase 1 and Phase 2 toxicity identification evaluations (TIEs) that will lead to better understanding of causation if toxicity is present

The statements above are a true and accurate statement of my analyses and opinions of this matter. If the agencies require further information on this matter or would like to speak with me directly, please let me know. I can be reached through Chuck Anthony at Latham and Watkins, LLP at 714-540-1235.

Table 1. Effect range-low (ERL) and effects range-high (ERM) sediment guideline values for trace metals (mg/kg sediment dry weight).

Metal	Effects Range-Low (ERL)	Effects Range-High (ERM)
Arsenic	8.2	70
Cadmium	1.2	9.6
Chromium	81	370
Copper	34	270
Lead	46.7	218
Mercury	0.15	0.71
Nickel	20.9	51.6
Silver	1.0	3.7
Zinc	150	410

Table 2. Effects range-low (ERL) and effects range-median (ERM) for organic constituents (µg/kg sediment dry weight).

Constituent	Effects Range-Low (ERL)	Effects Range-High (ERM)
Acenaphthene	16	500
Acenaphthylene	44	640
Anthracene	85.3	1100
Fluorene	19	540
2-methyl naphthalene	70	670
Naphthalene	160	2100
Phenanthrene	240	1500
Sum LPAH [1]	552	3160
Benz(a)anthracene	261	1600
Benzo(a)pyrene	430	1600
Chrysene	384	2800
Dibenzo(a,h)anthracene	63.4	260
Fluoranthene	600	5100
Pyrene	665	2600
Sum HPAH [2]	1700	9600
Sum Total PAH	4022	44792
ρ,ρ'-DDE	2.2	27
Sum Total DDTs	1.58	46.1
Total PCBs	22.7	180

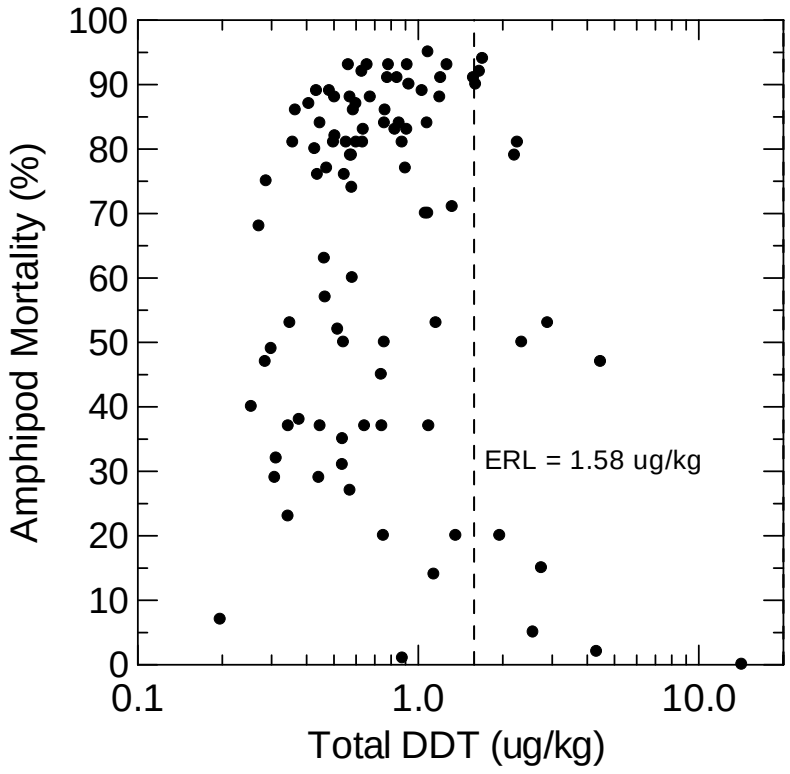
[1] Low molecular weight PAHs
[2] High molecular weight PAHs

Table 3. Four priority categorizations of sediment samples based on ERM, PEL, TEL and ERL values and the associated probabilities of amphipod toxicity (Long and MacDonald (1998).

Site Priority	Chemical Characteristics	Probability of Amphipod Toxicity [1]
Highest	Mean ERM Quotients > 1.5 Mean PEL Quotients > 2.3 > 10 ERMs Exceeded > 21 PELs Exceeded	74% 76% 85% 88%
Medium-High	Mean ERM Quotients 0.51 – 1.5 Mean PEL Quotients 1.51 – 2.3 6 – 10 ERMs Exceeded 6 – 20 PELs Exceeded	46% 50% 52% 53%
Medium-Low	Mean ERM Quotients 0.11 – 0.5 Mean PEL Quotients 0.11 – 1.5 1 – 5 ERMs Exceeded 1 – 5 PELs Exceeded	30% 25% 32% 24%
Lowest	Mean ERM Quotients < 0.1 Mean PEL Quotients < 0.1 No ERLs Exeeded No TELs Exceeded	12% 10% 11% 9%

[1] Data from Long *et al.* 1998

Figure 1. The relationship between concentrations of total DDT and amphipod mortality in sediments from San Diego Harbor. There are 94 observations in this plot and the Pearson correlation coefficient is -0.304.



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Shaping the Future

February 22, 2011

TO: California Regional Water Quality Control Board, Los Angeles Region;
United States Environmental Protection Agency, Region 9
CC: Paul Singarella, Latham & Watkins, LLP
FROM: David Sunding, Director, The Brattle Group and Professor, College of Natural Resources, UC Berkeley

RE: Comments on the cost consideration of, "Dominguez Channel and Greater Los Angeles and Long Beach Harbor Water Toxic Pollutants Total Maximum Daily Loads Draft"

I appreciate the opportunity to submit this comment to the California Regional Water Quality Control Board, Los Angeles Region, in response to the Board's issuance of a draft TMDL for toxic pollutants in the Dominguez Channel and Greater Los Angeles and Long Beach Harbor waters. I am submitting these comments on behalf of Montrose Chemical Corporation of California.

My background and qualifications are listed on the curriculum vitae attached as an exhibit to this comment. I am a principal at The Brattle Group, a global firm providing consulting and expert testimony in economics, finance and regulation to corporations, law firms and governments. I am also the Thomas J. Graff Professor of Natural Resource Economics at UC Berkeley, where I am also the Co-Director of the Berkeley Water Center. Prior to joining the faculty at Berkeley, I served as senior economist at President Clinton's Council of Economic Advisers. I am currently a Visiting Professor in the Woods Institute of the Environment at Stanford University.

The staff of the Regional Board has not met its burden under Porter-Cologne and U.S. EPA guidance to consider economics in the development of the TMDL. The Staff Report does not attempt to gauge the significance of the TMDL in that it does not consider or even calculate the benefits of the action relative to current water quality levels. The Staff Report does not discuss benefits of the TMDL in relation to the costs of implementation, which is the only logical way to assess economic reasonableness. The plan for implementing the proposed regulation is not described in enough detail to permit an adequate calculation of costs. The report makes no mention of who will bear the costs of complying with the regulation, or of the regional economic implications of the action. The report does not acknowledge the potential employment impacts of the proposed TMDL, or the effect of the cleanup plan on competitiveness of California businesses.

The costs of the proposed regulation are not adequately described in the staff report. Available information demonstrates that the assertions of the Staff Report regarding the costs of compliance are misleading. For example, the report makes assertions about the costs of dredging based on two projects completed more than a decade ago. These assertions are not consistent with the actual costs of dredging at other locations in California. The report also mischaracterizes the actual costs of impounding and treating stormwater to the levels required by the TMDL. Using more realistic assumptions on the cost of dredging ($200/cy based on a sample of projects around California instead of the $60/cy used in the Staff Report), the present value of dredging over 11 million cubic yards to fulfill the requirements of the Sediment Quality Objective could total $2.2 billion in 2011 dollars, equivalent to an annual expenditure over 20 years of $150.2 million. The cost of dredging over 35 million cubic yards to meet the Effect Range Low requirements would total $6.9 billion, or an annual expenditure of $476.9 million.

The Regional Board staff made no attempt to evaluate the benefits of the proposed TMDL. There is no mention of the number of people participating in recreational fishing activities in the area of the TMDL, nor for any other type of recreational use (sailing, hiking, bird watching, etc.). There is no mention in the Staff Report of how any of these uses would be improved by the proposed action, if they would be affected at all. There is

no mention of the value to residents of Los Angeles County, or any other area of California, of the wildlife benefits achieved by the TMDL. This circumstance is in violation of the State requirement that major regulations are subject to a demonstration of economic value.

The proposed action is likely to result in an unacceptably high level of costs in relation to the actual benefits achieved. The staff report fails to demonstrate that the Regional Board considered alternatives to the proposed TMDL that would be less burdensome, or that it considered the relative cost effectiveness of alternative standards. This is inconsistent with basic principles of economic analysis of regulation, and in contradiction to established federal guidelines promulgated by the US Environmental Protection Agency and the Office of Management and Budget.

2. Failure to Consider Economics

Under the Porter-Cologne Water Quality Control Act, the State Water Resources Control Board has the ultimate authority over State water rights and water quality policy. Porter-Cologne also establishes that the nine Regional Water Quality Control Boards shall oversee water quality on a day-to-day basis at the local and regional level. The Regional Boards engage in a number of water quality functions in their respective regions. One of the most important is preparing and periodically updating Basin Plans. Each Basin Plan establishes beneficial uses of water designated for each water body to be protected; water quality objectives for both surface water and groundwater; and actions necessary to maintain these standards in order to control nonpoint and point sources of pollution to the State's waters.

Porter-Cologne requires that when determining water quality targets the Regional Boards shall consider the following factors: "the beneficial uses to be protected, the water quality objectives reasonably required for that purpose, other waste discharges, the need to prevent nuisance, and the provisions of Section 13241." Section 13241 in turn lists six "factors to be considered," including "economic considerations" and "water quality conditions that could reasonably be achieved through the coordinated control of all factors which affect water quality in the area."

CEQA also requires the Regional Boards to consider economic impacts when establishing a performance standard. Discussing the application of CEQA to TMDLs, the State Board has acknowledged that "numeric targets and load allocations would probably fall into the category of performance standards." Thus, CEQA requires that the Regional Board should detail the likely methods and costs of compliance with the proposed TMDL.

In addition to these California requirements, the U.S. EPA has published guidelines for the preparation of TMDLs in California.[1] In particular, the EPA states that the State may consider a mix of allocation criteria (see Technical Support Document for Water Quality Based Permit Decisions (EPA, 1991) for more information). These criteria include technical and engineering feasibility, cost or relative cost, economic impacts/benefits, cost effectiveness and fairness/equity. Based on the Staff Report, there is no evidence that staff considered any of these factors in developing the TMDL.

It is worth noting that under federal law, economics can be considered in the basin planning process in designating potential beneficial uses. Specifically, the federal water quality standards regulations allow a state to dedesignate, to decide not to designate, or to establish a subcategory of a potential beneficial use on economic grounds. To rely on this basis, the state must demonstrate that attaining the use is infeasible

[1] *Guidance for Developing TMDLs in California*. U.S. EPA, Region 9. January 7, 2000.

because the controls necessary to attain the use "would result in substantial and widespread economic and social impact." The reason for this provision is to avoid the type of situation evidenced by this proposed TMDL, namely to require the expenditure of resources in pursuit of small or nonexistent benefits.

Over many decades, economists have developed a rigorous methodology to assess the impacts of government actions. The approach derives from the basic principles of public finance and welfare economics. It takes a holistic perspective by considering many groups in society, and articulates the tradeoffs among policy alternatives. The economist's approach to assessing government actions also combines considerations of efficiency and equity, and has been widely applied to problems of environmental regulation. At its heart, economic analysis of regulation is an accounting of the consequences of a governmental action. This accounting is often quantitative, but many first-rate economic analyses also treat impacts qualitatively, especially for nonstandard commodities. Ideally, economic analysis will also give information on the distributional impacts of the intervention, or a description of which groups in society are affected by the action, and how much.

A requirement to "consider economics" is not the same as a directive to adopt only those regulations that pass a cost-benefit test. Agencies can use the results of economic analysis, but not be bound by "bottom-line" numbers. Most economists would not argue that quantified costs and benefits tell the whole story, or that precise measurements of either are always possible. But when economic analysis reveals low or nonexistent benefits and high costs, something seems amiss. Indeed, the California legislature sought to avoid just such a socially undesirable outcome by mandating a consideration of economics when setting water quality standards.

The federal government has maintained a decades-long commitment to economic analysis of regulation. This practice began in the Nixon Administration, which initiated Quality of Life Reviews of federal regulations in 1970. The two main events in the history of economic analysis at the federal level, however, occurred in the Reagan and Clinton Administrations. President Reagan issued Executive Order 12,291, perhaps the most decisive step in the cost-benefit record. This Executive Order established a set of principles for agencies to follow to the extent permitted by law, including a commitment to cost-benefit analysis. The order required Regulatory Impact Analysis of major rules, and also established a formal mechanism for OMB oversight of interventions. President Clinton issued Executive Order 12,866, which reaffirmed the basic commitments to economic analysis and conferred bipartisan legitimacy. This order also introduced some reforms to the economic analysis process that were designed primarily to assuage fears of industry capture. These reforms included procedures for conflict resolution and inclusion of equity considerations.

3. Implementation Costs

The Draft TMDL describes the proposed implementation plan to meet numeric targets for toxic pollutants in the Dominguez Channel and greater Los Angeles and Long Beach Harbor Waters. Compliance with the TMDL for metals and PAHs is based on achieving the load and waste load allocations and / or demonstrating attainment of the Sediment Quality Objectives (SQOs) as multiple lines of evidence. Compliance with the TMDLs for bioaccumulative compounds shall be based on achieving the assigned loads and waste load allocations or, alternatively, by meeting fish tissue targets.[2]

Proposed compliance measures include the elimination of toxic pollutants being loaded into Dominguez Channel and the harbors, and cleanup of contaminated sediments lying on the bottom of greater Los Angeles

[2] Draft TMDL, p. 102.

and Long Beach Harbors. In most areas of the harbors, contaminant concentrations in sediment are above the proposed numeric targets for sediment. WLAs and LAs may not be attainable without removal of contaminated sediment areas including identified hotspots within the Dominguez Channel Estuary and the Los Angeles and Long Beach Harbors. The Draft TMDL recommends the following implementation processes:

1. Implement (and evaluate effectiveness of) best management practices (BMPs) and source control in conjunction with the remediation actions to remove contaminated sediment as necessary;
2. Evaluate effectiveness of controlling sediment loading from Los Angeles River, San Gabriel River and Machado Lake through implementation of effective TMDLs.
3. Conduct monitoring to evaluate compliance with targets during implementation and after implementation actions are in place.
4. Determine if reductions in loadings from controllable sources from Los Angeles River and San Gabriel River will be required and addressed through revision of the TMDL.
5. Re-evaluate the WLAs and LAs, if necessary.[3]

Staff finds it may be feasible to dredge Harbors for contaminated sediment removal as part of the existing practices.

The Staff Report cites a 1998 study conducted for sediment contamination mitigation at the mouth of Ballona Creek and Marina del Rey. According to this study, the cost of dredging at that location ranges from $10.95 per cubic yard to $74.4 per cubic yard (Moffat & Nichol Engineers, 1998). The Staff Report estimates the unit cost of dredging by averaging these two bookend figures to arrive at a unit cost of $42.68 per cubic yard in 1998 dollars. Assuming an inflation rate of 3% each year, the Staff Report then adjusts the unit dredging cost to current dollars, arriving at a figure of $60.84 per cubic yard.

Based on the December 10, 2010 draft memorandum to the Regional Board Staff prepared by the Ports of Long Beach and Los Angeles, and its associated discussion, areas where dredging activities may be necessary to remove contaminated sediment to fulfill requirements of Effect Range Low (ERL) or SQOs were analyzed. The Draft TMDL cites five primary locations (see Table 9) as having concentrations exceeding ERLs.[4]

Staff Report Table 7-3: Estimated volume of dredged materials with respect to SQO and ERL

Waterbody	Estimated Volume of Dredged Materials (cy)	
	SQO	ERL
Fish Harbor	1,120	1,111,701
Los Angeles Harbor Cabrillo Marina	1,156,131	1,159,768
Los Angeles Harbor Consolidated Slip	475,910	478,294
Los Angeles Harbor Inner Cabrillo Beach Area	196,560	238,138
Los Angeles Harbor Beach Inner Harbor	6,692,551	21,864,948
Los Angeles Harbor Beach Outer Harbor	2,645,954	10,669,544
San Pedro Bay outside Harbors Outlet of Los Angeles River	4,840	4,840
Total	**11,173,066**	**35,527,233**

[3] Draft TMDL, p. 103.
[4] Draft TMDL, p. 124.

Staff estimates that the total cost of dredging at Harbors $679.8 million in present dollars. Given a compliance schedule of 20 years, and an assumed annual interest rate of 6%, the staff calculates that the amortized cost for each year is $59.3 million dollars. Basing the cost calculations instead on the Effect Range Low scenario, the total cost of dredging at Harbors is $2.16 billion, or $188.1 million annually.

Staff's quantification of dredging costs is flawed for several reasons. Most important, the use of a single study from a single location is not making use of the best available information, particularly when the study in question did not report actual dredging costs, but rather ex ante engineering estimates. Further, simply averaging the upper and lower cost estimates is crude in the extreme.

Staff's $60.84 per cy estimate of dredging is far lower than the actual cost of other similar remediation projects. We surveyed several similar soil removal sites in California to demonstrate that the cost of dredging is in the range of $120 - $1,320 per cy. The following discussion summarizes some of the available information on dredging costs in California.

United Heckathorn

This site, located in Richmond CA, consists of a former manufacturing site that was used from approximately 1947 to 1966 by a number of companies (collectively referred to as "United Heckathorn"). The primary contaminants of concern at this site are DDT and dieldrin. The 1994 Record of Decision (ROD) for this site proposed dredging 65,000 cy of pesticide-contaminated sediment. Mechanical dredging in the Lauritzen Canal started in September 1996 and finished in April 1997 and removal using long-stick excavators started in the Parr Canal in August 1996 and finished in April 1997. A total of 108,000 cy of sediment were actually removed, solidified, and disposed offsite by rail to landfills in Arizona and Utah. Dredged areas were backfilled with 18 inches of sand (15,700 cy). Actual costs have not been released; however the bid cost for the original target volume of 65,000 cy was reportedly $7.3 - $7.5 million ($112 - $115 per cy) in 1994 dollars. Actual combined transport and disposal cost to the ECDC landfill was about $48 per ton.[5] Using the same rate of inflation as the Draft TMDL, 3%, the estimated cost of dredging in 2010 dollars is $180 - $185 per cy.

Seaplane Lagoon

Seaplane Lagoon underwent a thorough examination in 2005 and, based on the 2007 ROD, the sediment remediation alternative chosen was a combination of dredging, monitoring, dewatering, and upland confinement. In addition, the 2005 feasibility study estimated the costs of this alternative to be between $7.6 million and $8.9 million.[6] However, according to the ROD the actual costs for the remediation alternative selected were $24.6 million (2007 dollars) which is approximately 30-35% higher than the totals estimated in the 2005 feasibility study.[7]

Moffett Field

The feasibility study for Moffett Field was completed in 2005. The remediation alternatives were different than the previously discussed sites, primarily due to the area being described as a tidal marsh or wetland. The remediation activities best suited to Moffett Field were determined to be in situ/ex situ treatment, excavation,

[5] Applied Environmental Management, *Site Status Summary – United Heckathorn Superfund Site (Richmond, CA)*, Major Contaminated Sediment Sites Database, accessed at: http://www.smwg.org/MCSS_Database/MCSS_Database_Docs.html on January 27, 2011.

[6] Prepared by Battelle for Base Realignment and Closure Program Management Office West, *Final Feasibility Study Report, Seaplane Lagoon, Alameda Point, California, Appendix E*, July 22, 2005 (P. 20, 24)

[7] *Record of Decision for Site 17 Seaplane Lagoon*. U.S. Environmental Protection Agency, October 2006 (P. 12-2)

off-site disposal, restoration, and ecological monitoring. The costs associated with this suite of alternatives are estimated to be $6.7 million to $6.8 million (2007 dollars).[8]

Hunters Point

The final feasibility study for Hunters Point was finished in 2007. The sediment remediation alternatives developed were similar to Seaplane Lagoon but the actions suggested to best meet the desired remediation goal combined focused removal, off-site disposal, armored cap, monitored natural recovery, and institutional controls. The costs associated with this remediation alternative range from $26.9 to $29.0 million (2007 dollars).[9] The final costs of this cleanup have yet to be determined as this feasibility study is currently under review.

Oyster Point

This site remediation was completed in 2001 and significant sediment removal took place. Approximately two acres were removed at a depth of 2-3 feet with a twelve inch cap put in place for a total cost of $10 million.[10] According to the TMDL, there was at least one sample observed at some point in time with total PCB concentrations of greater than 1000µg/kg.[11] No post-remediation measurements were available to compare the effectiveness of the remediation activities.

San Diego Shipyard

The California Regional Water Quality Control Board, San Diego Region released a tentative cleanup and abatement order for the San Diego Shipyard Sediment Site on September 15, 2010. Proceedings, public comments, and public hearings are currently underway. Under the cleanup and abatement order, over 140,000 cubic yards of contaminated sediments will be removed from approximately 15.2 acres of the site with dredge buckets.[12] Dredging will involve stockpiling and dewatering the dredged material at an off-site sediment staging area in the vicinity of the project area and then transporting and disposing of the material at a regional hazardous waste landfill outside San Diego County (i.e. Copper Mountain, Nevada).[13]

In addition to the 15.2 acres targeted for dredging, approximately 2.3 acres of the project site are inaccessible or under-pier areas that will be remediated by other methods, most likely by sand cover. Sand capping would involve the transport of capping material to the site (possibly via truck or barge) and placement of the materials over contaminated sediment. Before the cleanup begins, an Environmental Impact Report (EIR) will be written to analyze the environmental impacts of sediment management, including the impacts of the

[8] Prepared by SulTech and Tetra Tech EM, Inc. for Base Realignment and Closure Program Management Office West, *Draft Addendum to the Revised Final Station-Wide Feasibility Study Site 25*, June 21, 2005, (P. D-8.)

[9] Prepared by Barajas & Associates, Inc. for Base Realignment and Closure Program Management Office West, *Revised Draft Feasibility Study Report for Parcel F Hunters Point Shipyard, San Francisco, California,* May 11, 2007 (P. 5-2, 5-5)

[10] Correspondence with Randy Lee SFRWQCB July 25, 2007

[11] *San Francisco Regional Water Quality Control Board Final TMDL June 2007* (P.36)

[12] California Regional Water Quality Control Board, San Diego Region website, accessed at: http://www.waterboards.ca.gov/sandiego/water_issues/programs/shipyards_sediment/index.shtml on February 17, 2011.

[13] California Water Resources Control Board - San Diego Region, "Revised Tentative Cleanup and Abatement Order and Draft Technical Report," September 15, 2010, Appendix for Section 32.

proposed dredging activities, handling of the dredged material, dewatering and potential treatment of the dredged material, and transport to the disposal site.[14]

The total cost of cleanup is estimated to be $58 million and includes the cost items in the table below.

Item	**Quantity**	**Unit**	**Unit Cost**	**Cost**
Design and Permitting				$2,323,000
Construction and Preparation				$1,400,000
Dredging subtotal	*178,670*	*cy*		*$3,900,000*
Unconstrained open-water dredging	17,925	cy	$10	$179,250
Constrained dredging from inner shipyard	125,475	cy	$18	$2,258,550
Dredging surface/subsurface debris	7,170	cy	$120	$860,400
Additional dredging	28,100	cy	$18	$505,800
Engineering controls				$96,000
Marine structures				$984,915
Sediment offloading and disposal				$24,781,250
Underpier remediation				$4,799,572
SW04 cleanout, BMP installation, investigation				$703,048
Other non-construction costs				$19,169,473
Total				**$58,061,258**
Total per cy dredged				***$325***

Source: California Water Resources Control Board - San Diego Region, "Revised Tentative Cleanup and Abatement Order and Draft Technical Report," September 15, 2010, Appendix for Section 32.

The following table summarizes dredging cost data for the locations listed above, including data from the Feasibility Studies and Records of Decision. These costs are all significantly larger than the $60.84/cy estimate provided in the Staff Report.

Summary of Remediation Costs for Selected Sites in California

Site	Sediment Removed in cy	Total Cost (2010 dollars)	Cost per cy (2010 dollars)
United Heckathorn	108,000	$19.1 - $19.4 M	$180 - $185
Sea Plane Lagoon	63,000	$27 M	$430
Moffett Air Field	47,400 - 61,500	$5.7 - $9.8 M	$120 - $160
Oyster Point[1]	9,860	$13 M	$1,320
Hunters Point Shipyard[2]	51,910 - 161,000	$12.8 - $112.4 M	$247 - $698
San Diego Shipyard	178,670	$58.1 M	$325

Notes:
(1) The costs per cubic yard for Oyster Point are for all dredging and capping activities
(2) Hunters Point Shipyard are composite costs that range from the complete dredging scenario to comparative unit cost of sediment removed for other alternatives.

[14] California Regional Water Quality Control Board, San Diego Region website, accessed at: http://www.waterboards.ca.gov/sandiego/water_issues/programs/shipyards_sediment/index.shtml on February 17, 2011.

The weighted average of dredging costs in this sample is roughly $200 per cy, which is over three times the unit cost assumed in the Staff Report. It is more appropriate to use this figure for estimating dredging costs since it is based on a sample of dredging projects in other locations, rather than a single, ex ante study.

4. Benefits

The Staff Report contains virtually no discussion of the incremental benefits of the TMDL relative to the status quo. The Regional Board staff have made no attempt to quantify the number of lives saved, illnesses avoided, improvements in wildlife populations, or other types of benefits that typically would be associated with major regulatory action. Indeed, there is no evidence in the Staff Report or other available documents that the actual benefits of implementing the TMDL would be commensurate with even the staff's low estimate of implementation costs.

The Draft TMDL cites the 1994 Water Quality Control Plan for the Los Angeles Region in defining the beneficial uses for the Dominguez Channel and greater Los Angeles and Long Beach Harbor waters. According to the Draft TMDL, Los Angeles and Long Beach harbor waters have designated uses to protect aquatic life including marine (MAR) and rare, threatened or endangered species habitat (RARE). There are also beneficial uses associated with human use of these waters, including recreational use for water contact (REC1), non-contact water recreation (REC2), navigation (NAV), industrial service supply (IND), commercial and sport fishing (COMM), and shellfish harvesting (SHELL). The estuaries (EST) are recognized as areas for spawning, reproduction and/or early development (SPWN), migration of aquatic organisms (MIGR) and wildlife habitat (WILD). Dominguez Channel also has an existing designated use of warm freshwater habitat (WARM) and the Los Angeles River estuary has the designated use of wetland habitat (WET).[15] There are also potential beneficial uses of the Dominguez Channel Estuary (MUNI, REC1, WARM, WILD), the Los Angeles River Estuary (SHELL), and Los Angeles – Long Beach Harbor (SHELL) and San Pedro Bay (SHELL). As noted in the Staff Report, recreation is listed as an actual or potential beneficial use of the Dominguez Channel Estuary even though access to it is currently prohibited by the Los Angeles County Department of Public Works.

The Staff Report does not adequately address the benefits of the proposed regulation. For example, although the Draft TMDL mentions recreational fishing as a beneficial use, it does not elaborate on how many anglers might be consuming fish in the greater Los Angeles and Long Beach Harbors area. Based on data of observed fish catches from the Pacific Recreational Fisheries Information Network, only a handful of species of fish are caught in any significant numbers from the Los Angeles Harbor. Those fish include barred sandbass, California scorpionfish, halfmoon, kelp bass, ocean whitefish, Pacific bonito, Pacific sanddab, and vermillion rockfish.

Number of fish caught at LA Harbor as compared to all of Los Angeles County, 2008

Fish Name	LA Harbor Site	LA County
Bank Rockfish	1	10
Barred Sandbass	**123**	2,502
Black Perch	3	331
Blacksmith	67	806
Boccaccio	93	826
Brown Rockfish	13	171
Cabezon	3	40

[15] Draft TMDL, p. 17.

Calico Rockfish	3	29
California Scorpionfish	**595**	3,552
California Sheephead	50	508
Canary Rockfish	1	4
Chub (Pacific) Mackerel	85	2,972
Copper Rockfish	26	292
Flag Rockfish	17	350
Gopher Rockfish	1	55
Grass Rockfish	2	19
Greenspotted Rockfish	2	509
Halfbanded Rockfish	8	166
Halfmoon	**199**	995
Honeycomb Rockfish	64	781
Jack Mackerel	5	129
Jacksmelt	2	566
Kelp Bass	**218**	2,377
Kelp Rockfish	7	44
Lingcod	4	81
Ocean Whitefish	**107**	801
Olive Rockfish	8	69
Opaleye	2	294
Pacific Barracuda	19	567
Pacific Bonito	**326**	1,687
Pacific Sanddab	**299**	8,706
Rockfish Genus	10	90
Rosy Rockfish	11	184
Rubberlip Seaperch	1	43
Speckled Rockfish	3	268
Spotted Sandbass	1	8
Squarespot Rockfish	11	763
Starry Rockfish	47	442
Treefish	30	152
Vermillion Rockfish	**183**	838
White Croaker	4	664
White Seaperch	2	106
Yellowfin Croaker	1	310
Yellowtail	14	52

Notes and Sources:

(1) 2008 was the most recent year for which complete data were available.

(2) If a fish is not shown in the table, it was not reported as being caught at LA Harbor in 2008.

(3) The Pacific Recreational Fisheries Information Network, accessed at: http://www.recfin.org/data/sampletools/tabulate-sample-data-refined-variable-choices on January 27, 2011.

Of the fish species most commonly mentioned in the Staff Report's survey of the limited fish tissue data available, there are only four reported instances of white croaker being caught in the Los Angeles Harbor in 2008, none for queenfish, none for spotted turbot, and none for halibut. Based on the best available survey data for recreational anglers, it is highly unlikely that there will be significant human health benefits relating to fish consumption as a result of implementing the TMDL.

As mentioned at the outset of this letter, the Staff Report does not contain any information on participation rates for any water-related activities. Thus, it is impossible to know whether staff actually considered the benefits of implementing the TMDL related to these activities.

Other parts of the Staff Report reinforce the conclusion that the TMDL will not produce benefits anywhere near commensurate with its costs. The analysis of pollutant loadings contained in the report shows that staff has concluded that air deposition of pollutants is a major contributor to water quality degradation. This observation calls into question the wisdom of a policy to require dredging since DDT and other contaminants removed by dredging will simply be redeposited by air.

Similarly, the Staff Report does not treat pollutant loading from the San Gabriel and Los Angeles Rivers, but rather calls for a series of "special studies" to analyze the impact of these inflows. As with air deposition, the likely influx of pollutants from an external source raises the potential that the area may be recontaminated after dredging has been completed. Such an outcome would be inefficient in the sense that tremendous resources would have been expended on dredging and other remediation activities as a result of the TMDL, but ongoing deposition would prevent its water quality targets from being attained.

In conclusion, the staff of the Regional Board has not met its burden under Porter-Cologne and U.S. EPA guidance to consider economics in the development of the TMDL. The Staff Report does not evaluate or consider the benefits of improving water quality relative to current water quality levels. The discussion of implementation costs fails to use the best available information, and contains important calculation errors. Actual costs of dredging are likely to be much higher than described. Finally, the Staff Report does not discuss benefits of the TMDL in relation to the costs of implementation, which is the only logical way to assess whether the action is reasonable.

Sincerely,
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Bioavailability to Earthworms of Aged DDT, DDE, DDD, and Dieldrin in Soil

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A study was conducted to determine the bioavailability of several pesticides that have persisted for various periods in soils in the field and the laboratory. Based on the concentrations or the percentages of the compound in soil samples that were found in the earthworm *Eisenia foetida*, ca. 30, 12, 34, and 20% of DDT, DDE, DDD, and a total of the three compounds were bioavailable in a soil treated in the field with DDT 49 years earlier. Only 28 or 43% of dieldrin aged for 49 years was bioavailable based on concentrations in *E. foetida* or percentages of the compound assimilated by the worms, respectively. Comparably low percentages of DDT, DDE, and DDD but not dieldrin were assimilated by the worms from samples of soil from a waste-disposal site receiving the insecticide ca. 30 years earlier. Aging for 190 days in Kendaia loam in the laboratory markedly reduced the availability to *E. foetida* of DDT and DDE but not DDD. The amounts of aged or unaged DDT, DDE, and DDD but not dieldrin that were removed from the soils by solid-phase extraction with Tenax TA beads were generally greater with increasing amounts assimilated by the earthworms. The results show that aging markedly reduces the bioavailability of these compounds.

Considerable evidence exists that organic compounds may undergo a time-dependent sequestration in soil that results in a decline in bioavailability without a parallel decline in the concentration of the compounds determined by vigorous extraction with organic solvents. For example, the toxicity of DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane] and dieldrin to three species of insects (*1*), the inhibition of *Drosophila melanogaster* by lindane (*2*), the assimilation of phenanthrene by earthworms (*3*), the toxicity of atrazine to plants (*4*), and the biodegradability of phenanthrene by bacteria (*5*) decline with time in soil with either little or no diminution in the concentrations determined by vigorous extraction or decreases far less than are evident by biological tests. Such data suggest that current procedures for analyzing organic pollutants that have persisted in soil do not accurately predict the availability of those toxicants to living organisms.

DDT and dieldrin are highly persistent insecticides. For example, more than 50% of the initially applied DDT was present in some soils after more than 15 years (*6*), and DDT, DDE [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane], and DDD [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene] were still in the soil 24 years after addition of DDT (*7*). Similarly, dieldrin was still detected in soil after 15 years (*6*). A reexamination of the data from long-term monitoring of the persistence of these pesticides and a number of other organic compounds suggested that this aging led to a decline in their bioavailability to indigenous soil microorganisms (*8*). Although other studies have shown that the bioavailability of organic compounds to animals is reduced as result of their persistence in soil (*9*−*11*), few data are available that show the quantitative reduction in availability.

An investigation was therefore conducted to assess the decline in bioavailability as a result of aging of DDT, DDE, DDD, and dieldrin in soils in the laboratory and taken from the field. Because the availability to animals of chemicals in solvents is less, often appreciably so, than the same compounds added to soil even without aging (*8*), the assimilation of the unaged insecticides was measured using soil treated with the test chemicals shortly before the bioassays were conducted. This was not generally done in previous studies. The existence of experimental plots treated in 1949 with known concentrations of individual insecticies (*6*) provided a unique opportunity to measure the effect of aging in the field. In addition, data are presented to assess the feasibility of using a solid-phase extractant to determine bioavailability by a chemical assay.

Materials and Methods

Chemicals. DDT (75% *p,p′*-isomer, 18% *o,p′*-isomer), DDE (99.5% pure), DDD (99.5% pure), and dieldrin (99.5% pure) were obtained from ChemService (West Chester, PA). Hexane (HPLC grade), acetone (ACS grade), diethyl ether (reagent grade), and Florisil (60/100 mesh) were obtained from VWR Scientific Products (Bridgeport, NJ). Tenax TA 20/35 mesh, a porous polymer based on 2,6-diphenyl-*p*-phenylene oxide, was purchased from Alltech Associates (Deerfield, IL).

Soils. Samples of Chester loam (pH 5.5, 6.5% organic matter) and Sassafras silt loam (pH 5.2, 4.4% organic matter) that had been treated with DDT and dieldrin in 1949 or that did not receive the insecticides were obtained at depths of 0−25 cm from experimental plots at the Beltsville Agricultural Research Center (U.S. Department of Agriculture, Beltsville, MD). DDT and dieldrin initially had been mixed in separate plots at rates of 448 and 11.2 kg/ha of soil, respectively (*6*).

Samples of Kendaia loam (pH 6.6, 12.5% organic matter) were obtained from Aurora, NY. Samples of a sandy loam (pH 6.6, 6.0% organic matter) that had been contaminated with DDT and dieldrin approximately 30 years earlier and an adjacent uncontaminated silt loam (pH 6.4, 5.4% organic matter) from a remediation site at the U.S. Navy Surface Weapons Testing Center in Dahlgren, VA, were provided by Remediation Technologies.

Earthworms. Mature redworms (*Eisenia foetida*) from Carolina Biological Supply (Burlington, NC) were maintained in an aerated Styrofoam box containing a commercial worm bedding (Magic Products, Amherst Junction, WI), which was kept moist with Cl_2-free deionized water. The worms were fed a mixture of crude protein and carbohydrate (Magic Worm Food) and were active when introduced into the soils.

Determination of Pesticide Residues. Worms were frozen at −10 °C and ground with a mortar and pestle. The ground tissue (ca. 2 g) or 10 g of soil was subject to Soxhlet extraction by EPA Method 3540 (*12*) except that 150 mL of a 1:1 hexane: acetone mixture in a 25-mL round-bottom flask was used for the extraction. The tissues or soil samples were mixed with

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10 g of anhydrous Na_2SO_4 in paper extraction thimbles and then extracted for approximately 16 h. The extracts were concentrated to 10 mL in a vacuum evaporator and then cleaned on a Florisil column by EPA Method 3620 (*12*). The fractions containing DDT congeners or dieldrin were concentrated under vacuum to 2 mL and then diluted to 10 mL in hexanes.

The chemicals were analyzed with a gas chromatograph (model 5880, Hewlett-Packard) equipped with an electron-capture detector according to EPA Method 8080 (*12*) except that an HP-608 column (30 m, 0.53-mm i.d., 0.5-μm film thickness; Hewlett-Packard) was used, the N_2 (99.99% pure) flow rate was 10 mL/min, and the temperatures of the column, injector, and detector were 225, 275, and 275 °C, respectively. The retention times of DDT, DDE, DDD, and dieldrin were 4.7, 2.8, 4.0, and 3.0 min, respectively. The concentrations found in Chester loam treated in 1949 with DDT were 10.0 mg of DDT, 5.38 mg of DDE, and 4.10 mg of DDD/kg. Chester loam treated in 1949 with dieldrin contained 9.56 mg of the insecticide per kilogram. The soil from the Dahlgren remediation site contained 81.6 mg of DDT, 9.64 mg of DDE, 33.2 mg of DDD, and 9.35 mg of dieldrin per kg.

Aging of Chemicals in Laboratory. DDT, DDE, and DDD in hexanes were added to 2 kg of Chester loam that had not previously been treated and contained no pesticides. The final concentrations were 13.6, 5.28, and 3.26 mg/kg of soil, respectively. The soil was thoroughly mixed with a Teflon-coated spatula and stored in an EPA-certified ultraclean 1-L glass jar with a Teflon-lined screwcap. The height of the headspace above the soil was 5 cm. The soil was stored in the dark for 90 days at approximately 22 °C, and then earthworm uptake of the compounds was determined.

Prior to aging, 3.0−3.2 kg of Kendaia loam (in a 4-L glass jar with a Teflon-lined cap), which had been sterilized by 2.5 Mrad of γ-irradiation, was amended with DDT, DDD, DDE, or dieldrin under aseptic conditions. The first three compounds dissolved in hexane or dieldrin dissolved in acetone were added to soil, which was then mixed thoroughly with a Teflon-coated spatula. The final concentrations of DDT, DDD, DDE, and dieldrin were 46.6, 11.2, 19.8, and 13.6 mg/kg, respectively. The soils were placed in a hood for 2−3 days with the caps of the jars slightly loosened to allow the solvent to evaporate. The moisture content was then adjusted to 80% of field capacity. The bottles were stored at approximately 22 °C in the dark for aging. No microbial growth was observed in 7 days on nutrient agar to which 0.1-g samples of soil were added prior to and after the aging period.

Bioavailability. Six redworms were placed in 60 g (dry wt) of pesticide-amended soil contained in 250-mL glass jars. The soil had been adjusted to 90% of field capacity with Cl_2-free deionized water before the worms were added, and the jars were covered with Saran wrap bearing holes for air entry. The soils were kept under constant room lighting, and after 8 days, the worms were carefully removed, rinsed, and allowed to purge their gut contents for 24 h on moistened filter paper. All worms were active after the 8-day period. The worms in each soil sample were then weighed, the mass varying from 1.5 to 2.5 g of fresh weight per replicate. The worms were sealed in glass Petri dishes, frozen at −10 °C for 25−48 h, and ground with approximately 10 g of anhydrous Na_2SO_4 with a mortar and pestle. The tissue was transferred to paper thimbles and subject to Soxhlet extraction, and the extracts were cleaned with a Florisil column and analyzed.

To compare the bioavailabilities of compounds that had aged in Chester loam for 49 years in the field or 90 days in the laboratory relative to the bioavailabilities of these pesticides when freshly added to soil, DDT, DDE, and DDD were thoroughly mixed into pesticide-free soil to give 13.6, 5.28, and 3.26 mg/kg of soil, respectively. These were the concentrations found after 49 years in the soil. Each treatment was replicated four times.

In a study of the relative bioavailabilities of compounds aged in Kendaia loam in the laboratory for 0 and 190 days, triplicate soil samples were amended with DDT, DDE, or DDD to give 46.6, 19.8, and 11.2 mg/kg.

A determination was also made of the uptake by worms of dieldrin that had aged for 49 years in the field and that had been freshly added. The labels in the experimental plots that gave the original soil types had deteriorated, and sometimes were illegible. Consequently, although the aged dieldrin was in Chester loam, Sassafras silt loam was inadvertently used for the unaged dieldrin. The soil in which the dieldrin was not aged was amended to give 9.56 mg/kg, which was the concentration of aged dieldrin that was found in the field. Four replicate samples were used.

Measurements were also made of the bioavailabilities of DDT, DDE, DDD, and dieldrin present for approximately 30 years in soil from the remediation site in Dahlgren and in an adjacent soil to which the compounds were freshly added. DDT, DDE, DDD, and dieldrin were added to the uncontaminated soil to give 45.9, 11.0, 15.6, and 13.6 mg/kg, respectively.

The data for earthworm uptake are expressed on the basis of fresh weight of tissue.

Solid-Phase Extraction. Tenax TA was used by the method of Cornelissen et al. (*13*) with slight modification. A single extraction rather than consecutive extractions was performed. Soil (0.2 or 0.5 g) was placed in a 30-mL glass separatory funnel equipped with a Teflon stopcock and stopper. Sterile inorganic salts solution (25 mL) containing 10 mg of Na azide to prevent biodegradation and 0.1 or 0.2 g of Tenax TA beads were then added to the funnels. The salts solution contained 0.8 g each of K_2HPO_4 and NH_4NO_3, 0.1 g each of $MgSO_4 \cdot 7H_2O$ and $CaCl_2 \cdot 2H_2O$, and 0.1 g of $FeCl_3 \cdot 6H_2O$/L. The beads had been initially conditioned by washing with acetone (1 g in 10 mL solvent) followed by rinsing three times in hexane (1 g in 10 mL solvent) and then drying. The funnel was placed inside a rotary extractor designed in accordance with EPA specifications (*12*). The sample was shaken end-over-end at 20 rpm for 14−16 h. Tenax TA beads were found to be an adequate sink for organic pollutants in sediments (*14*). Subsequent extractions did not show a detectable level of the compounds. The single extractions appeared to be complete in <14 h.

The soil particles were allowed to settle, and the Tenax beads, which floated at the top of the liquid or adhered to the walls of the funnel, were removed and extracted with 10 mL of hexane. Because the beads float in aqueous solution, they were easily separated from the solids and surfaces, and all were recovered. The extracts were concentrated under N_2 to 1 mL and analyzed by gas chromatography using an electron-capture detector.

To ascertain the recovery by the beads of unaged DDT or dieldrin in soil, known amounts of the compounds were added to Kendaia loam, and 1 g of amended soil was mixed with various amounts of beads in 25 mL of inorganic salts solution contained in separatory funnels as previously described. The beads were separated from the water and extracted with hexane or acetone. The recoveries of the two compounds from soil ranged from 90 to 95%.

The results showed that 0.1 to 0.2 g of Tenax TA beads had sufficient sorption capacity to extract DDT or dieldrin from 0.2 to 0.5 g of soil containing up to 12% organic matter.

Results

Earthworm Bioassays. A determination was made of the availabilities to redworms of DDT, DDE, DDD, and dieldrin aged in soil for 49 years, 90 days, or 0 days. The concentrations of DDT, DDE, DDD, total of DDT and metabolites (including

TABLE 1. Uptake of Aged and Unaged DDT, DDE, DDD, and Dieldrin in Chester Loam by *Eisenia foetida*

compound	aging period	concn (mg/kg tissue)	uptake (%)[a]
DDT	49 years	4.54 C[b]	1.40 C
	90 days	27.7 A	7.03 A
	0 days	14.0 B	4.98 B
DDE	49 years	3.06 C	1.75 C
	90 days	14.4 B	9.23 B
	0 days	22.9 A	15.2 A
DDD	49 years	1.71 B	1.30 B
	90 days	5.12 A	5.41 A
	0 days	4.61 A	4.00 A
DDT + DDE + DDD	49 years	9.31 C	1.64 B
	90 days	47.2 A	7.32 A
	0 days	41.5 B	7.59 A
dieldrin[c]	49 years	15.1 B	4.48 B
	90 days	ND[d]	ND
	0 days	53.5 A	10.8 A

[a] Percentage in soil that was assimilated by the worms. [b] Values in a column for any one compound or group of compounds that are not followed by the same letter are significantly different ($P < 0.01$). [c] Unaged dieldrin in Sassafras silt loam. [d] ND, not done.

DDE and DDD), and dieldrin were consistently lower in earthworms exposed to compounds that had persisted for 49 years than in worms exposed to soil containing the insecticides freshly added at the same concentrations (Table 1). If percentage sequestered is calculated as the difference in concentration between worms exposed to unaged and aged compound divided by the former concentration, then the percentages of DDT, DDE, DDD, DDT plus metabolites, and dieldrin sequestered are 68, 87, 63, 78, and 72%, respectively. The percentage of each of the compounds in soil that was assimilated by the worms was consistently and significantly lower for each of the aged than for the unaged insecticides. No such effect was evident with the compounds aged for 90 days, except for DDE. The lack of a detectable aging effect in these instances may be a result of the short time period, especially since aging of DDT sometimes is quite slow (*8*). The reason for greater uptake of DDT aged for 90 days than of unaged DDT is unknown. Based on the percentage of the compound assimilated by the animals, 72, 89, 68, 79, and 57% of the DDT, DDE, DDD, DDT plus metabolites, and dieldrin had been sequestered in the 49-year period. If the percentage sequestered in samples aged for 49 years was calculated for the data from the Tenax extractions following the procedure used for the data on earthworm uptake, the percentages of DDT, DDE, DDD, and total DDT and metabolites sequestered are 67, 74, 75, and 70%, respectively. The amount of the aged compound that was extracted by the Tenax beads was consistently less than the amount of the unaged compound. An aging effect was not evident for dieldrin.

A marked aging effect was observed when DDT and DDE were aged for 190 days in Kendaia loam (Table 2). The decline in bioavailability of DDT, DDE, and DDT plus metabolites was evident in measurements of concentration in the worm tissue and percentage of the compound assimilated. Aging of DDD was not evident by measurement of tissue concentration or percentage of the compound assimilated; indeed, the concentration of DDD aged for 190 days was higher in the worms in Kendaia loam. The percentage of DDT, DDE, and DDT plus metabolites sequestered were 46, 82, and 36%, respectively, based on tissue concentrations and 85, 73, and 74%, respectively, based on percentage of the compound assimilated.

The bioavailability of the insecticides after approximately 30 years of aging in the Dahlgren soil is shown in Table 3. Because the texture and organic matter and clay contents of

TABLE 2. Uptake by *Eisenia foetida* of DDT, DDE, and DDD Aged for 0 and 190 Days in Kendaia Loam

compound	aging period (days)	concn (mg/kg tissue)	uptake (%)
DDT	190	99.0 B[a]	1.13 B
	0	183. A	7.33 A
DDE	190	4.7 B	0.62 B
	0	26.4 A	2.29 A
DDD	190	45.6 A	5.90 A
	0	24.4 B	4.04 A
DDT + DDE + DDD	190	149. B	1.45 B
	0	234. A	5.57 A

[a] Values in a column for any one compound or group of compounds that are not followed by the same letter are significantly different ($P < 0.05$).

TABLE 3. Bioavailability of DDT, DDE, DDD and Dieldrin to Earthworms after Approximately 30 Years of Aging and with No Aging in a Silt Loam from Dahlgren, VA

compound	aging period[a]	concn (mg/kg tissue)	uptake (%)
DDT	30 years	38.4 ± 0.9[b]	2.02 ± 0.11
	unaged	28.3 ± 8.4	2.85 ± 0.88
DDE	30 years	3.50 ± 0.24	1.56 ± 0.11
	unaged	3.77 ± 0.48	1.11 ± 0.15
DDD	30 years	8.83 ± 0.61	1.15 ± 0.10
	unaged	11.5 ± 1.8	4.80 ± 0.77
DDT + DDE + DDD	30 years	50.7 ± 1.7	1.75 ± 0.10
	unaged	43.6 ± 10.0	2.77 ± 0.67
dieldrin[b]	30 years	6.13 ± 2.1	19.9 ± 1.7
	unaged	40.0 ± 5.3	12.8 ± 2.8

[a] Aged for ca. 30 years in sandy loam. Unaged in silt loam. [b] Mean ± standard error.

the two soils from the Dahlgren site differed appreciably, a comparison could not be made of the effect of aging in the contaminated soil, although the data from both soils were used to determine the utility of Tenax TA beads for assessing bioavailability. Nevertheless, a comparison of the data from this soil with the data in Table 1 from the soil samples aged for 49 years in Chester loam shows uniformly low values for percentages of the compound assimilated for the DDT species but not dieldrin. In contrast, the concentrations in the worm tissue were consistently higher for samples of Dahlgren soil, but this is not unexpected because the total concentrations of the various DDT species (but not dieldrin) were higher in the Dahlgren soil.

Assay with Tenax Beads. The quantity of aged and unaged DDT, DDE, and DDD extracted in triplicate by the beads from Chester loam, Sassafras silt loam, Kendaia loam, and the two soils from the Dahlgren site was determined. The standard errors for the values for all soils were <4% except that the standard errors for DDT aged in Chester loam were up to 14%. An analysis of variance ($P < 0.05$) showed that the values from soils containing aged DDT and metabolites were significantly different from the values from soils in which the compounds were freshly added except the values were not significantly different for DDD in Dahlgren soil and DDE in Kendaia loam.

A comparison was made of the quantities of DDT, DDE, DDD, and a total of the three compounds assimilated by the worms with the amounts retained by the Tenax TA beads (Figure 1). In soils from which the animals assimilated only small amounts of each compound, little was sorbed by the beads. Similarly, when worm uptake was high, a large amount was retained by the beads. As a result, the values for linear regression were high; i.e., the correlation coefficients (r) for the assays for DDT, DDE, and DDD were 0.933, 0.980, and

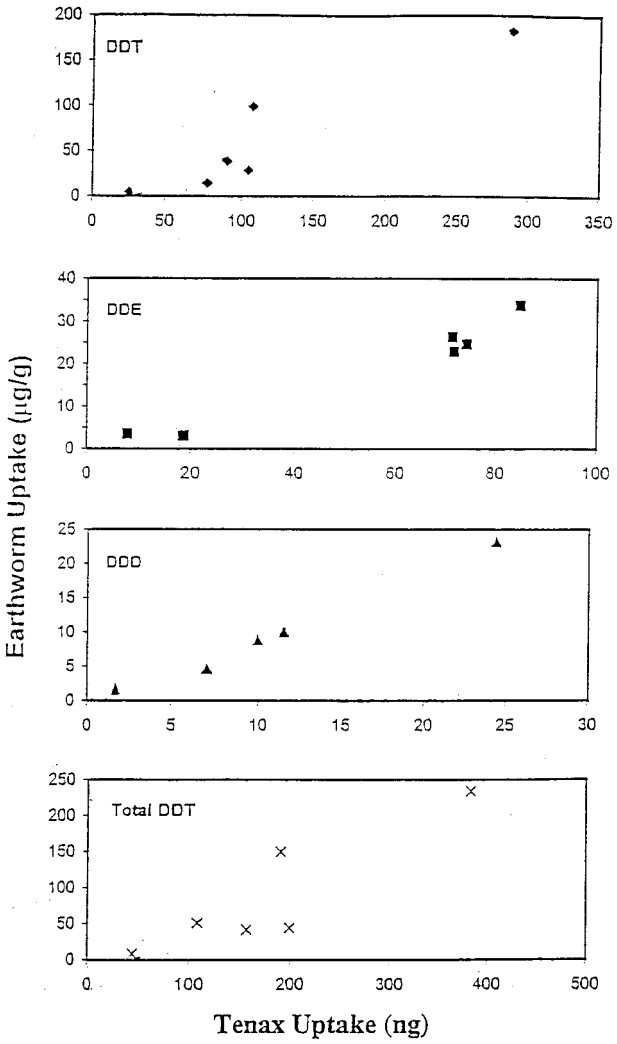

FIGURE 1. Correlation between earthworm uptake of DDT, DDE, DDD, and total of the three compounds from soil and the quantities extracted by Tenax TA beads.

0.995, respectively. The corresponding values for the adjusted R^2 were 0.850, 0.954, and 0.982, respectively. The results of the Tenax assay for DDT plus metabolites were also correlated ($r = 0.960$) with the concentration in earthworms. The corresponding value for the adjusted R^2 was 0.855. The value for total DDT species extracted was based upon three results from a single replicate set of beads that was assayed for DDT, DDE, and DDD. Nevertheless, a relationship between worm uptake and bead retention of DDT is not evident at the intermediate levels of assimilation. This is also evident in the totals of the three compounds since the values for totals are dominated by DDT as the major substance being analyzed. A clear correlation is shown between worm assimilation and bead retention of DDD, but it was not the major substance in these soils.

In contrast, the amount of dieldrin extracted from the soils by the Tenax resins was not correlated ($r = -0.215$) with the concentration of the insecticide in the earthworms after assimilation. The corresponding value for the adjusted R^2 was 0.036. The data are from analyses of Chester loam, Sassafras silt loam, and the two soils from the Dahlgren site.

Discussion

The results demonstrate the extensive decline in bioavailability to earthworms as a result of the aging of DDT, DDE, DDD, and dieldrin in field soils. The finding that more than half to >85% of the pesticides was not in a form accessible to the test species is of considerable importance in assessing the exposure and risk from these compounds. The observations that the availability to animals in field soils has diminished as a result of aging agree with laboratory studies of the decline in acute toxicity. Thus, as early as 1971, Peterson et al. (*15*) reported that the toxicity of DDT to *Drosophila melanogaster* declined as a soil was stored for a 108-day period, and recent tests with three insect species revealed the marked diminution in toxicity of both DDT and dieldrin as a result of aging, although the concentration of the two insecticides determined following vigorous extraction of the soils changed only slightly (*1*). The decline in bioavailabilty to animals is also consistent with the apparent reduced accessibility for biodegradation by the indigenous microflora of soils, as suggested by a reevaluation (*8*) of the patterns of disappearance in the field shown by repeated measurements of the concentrations of DDT (*6, 7, 16, 17*), DDE, DDD (*7*), dieldrin (*6*), and dieldrin and its aldrin precursor (*17*−*19*). Unavailability for microbial utilization is also shown by the resistance to bioremediation of 25% of the DDT and 65% of a breakdown product in a contaminated soil (*20*).

Although the bioavailability of each of the compounds was low after aging, some was still assimilated by *E. foetida* even after an aging period of 49 years. Thus, despite the fact that the accessibility has fallen sharply, the insecticides still can enter the animal and some exposure and risk remain. This view is consistent with the observation that DDT used in the field for termite control was still suppressing the termites after 33 years (*21*) and that the pesticide was still assimilated by aquatic animals many years after it entered coastal sediments (*22*). In addition, dieldrin present in a field site contaminated many years earlier was assimilated by rats that were given the contaminated soil by gavage (*23*).

Inasmuch as the data show differences in bioavailability among different soils, it is important to devise a useful method to assess bioavailability. Biological assays would serve this purpose, but they are typically slow, expensive, and lack precision. Thus, a chemical assay would be useful. A number of procedures have been proposed, including organic extractants (*24*), equilibrium partitioning (*25, 26*), high-temperature desorption (*13*), and analyses of pore water (*27*). In only some of the studies have correlations been made with bioavailability; for example, to earthworms (*24, 26*), nematodes (*27*), and microorganisms (*24*). The present investigation considered a different procedure, and it includes testing of soils with dissimilar properties as well as compounds that had been freshly added to soil or sequestered under both field and laboratory conditions for varying time periods.

The data show that the values for uptake by worms from soils with low or high bioavailability percentages of DDT, DDE, and DDD were correlated with retention with Tenax TA beads. However, a correlation was not evident at intermediate bioavailability percentages. It is not clear whether this lack of relationship is a result of soil properties, aging time, or some other factor. Although Tenax TA beads might thus be useful to distinguish among soils with low and high bioavailabilities, a more useful procedure appears to be our recently described technique using C18 membrane disks (*28*). With certain test conditions, this method gave correlations with *r* values of 0.967, 0.984, and 0.940 for DDT, DDE, and DDE, respectively, and the relationship between bioavailability and retention by the disks was clear even at intermediate bioavailabilities.

Acknowledgments

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How Toxic Are Toxic Chemicals in Soil?

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Assessments of the hazard of toxic chemicals in soil are made without concern about the possibility that their bioavailability may change with time. The patterns of disappearance of persistent compounds in the field and laboratory studies show a declining availability to microorganisms with residence time in soil. Changes in extractability with residence time and the kinetics of sorption and desorption suggest that the compounds are becoming sequestered in inaccessible microsites within the soil matrix. Diminishing toxicity as chemicals age in soil is evident in a limited number of assessments. Such findings suggest that the hazard and risk from toxic chemicals diminish as the compounds persist in soil.

Most polluted soils or subsoils that are currently being considered for remediation were contaminated many years ago. These soils were contaminated before there was widespread concern with environmental deterioration and before strict regulations were established and the high cost of remediation became evident. Nevertheless, assessments of the hazards from those sites have not taken into account slow processes that may take place and possibly reduce the impact of toxic compounds deposited in the soil, particularly those compounds that do not leach out to contaminate underlying aquifers. In recent years, evidence has accumulated that the availability of certain organic compounds changes as the compounds reside in soil for some time, a process that has been termed aging. Data have also been collected suggesting that organic molecules slowly become sequestered within the soil matrix. The declining availability and sequestration appear to be related, and a consideration of the declining bioavailability and the occurrence of chemical sequestration has great relevance to assessing toxicity, determining risk, and establishing meaningful regulations for the cleanup of sites containing hazardous wastes.

Three lines of evidence point to the sequestration of organic molecules that persist in soil: (a) field and laboratory studies demonstrating a diminishing availability to microorganisms; (b) investigations of the extractability of aged chemicals and the kinetics of sorption and desorption; and (c) assessments of toxicity. Although the issue of toxicity is the most relevant for decisions on risk and for environmental regulations, that line of evidence is based on few studies. However, all three lines of evidence are consistent and point to the need for a modified approach to assessing risk.

Diminishing Availability to Microorganisms

Long-term monitoring of organic compounds in soils has been chiefly restricted to pesticides, especially the chlorinated hydrocarbons that were once widely used as insecticides. These field measurements do not distinguish among losses resulting from biodegradation, volatilization, or abiotic decomposition. However, all the pesticides are biodegradable so that a chemical which disappeared initially but not in later periods must be less susceptible to all loss mechanisms, including degradation by microorganisms in the soil. The disappearance of appreciable amounts of these insecticides from the field sites was not a result of leaching because all are extensively sorbed and little vertical movement has been detected even after many years.

These persistent pesticides initially disappear from soil at reasonable rates, but frequently the rate subsequently slows markedly. A typical example is DDT [1,1,1-trichloro-2,2-*bis*(*p*-chlorophenyl)ethane]. Almost three decades ago, Nash and Woolson (*1*) reported a slow but continuous disappearance of the compound in the 10 years after its addition, but little or none of the compound was lost in subsequent years (Figure 1). Because this insecticide is metabolized by many microorganisms and microbial activity is a major contributor to its disappearance (*2*, *3*), prevailing conditions were conducive to their activity. However, some slow change occurred with the passage of time that rendered the compound increasingly less available to the microflora. Low winter temperatures and occasional drought periods can be ruled out for the lack of detectable biodegradation because the persistence curve extends for numerous seasons and many years. Similar curves, each with an initial phase of loss followed a period of little or no detectable loss, have been reported for DDT added to many soils in many areas of the world from the 1960s to the present time (*3*–*6*).

Field monitoring has shown analogous "hockey-stick" shaped curves for a variety of other chlorinated hydrocarbons; e.g., aldrin and its epoxide, dieldrin (*1*, *4*, *7*, *8*), heptachlor and its epoxide (*1*, *9*, *10*), chlordane (*1*, *4*), kepone (*11*), nonylphenol, and a linear alkylbenzene sulfonate (*12*). Examination of the data from these field monitoring

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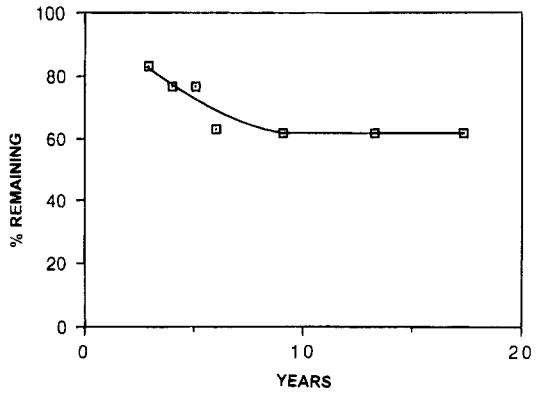

FIGURE 1. Concentration of DDT in Chester loam amended with 200 mg of the insecticide/kg of soil. Replotted from the data of Nash and Woolson (*1*).

programs reveals enormous differences in the time at which the initial phase of decline ends and the percentage of the original compound that remains following that decline. The time is sometimes as short as 1 year, but it may be longer than 10 years. From 10 to about 60% of the original compound remains in the soil during the period when there is little or no loss. Even with a single compound, the times and percentages may vary greatly; e.g., for DDT, aldrin, and dieldrin. A relationship to soil type or climate is not evident from the available data, but a systematic study was never conducted to establish such a relationship. It is possible that these differences in times and percentages can be attributed to the relative rates of degradation (or loss by volatilization) and the sequestration of the compounds in unavailable forms. If the loss is rapid compared to sequestration, little will remain. If the loss is slow and sequestration is rapid, a higher percentage will remain. Moreover, if the loss is so rapid that little time elapses for the slow sequestration, a residual, persistent amount will not be detected; this is the case for the many pesticides and other organic compounds that rapidly disappear from soil because of their degradation, volatilization, or leaching.

Laboratory studies confirm the unavailability to microorganisms of molecules that have been in soil for long periods. Thus, little or no loss was detected of 1,2-dibromoethane in a soil treated in the field 3 years earlier and of simazine [2-chloro-4,6-*bis*(ethylamino)-*s*-triazine] from a field that received the herbicide for 20 consecutive years, although newly added ^{14}C-labeled 1,2-dibromoethane and simazine were extensively metabolized in several weeks (*13*, *14*). Similarly, polycyclic aromatic hydrocarbons present in a soil at a manufactured gas site did not disappear in a 3-month period in the laboratory, but naphthalene and phenanthrene freshly added to the soil were rapidly metabolized (*15*).

A somewhat different approach to demonstrate aging was used by Hatzinger and Alexander (*16*). They introduced phenanthrene, a hydrocarbon that is not readily lost by abiotic mechanisms, into sterile soil and added a phenanthrene-degrading bacterium after the hydrocarbon had aged for different periods of time. The data with a soil rich in organic matter show that the extent of microbial conversion of phenanthrene to CO_2 and the rate of biodegradation decline with increasing time of contact with the soil (Figure 2). Similar observations were made with a soil with a lower level of organic matter, although aging appeared to be slower.

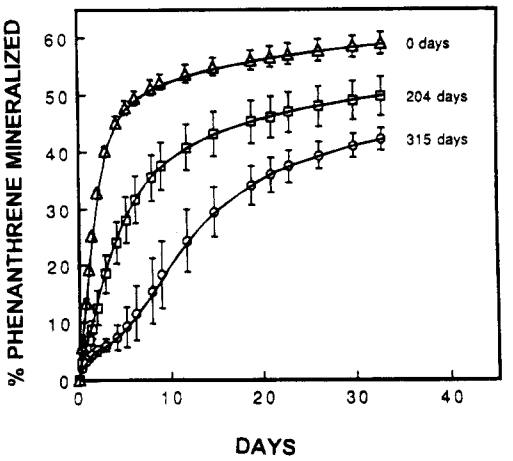

FIGURE 2. Biodegradation by a strain of *Pseudomonas* of phenanthrene at 10 mg/kg that had aged for 0, 204, or 315 days in Edwards muck. The error bars represent the standard deviations (*16*).

These field and laboratory investigations show that some abiotic process(es) make(s) organic compounds less readily available to microorganisms. The impact of that process or those processes becomes greater as the residence time of the chemicals increases. The fact that such a change in bioavailability affects microorganisms is of relevance to higher organisms because presumably bacteria are more able than plants and animals to assimilate chemicals in soil in view of their small size, their high population densities, and the physiological versatility of the indigenous microflora.

Extractability, Sorption, and Desorption

Little difficulty is usually encountered in finding an extractant that gives quantitative recoveries of organic compounds shortly after their introduction into soil. However, if a chemical persists and thus remains in contact with the particulate matter for some time, it becomes more and more resistant to extraction by many solvents. This has been known for many years by chemists endeavoring to obtain quantitative recoveries of insecticides and herbicides used for pest control (*17–19*). A diminished availability for solvent removal also has been noted in a sterile organic matter-rich soil and a loam that was incubated with phenanthrene under aseptic conditions (*16*). The organic solvents that extracted all of the newly added compound removed increasingly smaller amounts as the chemical persisted. More recent studies show that phenanthrene aged in sterile soil similarly becomes, with time, progressively less extractable with acetonitrile/water (1:1) and various ethanol/water mixtures (B. D. Kottler, J. W. Kelsey and M. Alexander, unpublished data). This represents another line of evidence that a change is occurring that renders persistent molecules less accessible, whether accessibility is assessed in microbiological or chemical terms.

Molecules that behave in this way are not to be confused with bound residues. Such bound residues, which have been well studied for pesticides, are those residual fractions that are not extracted by procedures that do not appreciably alter the nature of those residues; these molecules are changed in some manner and are usually converted to the original compound by vigorous hydrolysis. In contrast, aged compounds can be extracted by some organic solvents, often under vigorous conditions, and are thus subject to regulation if the original compounds are toxic in solution.

Studies of kinetics of sorption and desorption provide another perspective on the behavior of toxicants that is relevant to issues of risk. For hydrophobic as well as some other compounds, the attainment of apparent equilibrium in sorption may require weeks, months, and sometimes possibly even years; i.e., more and more of the organic compound is sorbed by soil particles with the passage of time. The initial phase of sorption is rapid, and frequently about half the chemical in aqueous solution is removed by the soil in a few minutes or hours. This first phase is followed by a considerably slower uptake, which can be of prolonged duration. Such kinetics have been observed not only with soil but also with aquifer solids (*20–24*), and they suggest that the sorption involves not only the external surface of the particles but also a slow and continuing diffusion of the molecules to sites within the particles. The internal and more remote sites continue to bind more of the compound with increasing time.

The desorption of many chemicals similarly shows a rapid phase followed by a period of slow desorption. Such behavior has been noted for polychlorinated biphenyls, trichloroethylene, tetrachloroethylene, toluene, xylene, picloram (4-amino-3,5,6-trichloropicolinic acid), and atrazine (2-chloro-4-ethylamino-6-isopropylamino-*s*-triazine). Furthermore, the longer the compound remains in soil, the less is the amount released (*24–27*). Such data suggest that, following the initially rapid release from sites very near to the particle–water interface, the slow appearance of the chemical in solution results from molecules originating from sites at some distance from the interface. The longer aging time allows more of the molecules to diffuse to ever more remote sites. Furthermore, the fraction of a compound that is not readily desorbed under natural conditions will increase with time because that is the fraction that will be less readily biodegraded, leached, or volatilized.

Sorption of nonionic compounds in soil is viewed as a three-dimensional process. The longer a chemical remains in the soil, the greater is the amount present in remote sites and the further is the bulk of the molecules from the more accessible, outer sites (*28*). Aging thus is presumably associated with a continuous diffusion into more remote sites, where the molecules are retained. Such a conceptualization is consistent with the findings of initially rapid followed by slow phases of both sorption and desorption. Furthermore, this view may explain the decreasing availability to microorganisms of organic compounds as they undergo aging, and it is of direct relevancy to the issue of toxicity to higher organisms of chemicals that have been in soil for extended periods.

Intraparticle or intraorganic matter diffusion is believed to account for the slow phases of sorption and desorption of nonionic molecules. According to the intraparticle diffusion model, the solute is within micropores inside of soil particles. Diffusion is greatly retarded by (a) the partitioning of the compound between the liquid in the pore and the pore wall and (b) the tortuous path between micropores before the compound reaches the outer surface of the micropore-filled soil particle (*29, 30*). Although the diameters of micropores within soil particles extend over a wide range, a considerable part of the pore volume is made up of pores with effective diameters of $<1.0\ \mu$m, and examination of several soils and solids from aquifers reveals an abundance of pores with diameters of 20 nm or smaller (*31–34*). Such pores are so small that even the smallest bacterium, animal, root, or root hair could not penetrate.

Intraorganic matter diffusion of hydrophobic molecules may essentially reflect a partitioning into the native organic matter of soil, the molecules moving into the amorphous humic polymers in a fashion similar to the movement of a hydrophobic compound from water to an organic solvent. However, the importance of partitioning into soil organic matter to slow sorption and desorption has been questioned (*35, 36*). Nevertheless, intraorganic matter diffusion is cited as being important for the slow sorption of a number of hydrophobic compounds by soil (*35*).

Regardless of which mechanism applies to a particular site or which is more important for a given soil, the outcome for bioavailability and toxicity of molecules is, for all intents and purposes, the same. Whether the molecule is present in a remote micropore, has partitioned into some solid organic phase in the soil, or both, it has become sequestered. In this sequestered state, it is inaccessible to microorganisms, plants, and animals. This physical remoteness of aged compounds and their very slow diffusion to locations that are biologically inaccessible are key considerations in assessing risk of toxicants in soil.

Assessments of Toxicity

The line of argumentation presented above—which follows from knowledge of the chemistry of sorption and the effect of aging on the availability of biodegradable substrates to microorganisms—is scarcely reflected in the literature of toxicology. Indeed, not a single investigation has been conducted of the possibly diminishing effect on mammals as toxicants age in soil, although in several studies, samples were taken from soils that were contaminated long before the assay was conducted. The difficulty in interpreting these investigations in terms of an aging effect is the inability to determine whether the diminished toxicity occurred only immediately after the compound came into contact with soil or whether there was an increased diminution of toxicity as the compound became sequestered in soil. Many toxicants become less hazardous to test mammals within a short time after they are added to soil or aquifer solids, regardless of whether exposure is by oral or dermal routes; e.g., trichloroethylene, benzo[*a*]pyrene, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), and *m*-xylene (*37–40*).

Such reservations on the possible involvement of aging must thus be considered in interpreting data showing that compounds present in soil for long periods are less toxic than the same compounds provided to test animals in the absence of soil. For example, less TCDD was taken up by rabbits and guinea pigs and far fewer of the guinea pigs were killed following their oral exposure to soils that had been contaminated years earlier than following exposure to newly treated soils (*41, 42*). A surprising effect of short-term aging was noted by Poiger and Schlatter (*40*), who found that less TCDD was absorbed by mammals from soil that had been in contact with this dioxin for 8 days than for 10–15 h. The work of Edwards et al. (*43*), although with insects rather than mammals, is noteworthy. Chemical determination and quantitative bioassays of the toxicity of lindane (γ-isomer of hexachlorocyclohexane) to *Drosophila melanogaster* were in good agreement shortly after adding this insecticide to the soil, but the results of the bioassays suggested that less of the toxicant was present after 22 months than shown by chemical analysis; i.e., although all of the compound was initially bioavailable, only a part was affecting the fruit flies in soil in which the molecule had

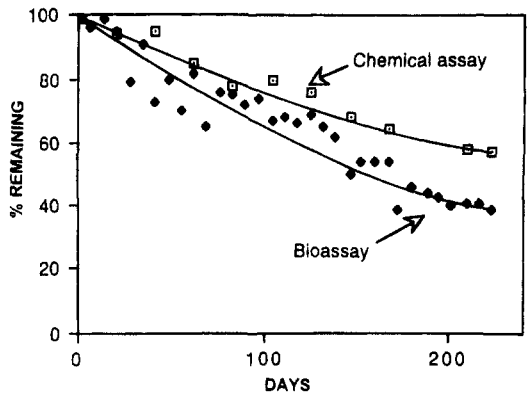

FIGURE 3. Changes with time in concentration of napropamide in soil as measured by bioassay and by extraction followed by gas chromatogrpahy. Calculated from the data of Hurle (45).

persisted for almost 2 years. An effect of aging also is suggested by a report that sugarbeet (*Beta vulgaris*) seedlings were not inhibited when grown in a fine loam that contained 0.22 mg of simazine/kg of soil derived from a field that was amended with the herbicide for 20 consecutive years, although 26 of 51 sugarbeet seedlings when grown in the same soil freshly amended with simazine at 0.22 mg/kg were injured by the newly introduced chemical (*14*).

Additional evidence that some compounds become progressively less toxic with increasing residence time comes from several old and largely forgotten studies. In one, the toxicity of DDT was found to decrease with an increase in the time during which the compound remained in soil (*44*). In another study, the rate of disappearance of napropamide [2-(α-naphthoxy)-*N,N*-diethylpropionamide] was faster when measured by quantitative bioassays of toxicity of the herbicide-treated soil to oats (*Avena sativa*) than when determined by gas chromatography (Figure 3); i.e., decreasing percentages of the herbicide were toxic with time (*45*). Similarly, bioassays with *Sinapis arvensis* (mustard) of soil to which was applied chlorbromuron [1-(3-chloro-4-bromophenyl)-3-methoxy-3-methylurea] gave higher values at approximately 30 days, the same values at approximately 60 days, and lower values at 85, 110, and 140 days, thereby showing that not all of the herbicide was available for toxicity (*46*). Aging also appears to occur in lake sediments, as witnessed by the decline in the rate of uptake of pyrene by the amphipod *Diporeia* sp. (*47*). Furthermore, we have recently found that phenanthrene aged for up to 150 days in samples of two sterile soils becomes progressively less available to earthworms subsequently added to these soils (J. W. Kelsey and M. Alexander, unpublished data).

A diminishing or reduced bioavailability with time is not always evident. The absence of an aging effect has been noted in investigations of the dermal availability to rats of TCDD (*48*), the toxicity of some herbicides to plants (*46*), and the biodegradation of a number of persistent pesticides by microorganisms (*14*). The reasons for the absence of an effect are as yet unknown, but they may be related to such properties of the soil as organic matter content or abundance of micropores or to such properties of the compound as hydrophobicity.

Perspective

The information that has been presented shows that (a) the availability of long-lived organic chemicals to microorganisms in soil in the field declines markedly with time, (b) some freshly added chemicals are readily available to microorganisms in soil in which the identical but aged compounds are not metabolized by indigenous microorganisms, (c) organic compounds incubated in sterile soil become increasingly less available to subsequently added microorganisms, (d) some compounds become increasingly resistant with time to extraction, and (e) sorption and desorption of hydrophobic compounds often require long time periods to reach equilibrium. These five lines of evidence are consistent with a sequestration of the molecules within particulate matter, presumably because the molecules diffuse into internal micropores or sites that are spatially remote. Because of their unavailability to even minute organisms and many extractants, it is likely that the compounds are also unavailable to humans, animals, and plants. These five lines of evidence are also consistent with a small but growing body of information showing that bioremediation of hydrocarbon-contaminated soils markedly reduces their toxicity to test species, despite the presence of presumably toxic concentrations of hydrocarbons as determined by exhaustive extraction (*49*).

Such a conclusion in regard to sequestration has major implications for assessments of the toxicity of compounds in soil that are known to be harmful in the absence of soil. The small base of information cited above shows that, at least for a few organic compounds, the toxicity to animals and plants disappears faster than the chemical itself. Thus, sequestration appears to alter the hazard to higher organisms. Indeed, the current views of the processes underlying slow sorption and desorption and those that give rise to ever increasing resistance to extraction implicitly suggest that toxic molecules will be less hazardous because they move to internal sites in soil particles that are too remote to impact living organisms.

The processes occurring may be envisioned in the following fashion. A toxic compound in solution is initially sorbed rapidly to the external surfaces of soil. This fraction is available for rapid desorption, to many organic solvents, to microorganisms for biodegradation, and to have some detrimental effect on susceptible organisms. That available fraction is slowly converted to an unavailable fraction, which is remote from the external surface. This portion of the compound is essentially irreversibly sorbed, is only extracted by highly vigorous means, and is neither available to microorganisms nor to cause injury to susceptible humans, animals, and plants. Not all of the compound will be sequestered and become nontoxic because soils into which 2,3,7,8-TCDD, polychlorinated biphenyls, or polybrominated biphenyls had been introduced years earlier still have considerable mammalian toxicity (*41, 42, 48, 50–52*). This toxicity is unlikely to have resulted from chemicals in solution because each of those compounds is extensively sorbed.

From the information presented, it is clear that regulatory decisions based on the currently used vigorous extraction procedures, which are designed to remove as much of the toxicant from soil as possible, may overestimate risk. This is not surprising, not only in view of the information cited above but also from an extensive literature that shows that the toxicity of the same concentration of an insecticide or herbicide may be vastly different in soils with dissimilar properties (*43, 44, 53, 54*).

Because of the sequestration of toxicants in soil, evaluations need to be made of the possibly reduced risk to humans, animal populations, plant populations, and

ecosystems. The very fact that the organic compounds in Superfund and most other hazardous waste sites have been in the contaminated soils for long periods of time emphasizes the need for assessing the significance of aging to toxicity. Tests of toxicity with pure chemicals in the absence of soil or chemicals freshly added to soil probably overestimate risk. Exhaustive extraction for determining toxicants in soil may be unrealiable for assessing the need for and effectiveness of remediation. An approach more meaningful than the ones used presently to establish priorities for remediation would include an assessment of the degree to which sequestering in soil alters the availability of environmental pollutants.

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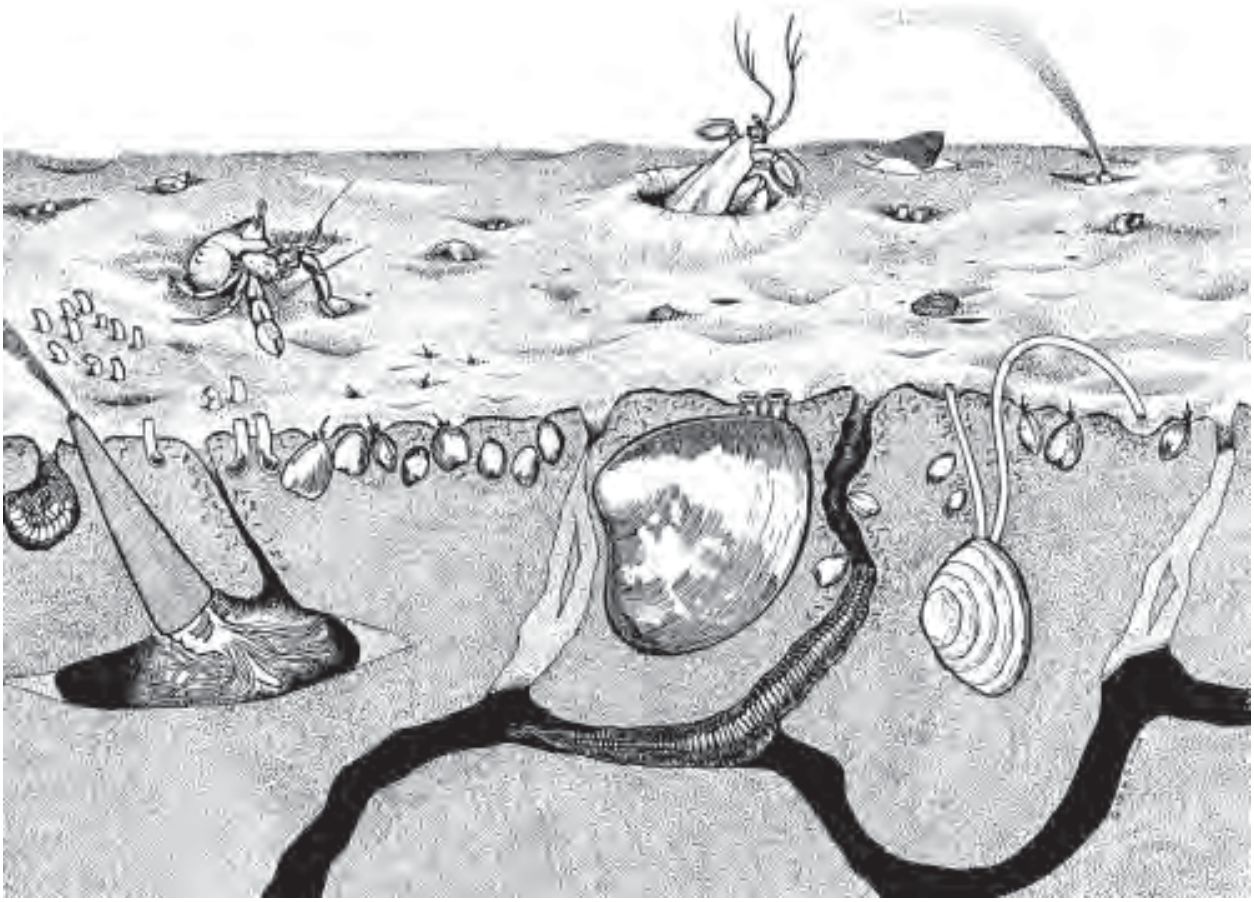
Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures



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Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures

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Notice

The Office of Research and Development (ORD) has produced this document to provide procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for mixtures of polycyclic aromatic hydrocarbons (PAHs). ESBs may be useful as a complement to existing sediment assessment tools. This document should be cited as:

U.S. EPA. 2003. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures. EPA-600-R-02-013. Office of Research and Development. Washington, DC 20460

The information in this document has been funded wholly by the U.S. Environmental Protection Agency. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document.

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Abstract

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations of PAH mixtures in sediment which are protective of the presence of benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration. This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any specific effect endpoint. In this document, the Final Chronic Value (FCV) for PAHs derived using the National Water Quality Criteria (WQC) Guidelines was used as the toxicity endpoint for this ESB. This value is intended to be the concentration of a chemical in water that is protective of the presence of aquatic life. For this PAH mixtures ESB, narcosis theory was used to (1) demonstrate that the slope of the acute toxicity-octanol water partition coefficient (K_{OW}) relationship was similar across species; (2) normalize the acute toxicity of all PAHs in water to an aquatic species using a reference K_{OW} of 1.0 (where the concentration in water and lipid of the organism would be essentially the same); (3) establish an acute sensitivity ranking for individual species at the K_{OW} of 1.0 and to use the rankings to calculate a Final Acute Value (FAV) following the WQC Guidelines; (4) calculate the final acute-chronic ratio (ACR) from water-only acute and chronic toxicity tests; (5) calculate the Final Chronic Value (FCV) at the reference K_{OW} of 1.0 from the quotient of the FAV and ACR; and (6) to calculate the PAH-specific FCV in µg/L using the FCV at the reference K_{OW} of 1.0, the PAH-specific K_{OW}, the slope of the K_{OW}-K_{OC} relationship and the universal narcotic slope of the K_{OW}-acute toxicity relationship. The EqP approach and the slope of the K_{OW}-K_{OC} relationship was then used to calculate, from the product of the PAH-specific FCV and $K_{OC,}$ the FCV concentration for each specific PAH in sediment ($C_{OC,PAHi,FCVi}$, µg/g organic carbon). Based on this approach, the recommended ESB for total PAH should be the sum of the quotients of a minimum of each of the suggested 34 individual PAHs in a specific sediment divided by the $C_{OC,PAHi,FCVi}$ of that particular PAH. This sum is termed the Equilibrium Partitioning Sediment Benchmark Toxic Unit ($\Sigma ESBTU_{FCV}$). Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected.

The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtures or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

Foreword

Under the Clean Water Act (CWA), the U.S. Environmental Protection Agency (EPA) and the States develop programs for protecting the chemical, physical, and biological integrity of the nation's waters. To support the scientific and technical foundations of the programs, EPA's Office of Research and Development has conducted efforts to develop and publish equilibrium partitioning sediment benchmarks (ESBs) for some of the 65 toxic pollutants or toxic pollutant categories. Toxic contaminants in bottom sediments of the nation's lakes, rivers, wetlands, and coastal waters create the potential for continued environmental degradation even where water column contaminant levels meet applicable water quality standards. In addition, contaminated sediments can lead to water quality impacts, even when direct discharges to the receiving water have ceased.

The ESBs and associated methodology presented in this document provide a means to estimate the concentrations of a substance that may be present in sediment while still protecting benthic organisms from the effects of that substance. These benchmarks are applicable to a variety of freshwater and marine sediments because they are based on the biologically available concentration of the substance in the sediments. These ESBs are intended to provide protection to benthic organisms from direct toxicity due to this substance. In some cases, the additive toxicity for specific classes of toxicants (e.g., metal mixtures or polycyclic aromatic hydrocarbon mixtures) is addressed. The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtues or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

ESBs may be useful as a complement to existing sediment assessment tools, to help assess the extent of sediment contamination, to help identify chemicals causing toxicity, and to serve as targets for pollutant loading control measures.

This document provides technical information to EPA Regions, States, the regulated community, and the public. It does not substitute for the CWA or EPA's regulations, nor is it a regulation itself. Thus, it cannot impose legally binding requirements on EPA, States, or the regulated community. EPA and State decisionmakers retain the discretion to adopt approaches on a case-by-case basis that differ from this technical information where appropriate. EPA may change this technical information in the future. This document has been reviewed by EPA's Office of Research and Development (Mid-Continent Ecology Division, Duluth, MN; Atlantic Ecology Division, Narragansett, RI), and approved for publication.

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Front cover image provided by Wayne R. Davis and Virginia Lee.

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Contents

Tables

Figures

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Executive Summary

This equilibrium partitioning sediment benchmark (ESB) document recommends an approach for summing the toxicological contributions of mixtures of 34 polynuclear aromatic hydrocarbons (PAHs) in sediments to determine if their concentrations in any specific sediment would be protective of benthic organisms from their direct toxicity. The combination of the equilibrium partitioning (EqP), narcosis theory, and additivity provide the technical foundation for this benchmark. These approaches were required because PAHs occur in sediments in a variety of proportions as mixtures and can be expected to act jointly under a common mode of action. Therefore, their combined toxicological contributions must be predicted on a sediment-specific basis. This overall approach provides for the derivation of this Tier 1 ESB that is causally linked to the specific mixtures of PAHs in a sediment, yet is applicable across sediments and appropriately protective of benthic organisms.

EqP theory holds that a nonionic chemical in sediment partitions between sediment organic carbon, interstitial (pore) water and benthic organisms. At equilibrium, if the concentration in any one phase is known, then the concentrations in the others can be predicted. The ratio of the concentration in water to the concentration in sediment organic carbon is termed the organic carbon partition coefficient (K_{OC}), which is a constant for each chemical. The ESB Technical Basis Document (U.S. EPA, 2003a) demonstrates that biological responses of benthic organisms to nonionic organic chemicals in sediments are different across sediments when the sediment concentrations are expressed on a dry weight basis, but similar when expressed on a μg chemical/g organic carbon basis ($\mu g/g_{OC}$). Similar responses were also observed across sediments when interstitial water concentrations were used to normalize biological availability. The Technical Basis Document (U.S. EPA, 2003a) further demonstrates that if the effect concentration in water is known, the effect concentration in sediments on a $\mu g/g_{OC}$ basis can be accurately predicted by multiplying the effect concentration in water by the chemical's K_{OC}.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any specific effect endpoint. In this document, the Final Chronic Value (FCV) for PAHs derived using the National Water Quality Criteria (WQC) Guidelines (Stephan et al., 1985) was used as the toxicity endpoint for this ESB. This value is intended to be the concentration of a chemical in water that is protective of the presence of aquatic life. For this PAH mixtures ESB, narcosis theory was used to (1) demonstrate that the slope of the acute toxicity-octanol water partition coefficient (K_{OW}) relationship was similar across species; (2) normalize the acute toxicity of all PAHs in water to an aquatic species using a reference K_{OW} of 1.0 (where the concentration in water and lipid of the organism would be essentially the same); (3) establish an acute sensitivity ranking for individual species at the K_{OW} of 1.0 and to use the rankings to calculate a Final Acute Value (FAV) following the WQC Guidelines (Stephan et al., 1985); (4) calculate the final acute-chronic ratio (ACR) from water-only acute and chronic toxicity tests; (5) calculate the Final Chronic Value (FCV) at the reference K_{OW} of 1.0 from the quotient of the FAV and ACR; and (6) to calculate the PAH-specific FCV in μg/L using the FCV at the reference K_{OW} of 1.0, the PAH-specific K_{OW} and the universal narcotic slope of the acute-K_{OW} toxicity relationship. The EqP approach and the slope of the K_{OW}-K_{OC} relationship was then used to calculate, from the product of the PAH-specific FCV and $K_{OC,}$ the FCV concentration for each specific PAH in sediment ($C_{OC,PAHi,FCVi}$, μg/g organic carbon).

Importantly, because PAHs occur in sediments as mixtures and their toxicities in water, tissues, or sediments are additive or nearly additive, their combined toxicities must be considered so that the benchmark is appropriately protective. For this reason, the combined toxicological contributions of the PAH mixture must be used. In this document, the 34 PAHs monitored in the EMAP program are used to derive a concentration of "total PAH." Many monitoring and assessment efforts measure a smaller group of PAHs, such as 13 or 23 PAHs. While adjustment factors have been calculated to relate these smaller subsets to the expected concentration of the 34 PAHs, their imprecision precludes their use in critical sediment assessments. Therefore, this document recommends that the ESB for total PAH should be the sum of the quotients of the concentrations of each of the 34 individual PAHs in a specific sediment divided by the $C_{OC,PAHi,FCVi}$ of that particular PAH. This sum is termed the Equilibrium Partitioning Sediment Benchmark Toxic Unit ($\Sigma ESBTU_{FCV}$), which is based on the FCV. Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected. This provides for the derivation of a benchmark that is causally linked to the specific mixtures of PAHs in a sediment, applicable across sediment types, and appropriately protective of benthic organisms. A sediment-specific site assessment would provide further information on PAH bioavailability and the expectation of toxicity relative to the $\Sigma ESBTU_{FCV}$ and associated uncertainty.

These ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAHs or the potential for bioaccumulation and trophic transfer of PAHs to aquatic life, wildlife or humans. Consistent with the recommendations of EPA's Science Advisory Board, publication of these documents does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, ESB exceedances could be used to trigger the collection of additional assessment data. ESBs apply only to sediments having $\geq 0.2\%$ organic carbon by dry weight.

Tier 1 and Tier 2 ESB values were developed to reflect differing degrees of data availability and uncertainty. Tier 1 ESBs have been derived for polycyclic aromatic hydrocarbon (PAH) mixtures in this document, and for the nonionic organic insecticides endrin and dieldrin, and metal mixtures in U.S. EPA (2003c,d,e). Tier 2 ESBs are reported in U.S. EPA (2003f).

Glossary of Abbreviations

ACR	Acute-Chronic Ratio
AR	Approximate Randomization
ASTM	American Society for Testing and Materials
BaP	Benzo[a]pyrene
BCF	Bioconcentration factor
C_{d}	Freely-dissolved interstitial water concentration of contaminant
C_{L}	Chemical concentration in target lipid
C_{*L}	Critical body burden in the target lipid fraction of the organism
C_{OC}	Chemical concentration in sediments on an organic carbon basis
$C_{OC,PAHi}$	PAH-specific chemical concentration in sediment on an organic carbon basis
$C_{octanol}$	Chemical concentration in octanol
C_{Org}	Chemical concentration in the organism
C_{*Org}	Critical body burden in the organism
C_{IW}	Total interstitial water concentration of contaminant
$C_{OC,PAHi,FCVi}$	Effect concentration of a PAH in sediment on an organic carbon basis calculated from the product of its FCV and K_{OC}
$C_{OC,PAHi,Rhepox,LC50}$	Sediment LC50 concentration on an organic carbon basis for a specific PAH for *Rhepoxinus* calculated from the product of its LC50 value at a K_{OW} of 1.0 and K_{OC}
$C_{OC,PAHi,Maxi}$	Maximum solubility limited PAH concentration in sediment on an organic carbon basis
CV	Coefficient of Variation
CWA	Clean Water Act
DOC	Dissolved Organic Carbon
EC50	Concentration affecting 50% of the test organisms
EMAP	Environmental Monitoring and Assessment Program
EPA	United States Environmental Protection Agency
EqP	Equilibrium partitioning
ESB	Equilibrium Partitioning Sediment Benchmark(s)

$ESBTU_{FCVi}$	Equilibrium Partitioning Sediment Benchmark Toxic Unit for PAH_i based on the FCV
$ESBTU_{Rhepox}$	Equilibrium Partitioning Sediment Benchmark Toxic Unit for PAH_i based on the LC50 of *Rhepoxynius abronius*.
$\Sigma ESBTU_{FCV}$	Sum of Equilibrium Partitioning Sediment Benchmark Toxic Units, where the units are based on FCV values
f_{Lipid}	Fraction of lipid in the organism
f_{OC}	Fraction of organic carbon in sediment
f_{SC}	Fraction of soot carbon in sediment
FACR	Final Acute-Chronic Ratio
FAV	Final Acute Value
FCV	Final Chronic Value
GMAV	Genus Mean Acute Value
IWTU	Interstitial Water Toxic Unit
$IWTU_{FCV}$	Interstitial water toxic unit calculated by dividing the dissolved interstitial water concentration by the FCV
K_{DOC}	Dissolved organic carbon: water partition coefficient
K_{LW}	Lipid: water partition coefficient
K_{OC}	Organic carbon: water partition coefficient
K_{OW}	Octanol: water partition coefficient
K_P	Sediment: water partition coefficient
K_S	Setschenow constant
K_{SC}	Soot carbon: water partition coefficient
LC50	Concentration estimated to be lethal to 50 % of the test organisms within a specified time period
LFER	Linear free energy relationship
MV	Molar Volume
NA	Not Applicable, Not Available
NAPL	Non-aqueous Phase Liquid
ND	Not Determined, Not Detected
NOAA	National Oceanographic and Atmospheric Administration

NOEC	No Observed Effect Concentration
NTU	Narcotic Toxic Units
OEC	Observable Effect Concentration
PAH	Polycyclic aromatic hydrocarbon
PAH_{OC}	Organic carbon-normalized PAH concentration in sediment
PCB	Polychlorinated Biphenyl
POC	Particulate Organic Carbon
PSTU	Predicted Sediment Toxic Units
QSAR	Quantitative Structure Activity Relationship
REMAP	Regional Environmental Monitoring and Assessment Program
S	Aqueous Solubility
SAB	U.S. EPA Science Advisory Board
SCV	Secondary Chronic Value
SE	Standard Error
SMAV	Species Mean Acute Value
SPARC	SPARC Performs Automated Reasoning in Chemistry
TOC	Total Organic Carbon
TU	Toxic Unit
WQC	Water Quality Criteria
$WQCTU_{FCVi}$	Water Quality Criteria Toxic Unit based on the FCV

Section 1

Introduction

1.1 General Information

Toxic pollutants in bottom sediments of the Nation's lakes, rivers, wetlands, estuaries, and marine coastal waters create the potential for continued environmental degradation even where water column concentrations comply with established WQC. In addition, contaminated sediments can be a significant pollutant source that may cause water quality degradation to persist, even when other pollutant sources are stopped (Larsson, 1985; Salomons et al., 1987; Burgess and Scott, 1992). The absence of defensible equilibrium partitioning sediment benchmarks (ESBs) make it difficult to accurately assess the extent of the ecological risks of contaminated sediments and to identify, prioritize, and implement appropriate cleanup activities and source controls (U.S. EPA 1997a, b, c).

As a result of the need for a procedure to assist regulatory agencies in making decisions concerning contaminated sediment problems, the U.S. Environmental Protection Agency (EPA) Office of Science and Technology, Health and Ecological Criteria Division (OST/HECD) and Office of Research and Development National Health and Environmental Effects Research Laboratory (ORD/NHEERL) established a research team to review alternative approaches (Chapman, 1987). All of the approaches reviewed had both strengths and weaknesses, and no single approach was found to be applicable for the derivation of benchmarks in all situations (U.S. EPA, 1989, 1992). The equilibrium partitioning (EqP) approach was selected for nonionic organic chemicals because it presented the greatest promise for generating defensible, national, numeric chemical-specific benchmarks applicable across a broad range of sediment types. The three principal observations that underlie the EqP approach to establishing sediment benchmarks are as follows:

1. The concentrations of nonionic organic chemicals in sediments, expressed on an organic carbon basis, and in interstitial waters correlate to observed biological effects on sediment-dwelling organisms across a range of sediments.

2. Partitioning models can relate sediment concentrations for nonionic organic chemicals on an organic carbon basis to freely-dissolved concentrations in interstitial water.

3. The distribution of sensitivities of benthic organisms to chemicals is similar to that of water column organisms; thus, the currently established water quality criteria (WQC) final chronic values (FCV) or secondary chronic values (SCV) can be used to define the acceptable effects concentration of a chemical freely-dissolved in interstitial water.

The EqP approach, therefore, assumes that (1) the partitioning of the chemical between sediment organic carbon and interstitial water is at or near equilibrium; (2) the concentration in either phase can be predicted using appropriate partition coefficients and the measured concentration in the other phase (assuming the freely-dissolved interstitial water concentration can be accurately measured); (3) organisms receive equivalent exposure from water-only exposures or from any equilibrated phase: either from interstitial water via respiration, from sediment via ingestion or other sediment-integument exchange, or from a mixture of exposure routes; (4) for nonionic chemicals, effect concentrations in sediments on an organic carbon basis can be predicted using the organic carbon partition coefficient (K_{OC}) and effects concentrations in water; (5) the FCV or SCV concentration is an appropriate effects concentration for freely-dissolved chemical in interstitial water; and (6) ESBs derived as the product of the K_{OC} and FCV are protective of benthic organisms. ESB concentrations presented in this document are expressed as µg chemical/g sediment organic carbon ($\mu g/g_{OC}$) and not on an interstitial water basis because (1) interstitial water

is difficult to sample and (2) significant amounts of the dissolved chemical may be associated with dissolved organic carbon; thus, total concentrations in interstitial water may overestimate exposure.

Sediment benchmarks generated using the EqP approach are suitable for use in providing technical information to regulatory agencies because they are:

1. Numeric values
2. Chemical specific
3. Applicable to most sediments
4. Predictive of biological effects
5. Protective of benthic organisms

ESBs are derived using the available scientific data to assess the likelihood of significant environmental effects to benthic organisms from chemicals in sediments in the same way that the WQC are derived using the available scientific data to assess the likelihood of significant environmental effects to organisms in the water column. As such, ESBs are intended to protect benthic organisms from the effects of chemicals associated with sediments and, therefore, only apply to sediments permanently inundated with water, to intertidal sediment, and to sediments inundated periodically for durations sufficient to permit development of benthic assemblages. ESBs should not be applied to occasionally inundated soils containing terrestrial organisms, nor should they be used to address the question of possible contamination of upper trophic level organisms or the synergistic, additive, or antagonistic effects of multiple chemicals. The application of ESBs under these conditions may result in values lower or higher than those presented in this document.

ESB values presented herein are the concentrations of PAH mixtures in sediment that will not adversely affect most benthic organisms. It is recognized that these ESB values may need to be adjusted to account for future data. They may also need to be adjusted because of site-specific considerations. For example, in spill situations, where chemical equilibrium between water and sediments has not yet been reached, sediment chemical concentrations less than an ESB may pose risks to benthic organisms. This is because for spills, disequilibrium concentrations in interstitial and overlying water may be proportionally higher relative to sediment concentrations. In systems where biogenic organic carbon dominates, research has shown that the source or "quality" of total organic carbon (TOC) in natural sediments does not affect chemical binding when sediment toxicity was measured as a function of TOC concentration (DeWitt et al., 1992). K_{OC}s have also been demonstrated to not vary in gradients of chemicals across estuarine sediments (Burgess et al., 2000a). However, in systems where other forms of carbon are present at elevated levels, the source or 'quality' of TOC may affect chemical binding despite expressing toxicity as a function of TOC concentration. At some sites, concentrations in excess of an ESB may not pose risks to benthic organisms because the compounds are partitioned to or a component of a particulate phase such as soot carbon or coal or exceed solubility such as in the case of undissolved oil or chemical (e.g. conditions at a manufactured gas plant site). In these situations, an ESB would be overly protective of benthic organisms and should not be used unless modified using the procedures outlined in "Procedures for the Derivation of Site-Specific Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms" (U.S. EPA, 2003b). If the organic carbon has a low capacity (e.g., hair, sawdust, hide), an ESB would be underprotective. An ESB may also be underprotective where the toxicity of other chemicals are additive with an ESB chemical or where species of unusual sensitivity occur at the site.

This document presents the theoretical basis and the supporting data relevant to the derivation of ESBs for PAH mixtures. The data that support the EqP approach for deriving ESBs for nonionic organic chemicals are reviewed by Di Toro et al. (1991) and EPA (U.S. EPA, 2003a). Before proceeding through the following text, tables, and calculations, the reader should also consider reviewing Stephan et al. (1985).

1.2 General Information: PAH Mixtures

The EPA developed ESBs for metal mixtures (Cd, Cu, Pb, Ni, Ag, Zn) (U.S. EPA 2003c) and the insecticides endrin and dieldrin (U.S. EPA 2003d,e) and proposed ESBs for the individual polycyclic aromatic hydrocarbons (PAHs) acenaphthene, fluoranthene and phenanthrene (U.S. EPA 1993a,b,c). Because PAHs occur in the environment as mixtures, rather than single chemicals, ESBs for individual PAHs have the potential to be substantially under-protective because they do not account for other co-occurring PAHs. This ESB for PAH mixtures replaces the earlier draft individual PAH documents.

Numerous efforts have previously sought to address and estimate the toxicity of PAH mixtures in sediments (Barrick et al., 1988; Long and Morgan, 1991; PTI Environmental Services, 1991; Long et al., 1995; Swartz et al., 1995; Ingersoll et al., 1996; MacDonald et al., 1996, 2000; Cubbage et al., 1997; Di Toro and McGrath, 2000; Di Toro et al., 2000; Ozretich et al., 1997, 2000). The resultant sediment benchmarks have engendered considerable controversy over such issues as the correlative versus causal relations between dry weight sediment chemistry and biological effects, the bioavailability of sediment contaminants, the effects of covarying chemicals and mixtures, and ecological relevance. Overviews of the various approaches are useful (Mount et al., 2003; Swartz et al., 1999). The use of sediment benchmarks derived in a variety of ways must be linked to the derivation procedure and specific intent of the methodology. The U. S. EPA research team has concluded, based upon additional investigation, that recommendation of sediment benchmarks for PAHs based on EqP, narcosis theory and additivity was necessary to resolve outstanding issues related to causality. Sediment benchmarks for mixtures of PAHs that are derived using these approaches are adequately protective of benthic organisms, as well as ecologically relevant.

The ΣPAH model developed by Swartz et al. (1995) and based upon a combination of the EqP approach, quantitative structure activity relationships (QSAR), narcosis theory, and additivity models provided initial insight into a technical approach for resolving these complexities. This EqP-based ΣPAH model provides a method to address causality, account for bioavailability, consider mixtures, and predict toxicity and ecological effects. The most significant contribution to the development of the scientific basis for deriving ESBs for PAH mixtures is described by Di Toro et al. (2000) and Di Toro and McGrath (2000). This pioneering research in developing a methodology for deriving ESBs for mixtures of narcotic chemicals and PAHs forms major portions of this document.

1.2.1 PAH Chemistry

Portions of the following overview of PAH chemistry are directly, or in part from, Neff's1979 classic book "Polycyclic Aromatic Hydrocarbons in the Aquatic Environment" and to a lesser extent Schwarzenbach et al. (1993). PAHs are composed of two or more fused aromatic or benzene rings. Two aromatic rings are fused when a pair of carbon atoms is shared. The resulting structure is a molecule with all carbon and hydrogen atoms lying in a single plane. Naphthalene ($C_{10}H_8$), which consists of two fused aromatic rings, is the lowest molecular weight PAH. The ultimate fused-ring aromatic system is graphite, an allotropic form of elemental carbon. Of primary environmental concern are mobile compounds ranging in molecular weight from naphthalene ($C_{10}H_8$, molecular weight 128.17) to coronene ($C_{24}H_{12}$, molecular weight 300.36). Within this range is an extremely large number of PAHs differing in the number and positions of aromatic rings and in the number, chemistry, and position of substituents on the ring system. Figure 1-1 presents a selection of PAH structures.

Physical and chemical characteristics of PAHs vary in a more or less regular fashion with molecular weight. Resistance to oxidation and reduction tends to decrease with increasing molecular weight. Vapor pressure and aqueous solubility decrease almost logarithmically with increasing molecular weight. As a consequence of these differences, PAHs of different molecular weights vary substantially in their behavior and

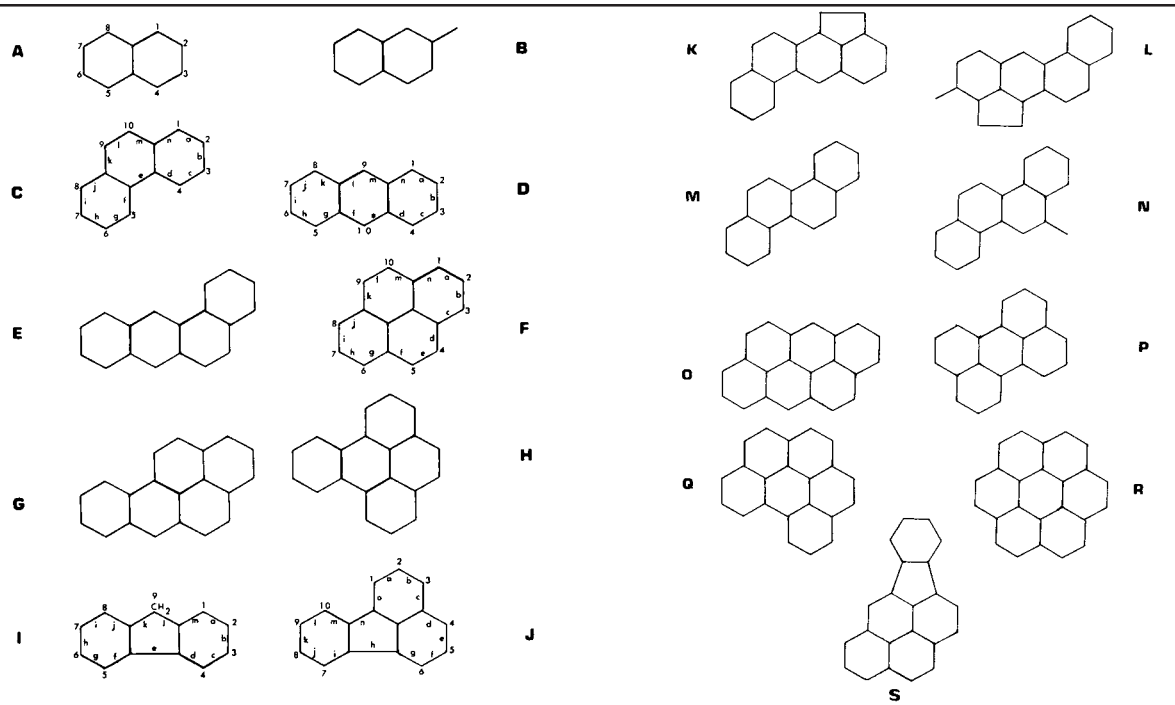

Figure 1-1. Ring structures of representative polycyclic aromatic hydrocarbons. The numbering and lettering system for several PAHs is also given. A, naphthalene; B, 2-methylnaphthalene; C, phenanthrene; D, anthracene; E, benz[*a*]anthracene; F, pyrene; G, benzo[*a*]pyrene; H, benzo[*e*]pyrene; I, fluorene; J, fluoranthene; K, benz[*j*]aceanthrylene = cholanthrene; L, 3-methylcholanthrene; M, chrysene; N, 5-methylchrysene; O, dibenzo[*cd,jk*]pyrene = anthranthrene; P, perylene; Q, benzo[*ghi*]perylene; R, coronene; S, indeno[1,2,3-*cd*]pyrene (from Neff 1979).

distribution in the environment and their toxic effects. PAHs undergo three types of chemical reactions characteristic of aromatic hydrocarbons - electrophilic substitution, oxidation, and reduction. Oxidation and reduction reactions destroy the aromatic character of the affected benzene ring but electrophilic substitution does not.

Several systems of nomenclature have been used to describe PAH ring structures. Nomenclature used in this document is that adopted by the International Union of Pure and applied Chemistry (IUPAC) and described in detail in *The Ring Index* (Patterson et al., 1960).

As noted above, there is an extremely large number of possible PAH structures (>10,000). Later in this document, 34 PAH structures (specific non-alkylated compounds and generic alkylated forms) are identified as representing a minimum for 'total PAHs'. It is recognized that this subset of all possible PAHs is not complete; however, the 34 PAHs identified are the ones that are generally most abundant and commonly measured as part of environmental monitoring programs. As analytical techniques improve, the number of PAHs composing 'total PAHs' will most certainly increase and users of this document are encouraged to include newly quantified PAHs in the derivation of benchmark values assuming good supporting data are available (e.g., K_{OW}s, solubilities).

PAHs found in aquatic environments originate from three possible sources: pyrogenic, petrogenic and diagenic. Pyrogenic PAHs result from the incomplete but high temperature, short-duration combustion of organic matter including fossil fuels and biomass (Neff 1979; Meyers and Ishiwatari 1993). These pyrogenic PAHs are believed to form from the breakdown or 'cracking' of organic matter to lower molecular weight radicals during pyrolysis, followed by rapid reassembly into non-alkylated PAH structures (Neff 1979). Petrogenic PAHs are created by diagenic processes at relatively low temperatures over geologic time scales, leading to the formation of petroleum and other fossil fuels containing PAHs (Meyers and

Ishiwatari 1993; Boehm et al., 2001). PAHs formed at relatively low temperatures (~150 °C) over long periods of time will be primarily alkylated molecules. The alkylated structure of petrogenic PAHs reflects the ancient plant material from which the compounds formed (Neff 1979). Diagenic PAHs refer to PAHs from biogenic precursors, like plant terpenes, leading to the formation of compounds such as retene and derivatives of phenanthrene and chrysene (Hites et al., 1980; Meyers and Ishiwatari 1993; Silliman et al., 1998). Perylene is another common diagenic PAH. Although its exact formation process remains unclear, an anaerobic process appears to be involved (Gschwend et al., 1983; Venkatesan 1988; Silliman et al., 1998). While diagenic PAHs are frequently found at background levels in recent sediments (i.e., deposited over the last 150 years), they often dominate the assemblage of PAHs present in older sediments deposited before human industrial activity (Gschwend et al., 1983). A potential fourth source of PAHs is biogenic; that is, purely from bacteria, fungi, plants or animals in sedimentary environments without any contributions from diagenic processes. However, attempts to produce biogenic PAHs have arguably failed, indicating this source is not significant (Hase and Hites 1976; Neff 1979).

The majority of PAHs found in aquatic environments originate from pyrogenic sources (Blumer 1976; Suess 1976; Hites et al., 1977; LaFlamme and Hites, 1978; NRC 1985; Wu et al., 2001). However, petrogenic PAHs do also occur alone or in combination with pyrogenic PAHs (Lake et al., 1979; Wakeham et al., 1980; NRC 1985; Gschwend and Hites 1981; Readman et al., 1992). In general, petrogenic PAHs appear to be associated with local or point sources, such as refineries and other petroleum industries, and adjacent to roads and navigational routes. This contrasts with the distribution of pyrogenic PAHs, which occur on a broader geographic scale. These distribution are also affected by the relative persistence of pyrogenic and petrogenic PAHs in the environment. As compared to petrogenic PAHs, pyrogenic PAHs are found more extensively in the sediment core record and appear to be less vulnerable to biotic and abiotic degradation (Burgess et al., 2003). Finally, diagenic PAHs occur at background levels although anthropogenic sources (e.g., perylene) can contribute to these types of PAHs.

1.2.2 PAH Mixtures

Unlike most other organic chemicals in the environment, PAHs are not released in a 'pure' or well-characterized form. Rather, because PAHs consist of thousands of structures originating from at least three sources, they always occur in the environment as complex mixtures (Burgess et al., 2003). As discussed above, pyrogenic PAHs, although not generally alkylated, are produced as mixtures of parent PAHs based on the conditions of their combustive formation (e.g., temperature, presence of oxygen, original organic matter). Similarly, the composition of petrogenic PAHs is a function of the diagenic conditions under which the original organic matter was exposed for thousands of years (e.g., pressure, temperature). Of course, human industrial practices convert some crude petrogenic PAH mixtures into more purified forms (e.g., fuel oils, creosote). These purified forms also contain complex mixtures of PAH molecules. As a consequence of these factors, when PAHs are released into the aquatic environment from the burning of fossil fuels and biomass, discharge of industrial chemicals, and transport of petroleum products they eventually accumulate in the sediments as complex mixtures (Neff 1979).

1.3 Application of Sediment Benchmarks

ESBs as presented in this document are meant to be used with direct toxicity testing of sediments as a method of sediment evaluation, assuming the toxicity testing species is sensitive to the chemical(s) of interest. They provide a chemical-by-chemical specification of sediment concentrations protective of benthic aquatic life. The EqP method should be applicable to nonionic organic chemicals with a K_{OW} above 3.0.

For the toxic chemicals addressed by the ESB documents Tier 1 (U.S. EPA, 2003c, d, e, and this document) and Tier 2 (U.S. EPA, 2003f) values

were developed to reflect the differing degrees of data availability and uncertainty. Tier 1 ESBs are more scientifically rigorous and data intensive than Tier 2 ESBs. The minimum requirements to derive a Tier 1 ESB include: (1) Each chemical's organic carbon-water partition coefficient (K_{OC}) is derived from the octanol-water partition coefficient (K_{OW}) obtained using the SPARC (SPARC Performs Automated Reasoning in Chemistry) model (Karickhoff et al., 1991) and the K_{OW}-K_{OC} relationship from Di Toro et al. (1991). This K_{OC} has been demonstrated to predict the toxic sediment concentration from the toxic water concentration with less uncertainty than K_{OC} values derived using other methods. (2) The FCV is updated using the most recent toxicological information and is based on the National WQC Guidelines (Stephan et al., 1985). (3) EqP-confirmation tests are conducted to demonstrate the accuracy of the EqP prediction that the K_{OC} multiplied by the effect concentration from a water-only toxicity test predicts the effect concentration from sediment tests (Swartz, 1991a; DeWitt et al., 1992). Using these specifications, Tier 1 ESBs have been derived for PAH mixtures in this document, metals mixtures (U.S. EPA, 2003c) and, the nonionic organic insecticides endrin and dieldrin (U.S. EPA, 2003d, e). In comparison, the minimum requirements for a Tier 2 ESB (U.S. EPA, 2003f) are less rigorous: (1) The K_{OW} for the chemical that is used to derive the K_{OC} can be from slow-stir, generator column, shake flask, SPARC or other sources (e.g., Site 2001). (2) FCVs can be from published or draft WQC documents, the Great Lakes Initiative or developed from AQUIRE. Secondary chronic values (SCV) from Suter and Mabrey (1994) or other effects concentrations from water-only toxicity tests can be used. (3) EqP cconfirmation tests are recommended, but are not required for the development of Tier 2 ESBs. Because of these lesser requirements, there is greater uncertainty in the EqP prediction of the sediment effect concentration from the water-only effect concentration, and in the level of protection afforded by Tier 2 ESBs. Examples of Tier 2 ESBs for nonionic organic chemicals are found in U.S. EPA (2003f).

1.4 Data Quality Assurance

All data used to derive the FCV used to calculate the ESB for PAHs from water-only toxicity tests were obtained from a comprehensive literature search completed in 1995. Discussions in other sections of this document utilized literature obtained up to 2003. Data were evaluated for acceptability using the procedures in the Stephan et al. (1985): *Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses*. Data not meeting the criteria for acceptability were rejected. All calculations were made using the procedures in Stephan et al. (1985). All data and intermediate values are presented in tables or appendices in the document. Four significant figures were used in intermediate calculations to limit the effect of rounding error, and are not intended to indicate the true level of precision. The document was reviewed as part of a formal peer review and all original data were made available as part of the review process. Any errors of omission or calculation discovered during the peer review process were corrected. The document was revised according to the comments of peer reviewers and additional scientific literature and significant data identified by reviewers were incorporated into the document. Hard copies of peer-review comments and responses to these comments are available from the ORD/NHEERL Atlantic Ecology Division - Narragansett, Rhode Island. Hard copies of all literature cited in this document reside at ORD/ NHEERL Atlantic Ecology Division - Narragansett, Rhode Island.

1.5 Overview

This document presents the theoretical basis and supporting data relevant to the derivation of ESBs for mixtures of PAHs.

Section 2 of this document "Narcosis Theory: Model Development and Application for PAH Mixtures" contains an analysis of the narcosis and EqP models to demonstrate the scientific basis for the derivation of WQC and ESBs for mixtures of narcotic chemicals, including PAHs. Data are

presented that demonstrate that the toxicity of narcotic chemicals when based on concentration in water increase with their K_{OW} and that the slope of the K_{OW}-toxicity relationship is not different across species. The universal slope of this relationship (-0.945) is applicable for all narcotic chemical classes, whereas the intercept is chemical class-specific. The intercept of this slope at a K_{OW} of 1.0 predicts the tissue effect concentration. The toxicities of mixtures of narcotic chemicals in water are shown to be approximately additive, thus the toxic unit concept is applicable to mixtures. The toxicities of narcotic chemicals are shown to be limited by their solubilities in water, hence their toxicities in sediments are limited.

Section 3 of this document "Toxicity of PAHs in Water Exposure and Derivation of PAH-specific FCVs" presents an analysis of acute and chronic water-only toxicity data for freshwater and saltwater aquatic organisms exposed to individual PAHs. It examines (1) the relative sensitivities of freshwater and saltwater organisms to determine if separate FCVs are required, and (2) the relative sensitivities of benthic organisms and organisms used to derive WQC to determine if the WQC FCV should be based only on benthic organisms. These data are used with the narcosis model presented in Section 2, the EqP approach (U.S. EPA, 2003a), and the U.S. EPA National WQC (Stephan et al., 1985) to derive the FCV for individual PAHs (PAH-specific FCV).

Section 4 "Derivation of PAH $\Sigma ESBTU_{FCV}$" contains the approach used for deriving the ΣESBs for mixtures of PAHs. The $C_{OC,PAHi,FCVi}$ is derived for each individual PAH as the product of the PAH-specific FCV and the respective K_{OC} value as recommended by the EqP approach. The use of the $C_{OC,PAHiFCVi}$ value for individual PAHs is inappropriate for use as the ESB because PAHs occur as mixtures. The toxicities of mixtures of narcotic chemicals has been shown to be approximately additive, therefore, combined toxic contributions of all PAHs in the mixture can be determined by summing the quotients of the concentration of each PAH in the sediment divided by its $C_{OC,PAHi,FCVi}$ to determine the sum of these Equilibrium Partitioning Sediment Benchmark Toxic Units ($\Sigma ESBTU_{FCV}$). If the $\Sigma ESBTU_{FCV}$ is ≤1.0, the sediment benchmark for the PAH mixture is not exceeded and the PAH concentration in the sediment is protective of benthic organisms. If the $\Sigma ESBTU_{FCV}$ exceeds 1.0, the sediment benchmark for the PAH mixture is exceeded and sensitive benthic organisms may be affected by the PAHs. The $\Sigma ESBTU_{FCV}$ is derived for PAH mixtures in sediments from national monitoring programs to reveal the incidence of sediment benchmark exceedences.

Section 5 "Actual and Predicted Toxicity of PAH Mixtures in Sediment Exposures" examines the applicability of the EqP methodology for $C_{OC,PAHi,FCVi}$ and ESB derivation. The $C_{OC,PAHi,FCVi}$ and ESB are compared to (1) databases of observed sediment toxicity, and (2) amphipod abundance in sediments from the field where PAHs are the probable contaminants of concern.

Section 6 "Implementation" defines the PAHs to which the ESB apply. An example calculation is provided to explain the conversion of concentrations of individual PAHs on a dry weight basis into the benchmark. The photo-activation of PAHs in UV sunlight and teratogenicity and carcinogenicity of certain PAHs in the mixture are examined. The importance of equilibrium and the partitioning of PAHs to other organic carbon phases (e.g., soot and coal) is described. An approach for calculating PAH solubilities for temperatures or salinities at specific sites is provided.

Section 7 "Sediment Benchmark Values: Application and Interpretation" presents the sediment benchmark values and lists several factors to consider when applying and interpreting these values.

Section 8 "References" lists references cited in all sections of this document.

Appendices provide supplementary tabulated information.

Section 2

Narcosis Theory: Model Development and Application for PAH Mixtures

2.1 Section Overview

This section of the ESB document presents a model of the toxicity of narcotic chemicals to aquatic organisms that is applicable to the derivation of WQC and ESBs for mixtures of narcotic chemicals, including PAHs. Both the model and this section of the document are largely excerpted from the publications of Di Toro et al. (2000) and Di Toro and McGrath (2000) which should be consulted for information on components of the overall model that are not included in this ESB document. The narcosis model includes a scientific analysis of the toxicities of narcotic chemicals fundamental to the derivation of WQC and ESBs for their mixtures. The ESB for PAH mixtures described in Section 4 of this document is derived using this model and toxicity data exclusively for PAHs (see Section 3).

The narcosis model is used to describe the toxicity of all type I narcotic chemicals. Since PAHs are expected to be type I narcotic chemicals (Hermens, 1989; Verhaar et al., 1992), the toxicological principles that apply to them should be more accurately characterized by an analysis of the principles that apply to narcotic chemicals overall. Model development utilizes a database of LC50 values comprising 156 chemicals and 33 aquatic species, including fish, amphibians, arthropods, molluscs, annelids, coelenterates and echinoderms. The analysis detailed in this section is used to demonstrate that (1) the toxicities of narcotic chemicals, and therefore PAHs, are dependant on the chemical's K_{OW}; (2) the slope of the K_{OW}-toxicity relationship is the same for all species of aquatic organisms and classes of narcotic chemicals with the intercepts being species and chemical class-specific; (3) the species-specific LC50 values normalized to a K_{OW} =1.0 permit ranking of species sensitivities and are equivalent to the body burden LC50 on a lipid basis; and (4) the toxicities of mixtures of narcotic chemicals are additive.

The analysis of narcotic chemical toxicity data presented in this section shows that the proposed model accounts for the variations in toxicity due to differing species sensitivities and chemical differences. The model is based on the idea that the target lipid is the site of action in the organism. Further, it is assumed that target lipid has the same lipid-octanol linear free energy relationship for all species. This implies that the $\log_{10}$LC50 vs $\log_{10}K_{OW}$ slope is the same for all species. However, individual species may have varying target lipid body burdens of narcotic chemicals that cause mortality. The target lipid LC50 body burdens estimated by extrapolations from the water-only acute toxicity data and K_{OW} values are compared to measured total lipid LC50 body burdens for five species. They are essentially equal, indicating that the extrapolation in the model is appropriate for estimation of LC50 body burdens, i.e., that the target lipid concentration is equal to the total extracted lipid concentration. The precise relationship between target lipid and octanol is established.

2.2 Narcosis Model Background

A comprehensive model of type I narcosis chemicals which considers multiple species has been presented by Van Leeuwen et al. (1992).

They developed QSARs for individual species and performed species sensitivity analysis. The analysis and model presented below and in Di Toro et al. (2000) and that of Van Leeuwen et al. (1992) are similar. The key differences in the Di Toro et al. (2000) model are the use of a single universal slope for the $\log_{10}$LC50 versus $\log_{10}K_{OW}$ QSAR for all the species, the inclusion of corrections for chemical classes, such as PAHs, that are slightly more potent than reference narcotics, and the interpretation of the y-intercepts as the species-specific critical body burdens for narcosis mortality.

2.3 Body Burden Model

The initial QSAR models for narcotic toxicity relied on correlations of $\log_{10}$LC50 and $\log_{10}K_{OW}$ (Konemann, 1981; Veith et al., 1983). An interesting and important interpretation of this inverse relationship which relates the toxicity to chemical body burden has been presented by McCarty et al. (1991), and proceeds as follows. The relationship between the LC50 (mmol/L) and K_{OW} for the narcosis LC50 for fish is approximately

$$\log_{10}\text{LC50} \simeq -\log_{10}K_{OW} + 1.7 \qquad (2\text{-}1)$$

For each LC50, a fish body burden, on a wet weight basis, corresponding to narcosis mortality can be computed using a bioconcentration factor BCF (L/kg) which is defined as the ratio of the chemical concentration in the organism C_{Org} (mmol/kg wet weight) to the chemical concentration dissolved in the water C_d (mmol/L)

$$\text{BCF} = \frac{C_{Org}}{C_d} \qquad (2\text{-}2)$$

Using the BCF, the organism concentration corresponding to the LC50, which is referred to as the critical body burden and denoted by C^*_{Org}, can be computed using

$$C^*_{Org} = \text{BCF} \times \text{LC50} \qquad (2\text{-}3)$$

The superscript * indicates that it is a critical body burden corresponding to the LC50. The BCF also varies with K_{OW}. For fish, the relationship is

$$\log_{10}\text{BCF} \simeq \log_{10}K_{OW} - 1.3 \qquad (2\text{-}4)$$

Therefore, the critical body burden corresponding to the LC50 for fish narcosis can be computed using the narcosis LC50 and the BCF

$$\log_{10} C^*_{Org} = \log_{10}\text{BCF} + \log_{10}\text{LC50}$$

$$\simeq \log_{10}K_{OW} - 1.3 - \log_{10}K_{OW} + 1.7$$

$$\simeq 0.4 \qquad (2\text{-}5)$$

or

$$C^*_{Org} \simeq 2.5\ \mu\text{mol/g wet wt} \qquad (2\text{-}6)$$

Thus, McCarty et al. (1991) rationalized the relationship between LC50 values and K_{OW} by suggesting that mortality is caused as a result of a constant body burden of the narcotic chemical.

The reason the critical body burden is a constant concentration for all the narcotic chemicals represented by the narcosis LC50 is a consequence of the unity slopes for $\log_{10}K_{OW}$ in Equations 2-1 and 2-4. For example, if the fraction of lipid in the fish is ass umed to be 5% ($f_{Lipid} = 0.05$), then the critical body burden in the lipid fraction of the fish is

$$C^*_L = \frac{C^*_{Org}}{f_{Lipid}} = \simeq 50\ \mu\text{mol / g lipid} \qquad (2\text{-}7)$$

which is the estimate of the chemical concentration in the lipid of these fish that causes 50 % mortality. The model presented below is an extension of this idea.

2.4 Target Lipid Model

The body burden model relates the narcosis concentration to a whole body concentration using a BCF. If different species are tested, then species-specific BCFs and lipid concentrations would be required to convert the LC50 concentration to a body burden for each species. A more direct approach is to relate narcotic

lethality to the concentration of the chemical in the target tissue of the organism, rather than to the concentration in the whole organism. If the partitioning into the target tissue is independent of species, then the need for species-specific BCFs is obviated. The identity of the target tissue is still being debated (Abernethy et al., 1988; Franks and Lieb, 1990), but we assume that the target is a lipid fraction of the organism. Hence the name, target lipid.

The target lipid model is based on the assumption that mortality occurs when the chemical concentration in the target lipid reaches a threshold concentration. This threshold is assumed to be species-specific rather than a universal constant that is applicable to all organisms (e.g., 50 µmol/g lipid, see Equation 2-7). The formulation follows the body burden model (McCarty et al., 1991). The target lipid-water partition coefficient K_{LW} (L/kg lipid) is defined as the ratio of chemical concentration in target lipid, C_L (µmol/g lipid = mmol/kg lipid), to the freely-dissolved aqueous concentration C_d, (mmol/L)

$$K_{LW} = \frac{C_L}{C_d} \qquad (2\text{-}8)$$

This equation can be used to compute the chemical concentration in the target lipid phase producing narcotic mortality, i.e., the critical body burden in the lipid fraction C_L^*, when the chemical concentration in the water phase is equal to the LC50

$$C_L^* = K_{LW} \times LC50 \qquad (2\text{-}9)$$

Assuming the narcosis hypothesis is true, i.e., that 50% mortality occurs if any narcotic chemical reaches the concentration C_L^*, then the LC50 for any chemical can be calculated using the same critical target lipid concentration C_L^* and the chemical-specific target lipid-water partition coefficient

$$LC50 = \frac{C_L^*}{K_{LW}} \qquad (2\text{-}10)$$

or

$$\log_{10}LC50 = \log_{10}C_L^* - \log_{10}K_{LW} \qquad (2\text{-}11)$$

The problem is determining the K_{LW} for narcotic chemicals. It is commonly observed for many classes of organic molecules that the logarithms of the partition coefficient between two liquids are related by a straight line (Leo, 1972). For target lipid and octanol, the relationship would be

$$\log_{10}K_{LW} = a_0 + a_1 \log_{10}K_{OW} \qquad (2\text{-}12)$$

Such a relationship is called a linear free energy relationship (LFER) (Leo et al., 1971; Brezonik, 1994). Combining Equations 2-11 and 2-12 yields the following linear relationship between $\log_{10}LC50$ and $\log_{10}K_{OW}$

$$\log_{10}LC50 = \log_{10}C_L^* - a_0 - a_1 \log_{10}K_{OW} \qquad (2\text{-}13)$$

where $\log_{10}C_L^* - a_0$ is the y intercept and $-a_1$ is the slope of the line.

This derivation produces the linear relationship between $\log_{10}LC50$ and $\log_{10}K_{OW}$ which is found experimentally (see, for example, Table 6 in Hermens et al., 1984)

$$\log_{10}LC50 = m \log_{10}K_{OW} + b \qquad (2\text{-}14)$$

where m and b are the slope and intercept of the regression, respectively. In addition, it identifies the meanings of the parameters of the regression line. The slope of the line m is the negative of the slope of the LFER between target lipid and octanol, a_1. The intercept of the regression $b = \log_{10}C_L^* - a_0$ is composed of two parameters: C_L^* is the target lipid concentration at narcosis mortality, and a_0 is the constant in Equation 2-12.

The difference between the target lipid model and the McCarty et al. (1991) body burden model is that for the latter, the coefficients a_0 and a_1 for fish are assumed to be known: $a_0 = -1.3$ and $a_1 = 1.0$. It is interesting to examine the consequences of a similar assumption applied to the target lipid model. If it is assumed that the partitioning of narcotic chemicals in lipid and octanol are equal, i.e., that lipid is octanol, a common first approximation, then $a_1 = 1$ and $a_0 = 0$ and the y-intercept becomes

$$b = \log_{10}C_L^* \qquad (2\text{-}15)$$

which is the target-lipid concentration producing 50% narcosis mortality.

This result can be understood by examining Figure 2-l. The y-intercept b is the LC50 value for a chemical with a $\log_{10}K_{OW} = 0$ or $K_{OW} = 1$. The K_{OW} is the ratio of the chemical's concentration in octanol to its concentration in water. Hence, for this hypothetical chemical (an example would be 2-chloroethanol for which $\log_{10}K_{OW} = -0.048$l 0 the chemical's concentration in water is equal to its concentration in octanol. However, if the K_{LW} equals the K_{OW}, i.e., lipid is octanol, then its concentration in water must be equal to its concentration in the target lipid of the organism. Therefore, the y-intercept is the target lipid phase concentration at which 50% mortality is observed. That is

$$LC50|_{K_{OW}=1} = b = C^*_{octanol} = C^*_L \qquad (2\text{-}16)$$

Note that this interpretation is true only if $a_0 = 0$ (see Equation 2-13).

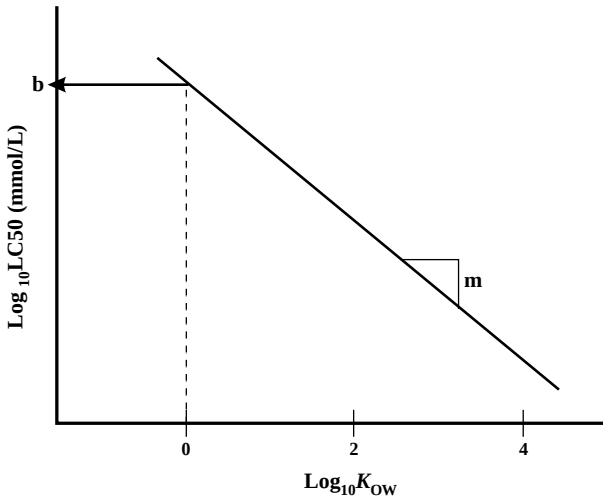

Figure 2-1. Schematic diagram of the $\log_{10}$LC50 versus $\log_{10}K_{OW}$ relationship. At $\log_{10}K_{OW} = 0$ ($K_{OW} = 1$), the concentration in water equals the concentration in octanol.

Thus, the target lipid narcosis model differentiates between the chemical and biological parameters of the $\log_{10}$LC50 - $\log_{10}K_{OW}$ regression coefficients in the following way

$$\begin{aligned} \text{slope} \quad &= \quad \text{chemical} \\ m \quad &= \quad -a_1 \\ \text{intercept} \quad &= \quad \text{chemical} \quad \text{biological} \\ b \quad &= \quad -a_0 \quad + \quad \log_{10}C^*_L \end{aligned} \qquad (2\text{-}17)$$

The chemical parameters a_0 and a_1 are associated with the LFER between octanol and target lipid (Equation 2-12). The biological parameter is the critical target lipid concentration C^*_L. This result is important because it suggests that the slope $m = -a_1$ of the $\log_{10}$LC50- $\log_{10}K_{OW}$ relationship should be the same regardless of the species tested since it is a chemical property of the target lipid - the slope of the LFER. Of course this assumes that the target lipid of all species have the same LFER relative to octanol. This seems to be a reasonable expectation since the mechanism of narcosis is presumed to involve the phospholipids in the cell membrane and it appears to be a ubiquitous mode of action. However, the biological component of the intercept C^*_L (Equations 2-13 and 2-17) should vary with species sensitivity to narcosis since it is commonly found that different species have varying sensitivity to the effects of exposure to the same chemical. The expectations that follow from the target lipid model - that the slope should be constant among species and that the intercepts should vary among species - is the basis for the data analysis presented below.

2.5 Acute Lethality Database Compilation

An acute lethality (LC50) database for type I narcotics from water-only toxicity tests was compiled from available literature sources. The principal criterion for acceptance was that a number of chemicals were tested using the same species so that the slope and intercept of the $\log_{10}$LC50 - $\log_{10}K_{OW}$ relationship could be estimated. The data were restricted to acute exposures and a mortality end point to limit the sources of variability. A total of 33 aquatic species including amphibians, fishes, arthropods (insects

and crustaceans), molluscs, annelids, coelenterates and protozoans were represented. Seventy-four individual datasets were selected for inclusion in the database which provided a total of 796 individual data points. Details are provided in Appendix A. The individual chemicals which comprise the database are listed in Appendix B. There are 156 different chemicals including halogenated and non-halogenated aliphatic and aromatic hydrocarbons, PAHs, alcohols, ethers, furans, and ketones.

The $\log_{10}K_{OW}$ values and aqueous solubilities of these chemicals were determined using SPARC (SPARC Performs Automated Reasoning in Chemistry) (Karickhoff et al., 1991), which utilizes the chemical's structure to estimate various properties. The reliability of SPARC was tested using $\log_{10}K_{OW}$ values measured using the slow stir flask technique (de Bruijn et al., 1989). Fifty three compounds such as phenols, anilines, chlorinated monobenzenes, PAHs, PCBs and pesticides were employed. A comparison of the $\log_{10}K_{OW}$ values measured using the slow stir flask technique to the SPARC estimates demonstrates that SPARC can be used to reliably estimate measured $\log_{10}K_{OW}$ values over nearly a seven order of magnitude range of $\log_{10}K_{OW}$ (Figure 2-2A). Note that this comparison tests both SPARC and the slow stir measurements, since SPARC is not parameterized using octanol-water partition coefficients (Hilal et al., 1994).

2.5.1 Aqueous Solubility

The toxicity data were screened by comparing the LC50 value to the aqueous solubility, S, of the chemical (Figure 2-2B). (Note: For this and other figures in this document where a large number of data points are available, the plotting procedure limits the actual number of data plotted.) Individual LC50 values were eliminated from the database if the LC50 > S, which indicated the presence of a separate chemical phase in the experiment. For these cases, mortality must have occurred for reasons other than narcosis - for example, the effect of the pure liquid on respiratory surfaces - since the target lipid concentration cannot increase above that achieved at the water solubility concentration. A total of 55 data points were eliminated, decreasing

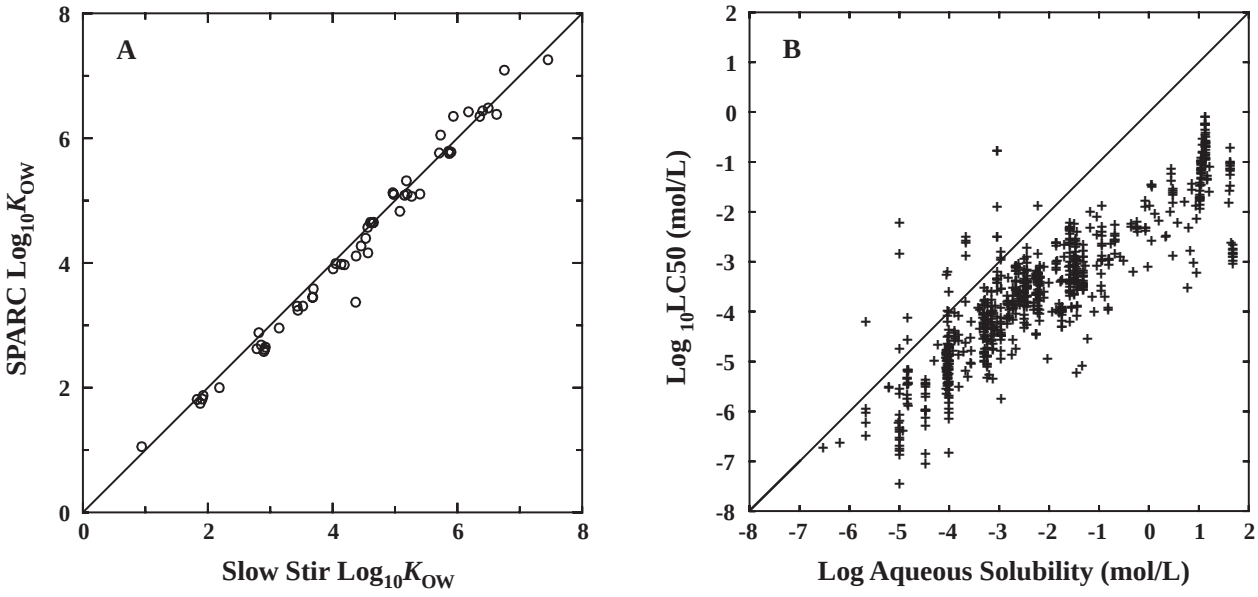

Figure 2-2. Comparisons of (A) $\log_{10}K_{OW}$ predicted by SPARC versus measured $\log_{10}K_{OW}$ using slow stir method and (B) reported $\log_{10}$LC50 values versus the aqueous solubility estimated by SPARC. The diagonal line represents equality.

the number to 736 and the number of individual chemicals to 145 (Appendix B).

2.5.2 Exposure Duration

The duration of exposure varied in the dataset from 24 to 96 hours (Appendix A). Before the data could be combined for ana ysis, the individual datasets should be adjusted to account for this difference. The required equilibration time may vary with both organism and chemical. An increase in either organism body size or chemical hyodrphobicity may increase the time to reach equilibrium.

To determine if acute lethality for narcotic chemicals varied with exposure time, cata were selected where toxicity was reported at multiple exposure times for the same organism and the same chemical. For seven fish species, data were available for 96 hours and either 24, 48 or both 24 and 48 hours of exposure. Arithmetic ratios of the LC50 values for 48 to 96 hours and for the 24 and 96 hours exposure are compared to log 10 K_{OW}. The 48 to 96 hour ratio is 1.0 for essentially all the data (Figure 2-3A). The 24 to 96 hour ratio is larger, approaching 1.4 for the higher Kow chemicals (Figure 2-3B). A linear regression is used to fit the relationship in Figure 2-3B.

$$LC50_{(24)}/LC50_{96} = 0.0988 \log_{10} K_{OW} + 0.9807 \qquad (2\text{-}18)$$

where $LC50_{24}$ and $LC50_{96}$ are the LC50 values for 24 and 96 hour exposures. Since approximately 46% of the data points in the overall database represent narcosis mortality after exposure of fish to a chemical for 24 hours, these data were converted to 96 hour LC50 values using Equation 2-18 for chemicals having $\log_{10}K_{OW}$ values of >1. No correction factor is applied to 24 hour toxicity data for invertebrates and fishes exposed to chemicals having $\log_{10}K_{OW}$ values of <1 (Di Toro et al., 2000).

2.6 Data Analysis

The analysis of the toxicity data is based on the target lipid model assumption that the slope of the $\log_{10}K_{OW}$ is the same for all species. This assumption was tested using a linear regression model to estimate the species-specific body burdens and the universal narcosis slope.

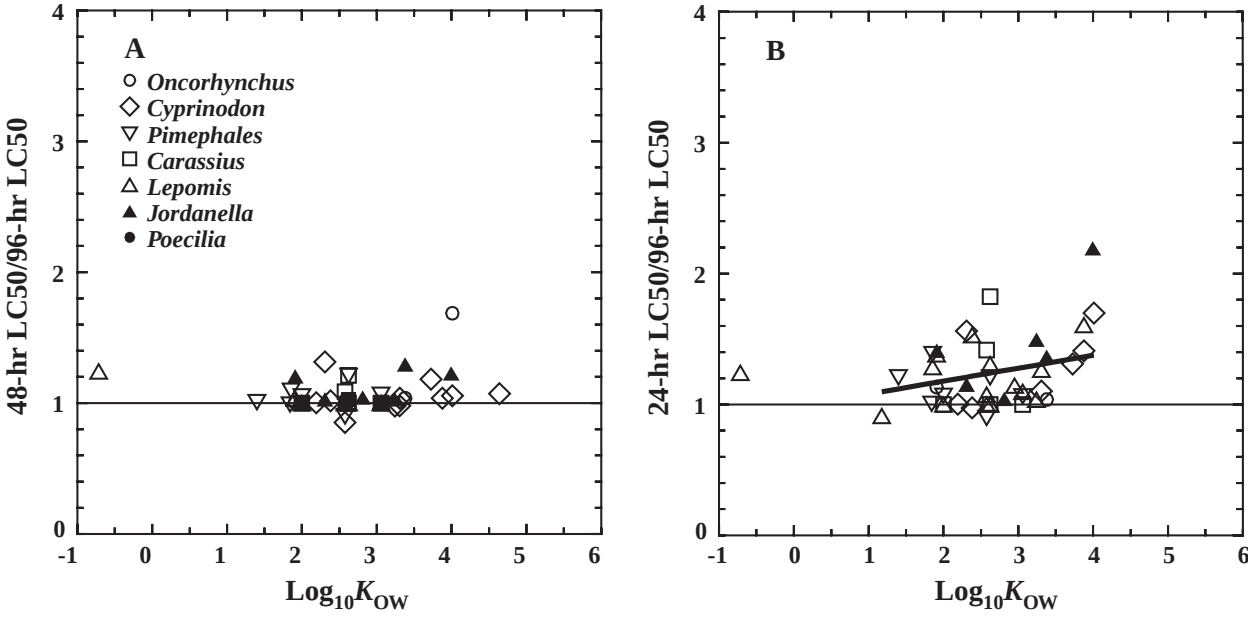

Figure 2-3. Ratios of (A) 48- to 96-hour LC50 values and (B) 24- to 96-hour LC50 values versus $\log_{10}K_{OW}$. The line in (B) is the regression used to correct the 24-hour LC50 to 96-hour LC50.

2.6.1 Regression Model

Consider a species k and a chemical j. The $LC50_{k,j}$ for that species-chemical pair is

$$\log_{10}LC50_{k,j} = \log_{10}C_L^*(k) - a_0 - a_1 \log_{10}K_{OW}(j) \quad (2\text{-}19)$$

$$= b_k - a_1 \log_{10}K_{OW}(j) \quad (2\text{-}20)$$

where

$$b_k = \log_{10}C_L^*(k) - a_0 \quad (2\text{-}21)$$

is the y-intercept. The problem to be solved is: how to include all the b_k, k = 1,...,N_S corresponding to the N_s = 33 species and a single slope a_1 in one multiple linear regression model equation.

The solution is to use a set of indicator variables $*_{ki}$ that are either zero or one depending on the species associated with the observation being considered. The definition is

$$\delta_{ki} = 1 \qquad k = i$$

$$\delta_{ki} = 0 \qquad k \neq i \quad (2\text{-}22)$$

which is the Kronecker delta (Kreyszig, 1972). The regression equation can be formulated using δ_{ki} as follows

$$\log_{10}LC50_{i,j} = a_1 \log_{10}K_{OW}(j) + \sum_{k=1}^{N_S} b_k \delta_{ki} \quad (2\text{-}23)$$

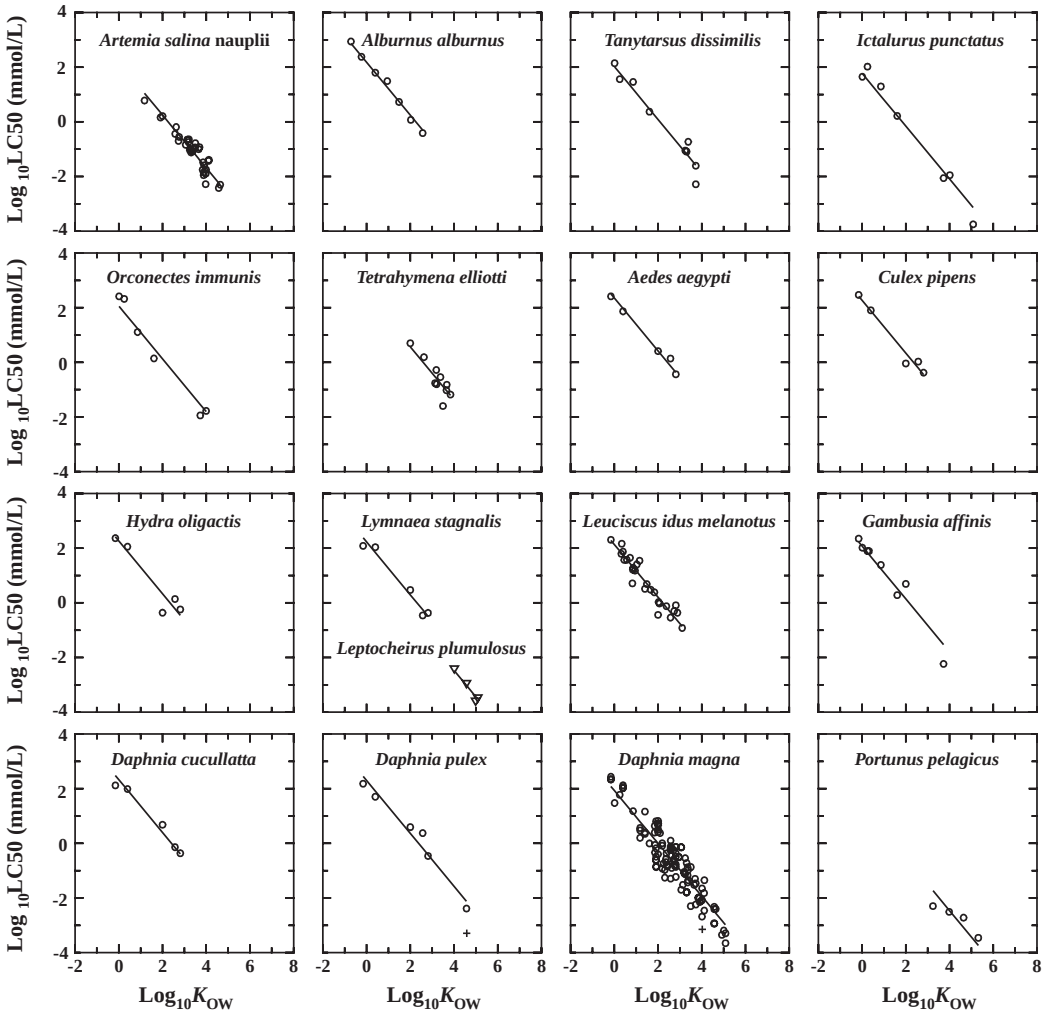

Figure 2-4. $Log_{10}LC50$ versus $log_{10}K_{OW}$ for the indicated species. The line has a constant slope of −0.945. The y-intercepts vary for each species. Outliers are denoted by a plus symbol (+).

Equation 2-23 is now a linear equation with N_s+1 independent variables: $\log_{10}K_{OW}(j)$ and δ_{ki}, k = 1,...,N_s. There are N_s+1 coefficients to be fit: a_1 and b_k, k = 1,...,N_S. For each $LC50_{ij}$ corresponding to species i and chemical j, one of the b_k corresponding to the appropriate species k = i has a unity coefficient $\delta_{ii} = 1$ while the others are zero. The way to visualize this situation is to realize that each row of data consists of the LC50 and these N_S+1 independent variables, for example for j = 1 and i = 3

$\log_{10}(LC50_{ij})$	$\log_{10}(K_{OW}(j))$	δ_{1i}	δ_{2i}	δ_{3i}	...	δ_{Nsi}
0.788	1.175	0	0	1	0	0

(2-24)

which is actually the first of the 736 records in the database. The result is that b_3 is entered into the regression equation as the intercept term associated with species i = 3 because that δ_{ki} is one for that record. By contrast, the slope term $a_1\log_{10}K_{OW}(j)$ is always included in the regression because there is always an entry in the $\log_{10}K_{OW}(j)$ column (Equation 2-24). Hence, the multiple linear regression estimates the common slope a_1 and the species-specific intercepts b_k, k = 1,...,N_s.

A graphical comparison of the results of fitting Equation 2-23 to the full dataset are shown in Figure 2-4 for each of the 33 species. The regression coefficients are tabulated and discussed subsequently after a further refinement is made to the model. The lines appear to be representative of the data as a whole. There appear to be no significant deviations from the common slope. A few outliers, which are plotted as +, were not included in the regression analysis. An outlier is identified if the difference between predicted and observed LC50 values are greater than one log unit when they are included in the regression. This decreases the total number of data points from 736 to 722.

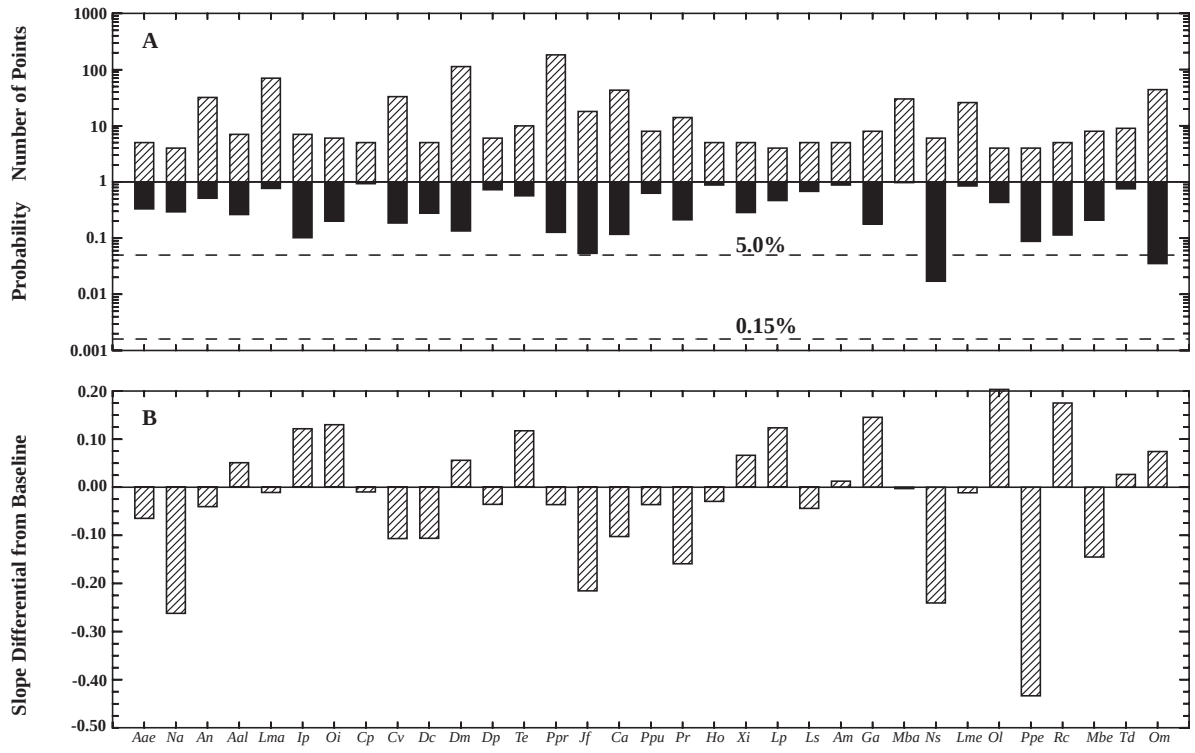

Figure 2-5 Statistical comparison of slopes fitted to individual species to the universal slope of −0.945 showing (A) the probability that the difference occurred by chance (filled bars) and number of data points in the comparison (hatched bars) for each species in the database, and (B) the deviations of the individual estimates from the universal slope.

2.6.2 Testing Model Assumptions

The adequacy of the regression model is tested by answering three questions:

1. Are the data consistent with the assumption that the slope is the same for each species tested?
2. Does the volume fraction hypothesis (Abernethy et al., 1988) provide a better fit?
3. Are there systematic variations for particular chemical classes?

The first assumption, that the slope estimated for a particular species is statistically indistinguishable from the universal slope a_1 = -0.97 without chemical class correction (see Section 2.5.4), can be tested using conventional statistical tests for linear regression analysis (Wilkinson, 1990). The method is to fit the data for each species individually to determine a species-specific slope. Then, that slope is tested against the universal slope a_1 = -0.97 without chemical class correction to determine the probability that this difference could have occurred by chance alone. The probability and the number of data points for each species are shown in Figure 2-5A. The slope deviations are shown in Figure 2-5B. Some of the slope deviations are quite large. However, only three species equal or exceed the conventional significance level of 5% for rejecting the equal slope hypothesis.

Testing at the 5% level of significance is misleading, however, because there is a one in twenty chance of rejecting one species falsely when 33 species are being tested simultaneously. The reason is that the expected number of rejections for a 5% level of significance would be 33 x 0.05 = 1.65, i.e., more than one species on average would be rejected due to statistical fluctuations even though all the slopes are actually equal. In fact, only 20 tests at 5% would, on average, yield one slope that would be incorrectly judged as different. The correct level of significance is (1/33)(1/20) = 0.152% so that the expected number of rejections is 33 x 0.00152 = 0.05 or 5% (Wilkinson, 1990). This level of significance is displayed together with the slope data presented in Figure 2-5A. As can be seen, there is no statistical evidence for rejecting the claim of equal slopes for the tested species. As would be expected, when 5% was used as the level of significance two species were identified as having unique slopes. When the current level of significance (0.00152) was used for the 33 samples none were significantly different.

2.6.3 Volume Fraction Hypothesis

The volume fraction hypothesis asserts that narcotic mortality occurs at a constant volume fraction of chemical at the target site of the organism (Abernethy et al., 1988). Basically, this involves expressing the LC50 as a volume fraction of chemical rather than a molar concentration. This is done using the molar volume of the chemicals (see column MV in Appendix B). The LC50 on a molar volume basis is

$$LC50(cm^3/L) = LC50\,(mmol/L) \times MV\,(cm^3/mmol) \quad (2\text{-}25)$$

The question is: does using molar volume as the concentration unit improve the regression analysis? The results are shown below

	Molar concentrations (mmol/L)	Molar volumes (cm^3/L)
Slope	-0.97 ±0.012	-0.90 ± 0.012
R^2	0.94	0.96

The coefficient of determination (R^2 value) for the volume fraction analysis (0.96) is slightly greater than that for the molar concentration (0.94). Because they are essentially the same, this document uses the molar concentration rather than those based on the volume fraction. Importantly, the slope for both volume and weight units of concentration is not unity.

2.6.4 Chemical Classes

The analysis presented above assumes that all of the 145 chemicals listed in Appendix B are narcotic chemicals. That is, the only distinguishing chemical property that affects their toxicity is K_{OW}.

A criteria has been suggested that can be used to determine whether a chemical is a narcotic (Bradbury et al., 1989), namely that it demonstrates additive toxicity with a reference narcotic. However, it is not practical to test each possible chemical. The more practical test is whether the toxicity can be predicted solely from the $\log_{10}LC50$ - $\log_{10}K_{OW}$ regression. In fact, this is used in methods that attempt to discriminate reference narcotics from other classes of organic chemicals (Verhaar et al., 1992).

Using this approach, differences in toxicity among chemical classes would be difficult to detect if differing species were aggregated or different slopes were allowed in the regression analysis. However, with the large dataset employed above, these differences can be seen by analyzing the residuals grouped by chemical class.

The criteria for choosing the relevant classes are not obvious without a detailed understanding of the mechanism of narcotic toxicity. Hence, the conventional organic chemical classes based on structural similarities, e.g. ethers, alcohols, ketones, etc., are used. The results are shown in Figure 2-6A. The means ±2 standard error (SE) of the means are shown for each class. Although not a rigorous test, the ±2 SE range does not encompass zero for certain classes. Thus, it is likely that there are statistically significant chemical class effects.

2.6.4.1 Statistical Analysis of K_{OW}-Toxicity Relationships

A rigorous test is conducted by including correction constants for each of the chemical classes in a manner that is analogous to Equation 2-23. The model equation is formulated using N_C - 1 corrections, Δc_ℓ, corresponding to the $\ell = 1,...,N_C$ - 1 chemical classes. These are interpreted as corrections relative to the reference class which is chosen to be aliphatic non-halogenated hydrocarbons. The regression equation is formulated as before with a variable $\xi_{\ell j}$ that is one if chemical j is in chemical class ℓ and zero otherwise

$\xi_{\ell j} = 1$ if chemical j is in class ℓ

$\xi_{\ell j} = 0$ otherwise (2-26)

The regression equation that results is

$$\log_{10}LC50_{i,j} = a_1\log_{10}K_{OW}(j) + \sum_{k=1}^{N_S} b_k\delta_{k_i} + \sum_{\ell=1}^{N_C-1}\Delta c_\ell\xi_{\ell j} \quad (2\text{-}27)$$

Each data record now contains t he dependent variable $\log_{10}LC50_{i,j}$, the independent variables $\log_{10}K_{OW}(j)$, and the δ_{ki}, $k = 1,...,N_s$ and $\xi_{\ell j}$, $\ell = 1,...,N_C$ - 1 indicator variables which are 0 or 1 depending on which species and which chemical class is represented by the $LC50_{i,j}$.

Only N_C - 1 chemical class corrections are required because including N_C class corrections under-determines the equation set with one too many unknowns. The reason is that every equation would have one b_i and one Δc_ℓ for species i and chemical j in chemical class ℓ. Since this condition would occur in every equation there is no unique solution for the b_k and the Δc_ℓ values. One of these constants could be adjusted by an arbitrary amount and the rest could then be adjusted to compensate while still achieving the same fit of the data. Thus, a reference chemical class is chosen: non-halogenated aliphatic hydrocarbons for which $\Delta c_\ell = 0$. The remaining regression constants Δc_ℓ, $\ell = 1,...,N_c$ - 1 are then the differential toxicity of chemical class ℓ relative to the reference chemical class. This is the reason for the Δc notation.

The requirement for a chemical class correction is decided using a statistical test that compares the Δc_ℓ values that result from the regression to the hypothesis $\Delta c_\ell = 0$. For the classes which are not statistically different, they are included in the reference class and the parameters are re-estimated. This is continued until all the remaining Δc_ℓ values are statistically different from zero. After a number of trials, it was found that treating halogen substitutions as a separate additive correction gave the least number of statistically significant class corrections. Thus, chemical class corrections are applied to the base structure, if necessary, and an additional correction is made if any substitute is a halogen. Therefore,

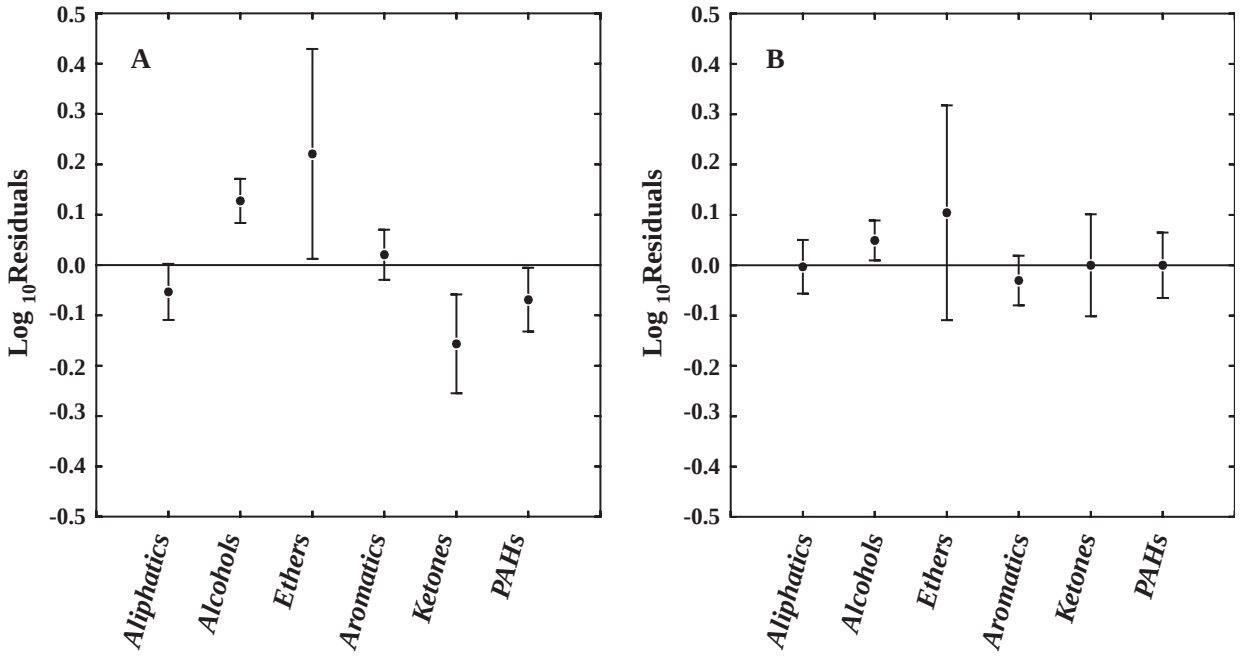

Figure 2-6. Chemical class comparisons of residuals from the regression grouped by class with (A) mean ± 2 standard errors and (B) chemical class corrections included in the regression.

for halogenated chemicals it is possible that two $\xi_{ij} = 1$ in Equation 2-27. The chemical classes are listed in Appendix B.

The results of the final regression analysis are listed in Table 2-1. Both the logarithmic b_i and arithmetic 10^{b_i} values of the intercepts are included together with their standard errors. Chemical classes which demonstrate higher potency than the reference class are ketones and PAHs. Halogenation increases the potency as well. After accounting for different potencies in the chemical classes, the mean residuals are statistically indistinguishable from zero (Figure 2-6B).

2.6.4.2 Standard Errors and Residuals

The standard errors of the body burdens $SE(b_i)$ found from the regression (Equation 2-27) are in an almost one-to-one correspondence with the number of data points for that species. Thus, the b_i for *Pimephales promelas* (fathead minnow) with 182 data points has a 10% coefficient of variation, $CV(b_i) = SE(b_i)/b_i$, while the b_i for *Neanthes arenaceodentata* (polychaete worm) with 4 data points has a 50% coefficient of variation (Table 2-1). The relationship of the sample size (N) to the coefficient of variation of the estimated critical body burden, $CV(b_i)$, is shown in Figure 2-7A.

The residuals are log normally distributed (Figure 2-7B) and exhibit no trend with respect to K_{OW} (Figure 2-7C) which confirms the assumption underlying the use of regression analysis. The reason they are restricted to ±1 order of magnitude is that 14 data points outside that range were originally excluded as outliers (for some values previously less than ± one order of magnitude, chemical class corrections produced values slightly greater than one order of magnitude as shown in Figure 2-7C).

2.6.4.3 Chemical Class Corrections

The corrections due to chemical classes reduce the critical body burden by a factor of approximately one-half for ketones and PAHs. Correction for halogenation reduces it further by

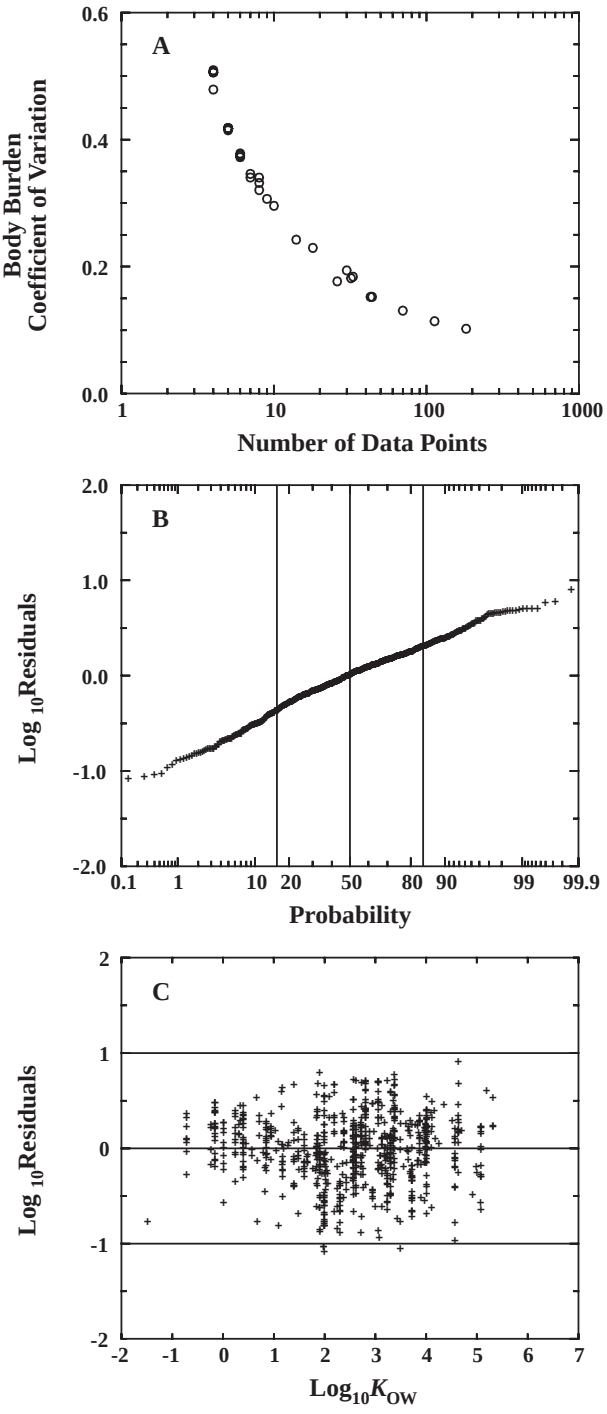

Figure 2-7. The coefficient of variation of the estimated species-specific body burdens versus (A) the number of data points for that species (B), the log probability plot of the residuals, and (C) the residuals versus $\log_{10}K_{OW}$.

0.570 (Table 2-1). Thus, a chlorinated PAH would exhibit a critical body burden of approximately one-third of a reference narcotic. The coefficients of variation for these corrections are approximately 10%.

The chemical class differences among the type I narcotics affect the LC50-K_{OW} relationship. The model no longer predicts a single straight line for the $\log_{10}$LC50-$\log_{10}K_{OW}$ relationship for all narcotic chemicals. What is happening is that the y-intercepts are changing due to the changing $)c_R$ values. The model (Equation 2-27) when applied to a single species k is

$$\log_{10}LC50_{k,j} = a_1 \log_{10}K_{OW}(j) + b_k + \sum_{\ell=1}^{N_C-1} \Delta c_\ell \xi_{\ell j} \qquad (2\text{-}28)$$

This is a straight line if only reference narcotics are considered Δc_ℓ, = 0 or if only one chemical class correction is involved, e.g., all halogenated reference narcotics. Otherwise, more than one Δc_ℓ, enters into Equation 2-28 and the line is jagged. Figure 2-8 presents three examples. The deviations from the reference narcosis straight line are caused by the different chemical class potencies.

2.7 Universal Narcosis Slope

The universal narcosis slope: m = -0.945±0.014 which results from the final analysis that includes chemical class corrections (Table 2-1) is smaller than that determined above without chemical class corrections (-0.97±0.012). It is close to unity, a value commonly found (Hansch and Leo, 1995), and larger than the average of individual slopes (-0.86±0.14) reported by Van Leeuwen et al. (1992), but comparable with a recent estimate for fathead minnows of -0.94 (Di Toro et al., 2000).

The fact that the slope is not exactly one suggests that octanol is not quite lipid. However, it is also possible that for the more hydrophobic chemicals in the database, the exposure time may not have been long enough for complete equilibration of water and lipid to have occurred. To test this hypothesis, the regression analysis is

restricted to successively smaller upper limits of $\log_{10}K_{OW}$. The results are listed below

Maximum $\log_{10}K_{OW}$	3.5	4.0	4.5	5.0	5.5
Slope	-0.959	-0.970	-0.958	-0.950	-0.945
Standard Error	0.018	0.015	0.015	0.014	0.015

The variation is within the standard errors of estimation, indicating that there is no statistically significant difference if the higher $\log_{10}K_{OW}$ data are removed from the regression. This suggests that the universal narcosis slope is not minus one but is actually -0.945 ± 0.014.

One consequence of the use of a universal narcosis slope is that the species sensitivity ranking derived from comparing either the water-only LC50 values or the critical body burdens of various species are the same. This occurs because the critical body burden is calculated from the LC50 value and the universal slope (Equations 2-14 and 2-15)

$$\log_{10}C_L^* = \log_{10}LC50 + 0.945\log_{10}K_{OW} \qquad (2\text{-}29)$$

If this were not the case, then the species sensitivity order could be reversed if LC50 values or C_L^* were considered.

Equation 2-29 is important because it can be used to compute the critical body burden of any type I narcotic chemical. Thus it predicts what the critical body burden should be for a particular species at its LC50 value. This would be the concentration that would be compared to a directly measured critical body burden. It can be thought of as a normalization procedure that corrects type I narcotics for the varying K_{OW} and places them on a common footing, namely, the critical body burden.

The motivation for the development of the target lipid model was to apply it to mixtures of PAHs and other persistent narcotic chemicals in sediments. The narcosis database used to determine the universal narcosis slope and the critical body burdens consists of 145 chemicals, of which 10 are un-substituted and substituted PAHs (Di Toro et al., 2000). A comparison of the LC50 data for just these chemicals and the target lipid

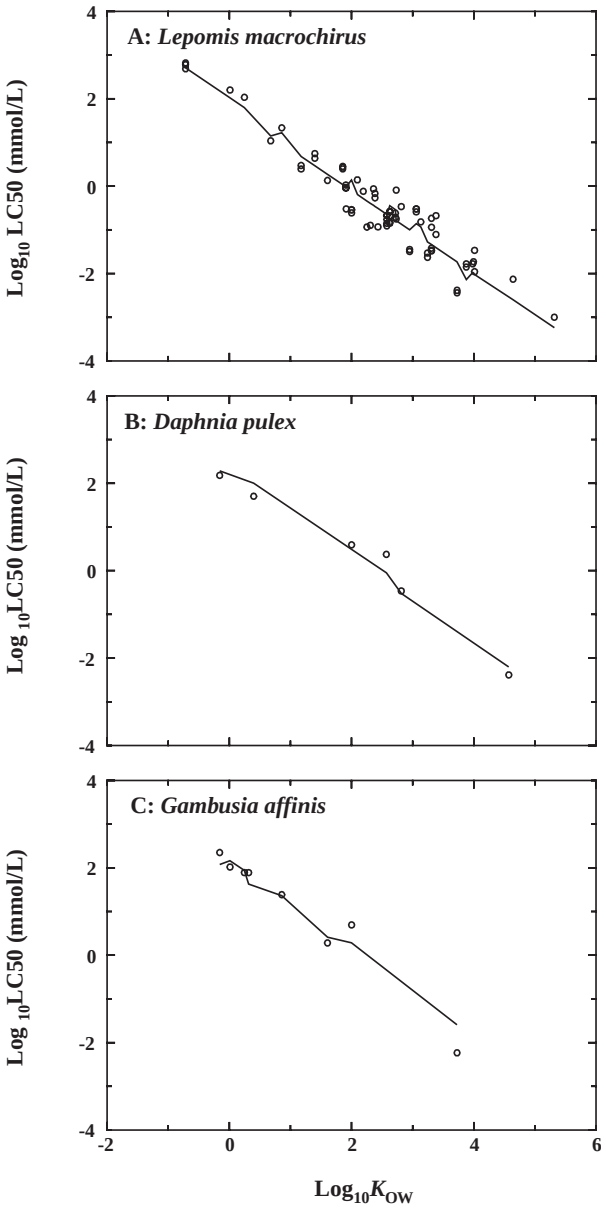

Figure 2-8. $\log_{10}$LC50 versus $\log_{10}K_{OW}$ for (A) *Lepomis macrochirus*, (B) *Daphnia pulex*, and (C) *Gambusia affinis*. The line connects the individual estimates of the $\log_{10}$LC50 values, including the chemical class correction.

model is shown in Figure 2-9. The solid $\log_{10}$LC50 - $\log_{10}K_{OW}$ lines are computed using the universal narcosis slope and the appropriate body burdens for PAHs for each organism listed. The dotted lines apply to the chloronaphthalenes which have a slightly lower critical body burden due to the halogen substitution. The lines are an adequate fit of the data, although the scatter in the *D. magna* data is larger than some of the other

Table 2-1. Regression results: y-intercepts and chemical class corrections‡ (Table from Di Toro et al., 2000).

Species i	N	b_i	SE(b_i)	10^{b_i} (µmol/g octanol)	SE(10^{b_i}) (µmol/g octanol)
Americamysis bahia	30	1.54	0.082	34.3	6.7
Portunus pelagicus	4	1.56	0.19	36.1	18.2
Leptocheirus plumulosus	4	1.56	0.191	36.2	18.4
Palaemonetes pugio	8	1.68	0.137	48.2	16.4
Oncorhynchus mykiss	44	1.79	0.065	61.7	9.4
Jordanella floridae	18	1.82	0.096	66.1	15.2
Ictalurus punctatus	7	1.87	0.139	74.8	25.9
Pimephales promelas	182	2.02	0.044	105	10.8
Lepomis macrochirus	70	2.03	0.0056	108	14.1
Daphnia magna	113	2.04	0.049	111	12.6
Cyprinodon variegatus	33	2.05	0.078	111	20.5
Oryzias latipes	4	2.05	0.182	112	53.9
Carassius auratus	43	2.13	0.065	134	20.5
Rana catesbian	5	2.13	0.162	135	55.9
Tanytarsus dissimilis	9	2.14	0.125	137	42
Orconectes immunis	6	2.14	0.149	139	52.3
Alburnus alburnus	7	2.16	0.137	144	49.1
Nitocra spinipes	6	2.17	0.148	147	54.7
Gambusia affinis	8	2.17	0.13	149	47.9
Leucisus idus melanotus	26	2.18	0.075	152	26.8
Neanthes arenaceodentata	4	2.23	0.19	168	85
Artemia salina nauplii	32	2.26	0.077	181	32.8
Lymnaea stagnalis	5	2.29	0.163	195	81.5
Xenopus laevis	5	2.33	0.163	213	88.9
Hydra oligactis	5	2.33	0.163	214	89.5
Culex pipiens	5	2.34	0.163	216	90.4
Poecilia reticulata	14	2.36	0.101	228	55.2
Menidia beryllina	8	2.37	0.134	233	77.3
Daphnia pulex	6	2.38	0.15	240	91

Table 2-1. Continued

Species i	N	b_i	SE(b_i)	10^{b_i} (µmol/g octanol)	SE(10^{b_i})
Ambystoma mexicanum	5	2.39	0.163	245	103
Daphnia cucullata	5	2.4	0.163	249	104
Aedes aegypti	5	2.42	0.163	261	109
Tetrahymena elliotti	10	2.46	0.121	286	85
Chemical Class ℓ	N	Δc_ℓ	SE(Δc_ℓ)	$10^{\Delta c_\ell}$	SE($10^{\Delta c_\ell}$)
Aliphatics	215	0	-	1	-
Ethers	13	0	-	1	-
Alcohols	134	0	-	1	-
Aromatics	241	0	-	1	-
Halogenated	319	-0.244	0.033	0.57	0.044
Ketones	49	-0.245	0.059	0.569	0.078
PAHs	84	-0.263	0.057	0.546	0.073
Slope		-0.945	0.014		

‡See Equation (2-27).
N = Number of data points.
b_i = y-intercept.
SE(b_i)=Standard error of b_i.
Δc_ℓ =chemical class correction to the y-intercept.
SE(Δc_ℓ)=standard error of Δc_ℓ.
†=Standard errors of 10^{b_i} and $10^{\Delta c_\ell}$ are based on the assumption that the estimation errors for b_i and Δc_ℓ are gaussian. The formulas follow from the standard error of a log normally distributed random variable (Aitchison and Brown, 1957). For $x = b_i$ or Δc_ℓ, $\mu_\ell = 2.303x$, $\sigma_\ell = 2.303\ SE(x)$, and $SE(10^x) = SE(e^{2.303x}) = e^{\mu_\ell}\sqrt{e^{2\sigma_\ell^2} - e^{2\sigma_\ell^2}}$.

species with multiple sources of data and there is a clear outlier for *Americamysis bahia*. It is for this reason that the slope representing all data for narcosis chemicals is used to derive the target lipid concentration from water-only toxicity data for PAHs in Section 3 of this document.

2.8 Comparison to Obsered Body Burdens

The target lipid model predicts the concentration in octanol (the y-intercept) that causes 50% mortality in 96 hours. The question is: how do these compare to measured critical body burdens? The species-specific y-intercepts, b_i, are related to the target lipid concentration by the relationship

$$\text{y-intercept} = b_i = \log_{10} C_L^*(i) - a_0 \qquad (2\text{-}30)$$

or, with chemical class corrections,

$$\text{y-intercept} = b_i + \Delta c_\ell = \log_{10} C_L^*(i) - a_0 \qquad (2\text{-}31)$$

for species i and chemical class ℓ, where a_0 is the parameter in the LFER between octanol and target lipid (Equation 2-12).

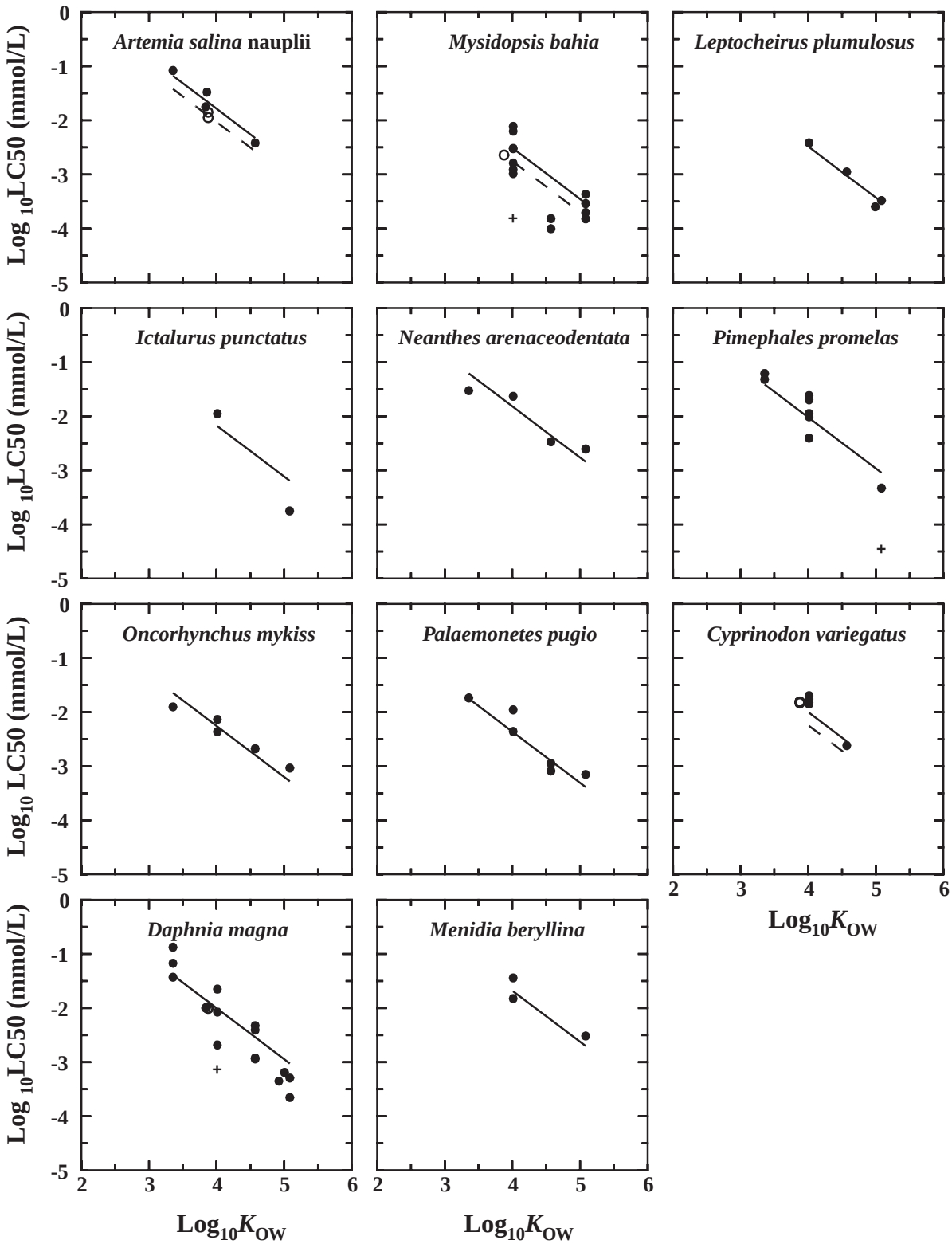

Figure 2-9. Comparison of target lipid model, line-of-fit and observed LC50 data for individual PAHs, by species.

The relationship between the predicted concentration in octanol, $b_i + \Delta c_\ell$, to the concentration measured in extracted lipid, $\log_{10}C_L^*$, is examined in Table 2-2 which lists observed LC50 body burdens (µmol/g lipid) and predicted critical body burdens (µmol/g octanol) for organisms in the database for which measured lipid-normalized critical body burdens were available. Three fish species: *Gambusia affinis* (mosquito fish), *Poecilia reitculata* (guppy) and *P. promelas*, and a crustacean: *Portunus pelagicus* (crab) are compared in Figure 2-10. The predicted and measured body burdens differ by less than a factor of 1.6. The fish were observed to have higher critical body burdens than the crustacean, which the model reproduces.

The apparent near equality between the estimated and measured critical body burdens, which come from two independent sets of data, strongly suggest that in fact

$$a_0 = 0 \qquad (2\text{-}32)$$

so that

$$\log_{10}C_L^*(i) = b_i + \Delta c_\ell = \text{y-intercept} \qquad (2\text{-}33)$$

This relationship implies that the target lipid is the lipid measured by the extraction technique used in the body burden datasets. This is an important practical result since it suggests that body burdens normalized to extracted lipid are expressed relative to the appropriate phase for narcotic toxicity. Since the intercepts appear to be the organism's lipid concentration, the y-intercepts ($b_i + \Delta c_\ell$) in the discussion presented below are referred to as body burden lipid concentrations although the units (µmol/g octanol) are retained since these are, in fact, the actual units of the intercepts.

2.9 Mixtures and Additivity

Narcotic chemicals, including PAHs, occur in the environment as mixtures, therefore, their mixture effects need to be appropriately resolved. If the toxicity of mixtures is additive, mixture effects can be assessed using the concept of toxic units. A toxic unit (TU) is defined as the ratio of the concentration in a medium to the effect concentration in that medium.

The additivity of the toxicity of narcotic chemicals in water has been demonstrated by a number of investigators. The results of mixture experiments which employed a large enough number of narcotic chemicals so that non-additive behavior would be detected is presented in Figure 2-11 as adopted from Hermens (1989). Three of the four experiments demonstrated essentially additive behavior and the fourth, a chronic exposure, was almost additive.

2.10 Aqueous Solubility Constraint

The existence of the need for a solubility cut-off for toxicity was suggested by Veith et al. (1983) based on data from fathead minnows (*P. promelas*) and guppies (*P. reticulata*). The highest dissolved concentration in water that can be achieved by a chemical is its aqueous solubility (S). Therefore, the maximum lipid concentration that can be achieved is limited as well. It is for this reason that the LC50 database is limited to chemicals with $\log_{10}K_{OW} \leq 5.3$. This is also the reason that the LC50 database that was used to generate the FCVs for specific PAHs in Section 3 of this document, was screened initially for LC50 values $\leq$ S, using the solubilities from Mackay et al. (1992), rather than $\log_{10}K_{OW} \leq 5.3$ used by Di Toro et al. (2000).

For sediments, a solubility constraint should be applied as well. This is readily calculated using the relationship between interstitial water and the organic carbon-normalized sediment concentration. Since the interstitial water concentration is limited by S, the sediment concentration should be limited by the concentration in sediment organic carbon that is in equilibrium with the interstitial water at the aqueous solubility. Therefore, observed sediment concentrations are limited by the condition

$$C_{OC} \leq C_{OC,max} = K_{OC}S \qquad (2\text{-}34)$$

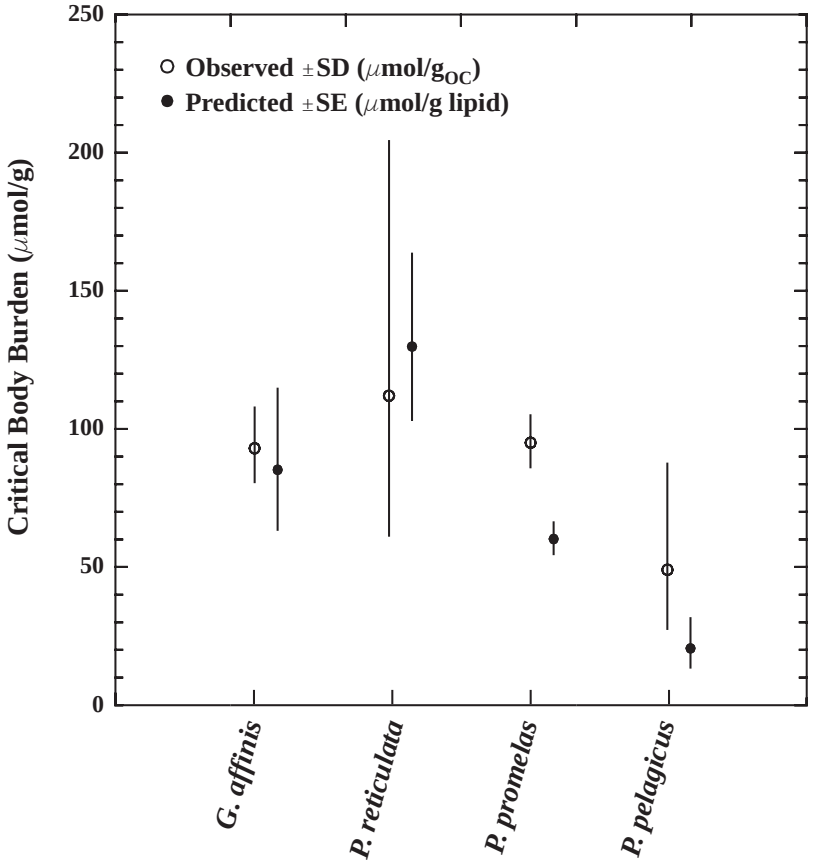

Figure 2-10. Predicted and observed body burdens for four species.

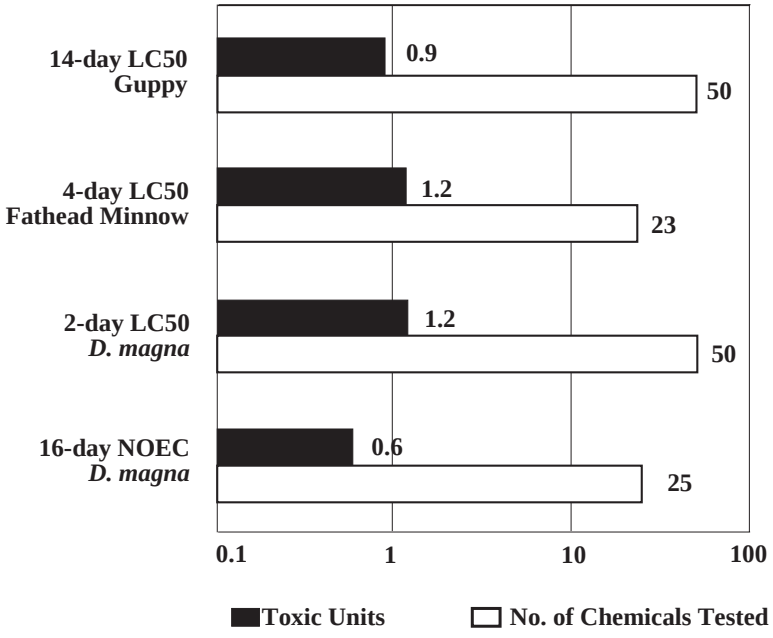

Figure 2-11. Additivity of type I narcosis toxicity. Comparison of the observed TU concentrations calculated from four studies to the predicted TU of 1.0.

Table 2-2. Comparison of body burdens observed in aquatic organisms acutely exposed to narcotic chemicals and body burdens predicted from target lipid narcosis theory (Table from Di Toro et al., 2000).

				C^*O_{rg}			
				Obs	Mean	Pred.	
Organism	Chemical	log K_{OW}	time (hr)	(µmol/ g lipid)		(µmol/g octanol)	References
Mosquitofish,	1,4-dibromonbenzene	3.55	96	85.0			Chaisuksant and
Gambusia affinis	1,2,3-trichlorobenzene	3.98	"	140.0			Connell, 1997
	1,2,4-trichlorobenzene	4.00	"	92.0			
	pentachlorobenzene	5.32	"	69.0	93.2	85.3	
Guppy,	1,4-difluorobenzene	2.11	1.5	444.0			Sijm et al., 1993
Poecilia reticulata	1,2-dichlorobenzene	3.31	91	34.0			
	1,4-dichlorobenzene	3.24	41	400.0			
	1,2-dibromobenzene	3.56	4	24.0			
	1,4-dibromobenzene	3.55	6	120.0	110	130	
Fathead minnow,	1,2-dichlorobenzene	3.31	18	78.0			Sijm et al., 1993
Pimephales promelas	1,4-dichlorobenzene	3.24	10	68.0			
	1,2-dibromobenzene	3.56	7	60.0			
	1,4-dibromobenzene	3.55	10	54.0			
	1,2,4-trichlorobenzene	4.00	50.2				van Wezel et al.,
	1,1,2,2-tetrachlorobenzene	2.31	57.2				1995
	dichlorobenzene	3.27	75.5				
	dichlorobenzene	3.27	129				
	1,2-dichlorobenzene	3.31	62.3				
	1,2-dichlorobenzene	3.31		98.9			van Wezel et al.,
	1,4-dichlorobenzene	3.24		173			1996
	1,4-dichlorobenzene	3.24		121			
	1,2-1,4-dichlorobenzene			107			
	1,2-1,4-dichlorobenzene			110			
	1,2-1,4-dichlorobenzene			138			
	1,2-1,4-dichlorobenzene			150			
	naphthalene	3.36		123			de Maagd et al.,
	1,2,4-trichlorobenzene	4.00		215	95	59.9	1996
Crab,	1,4-dichlorobenzene	3.24	96	9.6			Mortimer and
Portunus pelagicus	1,2,3-trichlorobenzene	3.98	96	45.0			Connell, 1994
	1,2,3,4-tetrachlorobenzene	4.64	96	119			
	pentachlorobenzene	5.32	96	111	49.9	20.6	

Section 3

Toxicity of PAHs in Water Exposures and Derivation of PAH-Specific FCVs

3.1 Narcosis Theory, EqP Theory and WQC Guidelines: Derivation of PAH-Specific FCVs for Individual PAHs

Polycyclic aromatic hydrocarbons occur in the environment as mixtures. Therefore, in order to adequately protect aquatic life the approach used to derive a WQC FCV or sediment benchmark for PAHs must account for their interactions as a mixture. In this section, we present an approach for deriving FCVs for individual PAHs which can be used to derive the ESB for mixtures of PAHs.

Concepts developed by Di Toro et al. (2000) and presented in Section 2 of this document provide the technical framework for screening and analyzing aquatic toxicity data on PAHs (Tables 3-1, 3-2). In particular, Section 2 demonstrated that: (1) the universal slope of the K_{OW}-toxicity relationship for narcotic chemicals is the same for all aquatic species; and (2) the intercept of the slope at a K_{OW} of 1.0 for each species provides the LC50/EC50 in µmol/g octanol that indicates the critical body burden in and relative sensitivities of each species.

These concepts permit the use of the U.S. EPA National WQC Guidelines (Stephan et al., 1985) to derive WQC FCVs for individual PAHs and PAH mixtures. The universal slope is used with PAH-specific LC50/EC50 values to derive test-specific K_{OW} normalized reference acute values at a K_{OW} of 1.0. This normalization was performed to put the data on the toxicities of narcotic chemicals on an internally consistent scale. This was also performed using hardness when WQC were derived for metals. These K_{OW} normalized reference acute values are used to calculate species mean acute values (SMAVs) and genus mean acute values (GMAVs): (1) because only acute and chronic toxicity data from water-only tests with freshwater and saltwater species exposed to individual PAHs are used, a PAH chemical class correction is not needed; (2) the data are screened for acceptability following the requirements for use of species resident to North America, test durations, test quality, etc. of the U.S. EPA National WQC Guidelines (Stephan et al., 1985); (3) the PAH-specific species mean acute values (PAH-specific SMAVs) from Appendix C are adjusted using the universal slope of the K_{OW}-toxicity relationship from the narcotic chemical analysis that was shown to apply to all aquatic species in Section 2 (Equation 2-29) to derive the acute value for that species at a K_{OW} of 1.0 (K_{OW} normalized PAH-specific SMAV) (Appendix C); (4) the intercept of the slope at a K_{OW} of 1.0 provides the LC50/EC50 in µmol/g octanol that indicates the relative sensitivity of each tested species and PAH, which was used to calculate SMAVs and GMAVs in µmol/g octanol, which are indicative of critical tissue concentrations in organisms on a µmol/g lipid basis. The GMAVs are used to calculate the final acute value (FAV) applicable to PAHs at a K_{OW} of 1.0 (Stephan et al., 1985). This FAV at a K_{OW} of 1.0, when divided by the Final Acute-Chronic Ratio (FACR), becomes the FCV at a K_{OW} of 1.0. Importantly, the FCV for any specific PAH can then be derived by back calculating using FAV at a K_{OW} of 1.0, the K_{OW} of the specific PAH and the universal narcosis slope. When the PAH-specific FCV exceeds the known solubility of that PAH, the maximum contribution of that PAH to the toxicity of the mixture is set at the K_{OC} multiplied by the solubility of that PAH.

Table 3-1. Summary of the chronic sensitivity of freshwater and saltwater organisms to PAHs; test specific data.

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Cladoceran, *Daphnia magna*	LC	W	Anthracene	21d		2.1	5.3% fewer broods	<2.1	Holst and Giesy, 1989
						4	8.0% fewer broods		
						8.2	13.8% fewer broods		
Cladoceran, *Daphnia magna*	LC	W	Fluoranthene	21d	6.9-17	35	17% reduction in length	24.5	Spehar et al., 1999
						73	25% reduction in length, 37% fewer young/adult		
						148	No survival		
Cladoceran, *Daphnia magna*	LC	W	Phenanthrene	21d	46-57	163	Survival reduced 83%, 98% fewer broods	96.39	Call et al., 1986
Midge, *Paratanytarsus sp.*	LC	B	Acenaphthene	26d	32-295	575	Survival reduced ~90%, ~60% reduction in growth, no reproduction	411.8	Northwestern Aquatic Sciences, 1982
Midge, *Paratanytarsus sp.*	LC	B	Acenaphthene	26d	27-164	315	Survival reduced ~20%, ~30% reduction in growth	227.3	Northwestern Aquatic Sciences, 1982; Thursby, 1991a
						676	Survival reduced ~60%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50	109	5% reduction in growth	73.82	Academy of Natural Sciences, 1981; Thursby, 1991a
						410	26% reduction in growth, Survival reduced 45%		
						630	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50-109	410	20% reduction in growth, Survival reduced 66%	211.4	Academy of Natural Sciences, 1981; Thursby, 1991a
						630	No survival		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32-35d	67-332	495	54% reduction in growth	405.4	Cairns and Nebeker, 1982
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32-35d	197-345	509	30% reduction in growth	419	Cairns and Nebeker, 1982
						682	52% reduction in growth, Survival reduced 45%		
						1153	87% reduction in growth, Survival reduced 97%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	64	98	Survival reduced 24%	79.2	ERCO, 1981
						149	Survival reduced 65%		
						271	Survival reduced 75%		
						441	Survival reduced 80%		
Fathead minnow, *Pimephales promelas*	ELS	W	Acenaphthene	32d	50-91	139	Survival reduced 20%	112.5	ERCO, 1981
						290	Survival reduced 50%		
						426	Survival reduced 52%		
Fathead minnow, *Pimephales promelas*	ELS	W	Fluoranthene	32d	3.7-10.4	21.7	Survival reduced 67%, 50% reduction in growth	15.02	Spehar et al., 1999
Rainbow trout, *Oncorhynchus mykiss*	ELS	B/W	Phenanthrene	90d	5	8	Survival reduced 41%, 33% reduced growth	6.325	Call et al., 1986
						14	Survival reduced 48%, 44% reduced growth		
						32	Survival reduced 52%, 75% reduced growth		
						66	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Mysid, *Americamysis bahia*	LC	B/W	Acenaphthene	35d	100-240	340	93% reduction in young	285.7	Horne et al., 1983
						510	No survival		
Mysid, *Americamysis bahia*	LC	B/W	Acenaphthene	25d	20.5-44.6	91.8	91% reduction in young	63.99	Thursby et al., 1989b
						168	No reproduction, 34% reduction in growth		
						354	Survival reduced 96%, no reproduction		
Mysid, *Americamysis bahia*	LC	B/W	Fluoranthene	28d	35926	21	Survival reduced 26.7%, 91.7% reduction in young	15.87	U.S. EPA, 1978
						43	No survival		
Mysid, *Americamysis bahia*	LC	B/W	Fluoranthene	31d	0.41-11.1	18.8	Survival reduced 23%, no reproduction	14.44	Spehar et al., 1999
Mysid, *Americamysis bahia*	LC	B/W	Phenanthrene	32d	1.5-5.5	11.9	No survival	8.129	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	LC	B/W	Pyrene	28d	3.82	5.37	46% reduction in young	4.53	Champlin and Poucher, 1992b
						6.97	47% reduction in young		
						9.82	73% reduction in young		
						15.8	85% reduction in young		
						20.9	90% reduction in young, Survival reduced 37%		
						38.2	No survival		

Table 3-1. Continued

Common Name, Species	Test[A]	Habitat[B]	PAH	Duration	NOEC[C] (µg/L)	OEC[D] (µg/L)	Observed Effects (Relative to Controls)	Chronic Value (µg/L)	Reference
Sheepshead minnow, *Cyprinodon variegatus*	ELS	B/W	Acenaphthene	28d	240-520	970	Survival reduced 70%	710.2	Ward et al., 1981
						2000	No survival		
						2800	No survival		

[A] Test: LC = life-cycle, PLC = partial life-cycle, ELS = early life-stage
[B] Habitat: I = infauna, E = epibenthic, W = water column
[C] NOEC = Concentrations where no significant effects were detected.
[D] OEC = Concentrations where significant effects were detected on survival, growth, or reproduction.

Table 3-2. Summary of acute and chronic values, acute-chronic ratios and derivation of the final acute values, final acute-chronic values and final chronic values.

Common Name, Species	PAH Tested	Value (µg/L)	Value (µg/L)	Chronic Ratio	PAH-Specific Mean Acute-Chronic Ratio	Species Mean Acute-Chronic Ratio	Reference
FRESHWATER SPECIES							
Cladoceran, *Daphnia magna*	Anthracene	-	<2.1	-	-	-	Holst and Giesy, 1989
Cladoceran, *Daphnia magna*	Fluoranthene	117	24.5	4.78	4.78	-	Spehar et al., 1999
Cladoceran, *Daphnia magna*	Phenanthrene	117	96.4	1.21	1.21	2.41	Call et al., 1986
Midge, *Paratanytarsus* sp.	Acenaphthene	2,040[A]	411	4.96	-	-	Northwestern Aquatic Sciences, 1982
Midge, *Paratanytarsus* sp.	Acenaphthene	2,040[A]	227	9	6.68	6.68	Northwestern Aquatic Sciences, 1982; Thursby,1991a
Fathead Minnow, *Pimephales promelas*	Acenaphthene	608	405	1.5	-	-	Cairns and Nebeker, 1982; Thursby, 1991a
Fathead Minnow, *Pimephales promelas*	Acenaphthene	608	419	1.45	1.48	-	Cairns and Nebeker, 1982
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	73.82	-	-	-	Academy of Natural Sciences, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	211	-	-	-	Academy of Natural Sciences, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	79.2	-	-	-	ERCO, 1981
Fathead Minnow, *Pimephales promelas*	Acenaphthene	-	112	-	-	-	ERCO, 1981
Fathead Minnow, *Pimephales promelas*	Fluoranthene	69[C]	15	4.6	4.6	2.61	Spehar et al., 1999
Rainbow trout, *Oncorhynchus mykiss*	Phenanthrene	50[C]	6.32	7.9	7.9	7.9	Call et al., 1986

Table 3-2. Continued

Common Name, Species	PAH Tested	Value (µg/L)	Value (µg/L)	Chronic Ratio	PAH-Specific Mean Acute-Chronic Ratio	Species Mean Acute-Chronic Ratio	Reference
SALTWATER SPECIES							
Mysid, *Americamysis bahia*	Acenaphthene	466	286	1.63	-	-	Horne et al., 1983
Mysid, *Americamysis bahia*	Acenaphthene	460	64	7.19	3.42	-	Thursby et al., 1989b
Mysid, *Americamysis bahia*	Fluoranthene	40	15.9	2.52	-	-	U.S. EPA, 1978
Mysid, *Americamysis bahia*	Fluoranthene	31	14.4	2.15	2.33	-	Spehar et al., 1999
Mysid, *Americamysis bahia*	Phenanthrene	27.1	8.13	3.33	3.33	-	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	Pyrene	28.3	4.53	6.24	6.24	3.59	Champlin and Poucher, 1992b
Sheepshead minnow, *Cyprinodon variegatus*	Acenaphthene	3,100[B]	710	4.36	4.36	4.36	Ward et al., 1981

[A] Geometric mean of two flow-through measured tests from the same laboratory as conducted the life-cycle tests.
[B] LC50 concentration slightly greater than acenaphthene's water solubility.
[C] EC50 based on immobilization used as the acute value instead of the LC50.

Final Acute Value = 9.31 µmol/g octanol
Final Acute-chronic Ratio = 4.16
Final Chronic Value = 2.24 µmol/g octanol

The FCV at a K_{OW} of 1.0 for PAHs derived in this section of the document differs slightly from that which would be derived for other narcotic chemicals according to Di Toro et al. (2000) in that it: (1) is derived using only acute and chronic toxicity data from water-only tests with freshwater and saltwater species exposed to individual PAHs, therefore, the data do not require the PAH chemical class correction; (2) the data are rigorously screened for acceptability following the requirements for the use of species resident to North America, test durations, test quality, etc. of the U.S. EPA National WQC Guidelines (Stephan et al., 1985). All other steps in the derivation of FCVs are the same as those used by Di Toro et al. (2000).

3.2 Acute Toxicity of Individual PAHS: Water Exposures

3.2.1 Acute Toxicity of PAHs

One hundred and four acute water-only toxicity tests with 12 different PAHs have been conducted on 24 freshwater species from 20 genera that meet the requirements of the U.S. EPA National WQC Guidelines (Stephan et al., 1985, see Appendix C). The tested life-stages of 15 of the genera were benthic (infaunal or epibenthic). The most commonly tested freshwater species were the cladocerans (*Daphnia magna* and *D. pulex*), rainbow trout (*O. mykiss*), fathead minnow (*P. promelas*) and bluegill (*Lepomis macrochirus*). The most commonly tested PAHs with freshwater organisms were acenaphthene, fluoranthene, fluorene, naphthalene, phenanthrene and pyrene.

Seventy-seven acute water-only toxicity tests with 8 different PAHs have been conducted on 30 saltwater species from 29 genera (Appendix C). The tested life-stages of 21 of the genera were benthic (infaunal or epibenthic). The most commonly tested saltwater species were the annelid worm (*N. arenaceodentata*), mysid (*Americamysis bahia*), grass shrimp (*Palaemonetes pugio*), pink salmon (*Oncorhynchus gorbuscha*), and sheepshead minnow (*Cyprinodon variegatus*). The most commonly tested PAHs with saltwater organisms were acenaphthene, fluoranthene, naphthalene, phenanthrene and pyrene.

3.2.2 Acute Values at a K_{ow} of 1.0

The rules for test acceptability of the National WQC Guidelines (Stephan et al., 1985) were used to identify the LC50 values or EC50 (µg/L) values from individual acute aquatic toxicity tests (Appendix C) and these values were used to derive the K_{OW} normalized GMAV (µmol/g octanol) in the following manner. The goal of this process was to convert individual LC50 or EC50 values that vary for a species across PAHs into a PAH-specific GMAV normalized to a K_{OW} of 1.0. The use of normalizing factors in FCV derivation is not unique to this ESB document. The use of K_{OW} to normalize the toxicity of PAHs to put the toxicity data on an internally consistent scale is analogous to the hardness normalization applied to the freshwater WQC for cadmium, copper, lead, nickel and zinc and the pH and temperature normalization applied to the freshwater WQC for ammonia. For multiple PAHs tested against one species, the K_{OW} normalization should result in similar PAH-specific SMAVs. The first step in the analysis of published LC50 or EC50 values was to compare them to the known solubility in water of the PAH tested. If the LC50 or EC50 concentration exceeded the solubility of the tested PAH, the published LC50/EC50 is in parentheses in Appendix C, the solubility is listed in bold in Appendix C as a "greater than" acute value to indicate that the actual toxicity of the dissolved PAH was unknown. For these tests, this greater than solubility value, and not the published LC50 or EC50 value, was used in further calculations only when there were no acute values for that species at concentrations less than the solubility. Next, the LC50, EC50 or greater than solubility value was converted to mmol of the tested PAH/L. When the same PAH was tested more than once against a species, the geometric mean of all LC50 or EC50 values was calculated to determine the PAH-specific SMAV using the rules in Stephan et

al. (1985). The -0.945 universal slope of the toxicity/K_{OW} relationship (Equation 2-29) was applied to the PAH-Specific SMAVs (µmol/L) to calculate the PAH-specific SMAV (µmol/g octanol) at a K_{OW}=1.0. The SMAV for all tested PAHs is the geometric mean of the PAH-Specific SMAVs at a K_{OW} of 1.0. The GMAV (µmol/g octanol) at a K_{OW} of 1.0 is the geometric mean of the SMAVs at a K_{OW} of 1.0.

The SMAVs at a K_{OW} of 1.0 were similar for multiple PAHs (Appendix C). For 18 freshwater and saltwater species, two to nine different PAHs were tested. The ratios of the highest to lowest acute values for multiple PAHs tested against an individual species before normalization was 1.37 to 1170; an average ratio of 105. In contrast, the range in the ratios of the highest to lowest PAH-specific SMAVs at a K_{OW} of 1.0 was 1.4 to 12.2; average ratio of 4.27. For 10 of the 18 (56%) species tested against multiple PAHs, the ratio of high to low SMAVs at a K_{OW} of 1.0 was 4.0 or less. This compares favorably with the factor of four or less difference in the acute values for 12 of 19 (63%) of the same species in multiple tests with the same PAH. Therefore, the variability of SMAVs at a K_{OW} of 1.0 across PAHs is similar to the variability inherent for these data in acute toxicity testing with only one PAH. This suggests that the GMAVs provide data across PAHs that indicate the relative sensitivity of that species that can be used to describe species at risk and to calculate the FAV.

The K_{OW}-normalized GMAVs (not including values greater than the solubility of the tested PAH) range from 7.63 µmol/g octanol for *Americamysis* to 187 µmol/g octanol for *Tanytarsus*, a factor of only 24. Saltwater genera constitute four of the five genera with GMAVs at a K_{OW} of 1.0 within a factor of two of the most sensitive genus (*Americamysis*). Of the 49 genera, the most sensitive one-third include a freshwater hydra, two amphipods, an insect, saltwater fish, a crab, two mysids, two shrimp, and three saltwater amphipods. All of these 16 genera have GMAVs at a K_{OW} of 1.0 that are within a factor of three, and 14 of the genera are benthic. Benthic and water column genera are distributed throughout the sensitivity distributions indicating that they have similar sensitivities. Genera that are benthic have been tested more frequently than water column genera.

3.3 Applicability of the WQC as the Effects Concentration for Benthic Organisms

The use of the FAV or FCV as the effects concentration for calculation of ESBs assumes that benthic (infaunal and epibenthic) species, taken as a group, have sensitivities similar to all aquatic (benthic and water column) species used to derive the WQC FCV. The data supporting the reasonableness of this assumption over all chemicals for which there were published or draft WQC documents were presented in Di Toro et al. (1991) and U.S. EPA (2003a). The conclusion of similarity of sensitivity was supported by comparisons between (1) acute values for the most sensitive benthic species and acute values for the most sensitive water column species for all chemicals; (2) acute values for all benthic species and acute values for all species in the WQC documents across all chemicals after normalizing the LC50 values; (3) FAVs calculated for benthic species alone and FAVs in the WQC documents; and (4) individual chemical comparisons of benthic species versus all species. The following analysis examines the data on the similarity of sensitivity of benthic and all aquatic species for PAHs.

For PAHs, benthic life-stages were tested for 15 of 20 freshwater genera and 21 out of 29 saltwater genera (Appendix C). An initial test of the difference between the freshwater and saltwater FAVs for all species (water column and benthic) exposed to PAHs was performed using the Approximate Randomization (AR) Method (Noreen, 1989). The AR Method tests the significance level of a test statistic when compared to a distribution of statistics generated from many random sub-samples. The test statistic in this case was the difference between the freshwater FAV (computed from the GMAVs at a K_{OW} of 1.0 for combined water column and benthic organisms) and the saltwater FAV (computed from the GMAVs at a K_{OW} of 1.0 for combined water

column and benthic organisms) (Appendix C). In the AR Method, the freshwater and the saltwater GMAVs at a K_{OW} of 1.0 were combined into one dataset. The dataset was shuffled, then separated back so that randomly generated "freshwater" and "saltwater" FAVs could be computed. The LC50 values were re-separated such that the number of GMAVs at a K_{OW} of 1.0 used to calculate the sample FAVs were the same as the number used to calculate the original FAVs. These two FAVs were subtracted and the difference used as the sample statistic. This was done iteratively so that the sample statistics formed a probability distribution representative of the population of FAV differences (Figure 3-1A). The test statistic was compared to this distribution to determine its level of significance. The null hypothesis was that the GMAVs at a K_{OW} of 1.0 that comprise the freshwater and saltwater data bases were not different. If this was true, the difference between the actual freshwater and saltwater FAVs should be common to the majority of randomly generated FAV differences. For PAHs, the test-statistic occurred at the 93.5 percentile of the generated FAV differences (Table 3-3). This percentile suggests that saltwater genera may be somewhat

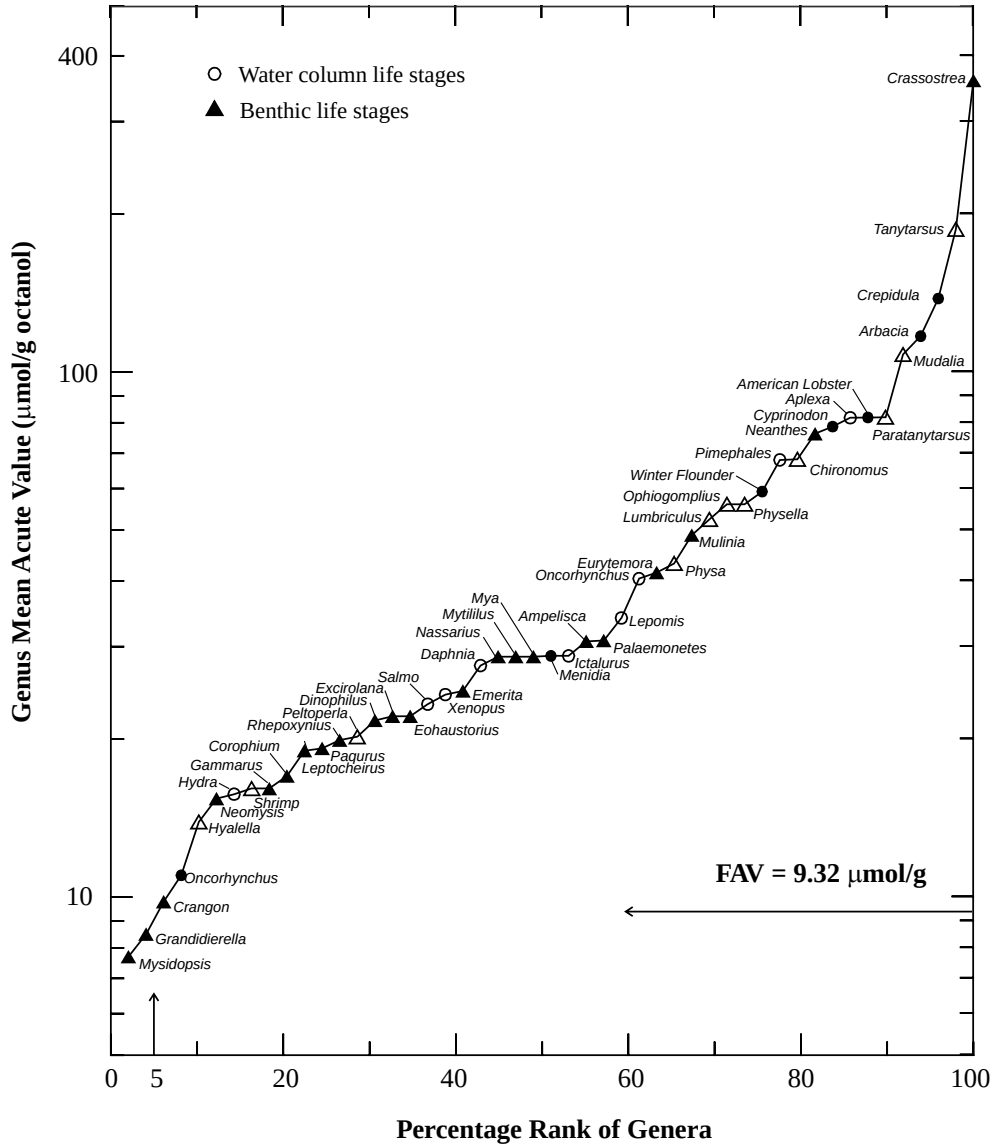

Figure 3-1 Probability distributions of FAV difference statistics to compare water-only toxicity data from (A) freshwater versus saltwater genera and (B) benthic versus WQC.

Table 3-3. Results of approximate randomization (AR) test for the equality of the freshwater and saltwater FAV distributions at a K_{OW} of 1.0 and AR test for the equality of benthic and combined benthic and water column FAVs for freshwater and saltwater distributions.

Comparison	Habitat or Water Type[A]		AR Statistic[B]	Probability[C]
Fresh vs Salt	Fresh (20)	Salt (29)	5.746	93.5
Freshwater: Benthic vs WQC[D]	WQC (49)	Benthic (33)	0.862	82.8

[A] Values in parantheses are the number of GMAVS at a K_{OW} of 1.0 used in the comparison.
[B] AR statistic = FAV difference between original compared groups.
[C] Probability that the theoretical AR statistic ≤ the observed AR statistic given that all samples came from the same population.
[D] Combined freshwater and saltwater.

more sensitive than freshwater genera as illustrated in Figure 3-2 and Appendix C. However, since the probability was less than 95% in the AR analysis, the null hypothesis of no significant difference in sensitivity for freshwater and saltwater species was accepted (Table 3-3).

Since freshwater and saltwater species showed no significant differences in sensitivity, the AR Method was applied jointly for the analysis of the difference in sensitivity for benthic and all aquatic organisms (benthic and water column species are always combined to derive WQC, therefore, the complete GMAV dataset is hereafter referred to as "WQC"). Using the criteria in U.S. EPA (2003a), each life stage of each test organism, hence each GMAV at a K_{OW} of 1.0, was assigned a habitat (Appendix C). The test statistic in this case was the difference between the WQC FAV, computed from the WQC GMAVs at a K_{OW} of 1.0, and the benthic FAV, computed from the benthic organism GMAVs at a K_{OW} of 1.0. The approach used to conduct this analysis was slightly different than that used in the previous test for freshwater and saltwater GMAVs. The difference was that freshwater and saltwater GMAVs in the first test represented two separate groups. In this test, the GMAVs at a K_{OW} of 1.0 for benthic organisms were a subset of the GMAVs at a K_{OW} of 1.0 in the entire WQC dataset. In the AR analysis for this test, the number of data points coinciding with the number of benthic organisms were selected from the WQC dataset to compute each "benthic" FAV. The original WQC FAV and the "benthic" FAV were then used to compute the difference statistic. This was done iteratively and the distribution that results was representative of the population of FAV difference statistics. The test statistic was compared to this distribution to determine its level of significance. The probability distributions of the computed FAV differences are shown in Figure 3-1B. The test statistic for this analysis occurred at the 82.8 percentile and the null hypothesis of no difference in the sensitivities between benthic species and species used to derive the WQC FCV was accepted (Table 3-3). This analysis supports the derivation of the FCV for PAHs based on all GMAVs at a K_{OW} of 1.0.

3.4 Derivation of the FAV at a K_{OW} of 1.0

The FAV is an estimate of the concentration corresponding to a cumulative probability of 0.05 in the GMAVs at a K_{OW} of 1.0. The analysis above demonstrates that the acute sensitivities of freshwater and saltwater genera and the sensitivities of benthic and benthic plus water column genera do not differ. Therefore, for calculation of the FAV, the GMAVs at a K_{OW} of 1.0 for all freshwater and saltwater genera can be grouped together to represent the relative sensitivities of all benthic organisms (Figure 3-2). The FAV at a K_{OW} of 1.0 is calculated using the procedure in Stephan et al. (1985), the GMAVs at

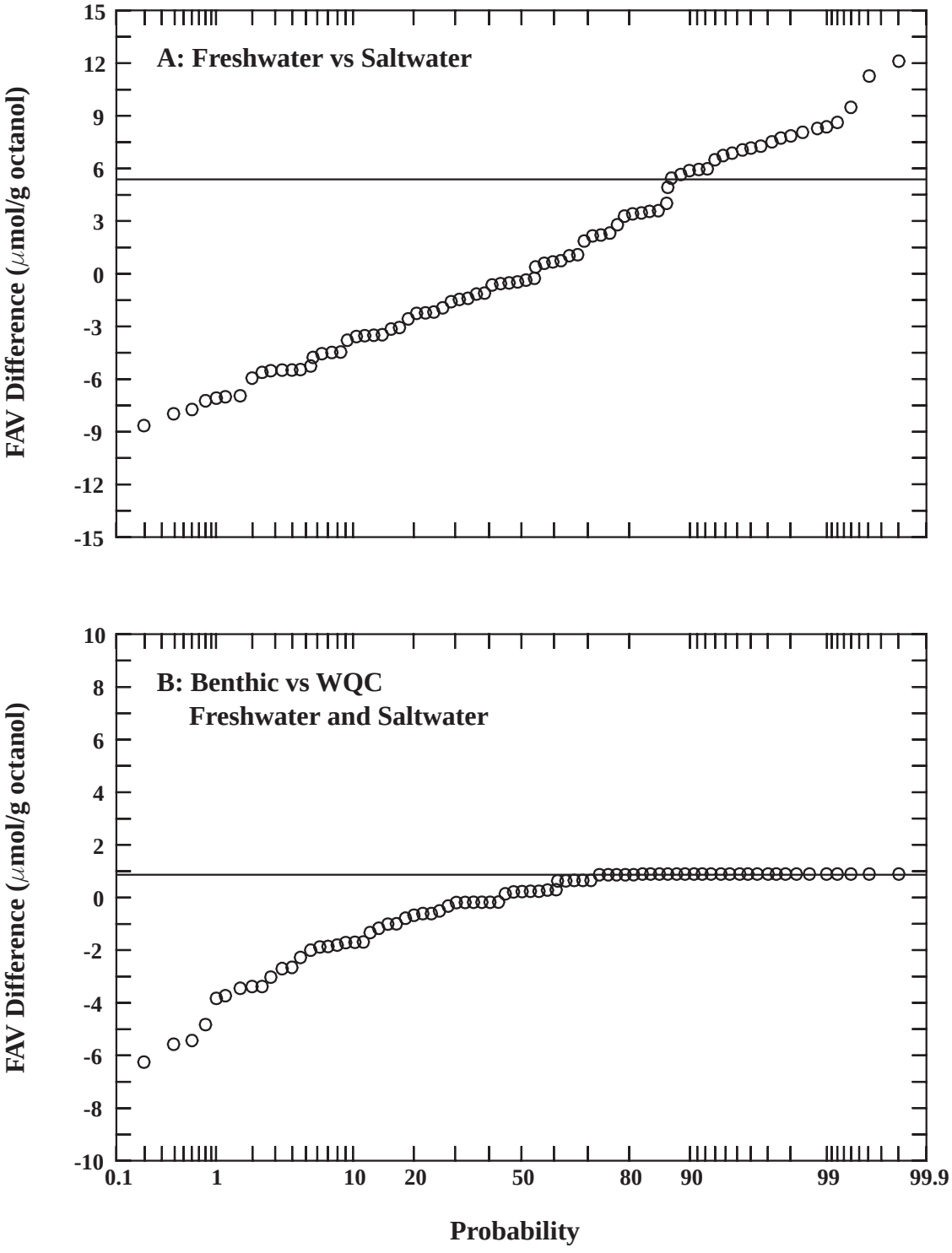

Figure 3-2. GMAVs at a $\log_{10}K_{OW}$ of 1.0 from water-only acute toxicity tests using freshwater and saltwater genera versus percentage rank of their sensitivity.

a K_{OW} of 1.0 of 7.63 µmol/g octanol for *Americamysis*, 8.51 µmol/g octanol for *Grandidierella*, 9.83 µmol/g octanol for *Crangon*, 11.0 µmol/g octanol for *Oncorhynchus* and the total number of genera tested (N = 49). The FAV at a K_{OW} of 1.0 is 9.31 µmol/g octanol. This FAV is greater than the GMAVs of the two most acutely sensitive genera as would be expected given the calculation procedure and the presence of 31 GMAVs.

3.5 Chronic Toxicity of Individual PAHs: Water Exposures

3.5.1 Acenaphthene

Chronic life-cycle toxicity tests have been conducted with acenaphthene with the freshwater midge (*Paratanytarsus* sp.) and the saltwater mysid (*A. bahia*), and early life-stage tests have been conducted with the fathead minnow (*P. promelas*) and sheepshead minnow (*C. variegatus*) (Table 3-1). For each of these species, one or more benthic life-stages were exposed. Other chronic toxicity tests have been conducted with the freshwater chironomid (*Paratanytarsus* sp.) and *P. promelas* (Lemke et al.,1983; Lemke, 1984; Lemke and Anderson, 1984) but insufficient documentation is available to permit use of these results (Thursby, 1991a).

Two acceptable life-cycle toxicity tests have been conducted with *Paratanytarsus* sp. (Northwestern Aquatic Sciences, 1982). In the first test, 575 µg/L reduced survival 90%, reduced growth 60%, and all eggs failed to hatch (Table 3-1). No adverse effects occurred at acenaphthene concentrations up to 295 µg/L acenaphthene. In the second test, survival was reduced 20% and growth 30% at 315µg/L. Egg hatchability was not affected in the highest concentration of 676 µg/L; although survival of hatched larvae was reduced ~60%. No significant effects were observed at acenaphthene concentrations up to 164 µg/L.

A total of six early life-stage toxicity tests have been conducted with *P. promelas* as part of a round-robin test series; two each from three laboratories (Table 3-1) (Academy of Natural Sciences, 1981; ERCO, 1981; Cairns and Nebeker, 1982). The lowest observed effect concentrations (LOEC) across laboratories and tests ranged from 98 to 509 µg/L, a factor of 5.2. Growth (dry weight), survival, or both growth and survival were reduced. Only one of these test pairs had a suitable measured acute value that allowed calculation of an ACR (Cairns and Nebeker, 1982). The concentration-response relationships were similar for the two tests of Cairns and Nebeker (1982). In the first test, the early life-stages of this fish were unaffected in acenaphthene concentrations ranging from 67 to 332 µg/L, but 495 µg/L reduced growth 54% relative to control fish. In the second test, growth was reduced 30% at 509 µg/L, but no effects were detected in fish exposed to 197 to 345 µg/L.

Data from saltwater chronic toxicity tests with acenaphthene are available for *A. bahia* and *C. variegatus*. Reproduction of *A. bahia* was affected by acenaphthene in two life-cycle tests from two different laboratories. In the first test (Horne et al., 1983), 340 µg/L reduced reproduction 93% relative to controls and all *A. bahia* died at 510 µg/L. No effects were observed on the parental generation at 100 to 240 µg/L and second generation juveniles were not affected at ≤ 340 µg/L. In the second test (Thursby et al., 1989b), no effects were observed at ≤ 44.6 µg/L, but a concentration of 91.8 µg/L reduced reproduction 91%. No reproduction occurred at higher concentrations, and growth was reduced 34% at 168 µg/L and survival 96% at 354 µg/L.

A test with early life-stages of *C. variegatus* showed that 240 to 520 µg/L had no effects, but that concentrations of 970, 2,000 and 2,800 µg/L reduced survival of embryos and larvae by ≥70% (Table 3-1; Ward et al., 1981).

In general, the above results show that the difference between acute and chronic toxicity of acenaphthene is small and differed minimally between species (Table 3-2). Species mean acute-chronic ratios for acenaphthene are 6.68 for *Paratanytarsus* sp., 1.48 for *P. promelas*, 3.42 for *A. bahia* and 4.36 for *C. variegatus*.

3.5.2 Anthracene

A single life-cycle toxicity test has been conducted with *D. magna* exposed to only three concentrations of anthracene (Holst and Geisy, 1989). Minimal decreases were observed on the number of broods produced in all three of the concentrations tested: 2.1 mg/L (5.3%), 4.0 µg/L (8.0%) and 8.2 µg/L (13.8%). No acute toxicity tests were conducted by the authors. Therefore, an ACR could not be derived for anthracene.

3.5.3 Fluoranthene

Fluoranthene has been tested in life-cycle toxicity tests with the freshwater cladoceran, *D. magna* (Spehar et al., 1999) and the saltwater mysid, *A. bahia* (U.S. EPA, 1978, Spehar et al., 1999), and early life-stage tests have been conducted with the fathead minnow (Spehar et al., 1999) (Table 3-1). No effects were observed with *D. magna* at ≤17 µg/L, but growth was reduced 17% at 35 µg/L and 25% at 73 µg/L. There were 37% fewer young per adult at 73 µg/L and no daphnids survived at 148 µg/L. An early life-stage toxicity test conducted with the fathead minnow showed no effects at ≤10.4 µg/L, but reduced survival (67%) and growth (50%) at 21.7 µg/L.

Saltwater mysids (*A. bahia*) were tested in two life-cycle toxicity tests. In the first test, the mysids were exposed to fluoranthene for 28 days (U.S. EPA, 1978). There was no effect on survival or reproduction (growth was not measured) in concentrations ranging from 5-12 µg/L. At a fluoranthene concentration of 21 µg/L, survival was reduced 26.7% and reproduction 91.7%, relative to the controls. At the highest concentration of fluoranthene, 43 µg/L, all *A. bahia* died. In the second test, *A. bahia* were exposed to fluoranthene for 31 days (Spehar et al., 1999). Effect concentrations were similar to those in the U.S. EPA (1978) test. *A. bahia* were not affected at fluoranthene concentrations from 0.41-11.1 µg/L. At the highest concentration tested, 18.8 µg/L, survival was reduced 23% relative to controls and there was no reproduction. Reproduction was reduced by 77% in 11.1 µg/L, but this was not significantly different from controls even at α=0.1.

The difference between acute and chronic sensitivity to fluoranthene varied minimally between species (Table 3-2). Three species mean ACRs are available for fluoranthene: 4.78 for *D. magna*, 4.60 for *P. promelas*, and 2.33 for *A. bahia*.

3.5.4 Phenanthrene

Phenanthrene has been tested in life-cycle toxicity tests with *D. magna* and *A. bahia* and an early life-stage test has been conducted with rainbow trout (*O. mykiss*) (Table 3-1). There were no effects of phenanthrene on *D. magna* at ≤57 µg/L, but survival was reduced 83% and reproduction 98% at 163 µg/L (Call et al., 1986). In a test with *O. mykiss*, no effects were observed at 5 µg/L. The percentage of abnormal and dead fry at hatch was significantly increased at the highest exposure concentration of 66 µg/L and survival of hatched fry was reduced with increase in exposure concentration (Call et al., 1986). Mortality was 41, 48, 52 and 100% at 8, 14, 32, and 66 µg/L, respectively. Wet weight was reduced 33, 44, and 75% at 8, 14 and 32 µg/L, respectively.

A life-cycle toxicity test with *A. bahia* exposed to phenanthrene showed that the effect concentrations were similar to those that affected *O. mykiss* (Kuhn and Lussier, 1987) (Table 3-1). Survival, growth and reproduction were not affected at ≤5.5 µg/L. However, at the highest test concentration of phenanthrene (11.9 µg/L), all mysids died.

The difference between acute and chronic sensitivity to phenanthrene varied minimally between *D. magna* (PAH-specific ACR= 1.21), *O. mykiss* (ACR=7.90) and *A. bahia* (ACR= 3.33). The ACR for *O. mykiss* (Call et al., 1986) was derived using the EC50 for immobilization (50 µg/L) and not the 96-hour LC50 of 375 µg/L as was required in Stephan et al. (1985).

3.5.5 Pyrene

A life-cycle toxicity test with *A. bahia* exposed to pyrene was conducted by Champlin and Poucher (1992b). There were no effects at 3.82 µg/L, but

20.9 µg/L reduced survival 37% and no mysids survived at the next higher concentration of 38.2 µg/L (Table 3-1). Reproduction was significantly reduced in ≥5.37 µg/L. The ACR from this test was pyrene is 6.24.

3.5.6 Naphthalene

Fathead minnows were exposed to naphthalene in an early life-stage toxicity test (DeGraeve et al., 1982). Hatching of fry was significantly reduced in 4.38 and 8.51 µg/L and none were alive in these concentrations at the end of the 30-day test. Weight and length of fish surviving the test were significantly reduced in 0.85 and 1.84 µg/L. No significant effects were detected in concentrations ≤0.45 µg/L. Control survival was only 42%, which does not meet requirements according to the American Society of Testing and Materials (ASTM, 1998). Also, the carrier methanol was absent from the control. These data are summarized in the text for completeness, but the ACR of 12.7, chronic value of 0.62 µg/L, and 96-hour LC50 of 7.9 µg/L for naphthanlene are not included in Tables 3-1 and 3-2.

The calanoid copepod (*Eurytemora affinis*) was exposed indvidually to 14.21 µg/L naphthalene, 15.03 µg/L 2-methylnaphthalene, 8.16 µg/L 2,6-dimethylnaphthalene and 9.27 µg/L 2,3,5-trimethylnaphthalene in life-cycle toxicity tests (Ott et al., 1978). Survival and reproduction were affected by each of the naphthalenes, but ACRs could not be derived because the duration of the acute test was too short (24 hours) according to WQC Guidelines (Stephan et al., 1985), and no other concentrations were tested chronically.

3.5.7 Derivation of the Final Acute Chronic Ratio

The FACR for the six PAHs is 4.16. This FACR is the geometric mean of all species mean ACRs for *Daphnia* (2.41), *Paratanytarsus* (6.68), *Pimephales* (2.61), *Oncorhynchus* (7.90), *Americamysis* (3.59), and *Cyprinodon* (4.36) (Table 3-2).

3.6 Derivation Of FCVs

3.6.1 Derivation of the FCV at a K_{OW} of 1.0

The FCV is the value that should protect 95% of the tested species. The FCV is the quotient of the FAV and the FACR for the substance. The FAV at a K_{OW} of 1.0 is 9.31 mmol/g octanol. It is an estimate of the acute LC50 or EC50 concentration corresponding to a cumulative probability of 0.05 for the GMAVs at a K_{OW} of 1.0. The FACR of 4.16 is the mean ratio of acute to chronic toxicity for six species exposed exposed both acutely and chronically to one or more of six individual PAHs in 15 experiments. (For more information on the calculation of ACRs, FAVs, and FCVs see the U.S. EPA National WQC Guidelines (Stephan et al., 1985.))

The FAV at a K_{OW} of 1.0 of 9.31 µmol/g octanol is divided by the FACR of 4.16 to obtain a FCV at a K_{OW} of 1.0 of 2.24 µmol/g octanol (Table 3-3). Because nonionic organic chemicals partition similarly into octanol and lipid of organisms, the FCV at a K_{OW} of 1.0 in µmol/g octanol approximately equals tissue-based "acceptable" concentration of about 2.24 µmol/g lipid.

3.6.2 Derivation of the PAH-Specific FCVs

The PAH-specific FCVs (mg/L) (Table 3-4, Appendix D) are calculated from the FCV at a K_{OW} of 1.0 (µmol/g octanol), the slope of the K_{OW}-K_{OC} relationship, the universal narcotic slope of the K_{OW}-acute toxicity relationship, and the PAH-specific K_{OW} values (Equation 3-1, 3-2, and 3-3).

$$\log_{10}\text{PAH-specific FCV} = (\text{slope})\log_{10}K_{OW} + \log_{10}\text{FCV at a } K_{OW} \text{ of 1.0} \quad (3\text{-}1)$$

$$\log_{10}\text{PAH-specific FCV} = -0.945\log_{10}K_{OW} + \log_{10}(2.24) \quad (3\text{-}2)$$

$$\text{PAH-specific FCV (mmol/L)} = 1000(\text{antilog}(-0.945\log_{10}K_{OW} + 0.3502)) \quad (3\text{-}3)$$

Table 3-4. $C_{OC,PAHi,FCVi}$ concentrations and properties required for their derivation[A].

PAH[B]	SPARC[C] $\log_{10}K_{ow}$	$\log_{10}K_{oc}$	FCV_i (µmol/g octanol)	PAH specific FCV_i (µmol/L)	PAH specific FCV_i (µg/L)	$C_{OC,PAHi,FCVi}$ (µg/goc)	$C_{OC,PAHi,MAXi}$[D] (µg/goc)
indan	3.158	3.105	2.24	2.322	274.5	349	127200
naphthalene	3.356	3.299	2.24	1.509	193.5	385	61700
C1-naphthalenes	3.8	3.736	2.24	0.5744	81.69	444	-
1-methylnaphthalene	3.837	3.772	2.24	0.53	75.37	446	165700
2-methylnaphthalene	3.857	3.792	2.24	0.5074	72.16	447	154800
acenaphthylene	3.223	3.168	2.24	2.016	306.9	452	24000
acenaphthene	4.012	3.944	2.24	0.3622	55.85	491	33400
1-ethylnaphthalene	4.221	4.15	2.24	0.2298	35.91	507	142500
2-ethylnaphthalene	4.283	4.21	2.24	0.2008	31.37	509	129900
C2-naphthalenes	4.3	4.227	2.24	0.1935	30.24	510	-
1,4-dimethylnaphthalene	4.3	4.227	2.24	0.1935	30.24	510	192300
1,3-dimethylnaphthalene	4.367	4.293	2.24	0.1673	26.13	513	157100
2,6-dimethylnaphthalene	4.373	4.299	2.24	0.1651	25.79	513	33800
2,3-dimethylnaphthalene	4.374	4.3	2.24	0.1647	25.74	513	49900
1,5-dimethylnaphthalene	4.378	4.304	2.24	0.1633	25.52	514	62400
fluorene	4.208	4.137	2.24	0.2364	39.3	538	26000
C3-naphthalenes	4.8	4.719	2.24	0.0652	11.1	581	-
2,3,5-trimethylnaphthalene	4.858	4.776	2.24	0.05747	9.785	584	-
1,4,5-trimethylnaphthalene	4.872	4.789	2.24	0.05575	9.488	584	129300
anthracene	4.534	4.457	2.24	0.1163	20.73	594	1300
phenanthrene	4.571	4.494	2.24	0.1073	19.13	596	34300
C1-fluorenes	4.72	4.64	2.24	0.0776	13.99	611	-
1-methylfluorene	4.739	4.659	2.24	0.07445	13.42	612	49700
C4-naphthalenes	5.3	5.21	2.24	0.02197	4.048	657	-
2-methylanthracene	4.991	4.906	2.24	0.04303	8.273	667	2420
1-methylanthracene	4.998	4.913	2.24	0.04238	8.148	667	-
9-methylanthracene	5.006	4.921	2.24	0.04165	8.007	668	21775
2-methylphenanthrene	5.029	4.944	2.24	0.03961	7.616	669	-
1-methylphenanthrene	5.037	4.952	2.24	0.03893	7.485	670	24100
C1-phenanthrene/anthracenes	5.04	4.955	2.24	0.03868	7.436	670	-
9-ethylfluorene	4.973	4.889	2.24	0.04475	8.693	673	-
C2-fluorenes	5.2	5.112	2.24	0.02731	5.305	686	-
pyrene	4.922	4.839	2.24	0.05	10.11	697	9090
fluoranthene	5.084	4.998	2.24	0.03515	7.109	707	23870
2-ethylanthracene	5.357	5.266	2.24	0.0194	4.003	739	-
C2-phenanthrene/anthracenes	5.46	5.367	2.24	0.01551	3.199	746	-
9,10-dimethylanthracene	5.494	5.401	2.24	0.0144	2.971	748	14071
3,6-dimethylphenanthrene	5.515	5.422	2.24	0.01376	2.838	749	-
C3-fluorenes	5.7	5.603	2.24	0.009199	1.916	769	-
C1-pyrene/fluoranthenes	5.287	5.197	2.24	0.0226	4.887	770	-
2,3-benzofluorene	5.539	5.445	2.24	0.01306	2.824	787	558

Table 3-4. Continued

PAH[B]	SPARC[C] $log_{10}K_{ow}$	$log_{10}K_{oc}$	FCV_i (µmol/g octanol)	PAH specific FCV_i (µmol/L)	PAH specific FCV_i (µg/L)	$C_{OC,PAHi,FCVi}$ (µg/goc)	$C_{OC,PAHi,MAXi}$[D] (µg/goc)
benzo(a)fluorene	5.539	5.445	2.24	0.01306	2.824	787	12500
C3-phenanthrene/anthracenes	5.92	5.82	2.24	0.0057	1.256	829	-
naphthacene	5.633	5.538	2.24	0.01064	2.43	838	207
benz(a)anthracene	5.673	5.577	2.24	0.009756	2.227	841	4153
chrysene	5.713	5.616	2.24	0.008943	2.042	844	826
triphenylene	5.752	5.654	2.24	0.008215	1.875	846	19400
C4-phenanthrenes/anthracenes	6.32	6.213	2.24	0.002387	0.5594	913	-
C1-benzanthracene/chrysenes	6.14	6.036	2.24	0.003531	0.8557	929	-
C3-pyrene/fluoranthenes	6.284	6.177	2.24	0.002581	0.6307	949	-
benzo(a)pyrene	6.107	6.003	2.24	0.003794	0.9573	965	3840
perylene	6.135	6.031	2.24	0.00357	0.9008	967	431
benzo(e)pyrene	6.135	6.031	2.24	0.00357	0.9008	967	4300
benzo(b)fluoranthene	6.266	6.16	2.24	0.002685	0.6774	979	2169
benzo(j)fluoranthene	6.291	6.184	2.24	0.002542	0.6415	981	3820
benzo(k)fluoranthene	6.291	6.184	2.24	0.002542	0.6415	981	1220
C2-benzanthracene/chrysenes	6.429	6.32	2.24	0.001883	0.4827	1008	-
9,10-dimethylbenz(a)anthracene	6.567	6.456	2.24	0.001395	0.3575	1021	124200
7,12-dimethylbenz(a)anthracene	6.575	6.464	2.24	0.00137	0.3513	1021	145300
7-methylbenzo(a)pyrene	6.537	6.426	2.24	0.001489	0.3965	1058	-
benzo(ghi)perylene	6.507	6.397	2.24	0.001589	0.4391	1095	648
C3-benzanthracene/chrysenes	6.94	6.822	2.24	0.0006194	0.1675	1112	-
indeno(1,2,3-cd)pyrene	6.722	6.608	2.24	0.0009953	0.275	1115	-
dibenz(a,h)anthracene	6.713	6.599	2.24	0.001015	0.2825	1123	2389
dibenz(a,j)anthracene	6.713	6.599	2.24	0.001015	0.2825	1123	47680
dibenz(a,c)anthracene	6.78	6.665	2.24	0.0008773	0.2442	1129	7400
C4-benzanthracene/chrysenes	7.36	7.235	2.24	0.0002483	0.07062	1214	-
C1-dibenz(a,h)anthracenes	7.113	6.992	2.24	0.0004251	0.1243	1221	-
coronene	6.885	6.768	2.24	0.0006981	0.2097	1230	821
C2-dibenz(a,h)anthracenes	7.513	7.386	2.24	0.000178	0.05454	1325	-
C3-dibenz(a,h)anthracenes	7.913	7.779	2.24	0.0000746	0.02389	1435	-

[A] Four significant figures are used even when fewer are appropriate for the parameter to limit the effects of rounding error when calculating $\Sigma ESBTU_{FCV}$ which has two significant figures.

[B] See Appendix E for solubilities.

[C] For C#-PAHs, reported $log_{10}K_{OW}$ values are the average $log_{10}K_{OW}$ values of all structures.

[D] $C_{OC,PAHi,Maxi}$ is based on solubility; if $C_{OC,PAHi,FCVi}$ is > $C_{OC,PAHi,Maxi}$, then $C_{OC,PAHi,Maxi}$ may be used to calculate ESB toxic units (see Section 6).

Section 4

Derivation of PAH ESB_{FCVS}

4.1 Derivation of Potencies for Individual PAHs in Sediments ($C_{OC,PAHi,FCVi}$)

The critical concentration of a PAH in sediment ($C_{OC,PAHi,FCVi}$) that is related to the FCV is derived following the EqP method (U.S. EPA, 2003a; Di Toro et al., 1991) because the interstitial water-sediment partitioning of PAHs follows that of other nonionic organic chemicals. Therefore, a sediment effects concentration for any measure of effect can be derived from the product of the water-only effects concentration for that effect and the K_{OC} for that particular PAH. The use of K_{OC} to derive a sediment effects concentration for PAHs is applicable because partitioning for these chemicals is primarily determined by the organic carbon concentration of the sediment.

The partitioning equation between the organic carbon-normalized sediment concentration, C_{OC} ($\mu mol/g_{OC} = \mu mol/kg_{OC}$), and the free interstitial water concentration, C_d (mmol/L), is given by the equation

$$C_{OC} = K_{OC}\, C_d \qquad (4\text{-}1)$$

where K_{OC} (L/kg_{OC}), defined above, can be calculated from a K_{OW} obtained from SPARC (Hilal et al., 1994) using the following equation from Di Toro (1985)

$$\log_{10}K_{OC} = 0.00028 + 0.983 \log_{10}K_{OW} \qquad (4\text{-}2)$$

$C_{OC,PAHi,FCVi}$ for individual PAHs are then calculated using Equation 4-1 with the FCV as the water concentration

$$C_{OC,PAHi,FCVi} = K_{OC}\, FCV_i \qquad (4\text{-}3)$$

Since K_{OC} is presumed to be independent of sediment type for nonionic organic chemicals, so also is $C_{OC,PAHi,FCVi}$.

Table 3-4 contains the $C_{OC,PAHi,FCVi}$ ($\mu g/g_{OC}$) for 74 PAHs found in sediments, including the 34 PAHs (in bold) analyzed by the U.S. EPA in their EMAP program (U.S. EPA, 1996a,b; 1998). $C_{OC,PAHi,FCVi}$ values for PAHs not in Table 3-4 can be calculated in a similar manner (see Section 7.2 for discussion on the PAHs to which the ESB applies). The range in the $C_{OC,PAHi,FCVi}$ values for the 74 PAHs listed in Table 3-4, which were derived using only data for PAHs, is from 349 to 1435 $\mu g/g_{OC}$. In contrast, the range of the same value, termed the $C_{S,OC}$ by Di Toro and McGrath (2000), was about the same (655 to 1940 $\mu g/g_{OC}$) for the 23 PAHs commonly measured when derived using the database for narcotic chemicals with a PAH correction.

4.2 Derivation of the ESB_{FCV} for PAH Mixtures

The correct derivation of the ESB for a mixture of PAHs is based on the approximate additivity of narcotic chemicals in water and tissue (Di Toro et al., 2000; Section 2.8 of this document) and in sediment (Section 5.2). Because WQC and ESBs are based on FCVs they are not intended to cause toxicity in water or sediments to most species, the term toxic unit could be misleading. Therefore, we refer to the quotient of the concentration of a specific chemical in water and its WQC FCV as water quality criteria toxic units ($WQCTU_{FCVi}$). Similarly, the quotient of the sediment concentration for a specific PAH ($C_{OC,PAHi}$) and the $C_{OC,PAHi,FCVi}$ in sediments should be termed equilibrium partitioning sediment benchmark toxic unit ($ESBTU_{FCVi}$). Thus, the ESB for the mixture of PAHs is the sum of the $ESBTU_{FCVi}$ for all of the PAHs in the particular sediment termed the $\Sigma ESBTU_{FCV}$

$$\Sigma ESBTU_{FCV} = \sum_{i} \frac{C_{OC,PAHi,}}{C_{OC,PAHi,FCVi}} \qquad (4\text{-}4)$$

For a particular sediment, if the $\Sigma ESBTU_{FCV}$ for "total PAHs" is less than or equal to 1.0, the concentration of the mixture of PAHs in the sediment is acceptable for the protection of benthic organisms (see Section 7.2 for the technical basis for defining total PAH as the $\Sigma ESBTU_{FCV}$ for the 34 PAHs monitored in the U.S. EPA EMAP). The equilibrium partitioning sediment benchmark is given by the equation

$$ESB = \Sigma ESBTU_{FCV} \leq 1.0 \quad (4\text{-}5)$$

For a particular sediment, if the $EESBTU_{FCV}$ is > 1.0, the concentration of the mixture of PAHs in the sediment may not acceptable for the protection of benthic organisms

$$ESB = \Sigma ESBTU_{FCV} > 1.0 \quad (4\text{-}6)$$

4.3 Aqueous Solubility Constraint

A solubility constraint is applied to sediment concentrations when computing their individual contributions to the effect of the PAH mixture because the $C_{OC,PAHi,FCVi}$ derived for each PAH is solubility limited, i.e., the interstitial water concentration of the PAH is limited by the solubility S. Therefore, $C_{OC,PAHi,FCVi}$ is limited by the concentration in sediment organic carbon that is in equilibrium with the interstitial water at the aqueous solubility (Equation 4-7). This is termed the maximum $C_{OC,PAHi,Max}$ (Table 3-4)

$$C_{OC,PAHi,FCVi} \leq C_{OC,PAHi,Maxi} = K_{OC}S \quad (4\text{-}7)$$

Thus, only the contribution up to the maximum $C_{OC,PAHi,Maxi}$ is counted in the $\Sigma ESBTU_{FCV}$ for the PAH mixture.

Narcosis theory suggests that highly insoluble PAHs should contribute fractional toxic units and $ESBTU_{FCVi}$, limited by the solubility constraint, to the sum of the effects of the mixture when these PAHs are present in mixtures. If so, then this points out the importance of knowing the aqueous solubility of these PAHS so that Equations 4-4 and 4-5 can be applied correctly.

The question of whether highly insoluble chemicals that are not by themselves acutely or chronically toxic, e.g., high molecular weight PAHs, contribute fractional toxic units to the total toxicity when present as mixtures is discussed in Section 5.2.8 of this document and in Spehar et al. (*In preparation*). Spehar et al. (*In preparation*) demonstrate that high K_{OW} PAHs do contribute to the total toxicity of the PAH mixture.

4.4 Comparison of the $\Sigma ESBTU_{FCV}$ for Mixtures of PAHS in Estuarine Sediments

Coastal and estuarine monitoring data were compiled from eight sources to obtain a preliminary assessment of the $\Sigma ESBTU_{FCV}$ values for PAHs in the sediments of the Nation's water bodies (NOAA, 1991; Adams et al., 1996; Anderson et al., 1996; Fairey et al., 1996; U.S. EPA, 1996a,b, 1998; Hunt et al., 1998). Data sources which were identified had measured concentrations for the 23 PAHs (18 parent and 5 alkylated groups) (see Table 6-2) as well as the corresponding sediment organic carbon measurements. Sediments analyzed were from randomly selected and specifically targeted locations, samples of surficial grabs and vertical profiles, and studies where the relative frequency and intensities of sampling varied. This analysis is presented as an aid in assessing the range of reported PAH concentrations, and the extent to which they may exceed 1.0 $\Sigma ESBTU_{FCV}$. The sediments analyzed were not randomly selected from the entire United States. Therefore, this analysis is not intended to reflect expected occurrence nationwide or at any specific site of concern. Sediments where 23 PAHs were analyzed will underestimate the $\Sigma ESBTU_{FCV}$ if 34 PAHs had been analyzed. $\Sigma ESBTU_{FCV}$ values were computed by summing the $ESBTU_{FCVi}$ for each PAH measured in the sediment sample. For insoluble PAHs, the $C_{OC,PAHi,Maxi}$ (Table 3-4) was used to calculate $\Sigma ESBTU_{FCV}$.

The probability distribution for the $\Sigma ESBTU_{FCV}$ data are shown on Figure 4-1. The number of data points used to generate each distribution is provided in the lower right hand corner of each graph. For visual effect, only non-

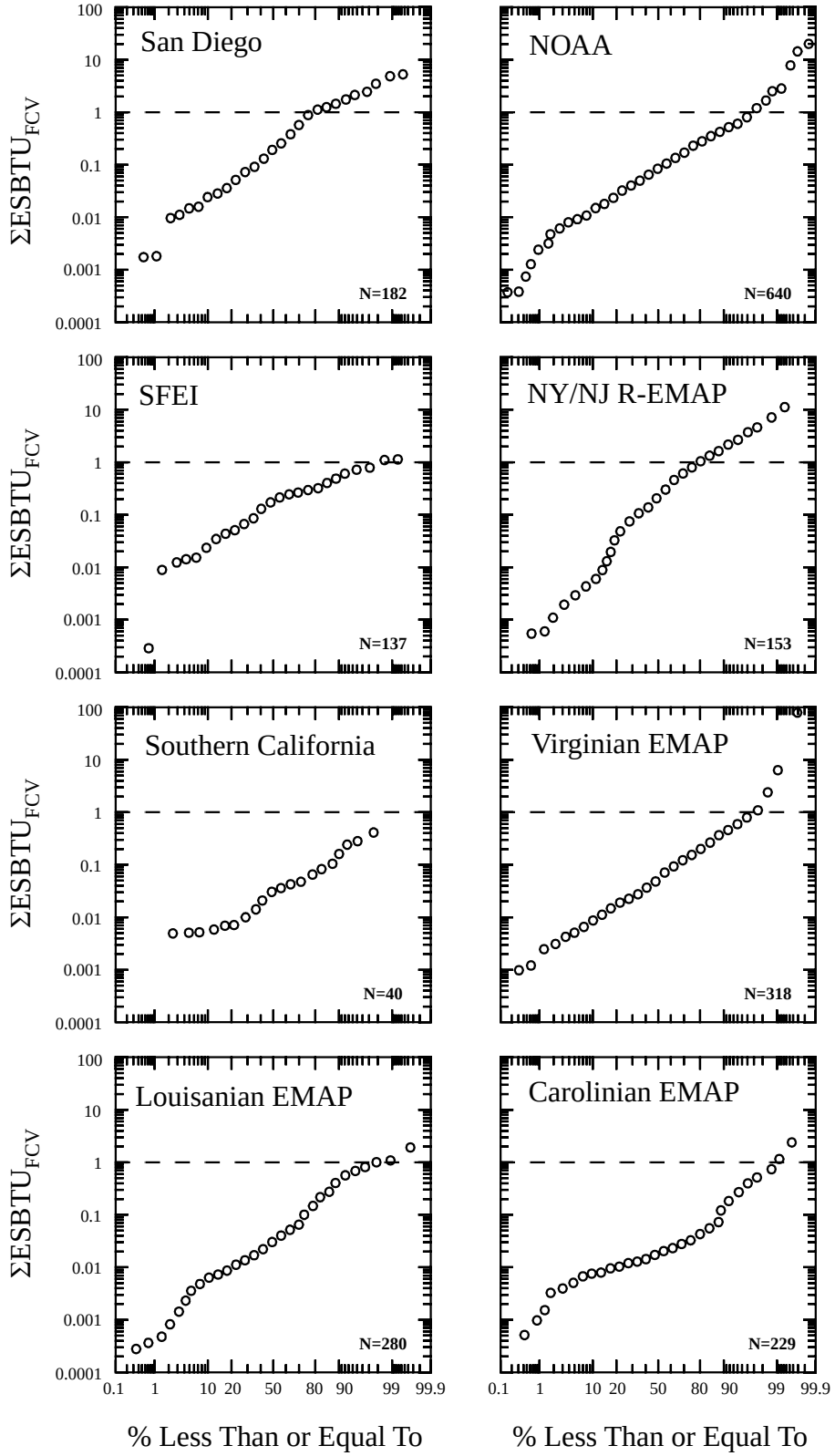

Figure 4-1. Probability distribution of the $\Sigma ESBTU_{FCV}$ for PAH mixtures in sediments from individual coastal and estuarine locations in the United States.

overlapping data are shown. For comparison purposes, a line indicating 1.0 $\Sigma ESBTU_{FCV}$ is also shown. Data presented are from sediments with 0.201 to 15.2% organic carbon. With the exception of the Louisianian and Carolinian Province EMAP datasets, all of the datasets had only 23 PAHs measured. The Louisianian and Carolinian Province EMAP datasets had a total of 34 measured PAHs (18 parent and 16 alkylated groups). The PAHs in addition to the 23 were the C1 through C4 alklyated forms of some of the parent PAHs. To assess the total number of PAHs, a C1-PAH series was considered as one PAH. Computed $\Sigma ESBTU_{FCV}$ values are based on the total number of PAHs measured. The distributions across the different locations are relatively similar. With the exception of the Southern Californian data, all of the datasets had $\Sigma ESBTU_{FCV}$ values greater than 1.0 at the 95th percentile. Although the $\Sigma ESBTU_{FCV}$ from the Lousianian and Carolinian Province EMAP data are computed from 34 PAHs, these sediments do not contain greater $\Sigma ESBTU_{FCV}$ values than sediments from the other studies which measured only 23 PAHs.

A single probability distribution using all of the data is shown in Figure 4-2. The total number of sediments is 1979. $\Sigma ESBTU_{FCV}$ values computed from 23 PAHs are denoted by open circles, and for the 34 PAHs, by open squares. The median $\Sigma ESBTU_{FCV}$ was about 0.06. Approximately 6% of the samples (109 sediments) had $\Sigma ESBTU_{FCV}$ values greater than 1.0.

Although the EqP-based ESBs for nonionic organic chemicals are not intended for use with largely sandy sediments having <0.2% TOC, the EMAP Lousianian and Carolinian Provinces (34 PAHs) and the Elliot Bay (31 PAHs) monitoring databases were examined to determine the frequency of ESB exceedences. A total of 115 of the 654 sediments in these databases had <0.2% TOC. Only two of these sediments (1.7 percent) exceeded the ESB of >1.0 $\Sigma ESBTU_{FCV}$.

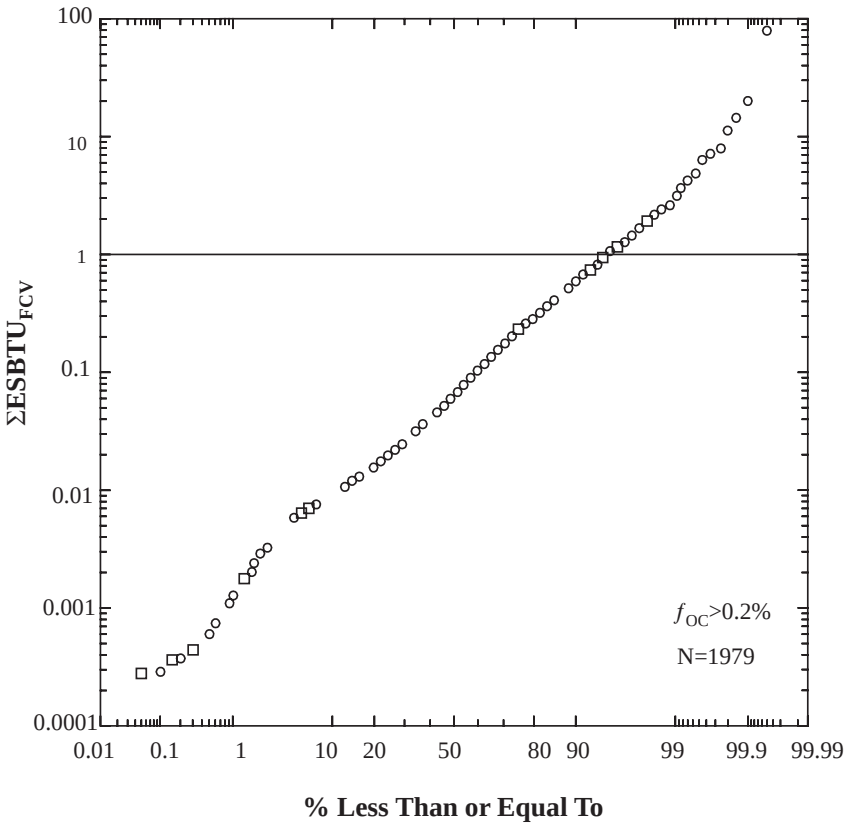

Figure 4-2. Probability distribution of the $\Sigma ESBTU_{FCV}$ for PAH mixtures in sediments from all of the coastal and estuarine locations in the United States from Figure 4-1.

Section 5

Actual and Predicted Toxicity of PAH Mixtures in Sediment Exposures

5.1 Introduction

The $C_{OC,PAHi,FCVi}$ for individual PAHs and ESBs for their mixtures were derived using water-only toxicity data (Appendix C) and both equilibrium partitioning (U.S. EPA, 2003a; Di Toro et al., 1991) and narcosis theory (Di Toro et al., 2000; Di Toro and McGrath, 2000). This section examines data from toxicity tests with spiked and field sediments contaminated with individual PAHs and their mixtures to demonstrate the strength of the technical approach used to derive ESBs and the applicability of ESBs to sediments from the field.

5.2 Spiked Sediment Toxicity Tests

5.2.1 Interstitial Water Concentrations and Sediment Toxicity: Relevance to Water-Only Toxicity Tests and WQC FCVs

The key hypothesis in the derivation of ESBs from EqP and narcosis theory is that effects concentrations from water-only aquatic toxicity tests data using benthic species are similar to effects concentrations in sediment toxicity tests based on interstitial water concentrations or sediment concentrations predicted to be toxic using EqP. This hypothesis has been tested in two ways: 1) by comparing LC50 values determined in water-only experiments to interstitial water LC50 values determined in spiked-sediment exposures, and 2) by comparing organic carbon-normalized sediment LC50 values observed in spiked-sediment exposures with those predicted from water-only LC50 values multiplied by the K_{OC} using the equilibrium partitioning model (Di Toro et al, 1991).

The interstitial water and water-only LC50 values for 28 experiments with a variety of PAHs and several freshwater and marine species are listed in Appendix F (Swartz et al., 1990; Swartz, 1991a; DeWitt et al., 1992; Suedel et al., 1993; Driscoll et al., 1997a,b, 1998). The mean ratio of the water-only LC50 to interstitial water LC50 from 20 experiments with definitive LC50 values was 1.60, indicating agreement generally within less than a factor of two. Interstitial water LC50 values almost always slightly exceeded water-only LC50 values. Three factors may contribute to that result: 1) some test species, especially epibenthic or tube-dwelling organisms, frequently encounter unspiked, overlying water and, thus, are not exclusively exposed to interstitial water; 2) interstitial water near the sediment surface may be slowly diluted by overlying water because of bioturbation and other transport processes; and 3) chemical analyses of interstitial water may include a portion of the non-bioavailable PAH fraction that is bound to dissolved organic matter. Despite these limitations, the interstitial water and water-only LC50 values are remarkably close, especially for sensitive, free-burrowing, infaunal species like *R. abronius*. These data support the evaluation of the risks of sediment-associated chemicals by comparisons between dissolved concentrations in interstitial water and water concentrations of concern from water-only toxicity tests.

A more comprehensive evaluation of the degree to which the response of benthic organisms can be predicted from contaminant concentrations in interstitial water can be made utilizing organism responses in each treatment from toxicity tests with sediments spiked with various

chemicals, including acenaphthene (Swartz, 1991a), phenanthrene (Swartz, 1991a), fluoranthene (Swartz et al., 1990; DeWitt et al., 1992), endrin (Nebeker et al., 1989; Schuytema et al., 1989), dieldrin (Hoke, 1992), DDT (Nebeker et al., 1989; Schuytema et al., 1989) or kepone (Adams et al., 1985) (Figure 5-1). Interstitial Water Toxic Units (IWTU) are calculated by dividing the concentration of a chemical in the interstitial water (µg/L) of a treatment by the water-only LC50 (µg/L). Theoretically, 50% mortality should occur at 1.0 IWTU. Mortality should be <50% at interstitial water concentrations < 1.0 IWTU, and > 50% at concentrations > 1.0 IWTU. Figure 5-1 presents the percent mortality in individual treatments for each chemical versus the IWTUs. Mortality was generally low at concentrations <1.0 IWTU, and increased sharply at ≥1.0 IWTU as would be expected if interstitial water concentrations account for the bioavailability of nonionic organic chemicals across sediments and water-only LC50 values are surrogates for interstitial water LC50 values.

5.2.2 Sediment Toxicity: Prediction Using Water-Only Toxicity and K_{OC}

The equilibrium partitioning model predicts the organic carbon-normalized sediment PAH concen-tration (PAH_{OC}) as the product of the PAH-specific partition coefficient between organic carbon and water (K_{OC}) and the water-only effect concentration for the PAH in water (example, 10-day LC50 or FCV)(Di Toro et al., 1991).

$$\text{Predicted LC50 } (\mu g/g_{OC}) = \text{water-only LC50 } (\mu g/L) \times K_{OC}\ (L/kg_{OC}) \quad (5\text{-}1)$$

Equation 5-1 was used with the water-only LC50 values in table 5-1 and the K_{OC}s in table 3-4 to predict the sediment LC50s ($\mu g/g_{OC}$) for 22 combinations of a variety of PAHs and test species (Table 5-1). Corresponding LC50 values were also determined for each combination in standard sediment toxicity tests. The mean ratio of observed/predicted LC50 values was 2.07, indicating that Equation 5-1 predicts PAH LC50 values $\mu g/g_{OC}$ in sediment with an accuracy within a factor of two (Table 5-1). This result is essentially equal to the ratio of the interstitial water and water-only LC50 values and may be the result of the same factors listed previously.

As in the case of IWTU, predicted sediment toxic units (PSTU) can be estimated by dividing the measured PAH concentration in sediments from individual treatments of spiked-sediment toxicity tests ($\mu g/g_{OC}$) by the predicted LC50 ($\mu g/g_{OC}$). This standardization allows a compre-hensive analysis of the efficacy of the EqP prediction of a sediment effect concentration from the product of the K_{OC} and water-only effects data for that chemical and duration of exposure. Figure 5-2 combines PSTU-response data for diverse chemicals including acenaphthene (Swartz, 1991a), phenanthrene (Swartz, 1991a), fluoranthene (Swartz et al., 1990; DeWitt et al., 1992), endrin (Nebeker et al., 1989; Schuytema et al., 1989), dieldrin (Hoke, 1992) or kepone (Adams et al., 1985) (Figure 5-2). As with the IWTU plot, 50% mortality should occur at about 1.0 PSTU. Figure 5-2 shows that mortality was generally low at PSTU < 1, increased rapidly at PSTU ≈ l, and was high for most samples with PSTU > 1.

These analyses support the concept that water-only LC50 values and K_{OC}s can be used to predict the sediment concentrations on an organic carbon basis that are toxic to benthic organisms. It seems probable that this EqP prediction of sediment effect concentrations from water-only effect data is applicable to other measures of aquatic toxicity, including WQC final chronic values. Therefore, an FCV for a specific PAH multiplied by its K_{OC} value should be applicable to the derivation of a value analogous to the FCV, but based on a sediment concentration. This concentration is the ESB.

5.2.3 Toxicity of Individual PAHs

Spiked-sediment toxicity tests have provided an important tool for investigating the effects of sediment-associated PAHs and the applicability of the EqP approach for the derivation of sediment benchmark concentrations. The toxicity test

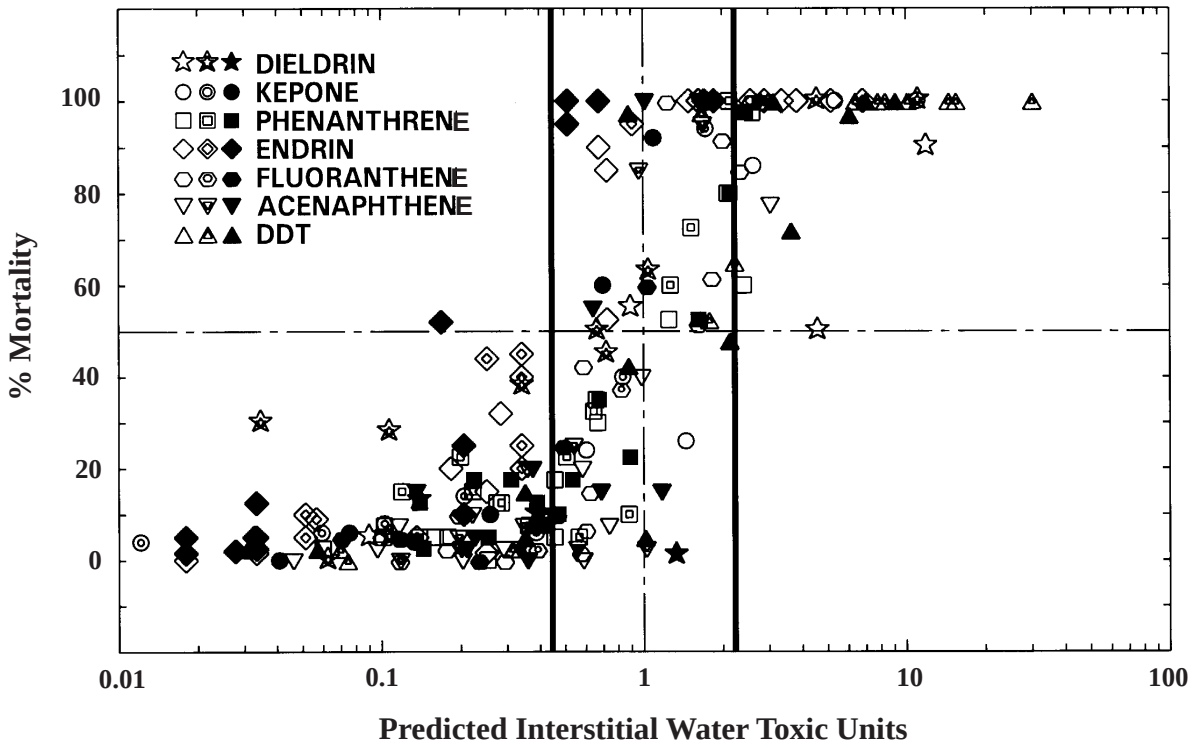

Figure 5-1. Percent mortality versus predicted interstitial water toxic units for seven chemicals and three sediments per chemical (each sediment represented by unique symbol).

method involves: 1) addition and thorough mixing of the PAH into a reference sediment that contains little or no background contamination and is not toxic, by itself, to the test species; 2) storage of the spiked sediment for up to 28d to allow the PAH to reach an equilibrium of the partitioning of the PAH between interstitial water and dissolved and particulate sedimentary materials; 3) conduct of a sediment toxicity test following standard U.S. EPA (1994) or ASTM (1993) procedures; and 4) analytical measurements, typically of the sediment/interstitial water concentration of the PAH, organic carbon, and other sediment variables. The method yields a dataset on the relation between the measured PAH concentration and the toxicity response, from which a LC50, IWTU, PSTU, and other statistical parameters can be calculated.

Sediment contaminant concentrations of nonionic organic chemicals are typically normalized to either the dry weight or organic carbon content of the sediment. To facilitate comparisons among the four PAHs from spiked sediment toxicity tests with *R. abronius*, PAH concentrations in sediments from each treatment in each spiked sediment toxicity test are normalized in this section to the PAH-specific $C_{OC,PAHi,FCVi}$ (see Table 3-4). This ratio is termed the $ESBTU_{FCVi}$, which is the ratio of the measured PAH concentration in sediments from the toxicity tests ($\mu g/g_{OC}$) to the $C_{OC,PAHi,FCVi}$ concentration ($\mu g/g_{OC}$) for that PAH, i.e., the fraction of $ESBTU_{FCV}$ represented by the observed PAH concentration in sediment. The $C_{OC,PAHi,FCVi}$ normalization does not alter the original variability in concentration-response but allows comparison of PAH effects among species, compounds, and response criteria. For example, the $C_{OC,PAHi,FCVi}$-normalized raw data for effects of individual PAHs on the amphipod, *R. abronius*, indicates similar patterns of concentration-response for acenaphthene, phenanthrene, fluoranthene, and pyrene (Figure 5-3). The individual LC50 values for the four PAHs ranged from 3.3 to 4.5 $ESBTU_{FCVi}$ (mean = 3.8)

Table 5-1. Water-only and spiked-sediment LC50 values used to test the applicability of narcosis and equilibrium partitioning theories to the derivation of ESBs for PAHs. See Appendix F for water-only and interstitial water LC50s (µg/L).

Chemical *Test Species*	Method[A]	Ratio: Interstitial Water LC50/Water-only LC50	Organic Carbon-Normalized LC50 ($\mu g/g_{OC}$) Observed	Predicted[B]	LC50 Ratio Obs/Pred	Reference
Freshwater						
Fluoranthene						
Diporeia sp.	FT,M/10	-	-	-	-	Driscoll et al., 1997a,b
Hyalella azteca	FT,M/10	> 0.58	-	-	-	Driscollet al., 1997a,b
Hyalella azteca	S,M/10	1.02[C]	500	4490	0.11[C]	Suedel et al., 1993
Hyalella azteca	S,M/10	5.27[C]	1480	4490	0.33[C]	Suedel et al., 1993
Hyalella azteca	S,M/10	2.17[C]	1250	4490	0.28[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	2.86[C]	1587	3190	0.50[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	7.87[C]	1740	3190	0.55[C]	Suedel et al., 1993
Chironomus tentans	S,M/10	2.37[C]	682	3190	0.21[C]	Suedel et al., 1993
Saltwater						
Acenaphthene						
Eohaustorius estuarius	FT,M/10	2.14	4330	2152	2.01	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.63	1920	2152	0.89	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.45	1630	2152	0.76	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	> 2.54	>23,500	3900	> 6.02	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.08	7730	3900	1.98	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.2	11200	3900	2.87	Swartz, 1991a
Fluoranthene						
Leptocheirus plumulosus	S/10	-	>21,200	3900	>5.44	Driscoll et al., 1998
Phenanthrene						
Eohaustorius estuarius	FT,M/10	1.05	4050	3778	1.07	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.06	3920	3778	1.04	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	1.11	3820	3778	1.01	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	2.09	8200	5335	1.54	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	1.65	6490	5335	1.22	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	1.95	8200	5335	1.54	Swartz, 1991a
2,6-dimethylnaphthlene						
Rhepoxynius abronius	S,M/10	-	8120	-	-	Ozretich et al., 2000a
2,3,5-trimethylnaphthlene						
Rhepoxynius abronius	S,M/10	-	3190	-	-	Ozretich et al., 2000a
1-methylfluorene						
Rhepoxynius abronius	S,M/10	-	1950	-	-	Ozretich et al., 2000a
2-methylphenanthrene						
Rhepoxynius abronius	S,M/10	-	2270	-	-	Ozretich et al., 2000a
9-methylanthracene						
Rhepoxynius abronius	S,M/10	-	6840	-	-	Ozretich et al., 2000a
Acenaphthene						
Rhepoxynius abronius	S,M/10	-	2110	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2310	-	-	Swartz et al., 1997

Table 5-1. Continued

Test Species	Method[A]	Ratio: Interstitial Water LC50/Water-only LC50	Organic Carbon-Normalized LC50 ($\mu g/g_{OC}$) Observed	Predicted[B]	LC50 Ratio Obs/Pred	Reference
Phenanthrene						
Rhepoxynius abronius	S,M/10	-	3080	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2220	-	-	Swartz et al., 1997
Pyrene						
Rhepoxynius abronius	S,M/10	-	1610	-	-	Ozretich et al., 2000a
Rhepoxynius abronius	S,M/10	-	1220	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	2810	-	-	Swartz et al., 1997
Fluoranthene						
Rhepoxynius abronius	S,M/10	-	2320	1390	1.66	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	3310	1390	2.38	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	1.63	1890	1390	1.36	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	2.12	2100	1390	1.51	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	1.74	2230	1390	1.6	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	> 22.66[D]	>4360	1390	4.04[D]	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.01	4410	1390	3.17	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.91	3080	1390	2.22	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	1.38	3150	1390	2.26	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	0.67	2790	1390	2.01	DeWitt et al., 1992
	Mean LC50 ratio =	1.6		Mean LC50 ratio =	2.07	

[A] Test conditions for water-only toxicity tests: S = static, FT = flow-through, M = measured, 10 = 10-d duration.
[B] Predicted LC50 ($\mu g/g_{OC}$) = water-only LC50 ($\mu g/L$) K_{OC} (L/kg_{OC}) 1 $kg_{OC}/1000g_{OC}$.
[C] Sediments spiked with fluoranthene by Suedel et al. (1993) were not at equilibrium, therefore, are not included in the mean.
[D] Source of organic carbon was fresh plant material, not naturally aged organic matter, therefore, value was not included in the mean.
[E] 10-day LC50 value from R. Swartz, Environmental Consultant (personal communication).

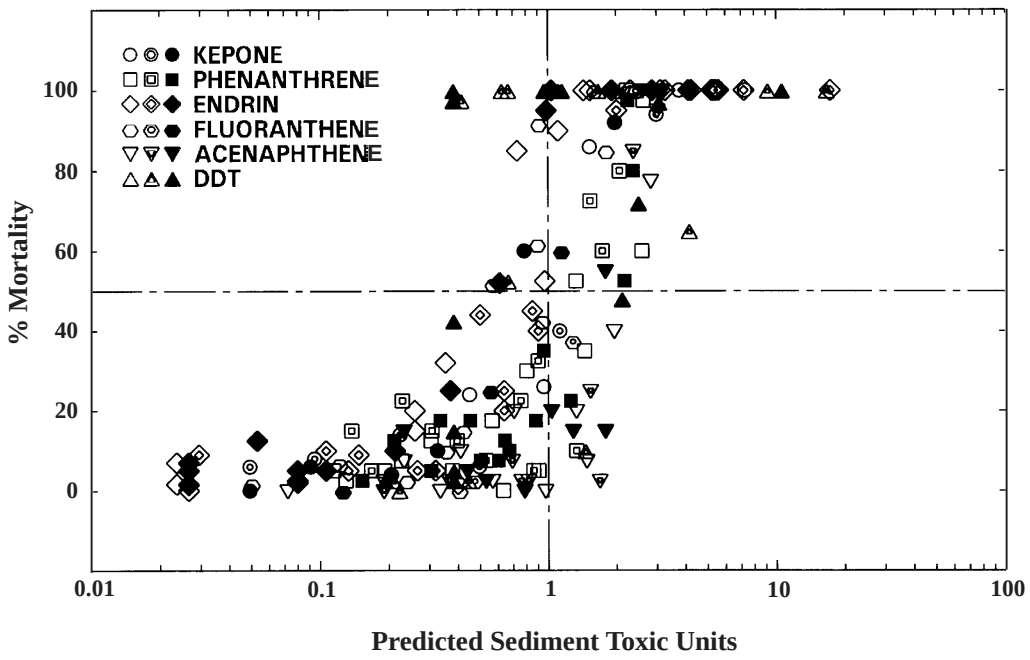

Figure 5-2. Percent mortality versus predicted sediment toxic units for six chemicals and three sediments per chemical (each sediment represented by unique symbol).

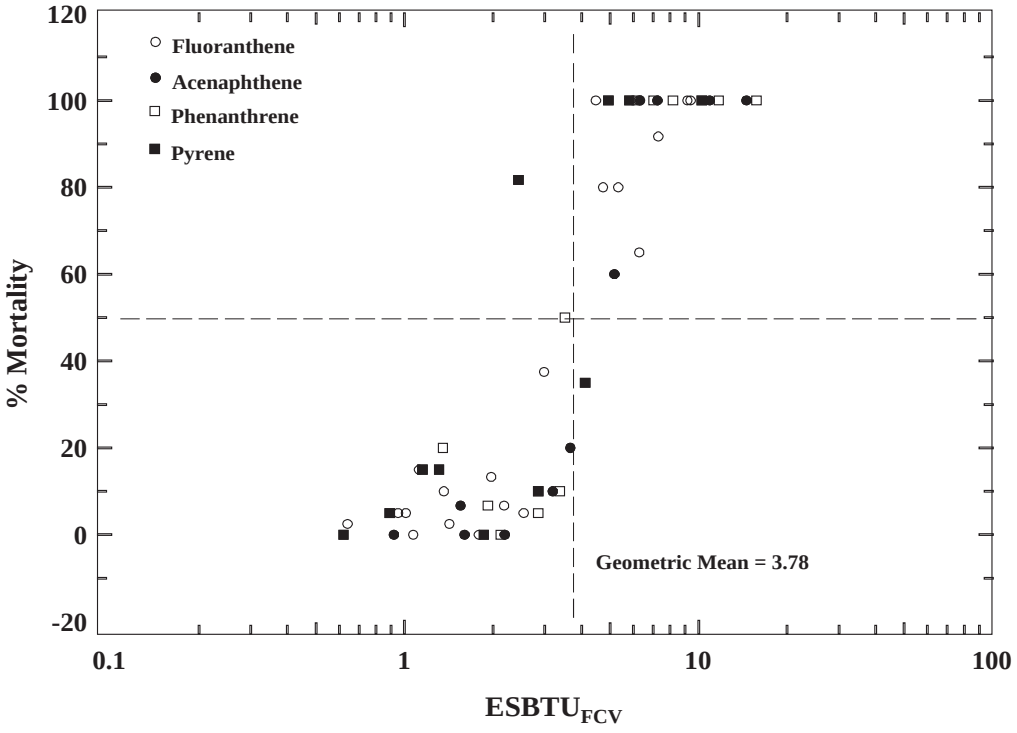

Figure 5-3. Percent mortality of *Rhepoxynius abronius* in sediments spiked with acenaphthene, phenanthrene, fluoranthene, or pyrene concentrations in sediment normalized to ESBTU$_{FCVi}$.

indicating that sediment concentrations would have to exceed the $C_{OC,PAHi,FCVi}$ by about a factor of four to cause 50% mortality in this amphipod during a 10-day exposure. The presence of mortality only at PAH concentrations in excess of the $C_{OC,PAHi,FCVi}$ would be expected.

5.2.4 Comparison of Sediment Toxicity to C_{OC,PAH_i,FCV_i}

The degree to which ESBs derived from narcosis and EqP theory and FCVs derived from water-only toxicity databases are appropriately protective of benthic organisms can be independently tested using data from spiked-sediment toxicity tests. The individual PAH concentrations in sediment (C_{OC}) affecting benthic organisms in toxicity tests were divided by the $C_{OC,PAHi,FCVi}$ to determine the $ESBTU_{FCVi}$. If most benthic organisms are sensitive at the $ESBTU_{FCVi}$ greater than 1.0 then the ESB for the PAH mixture may be appropriately protective of benthic organisms (see Section 4.2).

A review of the literature on spiked-sediment toxicity tests yielded 54 estimates of LC50, EC50 or EC25 (concentration affecting 25% of the test organisms) values for four individual PAHs (acenaphthene, phenanthrene, fluoranthene, pyrene; Appendix F). The duration of most of the tests was 10 days, but a few were longer-term tests that measured sublethal effects on reproduction or emergence (sediment avoidance). Over all the data, there was a substantial range (500 to 147,000 $\mu g/g_{OC}$) in the estimates of the median response concentrations. For example, the relative sensitivity of marine amphipods in this dataset was *Rhepoxynius abronius* > *Eohaustorius estuarius* > *Leptocheirus plumulosus*. This range in median response concentrations reflects differences in species sensitivity, PAH bioavailability and probably, most importantly, specific experimental conditions.

The data from some of the toxicity tests with individual PAHs spiked into sediments needed to be modified or not included in further analyses. Some tests with *Diporeia* sp., *Lumbriculus variegatus*, *Limnodrilus hoffmeisteri* and *Hyalella azteca* were conducted at concentra-tions in the sediment that could not have been at equilibrium with the concentration of the PAH at solubility in interstitial water (Kukkonen and Landrum, 1994; Landrum et al., 1994; Lotufo and Fleeger, 1996; Driscoll et al., 1997a,b). The reported median effect concentration is in parenthesis and maximum sediment concentration at water solubility (given in Table 3-4) for each PAH is indicated in bold in Appendix D. To facilitate comparisons of species sensitivity and to account for bioavailability, median response concentrations were divided by the $C_{OC,PAHi,FCVi}$ values to obtain the test-specific $ESBTU_{FCVi}$ values. Then PAH-specific SMAVs and GMAVS across PAHs were calculated only for 10-day lethality tests. The maximum solubility-limited sediment concentration was used to calculate the test-specific $ESBTU_{FCVi}$ and PAH-specific SMAVs and GMAVs only if there insufficient no data from tests that lacked this solubility constraint. Some tests were conducted with newly spiked sediments where time was likely insufficient to permit equilibrium to be achieved between the interstitial water and organic carbon and other sediment partitioning phases (Suedel et al., 1993). Data from these tests were not used because the median effect concentration in sediments would be lower than that expected if sediments and interstitial water were at equilibrium.

For the seven species tested acceptably against one or more PAH, the 43 test-specific $ESBTU_{FCVi}$ ranged from 1.47 to 57.8, a factor of 39.3, with no values below 1.0 $ESBTU_{FCVi}$ (Figure 5-4; Appendix D). Within each individual species, the range of test-specific $ESBTU_{FCVi}$ across multiple tests with one or more PAH, based on 10-day LC50 values, was within only a factor of 1.5 to 4.1 (mean 3.0). For the three saltwater amphipods tested against multiple PAHs the range of PAH-specific SMAVs was within a factor of 1.4 to 2.0 (mean 1.7). These observations indicate that the species tested differed in their sensitivities to PAHs, but that within a species there was a similarity of response across tests with the same or multiple PAHs. The range and frequency

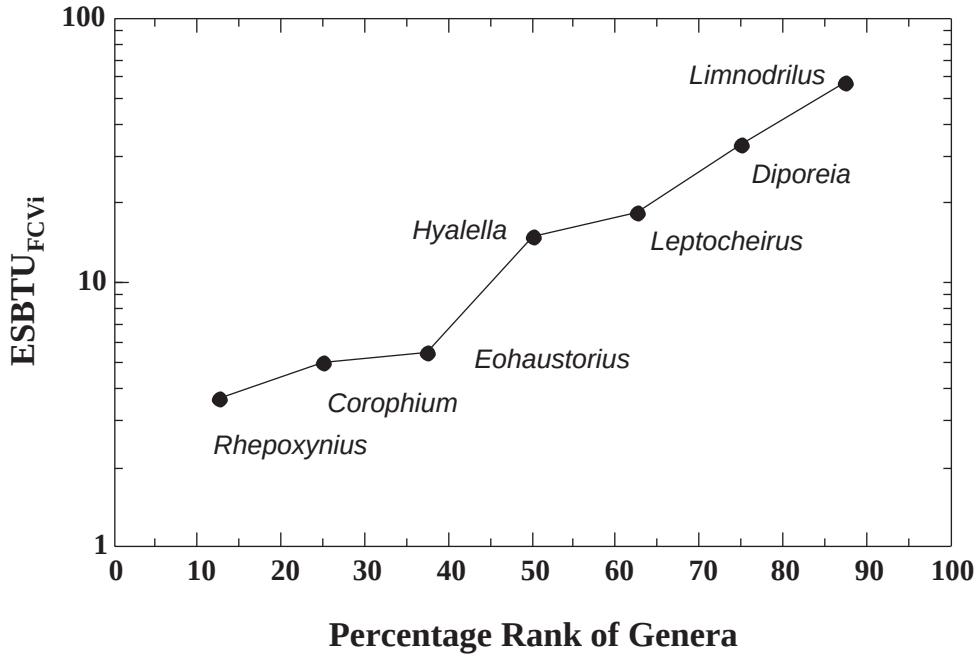

Figure 5-4. Percentage rank, based on ESBTU$_{FCVi}$, of the sensitivities of genera of benthic organisms from spiked sediment toxicity tests.

distribution of contaminant sensitivity among aquatic species is comparable to that of benthic species in water-only tests (see Section 3.4).

This analysis of data from spiked-sediment toxicity tests with individual PAHs supports the conclusion that the $C_{OC,PAHi,FCVi}$ derived from water-only toxicity tests, narcosis theory and national WQC are appropriately protective of benthic organisms. These comparisons between sediments spiked with individual PAHs and their respective $C_{OC,PAHi,FCVi}$ have value in suggesting the validity of the EqP and narcosis approaches. However, PAHs occur in nature not as individual compounds, but as mixtures.

5.2.5 PAH Mixtures

Sediments spiked with PAH mixtures have been used to resolve two issues that are relevant to the validation of the ESB for PAHs (Swartz et al., 1997; Landrum et al., 1991; Boese et al., 1999; Burgess et al., 2000b; Spehar et al., In preparation). The first concerns the toxicological additivity of the effects of the individual components of the mixture. If effects are additive, relatively simple models can be used to predict the effects of mixtures. The second issue concerns the low solubility of PAHs with high octanol-water partitioning coefficients (i.e., PAHs with $K_{OW} > 5.5$). The predicted LC50 of many high K_{OW} compounds exceeds their solubility limit. Accordingly, experimental attempts to establish the LC50 for individual high K_{OW} PAHs spiked into sediment have observed little or no acute or chronic toxicity. High K_{OW} PAH mixtures have been recently tested to see if individual high K_{OW} PAHs contribute fractional toxic units that are additive with effects of other PAHs (Spehar et al., *In preparation*).

5.2.6 Additivity of PAH Mixtures

There is a wealth of aquatic toxicological data that supports the additivity of PAHs and other narcotic chemicals in water (Konemann, 1980;

Hermens et al., 1984; Broderius and Kahl, 1985; Fig. 2-11). The additivity of sediment-associated contaminants is less well documented, although several publications indicate that PAHs in sediment are either additive or slightly less than additive (Swartz et al., 1995, 1997; Landrum et al., 1991, 1994). Landrum et al. (1991) found that the effects of a mixture of 11 sediment-associated PAHs on the freshwater amphipod, *Diporeia* sp. were "approximately additive with no overt evidence of synergism or antagonism." Landrum et al. (1991) also noted that additivity is further supported by the fact that LD50 values, expressed as PAH molar concentration in amphipod tissue, were the same for a single compound (pyrene) and the mixture of 11 compounds.

The results from some of the above 10-day studies were analyzed by dividing the concentrations of each of the PAHs in the sediments by the $C_{OC,PAHi,FCVi}$ and summing the quotients to derive the $\Sigma ESBTU_{FCV}$ for the mixture (Table 5-2). No acute toxicity was observed with *Diporeia* exposed to a $\Sigma ESBTU_{FCV}$ for all PAHs up to 3.08 (Landrum et al., 1991), but none would be expected given the 10-day LC50 value of >34.0 $\Sigma ESBTU_{FCV}$ for this species (Table 5-2). Toxicity to *R. abronius* was absent in several tests with mixtures of PAHs in treatments from 1.42 to 27.8 $\Sigma ESBTU_{FCV}$ and occurred in treatments with 5.80 and 10.3 $\Sigma ESBTU_{FCV}$ (Swartz et al., 1997; Boese et al., 1999). For *R. abronius*, the GMAV from 10-day spiked sediment tests with individual PAHs was 3.67 $\Sigma ESBTU_{FCV}$ (Table 5-2). This suggests a less than additive toxicity of the PAH mixtures tested. The amphipod *A. abdita* was exposed to a total of 2.58 and 6.05 $\Sigma ESBTU_{FCV}$ by Burgess et al. (2000b). Toxicity was absent from both treatments, and none probably should have been expected given the 4-day LC50 at 13.8 $\Sigma ESBTU_{FCV}$ (Table 5-2).

Additivity of mixtures of 13 PAHs was assumed in the development of the ΣPAH model that was used to accurately classify PAH-contaminated, field-collected sediment as toxic or not toxic (Swartz et al., 1995). Swartz et al. (1997) concluded that sediment spiked with a mixture of acenaphthene, phenanthrene, fluoranthene and pyrene caused effects on *R. abronius* that were slightly less than additive. Di Toro and McGrath (2000) reanalyzed these data and concluded that the mixture was additive (also see Section 5.2.7). Even if PAH interactions are slightly less than additive, the potential error introduced by the assumption of additivity in the derivation of an ESB for PAH mixtures would be relatively small and would be environmentally protective (i.e., the toxicity of mixtures would be slightly over-estimated).

5.2.7 PAH Additivity Demonstrated Using the Universal Narcosis Slope

The additivity of mixtures of PAHs spiked into sediments was tested using narcosis theory to calculate PAH-specific 10-day LC50 values in sediments for *R. abronius*. The experimental data from Swartz et al., (1997) was reexamined using predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. The narcosis methodology was used to test additivity, rather than the actual sediment LC50 values as was presented above and by Swartz et al., (1997). This is because the predicted sediment LC50 values were derived using data from many tests with a variety of PAHs. Also, because sediment LC50 values could be predicted for the 31 or 34 PAHs analyzed from field sediments used in 10-day toxicity tests with data from toxicity tests with *R. abronius* to test narcosis and EqP predictions (See Section 5.3.1).

Interstitial water concentrations were used in place of water-only LC50 values in this process because water-only toxicity data were not available. This is justified because interstitial water and water-only LC50 values have been shown to be nearly the same (see Section 5.2.1). The 10-day interstitial water LC50 values were for eight PAHs (fluoranthene, naphthalene, pyrene, 1-methylfluorene, 2-methylphenanthrene, 9-methylanthracene, 2,6-dimethylnaphthlene, and 2,3,5-trimethylnaphthlene) tested in separate experiments. The interstitial water LC50 values for fluoranthene were from seven separate experiments (mean LC50 = 19.5 µg/L) (Swartz et al., 1990; DeWitt et al., 1992), whereas the LC50

Table 5-2. Percent mortality of benthic invertebrates in relation to the ΣESBTU$_{FCV}$ values of mixtures of polycyclic aromatic hydrocarbons spiked into sediment.

Species[A]	ΣESBTU$_{FCV}$ PAH K_{ow} <5.5	ΣESBTU$_{FCV}$ PAH K_{ow} >5.5	ΣESBTU$_{FCV}$ All PAHs	Percent Mortality	PAH Mixture[B]	Reference
Diporeia sp.	0.01	0.02	0.03	3	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	0.21	0.36	0.57	10	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	0.49	0.6	1.1	0	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
Diporeia sp.	1.37	1.71	3.08	12	fluor, phen, anthr, flu, pyr, chry, b(b)flu, b(e)pyr, b(a)pyr, pery, b(ghi)pery	Landrum et al., 1991
R. abronius	10.32	0	10.3	100	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	5.8	0	5.8	38	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	5.12	0	5.12	8	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	3.25	0	3.25	11	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	2.5	0	2.5	4	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	1.8	0	1.8	2	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	1.42	0	1.42	3	ace; phen; flu; pyr	Swartz et al., 1997
R. abronius	2.77	0	2.77	5	anthr; flu	Boese et al., 1999
R. abronius	4.91	5.02	9.93	3	b(a)anthr; flu	Boese et al., 1999
R. abronius	5.88	0	5.88	5	2-methylanthr; flu	Boese et al., 1999
R. abronius	5.71	0	5.71	2	9,10-dimethylanth; flu	Boese et al., 1999
R. abronius	2.71	2.23	4.94	3	b(b)flu; flu	Boese et al., 1999
R. abronius	2.06	0.79	2.84	2	chr; flu	Boese et al., 1999
R. abronius	0.63	1.57	2.2	1	3,6-dimethylphen; flu	Boese et al., 1999
R. abronius	1.91	25.89	27.8	4	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chr; 3,6-dimethylphen	Boese et al., 1999
R. abronius	0.58	8.03	8.61	5	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chr; 3,6-dimethylphen	Boese et al., 1999
R. abronius	1.55	8.03	9.58	9	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chry; 3,6-dimethylphen; flu	Boese et al., 1999
R. abronius	0.9	3.4	4.3	0	anthr; b(a)anthr; 2-methylanthr; b(b)flu; chry; 3,6-dimethylphen; flu	Boese et al., 1999
A. abdita	5.41	0.64	6.05	7	9,10-dimethylanthr; chry	Burgess et al., 2000b
A. abdita	0	2.58	2.58	7	b(a)pyr; cor	Burgess et al., 2000b
A. abdita	5.41	3.22	8.63	10	9,10-dimethylanthr; chry; b(a)pyr; cor	Burgess et al., 2000b
A. bahia	5.41	0.64	6.05	3	9,10-dimethylanthr; chry	Burgess et al., 2000b
A. bahia	0	2.58	2.58	7	b(a)pyr; cor	Burgess et al., 2000b
A. bahia	5.41	3.22	8.63	7	9,10-dimethylanthr; chry; b(a)pyr; cor	Burgess et al., 2000b

[A] Test Species: amphipods: *Diporeia* sp., *Rhepoxynius abronius*, *Ampelisca abdita*; mysids: *Americamysis bahia*
[B] PAH Code: ace - acenaphthene; anthr - anthracene; b(a)anthr - benz(a)anthracene; b(a)pry - benzo(a)pyrene; b(ghi)pery - benzo(ghi)perylene; b(b)flu - benzo(b)fluoranthene; chry - chrysene; cor - coronene; 9,10-dimethylanth - 9,10-dimethylanthracene; 3,6-dimethylphen - 3,6dimethylphenanthrene; flu - fluoranthene; fluor-fluorene; 2-methylanthr - 2-methylanthracene; pery - perylene; phen - phenanthrene; pyr - pyrene.

$$\log_{10}\text{PAH-specific LC50}_{R.\ abronius} = -0.945 \log_{10}K_{OW} + \log_{10}(15.8\ \mu\text{mol/g octanol}) \quad (5\text{-}2)$$

The PAH-specific $LC50_{R.\ abronius}$ is used to calculate the PAH-specific sediment LC50 ($\mu g/_{OC}$) for *R. abronius* (equation 5-3).

$$\text{PAH-specific sediment LC50}_{R.\ abronius} = K_{OC} \times \text{PAH-specific LC50}_{R.\ abronius} \quad (5\text{-}3)$$

values for the remaining seven PAHs are from single experiments (Ozretich et al., 1997) (Table 5-1). The individual LC50 values, and mean value for fluoranthene, were normalized to a K_{OW} of 1.0 using the universal narcosis slope (Equation 2-29). The geometric mean of these LC50 values at a K_{OW} of 1.0 is the critical body burden of 15.8 µmol/g octanol (octanol serves as a surrogate for lipid). The critical body burden is used to calculate the PAH-specific 10-day LC50 values (µg/L) for *R. abronius* (Equation 5-2). This equation is analogous to Equation 3.2 which is used to calculate the PAH-specific WQC.

The mortality of *R. abronius* in the standard 10-day sediment tests where the sediments were spiked individually (acenaphthene, fluoranthene, phenanthrene or pyrene (open symbols)), or a mixture of these four PAHs (solid circles), is compared to the predicted sediment toxic units (PSTU) to test the utility of this approach to normalize the toxicity of individual PAHs and, most importantly, to test the additivity of the PAH mixture experiment of Swartz et al. (1997) (Figure 5-5A). PSTUs are the quotients of the concentration of each PAHs measured in the individual spiked sediment treatments divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. For the mixture, PSTUs were summed to obtain the total toxic unit contribution (in Section 5.3.1, sediments from the field are similarly analyzed; Figure 5-5B). The percent mortality-PSTU relationship is similar for the individual PAHs and the mixture. Apparent LC50 values are approximately within a factor of two of 1.0 PSTU. This analysis based on the universal narcosis slope and a similar analysis for narcotic chemicals in water-only experiments (Section 2.10), suggests that the assumption of near additivity of mixtures of PAHs is a reasonable approximation.

5.2.8 Additivity of Mixtures of High K_{OW} PAHs

The solubility of PAHs in water generally decreases with increasing K_{OW}, while the water column toxicity of PAH increases with increasing K_{OW}. Although the solubility of individual PAHs are a function of their structure and polarity rather than just K_{OW}, the general relationship between solubility and K_{OW} is such that solubility decreases with increasing K_{OW} slightly faster than toxicity increases. The net result of this relationship is that PAHs with high K_{OW} (roughly $\log_{10}K_{OW}$ of 5.5 and higher) have solubilities below their predicted LC50. This has led to the conventional wisdom that high K_{OW} PAHs are not toxic (at least on an acute basis) because they are insufficiently soluble to cause toxicity. For example, high K_{OW} PAHs are generally not toxic in water-only toxicity tests (Appendix C).

This argument is founded, however, on the basis of single chemicals. PAHs do not occur as single chemicals in the environment, and available experimental evidence indicates that their toxicities are additive, or slightly less than additive, when present in mixtures. This has special significance for the higher K_{OW} PAHs; although they may be too insoluble to cause toxicity individually, they could still contribute fractional toxic units to the overall toxicity of PAH mixtures.

Historically, toxicity experiments with mixtures have been conducted by testing the toxicity of individual chemicals to determine their potency, then testing mixtures of these chemicals to determine the potency of the mixture. Comparing the toxicity of the mixture to the toxic units contributed by each chemical allows evaluation of the interactive toxicity of the mixture. In the case of high K_{OW} PAHs, this experimental approach cannot be used, because the toxicity of the individual chemicals cannot be measured. Use of the narcosis model, however, allows prediction of toxicity for the mixture components and can be used to evaluate the overall toxicity of the mixture.

Spehar et al. (*In preparation*) conducted a

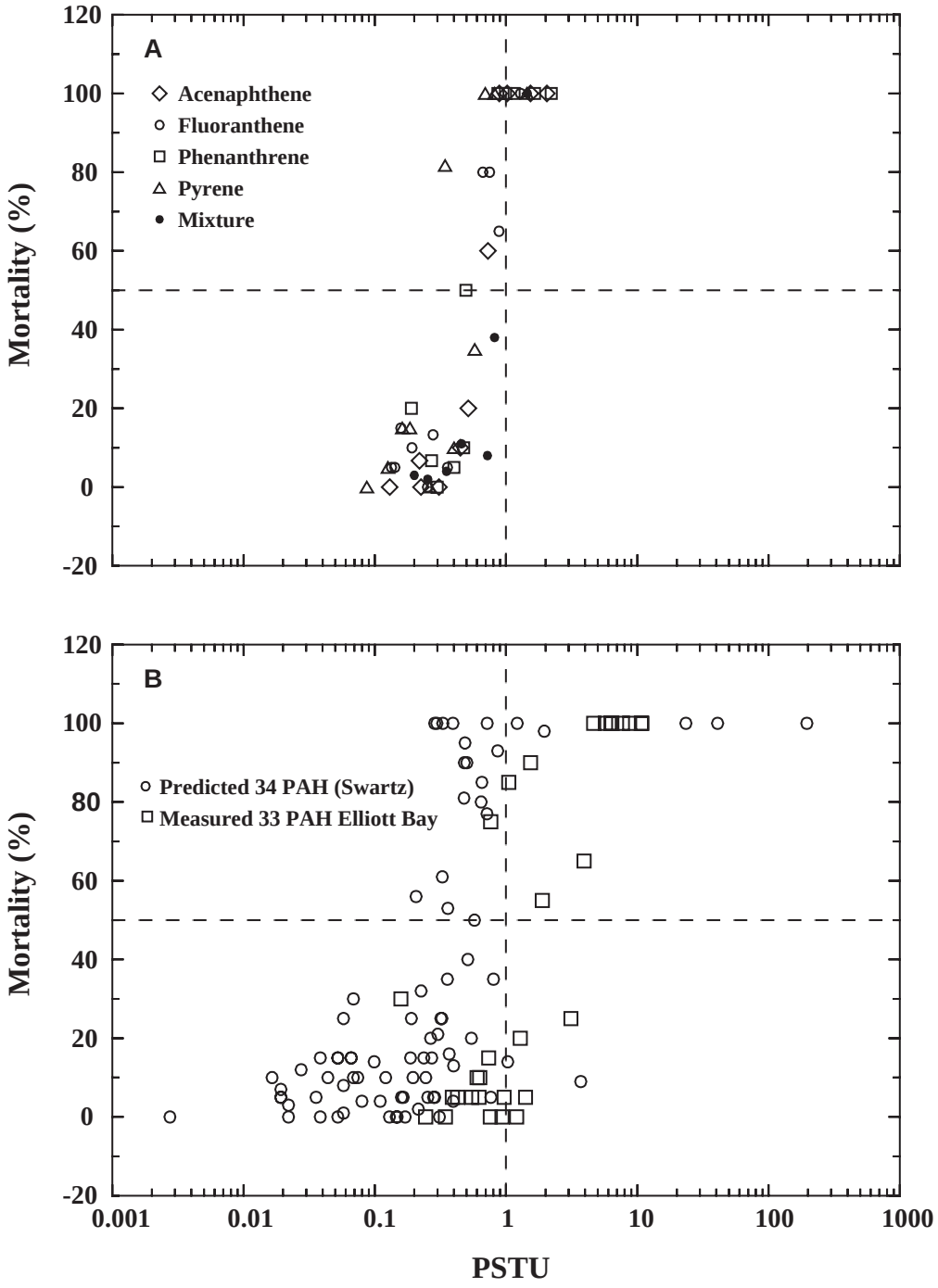

Figure 5-5. Mortality of the amphipod, *Rhepoxynius abronius*, from 10-day spiked sediment toxicity tests with four parent PAHs separately (open symbols) and in combination (closed circles) (A) and in tests with sediments from the field (B) versus predicted sediment toxic units (PSTUs). PSTUs are the quotients of the concentration of each PAH measured in sediments from the individual spiked sediment treatments, or individual sediments from the field, divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius.* The predicted PAH-specific 10-day sediment LC50 values for *R. abronius*is were calculated using the critical body burden of 15.8 Fmol/g octanol and Equation 5-2. PSTUs were summed to obtain the total toxic unit contribution of the mixture of PAHs in spiked or field sediments.

series of sediment toxicity tests using a mixture of 13 PAHs with $\log_{10}K_{OW}$ ranging from 5.36 to 6.76 (Table 5-3). Potency of each chemical was predicted using an earlier version of the narcosis model, and the concentration for each chemical in the highest concentration of the mixture was established at an estimated 0.5 TU for *Hyalella azteca* (re-analysis using current models and the *H. azteca* GMAV from Appendix C predicts more than 0.5 TU for most PAHs). For some of these chemicals, solubility would be expected to limit their TU contribution (Table 5-3). The PAH mixture was spiked into a clean freshwater sediment at several concentrations, and into a clean marine sediment at the highest concentration only.

Several toxicity tests were conducted. A 42-day survival, growth, and reproduction study with *H. azteca* (Spehar et al., *In preparation*) was conducted in a flow-through system (2x daily renewal of overlying water) using four concentrations of the PAH mixture. In this study, chemical analysis of the bulk sediment showed that about 80% of the nominal PAH spike was measured in the sediment at the start of the exposure, and concentrations of PAH in the interstitial water were generally within a factor of 2 of the concentrations predicted from K_{OC} and solubility. After 10 days of exposure, significant effects on the dry weight of the amphipods were observed in the three highest concentrations of the PAH mixture (Figure 5-6), but there were no effects on survival. After 28 days of exposure, survival was significantly reduced in the two highest treatments, although the growth effects observed at day 10 were no longer present (Figure 5-7). As per the test protocol, organisms were removed from the sediment at day 28 and held for 14 more days in clean water to assess reproduction. No further effects on survival, growth, or reproduction were observed between days 28 and 42.

Toxicity of the PAH mixture was lower than would have been predicted based on narcosis

Table 5-3. Chemicals included in the high K_{OW} PAH mixture experiment (Spehar et al., *In preparation*).

Chemical Name	Molecular Weight (g/mol)	$\log_{10}$ K_{OW}[A]	$\log_{10}$ K_{OC}[B]	Estimated Solubility[C] (µg/L)	Nominal Sediment Concentration (µmol/g_{OC})	Estimated porewater concentration (µg/L)	
						Nominal[D] (Sed. Conc./K_{OC})	Limited by Solubility
2-Ethylanthracene	206.29	5.36	5.27	59.62	39.32	43.94	43.94
3,6 Dimethylphenanthrene	206.29	5.52	5.42	77.98	42.38	33.12	33.12
2,3 Benzofluorene	216.28	5.54	5.44	25.30	42.88	33.27	25.30
Benzo(a)anthracene	228.29	5.67	5.58	12.28	45.80	27.70	12.28
Triphenylene	228.3	5.75	5.65	5.11	47.66	24.11	5.11
2-(tert-butyl)anthracene	234.34	5.88	5.78	33.04	50.91	19.78	19.78
Benzo(a)pyrene	252.31	6.11	6.00	2.88	57.46	14.38	2.88
Benzo(b)fluoranthene	252.32	6.27	6.16	8.28	62.75	10.96	8.28
Benzo(k)fluoranthene	252.32	6.29	6.18	8.35	63.64	10.50	8.35
9-Phenylanthracene	254.33	6.31	6.2	3.64	64.22	10.30	3.64
7-Methylbenzo(a)pyrene	266.35	6.54	6.43	1.46	73.37	7.32	1.46
7,12Dimethylbenz(a)anthracen	256.35	6.58	6.46	13.41	75.04	6.62	6.62
3-Methylcholanthrene	268.38	6.76	6.64	3.11	83.92	5.1	3.11
TOTAL PAH					749.4	247.1	173.9

[A] Predicted by SPARC.
[B] Predicted from Di Toro et al. (1991).
[C] Predicted by SPARC in distilled water at 25°C.
[D] Nominal concentration predicted by K_{OC}, regardless of solubility limits; highest concentration only.

theory. However, concentrations of PAH measured in the tissue of exposed *Hyalella* were considerably lower than would be in equilibrium with interstitial water, suggesting that the *Hyalella* may have avoided the test sediment, thereby reducing their exposure. Avoidance of toxic sediments by *Hyalella* has been reported previously (e.g., Whiteman et al., 1996). When 10-day growth and 28-day survival responses are compared on the basis of measured tissue burden, the thresholds for response fall in the same range as is predicted by narcosis theory (Figure 5-8). Thus, although *Hyalella* had lower uptake of these PAHs, they did show a response to the high K_{OW} PAHs suggesting that these chemicals can cause toxicity to benthic organisms. Moreover, the relationship of measured tissue concentrations to biological responses was consistent with that expected from a narcotic mode of action and additivity among PAHs in the mixture. It should be noted that because the toxicity of the individual mixture components was predicted rather than measured (which would not be possible if they are not individually toxic at solubility), we can only conclude that these results are consistent with the additivity, or approximate additivity, hypothesis, but they are not, by themselves, proof of additivity.

Because of concerns that *Hyalella* may have avoided exposure to PAH in the flow-through test by spending more time in the overlying water which was being replaced 2x daily, an additional test was conducted using the same PAH-spiked sediments, but conducting the test with renewal of overlying water-only three times during the entire 10-day test. This reduced frequency of renewal should have increased the concentrations of PAH in the overlying water (not measured), thereby increasing exposure of *Hyalella* to the PAH mixture. While the flow-through test showed effects only on growth after 10 days of exposure, results of the second test showed a concentration-dependent response of both survival and growth (Figure 5-9). When expressed on the basis of total PAH molar concentration in the sediment

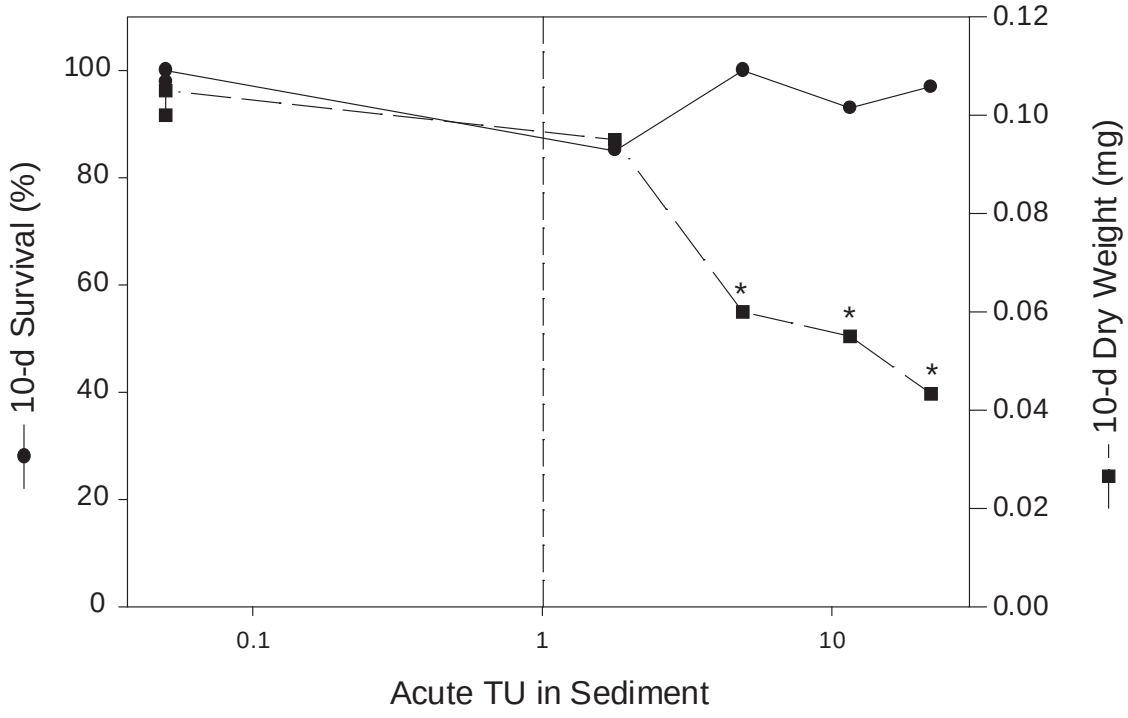

Figure 5-6. Response of *Hyalella azteca* exposed for 10 days under flow-through conditions to sediment spiked with a mixture of high K_{ow} PAH.

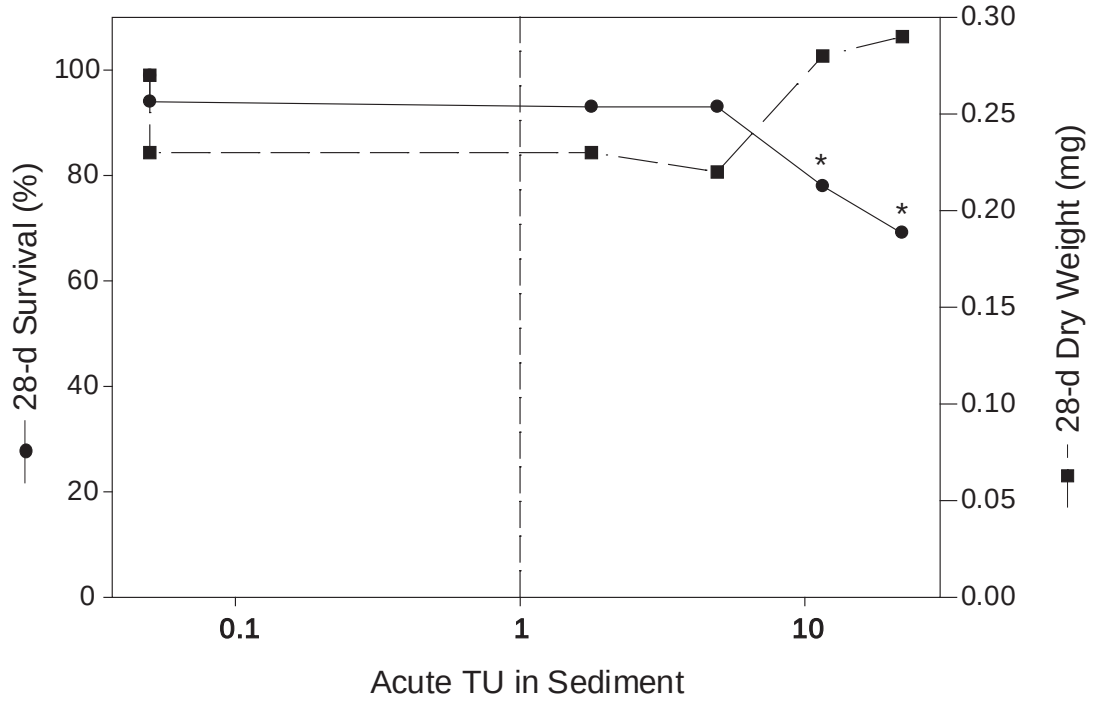

Figure 5-7. Response of *Hyalella azteca* exposed for 28 days under flow-through conditions to sediment spiked with a mixture of high K_{ow} PAH.

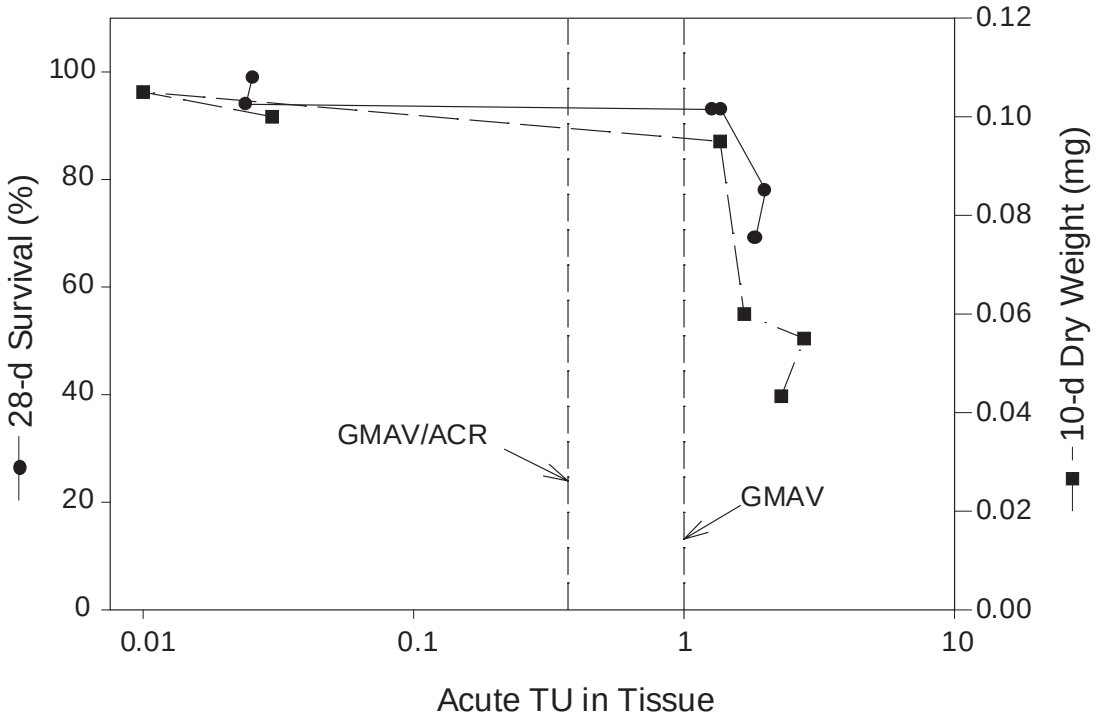

Figure 5-8. Survival (after 28 days) and growth (after 10 days) of *Hyalella azteca* expressed on the basis of measured PAH concentrations in tissues (lipid normalized).

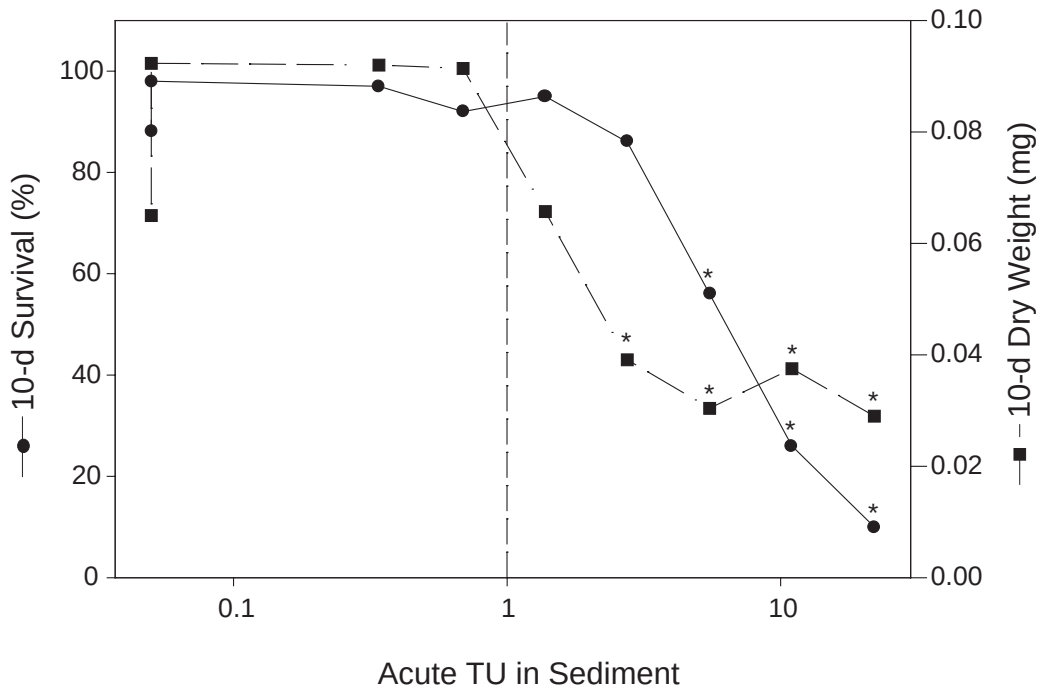

Figure 5-9. Response of *Hyalella azteca* exposed for 10 days (3 renewals) to sediment spiked with a mixture of high K_{ow} PAH.

(normalized to organic carbon), the threshold for survival and growth effects were close to the sediment concentration predicted to cause acute effects based on the narcosis model. Similarly, the tissue concentration of the mixture in the amphipods compared favorably with the critical body burden predicted to cause effects based on the narcosis model.

In addition to the freshwater experiments described above, additional experiments were conducted using marine organisms (Spehar et al., *In preparation*). Two marine organisms, a mysid (*Americamysis bahia*) and a marine amphipod (*Ampelisca abdita*), were exposed to a marine sediment spiked with the highest concentration of the PAH mixture. After 10 days of exposure to the sediment in a static system, both species showed marked mortality, with 85% mortality of mysids and 95% mortality of the amphipods. Because this sediment would be predicted to contain a large number of acute TU based on the GMAVs for these species (41 acute TU for mysids; 10 acute TU for *A. abdita*), these results cannot be used to evaluate accuracy of the narcosis model rigorously; however, they provide further support to emphasize that mixtures of high K_{OW} PAHs can cause toxicity.

In a separate test, another species of marine amphipod (*Leptocheirus plumulosus*) was exposed for 10 days to a series of concentrations of the PAH mixture spiked into the freshwater sediment used in the freshwater studies (*L. plumulosus* is tolerant of the lower salinity in the freshwater sediment, while *A. bahia* and *A. abdita* are not) (Spehar et al., In preparation). After 10 days under static conditions, *L. plumulosus* showed reduced survival in the four highest PAH concentrations (Figure 5-10). The observed toxic unit threshold for mortality was within a factor of 2 of that predicted using narcosis theory and the GMAVs in sediment from Appendix C for *L. plumulosus*.

Taken together, the results of these experiments with high K_{OW} PAHs clearly demonstrate that they can cause toxicity to benthic organisms when present in mixtures. Thresholds for toxicity in several experiments were slightly

higher than would be predicted directly from the narcosis model, though this may reflect uncertainties in the GMAV values as well as exposure-related factors (e.g., avoidance). Measured tissue concentrations in freshwater amphipods from treatments where toxicity was observed were consistent with those shown to be toxic for lower K_{OW} PAHs. Therefore, $\Sigma ESBTU_{FCV}$ for mixtures must include the partial contributions of high K_{OW} PAH in the mixture to insure that the ESB is not under protective.

5.3 Field Sediments Versus ESB_{FCV} for PAH Mixtures

The ultimate test of validity of sediment benchmarks is their predictive ability. That is, can they be used to predict effects seen in field collected samples. Unfortunately, the problem of validation using field collected samples has no straightforward solution. It is extremely difficult to separate actual cause and effect from simple correlation. The primary reason is the presence of covariation of many chemical contaminants in field collected sediments, some of which may be unmeasured. Therefore, it cannot be presumed that the response observed is due to only the chemical(s) being investigated.

However, if the PAH benchmark predicts an effect at a certain $\Sigma ESBTU_{FCV}$ for a mixture of PAHs (e.g., 50% mortality of a test organism), and the organism survives exposures significantly above the $\Sigma ESBTU_{FCV}$ value, then the benchmark may not be valid. No other comparison is more definitive. Of course, mortality at $\Sigma ESBTU_{FCV}$ values below those predicted to cause effects may be due to other causes, and provide no evidence for the validity or invalidity of the prediction.

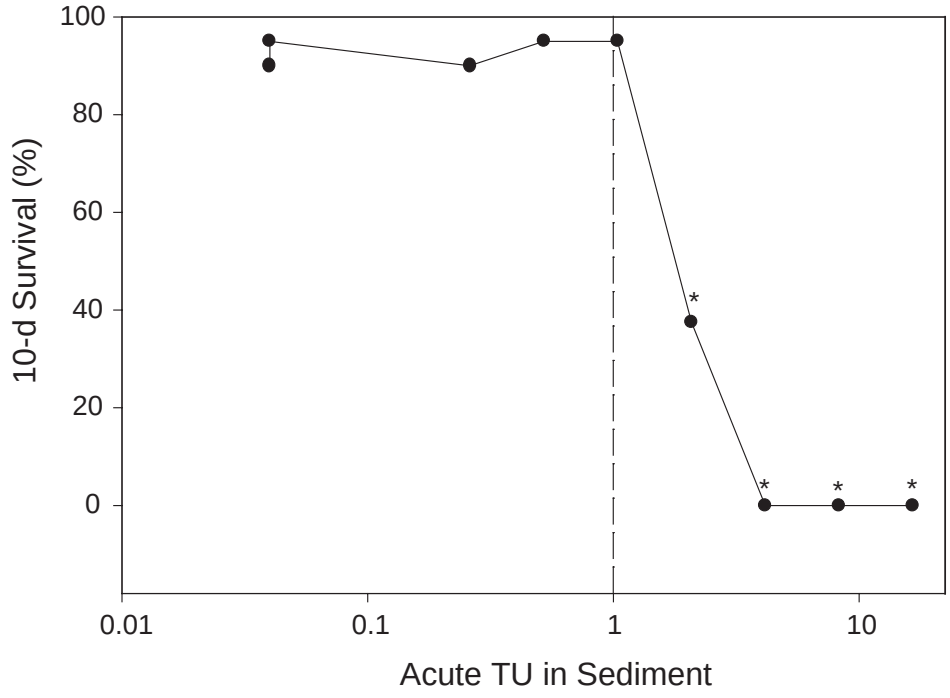

Figure 5-10. Response of *Leptocheirus plumulosus* exposed for 10 days under static conditions to sediment spiked with a mixture of high K_{ow} PAH.

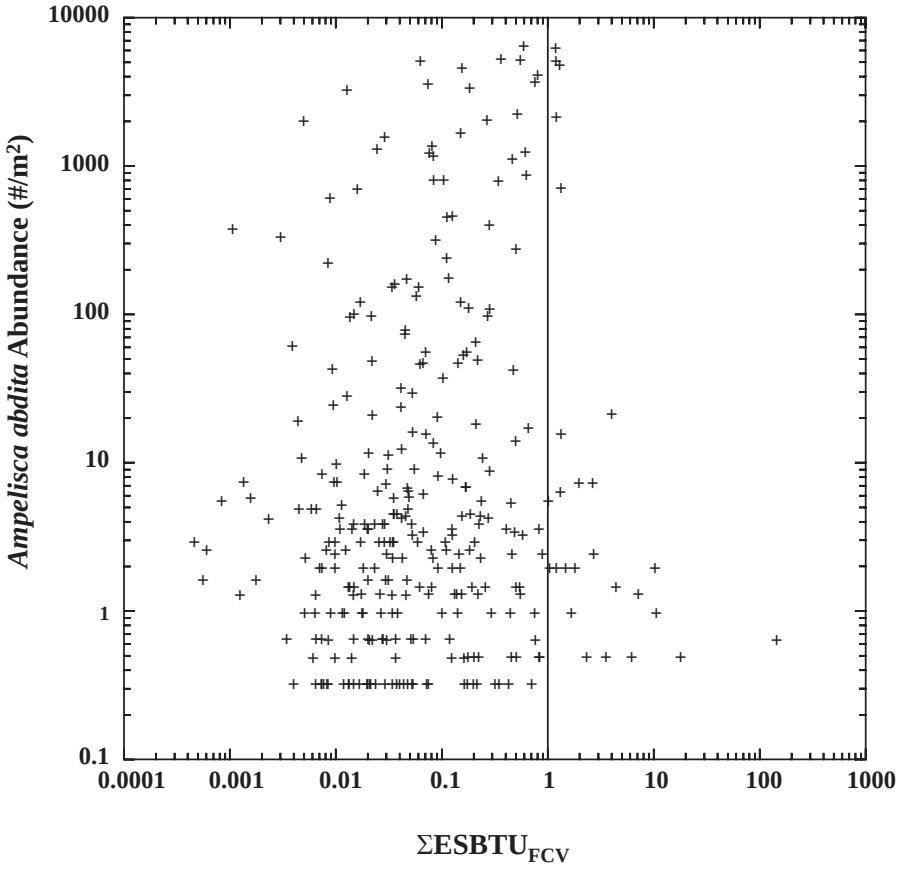

Figure 5-11. Amphipod (*Ampelisca abdita*) abundance versus $\Sigma ESBTU_{FCV}$.

5.3.1 Toxicity to R. abronius of Field Sediments Containing PAH Mixtures vs. ΣPSTUs Derived from Narcosis Theory

A set of 10-day toxicity data using *R. abronius* exposed to sediments from locations where 13 PAHs were measured and PAHs are suspected to be the primary cause of toxicity has been assembled by Swartz et al. (1995). A similar set of data from Elliott Bay where 32 PAHs (18 parent and 14 alkylated groups) were measured (Ozretich et al., 2000) is also available (See Table 3-4 for the list of PAHs). As explained in Section 5.2.7, predicted PAH-specific 10-day sediment LC50 values for *R. abronius* were derived using narcosis theory and 10-day LC50 values based on interstitial water concentrations of eight PAHs for *R. abronius*. The mortality of *R. abronius* in the standard 10-day sediment tests in each of these sediments from the field is compared to the sum of the PSTUs for that sediment (Figure 5-5B). PSTUs are the quotients of the concentration of each PAHs measured in the individual field sediments divided by the predicted PAH-specific 10-day sediment LC50 values for *R. abronius*. The sum of the PSTUs for the sediments where only 13 PAHs were analyzed were multiplied by the uncertainty factor of 2.75 (the mean ratio of the toxic contribution of the 34 PAHs analyzed by the U.S. EPA EMAP program to the 13 PAHs (see Table 6-1)). The uncertainty factor of 2.75, rather than the 95th percent uncertainty factor, was used to adjust for fewer than 34 PAHs because the goal was to use the best estimate of the sum of the toxic units to compare to the observed amphipod mortality in a specific sediment.

Consider, first, the data for which the sum of the PSTUs of the 13 PAHs (termed "predicted 34 PAH" in the figure as represented by the open circles in Figure 5-5B). There is only one

sediment where the sum of the PSTUs exceeds two where mortality was less than 50%. The important point here is that all, except one, of the sediments exceeding this concentration exhibited >50% mortality consistent with the prediction. There could be several explanations why the exception might occur in that one sediment. For example, the 13 PAHs multiplied by the mean uncertainty factor may have under-represented the true total PAH concentration.

For the remaining data, the total PAH concentrations are from field sediments where 31 of the 34 PAHs were measured (open squares). For all of these data there appears to be a concentration-response relationship with an apparent LC50 approximately at the predicted LC50 ± a factor of two, and only one sediment with less than 50% mortality had >2.0 PSTUs. This suggests that the assumption of near additivity of mixtures of PAHs is a reasonable approximation for predicting the toxicity of sediments from the field and for deriving ESBs for PAH mixtures.

5.3.2 Organism Abundance vs. ESB_{FCV} for PAH Mixtures

Another test of this sediment benchmark is the observations of the abundance of sensitive amphipods versus the total PAH concentrations in field collected sediments. Figure 5-11 presents the observed *A. abdita* abundance versus $\Sigma ESBTU_{FCV}$ when 34 PAHs were measured or estimated using the 50% uncertainty factor of 1.64 (see Table 6-1) when 23 PAHs were measured. The data are from sediments collected as part of the Virginian and Louisianian province EMAP (U.S. EPA, 1996a,b) and the New York/New Jersey Harbor REMAP (Adams et al., 1996) sediment sampling programs. The vertical line is at the ESB of 1.0 $\Sigma ESBTU_{FCV}$. The results are very encouraging. The absence of sediments having high abundances of *A. abdita* at slightly above 1.0 $\Sigma ESBTU_{FCV}$ and the decrease in amphipod abundance as the $\Sigma ESBTU_{FCV}$ increases above 1.0 is consistent with that predicted by this ESB for PAH mixtures.

It is tempting to conclude from the coincidence of $\Sigma ESBTU_{FCV}$ values >1.0 and the drop in amphipod abundance, that in fact, these data support the validity of the ESB. However, it should be pointed out again that these data can only be used to demonstrate that the ESB is not in conflict with observations. They cannot be used to validate the ESB. However, these data, and those in Figure 5-5A and B, might have cast doubt on the ESB if effects were predicted and none were observed.

The validation procedure requires sediments for which the nature of all the bioavailable chemicals are known and quantified. This is usually only satisfied with laboratory spiked sediments. This is why the experimental validity of the narcosis mixture theory as is demonstrated in Section 5.2 and illustrated in Figure 5-5A is so important.

Section 6

Implementation

6.1 Introduction

This section on implementation defines "total PAHs" for use with this ESB for PAH mixtures, presents an example ESB calculation, provides guidance on the interpretation of the ESB relative to sediment toxicity tests, describes the role of photo-activation of PAH toxicity by ultraviolet light and the relative importance of teratogenicity and carcinogenicity as a mode of toxic action for PAHs, and critically examines equilibrium of PAHs in sediments, including the presence of soot carbon, coal and similar materials as sediment binding phases other than natural organic carbon. The section ends with an approach for calculating PAH solubilities for temperatures or salinities at a specific site. This information is needed to apply this ESB and assess the risks of mixtures of sediment-associated PAHs based on the EqP methodology.

6.2 Defining Total PAH Concentration in Field Collected Sediments

"Total PAHs" required for deriving the ESB for PAH mixtures is defined in this subsection as the sum of the $ESBTU_{FCV}$ values for a minimum of the 34 PAHs (18 parents and 16 alkylated groups) measured in the U.S. EPA EMAP (U.S. EPA, 1996b, 1998) (Table 6-1). This pragmatic definition is required because databases from sediment monitoring programs that have measured a greater number of PAHs are rare, methodologies for quantification of greater than the 34 PAHs are not standard, and the use of fewer than 34 PAHs may greatly underestimate the total toxicological contribution of the PAH mixtures. We recommend that the uncertainty factors developed in this section for the 13 or 23 commonly quantified PAHs NOT be used to estimate the ESB for the 34 PAHs when important decisions are to be made based on the ESB. However, uncertainty values may be useful in specific non-ESB related decisions. The recommendation to not use the uncertainty factors for derivation of ESBs is intended to prevent the under- or over-estimation of an ESB acceptable for the protection of benthic organisms and to encourage the analysis of a minimum of the 34 PAHs using readily available analytical methodologies for new monitoring programs (NOAA, 1998)

It is expected that many sediment assessors may be in the position where available data are limited to only certain PAHs (e.g., 13 un-substituted compounds) and it is impractical to re-analyze all samples for the full suite of PAHs, but

Table 6-1. Relative distribution of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ and $\Sigma ESBTU_{FCV,23}$ for the combined EMAP dataset (N=488).

Percentile	$\Sigma ESBTU_{FCV,TOT}/\Sigma ESBTU_{FCV,13}$	$\Sigma ESBTU_{FCV,TOT}/\Sigma ESBTU_{FCV,23}$
50	2.75	1.64
80	6.78	2.8
90	8.45	3.37
95	11.5	4.14
99	16.9	6.57

also undesirable to accept uncertainties stemming from the incomplete PAH characterization. In this instance, an intermediate approach may be to analyze a subset of sediment samples for the full suite of PAHs and use these data to develop a site-specific correction factor. This approach requires the assumption that this correction factor is consistent across the site, but it seems likely that the uncertainty with this assumption will be less than the uncertainty involved in using the generic correction factors from Table 6-1.

The following subsection presents the analysis that led to the adoption of the 34 PAHs as total PAH. Hereafter, mention of "total PAHs" in this document refers to use of a minimum of the 34 PAHs to derive the $\Sigma ESBTU_{FCV}$.

6.2.1 Introduction

PAHs are present in sediments as mixtures rather than as single compounds. It has been shown that the toxicity of sediment associated PAHs is approximately additive, and that PAHs with both low and high K_{OW} values contribute to the total toxicity (Section 5). Therefore, assessment of the toxicological contribution from the total PAH concentration present in sediments would theoretically require the measurement in every sediment of all PAHs. If the compounds formed by the alkylation of parent PAHs are included, there are more than several hundred possible structures, and quantifying all of them is impractical and costly.

As an alternative to measuring all PAHs, it may be possible to estimate the total PAH concentration in sediments using a subset of the commonly measured PAHs. This is desirable because the number of individual PAHs measured in field sediment monitoring programs varies and if too few PAHs are measured, the toxicity of sediment-associated PAHs will be underestimated. For some historical sediment monitoring data, only 13 PAHs identified by the U.S. EPA as parameters of concern were measured (Table 6-2). The National Oceanic and Atmospheric Administration (NOAA, 1991) began to quantify 10 additional PAHs in sediments, bringing the total number of PAHs measured to 23. Since then, the majority of sediment monitoring programs have measured these 23 PAHs (Table 6-2). More recently, the U.S. EPA EMAP has increased the number of PAHs measured from 23 to 34 by quantifying the C1 through C4 alkylated series for some patent PAHs where the C# indicates an alkyl group substitution (Table 6-2). The C1 represents one methyl substitution at any location on the PAH. The C2 represents either two methyl substitutions at any two locations or one ethyl substitution at any one location. The C3 represents either three methyl groups, one methyl and one ethyl group or one propyl group substitution. Similarly, the C4 represents any combination of methyl, ethyl, propyl and butyl groups so that the total number of carbons added to the parent PAH is four (Table 6-2). Although a C# PAH series by itself represents several different structures, for simplicity a C# PAH series was considered as one PAH. In total, this C# PAH alkylated series represents 16 groups of compounds as listed in Table 6-2.

In this section, the uncertainty limits are derived for estimating the total PAH toxicological contribution of the 34 PAHs from the 13 or 23 commonly measured PAHs. Data are presented using $ESBTU_{FCVi}$ to sum the contributions of the individual PAHs and determine the total PAH toxicity of the mixture as represented by the $\Sigma ESBTU_{FCV}$.

6.2.2 Data Collection

Coastal and estuarine sediment data from the Nation's water bodies were compiled from nine sources (NOAA, 1991; Adams et al., 1996; Anderson et al., 1996; Fairey et al., 1996; U.S. EPA, 1996a,b,1998; Ozretich et al., 2000; Hunt et al., 1998). With the exception of the Elliott Bay data (Ozretich et al., 2000), all of the data sources were from state and/or government funded sediment monitoring programs. In Elliott Bay, the PAHs were suspected to be causing the toxicity due to their elevated levels. Data sources that were identified had measured concentrations for at least the 23 PAHs identified by NOAA and

Table 6-2. PAH measured in various sediment monitoring programs. See Di Toro and McGrath (2000) for data sources.

Parameter	NOAA	SFEI	San Diego	Southern California	NY/NJ REMAP[A]	Virginian EMAP[B]	Elliott Bay	Carolinian EMAP	Louisianian EMAP
Acenaphthene	x	x	x	x	x	x	x	x	x
Acenaphthylene	x	x	x	x	x	x	x	x	x
Anthracene	x	x	x	x	x	x	x	x	x
Chrysene	x	x	x	x	x	x	x	x	x
Fluoranthene	x	x	x	x	x	x	x	x	x
Fluorene	x	x	x	x	x	x	x	x	x
naphthalene	x	x	x	x	x	x	x	x	x
phenanthrene	x	x	x	x	x	x	x	x	x
pyrene	x	x	x	x	x	x	x	x	x
Benzo(k)fluoranthene	x	x	x	x	x	x	x	x	x
Benzo(b)fluoranthene	x	x	x	x	x	x	x	x	x
Benzo(a)pyrene	x	x	x	x	x	x	x	x	x
Benzo(a)anthracene	x	x	x	x	x	x	x	x	x
Benzo(e)pyrene	x	x	x	x	x	x	x	x	x
Benzo(g,h,i)perylene	x	x	x	x	x	x	x	x	x
Dibenz(a,h)anthracene	x	x	x	x	x	x	x	x	x
2,6-dimethylnaphthalene	x	x	x	x	x	x	x	x	x
Indeno(1,2,3-cd)pyrene	x	x	x	x	x	x	x	x	x
1-methylnaphthalene	x	x	x	x	x	x	x	x	x
2-methylnaphthalene	x	x	x	x	x	x	x	x	x
perylene	x	x	x	x	x	x	x	x	x
1-methylphenanthrene	x	x	x	x	x	x	x	x	x
2,3,5-trimethylnaphthalene	x	x	x	x	x	x	x	x	x
2-methylanthracene							x		
2-methylphenanthrene		x					x		
3,6-dimethylphenanthrene							x		
9-methylanthracene		x					x		
9,10-dimethylanthracene							x		
C1-benzo(a)anthracenes /chrysenes							x	x	x
C2-benzo(a)anthracenes /chrysenes							x	x	x
C3-benzo(a)anthracenes /chrysenes								x	x
C4-benzo(a)anthracenes /chrysenes								x	x
C1-fluoranthenes/pyrenes							x	x	x
C2-fluoranthenes/pyrenes							x		
C1-fluorenes							x	x	x
C2-fluorenes							x	x	x
C3-fluorenes							x	x	x
C1-naphthalenes							x	x	x
C2-naphthalenes							x	x	x
C3-naphthalenes							x	x	x
C4-naphthalenes							x	x	x
C1-phenanthrenes/anthracenes							x	x	x
C2-phenanthrenes/anthracenes							x	x	x
C3-phenanthrenes/anthracenes							x	x	x
C4-phenanthrenes/anthracenes								x	x
Total Number of PAHs[B]	23	25	23	23	23	23	32	34	34
Number of data points	640	137	182	40	153	318	30	280	229

[A] Benzo(b)fluoranthene and benzo(k)flouranthene were measured together.
[B] A specific C1-PAH was not included in the total if the C1 alkylated PAH series was measured. For example, 1-methylnaphthalene was not included in the total if the C1-naphthalenes were measured.

corresponding sediment organic carbon measurements. Three sources, Elliott Bay, EMAP Louisianian Province and EMAP Carolinian Province, had measurements for some of the alkylated PAH series. The two EMAP sources analyzed for the same alkylated PAH series. The Elliott Bay dataset had some alkylated PAHs that were similar to the EMAP sources and some alkylated PAHs that were not included in the EMAP sources. A listing of the PAHs measured in each dataset is provided in Table 6-2. The first 13 PAHs in the list are the initial 13 PAHs identified by the U.S. EPA as PAHs of concern. The first 23 PAHs in the list include the additional PAHs monitored by NOAA. The total number of PAHs measured in each dataset is also provided. To prevent duplicate counting, a specific C1, C2, C3 or C4 PAH was not included in the total number of PAHs if the alkylated PAH series was measured. As an example, for Carolinian EMAP, 1-methylnaphthalene was not included in the total, because the C1-naphthalenes were measured.

To screen for insoluble PAHs, interstitial water concentrations were computed from measured solid phase concentrations using EqP theory (Di Toro et al., 1991). If the resulting interstitial water concentrations were greater than the corresponding solubilities, insoluble PAHs were assumed to be present in the sediment. For these cases, the measured solid phase concentrations were replaced by solid phase concentrations based on the aqueous solubility of each compound ($C_{OC,PAHi,Maxi}$).

The data were converted to $ESBTU_{FCVi}$ for individual PAHs by dividing the concentration of the specific PAH in the sediment (C_{OC},µg/g_{OC}) by the $C_{OC,PAHi,FCVi}$. $\Sigma ESBTU_{FCV}$ for each sediment sample were computed by summing the $ESBTU_{FCVi}$ for each PAH measured. For purposes of this section, $\Sigma ESBTU_{FCV}$ for the 34 PAHs is denoted by $\Sigma ESBTU_{FCV,TOT}$. Equation 6-1 was used to compute $\Sigma ESBTU_{FCV,TOT}$

$$ESBTU_{FCV,TOT} = \Sigma ESBTU = \sum_{i} \frac{C_{OCi}}{C_{OC,PAHi,FCVi}} \leq 1.0 \qquad (6\text{-}1)$$

6.2.3 Methodology

The objective was to determine the uncertainty of using the 13 PAHs or the 23 PAHs to predict the $\Sigma ESBTU_{FCV,TOT}$. Only the monitoring databases containing 34 PAHs were used in this analysis. The 13 PAHs were selected since the majority of the existing sediment monitoring data include these PAHs. The uncertainty values for estimating total PAHs from datasets where 13 or 23 PAHs were measured were developed from a database of ratios of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ or $\Sigma ESBTU_{FCV,23}$. In addition, regression analyses of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ or to $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,23}$ on a log-log linear basis were conducted to demonstrate the utility of the ratio approach across the range of $\Sigma ESBTU_{FCV}$ values.

6.2.4 Uncertainty in Predicting $\Sigma ESBTU_{FCV,TOT}$

For use in determining the uncertainty in predicting $\Sigma ESBTU_{FCV,TOT}$ from datasets consisting of the 13 or 23 PAHs, the two EMAP data sources that measured the 34 PAHs were combined and treated as a single data source. In doing this, a larger dataset that represents both alkylated and parent PAHs, and therefore, inherently has the correlative relationships of both types of PAHs, was generated (N=488). The relative distributions of the $\Sigma ESBTU_{FCV,TOT}$ to the $\Sigma ESBTU_{FCV}$ for the 13 and 23 PAHs for this dataset are provided in Table 6-1. Based on the observed ratios, the measured $\Sigma ESBTU_{FCV,13}$ for the 13 PAHs must be multiplied by 11.5 to obtain an accurate estimation of the $\Sigma ESBTU_{FCV,TOT}$ with 95 % confidence. Similarly, the measured $\Sigma ESBTU_{FCV,23}$ for the 23 PAHs must be multiplied by 4.14 to obtain an estimate of the $\Sigma ESBTU_{FCV,TOT}$ with 95% confidence. High adjustment factors needed to estimate $\Sigma ESBTU_{FCV,TOT}$, particularly from 13 PAHs, indicate the importance of having real measurements of the 34 PAHs from sediments where the PAH concentrations are of likely toxicological significance. In contrast, for

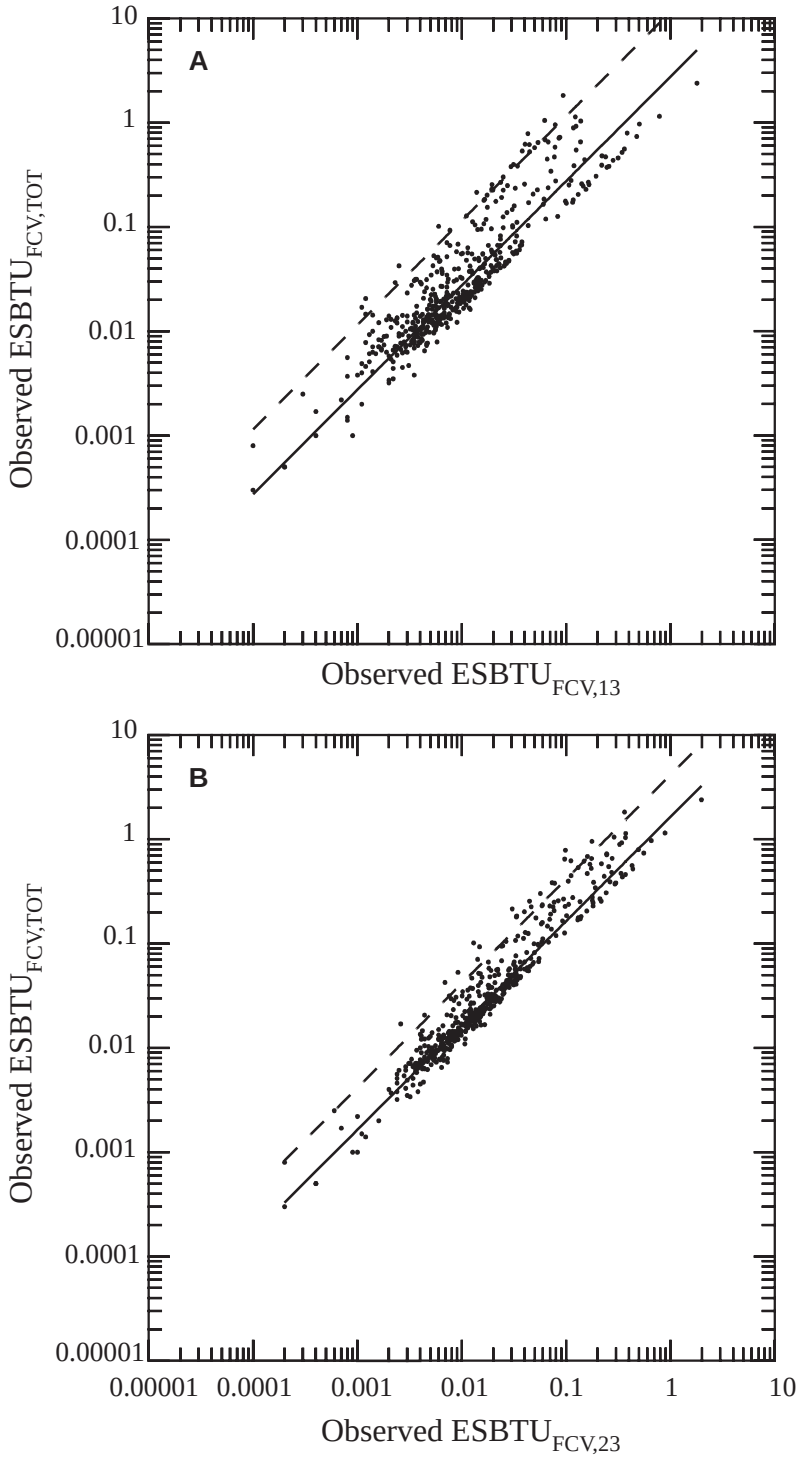

Figure 6-1. Comparison of observed ΣESBTU$_{FCV,TOT}$ to observed (A) ΣESBTU$_{FCV,13}$ from13 PAHs and (B) ΣESBTU$_{FCV,23}$ from 23 PAHs for the combined dataset including U.S. EPA EMAP Louisianian and Carolinian Provinces.

sediments where the $\Sigma ESBTU_{FCV,13}$ or $\Sigma ESBTU_{FCV,23}$ times the uncertainty factors does exceed the ESB, additional measurements including the 34 PAHs would not be warranted.

The $\Sigma ESBTU_{FCV,TOT}$ can be plotted against the $\Sigma ESBTU_{FCV,13}$ for the 13 PAHs and regression analysis conducted to show that the ratios can be used fairly well to estimate the $\Sigma ESBTU_{FCV}$ for the 34 PAHs from the sum of the 13 PAHs across a wide range of $\Sigma ESBTU_{FCV,TOT}$ because the slope is nearly 1.0 (0.9595) (Figure 6-1A). The solid line is the mean (50%) ratio of 2.75 from Table 6-1 and the dashed line is the line representing 95% of the data with a ratio of 11.5. The resulting linear regression equation from a log-log relationship is

$$\log_{10} \Sigma ESBTU_{FCV,TOT} = 0.9595 \log_{10} \Sigma ESBTU_{FCV,13} + 0.4251 \quad (R^2 = 0.8236) \qquad (6\text{-}2)$$

A similar analysis using the $\Sigma ESBTU_{FCV,TOT}$ plotted against the sum $\Sigma ESBTU_{FCV,23}$ of 23 PAHs with a regression analysis conducted to show that the slope of the regression is also nearly 1.0 (1.038) (Figure 6-1B). The solid line is the mean (50%) ratio of 1.64 from Table 6-1 and the dashed line represents 95% of the data with a ratio of 4.14. The resulting linear regression equation from a log-log relationship is

$$\log_{10} \Sigma ESBTU_{FCV,TOT} = 1.038 \log_{10} \Sigma ESBTU_{FCV,23} + 0.3576 \quad (R^2 = 0.9272) \qquad (6\text{-}3)$$

The regression approach has been used to derive uncertainty factors for estimating the $\Sigma ESBTU_{FCV,TOT}$ for the 34 PAHs using combinations of as few as three to as many as 13 or 23 PAHs (McGrath and Di Toro, 2000)

The probability distributions of the $\Sigma ESBTU_{FCV,13}$ and $\Sigma ESBTU_{FCV,23}$ values for each sediment from the databases in Table 6-2 were plotted in Figure 6-2 (A and B, respectively). The actual $\Sigma ESBTU_{FCV,13}$ values (triangles) exceeded 1.0 for 5.22% of the 1992 sediment samples (Figure 6-2A) and the $\Sigma ESBTU_{FCV,23}$ values (triangles) exceeded 1.0 for 6.55% of the 2001

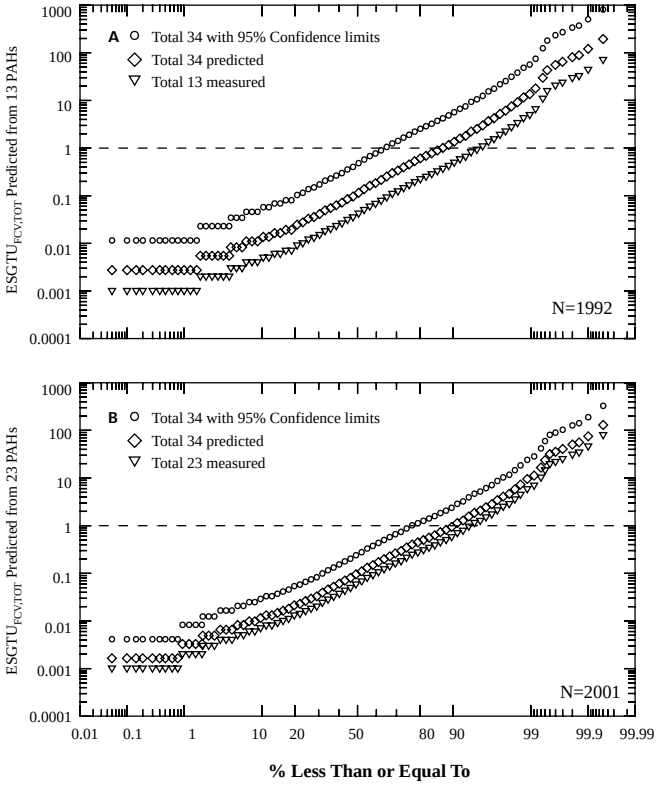

Figure 6-2. Probability distribution of the (A) $\Sigma ESBTU_{FCV,13}$ and (B) $\Sigma ESBTU_{FCV,23}$ values for each sediment from the entire database.

sediment samples (Figure 6-2B). To estimate the 50% uncertainty of $\Sigma ESBTU_{FCV,TOT}$ (plotted as diamonds in Figure 6-2), the mean ratio of the $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,13}$ (2.75; see Table 6-1) was applied to the sediment-specific $\Sigma ESBTU_{FCV,13}$ values and the mean ratio of $\Sigma ESBTU_{FCV,TOT}$ to $\Sigma ESBTU_{FCV,23}$ (1.64) was applied to the sediment-specific $\Sigma ESBTU_{FCV,23}$ values. The $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,13}$ exceeded 1.0 for 12.9% of the 1992 sediment samples and the $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,23}$ exceeded 1.0 for 9.85% of the 2001 sediment samples. The 95% uncertainty estimates of the $\Sigma ESBTU_{FCV,TOT}$ for each sediment (plotted as circles in Figure 6-2) was determined by multiplying the sediment-specific $\Sigma ESBTU_{FCV,13}$ values by 11.5 and by multiplying the sediment-specific $\Sigma ESBTU_{FCV,23}$ by 4.14 (Table 6-1). The 95% limits on the $\Sigma ESBTU_{FCV,TOT}$ estimated from the $\Sigma ESBTU_{FCV,13}$ exceeded 1.0 for 35.5% of the 1992 sediment samples and the 95% limits on the $\Sigma ESBTU_{FCV,TOT}$

estimated from the $\Sigma ESBTU_{FCV,23}$ exceeded 1.0 for 23.7% of the 2001 sediment samples. Therefore, if the 95% uncertainty ratios are applied to the $\Sigma ESBTU_{FCV,13}$ or the $\Sigma ESBTU_{FCV,23,}$ the predicted $\Sigma ESBTU_{FCV,TOT}$ for about one-third of the sediments are in excess of the ESB for PAH mixtures of 1.0 $\Sigma ESBTU_{FCV}$. This strongly suggests that new monitoring programs should quantify a minimum of the 34 PAHs monitored by the U.S. EPA EMAP program. In addition, field sediments containing PAHs of principally petrogenic origin will contain a greater proportion of alkalyated PAHs and PAHs not quantified in the 34 "total PAHs"(Bence et al., 1996; Means, 1998; Ho et al., 1999; Page et al., 2002). Therefore, the uncertainty factors derived above from sediments containing mostly pyrogenic PAHs, will underestimate the total PAH toxic unit contribution of the PAH mixture in sediments contaminated with mostly petrogenic PAHs. It is important to repeat that at present, the uncertainty of using the 34 PAHs to estimate the total toxicological contributions of the unmeasured PAHs is unknown and needs additional research.

For existing databases, individuals may wish to utilize uncertainty factors for sediment assessment applications other than the derivation of an ESB for PAH mixtures. An example, the use of the 50% uncertainty factors from Table 6-1 to provide the "best estimate" of the $\Sigma ESBTU_{FCV,TOT}$ for the 34 "total PAHs" in field sediments to compare with amphipod abundance (Section 5.3.1, Figure 5-11). If the number and kinds of PAHs are of similar proportions from a database from a specific site, uncertainty factors for adjusting the concentrations of the PAHs at that site may be derived using the approach detailed above. Research to determine the toxicological contributions of PAHs in sediments that are not included in the 34 PAHs is encouraged so that the uncertainty of this definition of "total PAHs" can be estimated.

6.3 Example Calculation of ESB_{FCV} for PAHs and EqP-based Interpretation

To assist the users of this ESB for mixtures of PAHs, example calculations for deriving the ESB are provided in Table 6-3. For each of three sediments, the calculations began with measured concentrations (in bold) of individual PAHs (µg/g dry wt.) and TOC (%) in each sediment. All other values were calculated. The specific concentrations in each sediment were selected to provide examples of how the chemical measurements are used with the ESB to determine the acceptability of the mixture of PAHs in a specific sediment and how the risks of sediment-associated PAHs can be evaluated within the technical framework of the EqP and narcosis approaches. The 34 PAHs constituting what is defined as "total PAH" in Section 6 are listed. Also listed are the critical concentrations in sediment of each of the 34 individual PAHs ($C_{OC,PAHi,FCVi}$) from Table 3-4.

Sediment A is provided as an example to demonstrate how to calculate the ΣESBTU when less than the required 34 PAHs have been chemically analyzed. It is important to remember that because of the uncertainty in such calculations the resultant ΣESBTU must not be considered as an ESB nor used in important sediment management decisions. Uncertainty factors applied to the ΣESBTU have value, for example, in determining if additional chemical analyses are required and prioritizing which sediments require the additional analyses.

Sediment A is from a historical monitoring database, it contains concentrations of 13 PAHs measured as µg PAH/g dry sediment and has 0.81% TOC. First, the dry weight concentrations for each PAH were converted to µg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) by dividing by the fraction organic carbon (f_{OC} = 0.0081), where f_{OC} = %TOC/100. Second, the organic carbon-normalized PAH concentrations in the sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the toxic unit-like $ESBTU_{FCVi}$ for each individual PAH. In this sediment, none of the measured C_{OCi} exceed the corresponding $C_{OC,PAHi,Maxi}$ so solubility constraints do not affect the calculation of $ESBTU_{FCVi}$ for this sediment. The $ESBTU_{FCVi}$ for the 13 PAHs were added to derive the ΣESBTU for the 13 PAHs ($\Sigma ESBTU_{FCV,13}$) which is 0.348

Table 6-3A. Example calculations of ESBs for PAH mixtures: three sediments.

			Sediment A (TOC=0.81%; f_{OC}=0.0081)		
PAH[A]	$C_{OC, PAHi, FCVi}$ ($\mu g/g_{OC}$)	$C_{OC, PAHi, Maxi}$ ($\mu g/g_{OC}$)	Conc. ($\mu g/g$ dry wt.)	C_{OC} ($\mu g/g_{OC}$)	$ESBTU_{FCVi}$
naphthalene	385	61700	**0.0894**	11	0.0287
C1 naphthalenes	444	-			
acenaphthylene	452	24000	**0.0348**	4.29	0.0095
acenaphthene	491	33400	**0**	0	0
C2 naphthalenes	510	-			
fluorene	538	26000	**0.0722**	8.91	0.0166
C3 naphthalenes	581	-			
anthracene	594	1300	**0.628**	77.6	0.1306
phenanthrene	596	34300	**0.139**	17.1	0.0287
C1 flourenes	611	-			
C4 naphthalenes	657	-			
C1 phenanthrenes	670	-			
C2 flourenes	686	-			
pyrene	697	9090	**0.171**	21.1	0.0303
flouranthene	707	23870	**0.0806**	9.96	0.0141
C2 phenanthrenes	746	-			
C3 flourenes	769	-			
C1 fluoranthenes	770	-			
C3 phenanthrenes	829	-			
benz(a)anthracene	841	4153	**0.0709**	8.75	0.0104
chrysene	844	826	**0.157**	19.4	0.023
C4 phenanthrenes	913	-			
C1 chrysenes	929	-			
benzo(a)pyrene	965	3840	**0.164**	20.3	0.021
perylene	967	431			
benzo(e)pyrene	967	4300			
benzo(b)fluoranthene	979	2169	**0.139**	17.2	0.0175
benzo(k)fluoranthene	981	1220	**0.139**	17.2	0.0175
C2 chrysenes	1008	-			
benzo(g,h,i)perylene	1095	648			
C3 chrysenes	1112	-			
indeno(1,2,3-cd)pyrene	1115	-			
dibenzo(a,h)anthracene	1123	2389			
C4 chrysenes	1214	-			
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,13} = 0.3479$

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

Table 6-3B. Continued.

			Sediment B (TOC=0.886%; f_{OC}=0.00886)		
PAH[A]	$C_{OC, PAHi, FCVi}$ (µg/g_{OC})	$C_{OC, PAHi, Maxi}$ (µg/g_{OC})	Conc. (µg/g dry wt.)	C_{OC} (µg/g_{OC})	$ESBTU_{FCVi}$
naphthalene	385	61700	**0.2703**	30.51	0.07925
C1 naphthalenes	444	-	**1.2084**	136.39	0.30719
acenaphthylene	452	24000	**0.0165**	1.86	0.00412
acenaphthene	491	33400	**0.0401**	4.53	0.00922
C2 naphthalenes	510	-	**3.2691**	368.98	0.72348
fluorene	538	26000	**0.3702**	41.78	0.07766
C3 naphthalenes	581	-	**5.1079**	576.51	0.99227
anthracene	594	1300	**0.0507**	5.72	0.00962
phenanthrene	596	34300	**0.5679**	64.09	0.1075
C1 flourenes	611	-	**0.9362**	105.67	0.17294
C4 naphthalenes	657	-	**3.3088**	373.46	0.56843
C1 phenanthrenes	670	-	**0.9267**	104.6	0.15611
C2 flourenes	686	-	**1.2384**	139.77	0.20375
pyrene	697	9090	**0.408**	46.05	0.06606
flouranthene	707	23870	**0.3244**	36.62	0.0518
C2 phenanthrenes	746	-	**1.0645**	120.15	0.16106
C3 flourenes	769	-	**1.2664**	142.94	0.18587
C1 fluoranthenes	770	-	**0.3824**	43.16	0.05605
C3 phenanthrenes	829	-	**0.81**	91.43	0.11028
benz(a)anthracene	841	4153	**0.2011**	22.69	0.02698
chrysene	844	826	**0.2574**	29.05	0.03442
C4 phenanthrenes	913	-	**0.5644**	63.71	0.06978
C1 chrysenes	929	-	**0.2987**	33.72	0.03629
benzo(a)pyrene	965	3840	**0.1817**	20.51	0.02125
perylene	967	431	**0.3511**	39.63	0.04098
benzo(e)pyrene	967	4300	**0.1673**	18.89	0.01953
benzo(b)fluoranthene	979	2169	**0.1708**	19.28	0.01969
benzo(k)fluoranthene	981	1220	**0.1962**	22.15	0.02258
C2 chrysenes	1008	-	**0.2242**	25.3	0.0251
benzo(g,h,i)perylene	1095	648	**0.1504**	16.97	0.0155
C3 chrysenes	1112	-	**0.0279**	3.15	0.00283
indeno(1,2,3-cd)pyrene	1115	-	**0.1473**	16.63	0.01491
dibenzo(a,h)anthracene	1123	2389	**0.0423**	4.77	0.00425
C4 chrysenes	1214	-	**0.1196**	13.5	0.01112
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,TOT}$ = 4.408

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

Table 6-3C. Continued.

PAH[A]	$C_{OC, PAHi, FCVi}$ (µg/g_{OC})	$C_{OC, PAHi, Maxi}$ (µg/g_{OC})	Sediment C (TOC=6.384%; f_{OC}=0.06384) Conc. (µg/g dry wt.)	C_{OC} (µg/g_{OC})	$ESBTU_{FCVi}$
naphthalene	385	61700	**2.193**	34.4	0.0892
C1 naphthalenes	444	-	**1.37**	21.9	0.0493
acenaphthylene	452	24000	**2.04**	32	0.0707
acenaphthene	491	33400	**0.806**	12.6	0.0257
C2 naphthalenes	510	-	**1.448**	22.7	0.0445
fluorene	538	26000	**1.387**	21.7	0.0404
C3 naphthalenes	581	-	**1.979**	31	0.0533
anthracene	594	1300	**3.695**	57.9	0.0974
phenanthrene	596	34300	**4.208**	65.9	0.1106
C1 flourenes	611	-	**1.03**	16.1	0.0264
C4 naphthalenes	657	-	**2.009**	31.5	0.0479
C1 phenanthrenes	670	-	**4.559**	71.4	0.1066
C2 flourenes	686	-	**1.928**	30.2	0.0440
pyrene	697	9090	**20.14**	315.5	0.4526
flouranthene	707	23870	**2.519**	39.5	0.0558
C2 phenanthrenes	746	-	**4.789**	75	0.1006
C3 flourenes	769	-	**3.419**	53.6	0.0696
C1 fluoranthenes	770	-	**11.73**	183.7	0.2386
C3 phenanthrenes	829	-	**5.378**	84.2	0.1016
benz(a)anthracene	841	4153	**8.293**	129.9	0.1545
chrysene	844	826	**9.197**	144.1	0.1707
C4 phenanthrenes	913	-	**4.674**	73.2	0.0802
C1 chrysenes	929	-	**5.24**	82.1	0.0884
benzo(a)pyrene	965	3840	**10.97**	171.8	0.1781
perylene	967	431	**28.23**	442.2	0.4457[B]
benzo(e)pyrene	967	4300	**8.92**	139.7	0.1445
benzo(b)fluoranthene	979	2169	**18.14**	284.1	0.2902
benzo(k)fluoranthene	981	1220	**5.5**	86.2	0.0878
C2 chrysenes	1008	-	**4.753**	74.5	0.0739
benzo(g,h,i)perylene	1095	648	**5.583**	87.5	0.0799
C3 chrysenes	1112	-	**0.398**	6.2	0.0056
indeno(1,2,3-cd)pyrene	1115	-	**10.8**	169.2	0.1517
dibenzo(a,h)anthracene	1123	2389	**2.499**	39.1	0.0349
C4 chrysenes	1214	-	**1.581**	24.8	0.0204
Sum total of $ESBTU_{FCVi}$					$\Sigma ESBTU_{FCV,TOT}$ = 3.831

[A] PAHs and corresponding $C_{OC,PAHi,FCVi}$ and $C_{OC,PAHi,Maxi}$ values are from Table 3-4 (bold).

[B] Because C_{OC} exceeds $C_{OC,PAHi,Maxi}$, $C_{OC,PAHi,Maxi}$ is substituted for C_{OC} to calculate $ESBTU_{FCVi}$ (see text).

(Table 6-3). Importantly, only 13 of the 34 individual PAHs defined as total PAH were measured. Because the toxicological contributions of all 34 PAHs must be considered if the ESB is to be protective of benthic organisms, some assumption must be made regarding the contribution of the unmeasured PAHs. For a confidence level of 95%, the uncertainty factor from Table 6-1 is 11.5, which is then multiplied by the calculated $\Sigma ESBTU_{FCV,13}$ of 0.348 for an estimated value of $\Sigma ESBTU_{FCV,34}$ of 4.00. Since this value is greater than one, it suggests the potential for adverse effects from PAHs. However, one must realize that this finding is, in part, a function of the correction factor selected to relate the data for 13 PAHs to an estimated ΣESBTU for 34 PAHs. If the value for 50% confidence was selected from Table 6-1 (2.75), the estimated $\Sigma ESBTU_{FCV,34}$ drops to 0.957, which is much lower than the value predicted for the 95% confidence interval. This difference illustrates the importance of measuring all 34 PAH compounds in order to eliminate unnecessary uncertainty in applying the PAH ESB.

Sediment B is a PAH-contaminated sediment from one of the U.S. EPA EMAP monitoring programs where all 34 of the PAHs in the sediment and TOC (0.886%) were measured. The concentrations of each PAH on a μg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) basis were derived by dividing the dry weight concentrations by the fraction organic carbon (f_{OC} = 0.00886), where f_{OC} = %TOC/100. The organic carbon-normalized PAH concentrations in sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the $ESBTU_{FCVi}$ for each individual PAH. As was the case for Sediment A, none of the measured C_{OC} exceeded $C_{OC,PAHi,Maxi.}$, so solubility constraints did not factor into the calculation of $ESBTU_{FCVi}$. The $ESBTU_{FCVi}$ values for the 34 PAHs were summed to determine the $\Sigma ESBTU_{FCV}$ which was 4.41, which exceeds the ESB ($\Sigma ESBTU_{FCV}$ >1.0) for PAH mixtures. Further examination of this sediment suggested that it is contaminated with primarily petrogenic PAHs; i.e., the ratio of $\Sigma ESBTU_{FCV,13}$ (which contains no alkylated PAHs) to $\Sigma ESBTU_{FCV}$ for the 34 PAHs is low (approximately 0.1). Chemical analysis of the PAHs in interstitial water indicated that this sediment may be unacceptably contaminated by the mixture of PAHs because it contained 5.6 interstitial water toxic units ($IWTU_{FCV}$). Ten day toxicity tests, which were part of the monitoring project, showed 64% mortality of *R. abronius* which is consistent with the $IWTU_{FCV}$ and the 10-day spiked sediment LC50 for *R. abronius* at 3.68 $\Sigma ESBTU_{FCV}$ values (Appendix D). This suggests the EqP- and narcosis-based ESB is appropriate to the sediment. The sediment is unacceptable for the protection of benthic organisms due to the PAH mixture present and additional studies to quantify the spatial extent of contamination are desirable.

Sediment C is also a PAH-contaminated sediment from an U.S. EPA EMAP monitoring program where the 34 PAHs and TOC of 6.38% were measured. The concentrations of each PAH on a µg PAH/g organic carbon (C_{OC}, $\mu g/g_{OC}$) basis were derived by dividing the dry weight concentrations by the fraction organic carbon (f_{OC} = 0.0638), where f_{OC} = %TOC/100. Except for perylene, the organic carbon-normalized PAH concentrations in sediment were divided by the PAH-specific sediment concentration of concern ($C_{OC,PAHi,FCVi}$) to derive the $ESBTU_{FCVi}$ for each individual PAH. The concentration of perylene 442.2 $\mu g/g_{OC}$ exceeded the solubility-constrained solid phase concentration ($C_{OC,PAHi,Maxi}$). Thus, the $ESBTU_{FCVi}$ for perylene was calculated as the quotient of the solubility-constrained solid phase concentration over the perylene-specific solid phase concentration equivalent to the FCV ($ESBTU_{FCV,Perylene} = C_{OC,Perylene,Maxi} / C_{OC,Perylene,FCV}$). The $ESBTU_{FCVi}$ values for the 34 PAHs were summed to determine the $\Sigma ESBTU_{FCV}$ which was 3.83, a similar value as in sediment B. The PAH mixture in sediment C exceeds the ESB ($\Sigma ESBTU_{FCV}$ >1.0) for PAH mixtures. In contrast to sediment B, sediment C was not toxic to *R. abronius* in 10-day sediment toxicity tests. This sediment is contaminated with primarily pyrogenic PAHs; i.e., the ratio of $\Sigma ESBTU_{FCV,13}$ (which contains no alkylated PAHs) to $\Sigma ESBTU_{FCV}$ for the 34 PAHs is high (approximately 0.5).

Because this PAH mixture appears to be combustion related, it suggests the potential for the presence of soot carbon, coal, or other carbon forms that show unusual partitioning behavior relative to normal diagenetic carbon (see Section 6.8). Indeed, chemical analysis of interstitial water from this sediment showed <0.12 $IWTU_{FCV}$ of PAHs. If normal partitioning behavior was occuring, one would expect the $IWTU_{FCV}$ to be very close to the calculated $\Sigma ESBTU_{FCV}$ (in this case, 3.89) is indicative of this unusual partitioning behavior. Physical examination of the sediment showed the presents of soot-like particles. The presence of soot and associated differences in chemical partitioning make the directly calculated $\Sigma ESBTU_{FCV}$ overly protective for this sediment. However, one could apply the general PAH ESB approach to the interstitial water using $IWTU_{FCV}$, or develop site-specific partition coefficients and recalculate $\Sigma ESBTU_{FCV}$ using site-specific $C_{OC,PAHi,FCVi}$ values calculated from the site-specific partition coefficients, as described in U.S. EPA (2003b).

6.4 Interpreting ESBs in Combination with Toxicity Tests

Sediment toxicity tests provide an important complement to ESBs in interpreting overall risk from contaminated sediments. Toxicity tests have different strengths and weaknesses compared to chemical-specific benchmarks, and the most powerful inferences can be drawn when both are used together.

Unlike chemical-specific benchmarks, toxicity tests are capable of detecting any toxic chemical, if it is present in toxic amounts; one does not need to know what the chemicals of concern are to monitor the sediment. Toxicity tests are also useful for detecting the combined effect of chemical mixtures, if those effects are not considered in the formulation of the applicable chemical-specific benchmark. However, if the sediment requirements of the test species are not met, observed mortality may not be due to chemical contaminants in the sediment.

On the other hand, toxicity tests have weaknesses also; they provide information only for the species tested, and also only for the endpoints measured. This is particularly critical given that most sediment toxicity tests conducted at the time of this writing measure primarily short-term lethality; chronic test procedures have been developed and published for some species, but these procedures are more resource-intensive and have not yet seen widespread use. In contrast, chemical-specific benchmarks are intended to protect most species against both acute and chronic effects.

Many assessments may involve comparison of sediment chemistry (e.g., using ESB values) and toxicity test results. In cases where results using these two methods agree (either both positive or both negative), the interpretation is clear. In cases where the two disagree, the interpretation is more complex; some investigators may go so far as to conclude that one or the other is "wrong," which is not necessarily the case.

Individual ESBs consider only the effects of the chemical or group of chemicals for which they are derived. For this reason, if a sediment shows toxicity but does not exceed the ESB for a chemical of interest, it is likely that the cause of toxicity is a different chemical or group of chemicals.

In other instances, it may be that an ESB is exceeded but the sediment is not toxic. As explained above, these findings are not mutually exclusive, because the inherent sensitivity of the two measures is different. ESBs are intended to protect relatively sensitive species against both acute and chronic effects, whereas toxicity tests are performed with specific species that may or may not be sensitive to chemicals of concern, and often do not encompass the most sensitive endpoints (e.g., chronic survival, growth or reproduction). It is also possible for a sediment above the ESB to be non-toxic if there are site-specific partitioning conditions that run counter to the equilibrium partitioning model and its assumptions (see Section 7.2).

The first step in interpreting this situation is to consider the magnitude of the ESB exceedance

and the sensitivity of the test organism and endpoint to the suspect chemical. For example, the acute-chronic ratio used for the PAH mixtures ESB is 4.16 (Section 3.3.7); as such, if $\Sigma ESBTU_{FCV} = 4$, one would anticipate lethal effects only for highly sensitive species. Between $\Sigma ESBTU_{FCV}$ of 1 and 4, one would expect only chronic effects, unless the test species was unusually sensitive. If $\Sigma ESBTU_{FCV}$ for PAHs was 2, for example, one would not generally expect to see lethality from PAHs in short term sediment lethality tests.

A more precise method for evaluating the results of toxicity tests is to calculate effect concentrations in sediment that are species specific. For species contained in the toxicity data for the PAH mixtures ESB (Section 3.2.1), effect concentrations in sediment can be calculated that are specific for that organism (using procedures in Section 4). These values could then be used to directly judge whether the absence of toxicity in the toxicity test would be expected from the corresponding level of sediment contamination.

If the exceedance of the PAH ESB is sufficient that one would expect effects in a toxicity test but they were not observed, it is prudent to initially evaluate the partitioning behavior of the chemical in the sediment based on sediment organic carbon content. Later evaluations may require evaluating partitioning based on other partitioning phases as described in Section 6.8. This is performed by isolation of interstitial water from the sediment and analyzing it for the same PAHs measured in the solid phase. Predicted concentrations of chemicals in the interstitial water can be calculated from the measured concentrations in the solid phase (normalized to organic carbon)

$$C_d = C_{OC} / K_{OC} \qquad (6\text{-}4)$$

For chemicals with $\log_{10}K_{OW}$ greater than 5.5, corrections for DOC binding in the interstitial water will be necessary

$$C_d = C_{OC}/K_{DOC} \qquad (6\text{-}5)$$

If the measured chemical in the interstitial water is substantially less (e.g., 2-3 fold lower or more), it suggests that the organic carbon in that sediment may not partition similarly to more typical organic carbon, and derivation of site-specific ESBs based on interstitial water may be warranted (U.S.EPA, 2003b).

6.5 Photo-Activation

6.5.1 Overview

Research over the last decade has shown that the presence of ultraviolet (UV) light can greatly enhance the toxicity of many PAHs. This "photo-activated" toxicity has been shown to cause rapid, acute toxicity to several freshwater and marine species including fish, amphibians, invertebrates, plants and phytoplankton (Bowling et al., 1983; Cody et al., 1984; Kagan et al., 1984; Landrum et al., 1984a,b; Oris et al., 1984; Allred and Giesy, 1985; Kagan et al., 1985; Oris and Giesy, 1985, 1986, 1987; Gala and Giesy, 1992; Huang et al., 1993; Gala and Giesy, 1994; Ren et al., 1994; Arfsten et al., 1996; Boese et al., 1997; Huang et al., 1997; McConkey et al., 1997; Pelletier et al., 1997; Diamond and Mount, 1998; Hatch and Burton, 1998; Boese et al.,1999; Monson et al., 1999; Spehar et al., 1999; Pelletier et al., 2000; Barron et al., 2003). Depending on the organism and exposure regime, photo-activation can increase toxicity of certain PAHs by one to four orders of magnitude over that caused by narcosis.

The mechanism for phototoxicity has been related to the absorption of ultraviolet radiation (UV) by the conjugated bonds of selected PAH molecules

$$PAH + UV \rightarrow PAH^* + O_2 \rightarrow PAH + O_2^* \qquad (6\text{-}6)$$

This excites the PAH molecules to a triplet state (PAH*) which rapidly transfers the absorbed energy to ground state molecular oxygen (O_2) forming excited singlet oxygen intermediaries (O_2*) (Newsted and Giesy, 1987). Although extremely short-lived (2 to 700 μs), oxygen intermediaries are highly oxidizing and can cause severe tissue damage upon contact. Despite the many different parent PAHs and related alkylated

forms, not all PAHs induce photo-activated toxicity. Those PAHs that are photo-activated can be predicted using various molecular physical-chemical variables (Newsted and Giesy, 1987; Oris and Giesy, 1987); however, the Highest Occupied Molecular Orbital - Lowest Unoccupied Molecular Orbital gap model (HOMO-LUMO) has been the most successful (Mekenyan et al., 1994a,b; Veith et al., 1995a,b; Ankley et al., 1996; Ankley et al., 1997). As research on the nature of photo-activated toxicity has evolved, certain key elements of this phenomena have been better defined including interactions of UV and PAH dose, effects of temperature, humic substances, organism behavior, turbidity, dissolved oxygen, mixtures, photoperiod and additivity (Oris et al., 1990; McCloskey and Oris, 1991; Ankley et al., 1995, 1997; Ireland et al., 1996; Hatch and Burton,1998, 1999; Erickson et al., 1999; Nikkilä et al., 1999; Weinstein and Oris 1999, Weinstein 2002).

Several studies have been performed with sediments contaminated with PAHs to assess the importance of photo-activated toxicity in the benthos (Davenport and Spacie, 1991; Ankley et al., 1994; Monson et al., 1995; Sibley et al., 1997; Swartz et al., 1997; Boese et al., 1998; Kosian et al., 1998; Boese et al., 1999; Spehar et al., 1999; Wernersson et al., 1999; Pelletier et al., 2000). These studies conclude that photo-activated toxicity may occur in shallow water environments; however, the magnitude of these effects are not as well characterized as in water-only exposures and are probably not as dramatic as those observed in the water column. Comparisons by Swartz et al. (1995) suggest that responses of benthic communities in PAH-contaminated sites correlate well with the toxicity that is predicted based on narcosis, suggesting that photo-activation was not a major confounding factor for those environments. However, Boese et al., (1997) and Pelletier et al. (In prepartion) show that life history of benthic organisms is critical to assessing whether or not photo-activated toxicity will occur. For example, several marine species which frequently encounter ultraviolet radiation during low tide are not vulnerable to photo-activated toxicity due to light protective adaptation (e.g., shells, pigments, borrowing). Additionally, there is evidence that maternal transfer of PAHs from benthic adult bivalves to pelagic embryos does occur (Pelletier et al., 2000).

6.5.2 Implications to Derivation of ESB

Because the PAH mixture ESB derived here is based on narcosis, if there is additional toxicity caused by photo-activation it may cause the ESB to be underprotective. At present, the magnitude of potential errors can not be specifically quantified, and are the subject of scientific debate (Swartz et al., 1997; Boese et al., 1999; Diamond and Mount, 1998; McDonald and Chapman, 2002). If photoactivation of PAHs is ecologically relevant, it is probably most significant primarily for organisms that inhabit very shallow or very clear water. This is because of the rapid attenuation of ultraviolet radiation in the water column (Pickard and Emery 1982; Wetzel, 1983). For example, <25% of incident UV penetrates below the first meter of water in productive aquatic systems. In areas where PAH-contaminated sediments are present in shallow environments the risk of photo-activated toxicity is greater and a site-specific ESB may need to be generated that considers this potential risk (U.S. EPA, 2003b).

6.6 Teratogenicity and Carcinogenicity

This subsection presents an analysis intended to determine if the ESB for PAH mixtures of <1.0 $\Sigma ESBTU_{FCV}$ is protective for non-narcosis modes of toxic action of individual PAHs. Published articles were screened for applicable data on teratogenic (Appendix G) and carcinogenic (Appendix H) effects of individual PAHs and their mixtures. Five laboratory studies with benzo(a)pyrene (BaP), predominantly water exposures, and one with anthracene were selected for analysis of teratogenic effects; two laboratory studies with BaP were selected for analysis of carcinogenic effects (Table 6-4). In the teratogen studies, typically radio-labeled BaP was used to quantify the accumulation of the PAH and its metabolites in fish ranging in lifestage from

Table 6-4. Teratogenic and carcinogenic effects of benzo(a)pyrene (BaP) and anthracene on freshwater and saltwater fishes. Measured concentrations of exposure are converted to sediment concentrations (C_{OC}) likely to result in the equivalent effect using EqP and SAR methodology.

Organism/ Chemical	Measured C_d[A] (µg/L)	C_d-derived C_{OC} (µg/g_{OC})	Measured C_{ORG}[B] (µg/g)	f_{Lipid}	C_L[B] (µg/g Lipid)	C_L-derived C_{OC} (µg/g_{OC})	References
TERATOGENIC EFFECTS							
FRESHWATER							
Fathead minnow eggs Anthracene	-	-	8.8	0.06	147	219	Hall and Oris, 1991
Topminnows BaP	>3.81[C] (1,000)	>3810	9	0.06	150	256	Goddard et al., 1987
Rainbow trout eggs BaP	0.21	210	1.9	0.05	38.6	66	Hannah et al., 1982 Hose et al., 1984
SALTWATER							
English sole eggs BaP	-	-	157	0.03	5233[D]	8,937[D]	Hose et al., 1981
Sand sole eggs BaP	0.1	100	2.1	0.03	70	120	Hose et al., 1982
Calif. grunion eggs	>3.81 (5)	>3810	1	0.03	33.3	57	Winkler et al., 1983
Calif. grunion eggs	>3.81 (24)	>3810	10.5	0.03	350	598	Winkler et al., 1983
Calif. grunion eggs	>3.81[C] (869)	>3810	20	0.03	666	1137	Winkler et al., 1983
CARCINOGENIC EFFECTS							
FRESHWATER							
Japanese medaka	>3.81[C] (261)	>3840	-	-	-	-	Hawkins et al., 1988, 1990
Guppy	>3.81[C] (209)	>3840	-	-	-	-	Hawkins et al., 1988, 1990

[A] If the concentration of BaP exceeded its solubility of 3.81 μg/L, the published concentration in water is listed in parenthesis with the solubility of 3.81 μg/L listed above as the concentration of exposure. Therefore the maximum C_{OC} value for these exposures is 3840μg BaP/g_{OC}.

[B] Concentrations in eggs on a wet weight basis are converted to concentrations on a lipid basis using lipid concentrations (f_{Lipid}) from Table 1 in Kamler (1992).

[C] Water concentrations of BaP were not stable throughout the duration of the experiment.

[D] The solubility of BaP in water theoretically limits the maximum concentration in eggs to ~3,840 μg/g lipid and in sediments to ~3,840 $\mu g/g_{OC}$, but metabolites of BaP will likely be included in radio-labeled quantification of total BaP equivalents.

embryo to adults. The water PAH concentrations associated with teratogenic and carcinogenic effects were generally high and steady-state was not always achieved. The solubility limit in water for BaP of 3.81 µg/L was exceeded in 6 of 8 experiments (Table 6-4). In contrast, for seven of the experiments, the BaP concentration in eggs or fish tissue was also listed as an observed effect concentration. The theoretical solubility-limited maximum of 3840 µg BaP/g lipid was exceeded only in one of the experiments. For these reasons, when the concentration of BaP plus metabolites was measured in the eggs or tissue of the organism, this concentration was considered the most valid representation of the true observed exposure concentration and the water concentration was not used in further analysis. Elutriates from crude oil contained non-PAH compounds and the relationship of total PAH concentrations in the study vs total PAH as defined in this document were difficult to determine in the Carls et al. (1999) study; therefore, these data were also excluded from this analysis. Although metabolism of PAHs is known to occur in invertebrates such as polychaetes, mollusks and crustaceans (McElroy et al., 2000), data on the potential carcinogenic effects of the metabolites is unknown.

As indicated in Table 6-4 and Appendix H, the database for carcinogenic effects of PAHs on aquatic (fish) species from laboratory studies is limited. Most of the available data are from studies of epizootic outbreaks of neoplasia (tumors) from highly contaminated field sites such as the Black River, Ohio (see Baumann and Harshbarger 1998 for a review) or Puget Sound, WA (Malins et al., 1987, Myers et al., 1990), to mention only a notable few. The applicability of these field studies to a causal relationship between carcinogenic effects observed and PAH concentrations is limited by the possible interactive effects of the PAHs with PCBs and other simultaneously occurring chemicals. The bulk of laboratory experimental evidence for carcinogenic effects of PAHs is based on the distribution of neoplasms in fish species exposed to PAH-enriched sediment extracts (Black, 1983; Metcalfe et al., 1988; Fabacher et al., 1991), dietary exposures or inter-peritoneal injection (Hendricks et al., 1985), or intermittent water exposures of 7,12-dimethylbenzanthracene (Schultz and Schultz, 1982). These studies are listed in Appendix H for completeness, but were not included in Table 6-4 for further analysis. This is because the exposure regime or concentrations of individual or mixtures of PAHs were not provided in sufficient detail to permit critical measured sediment concentrations, or sediment concentrations derived from concentrations in water or tissue, to be compared to the observed carcinogenic effects. The study with 7,12-dimethylbenzanthracene (Schultz and Schultz, 1982) was not considered for analysis because this PAH is not commonly measured as part of environmental monitoring programs (see Table 6-2).

A far more extensive database exists on the influence of PAHs on various aspects of tumor biology, such as PAH-DNA adduct formation and phase I (oxidation, reduction, and hydrolysis reactions) and phase II (glucuronidation and glutathione conjugation) metabolism of individual compounds. However, as indicative of cytotoxicity as these biomarkers may or may not be, they have been excluded from the analysis for the explicit purposes of this subsection. The methods of PAH exposure that were useful for this analysis were aqueous (Hannah et al., 1982; Hose et al., 1982, 1984; Winkler et al., 1983; Goddard et al., 1987; Hawkins et al., 1988, 1990), maternal (Hall and Oris, 1991), or inter-peritoneal injection of adult English sole (Parophrys vetulus) followed by measurement of concentrations in embryos (Hose et al., 1981).

6.6.1 Calculations

When the measured concentration of the PAH dissolved in water (C_d; µg/L) associated with a teratogenic or carcinogenic effect was available it was multiplied by its K_{OC} (L/kg_{OC}) x 10^{-3} to derive an equivalent effect concentration in sediment (C_d-derived C_{OC}; $\mu g/g_{OC}$), as per the EqP methodology (Table 6-4; Appendix G and H). When the measured concentration of the PAH in eggs or tissue (C_L; μg PAH/g lipid) associated with an

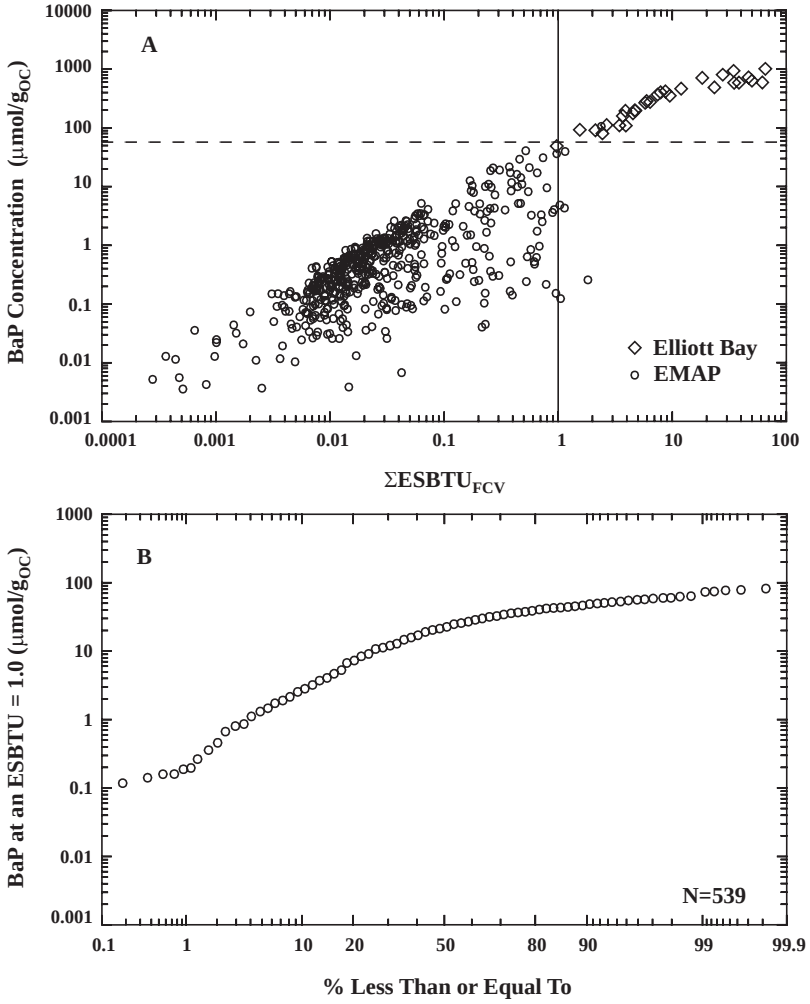

Figure 6-3. BaP concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the ΣESBTU$_{FCV}$ values of 34 PAHs and (B) a probability plot of these BaP concentrations at an ΣESBTU$_{FCV}$ = 1.0.

effect was available, its equivalent effect concentration in sediment (C_L-derived C_{OC}; μg/g_{OC}) was calculated using the equation.

$$\log_{10}C_{OC} = 0.00028 + \log_{10}C_L + 0.038\log_{10}K_{OW} \qquad (6\text{-}7)$$

6.6.2 Critical Sediment Concentrations for Teratogenic and Carcinogenic Effects versus ESBs for PAH Mixtures

The critical sediment concentrations (i.e., C_d- or C_L-derived C_{OC}) that would be expected to cause teratogenic or carcinogenic effects on the five freshwater and three saltwater fishes exposed to BaP ranged from 57 to 8,937 µg/g_{OC}; the only C_{OC} for anthracene was 219 µg/g_{OC} (Table 6-4). The majority of C_{OC} values were derived using concentrations measured in fish eggs. Six of the nine C_{OC} concentrations for BaP were less than the solubility-limited maximum concentration of 3,840 µg/g_{OC}. The C_{OC} value of 8,937 µg /g_{OC} is retained because the concentrations in the eggs probably included metabolites of BaP that are quantified as total BaP equivalents in the radio-label analysis. The C_{OC} values for individual PAHs in sediments were then compared to PAH concentrations in monitored field sediments to determine if teratogenic or carcinogenic effects might occur in sediments having <1.0 ΣESBTU$_{FCV}$. This analysis was used to determine if the ESB derived from the narcosis mode of action was protective of teratogenic or carcinogenic effects.

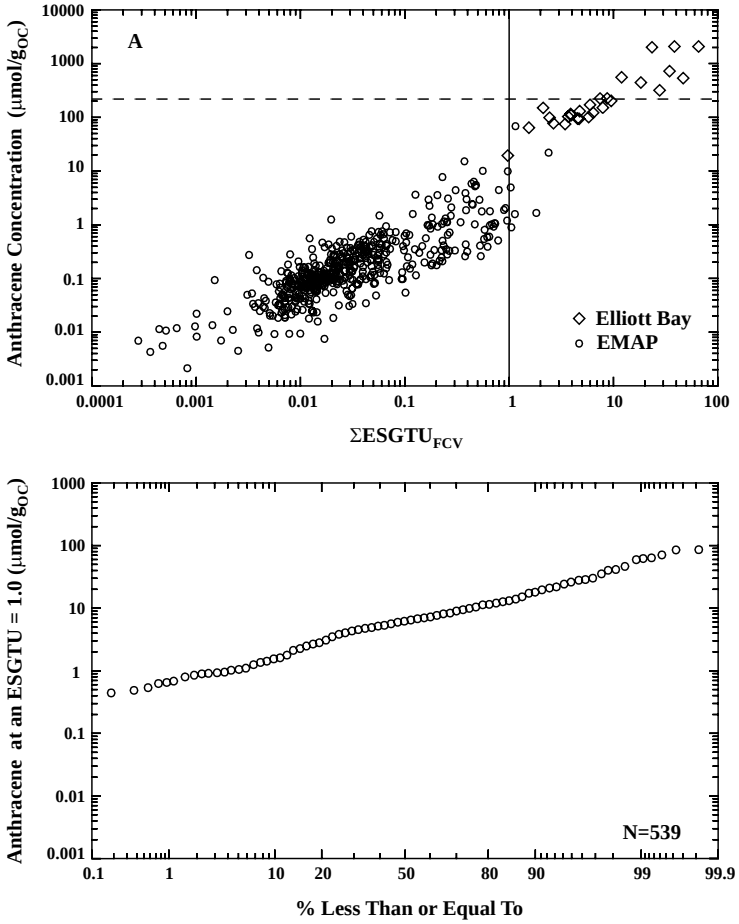

Figure 6-4. Anthracene concentration of 539 sediment samples from the EMAP and Elliott Bay datasets versus (A) the ΣESBTU$_{FCV}$ values of 34 PAHs and (B) a probability plot of these anthracene concentrations at an ΣESBTU$_{FCV}$ 1.0.

The database from the U.S. EPA EMAP (U.S. EPA 1996b, 1998) and Elliot Bay (Ozretich et al., 2000) sediment monitoring programs were used to compare the BaP (Figure 6-3A) or anthracene (Figure 6-4A) concentration of 539 sediment samples where 34 PAHs, or 33 of 34 PAHs for Elliott Bay, were measured versus the ΣESBTU$_{FCV}$ for all PAHs measured in those sediments. The lowest critical sediment concentration for teratogentic or carcinogenic effects is indicated with a solid line at 57 µg/g$_{OC}$ for BaP and at 219 μg/g$_{OC}$ for anthracene. None of the sediments having <1.0 ΣESBTU$_{FCV}$ contained BaP or anthracene at concentrations likely to cause the teratogenetic or carcinogenic effects reported in Table 6-4. The same database of PAH concentrations in field sediments was used to calculate the sediment-specific BaP:ΣESBTU$_{FCV}$ ratio and the sediment-specific anthracene: ΣESBTU$_{FCV}$ ratio. The total PAH concentration in each of the 539 sediments was multiplied by its sediment-specific ratio to determine the BaP or anthracene concentration for the sediment if the ΣESBTU$_{FCV}$ was equal to 1.0. Probability plots of the calculated concentrations for BaP and anthracene at 1.0 ΣESBTU$_{FCV}$ are in Figures 6-3B and 6-4B, respectively. The dashed lines represent the critical sediment concentration of 57 µg/g$_{OC}$ for BaP and 219 µg/g$_{OC}$ for anthracene. Based on this analysis, none of the sediments for anthracene and only 3.53% of the sediments for BaP would be expected to produce teratogenic or carcinogenic effects if the proportions of BaP or anthracene in these sediments were maintained and the concentrations of each of the other PAHs were increased so that all sediments contained 1.0

$\Sigma ESBTU_{FCV}$. The approach of examining these relationships individually with BaP or anthracene may be flawed because it may under-represent the teratogenic of carcinogenic contributions of other PAHs with the same mode of action in the PAH mixture. However, at present insufficient data are available to appropriately sum the contributions of multiple teratogenic or carcinogenic PAHs.

6.7 Equilibrium and ESBs

Care must be used in application of ESBs in disequilibrium conditions. In some instances site-specific ESBs may be required to address this condition (U.S. EPA, 2003b). Benchmarks based on EqP theory assume that nonionic organic chemicals are in equilibrium with the sediment and interstitial water, and that they are associated with the sediment primarily through absorption into sediment organic carbon. In order for these assumptions to be valid, the chemical must be dissolved in interstitial water and partitioned into sediment organic carbon. The chemical must, therefore, be associated with the sediment for a sufficient length of time for equilibrium to be reached. With PAHs, the absence of toxicity when the ESB is exceeded may be because of the presence of less available PAHs associated with soot, coal or similar materials in sediments (see discussion in Section 6.8). Alternatively, disequilibrium exists, and ESB may be over-protective, when PAHs occur in sediments as undissolved liquids or solids; although the use of solubility limited acceptable sediment concentrations should adequately account for this.

In very dynamic locations, with highly erosional or depositional sediments, the partitioning of nonionic organic chemicals between sediment organic carbon and interstitial water may only attain a state of near equilibrium. Likewise, nonionic organic chemicals with high $\log_{10}K_{OW}$ values may come to equilibrium in clean sediment only after a period of weeks or months. Equilibrium times are shorter for chemicals with low $\log_{10}K_{OW}$ values and for mixtures of two sediments with similar organic carbon-normalized concentrations, each previously at equilibrium. This is particularly relevant in tidal situations where large volumes of sediments are continually eroded and deposited, yet near equilibrium conditions between sediment and interstitial water may predominate over large spatial areas. For locations where times are sufficient for equilibrium to occur, near equilibrium is likely the rule and disequilibrium uncommon. In many environments, disequilibrium may occur intermittently, but in those cases ESBs would be expected to apply when the disturbance abates. In instances where long-term disequilibrium is suspected, application of site-specific methodologies may be desirable (U.S. EPA, 2003b).

6.8 Other Partioning Phases

6.8.1 Overview

In general, laboratory studies with PAHs have shown the same partitioning behavior demonstrated by many classes of nonpolar organic contaminants (Chiou et al., 1979,1983; Karickhoff et al., 1979; Means et al., 1980; Di Toro et al., 1991). However, there are some data indicating that PAHs do not always follow equilibrium partitioning behavior in the environment. Specifically, some studies have reported larger partitioning coefficients for PAHs in field-collected sediments than is predicted based on laboratory or theoretically-generated $\log_{10}K_{OW}/K_{OC}$ values (Prahl and Carpenter, 1983; Socha and Carpenter, 1987; Broman et al., 1990; McGroddy and Farrington, 1995; Maruya et al., 1996; McGroddy et al., 1996). The observed differences in partitioning of PAHs may relate to differences in PAH sources with the speculation that PAHs from pyrogenic sources (e.g., soot carbon, coal or similar materials) may be more strongly associated with the particulate phase than PAHs from some petrogenic sources (Readman et al., 1984; Socha and Carpenter, 1987; McGroddy and Farrington, 1995; Meador et al., 1995; Naes et al., 1995; Chapman et al., 1996; Maruya et al., 1996; McGroddy et al., 1996; Gustafsson and Gschwend, 1997; Gustafsson et al., 1997; Naes and Oug, 1997; de Maagd et al. 1998; Naes and Oug 1998;

Naes et al., 1998; Bucheli and Gschwend 2000; Jonker and Smedes 2000; Ozretich et al., 2000; Jonker and Koelmans 2001, 2002a,b; Accardi-Dey and Gschwend 2002, 2003). The result is that PAH concentrations in interstitial water are significantly lower compared to the organic carbon-based sediment concentration from laboratory or theoretically-predicted K_{OC} values and, presumably, exhibit correspondingly lower bioavailability. Several studies have proposed that the lack of observable biological effects from sediments (and other samples) containing high concentrations of presumably bioavailable PAHs is related to this phenomena (Farrington et al., 1983; Varanasi et al., 1985; Bender et al., 1987; Hickey et al., 1995; Knutzen, 1995; Chapman et al., 1996; Paine et al., 1996; Maruya et al., 1997; Oug et al., 1998; Lamoureux and Brownawell 1999; Naes et al., 1999; West et al., 2001; Talley et al., 2002).

The mechanisms causing these field observations of unusual PAH partitioning are not well understood. One explanation proposes that PAHs condense into the soot matrix during particle formation, and are thereby sterically inhibited from partitioning to interstitial water as would be expected under equilibrium conditions. A second perspective assumes that the soot fraction represents a second partitioning phase in addition to normal organic carbon. The partitioning of PAHs from this phase approximates the equilibrium behavior assumed for normal organic carbon, but have a much higher partition coefficient than biologically-derived organic carbon (represented by K_{OC}) (Gustafsson and Gschwend, 1997, 1999). Methods for measuring the soot carbon fraction in sediments (f_{sc}) continue to be developed and evaluated (Verardo 1997; Gustafsson et al., 1997; Karapanagioti et al., 2000; Gelinas et al., 2001; Currie et al., 2002; Gustafsson et al., 2001; Song et al., 2002) but no one method is recognized as most accurate, although those based on Gustaffson et al. (1997) are probably used most frequently.

Once partition coeffifients are available and f_{SC} can be measured, the soot phase can then be incorporated into an expanded partitioning equation with two partitioning terms

$$K_p = f_{OC} K_{OC} + f_{SC} K_{SC} \qquad (6\text{-}8)$$

where, K_p is the partition coefficient for the expanded partitioning equation, f_{OC} and f_{SC} are the fraction organic carbon and fraction soot carbon, respectively, and K_{OC} and K_{SC} are the organic carbon and soot carbon partition coefficients. Recently, Bucheli and Gustaffson (2000) and Accardi-Dey and Gschwend (2002; 2003) proposed a new version of Equation 6-8 which includes a non-linear term for the soot carbon contribution to partitioning

$$K_p = f_{OC} K_{OC} + f_{SC} K_{SC} C_d^{n-1} \qquad (6\text{-}9)$$

where, the exponential 'n' is the Freundlich term used to fit the non-linear relationship between particulate and dissolved PAH. This description of the interaction of PAHs and soot carbon is more accurate but is currently limited in practicality by the lack of values for K_{SC} and n for many PAHs.

Another phase for which there is less data available as compared to soot carbon but which may also alter the partitioning and bioavailability of PAHs is non-aqueous phase liquids (NAPLs) like coal tar found at manufactured gas plant sites (Lane and Loehr 1992; Luthy et al., 1994; Mahjoub et al., 2000). The significance of these liquids relative to the benthic toxicity of PAHs is not yet understood fully.

6.8.2 Implications to Derivation of ESB

Irrespective of the mechanisms, these issues have the potential to affect the predictive power and accuracy of the PAH mixtures ESB. For soot, coal and similar materials, their presence are associated with reduced concentrations of PAH in interstitial water, one would presume that this results in decreased bioavailability of PAHs, a phenomenon demonstrated by West et al. (2001). This, in turn, would make the PAH mixtures ESB derived here overprotective, because the K_{OC}-based partitioning model would overpredict chemical activity and, therefore, concentrations of PAH in interstitial water and in organisms.

Importantly, most sediments are expected to

contain insufficient concentrations of PAHs to exceed the ESB. Therefore, even if partitioning to soot, coal and similar materials reduces the interstitial water concentration and biological availability of the PAHs, the partitioning effect is not important because PAH concentrations in the sediment are judged by the ESB as acceptable without invoking complex measurements of partitioning to soot, coal and similar materials. Further, most sediments where empirical data on partitioning that demonstrates soot, coal and similar materials are important are sediments that relative to the ESB are uncontaminated. Also, for sediments that have concentrations of PAHs in excess of the ESB, data suggest minimal error in ignoring partitioning to soot, coal and similar materials and ascribing partitioning to only organic carbon. Most applications of the PAH mixture narcosis model to toxicity data for field-collected sediments show good predictive ability for the ESB (see Section 5.3). This may be because most sediments that are sufficiently contaminated to cause narcosis are contaminated by PAH sources that exhibit normal partitioning behavior, such as creosote and other petrogenic sources. In their study of PAH-contaminated sediments, Ozretich et al. (2000) found that discrepancies between measured and predicted partitioning behavior predominated in sediments with lower PAH concentrations, while those with higher PAH concentrations showed partitioning behavior closer to that predicted from published K_{OW}/K_{OC} relationships. This differential behavior was attributed to the presence of two PAH sources, with creosote being the source causing the highest levels of contamination and toxicity.

In cases where it is suspected that soot, coal, or other materials including coal tars and other NAPLs may be causing unusual partitioning, direct measurement of PAH concentrations in interstitial water may be used to evaluate this possibility and, where necessary, derive site-specific sediment benchmarks which account for local differences in partitioning behavior (see U.S. EPA 2003b).

6.9 Aqueous Solubility Under Non-Standard Conditions

It has been long established that organic compounds are generally less soluble in aqueous solutions at colder temperatures than at warmer, and in salt solutions such as seawater, than in freshwater, a phenomenon termed the salting-out effect (May, 1980; Schwarzenbach et al., 1993; Xie et al., 1997). Setschenow (1889) derived an empirical relationship for the magnitude of the salting-out effect

$$\log_{10}({}^{t}S_0 / {}^{t}S_{‰}) = K_S\, C_{salt} \qquad (6\text{-}9)$$

where ${}^{t}S_0$ and ${}^{t}S_{‰}$ are the aqueous solubilities of the solute in fresh and saltwater (mol/L) at a given temperature (t in the units °C), respectively, K_S is

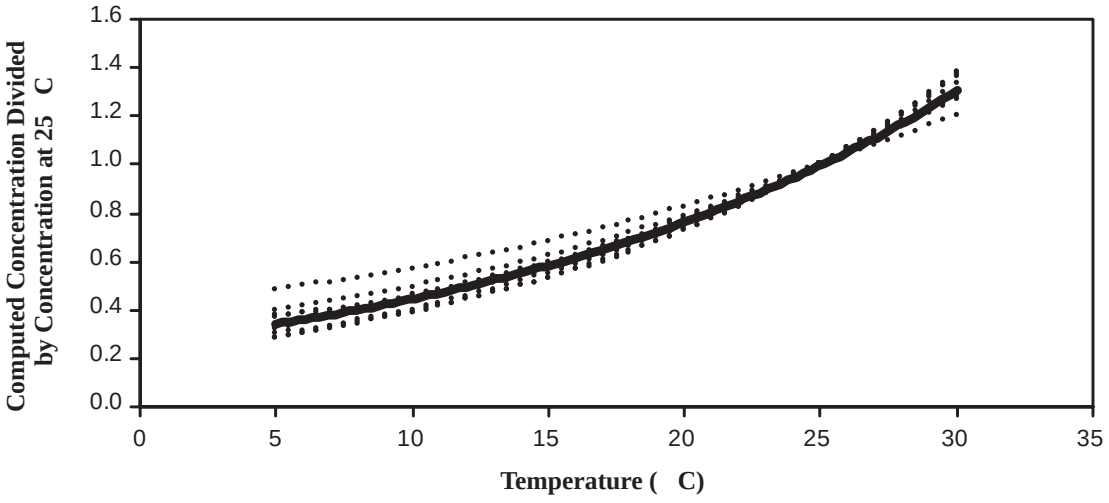

Figure 6-5. Computed solubilities of nine PAHs relative to their 25°C solubilities as a function of temperature.

the Setschenow constant (L/mol) for the salt solution and the solute of interest, and C_{salt} is the molar salt concentration. A one molar salt solution (NaCl) is approximately equivalent to 48‰ sea water (Owen and Brinkley, 1941), and K_S was found to be essentially invariant with temperatures from 1 to 30°C, averaging 0.28 ± 0.02 (mean ± SE) (May, 1980) for 9 PAHs. Temperature has been shown to have a non-linear effect on PAHs solubilities (May, 1980). Concentrations of nine PAHS (naphthalene, fluorene, phenanthrene, 1-methylphenanthrene, anthracene, fluoranthene, pyrene, benz(a)anthracene, and chrysene) were computed for distilled water at temperatures between 5 and 30°C using the relationships of May (1980) and are compared with the compound's concentrations at 25°C (Figure 6-5). The least-squares exponential representation of the data is as follows

$$({}^{t}S_0 = {}^{25}S_0) = 0.261\, e^{0.0536t},\ r^2 = 0.959 \qquad (6\text{-}10)$$

where ${}^{25}S_0$ is the commonly reported solubility of a compound at 25°C in freshwater. Although naphthalene's solubility has the least response to temperature of PAHs, estimates from Equation 6-10 are only +8% and -30% inaccurate for naphthalene at the temperature extremes (Figure 6-5).

The solubility of PAHs under environmental conditions can be estimated from the following relationship that is a combination of Equations 6-9 and 6-10 using the average Setschenow constant

$${}^{t}S_{‰} = {}^{t}S_0\ 10^{-0.000583‰} \qquad (6\text{-}11)$$

where ‰ is the salinity of the sea water. This correction for solubility can be used as part of the procedures to modify this ESB for site-specific conditions.

Section 7

Sediment Benchmark Values: Application and Interpretation

7.1 Benchmark Value

The procedures described in this document and in the "Technical Basis for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Nonionic Organics" (U.S. EPA, 2003a) indicate that, except possibly where a locally important species is very sensitive or benthic organisms are exposed to both significant amounts of PAHs and UV light, benthic organisms should be acceptably protected from the effects of PAH mixtures in freshwater and saltwater sediments if the $EESBTU_{FCV}$ is less than or equal to 1.0:

$$ESB = \Sigma ESBTU_{FCV} = \sum_{i} \frac{C_{OCi}}{C_{OC,PAHi,FCVi}} \leq 1.0 \qquad (7\text{-}1)$$

Freshwater or saltwater sediments containing ≤ 1.0 $\Sigma ESBTU_{FCV}$ of the mixture of the 34 PAHs or more PAHs are acceptable for the protection of benthic organisms, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be unacceptably affected. PAHs.

As indicated, this sediment-specific benchmark is the sum of the quotients of the concentrations of individual PAHs in a sediment, on an organic carbon basis, each divided by its respective $C_{OC,PAHi,FCVi}$. At a minimum, the definition of total PAHs for this ESB requires quantification of the 34 PAHs analyzed by the U.S. EPA as part of the EMAP and REMAP programs (PAHs are identified in bold in Table 3-4).

The ESB is intended to protect benthic organisms from direct toxicity associated with exposure to PAH-contaminated sediments. The ESB does not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAHs or the potential for bioaccumulation and trophic transfer of PAHs to aquatic life, wildlife or humans.

7.2 Special Considerations

To establish a national benchmark that is widely applicable, certain issues must be considered. It is possible that site-specific conditions may affect the broad applicability of such a benchmark. These include:

1. Fewer than 34 PAHs have been measured. Particularly in cases where historical data are being examined, chemistry data may be available for fewer than the 34 PAHs recommended for this benchmark. Calculating $\Sigma ESBTU_{FCV}$ directly using fewer PAHs will cause the benchmark to be underprotective because PAH mixtures found in the environment typically contain substantial concentrations of PAHs outside the suites of 13 or 23 PAHs commonly measured in monitoring programs. The analysis of PAH distributions across many geographic regions has been used to develop uncertainty factors that can be used to adjust $\Sigma ESBTU_{FCV}$ based on subsets of 13 or 23 PAHs with varying degrees of certainty (see Section 6.2). In some applications using these uncertainty factors, it may be important to minimize the frequency of false negatives (sediments judged to be acceptable when they are not). For these cases, the $\Sigma ESBTU_{FCV}$ calculated from a subset of 13 PAHs (see Table 6-1 for listing) can be multiplied by 11.5, or the $\Sigma ESBTU_{FCV}$ calculated from a subset of 23 PAHs

(see Table 6-1 for listing) can be multiplied by 4.14 to achieve 95% confidence that the actual $\Sigma ESBTU_{FCV}$ for all 34 PAHs would not be higher than the calculated value. In this case, the uncertainty for the 95% confidence level is applied. This means that most of the sediments may actually contain fewer $\Sigma ESBTU_{FCV}$ than indicated by the calculation. In cases where less conservative assumptions are appropriate, factors with lower confidence can be applied, as detailed in Section 6.2.

Use of the uncertainty factors from Section 6.2 assumes that the relative frequency distributions of PAHs in sediments used to calculate the factors are similar to those of the sediments to which the uncertainty factors are applied. This assumption is likely significantly violated for sediments containing predominately petrogenic PAHs. While the uncertainty factors can be used to derive the $\Sigma ESBTU_{FCV}$, this value should not be considered as an ESB. In principal, ESBs based on the $\Sigma ESBTU_{FCV}$ calculated using a minimum of the 34 specified PAHs and can be used to make important sediment decisions. In contrast, important sediment decisions should not be made using $\Sigma ESBTU_{FCV}$ values when fewer PAHs, such as the 13 or 23 PAHs commonly quantified, and uncertainty factors. To avoid errors introduced by the use of uncertainty factors, wherever possible, a more complete PAH chemical analysis should be undertaken with concentrations for a minimum of the 34 specified PAHs analyzed.

2. Interaction of PAHs with UV light. Benchmarks calculated in this document are based on narcotic toxicity only and do not consider enhanced toxicity that can occur if PAH-exposed organisms are simultaneously exposed to UV light. In environments where significant sunlight penetrates to the sediment and benthic organisms are exposed to UV light, the ESB may be underprotective. Consult Section 6.5 for additional details.

3. Influence of soot carbon and coal on PAH partitioning. PAHs may partition less to interstitial water in sediments that contain soot and/or coal particles or similar materials that expected with typical organic carbon partitioning. This could cause the benchmark to be overprotective. The influence of these phases can be assessed by measuring concentrations of PAHs directly in interstitial water and comparing these measures with concentrations predicted by EqP or through quantification of partitioning to these other sediment phases. See Section 6.8 and the site-specific ESBs (U.S. EPA, 2003b) for further discussion. NAPLs are not directly addressed by this document, but may be expected to result in reduced interstitial water concentrations of PAHs.

4. Unusual composition of organic carbon. Partition coefficients used for calculating the national PAH mixture ESB are based on measured partitioning from natural organic carbon in typical field sediments. Some sediments influenced heavily by industrial activities may contain sources of organic carbon whose partitioning properties are not similar, such as rubber, animal processing wastes (e.g., hair or hide fragments), or wood processing wastes (bark, wood fiber or chips). Relatively undegraded woody debris or plant matter (e.g., roots, leaves) may also contribute organic carbon that results in partitioning different from that of typical organic carbon. Sediments with large amounts of these materials may show higher concentrations of chemicals in interstitial water than would be predicted using generic K_{OC} values, making the ESB underprotective. Direct analysis of interstitial water can be used to evaluate this possibility (see U.S. EPA, 2003a,b).

5. Presence of additional narcotic compounds. The PAH mixture ESB is based on the additivity of the narcotic toxicity of PAHs. However, some sediments may contain additional nonionic narcotic chemicals that would contribute to narcotic toxicity, such as chlorobenzenes or PCBs (note: PCBs may also cause adverse effects through bioaccumulation and transfer to higher trophic levels; these bioaccumulative effects are not addressed by this narcosis-based ESB and should be evaluated separately). The presence of additional nonionic narcotic chemicals may make the PAH mixture ESB underprotective, because the ESB itself only addresses that part of the narcotic potency caused by PAHs. Di Toro et al.

(2000) and Di Toro and McGrath (2000) describe methods by which the contributions of other narcotic chemicals can be incorporated into an ESB-type assessment.

6. Site-specific temperature and salinity corrections. Temperature and salinity both affect solubility of PAHs and can therefore affect the solubility-constrained maximum contribution of individual PAHs to the overall ESB. Solubilities used in this document are calculated for 25°C and salinities less than 1‰. Solubilities can be recalculated to meet site specific conditions using procedures described in Section 6.9. Within a temperature range of 0 to 35°C and salinities from 0 to 35‰, solubility can be expected to decrease by a factor of about 30 to 40% with decrease in temperature or increase in salinity. Site-specific recalculation of solubilities will only affect $\Sigma ESBTU_{FCV}$ in cases where the contribution of one or more PAHs are solubility constrained (see Section 6.9).

7.3 Summary

Benthic organisms should be acceptably protected from the narcotic effects of PAH mixtures in freshwater and saltwater sediments if the $\Sigma ESBTU_{FCV}$ is less than or equal to 1.0, and if the $\Sigma ESBTU_{FCV}$ is greater than 1.0, sensitive benthic organisms may be adversely affected**.** This ESB is intended to protect benthic organisms from direct toxicity associated with exposure to PAH-contaminated sediments. This ESB does not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with PAH mixtures or the potential for bioaccumulation and trophic transfer of PAH mixtures to aquatic life, wildlife or humans.

Section 8

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Appendix A

Individual datasets which Comprise the Acute Lethality Database: Table from Di Toro et al. (2000).

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Freshwater					
Paramecium, *Tetrahymena elliotti*	24	S	U	10(12)	Rogerson et al., 1983
Hydra, *Hydra oligactis*	48	S	U	5	Slooff et al., 1983
Snail, *Lymnae stagnalis*	48	S	U	5	Slooff et al., 1983
Cladoceran, *Daphnia cucullata*	48	S	U	5	Canton and Adema, 1978
Cladoceran, *Daphnia magna*	24	S	U	21(28)	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	48	S	U	72(78)	Abernethy et al., 1988; U.S. EPA, 1978; Canton and Adema, 1978 Rogerson et al., 1983; Bringman and Kuhn, 1959; Eastman et al., 1984; Dill, 1980
Cladoceran, *Daphnia magna*	48	S	U	19	EG&G Bionomics, 1982; Thurston et al., 1985; Adema, 1978; Oris et al., 1991; Brooke, 1991; Millemann et al., 1984; Munkrittrick et al., 1991
Cladoceran, *Daphnia magna*	48	FT,R	M	1(2)	EG&G Bionomics, 1982; Brooke, 1994
Cladoceran, *Daphnia pulex*	48	S	M	(1)	Trucco et al., 1983
Cladoceran, *Daphnia pulex*	48	S	U	6	Canton and Adema, 1978; Passino and Smith, 1987
Brine shrimp, *Artemia salina*	24	S	N	32(34)	Abernethy et al., 1988; Abernethy et al., 1986

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Crayfish, *Orconectes immunis*	96	FT	M	6	Thurston et al., 1985; Holcombe et al., 1987
Mosquito, *Aedes aegypti*	48	S	U	5	Slooff et al., 1983
Mosquito, *Culex pipiens*	48	S	U	5	Slooff et al., 1983
Midge, *Tanytarsus dissimilis*	48	S	M	9	Thurston et al., 1985; Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	48	FT	M	7	Holcombe et al., 1987; Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	24	FT	M	6	Call et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	24	S	U	1(2)	Bently et al., 1975
Rainbow trout, *Oncorhynchus mykiss*	48	S	U	6	Slooff et al., 1983; Bently et al., 1975
Rainbow trout, *Oncorhynchus mykiss*	96	FT	M	22	Thurston et al., 1985; Call et al., 1983; Holcombe et al., 1987; Call et al., 1986; DeGraeve et al., 1982; Hodson et al., 1988
Rainbow trout, *Oncorhynchus mykiss*	96	S	M	1	Horne et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	96	S	U	1	Bently et al., 1975
Bleak, *Alburnus alburnus*	96	S	I	7	Bengtsson et al., 1984

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Goldfish, *Carasius auratus*	24	S	M	26(28)	Bridie et al., 1979
Goldfish, *Carasius auratus*	24	S	U	5(6)	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	24	FT	M	1(2)	Brenniman et al., 1976
Goldfish, *Carasius auratus*	96	S	U	4	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	96	FT	M	1(2)	Brenniman et al., 1976
Goldfish, *Carasius auratus*	48	S	U	5(6)	Pickering and Henderson, 1966
Goldfish, *Carasius auratus*	48	FT	M	1(2)	Brenniman et al., 1976
Golden orfe, *Leuciscus idus melanotus*	24	S	i(ns)	26	Juhnke and Ludemann, 1978
Fathead minnow, *Pimephales promelas*	24	S	U	6	Pickering and Henderson, 1966
Fathead minnow, *Pimephales promelas*	24	FT	M	8	Ahmad et al., 1984
Fathead minnow, *Pimephales promelas*	48	S	U	11	Pickering and Henderson, 1966
Fathead minnow, *Pimephales promelas*	48	FT	M	8	Ahmad et al., 1984

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Fathead minnow, *Pimephales promelas*	96	FT	M	141(146)	Veith et al., 1983; Thurston et al., 1985; Holcombe et al., 1987; Ahmad et al., 1984; Dill,1980; DeGraeve et al., 1982; Alexander et al., 1978; Broderius and Kahl, 1985; Cairns and Nebeker, 1982; Hall et al., 1989; Hall et al., 1984; Call et al., 1985; CLSES, 1984; CLSES, 1985; CLSES, 1986; CLSES, 1988; CLSES, 1990; Kimball, 1978
Fathead minnow, *Pimephales promelas*	96	S	M	3(4)	Bridie et al., 1979; EG&G Bionomics, 1982; Gendussa, 1990; Horne et al., 1983
Fathead minnow, *Pimephales promelas*	96	R	U	1	Academy Natural Sci., 1981
Fathead minnow, *Pimephales promelas*	96	S	U	4	Pickering and Henderson, 1966
Channel catfish, *Ictalurus punctatus*	96	FT,S	M	7	Thurston et al., 1985; Holcombe et al., 1983; Gendussa, 1990
Medaka, *Oryzias latipes*	48	S	U	4(5)	Slooff et al., 1983
American flagfish, *Jordanella floridae*	24	FT	M	6	Smith et al., 1991
American flagfish, *Jordanella floridae*	48	FT	M	6	Smith et al., 1991
American flagfish, *Jordanella floridae*	96	FT	M	6	Smith et al., 1991
Mosquitofish, *Gambusia affinis*	24	S	U	(3)	Thurston et al., 1985
Mosquitofish, *Gambusia affinis*	48	S	U	(3)	Thurston et al., 1985

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Mosquitofish, *Gambusia affinis*	96	FT	M	5(6)	Thurston et al., 1985; Wallen et al., 1957
Mosquitofish, *Gambusia affinis*	96	S	U	3	Wallen et al., 1957
Guppy, *Poecilia reticulata*	24	S	U	(1)	Pickering and Henderson, 1966
Guppy, *Poecilia reticulata*	48	S	U	10(11)	Slooff et al., 1983; Pickering and Henderson, 1966
Guppy, *Poecilia reticulata*	96	S	U	4	Slooff et al., 1983
Bluegill, *Lepomis macrochirus*	24	S	U	18(19)	Pickering and Henderson, 1966; Buccafusco et al., 1981; Bently et al., 1975
Bluegill, *Lepomis macrochirus*	24	FT	M	1	Call et al., 1983
Bluegill, *Lepomis macrochirus*	48	FT	M	1	Call et al., 1983
Bluegill, *Lepomis macrochirus*	48	S	U	6(7)	Pickering and Henderson, 1966; Bently et al., 1975
Bluegill, *Lepomis macrochirus*	96	FT	M	8	Thurston et al., 1985; Bently et al., 1975; Call et al., 1983; Holcombe et al., 1987
Bluegill, *Lepomis macrochirus*	96	S	U	36(40)	Pickering and Henderson, 1966; U.S. EPA, 1978; LeBlanc, 1980b; ; Buccafusco et al., 1981; Bently et al., 1975; Dawson et al., 1977.
Tadpole, *Rana catesbeiana*	96	FT	M	5	Thurston et al., 1985

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Clawed toad, *Xenopus laevis*	48	S	U	5	Slooff and Baerselman, 1980
Mexican axolotl, *Ambystoma mexicanum*	48	S	U	5	Slooff and Baerselman, 1980
Saltwater					
Annelid worm, *Neanthes arenaceodentata*	96	S	U	4(5)	Horne et al., 1983; Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	96	R	U	(1)	Thursby et al., 1989a
Copepod, *Nitocra spinipes*	96	S	I	6	Bengtsson et al., 1984
Amphipod, *Leptocheirus plumulosus*	96	FT	M	4	Swartz, 1991a; Champlin and Poucher, 1992a; Boese et al., 1997
Mysid, *Americamysis bahia*	96	S	U	20(23)	U.S. EPA, 1978; Champlin and Poucher, 1992a; Zaroogian et al., 1985
Mysid, *Americamysis bahia*	96	S	M	1	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	96	R	U	1 8(9)	Thursby et al., 1989b
Mysid, *Americamysis bahia*	96	FT	M	8(9)	Battelle, 1987; Champlin and Poucher, 1992a; Horne et al., 1983; EG&G Bionomics, 1978; U.S. EPA, 1978; Kuhn and Lussier, 1987; Thursby, 1991b
Grass shrimp, *Palaemonetes pugio*	96	R	U	2	Battelle, 1987; Thursby et al., 1989a

Common Name, *Scientific Name*	Test Conditions				References
	Test Duration (hr)	Method[A]	Concentration[B]	No. of Data Points[C]	
Grass shrimp, *Palaemonetes pugio*	96	S	U	4	Champlin and Poucher, 1992a; Horne et al., 1983; Thursby, 1991b; Tatem et al., 1978
Grass shrimp, *Palaemonetes pugio*	96	FT	M	1	Battelle, 1987
Grass shrimp, *Palaemonetes pugio*	96	S	M	1	Tatem, 1977
Crab, *Portunus pelagicus*	96	S	M	4	Mortimer and Connell, 1994
Inland silverside, *Menidia beryllina*	96	R	U	1	Thursby et al., 1989a
Inland silverside, *Menidia beryllina*	96	S	U	7(8)	Champlin and Poucher, 1992a; Dawson et al., 1977; Horne et al., 1983
Sheepshead minnow, *Cyprinodon variegatus*	24	S	U	7(8)	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	48	S	U	11(12)	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	96	S	U	13(15)	Heitmuller et al., 1981: U.S. EPA, 1978
Sheepshead minnow, *Cyprinodon variegatus*	96	FT	M	2	Ward et al., 1981; Battelle, 1987
Total Data Points				736 (796)	

[A]Method: S=static, FT=flow-through, R=renewal

[B]Concentration: U=unmeasured (nominal), M=chemical measured, I=initial

[C]Number of data points used; ()=number of data before screening for concentration>solubility and outliers.

Appendix B

Chemicals which Comprise the Acute Toxicity Database for Narcosis Chemicals in Section 2 of this Document: Table from Di Toro et al. (2000).

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
triethylene glycol	112276	ao	-1.48	150.17	131	-
methanol	67561	ao	-0.715	32.04	41.0	13.5
2,4-pentanedione*	123546	k	-0.509	100.12	100	7.87
ethanol	64175	ao	-0.234	46.07	59.0	11.9
acetone	67641	k	-0.157	58.08	74.0	13.71
2-chloroethanol*	107073	ao	-0.048	80.51	65.0	9.09
2-(2-ethoxyethoxy)ethanol	111900	ao	0.011	134.17	111	-
1-chloro-2-propanol*	127004	ao	0.156	94.54	84.0	44.8
1,3-dichloro-2-propanol*	96231	ao	0.165	128.99	91.0	6.30
2-methyl-2,4-pentanediol	107415	ao	0.246	118.17	120	43.0
2-butanone	78933	k	0.316	72.11	90.0	2.81
2-propanol	67630	ao	0.341	60.10	77.0	13.6
3-chloro-1-propanol*	627305	ao	0.363	94.54	82.0	2.00
1-propanol	71238	ao	0.399	60.10	75.0	11.2
cyclopentanone	120923	k	0.453	84.12	89.0	1.11
2-methyl-2-propanol	75650	ao	0.663	74.12	95.0	16.5
methyl chloride	74873	al,ha	0.677	50.49	56.0	0.0666
2-butanol	78922	ao	0.717	74.12	93.0	14.9
methyl bromide*	74839	al,ha	0.791	94.94	57.0	0.154
3-methyl-2-butanone	563804	k	0.792	86.13	108	1.32
2,3-dibromopropanol*	96139	ao	0.819	217.90	96.0	5.97
cyclohexanone	108941	k	0.827	98.14	103	0.445
cyclopentanol	96413	ao	0.849	86.13	89.0	5.19
2-methyl-1-propanol	78831	ao	0.858	74.12	93.0	10.6
4-methyl-3-pente-2-one	141797	k	0.867	98.14	118	2.68
2-pentanone	107879	k	0.877	86.13	107	1.03
1-butanol	71363	ao	0.946	74.12	92.0	3.03
3-pentanone	96220	k	0.954	86.13	108	0.849
2-methyl-2-butanol	75854	ao	1.03	88.15	110	1.62
2-n-butoxyethanol	111762	ao	1.05	118.17	131	8.78
diethyleneglycolmono-n-butylether	112345	et	1.09	162.23	170	40.0
3,3-dimethyl-2-butanone	75978	k	1.09	100.16	125	0.954

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
diethyl ether	60297	et	1.15	74.122	105	1.16
4-methoxy-4-methyl-2-pentane	107700	k	1.17	130.19	143	41.5
4-methyl-2-pentanone	108101	k	1.17	100.16	124	0.862
dichloromethane	75092	al,ha	1.18	84.93	65.0	0.211
t-butylmethyl ether	1634044	et	1.20	88.149	122	9.04
cyclohexanol	108930	ao	1.29	100.16	103	1.61
2-hexanone	591786	k	1.29	100.16	124	0.598
1,2-dichloroethane	107062	al,ha	1.40	98.96	79.0	0.114
1-pentanol	71410	ao	1.49	88.15	109	0.581
3-methyl-3-pentanol	77747	ao	1.49	102.18	125	3.79
2-phenoxyethanol	122996	ao	1.50	138.17	122	0.173
2,2,2-trichloroethanol	115208	ao	1.61	149.4	93.0	48.4
4-methyl-2-pentanol	108112	ao	1.66	102.18	126	2.25
3-hexanol	623370	ao	1.66	102.18	125	2.18
2-heptanone	110430	ke	1.67	114.19	141	0.312
5-methyl-2-hexanone	110123	ke	1.68	114.19	141	0.271
2,4-dimethyl-3-pentanol	600362	ao	1.78	116.2	140	3.05
6-methyl-5-heptene-2-one	110930	ke	1.82	126.2	151	0.487
2-hexanol	626937	ao	1.83	102.18	126	1.13
1,3-dichloropropane	142289	al,ha	1.84	112.99	97.0	0.0363
1,2-dichloropropane	78875	al,ha	1.86	112.99	99.0	0.0342
diisopropyl ether	108203	et	1.87	102.18	138	0.0918
chloroform	67663	al,ha	1.91	119.38	81.0	0.0319
1,1,2-trichloroethane	79005	al,ha	1.91	133.4	94.0	0.0369
1,4-dimethoxybenzene	150787	ar	1.95	138.165	132	0.0250
2,6-dimethoxytolunene	5673074	ar	1.99	152.19	147	0.0283
benzene	71432	ar	2.00	78.11	89.0	0.0260
1-hexanol	111273	ao	2.02	102.18	125	0.159
2-octanone	111137	ke	2.02	128.21	157	0.111
1-chloro-3-bromopropane	109706	al,ha	2.04	157.44	100	0.0184
5-methyl-3-heptanone	541855	ke	2.05	128.21	156	0.111
anisole	100663	ar	2.06	108.14	111	0.0148

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
2,6-dimethyl-2,5-heptadiene	504201	ke	2.07	138.21	164	0.0171
t-1,2-dichloroethylene	156605	al,ha	2.10	96.94	81.0	0.0202
1,2,3-trichloroepropane	96184	al,ha	2.13	147.43	107	0.0177
1,1-dichloroethylene	75354	al,ha	2.19	96.94	81.0	0.0141
1,3-dibromopropane*	109648	al,ha	2.24	201.9	103	0.00930
bromoform	75252	al,ha	2.25	252.73	88.0	0.00650
1,1,2,2-tetrachloroethane	79345	al,ha	2.31	167.85	106	0.0181
1,4-dichlorobutane	110565	al,ha	2.33	127.01	113	0.00990
1,1-dichloropropane	78999	al,ha	2.36	112.99	101	0.00790
2-nonanone	821556	ke	2.38	142.24	174	0.0801
1,1,1-trichloroethane	71556	al,ha	2.38	133.4	101	0.00662
1,1,1,2-tetrachloroethane	630206	al,ha	2.43	167.85	110	0.0050
5-nonanone	502567	ke	2.44	142.24	174	0.0740
1-heptanol	111706	ao	2.57	116.2	142	0.0487
chlorobenzene	108907	ar,ha	2.58	112.56	102	0.00320
2-ethyl-1-hexanol	104767	ao	2.58	130.23	155	0.132
bicyclo(2,2,1)hepta-2,5-diene	121460	al	2.60	92.14	102	0.00490
toluene	108883	ar	2.62	92.14	107	0.00600
styrene	100425	ar	2.72	104.15	116	0.00550
tetrachloromethane	56235	al,ha	2.73	153.82	97.0	0.00248
2-decanone	693549	ke	2.73	156.27	190	0.0599
bromobenzene	108861	ar,ha	2.75	157.01	106	0.00196
cyclopentane	278923	al	2.76	70.134	95.0	0.00260
1,5-dichloropentane	628762	al,ha	2.76	141.04	130	0.00286
1,3,5-cycloheptatriene	544252	al	2.77	92.14	104	0.00377
trichloroethylene	79016	al,ha	2.81	131.39	90.0	0.00360
di-n-butyl ether	142961	et	2.89	130.23	170	0.00614
t-1,2-dichlorocyclohexane	822866	al,ha	2.90	153.05	128	0.00162
pentachloroethane	76017	al,ha	2.95	202.29	121	0.00111
2,4-hexadiene	592461	al	2.98	82.145	115	0.00237
butylphenyl ether	1126790	et	3.00	150.22	160	0.000790
benzophenone	119619	ke	3.05	182.22	163	0.000480

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
ethylbenzene	100414	ar	3.06	106.17	123	0.00219
2,3-dimethyl-1,3-butadiene	513815	al	3.06	82.145	121	0.00162
2-undecanone	112129	ke	3.08	170.29	207	0.0459
1-octanol	118875	ao	3.10	130.23	158	0.0161
3-chlorotoluene	108418	ar,ha	3.12	126.59	118	0.000834
4-chlorotoluene	106434	ar,ha	3.13	126.59	118	0.000817
o-xylene	95476	ar	3.13	106.17	121	0.00191
m-xylene	108383	ar	3.19	106.17	124	0.00154
p-xylene	106423	ar	3.21	106.17	124	0.00146
1,4-dichlorobenzene	106467	ar,ha	3.24	147.00	113	0.000581
3,5,5-trimethyl-1-hexanol	3452979	ao	3.29	144.26	172	0.0117
1,2-dichlorobenzene	95501	ar,ha	3.31	147.00	113	0.000507
1,3-dichlorobenzene	541731	ar,ha	3.31	147.00	115	0.000524
napthalene	91203	pah	3.36	128.17	125	0.00110
cyclohexane	110827	al	3.38	84.16	109	0.000919
tetrachloroethylene	127184	al,ha	3.38	165.83	99.0	0.000710
2-dodecanone	6175491	ke	3.43	184.32	223	0.0357
cumene	98828	ar	3.49	120.19	140	0.000762
pentane	109660	al	3.50	72.15	116	0.000592
1,2-dibromobenzene	585539	ar,ha	3.56	235.92	119	0.000196
1,5-cyclooctadiene	111784	al	3.61	108.18	130	0.000386
1-nonanol	143088	ao	3.63	144.26	175	0.00552
1,2,4-trimethylbenzene	95636	ar	3.65	120.19	138	0.000487
n-propylbenzene	103651	ar	3.67	120.19	140	0.000467
dipentyl ether	693652	et	3.69	158.28	202	0.000757
1,3,5-trimethylbenzene	108678	ar	3.69	120.19	140	0.000414
hexachloroethane	67721	al,ha	3.73	236.74	132	0.0000936
2,4-dichlorotoluene	95738	ar,ha	3.79	161.03	129	0.000457
1-methylnaphthalene	90120	pah	3.84	142.20	140	0.000280
2-methylnaphthalene	91576	pah	3.86	142.20	141	0.000270
2-chloronaphthalene	91587	pah,ha	3.88	162.62	136	0.000100
1-chloronaphthalene	90131	pah,ha	3.88	162.62	136	0.000100
3,4-dichlorotoluene	95750	ar,ha	3.88	161.03	129	0.000120
biphenyl	92524	ar	3.91	154.21	150	0.000216

Chemical	CAS[A]	Class[B]	K_{OW}[C]	MW[D]	MV[E]	S[F]
1,3,5-trichlorobenzene	108703	ar,ha	3.97	181.45	125	0.0000933
1,2,3-trichlorobenzene	87616	ar,ha	3.98	181.45	124	0.0000870
1,2,4-trichlorobenzene	120821	ar,ha	4.00	181.45	126	0.0000886
acenaphthene	83329	pah	4.01	154.21	140	0.000100
2,5-dimethyl-2,4-hexadiene	764136	al	4.10	110.20	146	0.000133
methyl cyclohexane	108872	al	4.10	98.19	128	0.000155
1,2,4,5-tetramethylbenzene	95932	ar	4.11	134.22	152	0.000159
hexane	110543	al	4.12	86.18	132	0.000131
1,3-diethylbenzene	141935	ar	4.17	134.22	156	0.000135
1-decanol	112301	ao	4.19	158.28	192	0.00181
p-tert-butyltoluene	98511	ar	4.26	148.25	173	0.0000995
diphenylether	101848	et	4.36	170.21	152	0.0000595
amylbenzene	538681	ar	4.52	148.25	173	0.0000502
phenanthrene	85018	pah	4.57	178.23	161	0.0000340
1,2,4,5-tetrachlorobenzene	95943	ar,ha	4.64	215.89	136	0.0000151
1,2,3,4-tetrachlorobenzene	634662	ar,ha	4.64	215.89	136	0.0000145
1,2,3,5-tetrachlorobenzene	634902	ar,ha	4.64	215.89	136	0.0000148
1-undecanol	112425	ao	4.70	172.31	207	0.000640
pyrene	129000	pah	4.92	202.26	182	0.0000120
9-methylanthracene	779022	pah	5.01	192.26	175	0.00000980
fluoranthene	206440	pah	5.08	202.26	197	0.0000102
1-dodecanol	112538	ao	5.20	186.34	223	0.000238
pentachlorobenzene	608935	ar,ha	5.32	250.34	147	0.00000218
octane*	111659	al	5.34	114.23	164	0.00000625
1-tridecanol*	112709	ao	5.75	200.36	224	0.0000793
decane*	124185	al	6.56	142.28	229	0.000000300

*Chemical is not included: LC50>S.
[A]CAS=Chemical abstract number
[B]Class: ao=alcohol, ar=aromatic, ha=halogenated, et=ether, al=aliphatic, ke=ketone, pah=PAH
[C]$K_{OW}=\log_{10}(K_{OW})$;
[D]MW=molecular weight (gm/mol);
[E]V=molar volume (cm^3/mol);
[F]S=aqueous solubility(mol/L)

Appendix C

Summary of Data on the Acute Toxicity of PAHs to Freshwater and Saltwater Species and the Derivation of Genus Mean Acute Values.

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
FRESHWATER													
Hydra, *Hydra americana*	J	W,E	fluoranthene (206-44-0)	5.084	FT	M	70	0.3461	0.3461	22.06	22.06	_	Spehar et al., 1999
Hydra, *Hydra sp.*	X	W,E	phenanthrene (85-01-8)	4.571	FT	M	96	0.5386	0.5386	11.24	11.24	15.7	Call et al., 1986
Annelid, *Lumbriculus variegatus*	X	I	phenanthrene (85-01-8)	4.571	FT	M	>419	>2.351	>2.351	>49.07	_	_	Call et al., 1986
Annelid, *Lumbriculus variegatus*	A	I	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>52.46	>52.5	Spehar et al., 1999
Snail, *Mudalia potosensis*	X	E	fluorene (86-73-7)	4.208	S	U	**>1900**[G] (5600)	>11.42	>11.42	>108.2	>108.2	>108.2	Finger et al., 1985
Snail, *Aplexa hypnorum*	X	E	acenaphthene (83-32-9)	4.012	FT	M	>2040	>13.23	>13.23	>81.82	>81.82	>81.8	Holcombe et al., 1983
Snail, *Physa heterostropha*	X	E	fluoranthene (206-44-0)	5.084	S	U	137	0.6773	0.6773	43.17	43.17	43.2	Horne and Oblad, 1983
Snail, *Physella virgata*	A	E	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>56.09	>56.1	Spehar et al., 1999
Cladoceran, *Daphnia magna*	X	W	naphthalene (91-20-3)	3.356	S	U	8570	66.86	_	_	_	_	U.S. EPA, 1978
Cladoceran, *Daphnia magna*	J	W	naphthalene (91-20-3)	3.356	S	U	4723	36.85	_	_	_	_	Abemethy et al., 1986
Cladoceran, *Daphnia magna*	X	W	naphthalene (91-20-3)	3.356	S	M	2160	16.85	34.63	51.39	_	_	Millemann et al., 1984
Cladoceran, *Daphnia magna*	J	W	1-methyl naphthalene (90-12-0)	3.837	S	U	1420	9.986	9.986	42.20	_	_	Abemethy et al., 1986
Cladoceran, *Daphnia magna*	J	W	2-methyl naphthalene (91-57-6)	3.857	S	U	1491	10.49	10.49	46.29	_	_	Abemethy et al., 1986

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	U	3450	22.37	_	_	_	_	Randall and Knopp, 1980
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	U	**>3800** (41000)	>24.64	_	_	_	_	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	M	320	2.075	_	_	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	S	M	1300	8.430	_	_	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	acenaphthene (83-32-9)	4.012	FT	M	120	0.7782	0.7782	4.813	_	_	EG&G Bionomics, 1982
Cladoceran, *Daphnia magna*	X	W	fluorene (86-73-7)	4.208	S	U	430	2.585	2.585	24.49	_	_	Finger et al., 1985
Cladoceran, *Daphnia magna*	J	W	phenanthrene (85-01-8)	4.571	S	U	207	1.160	_	_	_	_	Abernethy et al., 1986
Cladoceran, *Daphnia magna*	X	W	phenanthrene (85-01-8)	4.571	S	U	843	4.730	_	_	_	_	Eastmond et al., 1984
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	S	M	700	3.928	_	_	_	_	Millemann et al., 1984
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	S,R	M	212	1.189	_	_	_	_	Brooke, 1994
Cladoceran, *Daphnia magna*	Neonate	W	phenanthrene (85-01-8)	4.571	FT	M	230	1.290	_	_	_	_	Brooke, 1993
Cladoceran, *Daphnia magna*	X	W	phenanthrene (85-01-8)	4.571	FT	M	117	0.6565	0.9204	19.21	_	_	Call et al., 1986
Cladoceran, *Daphnia magna*	J	W	pyrene (129-00-0)	4.922	S	U	90.9	0.4494	0.4494	20.13	_	_	Abernethy et al., 1986

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia magna*	J	W	9-methyl anthracene (779-02-2)	5.006	S	U	124.8	0.6491	0.6491	34.91	_	_	Abemethy et al., 1986
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (320000)	>1.285	_	_	_	_	LeBlanc, 1980a
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	S	M	45	0.2225	_	_	_	_	Oris et al., 1991
Cladoceran, *Daphnia magna*	J	W	fluoranthene (206-44-0)	5.084	R	M	117	0.5785	_	_	_	_	Spehar et al., 1999
Cladoceran, *Daphnia magna*	X	W	fluoranthene (206-44-0)	5.084	S	M	105.7	0.5226	0.4067	25.92	25.23		Suedel ad Rodgers, 1996
Cladoceran, *Daphnia pulex*	X	W	naphthalene (91-20-3)	3.356	S	U	4663	36.38	36.38	53.99	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	fluorene (86-73-7)	4.208	S	U	212	1.275	1.275	12.08	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	1,3-dimethyl naphthalene (575-41-7)	4.367	S	U	767	4.917	4.917	65.84	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	2,6-dimethyl naphthalene (581-42-0)	4.373	S	U	193	1.237	1.237	16.78	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	anthracene (120-12-7)	4.534	S	U	**>45** (754)	>0.2528	>0.2528	>4.869[L]	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	Neonate	W	phenanthrene (85-01-8)	4.571	S	U	734	4.118	_	_	_	_	Passino and Smith, 1987
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	U	**>1100** (>1150)	>6.172	_	_	_	_	Geiger and Buikema, 1981, 1982

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	U	350	1.964	_	_	_	_	Smith et al., 1988
Cladoceran, *Daphnia pulex*	X	W	phenanthrene (85-01-8)	4.571	S	M	100	0.5611	1.656	34.56	_	_	Trucco et al., 1983
Cladoceran, *Daphnia pulex*	X	W	2-methyl anthracene (613-12-7)	4.991	S	U	**>30** (96)	>0.1563	>0.1563	>8.134[L]	30.15	27.6	Smith et al., 1988
Amphipod, *Gammarus minus*	X	E	acenaphthene (83-32-9)	4.012	S	U	460	2.983	2.983	18.45	_	_	Horne et al., 1983
Amphipod, *Gammarus minus*	A	E	fluoranthene (206-44-0)	5.084	S	U	32	0.1582	0.1582	10.08	13.64	_	Horne and Oblad, 1983
Amphipod, *Gammarus pseudolimnaeus*	X	E	fluorene (86-73-7)	4.208	S	U	600	3.607	3.607	34.18	_	_	Finger et al., 1985
Amphipod, *Gammarus pseudolimnaeus*	X	E	phenanthrene (85-01-8)	4.571	FT	M	126	0.7070	0.7070	14.76	_	_	Call et al., 1986
Amphipod, *Gammarus pseudolimnaeus*	A	E	fluoranthene (206-44-0)	5.084	FT	M	43	0.2126	0.2126	13.55	18.98	16.1	Spehar et al., 1999
Amphipod, *Hyalella azteca*	J	E	fluoranthene (206-44-0)	5.084	FT	M	44	0.2175	0.2175	13.87	13.87	13.9	Spehar et al., 1999
Dragonfly, *Ophiogomphus* sp.	N	E	fluoranthene (206-44-0)	5.084	FT	M	>178	>0.8801	>0.8801	>56.09	>56.09	>56.1	Spehar et al., 1999
Stonefly, *Peltoperla maria*	X	E	acenaphthene (83-32-9)	4.012	S	U	240	1.556	1.556	9.626	_	_	Horne et al., 1983
Stonefly, *Peltoperla maria*	X	E	fluoranthene (206-44-0)	5.084	S	U	135	0.6675	0.6675	42.54	20.24	20.2	Horne and Oblad, 1983

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Midge, *Chironomus tentans*	L	I	naphthalene (91-20-3)	3.356	S	M	2810	21.92	21.92	32.53	_	_	Millemann et al., 1984
Midge, *Chironomus tentans*	L	I	phenanthrene (85-01-8)	4.571	S	M	490	2.749	2.749	57.39	_	_	Millemann et al., 1984
Midge, *Chironomus tentans*	L	I	fluoranthene (206-44-0)	5.084	S	M	>250	>1.236	>1.236	>78.78[L]	43.21	_	Suedel ad Rodgers, 1996
Midge, *Chironomus riparius*	L	I	fluorene (86-73-7)	4.208	S	U	**>1900** (2350)	>11.42	>11.42	>108.2	>108.2	>68.4	Finger et al., 1985
Midge, *Paratanytarsus sp.*	X	E	acenaphthene (83-32-9)	4.012	S	M	2000	12.97	_	_	_	_	Northwestern Aquatic Science Inc., 1982
Midge, *Paratanytarsus sp.*	X	E	acenaphthene (83-32-9)	4.012	S	M	2090	13.55	13.26	82.00	82.00	82.0	Northwestern Aquatic Science Inc., 1982
Midge, *Tanytarsus dissimilis*	L	I	naphthalene (91-20-3)	3.356	S	U	20700	161.5	_	_	_	_	Darville and Wilhm, 1984
Midge, *Tanytarsus dissimilis*	L	I	naphthalene (91-20-3)	3.356	S	U	12600	98.31	126.0	187.0	187.0	187	Darville and Wilhm, 1984
Coho salmon, *Oncorhynchus kisutch*	E	I	naphthalene (91-20-3)	3.356	R	M	>11800	>92.07	_	_	_	_	Korn and Rice, 1981
Coho salmon, *Oncorhynchus kisutch*	F	W	naphthalene (91-20-3)	3.356	R	M	5600	43.69	43.69	64.84	64.84	_	Korn and Rice, 1981
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	1800	14.04	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	6100	47.59	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	2600	20.29	_	_	_	_	Edsall, C.C., 1991

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	4400	34.33	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	naphthalene (91-20-3)	3.356	S	U	5500	42.91	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	J	W	naphthalene (91-20-3)	3.356	FT	M	1600	12.48	_	_	_	_	DeGraeve et al., 1982
Rainbow trout, *Oncorhynchus mykiss*	X	W	naphthalene (91-20-3)	3.356	FT	M	2300	17.94	14.97	22.21	_	_	DeGraeve et al., 1980
Rainbow trout, *Oncorhynchus mykiss*	J	W	acenaphthene (83-32-9)	4.012	FT	M	670	4.345	4.345	26.87	_	_	Holcombe et al., 1983
Rainbow trout, *Oncorhynchus mykiss*	J	W	fluorene (86-73-7)	4.208	S	U	820	4.930	4.930	46.71	_	_	Finger et al., 1985
Rainbow trout, *Oncorhynchus mykiss*	pre SU	I	1,3-dimethyl naphthalene (575-41-7)	4.367	S	U	1700	10.88	14.04	188.1[L]	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	L	W	phenanthrene (85-01-8)	4.571	S	U	**>1100** (3200)	>6.172	_	_	_	_	Edsall, C.C., 1991
Rainbow trout, *Oncorhynchus mykiss*	J	W	phenanthrene (85-01-8)	4.571	FT	M	375	2.104	2.104	43.92	_	_	Call et al., 1986
Rainbow trout, *Oncorhynchus mykiss*	X	W	fluoranthene (206-44-0)	5.084	S	M	187	0.9246	_	_	_	_	Horne and Oblad, 1983
Rainbow trout, *Oncorhynchus mykiss*	J	W	fluoranthene (206-44-0)	5.084	FT	M	26.0	0.1285	0.1285	8.193	25.13	40.4	Spehar et al., 1999
Brown trout, *Salmo trutta*	J	W	acenaphthene (83-32-9)	4.012	FT	M	580	3.761	3.761	23.26	23.26	23.3	Holcombe et al., 1983
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	S	M	1990	15.53	_	_	_	_	Millemann et al., 1984

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Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	7900	61.64	_	_	_	_	DeGraeve et al., 1982
Fathead minnow, *Pimephales promelas*	X	W	naphthalene (91-20-3)	3.356	FT	M	4900	38.23	_	_	_	_	DeGraeve et al., 1980
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	6140	47.91	_	_	_	_	Geiger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	8900	69.44	_	_	_	_	DeGraeve et al., 1980
Fathead minnow, *Pimephales promelas*	J	W	naphthalene (91-20-3)	3.356	FT	M	6080	47.44	51.77	76.82	_	_	Holcombe et al., 1984
Fathead minnow, *Pimephales promelas*	J	W	1-methyl naphthalene (90-12-0)	3.837	S	U	9000	63.38	63.38	267.9	_	_	Mattson et al., 1976
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	S	M	3100	20.10	_	_	_	_	Marine Bioassay Lab., 1981
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	S	M	1500	9.727	_	_	_	_	EG&G Bionomics, 1982
Fathead minnow, *Pimephales promelas*	A	W	acenaphthene (83-32-9)	4.012	R	U	3700	23.99	_	_	_	_	Academy of Natural Sci., 1981
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	1730	11.22	_	_	_	_	Geiger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	608	3.943	_	_	_	_	Cairns and Nebeker, 1982
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	>1400	>9.079	_	_	_	_	EG&G Bionomics, 1982
Fathead minnow, *Pimephales promelas*	J	W	acenaphthene (83-32-9)	4.012	FT	M	1600	10.38	7.713	47.71	_	_	Holcombe et al., 1983

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Fathead minnow, *Pimephales promelas*	X	W	fluorene (86-73-7)	4.208	S	U	**>1900** (100000)	>11.42	>11.42	>108.2[L]	_	_	Finger et al., 1985
Fathead minnow, *Pimephales promelas*	J	W	phenanthrene (85-01-8)	4.571	S	M	**>1100** (>1150)	>6.172	>6.172	>128.8[L]	_	_	U.S. EPA, 1978
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	S	M	95	0.4697	_	_	_	_	Horne and Oblad, 1983
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	S	M	7.71	0.0381	_	_	_	_	Gendusa, 1990
Fathead minnow, *Pimephales promelas*	A	W	fluoranthene (206-44-0)	5.084	FT	U	**>260** (>1000)	>1.285	_	_	_	_	Birge et al., 1982
Fathead minnow, *Pimephales promelas*	J	W	fluoranthene (206-44-0)	5.084	FT	M	69	0.3411	0.3411	21.74	67.97	68.0	Spehar et al., 1999
Channel catfish, *Ictalurus punctatus*	J	E	acenaphthene (83-32-9)	4.012	FT	M	1720	11.15	11.15	68.99	_	_	Holcombe et al., 1983
Channel catfish, *Ictalurus punctatus*	J	E	fluoranthene (206-44-0)	5.084	S	M	37.40	0.1849	0.1849	11.79	28.51	28.5	Gendusa, 1990
Bluegill, *Lepomis macrochirus*	J	W	acenaphthene (83-32-9)	4.012	S	U	1700	11.02	11.02	68.18	_	_	Buccafusco et al., 1981
Bluegill, *Lepomis macrochirus*	X	W	fluorene (86-73-7)	4.208	S	U	910	5.471	5.471	51.84	_	_	Finger et al., 1985
Bluegill, *Lepomis macrochirus*	J	W	phenanthrene (85-01-8)	4.571	FT	M	234	1.313	1.313	27.41	_	_	Call et al., 1986
Bluegill, *Lepomis macrochirus*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (4000)	>1.285	_	_	_	_	Buccafusco et al., 1981; EPA, 1978
Bluegill, *Lepomis macrochirus*	J	W	fluoranthene (206-44-0)	5.084	FT	M	44	0.2175	0.2175	13.87	34.04	34.0	Spehar et al., 1999

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South african clawed frog *Xenopus laevis*	L	W	naphthalene (91-20-3)	3.356	FT	M	2100	16.38	_	_	_	_	Edmisten and Bantle, 1982
South african clawed frog *Xenopus laevis*	L	W	naphthalene (91-20-3)	3.356	FT	M	2100	16.38	16.38	24.31	24.31	24.3	Edmisten and Bantle, 1982
SALTWATER													
Annelid worm, *Neanthes arenaceodentata*	J	I	naphthalene (91-20-3)	3.356	S	U	3800	29.65	29.65	44.00	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	X	I	acenaphthene (83-32-9)	4.012	S	U	3600	23.34	_	_	_	_	Horne et al., 1983
Annelid worm, *Neanthes arenaceodentata*	J	I	acenaphthene (83-32-9)	4.012	R	U	**>3800** (16440)	>24.64	23.34	144.4	_	_	Thursby et al., 1989a
Annelid worm, *Neanthes arenaceodentata*	A	I	phenanthrene (85-01-8)	4.571	S	U	600	3.366	3.366	70.27	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	J	I	fluoranthene (206-44-0)	5.084	S	U	**>260** (500)	>1.285	_	_	_	_	Rossi and Neff, 1978
Annelid worm, *Neanthes arenaceodentata*	J	I	fluoranthene (206-44-0)	5.084	S	U	**>260** (20000)	> 1.285	>1.285	>81.93[L]	76.43	76.4	Spehar et al., 1999
Archiannelid, *Dinophilus gyrociliatus*	J	I	phenanthrene (85-01-8)	4.571	R	U	185.40	1.040	1.040	21.71	21.71	21.7	Battelle Ocean Sciences, 1987
Mud snail, *Nassarius obsoletus*	A	I,E	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987
Blue mussel, *Mytilus edulis*	A	E,W	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987

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Pacific oyster, *Crassostrea gigas*	E/L	W	naphthalene (91-20-3)	3.356	S	U	**>31000** (199000)	>241.9	>241.9	>358.9	>358.9	>359	U.S. EPA, 1980
Coot clam, *Mulinia lateralis*	J	E	pyrene (129-00-0)	4.922	FT	M	**>132** (>240)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Coot clam, *Mulinia lateralis*	J	E	fluoranthene (206-44-0)	5.084	S	U	**>260** (10710)	>1.285	>1.285	>81.93	>48.94	>48.9	Spehar et al., 1999
Soft-shell clam, *Mya arenaria*	A	I	phenanthrene (85-01-8)	4.571	R	M	>245	>1.375	>1.375	>28.69	>28.69	>28.7	Battelle Ocean Sciences, 1987
Calanoid copepod, *Eurytemora affinis*	A	X	naphthalene (91-20-3)	3.356	S	U	3798	22.58	22.58	33.51			Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2-methyl naphthalene (91-57-6)	3.857	S	U	1499	7.741	7.741	34.17	_	_	Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2,6-dimethyl naphthalene (581-42-0)	4.373	S	M	852	3.860	3.860	52.37	_	_	Ott, et al., 1978
Calanoid copepod, *Eurytemora affinis*	A	X	2,3,5-trimethyl naphthalene (2245-38-7)	4.856	S	M	316	1.271	1.271	49.53	41.51	41.5	Ott, et al., 1978
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	S	U	970	6.290	_	_	_	_	U.S. EPA, 1978;Ward et al., 1981
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	S	M	160	1.038	_	_	_	_	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	R	U	1190	7.717	_	_	_	_	Thursby et al., 1989a
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	460	2.983	_	_	_	_	Thursby et al., 1989b

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Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	190	1.232	_	_	_	_	EG&G Bionomics, 1982
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	466.1	3.023	_	_	_	_	Horne et al., 1983;Thursby, 1991a
Mysid, *Americamysis bahia*	J	E	acenaphthene (83-32-9)	4.012	FT	M	271.9	1.763	2.104	13.01	_	_	Horne et al., 1983;Thursby, 1991a
Mysid, *Americamysis bahia*	J	E	phenanthrene (85-01-8)	4.571	FT	M	27.1	0.1521	_	_	_	_	Kuhn and Lussier, 1987
Mysid, *Americamysis bahia*	J	E	phenanthrene (85-01-8)	4.571	FT	M	17.7	0.0993	0.1229	2.565	_	_	Battelle Ocean Sciences, 1987
Mysid, *Americamysis bahia*	J	E	pyrene (129-00-0)	4.922	FT	M	28.28	0.1398	0.1398	6.264	_	_	Champlin and Poucher, 1992a
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	S	U	31	0.1533	_	_	_	_	Spehar et al., 1999
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	S	U	40	0.1978	_	_	_	_	U.S. EPA, 1978
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	FT	M	30.53	0.1509	_	_	_	_	Spehar et al., 1999
Mysid, *Americamysis bahia*	J	E	fluoranthene (206-44-0)	5.084	FT	M	87	0.4301	0.2548	16.24	7.633	7.63	EG&G Bionomics, 1978
Mysid, *Neomysis americana*	X	E	naphthalene (91-20-3)	3.356	S	M	1250	9.753	_	_	_	_	Hargreaves et al., 1982
Mysid, *Neomysis americana*	X	E	naphthalene (91-20-3)	3.356	S	M	1420	11.08	10.39	15.43	15.43	15.4	Hargreaves et al., 1982
Isopod *Excirolana vancouverensis*	J	I,E	fluoranthene (206-44-0)	5.084	R	M	>70	>0.3461	>0.3461	>22.06	>22.06	>22.1	Boese et al., 1997

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Amphipod, *Ampelisca abdita*	J	I	acenaphthene (83-32-9)	4.012	R	U	1125	7.295	7.295	45.12	_	_	Thursby et al., 1989a
Amphipod, *Ampelisca abdita*	J	I	fluoranthene (206-44-0)	5.084	S	U	67	0.3313	0.3313	21.11	30.86	30.9	Spehar et al., 1999
Amphipod, *Leptocheirus plumulosus*	A	E,I	acenaphthene (83-32-9)	4.012	FT	M	589.4	3.822	3.822	23.64	_	_	Swartz, 1991a
Amphipod, *Leptocheirus plumulosus*	A	E,I	phenanthrene (85-01-8)	4.571	FT	M	198.4	1.113	1.113	23.24	_	_	Swartz, 1991a
Amphipod, *Leptocheirus plumulosus*	J	E,I	pyrene (129-00-0)	4.922	FT	M	66.49	0.3287	0.3287	14.73	_	_	Champlin and Poucher , 1992a
Amphipod, *Leptocheirus plumulosus*	X	E,I	fluoranthene (206-44-0)	5.084	R	M	51	0.2522	0.2522	16.07	18.99	19.0	Boese et al., 1997
Amphipod, *Rhepoxynius abronius*	J	I	fluoranthene (206-44-0)	5.084	R	M	63	0.3115	0.3115	19.85	19.85	19.9	Boese et al., 1997
Amphipod, *Eohaustorius estuarius*	J	I	fluoranthene (206-44-0)	5.084	R	M	>70	>0.3461	>0.3461	>22.06	>22.06	>22.1	Boese et al., 1997
Amphipod, *Grandidierella japonica*	J	I	fluoranthene (206-44-0)	5.084	R	M	27	0.1335	0.1335	8.508	8.508	8.51	Boese et al., 1997
Amphipod, *Corophium insidiosum*	J	I	fluoranthene (206-44-0)	5.084	R	M	54	0.2670	0.2670	17.02	17.02	17.0	Boese et al., 1997
Amphipod, *Emerita analoga*	J	I,E	fluoranthene (206-44-0)	5.084	R	M	74	0.3659	0.3659	23.32	23.32	23.3	Boese et al., 1997
Kelp shrimp, *Eualis suckleyi*	X	W	naphthalene (91-20-3)	3.356	FT	M	1390	10.84	10.84	16.09	16.09	16.1	Rice and Thomas, 1989
Grass shrimp, *Palaemonetes pugio*	X	E,W	naphthalene (91-20-3)	3.356	S	M	2350	18.34	18.34	27.21	_	_	Tatem et al., 1978

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Grass shrimp, *Palaemonetes pugio*	X	E,W	acenaphthene (83-32-9)	4.012	S	U	676.8	4.389	_	_	_	_	Horne et al., 1983;Thursby, 1991b
Grass shrimp, *Palaemonetes pugio*	L	E,W	acenaphthene (83-32-9)	4.012	R	U	1697	11.00	6.950	42.98	_	_	Thursby et al., 1989a
Grass shrimp, *Palaemonetes pugio*	A	E,W	phenanthrene (85-01-8)	4.571	R	U	200.8	1.127	_	_	_	_	Battelle Ocean Sciences, 1987
Grass shrimp, *Palaemonetes pugio*	A	E,W	phenanthrene (85-01-8)	4.571	FT	M	145.4	0.8158	0.8158	17.03	_	_	Battelle Ocean Sciences, 1987
Grass shrimp, Palaemonetes pugio	J	E,W	fluoranthene (206-44-0)	5.084	S	U	142	0.7021	0.7021	44.75	30.72	30.7	Spehar et al., 1999
Sand shrimp, *Crangon septemspinosus*	X	E	acenaphthene (83-32-9)	4.012	S	U	245	1.589	1.589	9.826	9.826	9.83	Horne et al., 1983;Thursby , 1991b
American Lobster, *Homarus americanus*	L	_	fluoranthene (206-44-0)	5.084	R	U	**>260** (317)	1.285	1.285	81.93	81.93	81.9	Spehar et al., 1999
Hermit crab, *Paqurus longicarpus*	A	E	phenanthrene (85-01-8)	4.571	FT	M	163.7	0.9185	0.9185	19.17	19.17	19.2	Battelle Ocean Sciences, 1987
Slipper limpet, *Crepidula fornicata*	L	W	acenaphthene (83-32-9)	4.012	R	U	3426	22.28	22.28	137.8	137.8	138	Thursby et al., 1989a
Sea urchin, *Arbacia punctalata*	E	W	acenaphthene (83-32-9)	4.012	S	U	**>3800** (8163)	>24.64	>24.64	>152.4	_	_	Thursby et al., 1989a
Sea urchin, *Arbacia punctalata*	E	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (20000)	>1.285	>1.285	>81.93	>117.2	>117	Spehar et al., 1999
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	960	7.490	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	900	7.022	_	_	_	_	Rice and Thomas, 1989

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	990	7.724	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	1010	7.880	_	_	_	_	Rice and Thomas, 1989
Pink salmon, *Oncorhynchus gorbuscha*	Fry	W	naphthalene (91-20-3)	3.356	FT	M	890	6.944	7.40	10.99	10.99	11.0	Rice and Thomas, 1989
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	acenaphthene (83-32-9)	4.012	S	U	2200	14.27	_	_	_	_	Heitmuller et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	acenaphthene (83-32-9)	4.012	R	U	**>3800** (50000)	>25.00	_	_	_	_	Thursby et al., 1989a
Sheepshead minnow, *Cyprinodon variegatus*	A	E,W	acenaphthene (83-32-9)	4.012	FT	M	3100	20.10	20.10	124.3	_	_	Ward et al., 1981
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	phenanthrene (85-01-8)	4.571	R	U	>245	>1.375	_	_	_	_	Battelle Ocean Sciences, 1987
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	phenanthrene (85-01-8)	4.571	FT	M	429.4	2.409	2.409	50.29	_	_	Battelle Ocean Sciences, 1987
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	pyrene (129-00-0)	4.922	FT	M	**>132** (>640)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>20000)	>1.285	_	_	_	_	Spehar et al., 1999
Sheepshead minnow, *Cyprinodon variegatus*	J	E,W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>560000)	>1.285	>1.285	>81.93[L]	79.07	79.1	Heitmuller et al., 1981;U.S EPA, 1978
Inland silverside, *Menidia beryllina*	X	W	acenaphthene (83-32-9)	4.012	S	U	2300	14.91	_	_	_	_	Horne et al., 1983
Inland silverside, *Menidia beryllina*	J	W	acenaphthene (83-32-9)	4.012	R	U	**>3800** (5564)	>24.64	>19.17	>118.6	_	_	Thursby et al., 1989a

Common/scientific	Life-Stage[A]	Habitat[B]	PAH Tested (CAS #)	Log K_{ow}[C]	Method[D]	Concen-tration[E]	LC50/ EC50[F] (µg/l)	LC50/ EC50[F] (µmol/l)	PAH Specific SMAV[H] (µmol/l)	K_{ow} Normalized PAH Specific SMAV[I] (µmol/g_{oc})	Species SMAV[J] (µmol/g_{oc})	GMAV[K] (µmol/g_{oc})	References
Inland silverside, *Menidia beryllina*	J	W	pyrene (192-00-0)	4.922	FT	M	**>132** (>188.17)	>0.6526	>0.6526	>29.24	_	_	Champlin and Poucher, 1992a
Inland silverside, *Menidia beryllina*	J	W	fluoranthene (206-44-0)	5.084	S	U	**>260** (>616)	>1.285	>1.285	>81.93	>65.73	_	Spehar et al., 1999
Atlantic silverside, *Menidia menidia*	A	W	phenanthrene (85-01-8)	4.571	FT	M	108	0.6060	0.6060	12.65	12.65	28.8	Battelle Ocean Sciences, 1987
Winter flounder, *Pseudopleuronectes americanus*	J	_	fluoranthene (206-44-0)	5.084	S	M	>188	>0.9295	>0.9295	>59.24	>59.24	>59.2	Spehar et al., 1999

[A]Life-stage: A = adult, J = juvenile, L = larvae, E = embryo, U = life-stage and habitat unknown, X = life-stage unknown but habitat known.
[B]Habitat: I = infauna, E = epibenthic, W = water column.
[C]log K_{OW}: Predicted using SPARC (Karickoff et al, 1991).
[D]Method: S= static, R = renewal, FT= flow-through.
[E] Concentration: U = unmeasured (nominal), M = chemical measured.
[F] Acute Values: 96 hour LC50 or EC50, except for *Daphnia* and *Tanytarsus* which are 48 hours duration.
[G] Bolded acute values are the water solubilities of the PAH (Mackay et al., 1992). For these tests the acute values exceeded solubility. Therefore, solubilities are used instead of the acute value for further calculations.
[H] PAH-specific SMAV: Geometric mean of the acute values by PAH and species.
[I] PAH-specific SMAVs at a log K_{OW}=1.0; calculated as antilog ($\log_{10}$LC50 + 0.945$\log_{10}K_{OW}$)/1000 (see Equation 2-33).
[J] Species SMAV: Geometric mean of K_{OW}-normalized SMAVs for a species across PAHs.
[K] GMAV: Geometric mean of SMAVs for all species within a genus.
[L] Not used in calculations.

Appendix D

Comparison of PAH-specific Equilibrium Partitioning Sediment Benchmarks (ESBs) Derived from Narcosis Theory and the Median Response Concentration of Benthic Species for Individual PAHs in Spiked-sediment Toxicity Tests.

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Oligochaete, *Lumbriculus variegatus*	pyrene	7 d LC50	**>9090** (61100)	694	**>13.1**	-	-	Kukkonen and Landrum, 1994
Oligochaete, *Lumbriculus variegatus*	pyrene	7 d EC50-SA	**>9090** (51400)	694	**>13.1**	-	-	Kukkonen and Landrum, 1994
Oligochaete, *Limnodrilus hoffmeisteri*	phenanthrene	10 d LC50	**>34300** (42500)	593	**>57.8**	**>57.8**	**>57.8**	Lotufo and Fleeger, 1996
Oligochaete, *Limnodrilus hoffmeisteri*	phenanthrene	28 d EC25-R	5790	593	9.80	-	-	Lotufo and Fleeger, 1996
Oligochaete, *Limnodrilus hoffmeisteri*	pyrene	28 d EC25-R	8440	694	12.2	-	-	Lotufo and Fleeger, 1996
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	2380	704	-	-	-	Suedel et al., 1993
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	955	704	-	-	-	Suedel et al., 1993
Cladoceran, *Daphnia magna*	fluoranthene	10 d LC50	3260	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	**>23900** (37649)	704	-	-	-	Driscoll et al., 1997a
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	1250	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	1480	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	500	704	-	-	-	Suedel et al., 1993
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	22000	704	31.3	-	-	Harkey et al., 1997
Amphipod, *Hyalella azteca*	fluoranthene	10 d LC50	5130	704	7.29	15.1	15.1	DeWitt et al., 1989
Amphipod, *Corophium spinicorne*	fluoranthene	10 d LC50	2830	704	4.02	-	-	Swartz et al., 1990

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Corophium spinicorne*	fluoranthene	10 d LC50	4390	704	6.23	5.01	5.01	Swartz et al., 1990
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	10900	489	22.3	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	23500	489	48.1	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	acenapthene	10 d LC50	8450	489	17.3	26.4	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	6870	593	11.59	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	8080	593	13.63	-	-	Swartz et al., 1991a
Amphipod, *Leptocheirus plumulosus*	phenanthrene	10 d LC50	8180	593	13.8	13.0	18.5	Swartz et al., 1991a
Amphipod, *Rhepoxynius abronius*	acenapthene	10 d LC50	2310	489	4.72	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	acenapthene	10 d LC50	2110	489	4.31	4.51	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	phenanthrene	10 d LC50	3080	593	5.19	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	phenanthrene	10 d LC50	2220	593	3.74	4.41	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	pyrene	10 d LC50	1220	694	1.76	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	pyrene	10 d LC50	2810	694	4.05	2.67	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	>4360	704	>6.19	-	-	DeWitt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	4410	704	6.26	-	-	DeWitt et al., 1992

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	3080	704	4.38	-	-	DeWitt et al., 1992
Amphipod, Rhepoxynius abronius	fluoranthene	10 d LC50	2230	704	3.17	-	-	Swartz et al., 1990
Amphipod, Rhepoxynius abronius	fluoranthene	10 d LC50	3150	704	4.50	-	-	DeWitt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1890	704	2.68	-	-	Swartz et al., 1990
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2790	704	3.96	-	-	De Witt et al., 1992
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2320	704	3.30	-	-	Swartz et al., 1997
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1700	704	2.41	-	-	DeWitt et al., 1989
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	1030	704	1.47	-	-	Swartz et al., 1988
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	2100	704	2.98	-	-	Swartz et al., 1990
Amphipod, *Rhepoxynius abronius*	fluoranthene	10 d LC50	3310	704	4.70	3.56	3.67	Swartz et al., 1997
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	1630	489	3.33	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	4180	489	8.55	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	acenapthene	10 d LC50	1920	489	3.93	4.82	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	4210	593	7.10	-	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	3760	593	6.34	-	-	Swartz et al., 1991a

Common Name, *Scientific Name*	Chemical	Response	Median Response Conc.[A] (µg/goc)	$C_{OC,PAHi,FCVi}$ (µg/goc)	Test-Specific $ESBTU_{FCVi}$[B] (Unitless)	PAH-Specific SMAV[C]	GMAV[D]	References[E]
Amphipod, *Eohaustorius estuarius*	phenanthrene	10 d LC50	4060	593	6.85	6.75	-	Swartz et al., 1991a
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3100	704	4.40	-	-	DeWitt et al., 1989
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3930	704	5.59	-	-	DeWitt et al., 1989
Amphipod, *Eohaustorius estuarius*	fluoranthene	10 d LC50	3570	704	5.07	5.00	5.46	DeWitt et al., 1989
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	1590	704	-	-	-	Suedel et al., 1993
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	1740	704	-	-	-	Suedel et al., 1993
Midge, *Chironomus tentans*	fluoranthene	10 d LC50	682	704	-	-	-	Suedel et al., 1993
Amphipod, *Diporeia* sp.	pyrene	31 d LC50	**>9090**	694	**>13.1**	-	-	Landrum et al., 1994
Amphipod, *Diporeia* sp.	fluoranthene	10 d LC50	(147000) **>23900** (29300)	704	**>34.0**	**>34.0**	**>34.0**	Driscoll et al., 1997a

[A] Bolded median response concentration (acute) values are the $C_{OC,PAHi,Maxi}$ based on the water solubilities of the PAH (Mackay et al., 1992). For these tests the interstitial water concentration at the median response concentration exceeded solubility. Therefore, solubilities are used instead of the acute value for further calculations.

[B] Test-specific ESBTUs: Quotient of the median response concentration (µg/goc) and $C_{OC,PAHi,FCVi}$ (from Table 3-4).

[C] PAH-specific SMAV: Geometric mean of the test-specific $ESBTU_{FCVi}$ values from 10-d LC50 tests by species and PAH. Test-specific $ESBTU_{FCVi}$ values greater that solubility included only if they are the sole 10-d LC50 for the species.

[D] GMAV: Geometric mean of the PAH-specific SMAVs for all species within a genus.

[E] Spiked sediments from Suedel et al. (1993) were unlikely at equilibrium; i.e., organisms were tested after only 18 to 24 hours after spiking.

Appendix E

CAS#, Molecular Weight and Solid Solubility of Selected PAHs.

PAH	CAS #[A]	Molecular Weight (µg/µmol)	Mackay Solid Solubility[B] (µg/L)
indan	496117	118.18	100000
naphthalene	91203	128.17	30995
C1-naphthalenes	-	142.20	??
1-methylnaphthalene	90120	142.20	28001
2-methylnaphthalene	91576	142.20	25000
acenaphthylene	208968	152.20	16314
acenaphthene	83329	154.21	3800
1-ethylnaphthalene	1127760	156.23	10100
2-ethylnaphthalene	939275	156.23	8001
C2-naphthalenes	-	156.23	??
1,4-dimethylnaphthalene	571584	156.23	11400
1,3-dimethylnaphthalene	575417	156.23	8001
2,6-dimethylnaphthalene	581420	156.23	1700
2,3-dimethylnaphthalene	581408	156.23	2500
1,5-dimethylnaphthalene	571619	156.23	3100
fluorene	86737	166.22	1900
C3-naphthalenes	-	170.25	??
2,3,5-trimethylnaphthalene	2245387	170.26	??
1,4,5-trimethylnaphthalene	213411	170.20	2100
anthracene	120127	178.12	45.00
phenanthrene	85018	178.23	1100
C1-fluorenes	-	180.25	??
1-methylfluorene	1730376	180.25	1090
C4-naphthalenes	-	184.28	??
2-methylanthracene	613127	192.26	29.99
1-methylanthracene	610480	192.26	??
9-methylanthracene	779022	192.26	261.1
2-methylphenanthrene	2531842	192.26	??
1-methylphenanthrene	832699	192.26	269.9
C1-phenanthrene/anthracenes	-	192.26	??
9-ethylfluorene	2294828	194.28	??
C2-fluorenes	-	194.27	??
pyrene	129000	202.26	131.9
fluoranthene	206440	202.26	239.9
2-ethylanthracene	52251715	206.29	??
C2-phenanthrene/anthracenes	-	206.29	??
9,10-dimethylanthracene	781431	206.29	55.90
3,6-dimethylphenanthrene	1576676	206.29	??
C3-fluorenes	-	208.3	??
C1-pyrene/fluoranthenes	-	216.29	?
2,3-benzofluorene	243174	216.28	2.001

PAH	CAS #[A]	Molecular Weight (µg/µmol)	Mackay Solid Solubility[B] (µg/L)
benzo(a)fluorene	238843	216.29	45.00
C3-phenanthrene/anthracenes	-	220.32	??
naphthacene	92240	228.30	0.600
benz(a)anthracene	56553	228.29	11.00
chrysene	218019	228.29	2.000
triphenylene	217594	228.3	43.00
C2-pyrene/fluoranthenes	-	230.13	??
C4-phenanthrenes/anthracenes	-	234.23	??
C1-benzanthracene/chrysenes	-	242.32	??
C3-pyrene/fluoranthenes	-	244.32	??
benzo(a)pyrene	50328	252.31	3.810
perylene	198550	252.31	0.4012
benzo(e)pyrene	192972	252.32	4.012
benzo(b)fluoranthene	205992	252.32	1.501
benzo(j)fluoranthene	205822	252.32	2.500
benzo(k)fluoranthene	207089	252.32	0.7999
C2-benzanthracene/chrysenes	-	256.23	??
9,10-dimethylbenz(a)anthracene	56564	256.35	43.50
7,12-dimethylbenz(a)anthracene	57976	256.35	49.99
7-methylbenzo(a)pyrene	63041770	266.35	??
benzo(ghi)perylene	191242	276.23	0.2600
C3-benzanthracene/chrysenes	-	270.36	??
indeno(1,2,3-cd)pyrene	193395	276.23	??
dibenz(a,h)anthracene	53703	278.35	0.6012
dibenz(a,j)anthracene	58703	278.35	12.00
dibenz(a,c)anthracene	215587	278.35	1.601
C4-benzanthracene/chrysenes	-	284.38	??
C1-dibenz(a,h)anthracenes	-	292.37	??
coronene	191071	300.36	0.1400
C2-dibenz(a,h)anthracenes	-	306.39	??
C3-dibenz(a,h)anthracenes	-	320.41	??

[A] For C#-PAHs, a CAS is not available.
[B] Mackay et al. (1992).

Appendix F

Water-only and Interstitial Water LC50s used in Table 5-1.

Chemical Test Species	Method[A]	Water-only LC50 (µg/L)	Interstitial Water LC50 (µg/L)	References
Freshwater				
Fluoranthene				
Diporeia sp.	FT,M/10	>194	>381.3	Driscoll et al., 1997a,b
Hyalella azteca	FT,M/10	130.7	>75.4	Driscollet al., 1997a,b
Hyalella azteca	S,M/10	44.9	45.9	Suedel et al., 1993
Hyalella azteca	S,M/10	44.9	236.5	Suedel et al., 1993
Hyalella azteca	S,M/10	44.9	97.6	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	91.2	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	251	Suedel et al., 1993
Chironomus tentans	S,M/10	31.9	75.7	Suedel et al., 1993
Saltwater				
Acenaphthene				
Eohaustorius estuarius	FT,M/10	374	800	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	374	609	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	374	542	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	>1,720	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	1410	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	678	1490	Swartz, 1991a
Fluoranthene				
Leptocheirus plumulosus	S/10	39.2	-	Driscoll et al., 1998
Phenanthrene				
Eohaustorius estuarius	FT,M/10	131	138	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	131	139	Swartz, 1991a
Eohaustorius estuarius	FT,M/10	131	146	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	387	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	306	Swartz, 1991a
Leptocheirus plumulosus	FT,M/10	185	360	Swartz, 1991a
2,6-dimethylnaphthlene				
Rhepoxynius abronius	S,M/10	-	200	Ozretich et al., 2000a
2,3,5-trimethylnaphthlene				
Rhepoxynius abronius	S,M/10	-	153	Ozretich et al., 2000a
1-methylfluorene				
Rhepoxynius abronius	S,M/10	-	44	Ozretich et al., 2000a
2-methylphenanthrene				
Rhepoxynius abronius	S,M/10	-	70	Ozretich et al., 2000a
9-methylanthracene				
Rhepoxynius abronius	S,M/10	-	32	Ozretich et al., 2000a
Acenaphthene				
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997

Chemical Test Species	Method[A]	Water-only LC50 (µg/L)	Interstitial Water LC50 (µg/L)	References
Naphthalene				
Rhepoxynius abronius	S,M/10	-	10440	Ozretich et al., 2000a
Phenanthrene				
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Pyrene				
Rhepoxynius abronius	S,M/10	-	28.1	Ozretich et al., 2000a
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	-	-	Swartz et al., 1997
Fluoranthene				
Rhepoxynius abronius	S,M/10	13.9	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	13.9	-	Swartz et al., 1997
Rhepoxynius abronius	S,M/10	13.9	22.7	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	29.4	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	24.2	Swartz et al., 1990
Rhepoxynius abronius	S,M/10	13.9	> 315	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	14.1	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	26.6	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	19.2	DeWitt et al., 1992
Rhepoxynius abronius	S,M/10	13.9	9.38	DeWitt et al., 1992
		Mean LC50 ratio =	1.6	

[A] Test conditions for water-only toxicity tests: S = static, FT = flow-through, M = measured, 10 = 10-d duration.

Appendix G

Teratogenic Effects from Laboratory Exposure to PAHs.

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
fathead minnow (embryos), *Pimephales promelas*	maternal via water	lab; flow-through	Anthracene	6.66 µg/L 11.6 µg/L	6 wks 3 wks	-yolk-sac malformations -edema -eye deformities	8.8a µg/g (eggs)	Effects on embryos incubated with solar ultraviolet light radiation	Hall and Oris, 1991
freshwater topminnows, *Poeciliopsis monacha Poeciliopsis lucida*	water; acetone carrier	lab; static renewal	BaP	1,000 µg/L nominal; 1,250 µg/L was acutely lethal	24 h followed by 6 mo. of monitoring	-increased AHH and EROD activities	9.0 µg/g converted from 35.7 nmol/g wet wt.	Implied effect - increased AHH and EROD activity indicative of carcinogenic and teratogenic metabolites formed during metabolism of BaP by MFO-system	Goddard et al., 1987
English sole (embryos), *Parophrys vetulus*	matemal via oral	lab; wild-caught	BaP	8,000 µg/L (8 mg/kg force-fed)	-	-malformation of tail regions -insufficient yolk-sac -reduced fin-fold size -reduced hatching success	51.2 and 263 µg/g (eggs) - avg. = 157; Tissue conc. from 80 mg/kg i.p. maternal injection	-Eggs maintained 11 days until yolk-sac absorbed; static. -Incidence of effect 4 times greater than controls (Chai-square df=3.81)	Hose et al., 1981
Rainbow trout (embryos), *Oncorhynchus mykiss*	aqueous from BaP spiked to sediment	lab; static renewal (7-10d)	BaP	0.21 µg/L measured	through to 36 d post-hatch	-nuclear pycnosis -lack of body pigment -insufficient yolk-sac -abnormalities of eyes -increased mortality (at 2.40 µg/L in aqueous) -muscle necrosis -abnormal mitosis in eyes and brains	1.93 µg/g (eggs), 12.34 µg/g (alevins), from exposure to 2.40 µg/L BaP	Poor control survival (52% mortality)	Hannah et al., 1982; Hose et al., 1984
Sand sole (embryos), *Psettichthys melanostichus*	water; static	lab	BaP	0.1 µg/L measured; range (0.08 - 0.12)	through to yolk-sac absorption (7 -10 d)	-overgrowth of tissues -arrested development -twinning; Effects only after 48 h, i.e., during organogenesis	2.1 µg/g wet weight	effects only exhibited in 5% of animals; average hatching success of controls only 57% versus 28% BaP-treated	Hose et al., 1982

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Flathead sole (embryos), *Hippoglossoide selassodon*	water; static	lab	BaP bound to bovine serum albumin	4.2 µg/L decreasing to <0.05 µg/L (DL)	through to yolk-sac absorption (7 -10 d)	-hatching success sig. decrease -nuclear pycnosis and general disruption of neural and ocular tissues	-	very low hatching success in controls and experimentals; 5.5 and 11.5%, respectively	Hose et al., 1982
English sole (embryos), *Parophrys vetulus*	water	lab	BaP	2.1 µg/L measured	through to yolk-sac absorption (7 -10 d)	none	-	-	Hose et al., 1982
gizzard shad, *Dorosoma cepedianum*	water via treated sediment	lab; static	BaP	1.38 µg/g sediment (initial); 0.74 µg/g sediment (mean of days 4,8 and 15)	22 d	none	BDL in all but 2 fish on day 4 - (0.001 and 0.0002 µg/g wet weight)	-40 ligated shad in 250 L H_2O with 4.15 kg sediment -no sig. decline in sediment conc. after day 4.	Kolok et al., 1996
gizzard shad, *Dorosoma cepedianum*	water and/or sediment ingestion	lab; static	BaP	1.02 µg/g sediment (initial); 0.63 µg/g sediment (mean of days 4,8, and 15)	22 days	none	ligated fish: 0.010 µg/g wet weight (n=4) non- ligated: 0.012 µg/g wet weight (n=14)	-50 shad, 30 ligated; 20 non-ligated, in 500 L H_2O with 3.15 kg sediment -no sig. decline in sediment conc. after day 4 -all other tissue concs. BDL (n=26 ligated; n=6 non- ligated)	Kolok et al., 1996
estuarine clams, *Rangia cuneata*	water; acetone carrier	lab; static	BaP	30.5 µg/L	24 h	none	7.2 µg/g wet weight	-majority of BaP concentrated in the viscera (~75%) -n=5	Neff and Anderson, 1975
estuarine clams, *Rangia cuneata*	water; acetone carrier	lab; static	BaP	30.5 µg/L	24 h	none	5.7 µg/g wet weight	-majority of BaP concentrated in the viscera (~65%) -n=8	Neff and Anderson, 1975

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
coho salmon (24 h Post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	-	Effects on hatching, orientation, and foraging only.	Ostrander et al., 1988
coho salmon, (32 d post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	-	Effects on hatching, orientation, and foraging only.	Ostrander et al., 1988
coho salmon, (24 h Post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	0.54 decreasing to 0.15 nmol/mg protien from 2 to 68 d post fertilization	Conc. of BaP in tissue are not converted because wet weights were not given; only the mg protein/animal. Can possibly borrow weights from earlier paper.	Ostrander et al., 1989
coho salmon, (32 d post fertilization), *Oncorhynchus kisutch*	water; 0.5% DMSO	lab; static exposure then flow-through	BaP	25,000 µg/L	24 h	none	4.47 decreasing to 0.33 nmol/mg protien from 2 to 68 d post fertilization	Conc. of BaP in tissue are not converted because wet weights were not given; only the mg protein/animal. Can possibly borrow weights from earlier paper.	Ostrander et al., 1989
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 5 µg/L (steady-state); 24 µg/L (initial)	15 days	-reduction in % hatch -lateral folding of tail -absence of caudal fin folds -hemorrhagic lesion or congested vasculature in caudal region	day 15: 0.992 ppm (wet weight); 6.872 ppm (dry weight)	steady state concentration reached in 4 to 10 days	Winkler et al., 1983

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 5-24 µg/L (steady state); 24-361 µg/L (initial)	15 days	-retarded growth (14d) -sporadic heart beat -displaced head relative to yolk-sac -absence of melanophores near lateral lines -absence of lens formation -lesions as larvae (above)	day 15:0.92 to 10.48 µg/g wet weight; 6.87 to 62.80 µg/g (dry weight)	steady state concentration reached in 4 to 10 days	Winkler et al., 1983
Calif. grunion (embryos), *Leuresthes tenuis*	water	lab; static	BaP	measured: 869 ppb (initial); steady-state not reached	15 days	-retarded growth (14d) -lateral curvature mid-body -absent melanophores -unused yolk sac -lesions as larvae (above)	day 15 - 19.98 µg/g wet weight; 112.03 µg/g dry weight	steady-state concentration never reached	Winkler et al., 1983
Pacific herring (embryos), *Clupea pallasi*	seawater contaminated by contact with oiled gravel - experiment 1; less weathered	lab; static	Field Mixture[A]	9.1 µ/L	16 days	-yolk sac edema	13.7 µg/g wet weight	Crude Oil characterized for PAHs only; concentrations of individual PAHs not given	Carls et al., 1999
Pacific herring (embryos), *Clupea pallasi*	seawater contaminated by contact with oiled gravel - experiment 2; more weathered	lab; state	Field Mixture[A]	0.41 µ/L to 0.72 µ/L	16 days	- yolk sac edema -pericardial edema - skeletal, spinal, and craniofacial abnormalities - anaphase aberration	0.022 µg/g wet weight	Crude Oil characterized for PAHs only; concentrations of individual PAHs not given	Carls et al., 1999

[A]Artificially weathered Alaska North Slope crude oil.

Appendix H

Carcinogenic Effects from Laboratory and Field Exposure to PAHs and PAH Mixtures.

Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Japanese Medaka, *Oryzia latipes* (6-10 d old)	Water; dimethyl-formamide carrier.	Lab; static	BaP	261 µg/L	2 x 6h, 1 week apart	Neoplastic lesions in livers and other tissues after 36 weeks 36% vs 1% (controls); 20 fish with adenoma, 6 with hepatocellular carcinoma	-	Exposures carried out at 26°C in the dark; concentration exceeds saturation solubility of BaP	Hawkins et al, 1988; Hawkins et al., 1990
guppy, *Poecilia reticulata* (6-10 d old)	Water; dimethyl-formamide carrier.	Lab; static	BaP	209 µg/L	2 x 6h, 1 week apart	Neoplastic lesions in livers and other tissues after 52 weeks 23% vs 0% (controls); 1 altered foci, 5 adenoma, 4 with hepatocellular carcinoma	-	Studies carried out longer because tumorigenic response in guppy is slower than in medaka	Hawkins et al, 1988; Hawkins et al., 1990
Rainbow trout (fingerlings), *Oncorhynchus mykiss*	oral	Lab	BaP	1,000 ppm per feeding	12 and 18 months	Incidence of neoplasms on liver 15% (1.0/liver) at 12 months 25% (7.7/liver) at 18 months	-	MFO info also available 0% at 6 months 0% on other organs	Hendricks et al., 1985
Rainbow trout (juvenile), *Oncorhynchus mykiss* (10 mo)	ip injection	Lab	BaP	1 mg B(a)P in 0.4 ml PG (1/month for 12 months)	18 months (6 months after final injection)	Incidence of neoplasms in various organs = 46% (x = 7.7 tumors/organ)	-	Organs examined = gonads, swim bladder, liver, spleen, head and trunk kidneys, pancreas, intestines, and stomach	Hendricks et al., 1985
Poeciliopsis lucida and *Poeciliopsis monacha* (1-7 months old)	water; acetone carrier	Lab: (multiple exposures) 3 to 4 exposure periods of 5-20 hours each week	7,12-dimethylbenz(a)-anthracene	5 ppm (per exposure)	7 - 8 months (from initial exposure)	incidence of hepatic tumors = 48%	-	only survivors examined = (55% mortality in 5 ppm treatment) (13% mortality in control)	Schultz and Schultz 1982

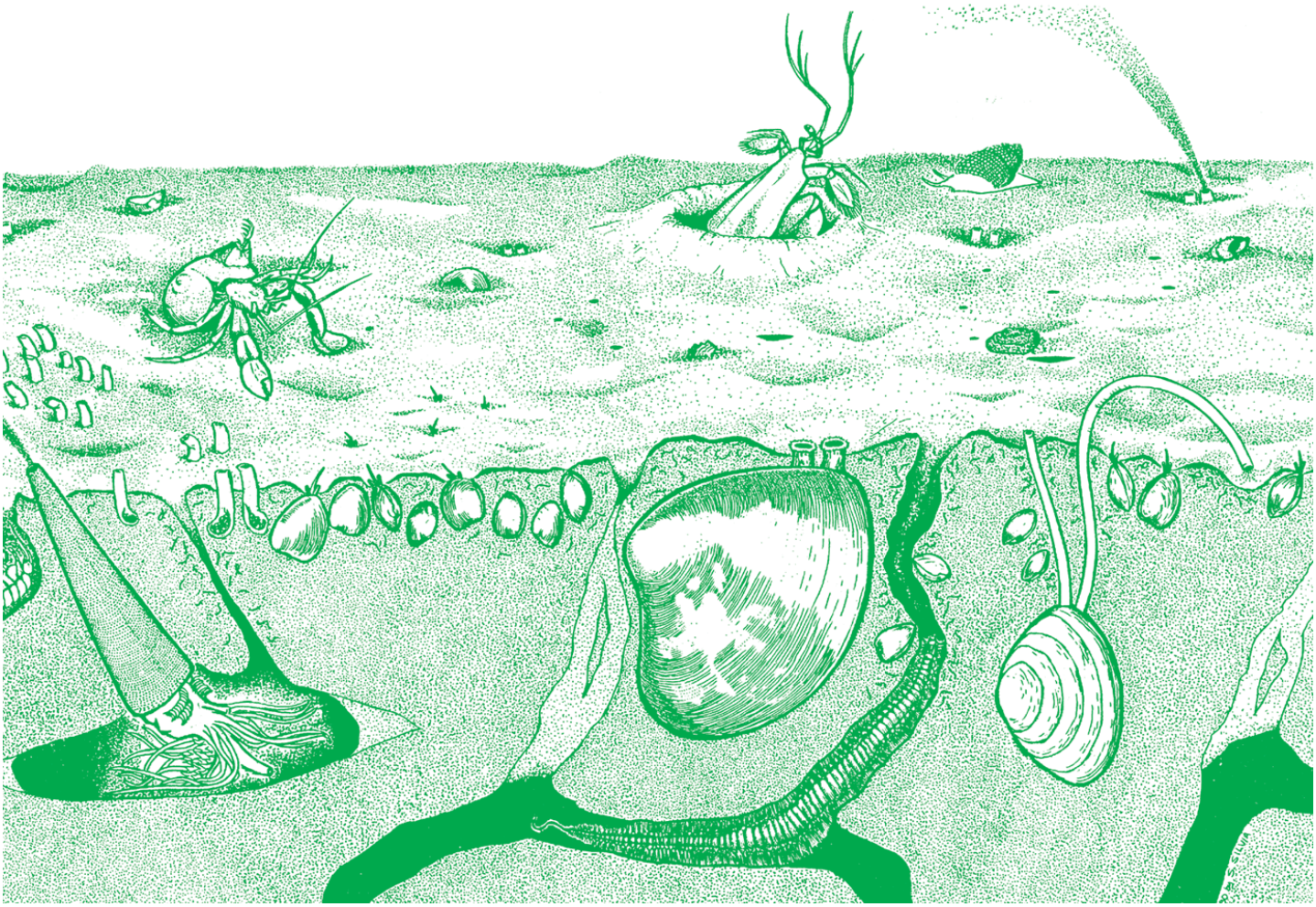
Species	Mode of Exposure	Method	PAH	Exposure Conc Associated with Effect	Exposure Time	Toxic Effect(s):	Tissue Conc	Comments:	References
Poeciliopsis lucida and *Poeciliopsis monacha* (1-6 weeks old)	water; acetone carrier	Lab: (multiple exposures) 5 exposures periods of 6 hours each week	7,12-dimethylbenz(a)-anthracene	5 ppm (per exposure)	6 - 7 months	Incidence of hepatic tumors = 41.8%	-	22% mortality in treatment 16% mortality in control Tumor-bearing livers enlarged, yellow-white to greenish and granular.	Schultz and Schultz 1982
Bullheads	Direct skin (river sediment extract)	Lab	Field Mixture[A]	5% RSE painted once per week	18 months	23% of survivors hyperplastic 9% with multiple papillomas	-	Survival of control and experimental fish was 31%.	Black, 1983
Japanese Medaka, *Poecilia reticulata* (6-10 d old)	Water via Sediment extract re-dissolved in acetone	Lab	Field Mixture[B]	182 ppb TPAH Black River, OH extract; 254 ppb TPAH Fox River, WI extract	24 h	hepatocellular carcinoma - Black River Ex. (2/15 fish); Pancreatic-duct cell adenoma - Fox River Ex. (1/15 fish)	-	No incidence of carcinomas in controls up to 270 days post-exposure; one incidence of lymphoma after 360 days of exposure.	Fabacher et al., 1991
Rainbow trout (embryos), *Oncorhynchus mykiss*	injection of sediment extract into yolk sac	Lab	Field Mixture[C]	Doses[D]: (Exp I) 0.006 g (Exp II) 0.012 g 0.006 g 0.003 g	1 year	Hepatic carcinomas (I) 8.9% (11/123) (II) 8.1% (12/148) 4.0% (5/148) 3.1% (2/65)	-	Note; PCBs also present sediment from Hamilton Harbour	Metcalfe et al 1988

[A] Buffalo River, NY; total no. PAHs measured = 13, total no. of carcinogenic PAHs = 6.
[B] Black River, OH. And Fox River, WI; full compliment of measured PAHs.
[C] Hamilton Harbor, ON, Canada; total no. PAHs measured = 13, total no. of carcinogenic PAHs = 6.
[D] Doses are calculated as gram equivalent wet weight of sediment represented by the volume of extract micro-injected into each trout sac-fry.

United States
Environmental Protection
Agency

Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms

Compendium of Tier 2 Values for Nonionic Organics



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Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Compendium of Tier 2 Values for Nonionic Organics

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Notice

The Office of Research and Development (ORD) has produced this compendium document to provide procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for several nonionic organic chemicals. ESBs may be useful as a complement to existing sediment assessment tools. This document should be cited as:

> U.S. EPA. 2008. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Compendium of Tier 2 Values for Nonionic Organics. EPA-600-R-02-016. Office of Research and Development. Washington, DC 20460

This document, and the other ESB documents, can also be found in electronic format at the following web address:

> http://www.epa.gov/nheerl/publications/

The information in this document has been funded wholly by the U.S. Environmental Protection Agency. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Abstract

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations for 32 nonionic organic chemicals in sediment which are protective of the presence of freshwater and marine benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration. This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any single effect endpoint. For the purposes of this document, ESBs for 32 nonionic organic chemicals, including several low molecular weight aliphatic and aromatic compounds, pesticides, and phthalates, were derived using Final Chronic Values (FCV) from Water Quality Criteria (WQC) or Secondary Chronic Values (SCV) derived from existing toxicological data using the Great Lakes Water Quality Initiative (GLI) or narcosis theory approaches. These values are intended to be the concentration of each chemical in water that is protective of the presence of aquatic life. For nonionic organic chemicals demonstrating a narcotic mode of action, ESBs derived using the GLI approach specifically for freshwater organisms were assumed to also be protective of marine organisms. This assumption is based on the similar sensitivity of freshwater and marine organisms to narcotic chemicals like some of the nonionic organics in this document. For this reason, SCVs derived using narcosis theory are protective of both freshwater and marine organisms. For chemicals with more specific modes of action, freshwater and marine organisms were not assumed to be similar in sensitivity, and separate freshwater and marine ESBs were derived as the available data allowed. Because of the lack of a comprehensive toxicity data set and other reasons discussed in this document in detail, values derived here are considered Tier 2 ESBs (ESB_{Tier2}). The presentation of these ESBs is such that updated values could be calculated as new toxicity data become available.

The ESB_{Tier2} is derived by multiplying the FCV or SCV by a chemical's K_{OC}, yielding the concentration in sediment that should provide the same level of protection that the FCV or SCV provides in water. The ESB_{Tier2} should be interpreted as a chemical concentration below which adverse effects are not expected. At concentrations above the ESB_{Tier2}, and assuming equilibrium between phases, effects may occur with increasing severity as the degree of exceedance increases. The document also includes examples demonstrating the calculation of conventionally-derived and narcosis-based ESBs that discuss an approach for addressing mixtures of narcotic chemicals.

ESB documents have also been developed for two pesticides (endrin, dieldrin), polycyclic aromatic hydrocarbon (PAH) mixtures, and metal mixtures.

The ESBs do not intrinsically consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with the individual nonionic organic chemicals discussed in this document or the potential for bioaccumulation and trophic transfer of these chemicals to aquatic life, wildlife or humans. However, for narcotic chemicals, an approach for considering the toxicity of mixtures is presented. Important assumptions and considerations for applying and interpreting the ESBs are also discussed.

Foreword

Under the Clean Water Act (CWA), the U.S. Environmental Protection Agency (EPA) and the States develop programs for protecting the chemical, physical, and biological integrity of the Nation's waters. To support the scientific and technical foundations of the programs, EPA's Office of Research and Development has conducted efforts to develop and publish equilibrium partitioning sediment benchmarks (ESBs) for some of the 65 toxic pollutants or toxic pollutant categories. Toxic contaminants in bottom sediments of the Nation's lakes, rivers, wetlands, and coastal waters create the potential for continued environmental degradation even where water column contaminant levels meet applicable water quality standards. In addition, contaminated sediments can lead to water quality impacts, even when direct discharges to the receiving water have ceased.

The ESBs and associated methodology presented in this document provide a means to estimate the concentrations of a substance that may be present in sediment while still protecting benthic organisms from the effects of that substance. These benchmarks are applicable to a variety of freshwater and marine sediments because they are based on the biologically available concentration of the substance in the sediments. These ESBs are intended to provide protection to benthic organisms from direct toxicity due to this substance. In some cases, the additive toxicity for specific classes of toxicants (e.g., metal mixtures or polycyclic aromatic hydrocarbon mixtures) is addressed. The ESBs do not intrinsically consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with the individual nonionic organic chemicals discussed in this document or the potential for bioaccumulation and trophic transfer of these chemicals to aquatic life, wildlife or humans. However, for narcotic chemicals, the ESBs can be used in a framework to evaluate the toxicity of mixtures.

ESBs may be useful as a complement to existing sediment assessment tools, to help evaluate the extent of sediment contamination, to identify chemicals causing toxicity, and to serve as targets for pollutant loading control measures. Both types of ESBs, Tier 1 and Tier 2, are intended for similar applications with the user's understanding that, because of limited data availability, Tier 2 ESBs are likely to have greater uncertainty associated with them as compared to Tier 1 ESBs. As new, high quality toxicological and geochemical data becomes available, it is encouraged that the ESB values are revised and updated.

This document provides technical information to EPA Program Offices, including Superfund, Regions, States, the regulated community, and the public. Decisions about risk management are the purview of individual regulatory programs, and may vary across programs depending upon the regulatory authority and goals of the program. For this reason, each program will have to decide whether the ESB approach is appropriate to that program and, if so, how best to incorporate this technical information into that program's assessment process. While it was necessary to choose specific parameters for the purposes of this document, it is important to realize that the basic science underlying this document can be adapted to a range of risk management goals by adjusting the input parameters. At the same time, the ESBs do not substitute for the CWA or other EPA regulations, nor are they regulation. Thus, they cannot impose legally binding requirements on EPA, States, or the regulated community. EPA and State decision makers retain the discretion to adopt approaches on a case-by-case basis that differ from this technical information where appropriate. It is recommended that the ESBs not be used alone but with other sediment assessment methods to make informed management decisions. EPA may change this technical information in the future. This document has been reviewed by EPA's Office of Research and Development (Atlantic Ecology Division, Narragansett, RI), undergone an external peer review, and approved for publication.

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Front cover image provided by Wayne R. Davis and Virginia Lee.

Contents

Tables

Figures

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**Series Editor

Executive Summary

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations of 32 nonionic organic chemicals in sediment which are protective of the presence of freshwater and marine benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration (U.S. EPA 2003a). This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

EqP theory holds that a nonionic chemical in sediment partitions between sediment organic carbon, interstitial (pore) water and benthic organisms. At equilibrium, if the concentration in any one phase is known, then the concentrations in the others can be predicted. The ratio of the concentration in water to the concentration in organic carbon is termed the organic carbon-water partition coefficient (K_{OC}), which is a constant for each chemical. The ESB Technical Basis Document (U.S. EPA 2003a) demonstrates that biological responses of benthic organisms to nonionic organic chemicals in sediments are different across sediments when the sediment concentrations are expressed on a dry weight basis, but similar when expressed on a µg chemical/g organic carbon basis ($\mu g/g_{OC}$). Similar responses were also observed across sediments when interstitial water concentrations were used to normalize biological availability. The Technical Basis Document (U.S. EPA 2003a) further demonstrates that if the effect concentration in water is known, the effect concentration in sediments on a $\mu g/g_{OC}$ basis can be accurately predicted by multiplying the effect concentration in water by the chemical's K_{OC}.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any single effect endpoint. For the purposes of this document, ESBs for 32 nonionic organic chemicals, including several low molecular weight aliphatic and aromatic compounds, pesticides, and phthalates, were derived using Final Chronic Values (FCV) from Water Quality Criteria (WQC) or Secondary Chronic Values (SCV) derived from existing toxicological data using the Great Lakes Water Quality Initiative (GLI) or narcosis theory approaches. These values are intended to be the concentration of each chemical in water that is protective of the presence of aquatic life. For nonionic organic chemicals demonstrating a narcotic mode of action, ESBs derived using the GLI approach specifically for freshwater organisms were assumed to also be protective of marine organisms. This assumption is based on the similar sensitivity of freshwater and marine organisms to narcotic chemicals like some of the nonionic organics in this document. For this reason, SCVs derived using narcosis theory are presumed to be protective of both freshwater and marine organisms. For chemicals with other specific modes of action, freshwater and marine organisms were not assumed to have similar sensitivity and separate freshwater and marine ESBs were derived as the available data allowed. For pesticides, only freshwater- and marine-specific FCVs or SCVs were used to derive ESBs because of likely differences between freshwater and marine organism sensitivities. Similarly, for the phthalates, which are not thought to be narcotic, SCVs were derived using the GLI approach and considered protective of freshwater species only. Because of the lack of a comprehensive toxicity data set and other reasons discussed in this document in detail, values derived here are considered Tier 2 ESBs (ESB_{Tier2}). Ancillary analyses conducted as part of this derivation suggest that the sensitivity of benthic/epibenthic organisms is not significantly different from pelagic organisms; for this reason, the FCV or SCV and the resulting ESB_{Tier2} should be fully applicable to benthic organisms. The ESB_{Tier2} is derived by multiplying the FCV or SCV by a chemical's K_{OC}, yielding the concentration in sediment that should provide the same level of protection that the FCV or SCV provides in water. The ESB_{Tier2} should be interpreted as a chemical concentration below which adverse effects are not expected. At concentrations above the ESB_{Tier2}, assuming equilibrium between phases, effects may occur with increasing severity as the degree of

exceedance increases. A sediment-specific site assessment (e.g., toxicity testing) would provide further information on chemical bioavailability and the expectation of toxicity relative to the ESB_{Tier2} along with associated uncertainties. The document also includes examples demonstrating the calculation of conventionally-derived and narcosis-based ESBs that discuss an approach for addressing mixtures of narcotic chemicals.

As discussed, while this document uses the FCV or SCV, the EqP methodology can be used by environmental managers to derive a benchmark with any desired level of protection, so long as the water-only concentration affording that level of protection is known. Therefore, the resulting benchmark can be species or site-specific if the corresponding water-only information is available. For example, if a water-only effects concentration is known for an economically important benthic species, that value could be used to derive a sediment benchmark commensurate with the protection of that species and endpoint. Another way to increase the site-specificity of an ESB would be to incorporate information on sediment-specific partitioning of chemicals, particularly for sites where the composition and partitioning behavior of the sediment organic carbon may be substantially different than for typical diagenic organic matter (see U.S. EPA 2003b). However, it should also be noted that the ability to predict partitioning based on additional partitioning factors like black carbon is still evolving and may serve to decrease partitioning-related uncertainties in future applications.

The ESBs do not intrinsically consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with the individual nonionic organic chemicals discussed in this document or the potential for bioaccumulation and trophic transfer of these chemicals to aquatic life, wildlife or humans. However, for narcotic chemicals, ESB values may be used in a framework to evaluate the potential effects of chemical mixtures. Consistent with the recommendations of EPA's Science Advisory Board, publication of these documents does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, ESB exceedances could be used to trigger the collection of additional assessment data. Similarly, ESBs are supportive of recent recommendations by Wenning et al. (2005), to apply a weight of evidence approach when evaluating contaminated sediments. These ESBs apply only to sediments having $\geq 0.2\%$ total organic carbon by dry weight and nonionic organic chemicals with log K_{OW}s ≥ 2.

Tier 1 and Tier 2 ESB values were developed to reflect differing degrees of data availability and uncertainty. Tier 1 ESBs have been derived for the nonionic organic pesticides endrin and dieldrin (U.S. EPA 2003c,d), polycyclic aromatic hydrocarbon (PAH) mixtures (U.S. EPA 2003e), and metal mixtures (U.S. EPA 2005a). Tier 2 ESBs for several nonionic organic chemicals for freshwater and marine sediments are reported in this document. Both types of ESBs are intended for similar applications with the user's understanding that Tier 2 ESBs are likely to have greater uncertainty associated with them as compared to Tier 1 ESBs. As new, high quality toxicological and geochemical data becomes available, recalculation of the Tier 2 ESB values is encouraged.

Uncertainties associated with ESB_{Tier2} values are discussed in detail through-out this document. They include unknown effects of antagonism, synergism and additivity, occurrence of chemical disequilibria, and presence of unusual types of sedimentary carbon, like black carbon, and large particles. Uncertainties for the ESB_{Tier2} values can be reduced by conducting additional acute and chronic water-only and spiked sediment toxicity tests to refine water-only effect concentrations and confirm predictions of sediment toxicity, respectively.

Glossary of Abbreviations

ACR	Acute–chronic ratio
AQUIRE	Aquatic Toxicity Information Retrieval
ASTER	ASsessment Tools for the Evaluation of Risk
ASTM	American Society for Testing and Materials
C_L*	Critical lipid concentration
CAS	Chemical Abstracts Service
CWA	Clean Water Act
DOC	Dissolved organic carbon
EC_{50}	Chemical concentration estimated to cause adverse effects to 50% of the test organisms within a specified time period
ECOTOX	ECOTOXicology databases
EMAP	Environmental Monitoring and Assessment Program
EPA	United States Environmental Protection Agency
EqP	Equilibrium partitioning
ESB	Equilibrium partitioning Sediment Benchmark; for nonionic organics, this term usually refers to a value that is organic carbon–normalized (more formally ESB_{OC}) unless otherwise specified
$ESB_{DRY\ WT}$	Equilibrium partitioning Sediment Benchmark; for nonionic organics, expressed on a sediment dry weight basis
ESB_{OC}	Equilibrium partitioning Sediment Benchmark; for nonionic organics, expressed on an organic carbon basis
ESB_{Tier2}	Equilibrium partitioning Sediment Benchmark; for nonionic organics, derived using Tier 2 data; specifically, the values in this document
$ESB_{Tier2DRY\ WT}$	Equilibrium partitioning Sediment Benchmark; for nonionic organics, derived using Tier 2 data, expressed on a sediment dry weight basis
$ESB_{Tier2OC}$	Equilibrium partitioning Sediment Benchmark; for nonionic organics, derived using Tier 2 data; expressed on organic carbon basis

ESBTU	Equilibrium Partitioning Sediment Benchmark Toxic Units
FACR	Final acute–chronic ratio
FAV	Final acute value
FCV	Final chronic value
f_{OC}	Fraction of organic carbon in sediment
f_{Solids}	Fraction of solids in sediment
GLI	Great Lakes Water Quality Initiative
GMAV	Genus mean acute value
GMCV	Genus mean chronic value
g_{OC}	Gram organic carbon
HECD	U.S. EPA, Health and Ecological Criteria Division
IC_{50}	Chemical concentration estimated to cause some form of inhibition to 50% of the test organisms within a specified time period
K_{BC}	Black carbon-water partition coefficient
K_{OC}	Organic carbon–water partition coefficient
K_{OW}	Octanol–water partition coefficient
K_P	Sediment–water partition coefficient
LC_{50}	Chemical concentration estimated to be lethal to 50% of test organisms within a specified time period
MC	Moisture content
MDR	Minimum data requirement
NHEERL	U.S. EPA, National Health and Environmental Effects Research Laboratory
OECD	Organization for Economic Cooperation and Development
ORD	U.S. EPA, Office of Research and Development
OST	U.S. EPA, Office of Science and Technology
OSWER	U.S. EPA, Office of Solid Waste and Emergency Response

PAH	Polycyclic aromatic hydrocarbon
PM	Particulate matter
QSAR	Quantitative structure-activity relationship
SACR	Secondary acute-chronic ratio
SAF	Secondary acute factor
SAV	Secondary acute value
SCV	Secondary chronic value
SCV_N	Secondary chronic value based on narcosis theory
SMACR	Species mean acute–chronic ratio
SMAV	Species mean acute value
SPARC	SPARC Performs Automated Reasoning in Chemistry
STORET	EPA's computerized database for STOrage and RETrieval of water-related data
TIE	Toxicity Identification Evaluation
TOC	Total organic carbon
WQC	Water Quality Criteria

Section 1

Introduction

1.1 General Information

Toxic pollutants in bottom sediments of the Nation's lakes, rivers, wetlands, estuaries, and marine coastal waters create the potential for continued environmental degradation even where water column concentrations comply with established WQC. In addition, contaminated sediments can be a significant pollutant source that may cause water quality degradation to persist, even when other pollutant sources are stopped (Larsson 1985, Salomons et al. 1987, Burgess and Scott 1992). The absence of defensible equilibrium partitioning sediment benchmarks (ESBs) make it difficult to accurately assess the extent of the ecological risks of contaminated sediments and to identify, prioritize, and implement appropriate cleanup activities and source controls (U.S. EPA 1997a, b, c, 2004).

As a result of the need for a procedure to assist regulatory agencies in making decisions concerning contaminated sediment problems, the U.S. Environmental Protection Agency (EPA) Office of Water Office of Science and Technology, Health and Ecological Criteria Division (OST/HECD) and Office of Research and Development National Health and Environmental Effects Research Laboratory (ORD/NHEERL) established a research team to review alternative approaches (Chapman 1987). All of the approaches reviewed had both strengths and weaknesses, and no single approach was found to be applicable for the derivation of guidelines in all situations (U.S. EPA 1989, 1993). The equilibrium partitioning (EqP) approach was selected for nonionic organic chemicals because it presented the greatest promise for generating defensible, national, numeric chemical-specific benchmarks applicable across a broad range of sediment types. The three principal observations that underlie the EqP approach to establishing sediment benchmarks are as follows:

1. The concentrations of nonionic organic chemicals in sediments, expressed on an organic carbon basis, and in interstitial waters correlate to observed biological effects on sediment-dwelling organisms across a range of sediments.

2. Partitioning models can relate sediment concentrations for nonionic organic chemicals on an organic carbon basis to freely-dissolved concentrations in interstitial water.

3. The distribution of sensitivities of benthic organisms to chemicals is similar to that of water column organisms; thus, the currently established water quality criteria (WQC) final chronic values (FCV) or secondary chronic values (SCV) can be used to define the acceptable effects concentration of a chemical freely-dissolved in interstitial water.

The EqP approach, therefore, assumes that (1) the partitioning of the chemical between sediment organic carbon and interstitial water is at or near equilibrium; (2) the concentration in either phase can be predicted using appropriate partition coefficients and the measured concentration in the other phase (assuming the freely-dissolved interstitial water concentration can be accurately measured); (3) organisms receive equivalent exposure from water-only exposures or from any equilibrated phase: either from interstitial water via respiration, from sediment via ingestion or other sediment-integument exchange, or from a mixture of exposure routes; (4) for nonionic chemicals, effect concentrations in sediments on an organic carbon basis can be predicted using the organic

carbon partition coefficient (K_{OC}) and effects concentrations in water; (5) the FCV or SCV concentration is an appropriate effects concentration for freely-dissolved chemical in interstitial water; and (6) ESBs derived as the product of the K_{OC} and FCV or SCV are protective of benthic organisms. ESB concentrations presented in this document are expressed as µg chemical/g sediment organic carbon (µg/g_{OC}) and not on an interstitial water basis because (1) interstitial water is difficult to sample and (2) significant amounts of the dissolved chemical may be associated with dissolved organic carbon; thus, total concentrations in interstitial water may overestimate exposure.

1.2 Development of Tier 2 Sediment Benchmarks

Aquatic toxicity values used in this compendium (Table 3-1) were developed in two possible ways: (1) conventionally using Water Quality Criteria (WQC) (when available) and Great Lakes Water Quality Initiative (GLI) generated values, and (2) narcosis theory. This compendium consists of Tier 2 ESBs for 32 chemicals including several low molecular weight aliphatic and aromatic compounds, pesticides and phthalates. Both types of ESBs, Tier 1 and Tier 2, are intended for similar applications with the user's understanding that Tier 2 ESBs are likely to have greater uncertainty associated with them as compared to Tier 1 ESBs. See Section 1.3 for further discussion of Tier 1 and Tier 2 ESBs.

The ESB values are reported in Tables 3-2 and 3-4. In the *References* section, along with the cited sources, the reference U.S. EPA (2001a) contains the sources and tables of data used to derive some of the Tier 2 ESBs.

For many of the chemicals in this document, the Tier 2 ESBs were developed using the GLI (1995) methodology for obtaining secondary chronic values (SCVs). As described in Section 2 and Appendix A, this methodology uses adjustment factors to allow derivation of chronic values when fewer toxicity data are available than are required under the National Ambient Water Quality Criteria methodology (Stephan et al. 1985). Because of these adjustment factors, SCVs are generally expected to be lower than would be likely if a complete data set were available. Consequently, Tier 2 ESBs would tend to be lower (i.e., be more conservative) compared to the Tier 1 ESBs developed exclusively from FCVs. The degree of conservatism will be a function of the database used to derive the SCVs. Further, the presence of these chemicals in mixtures will also affect the conservatism (see Section 4.2.5). The SCVs used in calculating most Tier 2 ESBs were derived using toxicity data primarily for freshwater species. In the toxicity data evaluation for the PAH mixtures ESB (U.S. EPA 2003e), there was no significant difference in sensitivity between freshwater and saltwater species when distributions of data for all species were compared using the approximate randomization (AR) method (Noreen 1989, U.S. EPA 2003e). Like PAHs, many of the Tier 2 ESB chemicals are also narcotics; from this, it is reasonable to presume that these ESBs would be applicable to both freshwater and saltwater sediments.

For pesticides, there are likely to be differences between FCVs or SCVs developed for freshwater and saltwater organisms (e.g., Thursby 1990, U.S. EPA 1980a,b, 1986, 1996, 2005b). Therefore, applying Tier 2 ESB values for pesticides derived using the GLI methodology to saltwater sediments is not recommended and would result in increased uncertainties. To address these uncertainties, Tier 2 ESBs are presented for pesticides for both freshwater and marine organisms based on FCVs from WQC (when available) or SCVs. Similarly, SCVs developed for phthalates in this document using the GLI approach were assumed to be protective only of freshwater species. Unlike the pesticides, WQC FCVs were not available for either freshwater or marine species for the phthalates.

As noted, many of the chemicals for which EPA has developed Tier 2 ESBs are known or suspected to affect aquatic organisms by a

narcotic mode of action (Russom et al. 1997). For these compounds, Tier 2 ESBs were also derived using the narcosis theory approach applied to develop ESBs for PAH mixtures (U.S. EPA 2003e). In contrast to the conventional GLI approach, the narcosis approach does not apply adjustment factors. As a consequence, narcosis-based values are often larger in magnitude compared to the GLI-derived values (discussed further in Section 2). In Table 3-1, narcosis-based SCVs are also reported for chemicals with other modes of actions in addition to narcosis (i.e., pesticides and phthalates). For these chemicals, potency via narcosis is generally small compared to the more specific mode(s) of action which would result in narcosis-based ESB values being considerably higher than the conventionally-derived values. Accepting these approaches for developing chronic toxicity values and the associated uncertainties, Tier 2 ESB values for narcotic chemicals, pesticides and phthalates should be meaningful interpretive tools for marine sediments as well as freshwater sediments (Tables 3-2 and 3-4).

With regard to using narcosis to derive ESB values, the approach applied in this document and U.S. EPA (2003e) uses narcosis theory to predict acute toxicity and then empirically based acute-chronic ratios (ACRs) to calculate chronic toxicity values. These chronic values (i.e., SCVs) are then used to calculate the ESBs. Strengthening our mechanistic understanding of the link between acute toxicity based on narcosis and chronic effects potentially caused by other forms of toxicity is an active area of research (e.g., Incardona et al. 2006). Users of this document should recognize deficiencies in our understanding of this link may introduce uncertainties into the narcosis based estimates of ESB values.

Regardless of the approach used to derive the Tier 2 toxicity values, these concentrations have been generated on a single chemical basis; that is, the benchmark addresses effects for that chemical only and does not consider additive effects from other chemicals that may be present in sediment. For that reason, as the number and concentration of other chemicals present increases, single chemical benchmarks would be expected to provide a lesser degree of protection than a mixtures-based approach. EPA has not yet recommended an approach for summing the particular chemicals in this document, but approaches for assessing the toxicity of narcotic mixtures in sediments have been published (Di Toro and McGrath 2000, DiToro et al. 2000), and the Agency has developed methodologies for deriving ESBs for mixtures of PAHs (U.S. EPA 2003e) and metals (U.S. EPA 2005a). The approach discussed in U.S. EPA (2003e) for addressing the toxicity of mixtures of PAHs may be useful for those interested in combining the toxic effects of narcotic chemicals in this compendium (see Section 4.3 for an example).

Values similar to some of those reported in this document were used to evaluate data for EPA's 1997 and 2004 National Sediment Quality Survey reports to Congress (USEPA 1997a,b,c, 2004). In those documents, the values were called sediment quality advisory levels (SQALs). These SQALs for nonionic organic chemicals were also included as "Ecotox Thresholds" in a 1996 ECO Update bulletin published by EPA's Office of Solid Waste and Emergency Response (OSWER) (U.S. EPA 1996). In some cases, the Tier 2 ESBs in this document may differ from the SQALs and Ecotox Thresholds because of different data sources. Further, the SQALs and Ecotox Thresholds did not include narcosis-based chronic toxicity values.

Sediment benchmarks generated using the EqP approach are suitable for use in providing technical information to regulatory agencies because they are:

1. Numeric values
2. Chemical specific
3. Applicable to most sediments
4. Predictive of biological effects
5. Protective of benthic organisms

ESBs are derived using the available scientific data to assess the likelihood of significant environmental effects to benthic organisms from chemicals in sediments in the same way that the WQC are derived using the available scientific data to assess the likelihood of significant environmental effects to organisms in the water column. As such, ESBs are intended to protect benthic organisms from the effects of chemicals associated with sediments and, therefore, only apply to sediments permanently inundated with water, to intertidal sediment, and to sediments inundated periodically for durations sufficient to permit development of benthic assemblages. ESBs should not be applied to occasionally inundated soils containing terrestrial organisms, nor should they be used to address the question of possible contamination of upper trophic level organisms or the generic synergistic, additive, or antagonistic effects of multiple chemicals. The application of ESBs under these conditions may result in values lower or higher than those presented in this document. It should be noted that under certain conditions with narcotic chemicals, additivity may be considered.

ESB values presented herein are the concentrations of 32 nonionic organic chemicals in sediment that are not expected to adversely affect most benthic organisms. Just as values in this document can be seen as an update of the SQALs and Ecotox Thresholds, it is recognized (and encouraged) that these ESB values may need to be adjusted to account for new data as they become available. They may also need to be adjusted because of site-specific considerations. For example, in spill situations, where chemical equilibrium between water and sediment has not yet been reached, sediment chemical concentrations less than an ESB may pose risks to benthic organisms. This is because for spills, disequilibrium concentrations in interstitial and overlying water may be proportionally higher relative to sediment concentrations. In systems where biogenic organic carbon dominates, research has shown that the source or 'quality' of total organic carbon (TOC) in natural sediments does not affect chemical partitioning when sediment toxicity was measured as a function of TOC concentration (DeWitt et al. 1992). K_{OC}s for several nonionic chemicals have also been shown to not vary significantly across estuarine sediments with differing organic carbon concentrations and quality (Burgess et al. 2000). However, in systems where other forms of carbon are present at elevated levels, the source or 'quality' of TOC may affect chemical binding despite expressing toxicity as a function of TOC concentration. At some sites, concentrations in excess of an ESB may not pose risks to benthic organisms because the compounds are partitioned to a component of a particulate phase such as black carbon or coal or exceed solubility such as in the case of undissolved oil or chemical (e.g., manufactured gas plant sites) (U.S. EPA 2003e, Cornelissen et al. 2005). In these situations, an ESB would be overly protective of benthic organisms and should not be used unless modified using the procedures outlined in "Procedures for the Derivation of Site-Specific Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Nonionic Organics" (U.S. EPA 2003b). It should also be noted that the ability to predict partitioning based on additional factors like black carbon is still evolving and may serve to decrease partitioning-related uncertainties in future applications. If the organic carbon has a low sorptive affinity (e.g., hair, wood chips, hide fragments), an ESB would be under protective. An ESB may also be under protective when the toxicity of other chemicals are additive with an ESB chemical or when species of unusual sensitivity occur at the site.

This document presents the derivation and calculation of Tier 2 ESBs for 32 nonionic organic chemicals. The data that support the EqP approach for deriving ESBs for nonionic organic chemicals are reviewed by Di Toro et al. (1991) and EPA (2003a). Before proceeding through the following text, tables, and calculations, the reader should also consider reviewing Stephan et al. (1985).

1.3 Application of Sediment Benchmarks

ESBs as presented in this document are meant to be used with direct toxicity testing of sediments as a method of sediment evaluation, assuming the toxicity testing species is sensitive to the chemical(s) of interest (e.g., ASTM 1998a,b,c, U.S. EPA 1994, 2000, 2001b). In this way, ESBs are supportive of recent recommendations by Wenning et al. (2005), to apply a weight of evidence approach when evaluating contaminated sediments. Specifically, the ESBs provide a chemical-by-chemical specification of sediment concentrations protective of benthic aquatic life (see Section 4.2.6 for more discussion). The EqP method should be most applicable to nonionic organic chemicals with a log $K_{OW} \geq 2$. However, for chemicals with log K_{OW} between 2 and 3, EqP will function but sedimentary conditions (i.e., f_{OC} and f_{Solids}) should be considered and adjustments to the derivation of the ESB maybe advisable (see Section 3.3). Examples of other chemicals to which the methodology applies include the pesticides endrin and dieldrin (U.S. EPA 2003c,d), metal mixtures (U.S. EPA 2005a), and PAH mixtures (U.S. EPA 2003e).

For the toxic chemicals addressed by the ESB documents, Tier 1 (U.S. EPA, 2003c, d, e, and 2005a) and Tier 2 (this document) values were developed to reflect the differing degrees of data availability and uncertainty. Tier 1 ESBs are more scientifically rigorous and data intensive than Tier 2 ESBs. The minimum requirements to derive a Tier 1 ESB include: (1) each chemical‘s organic carbon-water partition coefficient (K_{OC}) is derived from the octanol-water partition coefficient (K_{OW}) obtained using the SPARC model (Karickhoff et al. 1991) and the K_{OW}-K_{OC} relationship from Di Toro et al. (1991). This K_{OC} has been demonstrated to predict the toxic sediment concentration from the toxic water concentration with less uncertainty than K_{OC} values derived using other methods, (2) the FCV is updated using the most recent toxicological information and is based on the National WQC guidelines (Stephan et al. 1985), and (3) EqP-confirmation tests are conducted to demonstrate the accuracy of the EqP prediction that the K_{OC} multiplied by the effect concentration from a water-only toxicity test predicts the effect concentration from sediment tests (Swartz 1991, DeWitt et al. 1992, Hoke et al. 1994). Using these specifications, Tier 1 ESBs have been derived for the nonionic organic pesticides endrin and dieldrin (U.S. EPA 2003c,d), PAH mixtures (U.S. EPA 2003e), and metals mixtures (U.S. EPA 2005a). In comparison, the minimum requirements for a Tier 2 ESB (this document) are less rigorous: (1) the K_{OW} for the chemical that is used to derive the K_{OC} can be from slow-stir, generator column, shake flask, SPARC or other sources (e.g., Site 2001), (2) FCVs can be from published or draft WQC documents, the Great Lakes Water Quality Initiative (GLI 1995), or developed from AQUIRE (now ECOTOX). Secondary chronic values (SCV) from narcosis theory (Di Toro and McGrath 2000, Di Toro et al. 2000, U.S. EPA 2003e), Suter and Tsao (1996), or other effects concentrations from water-only toxicity tests can also be used. The U.S. EPA methodology for deriving water quality criteria SCVs required for the computation of Tier 2 ESBs is described in *Water Quality Guidance for the Great Lakes System: Supplementary Information Document (SID)* (U.S. EPA 1995a), and (3) EqP confirmation tests are recommended, but are not required for the development of Tier 2 ESBs. Because of these lesser requirements, there is greater uncertainty in the EqP prediction of the sediment effect concentration from the water-only effect concentration, and in the level of protection afforded by Tier 2 ESBs. This uncertainty can be decreased by conducting additional acute and chronic water-only and spiked sediment toxicity tests to evaluate effect concentrations and confirm predicted sediment concentrations, respectively.

1.4 Data Quality Assurance

Data sources, selections and manipulations used to generate K_{OW}s or K_{OC}s and SCV or FCVs are discussed in detail in Section 2. Toxicological data were selected from final and draft Water Quality Criteria, Suter and Tsao

(1996), U.S. EPA (1996), GLI (1995) and U.S. EPA (2001a) or derived using the approach described by Di Toro and McGrath (2000), Di Toro et al. (2000) and U.S. EPA (2003e). K_{OW} values were taken from Karickhoff and Long (1995) as well as other sources. Toxicity data were evaluated for acceptability using the procedures in Stephan et al. (1985), the Great Lakes Water Quality Initiative (GLI 1995), and the approach for deriving narcotic chronic toxicity values (Di Toro and McGrath 2000, Di Toro et al. 2000, U.S. EPA 2003e). Data not meeting criteria for acceptability were rejected. In general, three or four significant figures were used in intermediate calculations to limit the effect of rounding errors, and are not intended to indicate the true level of precision. The time periods covered in the literature searches associated with data in this document can be found in the cited source literature.

Literature searches supporting Suter and Tsao (1996), U.S. EPA (1996), GLI (1995) and U.S. EPA (2001a) were conducted in the mid-1990s. In order to capture more recent data, EPA's ECOTOX database (www.epa.gov/ecotox) was searched for any data pertaining to the chemicals evaluated in this document published after 1995. These data were then sorted to identify sources of acute toxicity data for North American species tested for a period appropriate to the species (Stephan et al. 1985) and for which test concentrations of chemical were measured. In addition, literature sources suggested by peer reviewers of this document were also consulted for data meeting minimum requirements. Fewer than 30 additional data points were identified, and only one of these affected the calculation of an SCV (see footnote in Table 3-1). As new, high quality toxicological and geochemical data becomes available, it is encouraged that the ESB values are revised and updated. See Section 2.5 for further discussion.

The document was reviewed as part of a formal external peer review coordinated at the U.S. EPA National Health and Environmental Effects Research Laboratory, Research Triangle Park, North Carolina and Atlantic Ecology Division, Narragansett, Rhode Island. Any errors of omission or calculation discovered during the peer review process were corrected.

1.5 Overview

This document presents the derivation and calculation of ESBs for 32 nonionic organic chemicals.

Section 2 reviews the toxicological and chemical data used to derive the ESB_{Tier2}s. Section 3 discusses the calculation of the ESB_{Tier2}s. Section 4 "Sediment Benchmark Values: Application and Interpretation" discusses the sediment benchmark values and lists several factors to consider when applying and interpreting these values. Section 5 lists references cited in all sections of this document. Appendix A discusses, in detail, the GLI approach for calculating chronic toxicity values.

Section 2

Derivation of Equilibrium Partitioning Sediment Benchmark Effects Concentrations

2.1 General Introduction

This section outlines the compilation of data used in the derivation of the Tier 2 ESBs presented in this compendium. The section follows the format for calculating the ESB values by first describing the derivation of the K_{OW} values, and then the derivation of the appropriate aquatic toxicity values. The derivation of the K_{OW} values follows procedures outlined in Karickhoff and Long (1996) and in many cases uses values summarized in Karickhoff and Long (1995). Because of the diversity of chemicals discussed in this compendium (i.e., narcotics, pesticides, phthalates), aquatic toxicity values were derived in two possible ways. Conventional aquatic toxicity values were derived either using the procedures detailed in the Great Lakes Water Quality Initiative (GLI, 1995) or taken from existing or draft WQC. For example, marine ESBs for pesticides were based only on FCVs from existing or draft WQC while freshwater ESBs for pesticides were derived using both WQC and GLI toxicity values. Similarly, ESBs for phthalates were derived only for freshwater species using the GLI approach as WQC values were not available. For chemicals designated as being narcotic, toxicity values were also derived using the narcosis theory used to develop ESBs for PAH mixtures (Di Toro et al. 2000, U.S. EPA 2003e). As discussed in Section 1, ESBs derived using either conventional or narcotic approaches, for narcotic chemicals in this document are applicable to both freshwater and marine species based on the concept that these organisms show similar sensitivity to narcotic chemicals. This concept was not exercised for pesticides and phthalates.

2.2 Determination of K_{OW} Values

The determination of K_{ow} values was based on experimental measurements taken primarily by the slow-stir, generator-column, and shake-flask methodologies. The SPARC properties calculator model (Karickhoff and Long 1995) was also used to generate K_{ow} values, when appropriate, for comparison with the measured values. Values that appeared to be considerably different from the rest were classified as outliers and were not used in the calculation. For each chemical, the available log K_{ow} value, based on one of the above mentioned methods, was given preference. If more than one such value was available, the log K_{ow} value was calculated as the arithmetic mean of those values (U.S. EPA 1995b). Most of the log K_{ow} values used in this document are summarized in an internal EPA report (Karickhoff and Long 1995). Subsequent to that evaluation, EPA has published a recommended procedure for selecting K_{ow} values, which can be seen in Karickhoff and Long (1996).

Log K_{ow} values were initially identified in summary texts on physical-chemical properties, such as Howard (1990) and Mackay et al. (1992a,b), and accompanying volumes. Additional compendia of log K_{ow} values were also evaluated including de Bruijn et al. (1989), De Kock and Lord (1987), Doucette and Andren (1988), Isnard and Lambert (1989), Klein et al. (1988), Leo (1993), Noble (1993), and Stephan (1993). To supplement these sources, on-line database searches were conducted in ChemFate,

TOXLINE, and Hazardous Substances Data Bank (HSDB) (National Library of Medicine); Internet databases such as EPA's ASsessment Tools for the Evaluation of Risk (ASTER) were also reviewed. Original references were located for the values, and additional values identified. In cases where log K_{ow} values varied over several orders of magnitude or measured values could not be identified, detailed on-line searches were conducted using TOXLIT, Chemical Abstracts, and DIALOG.

2.3 Selection and Determination of Aquatic Toxicity Values

For this discussion, all sources of toxicological information are considered 'conventionally-derived' approaches except for the narcosis source which will be referred to separately as the 'narcosis-based' approach.

A variety of sources were used for selecting conventional chronic toxicity values to be used in the derivation of the ESBs. The following were identified as possible sources to be used for determining chronic toxicity values:

1. Final Chronic Values from the Great Lakes Water Quality Initiative (GLI 1995, U.S. EPA 2001a)
2. Final Chronic Values from National Ambient Water Quality Criteria documents
3. Final Chronic Values from draft freshwater and marine National Ambient Water Quality Criteria documents
4. Final Chronic Values developed from data in AQUIRE (now ECOTOX) and other sources
5. Secondary Chronic Values from Suter and Tsao (1996)
6. Secondary Chronic Values developed from data in AQUIRE (now ECOTOX) and other sources (U.S. EPA 1996, 2001a)

2.3.1 Derivation of Conventional Chronic Toxicity Values

For the nine pesticides discussed in this document, values for freshwater ESBs for the following chemicals:

gamma-BHC/Lindane
diazinon
endosulfan (mixed isomers and alpha and beta forms)
toxaphene

were based on the FCVs from existing or draft National Ambient Water Quality Criteria documents (U.S. EPA 1980a,b, 1986, 2005b). Exceptions were the ESBs for BHCs other than Lindane, malathion and methoxychlor which were derived using SCVs with the GLI approach (GLI 1995, Suter and Tsao 1996, U.S. EPA 1996, 2001a). Marine ESBs for pesticides, in this document, were based only on WQC-derived FCVs. Consequently, marine ESBs for the following chemicals:

diazinon
endosulfan (mixed isomers and alpha and beta forms)
malathion
toxaphene

were derived from FCVs in existing or draft National Ambient Water Quality Criteria documents (Thursby 1990, U.S. EPA 1980b, 1986, 2005b). Similar FCVs for the pesticides BHCs other than Lindane, gamma-BHC/Lindane, and methoxychlor were unavailable and marine ESBs were not derived.

Twelve aquatic toxicity values, including three phthalates, used to develop freshwater SCVs were based on work conducted by Oak Ridge National Laboratories (Suter and Tsao 1996) using the GLI (1995) methodology. This methodology was developed to obtain whole-effluent toxicity screening values based on all available data, but the methodology can also be used to calculate SCVs with fewer toxicity data than are required for the WQC methodology. The SCVs are generally lower than values that are produced by the FCV methodology,

reflecting greater uncertainty and use of protective adjustment factors in the absence of additional toxicity data (see Section 2.4). According to GLI (1995), the minimum requirement for deriving an SCV is toxicity data from a single taxonomic family (Daphnidae), provided the data are acceptable. In general, those values from Suter and Tsao (1996), which included at least one daphnid test result in the calculation of the SCV, were included for the derivation of Tier 2 ESBs with the exception of ethylbenzene, toluene, 1,1,1-trichloroethane and trichloroethene. For these four chemicals, daphnids were not used for calculating the SCVs. SCVs from Suter and Tsao (1996) were used to develop Tier 2 ESBs for the following chemicals:

benzene
BHC (other than Lindane)
chlorobenzene
dibenzofuran
diethyl phthalate
di-n-butyl phthalate
ethylbenzene
tetrachloroethane, 1,1,2,2-
tetrachloroethene
toluene
trichloroethane, 1,1,1-
trichloroethene

A preliminary search of data records in the AQUIRE (now ECOTOX) database indicated that the following chemicals, which includes one phthalate, might have sufficient toxicity data for the development of SCVs using the GLI (1995) methodology:

biphenyl
4-bromophenyl phenyl ether
butyl benzyl phthalate
dichlorobenzene, 1,2-
dichlorobenzene, 1,3-
dichlorobenzene, 1,4-
hexachlorethane
malathion
methoxychlor
pentachlorobenzene
tetrachloromethane
tribromomethane
trichlorobenzene, 1,2,4-
m-xylene

The procedure used for deriving SCVs for other chemicals of concern using the GLI (1995) methodology and data from ACQUIRE (now ECOTOX) and other sources is described in detail in Appendix A and U.S. EPA (1996, 2001a).

2.3.2 Derivation of Narcotic Chronic Toxicity Values

Along with the derivation of aquatic toxicity values using conventional techniques (see discussion above), narcosis theory was used to derive SCVs for chemicals determined to be primarily narcotic in their mode of action by ASsessment Tools for the Evaluation of Risk (ASTER) (Russom et al. 1997). These chemicals include:

benzene
biphenyl
4-bromophenyl phenyl ether
chlorobenzene
dibenzofuran
1,2-dichlorobenzene
1,3-dichlorobenzene
1,4-dichlorobenzene
ethylbenzene
hexachloroethane
pentachlorobenzene
1,1,2,2-tetrachloroethane
tetrachloroethene
tetrachloromethane
toluene
tribromomethane
1,2,4-trichlorobenzene
1,1,1-trichloroethane
trichloroethene
m-xylene

It should be noted that for a given chemical multiple modes of action can affect an organism. Therefore, despite the categorization of these chemicals as primarily narcotics, other modes of action may be active. Section 4.3 discusses some of the implications of this issue.

Narcosis-based SCVs were derived using the approach discussed in the *Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH Mixtures* (U.S. EPA 2003e) and Di Toro et al. (2000). In this approach, the SCV for these narcotic chemicals is derived using Equation 2-1:

$$\log (SCV_N) = \log[C_L^* \Delta c_l \div ACR] - 0.945 \cdot \log (K_{OW}) \quad (2\text{-}1)$$

where, SCV_N is the narcosis-based SCV for a given chemical (mmol/L), C_L^* is the critical lipid concentration predicted to cause 50% mortality equaling 35.3 µmol/g octanol, Δc_l is the chemical class specific correction, ACR is the acute-chronic ratio equaling 5.09, -0.945 the universal narcosis slope, and K_{OW} is specific to the chemical being investigated (Di Toro et al. 2000). This equation can be simplified to:

$$\log (SCV_N) = \log (6.94) + \Delta c_l - 0.945 \cdot \log (K_{OW}) \quad (2\text{-}2)$$

For the narcotic chemicals in this document, the chemical class specific correction value (Δc_l) for halogenated compounds was -0.244. For all other compounds, a correction was not necessary (Di Toro et al. 2000).

Narcosis values were also calculated for chemicals with other toxicological modes of action; specifically, the pesticides and phthalates. In every instance, the narcosis SCV_N was larger in magnitude than the conventional FCV or SCV. For example, the range of the ratio of narcosis to conventional values was 2.4 for di-n-butyl phthalate to nearly 50,000 for alpha-endosulfan. In general, the ratio of narcosis to conventional values was greater than 1000 and thus the pesticides and phthalates contribute only a small amount of narcotic potency. Despite the utility of knowing the contribution of narcosis to the overall toxicity of the pesticides and phthalates, the narcosis values should be used with caution. The narcosis equation above (Equation 2-2) provides chemical class specific corrections (i.e., Δc_l) for halogenated functional groups. However, several of the pesticides and phthalates contain other functional groups not directly addressed in Equation 2-2 including ester and sulfur groups. At this time, the effects of these types of groups on predictions by Equation 2-2 are unknown.

2.4 Comparison of Narcosis and Conventional Chronic Toxicity Values

For every narcotic chemical in this document, the narcosis-based SCV is greater than the conventionally-derived SCV, although the magnitude of the difference varies among chemicals (also see Table 3-1). Figure 2-1 shows the ratio of the two values, which ranges from 1.1 (1,2,4-trichlorobenzene) to 220 (1,1,1-trichloroethane). Of the 20 chemicals evaluated, four chemicals had ratios below 10, 13 chemicals had ratios between 10 and 50, and three chemicals had ratios greater than 100. To interpret these differences, one must consider the differences in how the two values are derived. There are two features of the conventional SCV derivation that create discrepancies. The first is the use of secondary acute factors (SAFs) to estimate a SAV from existing data (see Section A.5 of Appendix A for more discussion of SAFs). The SAFs applied to the chemicals in question here range from 4 up to 242, depending on the number of minimum data requirements met by the available toxicity data, and is applied to the lowest reported mean acute value available (see Suter and Tsao (1996) and U.S. EPA (2001) for a description of how the conventional SCVs were calculated).

The SAFs were derived based on an analysis of a wide range of chemicals. However, narcotics tend to show a much narrower range in species sensitivity than do many other chemicals; in fact, the total range in species sensitivity reported by Di Toro et al. (2000) is only a factor of 8.3 across a total of 33 species. More importantly, the conventional GLI SCV methodology requires that data for *Daphnia magna* be included in the data set. As shown by Di Toro et al. (2000), the ratio of the estimated SMAV for *Daphnia magna* and the FAV for all species is only a factor 3.1. In the case of

rainbow trout, a species for which data were frequently available for the present analysis, that ratio is only 1.7. What this means in terms of SCV derivation for narcotic chemicals is that the generic SAFs are larger than is appropriate for narcotic chemicals in particular; while values of 4 to 242 were used, one would expect the true value to have never been higher than 3.1, and commonly 1.7 or less. This difference in extrapolation therefore accounts for as much as a factor of >10 difference between the conventionally-derived and narcosis-based SAVs, which is directly translated into differences in the SCVs (Figure 2-1).

The second major factor lies in the acute-chronic ratios (ACRs) used to translate the SAV into a SCV. In the conventional approach, calculation of the ACR was based on the geometric mean of at least three ACRs. However, wherever there were less than three species-specific ACRs available, a value of 18 was used to replace the missing data (see Section A.5 of Appendix A for more discussion of ACRs); this value was derived through an analysis of ACRs for a variety of chemicals. For the narcotic chemicals shown in Figure 2-1, availability of chronic toxicity data varied from no measured ACRs to three measured ACRs. Where there were no measured ACRs, the conventionally-derived secondary ACR (SACR) was 18.

In their analysis, Di Toro et al. (2000) calculated a much lower mean ACR of 5.09 for narcotic chemicals specifically. Because narcosis appears to result in a lower ACR than the default value of 18 used in the conventional Tier 2 SCV derivation, one can expect additional conservatism in the conventionally-derived Tier 2 SCVs for those chemicals where little or no chronic data were available. Examples include chemicals like 1,2 dichlorobenzene and pentachlorobenzene, both of which were derived using SACRs of 18 and have correspondingly high ratios of the narcosis-based and conventionally-derived SCV values (Figure 2-1). In contrast, 1,2,4 – trichlorobenzene had enough acute toxicity data to meet all 8 minimum data requirements (MDRs) (so no SAF was applied) and the SACR (with two measured ACRs) was only 6.7, very close to the 5.09 estimated for narcotic chemicals (Di Toro et al. 2000). As a result, the conventionally-derived SCV and the narcosis-based SCVs are very close (Figure 2-1).

The applicability of narcosis theory to the compounds designated here as narcotics can be evaluated by comparing the individual species mean acute values (SMAVs) for each of the compounds to the SMAV one would predict based on narcosis theory. To do this, the individual SMAV values were extracted from the SCV derivation for the 20 narcotic chemicals listed in Section 2.3.2. For those species which also appeared in the dataset compiled by Di Toro et al. (2000), the mean species sensitivity was used along with the K_{OW} of each chemical to predict an LC50 for that species and chemical. These predicted LC50s for all 20 chemicals were compared to the observed SMAVs as shown in Figure 2-2. To allow better discrimination of data for individual chemicals, this same data set was segregated into three groups of chemicals, and replotted as Figures 2-3 through 2-5.

The strong agreement between observed and predicted values, shown by alignment along the one to one line, clearly indicates that the observed toxicity of these chemicals is consistent with a narcosis mode of action. Most of the measured values fall within a factor of two of the predicted value (shown by the dashed lines in Figures 2-2 through 2-5) with no consistent bias from a 1:1 relationship. This in turn suggests that deriving SCVs for these chemicals using narcosis theory is appropriate, and that the differences in the conventionally-derived and narcosis-based SCVs is primarily due to conservatism in the SAFs and default SACRs as discussed above.

Finally, for the three phthalates discussed in this document, 'FCVs' derived using the quantitative structure-activity relationship (QSAR) described by Parkerton and Konkel (2000) were compared to conventional SCVs in Table 3-1. ASTER does not classify phthalates

as narcotics but there is some evidence they may demonstrate narcotic-like behavior. The QSAR values derived by Parkerton and Konkel (2000) were 60, 62 and 1173 µg/L for butyl benzyl phthalate, di-n-butyl phthalate and diethyl phthalate, respectively. These values compare relatively well to the conventional SCVs of 19, 35 and 270 µg/L for butyl benzyl phthalate, di-n-butyl phthalate and diethyl phthalate, respectively. From this comparison, the conventional values for phthalates in this document appear to be slightly more conservative than the QSAR based numbers but not tremendously different with ratios ranging from 2 to 4. See Adams et al. (1995), Rhodes et al. (1995), Staples et al. (1997), Parkerton and Konkel (2000), and Call et al. (2001) for further discussion of phthalate aquatic toxicity.

2.5 Selection of New and Alternate Aquatic Toxicity Values

As discussed in the *Foreword*, the ESBs are intended primarily as technical information, not as formal guidelines. As such, the aquatic toxicity values used to derive the Tier 2 ESBs reported in this document are principally recommendations. The conventional (based on WQC and GLI) and narcosis approaches were selected to generate aquatic toxicity values for the 32 chemicals in this document because of their wide usage and acceptance by the scientific, regulatory and regulated communities. As new high quality aquatic toxicity data becomes available, it is encouraged that these Tier 2 ESBs be updated and revised. The GLI approach, as discussed in Appendix A, is one method for performing these updates and revisions. Periodic review of aquatic toxicity databases like ECOTOX may provide new high quality aquatic toxicity values for some of the chemicals discussed in this ESB, especially those for which a limited data base was initially available (see Section 2.3.1).

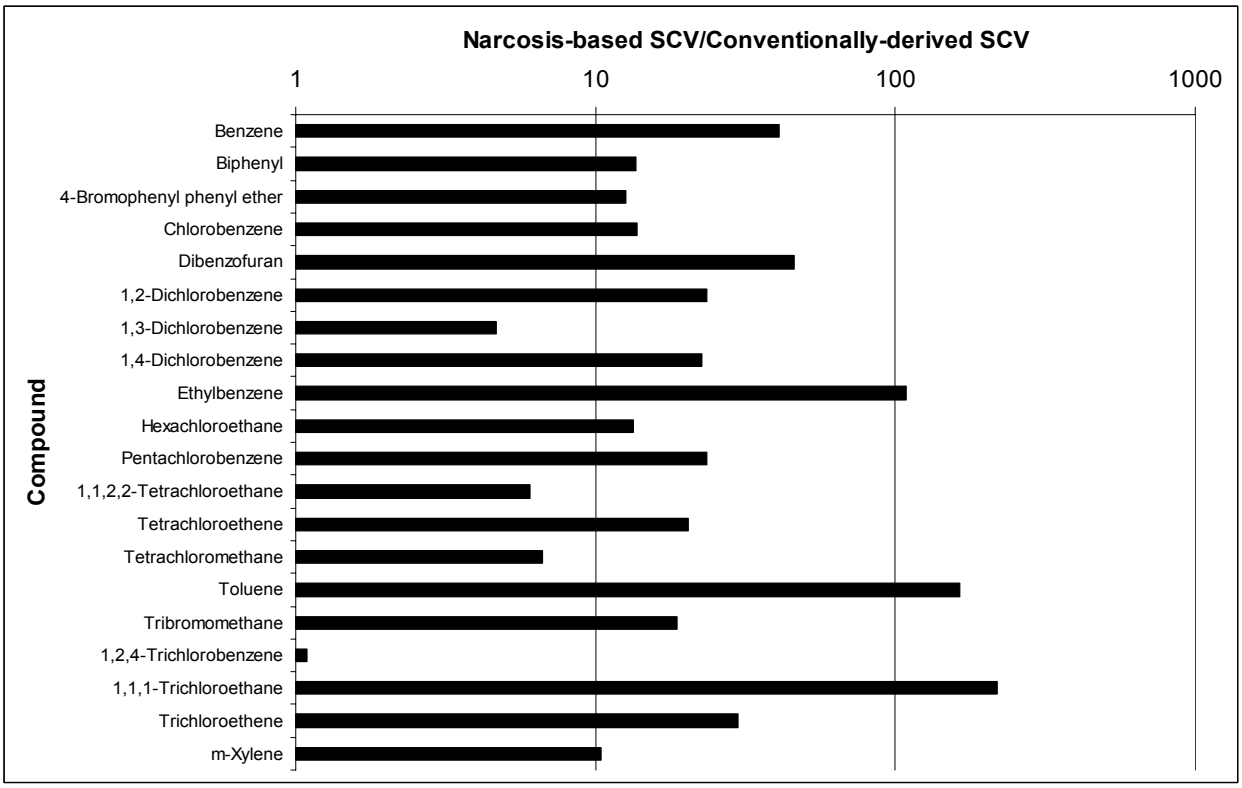

Figure 2-1 Comparison of narcosis-based and conventionally-derived chronic toxicity values. Chemicals with modes of action in addition to narcosis (i.e., pesticides and phthalates) are not shown.

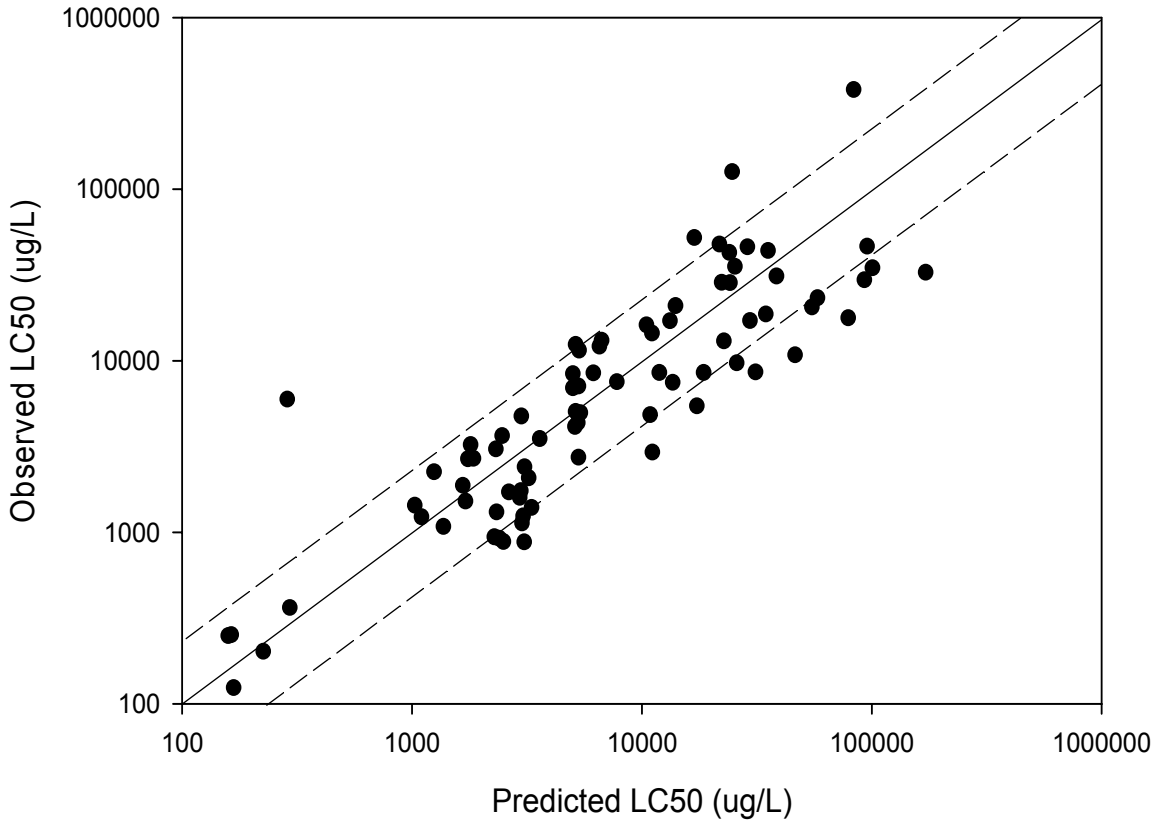

Figure 2-2 Comparison of observed LC_{50} values used in the calculation of secondary chronic values and LC_{50} values predicted using narcosis theory as described by Di Toro et al. (2000) for all 20 narcotic chemicals discussed in this document (including data from Chaisuksant et al. (1998)). Plot shows data for all species that had both measured LC50 values in the SCV derivation and have species-specific sensitivity data as calculated by Di Toro et al. (2000). See discussion in text for more details. The solid line is the one to one line and the dashed lines show ± a factor of two. Chemicals potentially having more specific modes of action (e.g., pesticides and phthalates) are not shown.

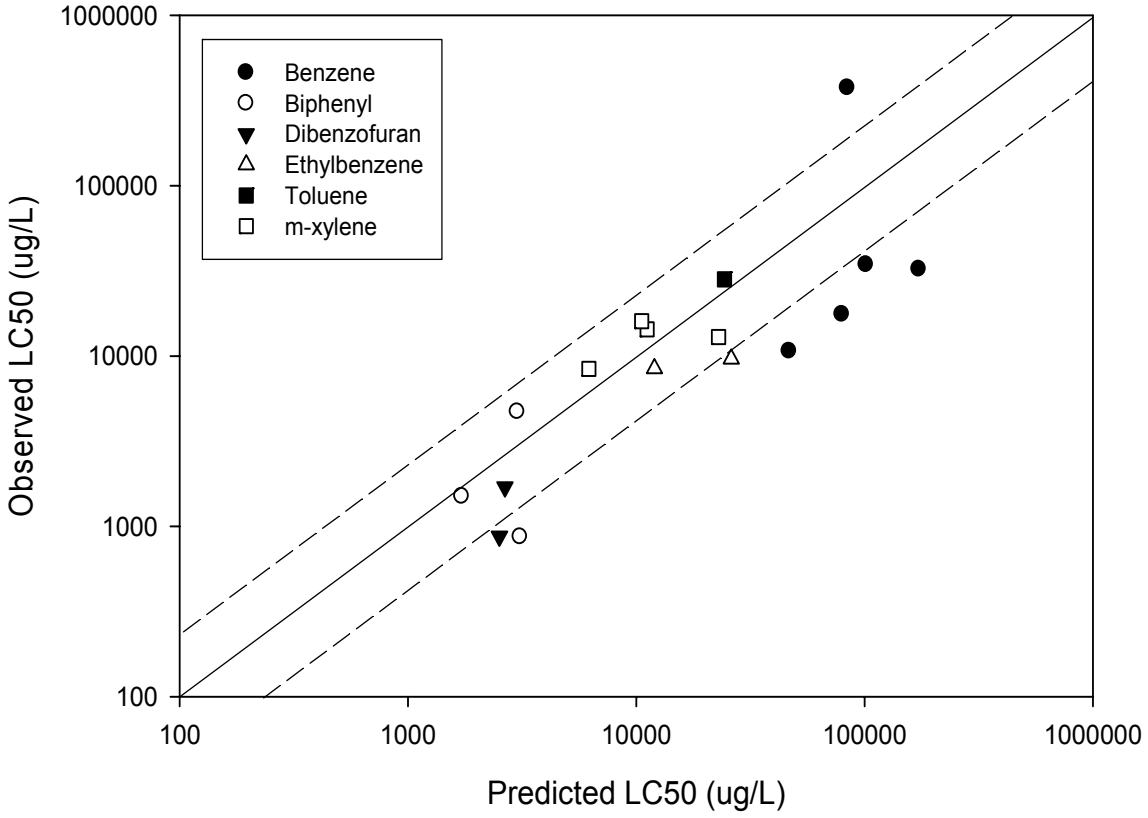

Figure 2-3 Comparison of observed LC_{50} values used in the calculation of secondary chronic values and LC_{50} values predicted using narcosis theory as described by Di Toro et al. (2000) for non-halogenated aromatic narcotic chemicals discussed in this document. Plot shows data for all species that had both measured LC50 data in the SCV derivation and have species-specific sensitivity data as calculated by Di Toro et al. (2000). See discussion in text for more details. The solid line is the one to one line and the dashed lines show ± a factor of two.

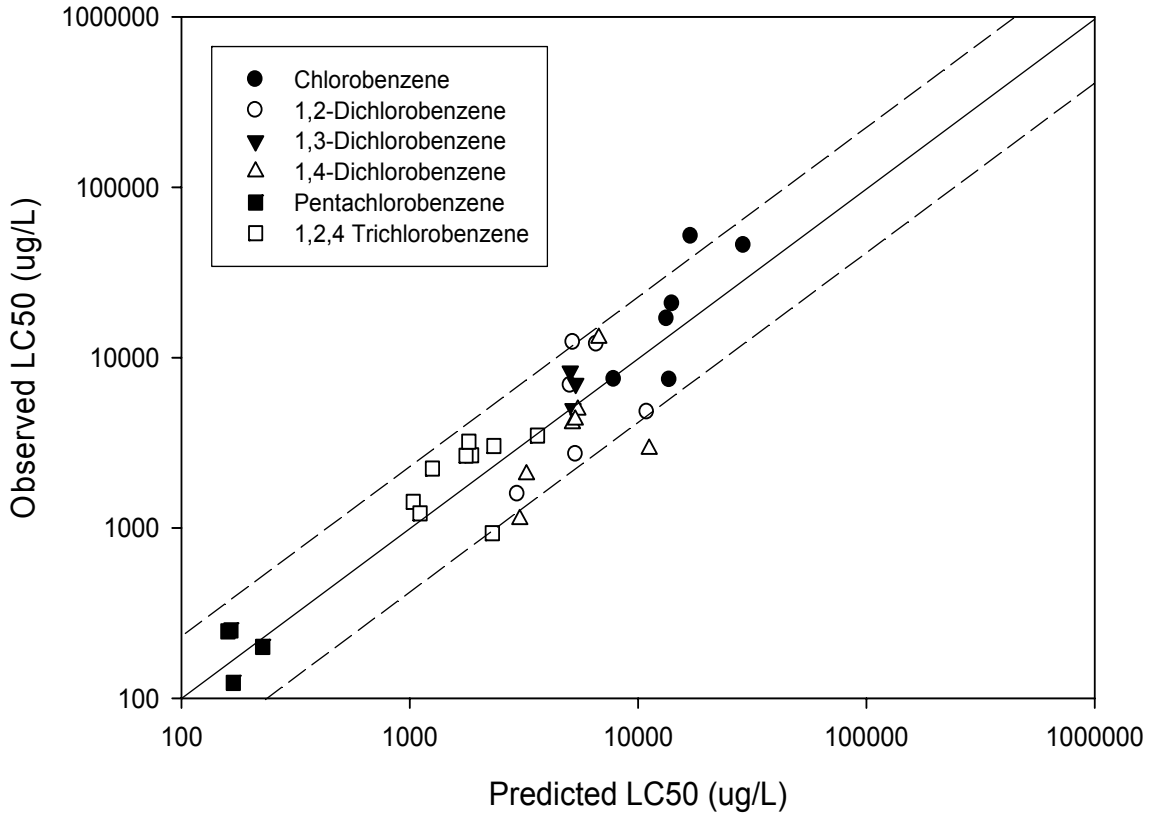

Figure 2-4 Comparison of observed LC_{50} values used in the calculation of secondary chronic values and LC_{50} values predicted using narcosis theory as described by Di Toro et al. (2000) for chlorobenzenes (including Chaisuksant et al. (1998)). Plot shows data for all species that had both measured LC50 data in the SCV derivation and have species-specific sensitivity data as calculated by Di Toro et al. (2000). See discussion in text for more details. The solid line is the one to one line and the dashed lines show ± a factor of two.

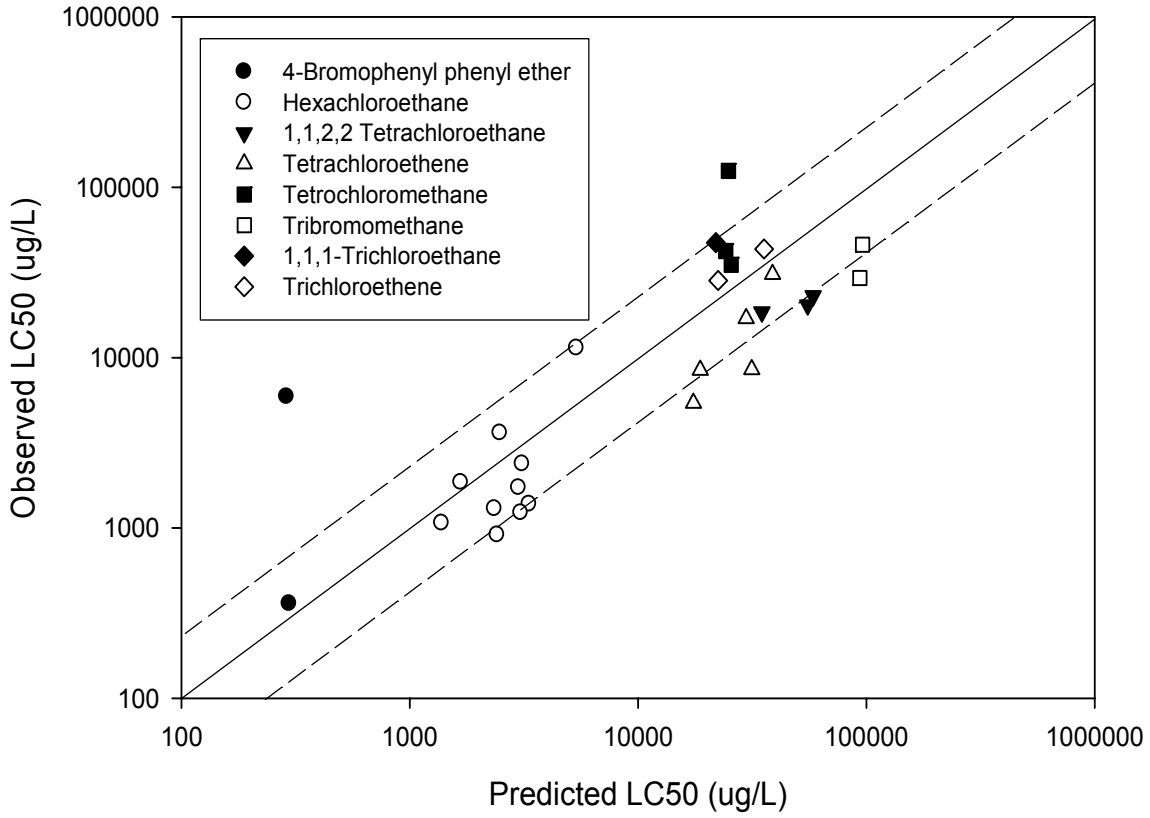

Figure 2-5 Comparison of observed LC_{50} values used in the calculation of secondary chronic values and LC_{50} values predicted using narcosis theory as described by Di Toro et al. (2000) for narcotic chemicals not shown in Figures 2-3 or 2-4, primarily halogenated hydrocarbons. Plot shows data for all species that had both measured LC50 data in the SCV derivation and have species-specific sensitivity data as calculated by Di Toro et al. (2000). See discussion in text for more details. The solid line is the one to one line and the dashed lines show ± a factor of two.

Section 3

Calculation of Equilibrium Partitioning Sediment Benchmarks

3.1 Overview of EqP Methodology

ESBs are the numeric concentrations of individual chemicals that are intended, based on the assumptions discussed in Section 1, to be predictive of biological effects, protective of the presence of benthic organisms, and applicable to the range of natural sediments from lakes, streams, estuaries, and near-coastal marine waters. For nonionic organic chemicals, ESBs are expressed as µg chemical/g_{OC} and apply to sediments having ≥ 0.2% organic carbon by dry weight. A brief overview follows of the concepts that underlie the EqP methodology for deriving ESBs. The methodology is discussed in detail in "Technical Basis for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Nonionic Organics" (U.S. EPA 2003a), hereafter referred to as the ESB Technical Basis Document.

Bioavailability of a chemical at a particular sediment concentration often differs from one sediment type to another. Therefore, a method is necessary to determine ESBs based on the bioavailable chemical fraction in a sediment. For nonionic organic chemicals, the concentration–response relationship for the biological effect of concern can most often be correlated with the interstitial water (i.e., pore water) concentration (µg chemical/L interstitial water) and not with the sediment chemical concentration (µg chemical/g sediment) (Di Toro et al. 1991). This does not mean that all of the exposure is from the interstitial waters but from a purely practical point of view, this correlation suggests that if it were possible to measure the interstitial water chemical concentration, or predict it from the total sediment concentration and the relevant sediment properties, then that concentration could be used to quantify the exposure concentration for an organism. Thus, knowledge of the partitioning of chemicals between the solid and liquid phases in a sediment is a necessary component for establishing ESBs. For this reason, the methodology described below is called the EqP method. As stated above, an ESB can be derived using any given level of protection, in the following discussion the SCVs or FCVs for several nonionic organic chemicals are applied. The EqP approach used here to derive ESBs functions most effectively for nonionic organic chemicals with log K_{OW}s ≥ 2. However, for chemicals with log K_{OW} between 2 and 3, EqP will function but sedimentary conditions (i.e., f_{OC} and f_{Solids}) should be considered and adjustments to the derivation of the ESB maybe advisable (see Section 3.3).

3.2 Derivation of Tier 2 Equilibrium Partitioning Sediment Benchmarks

The ESB Technical Basis Document (U.S. EPA 2003a) demonstrates that benthic species, as a group, have sensitivities similar to all benthic and water column species tested (taken as a group) to derive the WQC concentration for a wide range of chemicals. Thus, an ESB can be established using the FCV, calculated based on the WQC guidelines (Stephan et al. 1985), or a SCV calculated based on other sources like the water quality guidance originally derived for the Great Lakes Water Quality Initiative (GLI 1995), as the acceptable effect concentration in interstitial or overlying water. The appropriate partition coefficient can then be used to relate the interstitial water concentration (i.e., the calculated FCV or SCV) to the sediment concentration via the partitioning equation.

For chemicals discussed in this document, this acceptable concentration in sediment is termed an ESB_{Tier2}.

The methodology for deriving FCVs and SCVs used in the development of these ESBs were taken from existing or draft WQC, the approach developed for the Great Lakes Water Quality Initiative (Tier 1 and 2) and, when necessary, available data were obtained from EPA's AQUIRE database (now ECOTOX accessible at www.epa.gov/ecotox) and other literature (see Section 2).

In addition to deriving FCVs or SCVs based on chemical-specific toxicity data, the likelihood that each chemical would act as a narcotic toxicant (as opposed to a more specific mode of action) was evaluated using the ASTER model (Russom et al. 1997) which predicts mode of toxic action based on chemical structure. For chemicals in this document that were flagged by the ASTER model as acting through a narcotic mode of action, SCVs were also derived using the narcosis model described in U.S. EPA (2003e), Di Toro and McGrath (2000) and Di Toro et al. (2000).

For chemicals evaluated using conventionally-derived SCVs, separate ESB values were calculated for freshwater and marine organisms according to data availability. For chemicals flagged as narcotic toxicants, only single values were calculated, as it is believed that there is little difference in sensitivity between freshwater and marine organisms under this mode of action (U.S. EPA 2003e). A listing of SCVs and FCVs using conventional and narcosis approaches are shown in Table 3-1.

An ESB is calculated as follows. Establishing the SCV or FCV (µg/L) as the acceptable concentration in water for the chemical of interest, the ESB is computed using the partition coefficient, K_P (L/Kg), between sediment and water:

$$ESB_{Tier2} = K_P \cdot SCV \qquad (3\text{-}1)$$

This is the fundamental equation used to generate an ESB_{Tier2}. Its' utility depends on the existence of a methodology for quantifying K_P.

Organic carbon appears to be the dominant sorption phase for most nonionic organic chemicals in naturally occurring sediments and, thus, controls the bioavailability of these compounds in sediments. Evidence for this can be found in numerous toxicity tests, bioaccumulation studies, and chemical analyses of interstitial water and sediments (Di Toro et al. 1991, U.S. EPA 2003a). The organic carbon binding of a chemical in sediment is a function of that chemical's K_{OC} and the weight fraction of organic carbon (f_{OC}) in the sediment. The relationship is as follows:

$$K_P = f_{OC} \cdot K_{OC} \qquad (3\text{-}2)$$

It follows that:

$$ESB_{Tier2OC} = K_{OC} \cdot SCV \qquad (3\text{-}3)$$

where $ESB_{Tier2OC}$ is an ESB_{Tier2} expressed on a sediment organic carbon normalized basis. For nonionic organics, normalization of the "ESB_{Tier2}" to organic carbon is assumed (more formally $ESB_{Tier2OC}$) unless otherwise specified.

Although K_{OC} is not usually measured, it is closely related to the octanol-water partition coefficient (K_{OW}), which has been measured for many compounds, and can be measured very precisely. A chemical's K_{OC} is related to the K_{OW} by the following equation (Di Toro et al. 1991):

$$\text{Log } K_{OC} = 0.00028 + 0.983 \cdot (\log K_{OW}) \qquad (3\text{-}4)$$

Karickhoff and Long (1996) established a protocol for recommending K_{OW} values for nonionic organic chemicals based on the best available measured, calculated, and estimated data. The recommended $\log_{10}K_{OW}$ values from Karickhoff and Long (1995) were used to derive many of the K_{OC} values for ESB calculation in this document (Table 3-2).

Based on this derivation, ESB_{Tier2} values for 32 nonionic organic chemicals using conventional and narcosis approaches are listed in Table 3-2.

Table 3-1. Chronic toxicity values (µg/L), SCVs and FCVs, used to derive Tier 2 ESBs based on conventional and narcosis approaches. Narcosis values for chemicals with a toxicological mode of action in addition to narcosis are italicized and bolded (e.g., pesticides and phthalates) and are provided for comparison not for use. Values presented with two significant figures except FCVs.

CAS Number	Chemical	log K_{ow}	Conventional* FCV or SCV (µg/L)		Narcosis* SCV (µg/L)
			Freshwater	Marine	
71432	Benzene	2.13	SCV = 130	SCV = 130	5300
319868	BHC other than Lindane	3.78	SCV = 2.2	-	***310***
58899	Gamma-BHC, Lindane	3.73	FCV = 0.080	-	***340***
92524	Biphenyl	3.96	SCV = 14	SCV = 14	190
101553	4-Bromophenyl phenyl ether	5.00	SCV = 1.5	SCV = 1.5	19
85687	Butyl benzyl phthalate	4.84	SCV = 19	-	***58***
108907	Chlorobenzene	2.86	SCV = 64	SCV = 64	880
333415	Diazinon	3.70	FCV = 0.1699	FCV = 0.8185	***670***
132649	Dibenzofuran	4.07	SCV = 3.7	SCV = 3.7	170
95501	1,2-Dichlorobenzene	3.43	SCV = 14	SCV = 14	330
541731	1,3-Dichlorobenzene	3.43	SCV = 71	SCV = 71	330
106467	1,4-Dichlorobenzene	3.42	SCV = 15	SCV = 15	340
84742	Di-n-butyl phthalate	4.61	SCV = 35	-	***85***
84662	Diethyl phthalate	2.50	SCV = 270**	-	***6700***
115297	Endosulfan mixed isomers	4.10	FCV = 0.056	FCV = 0.0087	***210***
959988	Alpha-Endosulfan	3.83	FCV = 0.056	FCV = 0.0087	***390***
33213659	Beta-Endosulfan	4.52	FCV = 0.056	FCV = 0.0087	***86***
100414	Ethylbenzene	3.14	SCV = 7.3	SCV = 7.3	790
67721	Hexachloroethane	4.00	SCV = 12	SCV = 12	160
121755	Malathion	2.89	SCV = 0.097	FCV = 0.1603	***4300***

CAS Number	Chemical	log K_{ow}	Conventional* FCV or SCV (µg/L)		Narcosis* SCV (µg/L)
			Freshwater	Marine	
72435	Methoxychlor	5.08	SCV = 0.019	-	***22***
608935	Pentachlorobenzene	5.26	SCV = 0.47	SCV = 0.47	11
79345	1,1,2,2-Tetrachloroethane	2.39	SCV = 610	SCV = 610	3700
127184	Tetrachloroethene	2.67	SCV = 98	SCV = 98	2000
56235	Tetrachloromethane	2.73	SCV = 240	SCV = 240	1600
108883	Toluene	2.75	SCV = 9.8	SCV = 9.8	1600
8001352	Toxaphene	5.50	FCV = 0.039	FCV = 0.2098	***10***
75252	Tribromomethane (Bromoform)	2.35	SCV = 320	SCV = 320	6000
120821	1, 2, 4-Trichlorobenzene	4.01	SCV = 110	SCV = 110	120
71556	1, 1, 1-Trichloroethane	2.48	SCV = 11	SCV = 11	2400
79016	Trichloroethene	2.71	SCV = 47	SCV = 47	1400
108383	m-Xylene	3.20	SCV = 67***	SCV = 67***	700

- = Not Available.
* = See Section 2.3 for definition.
** = Data summary in Suter and Tsao (1996) did not include a 96-hour LC50 of 131,000 ug/L from Adams et al. (1995). Inclusion of this LC50 in the SCV calculation increased the SCV from 210 to 270 µg/L (Mount 2008).
*** = Value changed from original GLI SCV (Suter and Tsao 1996, U.S. EPA 1996), see Mount (2006).

Table 3-2. Tier 2 ESBs (µg/g_{oc}) based on toxicity values derived using conventional and narcosis approaches (from Table 3-1). K_{OC} based on Equation 3-4. Values presented with two significant figures.

CAS Number	Chemical	Log K_{OC}	Conventional* ESB (µg/g_{OC})		Narcosis* ESB (µg/g_{OC})
			Freshwater	Marine	
71432	Benzene	2.09	16	16	660
319868	BHC other than Lindane	3.72	11	-	^

CAS Number	Chemical	Log K_{OC}	Conventional* ESB ($\mu g/g_{OC}$)		Narcosis* ESB ($\mu g/g_{OC}$)
			Freshwater	Marine	
58899	Gamma-BHC, Lindane	3.67	0.37	-	^
92524	Biphenyl	3.89	110	110	1500
101553	4-Bromophenyl phenyl ether	4.92	120	120	1600
85687	Butyl benzyl phthalate	4.76	1100	-	^
108907	Chlorobenzene	2.81	41	41	570
333415	Diazinon	3.64	0.74	3.6	^
132649	Dibenzofuran	4.00	37	37	1700
95501	1,2-Dichlorobenzene	3.37	33	33	780
541731	1,3-Dichlorobenzene	3.37	170	170	780
106467	1,4-Dichlorobenzene	3.36	34	34	780
84742	Di-n-butyl phthalate	4.53	1200	-	^
84662	Diethyl phthalate	2.46	77	-	^
115297	Endosulfan mixed isomers	4.03	0.60	0.093	^
959988	Alpha-Endosulfan	3.77	0.33	0.051	^
33213659	Beta-Endosulfan	4.44	1.6	0.24	^
100414	Ethylbenzene	3.09	8.9	8.9	970
67721	Hexachloroethane	3.93	100	100	1400
121755	Malathion	2.84	0.067	0.11	^
72435	Methoxychlor	4.99	1.9	-	^
608935	Pentachlorobenzene	5.17	70	70	1600
79345	1,1,2,2-Tetrachloroethane	2.35	140	140	830
127184	Tetrachloroethene	2.62	41	41	840
56235	Tetrachloromethane	2.68	120	120	770
108883	Toluene	2.70	5.0	5.0	810

CAS Number	Chemical	Log K_{OC}	Conventional* ESB ($\mu g/g_{OC}$)		Narcosis* ESB ($\mu g/g_{OC}$)
			Freshwater	Marine	
8001352	Toxaphene	5.41	10	54	^
75252	Tribromomethane (Bromoform)	2.31	65	65	1200
120821	1, 2, 4-Trichlorobenzene	3.94	960	960	1100
71556	1, 1, 1-Trichloroethane	2.44	3.0	3.0	660
79016	Trichloroethene	2.66	22	22	650
108383	m-Xylene	3.15	94	94	980

* = See Section 2.3 for definition.
- = Not Available.
^ = Not Calculated.

3.3 Effects of Low K_{OW} on Derivation of ESB_{Tier2}

As noted above, the EqP approach used here to derive ESBs functions most effectively for nonionic organic chemicals with log K_{OW}s ≥ 2. However, Fuchsman (2003) demonstrated recently that equilibrium partitioning may inaccurately predict the bioavailable concentration of organic compounds with low log K_{OW}s (i.e., approximately 3). This is because the basic equilibrium partitioning equation (Equation 3-3) assumes that the measured contaminant is associated overwhelmingly with sediment organic carbon and that the amount in the dissolved phase is negligible. However, for chemicals with comparatively low K_{OW} a more substantial fraction of total chemical may be present in the dissolved phase. As a result, the ESB calculation as shown in Equation 3-3, may result in overly protective ESBs.

A modification of the equilibrium partitioning equation (Equation 3-3) can be determined (Fuchsman 2003):

$$ESB_{Tier2DRY\ WT} = SCV\ [(f_{OC}\ K_{OC}) + ((1 - f_{Solids}) \div f_{Solids})] \quad (3\text{-}5)$$

In which, $ESB_{Tier2DRY\ WT}$ is in units of µg chemical/g dry weight sediment and f_{Solids} is the fraction of sediment present as solids. In the U.S. EPA Environmental Monitoring and Assessment Program (EMAP) data set discussed below, f_{Solids} values for 1024 sediment samples ranged from 0.085 to 0.938 with an average value of 0.553 (U.S. EPA 2007a). In Equation 3-5, the proportion $((1 - f_{Solids}) \div f_{Solids})$, is used to adjust the magnitude of the $ESB_{Tier2DRY\ WT}$ as a function of the amount of solids in the sediment. As K_{OC} increases; that is, the chemical becomes more hydrophobic, the proportion becomes less important and has little effect on the $ESB_{Tier2DRY\ WT}$. Conversely, for low K_{OC} chemicals, the proportion may have a substantial effect on the magnitude of $ESB_{Tier2DRY\ WT}$. The $ESB_{Tier2DRY\ WT}$ is converted to $ESB_{Tier2OC}$ by the following:

$$ESB_{Tier2OC} = ESB_{Tier2DRY\ WT} \div f_{OC} \quad (3\text{-}6)$$

It should be noted that in aquatic environments, f_{Solids} and f_{OC} are often inversely correlated. For example, in depositional areas, where contaminants discussed in this document frequently accumulate, f_{Solids} is often low and f_{OC} elevated because of the abundance of carbon-rich small particles with large surface area to volume ratios. Conversely, sediments in dynamic areas tend to have low f_{OC} and elevated f_{Solids} because of the dominance of large mineral particles with low surface area to volume ratios and comparatively low carbon content.

An analysis of the effects of low K_{OW} on the ESB calculation is shown in Figure 3-1. The departure of the standard ESB (Equation 3-3) from the modified ESB (Equations 3-5 and 3-6) occurs most substantially at low f_{OC} and low f_{Solids} conditions, starting at a log K_{OW} of approximately 4. Conversely, at high f_{Solids} and high f_{OC} conditions, there is little difference between the calculated values (Figure 3-1a). When high f_{OC} is combined with low f_{Solids} as well as low f_{OC} combined high f_{Solids}, departure between the two approaches for calculating ESBs are observed but at log K_{OW}s of about 2.50 (Figure 3-1b).

Table 3-3 provides examples of the specific effects of f_{Solid} on the derivation of ESBs for four chemicals with a range of K_{OW}s. For this exercise, f_{Solids} was calculated using paired sand and moisture content data from sediment samples collected in several U.S. EPA EMAP estuarine provinces (i.e., Acadian, Carolinian, Virginian) (U.S. EPA 2007a). From the moisture content (MC) data (as %), f_{Solids} was calculated as:

$$f_{Solids} = (100 - MC) \div 100 \quad (3\text{-}7)$$

and regressed against the sand content (%) to derive the relationship:

$$f_{Solids} = 0.264 + 0.00487 \cdot \text{Sand Content} \quad (3\text{-}8)$$

For this example, f_{OC} values were set to the environmentally relevant range of 0.002 to 0.05. Examining the extremes, in a sandy sediment

(80% sand), the ESB for low K_{OW} benzene is shown to increase by a factor of three between the standard equation (Equation 3-3) and the modified equation (Equation 3-5) calculations. Conversely, for high K_{OW} toxaphene, there is no difference between the ways of calculating ESBs in the same sandy sediment. For a low sand content sediment (20% sand), benzene ESBs are different by only 20% and again no difference was observed between toxaphene ESBs. The other two chemicals, malathion and 1,2,4-trichlorobenzene with K_{OW}s between benzene and toxaphene, follow similar trends.

Of the 32 chemicals discussed in this document, only four have log K_{OW}s less than 2.5 while 22 have log K_{OW}s that are equal to or less than log 4. In situations where low f_{OC} and low f_{Solids} are known to occur, it is recommended that Equation 3-5 be used to modify the predicted ESB. However, it is most likely chemicals in this document will occur in environments at concentrations of concern when f_{Solids} are low and f_{OC} is high, conditions where departure between the standard and modified ESBs takes place at log K_{OW} of about 2.5, not affecting these chemicals too substantially. It maybe possible under conditions where a contaminated groundwater discharge is occurring into a sedimentary environment for f_{Solids} to be elevated, f_{OC} to be low, and for low K_{OW} chemicals to be present. Under such conditions, the use of Equation 3-5 maybe warranted.

Finally, the value f_{Solids} is not often reported in sediment investigations. In sediments suspected of contamination by low K_{OW} chemicals, it may be important to record this sediment characteris-tic (see Equation 3-8 for predicting f_{Solids} based on sediment sand content). The f_{Solids} values should be available from laboratories conducting chemical analyses on any contaminated sediment samples as part of the determination of moisture content (i.e., Percent Solids = 100% - moisture content (expressed as %)).

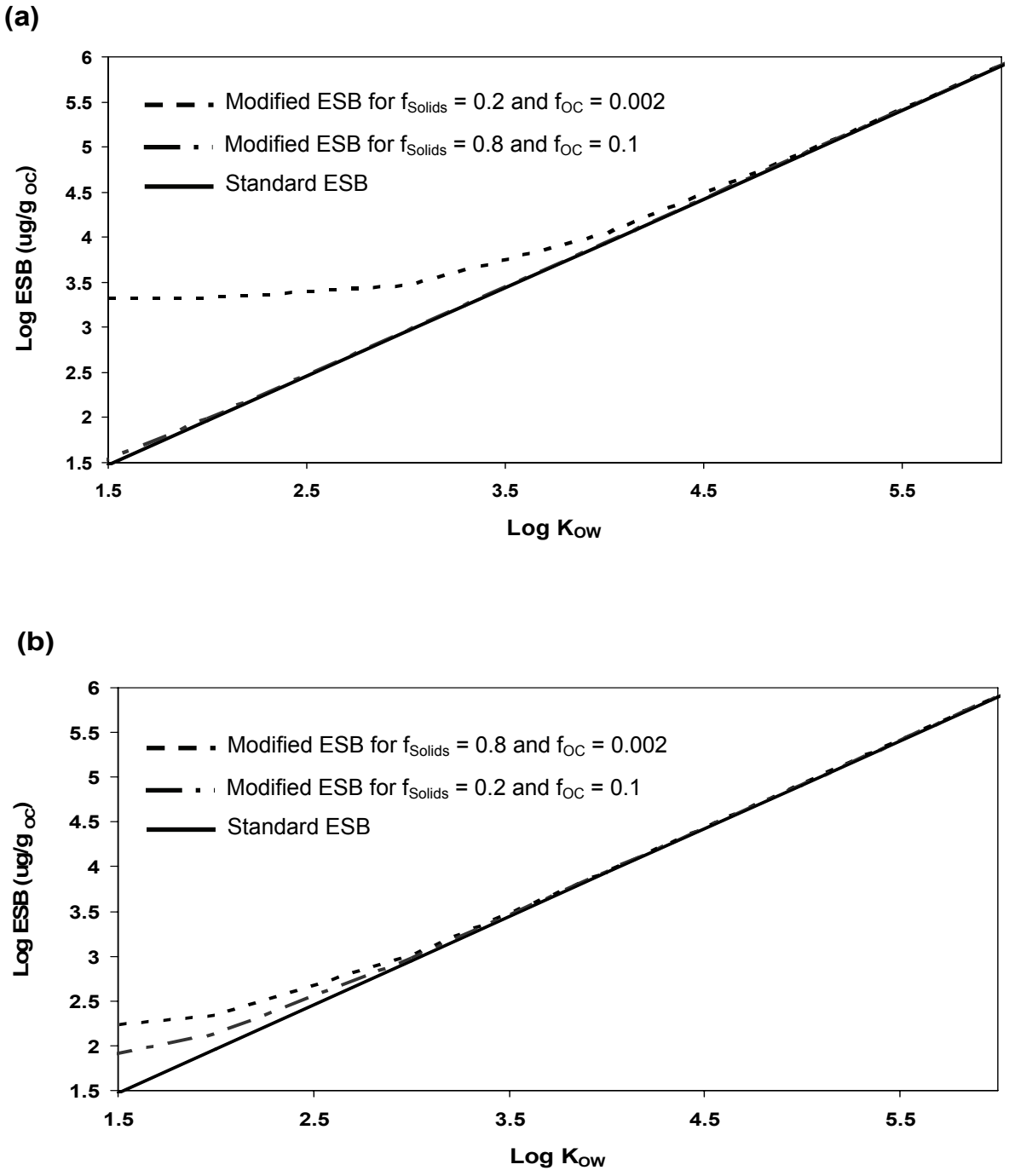

Figure 3-1 **Comparison of ESBs calculated using the standard equation (Equation 3-3) and modified equations which include the effects of low K_{OW} (Equations 3-5 and 3-6): (a) effects of low f_{Solids} and f_{OC} and high f_{Solids} and f_{OC} and (b) effects of high f_{Solids} and low f_{OC} and low f_{Solids} and high f_{OC}. In all cases, the FCV is 1000 ug/L.**

Table 3-3 Example calculations of conventional freshwater standard and modified $ESB_{Tier2DRY\ WT}$ values (µg/g dry weight) for four chemicals under different f_{OC} and f_{Solids} conditions. See text for discussion of the calculation of f_{Solids}. ESB values presented with two significant figures.

Chemical	FCV or SCV (µg/L)	Log K_{OW}:K_{OC}	Standard $ESB_{Tier2DRY\ WT}$: Modified $ESB_{Tier2DRY\ WT}$* (µg/g dry weight)		
			Sediment Characteristics		
			Sand = 80% Silt-Clay = 20% f_{OC} = 0.002 f_{Solids} = 0.65	Sand = 50% Silt-Clay = 50% f_{OC} = 0.025 f_{Solids} = 0.51	Sand = 20% Silt-Clay = 80% f_{OC} = 0.05 f_{Solids} = 0.36
Benzene	130	2.13:2.09	0.032:0.10	0.40:0.52	0.80:1.0
Malathion	0.097	2.89:2.84	0.00013:0.00019	0.0017:0.0018	0.0034:0.0035
1,2,4-Trichlorobenzene	110	4.01:3.94	1.9:2.0	24:24	48:48
Toxaphene	0.039	5.50:5.41	0.02:0.02	0.25:0.25	0.50:0.50

* = See Equation 3-5.

3.4 Conversion to Dry Weight Concentration

Since organic carbon is the major factor controlling the bioavailability of nonionic organic compounds in sediments, ESBs have been developed on an organic carbon normalized basis (e.g., $ESB_{Tier2OC}$) not on a dry weight basis. When the chemical concentrations in sediments are reported as dry weight concentration and organic carbon data are available, it is best to convert the sediment concentration to µg chemical/g organic carbon. These concentrations can then be directly compared to the ESB value. This facilitates comparisons between the ESB and field concentrations relative to identification of hot spots and the degree to which sediment concentrations do or do not exceed ESB values. Conversion from the dry weight to organic carbon-normalized concentration can be performed using the following equations:

$$\mu g\ Chemical/g_{OC} = \mu g\ Chemical/g_{DRY\ WT} \div (\%\ TOC \div 100) \quad (3\text{-}9)$$

or

$$\mu g\ Chemical/g_{OC} = \mu g\ Chemical/g_{DRY\ WT} \cdot 100 \div \%\ TOC \quad (3\text{-}10)$$

For example, sediment with a chemical concentration of 0.1 $\mu g/g_{DRY\ WT}$ and 0.5% TOC has an organic carbon-normalized concentration of 20 $\mu g/g_{OC}$ (0.1 $\mu g/g_{DRY\ WT} \cdot 100 \div 0.5 = 20\ \mu g/g_{OC}$). Another sediment with the same dry weight concentration (0.1 $\mu g/g_{DRY\ WT}$) but a TOC concentration of 5.0% would have an organic carbon-normalized concentration of 2.0 $\mu g/g_{OC}$ (0.1 $\mu g/g_{DRY\ WT} \cdot 100 \div 5.0 = 2.0\ \mu g/g_{OC}$).

In situations where TOC values for particular sediments are not available, a range of TOC values may be used in a 'worst case' or 'best case' analysis. In this situation, the organic carbon-normalized ESB values ($ESB_{Tier2OC}$) may be 'converted' to dry weight-normalized ESB values ($ESB_{Tier2DRY\ WT}$). This 'conversion' must be performed for each level of TOC of interest:

$$ESB_{Tier2DRY\ WT} = ESB_{Tier2OC}\ (\mu g/g_{OC}) \cdot (\%\ TOC \div 100) \quad (3\text{-}11)$$

where $ESB_{Tier2DRY\ WT}$ is the dry weight normalized ESB value. Examples of the Tier 2 ESB values ($ESB_{Tier2DRY\ WT}$) using conventional and narcosis approaches normalized to various organic carbon concentrations can be seen in Table 3-4.

Table 3-4. Example Tier 2 ESBs (µg/g dry weight) using freshwater conventional (C) and narcosis (N) approaches normalized to various total organic carbon (TOC) concentrations. Narcosis values for chemicals with a toxicological mode of action in addition to narcosis (e.g., pesticides and phthalates) are not presented. Values presented with two significant figures.

Chemical Name	Approach	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 0.2% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 1.0% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 5.0% TOC
Benzene	C	0.032	0.16	0.80
	N	1.3	6.6	33
BHC other than Lindane	C	0.022	0.11	0.55
Gamma-BHC, Lindane	C	0.00074	0.0037	0.019
Biphenyl	C	0.22	1.1	5.5
	N	3.0	15	75
4-Bromophenyl phenyl ether	C	0.24	1.2	6.0
	N	3.2	16	80
Butyl benzyl phthalate	C	2.2	11	55
Chlorobenzene	C	0.082	0.41	2.1
	N	1.1	5.7	29
Diazinon	C	0.0015	0.0074	0.037
Dibenzofuran	C	0.074	0.37	1.9
	N	3.4	17	85
1,2-Dichlorobenzene	C	0.066	0.33	1.7
	N	1.6	7.8	39
1,3-Dichlorobenzene	C	0.34	1.7	8.5
	N	1.6	7.8	39
1,4-Dichlorobenzene	C	0.068	0.34	1.7
	N	1.6	7.8	39
Di-n-butyl phthalate	C	2.4	12	60

Chemical Name	Approach	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 0.2% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 1.0% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 5.0% TOC
Diethyl phthalate	C	0.15	0.77	3.85
Endosulfan mixed isomers	C	0.0012	0.006	0.030
Alpha-Endosulfan	C	0.00066	0.0033	0.017
Beta-Endosulfan	C	0.0032	0.016	0.08
Ethylbenzene	C	0.018	0.089	0.45
	N	1.9	9.7	49
Hexachloroethane	C	0.20	1.0	5.0
	N	2.8	14	70
Malathion	C	0.00013	0.00067	0.0034
Methoxychlor	C	0.0038	0.019	0.095
Pentachlorobenzene	C	0.14	0.70	3.5
	N	3.2	16	80
1,1,2,2-Tetrachloroethane	C	0.28	1.4	7.0
	N	1.7	8.3	42
Tetrachloroethene	C	0.082	0.41	2.1
	N	1.7	8.4	42
Tetrachloromethane	C	0.24	1.2	6.0
	N	1.5	7.7	39
Toluene	C	0.01	0.05	0.25
	N	1.6	8.1	41
Toxaphene	C	0.02	0.10	0.50
Tribromomethane (Bromoform)	C	0.13	0.65	3.3
	N	2.4	12	60
1, 2, 4-Trichlorobenzene	C	1.9	9.6	48

Chemical Name	Approach	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 0.2% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 1.0% TOC	Dry Weight Sediment Concentration ($\mu g/g_{DRYWT}$) at 5.0% TOC
	N	2.2	11	55
1, 1, 1-Trichloroethane	C	0.006	0.03	0.15
	N	1.3	6.6	33
Trichloroethene	C	0.044	0.22	1.1
	N	1.3	6.5	33
m-Xylene	C	0.19	0.94	4.7
	N	2.0	9.8	49

Section 4

Sediment Benchmark Values: Application and Interpretation

4.1 Benchmarks

Based on the level of protection provided by FCVs or SCVs, the procedures described in this document indicate that benthic organisms should be comparably protected from the adverse effects of the 32 nonionic organic chemicals listed in Table 3-2, when their concentrations in sediment are below the ESB_{Tier2} values. These values are appropriate for the protection of both freshwater and marine sediments based on the assumptions discussed in Section 1, except possibly where a locally important species is very sensitive or sediment organic carbon is <0.2% or the nonionic organic chemical's log K_{OW} is <2 (see Section 3.3 to modify ESB_{Tier2} values).

The benchmarks presented in this document are the concentrations of a substance that may be present in sediment while still protecting benthic organisms from the effects of that substance. These benchmarks are applicable to a variety of freshwater and marine sediments because they are based on the biologically available concentration of the substance in those sediments.

The ESBs do not intrinsically consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with the individual nonionic organic chemicals discussed in this document or the potential for bioaccumulation and trophic transfer of these chemicals to aquatic life, wildlife or humans. However, for narcotic chemicals, the toxicity of mixtures can be considered (see discussion below). Consistent with the recommendations of EPA's Science Advisory Board, publication of this document does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, when used in a weight of evidence approach (Wenning et al. 2005), exceedances of ESBs could trigger collection of additional assessment data (e.g., benthic community composition, whole sediment toxicity testing, and other sediment quality guideline evaluations (e.g., Long et al. 1995, MacDonald et al. 1996, Long and MacDonald 1998, Swartz 1999, MacDonald et al. 2000a,b, Leung et al. 2005).

4.2 Considerations in the Application and Interpretation of ESBs

4.2.1 Relationship of ESB_{Tier2} to Expected Effects

The ESB_{Tier2} should be interpreted as a chemical concentration below which adverse effects are not expected. In contrast, at concentrations above the ESB_{Tier2}, assuming equilibrium between phases, effects may occur if the chemical is bioavailable as predicted by EqP theory. In general terms, the degree of effect expected increases with increasing exceedance of the ESB_{Tier2}. Because the FCV or SCV is derived as an estimate of the concentration causing chronic toxicity to sensitive organisms, effects of this type may be expected when sediment concentrations are near the ESB_{Tier2}. As sediment concentrations increase beyond the ESB_{Tier2}, one can expect chronic effects on less sensitive species and/or acute effects on sensitive species. See Section 4.2.6 for further discussion.

4.2.2 Use of EqP to Develop Alternative Benchmarks

The FCV or SCV is used to define a threshold for unacceptable effects based on its precedence in establishing unacceptable effects in the development of WQC. However, the use of EqP to assess sediment contamination is not limited to the ESB_{Tier2} and the associated level of protection discussed in this document. As discussed in earlier sections of this document, by substituting water-only effect values other than the FCV or SCV into the ESB equations, other benchmarks may be developed that are useful in evaluating specific types of biological effects, or that better represent the ecological protection goals for specific assessments.

4.2.3 Influence of Unusual Forms of Sediment Organic Carbon

Partition coefficients used for calculating these ESBs are based on estimated and measured partitioning from natural organic carbon in typical field sediments. Some sediments influenced heavily by anthropogenic activity may contain sources of organic carbon whose partitioning properties are not similar to natural organic carbon. The presence of rubber, animal or wood processing wastes, relatively undegraded woody debris or plant matter (e.g., roots, leaves) as well as black carbon (soot) and coal may alter contaminant partitioning and concentrations of chemicals in interstitial waters in unexpected ways (Iglesias-Jimenez et al. 1997, Grathwohl 1990, Xing et al. 1994). Sediments with substantial amounts of these materials may exhibit higher concentrations of chemicals in interstitial water than would be predicted using generic K_{OC} values, thereby making the ESBs under protective. If such a situation is encountered, the applicability of literature K_{OC} values can be evaluated by analyzing for the chemical of interest in both sediment and interstitial water. If the measured concentration in interstitial water is markedly greater (e.g., more than twofold) than that predicted using the K_{OC} values recommended herein (after accounting for dissolved organic carbon (DOC) partitioning in the interstitial water (U.S. EPA 2003b)), then the ESBs would be under protective and calculation of a site-specific ESB should be considered (see U.S. EPA 2001c, 2003b). Conversely, the presence of black carbon or coal in a sediment may result in reduced chemical activity in sediment and correspondingly reduced concentrations of chemical in interstitial water. Under these conditions, the ESB is likely to be over protective and a site-specific ESB may be warranted (U.S. EPA 2001c, 2003b). However, it should also be noted that the ability to predict partitioning based on additional partitioning factors like black carbon is still evolving and may serve to decrease partitioning-related uncertainties in future applications.

The presence of organic carbon in large particles may also influence the apparent partitioning. Large particles may artificially inflate the effect of the organic carbon because of their large mass, but comparatively small surface area; they may also increase variability in TOC measurements by causing sample heterogeneity. The effect of these particles on partitioning can be evaluated by analysis of interstitial water as described above (U.S. EPA 2001c), and site-specific ESBs may be used if required (U.S. EPA 2003b). It may be possible to screen large particles from sediment prior to analysis to reduce their influence on the interpretation of sediment chemistry relative to ESBs.

4.2.4 Relationship to Risks Mediated through Bioaccumulation and Trophic Transfer

As indicated above, ESBs are designed to address direct toxicity to benthic organisms exposed directly to contaminated sediment. They are not designed to address risks that may occur through bioaccumulation and subsequent exposure of pelagic aquatic organisms (e.g., predatory fish), terrestrial or avian wildlife, or humans. No inference can be drawn between attainment of the ESB_{Tier2} and the potential for risk via bioaccumulation and trophic transfer; the potential for those risks must be addressed by separate means.

4.2.5 Exposures to Chemical Mixtures

It is very important that users of this guidance are aware that the ESB_{Tier2} values provided here reflect the expected toxicity of that specific chemical individually; they do not consider the potential interactive toxicity of that chemical with other chemicals in the mixture, whether antagonistic, additive or synergistic. Thus, a sediment may have concentrations of several chemicals at concentrations below the individual ESB_{Tier2} values, but still cause toxicity because of the aggregate effects of the chemicals acting as a mixture. This potential is not explicitly incorporated into the derivation of the ESB_{Tier2} values because the types and concentrations of co-occurring chemicals is infinitely variable, and the expected interaction of those chemicals is therefore not predictable in a general case.

While the potential for mixture effects must be considered for all chemical mixtures, it is of special concern for the chemicals with a primarily narcotic mode of action discussed in this document. Published literature provides a convincing argument that narcotic chemicals do show additive toxicity with other narcotic chemicals (U.S. EPA 2003e). This is especially relevant for interpreting ESB_{Tier2} values because many, if not most, narcotic chemicals tend to co-occur with other narcotic chemicals because they have common sources. For example, benzene, xylene, toluene, and ethylbenzene commonly co-occur in refined petroleum products. Sources of chlorobenzenes often include multiple chlorobenzene compounds with differing levels of chlorination. Also common in sediments is contamination with narcotic chemicals outside those with ESB_{Tier2} values derived here, such as PAHs (see U.S. EPA 2003e).

For these reasons, it is *expected* that narcosis-based ESB_{Tier2} values will be *under protective* if applied as individual values in most sediments, because other narcotic chemicals are likely to co-occur. This issue can be addressed by using ESB_{Tier2} values in the context of a mixture assessment similar to that used for the ESB for PAH mixtures (U.S. EPA 2003e). In this approach, as shown in the examples in Section 4.3, the contribution of each individual narcotic chemical to the toxicity of the overall mixture is assessed by taking the ratio of the measured concentration of that individual chemical in the mixture by the corresponding single chemical ESB_{Tier2} value. This proportion is calculated individually for all narcotic chemicals in the mixture, then the proportions are summed. If the sum of these values is greater than one, then the expected toxicity of the mixture is greater than that associated with an ESB. If the sum of proportions is less than one, then the sediment would not be expected to be toxic to benthos as a result of that mixture of narcotic chemicals. If PAHs are present in the mixture, then the proportions calculated for PAHs according to the PAH mixture ESB (U.S. EPA 2003e) should be added to the proportions calculated for the narcotic ESB_{Tier2} chemicals. In addition, if there are other narcotic chemicals present in the sediment beyond PAHs and the narcotic chemicals with ESB_{Tier2} values given in this document, they can be incorporated into the analysis using parallel procedures as described by Di Toro and McGrath (2000) and Di Toro et al. (2000). Also, U.S. EPA (2003e), and the references within, provides information about narcotic chemicals. Finally, as discussed in Section 4.3, the narcotic contribution of chemicals with modes of action in addition to narcosis (i.e., the pesticides and phthalates) can be included.

While narcosis is generally discussed for chemicals without a more specific mode of action, theory would suggest that all nonionic organic chemicals would contribute to the overall narcotic potency of a mixture. While this is technically true, the impact of these other chemicals (e.g., pesticides) on the overall narcotic potency of a mixture would be dependent on the toxicity of the chemical acting through a specific mode of action compared to its narcotic potency. If a chemical has a very high conventional potency (low FCV/SCV) compared to its narcosis SCV, then it would exceed the conventional chemical-specific ESB before it was present in sufficient concentration to contribute significantly to the narcotic

potency of a mixture. One can make this comparison by examining the ratios between conventionally-derived FCV/SCV values and the narcosis SCV as given in Table 3-1. Most of these comparisons show that the conventional FCV/SCV is generally 100-fold or more lower than the narcosis SCV; accordingly, these chemicals could not contribute more than 1% to an exceedance of a narcosis mixture ESB without simultaneously violating the conventionally-derived ESB_{Tier2}. For this reason, the contribution of most non-narcotic chemicals discussed in this document can be ignored in the calculation of the narcotic potency of mixtures without substantial error. The exceptions are some of the phthalates, for which the conventionally-derived ESB_{Tier2} values are much higher relative to the narcosis SCV. Where these chemicals occur near their conventionally-derived ESB_{Tier2} concentrations, it may be worth considering the potential for them to contribute to the narcotic potency of that mixture.

4.2.6 Interpreting ESB_{Tier2}s in Combination with Toxicity Tests

Sediment toxicity tests provide an important complement to ESBs in interpreting overall risk from contaminated sediments. Toxicity tests have different strengths and weaknesses compared to chemical-specific guidelines, and the most powerful inferences can be drawn when both are used together.

Unlike chemical-specific guidelines, toxicity tests are capable of detecting any toxic chemical, if it is bioavailable in toxic amounts; one does not need to know what chemicals of concern are present to monitor the toxicity of sediment. Toxicity tests are also useful for detecting the combined effects of chemical mixtures, if those effects are not considered in the formulation of the applicable chemical-specific guideline or benchmark.

On the other hand, toxicity tests also have weaknesses; they provide information only for the species tested, and only for the endpoints measured. This is particularly critical given that a majority of the sediment toxicity tests conducted at the time of this writing primarily measure short-term lethality (in some cases growth), although the use of chronic sediment toxicity tests is becoming more common. Chronic sediment toxicity test procedures have been developed and published for some species (e.g., U.S. EPA 2001b), but these procedures are more resource-intensive as compared to acute tests. In contrast, the ESB_{Tier2} is intended to protect most species against both acute and chronic effects.

Many assessments may involve comparison of sediment chemistry (relative to ESBs or other sediment quality guidelines) and toxicity test results. In cases where results using these two methods agree (either both positive or both negative), the interpretation is clear. In cases where the two disagree, the interpretation is more complex and requires further evaluation.

Individual ESBs address only the effects of the chemical or group of chemicals for which they are derived. For this reason, if a sediment shows toxicity but does not exceed the ESB_{Tier2} value for a chemical of interest, it is likely that the cause of toxicity is a different chemical or chemicals (although the chemical of interest maybe contributing to observed toxicity as a component of a mixture). This result might also occur if the partitioning of the chemical in a sediment is different from that assumed by the K_{OC} value used (see Section 4.2.3 *Influence of Unusual Forms of Sediment Organic Carbon* above).

In other instances, it may be that an ESB_{Tier2} is exceeded but the sediment is not toxic. As explained above, these findings are not mutually exclusive, because the inherent sensitivity of the two measures is different. Four possible circumstances may account for this result. First, the ESB_{Tier2} is intended to protect relatively sensitive species against both acute and chronic effects, whereas toxicity tests are performed with species that may or may not be sensitive to chemicals of concern, and often do not encompass the most sensitive endpoints (e.g., growth or reproduction). As such, one may not expect a nonionic organic chemical concentration near the ESB_{Tier2} to cause lethality

in a short-term toxicity test. Second, a GLI-based SCV, because of the use of SAFs, may overestimate a contaminant's toxicity compared to the intended level of protection, as described in Section 2.4. Third, site-specific conditions may result in lower bioavailability than assumed based on equilibrium partitioning (see Section 4.2.3). Finally, the organism may avoid the sediment or have other mechanisms to reduce exposure relative to that assumed by the EqP approach. To distinguish these potential explanations, species- and endpoint-specific toxicity information could be used to better interpret toxicity test results, and SCV derivation could be reviewed. Spiked sediment tests could also be used to verify the exposure-response relationship for that particular organism and contaminant. If these lines of evidence do not account for the discrepancy between predicted and observed toxicity, then site-specific chemical partitioning could be investigated (U.S. EPA 2003b).

As discussed above, a good method for evaluating the results of toxicity tests is to calculate effect concentrations in sediment that are species and endpoint specific. For some species contained in the water-only toxicity data for the 32 nonionic organic chemicals discussed here, effect concentrations in sediment can be calculated that are specific for that organism (U.S. EPA 2003e). These values could then be used to directly judge whether the absence of toxicity in the test would be expected from the concentration of nonionic organics chemicals present. As noted above, the magnitude of error between toxicity test results and predicted effects is made larger because of the use of SCVs, and SAFs, to derive some ESB_{Tier2} values (see discussion in Section 2.4).

If the exceedance of an ESB is sufficient that one would expect effects in a toxicity test but they are not observed, it is prudent to evaluate the partitioning behavior of the chemical in the sediment. This is performed by isolating interstitial water from the sediment and analyzing it for the chemicals of interest. Predicted chemical concentrations in the interstitial water can be calculated from the measured concentrations in the solid phase (normalized to organic carbon) as follows:

$$\mu g\ chemical/L = (\mu g\ chemical/g_{OC}) \cdot (10^3 g_{OC}/Kg_{OC} \div K_{OC}) \quad (4\text{-}1)$$

For chemicals with log K_{OW} greater than 5.5, corrections for DOC partitioning in the interstitial water will be necessary (see Gschwend and Wu 1985, Burkhard 2000, U.S. EPA 2003b). See U.S. EPA (2003b) for a discussion of the effects of DOC on ESB derivation. If the measured chemical in the interstitial water is substantially less (e.g., 2-3 fold lower or more), it suggests organic carbon in that sediment may not partition similarly to more typical natural organic carbon, and derivation of site-specific ESBs based on interstitial water may be warranted (U.S. EPA 2003b).

Finally, in addition to the use of sediment toxicity tests for interpreting ESB_{Tier2} values, the generation of acute and chronic water-only data with benthic organisms for the nonionic organic chemicals discussed in this document would be very beneficial. Further, acute and chronic whole sediment toxicity data sets with these chemicals would also complement the interpretation of the ESBs.

4.2.7 Effects of Disequilibrium Conditions

As discussed throughout this document, the EqP is based on an assumption of chemical equilibrium between the solid phase of sediment and the interstitial water. In natural settings, equilibrium may not always exist or may be disturbed by episodic events. As such, the potential for disequilibrium and its impact on the interpretation of the equilibrium-based ESBs should be considered. For purposes of this discussion, two types of disequilibria are discussed: 1) disequilibrium between the solid phase sediment and interstitial water; and 2) disequilibrium between the sediment and overlying water column.

With regard to the first, ESBs are based on an assumption that nonionic organic chemicals

are in equilibrium with the sediment and interstitial water and are associated with sediment primarily through absorption to sediment organic carbon. When new chemical is introduced to a sediment, time is required for the chemical to distribute itself between interstitial water and sediment organic carbon. The time required for equilibrium to be achieved is dependent on the characteristics and concentration of the chemical. Sediment spiking experiments suggest that this is typically in the range of weeks.

In areas where sediment erosion and deposition are highly dynamic, equilibrium may be frequently disturbed. The degree to which this would affect the applicability of ESBs depends on the degree and frequency of equilibrium disruption. As noted above, even high K_{ow} nonionic organic compounds come to equilibrium in clean sediment in a period of days, weeks or months. Equilibrium times should be even shorter for mixtures of two sediments that each have previously been at equilibrium. This is particularly relevant in tidal situations where large volumes of sediments are eroded and deposited, even though near equilibrium conditions may predominate over large areas. While the potential for disequilibrium is recognized, it is probably unwise to deviate from the equilibrium assumption without strong evidence that disequilibrium exists over the long term to a sufficient degree to change the expected toxicity of sediment contamination. Recognize that even if there are short-term disturbances to equilibrium between sediment and interstitial water, conditions may quickly re-approach equilibrium between disturbances, such that an equilibrium-based approach is still reasonable, even if there are periods of disturbance/disequilibrium. If it is shown that disequilibrium exists to such an extent that equilibrium-based ESBs are inappropriate, site-specific experimentation may be useful in developing a modified approach (U.S. EPA 2003b).

Even where equilibrium exists between the solid phase sediment and interstitial water, there is often disequilibrium between the sediment and overlying water. This is particularly true for legacy pollution where input of new contamination to the water body has ceased or greatly decreased, and the sediment is now a source of contamination to the overlying water. Some have argued that such disequilibrium reduces exposure of sediment organisms, particularly for those that interact substantially with the overlying water. While the theoretical possibility is clear, the quantitative data from which an appropriate compensation could be calculated is lacking. Moreover, many toxicity test procedures used in the development and testing of EqP theory involve renewal of overlying water and thus include some degree of disequilibrium between the sediment and overlying water. Nonetheless, results from these tests are generally explicable through EqP predictions (e.g., Swartz et al. 1990, DeWitt et al. 1992, Hoke et al. 1994), suggesting that the degree to which this disequilibrium affects exposure is not exceptional, at least for those organisms. In instances where it is determined that EqP does not apply for a particular sediment because of the disequilibrium situations discussed above, site-specific experimentation may be useful in developing a modified approach (U.S. EPA 2003b).

A special case may be in spill situations, where there is a sudden, dramatic influx of new chemical into a system. Immediately following a spill, it can be expected that one or both types of disequilibrium might exist, that the overlying water might have higher chemical activity than in the sediment, and that the solid-phase sediment may not be in equilibrium with the interstitial water. In this situation there is a high potential for ESBs to be under protective.

In sediments where particles of undissolved chemical occur, disequilibrium exists and the benchmarks may be over protective in the sense that chemical concentrations in interstitial water may be lower than would be predicted based on chemical concentrations in sediment and f_{oc}. However, it is also true that in this situation basing an assessment solely on chemical concentrations in the interstitial water might

under-represent the degree of contamination. This is because sufficient chemical exists to contaminate a larger mass of sediment if the sediment containing as yet undissolved chemical is later mixed with other, less contaminated sediment.

Clearly, situations where substantial disequilibrium exists can result in several complexities for interpreting sediment chemistry in the context of ESBs. While it is true that ESBs may be less accurate for such situations, it is also important that an alternate assessment approach be developed that adequately accounts for the site-specific conditions. Disequilibrium should not be used as an excuse to dismiss ESB values without developing an alternate conceptual model on which to base the assessment.

4.3 Example Application of ESB_{Tier2}s Using Conventional and Narcosis Approaches and EqP-based Interpretation

Table 4-1 shows sediment chemistry data (in ug/g_{OC}) for four example marine sediments (i.e., A, B, C, D) along with the corresponding conventional and narcosis ESB_{Tier2} values. The sediment concentrations have been normalized for a TOC of 4.5% using Equation 3-9. Assuming a f_{Solids} of 0.20, ESB_{Tier2} values for benzene, 1,1,2,2-tetrachloroethane, and tetrachloroethene were adjusted using Equations 3-5 and 3-6. These values were compared to measured sediment chemistry. For each of the four sediments, Table 4-1 also shows the ratios of the measured concentration in sediment to the conventional and narcosis ESB_{Tier2}s. For the chemicals with modes of action in addition to narcosis (i.e., the pesticides in these examples), their narcosis contribution is not reported but was calculated to be very small and did not substantially affect the sum narcosis ESBTUs (see discussion in Section 2.3.2).

In sediment A (Table 4-1), all measured chemicals were below their conventional and narcosis ESB_{Tier2} values. In addition, the sum of the ratios of the measured concentrations to their narcosis ESB_{Tier2} (sum narcosis ESBTUs) was only 0.01, far below a value of 1 which would indicate concern for a narcotic effect caused by a mixture of chemicals. While these results themselves indicate no reason to suspect adverse effects to benthic organisms from these chemicals, it must be remembered that this conclusion is limited to the effects of these specific chemicals. It is, of course, still possible that other chemicals could be present in the sediment at concentrations that could cause adverse effects. Toxicity testing would be one way to address the potential for toxicity caused by unmeasured chemicals.

Sediment B (Table 4-1) has the same concentrations of all measured chemicals as in sediment A, except for diazinon and malathion, which exceed their conventional ESB_{Tier2} by factors of 3.9 and 11, respectively. These exceedances suggest concern for adverse effects of these chemicals on benthic organisms, subject to the assumptions underlying the ESB approach as discussed elsewhere in this document. Toxicity testing, particularly with species sensitive to these chemicals, could be used to further evaluate the presence of toxicity, as well as assessing the potential presence of toxicity from unmeasured chemicals. In addition, spiked sediment tests with these chemicals and/or sediment Toxicity Identification Evaluation (TIE) studies (U.S. EPA 2007b) may also be useful in evaluating the expected contribution of these chemicals at these concentrations to sediment toxicity.

For sediment C (Table 4-1), concentrations of the pesticides diazinon, alpha endosulfan and malathion are all below their conventional ESB_{Tier2} values, but three of the other measured chemicals, benzene, ethylbenzene and toluene, exceed their corresponding conventional ESB_{Tier2} values by factors of 4.3, 5.1, and 7.6, respectively. In contrast, these same chemicals do not exceed their narcosis ESB values, nor does the sum of narcosis ESBTUs exceed 1. The exceedance of the conventional ESB_{Tier2}s suggests that the levels of benzene, ethylbenzene, and toluene are high enough to be of potential concern when evaluated by the GLI

Tier 2 assessment approach (GLI 1995). However, the fact that the sum of narcosis ESBTUs does not exceed one raises the possibility that the exceedances for these chemicals may be influenced by conservatism in the GLI Tier 2 paradigm, particularly as it relates to narcotic chemicals (see Section 2.4 for additional discussion). Another issue to be considered relates to the likelihood that other narcotic chemicals, not listed in Table 4-1 may be present and contribute to an overall mixture toxicity. In particular, the elevated concentrations of benzene, ethylbenzene and toluene may suggest contamination with hydrocarbons such as refined petroleum products that may also contain PAHs or other hydrocarbons that could contribute to a narcotic mixture effect. Further analytical chemistry and toxicity testing would be logical supplements to the information in Table 4-1 for determining the overall likelihood of risk to benthic organisms. If PAHs are present, separate ESB guidance for PAH mixtures (U.S. EPA 2003e) can provide an approach to evaluate their potential contribution to narcotic toxicity. The theory underlying narcotic toxicity (Di Toro and McGrath 2000, Di Toro et al. 2000, U.S. EPA 2003e) suggests that the sum of ESBTUs for PAHs could be added to the sum of narcosis Tier 2 ESBTUs in Table 4.1 to assess the combined potency of those chemicals.

Finally, in sediment D (Table 4-1), concentrations of measured pesticides are again low, but concentrations of both BTEX compounds (i.e., benzene, toluene, ethylbenzene, xylene) and the measured chlorinated compounds are higher than for sediment C. Conventional ESB_{Tier2}s are exceeded for several compounds; although no individual narcosis ESB_{Tier2} values are exceeded, the sum of narcosis ESBTUs does exceed 1. In this case, both the conventional ESB_{Tier2}s and the narcosis mixture analysis suggests the potential for adverse effects to benthic organisms. Also, the finding that many compounds, including BTEX, chlorinated benzenes, and other chlorinated hydrocarbons are all present in concentrations approaching their narcosis ESB_{Tier2}s makes it likely that other, unmeasured chemicals in these families may also be present at toxicologically significant concentrations in this sediment, because typical sources of these chemicals to the environment often include many different related compounds (e.g., other di-, tri-, tetra-and hexachloro-benzenes). While this document does not address these additional compounds specifically, an approach for addressing their contribution in a way similar to that used in this document is provided by Di Toro and McGrath (2000) and Di Toro et al. (2000).

Table 4-1 Example applications of ESB_{Tier2} values with several nonionic organic chemicals using conventional and narcosis approaches. In this example, four marine sediments with 4.5% TOC and f_{Solids} of 0.20 are assessed. Sediment concentrations are shown with organic carbon normalization using Equation 3-9. ESB_{Tier2} values modified with Equations 3-5 and 3-6 to account for f_{Solids} for benzene, 1,1,2,2-tetrachloroethane and tetrachloroethene are shown rather than ESB_{Tier2} values in Table 3-1.

Sediment A	Conventional * ESB ($\mu g/g_{OC}$)	Narcosis* ESB ($\mu g/g_{OC}$)	Sediment Concentration ($\mu g/g_{OC}$)	Sediment Concentration/ Conventional ESB	Sediment Concentration/ Narcosis ESB
Benzene	28	1100	0.95	0.0339	0.0009
Ethylbenzene	8.9	970	0.23	0.0258	0.0002
Toluene	5	810	0.32	0.0640	0.0004
m-Xylene	94	980	0.42	0.0045	0.0004
Chlorobenzene	41	570	0.67	0.0163	0.0012
1,2-Dichlorobenzene	33	780	1.2	0.0364	0.0015
Pentachlorobenzene	70	1600	2.3	0.0329	0.0014
Tetrachloromethane	120	770	1.5	0.0125	0.0019
1,1,2,2-Tetrachloroethane	190	1200	1.3	0.0068	0.0011
Hexachloroethane	100	1400	0.89	0.0089	0.0006
Trichloroethene	22	650	0.51	0.0232	0.0008
Tetrachloroethene	50	1000	0.53	0.0106	0.0005
Diazinon	3.6	^	0.02	0.0056	^
Alpha-Endosulfan	0.051	^	0.01	0.1961	^
Malathion	0.11	^	0.01	0.0909	^
Sum Narcosis ESBTUs					0.0111

Sediment B	Conventional * ESB ($\mu g/g_{OC}$)	Narcosis* ESB ($\mu g/g_{OC}$)	Sediment Concentration ($\mu g/g_{OC}$)	Sediment Concentration/ Conventional ESB	Sediment Concentration/ Narcosis ESB
Benzene	28	1100	0.95	0.0339	0.0009
Ethylbenzene	8.9	970	0.23	0.0258	0.0002
Toluene	5	810	0.32	0.0640	0.0004
m-Xylene	94	980	0.42	0.0045	0.0004
Chlorobenzene	41	570	0.67	0.0163	0.0012
1,2-Dichlorobenzene	33	780	1.2	0.0364	0.0015
Pentachlorobenzene	70	1600	2.3	0.0329	0.0014

Tetrachloromethane	120	770	1.5	0.0125	0.0019
1,1,2,2-Tetrachloroethane	190	1200	1.3	0.0068	0.0011
Hexachloroethane	100	1400	0.89	0.0089	0.0006
Trichloroethene	22	650	0.51	0.0232	0.0008
Tetrachloroethene	50	1000	0.53	0.0106	0.0005
Diazinon	3.6	^	13.9	**3.8611**	^
Alpha-Endosulfan	0.051	^	0.01	0.1961	^
Malathion	0.11	^	1.2	**10.9091**	^
Sum Narcosis ESBTUs					0.0111

Sediment C	Conventional * ESB ($\mu g/g_{OC}$)	Narcosis* ESB ($\mu g/g_{OC}$)	Sediment Concentration ($\mu g/g_{OC}$)	Sediment Concentration/ Conventional ESB	Sediment Concentration/ Narcosis ESB
Benzene	28	1100	120	**4.2857**	0.1091
Ethylbenzene	8.9	970	45	**5.0562**	0.0464
Toluene	5	810	38	**7.6000**	0.0469
m-Xylene	94	980	31	0.3298	0.0316
Chlorobenzene	41	570	1.3	0.0317	0.0023
1,2-Dichlorobenzene	33	780	3.7	0.1121	0.0047
Pentachlorobenzene	70	1600	8.8	0.1257	0.0055
Tetrachloromethane	120	770	1.1	0.0092	0.0014
1,1,2,2-Tetrachloroethane	190	1200	0.66	0.0035	0.0006
Hexachloroethane	100	1400	0.43	0.0043	0.0003
Trichloroethene	22	650	0.19	0.0086	0.0003
Tetrachloroethene	50	1000	0.21	0.0042	0.0002
Diazinon	3.6	^	0.02	0.0056	^
Alpha-Endosulfan	0.051	^	0.01	0.1961	^
Malathion	0.11	^	0.01	0.0909	^
Sum Narcosis ESBTUs					0.2493

Sediment D	Conventional * ESB ($\mu g/g_{OC}$)	Narcosis* ESB ($\mu g/g_{OC}$)	Sediment Concentration ($\mu g/g_{OC}$)	Sediment Concentration/ Conventional ESB	Sediment Concentration/ Narcosis ESB
Benzene	28	1100	410	**14.6429**	0.3727
Ethylbenzene	8.9	970	320	**35.9551**	0.3299
Toluene	5	810	290	**58.0000**	0.3580

m-Xylene	94	980	360	**3.8298**	0.3673
Chlorobenzene	41	570	250	**6.0976**	0.4386
1,2-Dichlorobenzene	33	780	140	**4.2424**	0.1795
Pentachlorobenzene	70	1600	87	**1.2429**	0.0544
Tetrachloromethane	120	770	12	0.1000	0.0156
1,1,2,2-Tetrachloroethane	190	1200	16	0.0842	0.0133
Hexachloroethane	100	1400	31	0.3100	0.0221
Trichloroethene	22	650	27	**1.2273**	0.0415
Tetrachloroethene	50	1000	15	0.3000	0.0150
Diazinon	3.6	^	0.02	0.0056	^
Alpha-Endosulfan	0.051	^	0.01	0.1961	^
Malathion	0.11	^	0.01	0.0909	^
Sum Narcosis ESBTUs					**2.2081**

* = See Section 2.3 for definition.
^ = Not Reported.

Section 5

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Appendix A

Description of the Derivation of Conventional Freshwater Chronic Toxicity Values using the Great Lakes Water Quality Initiative (GLI 1995) Approach

A.1 Acquisition and Review of Conventional Aquatic Toxicity Data

As discussed above, when possible, conventional ESBs were based on FCVs for aquatic life (Stephan et al.1985). When FCVs could not be derived, the ESBs were calculated from SCVs for aquatic life using the GLI approach (Suter and Mabrey 1994, GLI 1995, Suter and Tsao 1996). The purpose of this section is to describe the procedure used to derive SCVs from data in AQUIRE (now ECOTOX) and other sources.

The following restrictions on toxicity data and reference sources used were applied:

1. Acute toxicity data for only freshwater species were used (GLI, 1995), whereas acute-chronic ratios (ACRs) for both freshwater and saltwater species were used in order to expand the number of available ACRs.

2. Only the following were used as sources of references:

a. U.S. Environmental Protection Agency's AQUIRE (now ECOTOX) database.
b. Tables in existing documents from EPA's Office of Research and Development.

A preliminary review was conducted on test results obtained by means of a search of AQUIRE (now ECOTOX). Only information that could be retrieved from AQUIRE (now ECOTOX) was used in this review. Each test result was rejected if one or more of the reasons listed below applied. The first three reasons for rejection given below were addressed in the search strategy used to find test results in AQUIRE (now ECOTOX). All pertinent test results were printed and reviewed manually using the "Reasons for Rejection of a Test Result Based on Information in AQUIRE." For each test result that was not rejected, a copy of the original report was reviewed as described in the next section of this report, "Data Rejection Checklist."

Reasons for Rejection of a Test Result Based on Information in AQUIRE (now ECOTOX):

___ The test was not conducted in the laboratory (i.e., **Site** was not LAB).
___ Poor documentation (the documentation code (**Dc**) was not 1 or 2).
___ The endpoint was not reported (i.e., **Endpt** was left blank or was "NR").
___ The purity of the test chemical was less than 80% (i.e., **Chem_char** < 80%).
___ The test species (**Latin, Species**) was not an aquatic animal.
___ The test species (**Latin, Species**) was not a resident North American species.
___ The test species was *Wyeomyia smithii* (i.e., the pitcher plant mosquito) or was in the genus *Artemia* (i.e., it was a brine shrimp).

The following reasons for rejection applied only to acute toxicity tests:

___ The test exposure was not static, renewal, or flow-through (i.e., **Extype** was not S, R, or F).
___ The test was not conducted in freshwater (i.e., **Media** was not FW).
___ If the test species was Cladoceran (CLAD, water flea), copepod (COPE), midge or phantom midge (insect, family Chironomidae, order Diptera, DIPT), the **Duration** was less than 2 days (48 hr).
___ For all other animal species, the **Duration** was less than 4 days (96 hrs).
___ The **endpoint** was not LC_{50} or EC_{50} or IC_{50}.
___ The **effect** was not EQU, IMM, and/or MOR, except that SHD (incompletely developed shells, change in the ability to grow a shell) was acceptable for bivalve molluscs.

The following reasons for rejection applied only to chronic toxicity tests:

___ The concentrations of test material were not measured (i.e., **Method** was not M) in the test solution.
___ The test exposure was not flow-through or renewal (i.e., **Extype** was not F or R).

If the test species was a Cladoceran (CLAD, water flea) or copepod (COPE):

___ The **Life stage** was older than 24 hr.
___ The **Duration** was less than 21 days (except less than 7 days for *Ceriodaphnia*).
___ For all other species, the **Duration** was less than 24 days.

Stephan et al. (1985), references cited therein, and other pertinent publications (e.g., the American Fisheries Society guidebook series for North American fishes, molluscs, and crustacea) were used to determine whether a vertebrate or invertebrate aquatic species is resident in North America. Because of various constraints, some species listed below were assumed to be nonresident if a limited search did not demonstrate that they were resident. Any species that was said to have been field-collected in North America was considered resident.

Examples of resident species not in Stephan et al. (1985):

Chironomus riparius	midge
Gila elegans	bonytail
Gillia attilis	buffalo pebblesnail
Lestes congener	damselfly
Sigara alternata	water boatman
Stenonema interpunctatum	mayfly
Umbra pygmaea	eastern mudminnow

Examples of nonresident species not in Stephan et al. (1985):

Anguilla anguilla	common eel (assumed nonresident)
Anodonta anatina	fresh-water mussel
Anodonta cyanea	swan mussel
Barbus ticto	two-spotted; tic tac toe barb
Carassius carassius	Crucian carp
Chana punctatus or *gachua*	snake-head catfish
Cirrhinus mrigala	carp, hawkfish
Heteropneustes fossilis	Indian catfish
Macrobrachiu rosenbergii	giant freshwater prawn
Mystus vittatus	catfish
Notopterus notopterus	featherback
Paratelphusa jacquemontii	crab (probably)
Rasbora heteromorpha	harlequinfish/red rasbora
Spicodiaptomus chilospinus	calanoid copepod (assumed nonresident)

Resident status of organisms for which only the genus and "sp." were provided as the scientific name (e.g., *Peltodytes* sp.) was based on the location where the organisms were collected.

This checklist was used to review the acceptability of results of aquatic toxicity tests on nonionic organic chemicals including all references that were obtained from AQUIRE (now ECOTOX) and passed the "Preliminary Review of Records from AQUIRE." Because this second review was performed on all test results regardless of whether the reference came from AQUIRE (now ECOTOX), all items on the AQUIRE (now ECOTOX) review were also included here. This review was performed using the original publication and sources of supplemental information; this review was not performed using only secondary sources.

This final review covered both the quality of the test result and whether it was the kind of result that had been specified for use in this document. A test result that was deemed unacceptable for use in this document might be acceptable for another use. A result that was deemed unacceptable was not necessarily an incorrect result; it just might have been too questionable to use. For example, an LC_{50} obtained using unacceptable methodology might have been the same as an LC_{50} using acceptable methodology. The LC_{50} from the test using the unacceptable methodology, however, was unacceptable because it was questionable. In many cases, some test results in a publication were acceptable, whereas others were unacceptable. Similarly, one result from a test (e.g., a 24-hr LC_{50}) might not have been acceptable although another (e.g., a 48-hr LC_{50}) was acceptable.

Each test result was placed in one of three categories for the purposes of this review:

1. A test result was assumed acceptable if the test was conducted at EPA laboratories in Corvallis (OR), Duluth (MN), Gulf Breeze (FL), or Narragansett (RI); was conducted at the U.S. Fish and Wildlife Service laboratory in La Crosse, Wisconsin; was contained in Mayer and Ellersieck (1986); was conducted at the U.S. Department of the Interior laboratory in Columbia, Missouri, after the period covered by the report published by Mayer and Ellersieck (1986); or was contained in the University of Wisconsin-Superior data summary volumes (Brooke et al.1984; Geiger et al.1985, 1986, 1988, 1990). Reports from these sources usually contained information concerning methodology, but the result was assumed acceptable even if little information was available concerning methodology. Results in this category were rejected only if a major problem was known to exist.

2. A test result was assumed acceptable if the test was reported to have been conducted according to procedures described by such American Society for Testing and Materials (ASTM) standards as:

   ASTM Standard E 729, Guide for Conducting Acute Toxicity Tests with Fishes, Macroinvertebrates, and Amphibians

   ASTM Standard E 1241, Guide for Conducting Early Life-Stage Toxicity Tests with Fishes

   ASTM Standard E 1193, Guide for Conducting Renewal Life-Cycle Tests with *Daphnia magna*

   ASTM Standard E 1295, Guide for Conducting Three-brood, Renewal Toxicity Tests with *Ceriodaphnia dubia*

   Or procedures described by *Standard Methods*, the European Economic Community (EEC), the International Organization for Standardization, or the Organization for Economic Cooperation and Development (OECD), and if the description of the methodology at least mentioned such factors as acclimation, temperature control, controls, solvent and solvent control (if used), source of water, randomization, and duplication. Results in this category were, however, rejected if a single major problem was identified.

3. All other test results were in a third category. Whether they were accepted or rejected depended on the information available concerning the methodology and results. The result was rejected if insufficient information was available to evaluate the test. Identification of a single major problem, or at least three minor problems, were grounds for rejection of a test result, and most results with this number of identified problems were rejected. Best professional judgment was; however, applied to determine whether identified problems warranted rejection of the result.

The review of test results required judgments, starting with decisions about what items to include on the following list, and whether each one was major or minor. Applying the list also required judgment. For example, a test result was always rejected if a surfactant was used in the preparation of a stock solution or the test solutions, even if the test was conducted by Mount and Stephan (1967). If no information was given concerning the use of surfactants, test results in the first category above were deemed acceptable, but it was identified as a problem for other test results.

Reasons for Rejection (Asterisks indicate major problems; all others are minor problems.)

Report

___ * The test results were not available for public distribution in a dated and signed hard copy (e.g., publication, manuscript, letter, memorandum, etc.).

___ * The test results were from a secondary publication, except those results contained in the *Manual of Acute Toxicity: Interpretation and Data Base for 410 Chemicals and 66 Species of Freshwater Animals* (Mayer and Ellersieck 1986) were considered acceptable.

___ * Methodology and/or results were not adequately and clearly described, except for category 1. In some cases, other papers by the same or different authors provided the necessary information.

Test chambers

___ All test chambers and any compartments within the chambers were not identical.

___ * The test result was from a microcosm or model ecosystem study.

___ * The test chambers were made from or lined with PVC, except that the presence of PVC in chambers was acceptable if the test material was miscible with or very soluble in water or concentrations of the test material in solutions were measured.

Test material

___ The test material was not adequately described.

___ * The organisms were exposed to the test material via food, sediment, injection, gavage, etc.; exposure was not via only the test solutions.

___ * The test material was a component of a drilling mud, effluent, fly ash, mixture, formulation, sediment, or sludge.

___ The purity of the test material was less than <u>80 percent</u> (e.g., the test material contained less than 80 percent active ingredient); <u>analytical-grade, reagent-grade, or technical-grade</u> materials were considered acceptable unless known to be unacceptable.

Exception: The test material could contain less active ingredient if data were available to show that tests on the material produced the same results as tests on material that was at least 80 percent pure.

___ * The test material was an emulsifiable concentrate, a wettable powder, or a specially prepared mixture that contained a surfactant and/or an organic solvent that was not miscible with water.

___ * A surfactant or an organic solvent that was not miscible with water was used in the preparation of a stock solution or the test solutions.

___ If a water-miscible solvent was used to prepare the stock solution and/or test solutions, its concentration exceeded 0.5 mL/L in the test solutions.

___ * The test material was introduced into the test chamber by evaporating it onto the test chamber and adding dilution water.

Exception: This procedure was acceptable if the concentrations of test material in the test solutions were measured.

___ * Concentrations of test material in the test solutions were not measured for chronic toxicity tests (measurement was not necessary for acute tests).

___ Measured concentrations of test material during a flow-through test varied too much.

___ * For highly volatile, hydrolyzable, or degradable materials, the test was static or renewal (i.e., not flow-through) and/or concentrations of test material were not measured often enough using acceptable analytical methods.

___ * Exposure to the test material was intermittent, not continuous.

Test organisms

___ * The test species was not an aquatic animal.

___ * The test species was a single-celled organism.

___ * The test species was not a resident North American species.

___ * The test species was *Wyeomyia smithii* (i.e., the pitcher plant mosquito) or was in the genus *Artemia* (i.e., it was a brine shrimp).

___ * The test was not conducted using "whole" organisms; for example, the test was conducted using tissues or cell cultures.

___ * The test result was calculated for a mixture of species, especially if the species were in different genera.

___ * At least some of the test organisms were in a life stage that is not aquatic for at least part of the test.

___ * The test organisms were cladocerans that were obtained from a stock culture in which ephippia were being produced.

___ The test organisms showed signs of stress or disease before the test.

___ * The test was begun with organisms within 10 days after they were treated to cure or prevent disease and/or the organisms were treated during the test.

___ * Test organisms were previously exposed to substantial concentrations of the test material or other contaminants and were not held in clean water for at least 10 days before the beginning of the test.

___ * The test organisms were not acclimated

to or were not maintained in the dilution water at the test temperature for at least 48 hours before the beginning of the test.

___ The test organisms were mishandled or excessively disturbed before or during the test.

___ * The test organisms were fed during an acute toxicity test.

Exceptions:

1. Saltwater annelids and mysids could be fed during acute tests.
2. The test material does not sorb or complex readily with food.
3. Data were available to show that the presence of food probably would not affect the results of the test.

___ There were fewer than 10 test organisms per treatment.

___ There were not two or more replicates (groups of individuals of a species) tested for each concentration for chronic tests.

___ The test organisms were crowded in the test chambers.

___ * The test organisms reproduced during the test and all of the new organisms could not be distinguished from the initial organisms at the end of the test. (This has been a problem in some tests with rotifers.)

Controls

___ * There was no control treatment.

___ * There was a control treatment, but it was not comparable to the other treatments.

___ No data were reported for the controls.

___ * More than 10 percent of the control organisms died or showed signs of stress or disease or were otherwise adversely affected, except that a higher percentage was acceptable for a few species.

___ * Survival, growth, or reproduction in the control treatment for chronic tests were unacceptably low. (The limits of acceptability depended on the species.)

Dilution water

___ * Distilled or deionized water was used without addition of appropriate salts.

___ * Chlorinated water was used without adequate dechlorination.

___ * River water was used as the dilution water without appropriate treatment.

___ * The concentration of total organic carbon (TOC) or particulate matter (PM) in the dilution water exceeded 5 mg/L.

Exceptions:

1. TOC or PM could exceed 5 mg/L if a relationship was developed between toxicity and TOC or PM.
2. Data were available to show that TOC or PM probably would not affect the results of the test.

___ The dilution water contained unusual amounts or ratios of inorganic ions.

Test conditions

___ Turbulence in the test chamber, resulting from aeration, stirring, or design (of flow-through chambers), was excessive.

___ The temperature, pH, etc., of the test solutions were not adequately controlled.

___ * The pH of the dilution water was below 6.5 or above 9.0.

___ * The concentration of dissolved oxygen in a renewal or flow-through test was less than 60 percent of saturation.

___ * The concentration of dissolved oxygen during a static test was less than 60 percent saturation during the first 48 hours, or less than 40 percent of saturation from 48 to 96 hours.

___ Treatments, test organisms, and experimental units were not appropriately randomized.

___ * The dilution factor was greater than 9.

The toxicity tests that were not rejected were next evaluated to determine whether they provided the kinds of acute and chronic results that were to be used, as described in the next two sections.

A.2 Compilation of Acute Values

The following kinds of results of acute toxicity tests were used:

1. For midges, phantom midges, daphnids, and other cladocerans, the result used was the 48-hr EC_{50} based on percentage of organisms immobilized plus percentage of organisms killed. If such an EC_{50} was not available from a test, the 48-hr LC_{50} was used in place of the desired 48-hr EC_{50}. An EC_{50} or LC_{50} of longer

than 48 hours was used as long as the animals were not fed and the control animals were acceptable at the end of the test. Tests with daphnids and other cladocerans should have been started with organisms less than 24 hours old, and tests with midges and phantom midges should have been started with second- or third-instar larvae.

2. For embryos and larvae of barnacles, bivalve molluscs (clams, mussels, oysters, and scallops), sea urchins, lobsters, crabs, shrimp, and abalones, the result used was the 96-hr EC_{50} based on the percentage of organisms with incompletely developed shells plus the percentage of organisms killed. If such an EC_{50} was not available from a test, the lower of the 96-hr EC_{50} based on percentage of organisms with incompletely developed shells and the 96-hr LC_{50} was used in place of the desired 96-hr EC_{50}. If the duration of the test was between 48 and 96 hours, the EC_{50} or LC_{50} at the end of the test was used.

3. For all other freshwater and saltwater animal species and older life stages of barnacles, bivalve molluscs, sea urchins, lobsters, crabs, shrimp, and abalones, the result used was the 96-hr EC_{50} based on the percentage of organisms exhibiting loss of equilibrium plus the percentage of organisms immobilized plus the percentage of organisms killed. If such an EC_{50} was not available from a test, the 96-hr LC_{50} was used in place of the desired 96-hr EC_{50}.

Acceptable freshwater acute test results were entered in taxonomic order. If the tests were conducted properly, acute values reported as "greater than" values and those that were above the solubility of the test material were entered because rejection of such acute values would unnecessarily lower the Final Acute Value (FAV) by eliminating acute values for resistant species. Reported results were not rounded off to fewer than four significant digits.

In the case of a species for which at least one acceptable acute value was available, the species mean acute value (SMAV) was calculated as the geometric mean of the results of all flow-through tests in which the concentrations of test material were measured. In the case of a species for which no such result was available, the SMAV was calculated as the geometric mean of all available acute values (i.e., results of flow-through tests in which the concentrations were not measured and results of static and renewal tests based on initial concentrations of test material). (Nominal concentrations were acceptable for most test materials if measured concentrations were not available.) If only one acceptable acute value was available for a species, the SMAV was that value. The following information was also considered:

1. If the available data indicated that one or more life stages were more resistant than one or more other life stages of the same species by at least a factor of 2, the data for the more resistant life stages were not used in the calculation of the SMAV. This procedure was followed because a species can be considered protected from acute toxicity only if all life stages are protected.

2. The agreement of the data within and between species was considered. Acute values that appeared to be questionable in comparison with other acute and chronic data for the same species and for other species in the same genus usually were not used in the calculation of a SMAV. For example, if the acute values available for a species or genus differed by more than a factor of 10, some or all of the values usually were not used in calculations.

SMAVs were not rounded off to fewer than four significant digits.

The geometric mean of N numbers was calculated as the Nth root of the product of the N numbers. Alternatively, the geometric mean was calculated by adding the logarithms of the N numbers, dividing the sum by N, and taking the antilog of the quotient. Either natural (base e) or common (base 10) logarithms were used to calculate geometric means as long as they were used consistently within each set of data (i.e., the antilog used matched the logarithm used). The geometric mean of two numbers was usually calculated as the square root of the product of the two numbers. The geometric mean of one number was that number.

A.3 Compilation of Chronic Values

Results of three kinds of chronic toxicity tests were used:

1. *Life-cycle toxicity tests*. These tests consist of exposures of each of two or more groups of individuals of a species to a different concentration of the test material throughout a life cycle. To ensure that all life stages and life processes are exposed, tests with fish begin with embryos or newly hatched young less than 48 hours old, continue through maturation and reproduction, and end not less than 24 days (90 days for salmonids) after the hatching of the next generation. Tests with daphnids begin with young less than 24 hours old and continue until 7 days past the median time of first brood release in the controls.

   For fish, data are obtained and analyzed on survival and growth of adults and young, maturation of males and females, eggs spawned per female, embryo viability (salmonids only), and hatchability. For daphnids, data are obtained and analyzed on survival and young per female. For mysids, data are obtained and analyzed on survival, growth, and young per female.

2. *Partial life-cycle toxicity tests*. These tests consist of exposures of each of two or more groups of individuals of a species of fish to different concentrations of the test material through most portions of a life cycle. Partial life-cycle tests are allowed with fish species that require more than a year to reach sexual maturity, so that all major life stages are exposed to the test material in less than 15 months (i.e., the tests begin with immature juveniles at least 2 months prior to active gonad development and end not less than 24 days (90 days for salmonids) after hatching of the next generation).

   Data are obtained and analyzed on survival and growth of adults and young, maturation of males and females, eggs spawned per female, embryo viability (salmonids only), and hatchability.

3. *Early life-stage toxicity tests*. These tests consist of 28- to 32-day (60-days post hatch for salmonids) exposures of the early life stages of a species of fish from shortly after fertilization through embryonic, larval, and early juvenile development. Results of early life-stage tests in which the incidence of mortalities or abnormalities increased substantially near the end of the test are not used because the results of such tests are probably not good predictions of the results of comparable life-cycle or partial life-cycle tests.

   Data are obtained and analyzed on survival and growth. Results of early life-stage tests were used as predictions of results of life-cycle and partial life-cycle tests with the same species. Therefore, when results of a life-cycle or partial life-cycle test were available, results of an early life-stage test with the same species were not used.

Acceptable freshwater and saltwater chronic test results were sorted by taxonomic order. Reported results were not rounded off to fewer than four significant digits.

A chronic value was obtained either by calculating the geometric mean of the lower and upper chronic limits from a chronic test or by analyzing chronic data using regression analysis. A lower chronic limit was the highest tested concentration (a) in an acceptable chronic test, (b) that did not cause an unacceptable amount of adverse effect on any of the specified biological measurements, and (c) below which no tested concentration caused an unacceptable effect. An upper chronic limit was the lowest tested concentration (a) in an acceptable chronic test, (b) that did cause an unacceptable amount of adverse effect on one or more of the specified biological measurements, and (c) above which all tested concentrations also caused such an effect.

Because various authors have used a variety of terms and definitions to interpret and report results of chronic tests, reported results were reviewed carefully. The amount of effect that was considered unacceptable was based on a statistical hypothesis test and/or the percent reduction from the controls. For example, a small percent reduction (e.g., 3 percent) was considered acceptable even if it was statistically significantly different from the control, whereas a large percent reduction (e.g., 30 percent) was considered unacceptable even if it was not statistically significant.

A.4 Compilation of Acute-Chronic Ratios

Acceptable freshwater and saltwater ACRs and the test results on which they were based were recorded.

1. For each chronic value for which at least one corresponding appropriate acute value was available, an ACR was calculated, using for the numerator the geometric mean of the results of all acceptable flow-through acute tests in the same dilution water and in which the concentrations were measured. Static and renewal tests were acceptable for daphnids. Acute tests with fish should have been started with juveniles, whereas acute tests with daphnids should have been started with organisms less than 24 hr old.
2. Acute test(s) that were part of the same study as the chronic test were used if available. If acute tests were not conducted as part of the same study, acute tests conducted in the same laboratory and dilution water, but in a different study, were used. If no such acute tests were available, results of acute tests conducted in the same dilution water in a different laboratory were used. If no such acute tests were available, an ACR was not calculated.
3. For fish, if chronic test data for life-cycle or partial life-cycle tests were available for a species, they were used for the denominator instead of an early life-stage test for the same species.

For each species, the species mean acute-chronic ratio (SMACR) was calculated as the geometric mean of all ACRs available for that species.

A.5 Calculation Procedures

For each genus for which one or more SMAVs were available, the genus mean acute value (GMAV) was calculated as the geometric mean of the SMAVs available for the genus. The GMAVs were ranked from highest to lowest, with the lowest GMAV assigned rank 1. The associated SMAVs and freshwater SMACRs were also entered.

To derive a freshwater FAV (Stephan et al., 1985), it was necessary to have results of acceptable acute toxicity tests with at least one species of freshwater animal in eight different families, such that all of the following requirements were satisfied:

1. The family Salmonidae in the Class Osteichthyes.
2. A second family in the Class Osteichthyes, preferably a commercially or recreationally important warm-water species (e.g., bluegill, channel catfish).
3. A third family in the phylum Chordata (may be in the class Osteichthyes or may be an amphibian, etc.).
4. A planktonic crustacean (e.g., cladoceran, copepod).
5. A benthic crustacean (e.g., ostracod, isopod, amphipod, crayfish).
6. An insect (e.g., mayfly, dragonfly, damselfly, stonefly, caddisfly, mosquito, midge).
7. A family in a phylum other than Arthropoda or Chordata (e.g., Rotifera, Annelida, Mollusca).
8. A family in any order of insect or any phylum not already represented.

If all eight of the minimum data requirements (MDRs) were satisfied, the FAV was calculated using the computer program given on page 98 of Stephan et al. (1985), using the total number of GMAVs and the four lowest. The calculated FAV was compared with the low SMAVs to determine whether the FAV should be lowered to protect a commercially or recreationally important species.

If all eight of the acute freshwater MDRs were not met, a freshwater secondary acute value (SAV) was calculated. To derive a freshwater SAV, it was necessary to have at least one acceptable acute toxicity test with a species in one of three genera (*Daphnia, Ceriodaphnia*, or *Simocephalus*) in the Family Daphnidae.

The SAV was calculated using the lowest GMAV and the secondary acute factor (SAF) corresponding to the number of minimum data requirements that were satisfied:

$$\textbf{SAV} = \frac{\text{lowest Genus Mean Acute Value}}{\text{Secondary Acute Factor}}$$

The SAFs from GLI (1995):

Number of MDRs Satisfied	SAF
1	21.9
2	13.0
3	8.0
4	7.0
5	6.1
6	5.2
7	4.3

If sufficient data are available, chronic values can be calculated in the same manner as acute values, without the use of an ACR. Genus mean chronic values (GMCVs) were then calculated as the geometric mean of available chronic values. If the necessary data were available, the chronic value was calculated using the computer program used to calculate the FAV. (This option is rarely used because the chronic MDRs are rarely satisfied.)

If the data were not available to allow use of the computer program (e.g., Stephan et al. 1985), a final acute-chronic ratio (FACR) was calculated if acceptable ACRs were available for at least one species of aquatic animal in at least three different families, and of the three species:

1. At least one was a fish.
2. At least one was an invertebrate.
3. At least one was an acutely sensitive freshwater species. (The other two could be saltwater species.)

If the MDRs for calculation of an FACR were satisfied, an FACR was calculated; otherwise an SACR was derived.

For some materials, the ACR seems to be the same for all species, but for other materials the ratio seems to increase or decrease as the SMAV increases. The FACR was obtained in one of four ways, depending on the data available:

1. If the SMACR seemed to increase or decrease as the SMAVs increased, the FACR was calculated as the geometric mean of the ACRs for species whose SMAVs were close to the FAV or SAV.
2. If no major trend was apparent and the ACRs for a number of species were within a factor of 10, the FACR was calculated as the geometric mean of the SMACRs that were within a factor of 10.
3. For acute tests conducted on metals and possibly other substances with embryos and larvae of barnacles, bivalve molluscs, sea urchins, lobsters, crabs, shrimp, and abalones, the ACR was usually assumed to be 2. Chronic tests are very difficult to conduct with most such species, but it is likely that the sensitivities of embryos and larvae would determine the results of life-cycle tests. Thus, if the lowest available SMAVs were obtained with embryos and larvae of such species, the FACR was assumed to be 2.
4. If the most appropriate SMACRs were less than 2.0, and especially if they were less than 1.0, acclimation had probably occurred during the chronic test. Because continuous exposure and acclimation cannot be assured to provide adequate protection in field situations, the FACR was assumed to be 2.

If the available SMACRs did not fit one of the above cases, an FACR could not be obtained and an SACR was derived if possible.

If the available ACRs did not satisfy the minimum data requirements for derivation of an FACR, sufficient ACRs of 18 were assumed so that the MDRs were satisfied. The SACR was then calculated as the geometric mean of the measured and assumed ACRs. If no experimentally determined ACRs were available, the SACR was 18 (GLI 1995).

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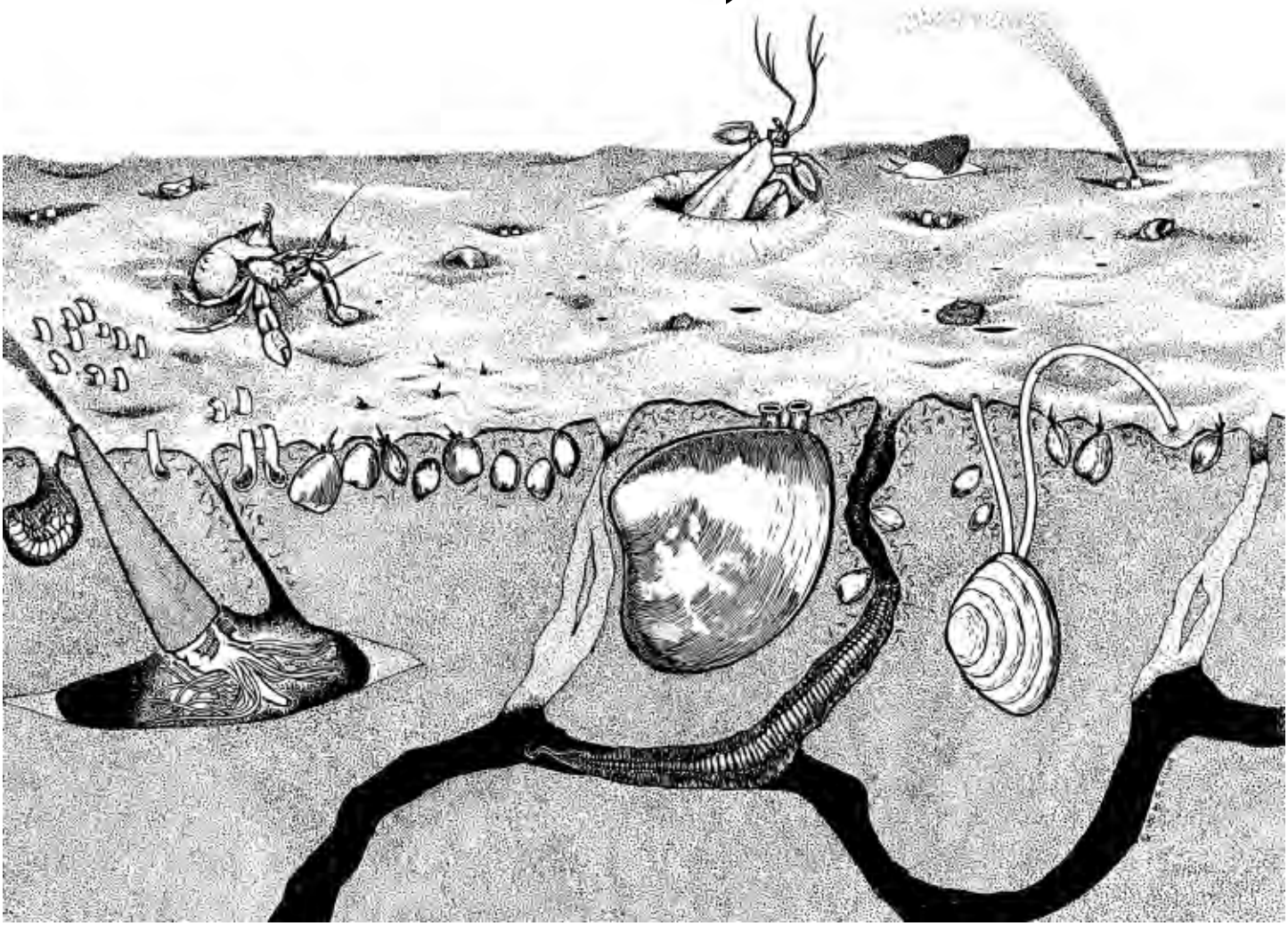
Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Metal Mixtures (Cadmium, Copper, Lead, Nickel, Silver, and Zinc)



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Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Metal Mixtures (Cadmium, Copper, Lead, Nickel, Silver and Zinc)

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Notice

The Office of Research and Development (ORD) has produced this document to provide procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for metal mixtures. ESBs may be useful as a complement to existing sediment assessment tools. This document should be cited as:

> U.S. EPA. 2005. Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Metal Mixtures (Cadmium, Copper, Lead, Nickel, Silver and Zinc). EPA-600-R-02-011. Office of Research and Development. Washington, DC 20460

This document can also be found in electronic format at the following web address:

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The information in this document has been funded wholly by the U.S. Environmental Protection Agency. It has been subject to the Agency's peer and administrative review, and it has been approved for publication as an EPA document.

Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Abstract

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations of metal mixtures in sediment which are protective of the presence of benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration. This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any single effect endpoint. For the purposes of this document, the ESB for mixtures of the metals cadmium, copper, lead, nickel, silver, and zinc, the $ESB_{AVS:WQC}$, is derived based on two complementary approaches. In the first approach, the $ESB_{AVS:WQC}$ is based on the solid phase and interstitial water phase of sediments. In sediments, these metals should not cause direct toxicity to benthic organisms if the ΣSEM-AVS is $\leq$ 0.0. In the second approach, sediments containing these metals should not cause direct toxicity to benthic organisms if the sum of the dissolved interstitial water concentrations for each of the metals ($\Sigma M_{i,d}$) divided by their respective Water Quality Criteria (WQC) Final Chronic Value (FCV) is $\leq$1.0. Uncertainty bounds on ΣSEM-AVS and (ΣSEM-AVS)/f_{OC} can be used to identify sediments where toxicity, because of these metals, is unlikely, uncertain, or likely. If the ΣSEM-AVS is > 0.0 or $\Sigma M_{i,d}$ divided by their respective FCVs is >1.0, effects may occur with increasing severity as the degree of exceedance increases. A procedure for addressing chromium toxicity in sediments is also included in an appendix.

The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans.

Foreword

Under the Clean Water Act (CWA), the U.S. Environmental Protection Agency (EPA) and the States develop programs for protecting the chemical, physical, and biological integrity of the nation's waters. To support the scientific and technical foundations of the programs, EPA's Office of Research and Development has conducted efforts to develop and publish equilibrium partitioning sediment benchmarks (ESBs) for some of the 65 toxic pollutants or toxic pollutant categories. Toxic contaminants in bottom sediments of the nation's lakes, rivers, wetlands, and coastal waters create the potential for continued environmental degradation even where water column contaminant levels meet applicable water quality standards. In addition, contaminated sediments can lead to water quality impacts, even when direct discharges to the receiving water have ceased.

The ESBs and associated methodology presented in this document provide a means to estimate the concentrations of a substance that may be present in sediment while still protecting benthic organisms from the effects of that substance. These benchmarks are applicable to a variety of freshwater and marine sediments because they are based on the biologically available concentration of the substance in the sediments. These ESBs are intended to provide protection to benthic organisms from direct toxicity due to this substance. In some cases, the additive toxicity for specific classes of toxicants (e.g., metal mixtures or polycyclic aromatic hydrocarbon mixtures) is addressed. The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans.

ESBs may be useful as a complement to existing sediment assessment tools, to help assess the extent of sediment contamination, to help identify chemicals causing toxicity, and to serve as targets for pollutant loading control measures.

This document provides technical information to EPA Program Offices, including Superfund, Regions, States, the regulated community, and the public. For example, ESBs when used in the Superfund process, would serve for screening purposes only, not as regulatory criteria, site specific clean-up standards, or remedial goals. The ESBs do not substitute for the CWA or EPA's regulations, nor is it a regulation itself. Thus, it cannot impose legally binding requirements on EPA, States, or the regulated community. EPA and State decision makers retain the discretion to adopt approaches on a case-by-case basis that differ from this technical information where appropriate. EPA may change this technical information in the future. This document has been reviewed by EPA's Office of Research and Development (Mid-Continent Ecology Division, Duluth, MN; Atlantic Ecology Division, Narragansett, RI), and approved for publication.

Mention of trade names or commercial products does not constitute endorsement or recommendation of use.

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Front cover image provided by Wayne R. Davis and Virginia Lee.

Contents

Tables

Figures

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**Series Editor

Executive Summary

This equilibrium partitioning sediment benchmark (ESB) document describes procedures to derive concentrations of metal mixtures in sediment which are protective of the presence of benthic organisms. The equilibrium partitioning (EqP) approach was chosen because it accounts for the varying biological availability of chemicals in different sediments and allows for the incorporation of the appropriate biological effects concentration U.S. EPA (2003a). This provides for the derivation of benchmarks that are causally linked to the specific chemical, applicable across sediments, and appropriately protective of benthic organisms.

Equilibrium partitioning theory predicts that these metals partition in sediment between acid volatile sulfide (AVS, principally iron monosulfide), interstitial (pore) water, benthic organisms, and other sediment phases such as organic carbon. Biological responses of benthic organisms to these metals in sediments are different across sediments when the sediment concentrations are expressed on a dry weight basis, but similar when expressed on a ΣSEM-AVS or interstitial water basis. The difference between the sum of the molar concentrations of simultaneously extracted metal (ΣSEM, the metal extracted in the AVS extraction procedure) minus the molar concentration of AVS accurately predicts which sediments are not toxic because of these metals. The use of (ΣSEM-AVS)/f_{OC} reduces variability associated with prediction of when sediments will be toxic.

EqP can be used to calculate ESBs for any toxicity endpoint for which there are water-only toxicity data; it is not limited to any single effect endpoint. For the purposes of this document, the ESB for mixtures of the metals cadmium, copper, lead, nickel, silver, and zinc is based on the solid phase and interstitial water phase of sediments. In sediments, these metals should not cause direct toxicity to benthic organisms if the ΣSEM-AVS is ≤ 0.0. Alternatively, sediments containing these metals should not cause direct toxicity to benthic organisms if the sum of the dissolved interstitial water concentrations for each of the metals ($\Sigma M_{i,d}$) divided by their respective water quality criteria final chronic value (FCV) is ≤ 1.0. Uncertainty bounds on ΣSEM-AVS and (ΣSEM-AVS)/f_{OC} can be used to identify sediments where toxicity, because of these metals, is unlikely, uncertain, or likely. If an FCV is not available, a secondary chronic value (SCV) can be substituted. Ancillary analyses conducted as part of this derivation suggest that the sensitivity of benthic/epibenthic organisms is not significantly different from pelagic organisms; for this reason, the FCV and the resulting $ESB_{AVS:WQC}$ should be fully applicable to benthic organisms. The $ESB_{AVS:WQC}$s should be interpreted as chemical concentrations below which adverse effects are not expected. At concentrations above the $ESB_{AVS:WQC}$s, effects may occur with increasing severity as the degree of exceedance increases. In principle, above the upper confidence limit effects are expected if the chemical is bioavailable as predicted by EqP theory. A sediment-specific site assessment would provide further information on chemical bioavailability and the expectation of toxicity relative to the $ESB_{AVS:WQC}$s and associated uncertainty limits. An appendix addresing chromium toxicity in sediments is also included in this document.

As discussed, while this document uses the WQC or AVS values, the EqP methodology can be used by environmental managers to derive a benchmark with any desired level of protection, so long as the water-only concentration affording that level of protection is known. Therefore, the resulting benchmark can be species or site-specific if the corresponding water-only information is available. For example, if a certain water-only effects concentration is known to be

economically important benthic species, the FCV or SCV for that benthic species could be used to derive the benchmark. Such a benchmark might be considered as providing "site-specific protection" for a species or endpoint, if the goal is to derive a benchmark for that particular site or species. Another way to make an ESB site-specific would be to incorporate information on unusual partitioning, if suspected, at the site (see U.S. EPA 2003b).

The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans. Consistent with the recommendations of EPA's Science Advisory Board, publication of these documents does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, ESB exceedances could be used to trigger the collection of additional assessment data. When using the AVS approach, the $ESB_{AVS:WQC}$ applies to sediments having AVS concentrations $\geq$ 0.1 µmol/g.

Tier 1 and Tier 2 ESB values were developed to reflect differing degrees of data availability and uncertainty. Tier 1 ESBs have been derived for metal mixtures in this document, and for the nonionic organic insecticides endrin and dieldrin, and polycyclic aromatic hydrocarbon (PAH) mixtures in U.S. EPA (2003c, d, e). Tier 2 ESBs are reported in U.S. EPA (2003f).

Glossary of Abbreviations

Ag	Silver
Ag_2S	Silver monosulfide
AVS	Acid volatile sulfide
CCC	Criteria continuous concentration
Cd	Cadmium
$\{Cd^{2+}\}$	Activity of ionic cadmium (mol/L)
$[Cd^{2+}]$	Concentration of ionic cadmium (mol/L)
$[Cd]_A$	Concentration of added cadmium (mol/L)
$[Cd]_B$	Concentration of bound cadmium (mol/L)
[CdS(s)]	Concentration of solid-phase cadmium sulfide (mol/L)
Cr	Chromium
C_S	Concentration of contaminant in sediment
C_s^*	Sediment LC50 Concentration
Cu	Copper
CWA	Clean Water Act
DOC	Dissolved organic carbon
EDTA	Ethlyenediaminetetra-acetic acid
EMAP	Environmental Monitoring and Assessment Program
EPA	U.S. Environmental Protection Agency
EqP	Equilibrium partitioning
ESB(s)	Equilibrium partitioning sediment benchmark(s)
$ESB_{AVS:WQC}$	Equilibrium partitioning sediment benchmark(s) for metal mixtures based on the Water Quality Criteria Final Chronic Values or Acid Volatile Sulfide
f_{OC}	Fraction of organic carbon in sediment
FCV	Final chronic value
Fe	Iron
$\{Fe^{2+}\}$	Activity of ionic iron (mol/L)
$[Fe^{2+}]$	Concentration of ionic iron (mol/L)
[FeS(s)]	Concentration of solid-phase iron sulfide (mol/L)

$[FeS(s)]_i$	Concentration of initial solid-phase iron sulfide (mol/L)
FeS	Iron monosulfide
GFAA	Gas Furnace Atomic Absorption Spectrophotometry
HECD	U.S. EPA, Health and Ecological Criteria Division
IW	Interstitial water
IWBU	Interstitial water benchmarks unit
IWTU	Interstitial water toxic unit
K_{FeS}	Solubility product for FeS(s) [$(mol/L)^2$]
K_{MS}	Solubility product for MS(s) [$(mol/L)^2$]
K_{OC}	Organic carbon–water partition coefficient
K_P	Sediment–interstitial water partition coefficient
K_{SP}	Solubility product constant
LC50	Concentration estimated to be lethal to 50% of the test organisms within a specified time period
M^{2+}	Divalent metal—cadmium, copper, lead, nickel, silver, or zinc
MOH^+	Metal hydroxide
MS	Metal sulfide
Mn	Manganese
$\{M^{2+}\}$	Divalent metal activity (mol/L)
$[M^{2+}]$	Concentration of ionic metal (mol/L)
$[M]_A$	Concentration of added metal (mol/L)
$[M]_B$	Concentration of bound metal (mol/L)
$[M_d]$	Dissolved metal concentration in the interstitial water
[MS(s)]	Concentration of solid-phase metal sulfide (mol/L)
$[M_T]$	Total cold extractable metal (mol/L)
NA	Not applicable, not available
NAS	National Academy of Sciences
Ni	Nickel
NOAA	National Oceanographic and Atmospheric Administration
NOEC	No observed effect concentration
NST	National Status and Trends monitoring program
NTA	Nitrilotriacetic acid

NTIS	National Technical Information Service
Pb	Lead
OEC	Observed effect concentration
ORD	U.S. EPA, Office of Research and Development
OST	U.S. EPA, Office of Science and Technology
POC	Particulate organic carbon
REMAP	Regional Environmental Monitoring and Assessment Program
S^{2-}	Sulfide ion
$\{S^{2-}\}$	Activity of sulfide (mol/L)
$[S^{2-}]$	Concentration of sulfide (mol/L)
SAB	U.S. EPA Science Advisory Board
SD	Standard deviation
SEM	Simultaneously extracted metals
$[SEM_T]$	Simultaneously extracted metals, concentration of the combined metals (μmol/g)
$[SEM_{Cd}]$	Simultaneously extracted metals, Cd concentration (μmol/g)
$[SEM_{Cu}]$	Simultaneously extracted metals, Cu concentration (μmol/g)
$[SEM_{Pb}]$	Simultaneously extracted metals, Pb concentration (μmol/g)
$[SEM_{Ni}]$	Simultaneously extracted metals, Ni concentration (μmol/g)
$[SEM_{Ag}]$	Simultaneously extracted metals, Ag concentration (μmol/g)
$[SEM_{Zn}]$	Simultaneously extracted metals, Zn concentration (μmol/g)
TIE	Toxicity identification evaluation
TOC	Total organic carbon
WQC	Water quality criteria
Zn	Zinc
$[\Sigma Cd(aq)]$	Concentration of total dissolved Cd^{2+} (mol/L)
$[\Sigma Fe(aq)]$	Concentration of total dissolved Fe^{2+} (mol/L)
$[\Sigma M(aq)]$	Concentration of total dissolved M^{2+} (mol/L)
$[\Sigma S(aq)]$	Concentration of total dissolved S^{2-} (mol/L)

Section 1

Introduction

1.1 General Information

Toxic pollutants in bottom sediments of the Nation's lakes, rivers, wetlands, estuaries, and marine coastal waters create the potential for continued environmental degradation even where water column concentrations comply with established WQC. In addition, contaminated sediments can be a significant pollutant source that may cause water quality degradation to persist, even when other pollutant sources are stopped (Larsson, 1985; Salomons et al., 1987; Burgess and Scott, 1992). The absence of defensible equilibrium partitioning sediment benchmarks (ESBs) make it difficult to accurately assess the extent of the ecological risks of contaminated sediments and to identify, prioritize, and implement appropriate cleanup activities and source controls (U.S. EPA 1997a, b, c).

As a result of the need for a procedure to assist regulatory agencies in making decisions concerning contaminated sediment problems, the U.S. Environmental Protection Agency (EPA) Office of Science and Technology, Health and Ecological Criteria Division (OST/HECD) and Office of Research and Development National Health and Environmental Effects Research Laboratory (ORD/NHEERL) established a research team to review alternative approaches (Chapman, 1987). All of the approaches reviewed had both strengths and weaknesses, and no single approach was found to be applicable for the derivation of benchmarks in all situations (U.S. EPA, 1989, 1992). The equilibrium partitioning (EqP) approach was selected for nonionic organic chemicals because it presented the greatest promise for generating defensible, national, numeric chemical-specific benchmarks applicable across a broad range of sediment types. The three principal observations that underlie the EqP approach to establishing sediment benchmarks are as follows:

1. The concentrations of nonionic organic chemicals in sediments, expressed on an organic carbon basis, and in interstitial waters correlate to observed biological effects on sediment-dwelling organisms across a range of sediments.

2. Partitioning models can relate sediment concentrations for nonionic organic chemicals on an organic carbon basis to freely-dissolved concentrations in interstitial water.

3. The distribution of sensitivities of benthic organisms to chemicals is similar to that of water column organisms; thus, the currently established water quality criteria (WQC) final chronic values (FCV) or secondary chronic values (SCV) can be used to define the acceptable effects concentration of a chemical freely-dissolved in interstitial water.

Because of their widespread release and persistent nature, metals such as cadmium, copper, lead, nickel, silver, and zinc are commonly elevated in aquatic sediments. These metals, in addition to nonionic organic chemicals, are of potential concern to aquatic environments. Thus, there have been various proposals for deriving sediment benchmarks for protecting benthic communities using measurement of total sediment metals followed by comparison with background metal concentrations, or in some cases, an effects-based endpoint (Sullivan et al., 1985; Persaud et al., 1989; Long and Morgan, 1990; Ingersoll et al., 1996; MacDonald et al., 1996). An important limitation to these types of approaches is that the causal linkage between the measured concentration of metals and the observed toxicity cannot be established, in part because of the procedures used to derive correlative values, and because values derived are based on total rather than bioavailable metal concentrations. That is, for any given total metal concentration, adverse toxicological effects may or may not occur, depending on the physicochemical characteristics of the sediment of concern (Tessier and Campbell, 1987; Luoma, 1989; Di Toro et al., 1990).

Many researchers have used elaborate sequential extraction procedures to identify sedimentary physicochemical fractions with which metals are associated in an attempt to understand the biological availability of metals in sediments (Tessier et al., 1979; Luoma and Bryan, 1981). Key binding phases for metals in sediments included iron and manganese oxides and organic carbon. Shortcomings with these approaches have limited their application largely to aerobic sediments instead of anaerobic sediments, where metals are often found in the greatest concentrations (see Section 2).

In developing ESBs for metals that causally link metals concentrations to biological effects and that apply across all sediments, it is essential that bioavailability be understood. Therefore, the EqP approach was selected as the technical basis for deriving ESBs for metals. Different studies have shown that although total (dry weight) metal concentrations in anaerobic sediments are not predictive of bioavailability, metal concentrations in interstitial water are correlated with observed biological effects (Swartz et al., 1985; Kemp and Swartz, 1986). However, as opposed to the situation for nonionic organic chemicals and organic carbon (see Di Toro et al., 1991), sediment partitioning phases controlling interstitial water concentrations of metals were not readily apparent. A key partitioning phase controlling cationic metal activity and metal-induced toxicity in the sediment–interstitial water system is acid volatile sulfide (AVS) (Di Toro et al., 1990, 1992). AVS binds, on a molar basis, a number of cationic metals of environmental concern (cadmium, copper, lead, nickel, silver, and zinc), forming insoluble sulfide complexes with minimal biological availability. (Hereafter in this document, the use of the term "metals" will apply only to these six metals.)

The data that support the EqP approach for deriving sediment benthmarks for nonionic organic chemicals were reviewed by Di Toro et al. (1991) and U.S. EPA (1997a; 2003a). The utility of the EqP approach for deriving sediment benchmarks for metals (U.S. EPA, 1994a) was reviewed and endorsed by EPA's Science Advisory Board (SAB) in 1994 and 1999 (U.S. EPA, 1995a, 1999). The data that support the EqP approach for deriving sediment benchmarks for metals presented in this document were taken largely from a series of papers published in the December 1996 issue of *Environmental Toxicology and Chemistry* by Ankley et al. (1996), Berry et al. (1996), DeWitt et al. (1996), Di Toro et al. (1996a,b), Hansen et al. (1996a,b), Leonard et al. (1996a), Liber et al. (1996), Mahony et al. (1996), Peterson et al. (1996), and Sibley et al. (1996). In addition, publications by Di Toro et al. (1990, 1992), Ankley et al. (1994), U.S. EPA (1995a), and Berry et al. (1999) were of particular importance in the preparation of this document.

The same three general principles observed in applying the EqP approach to nonionic organic chemicals listed above also apply with only minor adjustments to deriving ESBs for mixtures of the cationic metals—cadmium, copper, lead, nickel, silver, and zinc:

1. The concentrations of these six metals in sediments, normalized to the concentration of AVS and simultaneously extracted metals (SEM) (the metals extracted with AVS) in sediments and dissolved in interstitial waters, correlate with observed biological effects to sediment-dwelling organisms across a range of sediments (Di Toro et al., 1992).

2. Partitioning models can relate sediment concentrations for cationic divalent metals (and monovalent silver) on an AVS basis to the absence of freely-dissolved concentrations in interstitial water.

3. The distributions of sensitivities of benthic and water column organisms to organic chemicals and metals are similar (U.S. EPA, 2003a); thus, the currently established WQC FCVs can be used to define the acceptable effects concentration of the metals freely dissolved in interstitial water.

The EqP approach, therefore, assumes that (1) the partitioning of the metal between sediment AVS (or any other binding factors controlling

bioavailability) and interstitial water approximates equilibrium; (2) organisms receive equivalent exposure from interstitial water–only exposure or from exposure to any other equilibrated sediment phase: either from interstitial water via respiration, sediment via ingestion, or sediment-integument exchange, or from a mixture of exposure routes; (3) for the cationic metals cadmium, copper, lead, nickel, zinc, and silver, partitioning of metal between the solid phase and interstitial water can be predicted based on the relative concentrations of AVS and SEM; (4) the WQC FCV concentration is an appropriate effects concentration for freely-dissolved metal in interstitial water; and (5) the toxicity of metals in interstitial water is no more than additive.

For the first time, the Agency is publishing ESBs that account for bioavailability in sediments and the potential for effects of a metal mixture in the aquatic environment, thus providing an ecologically relevant benchmark. Two equally applicable ESBs for metals, a solid phase and an interstitial water phase, are described. The solid-phase AVS ESBs is defined as the $\Sigma_i[SEM_i] \leq [AVS]$ (total molar concentration of simultaneously extracted metal is less than or equal to the total molar concentration of acid volatile sulfide). Note that cadmium, copper, lead, nickel, and zinc are divalent metals so that one mole of each metal can bind only with one mole of AVS. The molar concentrations of these metals are compared with AVS on a one-to-one basis. Silver, however, exists predominantly as a monovalent metal, so that silver monosulfide (Ag_2S) binds two moles of silver for each mole of AVS. Therefore, SEM_{Ag} by convention will be defined as the molar concentration of silver divided by two, [Ag]/2, which is compared with the molar AVS concentration. The interstitial water phase ESB is $\Sigma[M_{i,d}]/[FCV_{i,d}] \leq 1$ (the sum of cadmium, copper, nickel, lead, and zinc of the concentration of each individual metal dissolved in the interstitial water divided by the metal-specific FCV based on dissolved metal is less than or equal to one; note that at present EPA does not have an FCV for silver). This latter value is termed an interstitial water benchmark unit (IWBU). A requirement of the IWBUapproach is that the toxicities of interstitial water metal concentrations be additive. The data presented in this document support the additivity of the toxicity of metal mixtures in water.

Importantly, both the solid-phase AVS ESB and interstitial water ESB are no-effect benchmarks; that is, they predict sediments that are acceptable for the protection of benthic organisms. These ESBs, when exceeded, do not unequivocally predict sediments that are unacceptable for the protection of benthic organisms. The solid-phase AVS benchmark avoids the methodological difficulties of interstitial water sampling that may lead to an overestimate of exposure and provides information on the potential for additional metal binding. Because the AVS benchmark does not include other metal-binding phases of sediments, the interstitial benchmark is also proposed. The use of both the AVS and interstitial water benchmarks will improve estimates of risks of sediment-associated metals. For example, the absence of significant concentrations of metal in interstitial water in toxic sediments having SEM≤AVS and in nontoxic sediments having SEM>AVS demonstrates that metals in these sediments are unavailable. The $(\Sigma SEM\text{-}AVS)/f_{OC}$ correction, although not an ESB, can be used to refine the prediction of sediments where protection of benthic organisms is acceptable, uncertain, or unacceptable.

ESBs based on the EqP approach are developed using the latest available scientific data and are suitable for providing guidance to regulatory agencies because they are

- Numeric values
- Chemical-specific
- Applicable to most sediments
- Predictive of biological effects
- Protective of benthic organisms

It should be emphasized that these benchmarks are intended to protect benthic organisms from the direct effects of these six metals in sediments that are permanently inundated with water, intertidal, or inundated periodically for durations sufficient to permit

development of benthic assemblages. They do not apply to occasionally inundated soils containing terrestrial organisms. The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans. The ESBs presented in this document are the recommended concentrations of cadmium, copper, lead, nickel, silver, and zinc in sediment that will not adversely affect most benthic organisms. ESB values may be adjusted to account for future data or site-specific considerations (U.S. EPA, 2003b).

This document includes the theoretical basis and the supporting data relevant to the derivation of an ESBfor cadmium, copper, lead, nickel, silver, and zinc and their mixture. An understanding of the "Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses" (Stephan et al., 1985); Response to Public Comment (U.S. EPA, 1985a); "Ambient Water Quality Criteria for Cadmium" (U.S. EPA, 1985b); "Ambient Water Quality Criteria for Copper" (U.S. EPA, 1985c); "Ambient Water Quality Criteria—Saltwater Copper Addendum" (U.S. EPA, 1995c); "Ambient Water Quality Criteria for Lead" (U.S. EPA, 1985d); "Ambient Water Quality Criteria for Nickel" (U.S. EPA, 1986); "Ambient Water Quality Criteria for Silver" (U.S. EPA, 1980); and "Ambient Water Quality Criteria for Zinc" (U.S. EPA, 1987) is necessary in order to understand the following text, tables, and calculations.

1.2 Applications of Sediment Benchmarks

ESBs are meant to be used with direct toxicity testing of sediments as a method of evaluation assuming the toxicity testing species is sensitive to the chemical of interest. They provide a chemical-by-chemical specification of what sediment concentrations are protective of benthic aquatic life. The EqP method should be applicable to nonionic organic chemicals with a K_{OW} above 3.0. Examples of other chemicals to which this methodology applies include endrin, dieldrin, and polycyclic aromatic hydrocarbon (PAH) mixtures.

For the toxic chemicals addressed by the ESB documents Tier 1 (U.S. EPA, 2003c, d, e, and this document) and Tier 2 (U.S. EPA, 2003f) values were developed to reflect the differing degrees of data availability and uncertainty. Tier 1 ESBs are more scientifically rigorous and data intensive than Tier 2 ESBs. The minimum requirements to derive a Tier 1 ESB include: (1) Each chemical's organic carbon-water partition coefficient (K_{OC}) is derived from the octanol-water partition coefficient (K_{OW}) obtained using the SPARC (SPARC Performs Automated Reasoning in Chemistry) model (Karickhoff et al., 1991) and the K_{OW}-K_{OC} relationship from Di Toro et al. (1991). This K_{OC} has been demonstrated to predict the toxic sediment concentration from the toxic water concentration with less uncertainty than K_{OC} values derived using other methods. (2) The FCV is updated using the most recent toxicological information and is based on the National WQC Guidelines (Stephan et al., 1985). (3) EqP-confirmation tests are conducted to demonstrate the accuracy of the EqP prediction that the K_{OC} multiplied by the effect concentration from a water-only toxicity test predicts the effect concentration from sediment tests (Swartz, 1991; DeWitt et al., 1992). Using these specifications, Tier 1 ESBs have been derived for metal mixtures in this document, the nonionic organic insecticides endrin and dieldrin (U.S. EPA, 2003c, d) and PAH mixtures (U.S. EPA, 2003e). In comparison, the minimum requirements for a Tier 2 ESB (U.S. EPA, 2003f) are less rigorous: (1) The K_{ow} for the chemical that is used to derive the K_{OC} can be from slow-stir, generator column, shake flask, SPARC or other sources. (2) FCVs can be from published or draft WQC documents, the Great Lakes Initiative or developed from AQUIRE. Secondary chronic values (SCV) from Suter and Mabrey (1994) or other effects concentrations from water-only toxicity tests can be used. (3) EqP cconfirmation tests are recommended, but are not required for the development of Tier 2 ESBs. Because of these lesser requirements, there is greater uncertainty in the EqP prediction of the sediment effect concentration from the water-only effect concentration, and in the level of protection afforded by Tier 2 ESBs. Examples of Tier 2 ESBs for nonionic organic chemicals are found in U.S. EPA (2003f).

1.3 Overview

Section 1 provides a brief review of the EqP methodology as it applies to the individual metals cadmium, copper, lead, nickel, silver, and zinc and their mixture. Section 2 reviews published experimental results that describe the toxicity associated with the partitioning and bioavailability of these metals in interstitial water of freshwater and marine sediments. Section 3 reviews the results of acute and chronic toxicity tests conducted with spiked and field sediments that demonstrate that the partitioning and bioavailability of metals in sediments can be used to accurately predict the absence of toxicity of sediment-associated metals. Section 4 describes the AVS benchmark and interstitial water benchmark approaches for the derivation of the ESB for individual metals and mixtures of metals. Published WQC values for five of these six dissolved metals (the silver FCV is not available) are summarized for use in calculating IWBUs as required in the interstitial water ESB approach. The $ESB_{AVS:WQC}$ for metals is then compared with chemical monitoring data on environmental occurrence of SEM, AVS, and interstitial metals in sediments from Lake Michigan, the Virginian Province from EPA's Environmental Monitoring and Assessment Program (EMAP), and the National Oceanic and Atmospheric Administration (NOAA) National Status and Trends monitoring program (NST). Section 5 describes recommended procedures for sampling, handling, and analysis of metals in sediments and interpretation of data from the sediment samples that is needed if the assessments of risks of sediment-associated metals are to be appropriately based on the EqP methodology. Section 6 concludes with the $ESB_{AVS:WQC}$ for a mixture of the metals: cadmium, copper, nickel, lead, silver, and zinc and discussion of their application and interpretation. The references cited in this document are listed in Section 7. Appendices A and B provide additional monitoring data. Appendix C reports on quality assurance for this document and Appendix D addresses chromium toxicity in sediments.

Section 2

Partitioning of Metals in Sediments

2.1 Metal Toxicity in Water-Only and in Interstitial Water of Sediment Exposures

The EqP approach for establishing sediment benchmarks (i.e., ESBs) requires that the chemicals be measured in phases that relate to chemical activity in sediment. The information provided in this section demonstrates that biological effects correlate to metal activity. Also, it demonstrates that biological response in sediment exposures is the same as in water-only exposures when sediment exposure is assessed on the basis of interstitial water concentrations. This is fundamental to satisfying the EqP approach for both metals and nonionic organic chemicals.

A direct method for establishing sediment benchmarks for metals would be to apply the WQC FCV to measured interstitial water concentrations. The validity of this approach depends both on the degree to which the interstitial water concentration represents free metal activity, and on whether free metal activity can be accurately measured in surface waters and water-only toxicity tests used to derive WQC, and in interstitial water of field sediments and sediments spiked with metals in the laboratory. For most metals, free metal activity cannot be directly measured at WQC concentrations. Therefore, present WQC are not based on free metal activity; rather, they are based on dissolved metals. However, many dissolved metals readily bind to dissolved (actually colloidal) organic carbon (DOC) forming complexes that do not appear to be bioavailable (Bergman and Dorward-King, 1997). Hence, sediment guidelines or benchmarks based on interstitial water concentrations of metals may be overly protective in cases where not all dissolved metal is bioavailable.

By implication, this difficulty extends to any complexing ligand that is present in sufficient quantity. Decay of sediment organic matter can cause substantial changes in interstitial water chemistry. In particular, bicarbonate increases because of sulfate reduction, which increases the importance of metal-carbonate complexes and further complicates the question of the bioavailable metal species (Stumm and Morgan, 1996).

Sampling sediment interstitial water for metals is not a routine procedure. The least invasive technique employs a diffusion sampler that has cavities covered with a filter membrane (Hesslein, 1976; Carignan, 1984; Carignan et al., 1985; Allen et al., 1993; Bufflap and Allen, 1995). The sampler is inserted into the sediment and the concentrations on either side of the membrane equilibrate. Because the sampler is removed after equilibration, the concentrations of metals inside the sampler should be equal to the concentrations of freely-dissolved metals in the interstitial water. The time required for equilibration, typically several days, depends on the size of the filter membrane and the geometry of the cavity.

An alternative technique for separating interstitial water is to obtain an undisturbed sediment sample as a whole sediment or core that can be sliced for vertical resolution, filter or centrifuge the sample, and then filter the resultant interstitial water twice. For anaerobic sediments, this must be done in a nitrogen atmosphere to prevent precipitation of iron hydroxide, which would scavenge the metals and yield artificially low dissolved concentrations of metals (Troup, 1974; Allen et al., 1993).

Although either technique is suitable for research investigations, they require more than the normally available sampling capabilities. If solid-phase chemical measurements were available

from which interstitial water metal activity could be deduced, this would obviate the need for interstitial water sampling and analysis, circumvent the need to deal with complexing ligands, and provide fundamental insight into metal-binding phases in sediments needed to predict bioavailability. The recommended procedures for suitable sampling, handling, and analytical techniques for interstitial water and sediments are provided in Section 5 of this document.

2.1.1 *Toxicity Correlates to Metal Activity*

A substantial number of water-only exposures indicate that biological effects can be correlated to divalent metal activity $\{M^{2+}\}$. Although other forms of metal may also be bioavailable (e.g., MOH^{+}), DOC and certain other ligand-complexed fractions of the metal render it unavailable to organisms. Results from some of these exposures are summarized below.

Acute toxicity of various concentrations of cadmium to grass shrimp (*Palaemonetes pugio*) has been determined in water containing the complexing ligand nitrilotriacetic acid (NTA) or chloride (as salinity), each of which forms cadmium complexes (Sunda et al., 1978). The concentration response curves as a function of total cadmium are quite different at varying concentrations of NTA and chloride (Figure 2-1, A and B). However, if the organism response is evaluated with respect to measured Cd^{2+} activity, a single concentration–response relationship results (Figure 2-1, C and D). Comparable results have been reported by Anderson and Morel (1978) for the dinoflagellate *Gonyaulax tamarensis* exposed to copper-ethylene diamine tetra-acetic acid (EDTA) complexes (Figure 2-2, A and C). Likewise, Allen et al. (1980) observed that when the concentration of zinc is held constant and the concentration of the complexing ligand NTA is varied, growth (cells/mL) of *Microcystis aeruginosa* decreases as the addition of NTA is

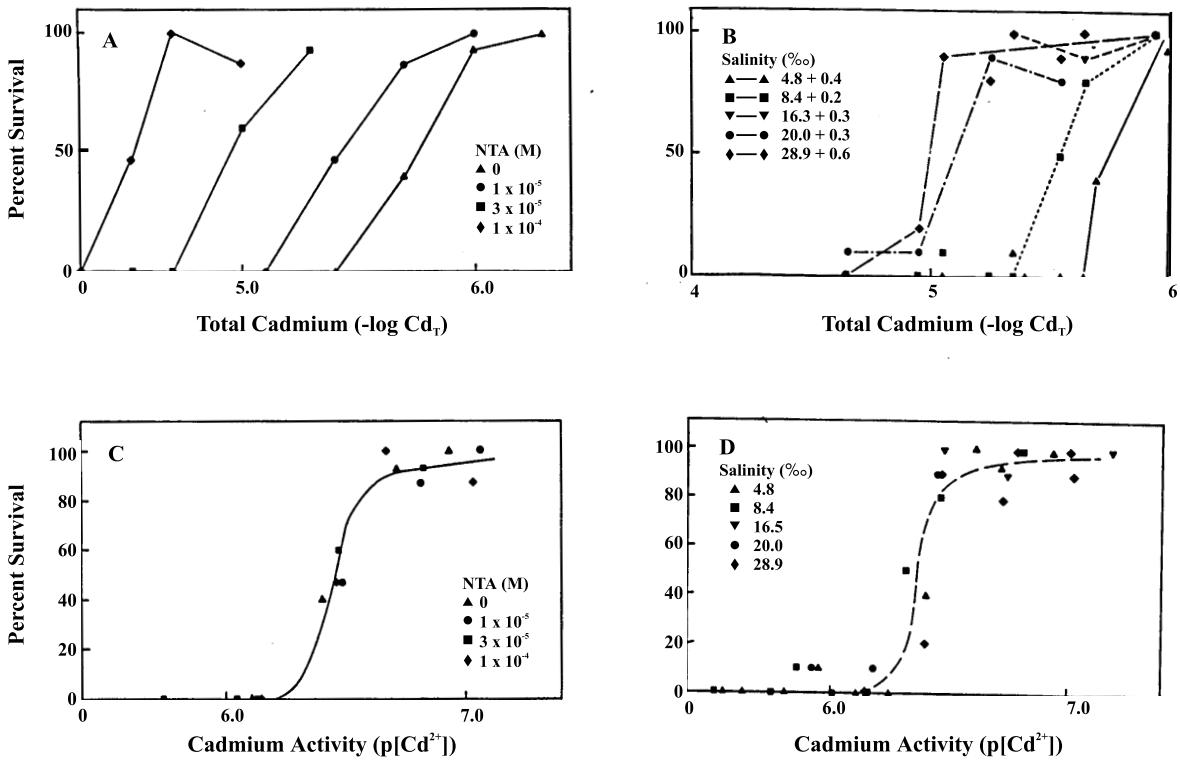

Figure 2-1. Acute toxicity to grass shrimp (*Palaemonetes pugio*) of total cadmium (top) and cadmium activity (bottom) with different concentrations of the complexing ligands NTA (left) and chloride as salinity (right) (figures from Sunda et al., 1978).

increased (Figure 2-2B). The authors correlated the effect to free zinc activity as shown in Figure 2-2D. A single concentration–response relationship is shown for the diatom, *Thalassiosira pseudonana*, and the unicellular alga, *Monochrysis lutheri*, exposed to copper and the complexing ligand Tris (Sunda and Guillard, 1976) as well as copper and DOC from natural river water (Sunda and Lewis, 1978) when exposure concentration is expressed as metal activity (Figure 2-3, A, B, C, and D, respectively).

Metal bioavailability, as measured by metal accumulation into tissues of organisms, has also

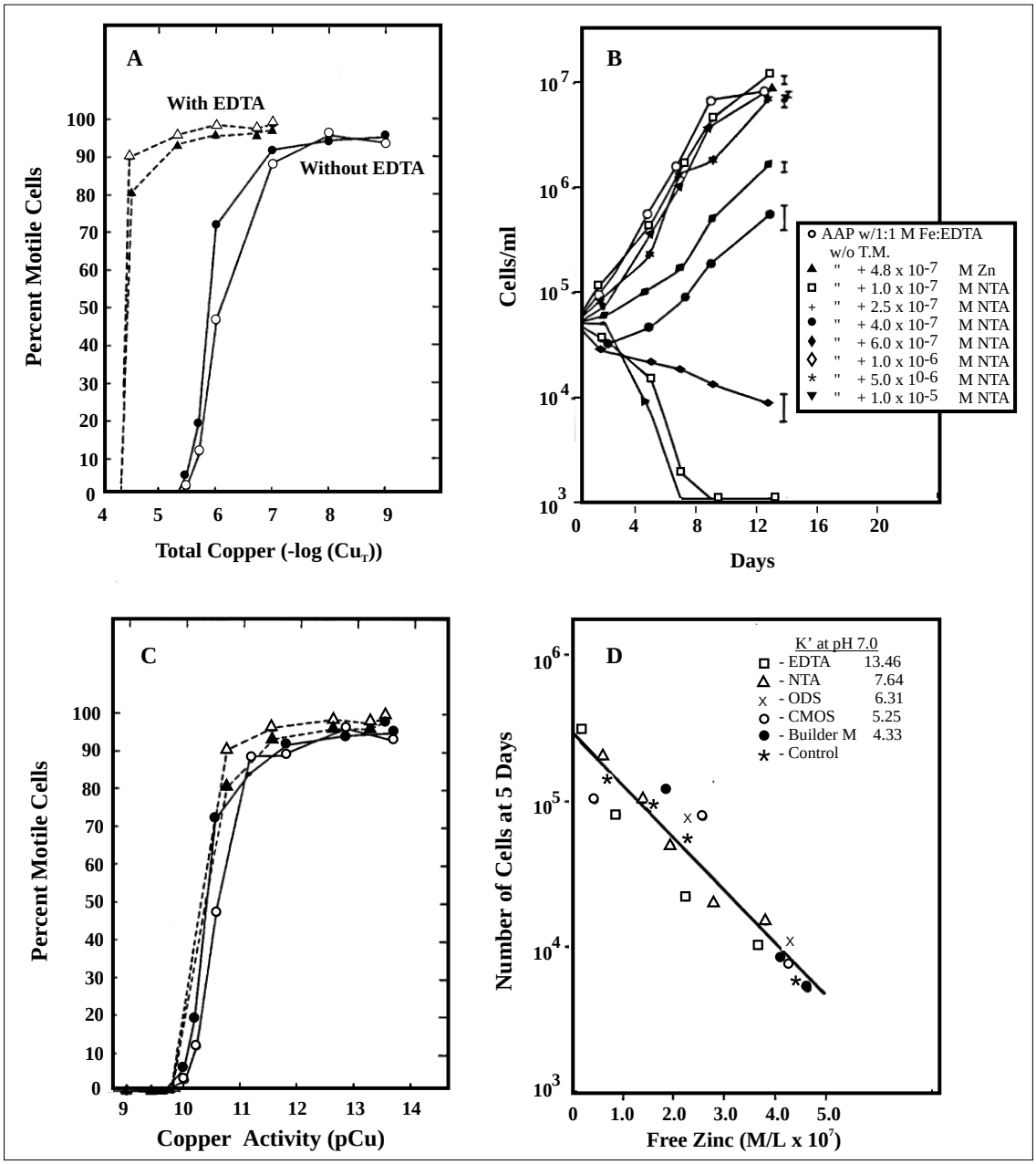

Figure 2-2. Acute toxicity of total copper (A) and copper activity (C) to the dinoflagellate *Gonyaulax tamarensis* with and without the complexing ligand EDTA (figures from Anderson and Morel, 1978). Toxicity of zinc to *Microcystis aeruginosa* showing growth of cells/mL versus time with different levels of the complexing ligands EDTA and NTA (B) and number of cells at 5 days as a function of free zinc concentration (D) (figures from Allen et al., 1980).

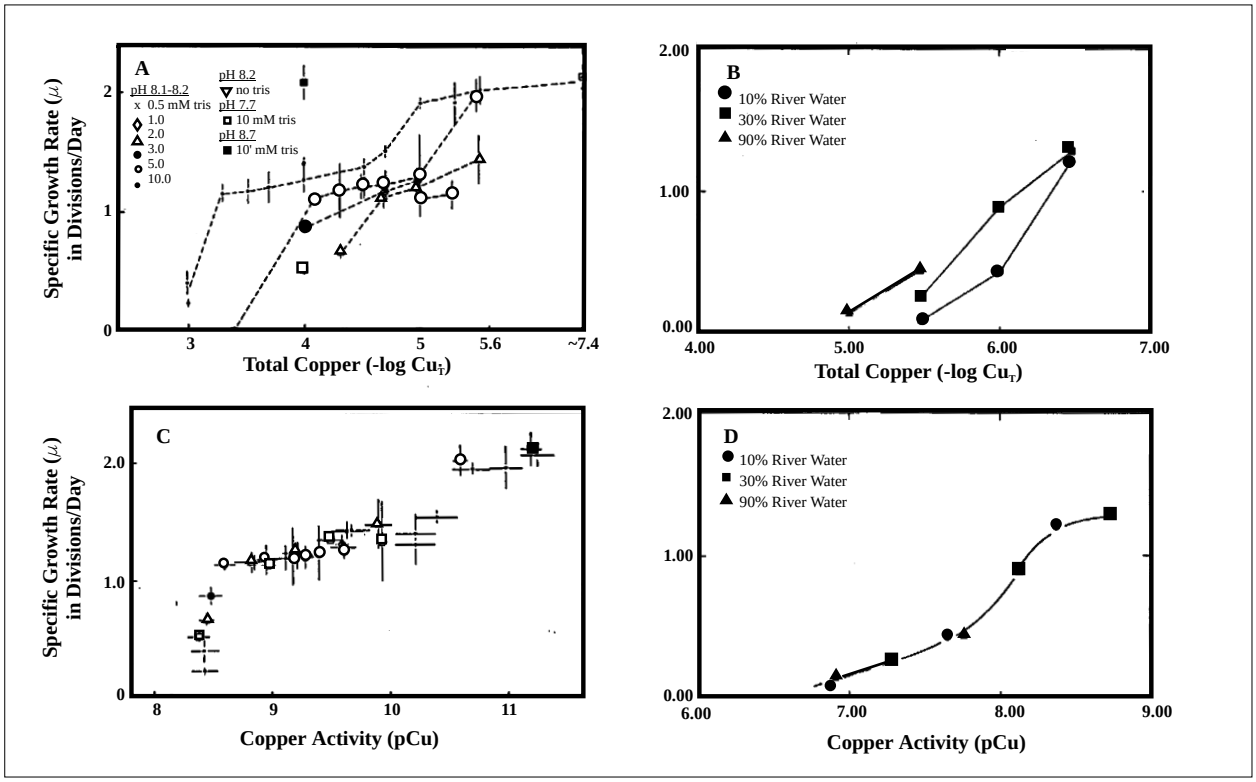

Figure 2-3. Specific growth rates of a diatom (*Thalassiosira pseudonana*) (left) and a unicellular algae (*Monochrysis lutheri*) (right) versus total copper (top) and copper activity (bottom) for a range of concentrations of the complexing ligands Tris (left; from Sunda and Guillard, 1976) and natural DOC in river water (right; from Sunda and Lewis, 1978).

been examined (Zamuda and Sunda, 1982). Uptake of copper by oysters is correlated not to total copper concentration (Figure 2-4A), but to copper activity (Figure 2-4B).

The implication to be drawn from these experiments is that the partitioning model required for establishing a sediment benchmark should predict dissolved metal activity in interstitial water, and that the benchmark based on dissolved metal would be conservative. The following subsection examines the utility of this idea.

2.1.2 *Toxicity Correlates to Interstitial Water Concentration*

This subsection presents early data that first indicated the equivalence of interstitial water concentrations and water-only exposures. Many more data of this sort are presented in Section 3. Swartz et al. (1985) tested the acute toxicity of cadmium to the marine amphipod *Rhepoxynius abronius* in sediment and water. An objective of the study was to determine the contributions of interstitial and particle-bound cadmium to toxicity. A comparison of the 4-day LC50 value of cadmium in interstitial water (1.42 mg/L) with the 4-day LC50 value of cadmium in water without sediment (1.61 mg/L) indicated no significant difference between the two (Figure 2-5). The LC50 represents the chemical concentration estimated to cause lethality to 50% of the test organisms within a specified time period.

Experiments were performed to determine the role of AVS in cadmium-spiked sediments using the amphipods *Ampelisca abdita* and *Rhepoxynius hudsoni* (Di Toro et al., 1990). Three sediments were used: a Long Island Sound sediment with high AVS, a Ninigret Pond sediment with low AVS concentration, and a 50/50 mixture of the two sediments Figure 2-6 presents a

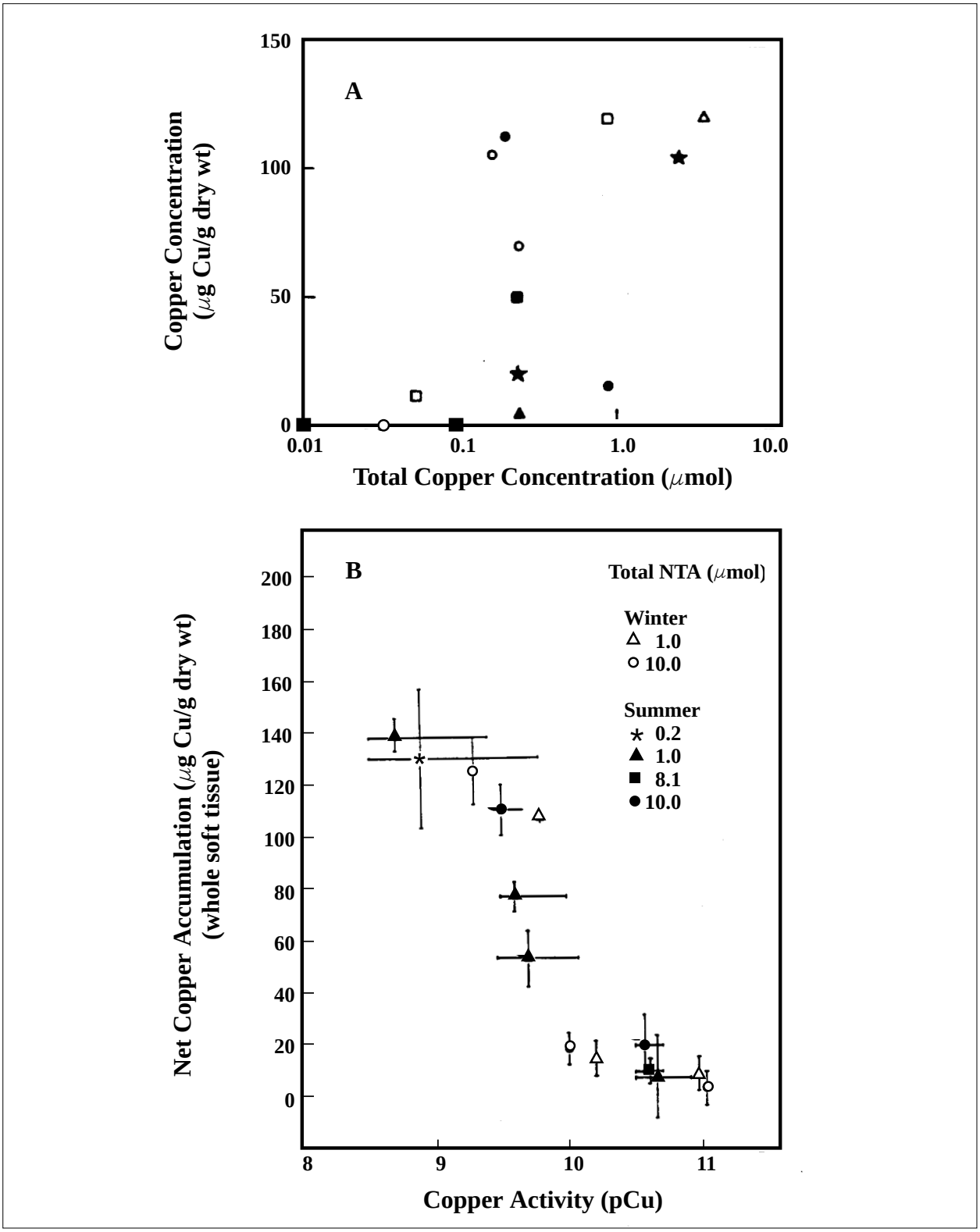

Figure 2-4. Copper accumulation in oysters (*Crassostrea virginica*) versus total copper (A) and copper activity (B) with different levels of the complexing ligand NTA (figures from Zamuda and Sunda, 1982).

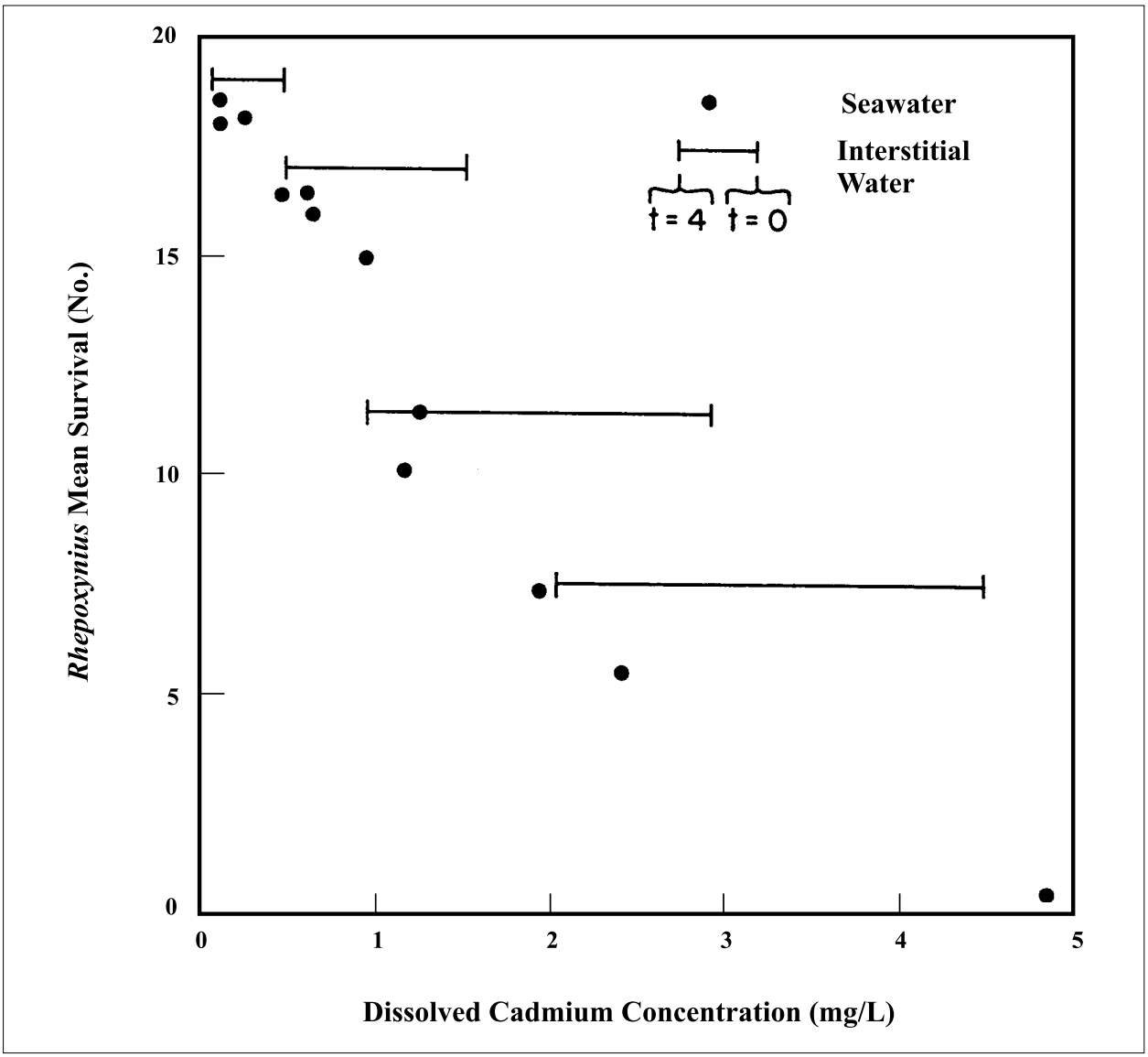

Figure 2-5. Mean survival of the amphipod *Rhepoxynius abronius* versus dissolved cadmium concentration for 4-day toxicity tests in seawater (symbols) and 0- and 4-day tests (bars) in interstitial water (figure from Swartz et al., 1985).

comparison of the observed mortality in three sediments with the interstitial water cadmium activity measured with a specific ion electrode. Four-day water-only and 10-day sediment toxicity tests were performed. The water-only response data for *A. abdita* and *R. hudsoni* are included for comparison although these data represent a shorter duration exposure. These experiments also demonstrate the equivalence of organism response to metal concentrations in interstitial water and in water-only exposures.

An elegant experimental design was employed by Kemp and Swartz (1986) to examine the relative acute toxicity of particle-bound and dissolved interstitial cadmium. They circulated water of the same cadmium concentration through different sediments. This resulted in different bulk sediment concentrations, but the same interstitial water concentrations. They found no statistically significant difference in organism response for the different sediments. Because the interstitial water concentrations were the same in each treatment,

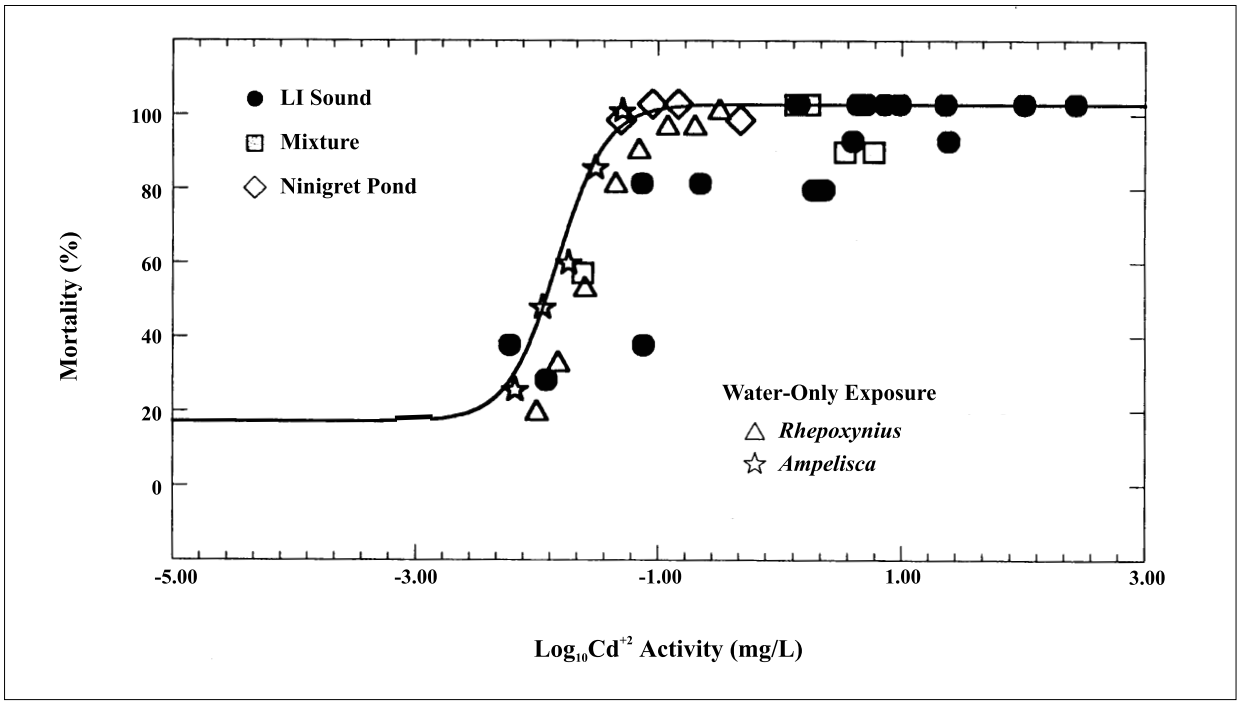

Figure 2-6. **Mortality versus interstitial water cadmium activity for sediments from Long Island Sound, Ninigret Pond, and a mixture of these two sediments. Water-only exposure data are from separate experiments with both *Ampelisca abdita* and *Rhepoxynius hudsoni*. The line is a joint fit to both water-only data sets (figure from Di Toro et al., 1990).**

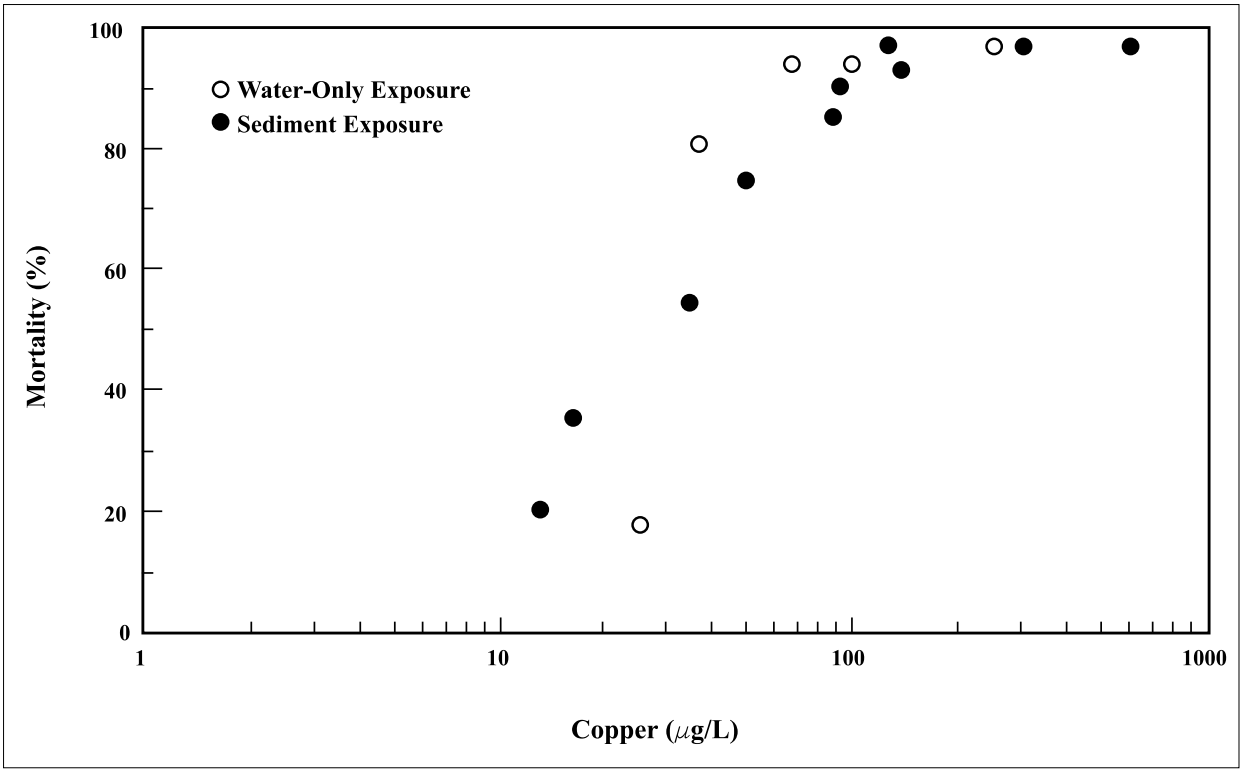

Figure 2-7. **Toxicity of copper to *Hyalella azteca* versus copper concentrations in a water-only exposure (○) and interstitial water copper concentrations in sediment exposures (●) using Keweenaw Watershed sediments (figure from Ankley et al., 1993).**

that is, the circulating water concentrations established the interstitial water concentrations, these experiments confirmed the hypothesis of equal response to concentrations in water-only and interstitial water.

A series of 10-day toxicity tests using the amphipod *Hyalella azteca* was performed to evaluate bioavailability of copper in sediments from two sites highly contaminated with this metal: Steilacoom Lake, WA, and Keweenaw Watershed, MI (Ankley et al., 1993). A water-only, 10-day copper toxicity test also was conducted with the same organism. The mortality resulting from the water-only test was strikingly similar to that from the Keweenaw sediment tests when related to interstitial water (Figure 2-7). The LC50 values show strong agreement for the water-only (31 μg/L) and the Keweenaw sediment test (28 μg/L) using the average of day 0 and day 10 interstitial water concentrations. Steilacoom Lake 10-day interstitial water concentrations were less than the 7 μg/L detection limit and were consistent with the observed lack of toxicity to *H. azteca*.

The data presented in this subsection, and the data in Section 3, demonstrate that in water-only exposures, metal activity and concentration can be used to predict toxicity. The results of the four experiments above demonstrate that mortality data from water-only exposures can be used to predict sediment toxicity using interstitial water concentrations. Therefore, the metal activity or dissolved concentration in interstitial water would be an important component of a partitioning model needed to establish sediment benchmarks. To complete the partitioning model, one would need to identify the solid metal-binding phase(s). The following subsection presents data that identifies solid-phase sulfides as the important metal-binding phase.

2.2 Solid-Phase Sulfide as the Important Binding Component

Modeling metal sorption to oxides in laboratory systems is well developed, and detailed models are available for cation and anion sorption (see Stumm [1987] and Dzombak and Morel [1990] for summaries). The models consider surface complexation reactions as well as electrical interactions by means of models of the double layer. Models for natural soil and sediment particles are less well developed. However, studies suggest that the models available for cation and anion sorption can be applied to soil systems (Allen et al., 1980; Barrow and Ellis, 1986a,b,c; Sposito et al., 1988). Because the ability to predict partition coefficients is required if interstitial water metal concentrations are to be inferred from the total concentration, some practical model is required. This subsection presents the state of the science in theoretical development of metal partitioning behavior in sediments.

2.2.1 *Metal Sorption Phases*

The initial difficulty selecting an applicable sorption model is that available models are complex and many of the parameter estimates may be specific to individual soils or sediments. However, the success of nonionic chemical sorption models based on organic carbon suggests that some model of intermediate complexity based on an identification of the dominant sorption phases may be more generally applicable.

A development in this direction has already been presented (Jenne et al., 1986; Di Toro et al., 1987). The basic idea was that instead of considering only one sorption phase, as is assumed for nonionic hydrophobic chemical sorption, multiple sorption phases must be considered. The conventional view of metal speciation in aerobic soils and sediments is that metals are associated with the exchangeable, carbonate and iron (Fe) and manganese (Mn) oxide forms, as well as organic matter, stable metal sulfides, and a residual phase. In oxic soils and freshwater sediments, sorption phases have been identified as particulate organic carbon (POC) and the oxides of Fe and Mn (Jenne, 1968, 1977; Oakley et al., 1980; Luoma and Bryan, 1981). These phases are important because they have a large sorptive capacity. Furthermore, they appear as coatings on the particles and occlude the other mineral

components. It was thought that they provided the primary sites for sorption of metals. These ideas have been applied to metal speciation in sediments. However, they ignore the critical importance of metal sulfide interactions, which dominate speciation in the anaerobic layers of the sediment.

2.2.2 *Titration Experiments*

The importance of sulfide in the control of metal concentrations in the interstitial water of marine sediments is well documented (Boulegue et al., 1982; Emerson et al., 1983; Davies-Colley et al., 1985; Morse et al., 1987). Metal sulfides are very insoluble, and the equilibrium interstitial water metal concentrations in the presence of sulfides are small. If the interstitial water sulfide

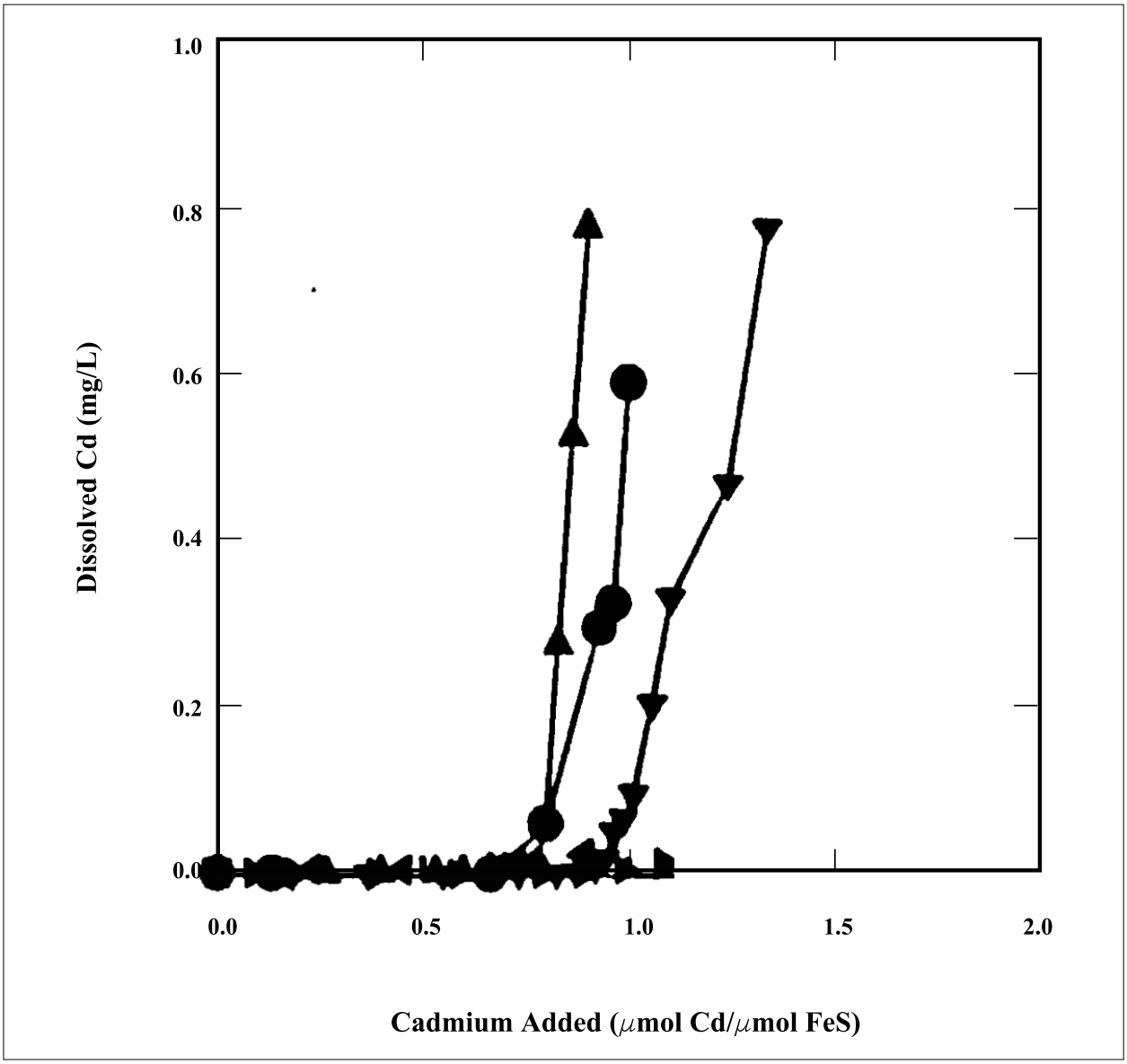

Figure 2-8. Cadmium titrations of amorphous FeS. The x-axis is the amount of cadmium added normalized by FeS initially present. The y-axis is total dissolved cadmium. The lines connecting the data points are an aid to visualizing the data. The different symbols represent replicate experiments (figure from Di Toro et al., 1990).

concentration, S^{2-}, in sediments is large, then the addition of metal, M^{2+}, to the sediment would precipitate metal sulfide (MS) following the reaction

$$M^{2+} + S^{2-} \rightleftharpoons MS(s) \qquad (2\text{-}1)$$

This appeared to be happening during a spiked cadmium sediment toxicity test (Di Toro et al., 1990) because a visible bright yellow cadmium sulfide precipitate formed as cadmium was added to the sediment. However, interstitial water sulfide activity, $\{S^{2-}\}$, measured with a sulfide electrode unexpectedly indicated that there was insufficient dissolved sulfide present in the unspiked sediment.

The lack of a significant quantity of dissolved sulfide in the interstitial water and the evident formation of solid-phase cadmium sulfide suggested the following possibility. The majority of the sulfide in sediments is in the form of solid-phase iron sulfides. Perhaps the source of the sulfide is from the solid-phase sulfide initially present. As cadmium is added to the sediment,

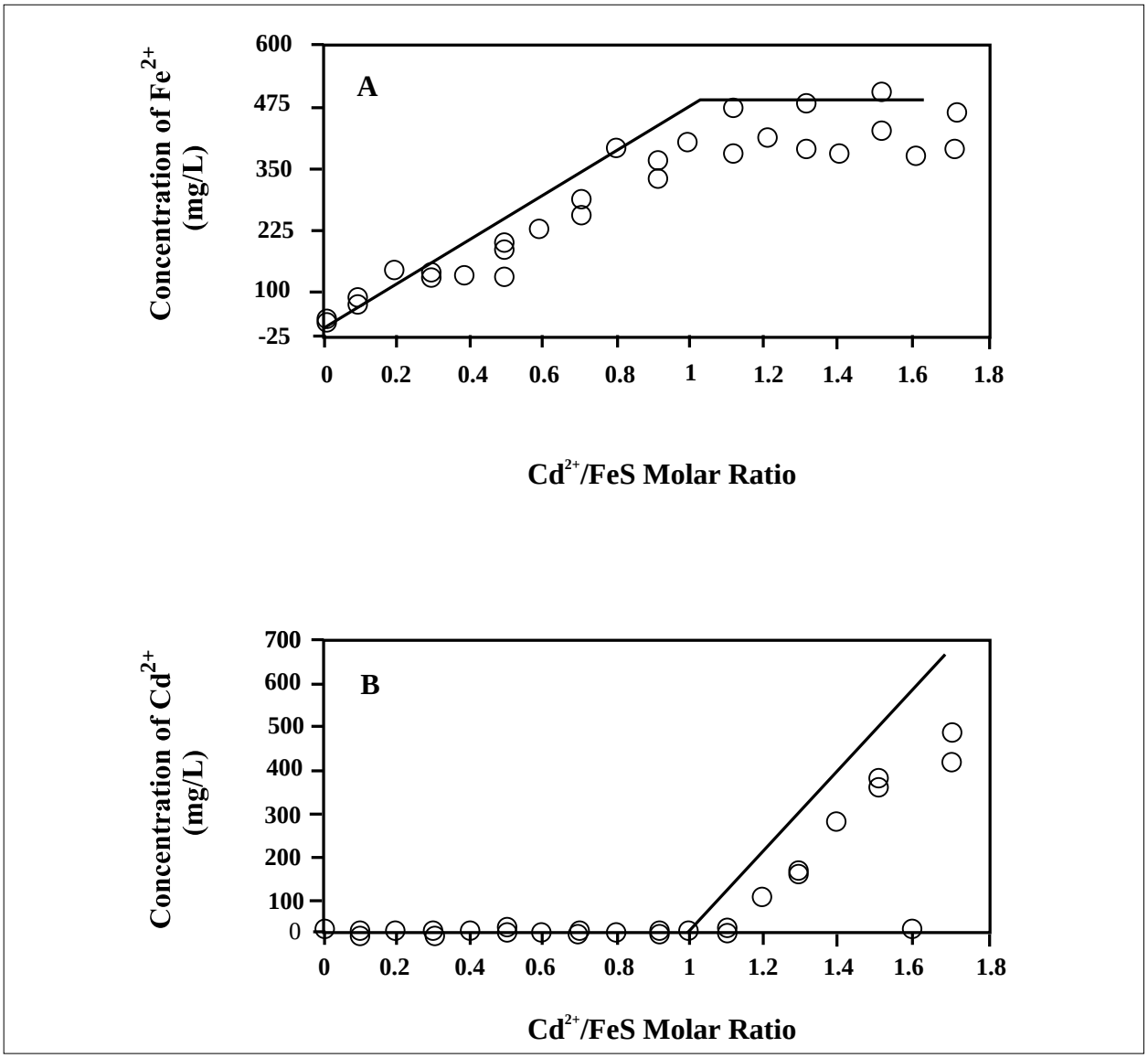

Figure 2-9. Concentrations of ionic iron (A) and cadmium (B) in the supernatant from titration of FeS by Cd^{2+} (Di Toro, unpublished data). The solid line represents the result expected from theory.

this causes the solid-phase iron sulfide to dissolve, releasing sulfide that is available for formation of cadmium sulfide. The reaction is

$$Cd^{2+} + FeS(s) \rightarrow CdS(s) + Fe^{2+} \quad (2\text{-}2)$$

Cadmium titrations with amorphous FeS and with sediments were performed to examine this possibility.

2.2.2.1 *Amorphous FeS*

A direct test of the extent to which this reaction takes place was performed (Di Toro et al., 1990). A quantity of freshly precipitated iron sulfide was titrated by adding dissolved cadmium. The resulting aqueous cadmium activity, measured with the cadmium electrode, versus the ratio of cadmium added $[Cd_A]$ to the amount of FeS initially present $[FeS(s)]_i$ is shown in Figure 2-8. The plot of dissolved cadmium versus cadmium added illustrates the increase in dissolved cadmium that occurs near $[Cd_A]/[FeS(s)]_i = 1$. It is interesting to note that these displacement reactions among metal sulfides have been observed by other investigators (Phillips and Kraus, 1965). The reaction was also postulated by Pankow (1979) to explain an experimental result involving copper and synthetic FeS.

These experiments plainly demonstrate that solid-phase amorphous iron sulfide can be readily displaced by adding cadmium. As a consequence, the source of available sulfide must be taken into account when evaluating the relationship between solid-phase and aqueous-phase cadmium in sediments.

A direct confirmation that the removal of cadmium was through the displacement of iron sulfide is shown in Figure 2-9. The supernatant from a titration of FeS by Cd^{2+} was analyzed for both iron and cadmium. The solid lines are the theoretical expectations based on the stoichiometry of the reaction.

2.2.2.2 *Sediments*

A similar titration procedure has been used to evaluate the behavior of sediments taken from four different marine environments: sediments from Black Rock Harbor and the Hudson River, and the sediments from Long Island Sound and Ninigret Pond used in the toxicity tests (Di Toro et al., 1990). The binding capacity for cadmium is estimated by extrapolating a straight line fit to the dissolved cadmium data. The equation is

$$[\Sigma Cd(aq)] = \max \{m([Cd_A] - [Cd_B])\} \quad (2\text{-}3)$$

where $[\Sigma Cd(aq)]$ is the total dissolved cadmium, $[Cd_A]$ is the cadmium added, $[Cd_B]$ is the bound cadmium, and m is the slope of the straight line. The different sediments exhibit quite different

Table 2-1. Cadmium binding capacity and AVS of sediments

Sediment	Initial AVS[a] (μmol/g)	Final AVS[b] (μmol/g)	Cd Binding Capacity[c] (μmol/g)
Black Rock Harbor	175 (41)	—	114(12)
Hudson River	12.6 (2.80)	—	8.58 (2.95)
LI Sound[d]	15.9 (3.30)	13.9 (6.43)	4.57 (2.52)
Mixture[d,e]	5.45 (—)	3.23 (1.18)	—
Ninigret Pond[d]	2.34 (0.73)	0.28 (0.12)	1.12 (0.42)

[a]Average (SD) AVS of repeated measurements of the stock.
[b]Average (SD) AVS after the sediment toxicity experiment.
[c]From Equation 2-3.
[d]From original cadmium experiment.
[e]50/50 mixture of LI Sound and Ninigret Pond.

Source: Di Toro et al., 1990.

binding capacities for cadmium, listed in Table 2-1, ranging from approximately 1 μmol/g to more than 100 μmol/g. The question as to whether this binding capacity is explained by the solid-phase sulfide present in the samples is addressed in subsequent sections of this document.

2.2.3 *Correlation to Sediment AVS*

The majority of sulfide in sediments is in the form of iron monosulfides (mackinawite and greigite) and iron bisulfide (pyrite), of which the former is the most reactive. These sediment sulfides can be classified into three broad classes that reflect the techniques used for quantification (Berner, 1967; Goldhauber and Kaplan, 1974; Morse et al., 1987). The most labile fraction, AVS, is associated with the more soluble iron monosulfides. The more resistant sulfide mineral phase, iron pyrite, is not soluble in the cold acid extraction used to measure AVS. Neither is the third compartment, organic sulfide, which is associated with the organic matter in sediments (Landers et al., 1983).

The possibility that acid volatile sulfide is a direct measure of the solid-phase sulfide that reacts with cadmium is examined in Table 2-1, which lists the sediment-binding capacity for cadmium and the measured AVS for each sediment, and in Figure 2-10, which indicates the initial AVS concentration. The sediment cadmium-binding capacity appears to be somewhat less than the initial AVS for the sediments tested. However, a comparison between the initial AVS of the sediments and that remaining after the cadmium titration is completed suggests that some AVS is lost during the titration experiment (Table 2-1). In any case, the covariation of sediment-binding capacity and AVS is clear. This suggests that

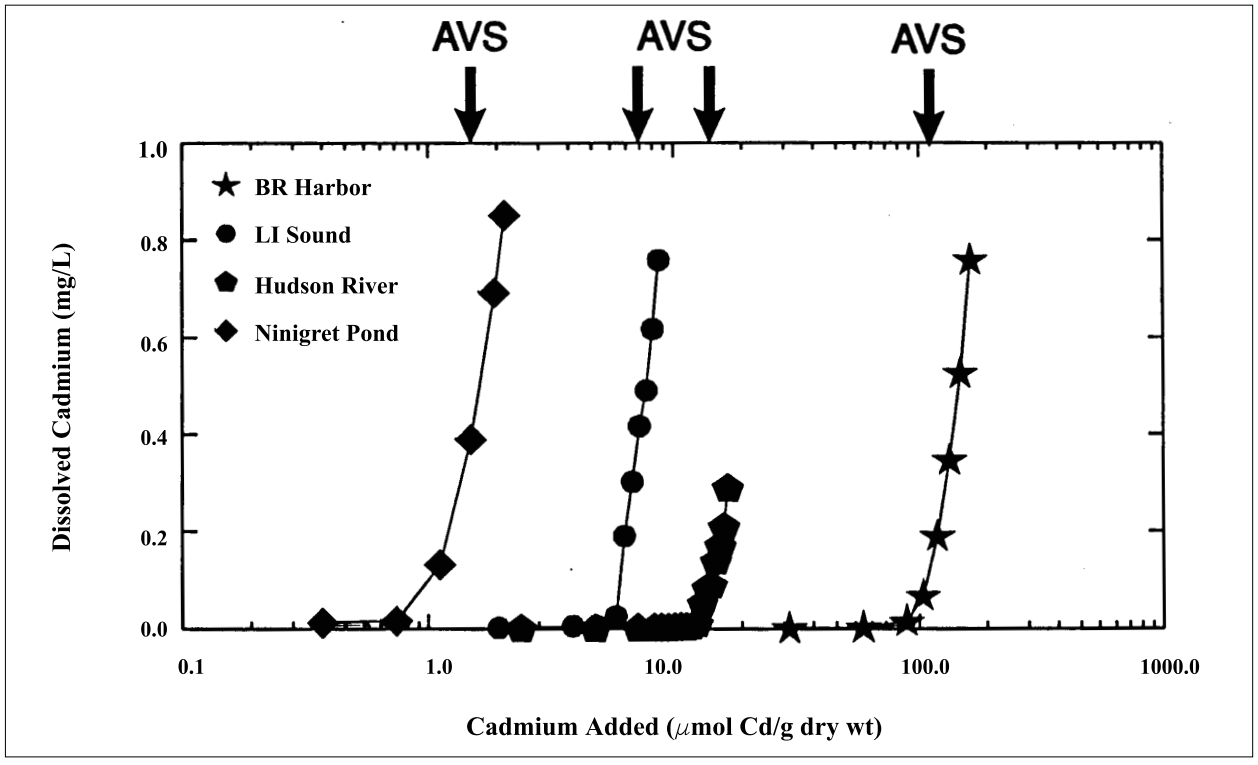

Figure 2-10. Cadmium titration of sediments from Black Rock Harbor, Long Island Sound, Hudson River, and Ninigret Pond. Cadmium added per unit dry weight of sediment versus dissolved cadmium. Arrows are the measured AVS concentrations for the four sediments (figure from Di Toro et al., 1990).

Table 2-2. Metal sulfide solubility products and ratios

Metal Sulfide	$\text{Log}_{10}K_{sp,2}$ [a]	$\text{Log}_{10}K_{sp}$ [b]	$\text{Log}_{10}(K_{MS}/K_{FeS})$
FeS	−3.64	−22.39	—
NiS	−9.23	−27.98	−5.59
ZnS	−9.64	−28.39	−6.00
CdS	−14.10	−32.85	−10.46
PbS	−14.67	−33.42	−11.03
CuS	−22.19	−40.94	−18.55
Ag_2S	−36.14	−54.71	−32.32

[a]Solubility products, $K_{sp,2}$ for the reaction $M^{2+} + HS^- \leftrightarrow MS(s) + H^+$ for FeS (mackinawite), NiS (millerite), and CdS (greenockite) from Emerson et al. (1983). Solubility products for ZnS (wurtzite), PbS (galena), CuS (covellite), and Ag_2S (acanthite) and $pK_2 = 18.57$ for the reaction $HS^- \leftrightarrow H^+ + S^{2-}$ from Schoonen and Barnes (1988).
[b]K_{sp} for the reaction $M^{2+} + S^{2-} \leftrightarrow MS(s)$ is computed from log $K_{sp,2}$ and pK_2.

measurement of AVS is the proper quantification of the solid-phase sulfides that can be dissolved by the addition of ionic cadmium. The chemical basis for this is examined below.

2.2.4 *Solubility Relationships and Displacement Reactions*

Iron monosulfide, FeS(s), is in equilibrium with aqueous-phase sulfide and iron via the reaction

$$FeS(s) \leftrightarrow Fe^{2+} + S^{2-} \quad (2\text{-}4)$$

If cadmium is added to the aqueous phase, the result is

$$Cd^{2+} + FeS(s) \leftrightarrow Cd^{2+} + Fe^{2+} + S^{2-} \quad (2\text{-}5)$$

As the cadmium concentration increases, $[Cd^{2+}][S^{2-}]$ will exceed the solubility product of cadmium sulfide and CdS(s) will start to form. Since the cadmium sulfide is more insoluble than iron monosulfide, FeS(s) should start to dissolve in response to the lowered sulfide concentration in the interstitial water. The overall reaction is

$$Cd^{2+} + FeS(s) \leftrightarrow CdS(s) + Fe^{2+} \quad (2\text{-}6)$$

The iron in FeS(s) is displaced by cadmium to form soluble iron and solid cadmium sulfide, CdS(s). The consequence of this replacement reaction can be seen using the analysis of the M(II)-Fe(II)-S(-II) system with both MS(s) and FeS(s) presented in Di Toro et al. (1992). M^{2+} represents any divalent metal that forms a sulfide that is more insoluble than FeS. If the added metal, $[M]_A$, is less than the AVS present in the sediment then the ratio of metal activity to total metal in the sediment–interstitial water system is less than the ratio of the MS to FeS solubility product constant

$$\{M^{2+}\}/[M]_A < K_{MS}/K_{FeS} \quad (2\text{-}7)$$

This general result is independent of the details of the interstitial water chemistry. In particular, it is independent of the Fe^{2+} activity. Of course, the actual value of the ratio $\{M^{2+}\}/[M]_A$ depends on aqueous speciation, as indicated by Equation 2-6. However, the ratio is still less than the ratio of the sulfide solubility products.

This is an important finding because the data presented in Section 2.1.1 indicate that toxicity is related to metal activity, $\{M^{2+}\}$. This inequality guarantees that the metal activity, in contrast to the total dissolved metal concentration, is regulated by the iron sulfide–metal sulfide system.

The metal sulfide solubility products and the ratios are listed in Table 2-2. For example, the ratio of cadmium activity to total cadmium is less

than $10^{-10.46}$. For nickel, the ratio is less than $10^{-5.59}$. By inference, this reduction in metal activity will occur for any other metal that forms a sulfide that is significantly more insoluble than iron monosulfide. The ratios for the other metals in Table 2-2 (Cu, Pb, Ag, and Zn) indicate that metal activity for these metals will be very small in the presence of excess AVS.

2.2.5 *Application to Mixtures of Metals*

A conjecture based on the sulfide solubility products for the metals listed in Table 2-2 is that the sum of the molar concentrations of metals should be compared with AVS. Because all these metals have lower sulfide solubility parameters than FeS, they would all exist as metal sulfides if their molar sum (and using [Ag]/2 because it is monovalent) is less than the AVS. For this case

$$\Sigma_i[M_T]_i < AVS \qquad (2\text{-}8)$$

no metal toxicity would be expected, where $[M_T]_i$ is the total cold acid extractable i^{th} metal molar concentration in the sediment (divided by 2 for silver). On the other hand, if their molar sum is greater than the AVS concentration, then a portion of the metals with the largest sulfide solubility parameters would exist as free metal and potentially cause toxicity. For this case the following would be true

$$\Sigma_i[M_T]_i > AVS \qquad (2\text{-}9)$$

These two equations are precisely the formulas that could be employed to determine the extent of metal toxicity in sediments assuming additive behavior and neglecting the effect of partitioning to other sediment phases. Whether the normalized sum is less than or greater than 1.0 discriminates between nontoxic and potentially toxic sediments. The additivity does not come from the nature of the mechanism that causes toxicity. Rather, it results from the equal ability of the metals to form metal sulfides with the same stoichiometric ratio of M and S (except silver).

The appropriate quantity of metals to use in the metals and AVS comparison is referred to as SEM, that is, the metal extracted with the cold acid used in the AVS procedure. This is the appropriate quantity to use because some metals form sulfides that are not labile in the AVS extraction (e.g., nickel, copper). If a more rigorous extraction were used to increase the fraction of metal extracted that did not also capture the additional sulfide extracted, then the sulfide associated with the additional metal release would not be quantified. This would result in an erroneously high metal value relative to AVS (Di Toro et al., 1992).

The above discussion is predicated on the assumption that all the metal sulfides behave similarly to cadmium sulfide. Furthermore, it has been assumed that only acid-soluble metals are reactive enough to affect the free metal activity. That is, the proper metal concentration to be used is the SEM. Both of these hypotheses were tested directly with benthic organisms using sediment toxicity tests. Results of these sediment-spiking experiments with cadmium, copper, lead, nickel, silver, zinc, and a mixture of these metals are presented in Section 3.

Section 3

Toxicity of Metals in Sediments

3.1 General Information

This section summarizes data from acute and chronic toxicity tests that demonstrate that absence of sediment toxicity caused by metals can be predicted by (a) the use of interstitial water concentrations of metals or (b) comparison of molar concentrations of AVS and SEM. Furthermore, they demonstrate that use of $(\Sigma SEM\text{-}AVS)/f_{OC}$ reduces the variability associated with prediction of when sediments will be toxic. The ability to predict toxicity of metals in sediments, through a fundamental understanding of chemical bioavailability, is demonstrated using results of toxicity tests with benthic organisms in spiked or field sediments. A wide variety of individual benthic species having different habitat requirements have been tested in 10-day experiments in spiked and field sediments, including the following: an oligochaete (*Lumbriculus variegatus*), polychaetes (*Capitella capitata* and *Neanthes arenaceodentata*), amphipods (*A. abdita, R. hudsoni, Leptocheirus plumulosus,* and *Hyalella azteca*), a harpacticoid copepod (*Amphiascus tenuiremis*), a midge (*Chironomus tentans*), and a gastropod (*Helisoma* sp.). In addition, the approach was tested in life-cycle tests with *L. plumulosus* and *C. tentans*. Many other benthic species were tested in freshwater and saltwater benthic colonization studies.

3.1.1 *Terminology*

Early studies on use of AVS in prediction of biological effects (e.g., Di Toro et al., 1990) involved the ratio of SEM to AVS, expressed as SEM/AVS. The ratio appeared more useful in the early laboratory tests because it caused concentration-response data from spiking experiments with different sediments to fall on the same line (Di Toro et al., 1990, 1992; Casas and Crecelius, 1994; Pesch et al., 1995; Berry et al., 1996). Later studies, however, showed several advantages to the use of the difference, expressed as SEM-AVS (Hansen et al., 1996a). The two expressions—SEM/AVS ≤ 1 and SEM-AVS ≤ 0—are functionally equivalent. Both indicate an excess of AVS over SEM. The advantages to using SEM-AVS are that it does not get very large when AVS is very low (as the ratio does), and that it can be used to develop partitioning relationships that include other phases, such as total organic carbon (TOC) (see Section 3.4; see also the discussion in Section 3.2.5). For these reasons, the use of the SEM-AVS difference is the recommended method, and it will be used throughout the rest of this document except in the discussion of the historical development of AVS theory that follows. In the ensuing discussion, SEM/AVS ratios are presented because they were originally presented in this form.

3.2 Predicting Metal Toxicity: Short-Term Studies

3.2.1 *Spiked Sediments: Individual Experiments*

A key to understanding the bioavailability of sediment-associated contaminants was provided by Adams et al. (1985), who observed that the effects of kepone, a nonionic organic pesticide, were similar across sediments when toxicity was related to interstitial water concentrations. Swartz et al. (1985) and Kemp and Swartz (1986) first observed that metal concentrations in interstitial waters of different sediments were correlated with observed biological effects. However, as opposed to the situation for nonionic organic chemicals and organic carbon (see Di Toro et al., 1991), the sediment-partitioning phases that controlled interstitial water concentrations of metals and metal-induced sediment toxicity were initially not apparent.

Di Toro et al. (1990) first investigated the significance of sulfide partitioning in controlling metal bioavailability and metal-induced toxicity in marine sediments spiked with cadmium. In these experiments, the operational definition of Cornwell and Morse (1987) was used to identify that fraction of amorphous sulfide, or AVS, available to interact with cadmium in the sediments. Specifically, AVS was defined as the sulfide liberated from wet sediment when treated with cold 1N HCl acid. Di Toro et al. (1990) found that, when expressed on a dry weight basis, the toxicity of cadmium in sediments in 10-day tests with the amphipods *R. hudsoni* or *A. abdita* was sediment specific (Figure 3-1A; from Di Toro et al., 1990).

Toxicity increased with increasing cadmium concentration, but the concentration-response relationships were different for each sediment. Thus, it would not be possible to predict whether a particular sediment would be toxic or not. If the cadmium concentration is expressed on an interstitial water basis (Figure 3-1B), however, concentration response is not sediment specific. Similar results are observed when cadmium concentration is expressed as SEM/AVS (Figure 3-1C). Note that when the ratio of μmol Cd/μmol AVS was less than 1.0, the sediments were not toxic, and when the ratio was greater than 1.0, the sediments became increasingly toxic. Studies by Carlson et al. (1991) with cadmium-spiked freshwater sediments yielded similar results; when there was more AVS than total cadmium, significant toxicity was not observed in 10-day tests with an oligochaete (*L. variegatus*) or snail (*Helisoma* sp). Di Toro et al. (1992), in their studies with nickel-spiked sediments using *A. abdita* and field sediments contaminated with cadmium and nickel using the freshwater amphipod *H. azteca*, provided further support to the importance of AVS in controlling metal bioavailability in sediments. These studies suggested that it may be feasible to derive an ESB for mixtures of metals by direct comparison of molar AVS concentrations to the molar sum of the concentrations of cationic metals (specifically, cadmium, copper, lead, nickel, and zinc) extracted with the AVS (i.e., ΣSEM). They observed that expression of metals concentrations based on the sum of SEM concentrations is required because a significant amount of nickel sulfide is not completely soluble in the AVS extraction. Hence, AVS must be used as the measure of reactive sulfide and the sum of SEM as the measure of total reactive metal.

Casas and Crecelius (1994) further explored the relationship of SEM and AVS, interstitial water concentrations, and toxicity by conducting 10-day toxicity tests with the marine polychaete *C. capitata* exposed to sediments spiked with zinc, lead, and copper. As was true in earlier studies, elevated interstitial water metal concentrations were observed only when SEM concentrations exceeded those of AVS. Sediments were not toxic when SEM concentrations were less than AVS and when the concentrations in interstitial water were less than the water-only LC50 values. Green et al. (1993) reported results of another spiking experiment supporting this general EqP approach to deriving an ESB for metals. In their study, metal–sulfide partitioning was not directly quantified, but it was found that toxicity of cadmium-spiked marine sediments to the meiobenthic copepod *A. tenuiremis* was predictable based on interstitial water, but not sediment dry weight cadmium concentrations. Further spiking experiments by Pesch et al. (1995) demonstrated that 10-day survival of the marine polychaete *N. arenceodentata* was comparable to controls in cadmium- or nickel-spiked sediments with more AVS than SEM.

Berry et al. (1996) described experiments in which *A. abdita* were exposed for 10 days to two or three sediments spiked either singly, or in combination, with cadmium, copper, lead, nickel, and zinc. As in previous studies, significant toxicity to the amphipod did not occur when AVS concentrations exceeded those of SEM. They compared observed mortality with interstitial water metal concentrations expressed as interstitial water toxic units (IWTUs)

$$IWTU = [M_d]/LC50 \quad (3\text{-}1)$$

where $[M_d]$ is the dissolved metal concentration in the interstitial water, and the LC50 is the concentration of the metal causing 50% mortality of the test species in a water-only test. If interstitial water exposure in a sediment test is indeed equivalent to that in a water-only test, then 1.0 IWTU should result in 50% mortality of the test animals. Berry et al. (1996) reported that significant (>24%) mortality of the saltwater amphipod occurred in only 3.0% of sediments with less than 0.5 IWTU, whereas samples with greater than 0.5 IWTUs were toxic 94.4% of the time. Berry et al. (1996) also made an important observation relative to interstitial water metal chemistry in their mixed-metals test; chemical equilibrium calculations suggest that the relative affinity of metals for AVS should be silver> copper>lead>cadmium>zinc>nickel (Emerson et al., 1983; Di Toro et al., 1992); hence, the appearance of the metals in interstitial water as AVS is exhausted should occur in an inverse order. For example, zinc would replace nickel in a monosulfide complex and nickel would be liberated to the interstitial water, and so on. Berry et al. (1996) observed this trend in sediments spiked with cadmium, copper, nickel, and zinc (Figure 3-2). Furthermore, an increase in the concentration of a metal in a sediment with a low sulfide solubility product constant (K_{sp}) theoretically would displace a previously unavailable and nontoxic metal with a higher K_{sp}, making that metal available to bind to other sediment phases or enter interstitial water to become toxic. Berry et al. (1999) exposed the saltwater amphipod *A. abdita* to sediments spiked with silver. When AVS was detected in the sediments, they were not toxic and interstitial water contained no detectable silver. For sediments that contain no detectable AVS,

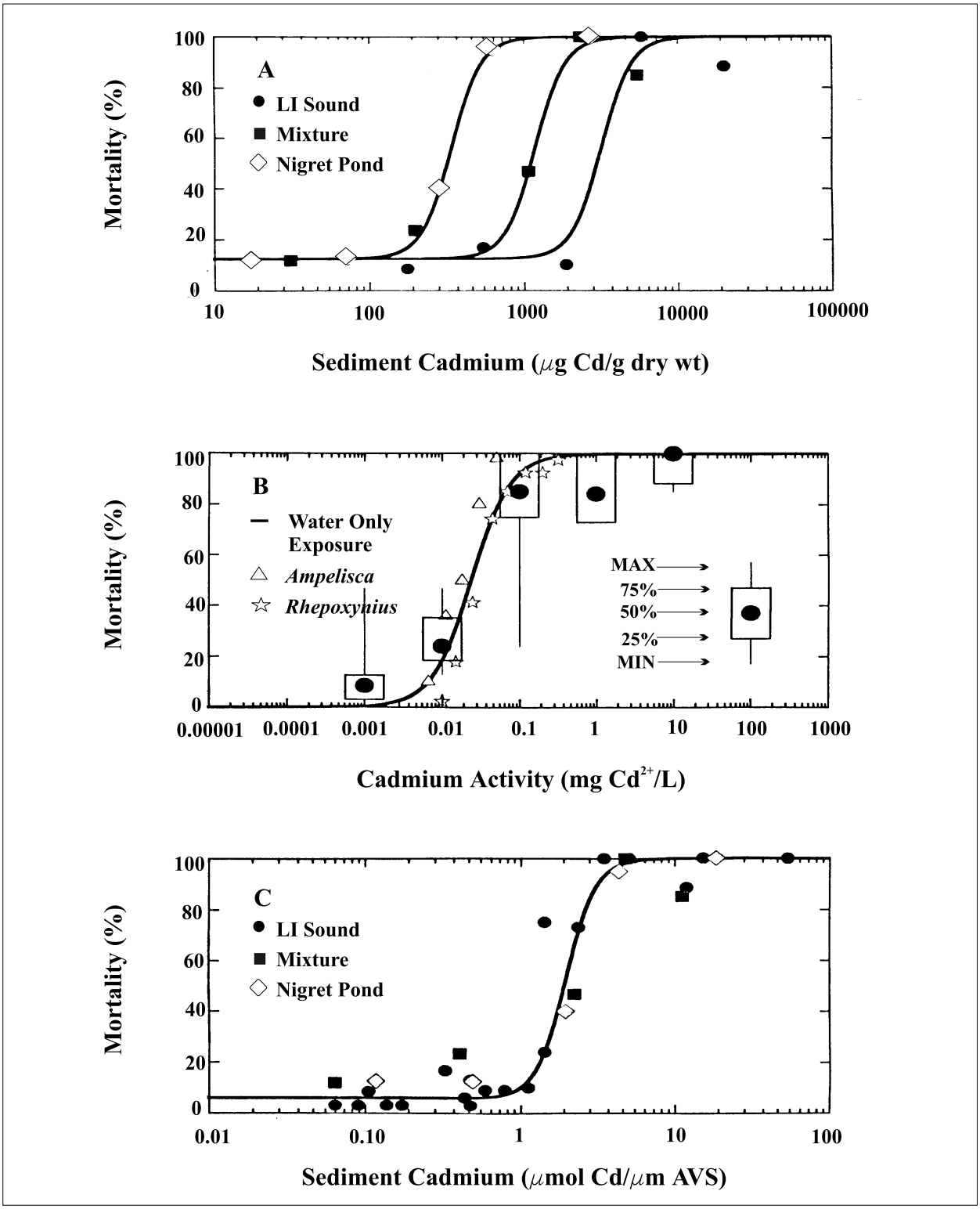

Figure 3-1. Percentage mortality of amphipods (*Ampelisca abdita* and *Rhepoxynius hudsoni*) exposed to sediments from Long Island Sound, Ninigret Pond, and a mixture of these two sediments as a function of the sum of the concentrations of metals in sediments expressed as: (A) dry weight, (B) interstitial water cadmium activity, and (C) the sediment cadmium/AVS ratio (figures from Di Toro et al., 1990).

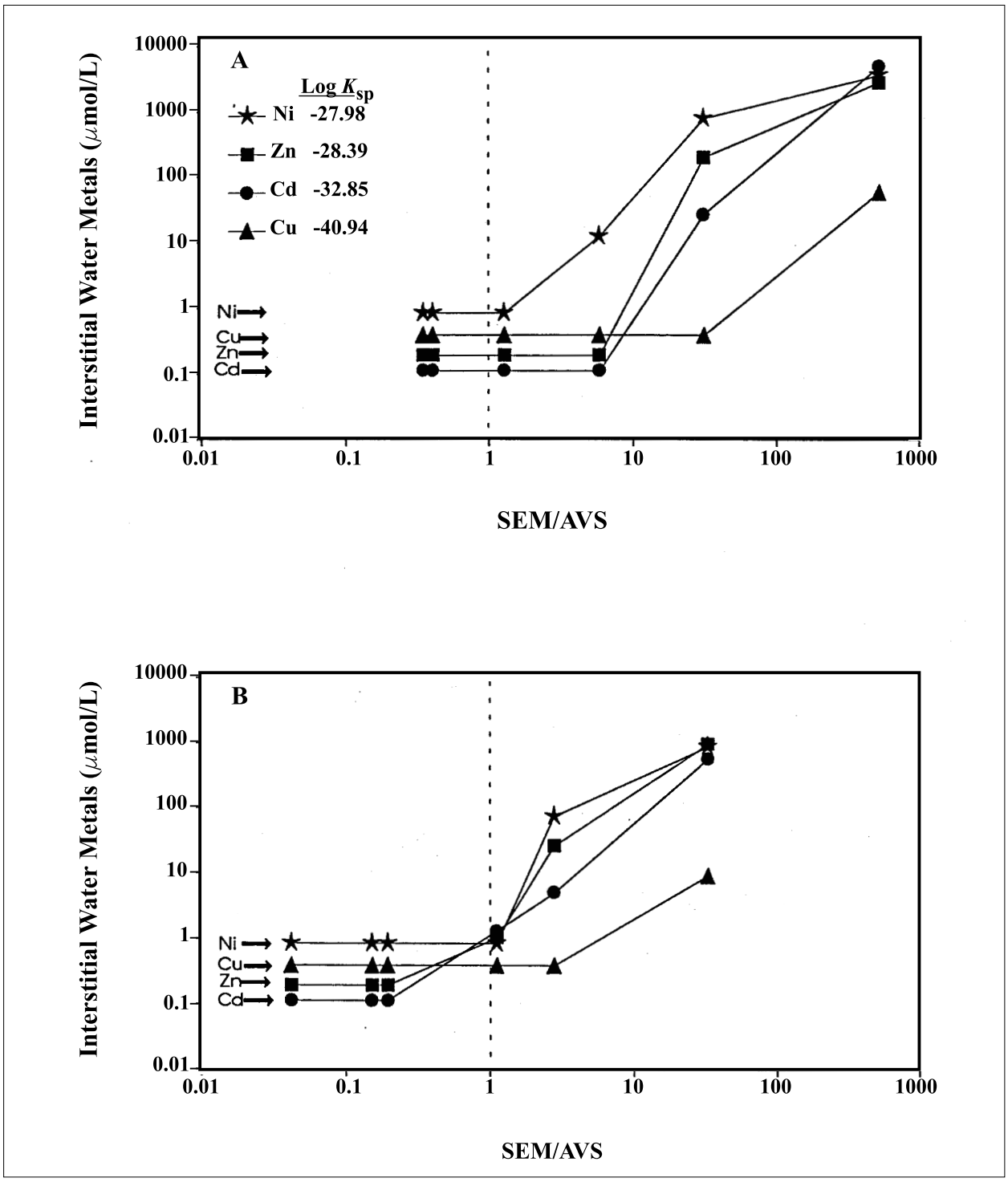

Figure 3-2. **Concentrations of individual metals in interstitial water of sediments from Long Island Sound (A) and Ninigret Pond (B) in the mixed metals experiment as a function of SEM/AVS ratio. Concentrations below the interstitial water detection limits, indicated by arrows, are plotted at one-half the detection limit. K_{SP} is the sulfide solubility product constant (figures from Berry et al., 1996).**

any SEM silver that is detected is dissolved interstitial silver, because silver sulfide and silver chloride precipitate are not extracted using the standard AVS procedure.

3.2.2 *Spiked Sediments: All Experimental Results Summarized*

This summary includes data from amphipods exposed in 10-day toxicity tests to saltwater sediments spiked with cadmium, copper, lead, nickel, silver, or zinc and their mixtures (Di Toro et al., 1990; Berry et al., 1996, 1999); polychaetes exposed to sediments spiked with cadmium, copper, lead, nickel, or zinc (Casas and Crecelius, 1994; Pesch et al., 1995); copepods exposed to sediments spiked with cadmium (Green et al., 1993; measured interstitial cadmium but not AVS); and freshwater tests using oligochaetes and snails exposed to sediments spiked with cadmium (Carlson et al., 1991). Seven species (freshwater and saltwater) and sediments from seven different locations were described. AVS concentrations ranged from 1.9 to 65.7 μmol/g dry weight, and TOC ranged from 0.15% to 10.6% in these sediments.

Overall, the results of these experiments demonstrate that predictions of the toxicity of sediments spiked with metals using the total metal concentration on a dry weight basis are not based on scientific theories of bioavailability and will have considerable error (Figures 3-3A and 3-4A). Sediments having $\leq$24% mortality are considered nontoxic as defined by Berry et al. (1996), which is indicated by the horizontal line in Figure 3-3. Furthermore, the concentration range where it is 90% certain that the sediment may be either toxic or nontoxic, shown as dashed lines in Figure 3-3, is almost two orders of magnitude for dry weight metals, a little over an order of magnitude for IWTUs, and only a half order of magnitude for SEM/AVS (see Section 3.4 for a description of the derivation of the uncertainty limits). The uncertain range for dry weight metals is approximately equal to the sum of the uncertainty range for SEM/AVS plus the range in the AVS concentrations of the spiked sediments in the database. If sediments with a lower AVS concentration had been tested, effects would have occurred at a lower dry weight concentration, and if sediments with lower or higher AVS concentrations had been tested, the uncertainty range would increase. Importantly, the uncertainty range for IWTUs or SEM/AVS would likely not be altered.

Even given the above, it is visually tempting to select a cutoff at a dry weight concentration of 1.0 μmol/g to indicate the separation of sediments that are toxic or nontoxic. This would be inappropriate because toxicity of metals in sediments when concentrations are expressed as dry weights have been shown to be sediment specific (Figure 3-1A). Also, had sediments with lower or higher AVS concentrations been tested, the cutoff would have been at lower or higher dry weight concentrations. However, to further demonstrate the risks of establishing a dry weight cutoff, the data from the 184 spiked sediments in Figure 3-3 were re-analyzed. A visually based cutoff of 1.0 μmol/g dry weight, and theoretically based cutoffs of 0.5 IWTU and 1.0 SEM/AVS were selected. Sediment concentrations were numerically ordered. Those with concentrations less than the cutoffs were divided into three groups containing approximately the same number of sediments (15, 22, or 25 sediments per group for dry weight metal concentrations, IWTUs, and SEM/AVS, respectively). Similarly, sediments containing greater concentrations were divided into six groups (21, 16, or 14 sediments per group for dry weight metal concentrations, IWTUs, and SEM/AVS, respectively). The percentages of nontoxic ($\leq$24% mortality) and toxic (>24% mortality) sediments in each group are plotted in a stacked bar plot (Figure 3-4). Not surprisingly, because the distribution was visually selected, most sediments having less than 1.0 μmol/g dry weight metal were not toxic. The same was true for the toxicologically selected cutoffs of 0.5 IWTUs and SEM/AVS ratios of 1.0. The advantage of using IWTUs and SEM/AVS becomes more clear when the sediments above the cutoffs are considered. For dry weight metal concentrations, more of the sediments in the first four sediment groups (up to 26.8 μmol/g dry weight) were nontoxic than were toxic. It was only in the two sediment groups that contained the highest concentrations, >27.6 μmol/g dry weight, that toxic sediments predominated after the first two sediment groups. In contrast, toxic sediments predominated in only the first two sediment groups above the IWTU cutoff and after the first sediment group above the SEM/AVS ratio cutoff.

In some cases, the dry weight metal concentrations required to cause acute mortality in these experiments were very high relative to those often suspected to be of toxicological significance in field sediments (e.g., Figures 3-1A and 3-3A). This has sometimes been interpreted as a limitation of the use of SEM and AVS to predict metal-induced toxicity. However, the range of AVS in these sediments spiked with metals is similar to

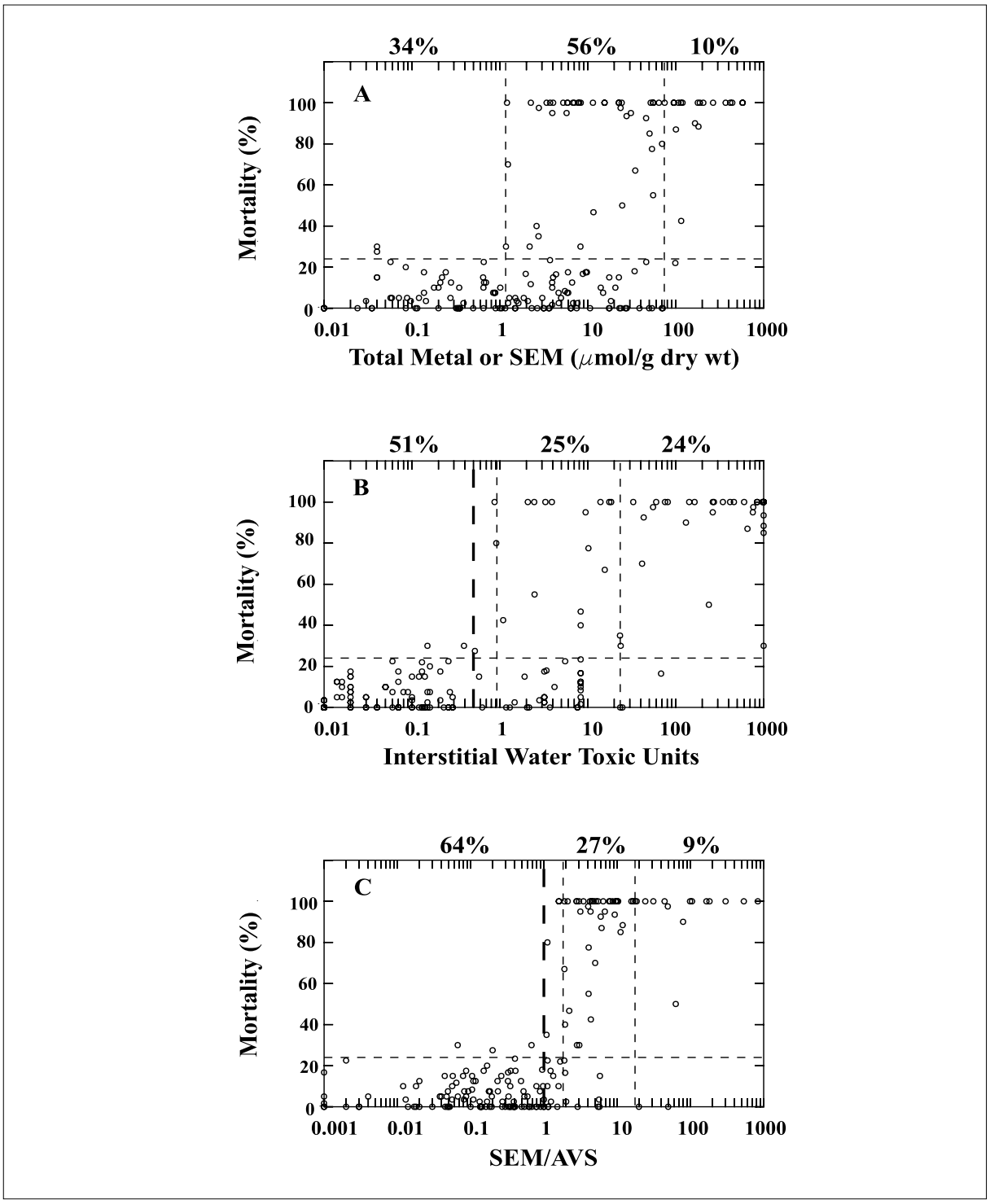

Figure 3-3. Percentage mortality of freshwater and saltwater benthic species in 10-day toxicity tests in sediments spiked with individual metals (Cd, Cu, Pb, Ni, Ag, or Zn) or a metal mixture (Cd, Cu, Ni, and Zn). Mortality is plotted as a function of: (A) the sum of the concentrations of the respective metal or metal mixture in μmol metal per gram dry weight of sediment; (B) IWTU; and (C) SEM/AVS ratio. Data below the detection limits are plotted at IWTU=0.01 and SEM/AVS=0.001. Heavy dashed lines are the theoretically based cutoffs of 0.5 IWTU and a SEM/AVS ratio of 1.0. Light vertical dashed lines are the 90% uncertainty bound limits derived as in Section 3.4. The percentage of the total number of sediments (n = 184) within the bounded limits is provided above each of the three panels for the purpose of comparison (silver data from Berry et al., 1999; all other data modified after Berry et al., 1996).

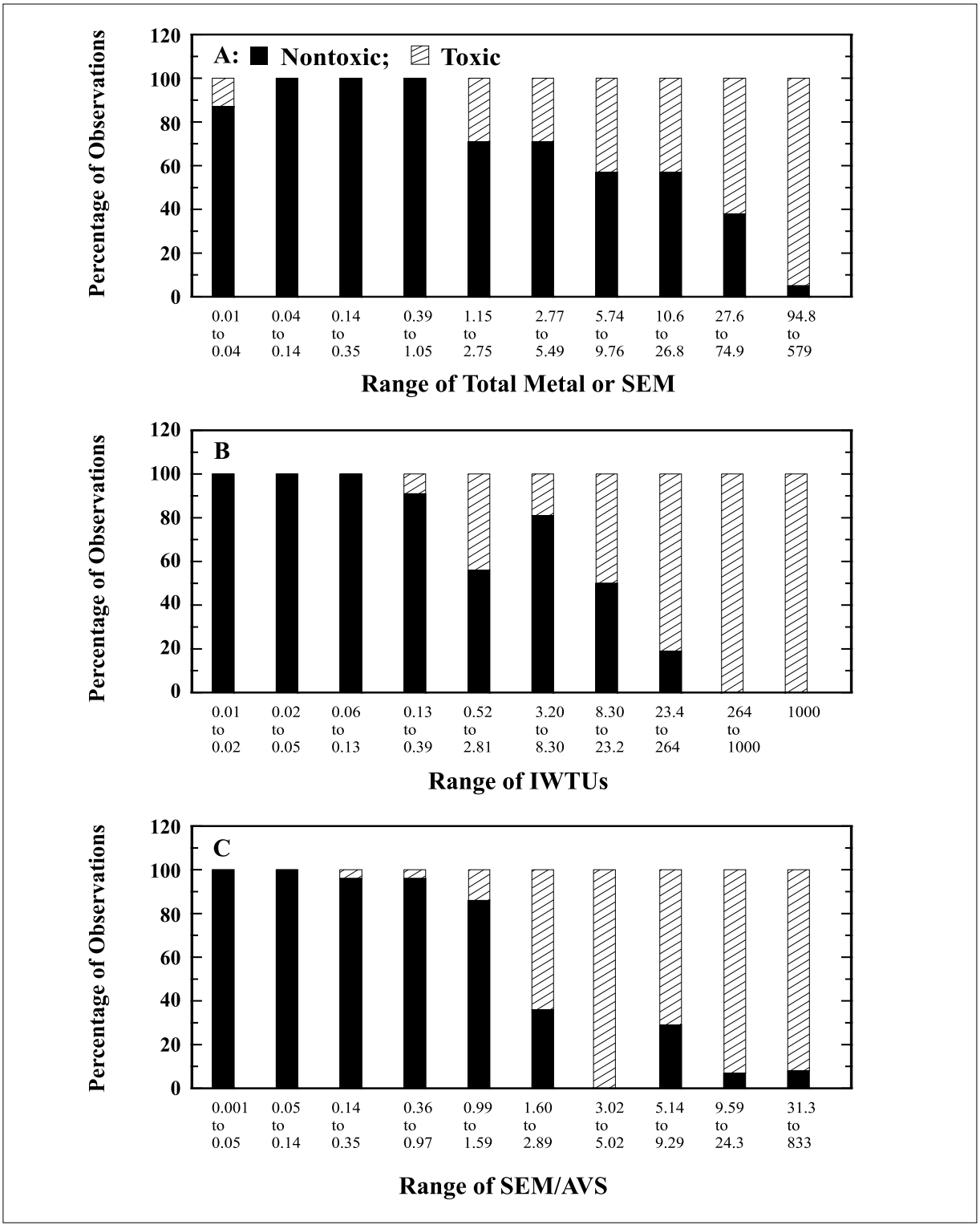

Figure 3-4. Percentage of the 184 spiked sediments from Figure 3-3 that were nontoxic or toxic over various intervals of (A) concentrations of metal based on sediment dry weight (μmol/g), (B) IWTU, and (C) SEM/AVS.

that of sediments commonly occurring in the field. The important point here is that even a sediment with only a moderate concentration of AVS has a considerable capacity for sequestering metals as a metal sulfide, a form that is not bioavailable (Di Toro et al., 1990).

In contrast, the combined data from all available freshwater and saltwater spiked-sediment experiments support the use of IWTUs to predict mortality of benthic species in spiked-sediment toxicity tests (Figure 3-3B). Mortality in these experiments was sediment independent when plotted against IWTUs. Sediments with IWTUs of <0.5 were generally not toxic. Of the 96 sediments with IWTUs <0.5, 96.9% were not toxic, whereas 76.4% of the 89 sediments with IWTUs ≥0.5 were toxic (Table 3-1). This close relationship between IWTUs and sediment toxicity in sediments spiked with metals was also observed in studies with field sediments contaminated with metals (see Section 3.2.3 below), as well as sediments spiked with nonionic organic chemicals (Adams et al., 1985; Swartz et al., 1990; Di Toro et al., 1991), and field sediments contaminated with nonionic organic chemicals (Hoke et al., 1994; Swartz et al., 1994).

Table 3-1. Toxicity of sediments from freshwater and saltwater lab-spiked sediment tests, field locations, and combined lab-spiked and field sediment tests as a function of the molar concentrations of SEM and AVS (SEM/AVS or the SEM-AVS), interstitial water toxic units (IWTUs), and both SEM/AVS or SEM-AVS and IWTUs

			Percent of Sediments	
Study Type/Parameter	Value	n	Nontoxic[a]	Toxic[b]
Laboratory Spike:				
SEM/AVS or SEM-AVS[c]	≤1.0 or ≤0.0	101	98.0	2.0
	>1.0 or >0.0	95	26.3	73.7
IWTU[d]	<0.5	96	96.9	3.1
	≥0.5	89	23.6	76.4
SEM/AVS or SEM-AVS[c]; IWTU[d]	≤1.0 or ≤0.0; <0.5	83	97.6	2.4
	>1.0 or >0.0; ≥0.5	78	14.1	85.9
Field:				
SEM/AVS or SEM-AVS[c]	≤1.0 or ≤0.0	57	98.2	1.8
	>1.0 or >0.0	79	59.5	40.5
IWTU[d]	<0.5	79	98.7	1.3
	≥0.5	53	45.3	54.7
SEM/AVS or SEM-AVS[c]; IWTU[d]	≤1.0 or ≤0.0; <0.5	49	100.0	0.0
	>1.0 or >0.0; ≥0.5	45	33.3	66.7
Lab-Spike and Field:				
SEM/AVS or SEM-AVS[c]	≤1.0 or ≤0.0	158	98.1	1.9
	>1.0 or >0.0	174	42.0	58.0
IWTU[d]	<0.5	175	97.7	2.3
	≥0.5	142	31.7	68.3
SEM/AVS or SEM-AVS[c]; IWTU[d]	≤1.0 or ≤0.0; <0.5	132	98.5	1.5
	>1.0 or >0.0; ≥0.5	123	21.1	78.9

[a]Nontoxic sediments ≤24% mortality.
[b]Toxic sediments >24% mortality.
[c]An SEM/AVS ratio of ≤1.0 or an SEM-AVS difference of ≤0.0 indicates an excess of sulfide and probable nontoxic sediments. An SEM/AVS ratio of >1.0 or an SEM-AVS difference of >0.0 indicates an excess of metal and potentially toxic sediments.
[d]An IWTU of <0.5 indicates a probable nontoxic interstitial water concentration of less than one-half of the water-only LC50 of the same duration. An IWTU of ≥0.5 indicates a possibly toxic interstitial water concentration of greater than one-half of the water-only LC50 of the same duration.

Source: Modified from Hansen et al., 1996a.

The interstitial water metal concentrations in spiked-sediment studies were most often below the limit of analytical detection in sediments with SEM/AVS ratios below 1.0 (Berry et al., 1996). Above an SEM/AVS ratio of 1.0, the interstitial metals concentrations increased up to five orders of magnitude with increasing SEM/AVS ratio. This increase of several orders of magnitude in interstitial water metals concentration with an increase of only a factor of two or three in sediment concentration is the reason why mortality is most often complete in these sediments, and why the chemistry of anaerobic sediments controls the toxicity of metals to organisms living in aerobic microhabitats. It also explains why toxicities of different metals in the same sediment to different species when expressed on the basis of sediment metals concentration are so similar. Interstitial water metals were often below or near detection limits when SEM/AVS ratios were only slightly above 1.0, indicating the presence of other metal-binding phases in sediments.

The combined data from all available freshwater and saltwater spiked-sediment experiments also support the use of SEM/AVS ratios to predict sediment toxicity to benthic species in spiked-sediment toxicity tests. All tests yield similar results when mortality is plotted against SEM/AVS ratios (Figure 3-3C). Mortality in these experiments was sediment independent when plotted on an SEM/AVS basis. With the combined data, 98.0% of the 101 metals-spiked sediments with SEM/AVS ratios $\leq$1.0 were not toxic, whereas 73.7% of the 95 sediments with SEM/AVS ratios >1.0 were toxic (Table 3-1).

The overall data show that when both SEM/AVS ratios and IWTUs are used, predictions of sediments that would be toxic were improved. Of the 83 sediments with SEM/AVS ratios $\leq$1.0 and IWTUs <0.5, 97.6% were not toxic, whereas 85.9% of the 78 sediments with SEM/AVS ratios >1.0 and IWTUs $\geq$0.5 were toxic (Table 3-1).

These results show that SEM/AVS and IWTUs are accurate predictors of the absence of mortality in sediment toxicity tests; however, predictions of sediments that might be toxic are less accurate. The fact that a significant number of sediments (26.3%) tested had SEM/AVS ratios of >1.0 but were not toxic indicates that other binding phases, such as organic carbon (Mahony et al., 1996), may also control bioavailability in anaerobic sediments.

Organism behavior may also explain why some sediments with SEM/AVS ratios of >1.0 were not toxic. Many of the sediments that had the highest SEM/AVS ratios in excess of 1.0 that produced little or no mortality were from experiments using the polychaete *N. arenaceodentata* (see Pesch et al., 1995). In these experiments, this polychaete did not burrow into some of the test sediments with the highest concentrations, thereby limiting its exposure to the elevated concentrations of metals in the interstitial water and sediments. This same phenomenon may also explain the low mortality of snails, *Heliosoma* sp., in freshwater sediments with high SEM/AVS ratios. These snails are epibenthic and crawl onto the sides of test beakers to avoid contaminated sediments (G.L. Phipps, U.S. EPA, Duluth, MN, personal communication). Increased mortality was always observed in sediments with SEM/AVS ratios >5.9 in tests with the other five species.

Similarly, a significant number of sediments (23.6%) with $\geq$0.5 IWTUs were not toxic. This is likely the result of interstitial water ligands, which reduces the bioavailability and toxicity of dissolved metals; sediment avoidance by polychaetes or snails; or methodological problems in contamination-free sampling of interstitial water. Ankley et al. (1991) suggested that a toxicity correction for the hardness of the interstitial water for freshwater sediments is needed to compare toxicity in interstitial water with that in water-only tests. Absence of a correction for hardness would affect the accuracy of predictions of metal-induced sediment toxicity using IWTUs. Furthermore, a significant improvement in the accuracy of metal-induced toxicity predictions using IWTUs might be achieved if DOC binding in the interstitial water is taken into account. Green et al. (1993) and Ankley et al. (1991) hypothesized that increased DOC in the interstitial water reduced the bioavailability of cadmium in sediment exposures, relative to the water-only exposures. Green et al. (1993) found that the LC50 value for cadmium in an interstitial water exposure without sediment was more than twice that in a water-only exposure, and that the LC50 value for cadmium in interstitial water associated with sediments was more than three times that in a water-only exposure.

3.2.3 *Field Sediments*

In addition to short-term laboratory experiments with spiked sediments, there have been several published studies of laboratory toxicity tests with metal-contaminated sediments from the field. Ankley et al. (1991) exposed *L. variegatus* and the amphipod *H. azteca* to 17 sediment samples along a gradient of cadmium and nickel contamination from a freshwater/

estuarine site in Foundry Cove, NY. In 10-day toxicity tests, *H. azteca* mortality was not significantly different from controls in all sediments where SEM (cadmium plus nickel) was less than AVS. Mortality was greater than controls only in sediments with more SEM than AVS. *L. variegatus* was far less sensitive to the sediments than *H. azteca*, which correlates with the differential sensitivity of the two species in water-only tests with cadmium and nickel.

In 10-day toxicity tests with the saltwater amphipod *A. abdita* in these same sediments, Di Toro et al. (1992) observed that metals concentrations ranging from 0.1 to 28 μmol SEM/g sediment were not toxic in some sediments, whereas metals concentrations ranging from 0.2 to 1,000 μmol SEM/g sediment were lethal in other sediments. These results indicate that the bioavailable fraction of metals in sediments varies from sediment to sediment. In contrast, the authors also observed a clearly discernible mortality-concentration relationship when mortality was related to the SEM/AVS molar ratio (i.e., there was no significant mortality where SEM/AVS ratios were <1.0, mortality increased in sediments having SEM/AVS ratios of 1.0 to 3.0, and there was 100% mortality in sediments with ratios >10). The sum of the IWTUs for cadmium and nickel ranged from 0.08 to 43.5. Sediments with ≤0.5 IWTUs were always nontoxic, those with >2.2 IWTUs were always toxic, and two of seven sediments with intermediate IWTUs (0.5 to 2.2) were toxic. Molar concentrations of cadmium and nickel in the interstitial water were similar. However, cadmium contributed over 95% to the sum of the toxic units because cadmium is 67 times more toxic to *A. abdita* than nickel. The latter illustrates the utility of interstitial water concentrations of individual metals in assigning the probable cause of mortality in benthic species (Hansen et al., 1996a).

In tests with the same sediments from Foundry Cove, Pesch et al. (1995) observed that 6 of the 17 sediments tested had SEM/AVS ratios <1.0 and IWTUs <0.5, and none of the 6 were toxic to the polychaete *N. arenaceodentata*. Interestingly, the other 11 sediments containing SEM/AVS ratios >1.0 were also not toxic. The results are not surprising given that in these particular tests only one sediment had >0.5 IWTUs, *N. arenaceodentata* is not sensitive to cadmium and nickel, and the polychaetes did not burrow into sediments containing toxic concentrations of these metals.

Ankley et al. (1993) examined the significance of AVS as a binding phase for copper in freshwater sediments from two copper-impacted sites. Based on interstitial water copper concentrations in the test sediments, the 10-day LC50 for *H. azteca* was 31 μg/L; this compared favorably with a measured LC50 of 28 μg/L in a 10-day water-only test. Sediments having SEM/AVS ratios <1.0 were not toxic. They also observed no toxicity in several sediments with markedly more SEM than AVS, suggesting that copper was not biologically available in these sediments. Absence of copper in interstitial water from these sediments corroborated this lack of bioavailability. This observation suggested the presence of binding phases in addition to AVS for copper in the test sediments. Two studies suggest that an important source of the extra binding capacity in these sediments was organic carbon (U.S. EPA, 1994a; Mahony et al., 1996).

Hansen et al. (1996a) investigated the biological availability of sediment-associated divalent metals to *A. abdita* and *H. azteca* in sediments from five saltwater locations and one freshwater location in the United States, Canada, and China using 10-day lethality tests. Sediment toxicity was not related to dry weight metals concentrations. In the locations where metals might be likely to cause toxicity, 49 sediments had less SEM than AVS and <0.5 IWTUs, and no toxicity was observed. In contrast, one-third of the 45 sediments with more SEM than AVS and >0.5 IWTUs were toxic (Table 3-1).

Hansen et al. (1996a) made an observation that is important to interpretation of toxicity of sediments from field locations, particularly those from industrial harbors. They observed that if sediments with SEM/AVS ratios <1.0 are toxic, even if metals concentrations on a dry weight basis are very high, the toxicity is not likely to be caused by metals. Furthermore, it is incorrect to use such data to reach the conclusion that the EqP approach is not valid. This is because when SEM/AVS ratios were <1.0, there was an almost complete absence of toxicity in both spiked sediments and field sediments where metals were the only known source of contamination and IWTUs for metals were <0.5. When metals concentrations expressed as the sum of the IWTUs are used in conjunction with SEM/AVS ratios, they together provide insight that can explain apparent anomalies between SEM/AVS ratios <1.0 and sediment toxicity in field sediments. Joint use of both SEM/AVS ratios and interstitial water concentrations is also a powerful tool for explaining absence of toxicity when SEM/AVS ratios are >1.0. Overall, when freshwater and saltwater field sediments were tested in the laboratory, 100% were not toxic when SEM/AVS was ≤1.0 and IWTUs were <0.5, and 66.7% were toxic

when SEM/AVS was >1.0 and IWTUs were ≥0.5 (Table 3-1).

Therefore, because AVS can bind divalent metals in proportion to their molar concentrations, Hansen et al. (1996a) proposed the use of the difference between the molar concentrations of SEM and AVS (SEM-AVS) rather than SEM/AVS ratios used previously. The molar difference provides important insight into the extent of additional available binding capacity and the magnitude by which AVS binding has been exceeded (Figure 3-5). Further, absence of organism response when AVS binding is exceeded can indicate the potential magnitude of other important binding phases in controlling bioavailability. Figure 3-5 shows that for most nontoxic freshwater and saltwater field sediments, 1 to 100 μmol of additional metal would be required to exceed the sulfide-binding capacity (i.e., SEM-AVS = −100 to −1 μmol/g). In contrast, most toxic field sediments contained 1 to 1,000 μmol of metal beyond the binding capacity of sulfide alone. Data on nontoxic field sediments whose sulfide-binding capacity is exceeded (SEM-AVS is >1.0 μmol/g) indicate that other sediment phases, in addition to AVS, have significance in controlling metal bioavailability. In comparison to SEM/AVS ratios, use of SEM-AVS differences is particularly informative where AVS concentrations are low, such as those from Steilacoom Lake and the Keweenaw Watershed, where the SEM-AVS difference is numerically low and SEM/AVS ratios are high (Ankley et al., 1993). For these reasons, SEM-AVS is used instead of the SEM/AVS ratio almost exclusively for the remainder of this document.

3.2.4 *Field Sites and Spiked Sediments Combined*

Figure 3-6 and Table 3-1 summarize available data from freshwater and saltwater sediments spiked with individual metals or metal mixtures, freshwater field sites, and saltwater field sites on the utility of metals concentrations in sediments normalized by dry weight, IWTUs, and SEM-AVS. These data explain the

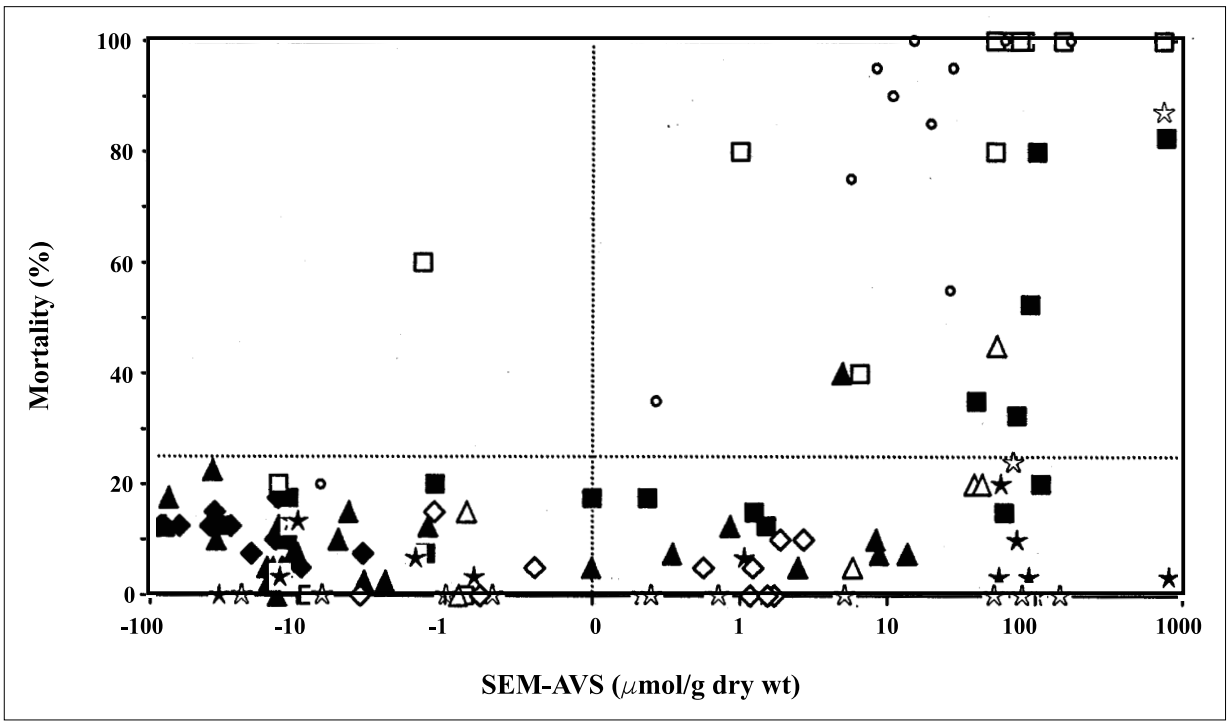

Figure 3-5. Percentage mortality of amphipods, oligochaetes, and polychaetes exposed to sediments from four freshwater and three saltwater field locations as a function of the sum of the molar concentrations of SEM minus the molar concentration of AVS (SEM-AVS). Sediments having ≤24% mortality are considered nontoxic as defined by Berry et al. (1996), which is indicated by the horizontal dotted line in the figure. The vertical dotted line at SEM-AVS = 0.0 μmol/g dry wt indicates the boundary between sulfide-bound unavailable metal and potentially available metal. The different symbols represent field sediments from different locations (figure from Hansen et al., 1996a).

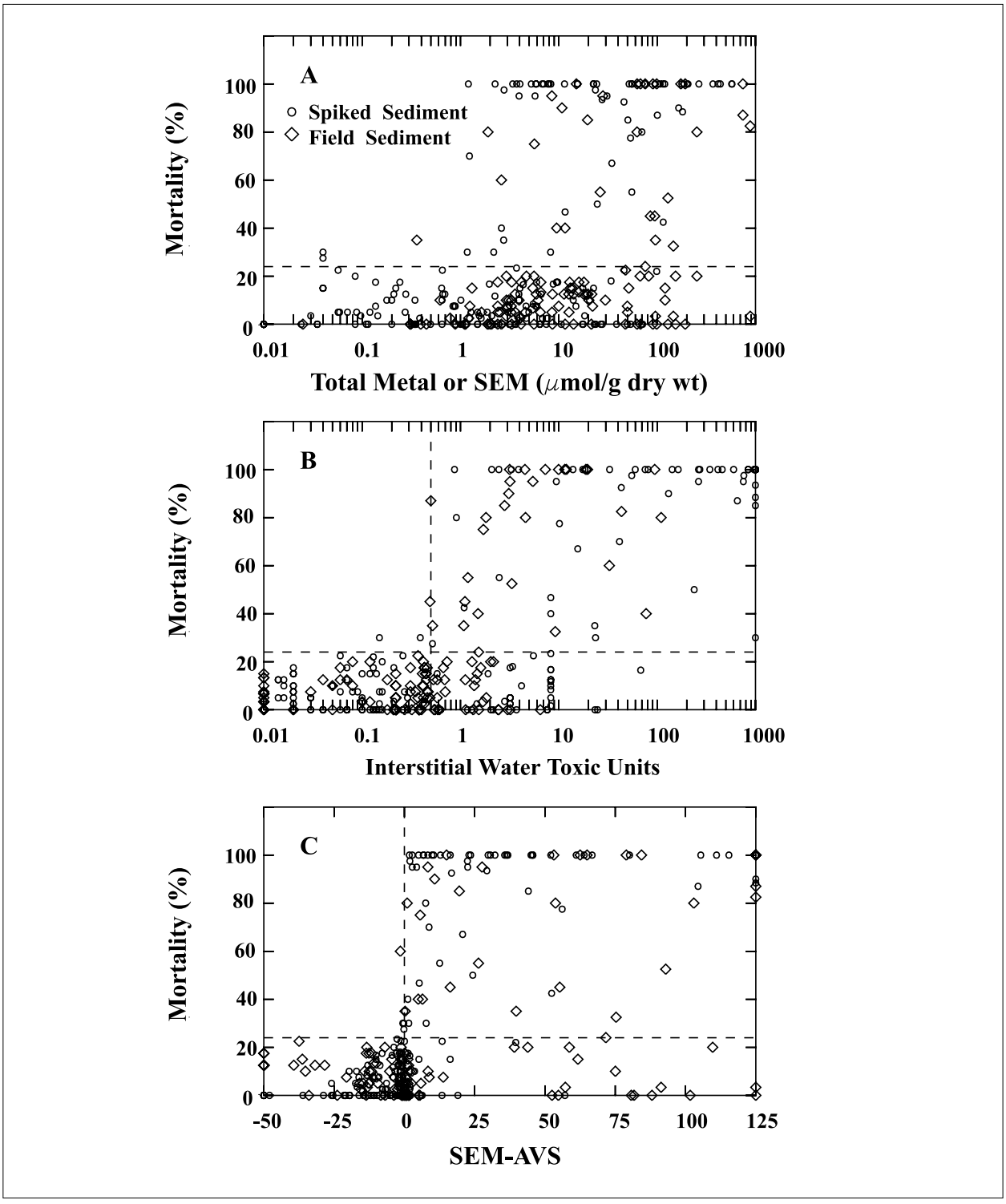

Figure 3-6. Percentage mortality of freshwater and saltwater benthic species in 10-day toxicity tests in spiked sediments and sediments from the field. Mortality is plotted as a function of: (A) the sum of the concentrations of the respective metal (Cd, Cu, Pb, Ni, Ag, or Zn) or metal mixture in μmol metal per gram dry weight of sediment; (B) IWTU; and (C) SEM-AVS difference. Data below the detection limits are plotted at IWTU = 0.01 and SEM-AVS = -50 μmol/g dry wt (silver data from Berry et al., 1999; all other data modified after Hansen et al., 1996a).

bioavailability and acute toxicity of metals in sediments (Hansen et al., 1996a; Berry et al., 1999). This analysis contains all available data from 10-day lethality tests where mortality, IWTUs, SEM, and AVS are known from experiments with sediments toxic only because of metals. The relationship between benthic organism mortality and total dry weight metals concentrations in spiked and field sediments is not useful to causally relate metal concentrations to organism response (Figures 3-4A and 3-6A). The overlap is almost four orders of magnitude in the bulk metals concentrations that cause no toxicity and those that are 100% lethal for these sediments where metals are the only source of toxicity (see discussion in Section 3.2.2).

Data in Figure 3-6B show that over all tests, the toxicity of sediments whose concentrations are normalized on an IWTU basis are typically consistent with the IWTU concept; that is, if IWTUs are $\leq$1.0, then sediments should be lethal to $\leq$50% of the organisms exposed, and significant mortality probably should be absent at <0.5 IWTUs. Of the spiked and field sediments evaluated that had IWTUs <0.5, 97.7% of 175 sediments were nontoxic (Table 3-1). For the 142 sediments having IWTUs $\geq$0.5, 68.3% were toxic. However, and as stated above, given the effect on toxicity or bioavailability of the presence of other binding phases (e.g., DOC) in interstitial water, water quality (hardness, salinity, etc.), and organism behavior, it is not surprising that many sediments having IWTUs $\geq$0.5 are not toxic.

Data in Figure 3-6C show that over all tests, organism response in sediments whose concentrations are normalized on an SEM-AVS basis is consistent with metal-sulfide binding on a mole to mole basis as first described by Di Toro et al. (1990), and later recommended for assessing the bioavailability of metals in sediments by Ankley et al. (1994). Saltwater and freshwater sediments either spiked with metals or from field locations with SEM-AVS differences $\leq$0.0 were uniformly nontoxic (98.1% of 158 sediments) (Table 3-1). The majority (58.0%) of 174 sediments having SEM-AVS >0.0 were toxic. It is not surprising that many sediments having SEM-AVS >0.0 are not toxic given the effect on toxicity or bioavailability of the presence of other sediment phases that also affect bioavailability (see Section 3-4; Di Toro et al., 1987, 2000; Mahony et al., 1996).

Over all tests, the data in Figure 3-6 indicate that use of both IWTUs and SEM-AVS together did not improve the accuracy of predictions of sediments that were nontoxic (98.5% of 132 sediments; Table 3-1). However, it is noteworthy that 78.9% of the 123 sediments with both SEM-AVS >0.0 and IWTUs $\geq$0.5 were toxic. Therefore, the approach of using SEM-AVS, IWTUs, and especially both indicators to identify sediments of concern is very useful.

The results of all available data demonstrate that using SEM, AVS, and interstitial water metals concentrations to predict the lack of toxicity of cadmium, copper, lead, nickel, silver, and zinc in sediments is certain. This is very useful, because the vast majority of sediments found in the environment in the United States have AVS concentrations that exceed the SEM concentration (SEM-AVS <0.0) (see Section 4.4). This may incorrectly suggest that there should be little concern about metals in sediments on a national basis, even though localized areas of biologically significant metal contamination do exist (Wolfe et al., 1994; Hansen et al., 1996a; Leonard et al., 1996a). It is potentially important that most of these data are from field sites where sediment samples were collected in the summer. At this time of year, the seasonal cycles of AVS produce the maximum metal-binding potentials (Boothman and Helmstetter, 1992; Leonard et al., 1993). Hence, sampling at seasons and conditions when AVS concentrations are at a minimum is a must in establishing the true overall level of concern about metals in the nation's sediments and in evaluations of specific sediments of local concern.

Predicting which sediments with SEM-AVS >0.0 will be toxic is presently less certain. Importantly, the correct classification rate seen in these experiments is high; that is, the accuracy of predicting which sediments were toxic was 58.0% using the SEM and AVS alone, 68.3% using IWTUs, and 78.9% using both indicators. An SEM-AVS >0.0, particularly at multiple adjacent sites, should trigger additional tiered assessments. These might include characterization of the spatial (both vertical and horizontal) and temporal distribution of chemical concentration (AVS and SEM) and toxicity, measurements of interstitial water metal, and toxicity identification evaluations (TIEs). In this context, the combined SEM-AVS and IWTU approach should be viewed as only one of the many sediment evaluation methodologies.

3.2.5 *Conclusions from Short-Term Studies*

Results from tests using sediments spiked with metals and sediments from the field in locations where toxicity is associated with metals demonstrate the value of explaining the biological availability of metals

concentrations normalized by SEM-AVS and IWTUs instead of dry weight metal concentrations. Importantly, data from spiked-sediment tests strongly indicate that metals are not the cause of most of the toxicity observed in field sediments when both SEM-AVS is $\leq$0.0 and IWTUs are <0.5 (Table 3-1). Expressing concentrations of metals in sediments on an SEM-AVS basis provides important insight into the available additional binding capacity of sediments and the extent to which sulfide binding has been exceeded.

SEM-AVS and interstitial water concentrations of metals can aid in identifying the specific metal causing toxicity. For example, the metal(s) in excess of AVS can be identified by subtracting from the molar concentration of AVS the molar concentrations of specific metals in the SEM in order of their sulfide solubility product constants ($K_{sp,2}$) in the SEM. Alternatively, interstitial water concentrations of metals can be used to identify a specific metal causing sediment toxicity using the toxic unit concept, if appropriate water-only toxicity data for the tested species are available (Hansen et al., 1996a).

Predictions of sediments not likely to be toxic, based on use of SEM-AVS and IWTUs for all data from freshwater or saltwater field sediment and spiked-sediment tests, are extremely accurate (98.5%) using both parameters. Predictions of sediments likely to be toxic are less accurate. Nevertheless, SEM-AVS is extremely useful in identifying sediments of potential concern. Data were summarized from amphipod tests using freshwater and saltwater laboratory metals-spiked sediments and field sediments where metals were a known problem by comparing the percentage of sediments that were toxic with the SEM-AVS concentration (tests with polychaetes and gastropods were excluded because these organisms avoid exposure) (Hansen, 1995). Seventy percent of the sediments in these amphipod studies with an SEM-AVS concentration of $\geq$0.76 μmol of excess SEM/g were toxic. The corresponding values for 80%, 90%, and 100% of the sediments being toxic were 2.7, 16, and 115 μmol of excess SEM/g, respectively.

Of course, SEM, AVS, and IWTUs can only predict toxicity or the lack of toxicity caused by *metals* in sediments. They cannot be used alone to predict toxicity of sediments contaminated with toxic concentrations of other contaminants. However, SEM and AVS have been used in sediment assessments to rule out metals as probable causative agents of toxicity (Wolfe et al., 1994). Also, the use of SEM and AVS to predict biological availability and toxicity of cadmium, copper, lead, nickel, silver, and zinc is applicable only to anaerobic sediments that contain AVS; binding factors other than AVS control bioavailability in aerobic sediments (Di Toro et al., 1987; Tessier et al., 1993). Measurement of interstitial water metal may be useful for evaluations of these and other metals in aerobic and anaerobic sediments (Ankley et al., 1994). Even with these caveats, the combined use of SEM, AVS, and interstitial measurements is preferable to all other currently available sediment evaluation procedures to causally assess the implications to benthic organisms of these six metals associated with sediments (see discussion in Section 5, Sampling and Analytical Chemistry, for further guidance).

3.3 Predicting Metal Toxicity: Long-Term Studies

Taken as a whole, the short-term laboratory experiments with metal-spiked and field-collected sediments present a strong argument for the ability to predict the absence of metal toxicity based on sediment SEM and AVS relationships and/or interstitial water metal concentrations. However, if this approach is to serve as a valid basis for ESB derivation, comparable predictive success must be demonstrated in long-term laboratory and field experiments where chronic effects could be manifested (Luoma and Carter, 1993; Meyer et al., 1994). This demonstration was the goal of experiments described by Hare et al. (1994), DeWitt et al. (1996), Hansen et al. (1996b), Liber et al. (1996), and Sibley et al. (1996). An important experimental modification to these long-term studies, as opposed to the short-term tests described in Section 3.2, was the collection of horizon-specific chemistry data. This is required because AVS concentrations often increase, and SEM-AVS differences decrease, with an increase in sediment depth (Howard and Evans, 1993; Leonard et al., 1996a); hence, chemistry performed on homogenized samples might not reflect the true exposure of benthic organisms dwelling in surficial sediments (Luoma and Carter, 1993; Hare et al., 1994; Peterson et al., 1996).

3.3.1 *Life-Cycle Toxicity Tests*

DeWitt et al. (1996) conducted an entire life-cycle toxicity test with the marine amphipod *L. plumulosus* exposed for 28 days to cadmium-spiked estuarine sediments (Table 3-2). The test measured effects on survival, growth, and reproduction of newborn amphipods relative to interstitial water and SEM/AVS

Table 3-2. Summary of the results of full life-cycle and colonization toxicity tests conducted in the laboratory and field using sediments spiked with individual metals and metal mixtures

Toxicity Test	Metal(s)	Duration (days)	Measured SEM-AVS[a] (μmol/g) NOEC(s)[b]	OEC(s)[c]	Effect	Reference
Life Cycle:						
Leptocheirus plumulosus	Cadmium	28	-3.5, -2.0, 0.78, 2.0	8.9, 15.6	Mortality 100%	DeWitt et al., 1996
Chironomus tentans	Zinc	56	-2.6, -1.4, 6.4	21.9, 32.4	Larval mortality 85%-100% Weight, emergence, and reproduction reduced	Sibley et al., 1996
Colonization:						
Laboratory-saltwater	Cadmium	118	-13.4	8.0, 27.4	Fewer polychaetes, shifts in community composition, fewer species, bivalves absent, tunicates increased	Hansen et al., 1996b
Field-saltwater	Cadmium, copper, lead, nickel, zinc	120	-0.31, -0.06, 0.02	—	No effects observed	Boothman et al., 2001
Field-freshwater	Cadmium	~365	-0.07, 0.08, 0.34	2.2	Reduced *Chironomus salinarius* numbers Bioaccumulation	Hare et al., 1994
Field-freshwater	Zinc	368	-3.6, -3.5, -2.9, -2.0, 1.0[d]	—	No effects observed	Liber et al., 1996

[a]SEM-AVS differences are used instead of SEM/AVS ratios to standardize across the studies referenced. An SEM-AVS difference of ≤0.0 is the same as an SEM/AVS ratio of ≤1.0. An SEM-AVS difference of >0.0 is the same as an SEM/AVS ratio of >1.0.
[b]NOECs = no observed effect concentration(s); all concentrations where response was not significantly different from the control.
[c]OECs = observed effect concentration(s); all concentrations where response was significantly different from the control.
[d]Occasional minor reductions in oligochaetes (Naididae).

normalization. Seven treatments of Cd were tested: 0 (control), −3.5, −2.0, 0.78, 2.0, 8.9, and 15.6 SEM_{Cd}-AVS differences (measured concentrations). Gradients in AVS concentration as a function of sediment depth were greatest in the control treatment, decreased as the SEM_{Cd} ratio increased, and became more pronounced over time. Depth gradients in SEM_{Cd}-AVS differences were primarily caused by the spatial and temporal changes in AVS concentration, because SEM_{Cd} concentrations changed very little with time or depth. Thus in most treatments SEM_{Cd}-AVS differences were smaller at the top of sediment cores than at the bottom. This is expected because the oxidation rate of iron sulfide in laboratory experiments is very rapid (100% in 60 to 90 minutes) but for cadmium sulfide it is slow (10% in 300 hours) (Mahony et al., 1993; Di Toro et al., 1996a). Interstitial cadmium concentrations increased in a dramatic stepwise fashion in treatments having a SEM-AVS difference of ≥8.9 μmol of excess SEM, but were below the 96-hour LC50 value for this amphipod in lesser treatments. There were no significant effects on survival, growth, or reproduction in sediments containing more AVS than cadmium (−3.5 and −2.0 μmol/g) and those with a slight excess of SEM_{Cd} (0.78 and 2.0 μmol/g), in spite of the fact that these samples contained from 183 to 1,370 μg cadmium/g sediment. All amphipods died in sediments having SEM-AVS differences ≥8.9 μmol excess SEM/g. These results are

consistent with predictions of metal bioavailability from 10-day acute tests with metal-spiked sediments (i.e., that sediments with SEM_{Cd}-AVS differences $\leq$0.0 are not toxic, interstitial water metal concentrations are related to organism response, and sediments with SEM_{Cd}-AVS differences >0.0 may be toxic).

Sibley et al. (1996) reported similar results from a 56-day life-cycle test conducted with the freshwater midge *C. tentans* exposed to zinc-spiked sediments (Table 3-2). The test was initiated with newly hatched larvae and lasted one complete generation, during which survival, growth, emergence, and reproduction were monitored. In sediments where the molar difference between SEM and AVS (SEM-AVS) was <0.0 (dry weight zinc concentrations were as high as 270 mg/kg), concentrations of zinc in the sediment interstitial water were low and no adverse effects were observed for any of the biological endpoints measured. Conversely, when SEM-AVS was 21.9 and 32.4 μmol of excess SEM/g, interstitial water concentrations of zinc increased (being highest in surficial sediments), and reductions in survival, growth, emergence, and reproduction were observed. Over the course of the study, the absolute concentration of zinc in the interstitial water in these treatments decreased because of the increase in sediment AVS and loss of zinc from twice-daily renewals of the overlying water.

3.3.2 *Colonization Tests*

Hansen et al. (1996b) conducted a 118-day benthic colonization experiment in which sediments were spiked to achieve nominal cadmium/AVS molar ratios of 0.0 (control), 0.1, 0.8, and 3.0 and then held in the laboratory in a constant flow of unfiltered seawater (Table 3-2). Oxidation of AVS in the surficial 2.4 cm of the control treatment occurred within 2 to 4 weeks and resulted in sulfide profiles similar to those occurring in sediments in nearby Narragansett Bay, RI (Boothman and Helmstetter, 1992). In the nominal 0.1 cadmium/AVS treatment, measured SEM_{Cd} was always less than AVS (SEM-AVS = $-$13.4 μmol AVS/g in the surficial 2.0 cm), interstitial cadmium concentrations (<3 to10 μg/L) were less than those likely to cause biological effects, and no significant biological effects were detected. In the nominal 0.8 cadmium/AVS treatment (SEM-AVS = 8.0 μmol SEM/g), measured SEM_{Cd} commonly exceeded AVS in the surficial 2.4 cm of sediment, and interstitial cadmium concentrations (24 to 157 μg/L) were sufficient to be of toxicological significance to highly sensitive species. In this treatment, shifts in the presence or absence of organisms were observed over all taxa, and there were fewer macrobenthic polychaetes (*Mediomastus ambiseta*, *Streblospio benedicti*, and *Podarke obscura*) and meiofaunal nematodes. In the nominal 3.0 cadmium/AVS treatment (SEM-AVS of 27.4 μmol SEM/g), concentrations of SEM_{Cd} were always greater than AVS throughout the sediment column. Interstitial cadmium ranged from 28,000 to 174,000 μg/L. In addition to the effects observed in the nominal 0.8 cadmium/AVS treatment, the following effects were observed: (a) sediments were colonized by fewer macrobenthic and polychaete species and harpacticoids, (b) the sediments had lower densities of diatoms, and (c) bivalve molluscs were absent. Over all treatments, the observed biological responses were consistent with predicted possible adverse effects resulting from elevated SEM_{Cd}-AVS differences in surficial sediments and interstitial water cadmium concentrations.

Boothman et al. (2001) conducted a field colonization experiment in which sediments from Narragansett Bay, RI, were spiked with an equimolar mixture of cadmium, copper, lead, nickel, and zinc at nominal SEM/AVS ratios of 0.1, 0.8, and 3.0; placed in boxes; and replaced in Narragansett Bay (Table 3-2). The AVS concentrations decreased with time in surface sediments (0 to 3 cm) in all treatments where the nominal SEM/AVS ratio was <1.0 (SEM-AVS decreased from $-$0.31 to $-$0.06 μmol SEM/g in the surficial 2.0 cm) but did not change in subsurface (6 to 10 cm) sediments or in the entire sediment column where nominal SEM/AVS ratios exceeded 1.0 (SEM-AVS = 0.02 μmol AVS/g). SEM decreased with time only where SEM exceeded AVS. The concentration of metals in interstitial water was below detection limits when there was more AVS than SEM. When SEM exceeded AVS, significant concentrations of metals were present in interstitial water, and appeared in the order of their sulfide solubility product constants. Interstitial water concentrations in these sediments decreased with time, although they exceeded the WQC in interstitial water for 60 days for all metals, 85 days for cadmium and zinc, and 120 days for the entire experiment for zinc. Benthic faunal assemblages in the spiked-sediment treatments were not different from those of the control treatment. Lack of biological response was consistent with the vertical profiles of SEM and AVS. AVS was greater than SEM in all surface sediments, including the top 2 cm of the 3.0 nominal SEM/AVS treatment, because of oxidation of AVS and loss of SEM. The authors speculated that interstitial metal was likely absent in the surficial sediments in spite of data demonstrating the presence of significant measured concentrations.

Interstitial water in the 3.0 nominal SEM/AVS treatment was sampled from sediment depths where SEM was in excess, rather than in the surficial sediments. Important to the biological data are the surficial sediments, where settlement by saltwater benthic organisms first occurs. Also, there was a storm event that allowed a thin layer of clean sediment to be deposited on top of the spiked sediment (W.S. Boothman, U.S. EPA, Narragansett, RI, personal communication). These data demonstrate the importance of sampling sediments and interstitial water in sediment horizons where benthic organisms are active.

Hare et al. (1994) conducted an approximately 1-year field colonization experiment in which uncontaminated freshwater sediments were spiked with cadmium and replaced in the oligotrophic lake from which they originally had been collected (Table 3-2). Cadmium concentrations in interstitial waters were very low at cadmium-AVS molar differences <0.0, but increased markedly at differences >0.0. The authors reported reductions in the abundance of only the chironomid *Chironomus salinarius* in the 2.2 μmol excess SEM/g treatment. Cadmium was accumulated by organisms from sediments with surficial SEM concentrations that exceeded those of AVS. These sediments also contained elevated concentrations of cadmium in interstitial water.

Liber et al. (1996) performed a field colonization experiment using sediments having 4.46 μmol of sulfide from a freshwater mesotrophic pond (Table 3-2). Sediments were spiked with 0.8, 1.5, 3.0, 6.0, and 12.0 μmol of zinc, replaced in the field, and chemically and biologically sampled over 12 months. There was a pronounced increase in AVS concentrations with increasing zinc concentration; AVS was lowest in the surficial 0 to 2 cm of sediment with minor seasonal variations. With the exception of the highest spiking concentration (approximately 700 mg/kg, dry weight), AVS concentrations remained larger than those of SEM. Interstitial water zinc concentrations were rarely detected in any treatment, and were never at concentrations that might pose a hazard to benthic macroinvertebrates. The only observed difference in benthic community structure across the treatments was a slight decrease in the abundance of Naididae oligochaetes at the highest spiking concentration. The absence of any noteworthy biological response was consistent with the absence of interstitial water concentrations of biological concern. The lack of biological response was attributed to an increase in concentrations of iron and manganese sulfides produced during periods of diagenesis, which were replaced by the more stable zinc sulfide, which is less readily oxidized during winter months. In this experiment, and theoretically in nature, excesses of sediment metal might be overcome over time because of the diagenesis of organic material. In periods of minimal diagenesis, oxidation rates of metal sulfides, if sufficiently great, could release biologically significant concentrations of the metal into interstitial waters. The phenomenon should occur metal by metal in order of their sulfide solubility product constants.

3.3.3 *Conclusions from Chronic Studies*

Over all full life-cycle and colonization toxicity tests conducted in the laboratory and field using sediments spiked with individual metals and metal mixtures (Table 3-2), no sediments with an excess of AVS (SEM-AVS $\leq$ 0.0) were toxic (Figure 3-7). Conversely, all sediments where chronic effects were observed, and 7 of 19 sediments where no effects were observed, had an excess of SEM (SEM-AVS >0.0) (Table 3-2; Figure 3-7). Therefore, the results from all available acute and chronic toxicity tests support the use of SEM-AVS $\leq$ 0.0 as an ESB that can be used to predict sediments that are unlikely to be toxic.

3.4 Predicting Toxicity of Metals in Sediments

3.4.1 *General Information*

The SEM-AVS method for evaluating toxicity of metals in sediments (Di Toro et al., 1990, 1992) has proven to be successful at predicting the lack of metal toxicity in spiked and field-contaminated sediments (Berry et al., 1996; Hansen et al., 1996a). However, because SEM-AVS does not explicitly consider the other sediment phases that influence interstitial water–sediment partitioning, and in spite of its utility in identifying sediments of possible concern, it was never intended to be used to predict the occurrence of toxicity. The proposed sediment quality criteria for metals using SEM, AVS, and IWTUs in Ankley et al. (1996)—now referred to as ESBs or equilibrium partitioning sediment benchmarks—were constructed as "one-tailed" guidelines. They should be used to predict the lack of toxicity but not its presence. Thus the problem of predicting the onset of toxicity in metal-contaminated sediments remained unsolved.

This section introduces a modification of the SEM-AVS procedure in which the SEM-AVS difference is

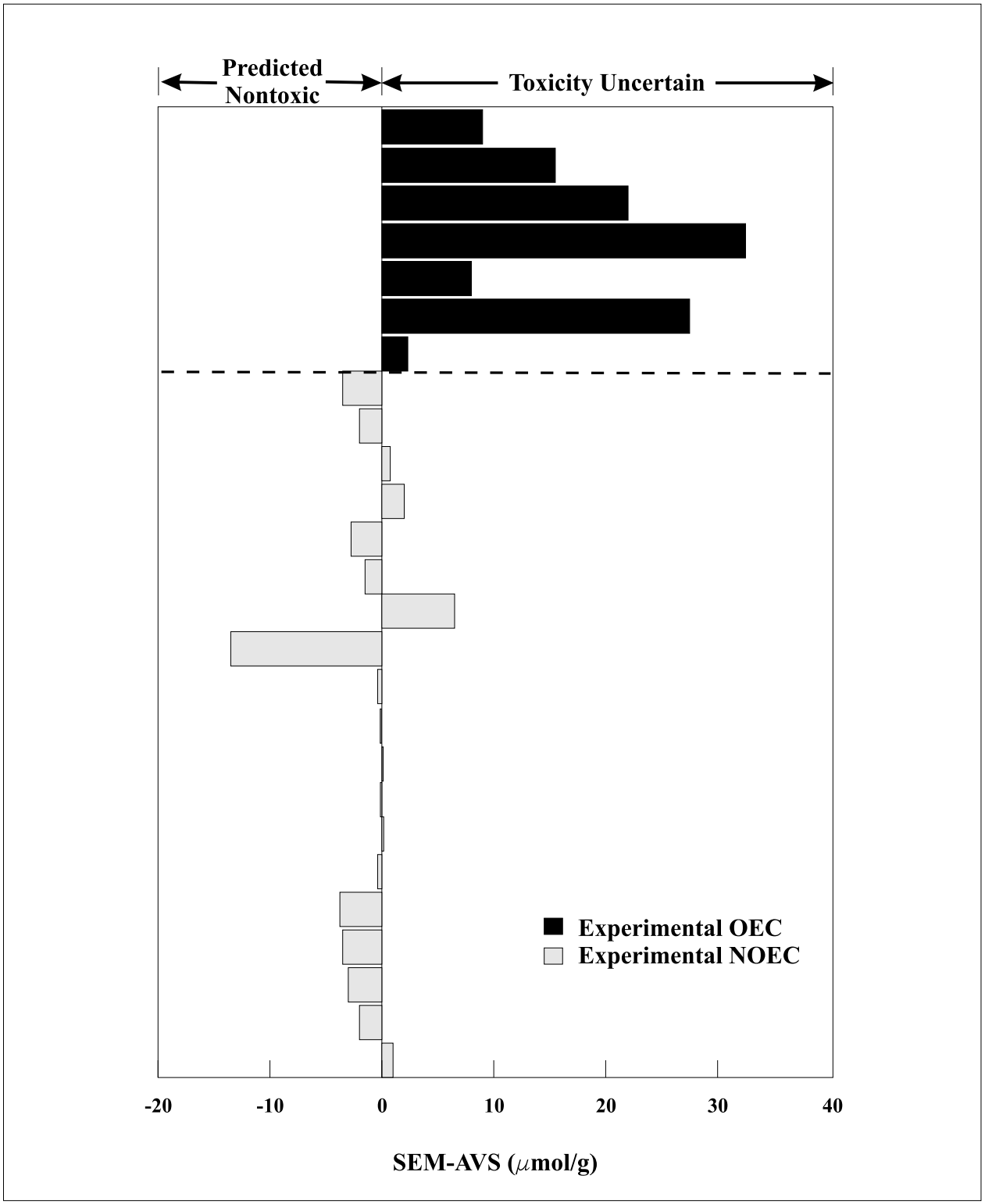

Figure 3-7. **Comparison of the chronic toxicity of sediments spiked with individual metals or metal mixtures to predicted toxicity based on SEM-AVS (data from Table 3-2). Horizontal dashed line separates experimental observed effect concentrations (solid columns) from no observed effect concentrations (shaded columns). Values at SEM-AVS ≤ 0.0 μmol/g_{OC} are predicted to be nontoxic. Values at SEM-AVS >0.0 μmol/g_{OC} are indicative of sediments that are likely to be toxic or toxicity is uncertain.**

normalized by the fraction of organic carbon, f_{OC}, in a sediment. This section is largely taken from Di Toro et al. (2000). Their publication should be consulted for additional information about the utility of the f_{OC} procedure and comparison of this procedure with the sediment guidelines of Long et al. (1995a) and MacDonald et al. (1996). The $(\Sigma SEM\text{-}AVS)/f_{OC}$ procedure significantly improves prediction of mortality by accounting for partitioning of metals to sediment organic carbon, as well as the effect of AVS. In addition, the approach used by Di Toro et al. (2000) to derive $(\Sigma SEM\text{-}AVS)/f_{OC}$ uncertainty bounds for identifying sediments that are likely to be toxic, are of uncertain toxicity, or are nontoxic has applicability to SEM/AVS ratios, SEM-AVS differences, and IWTUs. Although not used as an ESB, the uncertainty bounds should be useful in prioritizing sediments of concern for further evaluations.

3.4.2 *EqP Theory for SEM, AVS, and Organic Carbon*

The EqP model provides for the development of causal sediment concentrations that predict toxicity or lack of toxicity in sediments (Di Toro et al., 1991). The sediment concentration C_S that corresponds to a measured LC50 in a water-only exposure of the test organism is

$$C_S^* = K_p \, LC50 \tag{3-2}$$

where C_S^* is the sediment LC50 concentration (μg/kg dry wt), K_p (L/kg) is the partition coefficient between interstitial water and sediment solids, and LC50 is the concentration causing 50% mortality (μg/L). For application to metals that react with AVS to form insoluble metal sulfides, Equation 3-2 becomes

$$C_S^* = AVS + K_p \, LC50 \tag{3-3}$$

where AVS is the sediment concentration of acid volatile sulfides. Equation 3-3 simply states that because AVS can bind the metal as highly insoluble sulfides, the concentration of metal in a sediment that will cause toxicity is at least as great as the AVS that is present. The sediment metal concentration that should be employed is the SEM concentration, because any metal that is bound so strongly that 1N of hydrochloric acid cannot dissolve it is not likely to be bioavailable (Di Toro et al., 1992). Of course, this argument is theoretical, which is why so much effort has been expended to demonstrate experimentally that this is actually the case (Di Toro et al., 1992; Hare et al., 1994; Berry et al., 1996; Hansen et al., 1996a; Sibley et al., 1996). Therefore, the relevant sediment metal concentration is SEM, and Equation 3-3 becomes

$$SEM = AVS + K_p \, LC50 \tag{3-4}$$

The basis for the AVS method is to observe that if the second term in Equation 3-4 is neglected, then the critical concentration is SEM = AVS, and the criterion for toxicity or lack of toxicity is SEM-AVS $\leq$ 0.0 (μmol/g dry wt).

The failure of the difference to predict toxicity when there is an excess of SEM is due to neglect of the partitioning term K_pLC50. Note that ignoring the term does not affect the prediction of lack of toxicity in that it makes the condition conservative (i.e., smaller concentrations of SEM are at the boundary of toxicity and no toxicity).

The key to improving prediction of toxicity is to approximate the partitioning term rather than ignore it (Di Toro et al, *in prep.*). In sediments, the organic carbon fraction is an important partitioning phase, and partition coefficients for certain metals at certain pHs have been measured (Mahony et al., 1996). This suggests that the partition coefficient K_p in Equation 3-4 can be expressed using the organic carbon–water partition coefficient, K_{OC}, together with the fraction organic carbon in the sediment, f_{OC}

$$K_p = f_{OC} \, K_{OC} \tag{3-5}$$

Using this expression in Equation 3-4 yields

$$SEM = AVS + f_{OC} \, K_{OC} \, LC50 \tag{3-6}$$

Moving the known terms to the left side of this equation yields

$$\frac{SEM - AVS}{f_{OC}} = K_{OC} LC50 \tag{3-7}$$

If both K_{OC} and LC50 are known, then Equation 3-7 can be used to predict toxicity.

The method evaluated below uses $(\Sigma SEM\text{-}AVS)/f_{OC}$ as the predictor of toxicity and evaluates the critical concentrations (the right side of Equation 3-7) based on observed SEM, AVS, f_{OC}, and toxicity data (Di Toro et al, *in prep.*). If multiple metals are present, it is necessary to use the total SEM

$$\Sigma SEM = \sum_{i=1}^{N} [SEM_i] \tag{3-8}$$

to account for all the metals present. Note that (ΣSEM-AVS)/f_{OC} is the organic carbon–normalized excess SEM for which we use the notation

$$\left(\sum \text{SEM} - \text{AVS}\right)/f_{OC} = \left(\sum \frac{\text{SEM} - \text{AVS}}{f_{OC}}\right) \qquad (3\text{-}9)$$

3.4.3 *Data Sources*

Data from toxicity tests using both laboratory-spiked and field-collected sediments were compiled from the literature. Four sources of laboratory-spiked tests using marine sediments (Casas and Crecelius, 1994; Pesch et al., 1995; Berry et al., 1996, 1999) and one using freshwater sediments (Carlson et al., 1991) were included. Two sources for metal-contaminated field sediments were included (Hansen et al., 1996a; Kemble et al., 1994). The field data from the sediments where metals were not the probable cause of toxicity (Bear Creek and Jinzhou Bay) (Hansen et al., 1996a) were excluded. Data reported included total metals, SEM, AVS, f_{OC}, and 10- or 14-day mortality. In Hansen et al. (1996a), data were reported for five saltwater and four freshwater locations, but organic carbon concentrations were not available for freshwater field sediments from three locations. Organic carbon data for the Keweenaw Watershed were obtained separately (E.N. Leonard, U.S. EPA, Duluth, MN, personal communication).

Laboratory-spiked and field sediment data were grouped for analysis. Mortality data were compared against the SEM-AVS difference and the SEM-AVS difference divided by the f_{OC}. For each comparison, two uncertainty bounds were computed: a lower-bound concentration equivalent to a 95% chance that the mortality observed would be less than 24% (the percentage mortality considered to be toxic) (see Berry et al., 1996) and an upper-bound concentration equivalent to a 95% chance that the observed mortality would be greater than 24%. The lower-bound uncertainty limit was computed by evaluating the fraction of correct classification starting from the lowest x-axis value. When the fraction correct dropped to below 95%, the 95th percentile was interpolated. The same procedure was applied to obtain the upper-bound uncertainty limit. These uncertainty bounds are the concentration range where it is 90% certain that the sediment may be either toxic or not toxic.

3.4.4 *Acute Toxicity Uncertainty*

Mortality in the laboratory-spiked and field-contaminated sediment tests were both organism and metal independent when plotted against the SEM-AVS difference (Figure 3-8A). The horizontal dashed line indicating 24% mortality is shown for reference. The 90% lower and upper uncertainty bound limits for the SEM-AVS difference are from 1.7 and 120 μmol/g, a factor of 70. Thus, it appears that for both laboratory and spiked-sediment data, toxicity is likely when the SEM-AVS difference is >120, uncertain when the difference is from 1.7 to 120 μmol/g, and not likely when the difference is <1.7 μmol/g.

Although use of SEM-AVS differences to predict toxicity is not based on any theoretical foundation, use of SEM-AVS $\leq$0.0 to predict lack of toxicity is based on the equilibrium partitioning model (Di Toro et al., 1991) and the chemistry of metal–sulfide interactions. The stoichiometry of the uptake of divalent metals by AVS is such that 1 mol of AVS will stabilize 1 mol of SEM, except for silver, where the ratio is 2:1, hence the use of the difference of 0.0 μmol/g dry weight to predict lack of toxicity. In fact it is the very low solubility of the resulting metal sulfides that limits the interstitial water concentrations to below toxic levels regardless of the details of the sediment chemistry (e.g., pH, iron concentration) as has been demonstrated in this document and detailed in the Appendix in Di Toro et al. (1992).

The (ΣSEM-AVS)/f_{OC} approach provides an equivalent theoretical basis that is needed to derive an appropriately normalized sediment concentration that predicts occurrence of toxicity that is causally linked to bioavailable metal. When percent mortality is plotted against the organic carbon–normalized excess SEM (ΣSEM-AVS)/f_{OC}) for the same data as contained in Figure 3-8A, toxicity is likely when the (ΣSEM-AVS)/f_{OC} is >3,000 μmol/g_{OC}, uncertain when the concentration is between 130 and 3,000 μmol/g_{OC}, and not likely when the concentration is <130 μmol/g_{OC} (Figure 3-8B). Thus, the width of the uncertainty bound is a factor of 70 for SEM-AVS differences and 23 for (ΣSEM-AVS)/f_{OC}.

If the (ΣSEM-AVS)/f_{OC} approach improves predictions of sediment toxicity caused by metals, the uncertainty bounds should narrow and the percentages of sediments where toxicity predictions are uncertain should decrease. If the uncertainty bound analysis is not conducted, and SEM-AVS>0.0 is used as proposed in Sections 3.2 and 3.3, predictions of sediment toxicity

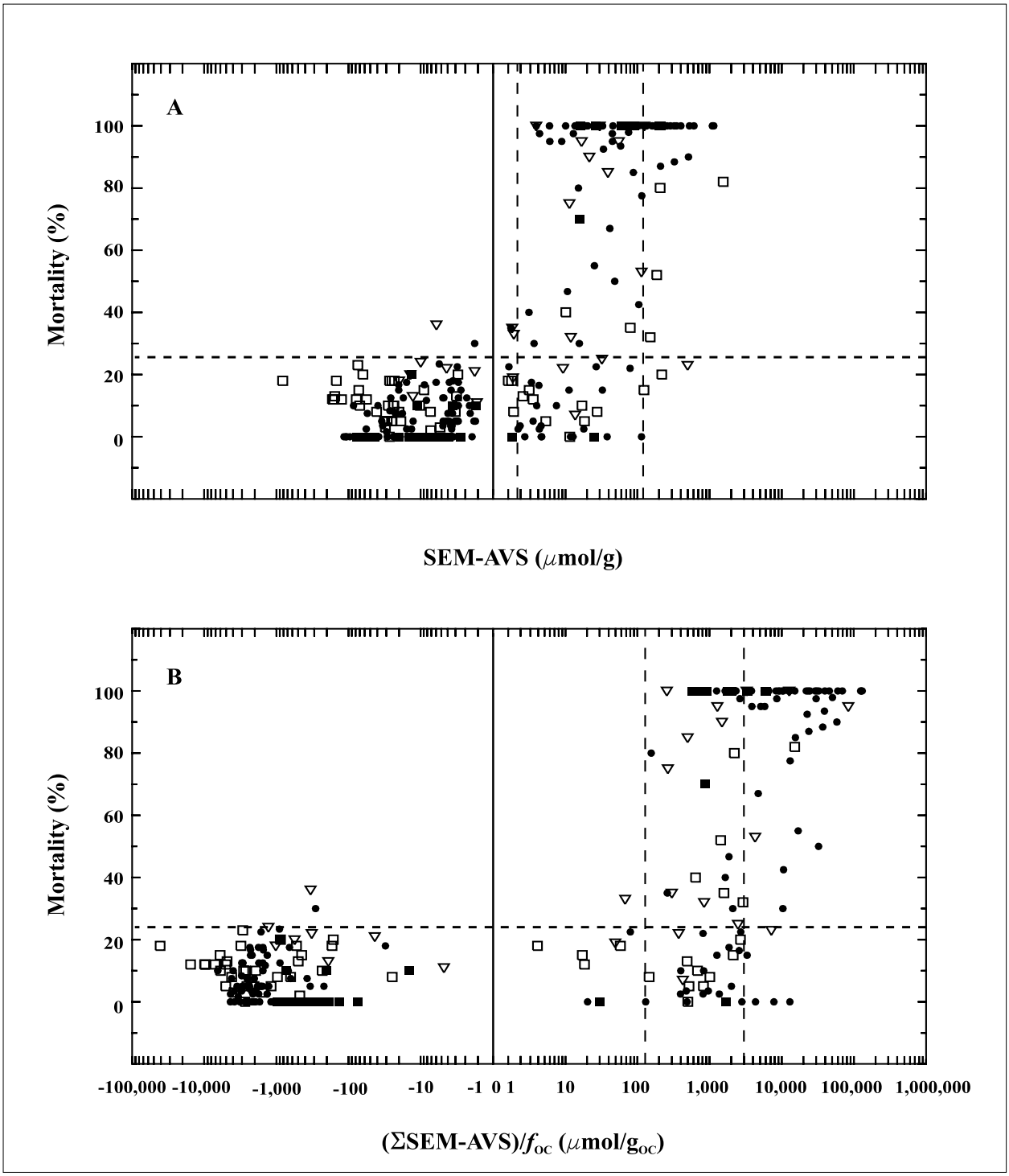

Figure 3-8. Percent mortality versus SEM-AVS (A) and (ΣSEM-AVS)/f_{oc} (B) for saltwater field data without Bear Creek and Jinzhou Bay (□), freshwater field data (▽), freshwater spiked data (≃), and saltwater spiked data (●); silver data excluded. Vertical dashed lines are the 90% uncertainty bound limits (figure from Di Toro et al., 2000).

for the 267 spiked sediments are classified as uncertain for 47.2% of the sediments. Using the uncertainty bounds on SEM-AVS of 1.7 to 120 μmol/g as described in this section results in reduction in the percentage of sediments where toxicity predictions are uncertain, to 34.1%. Use of $(\Sigma SEM\text{-}AVS)/f_{OC}$ with uncertainty bounds of 130 to 3,000 $\mu mol/g_{OC}$ results in further reduction in the percentage of sediments where toxicity predictions are uncertain, to 25.5%. Therefore, use of the uncertainty limits of the $(\Sigma SEM\text{-}AVS)/f_{OC}$ approach classifies 33.7% more sediments as toxic or nontoxic than using the uncertainty limits of SEM-AVS, and 85% more than use of SEM-AVS without uncertainty limits. This improvement highlights the advantages of using $(\Sigma SEM\text{-}AVS)/f_{OC}$ in assessing toxicity of metal-contaminated sediments.

Use of $(\Sigma SEM\text{-}AVS)/f_{OC}$ uncertainty limits applies to all the metals regardless of their identity. Figure 3-9 presents the spiked-sediment data categorized by identity of the metal. The field-contaminated data cannot be included because the identity of the metal causing toxicity cannot be unambiguously determined. There is no apparent difference for any of the metals in the region of overlapping survival and mortality data between 130 and 3,000 $\mu mol/g_{OC}$.

It is interesting to note that organic carbon normalization appears not to work for silver. The spiked-sediment test data are presented in Figure 3-10A (Berry et al., 1999). Note that there is almost a complete overlap of mortality and no mortality data. This suggests that organic carbon is not a useful normalization for silver partitioning in sediments. Perhaps this is not surprising because the role of sulfur groups is so prominent in the complexation chemistry of silver (Bell and Kramer, 1999).

To not depend on the identity of the metal is an advantage in analyzing naturally contaminated sediments in that it is difficult to decide which metal is potentially causing the toxicity. Of course it can be done using the sequence of solubilities of the metal sulfides or interstitial metal concentrations (Di Toro et al., 1992; Ankley et al., 1996). The metal-independent method can be tested using the results of an experiment with an equimolar mixture of cadmium, copper, nickel, and zinc (see Figure 3-10B). The area of uncertainty falls within the carbon-normalized excess SEM boundaries above.

3.4.5 *Chronic Toxicity Uncertainty*

The results of chronic toxicity tests with metals-spiked sediments can also be compared to $(\Sigma SEM\text{-}AVS)/f_{OC}$ (Figure 3-11; Table 3-3). Note that Figure 3-11 indicates a category for "predicted toxic." Significant chronic effects were observed in only 1 of the 19 sediments, where the uncertainty analysis of acute toxicity tests indicated that effects were not expected at $(\Sigma SEM\text{-}AVS)/f_{OC} < 130\ \mu mol/g_{OC}$. The concentration in the sediment where chronic effects were observed but not expected, i.e., $(\Sigma SEM\text{-}AVS)/f_{OC} = 28\ \mu mol$ excess SEM/g_{OC}. The previous analysis of the results of chronic toxicity tests using SEM-AVS indicated that concentrations of SEM exceeded AVS in 7 of 19 nontoxic sediments. Sediment concentrations based on $(\Sigma SEM\text{-}AVS)/f_{OC}$ placed these sediments in the uncertain toxicity category. Importantly, use of $(\Sigma SEM\text{-}AVS)/f_{OC}$ to classify sediments resulted in six of these same seven sediments being correctly classified as probably nontoxic. Chronic effects were observed in six of the seven sediments where predictions of effects are uncertain (130 to 3,000 $\mu mol/g_{OC}$). This suggests that chronic toxicity tests with sensitive benthic species will be a necessary part of the evaluations of sediments predicted to have uncertain effects.

3.4.6 *Summary*

The uncertainty bounds on SEM-AVS differences and organic carbon–normalized excess SEM ($(\Sigma SEM\text{-}AVS)/f_{OC}$) can be used to identify sediments that are likely to be toxic, are of uncertain toxicity, or are nontoxic. Use of $(\Sigma SEM\text{-}AVS)/f_{OC}$ as a correction factor for excess SEM is attractive because it is based on the theoretical foundation of equilibrium partitioning. Likewise, it reduces the uncertainty of the prediction of toxicity over that of SEM-AVS differences.

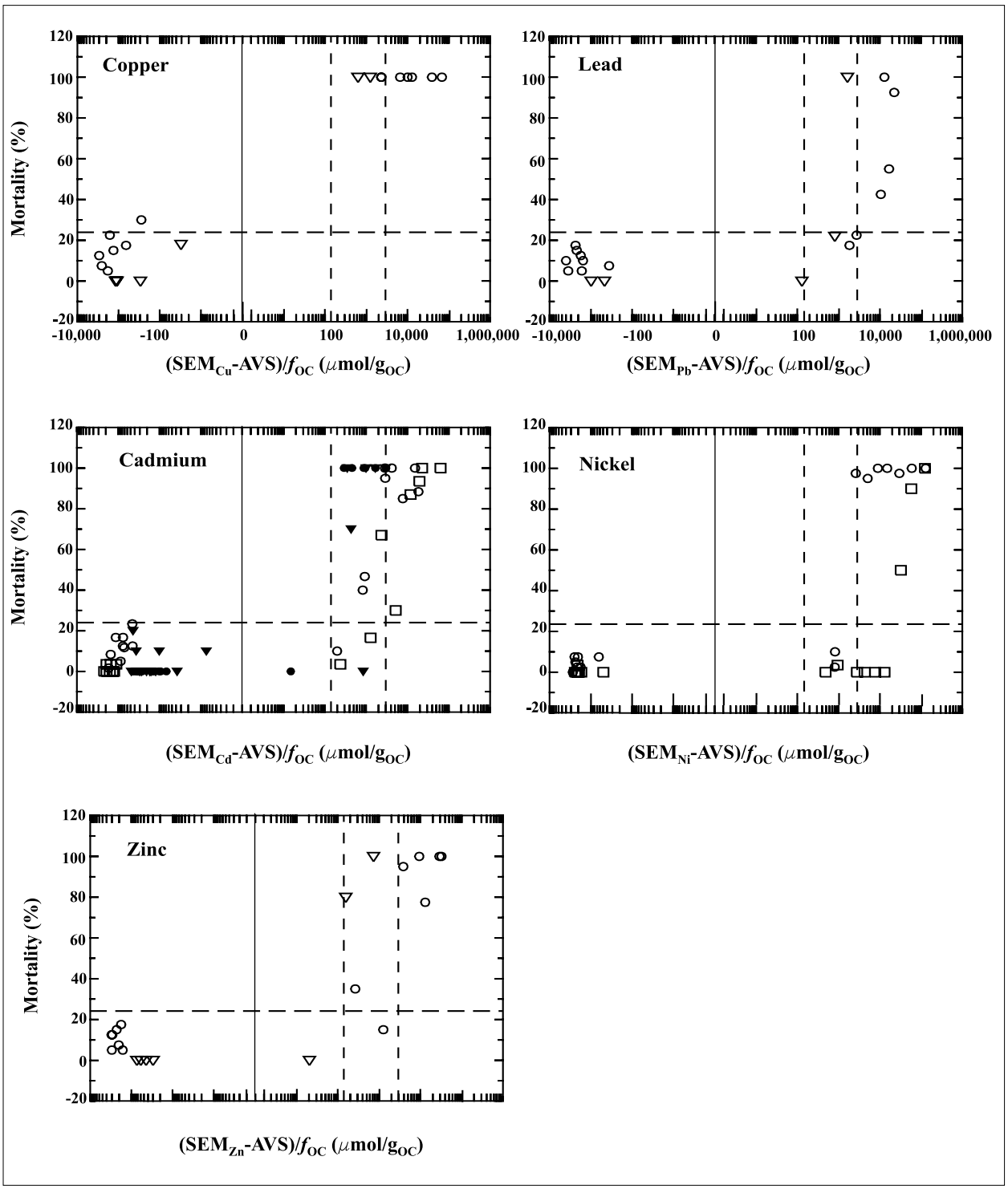

Figure 3-9. Percent mortality versus (SEM_{Metal}-AVS)/f_{OC} for each metal in spiked sediment tests using *Ampelisca* (○), *Capitella* (▽), *Neanthes* (□), *Lumbriculus* (●), and *Helisoma* (▾). Vertical dashed lines are the 90% uncertainty bound limits (figure from Di Toro et al., 2000).

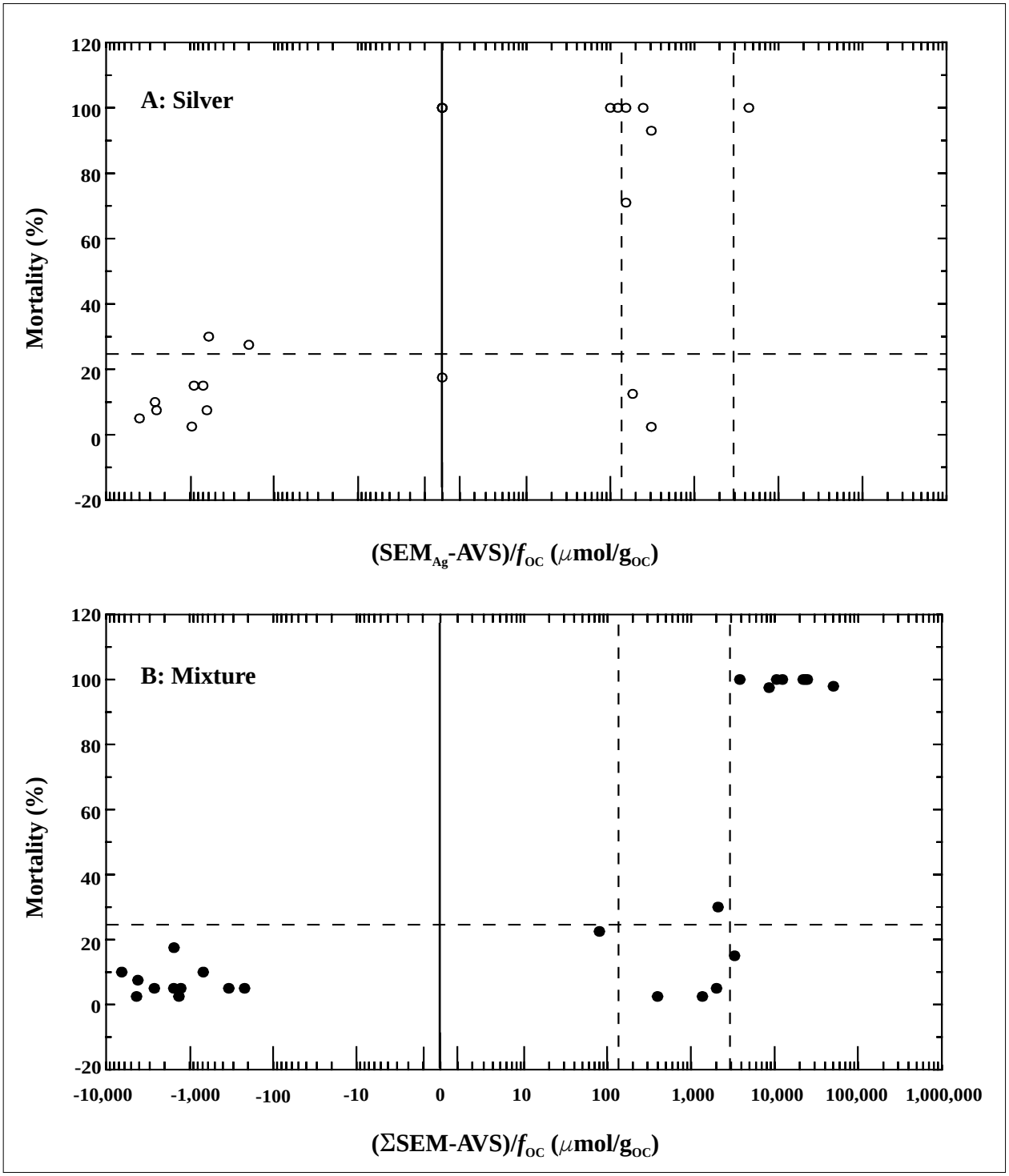

Figure 3-10. Percent mortality versus (SEM$_{Ag}$-AVS)/f_{OC} for silver (A) and (ΣSEM-AVS)/f_{OC} for a mixture experiment using Cd, Cu, Ni, and Zn (B; see Berry et al., 1996). Vertical dashed lines are the 90% uncertainty bound limits determined from Figure 3-8B (figures from Di Toro et al., 2000).

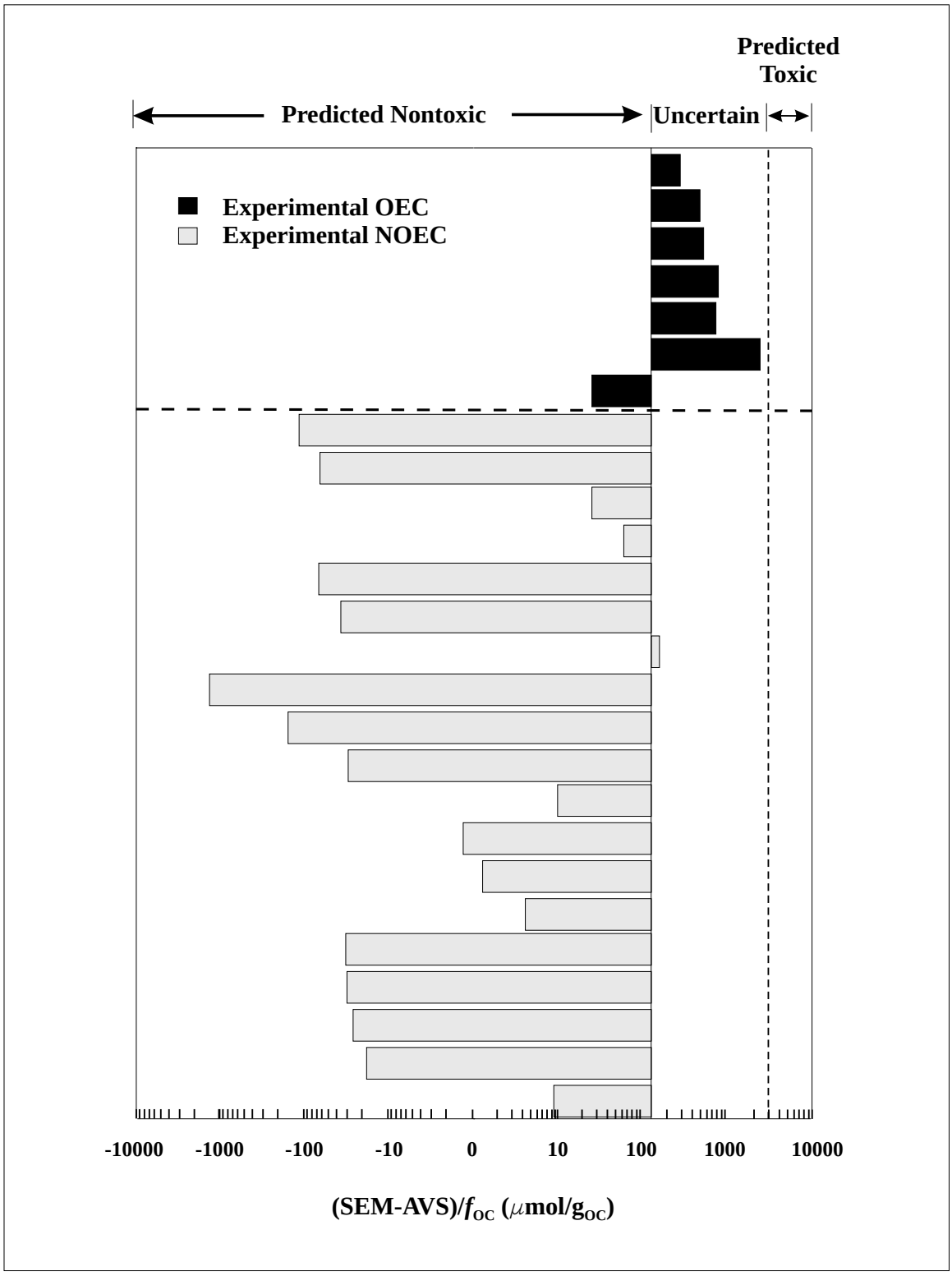

Figure 3-11. Comparison of the chronic toxicity of sediments spiked with individual metals or metal mixtures to predicted toxicity based on (SEM-AVS)/f_{OC} (data from Table 3-3). Horizontal dashed line separates experimental observed effect concentrations (solid columns) from no observed effect concentrations (shaded columns). Values at (SEM-AVS)/f_{OC} ≤ 130 μmol/g_{OC} are predicted to be nontoxic. Values between 130 and 3,000 μmol/g_{OC} lie where the prediction of toxicity is uncertain, and values greater than 3,000 μmol/g_{OC} are predicted to be toxic.

Table 3-3. Test-specific data for chronic toxicity of freshwater and saltwater organisms compared to (ΣSEM-AVS)/f_{OC}

Toxicity Test	Metal(s)	f_{OC} (g_{OC}/g)	(ΣSEM-AVS)/f_{OC}[a] (μmol/g_{OC}) NOEC(s)[b]	(ΣSEM-AVS)/f_{OC}[a] (μmol/g_{OC}) OEC(s)[c]	Reference
Life Cycle:					
Leptocheirus plumulosus	Cadmium	0.030	-117, -66.7, 26, 63.3	**297, 520**	DeWitt et al., 1996
Chironomus tentans	Zinc	0.038	-68, -36.8, **168**	**576, 847**	Sibley et al., 1996
Colonization:					
Laboratory-saltwater	Cadmium	0.010	-1340	**800**, **2740**	Hansen et al., 1996b
Field-saltwater	Cadmium, copper, lead, nickel, zinc	0.002	-155, -30, 10	—	Boothman et al., 2001
Field-freshwater	Cadmium	0.079	-0.92, 1.08, 4.30	*28*	Hare et al., 1994
Field-freshwater	Zinc	0.111	-32.7, -31.8, -26.4, -18.2, 9.1	—	Liber et al., 1996

[a](ΣSEM-AVS)/f_{OC} concentrations in bold type are those between 130 and 3,000 μmol/g_{OC} for which the expectation of effects is uncertain. Italics indicates concentrations where effects were observed but not expected.
[b]NOECs = no observed effect concentration(s); all concentrations where response was not significantly different from the control.
[c]OECs = observed effect concentration(s); all concentrations where response was significantly different from the control.

Section 4

Derivation of Metal Mixtures $ESB_{AVS:WQC}s$

4.1 General Information

Section 4 of this document presents the technical basis for establishing the ESB for cadmium, copper, lead, nickel, silver, and zinc. The basis of the overall approach is the use of EqP theory linked to the concept of maintaining metal activity for the sediment interstitial water system below concentrations that cause adverse effects. Extensive toxicological concentration-response data from short-term and chronic laboratory and field experiments, with both marine and freshwater sediments and a variety of species, indicate that it is possible to reliably predict absence of metal toxicity based on EqP theory and derive ESBs for metals in sediments using either of two approaches referred to as $ESB_{AVS:WQC}s$. The $ESB_{AVS:WQC}s$ for the six metals that collectively predicts absence of their toxicity in sediments can be derived by (a) comparing the sum of their molar concentrations, measured as SEM, with the molar concentration of AVS in sediments (solid-phase AVS benchmark); or (b) summing the measured interstitial water concentrations of the metals divided by their respective WQC FCVs (interstitial water benchmark). Lack of exceedence of the $ESB_{AVS:WQC}$ based on either of these two procedures indicates that metal toxicity should not occur.

At present, the technical basis for implementing these two approaches is supportable. The approaches have been presented to and reviewed by the SAB (U.S. EPA, 1994a, 1995a, 1999).

Additional research required to fully implement other approaches for deriving an $ESB_{AVS:WQC}$ for these metals and to derive an ESB for other metals such as mercury, arsenic, and chromium includes the development of uncertainty estimates; part of this would include their application to a variety of field settings and sediment types. Finally, the ESB approaches are intended to protect benthic organisms from direct toxicity associated with exposure to metal-contaminated sediments. The ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans. They are not designed to protect aquatic systems from metal release associated with, for example, sediment suspension, or the transport of metals into aquatic food webs. In particular, studies are needed to understand the toxicological significance of the biomagnification of metals that occurs when predators consume benthic organisms that have accumulated metals from sediments with more AVS than SEM (Ankley, 1996).

The following nomenclature is used in subsequent discussions of the $ESB_{AVS:WQC}s$ derivation for metal mixtures. The $ESB_{AVS:WQC}$ for the metals, based on AVS, is expressed in molar units because of the molar stoichiometry of metal binding to AVS. Thus, solid-phase constituents (AVS, SEM) are in μmol/g dry weight. The interstitial water metal concentrations are expressed in μmol/L or μg/L, either as dissolved concentrations $[M_d]$ or activities $\{M^{2+}\}$ (Stumm and Morgan, 1981). The subscripted notation, M_d, is used to distinguish dissolved aqueous-phase molar concentrations from solid-phase molar concentrations with no subscript. For the combined concentration, $[SEM_T]$, the units are μmol of total metal per gram of dry weight sediment. Note also that when $[SEM_{Ag}]$ is summed and/or compared with AVS, one-half the molar silver concentration is applied.

One final point should be made with respect to nomenclature. The terms *nontoxic* and *having no effect* are used only with respect to the six metals considered in this document. Toxicity of field- collected sediments can be caused by other chemicals. Therefore, avoiding exceedences of the $ESB_{AVS:WQC}$ for metal mixtures does not mean that the sediments are nontoxic. It only ensures that the six metals being considered should not cause direct toxicity to benthic organisms. Moreover, as discussed in detail below, exceedence of the benchmarks for the six metals does not necessarily indicate that metals will cause toxicity. For these reasons,it is strongly recommended that the combined use of both AVS and interstitial water measurements; toxicity tests; TIEs; chemical monitoring in vertical, horizontal, and temporal scales; and other assessment methodologies as integral parts of any evaluation of the effects of sediment-associated contaminants (Ankley et

al., 1994; Lee et al., 2000).

4.2 Sediment Benchmarks for Multiple Metals

It is neither sufficient nor appropriate to derive an ESB that considers each metal separately, because metals almost always occur as mixtures in field sediments and metal–sulfide binding is interactive.

4.2.1 *AVS Benchmarks*

Results of calculations using chemical equilibrium models indicate that metals act in a competitive manner when binding to AVS. That is, the six metals—silver, copper, lead, cadmium, zinc, and nickel— will bind to AVS and be converted to their respective sulfides in this sequence (i.e., in the order of increasing solubility). Therefore, they must be considered together. There cannot be a benchmark for just nickel, for example, because all the other metals may be present as metal sulfides, and therefore, to some extent, as AVS. If these other metals are not measured as a mixture, then the ΣSEM will be misleadingly small, and it might appear that Σ[SEM]<[AVS] when in fact this would not be true if all the metals are considered together. It should be noted that this document currently restricts this discussion to the six metals listed above; however, in situations where other sulfide-forming metals (e.g., mercury) are present at high concentrations, they also must be considered.

The equilibrium model used to derive the $ESB_{AVS:WQC}$ for a mixture of the metals is presented below (see Ankley et al., 1996, for details). If the molar sum of SEM for the six metals is less than or equal to the AVS, that is, if

$$\Sigma_i [SEM_i] \leq [AVS] \quad (4\text{-}1)$$

where

$$\Sigma_i [SEM_i] = [SEM_{Cd}] + [SEM_{Cu}] + [SEM_{Pb}] + [SEM_{Ni}] + [SEM_{Zn}] + 1/2[SEM_{Ag}]$$

then the concentrations of the mixtures of metals in the sediment are acceptable for protection of benthic organisms from acute or chronic metal toxicity.

4.2.2 *Interstitial Water Benchmarks*

The application of the interstitial water benchmark to multiple metals is complicated, not by the chemical interactions of the metals in the sediment-interstitial water system (as in the case with the AVS benchmark), but rather because of possible toxic interactions. Even if the individual concentrations do not exceed the water quality final chronic value (FCV) of each metal

Table 4-1. Water quality criteria (WQC) final chronic value (FCV) based on the dissolved concentration of metal[a]

Metal	Saltwater FCV (μg/L)	Freshwater FCV (μg/L)[b]
Cadmium	9.3	CF[c] $[e^{(0.7852[\ln(\text{hardness})]-3.490}]$
Copper[d]	3.1	$0.960[e^{(0.8545[\ln(\text{hardness})]-1.465)}]$
Lead	8.1	$0.791[e^{(1.273[\ln(\text{hardness})]-4.705)}]$
Nickel	8.2	$0.997[e^{(0.8460[\ln(\text{hardness})]+1.1645)}]$
Silver	NA[e]	NA[e]
Zinc	81	$0.986[e^{(0.8473[\ln(\text{hardness})]+0.7614)}]$

[a] These WQC FCV values are for use in the interstitial water benchmarks approach for deriving $ESBs_{AVS:WQC}$ based on the dissolved metal concentrations in interstitial water (U.S. EPA, 1995b).

[b] For example, the freshwater FCV at a hardness of 50, 100, and 200 mg $CaCO_3$/L are 0.62, 1.0, and 1.7 μg cadmium/L; 6.3, 10, and 20 μg copper/L; 1.0, 2.5, and 6.1 μg lead/L; 87, 160, and 280 μg nickel/L; and 58, 100, and 190 μg zinc/L.

[c] CF = conversion factor to calculate the dissolved FCV for cadmium from the total FCV for cadmium: CF=1.101672–[(ln hardness)(0.041838)].

[d] The saltwater FCV for copper is from U.S. EPA (1995c).

[e] The silver criteria are currently under revision to reflect water quality factors that influence the criteria such as hardness, DOC, chloride, and pH, among other factors.

presented in Table 4-1, the metals could exert additive effects that might result in toxicity (Biesinger et al., 1986; Spehar and Fiandt, 1986; Enserink et al., 1991; Kraak et al., 1994). Therefore, in order to address this potential additivity, the interstitial water metal concentrations are converted to interstitial water benchmark units (IWBUs). This conversion is done by dividing the individual metal interstitial water concentrations by their respective WQC FCV and summing these values for all the metals. IWBUs are conceptually similar to toxic units; however, the term IWBU was adopted because it is derived using the FCV, which is intended to be a "no effect" concentration (i.e., toxicity would not usually be expected at 1.0 IWBUs).

For freshwater sediments, the FCVs are hardness dependent for all of the divalent metals under consideration, and thus, need to be adjusted to the hardness of the interstitial water of the sediment being considered. Because there are no FCVs for silver in freshwater or saltwater, this approach is not applicable to sediments containing significant concentrations of silver (i.e., ΣSEM>AVS). Because silver has the smallest solubility product (see Table 2-2) and the greatest affinity for AVS, it would be the last metal to be released from the AVS or the first metal to bind with AVS. Therefore, it is unlikely that silver would occur in the interstitial water of any sediment with measurable AVS (Berry et al., 1996).

For the ith metal with a total dissolved concentration, $[M_{i,d}]$, the IWBU is

$$\sum_i \frac{[M_{i,d}]}{FCV_{i,d}} \le 1.0 \qquad (4\text{-}2)$$

where

$$\sum_i \frac{[M_{i,d}]}{[FCV_{i,d}]} = \frac{[M_{Cd,d}]}{[FCV_{Cd,d}]} + \frac{[M_{Cu,d}]}{[FCV_{Cu,d}]} + \frac{[M_{Pb,d}]}{[FCV_{Pb,d}]} + \frac{[M_{Ni,d}]}{[FCV_{Ni,d}]} + \frac{[M_{Zn,d}]}{[FCV_{Zn,d}]}$$

4.2.3 *Summary*

In summary, the sediment benchmarks for these six metals are not exceeded, and benthic organisms are sufficiently protected, if the sediment meets either one of the following benchmarks.

$$\Sigma_i[SEM_i] \le [AVS] \qquad (4\text{-}1)$$

or

$$\sum_i \frac{[M_{i,d}]}{FCV_{i,d}} \le 1.0 \qquad (4\text{-}2)$$

If the AVS or interstitial water $ESB_{AVS:WQC}$s are exceeded, there is reason to believe that the sediment *might* be unacceptably contaminated by these metals. Further evaluation and testing would, therefore, be necessary to assess actual toxicity and its causal relationship to the metals of concern. If data on the sediment-specific SEM, AVS, and organic carbon concentrations are available, the uncertainty bounds for $(\Sigma SEM\text{-}AVS)/f_{OC}$ described in Section 3.4 could be used to further classify sediments as those in which metals are not likely to cause toxicity, metal toxicity predictions are uncertain, or metal toxicity is likely. For sediments in which toxicity is likely or uncertain, acute and chronic tests with species that are sensitive to the metals suspected to be of concern, acute and chronic sediment TIEs, in situ community assessments, and seasonal and spatial characterizations of the SEM, AVS, and interstitial water concentrations would be appropriate (Ankley et al., 1994).

4.3 Example Calculation of $ESB_{AVS:WQC}$s for Metals and EqP-Based Interpretation

To assist users of these $ESB_{AVS:WQC}$s for mixtures of metals, example calculations for deriving solid-phase and interstitial water $ESB_{AVS:WQC}$s are provided in Table 4-2. For each of the three sediments, the calculations began with measured concentrations (in bold) of AVS (μg/g), SEM_i (μg/g), and interstitial water metal (μg/L). All other values were calculated. The specific concentrations in each of the these sediments were selected to provide examples of how the chemical measurements are used with the $ESB_{AVS:WQC}$ to determine the acceptability of a specific sediment and how the risks of sediment-associated metals can be evaluated within the technical framework of the EqP approach. Sediments are arranged in the table in decreasing order of their sulfide solubility product constants (see Section 2.2.5).

Sediment A contains relatively high concentrations of metals in the SEM, between 14.2 and 16.5 μg/g for copper, lead, and zinc. However, because there is sufficient AVS (0.96 μmol/g) in the sediment, the solid-phase $ESB_{AVS:WQC}$ is –0.343 (μmol/g), and there is no

Table 4-2. Example calculations of $ESB_{AVS:WQC}$s for metal mixtures: three sediments.

	Sediment Concentration			Interstitial Water Concentration				$ESB_{AVS:WQC}$	
Sediment	Analyte	$\mu g/g^a$	$\mu mol/g$	Metal ($M_{i,d}$)	$\mu g/L$	FCV[b]	IWBU	SEM-AVS ($\mu mol/g$)	IW
A	AVS	**30.8**	0.96	—	—	—	—		
	SEM_{Ni}	**2.85**	0.048	Nickel	**ND[c] (<0.8)**	8.2	<0.10		
	SEM_{Zn}	**16.5**	0.25	Zinc	**ND[c] (<5.0)**	81	<0.06		
	SEM_{Cd}	**0.05**	0.001	Cadmium	**ND[c] (<0.2)**	9.3	<0.02		
	SEM_{Pb}	**14.2**	0.068	Lead	**ND[c] (<0.7)**	8.1	<0.09		
	SEM_{Cu}	**16.0**	0.25	Copper	**ND[c] (<0.6)**	3.1	<0.19		
	SEM_{Ag}	—	—	Silver	—	—	—		
ΣSEM = 0.617 μmol/g; SEM-AVS = -0.343 μmol/g							ΣIWBU <0.46	-0.34	<0.46
B	AVS	**1310**	40.8	—	—	—	—		
	SEM_{Ni}	**34.0**	0.58	Nickel	**4.8**	160	0.03		
	SEM_{Zn}	**2630**	40.2	Zinc	**43.2**	100	0.43		
	SEM_{Cd}	**82.9**	0.74	Cadmium	**ND[c] (<0.01)**	1.0	<0.01		
	SEM_{Pb}	**282**	1.36	Lead	**ND[c] (<0.10)**	2.5	<0.04		
	SEM_{Cu}	**227**	3.58	Copper	**ND[c] (<0.05)**	11	<0.005		
	SEM_{Ag}	**ND[c]**	NDc	Silver	**ND[c] (<0.01)**	—	—		
ΣSEM = 46.5 μmol/g; SEM-AVS = 5.71 μmol/g							ΣIWBU ~0.46	5.71	~0.46
C	AVS	**146**	4.57	—	—	—	—		
	SEM_{Ni}	**269**	4.58	Nickel	**26.3**	87	0.30		
	SEM_{Zn}	**12.4**	0.19	Zinc	**4.3**	58	0.07		
	SEM_{Cd}	**573**	5.12	Cadmium	**24.9**	0.62	40.1		
	SEM_{Pb}	**66.2**	0.32	Lead	**ND[c] (<0.10)**	1.0	<0.10		
	SEM_{Cu}	**4.44**	0.07	Copper	**ND[c] (<0.05)**	6.3	<0.008		
	SEM_{Ag}	**ND[c]**	NDc	Silver	**ND[c] (<0.01)**	—	—		
ΣSEM = 10.28 μmol/g; SEM-AVS = 5.71 μmol/g							ΣIWBU ~40.47	5.71	~40.5

[a] Molecular weights: sulfur, 32.06; nickel, 58.7; zinc, 65.4; cadmium, 112; lead, 207; copper, 63.5; silver, 108.
[b] Saltwater sediment: sediment A. Freshwater sediments: sediment B, interstitial hardness 100 mg/L; sediment C, 50 mg/L.
[c] ND = not detected.

metal detected in the interstitial water. This sediment is acceptable for protection of benthic organisms from direct toxicity of the metals in the sediment. Silver was not measured in this sediment. However, because AVS is present, any silver in the sediment is not of toxicological concern and none should occur in interstitial water. One final consideration is the need for detection limits for metals in the sediment that are significantly below their respective WQC FCVs. For this sediment there were no detectable metals in the interstitial water and ΣIWBU was <0.46.

Sediment B is from a Superfund site heavily contaminated with all of the metals (ΣSEM = 46.5

μmol/g), but most severely with zinc (2,630 μg/g). There is an excess of SEM in this sediment (SEM-AVS = 5.71 μmol/g). Importantly for sediment B, the interstitial concentrations of the metals were all less than the WQC FCVs and the ΣIWBU was <1.0 (~ 0.46). Therefore, this sediment is acceptable for protection of benthic organisms from direct toxicity of this mixture of metals in the sediment. It should be noted that, if interstitial metal concentrations had not been quantified, the sediment would have exceeded the $ESB_{AVS:WQC}$ and additional testing would be advisable. A possible explanation for the absence of significant metals in the interstitial water of this sediment is its higher organic carbon concentration (f_{OC} = 0.05). The (ΣSEM-AVS)/f_{OC} of 114 μmol excess SEM/g_{OC} for this sediment is, therefore, predicted to be nontoxic because it is <130 μmol excess SEM/g_{OC} (see Section 3.4.4).

Sediment C is heavily contaminated with approximately equimolar concentrations of cadmium and nickel. It exceeds the $ESB_{AVS:WQC}$ for metals for both solid and interstitial water phases. The ΣSEM (10.28 μmol/g) exceeds the AVS (4.57 μmol/g); therefore, SEM-AVS = 5.71 μmol excess SEM/g, a concentration identical to that of sediment B. Although lead and copper are found in the sediment, they are not found in detectable concentrations in the interstitial water. This is because they have the lowest sulfide solubility product constants and the sum of their SEM concentrations (0.39 μmol/g) is less than AVS. If the dry weight concentrations of metals had been analyzed, silver and additional copper and nickel might have been detected. Silver will not be detected in the SEM or interstitial water when AVS is present (see Section 3.2.1). Nickel, cadmium, and zinc occur in interstitial water because in the sequential summation of the SEM_i concentrations in order of increasing sulfide solubilities, the concentrations of these metals exceed the AVS. Therefore, these three metals are found in the SEM that is not a metal sulfide and in the interstitial water, and contribute to the ΣIWBU (~40.47) as well as to the overall exposure of benthic organisms. Because only cadmium concentrations exceed the WQC FCV, any effects observed in toxicity tests or in faunal analyses with this sediment should principally be a result of cadmium. This sediment is low in organic carbon concentration (TOC = 0.2%; f_{OC} = 0.002). The organic carbon–normalized concentration (ΣSEM-AVS/f_{OC}) of 2,855 μmol excess SEM/g_{OC} was within the uncertainty bounds of 130 to 3,000 μmol excess SEM/g_{OC}, suggesting that additional evaluations should be conducted (see Section 3.4.4).

4.4 $ESB_{AVS:WQC}$ for Metals vs. Environmental Monitoring Databases

This section compares the $ESB_{AVS:WQC}$ based on AVS or IWBUs with chemical monitoring data from freshwater and saltwater sediments in the United States. This comparison of AVS-SEM and interstitial water concentrations is used to indicate the frequency of sediments in the United States where metals toxicity is unlikely. When data were available in the monitoring programs, (ΣSEM-AVS)/f_{OC} is used to indicate sediments where toxicity is unlikely, likely, or uncertain. When toxicity or benthic organism community health data are available in conjunction with these concentrations it is possible to speculate as to potential causes of the observed effects. These data, however, cannot be used to validate the usefulness of the AVS approach because sediments that exceed the benchmarks are not always toxic, and because observed sediment toxicity may be the result of unknown substances.

4.4.1 *Data Analysis*

Three monitoring databases were identified that contain AVS, SEM, and f_{OC} information; one also had data on concentrations of metals in interstitial water. Toxicity tests were conducted on all sediments from these sources. The sources are the Environmental Monitoring and Assessment Program (EMAP) (Leonard et al., 1996a), the National Oceanographic and Atmospheric Administration National Status and Trends monitoring program (NOAA NST) (Wolfe et al., 1994; Long et al., 1995b, 1996), and the Regional Environmental Monitoring and Assessment Program (REMAP) (Adams et al., 1996).

4.4.1.1 *Freshwater Sediments*

The AVS and SEM concentrations in the 1994 EMAP database from the Great Lakes were analyzed by Leonard et al. (1996a). A total of 46 sediment grab samples and 9 core samples were collected in the summer from 42 locations in Lake Michigan. SEM, AVS, TOC, interstitial water metals (when sufficient volumes were present), and 10-day sediment toxicity to the midge *C. tentans* and the amphipod *H. azteca* were measured in the grab samples (the concentrations are listed in Appendix A).

The AVS concentrations versus SEM-AVS differences from Appendix A are plotted in Figure 4-1. Grab sediment samples containing AVS concentrations

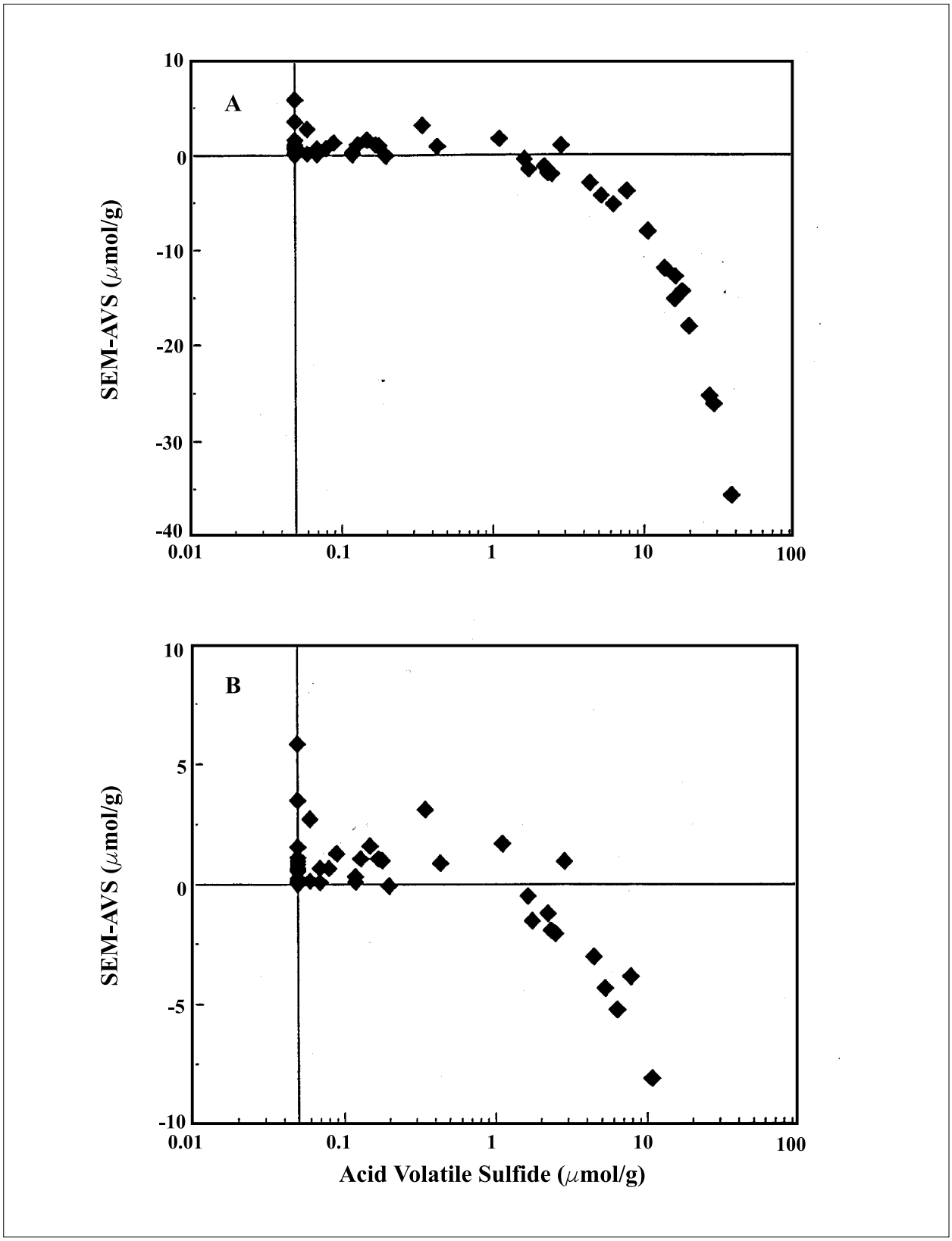

Figure 4-1. SEM-AVS values versus AVS concentrations in EMAP-Great Lakes sediments from Lake Michigan. Data are from surficial grab samples only. Plot (A) shows all values; plot (B) has the ordinate limited to SEM-AVS values between –10 and +10 μmol/g.

below the detection limit of 0.05 μmol/g AVS are plotted at that concentration. Forty-two of the 46 samples (91%) had SEM-AVS differences greater than 0.0. Thirty-six of these had less than 1.0 μmol of excess SEM/g sediment; and none had over 5.8 μmol excess SEM/g sediment. Sediments with SEM concentrations in excess of that for AVS have the potential to be toxic because of metals. However, the majority of sediments with an excess of SEM had low concentrations of both AVS and SEM. For 20 of these Lake Michigan sediments, interstitial water metals concentrations were measured. The sum of the IWBUs for cadmium, copper, lead, nickel, and zinc was always less than 0.4 (Leonard et al., 1996a). In 10-day toxicity tests using *C. tentans* and *H. azteca*, no toxicity was observed in 81% of the 21 sediments not exceeding the $ESB_{AVS:WQC}$, Leonard et al. (1996a) concluded that when toxicity was observed it was not likely from metals, because of the low interstitial water metals concentrations. These data demonstrate the value of using both SEM-AVS and IWBUs to evaluate the risks of metals in sediments.

4.4.1.2 *Saltwater Sediments*

Saltwater data from a total of 398 sediment samples from 5 monitoring programs representing the eastern coast of the United States are included in Appendix B. The EMAP Virginian Province database (U.S. EPA, 1996) consists, in part, of 127 sediment samples collected from August to mid-September 1993 from randomly selected locations in tidal rivers and small and large estuaries from the Chesapeake Bay to Massachusetts (Strobel et al., 1995). The NOAA data are from Long Island Sound, Boston Harbor, and the Hudson River Estuary. Sediments were collected from 63 locations in the coastal bays and harbors of Long Island Sound in August 1991 (Wolfe et al., 1994). Sediment samples from 30 locations in Boston Harbor were collected in June and July 1993 (Long et al., 1996). Sediment samples from 38 locations in the Hudson River Estuary were collected from March to May 1991 (Long et al., 1995b). Sediment samples were collected in the REMAP program from 140 locations from the New York/New Jersey Harbor Estuary System (Adams et al., 1996). All of the above sediment grab samples were from approximately the top 2 cm of undisturbed sediment.

For saltwater sediments, the molar concentration of AVS typically exceeds that for SEM (SEM-AVS $\leq$0.0 μmol/g) for most of the samples across the entire range of AVS concentrations (Figure 4-2). A total of 68 of the 398 saltwater sediments (17%) had an excess of metal, and only 4 of the 68 (6%) had over 2 μmol excess SEM/g. As AVS levels increase, fewer and fewer sediments have SEM-AVS differences that are positive; none occurred when AVS was >8.1 μmol/g. Interstitial water metal was not measured in these saltwater sediments. Only 5 of the 68 sediments (7%) having excess of up to 0.9 μmol SEM/g were toxic in 10-day sediment toxicity tests with the amphipod *A. abdita*, whereas 79 of 330 sediments (24%) having an excess of AVS were toxic. Toxicity was not believed to be metals related in the 79 toxic sediments where AVS was in excess over SEM. Metals were unlikely the cause of toxicity in those sediments having an excess of SEM because there was only $\leq$0.9 μmol excess SEM/g. Finally, the absence of toxicity in sediments having an excess of SEM of up to 4.4 μmol/g indicates significant metal-binding potential over that of AVS in some sediments. Organic carbon concentrations from 0.05% to 15.2% (average 1.9%) provide for some of this additional metal binding.

Organic carbon, along with SEM and AVS, was measured in these 398 saltwater sediments. Therefore, the $(\Sigma SEM\text{-}AVS)/f_{OC}$ concentrations of concern can be compared with the organic carbon–normalized concentrations of SEM-AVS differences (Figure 4-3). No sediments containing an AVS concentration in excess of 10 μmol/g had an excess of SEM; that is, all $(\Sigma SEM\text{-}AVS)/f_{OC}$ values were negative. Excess of SEM relative to AVS became more common as sediment AVS decreased. None of the sediments contained greater than 130 μmol excess SEM/g_{OC}, the lower uncertainty bound from Section 3.4. This indicates that metals concentrations in all of the sediments monitored in the summer by EPA EMAP and REMAP and by NOAA are below concentrations of concern for benthic organisms.

4.5 Bioaccumulation

The data appear to suggest that, for these sediments collected from freshwater and marine locations in the United States, direct toxicity caused by metals in sediments is expected to be extremely rare. Although this might be true, these data by themselves are inconclusive. Importantly, it would be inappropriate to use the data from the above studies to conclude that metals in sediments are *not* a problem. In all of the above studies, the sediments were conducted in the summer when the seasonal biogeochemical cycling of sulfur should produce the highest concentrations of iron monosulfide, which

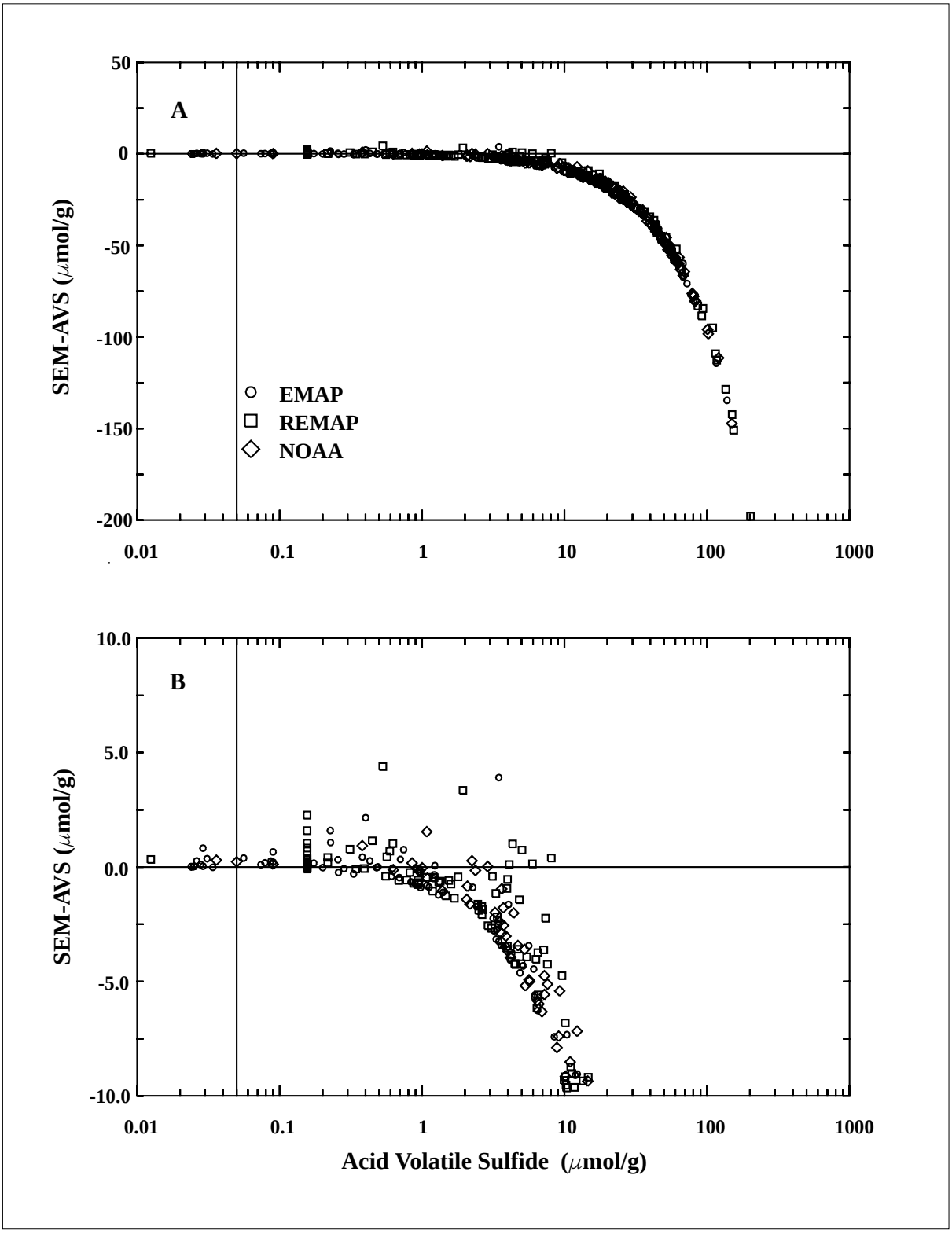

Figure 4-2. **SEM-AVS values versus AVS concentrations in EMAP-Estuaries Virginian Province (U.S. EPA, 1996); REMAP-NY/NJ Harbor Estuary (Adams et al., 1996); NOAA NST-Long Island Sound (Wolfe et al., 1994); Boston Harbor (Long et al., 1996); and Hudson-Raritan Estuaries (Long et al., 1995b). Plot A shows all values; plot B has the ordinate limited to SEM-AVS values between -10 and +10 μmol/g (see data in Appendix B).**

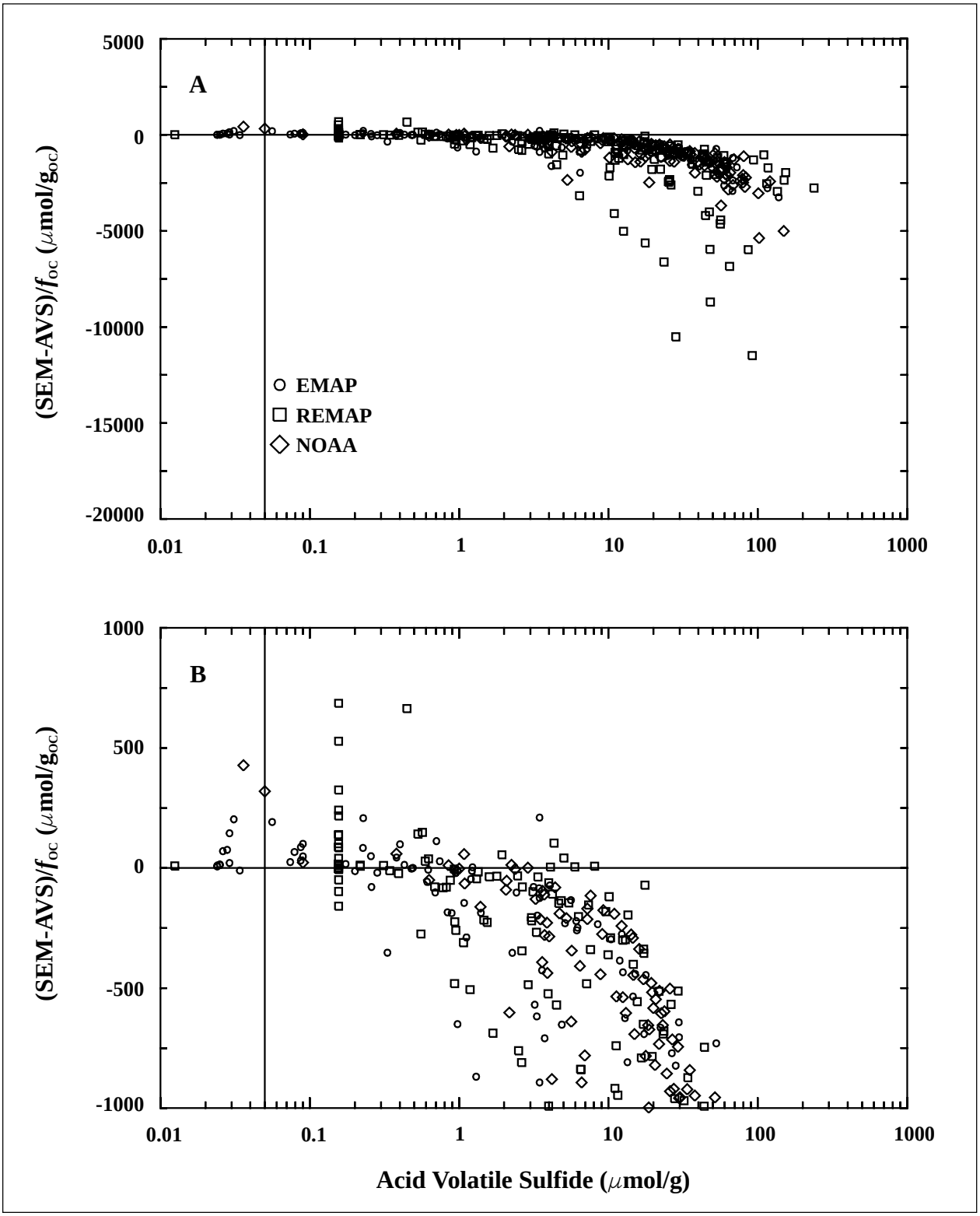

Figure 4-3. **(ΣSEM-AVS)/f_{OC} versus AVS concentrations in EMAP-Estuaries Virginian Province (U.S. EPA, 1996); REMAP-NY/NJ Harbor Estuary (Adams et al., 1996); NOAA NST-Long Island Sound (Wolfe et al., 1994); Boston Harbor (Long et al., 1996); and Hudson-Raritan Estuaries (Long et al., 1995b). Plot A shows all values; plot B has the ordinate limited to (ΣSEM-AVS)/f_{OC} values between -10 and +10 μmol/g (see data in Appendix B).**

might make direct metal-associated toxicity less likely than in the winter/spring months. Accurate assessment of the extent of the direct ecological risks of metals in sediments requires that sediment monitoring occur in the months of minimum AVS concentration; typically, but not always, in November to early May. These yet-to-be-conducted studies must monitor, at a minimum, SEM, AVS, f_{OC}, interstitial water metal, and toxicity.

Bioaccumulation of metals from sediments when SEM is less than AVS was not expected based on EqP theory. However, there is a significant database that demonstrates that metals concentrations in benthic organisms increase when metals concentrations in sediments on a dry weight basis increase (Ankley, 1996). This has caused considerable debate (Lee et al., 2000a,b) because it suggests that metal bioavailability may be related to dry weight metals concentrations, and if the increase in bioaccumulated metal is related to effects, then effects may be related to dry weight metals concentrations. Most importantly, these studies, and all other AVS-related testing, has overwhelming demonstrated that toxic effects of metals are absent in sediments when SEM is less than AVS, even when bioaccumulation is observed, and that toxicity is not related to dry weight metals concentrations. For example, careful evaluation of Lee et al. (2000b) results, demonstrates that in order to understand and predict metal toxicity AVS normalization is critical. Although Lee et al. (2000b) note the accumulation of metal by the test organisms, no adverse effects were reported. This suggests that the bioaccumulated metals may not be toxicologically available or of sufficient concentration in the organism to cause effects. In addition, these metals do not biomagnify to higher trophic levels in aquatic ecosystems (Suedel et al., 1994). Therefore, an $ESB_{AVS:WQC}$ based on the difference between the concentrations of SEM and AVS is appropriate for protecting benthic organisms from the direct effects of sediment-associated metals, and not for protecting against metal bioaccumulation.

Section 5

Sampling and Analytical Chemistry

5.1 General Information

This section provides guidance on procedures for sampling, handling, and analysis of metals in sediments, and on the interpretation of data from the sediment samples that are needed if the assessments of the risks of sediment-associated metals are to be appropriately based on the EqP methodology. The design of any assessment should match the goal of the specific assessment and how evaluation tools such as $ESB_{AVS:WQC}$s are to be applied.

Results of the short- and long-term laboratory and field experiments conducted to date using sediments spiked with individual metals and mixtures of metals represent convincing support for the conclusion that absence (but not necessarily presence) of metal toxicity can be reliably predicted based on metal–sulfide relationships or interstitial water metal concentrations. In contrast, much confusion exists on how to use this convincing evidence to interpret the significance of metals concentrations in sediments from the field. Using these observations as a basis for predicting metal bioavailability, or deriving an $ESB_{AVS:WQC}$, raises a number of conceptual and practical issues related to sampling, analytical measurements, and effects of additional binding phases. Many of these were addressed by Ankley et al. (1994). Those most salient to the proposed derivation of the $ESB_{AVS:WQC}$s are described below.

5.2 Sampling and Storage

Accurate prediction of exposure of benthic organisms to metals is critically dependent on sampling appropriate sediment horizons at appropriate times. This is because of the relatively high rates of AVS oxidation caused by natural processes in sediments and the requirement that oxidation must be avoided during sampling of sediments and interstitial water. In fact, the labile nature of iron monosulfides has led some to question the practical utility of using AVS as a basis for an EqP-derived ESB for metals (Luoma and Carter, 1993; Meyer et al., 1994). For example, there have been many observations of spatial (depth) variations in AVS concentrations, most of which indicate that surficial AVS concentrations are less than those in deeper sediments (Boothman and Helmstetter, 1992; Howard and Evans, 1993; Brumbaugh et al., 1994; Hare et al., 1994; Besser et al., 1996; Hansen et al., 1996b; Leonard et al., 1996a; Liber et al., 1996; Boothman et al., 2001). This is likely because of oxidation of AVS (principally FeS) at the sediment surface, a process enhanced by bioturbation (Peterson et al., 1996).

In addition to varying with depth, AVS can vary seasonally. For example, in systems where overlying water contains appreciable oxygen during cold-weather months, AVS tends to decrease, presumably because of a constant rate of oxidation of the AVS linked to a decrease in its generation by sulfate-reducing bacteria (Herlihy and Mills, 1985; Howard and Evans, 1993; Leonard et al., 1993). Because of potential temporal and spatial variability of AVS, it appears that the way to avoid possible underestimation of metal bioavailability is to sample the biologically "active" zone of sediments at times when AVS might be expected to be present at low concentrations. It is recommended that, at a minimum, AVS and SEM measurements be made using samples of the surficial (0 to 2.0 cm) sediments during the period from November to early May. Minimum AVS concentrations may not always occur during cool-weather seasons; for example, systems that become anaerobic during the winter can maintain relatively large sediment AVS concentrations (Liber et al., 1996). Therefore, AVS, SEM, and interstitial metal concentrations may need to be determined seasonally. Importantly, the biologically active zones of some benthic communities may be within only the surficial first few millimeters of the sediment, whereas other communities may be biologically active at depths up to a meter. In order to determine the potential for exposure to metals, sediment and interstitial water samples from multiple sediment horizons may be required.

The somewhat subjective aspects of these sampling recommendations have been of concern. Multiple sediment samples are necessary because of the dynamic nature of the metal-binding phases in sediments. Depending on the depth of bioturbation, the possible oxidation rates of specific metal sulfides, and the extent of possible metal concentrations, the horizontal and vertical resolution of the needed monitoring is likely to be site specific. Even if neither

of the sediment benchmarks is violated in extensive monitoring programs, metals concentrations on a dry weight basis may be high and widely distributed. This may be a good reason to conduct monitoring studies to determine the extent of metal bioaccumulation in benthic food chains. Furthermore, if the ultimate fate of the sediments is unknown, risk assessments to evaluate future risks caused by dynamic processes may be desirable.

Research suggests that the transient nature of AVS may be overstated relative to predicting the fate of all metal-sulfide complexes in aquatic sediments. Observations from the Duluth EPA laboratory made in the early 1990s indicate that AVS concentrations in sediments contaminated by metals such as cadmium and zinc tended to be elevated over concentrations typically expected in freshwater systems (G.T. Ankley, U.S. EPA, Duluth, MN, personal communication). The probable underlying basis for these observations did not become apparent, however, until a recent series of spiking and metal-sulfide stability experiments. The field colonization study of Liber et al. (1996) demonstrated a strong positive correlation between the amount of zinc added to test sediments and the resultant concentration of AVS in the samples. In fact, the initial design of their study attempted to produce test sediments with as much as five times more SEM_{Zn} (nominal) than AVS; however, the highest measured SEM_{Zn}/AVS ratio achieved was only slightly larger than 1. Moreover, the expected surficial depletion and seasonal variations in AVS were unexpectedly low in the zinc-spiked sediments. These observations suggested that zinc sulfide, which composed the bulk of AVS in the spiked sediments, was more stable than the iron sulfide present in the control sediments. The apparent stability of other metal sulfides versus iron sulfide also has been noted in laboratory spiking experiments with freshwater and saltwater sediments (Leonard et al., 1995; DeWitt et al., 1996; Hansen et al., 1996b; Peterson et al., 1996; Sibley et al., 1996; Boothman et al., 2001).

In support of these observations, metal-sulfide oxidation experiments conducted by Di Toro et al. (1996b) have confirmed that cadmium and zinc form more stable sulfide solid phases than iron. If this is also true for sulfide complexes of copper, nickel, silver, and lead, the issue of seasonal/spatial variations in AVS becomes of less concern because most of the studies evaluating variations in AVS have focused on iron sulfide (i.e., uncontaminated sediments). Thus, further research concerning the differential stability of metal sulfides, from both temporal and spatial perspectives, is definitely warranted.

5.2.1 *Sediments*

At a minimum, sampling of the surficial 2.0 cm of sediment between November and early May is recommended. A sample depth of 2.0 cm is appropriate for monitoring. However, for instances such as dredging or in risk assessments where depths greater than 2 cm are important, sample depths should be planned based on particular study needs. Sediments can be sampled using dredges, grabs, or coring, but mixing of aerobic and anaerobic sediments must be avoided because the trace metal speciation in the sediments will be altered (see Bufflap and Allen, 1995, for detailed recommendations to limit sampling artifacts). Coring is generally less disruptive, facilitates sampling of sediment horizons, and limits potential metal contamination and oxidation if sealed PVC core liners are used.

Sediments not immediately analyzed for AVS and SEM must be placed in sealed airtight glass jars and refrigerated or frozen. Generally, enough sediment should be added to almost fill the jar. If sediments are stored this way, there will be little oxidation of AVS even after several weeks. Sampling of the stored sediment from the middle of the jar will further limit potential effects of oxidation on AVS. Sediments experiencing oxidation of AVS during storage will become less black or grey if oxidized. Because the rate of metal-sulfide oxidation is markedly less than that of iron sulfide, release of metal during storage is unlikely.

5.2.2 *Interstitial Water*

Several procedures are available to sample interstitial water in situ or ex situ. Carignan et al. (1985) compared metals concentrations in interstitial water obtained by ex situ centrifugation at 11,000 rpm followed by filtration (0.45 μm and 0.2 or 0.03 μm) and in situ diffusion samplers with 0.2 μm polysulfone membranes. For the metals of concern in this benchmark document, concentrations of nickel and cadmium were equivalent using both methods, and concentrations of copper and zinc were higher and more variable using centrifugation. They recommended using in situ dialysis for studying trace constituents in sediments because of its inherent simplicity and the avoidance of artifacts that can occur with the handling of sediments in the laboratory.

More recently, Bufflap and Allen (1995) reviewed four procedures for collection of interstitial water for trace metals analysis. These included ex situ squeezing, centrifugation, in situ dialysis, and suction filtration. These authors observed that each method has its own advantages and disadvantages. Importantly, interstitial water must be extracted by centrifugation or squeezing in an inert atmosphere until acidified, because oxidation will alter metal speciation. Artifacts may be caused by temperature changes in ex situ methods that may be overcome by maintaining temperatures similar to those in in situ methods. Contamination of interstitial water by fine particles is important in all methods, because differentiation of particulate and dissolved metal is a function of the pore size of the filter or diffusion sampler membrane. The use of 0.45 μm filtration, although an often accepted definition of "dissolved" metals, may result in differences from laboratory to laboratory. Use of suction filtration devices is limited to coarser sediments, and they do not offer depth resolution.

Use of diffusion samplers is hampered by the time required for equilibrium (7 to 14 days) and the need for diver placement and retrieval in deep waters. Acidification of interstitial water obtained by diffusion or from suction filtration must occur immediately to limit oxidation. Bufflap and Allen (1995) conclude that in situ techniques have less potential for producing sampling artifacts than ex situ procedures. They concluded that, of the in situ procedures, suction filtration has the best potential for producing artifact-free interstitial water samples directly from the environment. Of the ex situ procedures, they concluded that centrifugation under a nitrogen atmosphere followed immediately by filtration and acidification was the simplest technique likely to result in an unbiased estimate of metal concentrations in interstitial water. At present, it is recommended filtration of the surface water through 0.40 to 0.45 μm polycarbonate filters to better define that fraction of aqueous metal associated with toxicity (Prothro, 1993). This guidance applies to interstitial water. Thurman (1985) equates the organic carbon retained on a 0.45 μm glass-fiber filter to suspended organic carbon, so that this filtration procedure under nitrogen atmosphere followed immediately by acidification is acceptable for interstitial waters. However, in studies comparing collection and processing methods for trace metals, sorption to filter membranes or the filtering apparatus does occur (Schults et al., 1992). These authors later presented a method combining longer centrifugation times with a unique single-step interstitial water withdrawal procedure that has potential for minimizing metal losses by eliminating the need for filtration (Ozretich and Schults, 1998).

Use of dialysis samplers to obtain samples of interstitial water is recommended for comparison of measured concentrations of dissolved metals with WQC. This is primarily because diffusion samplers obtain interstitial water with the proper in situ geochemistry, thus limiting artifacts of ex situ sampling. Furthermore, in shallow waters, where contamination of sediments is most likely, placement of diffusion samplers is easily accomplished and extended equilibration times are not a problem. Second, use of centrifugation under nitrogen and 0.45 μm filtration using polycarbonate filters for obtaining interstitial water from sediments in deeper aquatic systems. Care must be taken to ensure that filters or the filter apparatus do not remove metal from or add metal to the interstitial water sample to be analyzed. Perhaps most importantly, the extremely large database comparing interstitial metals concentrations with organism responses from spiked- and field-sediment experiments in the laboratory has demonstrated that, where the IWTU concept predicted that metals concentrations in interstitial water should not be toxic, toxicity was not observed when either dialysis samplers or centrifugation were used (Berry et al., 1996; Hansen et al., 1996a). Therefore, it is likely that when either methodology is used to obtain interstitial water for comparison with WQC, if metals concentrations are below 1.0 IWBU, sediments should be acceptable for protection of benthic organisms. The exception is for some silver-spiked freshwater and saltwater sediments that were toxic in spite of the absence of interstitial silver. It is for this reason that IWBUs are not used as $ESB_{AVS:WQC}$s for silver (see Sections 4.2.1 and 4.2.2).

5.3 Analytical Measurements

An important aspect to deriving ESB values is that the methods necessary to implement the approach must be reasonably standardized or have been demonstrated to produce results comparable to those of standard methodologies. From the standpoint of the $ESB_{AVS:WQC}$s, a significant amount of research has gone into defining methodologies to obtain interstitial water and sediments (see Section 5.2 above), to extract SEM and AVS from sediments, and to quantify AVS, SEM, and the metals in interstitial water.

5.3.1 *Acid Volatile Sulfide*

The SEM/AVS extraction method suggested is that of Allen et al. (1993). In terms of AVS quantification, a number of techniques have been successfully utilized, including gravimetric (Di Toro et al., 1990; Leonard et al., 1993), colorimetric (Cornwell and Morse, 1987), gas chromatography– photoionization detection (Casas and Crecelius, 1994; Slotton and Reuter, 1995), and specific ion electrodes (Boothman and Helmstetter, 1992; Brouwer and Murphy, 1994; Brumbaugh et al., 1994; Leonard et al., 1996b). Allen et al. (1993) report a detection limit for 50% accuracy of 0.01 μmol/g for a 10 g sediment sample using the colorimetric method. Based on several studies, Boothman and Helmstetter (1992) report a detection limit of 1 μmol AVS, which translates to 0.1 μmol/g dry weight for a 10 g sediment sample using the ion specific electrode method.

5.3.2 *Simultaneously Extracted Metals*

SEMs are operationally defined as metals extracted from sediment into solution by the AVS extraction procedure. The dissolved metals in this solution are also operationally defined as the metal species that pass through filter material used to remove the residual sediment. Common convention defines "dissolved" as metal species <0.45 μm in size. SEM concentrations measured in sediments are not significantly different, however, using Whatman #1 filter paper alone (<11 μm nominal interstitial size) or in combination with a 0.45 μm filter (W. Boothman, U.S. EPA, Narragansett, RI, personal communication). SEM solutions generated by the AVS procedure can be analyzed for metals, commonly including cadmium, copper, lead, nickel, silver, and zinc, by routine atomic spectrochemical techniques appropriate for environmental waters (e.g., inductively coupled plasma atomic emission or graphite furnace atomic absorption spectrophotometry [GFAA]) (U.S. EPA, 1994b). Because of the need to determine metals at relatively low concentrations, additional consideration must be given to preclude contamination during collection, transport, and analysis (U.S. EPA, 1995d,e,f).

5.3.3 *Total Organic Carbon*

Several methods for measuring organic carbon exist and are reviewed by Nelson and Sommers (1996). U.S. EPA (2001) summarizes the minimum requirements of acceptable methods for quantifying total organic carbon in sediments.

5.3.4 *Interstitial Water Metal*

Interstitial water can be analyzed for the metals cadmium, copper, lead, nickel, silver, and zinc by routine atomic spectrochemical techniques appropriate for environmental waters (e.g., inductively coupled plasma atomic emission or GFAA) (U.S. EPA, 1994b). Because of the need to determine metals at concentrations at or below the threshold of biological effects (i.e., WQC concentrations), additional consideration must be given to preclude contamination during collection, transport and analysis (U.S. EPA, 1995d,e,f; also see guidance on clean chemistry techniques in U.S. EPA, 1994c). Generally, detection limits should be at $\leq$0.1 IWBU because the contributions of each of the metals must be summed.

Section 6

Sediment Benchmark Values: Application and Interpretation

The procedures described in this document indicate that, except possibly where a locally, commercially, or recreationally important species is very sensitive, benthic organisms should be acceptably protected in freshwater and saltwater sediments if at least one of the following two conditions are satisfied: the sum of the molar concentrations of SEM cadmium, copper, lead, nickel, silver, and zinc is less than or equal to the molar concentration of AVS (Section 6.1), or the sum of the dissolved interstitial water concentration of cadmium, copper, lead, nickel, and zinc divided by their respective WQC FCV is less than or equal to 1.0 (Section 6.2). The AVS benchmark is intended to apply to sediments having ≥0.1 μmol AVS/g. The two conditions for deriving $ESB_{AVS:WQC}$ are detailed in Section 4.2 and are repeated below.

Consistent with the recommendations of EPA's Science Advisory Board, publication of these documents does not imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, exceedances of ESBs could trigger collection of additional assessment data.

As discussed in Section 3.4, a more accurate prediction of toxicity can be derived if the presence of organic carbon is considered along with AVS. For the multiple metals cadmium, copper, lead, nickel, silver and zinc, the following assumptions are useful in deriving a benchmark:

1) Any sediment in which (SEM - AVS)/f_{OC} < 130 μmols/g_{OC} should pose low risk of adverse biological effects due to cadmium, copper, lead, nickel and zinc.

2) Any sediment in which 130μmols/g_{OC} < (SEM - AVS)/f_{OC} < 3000 μmols/g_{OC} may have adverse biological effects due to cadmium, copper, lead, nickel or zinc.

3) In any sediment in which (SEM - AVS)/f_{OC} > 3000 μmols/g_{OC} adverse biological effects due to cadmium, copper, lead, nickel or zinc may be expected.

4) Any sediment with AVS > 0.0 will not cause adverse biological effects due to silver.

6.1 AVS ESB

$$\Sigma_i[SEM_i] \le [AVS]$$

where

$$\Sigma_i[SEM_i] = [SEM_{Cd}] + [SEM_{Cu}] + [SEM_{Pb}] + [SEM_{Ni}] + [SEM_{Zn}] + 1/2[SEM_{Ag}]$$

6.2 Interstitial Water ESB

$$\Sigma_i \frac{[M_{i,d}]}{FCV_{i,d}} \le 1.0$$

where

$$\Sigma_i \frac{[M_{i,d}]}{[FCV_{i,d}]} = \frac{[M_{Cd,d}]}{[FCV_{Cd,d}]} + \frac{[M_{Cu,d}]}{[FCV_{Cu,d}]} + \frac{[M_{Pb,d}]}{[FCV_{Pb,d}]} + \frac{[M_{Ni,d}]}{[FCV_{Ni,d}]} + \frac{[M_{Zn,d}]}{[FCV_{Zn,d}]}$$

It is repeated here that the interstitial water benchmark applies only to the five metals: cadmium, copper, lead, nickel, and zinc. Silver is not included in this benchmark because the FCV for silver is not available.

Arguably, the most important additional data needed for assessing contaminated sediments along with ESBs are the results of toxicity tests. Sediment toxicity tests provide an important complement to ESBs in interpreting overall risk from contaminated sediments. Toxicity tests have different strengths and weaknesses compared to chemical-specific guidelines,

and the most powerful inferences can be drawn when both are used together (see U.S. EPA 2003c,d for further discussion of using toxicity testing with ESBs to assess contaminated sediments).

The ESB approaches are intended to protect benthic organisms from direct toxicity associated with exposure to metal-contaminated sediments. They are not designed to protect aquatic systems from metals release associated, for example, with sediment suspension, or the transport of metals into the food web from either sediment ingestion or ingestion of contaminated benthos. Furthermore, the ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans.

Section 7

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Appendix A

Lake Michigan EMAP Sediment Monitoring Database

Concentrations of SEM, AVS, TOC, and IWBU for cadmium, copper, lead, nickel, and zinc in 46 surficial samples from Lake Michigan

					IWBU						% Survival	
Sample	TOC (%)	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Cadmium	Copper	Lead	Nickel	Zinc	Sum	*Hyalella azteca*	*Chironomus tentans*
1	0.18	0.53	0.03[a]	0.51	—[b]	—	—	—	—	—	92.5	40
2	4.63	3.46	0.35	3.11	0.029	0.003[d]	0.00004	0.005	0.003	0.040	90	90
3	3.36	2.78	0.06	2.72	0.018	0.308	0.002	0.003	0.029	0.360	92.5	90
4	4.89	3.55	0.05[a]	3.50	0.018[c]	0.266	0.0004	0.003	0.006	0.293	100	97.5
5	0.92	0.14	0.03[a]	0.12	0.0002[c]	0.034	0.0008	0.006	0.032	0.073	0	90
6	4.37	2.82	1.13	1.69	0.024	0.049[d]	0.0002[e]	0.004	0.020	0.097	97.5	100
7	5.27	1.20	0.13[a]	1.07	0.029	0.003[d]	0.0001[e]	0.006	0.020	0.058	92.5	100
8	0.08	0.17	0.03[a]	0.15	0.115	0.003[d]	0.001	0.006	0.055	0.180	95	87.5
9	4.27	1.47	4.49[a]	-3.02	0.050	0.034	0.0008	0.004	0.026	0.115	95	100
10	2.11	0.25	0.03[a]	0.23	—	—	—	—	—	—	77.5	87.5
11	1.89	1.12	0.03[a]	1.10	—	—	—	—	—	—	97.5	100
12	0.41	0.74	0.07	0.67	0.0002[c]	0.070	0.002	0.0005[f]	0.001	0.074	—	—
13	2.87	1.17	0.18[a]	0.99	—	—	—	—	—	—	97.5	97.5
14	3.68	1.56	0.03[a]	1.54	0.0002[c]	0.003	0.0004	0.006	0.015	0.025	96.5	92.5
15	0.28	1.32	0.44	0.88	0.0002[c]	0.119	0.0002	0.004	0.050	0.173	90	87.5
16	0.07	0.17	0.05	0.12	—	—	—	—	—	—	100	100
17	3.51	0.75	0.08[a]	0.67	0.018	0.060	0.0008	0.008	0.058	0.145	100	100
18	0.40	0.97	0.03[a]	0.95	—	—	—	—	—	—	95	100
19	1.73	1.74	0.15[a]	1.59	0.079	0.013	0.0008	0.010	0.020	0.123	97.5	97.5
20	0.69	0.70	0.03[a]	0.68	—	—	—	—	—	—	97.5	97.5
21	2.51	0.19	0.05[a]	0.14	—[b]	—	—	—	—	—	75	92.5
22	1.17	0.59	0.03[a]	0.57	—	—	—	—	—	—	97.5	100
23	0.13	0.21	0.03[a]	0.19	—	—	—	—	—	—	57.5	65
24	1.03	0.62	0.03[a]	0.60	—	—	—	—	—	—	72.5	57.5
25	0.63	0.13	0.20[a]	-0.07	—	—	—	—	—	—	95	90
26	0.30	0.15	0.03[a]	0.13	—	—	—	—	—	—	—	—
27	0.29	0.25	0.03[a]	0.23	—	—	—	—	—	—	35	35
28	0.21	0.12	0.03[a]	0.10	0.0002[c]	0.155	0.0001[e]	0.011	0.0003	0.167	75	72.5
29	0.11	0.20	0.06[a]	0.14	0.0002[c]	0.003	0.0004	0.007	0.0003	0.011	80	82.5
30	0.05	0.04	0.03[a]	0.02	—	—	—	—	—	—	97.5	100
31	0.27	0.85	0.03[a]	0.83	—	—	—	—	—	—	97.5	97.5
32	4.95	1.17	1.66	-0.49	0.012	0.036	0.0004	0.002	0.020	0.070	97.5	95
33	0.54	0.44	0.12	0.32	—	—	—	—	—	—	100	100
34	6.75	1.37	0.09[a]	1.28	0.018	0.041	0.0002	0.017	0.012	0.088	95	90
35	0.18	0.26	0.03[a]	0.24	—	—	—	—	—	—	95	100
36	0.15	0.06	0.05	0.01	—	—	—	—	—	—	95	92.5
37	0.56	0.17	0.05	0.12	—	—	—	—	—	—	—	—
38	0.10	0.22	0.12[a]	0.10	—	—	—	—	—	—	60	55
39	0.06	0.06	0.03[a]	0.04	—	—	—	—	—	—	97.5	100
40	2.68	5.83	0.03[a]	5.81	0.003	0.119	0.001	0.0005[f]	0.020	0.144	90	95
41	0.16	0.16	0.07[a]	0.09	—	—	—	—	—	—	62.5	65
42	1.80	0.56	0.03[a]	0.54	0.006[c]	0.003[d]	0.0006	0.008[f]	0.015	0.033	75	95
43	1.29	1.02	2.25[a]	-1.23	0.0002[c]	0.028	0.002	0.0005[f]	0.044	0.075	100	55
44	0.05	0.06	0.03[a]	0.04	—	—	—	—	—	—	82.5	72.5
45	0.14	0.16	0.05[a]	0.11	—	—	—	—	—	—	—	—
46	0.57	0.66	0.03[a]	0.64	—	—	—	—	—	—	70	67.5

[a] AVS Limit of Detection =0.03 μm S/g.
[b] Insufficient interstitial water volume for metals analysis.
[c] Cadmium LOD=0.01 μg/L (0.0002 IWBU).
[d] Copper LOD=0.2 μg/L (0.0003 IWBU).
[e] Lead LOD=0.1 μg/L (0.0001 IWBU).
[f] Nickel LOD=0.5 μg/L (0.0005 IWBU).

Source: Columns for Sample, TOC, SEM, AVS, SEM-AVS, and IWBU taken directly from Leonard et al. (1996a). Column for survival from personal communication with E.N. Leonard, U.S. EPA, Duluth, Minnesota.

Appendix B

Saltwater Sediment Monitoring Database

Concentrations of SEM, AVS, toxicity, and TOC for EMAP, NOAA NST, and REMAP databases

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
EMAP-VA	0.289	1.400	-1.111	100	0	0.60
EMAP-VA	1.500	0.742	0.758	98	0	2.68
EMAP-VA	0.066	0.029	0.037	99	0	0.17
EMAP-VA	0.134	0.028	0.106	103	0	0.14
EMAP-VA	0.266	3.740	-3.474	99	0	0.49
EMAP-VA	0.266	1.080	-0.814	102	0	0.56
EMAP-VA	1.292	1.230	0.062	107	0	1.80
EMAP-VA	0.347	0.087	0.260	102	0	0.30
EMAP-VA	0.750	0.948	-0.198	99	0	0.95
EMAP-VA	0.212	0.283	-0.071	108	0	0.37
EMAP-VA	0.497	0.490	0.007	103	0	1.00
EMAP-VA	0.624	13.400	-12.776	113	0	1.58
EMAP-VA	0.032	0.024	0.008	101	0	0.11
EMAP-VA	0.988	81.100	-80.112	101	0	3.36
EMAP-VA	0.604	3.340	-2.736	107	0	1.38
EMAP-VA	0.031	0.331	-0.300	98	0	0.09
EMAP-VA	1.597	72.400	-70.803	102	0	4.19
EMAP-VA	1.065	8.480	-7.415	93	0	3.17
EMAP-VA	0.189	6.460	-6.271	103	0	0.32
EMAP-VA	0.018	0.034	-0.016	99	0	0.15
EMAP-VA	0.079	0.976	-0.897	97	0	0.14
EMAP-VA	0.421	3.210	-2.789	111	0	0.49
EMAP-VA	0.798	68.000	-67.202	104	0	2.84
EMAP-VA	0.903	3.150	-2.247	99	0	2.85
EMAP-VA	1.202	67.700	-66.498	105	0	2.28
EMAP-VA	0.159	3.310	-3.151	104	0	0.51
EMAP-VA	0.246	4.870	-4.624	106	0	0.71
EMAP-VA	0.687	2.420	-1.733	93	0	1.70
EMAP-VA	0.699	0.430	0.269	91	0	2.05
EMAP-VA	1.663	116.000	-114.337	100	0	4.12
EMAP-VA	0.083	1.300	-1.217	99	0	0.14
EMAP-VA	0.740	0.976	-0.236	101	0	2.30
EMAP-VA	0.878	1.220	-0.342	98	0	2.84
EMAP-VA	0.044	0.025	0.019	106	0	0.15
EMAP-VA	0.910	3.430	-2.520	104	0	3.00
EMAP-VA	0.567	0.621	-0.054	104	0	0.76
EMAP-VA	0.734	25.000	-24.266	107	0	2.21
EMAP-VA	2.171	5.610	-3.439	102	0	2.57
EMAP-VA	3.423	138.000	-134.577	100	0	4.14
EMAP-VA	0.197	0.892	-0.695	107	0	0.37
EMAP-VA	0.162	3.590	-3.428	82	0	0.81
EMAP-VA	2.803	11.900	-9.097	101	0	2.36
EMAP-VA	0.472	12.500	-12.028	101	0	2.77
EMAP-VA	2.079	26.600	-24.521	94	0	3.18
EMAP-VA	0.445	0.056	0.389	106	0	0.20
EMAP-VA	2.228	15.100	-12.872	103	0	2.92
EMAP-VA	0.847	17.300	-16.453	99	0	2.38
EMAP-VA	1.402	52.700	-51.298	109	0	2.70
EMAP-VA	1.425	22.300	-20.875	88	0	3.14
EMAP-VA	0.263	0.079	0.184	84	0	0.27
EMAP-VA	2.936	29.600	-26.664	100	0	4.15
EMAP-VA	0.394	0.031	0.363	87	0	0.18
EMAP-VA	3.074	10.400	-7.326	104	0	2.47
EMAP-VA	2.555	0.402	2.153	96	0	2.18
EMAP-VA	0.452	0.480	-0.028	100	0	1.07
EMAP-VA	0.173	0.201	-0.028	98	0	0.22
EMAP-VA	0.578	0.257	0.321	101	0	0.65

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
EMAP-VA	0.209	3.460	-3.251	96	0	0.36
EMAP-VA	5.411	17.800	-12.389	100	0	2.78
EMAP-VA	1.298	0.228	1.070	100	0	0.51
EMAP-VA	1.039	0.705	0.334	102	0	0.30
EMAP-VA	0.960	12.900	-11.940	94	0	1.91
EMAP-VA	7.369	3.460	3.909	87	0	1.86
EMAP-VA	1.380	2.270	-0.890	97	0	0.25
EMAP-VA	4.259	54.600	-50.341	76	0	2.47
EMAP-VA	8.229	68.000	-59.771	43	0	4.98
EMAP-VA	3.535	61.800	-58.265	99	0	3.19
EMAP-VA	2.543	35.600	-33.057	33	0	2.50
EMAP-VA	2.124	35.600	-33.476	0	0	2.15
EMAP-VA	0.188	0.836	-0.648	108	0	0.35
EMAP-VA	0.229	0.692	-0.463	95	0	0.46
EMAP-VA	1.820	0.227	1.593	104	0	1.90
EMAP-VA	3.468	14.600	-11.132	102	0	2.08
EMAP-VA	1.622	6.080	-4.458	102	0	2.02
EMAP-VA	0.693	1.200	-0.507	99	0	1.11
EMAP-VA	0.294	0.026	0.268	95	0	0.38
EMAP-VA	0.178	0.074	0.104	81	0	0.42
EMAP-VA	0.223	0.087	0.136	104	0	0.43
EMAP-VA	0.239	1.120	-0.881	88	0	0.31
EMAP-VA	0.801	5.120	-4.319	92	0	1.88
EMAP-VA	0.751	0.090	0.661	102	0	0.66
EMAP-VA	0.299	0.090	0.209	104	0	0.43
EMAP-VA	0.341	0.174	0.167	105	0	0.99
EMAP-VA	0.205	0.611	-0.406	95	0	0.71
EMAP-VA	2.415	4.050	-1.635	100	0	2.25
EMAP-VA	0.632	28.200	-27.568	88	0	3.35
EMAP-VA	1.516	52.700	-51.184	85	0	7.01
EMAP-VA	3.249	12.300	-9.051	103	0	3.29
EMAP-VA	0.462	6.140	-5.678	108	0	2.19
EMAP-VA	0.043	0.024	0.019	100	0	0.18
EMAP-VA	0.050	0.025	0.025	102	0	0.17
EMAP-VA	1.177	3.460	-2.283	100	0	1.83
EMAP-VA	0.624	6.210	-5.586	104	0	2.25
EMAP-VA	0.799	29.700	-28.901	100	0	4.10
EMAP-VA	0.020	0.259	-0.239	96	0	0.30
EMAP-VA	0.088	4.150	-4.062	100	0	0.25
EMAP-VA	2.220	59.600	-57.380	74	0	2.18
EMAP-VA	0.813	0.381	0.432	93	0	0.98
EMAP-VA	0.851	0.029	0.822	87	0	0.57
NOAA- LI	0.701	3.600	-2.899	100	0	0.74
NOAA- LI	1.113	3.510	-2.397	96	0	1.12
NOAA- LI	0.601	6.440	-5.839	96	0	1.43
NOAA- LI	1.505	18.730	-17.225	93	0	2.56
NOAA- LI	0.701	5.630	-4.930	93	0	0.77
NOAA- LI	0.717	13.090	-12.373	93	0	2.05
NOAA- LI	2.163	65.310	-63.147	92	0	3.22
NOAA- LI	0.616	6.940	-6.324	92	0	0.81
NOAA- LI	2.368	19.990	-17.622	91	0	3.02
NOAA- LI	1.278	4.710	-3.432	91	0	1.81
NOAA- LI	2.253	59.590	-57.337	91	0	2.51
NOAA- LI	0.865	3.880	-3.015	91	0	1.32
NOAA- LI	0.950	16.520	-15.570	90	0	1.52
NOAA- LI	1.113	14.950	-13.837	89	0	2.00

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
NOAA- LI	1.026	0.850	0.176	88	0	1.63
NOAA- LI	1.446	12.480	-11.034	88	0	2.05
NOAA- LI	2.777	29.720	-26.943	87	0	2.81
NOAA- LI	0.211	0.090	0.121	87	0	0.54
NOAA- LI	2.665	78.900	-76.235	87	0	3.33
NOAA- LI	2.813	35.050	-32.237	86	0	3.83
NOAA- LI	1.235	2.080	-0.844	84	0	1.58
NOAA- LI	2.198	14.690	-12.492	84	0	2.80
NOAA- LI	3.624	21.800	-18.176	83	0	2.48
NOAA- LI	3.594	27.410	-23.816	82	0	2.59
NOAA- LI	1.342	37.970	-36.628	82	0	1.85
NOAA- LI	2.462	46.450	-43.988	82	0	3.18
NOAA- LI	0.964	1.000	-0.036	81	0	1.60
NOAA- LI	0.332	4.010	-3.678	81	0	1.29
NOAA- LI	2.311	79.890	-77.579	81	0	3.69
NOAA- LI	0.623	6.610	-5.987	80	0	0.67
NOAA- LI	0.896	16.370	-15.475	80	0	1.11
NOAA- LI	0.544	2.170	-1.626	79	1	0.27
NOAA- LI	0.641	2.060	-1.419	79	1	1.56
NOAA- LI	0.355	1.390	-1.035	79	1	0.64
NOAA- LI	0.222	4.180	-3.958	77	1	0.45
NOAA- LI	2.262	39.960	-37.698	77	1	2.67
NOAA- LI	1.307	0.380	0.927	76	1	1.56
NOAA- LI	1.963	51.820	-49.857	76	1	3.46
NOAA- LI	2.785	61.020	-58.235	76	1	3.81
NOAA- LI	4.333	16.080	-11.747	75	1	3.48
NOAA- LI	1.927	3.710	-1.783	75	1	1.60
NOAA- LI	0.004	24.580	-24.576	74	1	2.87
NOAA- LI	3.831	9.250	-5.419	73	1	3.08
NOAA- LI	0.808	0.960	-0.152	71	1	1.19
NOAA- LI	1.783	40.630	-38.847	70	1	2.50
NOAA- LI	2.622	61.840	-59.218	70	1	3.49
NOAA- LI	0.597	1.090	-0.493	69	1	0.76
NOAA- LI	1.181	3.730	-2.549	68	1	0.91
NOAA- LI	1.862	50.390	-48.528	67	1	2.81
NOAA- LI	2.726	62.760	-60.034	67	1	2.81
NOAA- LI	2.102	33.630	-31.528	64	1	3.42
NOAA- LI	2.471	7.220	-4.749	63	1	2.80
NOAA- LI	1.870	17.120	-15.250	61	1	3.29
NOAA- LI	1.607	17.810	-16.203	59	1	2.07
NOAA- LI	4.942	100.800	-95.858	54	1	3.15
NOAA- LI	2.705	83.010	-80.305	53	1	3.62
NOAA- LI	2.087	26.730	-24.643	47	1	3.45
NOAA- LI	1.514	30.880	-29.366	42	1	2.69
NOAA- LI	2.629	32.050	-29.421	39	1	2.68
NOAA- LI	3.194	35.390	-32.196	37	1	3.17
NOAA- LI	0.872	25.810	-24.938	34	1	1.83
NOAA- LI	1.080	11.300	-10.220	16	1	1.91
NOAA- LI	0.123	5.310	-5.187	10	1	0.22
NOAA- BO	2.914	2.893	0.021	8	1	3.05
NOAA- BO	2.218	2.369	-0.151	15	1	2.89
NOAA- BO	2.609	43.959	-41.350	26	1	3.74
NOAA- BO	3.650	101.984	-98.334	29	1	1.83
NOAA- BO	1.634	5.237	-3.603	36	1	1.72
NOAA- BO	1.267	3.256	-1.989	52	1	1.53
NOAA- BO	2.892	80.584	-77.692	83	0	6.98

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
NOAA- BO	2.511	2.241	0.270	86	0	2.12
NOAA- BO	0.661	13.490	-12.829	87	0	1.00
NOAA- BO	2.458	23.077	-20.619	87	0	3.15
NNOAA- BO	1.872	48.062	-46.190	89	0	3.25
NOAA- BO	0.959	53.288	-52.329	90	0	2.39
NOAA- BO	2.480	7.599	-5.119	90	0	4.45
NOAA- BO	0.784	22.486	-21.702	91	0	1.88
NOAA- BO	0.943	8.831	-7.888	91	0	1.78
NOAA- BO	1.683	42.399	-40.716	92	0	3.41
NOAA- BO	1.753	17.697	-15.944	94	0	1.41
NOAA- BO	2.447	10.958	-8.511	94	0	4.45
NOAA- BO	1.839	68.306	-66.467	95	0	2.54
NOAA- BO	1.296	56.838	-55.542	96	0	3.05
NOAA- BO	1.697	9.089	-7.392	97	0	2.68
NOAA- BO	1.390	43.801	-42.411	97	0	3.27
NOAA- BO	2.310	51.857	-49.547	97	0	3.35
NOAA- BO	0.399	3.899	-3.500	99	0	0.80
NOAA- BO	2.481	19.604	-17.123	99	0	3.31
NOAA- BO	1.736	148.969	-147.233	99	0	2.94
NOAA- BO	0.958	18.622	-17.664	99	0	1.77
NOAA- BO	9.192	120.622	-111.430	100	0	4.61
NOAA- BO	1.525	81.842	-80.317	102	0	2.96
NOAA- BO	0.678	5.679	-5.001	103	0	1.45
NOAA- HR	5.037	69.320	-64.283	0	1	5.02
NOAA- HR	4.202	21.980	-17.778	41	1	3.47
NOAA- HR	1.174	27.540	-26.366	11	1	1.88
NOAA- HR	1.855	14.170	-12.315	18	1	4.44
NOAA- HR	3.092	51.770	-48.678	101	0	3.86
NOAA- HR	2.997	79.710	-76.713	112	0	3.09
NOAA- HR	2.581	61.050	-58.469	119	0	2.86
NOAA- HR	2.869	28.080	-25.211	81	0	2.50
NOAA- HR	5.442	25.900	-20.458	95	0	2.20
NOAA- HR	2.618	1.080	1.538	109	0	2.67
NOAA- HR	5.061	12.240	-7.179	97	0	2.98
NOAA- HR	2.376	4.390	-2.014	108	0	2.49
NOAA- HR	6.998	63.450	-56.452	0	1	1.98
NOAA- HR	4.480	20.780	-16.300	20	1	2.98
NOAA- HR	4.662	23.720	-19.058	14	1	3.19
NOAA- HR	5.896	51.580	-45.684	2	1	4.78
NOAA- HR	3.103	59.780	-56.677	77	1	3.99
NOAA- HR	1.662	7.230	-5.568	19	1	2.61
NOAA- HR	3.512	25.840	-22.328	0	1	4.44
NOAA- HR	0.273	0.050	0.223	91	0	0.07
NOAA- HR	0.335	0.036	0.299	93	0	0.07
NOAA- HR	1.664	18.760	-17.096	69	1	0.69
NOAA- HR	2.674	3.630	-0.956	3	1	1.00
NOAA- HR	5.532	29.210	-23.678	96	0	3.18
NOAA- HR	4.029	18.440	-14.411	51	1	2.20
NOAA- HR	4.614	20.530	-15.916	91	0	1.94
NOAA- HR	3.379	30.120	-26.741	88	0	2.80
NOAA- HR	4.240	19.320	-15.080	101	0	3.15
NOAA- HR	4.303	22.570	-18.267	102	0	3.02
NOAA- HR	5.209	14.570	-9.361	101	0	3.21
NOAA- HR	4.801	35.370	-30.569	70	1	2.98
NOAA- HR	4.697	54.710	-50.013	38	1	3.47
NOAA- HR	2.600	56.730	-54.130	37	1	1.47

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
NOAA- HR	1.013	10.160	-9.147	29	1	0.77
NOAA- HR	1.527	15.130	-13.603	68	1	0.95
NOAA- HR	0.505	0.630	-0.125	105	0	0.25
NOAA- HR	3.341	43.920	-40.579	86	0	2.55
NOAA- HR	3.449	37.860	-34.411	76	1	3.63
NOAA- BA	0.270	0.950	-0.680	96	0	0.26
REMAP-BA	0.341	0.156	0.185	84	0	0.06
REMAP-BA	0.888	12.971	-12.083	92	0	4.05
REMAP-BA	0.722	4.948	-4.226	85	0	0.40
REMAP-BA	0.362	0.936	-0.574	98	0	0.26
REMAP-BA	2.138	3.295	-1.157	95	0	0.43
REMAP-BA	3.008	3.941	-0.933	95	0	0.18
REMAP-BA	0.151	0.555	-0.404	96	0	0.15
REMAP-BA	0.115	0.156	-0.041	99	0	0.08
REMAP-BA	0.543	0.156	0.387	94	0	0.07
REMAP-BA	0.103	0.156	-0.053	85	0	0.05
REMAP-BA	0.167	0.932	-0.765	97	0	0.16
REMAP-BA	0.073	0.156	-0.083	99	0	0.05
REMAP-BA	0.294	0.156	0.138	91	0	0.34
REMAP-BA	0.120	0.156	-0.036	84	0	0.83
REMAP-BA	0.109	0.156	-0.047	92	0	0.92
REMAP-BA	0.185	0.156	0.029	90	0	4.48
REMAP-BA	0.120	0.156	-0.036	88	0	0.83
REMAP-BA	0.347	0.156	0.191	89	0	1.26
REMAP-BA	0.120	0.156	-0.036	81	0	0.62
REMAP-BA	2.275	16.592	-14.317	69	1	1.81
REMAP-BA	0.344	0.012	0.332	91	0	3.85
REMAP-BA	0.258	0.343	-0.085	94	0	0.77
REMAP-BA	0.119	0.156	-0.037	84	0	2.23
REMAP-BA	0.258	0.156	0.102	91	0	0.88
REMAP-BA	0.494	0.156	0.338	86	0	2.10
REMAP-BA	0.109	0.156	-0.047	89	0	4.07
REMAP-BA	0.266	0.156	0.110	86	0	1.06
REMAP-JB	0.327	0.393	-0.066	93	0	0.29
REMAP-JB	0.230	6.400	-6.170	83	0	0.19
REMAP-JB	2.026	47.793	-45.767	51	1	0.77
REMAP-JB	14.550	389.857	-375.307	0	1	1.52
REMAP-JB	3.332	243.322	-239.990	37	1	0.83
REMAP-JB	3.763	201.687	-197.924	79	1	0.97
REMAP-JB	0.357	10.923	-10.566	95	0	0.26
REMAP-JB	0.524	3.974	-3.450	98	0	0.35
REMAP-JB	0.244	4.502	-4.258	84	0	0.27
REMAP-JB	1.247	48.130	-46.883	91	0	0.54
REMAP-JB	2.478	47.376	-44.898	36	1	1.12
REMAP-JB	1.744	0.156	1.588	69	1	1.14
REMAP-JB	0.131	1.184	-1.053	94	0	0.21
REMAP-JB	0.846	0.927	-0.081	73	1	1.58
REMAP-JB	4.399	116.954	-112.555	93	0	6.55
REMAP-JB	3.884	237.650	-233.766	89	0	8.45
REMAP-JB	0.673	21.769	-21.096	77	1	4.11
REMAP-JB	3.150	43.975	-40.825	91	0	5.47
REMAP-JB	0.270	4.491	-4.221	91	0	0.74
REMAP-JB	0.162	0.873	-0.711	98	0	1.40
REMAP-JB	2.880	153.755	-150.875	92	0	7.70
REMAP-JB	0.323	1.684	-1.361	93	0	0.20
REMAP-JB	0.413	3.056	-2.643	94	0	1.20

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
REMAP-JB	0.377	3.056	-2.679	92	0	1.30
REMAP-JB	0.099	0.686	-0.587	93	0	0.75
REMAP-JB	1.100	58.945	-57.845	96	0	3.86
REMAP-JB	0.209	1.466	-1.257	93	0	0.58
REMAP-JB	0.213	0.780	-0.567	95	0	0.69
REMAP-LS	0.954	1.542	-0.588	83	0	0.26
REMAP-LS	2.759	6.498	-3.739	96	0	0.45
REMAP-LS	0.711	10.240	-9.529	97	0	0.56
REMAP-LS	1.915	12.596	-10.681	97	0	0.21
REMAP-LS	2.186	17.605	-15.419	95	0	0.27
REMAP-LS	2.480	23.523	-21.043	99	0	0.32
REMAP-LS	0.606	2.501	-1.895	98	0	0.25
REMAP-LS	3.289	91.773	-88.484	95	0	0.77
REMAP-LS	3.241	56.100	-52.859	97	0	1.14
REMAP-LS	0.616	1.070	-0.454	95	0	0.15
REMAP-LS	1.506	26.201	-24.695	96	0	0.95
REMAP-LS	2.485	28.248	-25.763	96	0	0.25
REMAP-LS	1.894	25.394	-23.500	93	0	0.98
REMAP-LS	3.149	64.643	-61.494	93	0	0.90
REMAP-LS	0.632	1.310	-0.678	87	0	1.51
REMAP-LS	1.057	4.647	-3.590	90	0	2.44
REMAP-LS	0.638	0.218	0.420	92	0	3.52
REMAP-LS	1.087	0.312	0.775	90	0	7.36
REMAP-LS	3.711	17.184	-13.473	88	0	3.99
REMAP-LS	2.990	59.256	-56.266	80	0	5.24
REMAP-LS	8.894	60.816	-51.922	85	0	3.63
REMAP-LS	1.277	23.266	-21.989	92	0	3.18
REMAP-LS	3.925	42.727	-38.802	90	0	3.85
REMAP-LS	5.632	114.770	-109.138	86	0	4.29
REMAP-LS	6.809	135.354	-128.545	91	0	4.36
REMAP-LS	7.645	150.012	-142.367	92	0	6.04
REMAP-LS	4.012	43.663	-39.651	86	0	3.73
REMAP-LS	3.905	26.229	-22.324	89	0	3.93
REMAP-NB	0.942	6.531	-5.589	84	0	0.67
REMAP-NB	3.515	7.134	-3.619	87	0	0.75
REMAP-NB	2.216	11.243	-9.027	86	0	1.22
REMAP-NB	3.323	7.573	-4.250	85	0	1.25
REMAP-NB	3.391	4.820	-1.429	83	0	1.05
REMAP-NB	3.443	3.982	-0.539	95	0	0.88
REMAP-NB	2.466	20.273	-17.807	82	0	1.40
REMAP-NB	2.294	11.046	-8.752	84	0	0.95
REMAP-NB	5.768	5.028	0.740	75	1	1.77
REMAP-NB	1.013	11.079	-10.066	90	0	0.76
REMAP-NB	2.479	25.687	-23.208	83	0	0.99
REMAP-NB	0.554	2.634	-2.080	84	0	0.60
REMAP-NB	5.222	22.617	-17.395	83	0	1.48
REMAP-NB	5.116	7.352	-2.236	9	1	1.45
REMAP-NB	14.791	109.780	-94.989	8	1	9.15
REMAP-NB	4.917	0.530	4.387	89	0	3.10
REMAP-NB	0.398	0.218	0.180	94	0	2.42
REMAP-NB	4.855	9.606	-4.751	83	0	2.62
REMAP-NB	3.290	10.105	-6.815	60	1	5.70
REMAP-NB	5.822	51.460	-45.638	41	1	2.22
REMAP-NB	9.167	93.563	-84.396	25	1	6.48
REMAP-NB	6.214	42.415	-36.201	68	1	3.24
REMAP-NB	0.794	2.651	-1.857	93	0	2.36

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
REMAP-NB	4.985	43.663	-38.678	53	1	3.90
REMAP-NB	5.280	1.934	3.346	83	0	6.10
REMAP-NB	2.268	6.300	-4.032	16	1	1.99
REMAP-NB	6.678	17.559	-10.881	77	1	15.20
REMAP-NB	2.833	45.222	-42.389	54	1	2.02
REMAP-RB	0.333	22.315	-21.982	93	0	1.23
REMAP-RB	0.756	1.216	-0.460	92	0	0.33
REMAP-RB	0.582	0.821	-0.239	94	0	0.30
REMAP-RB	1.012	0.567	0.445	94	0	0.30
REMAP-RB	1.596	0.447	1.149	95	0	0.17
REMAP-RB	0.326	0.156	0.170	93	0	0.08
REMAP-RB	2.709	3.120	-0.411	70	1	0.42
REMAP-RB	5.485	14.666	-9.181	92	0	2.29
REMAP-RB	3.596	19.503	-15.907	62	1	0.88
REMAP-RB	5.329	4.321	1.008	91	0	0.97
REMAP-RB	0.337	2.901	-2.564	97	0	0.53
REMAP-RB	0.986	0.156	0.830	96	0	0.12
REMAP-RB	0.856	0.156	0.700	96	0	0.51
REMAP-RB	5.364	39.700	-34.336	91	0	1.17
REMAP-RB	1.706	23.515	-21.809	93	0	3.21
REMAP-RB	0.371	4.210	-3.839	91	0	3.54
REMAP-RB	0.193	0.156	0.037	92	0	2.52
REMAP-RB	0.869	19.617	-18.748	85	0	2.39
REMAP-RB	1.288	0.593	0.695	92	0	2.44
REMAP-RB	1.650	0.624	1.026	91	0	2.68
REMAP-RB	2.422	0.156	2.266	98	0	2.60
REMAP-RB	0.512	0.156	0.356	93	0	0.42
REMAP-RB	4.198	4.086	0.112	90	0	2.63
REMAP-RB	5.081	36.490	-31.409	89	0	2.08
REMAP-RB	6.095	5.957	0.138	4	1	3.03
REMAP-RB	8.471	8.078	0.393	91	0	5.30
REMAP-RB	3.370	17.247	-13.877	94	0	3.91
REMAP-RB	1.198	0.156	1.042	94	0	1.03
REMAP-UH	2.127	12.446	-10.319	83	0	3.43
REMAP-UH	1.360	1.790	-0.430	99	0	1.26
REMAP-UH	1.197	3.373	-2.176	92	0	5.85
REMAP-UH	1.975	17.136	-15.161	45	1	2.33
REMAP-UH	2.829	25.189	-22.360	84	0	0.91
REMAP-UH	2.830	56.401	-53.571	96	0	1.21
REMAP-UH	1.385	44.588	-43.203	88	0	1.03
REMAP-UH	1.519	11.549	-10.030	82	0	1.06
REMAP-UH	3.186	86.235	-83.049	93	0	1.39
REMAP-UH	2.086	11.713	-9.627	82	0	0.79
REMAP-UH	1.799	12.631	-10.832	37	1	1.06
REMAP-UH	0.930	10.093	-9.163	89	0	0.43
REMAP-UH	0.459	0.156	0.303	98	0	0.13
REMAP-UH	0.889	2.623	-1.734	95	0	0.21
REMAP-UH	0.833	2.464	-1.631	86	0	4.96
REMAP-UH	1.317	15.563	-14.246	88	0	2.56
REMAP-UH	2.480	32.123	-29.643	87	0	3.06
REMAP-UH	0.626	9.949	-9.323	97	0	2.58
REMAP-UH	1.500	5.427	-3.927	89	0	2.71
REMAP-UH	0.723	1.341	-0.618	89	0	3.89
REMAP-UH	4.158	13.504	-9.346	96	0	4.78
REMAP-UH	2.241	27.788	-25.547	70	1	2.66
REMAP-UH	2.907	29.285	-26.378	95	0	5.15

Study[a]	SEM (μmol/g)	AVS (μmol/g)	SEM-AVS (μmol/g)	Survival[b] %	Significance[c] %	TOC %
REMAP-UH	0.852	1.591	-0.739	93	0	2.03
REMAP-UH	2.294	53.955	-51.661	15	1	4.37
REMAP-UH	2.995	33.995	-31.000	88	0	3.55
REMAP-UH	2.981	44.910	-41.929	94	0	2.97
REMAP-UH	0.677	10.323	-9.646	91	0	3.32

[a] *Sources*: EMAP-VA is U.S. EPA, 1996. NOAA-LI is Wolfe et al., 1994. NOAA-BO is Long et al., 1996. NOAA-HR is Long et al., 1995b. REMAP is Adams et al., 1996.

[b] Conclusion of significance varies for three databases. EMAP significance based on percent survival of control. NOAA significance based on percent survival less than 80%. REMAP significance based on percent survival less than 80%.

[c] Significance: 0, no significant toxicity; 1, significant toxicity.

Appendix C

Quality Assurance Summary for the ESB Document: Procedures for the derivation of equilibrium partitioning sediment benchmarks (ESBs) for the protection of benthic organisms: Metal Mixtures (Cadmium, Copper, Lead, Nickel, Silver, and Zinc)

All data were obtained either from the WQC document for the metals cadmium, copper, lead, nickel, silver, and zinc (USEPA, 1980, 1985b, c, d, 1986, 1987) or from a comprehensive literature search completed in 1999 and updated in 2004. Data for the chromium appendix was obtained from a comprehensive literature search completed in 2004.

All data used in the example benchmark calculations were evaluated for acceptability using the procedures outlined in the Stephan et al. (1985): *Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses*. Data not meeting the criteria were rejected. The approach for deriving the values in this document were also reviewed by the U.S. EPA SAB (U.S. EPA, 1994a; 1995a; 1999). All calculations were made using the procedures in Stephan et al. (1985). This document was reviewed for scientific quality assurance by U.S. EPA Office of Water and Office of Research and Development scientists.

Hard copies of all literature cited in this document reside at ORD/NHEERL Atlantic Ecology Division - Narragansett, Rhode Island.

Appendix D

Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmark (ESBs) for the Protection of Benthic Organisms: Chromium

Executive Summary

Chromium exists in sediments primarily in two oxidation states: Cr(III), which is relatively insoluble and nontoxic, and Cr(VI), which is much more soluble and toxic. Cr(VI) is thermodynamically unstable in anoxic sediments and AVS is formed only in anoxic sediments; therefore sediments with measurable AVS concentrations should not contain toxic Cr(VI). If this "chromium hypothesis" holds true, measuring AVS could form the basis for an ESB for chromium in sediments.

A review of the literature and recently performed experiments with both freshwater and saltwater sediments support the chromium hypothesis.

In saltwater:

1) Survival of amphipods was decreased by waterborne Cr(VI), with a 10-day median lethal concentration (LC50) of 1850 µg/L Cr(VI).
2) Survival of amphipods was not decreased by waterborne Cr(III) at concentrations well above saturation.
3) In both laboratory-spiked sediments with Cr(III) and Cr(VI) and field-contaminated sediments, in sediments where detectable AVS was present, chromium concentrations in interstitial water were very low (<100 µg/L) and no significant lethality to *A. abdita* was observed. In sediments in which AVS was not significantly greater than zero, chromium concentrations in interstitial waters increased significantly, with greater than 90% of the chromium present as Cr(VI), and the mortality of *A. abdita* was elevated.

In freshwater:

1) Survival of amphipods was decreased by waterborne Cr(VI), with a 42-day LC50 of 40 µg/L.
2) Cr(VI) spiked into test sediments with differing levels of AVS resulted in graded decreases in AVS.
3) Sediments with low AVS concentrations (<1 µmol/g) after spiking with Cr(VI) caused 100% mortality of amphipods, but no toxic effects were observed in Cr(VI)-spiked sediments that maintained higher AVS concentrations.
4) Waterborne Cr(III) levels near solubility limits caused decreased survival of amphipods at pH 7 and pH 8, but not at pH 6.
5) Sediments spiked with high levels of Cr(III) had no effect on amphipod survival, but caused significant decreases in reproduction and/or growth.
6) Interstitial waters of some Cr(III)-spiked sediments contained measurable concentrations of Cr(VI), but observed toxic effects did not correspond closely to concentrations of aqueous Cr species.

Thus, although both Cr(VI) and Cr(III) could be toxic to *H. azteca* in water and sediment, risks of Cr toxicity were low in sediments containing substantial concentrations of AVS. Results presented in this appendix suggest that measurements of AVS and interstitial water chromium can be useful in predicting the absence of acute effects from chromium contamination in both freshwater and saltwater sediments. In sediments with substantial AVS, risks of chromium toxicity should be low, because the chromium will be present in the form of Cr (III). This should apply to any sediment with SEM-AVS < 0.0. Sediments with SEM-AVS > 0.0, but which have substantial AVS present may be toxic due to copper, cadmium, lead nickel, or zinc, but should not be toxic due to chromium or silver. The relationship, (SEM-AVS)/f_{OC}, should be used with caution (with regard to chromium toxicity) in sediments with little or no AVS, because a sediment with no appreciable AVS or SEM and substantial chromium might be toxic due to chromium, even though no toxicity due to the other metals would be expected. These findings form the basis for a chromium ESB.

Section 1

Introduction

Chromium is often found in contaminated sediments (Pawlitz et al., 1977). Elevated chromium concentrations in sediments are usually associated with tanneries, smelters, and plating facilities. However, without a good understanding of the adverse biological effects of chromium in sediments, it is difficult to know what concentration of chromium in sediment may present ecological risk to benthos.

Although there have been several studies on the bioaccumulation of chromium from laboratory-spiked sediments (Wang et al., 1997; Griscom et al., 2000; Fan and Wang., 2001), there are few published studies on biological effects of chromium in laboratory-spiked sediments other than uptake of chromium. There are also very few reports on effects of chromium in field sediments. Leslie et al. (1999) found that a tributary below a chromium salt processing plant was incapable of supporting benthic macrofauna, presumably because of chromium leaching from stock piles along the banks of the tributary, but concluded that much of the chromium might be coming from the water rather than the sediment. In a study of sediments associated with a tannery, some toxicity was observed in ten-day static toxicity tests with several sediments with chromium in excess of 4000 μg/g; the same sediments, however, exhibited no toxicity in 28-day flow-through tests, suggesting that the toxicity observed in the 10-day static test was related to test conditions and duration and not sediment chromium (HydroQual, 1994). Several other studies have found elevated chromium concentrations in the tissues of benthos from sediments contaminated with high levels of chromium from mining activities (Bervoets et al., 1998) or tannery wastes (Catsiki et al., 1994), but these tissue concentrations were not linked to biological effects.

Part of the difficulty in understanding the biological effects of chromium in sediment is that chromium exists in sediments in two oxidation states, Cr(III) and Cr(VI), each with very different geochemical properties and toxicological effects. Cr(VI) is highly oxidized and unstable in reducing and even moderately oxidizing environments (DeLaune et al., 1998, Masscheleyn et al., 1992). Cr(VI) is also very soluble and highly toxic, while Cr(III) has very low solubility at environmentally relevant pH (DeLaune et al., 1998; Barnhart., 1997) and is generally thought to have relatively low toxicity (Wang et al., 1997; Thompson et al., 2002). For example, Leslie et al. (1999) assumed that the effects they saw due to chromium must have been caused by Cr(VI). However, they did not measure the chromium speciation.

This appendix provides the technical basis for the derivation of an ESB for chromium analogous to the ESB for the cationic metals cadmium, copper, lead, nickel, silver, and zinc discussed earlier. Determining the relationship between AVS and chromium in sediments would extend the utility of AVS measurements as a part of sediment assessments. Chromium should not necessarily be included among the SEM metals because its interaction with AVS is not via formation of an insoluble sulfide, but rather oxidation of sulfide and concomitant reduction of chromium. However, the geochemical relationship between AVS and chromium and the toxicological differences between oxidation states of chromium might be used to develop a theoretically-derived benchmark through what is called the "chromium hypothesis." The hypothesis is based on the concepts that Cr(III) is much less soluble and toxic than Cr(VI) and that Cr(VI) is not stable in reducing environments such as anoxic sediments in which AVS is formed. Thus, in a sediment where AVS is present, chromium will exist solely as Cr(III), and therefore the interstitial water should contain little chromium and the sediment should not be toxic due to chromium.

Although there is literature discussing chromium toxicity and geochemistry, no studies were available which had tested the "chromium hypothesis" directly. To this end, recently,

experiments have been carried out with both freshwater (Besser et al., *in press*) and saltwater sediments (Berry et al., *in press*) to verify the chromium hypothesis. In the saltwater test series ten-day water-only and ten-day spiked sediment toxicity tests with the amphipod *Ampelisca abdita* were performed with Cr(VI) and Cr(III). Ten-day sediment tests with saltwater sediments collected from a site contaminated with high concentrations of chromium were also performed. In freshwater sediments, chronic (28- to 42-d) water-only and spiked sediment toxicity tests with the amphipod *Hyalella azteca* were performed with Cr(VI) and Cr(III).

Section 2

Chemistry of Chromium in Sediment

2.1 Valence States of Chromium in Sediments

Studies by many researchers have provided a generalized model of the cycling of chromium between redox states in various aquatic soils/ sediments and their interstitial and overlying waters (Masscheleyn et al., 1992; Kozuh et al., 2000; Hassan and Garrison., 1996; Mattuck and Nikolaidis., 1996). This model is characterized by the relative stability of Cr(VI) in oxygenated overlying waters, particularly in marine waters, and rapid removal of Cr(III) through precipitation of the insoluble hydroxide and adsorption onto particulate matter. In freshwater systems with elevated dissolved organic carbon concentrations, such as in wetland soils and waters, a considerable amount of Cr(III) may be organically complexed, which slows the rate of removal to the particulate matter. In some circumstances, Cr(III) may be oxidized to Cr(VI) by Fe/Mn-rich films on the air-water interface where reduced Mn(II) and Fe(II) diffuses from sediments into oxic overlying water (Masscheleyn et al., 1992).

In sediments and soils, the reactivities of Cr(III) and Cr(VI) are somewhat reversed. Cr(III) may be oxidized to Cr(VI) in soils or sediments with high concentrations of MnO_2 and low organic content apparently by oxidation at MnO_2 surfaces (Hassan and Garrison., 1996). Similar oxidation by resuspended sediments rich in manganese oxides has also been postulated as the cause for relatively higher concentrations of Cr(VI) in deep ocean seawater relative to seawater overlying reduced coastal sediments (Nakayama et al., 1981). If organic content is elevated, however, Cr(III) is not oxidized, even in highly oxidizing sediments (Masscheleyn et al., 1992; Kozuh et al., 2000). On the other hand, Cr(VI) is reduced to Cr(III) and almost completely removed from solution in even moderately oxidizing sediments (redox potential E_h < 300 mV). In more reducing sediments (E_h < 200 mV), reduction is significantly more rapid due to reaction with ferrous ionic Fe(II). In such reduced sediments, very high partitioning constants indicate that almost all chromium is bound to the sediment, presumably as Cr(III), with very little mobile in interstitial waters (Mattuck and Nikolaidis., 1996). In wetland sediments, much of the dissolved chromium may be organically complexed Cr(III) (Icopini and Long, 2002). Once the reductive capacity of soils or sediments is exceeded, concentrations of dissolved Cr(VI) increase sharply and remain stable. Although the reductive capacity of sediments is generally proportional to organic content, the primary reductant is more likely Fe(II) or, in sulfidic sediments, sulfide.

2.2 Geochemical Distribution of Chromium in Toxicological Exposures

The geochemical distributions found in the recent experiments with marine sediments (Berry et al., *in press*) amended with Cr(VI) and Cr(III) reflected the behavior described in the previous section. Although the sediments had differing characteristics such as silt/clay and organic contents, they were both reducing sediments, as evidenced by the presence of AVS. Cr(III) added to these sediments in massive quantities was essentially inert to redox transformation: no Cr(VI) was evident in sediments, interstitial waters or overlying waters throughout the experiment, as was the case with organic-rich peat soils and wetland sediments. Cr(VI) added to the sediments was reduced completely and rapidly (<1 day), regardless of the amount added, up to the reductive capacity of the sediments. No significant amount of chromium was evident in either interstitial or overlying waters, indicating that Cr(III) complexed by dissolved organic carbon was not important in these sediments. Once the capacity of the sediments were exceeded, very high concentrations of chromium, almost entirely Cr(VI), were evident in interstitial

waters (Figure D-1). Concentrations of chromium in overlying waters decreased throughout the experiment and remained primarily as Cr(VI). These geochemical controls on the concentrations and redox speciation of chromium in sediments constrain exposure and consequent biological effects of the chromium on benthic organisms, such as the amphipods used in the saltwater tests.

In a recent study, spiking of freshwater sediments (Besser et al., *in press*) with several levels of Cr(VI) resulted in graded decreases in AVS concentrations and changes in POC concentrations in sediment and interstitial water. For example, mean AVS concentrations decreased by up to 97% in study sediments on day 0 of the test. Sediment TOC also decreased slightly in Cr(VI)-spiked sediments. Cr(VI) spikes were associated with increased DOC, increased alkalinity, and decreased hardness in interstitial waters.

Overlying and interstitial water Cr(VI) concentrations reflected differences in AVS concentrations among treatments. Initial concentrations of Cr(VI) in interstitial water samples were greater than 10,000 µg/L in treatments with the lowest AVS concentrations while interstitial water Cr(VI) concentrations remained low (≤20 µg/L) in treatments with higher AVS concentrations. In all three treatments with quantifiable Cr(VI) in interstitial water, concentrations decreased during the test. Cr(VI) concentrations in overlying water were much lower than those in interstitial water, but followed similar trends among treatments and over time. Decreases in Cr(VI) concentrations during the course of the study may have resulted from reactions with AVS and POC and from dilution due to replacement of overlying water. The smallest proportional decrease of Cr(VI) in interstitial and overlying water occurred in the treatment which had no AVS and low POC.

These results are consistent with the hypothesis that Cr(VI) concentrations remain low in sediments containing substantial concentrations of AVS. Berry et al. (*in press*), in studies with Cr(VI)-spiked marine sediments, did not detect Cr(VI) in interstitial waters of sediments spiked with Cr(VI) at Cr:AVS ratios of 2.2 or less. In contrast, substantial Cr(VI) concentrations were measured in sediments spiked at 3:1 Cr:AVS ratios (Besser et al., *in press*). AVS may have persisted in these treatments due to regeneration of AVS during the test, at least in some treatments. However, the data also suggested that some added Cr(VI) reacted with sediment POC, as has been reported in several previous studies (Wittbrodt and Palmer., 1995; Elovitz and Fish., 1995; U.S.EPA., 2002; Poleo., 1995). The relationship between Cr(VI) spikes and AVS depletion in one freshwater sediment was similar to the 2:1 ratio reported by Berry et al. (*in press*). However, AVS concentrations in a high-POC sediment decreased in a proportion of about one mole of AVS per eight moles of added Cr(VI). Reaction of Cr(VI) spikes with sediment POC is also suggested by decreases in organic carbon in several sediments and increases in interstitial water DOC in all three sediments. These results suggest that sediment POC also provides protection against Cr(VI) lethality in benthic environments, although Cr(VI) lethality occurred in some of the spiked sediments despite high levels of OM.

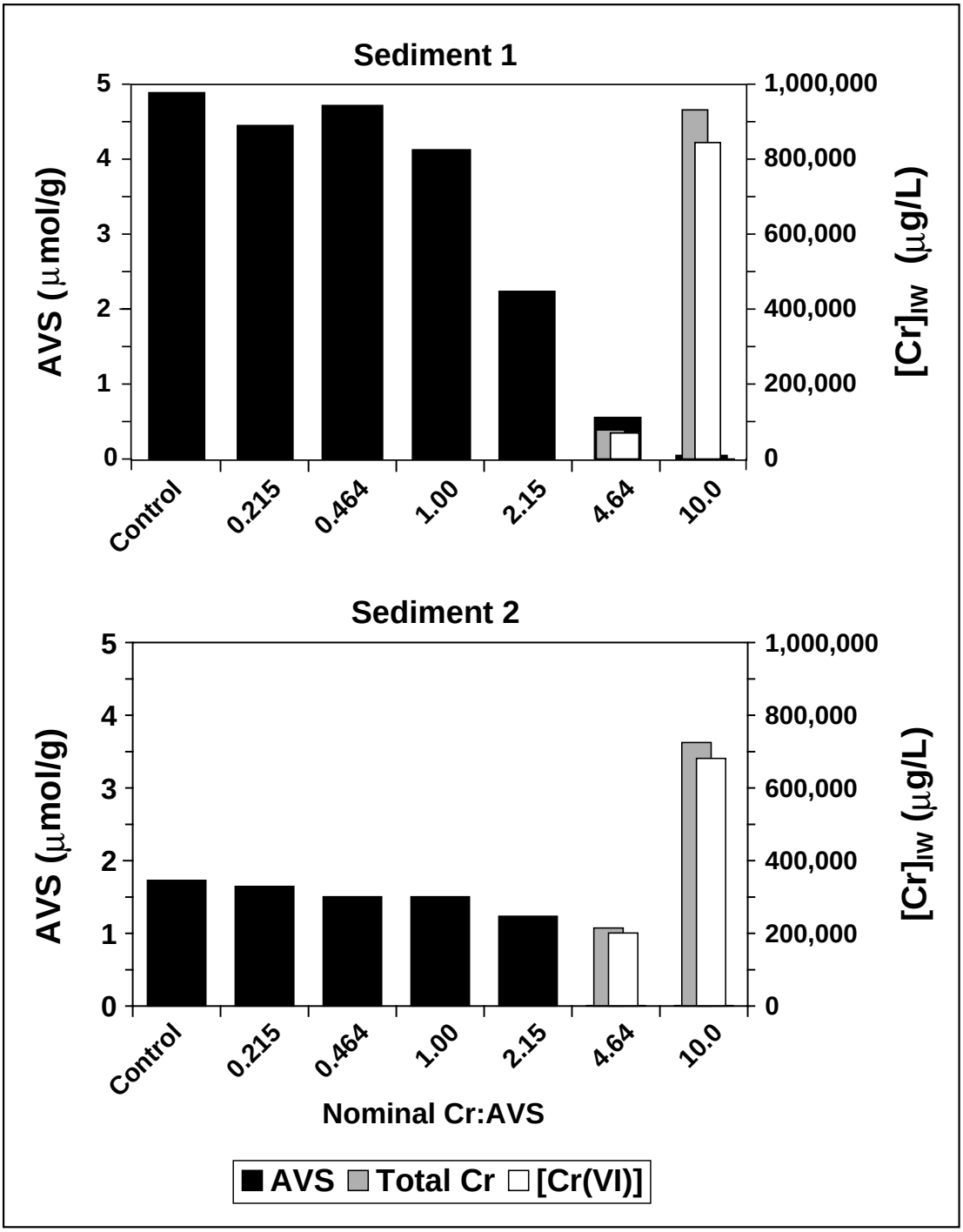

Figure D-1. Concentrations of AVS in sediment, and total Cr and Cr(VI) in interstitial waters of two saltwater sediments spiked with Cr (VI).

Section 3

Chromium Toxicity in Water and Sediment

3.1 Chromium Toxicity in Water-only Tests

Water-only tests were performed to complement the sediment toxicity tests conducted by Berry et al. (*in press*) and Besser et al. (*in press*). Survival of amphipods was decreased by Cr(VI) in water-only tests in saltwater, calculated 10-day LC50s were 1980 and 1854 µg Cr/L based on dissolved and hexavalent concentrations, respectively (Berry et al., *in press*). Survival of amphipods was not decreased by Cr(III) in water-only tests in saltwater at concentrations well above saturation.

Exposure to water-only Cr(VI) in freshwater caused decreased survival of *H. azteca* (Besser et al., *in press*) (Table D-1). Identical LC50s (40 µg/L) were determined for the 28- and 42-d exposure periods suggesting that lethal effects of Cr(VI) occurred early in the exposure. Evidence of sublethal effects of Cr(VI) on amphipods was less conclusive. Amphipod growth was not significantly decreased at any Cr(VI) exposure level, but reproduction in all Cr(VI) treatments was at least one-third less than controls. These results indicate that *H. azteca* is highly sensitive to chronic toxicity of Cr(VI). Excluding the reproduction data, the threshold for chronic Cr(VI) toxicity to *H. azteca* was 15 µg/L (geometric mean of Cr concentrations bracketing the lowest significant toxic effect) slightly greater than the current U.S.EPA water quality criterion for Cr(VI) of 10 µg/L (Richard and Bourg., 1991, U.S.EPA., 1995). Previous studies have reported chronic values for Cr(VI) between 6 µg/L and 40 µg/L for crustacean zooplankton and between 264 µg/L and 1987µg/L for fish (U.S.EPA, 1986).

Toxicity of Cr(III) in freshwater water-only tests was measured at 3 pHs: 6,7, and 8 (Besser et al., *in press*). Cr concentrations in the Cr(III) water-only test were less than the nominal concentration of 100 µg/L, indicating that Cr(III) concentrations were limited by solubility (Table D-2). Filterable Cr concentrations were highest at

Table D-1. Results of a toxicity test with the amphipod *H. azteca* exposed to Cr(VI) in water. Means with standard error in parentheses. Asterisks indicate significant difference between treatment and control (p<0.05; ANOVA and Dunnett's test with log-transformed data). From Besser et al. (*in press*).

Cr (ug/L) (n=4)	Survival (%) Day 28 (n=12)	Survival (%) Day 42 (n=8)	Length (mm) Day 42 (n=8)	Reproduction (young per female) (n=8)
<2.0	100 (0)	100 (0)	4.87 (0.11)	8.4 (2.4)
2.0 (0.3)	90 (7)	90 (7)	4.90 (0.07)	2.3 (1.1)*
4.7 (1.1)	95 (3)	95 (5)	4.84 (0.09)	2.9 (1.1)
10 (1.0)	98 (3)	95 (3)	5.27 (0.08)	5.4 (1.5)
18 (6)	88 (5)*	80 (4)*	5.05 (0.07)	3.3 (1.0)
48 (2)	38 (5)*	40 (9)*	5.22 (0.17)	1.6 (1.0)*

pH 6 and lowest at pH 8. Amphipod survival was high (≥90%) in controls at all three pH levels, but control growth and reproduction were significantly lower in pH 6 and pH 8 as compared to pH 7. Poor performance of amphipods in the pH 6 controls may indicate that this is near the lower limit of pH tolerance for this species, but growth and reproduction were also significantly decreased at pH 8, relative to the pH 7 control.

3.2 Spiked Sediments: Saltwater

Mortality of amphipods exposed to Cr(VI) in saltwater sediments increased with increasing chromium concentration, but the response was sediment dependent (Figure D-2a) (Berry et al., *in press*). In sediments where detectable AVS was present, chromium concentrations in interstitial water were very low (<100 μg/L). No significant lethality to *A. abdita* was observed in sediments with less than 0.5 interstitial water toxic units (IWTU) (Figure D-2b). In sediments in which AVS was not significantly greater than zero, chromium concentrations in interstitial waters increased significantly, with greater than 90% of the chromium present as Cr(VI), and *A. abdita* mortality was elevated (Figure D-2c). In a single treatment spiked with a high concentration of Cr(III) there was no chromium in the interstitial water, and the sediment was not toxic (Figures D-2a and D-2b). The results in these tests are consistent with the chromium hypothesis, and are similar to those for the other metals discussed in the main document.

3.3 Field Sediments: Saltwater

Berry et al. (*in press*) exposed amphipods for ten days to field sediments collected from Shipyard Creek, a tidal creek adjacent to a former ferrichromium alloy production facility in Charleston, SC, USA (Breedlove et al., 2002). The relationship between geochemical fractions and amphipod mortality in the field sediments was similar to that found with spiked sediments. AVS was measured at concentrations well above detection limits in all sediments, and despite some exceptionally high concentrations of total chromium (> 3000 μg Cr/g), only traces of Cr(VI) were detected (<4 μg/g) in sediments, and these concentrations were likely artifacts of the Cr(III)/

Table D-2. Results of toxicity test with the amphipod *H. azteca* exposed to Cr(III) in water at three pHs. Means with range (for pH) or standard error in parentheses. Within a pH level, asterisks indicate significant decreases in test endpoints in the Cr(III) treatment, relative to the control. For control sediments, means followed by the same letter are not significantly different (p≤0.05; ANOVA and Fisher's LSD test with log-transformed data). From Besser et al. *(In press*).

			Survival (%)		Length (mm)		Reproduction
Treatment	Chromium (μg/L)	pH	Day 28 (n=12)	Day 42 (n=8)	Day 28 (n=4)	Day 42 (n=8)	(young/female) (n=8)
Control - pH 6	<2	6.44 (6.00-7.12)	94 (1) ab	93 (2)	3.8 (0.1) b	3.3 (0.03) c	0 (0) c
Cr(III) - pH 6	76 (63-90)	6.41 (6.00-7.00)	98 (2)	95 (3)	4.3 (0.4)	4.3 (0.4)	1.0 (0.3)
Control - pH 7	<2	7.11 (6.90-7.34)	90 (2) b	93 (3)	4.4 (0.1) a	3.9 (0.04) a	1.4 (0.2) a
Cr(III) - pH 7	48 (38-54)	7.24 (6.96-7.42)	63 (5) *	60 (7) *	4.1 (0.1)	4.1 (0.1)	1.3 (0.6)
Control - pH 8	<2	7.98 (7.79-8.20)	95 (3) a	93 (4)	4.0 (0.2) b	3.6 (0.04) b	0.8 (0.2) b
Cr(III) - pH 8	29 (23-35)	7.94 (7.81-8.12)	63 (4) *	53 (5) *	3.9 (0.1)	3.9 (0.1)	2.0 (0.6)

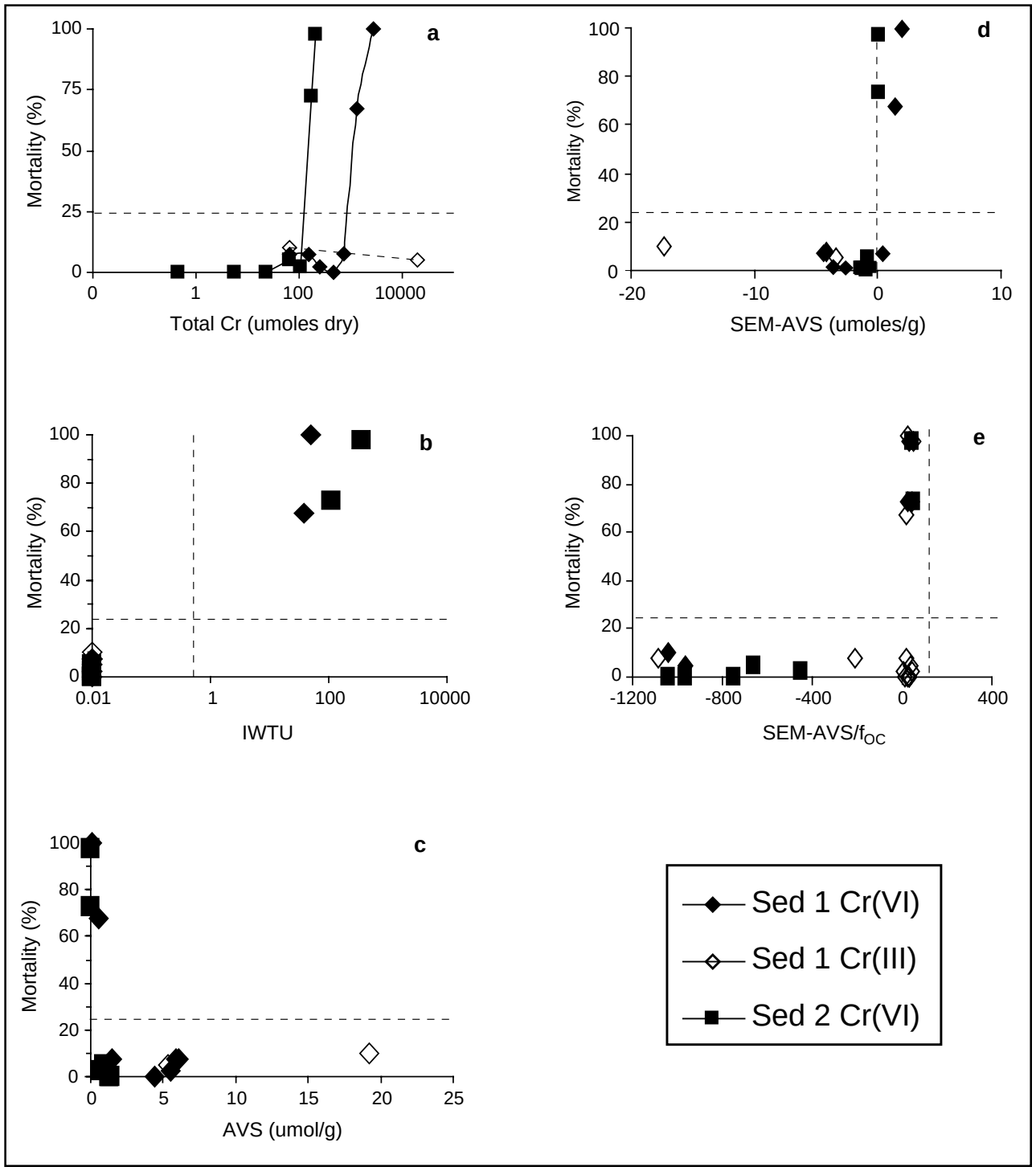

Figure D-2. Mortality in chromium-spiked saltwater sediment experiments (Berry et al., *in press*) vs. total chromium (a), IWTU (b), AVS (c), SEM – AVS (d), and SEM –AVS/f_{OC} (e). Where IWTU = interstitial water toxic units, AVS = acid volatile sulfide, SEM = simultaneously extracted metal, and f_{OC} = fraction of organic carbon. For illustrative purposes, sediments which caused greater than 24% mortality were classified as toxic (horizontal line), (Mearns et al., 1986). Vertical lines are drawn at 0.5 IWTU (b), 0.0 SEM-AVS (d), and 130 (SEM-AVS)/f_{OC} (e).

Cr(VI) separation technique (Berry et al., *in press*). No metals, including Cr(VI), were detected in interstitial waters of any of the sediments, which was expected given the large excess of AVS over SEM measured in all of the sediments. Despite concentrations of chromium exceeding 1700 to 3000 µg/g in some Shipyard Creek sediments, amphipod mortality in those sediments (5-25%) was no greater than in sediments from reference sites (5-20%) or a control sediment performed in conjunction with them (5-15%) (Berry et al., *in press*). These results are also consistent with the chromium hypothesis.

3.4 Spiked Sediments: Freshwater

The 28 and 42-day mortality results from the freshwater Cr(VI)-spiked sediment tests from Besser et al. (*in press*) were very similar to those from the 10-day saltwater Cr(VI) and Cr(III)-spiked sediment tests described by Berry et al. (*in press*). Mortality of amphipods exposed to Cr(VI) in freshwater sediments increased with increasing chromium concentration, but the response was sediment dependent (Figure D-3a) (Besser et al., *in press*). In sediments where detectable AVS was present, chromium concentrations in interstitial water were generally very low (Besser et al., *in press*). No significant toxicity to *H. azteca* was observed in sediments with less than 0.5 interstitial water toxic units (IWTU) (Figure D-3b). In sediments in which AVS was not significantly greater than zero, chromium concentrations in interstitial waters increased significantly (Besser et al., *in press*), with greater than 90% of the chromium present as Cr(VI), and mortality of *H. azteca* was elevated (Figure D-3c). Growth and reproduction were not significantly affected in any Cr(VI) -spiked treatment that did not show significant effects on survival (Besser et al., *in press*) (Figures D-3a ,D-3b, and D-3c)

The 28 and 42-day mortality results from the freshwater spiked sediment tests from Besser et al. (*in press*) with Cr(III) were also similar to those from the 10-day saltwater spiked sediment tests described by Berry et al. (*in press*) in that there was no increased mortality, even at high concentrations of Cr(III). However, the chemistry and sublethal results from the freshwater spiked sediment tests were different from the 10-day saltwater spiked sediment tests and the exposures with Cr(VI)-spiked freshwater sediment in several important respects. First, there was measurable chromium in the interstitial water of all three sediments spiked with a high concentration of Cr(III) (Figure D-3b). Also, there was significantly reduced growth in three of these sediments (Figures D-4a, D-4b, and D-4c) and reduced reproduction in one (Figures D-5a, D-5b, and D-5c). Finally, the reduced growth and reproduction was seen in some sediments which had less than 0.5 IWTU and/or significant amounts of AVS.

Besser et al. (*in press*) concluded that it was difficult to ascribe growth and reproductive effects in the Cr(III) - spiked sediments to chromium toxicity, because t he measured effects did not correspond with dissolved chromium concentrations, or with amphipod mortality. They hypothesized that the effects may have been a result of the physical effect of large amounts of chromium (presumably hydroxide) precipitate which forms when the Cr(III) solutions are pH-neutralized, prior to spiking (Besser et al., *in press*).

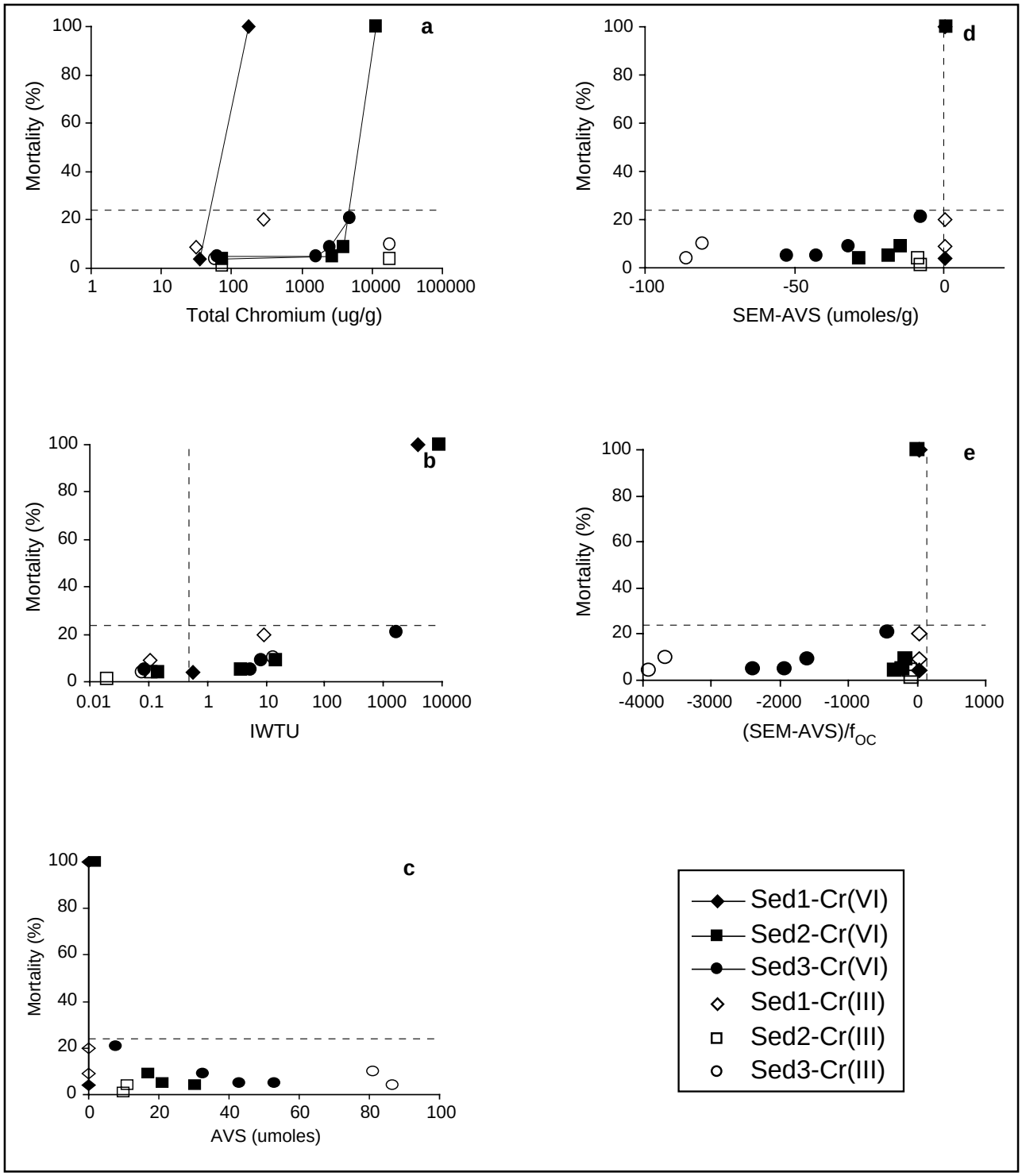

Figure D-3. Mortality in chromium-spiked freshwater sediment experiments (Besser et al., *in press*) vs. total chromium (a), IWTU (b), AVS (c), SEM – AVS (d), and SEM –AVS/f_{OC} (e). Where IWTU = interstitial water toxic units, AVS = acid volatile sulfide, SEM = simultaneously extracted metal, and f_{OC} = fraction of organic carbon. For illustrative purposes, sediments which caused greater than 24% mortality were classified as toxic (horizontal line) (Mearns et al., 1986). Vertical lines are drawn at 0.5 IWTU (b), 0.0 SEM-AVS (d), and 130 (SEM-AVS)/f_{OC} (e).

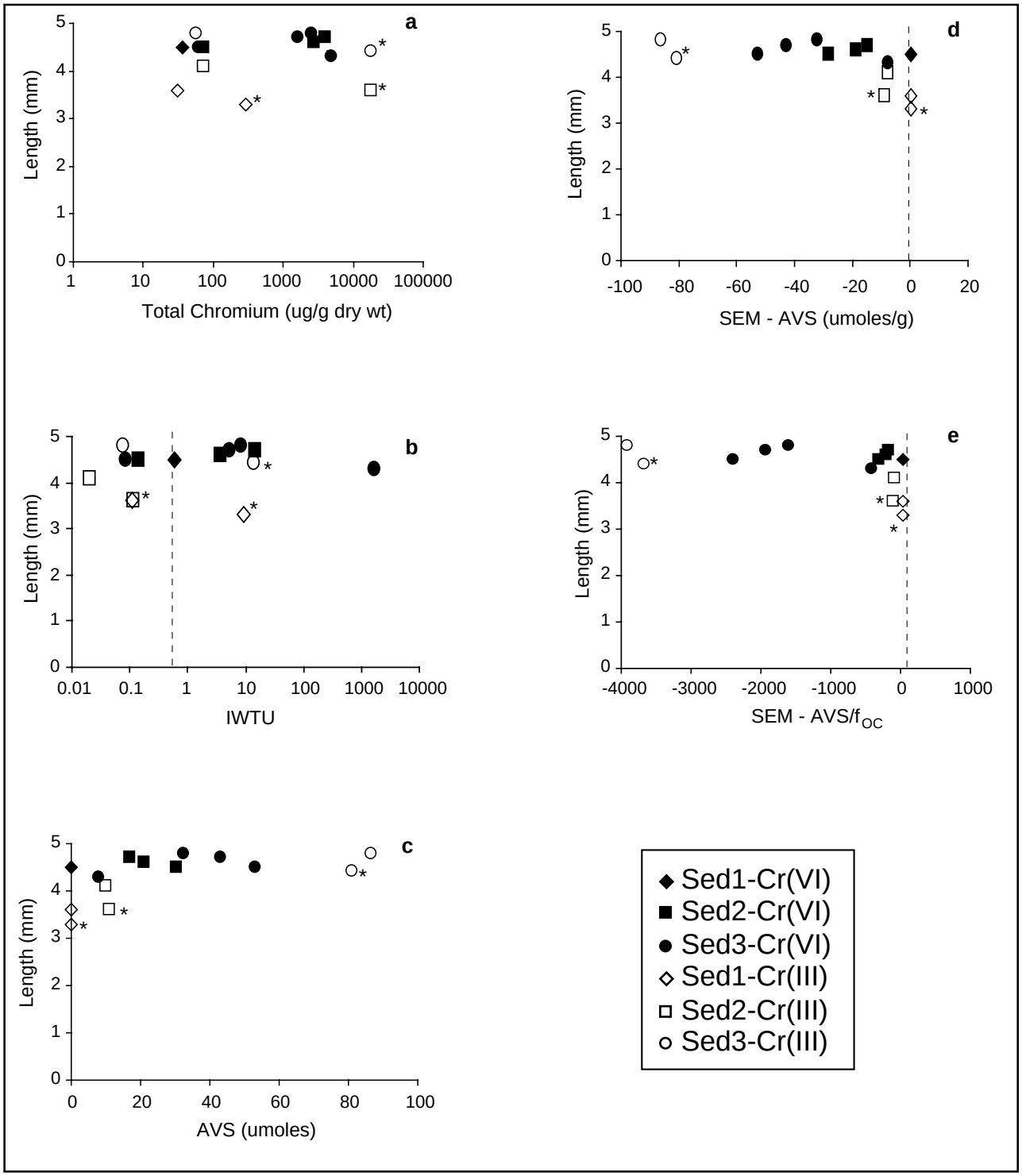

Figure D-4 Growth (length in mm) in chromium-spiked freshwater sediment experiments (Besser et al., *in press*) vs. total chromium (a), IWTU (b), AVS (c), SEM – AVS (d), and SEM –AVS/f_{OC} (e). Where IWTU = interstitial water toxic units, AVS = acid volatile sulfide, SEM = simultaneously extracted metal, and f_{OC} = fraction of organic carbon. Treatments significantly different from control are indicated with an asterisk. Vertical lines are drawn at 0.5 IWTU (b), 0.0 SEM-AVS (d), and 130 (SEM-AVS)/f_{OC} (e).

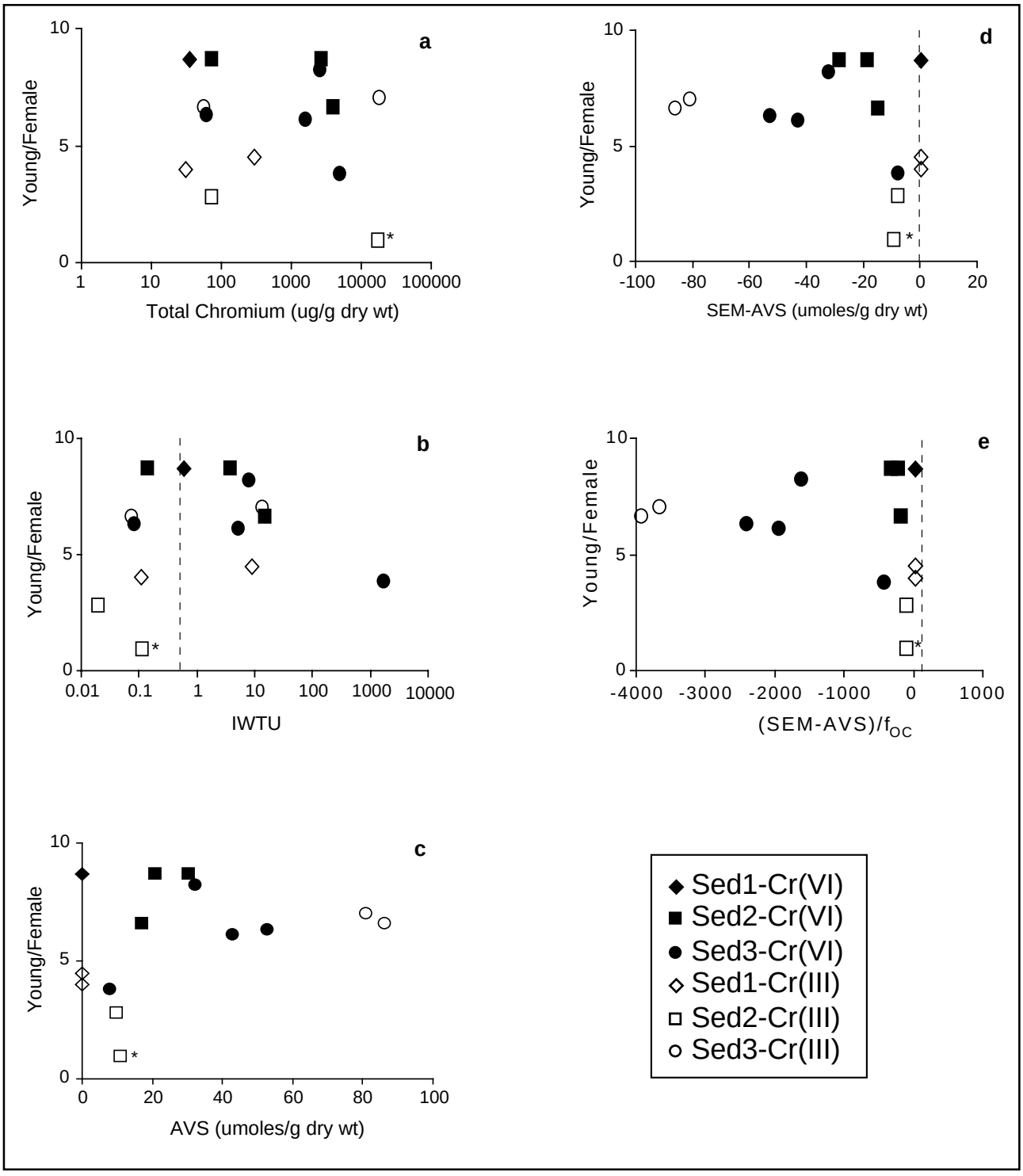

Figure D-5. Reproduction (young per female) in chromium-spiked freshwater sediment experiments (Besser et al., *in press*) vs. total chromium (a), IWTU (b), AVS (c), SEM – AVS (d), and SEM –AVS/f_{OC} (e). Where IWTU = interstitial water toxic units, AVS = acid volatile sulfide, SEM = simultaneously extracted metal, and f_{OC} = fraction of organic carbon. Treatments significantly different from control are indicated with an asterisk. Vertical lines are drawn at 0.5 IWTU (b), 0.0 SEM-AVS (d), and 130 (SEM-AVS)/f_{OC} (e).

Section 4

Derivation of ESB for Chromium

4.1. General Information

Mortality results of the toxicity tests conducted in both fresh and saltwater, with both spiked and field sediments, were generally consistent with the chromium hypothesis. They indicated that sediments with measurable amounts of AVS will not have acute toxicologically significant concentrations of chromium in the interstitial water, and that the sediments will not be acutely toxic due to chromium. Therefore, if measured sediment chemistry is being used as part of a sediment assessment, the presence of measurable AVS could be used to rule out chromium as the cause of observed acute toxicity. The chromium hypothesis can also serve as a foundation for a theoretically-derived sediment ESB for chromium.

The growth and reproduction results of the chronic tests conducted in freshwater with Cr(VI) were also consistent with the chromium hypothesis. The growth and reproduction results of the chronic tests conducted in freshwater with Cr(III) were more ambiguous. It is possible that these effects were observed as a result of the unrealistic conditions in the Cr(III)-spiked sediments, but more testing may have to be performed before the presence of growth and reproductive effects in sediments with large amounts of Cr(III) present can be ruled out.

4.2 Limitations of the chromium hypothesis

For the chromium hypothesis to work, and a Cr ESB to be useful, Cr(III) must not be toxic in interstitial water; however, many studies have reported on the toxic effects of Cr(III). Both the U.S. and Canada have water quality criteria (WQC) for Cr(III), although the criteria for Cr(III) are much higher than those for Cr(VI) (Pawlitz et al., 1997; U.S.EPA., 1985). Confounding factors in many of the tests used to develop these criteria, such as pH values outside of the tolerance range of test organisms (Dorfman., 1997) or reported LC50s orders of magnitude above limits of solubility for Cr(III) (Calabrese et al., 1973), make interpretation of the results of these tests difficult. Nonetheless, some of the tests used to develop the criteria demonstrate biological effects of Cr(III) at environmentally reasonable pH values and within limits of solubility (e.g., Stevens and Chapman (1984) showed chronic effects of Cr(III) on salmonid larvae).

Several recent studies have also shown biological effects due to Cr(III), including DNA damage and other sublethal effects associated with exposure to sediments from some of the same field sites from which Berry et al., (*in press*) collected sediments (Breedlovee et al., 2002), reduced growth of cyanobacteria (Thompson et al., 2002) and reduction in population growth rate of polychaetes (Mauri et al., 2002). Lastly, Besser et al. (2002) report reduced survival of the amphipod *Hyalella azteca* after 28 days in water-only exposures to Cr(III) at concentrations below solubility limits at a range of environmentally reasonable pHs. All of these reported effects are either sublethal or occurred after 10 days, so none of them would be expected to occur in the acute assays of Berry et al., (*in press*).

Another important fact to consider when deriving an ESB for chromium is that benthic animals, particularly tube and burrow dwellers such as *A. abdita*, modify the sediment around them by irrigation of their tubes and burrows, leading to changes in the sediment environment, and particularly in the redox condition of sediments near the animal (Wang et al., 2001). Thus, bulk sediment might have measurable AVS, while Cr(VI) might be present in oxic microenvironments within the sediment. The geochemistry of chromium argues against this,

however, because direct oxidation of Cr(III) to Cr(VI) by dissolved oxygen is slow (DeLaune et al., 1998), significant oxidation of Cr(III) to Cr(VI) occurs only in soils and sediments with elevated concentrations of manganese oxides and low organic content, conditions under which AVS would not be formed (Masscheleyn et al., 1992, Kozuh et al., 2000), and Cr(III) is very slow to react even in environments where it is thermodynamically unstable (Barnhart, 1997).

4.3 Incorporation into Multiple Metals Benchmark

In sediments where chromium is the only major metal of concern the AVS and interstitial water ESBs may be used as listed below. However, in many cases chromium will be present along with other metals, and will need to be evaluated along with them. One of the major objectives of this appendix is to expand the utility of the AVS methodology used with cadmium, copper, lead, nickel, silver, and zinc to include chromium. See sections 4 and 6 of the metals ESB for more detail on the benchmarks for cadmium, copper, lead, nickel, silver and zinc, and exact definitions of the AVS and interstitial water benchmarks.

Any sediment in which SEM -AVS < 0.0 should have low risk of adverse biological effects due to chromium, because measurable AVS must be present for this to be true (Figures D-2d, D-3d, D-4d, and D-5d). It should also have low risk of adverse biological effects due to cadmium, copper, lead, nickel, and zinc. Any sediment in which SEM - AVS > 0.0, but AVS > 0.0 should have low risk of adverse biological effects due to chromium or silver, but may have adverse biological effects due to cadmium, copper, lead, nickel or zinc. Sediments with SEM - AVS > 0.0 in which AVS does not exceed 0.0 may have adverse biological effects due to cadmium, copper, lead, nickel, silver, zinc and chromium.

The use of the (SEM - AVS)/f_{OC} benchmark in sediments contaminated with chromium is complicated slightly by the fact that a sediment with a slight excess of SEM - AVS may have AVS = 0.0, and thus be at risk to adverse biological effects of chromium, while at the same time not posing a risk due to cadmium, copper, lead, nickel, or zinc because of organic carbon binding. However, with an understanding of the chemistry of AVS, organic carbon, and metals it is possible to use the benchmark in sediment containing a mixture of metals including chromium. The interpretation of the benchmark with respect to cadmium, copper, lead, nickel, silver, zinc, and chromium is driven by four assumptions:

1) Any sediment with AVS > 0.0 will not cause adverse biological effects due to chromium or silver.
2) Any sediment in which (SEM -AVS)/f_{OC} < 130 μmols/g_{OC} should pose low risk of adverse biological effects due to cadmium, copper, lead, nickel and zinc.
3) Any sediment in which 130 μmols/g_{OC} < (SEM - AVS)/f_{OC} < 3,000 μmols/g_{OC} may have adverse biological effects due to cadmium, copper, lead, nickel or zinc.
4) In any sediment in which (SEM - AVS)/f_{OC} > 3,000 μmols/g_{OC} adverse biological effects due to cadmium, copper, lead, nickel or zinc may be expected.

Section 5

Sampling and Analytical Chemistry

5.1 General Information

All of the issues regarding proper sampling and analytical methods described for other metals (e.g., sampling biologically active zone, seasonal variation) are equally pertinent when chromium is an analyte of interest. Therefore, the guidance given on these topics earlier in this document are similarly appropriate. However, the differing physical and chemical characteristics of chromium in various oxidation states create additional concerns, both in sampling and analysis. For example, samples need to be collected and stored to preserve and minimize disturbance of existing redox conditions and thereby retain the distribution of solid and aqueous phase Cr(III) and Cr(VI) as much as possible. These chromium specific concerns are discussed below.

5.2 Sampling Sediment and IW

Normal procedures used to collect and preserve sediments for analysis of AVS and SEM are sufficient to preserve chromium speciation as well. Potential artifacts of sample handling and storage might include reduction of Cr(VI) or oxidation of Cr(III). The former should be addressed by keeping a sample cold, or even frozen, to inhibit *in situ* microbial reduction, while isolating a sediment sample from air, as well as chilling and freezing the sample, should eliminate the likelihood of oxidation of Cr(III) to Cr(VI) in sediments. Preservation of redox conditions in water samples, however; is significantly more problematic, and requires greater diligence.

As with sediment sampling, the guidance provided earlier in this document regarding collection of interstitial water is appropriate for samples in which chromium is an analyte of interest. Because of the potential of reduction of Cr (VI) to insoluble Cr(III) species within the sampler during the course of the experiment (Berry et al., *in press*), interstitial water samples should be filtered immediately after removal from the sampler, whether collected using centrifugation or *in situ* diffusion samplers (Berry et al., 1996). If centrifugation is used to isolate interstitial water, temperature should be kept low and the overlying atmosphere rendered inert to prevent possible oxidation of Cr(III) to Cr(VI) by Fe/Mn-rich films at the air-water interface (Masscheleyn et al., 1992).

Techniques that separate Cr species of different redox states should be applied to water samples as soon after collection as possible; if such separation cannot be obtained rapidly, samples should be frozen to preserve chemical speciation until such time as separation is practical. For example, Cr(III) and Cr(VI) species in overlying and interstitial water samples can be separated using a modified Fe(OH)3 coprecipitation technique (Berry et al., *in press*, Cranston and Murray, 1978) within hours of collection. Treatment with ion exchange resins to isolate Cr species has also been used (Besser et al., *in press*).

5.3 Chemical Analyses

5.3.1 Sediment Analysis

Techniques recommended for analysis of AVS in sediment samples are appropriate when chromium is a concern, with only slight modification of techniques for analyzing simultaneously extracted metals (SEM). As with water samples, if Cr(VI) is to be measured in the SEM solution, separation of redox species should be conducted as soon after filtration of the extract as possible. If Cr(VI) is expected to be a problem, it should be determined in an aliquot of the SEM extracts by using a modified $Fe(OH)_3$ coprecipitation technique to remove Cr(III) (Wang et al., 1997, Berry et al., *in press*) and analyzing Cr in the

supernatent by atomic spectrochemical means (e.g., inductively coupled plasma atomic emission spectrometry (ICP-AES) and graphite furnace atomic absorption spectrophotometry (GFAAS)).

5.3.2 Water analysis

Interstitial waters and overlying seawater from sediment tests may be analyzed for total and dissolved chromium and Cr(VI); however, analysis of chromium in saline waters at low concentrations can be problematic, so separation of redox species should only be conducted when evidence suggests the presence of Cr(VI) (i.e., AVS concentrations are near or below detection limits). Aliquots of water samples to be analyzed for dissolved metals should be filtered through a 0.4-micron polycarbonate membrane and then acidified with concentrated nitric acid (1% v/v), with Cr(VI) determined in subsamples of the dissolved sample using a modified $Fe(OH)_3$ coprecipitation technique (Cranston and Murray, 1978) as appropriate. Analysis of the various fractions may be conducted by GFAAS.

Section 6

Benchmark Sediment Values: Application and Interpretation

6.1 AVS Benchmark

The AVS benchmark for chromium is different from the SEM-AVS used for cadmium, copper, lead, nickel, silver and zinc because chromium does not form an insoluble sulfide. However, an AVS measurement is still useful in predicting the toxicity of chromium in a sediment, because sediments which have measurable AVS should be reducing in nature; therefore, most chromium should be present in the form of Cr(III), and the risk from acute toxicity due to chromium exposure should be low.

6.2 Interstitial Water Benchmark

The interstitial water benchmark is similar to that for cadmium, copper, lead, nickel, silver and zinc. If the interstitial water concentration of chromium does not exceed the chronic WQC FCV for Cr(VI) (10 µg/L in freshwater and 50 µg/L in saltwater (U.S.EPA., 1995)), the risk from chromium exposure should be low. The Cr(VI) WQC is used because most of the dissolved chromium in sediments should be in the form of Cr(VI), the freshwater benchmark for Cr(VI) is lower than that for Cr(III), and there is no chronic benchmark for Cr(III) in saltwater.

6.3 Incorporation into Multiple Metals Benchmark

The metals benchmark with respect to cadmium, copper, lead, nickel, silver, zinc and chromium is driven by four assumptions:

1) Any sediment with AVS > 0.0 will not cause adverse biological effects due to chromium or silver.
2) Any sediment in which (SEM -AVS)/f_{OC} < 130 µmols/g_{OC} should pose low risk of adverse biological effects due to cadmium, copper, lead, nickel and zinc.
3) Any sediment in which 130 µmols/g_{OC} < (SEM - AVS)/f_{OC} < 3,000 µmols/g_{OC} may have adverse biological effects due to cadmium, copper, lead, nickel or zinc.
4) In any sediment in which (SEM - AVS)/f_{OC} > 3,000 µmols/g_{OC} adverse biological effects due to cadmium, copper, lead, nickel or zinc may be expected.

These four assumptions should prove useful in the application of the chromium ESB in sediment assessments. However, the relationship (SEM - AVS)/f_{OC} should be used with caution (with regard to chromium toxicity) in sediments with little or no AVS. This is because a sediment with no appreciable AVS or SEM and substantial chromium might be toxic due to chromium, even though no toxicity due to other metals would be expected. Other potential limitations to the use of the chromium ESB are outlined in Section 4 of this appendix.

Use of an AVS based benchmark for assessing and predicting mortality in sediments due to chromium was successful in both freshwater and saltwater. Assessing and predicting sublethal toxicity in freshwater sediments was hindered by the observation of significant growth and reproductive effects in treatments where such effects were not expected. Causes of these effects remain ambiguous and may reflect sublethal chromium toxicity or experimental artifacts. Further study is needed to resolve these questions. Consequently, consistent with the recommendations of EPA's Science Advisory Board, publication of this document does not

imply the use of ESBs as stand-alone, pass-fail criteria for all applications; rather, exceedances of ESBs could trigger collection of additional assessment data.

Arguably, the most important additional data needed for assessing contaminated sediments along with ESBs are the results of toxicity tests. Sediment toxicity tests provide an important complement to ESBs in interpreting overall risk from contaminated sediments. Toxicity tests have different strengths and weaknesses compared to chemical-specific guidelines, and the most powerful inferences can be drawn when both are used together (see U.S. EPA 2003a,b for further discussion of using toxicity testing with ESBs to assess contaminated sediments).

The ESB approaches are intended to protect benthic organisms from direct toxicity associated with exposure to metal-contaminated sediments. They are not designed to protect aquatic systems from metals release associated, for example, with sediment suspension, or the transport of metals into the food web from either sediment ingestion or ingestion of contaminated benthos. Furthermore, the ESBs do not consider the antagonistic, additive or synergistic effects of other sediment contaminants in combination with metal mixtures or the potential for bioaccumulation and trophic transfer of metal mixtures to aquatic life, wildlife or humans.

Section 7

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Predicting Bioavailability of Sediment Polycyclic Aromatic Hydrocarbons to *Hyalella azteca* using Equilibrium Partitioning, Supercritical Fluid Extraction, and Pore Water Concentrations

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Polycyclic aromatic hydrocarbon (PAH) bioavailability to *Hyalella azteca* was determined in 97 sediments from six former manufactured-gas plants and two aluminum smelter sites. Measurements of Soxhlet extractable, rapidly released based on mild supercritical fluid extraction, and pore water dissolved concentrations of 18 parent and 16 groups of alkyl PAHs (PAH_{34}) were used to predict 28 day survival based on equilibrium partitioning and hydrocarbon narcosis models. Total PAH concentrations had little relationship to toxicity. Amphipods survived in sediments with PAH_{34} concentrations as high as 2990 μg/g, while sediments as low as 2.4 μg/g of PAH_{34} resulted in significant mortality. Equilibrium partitioning using either total extractable or rapidly released concentrations significantly improved predictions. However, pore water PAH_{34} concentrations were best for predicting amphipod survival and correctly classified toxic and nontoxic sediment samples with an overall model efficiency of 90%. Alkyl PAHs accounted for 80% of the toxicity, demonstrating that careful measurement of the 16 alkyl clusters in pore water is required. Regression analysis of the pore water PAH_{34} data from 97 field sediments against amphipod survival resulted in a mean 50% lethal residue value of 33 μmol/g of lipid, consistent with 32 μmol/g of lipid for fluoranthene determined by others in controlled laboratory conditions, thus demonstrating the applicability of EPA's hydrocarbon narcosis model when using pore water PAH_{34} concentrations.

Introduction

Industries that historically produced or utilized coal tars have been a major source of polycyclic aromatic hydrocarbons (PAHs) to the environment. Many such industries were (or are) located on waterways, with the result that PAH-contaminated sediments are often associated with these sites. In addition, sediments that have not been impacted by these industries contain background levels of PAHs from other sources such as atmospheric deposition of combustion particulates and background levels of PAHs that typically exceed baseline regulatory concentrations. For example, the regulatory based probable effects concentration (PEC, the total concentration of 13 parent PAHs above which toxicity is expected to be likely) and the threshold effects concentration (TEC, below which toxicity is considered unlikely) are 22.8 and 1.6 μg/g, respectively (*1*), but few urban sediments have PAH concentrations below either criteria. In a recent study of 114 field-collected sediments (both background and impacted), only 27 had PAH_{13} concentrations below the PEC, and only four sediments were below the TEC (*2*).

Efforts to improve predictive methods for the risks posed by sediments contaminated with PAHs have used equilibrium partitioning models to predict the partitioning of PAHs from sediments to water based on natural organic carbon–water partitioning coefficients (K_{OC}) (*3–6*). However, historically contaminated sediments often have much lower pore water concentrations of PAHs than predicted using literature K_{OC} values (*2, 7*), supposedly because of a greatly reduced availability of PAHs as sequestration processes occur. Measured K_{OC} values for historically contaminated sediments from manufactured-gas plant (MGP) sites have been reported to be as much as 3 orders of magnitude higher than literature K_{OC} values used in equilibrium partitioning models (*2, 7*). There are also an increasing number of reports demonstrating that many different carbon types (such as coal, combustion soots, charcoal, and coal tar pitch) are present in many sediments that bind PAHs much more tightly (and result in much less partitioning to water) than predicted by equilibrium partitioning models using K_{OC} values based on natural organic carbon (*2, 7–12*).

Recently, there has been an increasing amount of evidence that the bioavailability of PAHs can also be overestimated using equilibrium partitioning models based on K_{OC} values that are normally used to describe partitioning with natural organic carbon. Several investigators have reported the lack of observable toxicity to aquatic organisms, despite high sediment concentrations of PAHs (*13–16*), and PAH uptake in *Lumbriculus variegatus* was found to be as much as 1000-fold less than predicted by sediment PAH concentrations and equilibrium partitioning models (*17*). In a recent report, the toxicity of PAHs in sediments from MGP sites to *Hyalella azteca* was found to be much lower than predicted by the equilibrium partitioning model but was more accurately predicted when either pore water PAH concentrations or the rapidly released PAH concentrations measured by mild supercritical fluid extraction (SFE) were used rather than sediment concentrations (*18*). The use of mild SFE to obtain rapidly released PAH concentrations has also previously been reported to correlate with water desorption of PAHs from soils (*19, 20*) and to improve the prediction of earthworm uptake of PAHs from soil (*21*).

In the present study, we compare three approaches to predict the availability of PAHs to *H. azteca* based on a 28 day chronic toxicity test. Ninety-seven background and industrially impacted sediments were studied from six different MGP sites and from two aluminum smelting operations. Both 18 parents and 16 groups of alkyl PAHs (PAH_{34}) suggested by the U.S. EPA (*5*) were measured in the sediment, the pore water, and the rapidly released or available fraction based on mild SFE (*19–21*). Predictions of the internal

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PAH concentrations expressed on a lipid basis (and thus, toxicity to the organism) were based on the U.S. EPA's PAH equilibrium partitioning/hydrocarbon narcosis model (*3*–*6*), which uses generally accepted K_{OC} values to predict pore water PAH concentrations from sediment concentrations, followed by the use of octanol–water coefficients (K_{OW}) to predict the organism lipid PAH concentrations. Biota lipid PAH concentrations were also predicted using the rapidly released sediment PAH_{34} concentrations and the measured pore water PAH_{34} concentrations as input data to the equilibrium partitioning model proposed by the U.S. EPA (*3*–*6*).

Experimental Procedures

Sediment Collection and Characterization. Sediment collection procedures and analytical methods have been described in detail in earlier reports (*2*, *22*, *23*). In brief, approximately 150 sediments were collected using a Ponar grab sampler or, in a few cases, with a shovel. Approximately 15 L of the sediment–water slurry was transferred to a clean bucket, sieved through a 2 mm screen, briefly mixed, transferred to new glass jars with Teflon-lined lids, and immediately placed on ice. This procedure resulted in sediment–water slurries with approximately 40–70% water content. Samples were shipped by overnight air to the analytical and toxicology laboratories and stored at ca. 4 °C until use. All analytical tests were completed, and all biological tests were begun within 28 days of sample collection. All sediments were initially screened for PAH concentrations and total organic carbon (TOC) to select 97 sediments that best represented the range of PAH concentrations, organic carbon contents, and sediment textures existing at each site, as well as to spatially represent the site. Field reference sediments were also selected from each site that were not contaminated by the MGP or aluminum smelter activities (based on their PAH concentrations).

TOC and BC (black or soot carbon) were determined by elemental analysis (C,H,N) after acidification with HCl to remove inorganic carbonates. Samples for BC were prepared by oxidation under air at 375 °C for 24 h in a gas chromatographic oven (*24*). Pore water dissolved organic carbon (DOC) was determined after alum flocculation using the U.S. EPA method 415.1.

The 34 PAHs (18 parent and 16 groups of alkyl PAHs) listed for the sediment PAH narcosis model by the U.S. EPA (*5*) were determined in all sediment Soxhlet extracts, SFE extracts, and pore water samples. Each method used several 2- to 6-ring perdeuterated PAHs as analytical internal standards to aid in quantitation (*22*, *23*). Pore water PAH concentrations were obtained in quadruplicate by briefly centrifuging the sediment–water slurry, flocculating with alum twice to remove colloids, adding the perdeuterated internal standards, and quantitative analysis of the dissolved pore water PAH concentrations with solid-phase microextraction (SPME) and GC-MS with selected ion monitoring (*22*). SFE available and total extractable sediment PAH concentrations were based on extractions of quadruplicate 2 g samples of the sediment, after the pore water fraction was removed by centrifugation, and the sediments were mixed with 4 g of sodium sulfate (*23*). SFE was performed for 40 min with pure carbon dioxide at 200 bar, 50 °C, and a flow rate of 1.0 mL/min (*19*, *20*). Soxhlet extraction was performed for 18 h with 150 mL of acetone–methylene chloride (1:1).

Toxicity Testing. Toxicity to *H. azteca* was determined using EPA method 100.4 for a 28 day exposure period as previously described (*18*).

Data Analysis. Statistical analysis of amphipod survival data was performed to estimate the dose response for each of the three chemical measurements of PAHs. A Probit regression model was run in SAS (SAS Institute, Cary, NC) using the Probit procedure to estimate the mean amphipod survival. The statistical fits (χ^2 test of significance) of the Probit regression models were evaluated for significance ($\alpha = 0.05$), and the mean dose and probability were presented as the Probit regression line. The 85% survival dose was determined by the lower 95% confidence interval for a probability of 85% survival, and the 15% survival dose was represented by the upper 95% confidence interval for a probability of 15% survival. The modeled dose response for each of the three chemical measurements of PAHs was evaluated for statistical fit using a binary logistic regression model and the Goodman–Kruskal γ (*25*). The Goodman–Kruskal γ is a rank-order correlation statistic used as a measure of association for the ability of a predictor variable (e.g., pore water concentrations) to explain the response variable (the binary variable toxic or nontoxic). The γ value ranges from −1.0 (no predictive ability) to 1.0 (perfect predictor).

TABLE 1. Summary of Sediment and Pore Water Characteristics

	units	minimum	maximum	median
Bulk Sediment[a]				
total PAH_{34}	μg/g	1.31	17 600	277
total PAH_{16}	μg/g	0.22	8580	128
TOC	wt %	0.3	42.4	3.3
BC	wt %	0.1	39.7	0.8
Fraction (BC/TOC)	%	5.3	100	33.4
% alkylated PAHs[b]	%	25	94	53
2- and 3-ring PAHs/ total PAH_{34}[c]	%	6	96	38
Sediment Pore Water				
total PAH_{34}	ng/mL	0.02	10 900	17.4
total PAH_{16}	ng/mL	0.02	9250	5.84
DOC	mg/L	1.4	114	4.35

[a] Sediment PAH concentrations are on a dry weight basis. [b] Total concentration of alkyl PAHs divided by total PAH_{34} concentration. [c] Sum concentration of all 2- and 3-ring PAHs divided by total PAH_{34} concentration.

Results and Discussion

Sediment Characteristics and PAH Concentrations. Sediment textures ranged from coarse sand to fine-grained silts and clays. The TOC ranged from 0.3 to 42 wt %, and BC ranged from 0.1 to 40 wt %, indicating both low and high impact from industrial carbon residues. The fraction of BC as compared to TOC ranged from 5% (indicating no significant contribution of BC) to 100% (indicating that all organic carbon in that sediment was present as BC).

PAH concentrations for the 97 test sediments are summarized in Table 1 and Table S1. PAH concentrations ranged from very low background concentrations (<μg/g) to sediments contaminated as high as 1.8 wt % (17 600 μg/g) total PAH_{34}. The fraction of low molecular weight PAHs (sum of 2- and 3-ring PAHs) as compared to the total EPA_{34} PAHs (2–6-ring) ranged from 6% (indicating highly weathered PAHs from coal tar or coal tar pitch) to 96% (indicating unweathered coal tar PAHs) (Table 1). Pore water concentrations also ranged over several orders of magnitude, from a lowest detected concentration of 0.02 ng/mL to a highest detected concentration of 10 900 ng/mL (PAH_{34}). A more complete description of the individual 34 PAH concentrations for sediment, the SFE rapidly released fractions, and the pore water concentrations is given in Table S1.

Approximately 10 impacted sediments showed significant heterogeneity in the quadruplicate Soxhlet extracts and in the SFE extracts. For these sediments, the relative standard deviations (RSDs) for individual and total PAH_{34} concentrations in the quadruplicate extracts sometimes exceeded 50

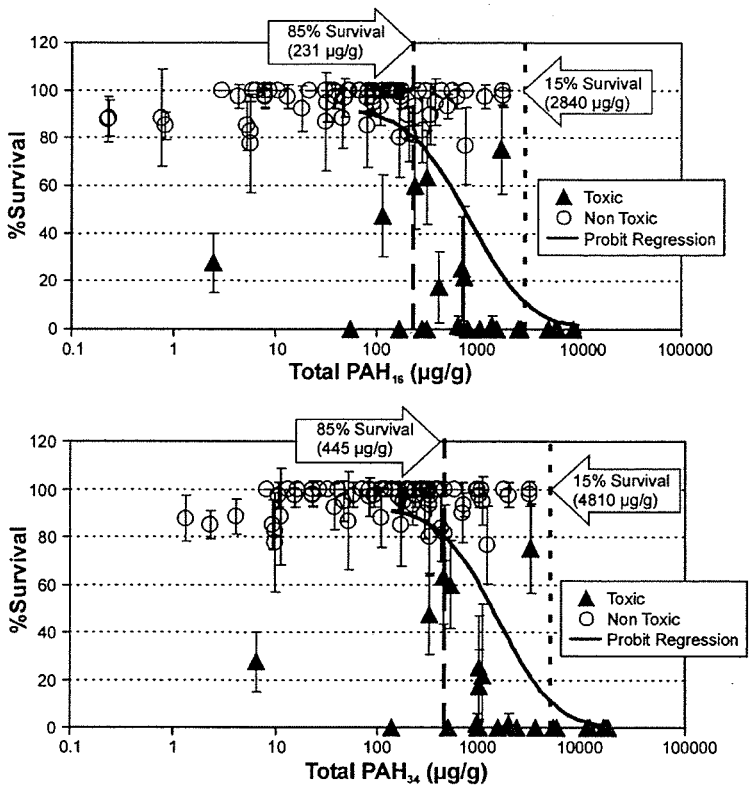

FIGURE 1. ***H. azteca*** **survival as compared to total extractable PAH_{16} and PAH_{34} sediment concentrations.**

or 100%, and for such sediments, the replicate extracts usually ranged from light yellow to dark brown. When this occurred, a second set of quadruplicate samples was extracted and analyzed, and in all cases, the sediment PAH heterogeneity was confirmed, demonstrating that the sediments contained blebs of nonaqueous-phase coal tars, pieces of coal tar pitch, or pieces of other material that were highly contaminated with PAHs. In many cases, visual observation under a low-power microscope confirmed the presence of pitch particles and/or tar droplets. In contrast, the quadruplicate pore water samples from each sediment showed good reproducibility, with % RSDs typically less than 10% for low- and mid-molecular weight PAHs and typically less than 15% for higher molecular weight PAHs.

Survival Predictions Based on Sediment Total Extractable PAH Concentrations. Out of the 97 sediments used to compare toxicity predictions, 25 were found to result in reduced survival to *H. azteca* following 28 day exposures. Both MGP and aluminum smelter sites had toxic sediments, with 17 out of the 73 MGP sediments causing reduced survival and eight of the 24 aluminum smelter sediments causing reduced survival.

The poor relationship between total PAH concentrations expressed as micrograms per gram (whether EPA_{16} or EPA_{34}) and *H. azteca* survival is shown in Figure 1. When compared to the probable effects concentration (PEC) of 22.8 µg/g (PAH_{13}) (*1*), 73 of the sediments have total PAH_{13} concentrations that exceed the PEC, yet 67% of those that exceed the PEC value are nontoxic. Similarly, the total extractable PAH_{34} concentration shows little relationship to *H. azteca* survival (Figure 1). Except for sediments with very low or very high PAH concentrations, total PAH concentrations have little or no ability to predict toxicity. (Note that the plot for total PAH_{13} versus toxicity is essentially identical to that shown in Figure 1 for total PAH_{16} since PAH_{13} concentrations are only a few percent lower than the PAH_{16} values for all of these sediments.)

The use of equilibrium partitioning models as developed by DiToro and others and proposed as a regulatory sediment guidance approach by the U.S. EPA (*3*–*6*) to improve toxicity predictions is explored in Figure 2. This model first predicts pore water concentrations from sediment concentrations using literature and modeled K_{OC} values. The resultant pore water PAH concentrations are then used to predict biota lipid concentrations using K_{OW} values for each of the 34 parent and groups of alkyl PAHs (*3*–*6*). Thus, proper prediction of the final lipid total molar PAH concentration depends heavily on the assumption that K_{OC} values used for each individual PAH (and each group of alkyl clusters) are correct and that each PAH has a single K_{OC} value that applies to all sediments.

As shown in Figure 2 (top), the use of total extractable PAH_{34} sediment concentrations and the equilibrium partitioning model (*5*, *6*) significantly improves the ability to predict survival to *H. azteca* over the simple use of total extractable (PAH_{34} or PAH_{16}) concentrations shown in Figure 1. The overall model efficiency (% of correct predictions) improves from 48 to 80% (Table 2). However, substantial overlap between toxic and nontoxic sediments still exists, and 26 of the 97 sediment samples lie within the region of 85–15% survival where it is difficult to make statistically strong predictions of toxicity (Table 2). The scatter in predicted versus actual survival shown in the top of Figure 2 might be expected based on a recent report where the K_{OC} values for each individual PAH from a similar set of sediments (including most of the sediments reported here) varied by nearly 3 orders of magnitude for the same PAH from different sediments. For example, while a log K_{OC} value for pyrene of 4.84 is used for all sediments in the model (*5*), measured values of log K_{OC} in 114 sediments ranged from 4.17 to 7.40, and the median log K_{OC} was 5.81, nearly one log unit higher

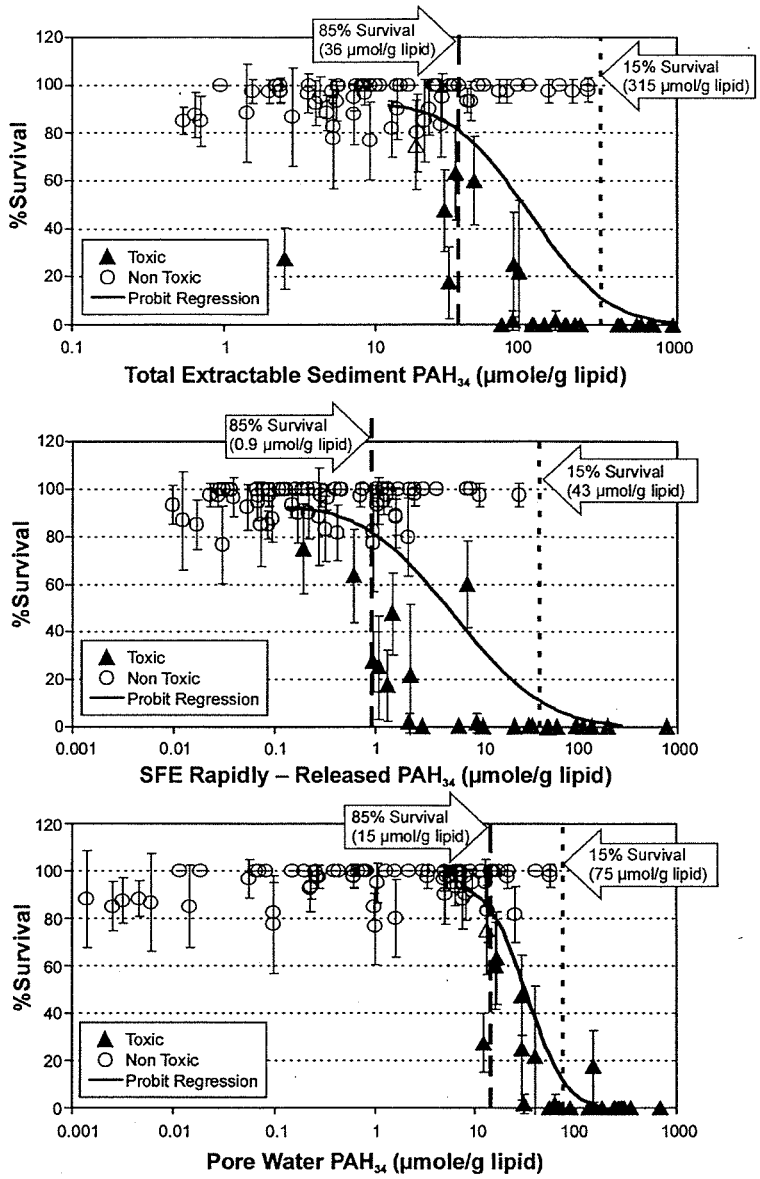

FIGURE 2. ***H. azteca*** **survival (28 day) as compared to predicted micromol per gram of lipid for 97 sediments based on total extractable sediment, SFE rapidly released, and pore water PAH_{34} concentrations.**

than the value used in the model (*2*, *5*). Thus, the essential assumption in the equilibrium partitioning model that K_{OC} values for a single PAH are constant for all sediments is not supported by experimental data on field sediments (*2*, *7*).

Survival Predictions Based on SFE Rapidly Released PAH Concentrations. The next approach we investigated was to replace the total Soxhlet extractable concentrations for each PAH by the concentration that was measured to be rapidly released by mild SFE, then predicting the lipid PAH concentrations in the same manner as that used for the total extractable concentrations using equilibrium partitioning modeling based on literature K_{OC} and K_{OW} values (*3*–*6*). As shown in Figure 2 (middle), replacing the total extractable PAH concentrations with rapidly available PAH concentrations does not appear to significantly differentiate toxic and nontoxic sediment samples. The Goodman–Kruskal γ increases modestly from 0.78 to 0.80, and the model sensitivity (correctly identifying toxic samples) increases from 80 to 92%. However, in gaining sensitivity, the model specificity (correctly identifying nontoxic samples) decreased significantly to 64%, with an even greater number of samples (forty) within the uncertain region of 85–15% survival, forcing the overall model efficiency to 71% (Table 2).

Survival Predictions Based on Pore Water PAH Concentrations. Finally, the predicted biota lipid PAH concentrations were determined based on measured pore water concentrations for PAH_{34}. The pore water approach has the obvious advantage that only K_{OW} values for each PAH need to be accurate (and apply to all water samples) (*3*–*6*), which is certainly more valid than the assumption that K_{OC} values are correct and apply to all sediment samples (*2*, *7*–*12*). As shown in Figure 2 (bottom), the use of pore water concentrations and lipid–water partitioning coefficients based on K_{OW} values significantly improves the prediction of toxic and nontoxic sediments over the other two approaches discussed previously. The Goodman–Kruskal γ improves dramatically, to 0.95. In addition, the model sensitivity (correctly predicted toxicity) reaches 92%, and the specificity (correctly predicted

TABLE 2. Survival Predictions for *H. azteca* using Total Extractable, SFE Rapidly Released, and Pore Water PAH_{34} Concentrations from 97 Field Sediments

method	15–85% survival range (μmol/g of lipid)[a]	no. of sediments in 15–85% range	prediction efficiencies: sensitivity[b] (%)	specificity[c] (%)	overall[d] (%)	Goodman–Kruskal γ
PAH_{13} concn > 1.6 mg/kg (TEC)[e]			100	6	30	0.73
PAH_{13} concn > 22.8 mg/kg (PEC)[f]			96	32	48	0.75
PAH_{34} concn	36–315	26	80	81	80	0.78
SFE rapidly released PAH_{34}	0.9–43	40	92	64	71	0.80
pore water PAH_{34}	15–75	17	92	89	90	0.95

[a] Lower 95% confidence interval for 85% survival and upper 95% confidence interval for 15% survival. [b] Sensitivity is the extent to which a test correctly classifies a toxic sample as toxic and is therefore protective of the environment. [c] Specificity is defined as the rate at which a test correctly classifies a nontoxic sample as nontoxic. [d] Overall efficiency is the fraction of correct predictions for all samples. [e] TEC is the sum of 13 parent PAH concentrations below which toxicity is considered unlikely (*1*). [f] PEC is the sum of 13 parent PAH concentrations above which toxicity is considered likely (*1*).

nontoxic) reaches 89%, yielding an overall model efficiency of 90% (Table 2).

Since the data presented in Figure 2 come from sediments from eight different MGP and aluminum smelter sites, and since they encompass such a large range of carbon concentrations and types (as well as PAH concentrations), it would seem likely that specific sites or sediment characteristics may strongly relate to the toxicity and/or ability of the models to predict toxicity. However, there is no apparent relationship between toxicity and model behavior among the different sites or between MGP and aluminum sites (Supporting Information Figure S1). In addition, there appears to be no relationship between total TOC (Supporting Information Figure S2), BC (Supporting Information Figure S3), the fraction of BC as compared to TOC (Supporting Information Figure S4), or sediment texture (not shown) on the predictive abilities of the models based on total extractable PAH, SFE available PAH, or porewater PAH concentrations.

As shown in Table 2 and Figure 2, Probit regression analysis shows that the number of sediment samples within the 95% confidence interval for 15–85% survival was significantly fewer for the pore water predictions than for either SFE rapidly released or total extractable concentrations. With the pore water data, only two toxic sediments were incorrectly predicted to have less than 85% survival (i.e., had a predicted μmol/g of lipid PAH concentration below the 85% survivability line). However, it should be noted that the toxicity for the sediment with ca. 30% survival (Figure 2, bottom) is unlikely to be caused by PAHs since the total sediment concentrations were only 2.4 μg/g (PAH_{16}) and 6.4 μg/g (PAH_{34}). In fact, only three sediments out of the 97 shown in Figure 2 had lower total extractable PAH concentrations. This sediment also had only 0.2 wt % natural organic carbon (0.8 wt % TOC minus 0.6 wt % SOC) and was composed primarily of sand. Recent studies with uncontaminated sandy sediments show that the lack of essential nutrients may cause mortality, even with the addition of food as per EPA method 100.4 (Francis Doherty, personal communication). Unidentified toxic agents may have also been present. However, none could be found by full-scan GC-MS analysis of the pore water and sediment extracts. The other sample defined as toxic (by failing the 85% survival criteria) but predicted as nontoxic had a survival of 75 ± 19% (Figure 2, bottom), which is very close to the conservative 85% survival value used in our study as the cutoff for nontoxic sediments.

Ideally, any method used to predict biological effects of pollutants would be conservative (i.e., tend to overpredict rather than underpredict toxicity). The pore water predictions of toxicity had a sensitivity of 92%, classifying only two sediments (just discussed previously) out of 25 toxic sediment samples as nontoxic (Table 2). The pore water predictions also had fewer sediments in the uncertain region between 85 and 15% survival as compared to the rapidly released and total extractable PAH_{34} approaches (Table 2). These results from Probit regressions show that (to a 95% confidence level) sediments falling outside the 85–15% survival range would not need to be tested during field surveys but could be directly classified as toxic or nontoxic based on pore water PAH concentrations. The sediments falling between the predicted 85 and 15% survival range would then require toxicity testing. Thus, for the 97 sediments, 26 sediments would require biological testing based on total extractable sediment PAH_{34} concentrations, 40 sediments for rapidly available sediment concentrations, while only 17 sediments would need biological testing for the predictions based on pore water concentrations (Table 2).

The relatively poor ability of rapidly released PAH concentrations to predict toxicity was a surprise since our initial report comparing pore water and SFE predictions showed both methods to improve toxicity predictions over using total extractable concentrations and the equilibrium partitioning model (*18*). All of the sediments studied in the initial report were from MGP sites, while the present study includes sites near aluminum smelters that had higher relative proportions of high molecular weight PAHs (likely associated with coal tar pitch used for anode production) than the MGP sites (likely contaminated with MGP tars having a lower molecular weight distribution). There are no apparent differences in toxicity (or the ability to predict toxicity) between MGP and aluminum sites as shown in Figure S1.

The poorer predictive ability of mild SFE is likely a result of the types of available PAH molecules measured by the two techniques. Pore water PAH measurements determine the equilibrium (or near equilibrium) pore water PAH concentrations found in the sediment–water slurry. In contrast, SFE measures the capacity of the sediment to rapidly release PAHs to water (*19*). Since *H. azteca* presumably absorbs solvated PAHs from the water phase, it would seem logical that pore water concentrations may more closely reflect exposure in the toxicity tests. It should also be noted that sediment heterogeneity (discussed previously) also likely reduces the predictive abilities of both total extractable and rapidly released PAH concentrations, as compared to the good homogeneity shown by pore water PAH concentrations. In any case, the results in Figure 2 and Table 2 clearly demonstrate that pore water concentrations are superior to either total extractable PAH_{34} or SFE rapidly available PAH_{34} concentrations in predicting *H. azteca* mortality.

Since the two methods measure different phenomena (capacity to rapidly release PAHs vs equilibrium pore water concentrations), it was hoped that a combination of the two approaches would further increase the ability to predict

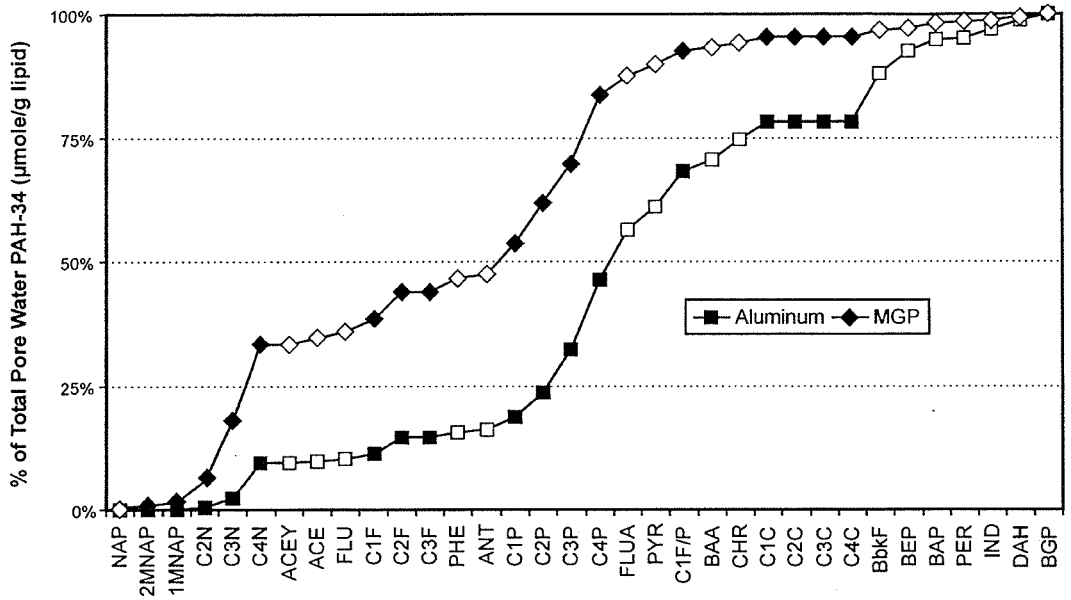

FIGURE 3. Cumulative average relative contribution to lipid PAH burden of each of the 34 PAHs in MGP and aluminum smelter sediment pore water samples. Alkyl PAHs are designated with a solid symbol and parent PAHs with an open symbol.

toxicity. As would be expected, the molar PAH lipid concentrations predicted by SFE and pore water measurement concentrations are correlated, but not strongly, and a linear regression of the log SFE µmol/g of lipid versus log pore water µmol/g of lipid only has an r^2 value of 0.55. A binary logistic regression model using the molar PAH lipid concentrations predicted by pore water as the predictor and toxic/nontoxic as the response was statistically significant ($p < 0.0001$) and explained approximately 95% of the variance in toxicity (Goodman–Kruskal $\gamma = 0.95$). Unfortunately, adding the molar PAH lipid concentrations predicted by SFE as an additional predictor was not statistically significant ($p = 0.657$), nor did it change the Goodman–Kruskal γ, indicating that adding SFE to the pore water measurements did not improve the predictions of toxicity. Thus, based on the results from the 97 sediments, there was no added value in obtaining the SFE rapidly released PAH_{34} concentrations for the prediction of *H. azteca* toxicity in freshwater sediments.

Effect of Nonaqueous-Phase Hydrocarbon Liquids (NAPL). As might be expected for sediments collected in industrial waterways, approximately one-third of the samples had a sheen or NAPL phase observed during sample collection, which was later confirmed by independent observation at the analytical laboratory. Any significant amount of a NAPL hydrocarbon phase (whether PAHs or other hydrocarbons such as petroleum alkanes) could change the mechanism from sediment–water partitioning to liquid–liquid partitioning. Since the equilibrium partitioning model assumes that PAH partitioning occurs between natural organic carbon on the sediment and pore water, the presence of the NAPL phase in one-third of our test sediments may contribute to the model's inability to predict pore water concentrations and thus reduce its ability to predict toxicity.

Surprisingly, the presence or absence of NAPL did not have a significant relationship to toxicity or on the predicted µmol/g of lipid PAH concentrations, as shown in Supporting Information Figure S5. Out of the 97 sediments tested, 27 had NAPL phases, of which 17 were toxic and 10 were nontoxic. Note also that removing the NAPL containing sediments from the sample set did not improve the prediction of toxicity based on the total Soxhlet extractable PAH_{34} concentrations or the rapidly available PAH_{34} concentrations.

Field versus Laboratory Determination of Threshold Toxicity Concentration. As shown in Figure 2, the sediment samples used in this study had a fairly broad distribution of nontoxic, moderately toxic, and toxic samples that allows a lethal residue (LR_{50}) to be calculated using Probit regression modeling. For the field sediments used in this study, the LR_{50} lipid concentration (total PAH_{34} molar concentration) was 33.0 µmol/g of lipid (31–35 µmol/g of lipid, 95% CI). This value is in good agreement with a laboratory-determined value recently reported by Schuler et al. (*26*) for fluoranthene with 28 day exposures to *H. azteca* of 32 µmol/g of lipid (26–40 µmol/g of lipid, 95% CI). This good agreement from controlled laboratory exposure and from 97 field sediments validates the EPA's narcosis model with pore water concentrations for describing PAH toxicity to sensitive benthic organisms such as *H. azteca*.

Relative Contributions of Alkyl and Parent PAHs to Toxicity Predictions. Historically, only 16 (or 13) parent PAHs determined by EPA method 8270 are considered in regulatory processes, as well as in the majority of scientific studies reported in the literature. This is potentially misleading since the majority of PAHs found in the environment are likely to be alkylated rather than parent PAHs. For example, a recent report showed that ca. 99% of the PAHs in a petroleum crude oil was alkylated and that ca. 60–70% of the PAHs in MGP coal tars was alkylated (*23*). In recognition of the potential importance of alkylated PAHs, the EPA's PAH hydrocarbon narcosis model suggests that a total of 18 prominent parent PAHs and 16 groups of alkyl PAHs be measured as was done in the present study (*5*). (Note that, with the exception of the two methylnaphthalene isomers, each group of alkyl PAHs can contain a few to nearly 100 isomers, so that this list of 16 groups of alkyl PAHs represents several hundreds of PAHs (*23*).) The potential impact of the alkyl PAHs on the predicted toxicity is potentially quite large since their modeled K_{OW} values are significantly larger than the related PAH (*3–5*), and therefore, the concentration of a particular group of alkyl PAHs can be much lower to account for one predicted toxic unit than the concentration required for the related

parent PAH. For example, the pore water concentrations required for one toxic unit in the EPA model (equivalent to a concentration of 2.24 μmol/g of lipid) for naphthalene, and its C1, C2, C3, and C4 isomers, were 194, 82, 30, 11, and 4 ng/mL, respectively (*5*, *22*). Therefore, C4 naphthalenes contribute nearly 50-fold higher toxic units than the same concentration of naphthalene in pore water. In contrast, the concentrations of sediment PAHs that account for one toxic unit only vary by a factor of 2, essentially since the partitioning coefficients (K_{OC} and K_{OW}) used in the model tend to cancel each other's effect on the predicted lipid concentration (*5*).

Figure 3 shows the average relative contributions of the different PAHs and groups of alkyl PAHs in pore water to predicted bioaccumulation and related PAH narcosis for all non-background sediments (i.e., sediments that had the majority of PAHs as nondetected are not included). Two features are important to note in these data. First, an average of 96% (MGP) and 78% (aluminum) of the predicted toxicity is contributed by 2–4 four-ring PAHs (e.g., naphthalene through chrysene), and the measurement of 5- and 6-ring PAHs in pore water does little to change the predicted lipid molar PAH concentration. This is fortunate since the measurement of 5- and 6-ring PAHs at the required picogram per milliliter detection limits is difficult (*22*). The results in Figure 3 also show that, since only the higher molecular weight PAHs have significant partitioning into dissolved organic matter (DOM) (*22*), there is no significant difference in the predicted lipid PAH concentrations (or the EPA toxic units (*5*)), whether freely dissolved (PAHs associated only with the pore water phase) or total dissolved (freely dissolved PAHs and PAHs associated with DOM). For the 97 sediments used in the present study, predicting the lipid PAH concentrations using freely dissolved or total dissolved PAH concentrations did not yield significantly different results in model interpretations. Therefore, the distinction of freely dissolved and total dissolved PAH concentrations for predicting PAH toxicity does not appear to be important as long as PAHs associated with colloids are removed by flocculation prior to analysis of the pore water (*22*).

The second observation from Figure 3 is that alkyl PAHs contribute an average of 81% (MGP) and 55% (aluminum) of the total predicted lipid molar PAH concentrations (and therefore 81 and 55% of the predicted toxicity, respectively). For the combined sites, 69% of the total toxicity was caused by the C1 to C4 alkyl naphthalene and phenanthrene/anthracene isomers. The importance of the alkyl PAHs to toxicity clearly demonstrates the need to apply methods capable of measuring both parent and alkyl PAHs (*22*, *23*) rather than relying on methods that only determine parent PAHs.

The results of this study involving 97 field sediments clearly demonstrate that the measurement of pore water PAHs is superior to the use of total extractable or SFE rapidly available PAH_{34} concentrations to accurately predict the bioavailability and toxicity of PAHs from background and impacted sediments using the EPA's hydrocarbon narcosis model. Considering sediment matrix characteristics such as TOC, BC, texture, and presence of NAPL did not appear to improve predictions based on total sediment PAH concentrations. In addition, it is clear that the common practice of measuring only the parent PAHs is not sufficient to describe environmental effects and that the high contribution of alkyl PAHs to the predicted sediment toxicity emphasizes the need for consistent and accurate methods to calibrate for, and quantitate, the complex clusters of isomers that make up most alkylated groups on the PAH_{34} list.

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Supporting Information Available

Five additional figures and a Table. This material is available free of charge via the Internet at http://pubs.acs.org.

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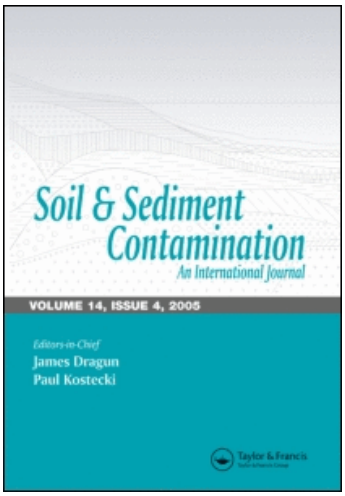

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Predicting Sediment Toxicity at Former Manufactured Gas Plants Using Equilibrium Partitioning Benchmarks for PAH Mixtures

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Predicting Sediment Toxicity at Former Manufactured Gas Plants Using Equilibrium Partitioning Benchmarks for PAH Mixtures

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This study was conducted to examine the application of Equilibrium Partitioning Sediment Benchmarks (ESBs) for assessing the toxicity of polycyclic aromatic hydrocarbons (PAHs) in sediments at former manufactured gas plant (MGP) and coke sites. Samples of freshwater sediment from four MGP and coke sites in the U.S. Northeast and Midwest were analyzed for 34 individual PAHs, total organic carbon, "black" carbon (potentially composed of pitch, soot, and other forms of pyrogenic carbon), and sediment toxicity (28-day Hyalella azteca *toxicity test). The sum of the Toxic Units in each sample was calculated from a one-phase model that accounts for sorption of PAHs to total sediment organic carbon, and a two-phase model that accounts for sorption to black carbon as well as to natural organic carbon. Although both the one-phase and two-phase models accurately predicted concentrations of PAHs that were not toxic to aquatic invertebrates, the two-phase model was more often in agreement with results of sediment toxicity tests. While the bioavailability and toxicity of PAHs may vary at other sites, the two-phase model correctly predicted that sediments from these sites with concentrations of total PAHs as high as 52 mg/kg were not toxic to invertebrates.*

Keywords Black carbon, equilibrium partitioning, MGP, PAHs, sediment

1. Introduction

A manufactured gas plant (MGP) is an industrial facility at which gas for lighting and other purposes was produced from coal, oil, and other feedstock. Sometimes, coke was the primary product, and the gas was a by-product, and the facility was called a coke plant. From the early 1800s, manufactured gas plants (MGPs) produced byproducts that included commodities such as coal tars containing high concentrations of polycyclic aromatic hydrocarbons (PAHs) (Hayes et al., 1996). Because MGPs were often located close to water bodies, contamination of sediment with PAHs is common at these sites. The distribution of PAHs in the aquatic environment is of concern because of their toxicity, carcinogenicity, and persistence (Neff, 1979). Many state regulatory authorities use empirical Sediment Quality Guidelines (SQGs), such as the Effects Range Low and Median (Long and Morgan, 1990; Long et al., 1995), Ontario Lowest and Severe Effect Levels (Persaud et al., 1993),

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and Consensus Threshold and Probable Effect Concentrations (MacDonald et al., 2000), for predicting toxicity or as remediation goals for sediments. However, one of the greatest sources of uncertainty associated with use of empirical SQGs is that they do not take into account site-specific bioavailability of sediment-associated contaminants. The National Research Council states that sediment cleanup goals should be based on site-specific risk considerations (NRC, 2001), and information on the bioavailability of PAHs is an important site-specific consideration that can be used to guide remedial strategies and focus clean-up actions at MGP sites.

U.S. EPA recently published Equilibrium Partitioning Sediment Benchmarks (ESBs) for PAH mixtures (U.S. EPA, 2003) and is developing a site-specific procedure (U.S. EPA, 2000) that can be used to estimate the bioavailability and toxicity of sediment-associated PAHs to benthic (bottom-dwelling) aquatic organisms. To develop the ESBs, U.S. EPA used a narcosis mode of action model incorporating water-only acute toxicity test data to estimate a lipid-normalized final chronic value (FCV) of 2.24 μmol PAH/g lipid, which is expected to be protective of 95% of the species tested (U.S. EPA, 2003; Di Toro and McGrath, 2000; Di Toro et al., 2000). Under the assumptions of equilibrium partitioning (EqP), the FCV is used to estimate the corresponding critical concentrations of individual PAHs in other phases (e.g., sediment organic carbon and pore water). The ESB approach calculates a Toxic Unit for each PAH as either:

$$\mathrm{TU} = \frac{C_{\mathrm{oci}}}{\mathrm{FCV_i}}$$

or

$$\mathrm{TU} = \frac{\mathrm{C_{pw}}}{\mathrm{FCV_i}}$$

where:

$\mathrm{C_{oci}}$ = the concentration of a specific PAH in sediment (ug/kg organic carbon)

$\mathrm{C_{pw}}$ = the concentration of a specific PAH in pore water (ug/L pore water)

$\mathrm{FCV_i}$ = the PAH-specific final chronic value (expressed either as ug/kg organic carbon or ug/L pore water).

If the sum of the Toxic Units for "total PAHs" in the sediment or pore water is less than or equal to 1.0, the concentration of the mixture of PAHs in the sediment is acceptable for the protection of benthic organisms from chronic effects. Previous studies have demonstrated that ESBs are useful in identifying concentrations of PAHs in sediment samples that are not likely to be toxic to invertebrates (Kane Driscoll et al., 2004; Kreitinger et al., 2007).

U.S. EPA (U.S. EPA, 2003) defines "total PAHs" as comprising, at a minimum, the 34 parent and alkylated PAHs that were measured in the U.S. EPA Environmental Monitoring and Assessment Program (U.S. EPA, 1996). This definition is used because few databases are available that have measured a greater number of PAHs, and because the use of fewer PAHs could underestimate the total toxicity of the PAH mixture. However, U.S. EPA recognizes most historical data consist of either the 13 PAHs identified by U.S. EPA as among the priority pollutants, or the 23 PAHs typically monitored by the National Oceanic and Atmospheric Administration (NOAA) for the Status and Trends Program. If data on only 13 PAHs are available, U.S. EPA (2003) states that an uncertainty factor (UF) can be applied to the sum of Toxic Units for 13 PAHs to estimate the sum of Toxic Units for 34 PAHs. U.S. EPA used a data set of 488 sediment samples to calculate a UF of 2.75 (the 50th percentile of the data set) or 11.5 (the 95th percentile of the data set) to account for the presence of the unmeasured PAHs. The data used by U.S. EPA to derive the UF

were primarily from sites contaminated with pyrogenic PAHs although a subset of samples contained more petrogenic PAHs (USEPA, 2003). Pyrogenic PAHs, which are formed under high temperatures and elevated concentrations of oxygen, are dominated by parent non-alkylated molecules. Conversely, petrogenic PAHs, which are formed under relatively low temperature and low oxygen conditions over thousands of years, are typically highly alkylated. Uncertainty Factors calculated for sites affected by petrogenic sources will tend to be larger than sites affected primarily by pyrogenic PAHs. Because of the fundamental differences between petrogenic PAHs and pyrogenic PAHs, using the uncertainty factors from the ESB (USEPA, 2003) for every site is not advisable, and the development of a source-specific UF is recommended.

U.S. EPA recognizes that a comparison of the EqP-based concentration of PAHs in bulk sediment (or pore water) to FCVs, as described above, may be overprotective at some sites if the characteristics of the sediment or of the PAHs reduce the partitioning of PAHs into pore water, thereby reducing their bioavailability and toxicity. For example, numerous studies have demonstrated that partitioning of PAHs cannot always be explained by standard models of equilibrium partitioning to sediment organic carbon (McGroddy and Farrington, 1995; Maruya et al., 1996; Ghosh et al., 2000). Additional studies suggest PAHs occlude in or adsorb to forms of pyrogenic carbon partition differently than in natural sediment organic carbon (Gustafsson et al., 1997; Buchelli and Gustafsson, 2000; Accardi-Dey and Gschwend, 2002). The presence of pyrogenic carbonaceous particles in sediment (collectively termed "black carbon") have been shown to reduce the bioavailability and bioaccumulation of PAHs in sediment by benthic invertebrates (Vinturella et al., 2004; Rust et al., 2004), which is expected to result in reduced toxicity. The draft U.S. EPA site specific procedure (2000) assumes that the bioavailable concentration in sediment can be reasonably measured or estimated from the "freely dissolved" chemical in pore water. An approach that is based on concentrations of bioavailable PAHs in pore water has the potential to help MGP site managers negotiate cleanup values that are appropriately based on actual bioavailability and toxicity. We use data from several MGP sites to examine the ability of the ESB site-specific procedure to assess the bioavailability and predict the chronic toxicity of sediment-associated PAHs.

The primary goal of this study is to examine whether the ESB approach can be used as a conservative predictor of toxicity (or lack of toxicity) of sediment-associated PAHs at MGP sites. A second goal is to examine whether a one-phase model that accounts for sorption of PAHs to total natural sediment organic carbon, or a two-phase model that accounts for sorption to black carbon as well as to natural organic carbon, is a better predictor of the bioavailability and toxicity of PAHs in sediments from MGP or coke sites. A third goal is to calculate an MGP-specific UF that can be used to predict toxicity associated with the specific suite of PAHs likely to be present at MGP sites if data are available for only a subset of PAHs.

2. Materials and Methods

2.1. Field Collection

Freshwater sites used in this study include a former coking plant in New Jersey, two MGP sites in central New York, and an MGP site in northern Indiana, USA. Samples of sediment were collected from 22 locations across these sites with concentrations of PAHs ranging from approximately 10 mg/kg to several thousand mg/kg total PAHs. Composite surficial

sediment samples (0–15 cm) were made from approximately six individual grab samples at each sample location. The composite sample was mixed in a large stainless-steel bowl, and subsamples taken for analysis of sediment chemistry, sediment characteristics, and toxicity. Field duplicates for sediment chemistry were collected as aliquots of the composite.

2.2. Sediment Analyses

Sediment samples collected at the New York and New Jersey sites (SD-1 to SD-4, K-1, P-1 to P-5) were analyzed for 34 PAHs using U.S. EPA SW846 Method 8270C, with the modification of the GC/MS in the selected ion monitoring (SIM) mode of operation. The sediment samples from the Indiana site (identified as SMR-1 to –12) were analyzed for 34 PAHs (parent and alkylated) by GC/FID using U.S. EPA Method 8100M, GC/MS using U.S. EPA Method 8270M, and GC/IRMS (U.S. EPA, 1986). All samples were analyzed for total organic carbon (TOC) using U.S. EPA SW846 Method 9060 (U.S. EPA, 1986). Samples were also analyzed for "black carbon," which is procedurally defined as carbon remaining after high-temperature combustion (375°C) and acid treatment of sediments to remove other forms of carbon (Gustaffson et al., 1997; Accardi-Dey and Gschwend, 2003). Total organic carbon and black carbon were also analyzed in a standard reference material, SRM-1944 (NIST 1999), which is an urban marine sediment.

2.3. Sediment Toxicity Tests

A 28-day sediment toxicity test with the freshwater amphipod, *Hyalella azteca,* was conducted on each sediment sample. Endpoints examined included survival and growth. This test was selected because its results are reported to be less variable than those obtained with other standard tests (Ingersoll et al., 2000). If the data met assumptions of normality and homogeneity of variance, then a parametric two-sample, equal-variance t-test was used to compare the sample response to the laboratory control response. If the assumptions of normality or homogeneity were not met, the non-parametric Wilcoxon Rank Sum test was used. Results for the site samples were compared to results for a laboratory control sediment, as well as to results for sediments collected from local reference sites.

2.4. Calculation of Toxic Units

As described in the Introduction, toxic units for individual PAHs were calculated as the concentration of bioavailable PAH in pore water divided by the corresponding FCV for that PAH. The one-phase model estimates the pore-water concentration from the sediment concentration of PAHs and the fraction of total organic carbon (f_{TOC}) in sediment, using an organic carbon normalized partition coefficient (Koc) that describes the partitioning from sediment organic carbon in sediment to pore water (US EPA, 2003). The two-phase model uses an additional term to account for the partitioning of PAHs from black carbon in sediment to pore water.

2.4.1. One-phase model for calculating the sum of toxic units. The one-phase model is used to estimate the freely dissolved concentration of each PAH in pore water using the following relationship:

$$C_{SED}/C_W = f_{TOC} * Koc$$

where:

C_{SED} = the concentration of each PAH in sediment(μg/kg dry wt)

C_W = the concentration of truly dissolved PAH in pore water(μg/L)

f_{TOC} is the weight fraction of total organic carbon in sediment (kg organic carbon/ kg dry wt)
K_{OC} is the organic carbon-water partition coefficient for a specific PAH (L/kg-OC)
The equation is rearranged and used to solve for C_W. C_W for each PAH is divided by its corresponding FCV to calculate Toxic Units. If the sum of the Toxic Units for 34 PAHs is less than or equal to 1.0, the concentration of the mixture of PAHs in sediment is predicted to be acceptable for the protection of benthic organisms from chronic effects.

2.4.2. Two-phase model for calculating the sum of toxic units. The two-phase model is used to estimate the freely dissolved concentration of each PAH in pore water using the following relationship:

$$C_{SED}/C_W = f_{NPOC} * K_{OC} + f_{BC} * K_{BC} C_W^{n-1}$$

where:

f_{NPOC} = the weight fraction of non-pyrogenic organic carbon in sediment (kg non-pyrogenic organic carbon/kg dry wt, calculated from the difference between total organic carbon and black carbon)

f_{BC} = the weight fraction of black carbon in sediment (kg black carbon/kg dry wt)

K_{BC} = the black-carbon-to-pore-water partition coefficient for each PAH (L/kg-BC)

n = the Freundlich exponent, which accounts for nonlinear sorption behavior of black carbon (n=0.6) (Accardi-Dey and Gschwend 2002)

We use an iterative approach to solve for C_W. C_W for each PAH is divided by its corresponding FCV to calculate a Toxic Unit.

Black carbon distribution coefficients, K_{BC}, were not available for all 34 PAHs; therefore, a regression relationship was used to estimate these values for the other PAHs based on data presented in Table 1. The regression relationship between K_{BC} values and K_{ow} for 17 PAHs that were determined experimentally (Accardi-Dey and Gschwend, 2003) is presented in Figure 1, and the estimated K_{BC} values are presented in Table 1. While this is an approximation, our study is designed to determine whether this relatively simple approach can be used to reduce the uncertainty associated with predictions of the potential toxicity of mixtures of PAHs in sediment.

2.5. Calculation of an MGP-Specific Uncertainty Factor

Data collected for this study are used to develop an MGP-specific UF that is likely to be a more accurate predictor of chronic toxicity to benthic invertebrates than the U.S. EPA UF (U.S. EPA, 2003). The ratio of the sum of Toxic Units for 34 PAHs to that of the sum of Toxic Units for 13 PAHs was calculated for each of the 22 sediment samples collected for this study.

Table 1
Partition coefficients and Final Chronic Values (FCV) for 34-PAHs from U.S. EPA (2003), except for log K_{BC}, which are estimated from the relationship presented in Figure 1

Alkylated PAH	Limit of Water Water Solubility (mg/L)	Molecular Weight	Log K_{OW}	Log K_{OC}	Log K_{BC}	FCV (ug/L)
Naphthalene	3.10E+01	128.19	3.36	3.30	5.10	193.5
C1-Naphthalenes		142.20	3.80	3.74	5.24	81.69
C2-Naphthalenes		156.23	4.30	4.23	5.48	30.24
C3-Naphthalenes		170.25	4.80	4.72	5.75	11.10
C4-Naphthalenes		184.28	5.30	5.21	6.02	4.048
Acenaphthylene	1.63E+01	152.20	3.22	3.17	4.80	306.9
Acenaphthene	3.80E+00	154.21	4.01	3.94	5.40	55.85
Fluorene	1.90E+00	166.20	4.21	4.14	6.00	39.30
C1-Fluorenes		180.25	4.72	4.64	5.70	13.99
C2-Fluorenes		194.27	5.20	5.11	5.98	5.305
C3-Fluorenes		208.30	5.70	5.60	6.24	1.916
Phenanthrene	1.10E+00	178.20	4.57	4.49	6.30	19.13
Anthracene	4.50E−02	178.20	4.53	4.46	6.10	20.73
C1-Phenanthrenes/ Anthracenes		192.26	5.04	4.96	5.88	7.436
C2-Phenanthrenes/ Anthracenes		206.29	5.46	5.37	6.15	3.199
C3-Phenanthrenes/ Anthracenes		220.32	5.92	5.82	6.38	1.256
C4-Phenanthrenes/ Anthracenes		234.23	6.32	6.21	6.63	0.5594
Fluoranthene	2.40E−01	202.26	5.08	5.00	6.70	7.109
Pyrene	1.32E−01	202.26	4.92	4.84	6.20	10.11
C1-Fluoranthenes/ Pyrenes		216.29	5.29	5.20	6.09	4.887
Benz[a]anthracene	1.10E−02	228.29	5.67	5.58	6.90	2.227
Chrysene	2.00E−03	228.29	5.71	5.62	6.80	2.042
C1-Chrysenes		242.32	6.14	6.04	6.52	0.8557
C2-Chrysenes		256.23	6.43	6.32	6.75	0.4827
C3-Chrysenes		270.36	6.94	6.82	6.91	0.1675
C4-Chrysenes		284.38	7.36	7.24	7.19	0.0706
Benzo[b]fluoranthene	1.50E−03	252.32	6.27	6.16	6.00	0.6774
Benzo[k]fluoranthene	8.00E−04	252.32	6.29	6.18	6.80	0.6415
Benzo[a]pyrene	3.81E−03	252.31	6.11	6.00	6.90	0.9573
Perylene	4.01E−04	252.31	6.14	6.03	6.73	0.9008
Benzo[e]pyrene	4.01E−03	252.32	6.14	6.03	6.90	0.9008
Indeno[1,2,3-cd]pyrene		276.23	6.72	6.61	6.75	0.2750
Dibenz[a,h]anthracene	6.01E−04	276.23	6.71	6.60	6.80	0.2825
Benzo[g,h,i]perylene	2.60E−04	278.35	6.51	6.40	7.06	0.4391

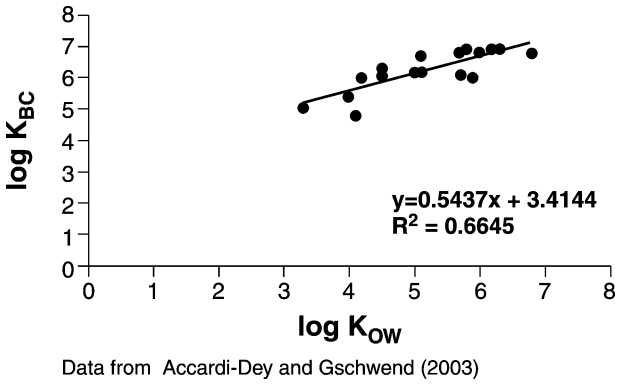

Figure 1. Relationship of log K_{ow} to log K_{BC}.

3. Results and Discussion

3.1. Sediment Chemistry

Concentrations of total PAHs in sediments ranged from 9 mg/kg to greater than 5,000 mg/kg (Table 2). Concentrations of total organic carbon ranged from 0.7 to 6.8% of sediment dry weight. Concentrations of black carbon ranged from 0.06% to 2.8% of sediment dry weight, and from 3% to 81% as a percent of TOC. Results reported for the standard reference material, SRM-1944 (0.0059 g black C/g dry wt) agree reasonably well with values reported by other researchers for this reference material: 0.0066 and 0.0088 g black C/g dry wt, (Gustaffson et al., 2001).

3.2. Sediment Toxicity

Four sediment samples, with total PAH concentrations of 60, 1,730, 3,363, and 5,160 mg/kg showed significantly reduced survival of *H. azteca* in comparison to 91% survival observed in laboratory controls, while samples with concentrations as high as 325 mg/kg were not significantly toxic in comparison to controls (Table 2). None of the other samples had significantly reduced survival, and none of the samples had significantly reduced growth (Table 2).

The concentration of PAHs in one toxic sample (60 mg/kg, 9% survival) was considerably lower than in other toxic samples that exhibited reduced survival (1,730 to 5,160 mg/kg). Field notes state that sheens were observed when this sample was collected. A review of the analytical data for this sample showed elevated levels of benzene, toluene, ethylbenzene, and xylenes (BTEX) in comparison to other samples, which may have contributed to the observed toxicity (data not shown).

3.3. Results of the One-Phase Model

The one-phase model correctly predicted a lack of toxicity for three samples with concentrations of total PAHs ranging from 9 to 21 mg/kg and a sum of Toxic Units <1.0, but incorrectly predicted (with one exception) that 16 samples with concentrations of total PAHs ranging from 18 to 325 mg/kg (Table 2, Figure 2) and a sum of Toxic Units >1.0 would be toxic. The one-phase model correctly predicted that three samples with

Table 2

Concentrations of PAHs, fraction total organic carbon (f_{TOC}), fraction black carbon (f_{BC}), corresponding Sum-Toxic Units and sediment toxicity. Results for f_{OC} and f_{BC} represent mean and standard deviation (n=2), except for samples for which a single analysis was conducted. Asterix denotes significant difference from laboratory control.

	Sediment				Sum of Toxic Units		Biotoxicity	
Sample	Total PAHs mg/Kg	f_{TOC} % (SD)	f_{BC} % (SD)	fBC as % of fOC	Sum-TU 1-phase Model	Sum-TU 2-phase model	Mean Survival %	Mean Growth % of Control
K-1	9	1.8 (0.21)	0.06(0.014)	3	0.7	0.4	99%	101%
SMR-12	18	1.6(0.21)	0.3(0.01)	19	1.6	0.2	66%	91%
SD-1	20	3.1(0.21)	0.88	28	0.9	0.05	94%	138%
P-5	21	6.8(6.0)	1.8(1.1)	26	0.7	0.02	99%	119%
SMR-11	26	1.7(0.07)	0.3(0.03)	18	2.3	0.4	91%	126%
SMR-7	26	1.1(0)	0.3(0.01)	27	3.6	0.5	71%	109%
SMR-10	33	1.7(0.14)	0.6(0)	35	2.8	0.2	81%	118%
SMR-6	36	1.5(0.14)	0.6(0.04)	40	3.7	0.3	78%	97%
SD-2	37	4.4(0.57)	2.8	64	1.2	0.03	85%	171%
SMR-9	37	2.3(0.28)	1.5(0.07)	65	2.3	0.1	70%	111%
P-4	39	3.2(1.10)	0.49(0.05)	15	1.8	0.3	98%	106%
SMR-5	40	1.6(0.14)	1.3(0)	81	3.9	0.1	86%	98%
SD-4	44	4.8(0.28)	2.2	46	1.3	0.05	79%	122%
SMR-3	52	2.5(0.14)	0.5(0.04)	20	3.0	0.5	65%	146%
SMR-1	60	2.4(0.42)	0.96(0.06)	40	4.3	0.5	9%*	72%
SMR-8	93	0.7(0.05)	0.4(0.01)	57	28	7.7	78%	120%
SD-3	257	3.8(0.07)	1.2	32	9.5	1.6	89%	141%
P-3	272	5.4(0.28)	1.6(0.14)	30	7.3	1.2	97%	110%
P-2	325	3.5(0.00)	0.67(0.13)	19	15	4.1	94%	136%
SMR-2	1730	4.5(0.35)	0.8(0.02)	18	70	44	49%*	121%
SMR-4	3363	3.0(0.14)	0.9(0.11)	20	192	132	46%*	126%
P-1	5160	6.2(0.0)	1.1(0.28)	18	134	90	64%*	175%

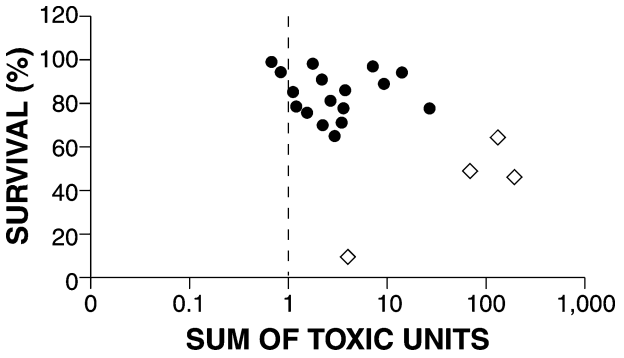

Figure 2. Sum of Toxic Units estimated using 1-phase model versus survival in sediment toxicity tests. Samples shown as diamonds had significantly reduced survival in comparison to laboratory controls.

high concentrations of PAHs in sediment (1,730 to 5,160 mg/kg) would be toxic (Table 2, Fig. 2).

3.4. Results of the Two-Phase Model

Predictions of the two-phase model were in closest agreement with the results of the sediment toxicity tests. The two-phase model correctly predicted (with one exception) that 15 samples with a sum of Toxic Units <1.0 and concentrations of PAHs in sediment ranging from 9 to 60 mg/kg (Table 2, Figure 3) would not be toxic, but incorrectly predicted that four samples with concentrations of PAHs ranging from 93 to 325 mg/kg and a sum of Toxic Units >1.0 would be toxic. The two-phase model also correctly predicted that three samples with high concentrations of PAHs in sediment (1,730 to 5,160 mg/kg) would be toxic (Table 2, Figure 3).

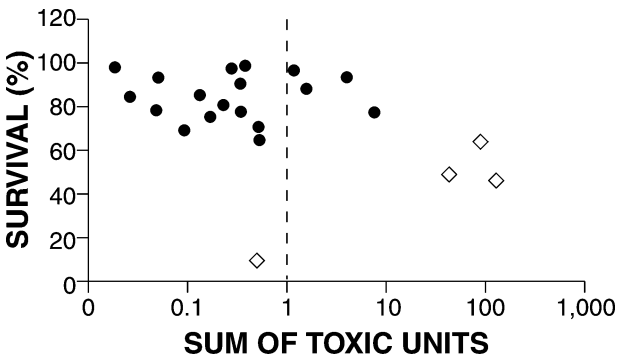

Figure 3. Sum of Toxic Units estimated using 2-phase model versus survival in sediment toxicity tests. Samples shown as diamonds had significantly reduced survival in comparison to laboratory controls.

3.5. Uncertainty Factor

We used the Excel^{TM} percentile function to calculate source-specific UF values of 1.98 (the 50th percentile of our data set) and 2.71 (the 95th percentile of our data set). The UF calculations indicate that source-specific UF for coke and MGP sites are lower than those recommended by U.S. EPA for use when source-specific data are lacking and sources are not known. The 50th percentile UF of 1.59, previously reported for four MGP site samples (Kane Driscoll et al., 2003), and the mean UF of 2.9 and 95th percentile UF of 4.2 that were calculated for 45 MGP-affected sediments from six sites (Hawthorne et al., 2006) are also lower than the U.S. EPA UF. Collectively, these studies demonstrate that application of the U.S. EPA UF is likely to overestimate the sum of Toxic Units for sediments affected by MGP and coke sites.

4. Conclusions

The present study demonstrates that, although both the one-phase and two-phase models can be used to predict concentrations of PAHs that are not toxic to aquatic invertebrates, the two-phase model is a better predictor of toxicity at these sites. Specifically, the two-phase model, which takes into account the influence of black carbon on the bioavailability of PAHs, demonstrated that sediments from these sites with concentrations of total PAHs as high as 52 mg/kg and less than 1.0 Toxic Unit are not toxic to benthic invertebrates. Results further demonstrate that the two-phase model is a conservative predictor of toxicity, because sediments estimated to have a sum of Toxic Units as high as 10 were not toxic, a result that has also been observed for other studies on MGP-affected sediments (Kreitinger et al., 2007). Thus, this study demonstrates that both models can accurately identify concentrations of PAHs in sediment at MGP sites that are not toxic, but the two-phase model is less conservative and provides more accurate predictions.

Although we recognize that estimates of black carbon include a heterogeneous mixture of materials, such as coal, coke, and coal tar pitch (Khalil et al., 2006), this study demonstrates that the simple measurement of black carbon can be used to reduce uncertainty and make conservative predictions of toxicity. This work also demonstrates that measurements of the full suite of 34 PAHs will reduce the uncertainty associated with the characterization of the source-specific suite of PAHs that are likely to be present at MGP or coke sites. In particular, the use of overly conservative UF in the calculation of the sum of Toxic Units should be avoided at MGP sites, because this and other studies demonstrate that the application of an overly conservative UF can result in the overestimation the concentration of alkylated PAHs in these sediments and their contribution to toxicity (Kreitinger et al., 2007).

Although the use of the two-phase model showed improved predictive ability in comparison to the one-phase model at these sites, uncertainties associated with the use of the two-phase model must be considered for future applications. For example, levels of black carbon were quite high in these samples (up to 81% of the total organic carbon) in comparison to "normal" sediment with black carbon levels that range from 1 to 10% of total organic carbon (Gustafsson and Gschwend, 1998). In particular, the K_{BC} values (Fig. 1) and the non-linear isotherm exponent are additional sources of uncertainty that may vary as the nature of the sediments and types of black carbon varies among sites (Jonker and Koelmans, 2001a; Ghosh et al., 2000; Cornelissen and Gustafsson, 2004; Hawthorne et al., 2007a). In addition, the potential for the presence of lighter aromatic hydrocarbons (e.g.,

BTEX) and other contaminants to confound results will need to be considered at other sites as well as sites discussed in the study.

Additional research that collects synoptic measurements of bioavailability and toxicity using various analytical techniques will most effectively demonstrate the factors controlling bioavailability of PAHs at MGP and other sites. For example, low-density polyethylene device samplers (Vinturella et al., 2004), polyoxymethylene extraction (Jonker and Koelmans, 2001b), and desorption to XAD resin (Lamoreaux and Brownawell, 1999) have been used to measure the bioavailable fraction of PAHs in sediments, and supercritical fluid extraction and solid phase microextraction (Kreitinger et al., 2007; Hawthorne et al., 2007b) have been used to demonstrate the relationship between bioavailable PAHs in sediments and toxicity to benthic invertebrates. Concordance among various lines of evidence, including bioaccumulation and toxicity tests with additional test organisms, along with validation and standardization of analytical techniques for measuring freely dissolved concentrations in pore water and black carbon in sediment, will increase confidence in the reliability of the two-phase model for use in environmental risk assessments and in the development of remedial strategies at MGP sites and other PAH-contaminated sites.

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AN ECOLOGICAL RISK-BASED CLEANUP STRATEGY FOR CONTAMINATED SEDIMENTS IN A FRESHWATER BROOK

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ABSTRACT

An ecological risk-based approach was used to define the extent of remediation in a brook adjacent to a former manufacturing and assembly plant. Sediment contained concentrations of metals, polycyclic aromatic hydrocarbons (PAHs), phthalates, polychlorinated biphenyls (PCBs), and pesticides above sediment quality benchmarks. Samples of sediment were analyzed for metals, 34 individual PAHs, phthalates, PCBs, pesticides, total organic carbon, black carbon, and sediment toxicity using the 42-day *Hyalella azteca* toxicity test. In addition, freely dissolved concentrations of PAHs in pore water from a subset of samples were determined using a solid phase microextraction (SPME) technique. Concentrations of freely dissolved PAHs in pore water and bioavailable PAHs in sediment were below levels of concern for aquatic organisms. Further evaluations indicated that lead was the contaminant most closely associated with sediment toxicity. A site-specific sediment cleanup level for lead in sediment was developed to define areas for sediment removal in the brook. Using the site-specific sediment cleanup level for lead resulted in a substantially smaller remediation footprint in the brook (24,434 ft^2; 2,270 m^2) than that originally proposed (64,799 ft^2; 6,020 m^2) based on exceedance of sediment quality benchmarks.

Key words: lead, polycyclic aromatic hydrocarbons, ecotoxicity, sediment cleanup level

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1. INTRODUCTION

An investigation was conducted to determine the extent of remediation in a brook (the Brook), located along the western property line of a former manufacturing and assembly facility (the facility) in eastern Massachusetts. The work described here is a part of the comprehensive response actions that were conducted in compliance with the Massachusetts Contingency Plan. Other components of the response actions included a comprehensive site assessment, human health risk assessment, ecological risk assessment, remedial feasibility study, and remedial action plan. The focus of this paper is on ecological risks, which need to be reduced through remediation of sediment in portions of the Brook.

The facility and the Brook are located in an urban setting with mixed industrial, commercial, and residential uses. The headwaters are surrounded by undeveloped wooded and emergent wetlands in an area immediately upstream of the facility. Approximately 3.5 miles downstream of the facility, the Brook discharges into a pond, which is located in a municipality. Historically, four sources of discharges entered the Brook within the study area: an outfall from the wetlands that discharges to the upstream reaches of the Brook near the facility, a roof drain from facility buildings, an outfall from a settling lagoon that was used to treat process and non-process wastewater, and a storm water drainage outfall from paved areas of the facility.

Habitat quality in the Brook reflects the influence of stressors that are typical in an urban setting, such as high peak flows caused by the preponderance of impervious surfaces in the drainage area, encroachment of development and subsequent riparian habitat loss, and water quality degradation resulting from urban storm water discharges. The Brook is shallow and, under normal flow conditions, ranges from 0.5 to 2.5 ft (2 ft average) in depth and from 15 to 28 ft (20 ft average) in width. These physical and hydrologic conditions are generally adequate to support benthic macroinvertebrates and seasonal populations of fish and semi-aquatic organisms, such as reptiles and amphibians. In addition, habitat features that support aquatic life, such as submerged or emergent aquatic vegetation, leaf litter, and submerged terrestrial vegetation (deadfall) are also present in the Brook at various locations.

Previous site studies indicated that sediment in the Brook contained elevated concentrations of metals, PAHs, phthalates, PCBs, and pesticides that exceeded sediment quality benchmarks (SQBs) (MacDonald et al., 2000; Long and Morgan, 1990; Jones et al., 1997). Toxicity testing conducted in November 1998 revealed that some sediment samples from the Brook were toxic to test organisms in 10-day toxicity tests using the amphipod, *Hyalella azteca,* and the midge, *Chironomous tentans* (USEPA, 2000; ASTM, 2005). No station exhibited lethal toxicity in the chironomid test, while 4 of the 12 stations displayed lethal toxicity in the amphipod test. Two stations downstream of the lagoon exhibited sublethal toxicity (reduced growth) for the chironomid test, and one of these stations also exhibited sublethal toxicity (reduced biomass) for the amphipod

test. Sublethal toxicity was not observed in the furthest downstream reach. The lack of observed toxicity in sediments from the furthest downstream station suggested that toxic effects are confined to the areas of the Brook adjacent and just downstream of the lagoon. Because state agencies currently recommend the use of long-term toxicity tests, the current study re-evaluated toxicity of the Brook sediment using a 42-day toxicity test with *H. azteca* (USEPA, 2000), with survival, growth, and reproduction endpoints.

The Sediment Quality Triad is commonly used to evaluate risk to benthic organisms from sediment-associated contaminants. This approach uses three types of measurements: concentrations of contaminants in bulk sediment, laboratory toxicity tests, and characterization of the benthic community, to reach conclusion regarding risk. Although the utility of the Sediment Quality Triad approach has been clearly demonstrated (Chapman, 1996; Krantzberg et al., 2000; Borgmann et al., 2001; Grapentine et al., 2002; Reynoldson et al., 2002a, b; Chapman, 2002), at some contaminated sites, the presence of non-contaminant factors such as nutrient enrichment, changes in dissolved oxygen or water temperature, or streambed sedimentation may mask the contaminant-related impacts on the benthic invertebrate community. In such cases, more reliance may be placed on the other two types of measurements, the bulk sediment chemistry and laboratory toxicity tests, and the benthic invertebrate community field study may not be considered. A benthic invertebrate community field study was not conducted as part of the current investigation; it was determined that this kind of investigation would not yield meaningful results because the high spatial and temporal variability associated with non-contaminant stressors discussed above are likely impacting the habitat quality of the Brook study site.

The main objective of the current study was to define the extent to which facility-related chemicals pose a risk to benthic invertebrates exposed to sediment in the Brook. Benthic invertebrates were selected as the potential receptor community in the Brook, because they are relatively immobile and would be highly exposed to contaminants in the sediment of the Brook. No fish were observed in the Brook during the Site visits and sediment sampling, probably because of low flow conditions. In addition, aquatic habitat characteristics of the Brook are not conducive to the establishment and maintenance of a permanent fish community. Because the Brook does not contain abundant prey items for wildlife, such as freshwater mussels and fish, wildlife also were not selected as receptors for this assessment. The results of the study were used to determine whether remedial actions are necessary to protect benthic invertebrates, and to assist in developing a remedial design plan that is appropriate for the Brook.

2. MATERIALS AND METHODS

2.1 Study Design

Sediment was collected from 25 stations along the length of the Brook. Sampling locations included an upstream reference location (i.e., outside of the influence of the facility); several locations immediately downstream from outfalls, which once released process and non-process wastewater but currently release runoff from parking lots adjacent to the Brook or are sealed; and six locations in the Brook but downstream of the property boundary (Figure 1). Using existing data on sediment chemistry, stations were selected to represent a broad range of concentrations of potentially facility-related chemicals.

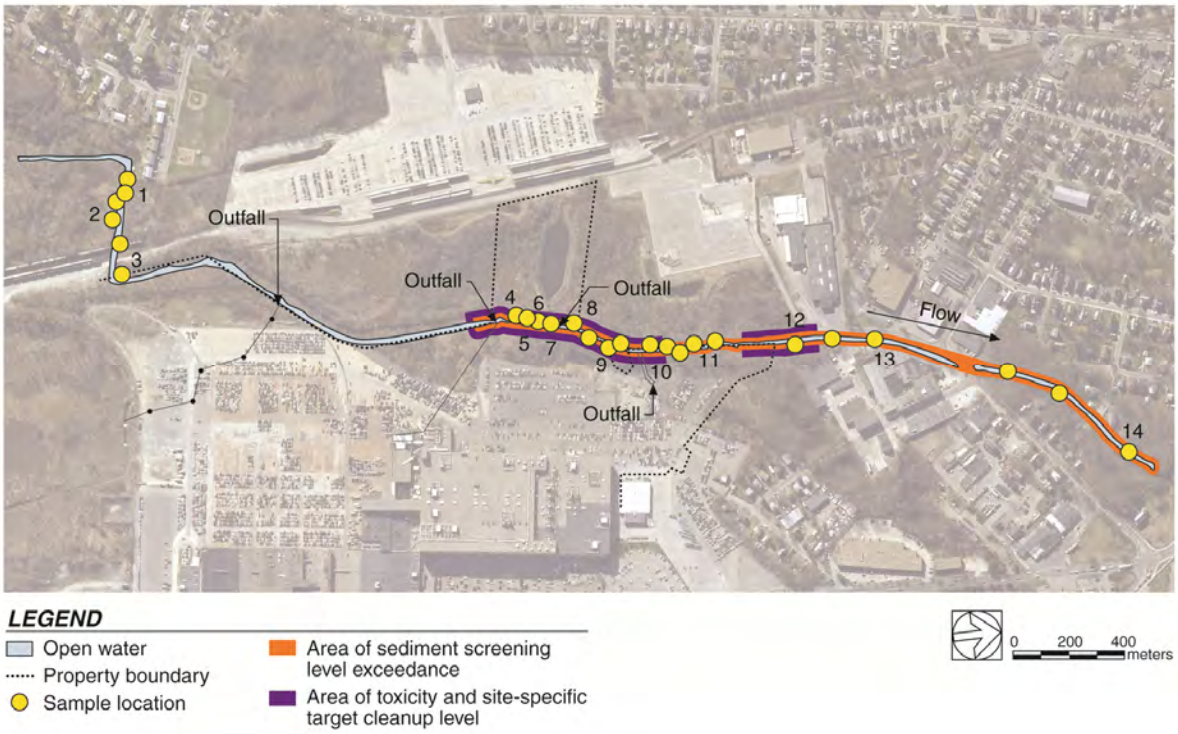

Figure 1. Sampling locations in a brook along a former manufacturing and assembly facility in eastern Massachusetts (USA). Numbered locations were submitted for the full suite of chemical analysis and sediment toxicity testing. Also shown are the two excavation footprints based on exceedance of sediment quality benchmarks and observed toxicity and exceedance of site-specific target cleanup level.

Expedited analyses of metals and semi-volatile compounds (SVOCs) were conducted to identify a subset of samples for toxicity testing and additional chemical testing. A total of 14 sediment samples, with a wide range of contaminant concentrations, were

submitted for toxicity testing. The goal was to bracket the upper and lower effects concentrations for potentially facility-related chemicals and establish quantitative relationships between exposure and effects. Samples included: three samples from the upstream reference area (sample locations 1, 2 and 3), six samples from the reach adjacent to the facility (sample locations 4, 5, 6, 7, 8 and 9), two samples from the reach downstream of the facility but within property boundaries (sample locations 10 and 11), and three samples (sample locations 12, 13, and 14) from downstream of the property boundaries (Figure 1). The selected subset of sediment samples were analyzed for target analyte list (TAL) metals, target compound list (TCL) SVOCs, TCL pesticides, PCB Aroclors®, TOC, black carbon, and sediment grain size.

A subset of five samples (sample locations 5, 6, 7, 11 and 13) was selected for analysis of the bioavailable fraction of PAHs using a laboratory-based solid-phase microextraction technique. The selected sediment samples represented typical physical sediment conditions (e.g., grain size, presence of organic material, texture, and color) and were not associated with any anomalous field observations, such as presence of a sheen or petroleum odor that might confound data interpretation.

2.2 Sediment Collection and Processing

Sediment at the 25 stations was sampled between July 23 and 25, 2007. Samples of sediment were collected using a 0.023-m^2 Eckman grab sampler (Wildlife Supply Company, Buffalo, NY, USA) or 0.0071-m^2 hand-auger sampler (Ben Meadows Company, Janesville, Wisconsin, USA). The top 15 cm of sediment, where most benthic organisms live in this freshwater system, were sampled at each station. Five to seven grabs from each station were collected and homogenized to obtain sufficient sample size for all analyses and sediment toxicity tests. Sediment samples were collected in a downstream-to-upstream fashion, to avoid disturbing the sediment prior to sampling. Samples for SPME analysis were sieved in the field through 2-mm mesh to remove debris. Sediment sample containers for chemical analysis, SPME analysis, and toxicity testing were placed on ice immediately after collection and maintained at about 4°C in coolers during transport to the receiving laboratories.

2.3 Sediment Chemical Analysis

Sediment samples from all 25 stations were analyzed within one week from the date of collection for TAL metals by EPA Methods 6020 and 7471A, and TCL SVOCs by EPA Method 8270C (http://www.epa.gov/osw/hazard/testmethods/sw846/ online/index.htm). Prior to metals analysis, two extractions were used. EPA Method 3050 without the addition of hydrochloric acid (HCl) was used for all metals, except silver and antimony. A separate extraction, EPA Method 3050 with HCl, was used for analysis of silver and antimony.

Following review of metals and SVOC data, 14 stations were selected for further chemical analysis and toxicity testing. Sediment from these 14 stations was analyzed by Alpha Analytical, Inc. (Mansfield, MA) for 34 PAHs by EPA Method 8270C (mass spectrometry in the selected ion-monitoring mode), TCL pesticides by EPA Method 8081A, PCB Aroclors® by EPA Method 8082, TOC by EPA Method 9060, black carbon by the Arccardi-Dey and Gschwend (2002) method, and grain size by Method D 422-63 (ASTM, 2007a).

2.4 Sediment Toxicity Testing

Standard sediment toxicity tests were conducted using the 42-day test with *H. azteca* (USEPA, 2000; ASTM, 2005) by Springborn-Smithers Laboratories (Wareham, MA). *H. azteca* was selected as the test organism because previous short-term toxicity testing conducted on Brook sediments suggested that *H. azteca* was more sensitive than *C. tentans* to site chemicals. Prior to test initiation, the toxicity laboratory sieved the samples through 0.5-mm mesh to remove larger indigenous organisms that could prey on the test organisms, a problem that was previously encountered in toxicity tests of sediments from this site. The tests were initiated 23 to 25 days after sample collection, within the recommended holding time of 2–8 weeks (USEPA, 2000; ASTM, 2005). The 42-day toxicity test with *H. azteca* measures the number of surviving amphipods and dry weight in milligrams after 28 days, and the number of progeny per female amphipod at day 42. Twelve replicates were evaluated for each tested sediment, with 10 amphipods in each replicate. Twelve replicates were used for the survival endpoint, four replicates were used for the growth endpoint, and eight replicates were used for the reproduction endpoint. Overlying water was renewed at a rate of two volume replacements per day. An artificial sediment prepared according to OECD Guideline NO. 219 (OECD, 2001) was used as a negative control. Potassium chloride was used as a positive control.

Overlying water quality characteristics were measured in accordance with guidance in USEPA (2000). Total hardness, alkalinity, conductivity, dissolved oxygen, pH, and total ammonia-N were measured in the overlying water from each test chamber on days 0, 28, 29, and 42 of the test. Conductivity was monitored weekly from a composite sample. Dissolved oxygen and pH were monitored three times per week in one alternating replicate throughout the course of the study. Temperature was monitored daily in one alternating replicate throughout the course of the study.

2.5 Solid Phase Microextraction and Analysis of 34 PAHs

For PAHs, various methods have been developed to estimate or measure the bioavailable fraction of PAHs in sediment, including the USEPA bioavailability procedure for measuring the bioavailable concentration in pore water (USEPA, 2003), equilibrium partitioning (EqP) methods that estimate the bioavailable fraction in pore water by accounting for association to non-pyrogenic and pyrogenic organic carbon in sediment

(Accardi-Dey and Gschwend, 2003; Gustafsson et al., 1997), supercritical fluid extraction (Hawthorne et al., 2007), and SPME (Hawthorne et al., 2005; Hawthorne et al., 2007; Kreitinger et al., 2007). Recent work has demonstrated that freely dissolved concentrations of PAHs in pore water as determined by SPME is a good predictor of sediment toxicity for PAH-contaminated sediments (Hawthorne et al., 2007; Kreitinger et al., 2007).

The SPME measurement of 34 parent and alkylated PAHs (18 parent and 16 groups of alkyl PAHs listed for EPA's equilibrium partitioning sediment benchmark (ESB) model [2003]) in pore water of sediment samples was conducted by TestAmerica (Knoxville, TN) using the ASTM Method D 7363 (ASTM, 2007b). The SPME analysis began within 28 days of sample collection. Briefly, sediment samples were centrifuged at 1,000 g for 30 min to collect pore water. Pore water samples were re-centrifuged after addition of alum to precipitate colloids. PAHs were extracted using SPME with fibers coated with poly(dimethylsiloxane). SPME sorption was performed for 30 minutes, after which the fiber was desorbed directly into the gas chromatography/mass spectrometer injection port.

2.6 Estimating Sum-TU

Recent EPA guidance (USEPA, 2003) establishes ESBs to protect benthic organisms from the narcotic effects of PAHs. To more accurately reflect the potential exposure of biota in the environment, the guidelines are applied to PAH mixtures, as opposed to individual PAHs. This guidance established final chronic values (FCVs) for individual PAHs that are expected to be protective of aquatic species. A toxic unit (TU) is the quotient of an individual PAH concentration in sediment or pore water (predicted or measured) divided by its corresponding FCV. If the sum of the TUs for 34 individual PAHs (Sum-TU) is ≤ 1.0, it can be concluded that the concentrations of PAHs in the bulk sediment sample are below the toxicity threshold for benthic organisms. Because of the environmentally conservative nature of the FCVs, a Sum-TU > 1.0 does not necessarily mean that bulk sediment PAHs will result in adverse effects to benthic organisms.

Three methods were used to calculate the Sum-TUs in the present study. The first method is referred to as a one-phase model, which estimates concentrations of PAHs in pore water from the concentrations of PAHs in bulk sediment and TOC. The second method, referred to as a two-phase model, estimates concentrations of PAHs in pore water from the concentrations of PAHs in bulk sediment, TOC, and black carbon (an additional phase that can also adsorb PAHs). The third method is the SPME method, which directly measures freely dissolved concentrations of PAHs in pore water.

The one-phase model is used to estimate the freely dissolved concentration of each PAH in pore water using the following relationship:

$$C_{SED}/C_W = f_{OC} * K_{OC} \quad \text{(Equation 1)}$$

where:

C_{SED} = the concentration of each PAH in sediment (μg/kg dry weight)

C_W = the concentration of freely dissolved PAH in pore water (μg/L)

f_{OC} = the weight fraction of total organic carbon in sediment (kg organic carbon/kg dry weight)

K_{OC} = the organic carbon-water partition coefficient for a specific PAH (L/kg organic carbon)

The equation is rearranged and used to solve for C_W. C_W for each PAH is divided by its corresponding FCV to calculate TUs.

The two-phase model is used to estimate the freely dissolved concentration of each PAH in pore water using the following relationship:

$$C_{SED}/C_W = f_{NPOC} * K_{OC} + f_{BC} * K_{BC} C_W^{n-1} \quad \text{(Equation 2; Accardi-Dey and Gschwend, 2002)}$$

where:

f_{NPOC} = the weight fraction of non-pyrogenic organic carbon in sediment (kg non-pyrogenic organic carbon/kg dry weight, calculated from the difference between total organic carbon and black carbon)

f_{BC} = the weight fraction of black carbon in sediment (kg black carbon/kg dry weight)

K_{BC} = the black carbon-pore water partition coefficient for each PAH (L/kg black carbon)

n = the Freundlich exponent, which accounts for nonlinear sorption behavior of black carbon (n=0.6) (Accardi-Dey and Gschwend, 2002)

We use an iterative approach to solve for C_W. C_W for each PAH is divided by its corresponding FCV to calculate a TU. Black carbon-pore water partition coefficients, K_{BC}, for 17 PAHs were taken from Accardi-Dey and Gschwend (2003). Because K_{BC} values were not available for all 34 PAHs, a regression relationship was used to estimate these values for the other PAHs (Kane Driscoll et al., 2009).

In order to calculate the sum of ESB TUs for the SPME method, the measured concentration of each PAH was divided by its corresponding ESB FCV (USEPA, 2003). The TUs for the individual PAH were summed in accordance with Hawthorne et al. (2007); if a peak was not detected above the method-specified signal-to-noise threshold, a zero was used in the TU calculation.

2.6 Statistical Data Analysis

Analysis of variance (ANOVA) followed by Dunnett's multiple comparison test and the Wilcoxon non-parametric test were used to detect significant differences among the toxicity test sample results. ANOVA followed by Dunnett's test is the most powerful analytical method for detecting statistical differences among multiple samples and a single control or reference group, but the data should meet the underlying assumptions of the method (equal variance and normality). Multiple residual and probability plots were used to test these assumptions. A non-parametric comparison was also conducted using an overall Kruskal-Wallis test followed by Wilcoxon pair-wise tests with an adjusted significance level to account for multiple tests. Additionally, an outlier evaluation using Dixon's test with a 0.05 significance level was completed to identify any samples that may be highly influencing the statistical comparison results. Statistical comparisons were conducted with and without these outlier samples. A sample was considered significantly lower than the upstream background samples or laboratory control sample if either statistical method indicated significance. An overall significance level of 0.05 was used, except for the non-parametric comparisons, which used adjusted significance levels.

Spearman correlation was used to evaluate the strength of relationships between each of the toxicity endpoints and the chemical (e.g., contaminant concentration, Sum-TU) and physical characteristics (e.g., TOC) of the sediment samples. Additionally, correlations among chemical and physical characteristics were used to examine the covariance among chemical contaminants in Brook sediments.

3. RESULTS AND DISCUSSION

3.1 Sediment Chemical Analyses

The 14 sediment samples contained a mixture of sands and fine-grained material (e.g., silt and clay). None of the samples contained any gravel or cobble. TOC content of sediments ranged from 0.6 to 3.2 percent (Table 1). Because *H. azteca* is tolerant to sediment grain size ranging from silt to predominantly sand, and TOC ranging from 0.3 to 9.6 percent (USEPA, 2000), grain size and TOC in these sediment samples probably did not adversely affect the results of the sediment toxicity tests.

Table 1. Physical characteristics of sediments.

Station	Total Organic Carbon (%)	Black Carbon (%)	Silt/Clay (%)
1	0.62	0.01	9.6
2	1.4	0.01	18
3	2.6	0.065	52
4	2.2	0.27	21
5	2.3	0.055	78
6	2.0	0.03	55
7	2.7	0.12	56
8	1.7	0.14	60
9	3.2	0.075	63
10	1.9	0.01	71
11	1.9	0.01	52
12	2.0	0.04	47
13	1.4	0.24	31
14	2.0	0.045	56

Sediments sampled near the facility had elevated concentrations of metals, PAHs, phthalates, and other organic chemicals (results of key chemical analyses are shown in Table 2). Concentrations of lead in sediment adjacent to and downstream of the facility ranged from 240 to 6,400 mg/kg dry, and upstream samples ranged from 7.9 to 52 mg/kg dry. Other metals, including antimony, cadmium, and zinc, were also elevated in sediment adjacent to and downstream of the facility relative to the upstream stations. Concentrations of total (34) PAHs in sediment adjacent to and downstream of the facility ranged from 48,000 to 890,000 μg/kg dry, and concentrations in upstream sediment samples ranged from 2,000 to 11,000 μg/kg dry. Similarly, concentrations of butyl

benzylphthalate in sediment adjacent to and downstream of the facility ranged from 32,000 to 1,000,000 μg/kg dry, and concentrations in upstream sediment samples were below detection limits (<440 μg/kg dry). In addition, total PCB Aroclors® up to 1,032 μg/kg dry and total DDT up to 90 μg/kg dry were detected in sediments near and downstream of the facility (Table 2).

Concentrations of metals, total PAHs, total PCB Aroclors®, and total DDT in sediment samples were compared to probable effect concentrations (PECs) and threshold effect concentrations (TECs) (MacDonald et al., 2000) to judge whether adverse biological effects to benthic macroinvertebrates could be occurring. Such comparisons alone do not indicate that adverse effects are occurring. The TECs for metals and the PECs for organic compounds have been adopted as the Massachusetts SQBs (http://www.mass.gov/dep/water/laws/ecoturss.pdf). Concentrations of metals, total PAHs, total PCB Aroclors®, and total DDT in upstream stations were below the PECs, and most of these chemicals were also below the TECs (Table 2). However, concentrations of lead and total PAHs in the sediment samples near and downstream of the facility were up to 50 and 40 times the PEC for lead and total PAHs, respectively. Concentrations of cadmium also exceeded the PEC at many locations near and downstream of the facility. Concentrations of zinc and total PCB Aroclors® occasionally exceeded their PECs near and downstream of the facility. Concentrations of total DDT in sediments exceeded the TEC at every location, but never exceeded the PEC. Although PECs were unavailable for butyl benzylphthalate and dibenzofuran, concentrations of these two chemicals were elevated in downstream sediment samples as compared to upstream sediment samples.

Concentrations of many of the chemicals co-vary in the Brook sediment (Table 3). In fact, all of the metals are significantly correlated with each other, as well as with total PCB Aroclors®, total PAHs, and butyl benzylphthalate (Spearman rank, $p<0.05$). Black carbon is also significantly correlated with all of the metals, total PCB Aroclors®, total PAHs, and butyl benzylphthalate. However, TOC is only significantly correlated with total DDT. The Sum-TUs did not significantly correlate with any of the metals or organic chemicals.

Table 2. Results of key chemical analyses and sediment toxicity tests for the Brook study site.

										Test Day 28				Test Day 42	
Station	Antimony (mg/kg)	Cadmium (mg/kg)	Lead (mg/kg)	Zinc (mg/kg)	Total PCB Aroclors® (µg/kg)[a]	Total DDTs (µg/kg)[a]	Butyl benzyl-phthalate (µg/kg)	Dibenzofuran (µg/kg)	Total 34-PAHs (µg/kg)[a]	Survival of *Hyalella azteca* (%)[b]	N	Dry Weight of *Hyalella azteca* (mg/survivor)[b]	N	Offspring of *Hyalella azteca* (number released per female)[b]	N
1	0.071	0.056	7.9	18	<56	12.4[c]	<240	<240	2,000[c]	96 (7)	12	0.37 (0.06)	4	8.18 (2.13)	8
2	0.14	0.2	26	55	<70[c]	24.7[c]	<280	<280	9,600[c]	94 (10)	12	0.31 (0.14)	4	7.32 (1.52)	8
3	0.54	0.44	52[c]	110	<98[c]	46.4[c]	<440	<250	11,000[c]	91 (17)	12	0.36 (0.16)	4	7.44 (2.87)	8
4	210	24[c,d]	6,400[c,d]	920[c,d]	570[c]	19[c]	140,000	1,600	660,000[c,d]	78 (19)[e,f,g,h]	12	0.28 (0.12)	4	2.36 (1.62)[e,f,g,h,i]	8
5	24	3.7[c]	1,100[c,d]	310[c]	380[c]	56[c]	32,000	370	68,000[c,d]	93 (11)	12	0.39 (0.03)	4	7.74 (4.21)	8
6	39	8.7[c,d]	1,900[c,d]	340[c]	612[c]	47[c]	290,000	420	110,000[c,d]	84 (13)[e,g,h]	12	0.24 (0.01)[e]	4	2.81 (1.22)[e,g,h,i]	8
7	47	7.0[c,d]	2,500[c,d]	420[c]	1,032[c,d]	40[c]	1,000,000	3,700	890,000[c,d]	63 (18)[e,f,g,h,i]	12	0.24 (0.09)[e]	4	3.22 (3.48)[e,g,h]	8
8	77	6.1[c,d]	2,600[c,d]	390[c]	421[c]	28[c]	140,000	2,200	150,000[c,d]	83 (15)[g,h]	12	0.36 (0.03)	4	4.49 (2.38)[g,h]	8
9	61	18[c,d]	2,700[c,d]	900[c,d]	664[c]	46[c]	380,000	1,500	230,000[c,d]	2 (4)[e,f,g,h,i]	12	0.5	1	0[e,f,g,h,i]	1
10	19	7.7[c,d]	710[c,d]	300[c]	504[c]	37[c]	220,000	360	84,000[c,d]	94 (5)	12	0.33 (0.06)	4	5.33 (2.38)	8
11	13	4.1[c]	420[c,d]	200[c]	318[c]	64[c]	76,000	4,600	72,000[c,d]	93 (10)	12	0.36 (0.04)	4	7.27 (2.34)	8
12	29	7.5[c,d]	1,200[c,d]	660[c,d]	1,000[c,d]	54[c]	600,000	190	57,000[c,d]	10 (11)[e,f,g,h,i]	12	0.40 (0.05)	3	1.00 (1.41)[e,g]	3
13	7.3	4.1[c]	340[c,d]	200[c]	340[c]	41[c]	140,000	<340	48,000[c,d]	90 (6)	12	0.32 (0.03)	4	6.13 (2.39)	8
14	4.9	3.1[c]	240[c,d]	220[c]	459[c]	90[c]	76,000	150	100,000[c,d]	92 (6)	12	0.31 (0.08)	4	4.72 (2.04)[g,h]	8
TEC[j]	NA	0.99	35.8	121	59.8	5.28	NA	NA	1,610	--		--		--	
PEC[k]	NA	4.98	128	459	676	572	NA	NA	22,800	--		--		--	

[a] For undetected values, one-half of the detection limits were used to calculate the sum concentrations.
[b] Values shown are the mean (with the standard deviation in parathenses). Laboratory control values were as follows: Mean survival = 93 (6)%; Mean dry weight = 0.37 (0.04) mg/survivor; Mean number of offspring = 5.93 (1.90) per female.
[c] Concentration exceeds its corresponding threshold effect concentration (TEC).
[d] Concentration exceeds its corresponding the probable effect concentration (PEC).
[e] Statistically significant when compared to the control group based on Kruskal-Wallis analysis ($p<0.05$).
[f] Statistically significant when compared to the control group based on ANOVA followed by Dunnett's test ($p<0.05$).
[g] Statistically significant when compared to the pooled reference samples based on ANOVA followed by Dunnett's test ($p<0.05$).
[h] Statistically significant when compared to the pooled reference samples based on Wilcoxon test ($p<0.05$).
[i] Statistically significant when compared to the control group based on Wilcoxon test ($p<0.05$).
[j] Threshold effect concentration (MacDonald et al. 2000). [k] Probable effect concentration (MacDonald et al. 2000).

Table 3. Summary of correlations between chemical variables.[a]

	Metals (mg/kg, dry)				Organic Analytes (μg/kg, dry)						Other (%)		Toxicity Units		
	Antimony	Cadmium	Lead	Zinc	Total Aroclor® PCBs	Total DDTs	Total 17 PAHs	Total 34 PAHs	Butyl benzylphthalate	Dibenzofuran	Black Carbon	Total Organic Carbon	Sum-TU (one-phase model)	Sum-TU (two-phase model)	Sum-TU (SPME)[b]
Metals (mg/kg, dry)															
Antimony		0.86[d]	1.00[c]	0.94[c]	0.77[d]	-0.05	0.79[d]	0.86[d]	0.73[d]	0.79[d]	0.65[e]	-0.51	-0.08	0.16	0.15
Cadmium	0.86[d]		0.88[d]	0.87[d]	0.82[d]	-0.02	0.65[e]	0.77[d]	0.82[d]	0.65[e]	0.73[d]	-0.39	-0.14	0.25	0.06
Lead	1.00[c]	0.88[d]		0.95[c]	0.79[d]	-0.04	0.78[d]	0.87[d]	0.74[d]	0.78[d]	0.64[e]	-0.52	-0.10	0.19	0.12
Zinc	0.94[c]	0.87[d]	0.95[c]		0.88[d]	0.05	0.66[e]	0.82[d]	0.79[d]	0.65[e]	0.70[e]	-0.44	-0.03	0.32	0.16
Organic Compounds (μg/kg, dry)															
Total Aroclor® PCBs	0.77[d]	0.82[d]	0.79[d]	0.88[d]		0.19	0.59[e]	0.78[d]	0.94[c]	0.54	0.85[d]	-0.26	0.27	0.40	0.33
Total DDTs	-0.05	-0.02	-0.04	0.05	0.19		0.13	0.04	0.13	0.13	0.04	0.69[e]	0.39	-0.18	0.14
Total 17 PAHs	0.79[d]	0.65[e]	0.78[d]	0.66[e]	0.59[e]	0.13		0.88[d]	0.60[e]	0.97[c]	0.51	-0.30	0.01	0.08	0.17
Total 34 PAHs	0.86[d]	0.77[d]	0.87[d]	0.82[d]	0.78[d]	0.04	0.88[d]		0.70[e]	0.79[d]	0.60[e]	-0.45	0.09	0.30	0.33
Butyl benzylphthalate	0.73[d]	0.82[d]	0.74[d]	0.79[d]	0.94[c]	0.13	0.60[e]	0.70[e]		0.57[e]	0.88[d]	-0.22	0.25	0.31	0.23
Dibenzofuran	0.79[d]	0.65[e]	0.78[d]	0.65[e]	0.54	0.13	0.97[c]	0.79[d]	0.57[e]		0.48	-0.27	-0.07	0.08	0.08
Other (%)															
Black Carbon	0.65[e]	0.73[d]	0.64[e]	0.70[e]	0.85[d]	0.04	0.51	0.60[e]	0.88[d]	0.48		-0.05	0.36	0.37	0.45
Total Organic Carbon	-0.51	-0.39	-0.52	-0.44	-0.26	0.69[e]	-0.30	-0.45	-0.22	-0.27	-0.05		0.43	-0.17	0.20
PAH Toxicity Units															
Sum-TU (one-phase model)	-0.08	-0.14	-0.10	-0.03	0.27	0.39	0.01	0.09	0.25	-0.07	0.36	0.43		0.01	0.85
Sum-TU (two-phase model)	0.16	0.25	0.19	0.32	0.40	-0.18	0.08	0.30	0.31	0.08	0.37	-0.17	0.01		0.20
Sum-TU (SPME)[b]	0.15	0.06	0.12	0.16	0.33	0.14	0.17	0.33	0.23	0.08	0.45	0.20	0.85[d]	0.20	

[a] Correlation estimate is based on Spearman non-parametric correlation. *P*-values have not been adjusted for the number of analyses.

[b] Solid-phase microextraction. Undetected results were included as zeros.

[c] $p<0.001$.

[d] $p<0.01$.

[e] $p<0.05$.

3.2 Sediment Toxicity Tests

In general, there were no unacceptable water quality deviations during the 42-day *H. azteca* test, and test results met the test acceptability criteria. The negative control had mean survival at 28 days of 93 percent, growth at day 28 of 0.37 mg per amphipod, and 5.93 offspring per female amphipod at day 42. The performance of the positive control suggested that the test organisms were suitably sensitive for testing.

Mean survival of *H. azteca* ranged from 2 percent at Station 9, which was downstream of the facility, to 96 percent at Station 1, which was upstream of the facility. Mean number of offspring per *H. azteca* female ranged from 0 at Station 9 to 8.18 at Station 1. No significant reductions in growth, which ranged from 0.24 to 0.50 mg dry weight per amphipod, were observed in any of the downstream samples in comparison to the upstream background samples (Table 2). Survival and reproduction were significantly reduced in five of the six sediment samples (Stations 4, 6, 7, 8, and 9) from areas adjacent to the facility and in one of the samples from downstream of the facility (Station 12) in comparison to the upstream background samples. Reproduction was also significantly reduced at Station 14 in comparison to the upstream background samples, although survival was not depressed at Station 14.

3.3 SPME for 34 PAHs

The SPME method was used on a subset of five sediment samples (Stations 5, 6, 7, 11, and 13) to measure concentrations of freely dissolved PAHs in pore water. Most of the 34 PAHs were detected in at least one of the five sediment samples by the SPME analysis, with the exception of benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenz[a,h]anthracene, C3–fluorenes, C4–phenanthrenes + anthracene, C1–chrysene + benz[a]anthracene, C2–chrysene + benz[a]anthracene, C3–chrysene + benz[a]anthracene and C4–chrysene + benz[a]anthracene. According to the approach adopted by Hawthorne et al. (2005), the Sum-TU for the SPME samples was calculated using zero as the concentration for PAHs below the practical quantitation limits for this method.

3.4 Prediction of PAH Toxicity

The Sum-TUs estimated from the one-phase model, which accounts for association of PAHs with TOC, were greater than 1.0 for the entire set of sediment samples including those from the upstream stations, ranging from 1.2 to 39 Sum-TU (Table 4). Sum-TUs estimated from the two-phase model, which accounts for association of PAHs with TOC and black carbon, were greater than 1.0 for all sediment samples, except for one upstream sample, ranging from 0.8 to 30. Although the results of the one- and two-phase models predict that concentrations of PAHs in the 14 sediment samples are not acceptable for the protection of benthic organisms, the toxicity results for 7 of the 14 samples (Stations 1, 2,

3, 5, 10, 11, and 13) were not significantly different from the laboratory control sample. In contrast, the Sum-TUs calculated from the results of the SPME for 5 samples were all less than 1.0, ranging from 0.22 to 0.56, a result that suggests that concentrations of freely dissolved PAHs in these samples are below a concentration that would be expected to result in adverse effects to benthic invertebrates. However, toxicity test results for two of the five samples (Stations 6 and 7) analyzed by SPME were significantly different from the laboratory control and upstream samples. Thus, none of the three Sum-TU models could adequately explain the observed toxicity in all samples. In addition, results of these three models were not significantly correlated with survival and reproduction of *H. azteca* (Table 5). Growth of *H. azteca* was negatively correlated with Sum-TU SPME results, but growth was not significantly different among the stations.

Table 4. Results of polycyclic aromatic hydrocarbon toxicity models.

Station	Sum-TU[a] (one-phase model)[b]	Sum-TU (two-phase model)[c]	Sum-TU (SPME)[d]
1	3.6	3.2	--[d]
2	2.2	2.0	--[d]
3	1.2	0.8	--[e]
4	36	19	--[e]
5	4.3	3.0	0.39
6	7.5	6.2	0.56
7	39	30	0.33
8	12	5.4	--[e]
9	9.4	7.0	--[e]
10	6.3	5.7	--[e]
11	5.7	5.3	0.17
12	4.6	3.4	--[e]
13	5.6	1.2	0.22
14	6.8	5.0	--[e]

[a] toxic unit

[b] This model estimates concentration of PAHs in pore water from the concentrations of PAHs in bulk sediment and TOC.

[c] This model estimates concentration of PAHs in pore water from the concentrations of PAHs in bulk sediment, TOC, and black carbon.

[d] Solid-phase microextraction technique directly measures the concentrations of PAHs in pore water. Undetected results were included as zeroes.

[e] Test was not run on sample.

Table 5. Summary of correlations between three toxicity endpoints from sediment toxicity tests with *Hyalella azteca* and chemical variables.

	Survival of *Hyalella azteca* (%)		Dry Weight of *Hyalella azteca* (mg/survivor)		Offspring of *Hyalella azteca* (number released per female)	
	Correlation[a]	*P*-value	Correlation	*P*-value	Correlation	*P*-value
Metals (mg/kg, dry)						
Antimony	-0.77	0.0053	--	ns[b]	-0.78	0.0048
Cadmium	-0.69	0.0124	--	ns	-0.86	0.0019
Lead	-0.79	0.0044	--	ns	-0.80	0.0037
Zinc	-0.84	0.0024	--	ns	-0.87	0.0016
Organic Compounds (µg/kg, dry)						
Total Aroclor® PCBs	-0.79	0.0044	--	ns	-0.88	0.0014
Total DDTs	--	ns	--	ns	--	ns
Total 17 PAHs	--	ns	--	ns	--	ns
Total 34 PAHs	-0.65	0.0183	--	ns	-0.74	0.0078
Butyl benzylphthalate	-0.78	0.0048	--	ns	-0.86	0.0019
Dibenzofuran	--	ns	--	ns	--	ns
Organic Compounds (µg/kg OC, dry)						
Total Aroclor® PCBs	--	ns	--	ns	--	ns
Total DDTs	-0.61	0.0278	--	ns	-0.71	0.0107
Total 17 PAHs	-0.59	0.0331	--	ns	--	ns
Total 34 PAHs	-0.61	0.0267	--	ns	-0.73	0.0081
Butyl benzylphthalate	--	ns	--	ns	-0.71	0.0098
Dibenzofuran	--	ns	--	ns	--	ns
Other (%)						
Black carbon	-0.58	0.0360	--	ns	-0.75	0.0071
TOC	--	ns	--	ns	--	ns
PAH Toxicity Units						
Sum-TU (one-phase model)	--	ns	--	ns	--	ns
Sum-TU (two-phase model)	--	ns	--	ns	--	ns
Sum-TU (SPME)[c]	--	ns	-0.55	0.0453	--	ns

[a] Correlation estimate is based on Spearman non-parametric correlation. *P*-values have not been adjusted for the number of analyses.

[b] Not significant, *P*-value greater than 0.05.

[c] Solid-phase microextraction. Undetected results were included as zeros.

Measured concentrations of PAHs in pore water by SPME and corresponding Sum-TUs were much lower than predicted from the one-phase or two-phase model. The

differences between measured and modeled concentrations of PAHs in pore water may relate to the partitioning coefficients used in the two-phase model. The K_{BC} values in the two-phase model were taken from Accardi-Dey and Gschwend (2003), which calculated K_{BC} values for 17 PAHs. Kane Driscoll et al. (2009) estimated the K_{BC} values for the other 17 PAHs using a regression relationship between the existing K_{BC} and K_{OW} values. However, K_{BC} values for individual PAHs in sediments can range nearly three orders of magnitude (Hawthorne et al., 2007). Rearranging Equation 2 to solve for K_{BC}, we calculated the K_{BC} values for individual PAHs in the Brook sediment samples using the pore water concentrations determined by SPME in five samples run in duplicate analysis (i.e., 10 results). The site-specific K_{BC} values for individual PAHs were compared to those found by Hawthorne et al. (2007) and those used in the two-phase model (Kane Driscoll et al., 2009) (Figure 2). The site-specific K_{BC} values for individual PAHs fell within the range observed by Hawthorne et al. (2007), but the site-specific K_{BC} values for several PAHs were greater than those used in the two-phase model. These comparisons suggest a greater than expected adsorption of PAHs to black carbon in Brook sediments. It is also possible that other sorbent phases were present in the sediment samples that were not accounted for in the black carbon and TOC analyses.

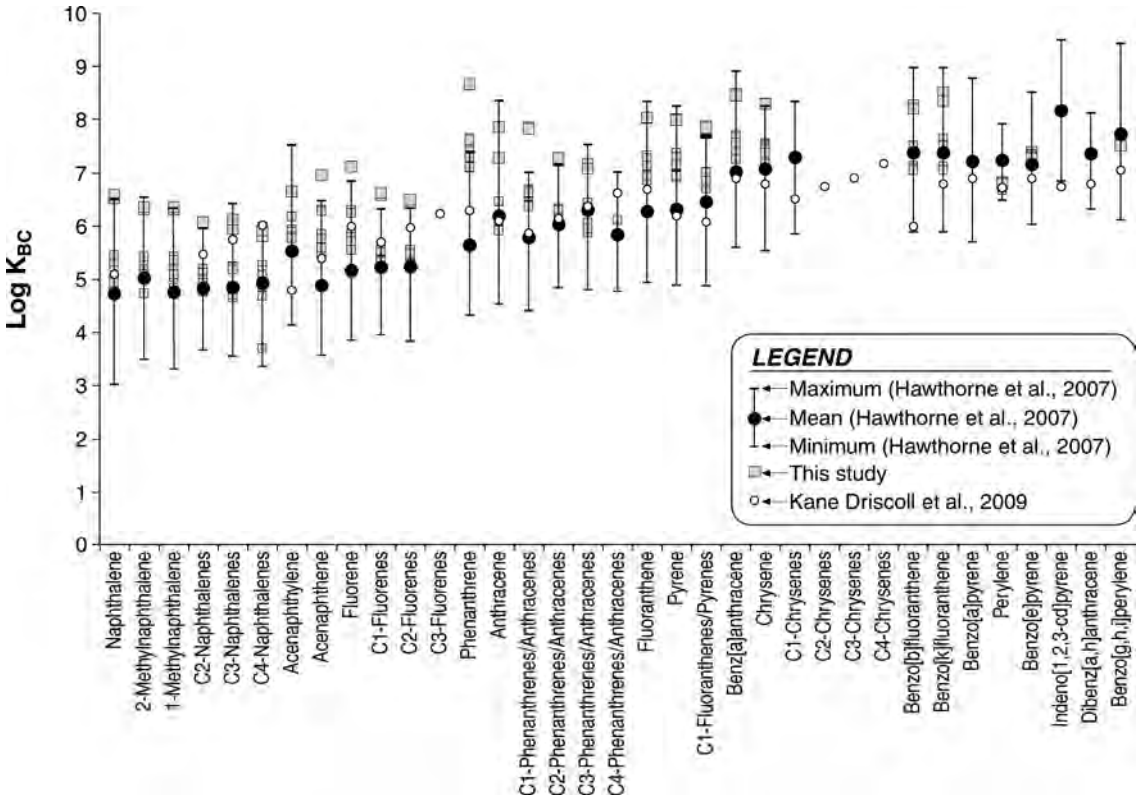

Figure 2. Black carbon-pore water partition coefficient (K_{BC}) values for parent and alkyl PAHs from 114 background and historically contaminated sediments (Hawthorne et al. 2007), Boston Harbor sediments (Kane-Driscoll et al. 2009), and Brook sediments (the present study).

3.5 Correlation Evaluation

The potential contribution of co-occurring metals and other organic sediment contaminants to toxicity was further evaluated. Spearman correlation was used to evaluate the strength of relationships between concentrations of chemicals and each of the toxicity endpoints. Chemicals included in the correlation analysis were those metals with sediment concentrations that were consistently greater than the PECs (cadmium, lead, and zinc) and organic compounds with sediment concentrations consistently greater than the TECs (total PCBs, total DDT, and the 34 PAH compounds). Antimony, butyl benzylphthalate, dibenzofuran, and the Sum-TUs calculated from the one- or two-phase models or SPME were also included in the correlation analysis because they were elevated relative to upstream samples. In addition, TOC and black carbon were included in the correlation analysis to determine whether these constituents were related to the observed toxicity. Correlations between concentrations of organic contaminants and the toxicity test endpoints were analyzed on both a dry-weight basis and an organic carbon-normalized basis.

Both survival and reproduction had significant negative correlations with concentrations of metals, black carbon, and the total PAHs on a dry-weight and organic-carbon basis (Table 5). Additionally, these endpoints were also correlated with organic-carbon normalized concentrations of DDT. Total PCBs and butyl benzylphthalate were also significantly correlated to survival and reproduction. Additional chemicals had significant correlations with only one of the toxicity endpoints (Table 5). Growth was significantly and negatively correlated with the Sum-TU for the SPME method, but growth was not significantly different among the stations. PAHs were ruled out as chemicals contributing to observed toxicity, because the Sum-TU SPME analysis indicated that concentrations of PAHs in these sediments would not result in toxicity to benthic organisms, and the Sum-TU determined by any method (one-phase, two-phase, or SPME) did not correlate strongly with survival and reproduction.

To further assess which chemical(s) may be contributing to the observed toxicity in the chronic sediment toxicity tests, the concentrations of antimony, cadmium, lead, zinc, total PCBs, total DDT, butyl benzylphthalate, and dibenzofuran in sediment samples were compared to the PECs, or other sources of sediment concentrations associated with effects if PECs were unavailable for a particular chemical. For example, the secondary chronic value (SCV) from Jones et al. (1997) was used for dibenzofuran after being corrected for 2% TOC. Studies on phthalate exposures to benthic organisms are limited; although the available data suggest that sediment effect concentrations for phthalates are in the range of >1,000 to >30,000 mg/kg dry weight for sediments with medium organic carbon content (approximately 5 percent TOC) (Staples et al., 1997). The low end of this range (1,000 mg/kg dry weight) was used as the SQB for butyl benzylphthalate after being corrected for 2% TOC.

Zinc, PCBs, DDT, butyl benzylphthalate, and dibenzofuran were eliminated as primary contributors to observed toxicity because concentrations did not exceed or only slightly exceeded sediment effect concentrations (Table 6). Toxicity appears to be most closely correlated with metals, especially lead (Figure 3). Other metals correlate well with toxicity, but have much lower PEC quotients than lead, and therefore are considered less likely than lead to contribute to toxicity. Because PEC quotients for lead, which range from 2 to 50, are much more consistent and much higher than PEC quotients for other metals, and because the severity of the effects on survival and reproduction increased with lead concentration, lead was considered to be the major contributor to observed sediment toxicity.

3.6 Cleanup Strategy for the Brook

A remedial feasibility study was conducted using the information from this investigation and other technical engineering investigations to develop a remedy for the Brook. Based on those studies, sediment removal is the planned remedial approach for the Brook. Sediment down to native material (i.e., pre-facility stream sediment) will be removed in areas where sediment toxicity was observed in the chronic sediment toxicity tests, and where that toxicity is strongly related to the presence of lead. These areas include Stations 4 through 9, which are adjacent and immediately downstream of the facility, and Station 12, which is a downstream hot spot. There was no toxicity observed at Stations 10 and 11. In certain areas (e.g., Station 12) that are not bounded by the presence of a sample with significant toxicity, the extent of contamination was delineated by the concentration of lead.

A target cleanup level for lead in sediment was developed by determining the concentration of lead at or below which no adverse effects are observed, referred to as the apparent effects threshold (AET). This approach used only "matched" chemical and biological effects data. Sediment samples were labeled "impacted" if survival, growth, or reproduction in facility samples was significantly lower than in the upstream samples. Note that survival, growth, and reproduction in the upstream background samples were not statistically different from the laboratory control. Using only the nonimpacted samples, the site-specific AET was set as the highest concentration that did not exhibit statistically significant effects on survival, growth, or reproduction. The AET for lead in these sediment samples was 1,100 mg/kg dry weight. The lowest concentration of lead at which significant effects were observed was 1,200 mg/kg dry weight. The putative toxicity threshold for lead in sediment is between 1,100 and 1,200 mg/kg dry weight, and the target cleanup level was set equal to the geometric mean of these values or 1,150 mg/kg dry weight.

Table 6. Comparison of sediment concentrations to sediment quality benchmarks.

	Sediment Concentrations				Sediment Quality Benchmark			Sediment Quality Benchmark Quotient			
Chemical	Range						Source	Mean	Range		
Antimony	4.9	–	210	mg/kg	25	mg/kg	ERM	1.9	0.2	–	8.4
Cadmium	3.1	–	24	mg/kg	4.98	mg/kg	PEC	1.7	0.6	–	4.8
Lead	240	–	6,400	mg/kg	128	mg/kg	PEC	14	2	–	50
Zinc	200	–	920	mg/kg	459	mg/kg	PEC	0.96	0.4	–	2.0
Total PCBs	318	–	1,032	µg/kg	676	µg/kg	PEC	0.85	0.5	–	1.5
Total DDT	19	–	90	µg/kg	572	µg/kg	PEC	0.08	0.03	–	0.2
Butyl benzylphthalate	32,000	–	1,000,000	µg/kg	500,000	µg/kg	Staples et al. 1997	0.56	0.1	–	2.0
Dibenzofuran	150	–	4,600	µg/kg	840	µg/kg	SCV	1.2	0.18	–	5.5

Note: ERM - effects range Median (Long and Morgan 1990),

PEC - probable effect concentration (MacDonald et al. 2000),

SCV - secondary chronic value (Jones et al. 1997).

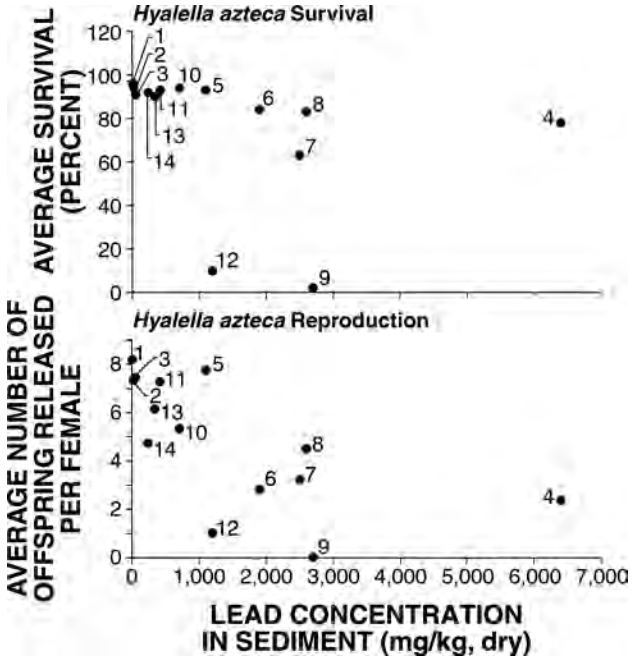

Figure 3. Relationship between lead concentrations in Brook sediments and *Hyalella azteca* survival (top) and reproduction (bottom).

While several samples were taken from the Brook to assess risk to benthic invertebrates, additional data were needed to delineate the removal area at Station 12, the one downstream station where toxicity was observed. The strategy for delineating the removal area at Station 12 was developed to protect benthic invertebrates from hazardous exposure to facility-related chemicals. First, the initial limits of sediment removal at Station 12 were defined as 50 ft upstream and 50 ft downstream of Station 12 (2,000 ft^2). One composite sediment sample was collected and submitted for analysis to confirm the presence of lead in excess of 1,150 mg/kg dry weight. Additional composite sediment samples were collected over 1,000-ft^2 sections of the Brook bed, starting with the closest 1,000-ft^2 sections upstream and downstream of the initial removal area, and extending to up to 3,000-ft^2 upstream and downstream of the initial limits of sediment removal at Station 12. Each sediment sample was composited from seven field-collected samples on a randomized basis and analyzed for lead. The area of approximately 1,000 ft^2 was selected to further define the limits of remediation around Station 12 because Massachusetts has set a regulatory precedent for evaluating exposure of benthic invertebrates to chemical concentrations in sediments over an area no greater than 1,000 ft^2. Analysis of the composite sediment sample from the initial limits of sediment removal at Station 12 showed a concentration of 1,980 mg/kg dry weight, confirming the presence of elevated lead concentrations in sediments at Station 12. The limit of excavation downstream of Station 12 was extended 50 ft from Station 12 to include an additional 1,000 ft^2 segment because the sediment sample from that segment contained a lead concentration of 1,310 mg/kg dry weight. Samples further downstream contained lead concentrations less than the site-specific target level for lead and will remain in place

(Figure 4). The sediment sample from the most distant upstream segment contained a lead concentration higher than the site-specific target cleanup level for lead. To address this area upstream of Station 12 and potentially contaminated areas just upstream of this segment, the excavation limit was extended 230 ft from Station 12, which is the midway point between Stations 12 and 11 (the nearest nontoxic station) (Figure 4).

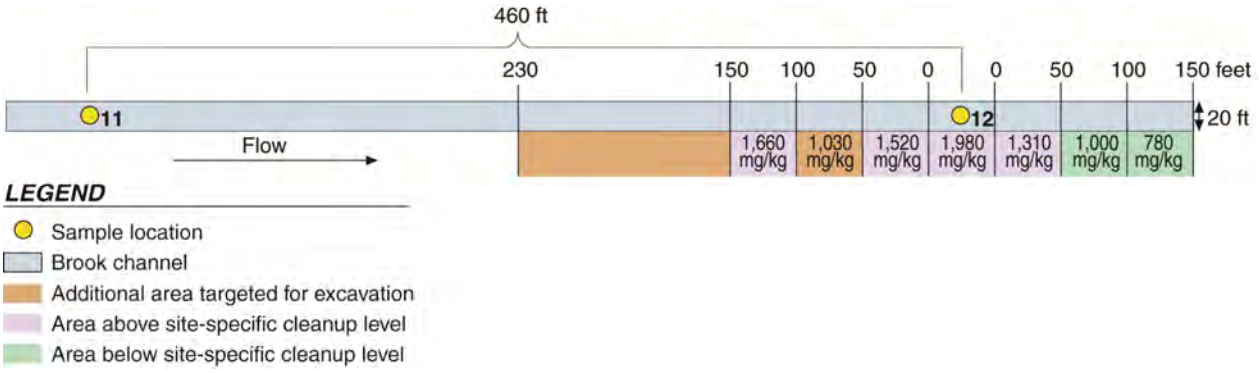

Figure 4. Delineation of sediment removal area at Station 12. Concentrations of lead in composite samples from 1,000 ft^2 segments are shown. Areas designated for sediment removal based on comparison of lead concentrations to the site-specific target cleanup level (1,150 mg/kg) are shown.

The sediment excavation limits at Station 12 will be limited to 6,600 ft^2 in total (1,000 ft^2 downstream and 4,600 ft^2 upstream of the center point at Station 12). However, confirmatory sampling and other verification activities will be conducted to demonstrate that cleanup objectives were met in that area.

4. CONCLUSIONS

The results of this study identified areas where sediment in the Brook exhibited significant toxicity and contained potentially facility-related contaminants at concentrations that were closely associated with observed toxicity. Use of SPME analysis of PAHs in sediment pore water provided evidence that the observed toxicity to *H. azteca* was not primarily caused by PAHs. In the SPME analysis, bulk sediment concentrations of total PAHs ranging from 48,000 to 890,000 μg/kg dry weight were predicted not to be toxic to benthic invertebrates. Freely dissolved concentrations of PAHs in pore water were much lower than predicted based on bulk sediment PAH concentrations. Further analysis of the co-located toxicity and sediment chemistry data indicated that lead was the primary contributor to the observed toxicity to *H. azteca*. Use of the site-specific target cleanup level (1,150 mg lead/kg sediment dry weight) resulted in a substantially smaller remediation footprint in the Brook (24,434 ft^2; 2,270 m^2) than that originally proposed (64,799 ft^2; 6,020 m^2) based on exceedance of SQBs (Figure 1).

5. ACKNOWLEDGEMENTS

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Designation: D 7363 – 07

Standard Test Method for Determination of Parent and Alkyl Polycyclic Aromatics in Sediment Pore Water Using Solid-Phase Microextraction and Gas Chromatography/Mass Spectrometry in Selected Ion Monitoring Mode[1, 2]

This standard is issued under the fixed designation D 7363; the number immediately following the designation indicates the year of original adoption or, in the case of revision, the year of last revision. A number in parentheses indicates the year of last reapproval. A superscript epsilon (ε) indicates an editorial change since the last revision or reapproval.

1. Scope

1.1 The U.S. Environmental Protection Agency (USEPA) narcosis model for benthic organisms in sediments contaminated with polycyclic aromatic hydrocarbons (PAHs) is based on the concentrations of dissolved PAHs in the interstitial water or "pore water" in sediment. This test method covers the separation of pore water from PAH-impacted sediment samples, the removal of colloids, and the subsequent measurement of dissolved concentrations of the required 10 parent PAHs and 14 groups of alkylated daughter PAHs in the pore water samples. The "24 PAHs" are determined using solid-phase microextraction (SPME) followed by Gas Chromatography/Mass Spectrometry (GC/MS) analysis in selected ion monitoring (SIM) mode. Isotopically labeled analogs of the target compounds are introduced prior to the extraction, and are used as quantification references.

1.2 Lower molecular weight PAHs are more water soluble than higher molecular weight PAHs. Therefore, USEPA-regulated PAH concentrations in pore water samples vary widely due to differing saturation water solubilities that range from 0.2 µg/L for indeno[1,2,3-cd]pyrene to 31 000 µg/L for naphthalene. This method can accommodate the measurement of milligram per litre concentrations for low molecular weight PAHs and nanogram per litre concentrations for high molecular weight PAHs.

1.3 The USEPA narcosis model predicts toxicity to benthic organisms if the sum of the toxic units (ΣTU_c) calculated for all "34 PAHs" measured in a pore water sample is greater than or equal to 1. For this reason, the performance limit required for the individual PAH measurements were defined as the concentration of an individual PAH that would yield 1/34 of a toxic unit (TU). However, the focus of this method is the 10 parent PAHs and 14 groups of alkylated PAHs (Table 1) that contribute 95 % of the toxic units based on the analysis of 120 background and impacted sediment pore water samples.[3] The primary reasons for eliminating the rest of the 5-6 ring parent PAHs are: *(1)* these PAHs contribute insignificantly to the pore water TU, and *(2)* these PAHs exhibit extremely low saturation solubilities that will make the detection of these compounds difficult in pore water. This method can achieve the required detection limits, which range from approximately 0.01 µg/L, for high molecular weight PAHs, to approximately 3 µg/L for high molecular weight PAHs.

1.4 The test method may also be applied to the determination of additional PAH compounds (for example, 5- and 6-ring PAHs as described in Hawthorne et al).[4] However, it is the responsibility of the user of this standard to establish the validity of the test method for the determination of PAHs other than those referenced in 1.1 and Table 1.

1.5 *This standard does not purport to address all of the safety concerns, if any, associated with its use. It is the responsibility of the user of this standard to establish appropriate safety and health practices and determine the applicability of regulatory limitations prior to use.* For specific hazard statements, refer to Section 9.

2. Referenced Documents

2.1 *ASTM Standards:* [5]

D 1192 Guide for Equipment for Sampling Water and

[1] This test method is under the jurisdiction of ASTM Committee D19 on Water and is the direct responsibility of Subcommittee D19.06 on Methods for Analysis for Organic Substances in Water.

Current edition approved Aug. 1, 2007. Published August 2007.

[2] Standard methods under the jurisdiction of ASTM Committee D19 may be published for a limited time preliminary to the completion of full collaborative study validation. Such standards are deemed to have met all other D19 qualifying requirements but have not completed the required validation studies to fully characterize the performance of the test method across multiple laboratories and matrices. Preliminary publication is done to make current technology accessible to users of Standards, and to solicit additional input from the user community.

[3] Hawthorne, S. B., Grabanski, C. B., and Miller, D. J., "Measured Partitioning Coefficients for Parent and Akyl Polycyclic Aromatic Hydrocarbons in 114 Historically Contaminated Sediments: Part I, Koc Values," *Environmental Toxicology and Chemistry*, 25, 2006, pp. 2901-2911.

[4] Hawthorne, S. B., Grabanski, C.B., Miller, D .J., and Kreitinger, J. P., "Solid Phase Microextraction Measurement of Parent and Alkyl Polycyclic Aromatic Hydrocarbons in Milliliter Sediment Pore Water Samples and Determination of K_{DOC} Values," *Environmental Science Technology*, 39, 2005, pp. 2795-2803.

[5] For referenced ASTM standards, visit the ASTM website, www.astm.org, or contact ASTM Customer Service at service@astm.org. For *Annual Book of ASTM Standards* volume information, refer to the standard's Document Summary page on the ASTM website.

TABLE 1 Relative Response Factors[A]

Analyte	SPME-GC/MS RRF[B] versus Parent	Basis for Performance Limit[C]
Naphthalene	1.00	B
2-Methylnaphthalene[D]	1.00	B
1-Methylnaphthalene	1.00	B
C2-Naphthalenes	1.44	B
C3-Naphthalenes	0.88	B
C4-Naphthalenes	0.71	C
Acenaphthylene	1.00	B
Acenaphthene	1.00	B
Fluorene	1.00	B
C1-Fluorenes	0.73	B
C2-Fluorenes	0.59	B
C3-Fluorenes	0.35	S
Phenanthrene	1.00	B
Anthracene	1.00	B
C1-Phenanthrenes/Anthracenes	0.57	B
C2-Phenanthrenes/Anthracenes	0.32	B
C3-Phenanthrenes/Anthracenes	0.29	B
C4-Phenanthrenes/Anthracenes	0.12	S
Fluoranthene	1.00	B
Pyrene	1.00	B
C1-Fluoranthenes/Pyrenes	0.51	C
Benz[a]anthracene	1.00	B
Chrysene	1.00	B
C1-Chrysenes/Benz[a]anthracenes	0.62	C

[A] From Hawthorne, S. B., Grabanski, C.B., Miller, D .J., and Kreitinger, J. P., "Solid Phase Microextraction Measurement of Parent and Alkyl Polycyclic Aromatic Hydrocarbons in Milliliter Sediment Pore Water Samples and Determination of K_{DOC} Values," *Environmental Science Technology*, 39, 2005, pp. 2795-2803.

[B] All relative response factors are based on the SPME-GC/MS peak area per ng of the alkyl PAH in a water standard compared to that of its parent PAH as determined by SPME followed by GC/MS.When several isomers were available, (for example, C2-naphthalenes), the mean relative response factor is reported. The relative response factors of alkyl PAHs for which no standards were available were estimated based on the closest analogous alkyl PAH as described in reference 2.1.

[C] Performance limits were determined as 3 times the background concentrations from the SPME fiber based on the analysis of water blanks ("B"), the lowest calibration standard which consistently yielded a signal to noise ratio of at least 3:1 ("C"), or (for when no calibration standard was available) for the lowest concentrations consistently found in pore water samples with a signal to noise ratio of at least 3:1 ("S"). Detection limits for alkyl PAHs are based on a single isomer.

[D] Alkyl PAHs used to determine the SPME-GC/MS relative response factors including alkyl naphthalenes (1-methyl-, 2-methyl-, 1,2-dimethyl-, 1,3-dimethyl-1,8-dimethyl-, 2,7-dimethyl-, 1-ethyl, 2-ethyl, 1,4,5-trimethyl-, 2,3,5-trimethyl-, and 2-isopropyl-), 1-methylfluorene, 2-methyl- and 9-methylanthracene, 1-methyl-, 2-methyl-, and 3-methylphenanthrene, 9,10-dimethylanthracene, 2-ethylanthracene, 2-tertbutylanthracene, 1-methyl-7-isopropylphenanthrene, 1-methylpyrene, 7-methylbenz[a]anthracene, and 7,12-dimethylbenz[a]anthracene.

Steam in Closed Conduits[6]
D 1193 Specification for Reagent Water
D 2777 Practice for Determination of Precision and Bias of Applicable Test Methods of Committee D19 on Water
D 3370 Practices for Sampling Water from Closed Conduits
D 5847 Practice for Writing Quality Control Specifications for Standard Test Methods for Water Analysis
E 178 Practice for Dealing With Outlying Observations

3. Terminology

3.1 *Definitions:*

3.1.1 *calibration standard*—a solution prepared from a secondary standard, stock solution, or both, and used to calibrate the response of the instrument with respect to analyte concentration.

[6] Withdrawn.

3.1.2 *calibration verification standard (VER)*—the mid-point calibration standard (CS3) that is analyzed daily to verify the initial calibration.

3.1.3 *CS1, CS2, CS3, CS4*—shorthand notation for calibration standards.

3.1.4 *data acquisition parameters*—parameters affecting the scanning operation and conversion of the analytical signal to digitized data files. These include the configuration of the ADC circuitry, the ion dwell time, the MID cycle time, and acquisition modes set up for the method. Examples of acquisition modes for the HP5973 include SIM mode, and Low Mass Resolution Mode.

3.1.5 *performance limit*—performance limit for individual PAH is defined as the concentration of an individual PAH that would yield 1/34 of a toxic unit. For performance limit of individual PAH, refer to Table 2 (see 4.6).

3.1.6 *deuterated PAH (d-PAH)*—polycyclic aromatic hydrocarbons in which deuterium atoms are substituted for all hydrogens (that is, perdeuterated). In this method, d-PAHs are used as internal standards.

3.1.7 *GC*—gas chromatograph or gas chromatography.

3.1.8 *HRGC*—high resolution GC.

3.1.9 *LRMS*—low resolution MS.

3.1.10 *internal standards*—isotopically labeled analogs (d-PAHs) of the target analytes that are added to every sample, blank, quality control spike sample, and calibration solution. They are added to the water samples immediately after completing the flocculation step and transferring the water aliquot to the autosampler vial, and immediately after adding the calibration PAH solution to water calibration standards, but before SPME extraction. The internal standards are used to calculate the concentration of the target analytes or estimated detection limits.

3.1.11 *laboratory blank*—see *method blank*.

3.1.12 *method blank*—an aliquot of reagent water that is extracted and analyzed along with the samples to monitor for laboratory contamination. Blanks should consistently meet concentrations at or less than one-third of the performance limits for individual PAHs stated in Table 2. Alternatively, if the PAH concentrations calculated from the water blank immediately preceding the test samples are <20 % of the test sample concentrations, the blank is acceptable.

3.1.13 *low calibration level (LCL)*—the level at which the entire analytical system must give a recognizable signal and acceptable calibration point for the analyte. It is equivalent to the concentration of the lowest calibration standard assuming that all method-specified sample weights, volumes, and cleanup procedures have been employed.

3.1.14 *high or upper calibration level (UCL)*—the concentration or mass of analyte in the sample that corresponds to the highest calibration level in the initial calibration. It is equivalent to the concentration of the highest calibration standard, assuming that all method-specified sample weights, volumes, and cleanup procedures have been employed.

3.1.15 *MS*—mass spectrometer or mass spectrometry.

3.1.16 *PAH*—polycyclic aromatic hydrocarbon, or alternately, polynuclear aromatic hydrocarbon.

TABLE 2 Toxic Unit Factors and Performance Limits[A]

Analyte	Added d-PAH Internal Standard	d-PAH Internal Std. for Calculation	SPME-GC/MS RRF versus Parent	Conc. for One Toxic Unit, C_{tu}, (ng/mL)	Performance Limit (ng/mL)
Naphthalene	A	A	1.00	193.47	5.69
2-Methylnaphthalene		B	1.00	81.69	2.40
1-Methylnaphthalene	B	B	1.00	81.69	2.40
C2-Naphthalenes		A	1.44	30.24	0.89
C3-Naphthalenes		A	0.88	11.10	0.33
C4-Naphthalenes		A	0.71	4.05	0.12
Acenaphthylene		C	1.00	306.85	9.03
Acenaphthene	C	C	1.00	55.85	1.64
Fluorene	D	D	1.00	39.30	1.16
C1-Fluorenes		D	0.73	13.99	0.41
C2-Fluorenes		D	0.59	5.30	0.16
C3-Fluorenes		D	0.35	1.92	0.06
Phenanthrene	E	E	1.00	19.13	0.56
Anthracene		E	1.00	20.72	0.61
C1-Phenanthrenes/Anthracenes		E	0.57	7.44	0.22
C2-Phenanthrenes/Anthracenes		E	0.32	3.20	0.09
C3-Phenanthrenes/Anthracenes		E	0.29	1.26	0.04
C4-Phenanthrenes/Anthracenes		E	0.12	0.56	0.02
Fluoranthene	F	F	1.00	7.11	0.21
Pyrene	G	G	1.00	10.11	0.30
C1-Fluoranthenes/Pyrenes		G	0.51	4.89	0.14
Benz[a]anthracene		H	1.00	2.23	0.066
Chrysene	H	H	1.00	2.04	0.060
C1-Chrysenes/Benz[a]anthracenes		H	0.62	0.86	0.025

[A] From Hawthorne, S. B., Grabanski, C.B., Miller, D .J., and Kreitinger, J. P., "Solid Phase Microextraction Measurement of Parent and Alkyl Polycyclic Aromatic Hydrocarbons in Milliliter Sediment Pore Water Samples and Determination of K_{DOC} Values," *Environmental Science Technology*, 39, 2005, pp. 2795-2803.

3.1.17 *percent difference (%D)*—the difference between the analyzed concentration and expected concentration, expressed as a percentage of the expected concentration.

3.1.18 *relative response factor (RRF)*—the empirically determined ratio between the area ratio (analyte to internal standard) and the unit mass of analyte in the calibration standard (area ratio/ng) for available alkyl PAHs in a given homolog and their parent PAH.

3.1.19 *selected ion monitoring (SIM)*—a mode of operation for the mass spectrometer in which specific ions are monitored. This mode of operation differs from the full scan mode, in which the MS acquires all ions within a range. Because the spectrometer is monitoring fewer ions in the SIM mode, more acquisition (dwell) time is possible for each ion. This results in greater instrument sensitivity for the selected ions. Spectral scanning and library searching, used for tentatively identified compounds, are not supported in this mode.

3.1.20 *signal-to-noise ratio*—the ratio of the mass spectrometer response of a GC peak to the background noise signal.

4. Summary of Test Method

4.1 Either the use of an autosampler, or a manual approach can be used to perform the SPME extraction and the subsequent injection of collected analytes into the GC/MS. An autosampler (Leap Technologies Compi-Pal or equivalent) is much preferred over the manual method because: *(1)* the autosampler yields lower and more reproducible blanks, *(2)* the manual method requires the use of a stir bar that can cause sample cross-contamination, *(3)* the manual method is highly labor-intensive and requires multiple timed manipulations per analysis leading to operator fatigue and resultant errors, and *(4)* the autosampler reduces the technician time required to prepare samples for a 24-h run sequence to approximately 3 h, while the manual method requires 24-h operator attendance. Therefore, the method procedures are written assuming the use of an autosampler, with modifications to the autosampler procedures listed for the manual method.

AUTOSAMPLER METHOD

4.2 *Pore Water Separation and Preparation*—The pore water is separated from wet sediment samples by centrifugation and supernatant collection. Colloids are removed from the separated pore water samples by flocculation with aluminum potassium sulfate (alum) and sodium hydroxide as described in Hawthorne et al.[4] A second flocculation and centrifugation, followed by supernatant collection completes the colloid removal. The prepared pore water samples are then split into the required number of replicate aliquots (1.5 mL each) and placed into silanized glass autosampler vials. The 8 perdeuterated PAH internal standards (d-PAHs) are then added immediately. All of the water preparation steps beginning with the centrifugation and ending with the addition of d-PAH internal standards should be conducted continuously and in the minimum amount of time possible.

4.2.1 The SPME fiber should be cleaned at the beginning of each sampling set (and after very contaminated samples) for 1 h by placing in the cleaning chamber under helium flow at 320°C. This can conveniently be performed while the pore waters are being prepared.

4.3 *Solid-Phase Microextraction*—The SPME extraction of the pore water samples is performed using a commercially available (available from Sigma-Aldrich, formerly Supleco, or equivalent) 7 µm film thickness polydimethylsiloxane (PDMS)-coated fused silica fiber for 30 min while the water sample is mixed by the precession of the autosampler mixing chamber at a rate of 250 revolutions per minute. The target

PAHs and d-PAH internal standards adsorb to the nonpolar PDMS phase at equivalent rates. The use of the d-PAHs (that is, isotopic dilution) to quantitate the target PAHs compensates for variations in equilibrium partitioning and kinetics.

4.4 *GC/MS SIM Analysis*—Following the sorption period, the SPME fiber is immediately desorbed to a GC/MS injection port in the splitless mode at 320°C for 5 min. The GC/MS system specified uses a 60 m narrow-bore (250 µm ID) HP5-MS or equivalent capillary column to achieve high resolution for PAHs. Following the 5 min desorption period, the SPME fiber is inserted into the cleaning port and additionally cleaned for 15 min under helium flow at 320°C. At the end of the cleaning period, sorption of the next water sample is begun.

MANUAL METHOD

4.5 *Alternate Procedures for Manual Method*—Samples are prepared as for the autosampler method, except that a small Teflon-coated stir bar is placed in the silanized autosampler vial prior to adding the water and d-PAH internal standard solution. A new stir bar should be used for each sample, calibration standard, and blank to avoid cross-contamination caused by carryover on the stir bar. To perform the SPME step, the vial is set on a stir plate and the stirring rate adjusted so that no large vortex is formed. The SPME fiber should be inserted into the water so that the entire 1-cm active length is exposed to the water sample, but not so low that the fiber comes into contact with the stir bar or that the metal needle sheath contacts the water. All time sequences should be the same as specified for the autosampler method. A spare GC split/splitless injection port at 320°C and under helium flow can be used for the 15-min cleaning step between samples as well as for the initial 1-h cleaning step at the beginning of each experimental day.

4.6 The mass spectrometer is operated in the SIM mode for the molecular ions of the target PAHs and d-PAHs to achieve low limits of detection. Analyte concentrations are quantified by three methods:

4.6.1 PAHs for which an exact deuterated analog is included in the internal standard mix are quantified by isotope dilution.

4.6.2 Parent PAHs (that is, unsubstituted PAHs) for which an exact deuterated analog is not included in the internal standard mix are quantified by reference to a deuterated analog of a PAH with the same number of rings as the analyte.

4.6.3 Alkyl PAHs are quantified using the experimentally and empirically-determined relative response factors from Hawthorne et al.[4] and as shown in Table 1. The laboratory may use updated response factors, if additional alkyl PAH standards become commercially available. However, the laboratory must correct for purities of less than 98 %.

4.7 *Conversion of Quantified Concentration to Toxic Units*—The USEPA narcosis model predicts toxicity to benthic organisms if the sum of the toxic units calculated for all "34 PAHs" measured in a pore water sample is greater than or equal to 1. For this reason, the performance limits required for the individual PAH measurements were defined as the concentration of an individual PAH that would yield 1/34 of a toxic unit. See Table 2. This distribution reflects the relative concentrations of PAHs expected to be found in pore water because the lower molecular weight PAHs are more soluble and have lower organic carbon partition coefficients (Koc), and reflects the lower partitioning of lower molecular weight PAHs to the receptor organism since they have smaller octanol/water coefficients (Kow). The performance limits are essentially benchmarks to ensure that the adequate sensitivity is achieved to predict toxicity.

5. Significance and Use

5.1 This method directly determines the concentrations of dissolved PAH concentrations in environmental sediment pore water samples. The method is important from an environmental regulatory perspective because it can achieve the analytical sensitivities to meet the goals of the USEPA narcosis model for protecting benthic organisms in PAH contaminated sediments. Regulatory methods using solvent extraction have not achieved the wide calibration ranges from nanograms to milligrams per litre and the required levels of detection in the nanogram-per-litre range. In addition, conventional solvent extraction methods require large aliquot volumes (litre or larger), use of large volumes of organic solvents, and filtration to generate the pore water. This approach entails the storage and processing of large volumes of sediment samples and loss of low molecular weight PAHs in the filtration and solvent evaporation steps.

5.2 This method can be used to determine nanogram to milligram per litre PAH concentrations in pore water. Small volumes of pore water are required for SPME extraction, only 1.5 mL per determination and virtually no solvent extraction waste is generated.

6. Interferences

6.1 Non-target hydrocarbons can cause peaks on selected ion current profiles (SICPs) intended for other PAHs. Pattern recognition must be employed for identifying interfering peaks, and peak series that should not be considered for the homolog or target PAH under consideration. Analysts should be intimately familiar with both parent and alkyl PAH analyses in complex environmental samples. Representative samples having higher PAH concentrations should periodically be analyzed by full scan GC/MS so that pattern recognition of alkyl PAHs (and interfering species) can be verified by their full mass spectra. This procedure is particularly important for newer operators.

6.2 Solvents, reagents, glassware and other sample processing hardware may yield discrete artifacts or elevated baselines that may cause misinterpretation of the chromatographic data. All of these materials must be demonstrated to be free from interferences under the conditions of analysis by performing laboratory method blanks. Analysts should avoid using PVC gloves, powdered gloves, or gloves with measurable levels of phthalates.

NOTE 1—The use of high purity reagents and solvents helps minimize interference problems.

7. Apparatus

7.1 *Centrifuge*, capable of sustaining 1000 g with cups for securing 40 mL and 20 mL vials.

7.2 *SPME Fiber Holder*, compatible with 7-µm SPME fiber and compatible with either the autosampler or the manual method.

TABLE 3 Primary Material Hazards

Material	Hazards	Exposure Limit[A]	Signs and Symptoms of Exposure
Alum (Aluminum Potassium Sulfate)	Irritant	2 mg/M^3 TWA	May cause skin irritation, especially under repeated or prolonged contact, or when moisture is present. May irritate or burn the eyes. Dust or mist inhalation at levels above the TLV may cause irritation to the respiratory tract. May irritate the gastrointestional tract.
Acetone	Flammable	1000 ppm-TWA	Inhalation of vapors irritates the respiratory tract. May cause coughing, dizziness, dullness, and headache.
Dichloromethane (DCM)	Carcinogen, Irritant	25 ppm-TWA, 125 ppm-STEL	Causes irritation to respiratory tract. Has a strong narcotic effect with symptoms of mental confusion, light-headedness, fatigue, nausea, vomiting and headache. Causes irritation, redness and pain to the skin and eyes. Prolonged contact can cause burns. Liquid degreases the skin. May be absorbed through skin.
Sodium Hydroxide	Corrosive	2 mg/M^3 TWA	Causes skin irritation, chemical burns, permanent injury or scarring, and blindness. Vinegar is a mild acid that will neutralize lye if it were to make contact with the skin. Harmful if inhaled or ingested. Causes Sore throat, cough labored breathing, shortness of breath, and abdominal pain. Symptoms may be delayed.

[A] Exposure limit refers to the OSHA regulatory exposure limit.

7.3 *SPME Fibers*, 7-µm diameter, coated with polydimethylsiloxane (PDMS).

7.4 *PTFE Coated Stir Bars (Stir Fleas)*, of a size effective for stirring 1.5 mL water without vortexing (for manual method only).

7.5 *Magnetic Stir Plate (for manual method only)*.

7.6 *SPME Holder Stand (for manual method only) or GC/MS Autosampler*, capable of SPME extraction and injection.

7.7 *Cleaning Port*, capable of purging SPME fibers in a helium-swept atmosphere at 320°C.

7.8 *GC/MS Analysis*:

7.8.1 *Gas Chromatograph* shall have split/splitless injection port for capillary column, temperature program with isothermal hold.

7.8.2 *GC Column*, 60 mm × 0.25 mm ID × 25 µm film thickness HP5-MS or equivalent.

7.8.3 *Inlet Liner*, 2 mm ID silanized glass.

7.8.4 *GC Inlet*, 320°C, splitless mode.

7.8.5 *Oven Program*—Isothermal 5 min hold at 40°C. Ramp at 50°C/min to 110°C, followed by a temperature ramp of 12°C/min to 320°C (hold for 10 min).

7.8.6 *Mass Spectrometer*—Electron impact ionization with the ionization energy optimized for best instrument sensitivity (typically 70 eV), stability and signal to noise ratio. Shall be capable of repetitively selectively monitoring at least 12 m/z during a period of approximately 1 s and shall meet all manufacturers' specifications.

7.8.7 *GC/MS Interface*—The mass spectrometer (MS) shall be interfaced to the GC such that the end of the capillary column terminates within 1 cm of the ion source but does not intercept the electron or ion beam.

7.8.8 *Data System*, capable of collecting, recording, and storing MS data.

8. Reagents and Materials

8.1 *Purity of Reagents*—Reagent grade chemicals must be used in all tests. Unless otherwise indicated, it is intended that all reagents shall conform to the specifications of the Committee on Analytical Reagents of the American Chemical Society, where such specifications are available.[7]

8.2 *Purity of Water*—Unless otherwise indicated, references to water shall be understood to mean reagent water that meets the purity specifications of Type I or Type II water, presented in Specification D 1193.

8.3 *40 mL Vials*, with Teflon-lined caps.

8.4 *20 mL Vials*, with Teflon-lined caps.

8.5 *Silanized 2.0 mL Autosampler Vials*.

8.6 *Internal Standard Stock Solution*—A dichloromethane solution of d-PAH internal standards used for preparing spiking solutions by dilution into acetone (see 12.2).

8.7 *Internal Standard Spiking Solution*—A dilution of the internal standard stock solution in acetone used to spike d-PAH internal standards into all sample, calibration, and blank water vials.

8.8 *Calibration Stock Solution*—A dichloromethane solution of PAHs used for preparing calibration standards (see 12.2).

8.9 *Calibration Spiking Solutions*—A series of solutions prepared by diluting the calibration stock solution with acetone (see 12.2).

8.10 *Calibration Standards*—Prepared by adding internal standard and calibration spiking solutions in reagent water (see 12.2).

8.11 *Acetone*.

8.12 *Dichloromethane* (DCM).

8.13 *Sodium Hydroxide* (NaOH).

8.14 *Aluminum Potassium Sulfate Dodecahydrate* ($AlK(SO_4)_2 \cdot 12H_2O$).

8.15 *Alum Solution*—Add 20 g ($AlK(SO_4)_2 \cdot 12H_2O$) to 80 mL reagent water.

9. Hazards

9.1 The effluents of sample splitters for the gas chromatograph and roughing pumps on the mass spectrometer must be

[7] *Reagent Chemicals, American Chemical Society Specifications*, American Chemical Society, Washington, DC. For suggestions on the testing of reagents not listed by the American Chemical Society, see *Analar Standards for Laboratory Chemicals,* BDH Ltd., Poole, Dorset, U.K., and the *United States Pharmacopeia and National Formulary,* U.S. Pharmacopeial Convention, Inc. (USPC), Rockville, MD.

vented to the laboratory hood exhaust system or must pass through an activated charcoal filter.

9.2 *Primary Materials Used*—The table contains a summary of the primary hazards listed in the MSDS. A complete list of materials used in the method can be found in the reagents and materials section. Practitioners must review the information in the MSDS for each material before using it for the first time or when there are major changes to the MSDS.

10. Sampling and Sample Preservation

10.1 Collect the sediment sample in accordance with Practices D 3370 and Specification D 1192, as applicable.

10.2 Prior to shipment, the samples should be mixed well. Sieve the slurry of sediment and site water through a 2-mm screen to remove debris. If the sieved slurry is to be stored or shipped before use, store in 250 mL to 1 L jars with PTFE-lined lids. Great care must be taken to clean the lid of the jar before capping with the lid to avoid leakage of the water during shipment.

10.3 Ship in an ice chest with adequate ice to maintain 0 to 6°C. Store at the laboratory in the dark at 0 to 6°C.

11. Preparation of Apparatus

11.1 Set up the GC system using the following parameters.

11.1.1 GC Column Agilent HP-5MS column (0.25 µm film thickness, 0.25 mm ID) or equivalent.

11.1.2 Inlet liner 2-mm ID silanized glass.

11.1.3 GC Inlet 320°C, splitless mode.

11.1.4 *Oven Program*—Isothermal 5 min hold at 40°C. Ramp at 50°C/min to 110°C, followed by a temperature ramp of 12°C/min to 320°C. (Hold for 10 min.)

MS Quad Temperature	150°C, maximum 200°C
MS Source Temperature	230°C, maximum 250°C

11.1.5 Set up SIM Groups to monitor the quantitation and internal standard ions shown in Table 4. Each ion dwell time should be set at 25 ms. Twelve ions are monitored in each group.

NOTE 2—Some ions (for example, m/z 184.1 for C4 naphthalenes) are included in two ion groups to ensure that the target peaks are adequately monitored. Table 4 should be used with the chromatograms in Appendix X1 to aid the analyst in setting proper retention time windows and recognition of target and contaminant peaks, especially for the alkyl clusters.

12. Calibration

12.1 Determine the absolute and relative retention times of the first and last characteristic peak in each homolog with the aid of the examples in Appendix X1.

12.1.1 Set up a SIM program with the necessary ions to acquire all the alkyl-PAH homologs using the ion groups shown in Table 4 and 25 ms dwell time per ion.

12.1.2 Update the expected retention times in the method section of the quantitation software using the d-PAH internal standards of previous runs as relative retention time markers and the representative chromatograms in Appendix X1. Assure that the SIM windows for the homologs are set to at least 8 s before the first, and 30 s after the last characteristic peaks to assure coverage of the elution range.

12.2 *Analyze Initial Calibration*:

TABLE 4 SIM Ion Groups and Retention Time Windows

NOTE—Retention times must be verified by the user.

Analyte	SIM Ion Group	Target m/z	Retention Time (min)	
			Start	Stop
Naphthalene	1	128.1	7	17
2-Methylnaphthalene	1	142.1	7	17
1-Methylnaphthalene	1	142.1	7	17
C2-Naphthalenes	1	156.1	7	17
C3-Naphthalenes	1	170.1	7	17
C4-Naphthalenes	1,2	184.1	7	21
Acenaphthylene	1	152.1	7	17
Acenaphthene	1	154.1	7	17
Fluorene	1	166.1	7	17
C1-Fluorenes	2	180.1	17	21
C2-Fluorenes	2	194.1	17	21
C3-Fluorenes	2,3	208.1	17	25
Phenanthrene	2	178.1	17	21
Anthracene	2	178.1	17	21
C1-Phenanthrenes/Anthracenes	2	192.1	17	21
C2-Phenanthrenes/Anthracenes	2,3	206.1	17	30
C3-Phenanthrenes/Anthracenes	2,3	220.1	17	30
C4-Phenanthrenes/Anthracenes	3	234.1	21	30
Fluoranthene	2,3	202.1	17	30
Pyrene	2,3	202.1	17	30
C1-Fluoranthenes/pyrenes	3	216.1	21	30
Benz[a]anthracene	3	228.1	21	30
Chrysene	3	228.1	21	30
C1-Chrysenes	3	242.1	21	30
d-PAH Internal Standards				
Naphthalene-d8	1	136.1	7	17
1-Methylnaphthalene-d10	1	152.1	7	17
Acenaphthene-d10	1	164.1	7	17
Fluorene-d10	1	176.1	7	17
Phenanthrene-d10	2	188.1	17	21
Fluoranthene-d10	2,3	212.1	17	30
Pyrene-d10	2,3	212.1	17	30
Chrysene-d12	3	240.2	21	30

12.2.1 Prepare stock solutions of PAHs and internal standard stock solutions of d-PAHs at approximately the concentrations shown in Table 5. These concentrations were based on the PAH distributions previously determined in 120 sediment pore water samples. Stocks are prepared in DCM. Spiking solutions are prepared by dilution of intermediate stocks in acetone. For calibration solutions, spiking solutions are added to reagent water.

12.2.1.1 Prepare calibration standard spiking solutions. These are prepared by adding acetone to the stock to give the calibration solution concentrations (CS1–CS4), as described below:

(1) For CS1, take 5 µL stock to 100 mL in acetone.

(2) For CS2 take 50 µL to 100 mL in acetone.

(3) For CS3, take 25 µL to 10 mL in acetone.

(4) For CS4, take 100 µL to 10 mL in acetone.

12.2.1.2 Spike 4 µL of each calibration solution into 1.5 mL of reagent water to give a calibration series with the low calibration limits (LCLs) and upper calibration limits (UCLs) shown in Table 5. Spike 10 µL of internal standard spiking solution at the concentrations shown in Table 5 into each vial.

12.2.1.3 Extract and analyze the calibration series.

(1) Extract and analyze two water blank solutions.

(2) Extract and analyze the water calibration solutions, as described in 13.4 and 13.5. Begin with the CS1-spiked sample,

TABLE 5 Initial Calibration Standard Series

Analyte	DCM Stock Conc. mg/mL	LCL			UCL
		CS1	CS2	CS3	CS4
		ng/1.5 mL	ng/1.5 mL	ng/1.5 mL	ng/1.5 mL
Naphthalene	41.5	8.3	83	415	1660
1-Methylnaphthalene	23.9	4.78	47.8	239	956
2-Methylnaphthalene	20.42	4.084	40.84	204.2	816.8
Acenaphthylene	9.02	1.804	18.04	90.2	360.8
Acenaphthene	11	2.2	22	110	440
Fluorene	7.55	1.51	15.1	75.5	302
Anthracene	0.6	0.12	1.2	6	24
Phenanthrene	5.5	1.1	11	55	220
Fluoranthene	2.11	0.422	4.22	21.1	84.4
Pyrene	1.8	0.36	3.6	18	72
Benz[a]anthracene	0.08	0.016	0.16	0.8	3.2
Chrysene	0.03	0.006	0.06	0.3	1.2
Deuterated Analogs of Mix A Compounds	Stock Solution	CS1	CS2	CS3	CS4
Naphthalene-d8	5	50.0	50.0	50.0	50.0
1-Methylnaphthalene-d10	6	60.0	60.0	60.0	60.0
Acenaphthene-d10	1.23	12.3	12.3	12.3	12.3
Fluorene-d10	1.2	12.0	12.0	12.0	12.0
Phenanthrene-d10	0.96	9.6	9.6	9.6	9.6
Fluoranthene-d10	0.93	9.3	9.3	9.3	9.3
Pyrene-d10	0.84	8.4	8.4	8.4	8.4
Chrysene-d12	0.033	0.33	0.33	0.33	0.33

followed by sequentially more concentrated calibration standards. Follow by two water blanks.

12.2.1.4 Calculate the performance parameters for the calibration.

(1) Generate ion chromatograms for the masses listed in Table 4 that encompass the expected retention windows of the target analytes. Integrate the selected ion current profiles of the quantitation ions shown in the table. Integration of alkyl clusters should be as the total area of the cluster integrated from the baseline before the first peak in the cluster to the baseline after the last peak in the cluster peaks. Cluster peaks should never be integrated using the valley-to-valley method. The peak areas of non-target peaks (see Appendix X1) must be removed from the alkyl cluster peak area before any calculation.

(2) Calculate the area ratio (analyte peak area divided by internal standard peak area) per unit mass of analyte, using the area of the appropriate internal standard listed in Table 1. Quantitative calculations are based on a comparison of the area ratio per ng from the calibration and sample waters. The area ratio per ng is calculated for calibration runs by dividing the calibration peak area by the peak area of its most closely associate d-PAH internal standard (the deuterated parent PAH, in most cases), and dividing this result by the ng of the calibration PAH present in the vial (that is, its mass in the vial, not its concentration). Calibration standards are given in Table 5.

$$area\ ratio\ per\ ng\ (ar\ rat/ng) = \frac{[(peak\ area\ cal.\ std)/(peak\ area\ d{-}PAH)]}{(mass\ of\ std\ in\ cal\ vial)} \quad (1)$$

(3) Calculate the mean ar rat/ng. The mean relative response factor for these duplicate daily calibration standards should agree with those from the 4-point (or 3-point) standard curve within 20 % for the two and three-ring PAHs, and within 25 % for the four-ring PAHs. No sample data will be reported if these calibration criteria are not met. Calculate the mean area ratio/ng and the standard deviation of the relative response factors for each calibration standard solution using the following equations:

$$\overline{ar\ rat/ng} = \frac{1}{n}\sum_{i=1}^{n}(ar\ rat/ng)_i \quad (2)$$

where:

$(ar\ rat/ng)_i$ = ar rat/ng calculated for calibration solution "i" using the equation in 12.2.1.4*(2)*, and

n = number of calibration points in the curve.

(4) Calculate the percent relative standard deviation:

$$\%RSD = \frac{SD}{\overline{ar\ rat/ng}} \times 100 \quad (3)$$

where:

$\overline{ar\ rat/ng}$ = mean ar rat/ng calculated above, and

SD = sample standard deviation of the replicate area rat/ng values used to calculate the mean ar rat/ng.

12.3 *Criteria for Acceptable Initial Calibration*—Prior to analyzing any samples, the standard curves are prepared using the identical analysis procedures as used for sample waters. To be acceptable, the linearity of each PAH standard curve should be $r^2 > 0.99$, and the relative response factor per ng for each concentration should show a relative standard deviation of <25 % for two- to three-ring PAHs, and <30 % for four-ring PAHs. See Section 16. If acceptable initial calibration is not achieved, identify the root cause, perform corrective action, and repeat the initial calibration. If the root cause can be traced to an abnormal disruption of an individual acquisition (for example, injector malfunction) repeat the individual analysis and recalculate the percent relative standard deviation. If the

calibration is acceptable, document the problem and proceed; otherwise repeat the initial calibration.

12.3.1 Because of the large range of calibration concentrations required, the wide range of water solubilities of the individual PAHs, and the desire to require only one stock calibration solution, some PAHs may only have a three point linear calibration curve that meets the above criteria. This is most likely to occur for the higher molecular weight PAHs, because the dilution of lowest calibration standard is likely to be below detection limits for many labs (and is also below the required detection limits needed for the method, so it does not negatively impact the analyses). In such cases, the lowest calibration standard is ignored, and the "J" level adjusted appropriately. Less frequently, the highest concentrations of the lowest molecular weight PAHs may exceed the linear dynamic range of the GC/MS response. In such cases the laboratory should investigate lowering the MS multiplier voltage to autotune voltage or slightly below and rerun the calibration curve. If the highest calibration standard still exceeds the detector linearity, it is acceptable to reject the highest concentration for those specific PAHs (and adjust the "E" value accordingly), as long as a minimum of a three-point standard curve is generated for each PAH.

12.3.1.1 It is recommended that a 4-point (or 3-point) initial calibration be established every two weeks, when continuing calibration criteria are not met, or when service is performed on the GC/MS instrument system.

12.3.2 The signal to noise ratio (S/N) for the GC signals present in every selected ion current profile (SICP) must be ≥10:1 for the labeled internal standards and unlabeled calibration compounds.

12.4 *Calibration Verification*—Continuing calibration is performed daily at the beginning of a 24-h period. The injection of the first continuing calibration begins the 24-h window, within which all pore water samples must be injected. Duplicate daily standards are analyzed.

12.4.1 Into 1.5 mL of reagent water, add 4 µL of the CS3 spiking solution and 10 µL of the d-PAH internal standards.

12.4.2 Analyze duplicate vials of the Calibration Standard Solution CS3. Use the same data acquisition parameters as those used during the initial calibration. Check for GC resolution and peak shape. If peak shape or retention times are unacceptable, perform column and injector maintenance. If this fails to correct the problem, the column must be replaced and the calibration repeated.

12.4.3 *Criteria for Acceptable Daily Calibration Check*—The criteria listed below for acceptable calibration must be met at the beginning of each 24-h period that samples are analyzed. The mean relative response factor for these duplicate daily calibration standards should agree with those from the 4-point (or 3-point) standard curve within 20 % for the two- and three-ring PAHs, and within 25 % for the four-ring PAHs. No sample data will be reported if these calibration criteria are not met. If the continuing calibration criteria are not met, identify the root cause, perform corrective action and repeat the continuing calibration. If the second consecutive continuing calibration does not meet acceptance criteria, additional corrective action must be performed.

12.4.4 The signal to noise ratio (S/N) for the GC signals present in every selected ion current profile (SICP) must be ≥10:1 for the labeled internal standards and unlabeled calibration compounds.

12.5 *Method Blanks*—Method blanks are prepared and analyzed daily in duplicate following the continuing calibration and between analysis of replicate sets of the same pore water sample. See 12.5.2.2.

12.5.1 For each method blank, add 10 µL of the d-PAH internal standards solution into 1.5 mL of reagent water.

12.5.2 Two types of sources of background PAHs must be considered. For the higher molecular weight PAHs, typical GC/MS criteria for signal to noise are appropriate, since their detection limits are normally controlled by GC/MS sensitivity. However, for lower molecular weight PAHs, atmospheric contaminants can cause significant background peaks, especially for low MW alkyl PAHs. This problem is most likely to be significant in urban areas impacted by atmospheric PAHs (for example, from diesel exhaust), and with laboratories using manual techniques, rather than the SPME autosampler.

12.5.2.1 *Background PAHs from Ambient Air*—Concentrations of each PAH in the water blanks should be calculated in the same manner as a sample. Should the blank prior to the subsequent pore water sample have detectable background concentrations more than ⅓ of the target detection limit given in Table 3, the analyses should not continue until the fiber is sufficiently cleaned as demonstrated by a clean water blank. The mean of the calculated concentrations of the PAHs in the blanks analyzed immediately before and immediately after sample pore waters should be subtracted from the sample pore water concentrations.

12.5.2.2 *Carryover from Highly Contaminated Samples*—Carryover blanks are analyzed between each new pore water sample (not including replicates). Significant carryover can occur if the previous sample was highly contaminated. Should the blank prior to the subsequent pore water sample have detectable background concentrations more than ⅓ of the target detection limit, the analyses should not continue until the fiber is sufficiently cleaned as demonstrated by a clean water blank. Alternatively, if the concentrations determined in the blanks are less than 20 % of those found in the related sample, the data can be accepted.

13. Procedure

13.1 At the laboratory, store samples and extracts in the dark at 0 to 6°C.

13.2 *Holding Times*:

13.2.1 Pore waters must be generated within 28 days of sediment sample collection.

13.2.2 Pore waters must be generated and flocculated as quickly as possible, and then immediately spiked with 10 µL of d-PAH solution.

13.2.3 Solid phase micro-extraction must be completed within 24 h of flocculation.

13.3 *Generation of Pore Water*:

13.3.1 Stir the slurry and transfer approximately 40 mL (containing a solids and liquids in proportion to the slurry provided) to a clean 40 mL vial. Cap the vial with a PTFE-lined cap. Place the vials in a centrifuge. Spin for 30 min at 1000 g.

TABLE 6 Example of a 24-h Analytical Sequence[A]

Example Analytical Sequence					
Run Type	Minutes	Cumulative Minutes to Start	Cumulative Minutes to End	Cumulative Hours to Start[A]	Cumulative Hours to End
Standard	50	0	50	0.0	0.8
Standard	50	50	100	0.8	1.7
Blank	50	100	150	1.7	2.5
Blank	50	150	200	2.5	3.3
Sample	50	200	250	3.3	4.2
Sample	50	250	300	4.2	5.0
Blank	50	300	350	5.0	5.8
Blank	50	350	400	5.8	6.7
Sample	50	400	450	6.7	7.5
Sample	50	450	500	7.5	8.3
Blank	50	500	550	8.3	9.2
Blank	50	550	600	9.2	10.0
Sample	50	600	650	10.0	10.8
Sample	50	650	700	10.8	11.7
Blank	50	700	750	11.7	12.5
Blank	50	750	800	12.5	13.3
Sample	50	800	850	13.3	14.2
Sample	50	850	900	14.2	15.0
Blank	50	900	950	15.0	15.8
Blank	50	950	1000	15.8	16.7
Sample	50	1000	1050	16.7	17.5
Sample	50	1050	1100	17.5	18.3
Blank	50	1100	1150	18.3	19.2

[A] The last pore water sample must be injected within 24 h of the flocculation step (that is, the value for cumulative hours to start must be ≤24).

Using a new, graduated serological pipette, transfer 10 mL of the supernatant to a new 20 mL vial.

13.3.2 *Flocculation of Pore Water*—Flocculation must be performed no more than 24 h prior to extraction.

13.3.2.1 If a flocculation blank is to be analyzed, create the blank by placing 10 mL of reagent water in clean a 40 mL vial. Process this blank along with pore water samples.

13.3.2.2 Add the working alum solution (see Section 9) to each vial of pore water (and QC samples). The volume of the alum solution should be 1/40th of the sample volume. After the addition, swirl the vial for several rotations to incorporate the solution.

13.3.2.3 Add 3 to 5 drops of NaOH working solution (see Section 9) to each vial. Swirl to incorporate the NaOH.

13.3.2.4 Shake the vial for 15 s.

13.3.2.5 Centrifuge for 30 min at 1000 g.

13.3.2.6 Collect the supernatant into a clean 20 mL vial.

13.3.2.7 Repeat 13.3.2.2 through 13.3.2.6 once.

13.3.2.8 Immediately transfer 1.5 mL aliquots to new silanized autosampler vials and immediately add the internal standard solution as described below. Vials are weighed before and after adding the water sample to determine the exact sample water mass.

13.4 *Extraction and Analysis of Flocculated Pore Water*:

13.4.1 Split the prepared pore water samples into the required number of replicate samples, placing 1.5 mL aliquots of each into a new silanized glass autosampler vial. For QC samples, add 1.5 mL of reagent water.

NOTE 3—The SPME fiber should be cleaned at the beginning of each sampling set (and after very contaminated samples) for 1 h by placing in the cleaning chamber under helium flow at 320°C. This can conveniently be performed while the pore waters are being prepared.

13.4.2 Immediately add 10 µL of the d-PAH solution to each sample and QC sample.

NOTE 4—All of the water preparation steps beginning with the centrifugation and ending with the addition of d-PAH internal standards should be conducted continuously and in the minimum amount of time possible.

13.4.3 Load the autosampler following the recommended analytical sequence in Table 6. Verify the sequence against documented sequence following the loading process.

13.5 The recommended analytical sequence described in Table 6 is based on a 24-h "clock."

13.5.1 Two calibration verification standards are analyzed (122 min). The sequence begins with analysis of the first continuing calibration standard.

13.5.2 Analyze two method blanks (61 min each).

13.5.3 Analyze pore water samples (in duplicate at a minimum) (61 min each).

14. Data Analysis and Calculations

14.1 Generate ion chromatograms for the masses listed in Table 4 that encompass the expected retention windows of the target analytes (see Appendix X1). Integrate the selected ion current profiles of the quantitation ions shown in the table.

14.1.1 *Qualitative Identification Criteria for Individual Analytes*—For a gas chromatographic peak to be identified as a target analyte, it must meet all of the following criteria:

14.1.1.1 The quantitation ion must be present, with a signal-to-noise ratio of at least 3:1 for environmental samples.

14.1.1.2 The relative retention time (RRT) of the parent PAHs (and the 2 and 1-methylnaphthalene compounds) compared to the RRT for the labeled-standards must be within ±3 s of the relative retention times obtained from the continuing calibration (or initial calibration if this applies). Alkyl clusters must be identified based on their relative retention times to the parent PAHs and related d-PAHs, and also by observation of their characteristic fingerprints by an experienced analyst.

14.1.2 *Qualitative Identification Criteria for Total Homolog Groups* (for example, total C2 or C3 alkylnaphthalenes)—Integration of the alkyl PAHs requires hands-on labor from a highly experienced analyst. Retention time windows, like those used for the parent PAHs are inadequate for identifying alkyl clusters (that can be minutes wide). Proper identification of alkyl clusters is critical, as is the proper identification of non-target species that occur at the same nominal mass. Mental pattern recognition must be used to avoid including non-target species that may occur at the same mass and retention time window as the target alkyl PAHs. All alkyl clusters should be integrated baseline to baseline to sum the total area of the cluster (adjusting the baseline for detector drift), but not valley to valley. Manual control of the integration is required for alkyl clusters.

14.1.2.1 Representative selected ion chromatograms from coal tar contaminated sediment pore water for all target species are shown in Appendix X1. The top chromatogram on each page is the d-PAH internal standard used for the parent and alkyl PAHs associated with that parent. For example, the first page shows d8-naphthalene (m/z 136) followed by naphthalene (m/z 128), the two methylnaphthalene isomers (m/z 142), the C2 naphthalene cluster (m/z 156), the C3-naphthalene cluster (m/z 170), and the C4 naphthalene cluster (m/z 184). The chromatogram also shows a typical interference that occurs in sediments for the C4-naphthalene cluster, that is, the dibenzothiophene isomers that occur in the same selected ion chromatogram as the C4-naphthalene cluster. These interfering dibenzothiopenes are crossed out, and the correct cluster for integration (based on full scan analyses of several different contaminated sediment pore waters) are indicated by brackets. Similar designations are used to indicate common interfering peaks and the correct target species in the subsequent chromatograms.

14.1.3 The retention time (RT) of the analyte must be no more than 5 s before the expected RT of the first isomer in the homolog, based on the continuing windowing solution analysis.

14.1.4 The retention time (RT) of the analyte must be no more than 5 s after the expected RT of the last isomer in the homolog, based on the continuing windowing solution analysis.

14.2 *Quantitation for Target Analytes*:

14.2.1 Sample water concentrations are calculated by dividing the peak area of the sample peak by the peak area of its d-PAH internal standard, and then dividing the result by the calibration area ratio per ng, and dividing that result by the sample water weight.

$$Concentration\ (ng/mL) = \frac{(area\ sample\ peak)/(area\ d-PAH\ peak)}{(area\ ratio\ per\ ng\ cal.\ std) \times (sample\ weight)} \quad (4)$$

14.2.2 The mean calibration area ratio per ng values from the daily calibration runs is used for sample concentration calculations (assuming QA/QC checks with the full calibration curve meet criteria).

14.2.3 The concentrations of alkyl PAH clusters are based on the calibration response of their parent PAH as adjusted for the relative response factor (rrf) for that cluster of species (including SPME and GC/MS responses) taken from Table 1. Thus, the concentrations of alkyl clusters are calculated by:

$$Concentration\ (ng/mL) = \frac{(area\ sample\ cluster)/(area\ d-PAH\ peak)}{(area\ ratio\ per\ ng\ parent\ cal\ std) \times (sample\ weight)} \quad (5)$$

NOTE 5—The two methylnaphthalene isomers are individual alkyl peaks (not clusters as in all other alkyl cases) and are treated as parent PAHs in the calculations.

14.2.4 If no peaks are present at a signal to noise value ≥3 to 1 in the region of the ion chromatogram where the compounds of interest are expected to elute, report the result as "Not Detected" (that is, ND) at the reporting limit.

14.2.5 Depending on project objectives, the results may be reported to TDLs or estimated detection limits (EDLs).

14.2.5.1 If project-specific guidance requires analysis-specific EDLs, calculate the detection limit for that compound according to the following equation:

$$Estimated\ Detection\ Limit = \frac{N \times 2.5}{His \times (ar\ rat/ng)} \quad (6)$$

where:

N = height of peak to peak noise of quantitation ion signal in the region of the ion chromatogram where the compound of interest is expected to elute,

His = peak height of quantitation ion for appropriate internal standard, and

ar rat/ng = mean ar rat/ng of compound obtained during daily calibration.

14.2.5.2 If project-specific guidance requires total toxic units (TTU) to be reported, calculate the detection limit for that compound according to the following equations:

$$TU_c = Ctu \times result(ng/mL)^{-1} \quad (7)$$

$$Total\ Toxic\ Units\ (TTU) = \sum_{1}^{34} TU_c \quad (8)$$

where:

TU_c = toxic unit concentration for each individual compound or homolog (ng/mL),

Ctu = concentration for one toxic unit (ng/mL), see Table 2,

result = individual pore water result for a compound or homolog (ng/mL), and

TTU = total toxic units for all 34 compounds and homologs.

14.2.6 Flag all compound results in the sample which were estimated below the lowest calibration level with a "J" qualifier.

14.2.7 Flag all compound results in the sample which were estimated above the upper calibration level with an "E" qualifier.

15. Precision and Bias

15.1 *Single Analyst Precision Statement*:

15.1.1 The recommendations of the ASTM task group members were followed in performing the single-laboratory study. Three environmental sediment samples were selected from archived sediments to represent low, medium, and high

TABLE 7 Precision Statement for SPME Pore Water PAHs

Target Analyte	Statistic/Parameter	Study Pore Water Samples		
		HP-24	HP-3	HP-4
		Low	Medium	High
Naphthalene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND[A]	130.9	975.3
	Single Operator Std. Deviation (So)		4.2	42.6
	Relative Standard Deviation (%)		3.2	4.4
2-Methylnaphthalene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	20.2	245.4
	Single Operator Std. Deviation (So)		0.64	9.89
	Relative Standard Deviation (%)		3.2	4.0
1-Methylnaphthalene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	81.7	209.6
	Single Operator Std. Deviation (So)		2.4	7.1
	Relative Standard Deviation (%)		3.0	3.4
C2-Naphthalenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.33	125.4	324.2
	Single Operator Std. Deviation (So)	0.0259	8.61	23.7
	Relative Standard Deviation (%)	7.8	6.9	7.3
C3-Naphthalenes	Number of Retained Values	7	7	6
	Mean Recovery (ng/mL)	0.41	124.9	212.5
	Single Operator Std. Deviation (So)	0.029	12.7	5.99
	Relative Standard Deviation (%)	7.1	10.2	2.8
C4-Naphthalenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.14	44.6	53.0
	Single Operator Std. Deviation (So)	0.025	6.05	5.3
	Relative Standard Deviation (%)	17.7	13.6	10.0
Acenaphthylene	Number of Retained Values	7	7	6
	Mean Recovery (ng/mL)	ND	0.16	7.52
	Single Operator Std. Deviation (So)		0.020	0.09
	Relative Standard Deviation (%)		12.5	1.3
Acenaphthene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.21	44.1	84.8
	Single Operator Std. Deviation (So)	0.0125	1.28	2.79
	Relative Standard Deviation (%)	6.1	2.9	3.3
Fluorene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.11	23.2	31.6
	Single Operator Std. Deviation (So)	0.0071	0.75	1.48
	Relative Standard Deviation (%)	6.7	3.2	4.7
C1-Fluorenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.11	22.4	25.8
	Single Operator Std. Deviation (So)	0.011	0.86	1.50
	Relative Standard Deviation (%)	10	3.8	5.8
C2-Fluorenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	12.7	16.1
	Single Operator Std. Deviation (So)		0.88	1.85
	Relative Standard Deviation (%)		6.9	11.5
C3-Fluorenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	ND	ND
	Single Operator Std. Deviation (So)			
	Relative Standard Deviation (%)			
Phenanthrene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.1	31.3	39.2
	Single Operator Std. Deviation (So)	0.0069	1.84	3.16
	Relative Standard Deviation (%)	6.8	5.9	8.1
Anthracene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.03	6.2	8.2
	Single Operator Std. Deviation (So)	0.0007	0.37	0.72
	Relative Standard Deviation (%)	2.6	5.9	8.9
C1-phenanthrenes/anthracenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.13	31.9	45.2
	Single Operator Std. Deviation (So)	0.0088	1.97	5.76
	Relative Standard Deviation (%)	6.9	6.2	12.7
C2-Phenanthrenes/Anthracenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.01	10.3	16.1
	Single Operator Std. Deviation (So)	0.0014	0.98	3.43
	Relative Standard Deviation (%)	11	9.5	21.3
C3-Phenanthrenes/Anthracenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	4.4	4.4
	Single Operator Std. Deviation (So)		0.62	1.55
	Relative Standard Deviation (%)		14.1	35.5
C4-Phenanthrenes/Anthracenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	1.2	ND
	Single Operator Std. Deviation (So)		0.24	

TABLE 7 *Continued*

Target Analyte	Statistic/Parameter	Study Pore Water Samples		
		HP-24	HP-3	HP-4
		Low	Medium	High
	Relative Standard Deviation (%)		20.6	
Fluoranthene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.04	5.6	5.8
	Single Operator Std. Deviation (So)	0.0028	0.61	0.87
	Relative Standard Deviation (%)	6.7	10.9	15.1
Pyrene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.06	6.2	7.7
	Single Operator Std. Deviation (So)	0.0038	0.75	1.28
	Relative Standard Deviation (%)	6.2	12.1	16.8
C1-Fluoranthenes/Pyrenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.04	5.0	6.1
	Single Operator Std. Deviation (So)	0.0033	0.78	1.79
	Relative Standard Deviation (%)	7.3	15.8	29.2
Benz[a]anthracene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	0.76	0.75
	Single Operator Std. Deviation (So)		0.16	0.33
	Relative Standard Deviation (%)		20.8	44.5
Chrysene	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	0.01	0.77	0.79
	Single Operator Std. Deviation (So)	0.0009	0.16	0.35
	Relative Standard Deviation (%)	10.7	20.5	44.7
C1-Chrysenes	Number of Retained Values	7	7	7
	Mean Recovery (ng/mL)	ND	0.54	0.50
	Single Operator Std. Deviation (So)		0.11	0.33
	Relative Standard Deviation (%)		21.2	64.9
Total Toxic Units	Number of Retained Values	7	7	7
	Mean Recovery (units)	0.15	50.4	81.4
	Single Operator Std. Deviation (So)	0.01	3.52	5.23
	Relative Standard Deviation (%)	4.8	7.0	6.4

[A] ND: Analyte not detected in the associated sample.

concentrations of pore water PAHs. Efforts were made to ensure that sediments were chosen that had a full distribution of target PAH ring sizes, a range of PAH concentrations found in environmental sediment samples, and a representative range in total organic carbon concentration and texture.

15.1.2 The quantitations were based on three- or four-point calibration curves as verified by daily analysis of duplicate calibration verification standards at the medium-high concentration level. Prior to sample analysis, the initial calibration curves must have a coefficient of determination greater than 0.990, and the relative response factors must have a relative standard deviation of less than 25 % for two to three-ring PAHs, and less than 30 % for four-ring PAHs. The calibration verification mean relative response factor must agree with those of the initial calibration curve within 20 % for two to three-ring PAHs, and less than 25 % for four-ring PAHs. No sample data were reported if these criteria were not met. All method blanks met the requirement that the concentrations be at or less than 20 % of the Performance Limits for individual PAHs.

15.1.3 As directed in section 10.3 of Practice D 2777, the data were evaluated for outliers. The data were evaluated using the one-sided t-test at the upper 5 % significance level as described in Practice E 178, Section 6. Two outlying observations were found for high-level sample HP-4. One C3-naphthalenes result and one acenaphthylene result for sample HP-4 were outliers. The mean and single operator standard deviation were recalculated for sample HP-4 C3-naphthalenes and acenaphthylene without the outlying observations (that is, n = 6).

15.1.4 The precision statements for each analyte are shown on Table 7. For this single-laboratory study, it was assumed that the calculated standard deviation is equivalent to the single operator standard deviation (S_o). Replicate determinations of sample PAH concentrations typically had relative standard deviations (RSDs) less than 10 %, with somewhat higher RSDs for higher molecular weight compounds. The only unusually high RSDs occurred for the highest molecular weight PAHs from high-level sample HP 4. The reason for this is that the saturation limits may have been reached for the high molecular weight PAHs (that is, C1-phenanthrenes/anthracenes through C1-chrysenes).

15.1.5 Finally, the variation of individual PAH determinations had no significant effect on the repeatability of the total toxic unit determinations. See Table 7. This was demonstrated even though the statistical outliers found in sample HP-4 were not omitted in the calculation of total toxic units. The RSDs for the total toxic unit results ranged from 5 to 7 %.

15.2 *Single Analyst Bias Statement*:

15.2.1 A single laboratory study was performed using the perdeuterated PAHs d12-benz(a)anthracene and d10-2-methylnaphthalene spiked at low, medium, and high levels into environmental sediment samples. The quality control statements for each analyte level sample, obtained from the perdeuterated spike study, are shown in Tables 8-10. The quality control statements can also be considered precision and bias statements because the true spiking levels of the perdeuterated PAHs were known. The graphs and regression equations show the relationship between single-operator standard deviation and concentration, and mean measured value and

TABLE 8 HP-24 Low Concentration Quality Control

Analyte	True Spiked Value (ng/mL)	Number of Retained Values	Mean Recovery (ng/mL)	Mean Recovery (%)	Single Standard Deviation (So)	Relative Standard Deviation (%)
2-Methylnaphthalene-d10	4.68	7	4.33	92.6	0.3161	7.3
Benz[a]anthracene-d12	0.0429	7	0.0352	81.9	0.0031	8.8

TABLE 9 HP-3 Medium Concentration Quality Control

Analyte	True Spiked Value (ng/mL)	Number of Retained Values	Mean Recovery (ng/mL)	Mean Recovery (%)	Single Standard Deviation (So)	Relative Standard Deviation (%)
2-Methylnaphthalene-d10	26.7	7	26.7	100.1	0.859	3.2
Benz[a]anthracene-d12	0.25	7	0.199	81.0	0.015	7.5

TABLE 10 HP-4 High Concentration Quality Control

Analyte	True Spiked Value (ng/mL)	Number of Retained Values	Mean Recovery (ng/mL)	Mean Recovery (%)	Single Standard Deviation (So)	Relative Standard Deviation (%)
2-Methylnaphthalene-d10	283.9	7	230.7	81.3	11.0	4.8
Benz[a]anthracene-d12	2.61	7	2.13	81.7	0.13	5.9

concentration for both perdeuterated PAHs (see Figs. 1-4). The figures show the linearity of precision and accuracy with increasing concentration. The d12-benz(a)anthracene recoveries were consistently around 80 %. This may possibly indicate the consistent suppression of the mass spectral signal by a near-eluting compound. The recoveries for d10-2-methylnaphthalene ranged from 81 to 112 %. The repeatability for the known spike recoveries was consistent; the known spike RSDs ranged from 3 to 9 %. PAH concentration had no significant effect on the repeatability of the technique.

16. Quality Control Criteria

16.1 *Initial Calibration*:

16.1.1 The following acceptance criteria will be used for initial calibration: *(1)* The signal to noise (S/N) ratio for the GC signals present in every selected ion current profile (SICP) must be ≥10:1 for the labeled internal standards and calibration compounds; *(2)* The percent relative standard deviation (RSD) for the mean area ratio/ng for labeled internal standards and the calibration compounds must be less than 30 % for high molecular weight PAHs and less than 25 % for low molecular weight PAHs, and the $r^2 > 0.99$. The calibration curve must not be forced through the origin; *(3)* The number of calibration standards may be reduced from four to three based on the criteria in 12.3 of this test method.

16.1.2 The following corrective action will be adopted for initial calibration: *(1)* Initial calibration must be re-established if the RSD(s) exceed the limit(s); *(2)* The calibration will not be re-established in response to a nonconforming RSD if the sample results are less than the PQL.

16.2 *Daily Duplicate Calibration Verifications*:

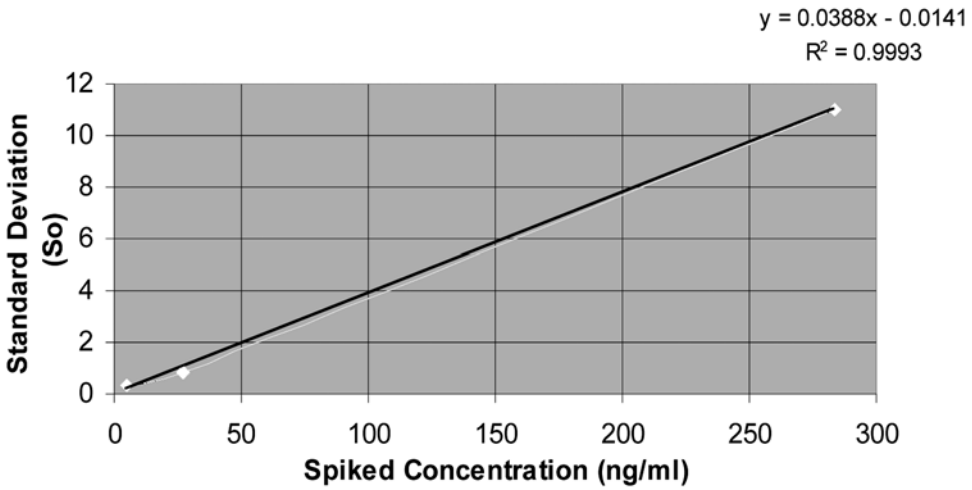

FIG. 1 2-Methylnaphthalene-d10 Single Standard Deviation versus Spiked Concentration

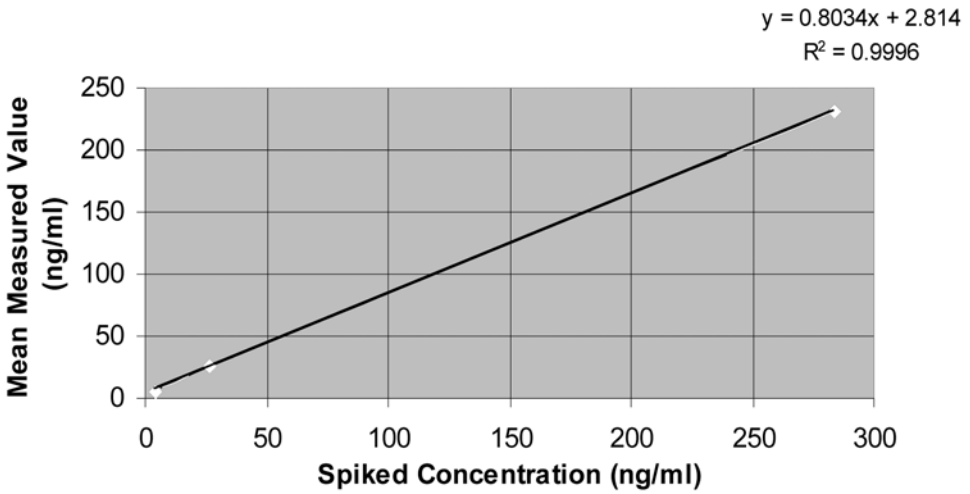

FIG. 2 Methylnaphthalene-d10 Mean Measured Value versus Spiked Concentration

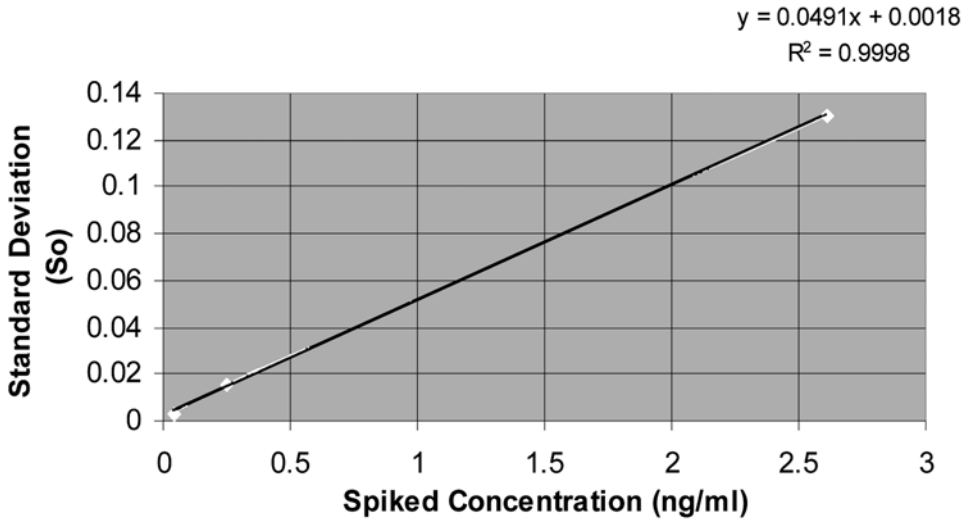

FIG. 3 Benz[a]anthracene-d12 Single Standard Deviation versus Spiked Concentration

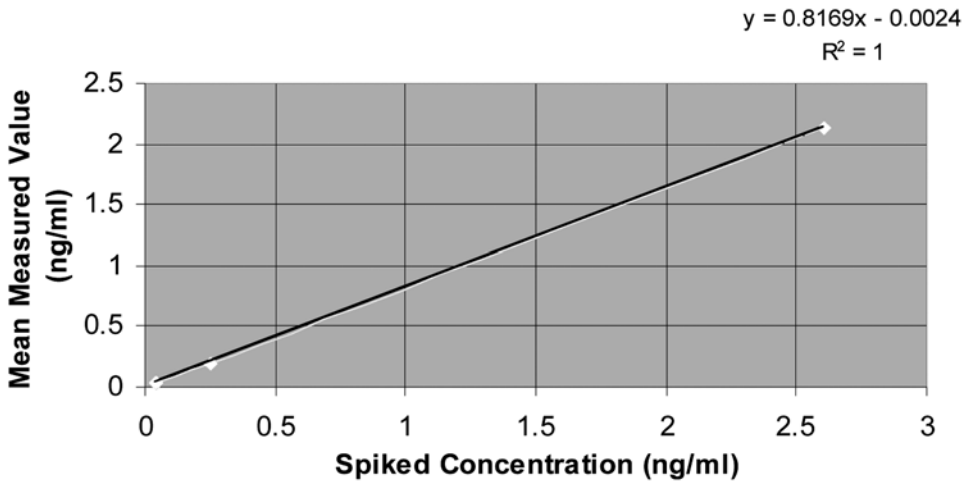

FIG. 4 Benz[a]anthracene-d12 Mean Measured Value versus Spiked Concentration

16.2.1 The following acceptance criteria will be used for daily duplicate calibration verifications: *(1)* The S/N ratio for the GC signals present in every SICP must be ≥10:1 for the labeled internal standards and the calibration compounds; *(2)*

The percent differences for the measured area ratio/ng of all analytes must be within ±25 % for high molecular weight PAHs and less than ±20 % for low molecular weight PAHs of the mean values established during the initial calibration.

16.2.2 The following corrective action will be adopted for daily duplicate calibration verifications if the first acceptance criterion is not satisfied: a new initial calibration curve must be established before sample extracts can be analyzed.

16.3 *Flocculation Blanks*:

16.3.1 The following acceptance criterion will be used for flocculation blanks: Prepared as needed to assess contamination from flocculation reagents and handling. Target analytes must not be detected above ⅓ of the target detection limits or >20 % of the associated sample result(s).

16.3.2 The following corrective action will be adopted for flocculation blanks: Locate the source of the contamination; correct the problem. Re-extract and reanalyze associated samples that are less than ten times the level of the contaminants present in the method blank.

16.4 *Extraction and Analytical Blanks*:

16.4.1 The following acceptance criterion will be used for extraction and analytical blanks: Analyzed between every sample to monitor the baseline. Target analytes must not be detected above ⅓ of the target detection limits or >20 % of the associated sample result(s).

16.4.2 The following corrective action will be adopted for extraction and analytical blanks: Locate the source of the contamination; correct the problem. Re-extract and reanalyze associated samples that are less than ten times the level of the contaminants present in the method blank.

16.5 *Signal to Noise Ration*:

16.5.1 The following acceptance criterion will be used for signal to noise ratio: The signal to noise (S/N) ratio for the GC signals present in every selected ion current profile (SICP) must be ≥3:1 for target compounds in environmental samples and ≥10:1 for the labeled internal standards.

16.5.2 The following corrective action will be adopted for signal to noise ratio: Reanalyze the sample unless obvious matrix interference is present.

APPENDIX

(Nonmandatory Information)

X1. ION PLOTS

X1.1 Selected ion chromatograms from a typical coal tar impacted pore water of d-PAH internal standards (top chromatogram of each page), and the related target parent and alkyl PAHs. Target species are indicated with brackets, and interfering species are marked with an “X.”

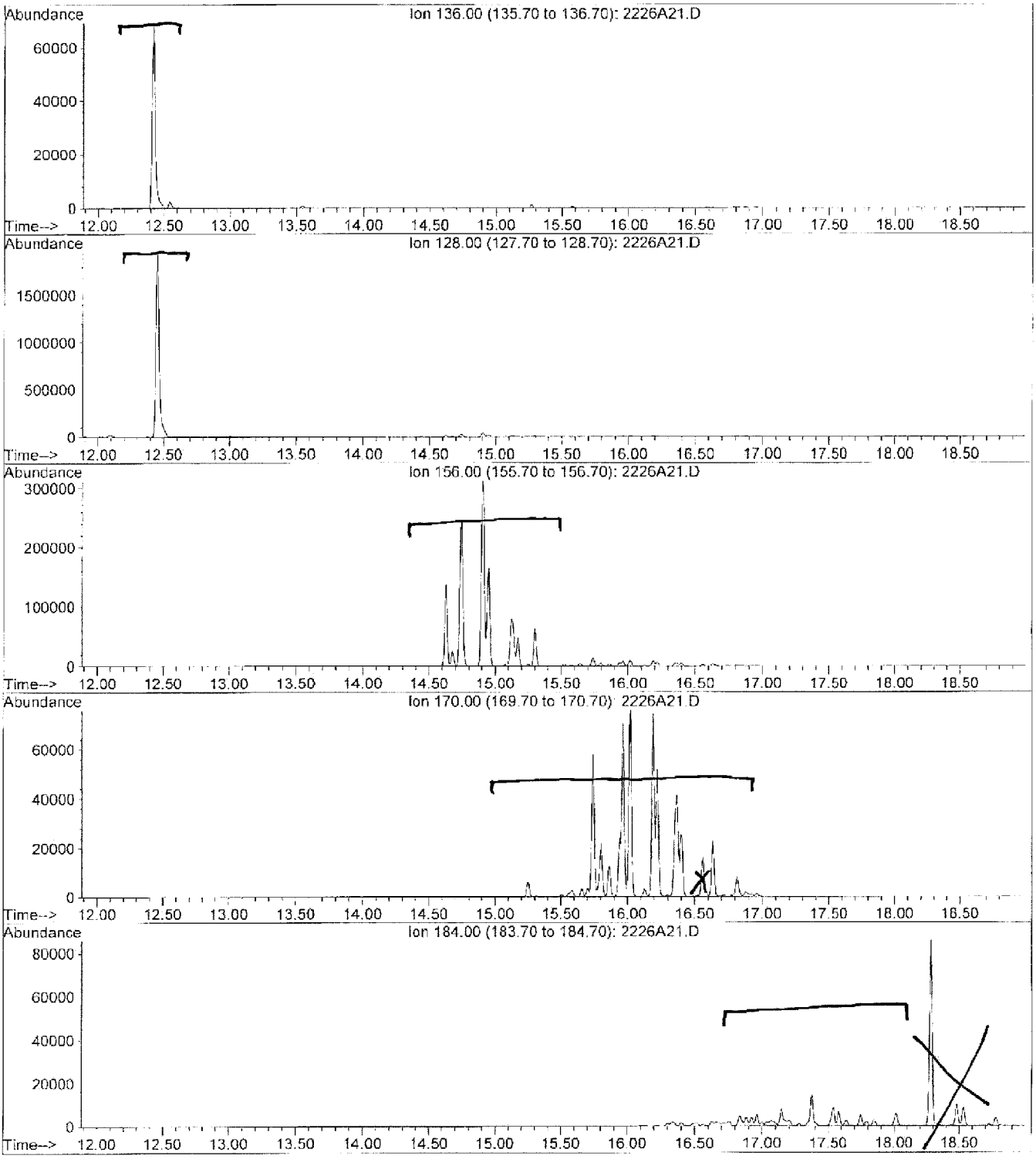

FIG. X1.1 Naphthalenes

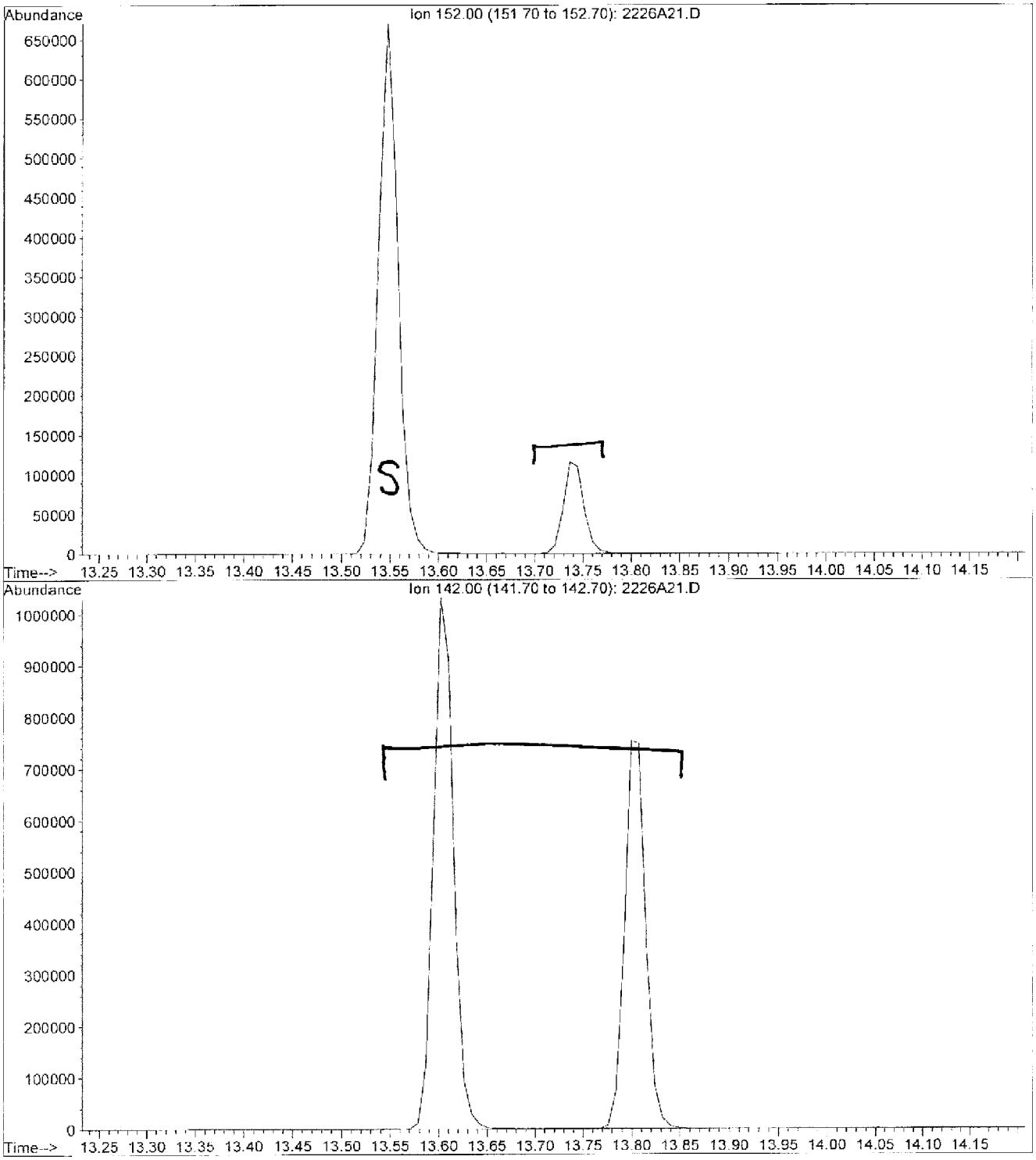

FIG. X1.2 Methylnaphthalenes
("s" is a spiked d_{10}-methylnaphthalene surrogate)

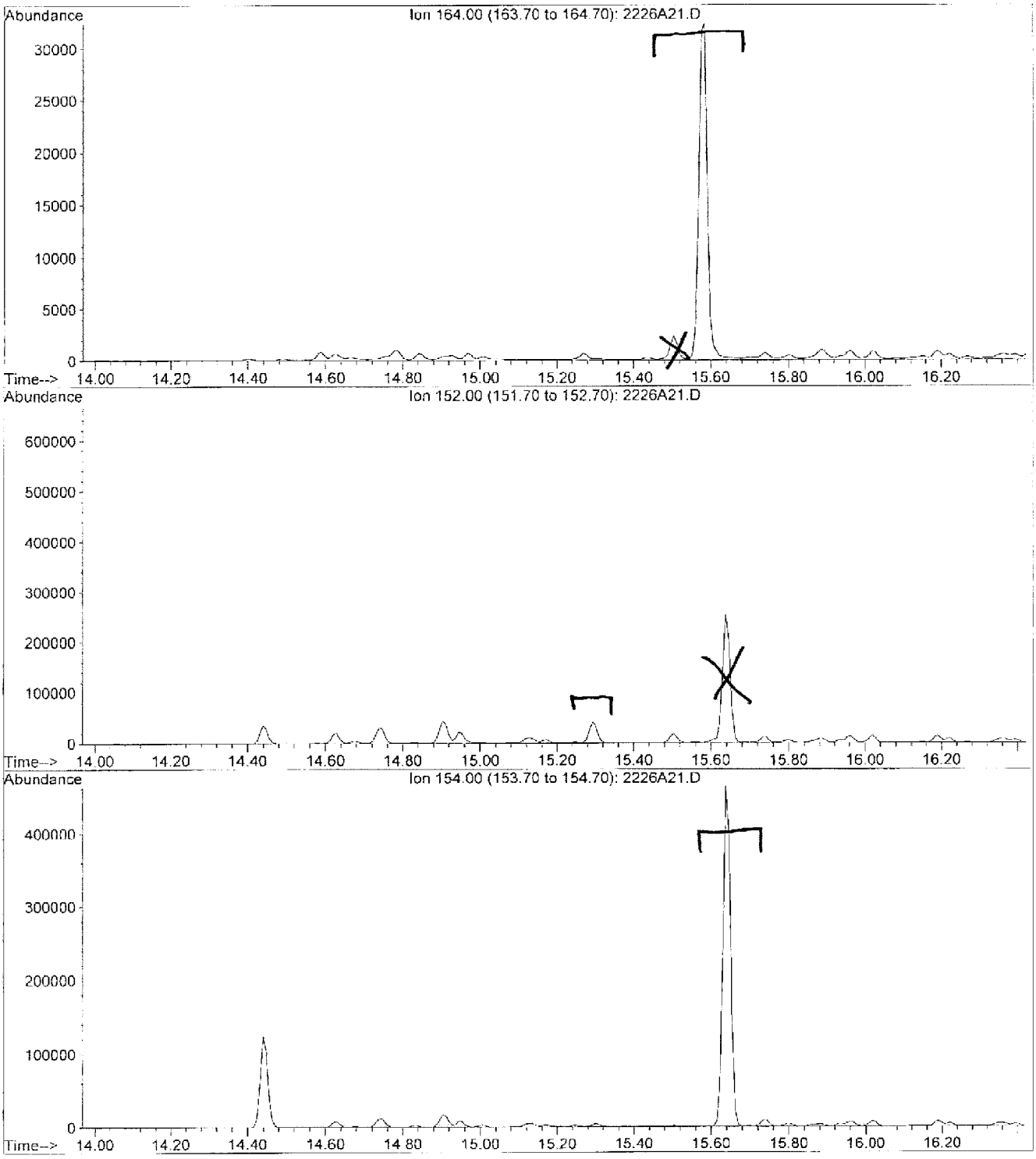

FIG. X1.3 Acenaphthylene/Acenaphthene

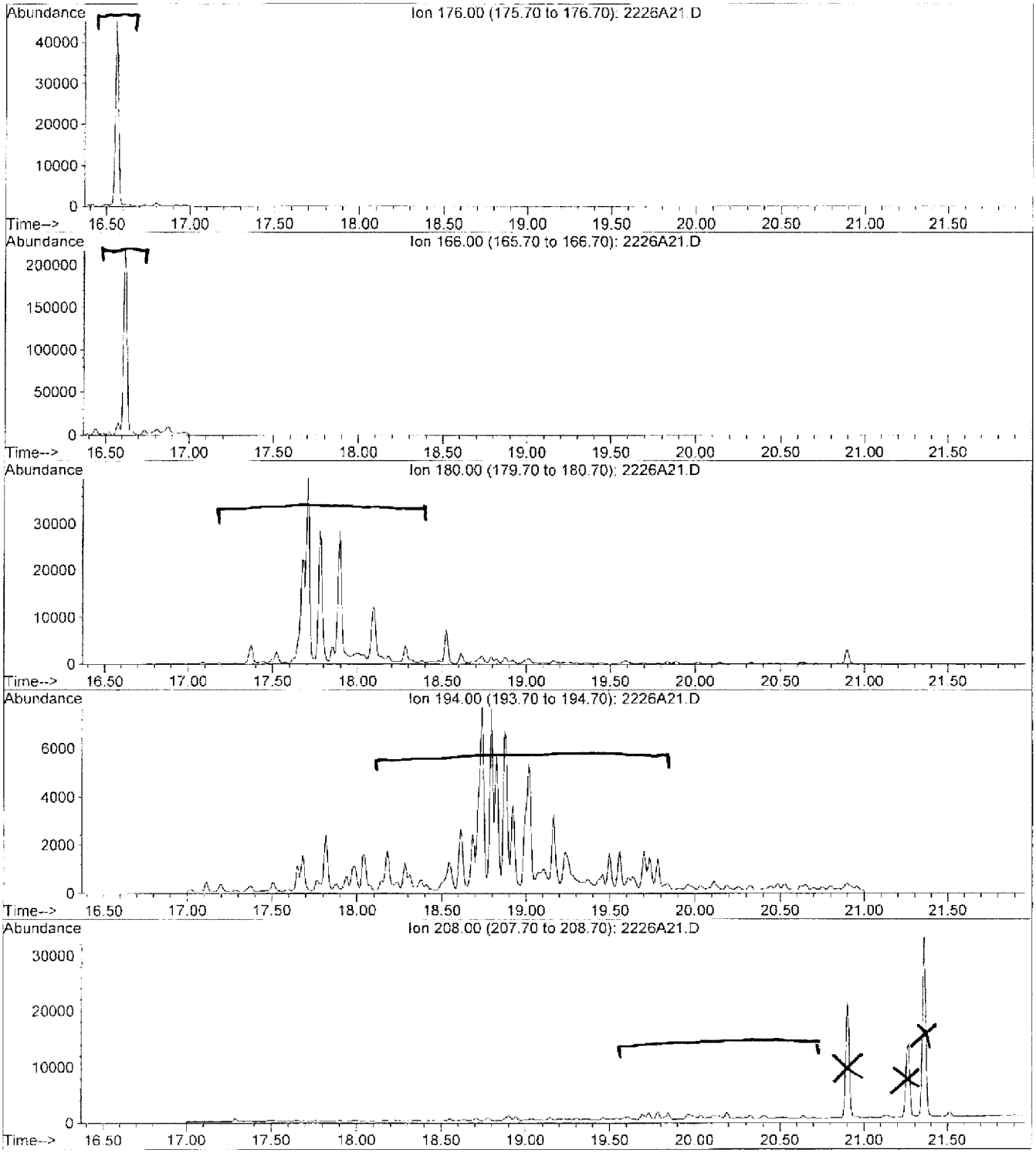

FIG. X1.4 Fluorenes

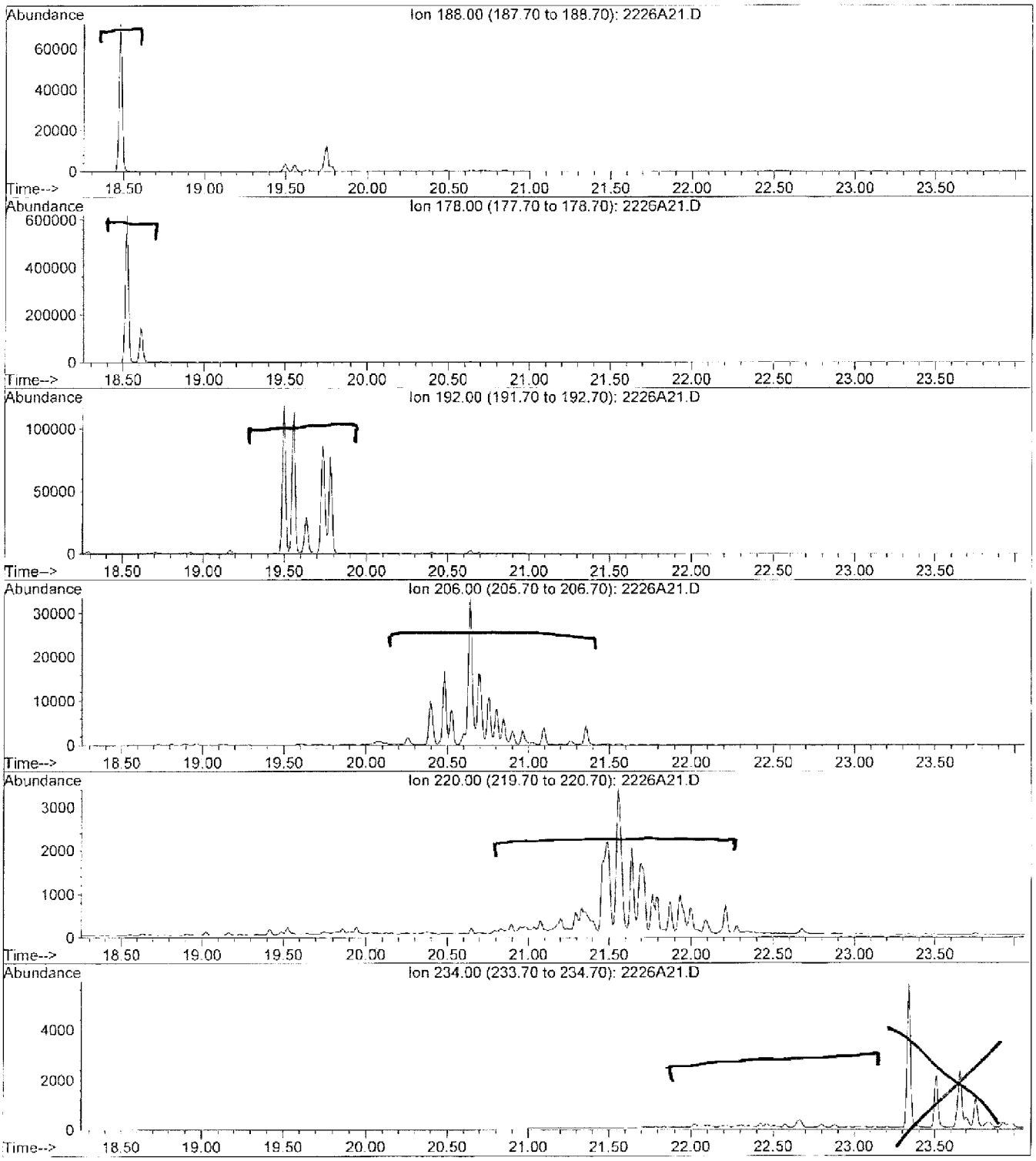

FIG. X1.5 Phenanthrenes/Anthracenes

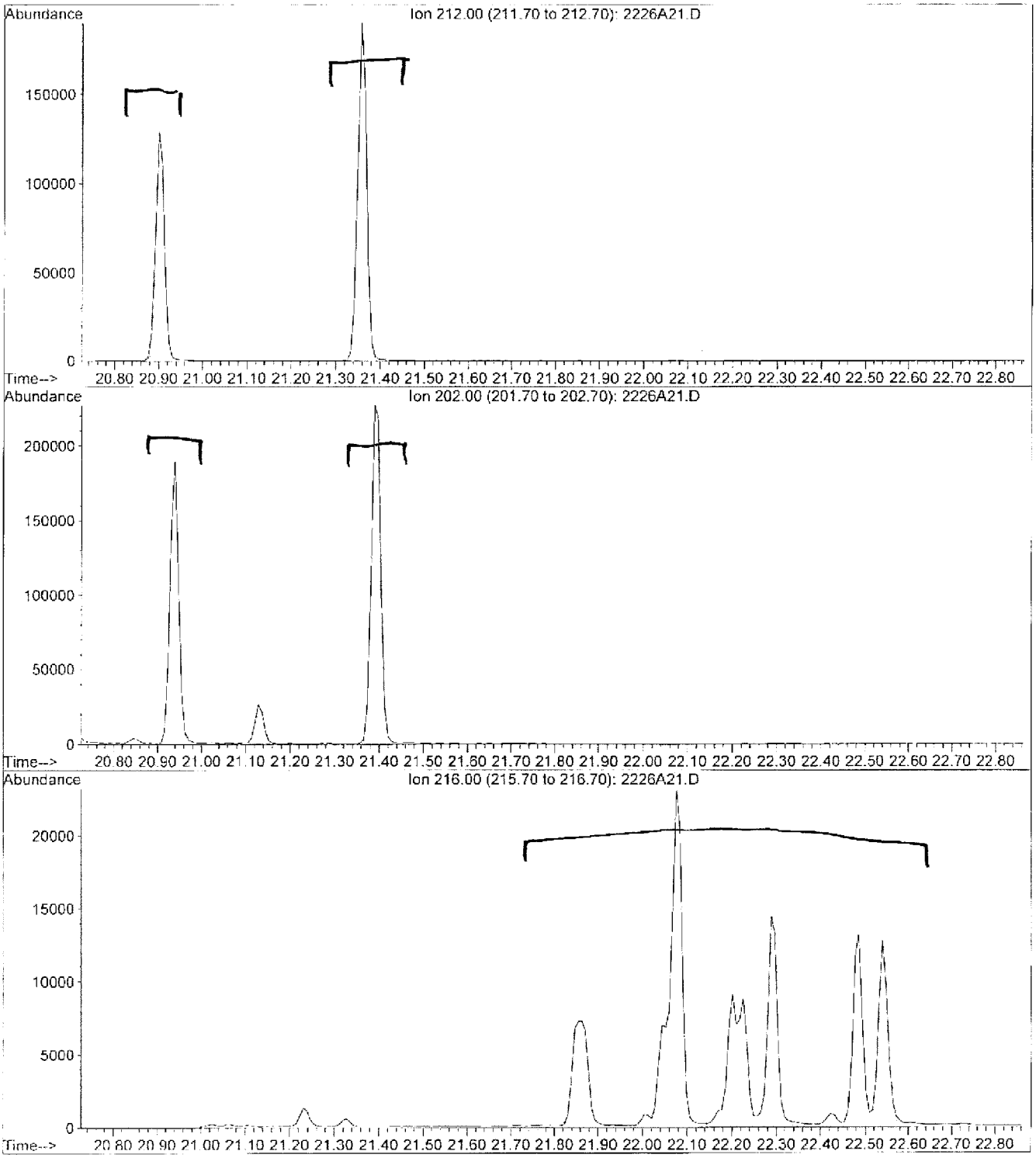

FIG. X1.6 Fluoranthenes/Pyrenes

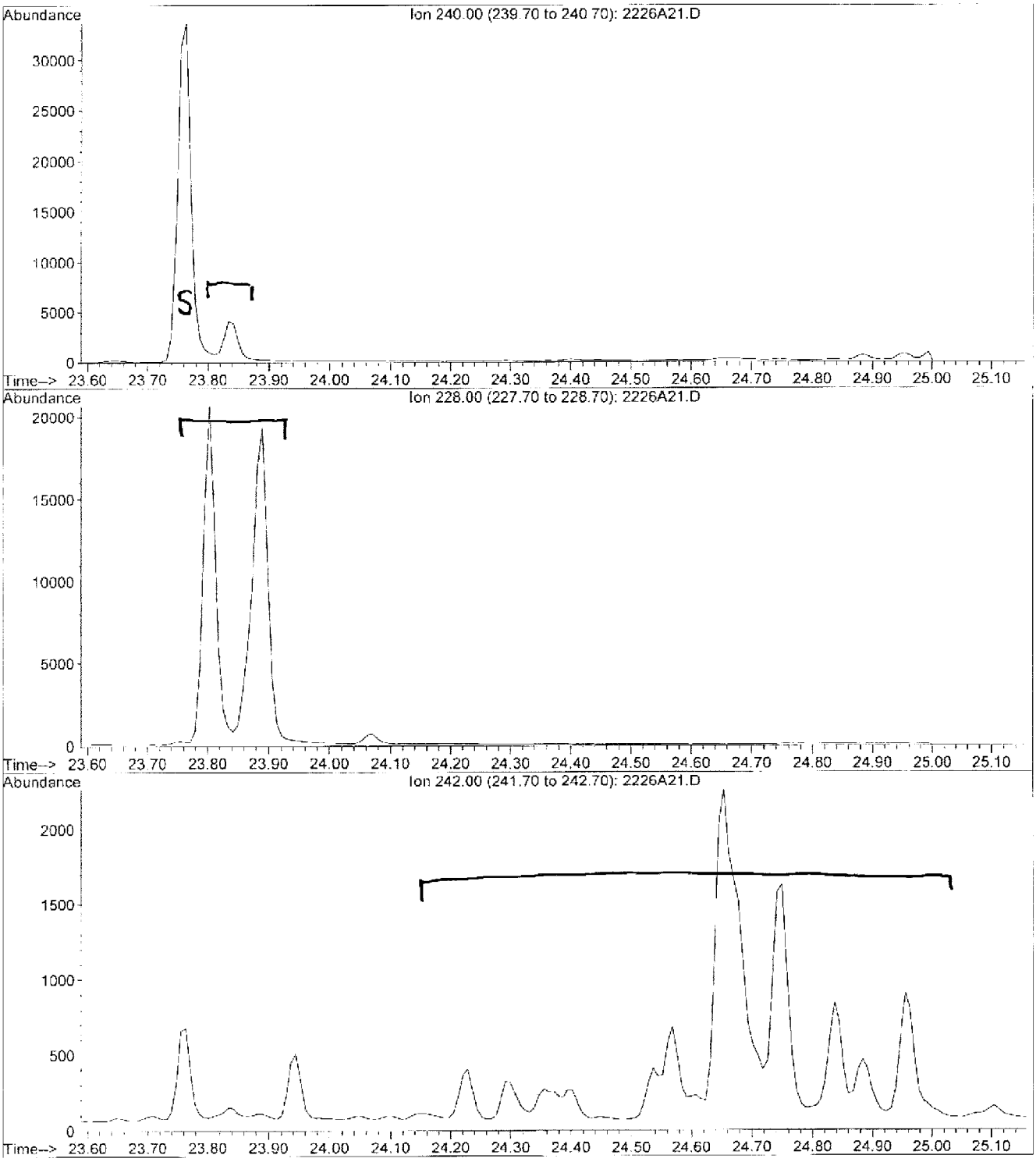

FIG. X1.7 Benz[a]anthracenes/Chrysenes
("s" is a spiked d_{12}-benz[a]anthracene surrogate)

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Black carbon in marine sediments

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Abstract

Concentrations of black carbon were determined for a number of marine sediments. A comparison of black carbon based on thermal oxidation and hot concentrated nitric acid pretreatments revealed that the latter significantly overestimates combustion derived carbon phases. Black carbon accounts for about 15 to 30% of total organic carbon and therefore reduces the fraction of unidentified sedimentary organic carbon. Examination of a relict oxidation front in a Madeira Abyssal Plain turbidite provided the first evidence for significant black-carbon degradation (about 64%) in marine sediments given time (10–20 kyr) and oxygen exposure.

Keywords: black carbon; soot carbon; organic carbon; sediments; degradation; Black Sea; turbidite; North Sea; Scheldt Estuary; Eastern Mediterranean

1. Introduction

Black carbon is usually defined as the highly condensed carbonaceous residue from incomplete combustion processes. It has been found omnipresent in the atmosphere, ice, soils and sediments due to its widespread production and its supposed chemical and microbiological inertness in the environment (Goldberg, 1985). Its distribution in the atmosphere has been investigated in detail because of its strong absorbance of solar radiation, its catalytic effects on chemical reactions and its importance to the global carbon cycle (Seiler and Crutzen, 1980; Kulbusch and Crutzen, 1995). Black carbon could represent a significant sink for carbon dioxide because vegetation fires and wood fuel combustion transfer carbon from the relatively fast biological-atmosphere carbon cycle to the long-term geological carbon cycle.

Estimates of global black carbon formation (0.05–0.270 Gt yr^{-1}; Kulbusch and Crutzen, 1995) are of the same order as those of riverine input of particulate organic carbon to the ocean (0.17 Gt yr^{-1}; Ludwig et al., 1996) and burial of organic carbon in marine sediments (0.13–0.6 Gt yr^{-1}; Berner, 1982; Middelburg et al., 1997). If these flux estimates are correct, black carbon should contribute considerably to the organic matter being buried in marine sediments.

The sedimentary record of black carbon has been used as a record of forest fires and fossil fuel emissions (Smith et al., 1973; Bird and Cali, 1998), and past atmospheric oxygen content, because the presence of charcoals provides a lower limit to atmospheric oxygen levels (Cope and Chaloner, 1980). Verardo and Ruddiman (1996) proposed that the

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black carbon record may improve estimates of marine organic carbon burial and may contain paleo-environmental information. The distribution of black carbon in surface sediments may significantly affect the distribution, speciation and bioavailability of polycyclic aromatic hydrocarbons (Gustafsson et al., 1997; Gustafsson and Gschwend, 1998).

Here we compare two recently developed techniques for black carbon determinations in sediments (Gustafsson et al., 1997; Verardo, 1997) and apply them to surface sediments from the North Atlantic Ocean, the North Sea, the Eastern Mediterranean, the Black Sea, and an intertidal flat in the Scheldt estuary (The Netherlands). The contribution of black carbon to total organic carbon will be discussed. We will also present results from the oxidised and reduced sediment zones from a relict turbidite in the Madeira abyssal Plain which indicate that black carbon was degraded significantly within 10–20 kyr.

2. Materials and methods

There is no unique definition of black carbon (Goldberg, 1985; Kulbusch, 1995) and many terms are in use (e.g. soot, black carbon, elemental carbon and charcoal). The definition chosen usually reflects the operational technique applied or the processes studied. The former three terms are usually applied to the combustion products formed at higher temperatures than charcoal (Lim and Cachier, 1996). Following a review of the literature we selected two methods: (1) a thermal oxidation method for soot carbon (Gustafsson et al., 1997) and (2) the HNO_3 method for charcoal (Verardo, 1997). Both methods have specifically been developed for determination of low quantities of black carbon in complex sedimentary matrices containing refractory macromolecular organic matter and large amounts of carbonate minerals. They both rely on in situ acidification to remove inorganic carbonate and carbon analysis of the residue by an elemental analyser, but they differ in the pretreatment procedure to remove non-black-carbon organic matter.

Samples were thoroughly ground in an agate mortar mill and very well homogenised to reduce variability between replicates and to reduce any potential charring during thermal treatments. Organic carbon (OC) was determined according to Nieuwenhuize et al. (1994) and involves the determination of total carbon using an automatic elemental analyser (Carlo Erba type NA-1500) following the partitioning of inorganic and organic carbon phases by acidification with 25% HCl in situ within precleaned silver cups (12 mm × 5 mm; Van Loenen Instruments, D2010). The soot carbon method of Gustafsson et al. (1997) follows that for organic carbon, except that there is a pretreatment in which the samples are thermally oxidised at 375°C for 14 h in the presence of oxygen at atmospheric partial pressure. The carbon remaining after thermal oxidation and HCl acidification is considered to be soot (SC1). Gustafsson et al. (1997) have extensively tested their method with synthetic samples and observed no interference but for corn pollen which were partly recovered as soot. The average blank value for OC and SC1 is 4.1 μg C, which mainly originates from the Ag containers used.

The charcoal method of Verardo (1997) is also similar to the organic carbon method, except that aluminium containers are used, instead of HCl, 10 times 30 μl of concentrated nitric acid (50°C) is added, and that the chromatographic column was kept at 35°C instead of 80°C to better separate the nitrogen (derived from nitric acid) and carbon dioxide peaks. Recovery and interference tests indicated that refractory carbon in coal, humic acids and *Lycopodium* spore tablets were removed by the hot nitric acid treatment and that natural charcoal and elemental carbon were quantitatively recovered as charcoal (Verardo, 1997). For reasons given below, we will refer to this fraction as nitric acid resistant carbon (NARC) and not charcoal. Samples were also subjected to hot nitric acid treatment after thermal oxidation (SC2). The average blank value for NARC and SC2 is 5.1 μg C, which mainly originates from the nitric acid. Reproducibility of carbon measurements is better than 5% at concentrations above 0.1 wt.%, but is on the order of 20% at concentrations less than 0.05 wt.%.

Sediment samples were obtained from various locations covering a range of environments (Table 1). Sediments from the Molenplaat (MOL1–5), an intertidal flat in the Scheldt estuary, are sandy and dynamic. Sediments from the Iberian Margin (OMEX1–5) are carbonate-rich and cover a range of water depths and grain sizes. Black Sea sediments

Table 1
Sample characteristics, organic carbon and black carbon

Sample	Depth in sediments (cm)	Water depth (m)	Latitude (N)	Longitude (E/W)	Median grain size (μm)	OC (wt.%)	SC1 (wt.%)	SC2 (wt.%)	NARC (wt.%)	SC/OC (%)
Molenplaat, Schelde estuary, The Netherlands										
MOL1	4–5	intertidal	51.26	03.57E	153	0.309	0.043	0.063	0.173	17
MOL2	4–5	intertidal	51.26	03.57E	109	0.372	0.056	0.077	0.280	18
MOL3	4–5	intertidal	51.26	03.57E	147	0.158	0.038	0.029	0.128	21
MOL4	4–5	intertidal	51.26	03.57E	153	0.093	0.041	0.037	0.160	42
MOL5	4–5	intertidal	51.26	03.57E	173	0.108	0.043	0.046	0.178	41
Iberian Margin, Atlantic Ocean										
OMEX	7–8	175	43.44	08.33W	83	0.470	0.095	0.085	0.445	19
OMEX	7–8	766	43.47	08.54W	127	0.240	0.075	0.065	0.201	29
OMEX	7–8	1522	43.41	09.27W	152	0.220	0.047	0.024	0.117	16
OMEX	7–8	2200	43.46	09.33W	12	0.630	0.163	0.152	0.528	25
OMEX	7–8	4909	44.01	09.54W	7	0.510	0.126	0.148	0.422	27
Northwestern Black Sea										
BS5	4–5	24	44.45	29.35E	13	1.262	0.220	0.242	1.206	18
BS6	4–5	54	43.45	28.48E	6	0.783	0.197	0.180	0.641	24
BS9	4–5	57	44.34	29.46E	11	1.638	0.233	0.254	1.388	15
BS10	4–5	72	44.18	30.05E	18	2.147	0.299	0.334	1.975	15
BS13	4–5	13	46.03	30.29E	19	1.111	0.274	0.260	1.034	24
BS15	4–5	13	46.33	31.25E	18	2.997	0.379	0.380	2.256	13
BS21	4–5	1997	43.22	32.10E	13	4.459	0.793	0.797	3.268	18
BS22	4–5	1494	43.18	30.02E	13	5.260	0.815	0.956	2.944	17
BS24	4–5	137	44.00	30.29E	24	2.288	0.337	0.383	1.913	16
North Sea										
GB	4–5	20	54.05	8.09E	38	2.093	0.457	0.441	1.443	21
SK	4–5	270	58.05	10.15E	12	2.424	0.367	0.417	1.834	16
FF	4–5	39	53.42	4.30E	77	0.559	0.090	0.119	0.458	19
BF	4–5	28	53.00	3.52E	233	0.078	0.032	0.041	0.101	47
BGA	4–5	4.9	51.45	3.48E	143	0.219	0.093	0.093	0.183	42
BGB	4–5	2.7	51.46	3.46E	285	0.055	0.028	0.039	0.084	61
Madeira Abyssal Plain			30.44	25.22W	< 10					
MAPox1	756–766	5400				0.182	0.102	0.045	0.205	40
MAPox2	767–777	5400				0.228	0.069	0.111	0.187	39
MAPox4	795–802	5400				0.361	0.123	0.122	0.283	34
MAPred1	807–817	5400				1.139	0.215	0.266	0.858	21
MAPred4	927–940	5400				1.137	0.277	0.292	0.917	25
Eastern Mediterranean			34.52	21.07E	< 10					
MED1	0–0.5	2539				0.417	0.142	0.154	0.314	35
MED2	9–11	2539				0.222	0.112	0.060	0.169	39
MED3	24.5–25	2539				2.598	0.729	0.596	2.396	25

OC: organic carbon concentration; SC1: carbon after thermal oxidation/HCl treatment; SC2: carbon after thermal oxidation/hot nitric acid treatment; NARC: hot nitric acid resistant carbon: SC/OC: contribution of soot carbon (average of SC1 and SC2) to organic carbon.

are from the north western shelf area including the mouths of the Danube (BS5), Dniestr (BS13) and Dnieper (BS15) and a transect from the Danube delta to the anaerobic, sulphide-containing deep basin (BS9, BS10, BS24, BS22, BS21). The samples from the North Sea, a continental shelf, have been investigated in detail for their biochemical composition and cover a range of degradation states and grain sizes

(Dauwe and Middelburg, 1998). Samples from the Madeira abyssal plain *f* turbidite (MAPox–red) have been studied in detail by Cowie et al. (1995) and Prahl et al. (1997) and will be used to estimate the extent of post-depositional oxidation of black carbon. Sediments from the Eastern Mediterranean comprise hemipelagic (MED1–2) muds and an organic-carbon rich sapropel (MED3; De Lange et al., 1994).

3. Results and discussion

3.1. Soot, charcoal and organic carbon contents

Soot concentrations determined with the thermal oxidation/HCl method (SC1) and thermal oxidation/HNO_3 method (SC2) are very well correlated ($r^2 = 0.96$; $n = 33$) with a slope (1.02 ± 0.04) and intercept (0.00 ± 0.04) not significantly different from 1 and 0, respectively (Fig. 1). This indicates that the fraction of carbon resistant to thermal oxidation (soot) is not removed by hot nitric acid. Although some pollen may survive thermal oxidation (Gustafsson et al., 1997), they are most likely degraded during acidification with hot concentrated nitric acid (Verardo, 1997).

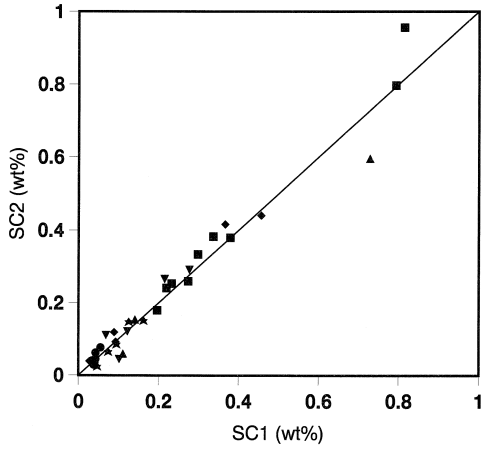

Fig. 1. Agreement between soot carbon results based on thermal oxidation/HCl treatment (SC1) and those based on thermal oxidation/hot nitric acid treatment (SC2). The solid line represents the best fit ($r^2 = 0.96$, $n = 33$): $SC2 = (1.02 \pm 0.04)SC1 + (0.00 \pm 0.04)$. Sample identification: dots: Molenplaat; stars: Iberian Margin; squares: Black Sea; rhombi: North Sea; inverted triangle: Madeira Abyssal Plain turbidite; triangle: Mediterranean.

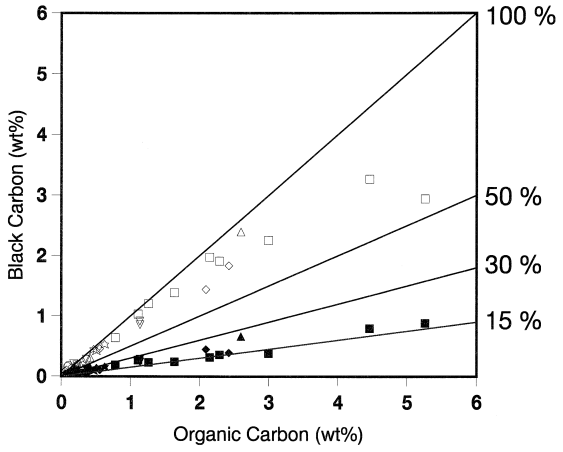

Fig. 2. Relation between organic carbon (OC), soot carbon (solid symbols, average of SC1 and SC2) and charcoal (NARC, open symbols). Lines representing 15, 30, 50 and 100% black carbon contributions to organic carbon are shown as well. Sample identification as in Fig. 1.

Soot contents range from 0.03 to 0.9 wt.% C (Table 1) and correlate with organic carbon contents (Fig. 2). Soot carbon contributes about 15 to 30% to total organic carbon with a tendency for deep-sea samples and other organic-carbon poor samples to contain a larger proportion of soot carbon (Fig. 3), perhaps an artefact of accumulated errors. Literature

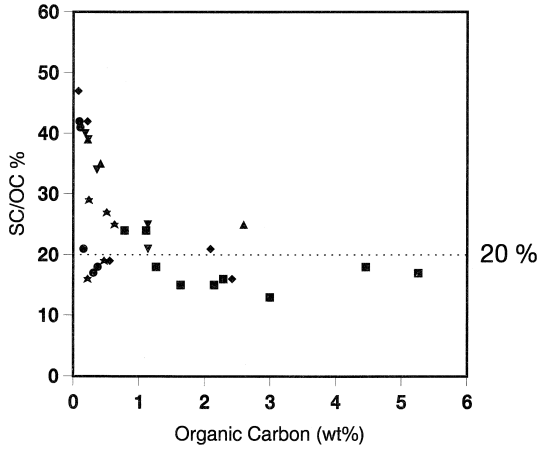

Fig. 3. Relation between organic carbon (OC) and the contribution of soot carbon (average of SC1 and SC2) to organic carbon (SC/OC). A line representing 20% black carbon contributions to organic carbon is shown as well. Sample identification as in Fig. 1.

data for the soot-to-organic carbon contribution range from 5 to 38% in Western Mediterranean sediments (Lim and Cachier, 1996) and 3 to 13% in North American shelf sediments (Gustafsson and Gschwend, 1998). The correlation between soot and organic carbon is probably due to their common dependence on grain size and hydrodynamic sorting during deposition.

Charcoal concentrations (NARC) based on the hot nitric acid digestion technique range from 0.08 to 3.2 wt.% and always account for more than 50% of the organic carbon (Fig. 2). Based on the same technique, Verardo and Ruddiman (1996) have reported that NARC was the dominant ($>50\%$) component of the organic carbon preserved in tropical Atlantic deep-sea sediments. If the NARC fraction indeed corresponds to charcoal, a product of terrestrial biomass burning, this would imply that the majority of organic carbon in marine sediments is terrestrial (charcoal and non-charcoal terrestrial carbon). This is clearly at odds with isotopic, elemental, NMR and molecular compositional constraints (Hedges and Oades, 1997; Bird and Cali, 1998). For instance, North Sea sediments contain at least 70% NARC and have $\delta^{13}C$ values ranging from -21.5 to -22.2‰ (Dauwe and Middelburg, 1998). If all NARC would be charcoal with a $\delta^{13}C$ of -27 to -26‰, this would imply that the marine organic carbon end-member should have $\delta^{13}C$ values heavier than -11‰. It is therefore more likely that hot nitric acid resistant carbon comprises not only charcoal (and soot), but also non-hydrolysable macromolecules from marine sources (De Leeuw and Largeau, 1993; Bird and Cali, 1998).

3.2. Black carbon and the unidentified fraction of sedimentary organic carbon

Despite significant improvements in analytical techniques and considerable efforts to characterise sedimentary organic carbon, a large fraction of it remains biochemically uncharacterised (Hedges and Oades, 1997). This unidentified fraction varies from about 40% in coastal sediments to more than 80% in deep-sea sediments. (e.g., Cowie et al., 1995; Wakeham et al., 1997; Dauwe and Middelburg, 1998). The major groups identified are amino acids, carbohydrates, lipids, and hexosamines (Cowie et al., 1995; Hedges and Oades, 1997). For a number of our samples there is additional information on the biochemical composition and the contributions of various biochemicals and soot carbon to total organic matter are shown in Fig. 4. The relative contributions of biochemical groups are remarkably similar: carbohydrates (8–19%), amino acids (8–19%) and hexosamines ($<2.2\%$). The total contribution of these hydrolysable fractions (17–41%) is similar to that of soot (16–42%), but smaller than the non-soot fraction (39–66%) that remains after hot nitric acid treatment ([NARC − SC]/OC). It therefore appears

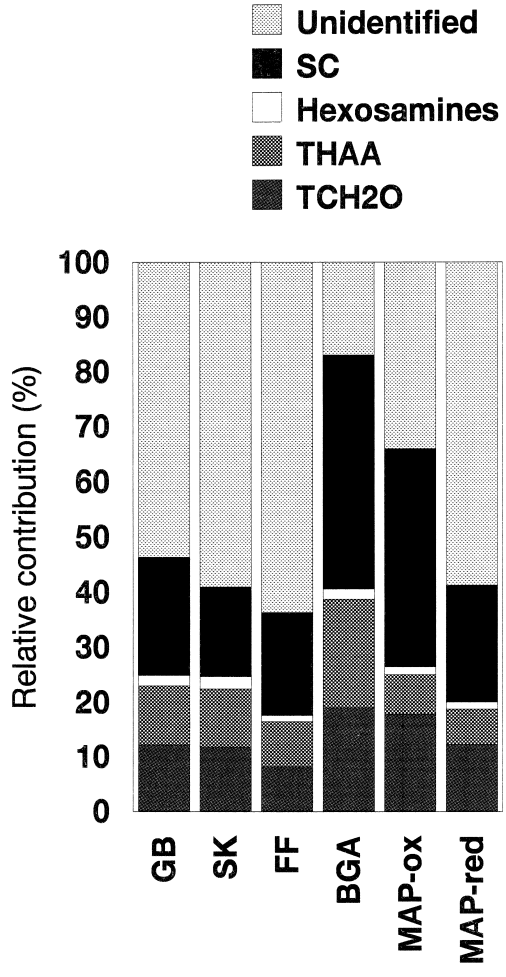

Fig. 4. Cumulative contribution of carbohydrates, total hydrolysable amino acids, hexosamines and soot carbon (SC) to total organic carbon. TCH_2O, THAA and hexosamine results for GB, SK, FF and BGA from Dauwe and Middelburg (1998) and for MAPox and MAPred from Cowie et al. (1995) and our own unpublished data.

that inclusion of black carbon in comprehensive analyses of sedimentary organic matter will significantly reduce the unidentified fraction.

3.3. Black carbon degradation

Black carbon is usually assumed to be chemically and biologically inert in the marine environment because (1) it has been identified in sediments of 65 Ma (Herring, 1985), (2) there is no down-core trend in size distribution of black carbon particles (Herring, 1985) and (3) it reacts very slowly towards chemical oxidation (Wolbach and Anders, 1989). Although microbiological breakdown during laboratory experiments has been reported (Potter, 1908; Shneour, 1966), there is as yet no information on degradation in marine sediments. The natural oxidation experiments provided by the Madeira Abyssal Plain *f* turbidite (Middelburg and De Lange, 1988; Prahl et al., 1989, 1997; Keil et al., 1994; Cowie et al., 1995) can be used to test the assumption of black carbon inertness. This distal turbidite originates from the northwestern African margin and was introduced to the Madeira Abyssal Plain about 140 kyr ago as a 4–4.5 m thick ungraded, uniform mud deposit (Middelburg and De Lange, 1988). The emplacement of this turbidite caused the exposure of labile and reduced components to oxic pelagic conditions. The post-depositional exposure to oxygen (and nitrate) under pelagic conditions during 10–20 kyr resulted in carbon removal in the uppermost section due to a downward progressing oxidation front (Wilson et al., 1985). A comparison between the organic matter concentrations and composition of the oxidised upper and reduced, unreacted lowermost section provides a unique opportunity to establish the extent of black carbon degradation.

Post-depositional oxidation of the upper section resulted in a decrease in total organic carbon from 1.14 to 0.25 wt.% and soot carbon from 0.27 to 0.10 wt.% (Fig. 5). Post-depositional oxidation removed about 77% of the organic carbon, but only about 64% of the soot carbon. Selective preservation of soot carbon increased its contribution to organic carbon from about 23 to 40% (Fig. 5) and is consistent with the preferential removal of marine organic matter and the selective preservation of terrestrial organic matter in these sediments (Prahl et al., 1997).

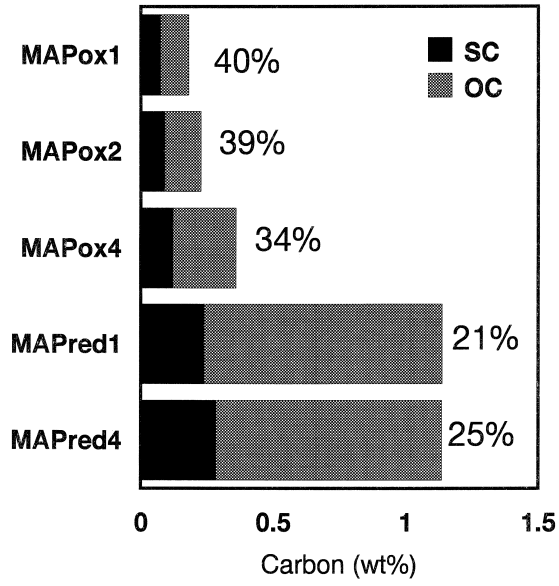

Fig. 5. Organic carbon (OC) and soot carbon (average of SC1 and SC2) in oxidised and reduced sections from the MAP *f* turbidite. MAPox1,2,4 have been subjected to extensive post-depositional oxidation, whereas MAPred1,4 have experienced little alteration since deposition. The relative contribution of soot carbon to organic carbon is also indicated.

These data definitely indicate that black carbon is degraded in marine sediments when exposed to oxygen (and nitrate), although the microbiological or chemical mechanisms involved remain unknown and require further study. Degradation of black carbon in marine sediments complicates the use of its sedimentary record to trace biomass burning (Herring, 1985; Verardo and Ruddiman, 1996; Bird and Cali, 1998) and to constrain past atmospheric oxygen levels (Cope and Chaloner, 1980). Our data also provide support for the oxygen exposure time impact on carbon preservation (Hartnett et al., 1998). It has been proposed that black carbon formation may represent a significant sink of atmospheric carbon dioxide and a minor source of oxygen (Kulbusch and Crutzen, 1995). This is based on the premise that black carbon is not degraded in the bio- and geosphere after formation. The observed degradation of black carbon in marine sediments during prolonged exposure to oxygen provides a small negative feedback between oxygen in the atmosphere (and ocean) and black carbon burial.

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ACTIVATED CARBON AMENDMENT AS A TREATMENT FOR RESIDUAL DDT IN SEDIMENT FROM A SUPERFUND SITE IN SAN FRANCISCO BAY, RICHMOND, CALIFORNIA, USA

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Abstract—Pesticide formulators formerly operating at Lauritzen Channel, a portion of San Francisco Bay near Richmond (CA, USA), caused contamination of sediment with dichlorodiphenyltrichloroethane (DDT). The present study evaluated the distribution of residual DDT in channel sediment six years following extensive remedial dredging. High DDT concentrations (up to 252 mg/kg) were found in Young Bay Mud sampled across the channel. Particle analyses showed most of the contamination is contained in the clay/silt sediment fraction, and desorption tests showed that availability is greater for DDT metabolites than parent DDT. The present study examined the feasibility of using activated carbon amendment to sequester DDT from sediment, including an evaluation of reactivated carbon as a less costly alternative to virgin activated carbons. Treatment success of activated carbon amendment to sediment collected from Lauritzen Channel was measured by reductions in aqueous equilibrium concentrations and uptake in semipermeable membrane devices (SPMDs). Four different activated carbons were tested and, after one month of treatment with 3.2 weight % carbon, DDT aqueous equilibrium concentrations were reduced up to 83% and SPMD uptake was reduced up to 91%. Reactivated carbon was comparable with virgin carbons in all tests. Reduction in SPMD uptake of DDT by treatment with 3.2% reactivated carbon increased to 99% after 26 months of treatment. The effectiveness of activated carbon was dependent on the type, size, dose, and contact time. The results show the potential usefulness of activated carbon amendment as a follow-up remedial technology for management of residuals after dredging contaminated sediment.

Keywords—DDT Sediment treatment Activated carbon amendment Dredging residuals Hydrophobic organic chemicals

INTRODUCTION

Dredging is the most prevalent ex situ approach for sediment remediation. However, problems with this approach include destruction of benthic habitat, incomplete removal of contaminated sediments, and resuspension of contaminated fines that may migrate to surrounding waters. Such limitations of dredging for management of contaminated sediments materialized at Lauritzen Channel, an active marine shipping terminal and industrial waterway connected to San Francisco Bay in Richmond, California, USA (Fig. 1). Several chemical processors conducted operations in this area from 1947 to 1966, including the pesticide formulator United Heckathorn. Chemical releases from processing and equipment washing caused DDT and dieldrin contamination in the embankment soil, sediment, and water of Lauritzen Channel [1]. The U.S. Environmental Protection Agency (U.S. EPA) added the United Heckathorn site to its National Priority List of Federal Superfund sites in March 1990, and a site history and description of embankment soil remedial actions can be found in Weston et al. [2]. The selected sediment remediation remedy was dredging and dewatering of all soft bay mud material with offsite disposal and placement of a clean material (sand) cap to enhance habitat value [3]. Remedial dredging was completed in 1997. Six years of postdredging marine monitoring of water and fauna by Pacific Northwest National Laboratory (PNNL) found residual DDT contamination levels exceeding the remediation goals of 0.59 ng/L and 0.59 mg/kg in water and sediment, respectively [4,5]. The highest sediment concentrations, greater than 1,000 mg/kg, were found under the eastern dock. Because the docks were not removed during dredging, contaminated sediment remained in this location. Total water DDT concentrations at the north end of the channel have remained consistently near 100 ng/L from 1991 to 2003 [5,6]. Several biological studies found remaining sediment toxicity to amphipods and DDT bioaccumulation in mussels [5,7]. After completion of remedial activities, Weston et al. [2] also found body burdens as great as or greater than before dredging.

The present study revisits the residual sediment contamination at Lauritzen Channel and proposes an in situ treatment strategy to sequester DDT and its metabolites. Sediment was collected at various locations in Lauritzen Channel to quantify the remaining DDT contamination residing at the sediment-water interface and at depth. Particle analysis and desorption kinetic testing further characterized the distribution of residual DDT and its metabolites in the sediment.

These results show the remedial action of environmental dredging proved ineffective for completely managing the DDT contamination at Lauritzen Channel. The remaining residual contamination continues to pose a risk to biota and human health more than six years after environmental dredging. The present study therefore investigates the in situ stabilization of residual DDT in sediment with activated carbon and tests the utility of reactivated carbon as a less costly alternative to virgin activated carbons. The efficacy of activated carbon amendment to sediment collected from Lauritzen Channel was measured using DDT aqueous equilibrium and uptake in semipermeable membrane devices (SPMDs) over two years of treatment in

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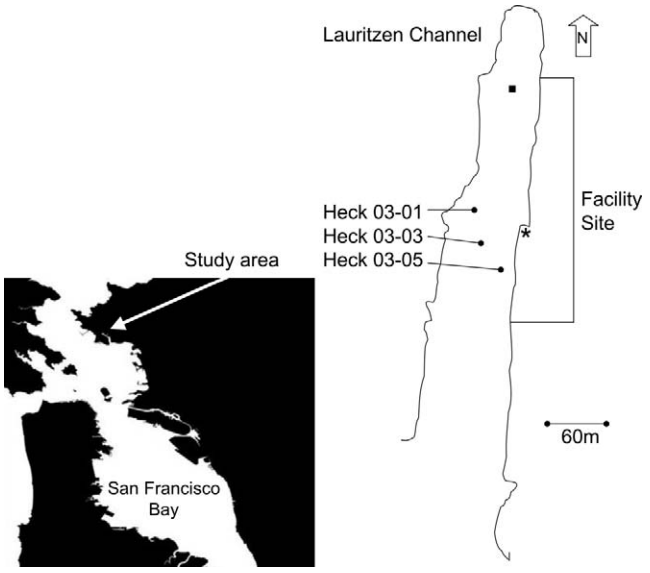

Fig. 1. Map of Lauritzen Channel (Richmond, CA, USA) and surrounding waterways including locations of vibracores (●), surface sampling using Eckman dredge (■), and contamination hotspot under pier (*). The approximate boundary of the former United Heckathorn facility is outlined on the eastern shore.

laboratory studies. These investigations derive from work showing that, compared to other types of sediment particles, polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons accumulate to a much greater extent in black carbonaceous particles and become more strongly bound and less bioavailable [8–10]. Black carbonaceous particles comprise a portion of the total organic carbon in sediments that strongly affects the partitioning of hydrophobic organic chemicals, via nonlinear, extensive, and competitive adsorption [11]. Zimmerman et al. [12] proposed that, by mixing activated carbon into the biologically active upper layer of sediment, PCBs would repartition and be sequestered in the carbon, thus reducing PCB bioavailability and release to water. Physicochemical [12,13] and 28- and 56-d benthic organism uptake studies [14–16] found high effectiveness in reducing polycyclic aromatic hydrocarbon and PCB availability after mixing sediment with activated carbon. These experiments and modeling [17] show activated carbon amendment effectiveness is dependent on size, dose, and contact time of activated carbon.

EXPERIMENTAL METHODS

Sediment characterization

Vibracore sampling and analysis. Sediment was collected at various locations in Lauritzen Channel to study the remaining DDT contamination within the sediment down to historic Old Bay Mud. In May 2003, six years after dredging of the channel, PNNL conducted vibracore sampling. The composition of strata in each core was identified and described by PNNL. We sampled distinct strata of three different cores collected near the hotspot at the north end of Levin Pier. The cores sampled included Heck 03-01, Heck 03-03, and Heck 03-05, at locations shown in Figure 1. After collection, all samples were placed in cold storage (4°C). Before analysis, deionized water containing 1 g/L sodium azide was added to each jar to inhibit microbial growth. The jars then were homogenized by rolling (2 rpm) for one week. The sediment samples were dried at room temperature and powdered with a clean mortar and pestle.

Dried sediment samples (2 g) were mixed with anhydrous sodium sulfate to form a free-flowing powder. Sediment DDT was extracted three times with an acetone:hexane mixture (1:1), following U.S. EPA Ultrasonic Extraction Method 3550B (http://www.epa.gov/sw-846/pdfs/3550b.pdf). Pesticide-grade solvents were used in all extractions and cleanups. Extracts were exposed to activated copper to remove sulfur and concentrated under nitrogen.

A variation of U.S. EPA Method 3620B (http://www.epa.gov/sw-846/pdfs/3620b.pdf) was followed for cleanup of extracts. A 1-cm diameter chromatographic column was packed with activated Florisil PR (3 g; Fluka, Buchs, Switzerland) and anhydrous sodium sulfate (1 cm). The column was preeluted with 40 ml of hexane. Sample extract was transferred into the column and eluted with 30 ml of 20% dichloromethane in hexane. The eluents were solvent switched to hexane and concentrated under nitrogen. Dibromooctafluorobiphenyl was added as an internal standard to correct for volume differences.

Concentration analyses were performed with an Agilent 6890N (Santa Clara, CA, USA) gas chromatograph with an electron capture detector and a fused silica capillary column (HP-5, 60 m × 0.25 mm i.d., 0.25-μm film thickness). Quantification of six desired peaks including *p,p′*-DDT, *o,p′*-DDT, *p,p′*-dichlorodiphenyldichloroethane (*p,p′*-DDD), *o,p′*-DDD, *p,p′*-dichlorodiphenyldichloroethylene (*p,p′*-DDE), and *p,p′*-dichlorodiphenylmonochloroethylene (*p,p′*-DDMU) was achieved using standard solutions prepared from solid standards (UltraSci, N. Kingstown, RI, USA). Standard curves were checked against a chlorinated pesticides standard (TCL) Mix (Supelco, Bellafonte, PA, USA). The breakdown of DDT on the gas chromatograph was monitored using a *p,p′*-DDT standard, and no samples were quantified unless breakdown was below 15%. In reporting data, ΣDDT represents the sum of the six identified compounds.

Surface sediment sampling. In February 2003, a surface-sediment sample was collected at the north end of Lauritzen Channel using an Eckman dredge (Fig. 1). The collected sediment was sieved through a 0.5-cm mesh to remove large objects (gravel, shells, worms) and stored in a cold room (4°C) until further analysis and use. The total organic carbon of the sediment was measured at 2.8 weight % by an elemental carbon analyzer after acid treatment to remove carbonates (Huffman Laboratories, Golden, CO, USA).

Particle analysis. Surface sediments were separated into size fractions by wet sieving. A saturated solution of cesium chloride was used to produce two density fractions termed light and heavy, as described elsewhere [18]. The light fraction contained coal- and wood-derived particles, and sand, silt, and clays comprised the heavy mineral fraction. Light microscopy identified the particle types within the fractions, and tweezers were used to remove individual types particle greater than 0.25 mm for analysis. Identified particles included heavy density particles of shell and mineral, and light density particles were comprised of black carbon, plant materials, and charred and uncharred wood. Particles then were ground in 3 g of clean sand (Fluka) and extracted using described methods. Petrographic analysis (R&D Carbon Petrography, Monroeville, CA, USA), used historically to identify different coal particles [19], also was used to identify the amount of coal-derived particles and wood and plant material in the various fractions.

Desorption kinetics. Following previously described meth-

Table 1. Properties of activated carbons used in amendment studies. Surface area data provided by manufacturer

Carbon	Manufacturer	Type	Specific surface area (m^2/g)	Sizes tested (mm)
TOG®	Calgon Carbon (Pittsburgh, PA, USA)	Virgin	935	0.074–0.177 0.074–0.297
Filtrasorb® 400 (F400)	Calgon Carbon	Virgin	1,100	0.074–0.177 0.5–1
Aquacarb® 830 (AC830)	Westates Carbon (Santa Fe Springs, CA, USA)	Virgin	900	0.074–0.177 0.595–2.38
Aquacarb RS (ACRS)	Westates Carbon	Reactivated	900	0.074–0.177 0.595–2.38

ods [8,18,20], untreated sediment from a control bottle of the series 1 contact experiment was used in desorption studies lasting for 55 d to determine the mass of readily desorbing DDT and metabolites.

Activated carbon amendment tests

Activated carbon. As in previous studies [12,16], contact experiments were set up to measure activated carbon amendment effectiveness. In this study, three virgin activated carbons, including Filtrasorb® 400 (Calgon Carbon, Pittsburgh, PA, USA), TOG® (Calgon Carbon), and Aquacarb® 830 (Westates Carbon, Santa Fe Springs, CA, USA), were tested at differing sizes and doses. Aquacarb RS (Westates Carbon), a reactivated carbon, also was tested. Carbon properties and sizes tested are shown in Table 1. Before use, the activated carbons were boiled for 5 min to remove air pockets from pores.

Sediment-activated carbon contact: Series 1 comparing types and size of carbons. In the first series of tests, the four different activated carbons were used both in the as-received size provided by the manufacturer and as a common size achieved by grinding to 0.074 to 0.177 mm. Grinding the activated carbons does not result in an appreciable change in surface area. Large glass bottles were filled with surface-sediment collected in February 2003 (90 g dry sediment/L) followed by the addition of 3.2 weight % activated carbon, on a dry mass basis. This dose corresponds to about 1.1 times the existing total organic carbon in the sediment. Untreated sediment controls were set up without addition of activated carbon. The bottles were rolled at 2 to 3 rpm for 31 d, and then the sediment was used for SPMD and aqueous equilibrium tests.

Sediment-activated carbon contact: Series 2 comparing dose of reactivated carbon. Aquasorb RS (ACRS) reactivated carbon was tested further at the as-received size and at a smaller size at three different doses each, for a total of six treatments plus an untreated control. Glass bottles were filled with surface-sediment collected in February 2003 (170 g dry/L) followed by the addition of either as-received sized (0.595–2.38 mm) at 3.2, 6.4, and 9.6 weight % or ground reactivated carbon (0.074–0.177 mm) at 0.8, 1.6, and 3.2 weight %, on a dry mass basis. The bottles were rolled at 2 to 3 rpm for over two years, with aliquots for SPMD tests removed after 1, 6, and 26 months.

Aqueous equilibrium concentration. The glass bottles from series 1 containing treated or untreated sediment were filled with deionized water and 1 g/L sodium azide. The bottles were rotated at 2 to 3 rpm for 14 d, after which the sediment/water mixture was allowed to settle. Duplicate water samples then were taken from the bottles, and colloids were removed using a flocculation technique described previously [21]. The aqueous phase was extracted with hexane following U.S. EPA method 3510C (http://www.epa.gov/sw-846/pdfs/3510c.pdf) for liquid-liquid extraction. All extracts were cleaned and analyzed for ΣDDT using the described method.

Semipermeable membrane devices. The SPMDs are biomimetic devices used for assessing the passive uptake of DDT in water [22]. Custom-made SPMDs 5-cm long and filled with 0.05 g triolein (EST Labs, St. Joseph, MO, USA) were used. Approximately 20 g of activated carbon treated or untreated sediment from series 1 and 2 experiments was added to a 40-ml clear glass vial. The vial was filled with deionized water and 1 g/L sodium azide and rotated for 14 d. Cleanup and dialysis of SPMDs followed previously described procedures [12]. All extracts were cleaned and analyzed for ΣDDT using the described method.

Quality assurance and control. Validation of procedures was completed prior to sample analysis. Spiked clean sediment matrices achieved ΣDDT recovery of 76 to 91% across six replicates. Blank sediment matrix and method replicates reported no ΣDDT. Liquid-liquid extraction achieved 93 to 100% recovery of ΣDDT across three replicates. The calculated method limit of detection was 0.1 μg/L in all final extracts. However, procedurally, sufficient sample was used to achieve levels above 1 μg/L for all metabolites in final extracts, which corresponds to 0.5 ng DDT/g dry sediment.

RESULTS AND DISCUSSION

Characterizing contamination in sediment

Vibracores. The vibracore sampling was used to quantify DDT concentration in soft, Young Bay Mud that overlies historic, hard Old Bay Mud. The vibracores also were used to assess whether the nominal 12-inch (30.5 cm) sand layer [23] applied after dredging was intact six years later. A typical vibracore collected by PNNL in May 2003 contained Young Bay Mud, which is indicated by softer consistency, fine grain size, and a dark gray to black color near the sediment-water interface [5]. Table 2 describes the consistency and measured DDT concentrations in the strata of the three sampled vibracores. Core Heck 03-01, located across the channel from the hotspot delineated by PNNL, contained 1 foot (30.5 cm) of Young Bay Mud with the highest concentration in all the samples (252 mg/kg ΣDDT) with tan Old Bay Mud below. No sand was noted between the Young Bay Mud and Old Bay Mud layers. The core taken from the middle of the channel, Heck 03-03, contained highly contaminated Young Bay Mud (12.7–62.1 mg/kg ΣDDT) overlying Old Bay Mud containing ΣDDT at levels less than the 0.59 mg/kg ΣDDT remediation goal (0.1–0.4 mg/kg ΣDDT). Again, no distinct sand layer was noted in the core. In the core Heck 03-05, located closest to

Table 2. Sediment descriptions and sum DDT (ΣDDT) concentrations in vibracores containing Young Bay Mud (YBM) and Old Bay Mud (OBM). Concentrations are an average of three replicates with a standard deviation (SD) or two replicates with the relative percent difference (RPD) reported

Core Heck 03-01		
Depth interval (cm)	Description	ΣDDT (mg/kg)
0–12 inches (0–30.5)	Black YBM, fine silt and clay	252 (SD: 1%)
12–27 inches (30.5–69)	Tan OBM with sand	
27–40 inches (69–102)	Tan OBM more clay, stiffer, denser	
Core Heck 03-03		
Depth interval (cm)	Description	ΣDDT (mg/kg)
0–10 inches (0–25)	YBM, silty clay, smooth, black	12.7 (SD: 25%)
10–16 inches (25–41)	YBM, black clay, some sandy texture	62.1 (SD: 9%)
16–20.5 inches (41–52)	OBM, gray clay	
20.5–24.5 inches (52–62)	OBM, brown	3.2 (RPD: 11%)
24.5–30 inches (62–76)	Sand mixed with clay, reddish brown	0.4 (RPD: 11%)
30–34 inches (76–86)	OBM, brown stiff smooth clay	0.1 (RPD: 4%)
34–36 inches (86–91)	Sandy clay, brown	
Core Heck 03-05		
Depth interval (cm)	Description	ΣDDT (mg/kg)
0–1 inches (0–2.5)	Black YBM, clay, soft	7.5 (SD: 4%)
1–24 inches (2.5–61)	Black YBM, clay, soft	14.7 (SD: 9%)
24–27 inches (61–69)	Olive green sand	15.4 (SD: 10%)
27–34 inches (69–86)	YBM, black	151 (SD: 7%)
34–48 inches (86–122)	OBM, gray clay, dry crumbly	0.9 (SD: 29%)

the hotspot, Young Bay Mud was found both above and below a 3-inch (7.6 cm) sand layer. In this location, a highly contaminated Young Bay Mud layer (151 mg/kg ΣDDT) lay between the sand layer and Old Bay Mud containing levels of ΣDDT slightly above the remediation goal.

These three cores show highly contaminated Young Bay Mud exists across the channel and up to 2 feet (61 cm) in depth. At the water-sediment interface, the Young Bay Mud contains DDT at levels ranging from 4 to 428 times the remediation goal. These high levels act as a DDT source to the water column and to the benthic biota in the area. A distinct sand layer was noted in only one of the three cores reported here, and in a total of only three of 16 vibracores collected throughout the channel by PNNL in 2003 [5]. Therefore, the sand layer applied after dredging with an expected depth of 12 inches (30.5 cm) [23] either was not applied evenly or has since washed away. Also, because highly contaminated sediment was found below the sand layer in core Heck 03-05, this sample suggests not all Young Bay Mud was removed during dredging prior to sand layer application.

Particle analysis

Sediment characterization quantified the amount of ΣDDT in different size fractions and types of particles. Analysis of sieved grain sizes in a mixed sample of surface sediment from Lauritzen Channel shows the sediment clay/silt (<63 μm) fraction comprises 82% of the sediment mass and contains 77% of the ΣDDT (in Supplemental Data; http://dx.doi.org/10.1897/07-179.S1). The light density sediment fraction comprises 6% of the total mass and contains 37% of ΣDDT. Investigations by Ghosh et al. [9] with Hunters Point sediment from South Basin, San Francisco Bay, found 5 to 7% of the total sediment mass resided in the light fraction but accounted for 68% of the PCBs. For Lauritzen Channel sediment, light particles certainly contain a greater absolute amount of DDT by mass, but not to nearly to the extent seen in other harbor sediments. Petrographic analysis of Lauritzen Channel sediment identified very little black carbon particles such as coal, coke, or byproduct-related materials (0.6% by volume). In contrast to the dominance of charcoal and coal in the Hunters Point light-density sediment fraction, the Lauritzen Channel light-density sediment fraction mostly consists of wood or humic plant material.

Extraction of different classes of particles from Lauritzen Channel sediment found that black and wood particles had the highest ΣDDT concentrations; minerals and shell had statistically lower concentrations by about one order of magnitude (in Supplemental Data; http://dx.doi.org/10.1897/07-179.S1). The light density particles therefore have significantly (t-test, $p = 0.05$) higher DDT concentrations, likely due to the higher sorptive capacity of plant-derived organic and black carbon particles. However, as Lauritzen Channel sediment lacks many black carbonaceous particles and mostly is comprised of fine-grained, inorganic minerals, 94% as revealed by both petrography and density separation, the majority of ΣDDT (61%) resides in the fine, heavy density mineral fraction. Because of these Lauritzen Channel sediment characteristics, adding small amounts of highly sorptive activated carbon to the sediment likely would have a significant effect on the partitioning and availability of DDT.

Desorption from sediment

Figure 2 shows the measured desorption of *p,p′*-DDT and its metabolites into the water over 55 d. At the end of the test period, the extent of desorption of DDT metabolites exceeded 57% of the total mass. In contrast, the *p,p′*-DDT desorbed to a much lesser extent (20%) in the same time interval. The same phenomenon of more rapid release and uptake of DDT metabolites compared to *p,p′*-DDT was noted in aqueous equilibrium concentration tests and SPMD uptake data, as sub-

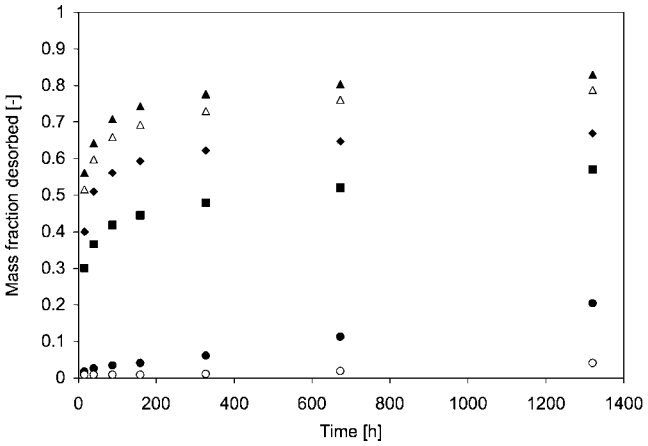

Fig. 2. Mass fraction of DDT and its metabolites desorbed from Lauritzen Channel (Richmond, CA, USA) untreated sediment over 55 d. Each point is an average of two replicates. Data are shown for *p,p′*-DDT (●), *o,p′*-DDT (○), *p,p′*-dichlorodiphenyldichloroethane (DDD) (▲), *o,p′*-DDD (△), *p,p′*-dichlorodiphenyldichloroethylene (DDE) (■), and *p,p′*-dichlorodiphenylmonochloroethylene (DDMU) (◆).

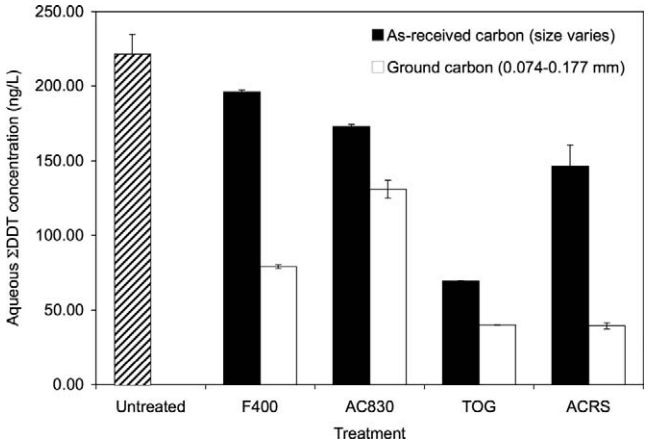

Fig. 3. Aqueous sum DDT (ΣDDT) concentrations for Lauritzen Channel sediment (Richmond, CA, USA) amended with 3.2 weight % of four types of activated carbon (F400, AC830, TOG, ACRS) for one month, comparing the as-received manufacturer size and a smaller size. The average and range of two or three replicates are represented.

sequently discussed. Desorption did not reach an equilibrium plateau during the 55-d test period as shown in Figure 2. These results show *p,p′*-DDT is desorbed less readily from Lauritzen Channel sediment in the time frame of the activated carbon tests. Jafvert et al. [24] noted a similar extreme desorption resistance for DDT from sediment over 46 d of gas-purged–induced desorption with DDT, DDD, and DDE averaging of 22, 58, and 75% of release, respectively. These data show DDT metabolites exhibit faster release and thus probably greater bioavailability than parent DDT.

With such widespread residual contamination and continued bioavailability of ΣDDT to the water column, the use of an alternative means for sediment remediation is appealing. Thus, activated carbon amendment was evaluated in feasibility tests with Lauritzen Channel sediment as a possible method to sequester residual DDT.

Activated carbon amendment

Series 1 comparing types and size of carbons. The results of this study show that both activated carbon type and size affect the extent of reduction in SPMD uptake or aqueous equilibrium concentration after sediment treatment. Figure 3 shows the aqueous equilibrium concentrations for the four different types of carbon at two different sizes. Untreated sediment has an aqueous equilibrium concentration of 222 ng/L ΣDDT. The as-received sized virgin coal carbons F400 and AC830 produce modest (15 and 19%, respectively) reduction in aqueous phase ΣDDT as compared to the average untreated control. In these short-term tests, the effect of grinding the activated carbon was significant, resulting in 66 and 43% reduction, respectively. Higher effectiveness was noted for type TOG carbon, which has been used in previous studies with PCBs [12], as well as ACRS, a reactivated carbon. Virgin type TOG activated carbon in its as-received and ground sizes showed 67 and 83% reduction in aqueous phase ΣDDT concentration. The ground, reactivated ACRS carbon showed a similar reduction in aqueous phase ΣDDT (83%), proving it as an alternative to more expensive virgin carbons for sediment treatment. If the 83% reduction noted in aqueous equilibrium studies was achieved for dissolved ΣDDT concentrations measured in the field at the north end of Lauritzen Channel in 2002 and 2003 [5], the remediation goal of 0.59 ng/L would be nearly met in 2002 (4.51 ng/L reduces to 0.77 ng/L ΣDDT), and water quality would have been much improved in 2003 (22 ng/L reduces to 3.7 ng/L ΣDDT). The overall reductions noted in ΣDDT concentrations are similar to the 87% reduction obtained for aqueous PCB concentration after one month of treatment of Hunters Point sediment with 3.4 weight % virgin carbon [12].

The same trends of reduced DDT availability in activated carbon–amended sediment were apparent in the SPMD uptake data. The results from batch experiments are presented as reduction in SPMD uptake compared to untreated sediment in Figure 4. The highest reduction in SPMD uptake was seen for the type TOG (80%) and ACRS (91%) ground carbons. The data in Figures 2 through 5 indicate the system is limited kinetically, because differing sizes of the same carbon do not perform alike. Longer treatment times, therefore, would increase effectiveness, as observed in the series 2 experiments

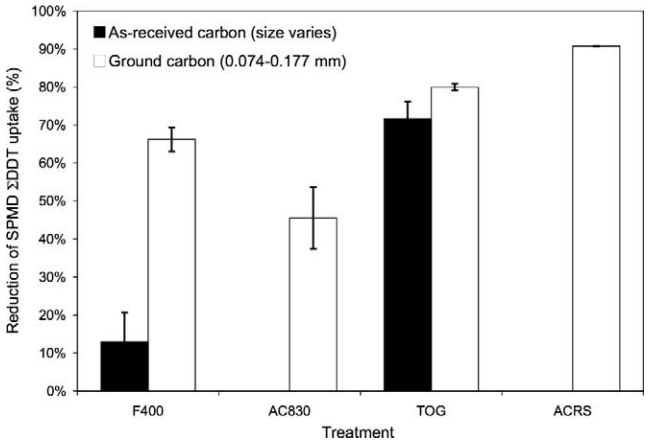

Fig. 4. Reduction in sum DDT (ΣDDT) uptake in semipermeable membrane device (SPMD) for Lauritzen Channel (Richmond, CA, USA) sediment after amendment with 3.2 weight % of activated carbon for one month using the as-received manufacturer size and a ground size. The average and standard deviation of three replicates are represented for four different activated carbons (F400, AC830, TOG, ACRS) and two particle sizes.

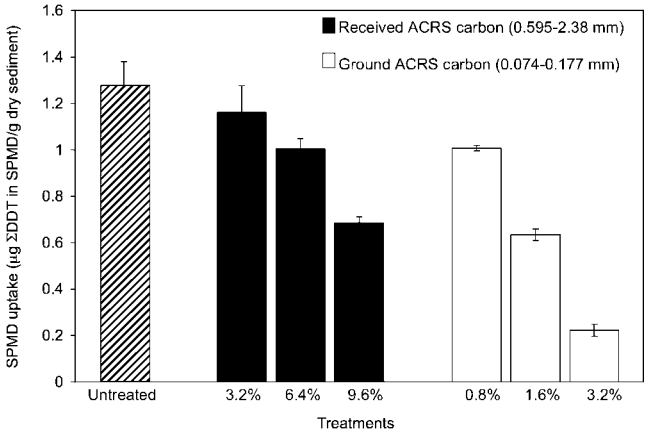

Fig. 5. Sum DDT (ΣDDT) semipermeable membrane device (SPMD) uptake for Lauritzen Channel (Richmond, CA, USA) sediment amended with different doses of Aquasorb® RS reactivated carbon (ACRS) for one month in the received manufacturer size and a grinded size. The average and standard deviation of three replicates are represented.

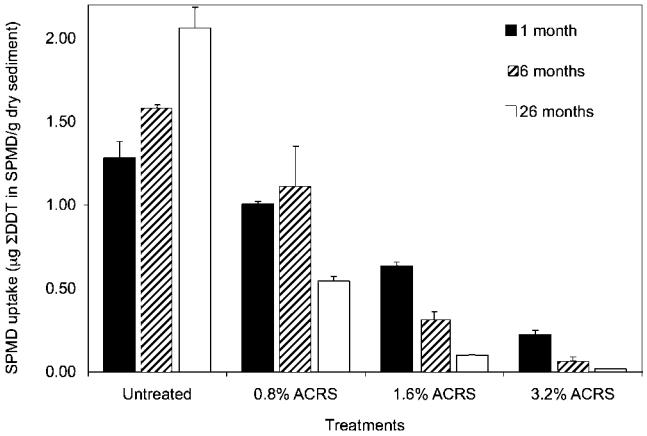

Fig. 6. Sum DDT (ΣDDT) uptake in semipermeable membrane device (SPMD) for Lauritzen Channel (Richmond, CA, USA) sediment amended with different doses of ground (0.074–0.177 mm) Aquasorb® RS reactivated carbon (ACRS) for one, six, and 26 months of contact time. The average and standard deviation of three replicates are represented.

described below (see *Series 2 comparing dose of reactivated carbon* section).

As shown in the desorption experiments, *p,p′*-DDT desorbs from Lauritzen Channel sediment to a much less extent than its metabolites. A similar trend was noted in the case of the mass of DDT partitioning into the SPMDs. A total of 24% of *p,p′*-DDD transferred from the untreated sediment to the SPMD over two weeks as compared to 1% of *p,p′*-DDT. Octanol-water partitioning coefficients (K_{OW}) and a linear free energy relationship, reported in Schwarzenbach et al. ([25], Eqn. 9–26a), were used to estimate organic carbon-normalized partition coefficients (K_{OC}) for the *p,p′* isomers of DDE, DDE, and DDT.

$$\log K_{OC}\ [\text{L/kg}] = 0.74 \cdot \log K_{OW} + 0.15$$

Apparent experimental K_{OC} values were calculated using

$$C_s/C_w = K_d = f_{OC} \cdot K_{OC}$$

where the distribution coefficient (K_d) is the quotient of the aqueous equilibrium concentration data for sediment (C_s) and water (C_w) for untreated Lauritzen Channel sediment. As reported in Table 3, apparent log K_{OC} values of 6.6, 6.3, and 8.3 for *p,p′*-DDE, DDD, and DDT, respectively, were calculated using the measured native organic carbon content (f_{OC} = 2.8%). The apparent K_{OC} values for *p,p′*-DDE and *p,p′*-DDD are two orders of magnitude greater than those estimated from literature [25]. The observed K_{OC} for *p,p′*-DDT is nearly four orders of magnitude greater than estimated values, again showing the low desorption potential of *p,p′*-DDT from Lauritzen Channel sediment.

Series 2 comparing dose of reactivated carbon. Because reactivated carbon was found highly effective in series 1 tests, complimentary tests examining different doses and treatment times with type ACRS reactivated carbon were completed. Figure 5 shows SPMD uptake results for dosing with 3.2, 6.4, and 9.6 weight % as-received ACRS carbon and 0.8, 1.6, and 3.2 weight % ground carbon in one-month treatment studies. For the larger carbon, as the dose was increased, SPMD uptake was reduced by 8, 20, and 46%. Doubling the dose corresponds to near doubling the effectiveness. For the ground ACRS carbon, an increase in SPMD uptake reduction with increasing dose also was noted (21, 50, 87%).

A numerical mass transfer model developed by Werner et al. [17] estimates reductions in aqueous and SPMD uptake upon addition of activated carbon. This model predicts that, at equilibrium, increasing the dose will increase proportionally the observed reduction in aqueous phase or SPMD PCB concentration. Experimental data from this study agrees with the trend suggested by the model's prediction of dose-effect.

Figure 6 shows ΣDDT uptake in a SPMD after 1, 6, or 26 months of treatment with increasing doses of ground (0.074–

Table 3. Apparent versus literature estimated partitioning coefficients of DDT compounds in untreated Lauritzen Channel (Richmond, CA, USA) sediment

	C_s mg/kg	C_w ng/L	Apparent Log K_d[a]	Apparent Log K_{OC}	Literature values Log K_{OW}[b]	Estimated Log K_{OC}[b]
p,p′-DDMU[c]	1,061.7	24.0	4.6	6.2	—	—
p,p′-DDE[d]	876.6	8.6	5.0	6.6	5.7	4.4
o,p′-DDD[e]	2,175.6	47.0	4.7	6.2	—	—
p,p′-DDD	7,843.7	128.8	4.8	6.3	5.5	4.2
o,p′-DDT	465.1	2.9	5.2	6.8	—	—
p,p′-DDT	5,716.3	1.0	6.7	8.3	6.4	4.9

[a] K values in L/kg.
[b] K_{OW} values and linear free energy relationship from Schwarzenbach et al. [25].
[c] DDMU = dichlorodiphenylmonochloroethylene.
[d] DDE = dichlorodiphenyldichloroethylene.
[e] DDD = dichlorodiphenyldichloroethane.

0.177 mm) type ACRS reactivated carbon. As the treatment time increases, the uptake of ΣDDT in a SPMD was reduced significantly. The effectiveness of reactivated carbon for sequestering DDT was not diminished over 26 months of treatment, demonstrating that DDT was not rereleased from the activated carbon. For the 3.2 weight % dose, a reduction of 99% in SPMD uptake was noted after 26 months of contact with reactivated carbon. This data shows the positive effect of dose and that a lower dose (1.6%) can produce high reduction (95%) at long treatment time.

CONCLUSION

Results from this work show that, despite extensive environmental dredging, high levels of available ΣDDT remain in Lauritzen Channel sediment. DDT metabolites are less strongly sorbed to the sediment than parent DDT. Amendment of field-collected residual sediments with activated carbon resulted in large reductions in aqueous equilibrium concentration and SPMD uptake. Aqueous phase concentration was reduced up to 83% with one month of treatment, and SPMD uptake reduced from 91 to 99% during 1 to 26 month treatment with 3.2 weight % reactivated carbon. Effectiveness of treatment increases with contact time and decreasing activated carbon particle size, and varies for different activated carbons with similar surface areas. Reactivated carbon was found to be as effective as virgin carbon in sequestering ΣDDT. Reactivated carbon is significantly less costly than virgin carbons, and thus it has a cost-advantage in field-scale treatment.

In total, the present study supports the use of activated carbon amendment to manage residual DDT contamination that may remain after environmental dredging. These results show the effectiveness of activated carbon amendment to reduce the availability of DDT in field sediments, as found previously for PCBs and polycyclic aromatic hydrocarbons. Hence, activated carbon amendment might be especially useful at sites with a mixture of legacy hydrophobic pollutant contamination to achieve the ideals of high containment effectiveness, limited taxing of the ecosystem, and low costs.

SUPPORTING INFORMATION

Table S1. Distribution of DDT in size and density fractions of mixed surface sediment sample from Lauritzen Channel collected February 2003.

Fig. S1. DDT concentrations on particles greater than 0.25 mm from mixed Lauritzen Channel surface sediment sample. Particles identified via light microscopy included heavy particles of shell (♦) and mineral (■), and light particles of plant materials (*), blacks (probably black carbon, ●), and charred (×) and uncharred (▲) wood.

Both found at DOI: 10.1897/07-179.S1 (127KB PDF).

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INFLUENCE OF SOOT CARBON ON THE BIOACCUMULATION OF SEDIMENT-BOUND POLYCYCLIC AROMATIC HYDROCARBONS BY MARINE BENTHIC INVERTEBRATES: AN INTERSPECIES COMPARISON

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Abstract—The sorption of polycyclic aromatic hydrocarbons (PAHs) to soot carbon in marine sediments has been hypothesized to reduce PAH bioavailability. This hypothesis was tested for eight species of marine benthic invertebrates (four polychaete worms, *Clymenella torquata*, *Nereis virens*, *Cirriformia grandis*, and *Pectinaria gouldii*, and four bivalve mollusks, *Macoma balthica*, *Mulinia lateralis*, *Yoldia limatula*, and *Mya arenaria*) that span a wide range of feeding behavior, ability to metabolize PAHs, and gut chemistry. Organisms were exposed for 20 d to two PAH-spiked sediments, one with soot and one without soot. The soot treatment generally resulted in lower bioaccumulation than the no soot treatment, though the differences between treatments were not significant for all species. All but one species accumulated significant PAH concentrations in their tissues from the soot treatment, indicating that soot-bound PAH cannot be dismissed as unavailable to infaunal benthic biota. Bioaccumulation factors were correlated negatively to both the organisms' ability to metabolize PAHs and the gut fluid contact angle, supporting the hypotheses that high PAH metabolism results in lower bioaccumulation factors and bioavailability of PAHs may be limited partially by PAH solubilization in the gut lumen. The variability in bioaccumulation due to the soot treatment was much less than the variability between species and between PAH analytes. Comparatively low bioaccumulation was observed in *Nereis virens*, a species commonly used in bioaccumulation tests. These results suggest that more effort is needed in understanding the salient characteristics of species present in a threatened environment, rather than focusing solely on the sediment geochemistry (e.g., soot and organic carbon content) and contaminant characteristics when predicting ecological risk of PAH-contaminated sediments.

Keywords—Polycyclic aromatic hydrocarbon Bioavailability Soot Gut fluid Metabolism

INTRODUCTION

Soot carbon, also referred to as black carbon, can be described as a carbonaceous residue resulting from the incomplete combustion of organic matter [1]. The term soot carbon has been used to describe many types of carbon residues resulting from high temperature alteration of organic carbon, including charcoal, fly ash, carbon black, and elemental carbon, which hereafter will be referred to as soot. Soot particles have a high degree of aromatic structure and contain reactive oxygenated surface functional groups (e.g., carbonyls, carboxyls, and ethers) [2]. Recent studies have suggested that the affinity of polycyclic aromatic hydrocarbons (PAHs) for soot carbon is linked to their planar aromatic structure and it is hypothesized that the two interact by establishing pi–pi bond interactions [3].

The presence of soot carbon in marine sediments has been proposed to explain observed PAH organic carbon normalized distribution coefficients (K_{oc}) that are much higher than predicted based on equilibrium partitioning to sedimentary organic carbon [4–6]. Research also has shown that a very high proportion of PAHs in sediment likely are associated with soot-like particles and that partitioning to the soot-carbon fraction of the sedimentary organic carbon can dominate equilibrium partitioning processes [7,8]. Soot is ubiquitous in marine sediments, with U.S. coastal waters containing from 0.11 to 6.6 mg/g dry sediment of black carbon (3–14% of the total organic carbon) [9,10]. Gustafsson et al. [4] have proposed that sediment quality criteria for PAHs should be revised to reflect the important role of soot carbon on PAH partitioning. This is accomplished through an extension of equilibrium partitioning to account for the strong sorption of PAHs to soot carbon using a soot carbon partition coefficient (K_{sc}). The model of Gustafsson et al. [4] has been further refined by Accardi-Dey and Gschwend [7] to include nonlinearity of PAH sorption to soot carbon:

$$K_d = f_{oc} \cdot K_{oc} + f_{sc} \cdot K_{sc} \cdot C_w^{n-1} \quad (1)$$

where K_d is the sediment–water partition coefficient, f_{oc} and f_{sc} are the fraction organic and soot carbon in the sediment, respectively, C_w is the truly dissolved PAH concentration in sediment pore waters, and n is the Freundlich exponent of the PAH-soot carbon relationship being investigated.

Little research has been conducted to directly evaluate the effect of soot carbon in sediments on PAH bioavailability to infaunal biota. Elevated petrogenic (e.g., characterized by a signature of high alkylated/parent PAH ratios) relative to pyrogenic PAH in organism tissues suggest that the combustion-derived pyrogenic PAH are less bioavailable [11]. However, the composition of PAHs in organism tissues also may be affected by differences in metabolism/depuration rates rather than differences in bioavailability [12,13]. The few studies that have endeavored to investigate directly the effect of soot carbon on PAH bioavailability suggest that the presence of soot

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does result in reduced bioavailability [2,14]. Lamoureux and Brownawell [15] found reduced PAH desorption and bioavailability to a deposit-feeding polychaete, *Nereis succinea*, from soot-amended sediments. However, the difference in bioaccumulation between the soot/no soot treatments was not as pronounced as the difference in the rates of desorption in seawater measured using XAD (Sigma-Aldrich, Milwaukee, WI, USA) resin.

High surfactant levels in the gut fluids of deposit-feeding invertebrates are one reason that soot-bound PAHs still may be bioavailable to some species despite strong binding. Solubilization of sediment-bound PAHs by gut fluids of deposit-feeding benthic invertebrates has been shown to correlate well with assimilation efficiency and bioaccumulation [16–18]. Furthermore, contact angle, a simple estimation of the surfactant strength of gut fluids, has been shown to correlate well with the extent of PAH desorption by organism gut fluids across several different phyla [19]. Polycyclic aromatic hydrocarbon desorption from various soot-like matrices in the gut fluid of *Arenicola marina*, a deposit-feeding polychaete, has been shown to exceed predictions by soot-corrected equilibrium partitioning [20].

Benthic invertebrates also vary in other important characteristics that may affect PAH bioaccumulation and account for deviations from equilibrium partitioning predictions derived from bulk sediment properties. Such factors include feeding behavior (e.g., head-down vs surface deposit feeders, filter feeders, carnivores, etc.), size-selective particle ingestion (e.g., selection for or against organic carbon and contaminant-enriched size fractions), gut passage time, depletion of PAH concentrations in the local environment (e.g., enhanced degradation and contaminant flux across sediment/water interface in infaunal burrows) [21–26]. The ability of organisms to metabolize and excrete PAH also has been shown to be related to bioaccumulation, where species with limited metabolic ability tend to accumulate higher PAH concentrations in their tissue [27–29]. Present approaches to bioaccumulation modeling generally ignore species-specific biology (other than total lipid content and, in some cases, growth rates) that may affect the apparent steady state concentration of PAHs in organism tissues.

The nature of the particular PAH being examined also is important to consider. Field studies show that bioaccumulation of PAHs generally either decreases with increasing octanol–water partition coefficient (K_{ow}) or exhibits a maximum at midranged logK_{ow} values of 5 to 5.5 (possible reasons for reduced accumulation of lower K_{ow} compounds include faster depuration rates and/or depletion of microhabitat in the exposure sediments) [12,30–32].

This study was designed to examine the influence of soot on the bioavailability of PAHs to benthic organisms that vary in their gut fluid surfactant strength and metabolic abilities. This was accomplished by exposing eight infaunal marine species to two PAH-spiked sediments, one with and one without soot. Bioaccumulation factors ([BAFs], from sediment to organism) for a diverse mixture of PAHs were then calculated and interpreted in relation to the treatment effect as well as other physiological parameters thought to affect PAH bioaccumulation (e.g., PAH metabolism, feeding mode, and gut fluid surfactant strength). We hypothesized that soot would limit the bioavailability of PAHs, that sediment-sorbed PAHs would be accumulated to greater extent by deposit-feeding as compared to filter-feeding organisms, the soot treatment effect would be minimized in deposit-feeding organisms with higher levels of gut surfactancy, and organisms with greater ability to metabolize PAHs would accumulate them to a lesser extent that those with limited metabolic capability.

MATERIALS AND METHODS

Organisms

Organisms used in this experiment were collected in September 2000 from intertidal areas adjacent to Ponquogue Bridge, Hampton Bays, New York, USA (*Clymenella torquata*, *Nereis virens*, *Cirriformia grandis*) and from intertidal areas in Stony Brook Harbor, New York, USA (*Mya arenaria*) or obtained from the Marine Biological Laboratory, Woods Hole, Massachusetts, USA (*Macoma balthica*, *Pectinaria gouldii*, *Mulinia lateralis*, *Yoldia limatula*). Taxonomic groups and feeding types are found in Table 1. The average wet weight of individual organisms ranged between 0.1 and 0.5 g for all species except *M. arenaria* and *M. balthica*, which averaged 1.0 each. Juvenile *M. arenaria* and *M. balthica* were used, but all the other species were adults. All organisms were considered to be from environments with limited hydrocarbon sources that were verified by chemical analysis of organism tissues prior to the start of each experiment. To avoid using organisms compromised by collection or transit to the laboratory, individuals were acclimated to laboratory conditions for at least 48 h prior to starting the experiment.

Gut fluid

Gut fluid from each species was sampled within 24 h of collection from the field by dissecting each organism and inserting a glass capillary tube into the lumen of the midgut to withdraw the gut contents. For bivalves, gut fluid was collected from the vicinity of the crystalline style. Gut fluid samples from between one and twenty individuals were pooled in acid-washed centrifuge tubes to obtain at least 10 μl, sufficient volume for repeated 2 μl analyses, and samples were frozen at −80°C until the time of contact angle measurement. Surfactancy of gut fluids was measured by drop contact angle analysis on a Parafilm™ (Royal Purple, Humble, TX, USA) coated slide following the methods described in detail by Mayer et al. [33] and Ahrens et al. [16].

Chemicals

The following compounds were used to spike exposure sediments: 1-methylfluorene, phenanthrene, fluoranthene, benzo[*a*]anthracene, chrysene, benzo[*a*]pyrene, and 7-methylbenzo[*a*]pyrene (Sigma-Aldrich) and 1-methylphenanthrene and 3,6-dimethylphenanthrene (Ultra Scientific, North Kingstown, RI, USA). LogK_{ow} values for these compounds were obtained from the literature [34], except for 3,6-dimethylphenanthrene and 7-methylbenzo[*a*]pyrene, which were estimated using LeBas molar volume as outlined by Mackay et al. [35] (See Table 2). Surrogate standards added before the extraction of all samples were d10-phenanthrene, d12-benzo[*a*]anthracene, d12-chrysene, and d12-perylene (Supelco, Bellefonte, PA, USA).

Sediment spiking and soot treatment

Exposure sediment was collected from the top 5 cm of benthic grab samples from a reference site in central Long Island Sound (41°08.036′N, 72°52.730′W, 1.8% organic carbon, 92% silt- and clay-size particles). Sediment was sieved immediately to 0.5 mm and stored at 4°C. Seawater used

Table 1. Contact angle, percent of benzo[*a*]pyrene (B[*a*]P) body burden metabolized after a 7-d sediment exposure, and feeding mode of organisms used in this study

	Contact angle[a]	% B[*a*]P metabolized	Feeding mode
Polychaetes			
Clymenella torquata	38 (±2)°	6%[b]	Subsurface deposit feeder
Pectinaria gouldii	39 (±3)°	7%[c]	Subsurface deposit feeder
Nereis virens	47 (±1)°	72%[b]	Carnivore/deposit feeder
Cirriformia grandis	55 (±2)°	14%[b]	Surface deposit feeder
Bivalves			
Mya arenaria	66 (±2)°	36%[b]	Filter feeder
Macoma balthica	68 (±3)°	10%[b]	Surface deposit/filter feeder
Yoldia limatula	72 (±2)°	26%[c]	Subsurface deposit feeder
Mulinia lateralis	77 (±2)°	42%[b]	Filter feeder

[a] Numbers in parentheses are 95% confidence intervals.
[b] Rust et al. [28].
[c] McElroy et al. [44].

throughout the experiment was collected from Flax Pond (Old Field, NY, USA) (28 ppt, ~23°C).

Diesel soot used in the soot treatment was collected from the tailpipes of diesel buses on the campus of Stony Brook University (Stony Brook, NY, USA). Soot was sieved through a 63-μm mesh sieve, placed in a beaker with excess 20% dichloromethane in hexane, and sonicated in a sonic bath for 5 min. Soot was then collected on a glass fiber filter, rinsed with clean hexane, and dried in a drying oven overnight at 65°C. Solvent extraction was performed to remove the majority of PAHs and other hydrocarbon residues that had condensed on the soot deposited in the tailpipes. Removal of most of the native PAHs should have acted to promote sorption of spiked PAHs due to the nonlinearity of PAH soot sorption isotherms [7,36]. The intention was to create a sorbed PAH phase that would be less chemically available than PAHs sorbed to natural sediment organic matter. As described below, the total concentration of PAHs spiked was then within a broad range observed in urban harbor sediments [15]. The sediment for the no soot treatment was spiked by adding a PAH stock solution to an empty glass jar and allowing the excess solvent to evaporate. A 50/50 mixture of sediment and seawater was then added, the jars sealed, and rolled in the dark for one week to ensure a homogenous distribution of PAH in the sediment. The nominal spiking concentration was approximately 1 μg/g dry sediment of each PAH (for a total of 9 μg PAH/g dry sediment).

The soot treatment sediment was prepared in a similar manner by equilibrating a PAH stock solution with extracted diesel soot and seawater for 6 d, adding sediment, and rolling for an additional 24 h. The final exposure sediment for the soot treatment had a nominal soot dosage of 1.93% diesel soot by dry weight. Lamoureux and Brownawell [30] found that diesel soot from these same buses was 36% soot carbon when measured by the thermal oxidation method of Gustafsson et al. [4]. Thus, the soot-amended sediment was 0.69% soot carbon, assuming no soot carbon was present in the control sediment. This soot concentration is on the upper end of the broad range measured in coastal and urban harbor sediments [9,10,15]. The final soot treatment sediment contained 3 μg/g dry sediment (nominal) of each PAH. The soot treatment was dosed at a higher level in anticipation that there would be reduced bioavailability of PAH.

Spiked sediments were allowed to equilibrate and settle in the dark for 5 d after rolling. Overlying water was then removed and discarded. Exposure chambers (solvent-rinsed, 60-ml glass jars) were filled to within 5 mm of the rim with exposure sediment (~85 g wet sediment/jar). Three replicate jars were placed in a plastic bucket and filtered seawater was dripped continuously into the bucket to create a flow-through system. Each exposure jar contained three individuals of each organism, except *M. arenaria* exposure chambers, which contained one organism in each jar due to their larger size.

Tissue and sediment samples from all treatments were collected on days 0 and 20. Organisms from the same exposure jar were combined as one tissue sample and samples from each of the three replicate exposure jars were collected at each time point. Sediment samples were composites of sediment from each of the three jars that were replicate exposures. Tissue and sediment samples were stored in glass at −20°C until extraction.

To determine whether or not 20-d exposures were sufficient to approach steady state body burdens, samples were collected at additional time points for three species, *C. grandis*, *Y. limulata*, and *M. balthica*, at days 5, 10, and 28. No additional PAH bioaccumulation was observed between days 20 and 28, indicating that in this system, 20 d appears to be a sufficient exposure period for small, infaunal benthic organisms to reach a steady PAH body burden.

Sample extraction and quantification

Sediments were extracted using a sonication method. Approximately 1 g of wet sediment was ground to a fine powder in a mortar and pestle with 5 g of anhydrous sodium sulfate to remove water. The dried sediments were then extracted in Teflon® centrifuge tubes using 15 ml of 1:1 acetone:hexane and a sonic probe (Cole-Parmer Instrument, Chicago, IL, USA). A second extraction using dichloromethane (15 ml) was done and the combined extracts concentrated and transferred into hexane using a combination of Kuderna Danish concentrators and nitrogen gas blow-down. Extracts were then purified using a column of 5% deactivated silica.

Tissues were homogenized using a VerTishear tissue homogenizer (Vertis, Gardiner, NY, USA) and then extracted in acetone with a vortex mixer. The extract was separated by centrifugation and removed, and the tissue re-extracted with fresh acetone. Water and hexane were added to the combined extracts to concentrate PAHs into hexane. A subsample of the

hexane was taken for lipid analysis and the remainder cleaned up for gas chromatography/mass spectrometry analysis using a Sep-Pak® Plus silica cartridge (Waters, Milford, MA, USA). Lipid analysis was performed on each tissue sample by gravimetric analysis on a Cahn C-26 automatic electrobalance (Thermo Orion, Beverly, MA, USA).

The PAH concentrations in tissue and sediment samples were quantified on a Hewlett-Packard (Avondale, PA, USA) 5890 series II gas chromatograph equipped with a 7673a autosampler and a 5971a mass selective detector (Agilent Technologies, Wilmington, DE, USA) [37]. Polycyclic aromatic hydrocarbons were quantified relative to the response of the most closely eluting deuterated surrogate standard. Coefficients of variation on replicate analyses of composite sediment samples averaged 12%. For separate samples of tissue or XAD resins, coefficients of variation were higher, averaging 27% and 23% respectively. Recovery of standard additions to tissue and XAD samples averaged 107% and 101%, respectively, of the nominal amount spiked. Total organic carbon content of sediments was determined after removal of inorganic carbon using an excess of 10% HCl. Sediment samples were then rinsed well in distilled water, dried, homogenized, and analyzed on a Carlo Erba EA1108 CHN Analyzer (CE Institute, Milan, Italy).

Accumulation factors and statistics

In this study, two types of accumulation factors were calculated following the nomenclature outlined by Meador et al. [31]. Lipid- and organic carbon–normalized bioaccumulation factors (BAF_{loc}s, g OrgC/g lipid) were calculated using the day 20 lipid-normalized tissue PAH concentrations and the average of days 0 and 20 organic carbon–normalized sediment PAH concentrations. For comparison, BAFs also were calculated without lipid and organic carbon normalization (BAFs g dry sediment/g wet tissue).

All statistical analyses were performed using the SAS System® 8E for Windows (SAS Institute, Cary, NC, USA). Accumulation factors and tissue PAH concentrations were log transformed prior to analysis to better approximate a normal distribution of the data. Three factor analysis of variance and tests for significant differences (e.g., Tukey and Dunnett's) were performed using the general linear model (proc GLM). A p-value of < 0.05 was considered statistically significant. Linear regressions were performed using proc REG.

RESULTS AND DISCUSSION

Sediment PAH analysis

Procedural blanks contained undetectable levels of PAHs. Control sediments had readily detectable PAH levels ranging from 5.5 (1-methylfluorene in control sediment) to 265 (phenanthrene in soot-added control) ng/g, but levels in control sediments were a relatively small fraction (<10%) of the total analyte signal present in day 0 exposure sediments (Table 1). Measured concentrations of analytes from day 0 sediments averaged 116% of the nominal (spiked) concentration for the no soot treatment and 106% of nominal for the soot treatment.

In both treatments, analysis of day 20 sediments showed good retention of PAHs with logK_{ow}s of >5.5, and significant losses of the smaller PAHs (Table 1). The loss of smaller PAHs was less in the soot-amended treatments (15% on average) as compared to the no soot treatments (36% on average). No species-specific effects were observed in day 20 sediment PAH concentrations except for the *Y. limatula* no soot treatment (data not shown), where sediment PAH concentrations were significantly lower than all other no soot treatment sediments ($p < 0.05$, Tukey test). The decrease in sediment PAH concentrations in the *Y. limatula* no soot treatment was likely due to a preferential loss of fine sediment caused by feeding-induced resuspension. To better represent the integrated exposure to sediment PAH concentrations over the time course of the experiment, accumulation factors were calculated using an average of day 0 and day 20 sediment concentrations.

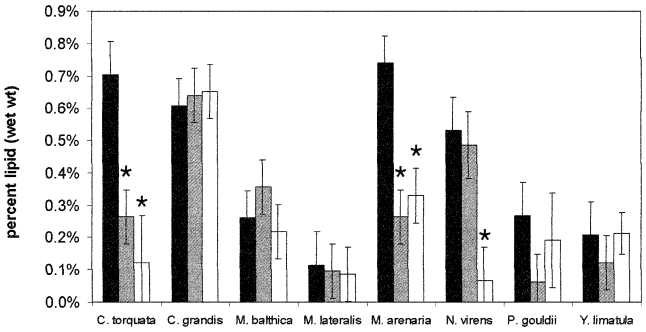

Fig. 1. Percent lipid in organism tissues. An asterisk (∗) over day 20 data indicates a significant change from day 0 ($p < 0.05$, Tukey test). Only *Nereis virens* showed a significant difference between treatments at day 20 ($p < 0.05$, Tukey test). Error bars represent 95% confidence intervals. *Clymenella torquata, Cirriformia grandis, Macoma balthica, Mulinia lateralis, Mya arenaria, N. virens, Pectinaria gouldii, Yoldia limatula.* ■, Day 0; ▨, day 20: no soot; □, day 20: soot.

Organism survival and lipid content

Survival of organisms at day 20 averaged 93 ± 8% (95% confidence interval—no soot) and 81 ± 16% (95% confidence interval—soot). In five of the eight species, lipid values did not vary significantly over time, and only in one species (*N. virens*) were lower values seen in the soot treatment (Fig. 1). Reduction in lipid content over time or with treatment may be an indication that organisms were not feeding/behaving normally during the exposure period (e.g., *C. torquata*, *M. arenaria*, and *N. virens*), which in turn may have affected bioaccumulation results.

Gut fluid analysis

Contact angle measurements indicate that gut fluid of the polychaetes generally is higher in surfactant strength (indicated by lower contact angle) than the bivalve mollusks, and that the surfactant strength of deposit feeders frequently is higher than that of filter feeders (Table 2). This is consistent with the trends observed in other crossphyletic studies of gut fluid characteristics [19,33]. The contact angle presented here for *N. virens* (47°) is very close to previously published results (44–42.5°) [33]. The values for *P. gouldii* (39°) and *M. balthica* (68°) also agree very well with previously reported results of 40° and approximately 67°, respectively [16,38]. The contact angle measured for *C. torquata* (38°), however, is very different from the result of 76° reported by Craig [39]. The methodology of Craig [39] generally was the same, both studies yielded internally consistent results, and the specimen collection site was the same for both studies. The source of this discrepancy, therefore, is unclear at this time.

PAH tissue concentrations

Tissue concentrations of PAH analytes are shown in Figure 2. Despite the variability of individual data points, a three-

Table 2. Results of sediment polycyclic aromatic hydrocarbon (PAH) analysis (ng PAH/g dry sediment)

		No soot treatment			Soot treatment		
Compound	Log K_{ow}[a]	Background[b]	Day 0[c]	Day 20[d]	Background[b]	Day 0[c]	Day 20[d]
1-Methylfluorene	4.97	5.5	891	620*	34	2,890	2,460*
Phenanthrene	4.52	73	973	429*	365	3,120	2,630*
Fluoranthene	5.07	18	1,151	761*	119	3,280	2,670*
1-Methylphenanthrene	5.14	9.5	1,434	981*	59	3,880	3,360*
3,6-Dimethylphenanthrene	5.40	202	1,367	1,020*	225	3,160	2,830*
Benzo[*a*]anthracene	5.91	41	1,033	958	50	2,570	2,300
Chrysene	5.73	153	1,246	1,070	289	3,170	2,820
Benzo[*a*]pyrene	6.20	142	818	793	142	2,380	2,360
7-Methylbenzo[*a*]pyrene	6.41	ND[e]	912	815	ND[e]	2,780	2,740
Total organic carbon (%)		—	1.9	1.8	—	3.2	3.1

[a] Log K_{ow} values taken from the literature [34], except for 3,6-dimethylphenanthrene and 7-methylbenzo[*a*]pyrene, which were estimated using LeBas Molar volume as outlined in Mackay et al. [35].
[b] Background PAH values for unspiked exposure sediment (incl. 1.93% soot for soot treatment sediment), $n = 1$.
[c] Nominal concentrations for each analyte at day 0 were 1,000 and 3,000 ng PAH/g dry sed for no soot and soot treatments, respectively.
[d] An asterisk (*) indicates day 20 sediment concentration is significantly lower than day 0 ($p < 0.05$, Dunnett's test).
[e] ND = not detected.

factor analysis of variance on the log-transformed tissue concentration data from each treatment revealed that, overall, PAH bioaccumulation increased significantly over the 20 d in both treatments for all species except *M. lateralis*. For individual PAHs, significant bioaccumulation was observed for PAHs with logK_{ow}s > 5.3. Absence of significant accumulation and high variability was most pronounced for the lower molecular weight PAHs, which also showed the greatest depletion in sediment concentrations over the 20-d experimental period (Table 1). In addition, bulk sediment concentrations of these more soluble compounds may overestimate exposure concentrations proximate to the organism due to local depletion caused by burrowing and feeding activities of the biota.

Filter feeders tend to bioaccumulate less from contaminated sediments than deposit-feeding species [40], as illustrated here by *M. lateralis*, which showed no significant PAH bioaccumulation from either treatment. However, *M. arenaria*, another filter feeder, did show significant uptake from both treatments. *Mulinia lateralis* has gut fluid and metabolic properties similar to those of *M. arenaria* (Table 2), suggesting that other species-specific factors may be responsible for the observed results. For example, it is known that *M. arenaria* uses post-ingestion particle selection, while *Mulinia edulis* has been shown to use pre-ingestion particle selection that may reduce contaminant exposure for organisms in this genus [41,42]. Exposure sediments also had extensive contact with soft tissues of *M. arenaria* due to the fact that its shell does not close fully and the siphons are very large. This may facilitate diffusive uptake from the sediment in *M. arenaria*, while *M. lateralis* has very little soft tissue in contact with exposure sediments.

Effect of K_{ow} on bioaccumulation

The BAF_{loc} values within a treatment often exhibit a maximum at log$K_{ow} = 5.4$ (3,6-dimethyl-phenanthrene) or show an inverse relationship with K_{ow} (Fig. 3). The notable exception is *M. lateralis*, which showed no obvious trend in BAF_{loc} when plotted against logK_{ow} (likely due to the fact that no significant bioaccumulation was observed in this species for any compound). The peak in BAF_{loc} and/or the inverse relationship between BAF_{loc} and K_{ow} also have been observed in other studies [13,30–32]. In exposures of PAH-contaminated sediments to benthic species, low K_{ow} compounds may show reduced BAF_{loc}s due to their shorter half-life in sediments (e.g., the exposure concentration drops over the course of the exposure), the depletion of the local environment due to burrow irrigation (e.g., diffusive loss from sediment immediately surrounding the organism, enhanced microbial activity near irrigated burrows, or loss from sediments due to bioaccumulation), and faster depuration rates of low K_{ow} compounds from organism tissues [22,23,31]. Reduced bioavailability of high K_{ow} PAHs also is observed consistently in literature reports and may be due to stronger sorption to the sediment and the tendency of organisms to more rapidly metabolize high K_{ow} PAHs [12,30]. The interaction of these many factors often results in lower BAF_{loc}s for low and high molecular weight PAHs and creates a peak in bioaccumulation of midsized compounds. For this study, BAF_{loc}s appear to be highest for compounds with a logK_{ow} between 5 and 5.5, with 3,6-dimethylphenanthrene typically having the highest BAF_{loc} (Fig. 3).

Effect of soot on bioaccumulation

Analysis of biota-sediment accumulation factors revealed that bioaccumulation of PAHs by benthic invertebrates is dependent on many interacting factors (Fig. 3). A three-factor analysis of variance of all the day 20 BAF_{loc}s, with species, compound, and soot treatment as explanatory variables, revealed that compound and species had a significant effect on BAF_{loc} results ($p < 0.0001$), while the soot treatment effect on BAF_{loc} generally was not significant ($p = 0.096$). Although treatment was not significant overall, the treatment interaction terms were all significant ($p < 0.05$ for species·treatment, compound·treatment, and compound·species·treatment). Bioaccumulation factors calculated without lipid and organic carbon normalization (BAFs) showed similar patterns except that the soot treatment variable became significant as well ($p < 0.0001$, $F = 59.6$ data not shown). The impact of lipid and organic carbon normalization on interpretation of BAF values is considered further below.

To examine the effect of soot on PAH bioaccumulation, we calculated the ratio of bioaccumulation factors between no soot and soot treatments for each species. Average ratios across all individual PAH analytes (*i*) between BAFs of no soot (NS) and soot (S) treatments (NS/S) are shown in Table 3 for each

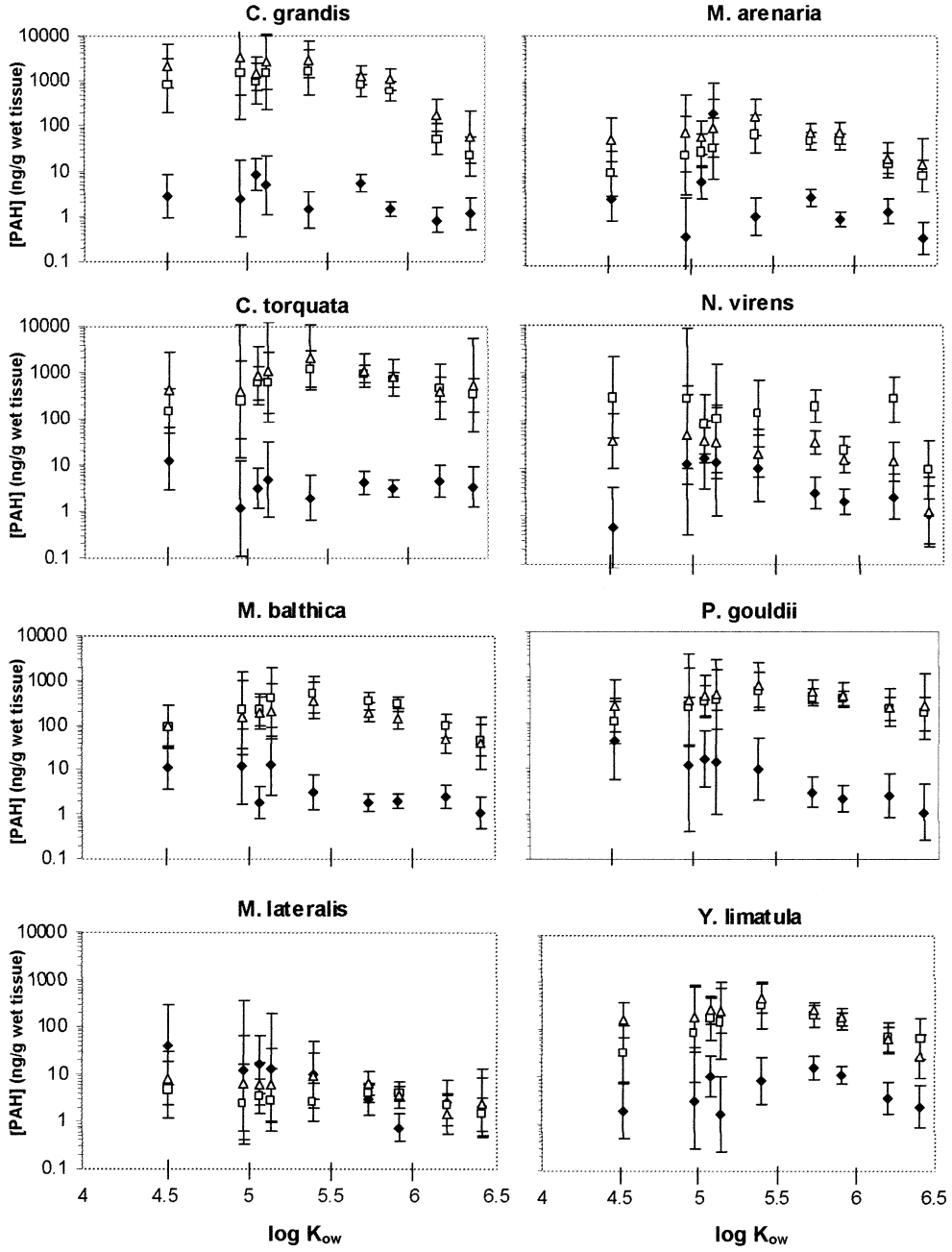

Fig. 2. Tissue concentrations of polycyclic aromatic hydrocarbons (PAHs) at days 0 and 20. All organisms except *Mulinia lateralis* showed significant uptake at day 20 in both treatments. Note that the same scale is used on all graphs and the *y*-axis is on a log scale. Error bars represent 95% confidence intervals. Refer to Figure 1 caption for names of species. ♦, Day 0; □, day 20: no soot; △, day 20: soot.

species and treatment (where n is the number of PAH analytes detected in both treatments)

$$\overline{\text{NS/S}} = \frac{\sum_{i=1}^{n} \frac{\text{BAFns}_i}{\text{BAFs}_i}}{n} \quad (2)$$

NS/S ratios calculated from BAF_{loc} data were greater than one for only five species and only two species (*Y. limatula* and *N. virens*) had a significant difference in BAF_{loc} between treatments. For both species, the NS/S values (2.7; 3.0) are interpreted as a reduction in PAH bioaccumulation in the presence of soot.

The apparent effect of soot is greater when the data are interpreted through analysis of the BAF values, those not normalized to lipid and organic carbon. When comparing BAF values, all species had NS/S ratios greater than one and the reduction in bioaccumulation from the soot treatment was significant for four species (Table 3). The differences between analyses using bioaccumulation factors normalized in different ways are caused in part by higher total organic carbon content (almost a factor of 2) in the soot treatments. Thus, the NS/S is lower when BAFs are normalized to organic carbon. In *N. virens*, decreased lipid in soot treatment organisms also leads to reducing the difference between NS/S when BAFs are lipid normalized. The reduction of soot treatment BAFs relative to the no soot treatment observed (for two species when using BAF_{loc} and for four species when using BAF) supports the results of previous studies that also have shown reduced as-

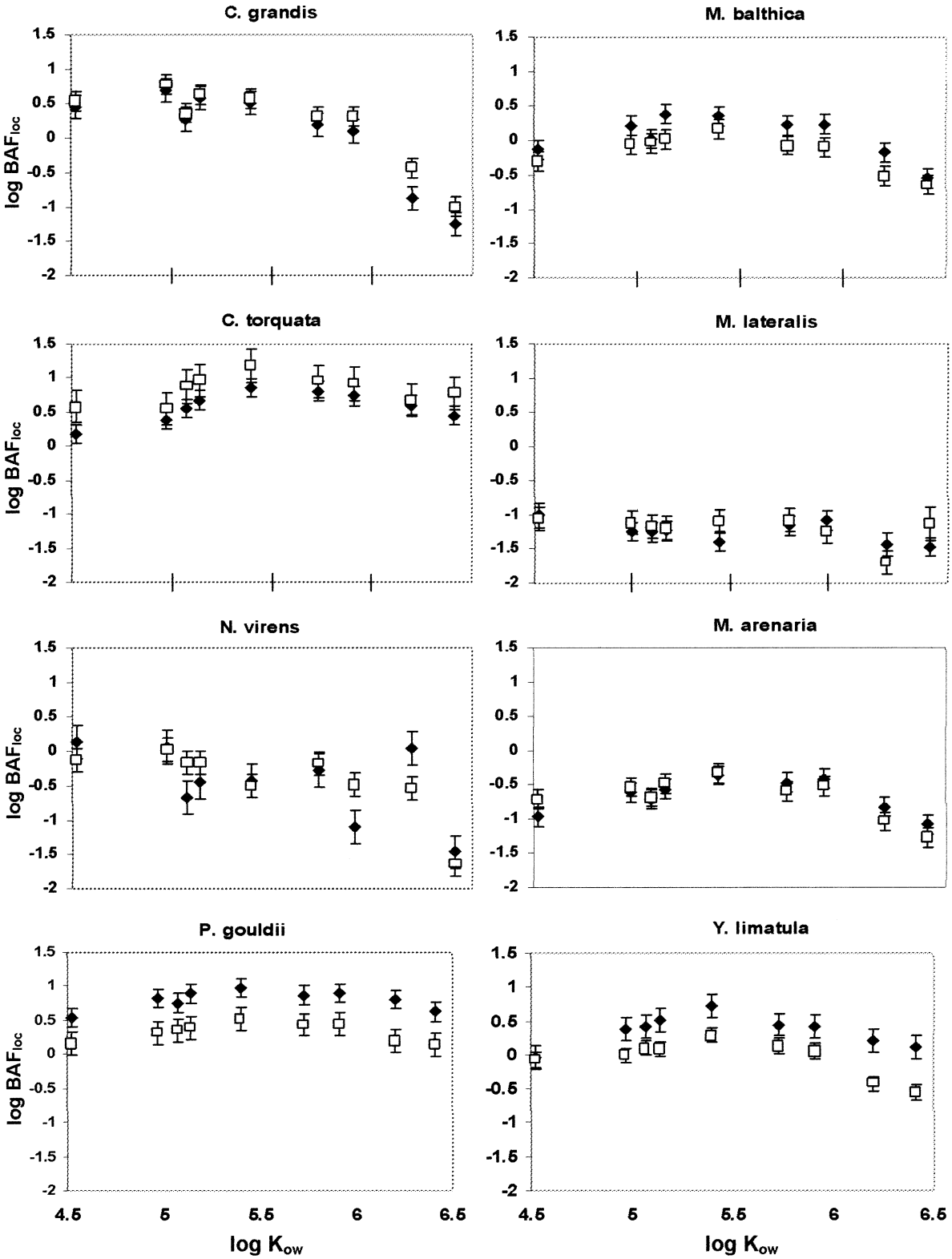

Fig. 3. Log BAF_{loc}s for day 20 data plotted against logK_{ow}, illustrating the difference between treatments for each analyte in each species. The BAF_{loc}s also tend to be highest for polycyclic aromatic hydrocarbons with midranged K_{ow} (e.g., 3,6-dimethylphenanthrene: K_{ow} = 5.4), though this pattern is not totally consistent. Error bars represent 95% confidence intervals. BAF = bioaccumulation factor. Refer to Figure 1 caption for names of species. ♦, No soot; □, soot.

similation efficiency or bioaccumulation of PAHs from soot-enriched sediments [2,15]. The fact that the effect of soot on bioaccumulation is relatively modest for all species is surprising in that it shows significant bioaccumulation of PAHs from sediments amended with high levels of soot (1.9% by weight). These data support the hypothesis of Voparil et al. [20] that digestive exposure to soot-bound PAHs may result in significant bioaccumulation. One cannot assume that soot-

Table 3. Statistical analysis of the effect of soot on bioaccumulation factor (BAF)

	BAF_{loc}			BAF		
Species	NS/S[a]	F[b]	p[b]	NS/S[a]	F[b]	p[b]
Cirriformia grandis	0.7 ± 0.2	1.25	0.27	1.3 ± 0.3	0.38	0.54
Clymenella torquata	0.6 ± 0.2	1.92	0.18	2 ± 0.5	2.6	0.12
Macoma balthica	1.8 ± 0.4	3.52	0.07	4.8 ± 1.2	29.41	<0.0001
Mulinia lateralis	1.0 ± 0.4	0.13	0.72	1.5 ± 0.7	1.01	0.33
Mya arenaria	1.1 ± 0.4	0.02	0.89	1.5 ± 0.5	1.35	0.26
Nereis virens	1.3 ± 1.1	0.05	0.83	22 ± 19	42.94	<0.0001
Pectinaria gouldii	3.0 ± 0.5	11.21	0.003	2.2 ± 0.4	6.07	0.021
Yoldia limatula	2.7 ± 1.1	9.5	0.005	2.6 ± 1.1	8.6	0.0071

[a] NS/S > 1 indicates higher bioaccumulation from the no soot (NS) treatment and NS/S < 1 indicates higher bioaccumulation from the soot (S) treatment (± standard deviation).

[b] Three-factor analysis of variation was used to determine whether the trend of elevated accumulation factors (BAF_{loc}s and BAFs) in the no soot treatment was significant. The F- and p-values are the results of slicing the species*treatment interaction term by species ($p < 0.05$ indicates there is a significant difference between treatments).

bound PAHs are not available to benthic species or that there is a uniform reduction in bioavailability for all benthic species. Further work is required to determine the extent to which these results can be generalized to other sources of soot carbon. It also should be noted that the soot-amended treatment differed not only in the amount of soot carbon present, but also in the amount of total sorbed PAHs (three times higher than the no soot treatment). It is not expected that this modest elevation in PAH concentration would appreciably change bioaccumulation from sediments.

Effects of biological properties on bioaccumulation

Approximately a two order of magnitude difference existed between species with the highest BAF_{loc}s, *C. torquata* and *P. gouldii*, and those with the lowest BAF_{loc}s, *M. lateralis* and *M. arenaria* (Fig. 3). This is consistent with expectations based on the contact angle, metabolic abilities [28], and feeding behavior for these species (Table 2). High surfactant strength, low metabolism, and exclusive deposit-feeding behavior in *C. torquata* and *P. gouldii* would be expected to result in high bioaccumulation of sediment-bound PAHs, while lower surfactant strength, intermediate metabolism, and filter feeding in *M. lateralis* and *M. arenaria* would be expected to result in very low bioaccumulation. As has been observed in other studies, these data illustrate that deposit feeders are at increased risk of accumulating PAHs from bedded sediment than are filter feeders [24,25,40]. The facultative deposit feeders (*M. balthica* and *N. virens*) and the possibly more selective deposit feeders (*C. grandis* and *Y. limatula*) show intermediate bioaccumulation (Fig. 3).

Bioaccumulation of PAHs by strict deposit feeders was influenced significantly by gut fluid surfactant strength. Contact angle is correlated negatively to the day 20 BAF_{loc}s and the slopes of linear regressions are significantly different from zero for both treatments ($p < 0.05$), indicating higher PAH bioaccumulation for species with stronger gut fluid surfactants (Fig. 4). These data are consistent with the findings of several studies that have shown that gut fluids and synthetic surfactants of increasing strength (lower contact angle) can solubilize more PAHs from sediments and that the amount solubilized is approximately equal to the amount assimilated [16–19]. Linear regressions yielded a similar correlation coefficient for the no soot (adjusted, $r^2 = 0.25$) and the soot (adjusted, $r^2 = 0.27$) treatments. Because the gut fluid of a deposit-feeding polychaete has been shown to solubilize more PAHs from soot and other black carbon matrices than predicted by soot-corrected equilibrium partitioning [20], we had hypothesized that the correlation of contact angle to BAF_{loc} results would be higher in the soot treatment due to surfactants playing a more important role. However, our data do not support this.

The extent of benzo[*a*]pyrene (B[*a*]P) metabolism, observed in vivo after 7-d exposures to radiolabeled sediments determined previously for these species [28], also is correlated negatively with BAF_{loc}. The slopes of the regressions are significantly different from zero for both treatments ($p < 0.05$ with adjusted r^2s being 0.49 for the soot and 0.27 for the no soot treatments; Fig. 4), consistent with previous work [27,29] and with results from a companion study evaluating short-term (7-d) bioaccumulation with many of the same species [28]. The more significant correlation between BAF_{loc} and B[*a*]P metabolism as compared to BAF_{loc} and contact angle, at least in the no soot treatments, suggests that metabolism may play a more important role than gut surfactancy in bioaccumulation, at least in some cases.

It is likely that other species-specific characteristics not taken into account by this study also are important in determining BAF_{loc}s. These may include particle selectivity, depletion of PAHs in the local environment (e.g., bioturbation/burrow irrigation), and differential metabolism of various PAHs. For example, Shull and Yasuda [21] found that *C. grandis* selects for particles in the 16- to 32-μm size range, and this is quite close to the size range of the soot particles used in this study (sieved to 63 μm). Self and Jumars [26] showed that *Yoldia scissurata* and *M. balthica* also selectively ingest particles of a certain size range. They hypothesize that most deposit feeders are highly selective for a similar size range of particles that carry the majority of bacteria and total organic carbon in the sediment (~3–17 μm). This additional variable may have an important effect on the BAF_{loc}s reported here, especially for the soot treatment. It also may explain why there are higher BAF_{loc}s in the soot treatment for *C. grandis* and *C. torquata*. Furthermore, the ability of an organism to metabolize PAHs can vary depending on the particular compound of interest [12,31]. The metabolic parameter used here, which is percent of B[*a*]P body burden represented as metabolites after 7-d sediment exposures, does not take into account variable rates of metabolism for different compounds and other factors influencing depuration rates. It also is known that bio-

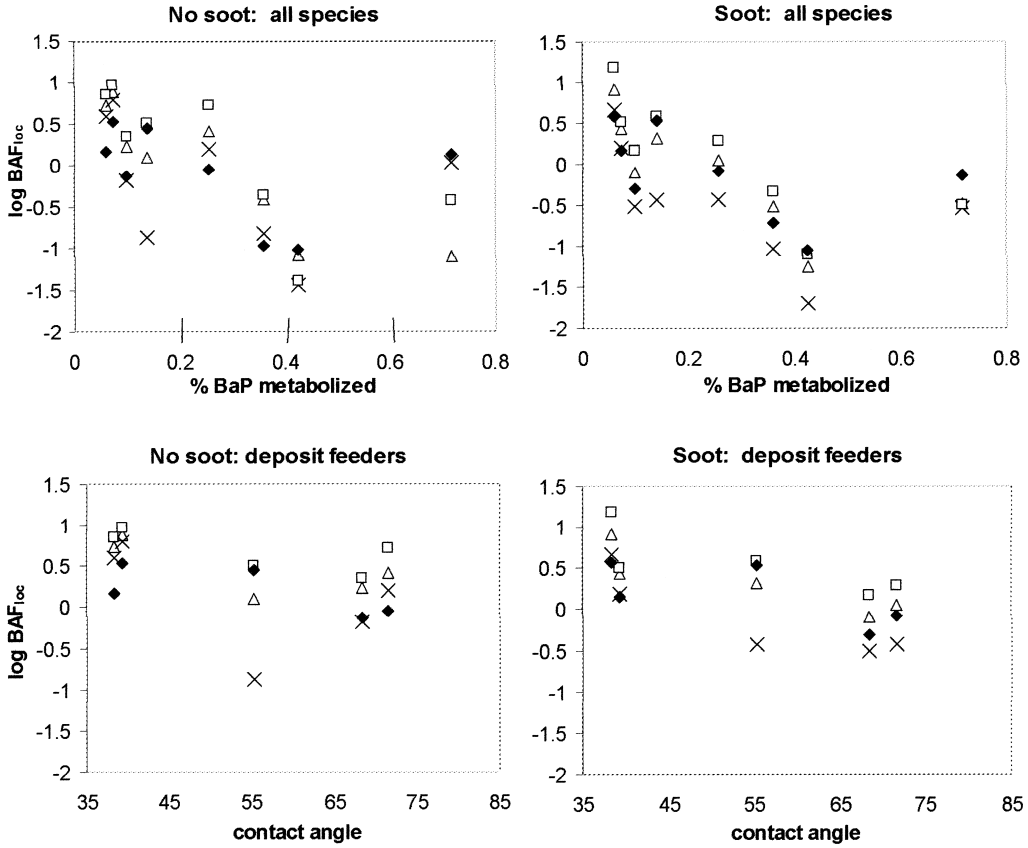

Fig. 4. The BAF_{loc}s of four representative polycyclic aromatic hydrocarbons plotted against contact angle and metabolism for each treatment. Contact angle plots exclude *Nereis virens*, *Mya arenaria*, and *Mulinia lateralis* because they are not primarily deposit feeders and contact angle is relevant only to species whose primary exposure pathway is through ingested sediment. Phen = phenthanthrene, diM phen = dimethylphenanthrene, BAA = benzo[*a*]pyrene, B[*a*]P = benzo[*a*]pyrene, BAF = bioaccumulation factor. ♦, Phen; □, diM phen; △, BAA; ×, B[*a*]P.

turbation and irrigation of bedded sediments caused by burrowing benthic organisms can enhance degradation and geochemical and contaminant fluxes across the sediment water interface [22,23]. Depending on the nature of the burrowing behavior for a particular species, this may mean that organisms actually are exposed to a microenvironment in an irrigated burrow that is depleted in contaminants relative to the bulk sediment, reducing their overall exposure to sediment contaminants.

Two of the test species *M. balthica* and *N. virens*, were chosen in part because they are recommended by the U.S. Environmental Protection Agency and commonly are used as model organisms to assess bioaccumulation of potentially toxic contaminants in site assessment, dredging management, or remediation studies [43]. This study suggests that, though *M. balthica* (intermediate metabolism and intermediate/low surfactant strength; among the higher BAF_{loc}s observed here for bivalves) may be a suitable monitoring species, *N. virens* (very high metabolism and intermediate/high surfactant strength; among the lowest BAF_{loc}s observed) clearly does not represent a sensitive indicator of PAH bioaccumulation among polychaetes (BAF_{loc}s for *C. torquata* and *P. gouldii* ~10× higher; Fig. 3). Effort should be focused on verifying the suitability of other current test species (as well as suitable replacement species) by measuring characteristics such as metabolic ability and contact angle.

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UBIQUITOUS OBSERVATIONS OF ENHANCED SOLID AFFINITIES FOR AROMATIC ORGANOCHLORINES IN FIELD SITUATIONS: ARE IN SITU DISSOLVED EXPOSURES OVERESTIMATED BY EXISTING PARTITIONING MODELS?

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Abstract—This paper investigates the ability of the traditional organic matter partitioning (OMP) model to predict the solid–water distribution, and hence the dissolved exposures, of hydrophobic organic compounds (HOCs) in real field situations. Observed organic-carbon-normalized partitioning coefficients ($(K_{oc})_{obs}$) of polychlorinated biphenyls, polychlorinated benzenes, polychlorinated dibenzo-dioxins and -furans, and *p,p′*-dichlorodiphenyltrichlorethane (DDT) with metabolites were selected from the literature and compared with their respective OMP model estimates. For all compound classes and in a majority of the investigated cases, $(K_{oc})_{obs}$ values were significantly larger than predicted. This translated into factors of overestimated dissolved exposures ranging from 1 to 1,000. Various reasons are discussed for the discrepancies between predictions and actual observations, such as the effect of the diagenetic state and other properties of the particulate organic matter. The greater enhancement in $(K_{oc})_{obs}$ of planar over nonplanar compounds suggests in certain cases that efficient interactions with aromatic soot phases may be significant. For an improved predictability of $(K_{oc})_{obs}$ and dissolved exposures of HOCs in the real environment, the inclusion of soot and possibly other distinct subfractions of bulk organic carbon into an extended solid–water partitioning model may be considered.

Keywords—Organic matter partitioning model Hydrophobic organic compounds Soot Sediment quality criteria Dissolved exposures

INTRODUCTION

The partitioning of hydrophobic organic compounds between water and various solid phases is a key process strongly influencing their environmental transport, reactivity, and dissolved exposures. The magnitude of the solid–water distribution coefficient (K_d) has been assumed to be linear and to be predictable from easily obtainable physicochemical properties of both the solid sorbent and the contaminant hydrophobic organic compound (HOC) sorbate [1,2], that is, as

$$K_d = \frac{c_s}{c_w} = f_{oc} \cdot K_{oc} \quad (1)$$

where c_s is the concentration of a sorbate in the solid phase (mol/kg solid), c_w is the concentration in the aqueous phase (mol/L water), f_{oc} is the organic carbon fraction of the sorbent (kg organic carbon/kg total solid), and K_{oc} is the organic-carbon-normalized partition coefficient (L water/kg organic carbon). According to this model, the f_{oc} represents the portion of the solid that matters in HOC sorption; HOCs may dissolve into nonpolar environments of the natural organic matter. The compound-specific value of K_{oc} represents the relative affinity of a HOC for this natural organic matter relative to that of water. Given that f_{oc} is easily measurable and since K_{oc} is obtainable from linear free energy relationships with direct measures of solvophilicity such as aqueous solubility and octanol/water partition coefficients (K_{ow}), the organic matter partitioning model of Equation 1 is a potentially powerful tool to predict solid–water distributions. This model has been successful in explaining the sorption behavior of many HOCs with a wide range of soils and sediments in laboratory studies. It has consequently won broad recognition (cf., [3]) and even formed the basis for development of sediment quality criteria (SQC) [4,5].

While the organic matter partitioning (OMP) model may still provide a useful reference point, results over the past several years from longer term sorption experiments spanning over wider concentration ranges as well as observations of the actual distribution in the field are at odds with the simple partitioning view of the OMP model: Sorption isotherms exhibit nonlinearity (cf., [6,7]), and K_ds of PAHs in the field are far in excess of OMP model expectations (cf., [8,9]). Two reviews of mechanisms that may account for these observations have been presented [10,11].

The ultimate test of our understanding of the solid–water partitioning process is to compare the model predictions with the actual phase distributions in the field along with collection of the relevant system descriptors. With this approach, it was realized that observed K_{oc} numbers ($(K_{oc})_{obs}$) of polycyclic aromatic hydrocarbons (PAHs) were systematically much underestimated (often by several orders of magnitude) by the OMP model in real field measurements [9,12]. For PAHs, it has been hypothesized that these elevated $(K_{oc})_{obs}$ values were caused by additional and efficient sorption to soot, a highly condensed subfraction of the total organic-matter pool [9,12]. Indeed, soot has been found to be ubiquitously present in modern day soils and sediments, where it may make up as much as 30% of the total organic carbon [13–15]. Recent laboratory determinations of the soot–water partition coefficients (K_{sc}) of PAHs demonstrated that the interaction with condensed soot carbon was about a factor of 100 stronger than with natural organic carbon [16]. The adsorption was rapid and the surface-normalized K_{sc} values agreed well with the-

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Table 1. Selected K_{oc} estimations from K_{ow} (log K_{oc} = a log K_{ow} + b)[a]

Reference	a	b	r^2	Compounds; sorbent type; method
Schwarzenbach and Westall [47]	0.72	0.49	0.95	Alkylated and chlorinated benzenes; soil and sediments; laboratory experiments
Chiou et al. [48]	0.90	−0.54	1.00	Mostly chlorinated benzenes and PCBs; soil; laboratory experiments
Chin et al. [49]	0.37	3.16	0.94	PCBs, polychlorinated benzenes, chlordane; lacustrine sediment; laboratory experiments
Chin et al. [49]	0.62	1.18	0.94	CCl_4, lindane, PCB, polychlorinated benzenes, chlordane; soil; laboratory experiments
Gerstl [50]	0.72	0.42	0.86	Halogenated aromatic hydrocarbons; soil; literature compilation, different methods
Sabljic et al. [51]	0.81	0.10	0.89	Alkylated and halogenated hydrocarbons, alkylated and halogenated benzenes and PAHs, PCBs, and chlorinated pesticides; soil; QSAR-modeling

[a] PCB = polychlorinated biphenyl; PAH = polycyclic aromatic hydrocarbon; QSAR = quantitative structure–activity relationship.

oretical estimates of PAH adsorption to both soot [12] and activated carbon (cf., [17]).

Given that activated carbon is a more efficient adsorbent also for other HOCs than PAHs and that its natural analogue (i.e., soot) is ubiquitous in the environment, it behooves us to investigate whether enhanced sorption may be systematically observed in presumed soot-rich environments for HOCs in general. To this end, it can be hypothesized that especially the highly planar compounds such as non-*ortho*-substituted polychlorinated biphenyls (PCBs) or polychlorinated dibenzodioxins and -furans (PCDDs and PCDFs) might exhibit similarly elevated affinities to soot as observed for PAHs. Hence, the objective of the present paper is to test the hypothesis of enhanced solid affinity of rigid organochlorine compounds by evaluating available literature data on $(K_{oc})_{obs}$ of PCBs, PCDDs, PCDFs, polychlorinated benzenes, *p,p′*-DDT with metabolites (hereafter collectively referred to as DDXs). The intention of this investigation is not limited to testing the scope of the soot supersorbent hypothesis but is aimed at evaluating the general need for an improved characterization of the speciation of HOCs and the necessity of considering specific subfractions of the bulk particulate organic matter.

DATA SELECTION AND ANALYSIS

Compilation of $(K_{oc})_{obs}$ *data*

The reporting criteria set up for data selection from the literature were field observations, individual compound concentrations, organic carbon content of the solid phase, and transparency and reliability of the analytical methods. Hence, these criteria apply to peer-reviewed articles that either explicitly report $(K_{oc})_{obs}$ values or from which $(K_{oc})_{obs}$ values could be derived from supplied information on concentrations in both the dissolved and particulate phase as well as the particulate organic carbon content. Consequently, the selected data sets do not include any laboratory-derived data such as from batch sorption experiments with spiked analytes. A considerable amount of field-observed K_d values have been published without any information on corresponding organic matter contents. Unfortunately, such data are not comparable with other studies and were therefore excluded from this compilation. We only include $(K_{oc})_{obs}$ numbers given for single compounds/congeners and do not consider any work that reported any kind of pooled values such as for PCBs distinguished only by numbers of chlorine substitution. Finally, for reasons of uncertainty and credibility, we did not attempt to extract any data from published $(K_{oc})_{obs}$ versus K_{ow} figures.

Selection of K_{ow} *data*

Considerable variability exists in reported K_{ow} values for individual compounds. For the more hydrophobic compounds, i.e., log $K_{ow} > 6$, this variability easily ranges over one order of magnitude (cf., Mackay et al. [18] for a thorough compilation). Any comparison of model-predicted K_{oc} versus $(K_{oc})_{obs}$ therefore depends to a certain extent on the K_{ow} values selected. As will be shown below, this variation does not affect the general conclusions of the present study. Our attempt was to use well-established K_{ow} values, with emphasis on the consistence and comparability of the data for individual compound classes. Hence, K_{ow} numbers were taken from Hawker and Connell [19] for PCBs, Govers and Krop [20] for PCDDs and PCDFs, De Bruijn et al. [21] for polychlorinated benzenes, and from Chiou et al. [22] and Baum [23] for DDXs.

Selection of K_{oc}–K_{ow} *linear free energy relationships*

From the ample laboratory-derived or quantitative structure–activity relationships and modeled log-linear K_{oc}–K_{ow} relationships, the ones using a data set with predominantly or only chlorinated HOCs were used for comparison with the actual $(K_{oc})_{obs}$ values from real environments. Because other compound classes such as modern pesticides and PAHs may exhibit different abilities of interaction with organic matter, K_{oc}–K_{ow} relationships based on data sets of such compounds were not considered. Table 1 compiles the various K_{oc}–K_{ow} estimates applied. The minimum and maximum log K_{oc} predictions are indicated in all subsequent log $(K_{oc})_{obs}$ versus log K_{ow} plots.

DATA EVALUATION

Comparison of field-observed versus model-predicted phase distributions

This compilation of actual solid–water distributions of aromatic organochlorines demonstrates a solid affinity that is, in a majority of the cases, significantly larger than predictions based on the widely employed OMP model. Observations from a large variety of aquatic environments suggest that the OMP model is underestimating the solid–water distribution of PCBs (Fig. 1), polychlorinated benzenes (Fig. 2), PCDDs and PCDFs (Fig. 3), and DDX (Fig. 4) by up to three orders of magnitude. Obviously, this consistently enhanced solid affinity corresponds to a proportional lowering of the actual dissolved exposures relative to predictions. For the compound classes where the congeners span a wider range of physicochemical

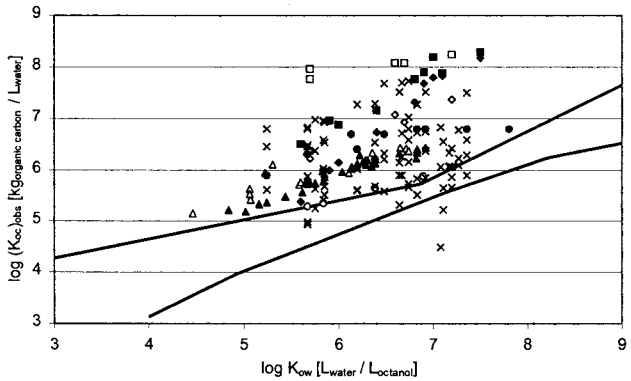

Fig. 1. Organic-carbon-normalized in situ partition coefficients (log $(K_{oc})_{obs}$) for PCBs, elevated relative to organic matter partitioning (OMP) model predictions (solid lines: upper and lower range of estimates given in Table 1), in different environmental compartments: planar (open squares) and nonplanar (filled squares) congeners in Ketelmeer surficial sediment [24], planar (open diamonds) and nonplanar (filled diamonds) congeners in deeper Ketelmeer sediment [24], St. Clair and Detroit suspended river sediment (filled circles; [25]), planar (open triangles) and nonplanar (filled triangles) congeners in Hudson River open water column [26], Lake Superior open water column (open circles; [27]), open North Atlantic Ocean waters (crosses; [33]).

properties (i.e., PCBs, polychlorinated benzenes, PCDDs, and PCDFs), there is a generally increasing trend in $(K_{oc})_{obs}$ with increasing K_{ow}. This suggests that the enhanced solid affinity is a result of active hydrophobicity-driven sorption to some supersorbent as opposed to a production-related permanent pollutant-to-particle association. The patterns and even magnitude of enhancements are reminiscent of the trends in previously reported compilations for PAHs (cf., [12]). The up to three orders of magnitude difference in reported values of $(K_{oc})_{obs}$ for a given compound may result from a combination of several factors, including a large variation in the abundance and availability of the postulated supersorbent media. Below, we attempt to discuss various factors that may contribute to the variations observed within the compiled data sets.

Effects of diagenetic state and properties of the particulate organic matter

For the PCBs (Fig. 1), the relative $(K_{oc})_{obs}$ enhancement is most pronounced in sediments [24], thereafter in suspended river sediments [25], but less so for pelagic water compartments [26,27]. This could be an indication that the origin and quality of particulate organic matter affects its overall sorptivity. Diagenetically younger organic matter (as observed in the pelagic water column) is richer in polysaccharides (cf., [28]), which is a poor sorbent matrix for HOCs [29]. The lipid fraction and the soot fraction are likely to be more resistant to degradation than bulk organic matter, and their relative importance for partitioning might thus increase with overall organic matter degradation increasing from the pelagic system to the sediment. Increasing values for individual PCB $(K_{oc})_{obs}$ with depth in a doubly stratified 400-m water column in the Baltic Sea was recently reported [30]. This is consistent with a dependency on the aging effect of the organic matter sorbent properties, as the increasing PCB $(K_{oc})_{obs}$ values were associated with increasing lipid fractions of the total particulate organic carbon. The notion of increased sorptivity of organic matter with aging is also supported by the polychlorinated benzenes data set (Fig. 2). For instance, the surface waters in

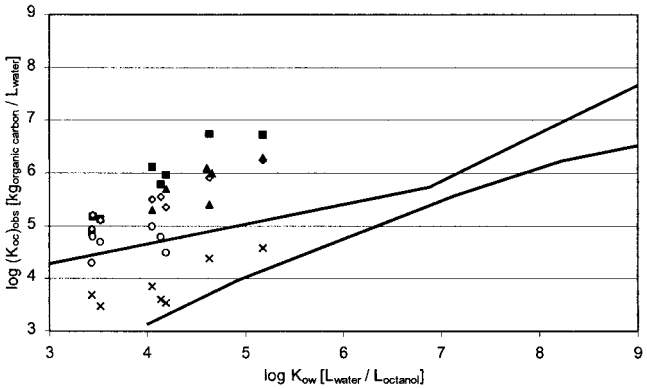

Fig. 2. Organic-carbon-normalized in situ partition coefficients (log $(K_{oc})_{obs}$) for polychlorinated benzenes, elevated relative to organic matter partitioning (OMP) model predictions (solid lines: upper and lower range of estimates given in Table 1) in different environmental compartments: surficial (filled squares) and deeper (open diamonds) Ketelmeer sediment [24], St. Clair and Detroit suspended river sediment (filled triangles; [25]), Ketelmeer sediment (open circles; [32]), Ise Bay (Japan) surface water (crosses; [31]).

the Ise Bay (Japan) exhibited considerably lower $(K_{oc})_{obs}$ values [31] than other studies with suspended river sediments or Ketelmeer sediments [24,32].

The open ocean PCB data set of Schulz-Bull et al. [33] is unique both in its abundance and in that it represents the only measurements of PCBs in both the dissolved and particulate phase from remote oceanic regimes far removed from anthro-

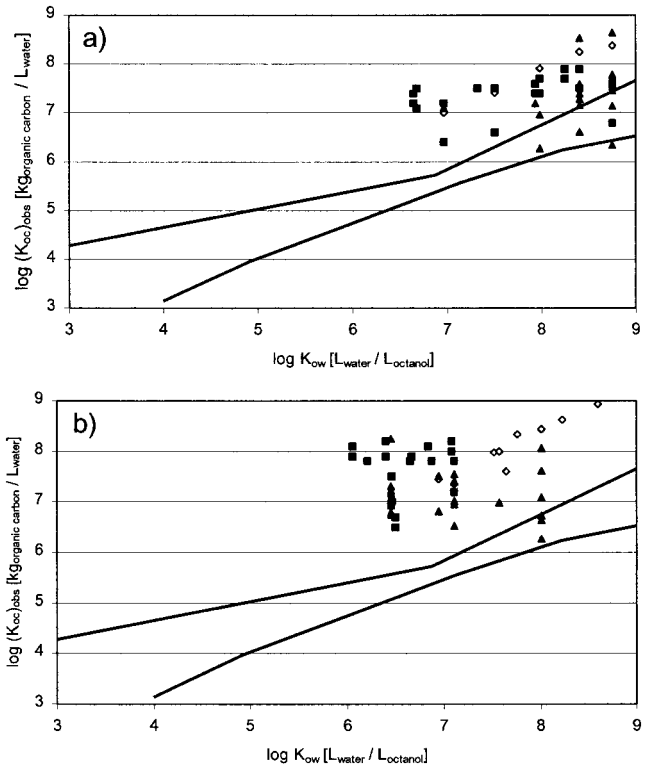

Fig. 3. Organic-carbon-normalized in situ partition coefficients (log $(K_{oc})_{obs}$) for (**a**) PCDDs and (**b**) PCDFs, elevated relative to organic matter partitioning (OMP) model predictions (solid lines: upper and lower range of estimates given in Table 1), in different environmental compartments: River Elbe up and downstream Hamburg Harbor (filled squares; [41]), Frierfjord (Grenlandsfjords, Norway) open water column (open diamonds; D. Broman, personal communication), Baltic Sea open water column (filled triangles; [40]).

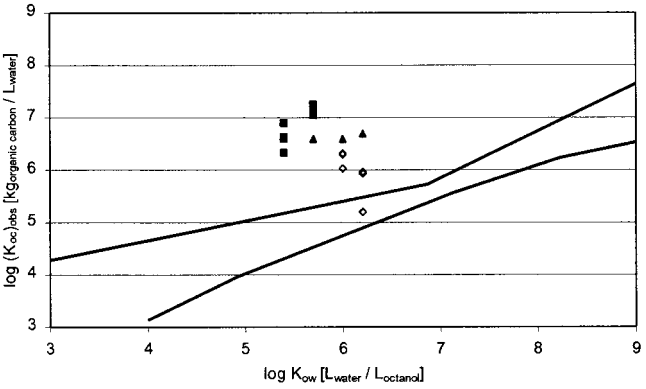

Fig. 4. Organic-carbon-normalized in situ partition coefficients (log $(K_{oc})_{obs}$) for *p,p′*-DDT with metabolites (DDXs), elevated relative to organic matter partitioning (OMP) model predictions (solid lines: upper and lower range of estimates given in Table 1), in different environmental compartments: planar *p,p′*-(dichlorodiphenyl)dichloroethylene (*p,p′*-DDE) and *p,p′*-(dichlorodiphenyl)monochloroethylene (filled squares); nonplanar *p,p′*-DDT and *p,p′*-(dichlorodiphenyl)dichloroethane (*p,p′*-DDD) (open diamonds) in Palos Verdes Shelf sediments (P.H. Santschi and Ö. Gustafsson, personal communication); *p,p′*-DDT, *p,p′*-DDD, and *p,p′*-DDE in St. Clair and Detroit suspended river sediment (filled triangles; [25]).

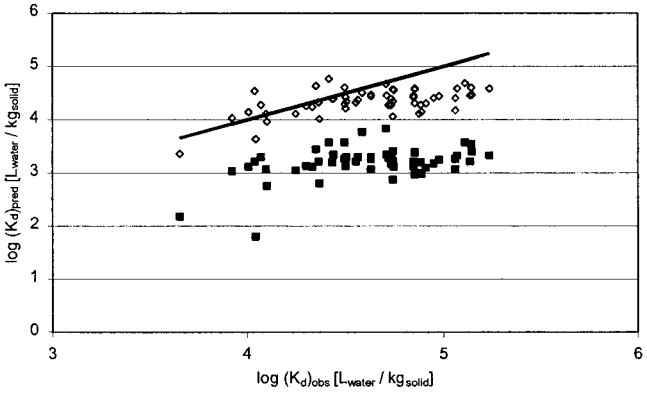

Fig. 5. Improved prediction of K_d values for pyrene in Humber Estuary sediments by application of the soot-inclusive organic matter partitioning (OMP) model. The solid line represents an ideal 1:1 line between $(K_d)_{obs}$ and $(K_d)_{pred}$. $(K_d)_{pred}$ from $f_{oc}K_{oc}$ (filled squares), $(K_d)_{pred}$ from $f_{oc}K_{oc} + f_{sc}K_{sc}$ (open diamonds). All $(K_d)_{obs}$ values from [46]. Numbers of the other parameters: $f_{oc} = 0.03 \pm 0.02$, $f_{sc} = 0.0022 \pm 0.0010$ (both from [46]), log $K_{oc} = 4.8$ [52], log $K_{sc} = 7.03$ [16].

pogenic sources. The North Atlantic has been proposed as a possibly dominant sink for PCBs (cf., [34]), and a correct understanding of their phase distribution in this regime is therefore warranted. Notably, in their study [33] with all samples collected on the same cruise using the same equipment and with analysis performed with identical procedures, there is a factor of >100 variability in individual congener $(K_{oc})_{obs}$ values between four different water masses investigated. While the data set in Figure 1 is not separated into these different water masses, somewhat contradictory to the above observed aging effect, there appeared to be a trend of increasing $(K_{oc})_{obs}$ values with younger halocarbon ages of the water masses [33,35].

In surface ocean systems, the particulate sorbents may be dominated by living cells, foremost phytoplankton, in which the organic matter is highly structured, contrasting to lyzed and diagenetically altered organic matter in settling particles and sediments. For instance, the lipid bilayer may be actively water decontaminated in living cells, making it a more ideal solvent for HOCs than water-contaminated octanol in binary octanol–water systems [11]. Further contributing to expectations of elevated K_{oc} values in living partitioning, one may expect a lower DOC contamination in the surrounding seawater than the octanol contamination in the water phase of the binary solvent system. Yet further complemented by salting out and freezing out effects (cf., [3]), this would lead to an increased fugacity in seawater relative to in the water compartment of the octanol–water system.

Effects of planarity and known presence of soot

The data of Jonker and Smedes [24] and Butcher et al. [26] are presented separately for ortho- and non–*ortho*-PCB congeners (Fig. 1). The preferentially elevated sorptivity for planar PCBs relative to their nonplanar counterparts observed in these two field studies cannot be rationalized with the sorbate hydrophobicity. Rather, it suggests interactions of these planar HOCs with specific adsorption sites. Elevated non–*ortho*-PCB binding with humic substances of a higher degree of aromaticity and polarizability was recently observed in laboratory studies [36]. A subfraction of the solid bulk organic matter that might offer adsorption sites preferentially favorable to planar HOCs is soot. The accessibility (e.g., by diffusion into narrow interplanar soot spaces) of and interaction (e.g., by efficient π-π overlap) with this highly condensed combustion residue might be enhanced for planar compounds due to steric advantages over the more bulky nonplanar compounds. Jonker and Smedes [24] in fact confirmed the presence of soot-like material in their sediment samples and brought up this finding as a plausible explanation for their observed elevated partition values. However, even the nonplanar PCBs regularly exhibit enhanced $(K_{oc})_{obs}$ values (Fig. 1), indicating that efficient sorption to soot seems not to be restricted to fully planar HOCs such as PAHs or non–*ortho*-substituted PCBs.

Interestingly, the PCDD/PCDF data (D. Broman, personal communication), with some of the largest ever observed log K_{oc} values of up to nine (Fig. 3), were obtained in the highly contaminated Frierfjord (recipient of releases from a magnesium smelter plant). The presence of soot-like solid phases in waters and sediments of smelter-affected fjords has previously been implicated to determine the speciation and distribution of PAHs [37,38]. Finally, soot partitioning was shown quantitatively to much better explain than the OMP model the sediment–porewater distribution of *p,p′*-DDT and its metabolites in Palos Verdes Shelf sediments (P.H. Santschi and Ö. Gustafsson, personal communication). At this soot-contaminated site [13], the more planar *p,p′*-(dichlorodiphenyl)dichloroethylene and *p,p′*-(dichlorodiphenyl)monochloroethylene exhibited higher $(K_{oc})_{obs}$ values than the more hydrophobic but nonplanar *p,p′*-DDT and *p,p′*-(dichlorodiphenyl)dichloroethane (Fig. 4; P.H. Santschi and Ö. Gustafsson, personal communication).

Due to the present absence of any K_{sc} values for aromatic organochlorines, it is not yet possible to test the potential for improved prediction of K_d by inclusion of a soot adsorption term into the OMP model, as recently performed for PAHs [9,12]. Figure 5 illustrates the beneficial application of the enhanced, soot-inclusive OMP model using the available K_{sc} values for PAHs [16]. Whereas the values of $(K_d)_{pred}$ for pyrene from the traditional OMP model are roughly two orders of magnitude lower than the observed ones, a much better agreement between predicted and observed K_d values is obtained when applying the enhanced, soot-inclusive model.

Other factors affecting the apparently dissolved fraction

Inclusion of a colloid-associated form in the operationally defined filter-passing dissolved fraction leads to an underestimation of K_d. This so-called particle-concentration effect (cf., [11,39]) is most pronounced for more hydrophobic compounds and in regimes where the colloid-to-large particle ratio is large. It can be expressed as

$$(K_{oc})_{obs} = \frac{K_{oc}}{1 + K_{coc} \cdot COC} \quad (2)$$

where the deviation in $(K_{oc})_{obs}$ from the true K_{oc} is a function of the product of the organic-carbon-normalized HOC partition coefficient with colloids (K_{coc}) and the abundance of colloidal organic matter (COC).

The phenomenon results in $(K_{oc})_{obs}$ versus K_{ow} plots with decreasing (i.e., nonlinear) slopes at higher K_{ow}, approaching in the limit apparently constant $(K_{oc})_{obs}$ values as the colloid partitioning overwhelms the truly dissolved form in the filtrate.

The potential presence of the particle concentration effect is indicated in several of the surveyed data sets in Figure 1, notably in Baker et al. [27], Lau et al. [25], and Butcher et al. [26]. Similarly, a decreasing $(K_{oc})_{obs}$ versus K_{ow} slope was observed for the highly hydrophobic PCDDs and PCDFs (Fig. 3a, and b; data from Broman et al. [40] and Götz et al. [41]) in the presumed colloid-rich waters of the Baltic Sea and the Elbe River. If the surveyed $(K_{oc})_{obs}$ data were corrected for this colloid effect, the true particle water distributions would be even more enhanced relative to OMP predictions than is currently depicted in Figures 1 through 4.

Extremely low log $(K_{oc})_{obs}$ values, on the order of only two to three, were reported for PCBs in organic-rich (total organic carbon, 4–6%; dissolved organic carbon, 14–87 mg/L) sediments of the outer New Bedford harbor [42]. These numbers are roughly one to three orders of magnitude lower than predicted from the OMP model and are even more divergent from all other field-observed numbers. The authors attributed their low $(K_{oc})_{obs}$ numbers to the overwhelming influence of colloidal sorption in their pore waters. This was suspected to be more important for larger PCBs due to both steric hindrance to solid-phase sorption sites and greater surface-to-volume ratio of the colloidal organic matter. As the K_{oc} values obtained in that study deviate to such a great extent from all other evaluated field data, they are not included in Figure 1.

Enhanced $(K_{oc})_{obs}$ values due to nonequilibrium caused by biodegradation of the dissolved fraction surpassing desorption cannot by totally ruled out. However, several studies have shown that the in situ degradation rates of these recalcitrant aromatic organochlorine compounds are not likely to create such large deficiencies of the dissolved concentrations (cf., [43]).

Overestimated dissolved exposures and sediment quality criteria

Because the ratio between observed and predicted K_{oc} values for a given compound is inversely correlated with the respective observed and predicted dissolved concentrations (Eqn. 3), the divergence between observed and predicted numbers is a direct measure of our current overestimation of the compounds' dissolved exposures,

$$FOE = \frac{(c_{water})_{pred}}{(c_{water})_{obs}} = \frac{(K_{oc})_{obs}}{(K_{oc})_{pred}} \quad (3)$$

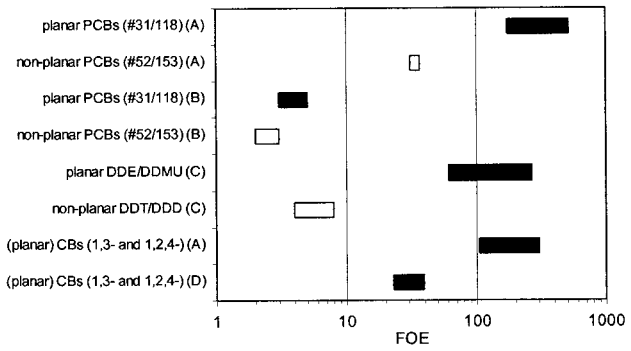

Fig. 6. Range of factors of overestimated dissolved exposures (FOEs) for selected compounds. Filled bars = planar compounds; open bars = nonplanar compounds. (**A**) Jonker and Smedes [24]; (**B**) Butcher et al. [26]; (**C**) Ö. Gustafsson, personal communication; (**D**) Ten Hulscher et al. [32]. CB = polychlorinated benzenes.

The factors of overestimated dissolved exposures (FOEs) observed in the data compiled in Figures 1 through 4 range from roughly one to three orders of magnitude. Selected examples for individual planar and nonplanar representatives of the different compound classes under investigation are presented in Figure 6. These ubiquitously observed large FOEs indicate that the mobility of HOCs in aqueous environments is likely to be significantly lower than currently expected. While ingestion may be an important uptake route for higher animals, the dissolved exposures constitute the directly bioavailable fraction of HOCs in the environment. Therefore, (enhanced) sorption to sediment reduces the bioavailability of organic pollutants (cf., [44,45]).

The separation and quantification of distinct subfractions of the bulk organic carbon pool such as soot and the determination of their specific affinities toward HOCs is likely to prove beneficial for a more accurate description of HOC solid–water-phase partitioning. An extension of the OMP model and sediment quality criteria (SQC) for aromatic organochlorines similar to the one earlier suggested for PAHs [9] might therefore be considered. The basis of SQCs is to ensure that the pore-water concentration, in equilibrium with the sediment, is not exceeding the final chronic water quality criteria values (FCV) [4,5]. Since the soot phase may be influencing the environmental speciation of organochlorines, a SQC for these compounds may, in analogy to the earlier formulation for PAHs [9], be defined as

$$SQC(\text{compound}_i) = (f_{oc}K_{oc} + f_{sc}K_{sc})FCV(\text{compound}_i) \quad (4)$$

where f_{sc} is the soot-carbon fraction of the sorbent (kg soot carbon/kg total solid) and K_{sc} is the soot-carbon normalized partition coefficient (L water/kg soot carbon).

Such an increased ability to anticipate the distribution of HOCs between particle-bound and dissolved forms would improve the possibility of society to prioritize and handle issues concerning these chemicals in the environment in many ways. The accurate quantitative prediction of phase speciation would, e.g., allow the a priori estimation of directly bioavailable, dissolved exposures and tendency for long-range dispersal. Large savings for society are to be made in using extended knowledge about pollutant speciation in risk evaluation. This would provide an efficient basis for setting priorities regarding which compounds and which contaminated sites are most efficiently and urgently remediated.

CONCLUSIONS

In summary, field-observed solid–water distributions of individual HOCs surveyed from the literature suggest that the traditional organic-carbon-normalized solid–water distribution model severely underestimates K_{oc} values for a wide range of compound classes (PCBs, polychlorinated benzenes, PCDDs and PCDFs, and DDXs). In several of the presented cases, the presence of soot-like subfractions of the bulk particulate organic matter was put forward as a likely explanation for these findings [24; D. Broman and P.H. Santschi and Ö. Gustafsson, personal communications]. This argument is additionally supported in certain cases by the preferential sorption of the planar representatives of these compound classes [24; P.H. Santschi and Ö. Gustafsson, personal communications]. For a given compound class, the divergence between observed and predicted K_{oc} values often appeared to be relatively small in surface water but much more pronounced in sediments. Based on this compilation, it appears likely that soot and possibly other subfractions of bulk organic matter with distinct structure and composition play a vital role for the distribution of not only PAHs but also of a wide range of other hydrophobic sorbates. The refined, soot-inclusive solid–water partitioning model has been demonstrated to significantly better explain the field-observed distributions than the OMP model [12,40,46]. The analysis presented here suggests that a soot-inclusive partitioning model may also improve prediction of the actual distributions for several other HOC compound classes. This would require the determination of soot–water distribution coefficients for the respective compounds either by theoretical estimations [12] or by laboratory-based soot partitioning experiments [16].

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THE BIOACCUMULATION OF POLYNUCLEAR AROMATIC HYDROCARBONS BY BENTHIC INVERTEBRATES IN AN INTERTIDAL MARSH

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Abstract—Biota-sediment accumulation factors (BSAF; concentration in organism lipid/concentration in sediment on an organic carbon basis) of polyaromatic hydrocarbons varied with season and along an intertidal gradient in a coastal marsh in San Francisco Bay. The BSAFs were lowest during the local rainy season. During the dry season, BSAFs were lowest in the high intertidal zone closest to shore. Significant differences among species groups were also observed; BSAFs were lowest in polychaetes and highest in the asian clam (*Potamocorbula amurensis*), varying over almost three orders of magnitude (0.0069–5.4 g sediment organic C/g lipid). The BSAFs decreased with increasing percent fines in the sediments and with PAH concentrations on an organic carbon basis. We suggest that a determining variable is the content of highly aromatic soot particles, which increases during periods of surface runoff and which is expected in the dry season to be highest in the high intertidal zone where these finer particles preferentially accumulate. Correlations of BSAFs with the ratio of the logarithm of the activity coefficients in porewaters to those in sediments were generally stronger than with log K_{ow}, indicating a limitation of octanol as a surrogate for sediment organic carbon or organism lipid. These observations qualify but also strengthen the concept of equilibrium partitioning as the determining factor in bioaccumulation by benthic organisms of nonpolar organic compounds from sediments; the assumption that ''organic carbon'' can be considered in generic terms without allowance for aromaticity and probably other factors as well, must, however, be reconsidered.

Keywords—Bioaccumulation Bioavailability PAHs Benthic invertebrates Sediment quality criteria

INTRODUCTION

In a study of the distribution of polynuclear aromatic hydrocarbons (PAHs) in sediments and their interstitial waters of a mudflat in San Francisco Bay, we measured an order of magnitude variation in values of the in situ sediment porewater partition coefficient (K_{oc}') for a suite of 3–6 ring PAHs. The K_{oc}'s increased with the organic carbon and silt content of surface sediments collected along an intertidal gradient during the dry and wet seasons in 1993–1994 and were higher during the wet period of high surface runoff than during the dry season. Moreover, K_{oc}' decreased from the high- to the low-intertidal zone during the dry season, indicating heterogeneity in partitioning behavior along this spatial gradient [1].

Several studies of the distribution of hydrophobic compounds between sediments and interstitial porewaters have examined the role of dissolved organic matter in enhancing solubilities and thereby increasing concentrations of these compounds in the porewaters [2–5]. On the basis of concentrations in sediments, however, our measured porewater concentrations during the rainy season and in the high intertidal during the dry season were lower than predicted by the octanol–water partition coefficients and by simple two-phase equilibrium partitioning models [6–12]. Conversely, PAH concentrations on an organic carbon basis in the sediments were higher than would be predicted by these equilibrium models.

We proposed that the observed heterogeneity resulted from variations in the content of soot particles, which enter the system during periods of elevated surface runoff and which are expected to accumulate preferentially with other fine material in the high intertidal zone in the dry season [13]. These would provide a highly aromatic matrix for the PAHs; consequently their activity coefficients [14] would be lower in this medium than in other organic carbon matrices, and the in situ K_{oc}'s would be correspondingly higher [1].

This interpretation would therefore qualify but not necessarily negate the utility of equilibrium partitioning models in formulating chemical-specific sediment quality criteria (SQC), particularly those in which SQC are expressed on a sediment organic carbon basis. Presently, these models do not make allowance for heterogeneity in the organic carbon matrix and corresponding differences in activity coefficients of contaminant chemicals, which would in turn affect bioavailability.

The biota-sediment accumulation factor (BSAF)

$$\text{BSAF} = C_{b,lip}/C_{s,oc} \quad (1)$$

where $C_{b,lip}$ = chemical concentration in organism lipid (μg/g lipid) and $C_{s,oc}$ = chemical concentration normalized to sediment organic carbon (f_{oc}) (μg/g OC). This parameter has been recommended by several researchers for the development of SQC based on a three-compartment (sediment-porewater-biota) equilibrium partitioning model [10,15–17]. To date, these models have assumed that the organic carbon matrix can be considered in generic terms; this assumption has been supported by data from a study that examined the partitioning and toxicity of fluoranthene in sediments containing organic matter from several diverse sources [18]. These media did not include, however, sediments from areas of recent surface runoff from urban areas.

It has been assumed that the attainment of equilibrium concentrations of a hydrophobic compound between the lipids of biota and sediment organic carbon, i.e., of equal fugacity capacities [19], or alternatively, of equal chemical potentials [20]

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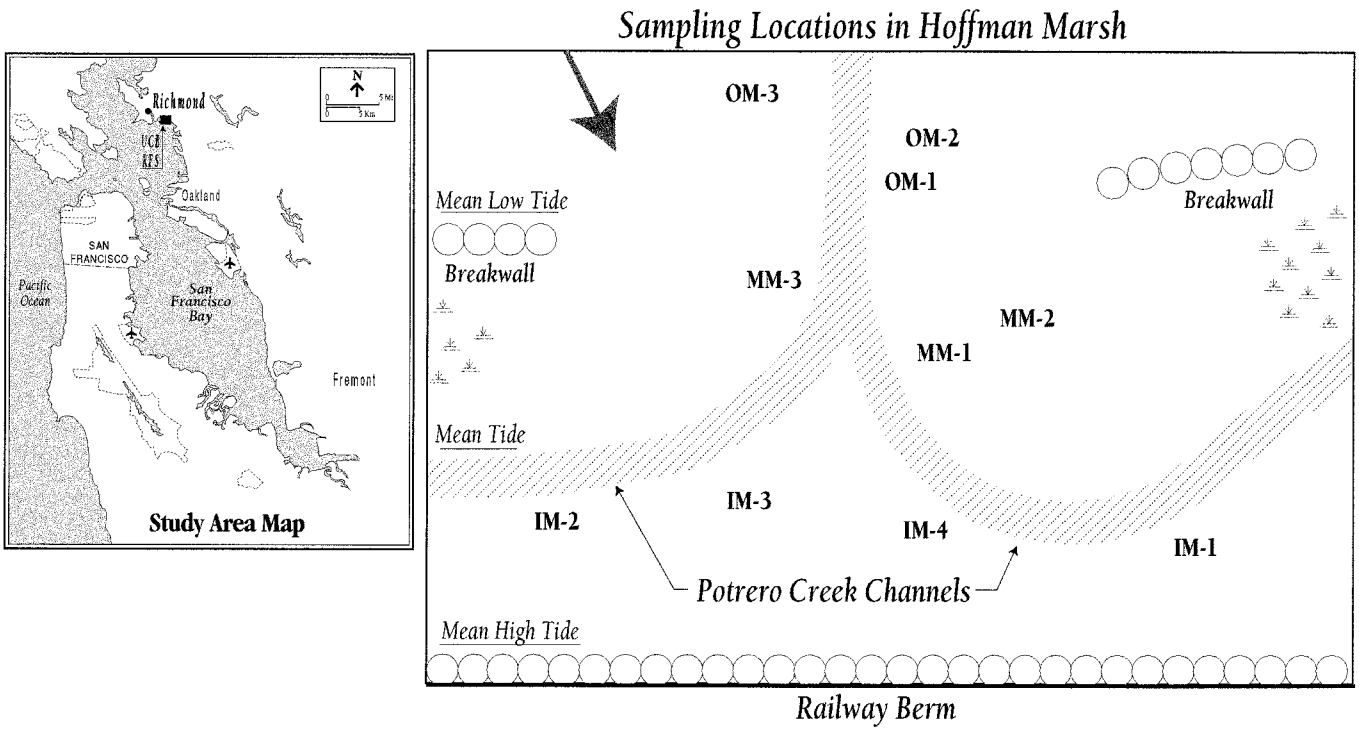

Fig. 1. Map of study area showing sampling locations in Hoffman Marsh, San Francisco Bay, California, USA.

would result in a value of approximately unity for the BSAF [10]. An abundance of both field and laboratory BSAF data, however, deviate significantly from unity [15,21,22].

In our study of the distribution of PAHs in the sediments and porewaters of an intertidal marsh in San Francisco Bay [1], we also measured PAHs in three groups of benthic intertidal organisms along an intertidal gradient during each season of a calendar year. In this paper, we examine how the order-of-magnitude variations in sediment-porewater partitioning that are associated with differences in the organic carbon matrix are related with the measured values of BSAF among the three species groups.

MATERIALS AND METHODS

All materials and methodologies utilized in this study were chosen to minimize contamination of all samples (and organic extracts thereof) by external sources. Prior to use, all sample handling/processing wares were carefully washed in hot, soapy water and rinsed thoroughly with tap water prior to use. After washing, borosilicate glasswares were kiln-fired (≥500°C) and stainless steel/teflon wares were rinsed with acetone. All organic solvents were of high purity (Optima grade; Fisher Scientific; Mountain View, CA, USA). A detailed description of the study site and all methods described herein is documented elsewhere [23].

Description of field site

Hoffman Marsh is a small (0.24 km^2) intertidal marsh along the eastern shore of central San Francisco Bay, California, USA, adjacent to the University of California Berkeley Richmond Field Station (UCB RFS), in the city of Richmond (Fig. 1). This marsh, whose area is roughly 50% brackish marsh vegetation, 30% mudflat, and 20% open bay water, has been modified by filling and installation of rock breakwalls. Two channels of a seasonally flowing creek meet near the center of the mudflats in the middle-intertidal zone. Both channels presently receive runoff from adjacent residential, commercial, and industrial developments and have historically received direct disposal of refuse and chemical contamination from neighboring munitions, lead paint and battery manufacturing, propane gas, and agrochemical manufacturing and research facilities.

The marsh supports a variety of flora and fauna including the macrophytes pickleweed and cordgrass (*Salicornia* and *Spartina* sp., respectively), benthic macro- and microalgae, and a host of benthic invertebrates that are consumed by large numbers of resident and migratory shorebirds. Several species of bivalves, dominated by the tiny gem clam (*Gemma gemma*), the Japanese littleneck clam (*Tapes japonica*), and the more recently introduced asian clam (*Potamocorbula amurensis*) can be found in the upper layers of mudflat sediments. Polychaetes are represented by small deposit-feeding species of the family Capitellidae and by larger omnivores/carnivores of the families Neptyidae and Nereidae. Several species of crustaceans (especially amphipods) are also plentiful in surface sediments.

Study design and sample collection/processing

Surface sediments and animals were collected in July and October of 1993 and again in January and April of 1994 at sites within three zones corresponding to the low, middle, and high intertidal. These zones, which correspond roughly to mean low, intermediate, and high tide zones (Fig. 1), are also referred to hereafter as the Outer, Middle and Inner Marsh (OM, MM, and IM, respectively). For each of the 10 sampling events, media were collected within a 10-m^2 quadrat. Quadrat locations are identified by their intertidal zone designation (i.e., OM) followed by a number (1–4) that signifies the sampling month (e.g., −1 = July 1993, −2 = October 1993, etc.). For the April 1994 event, only media from the Inner Marsh (IM-4)

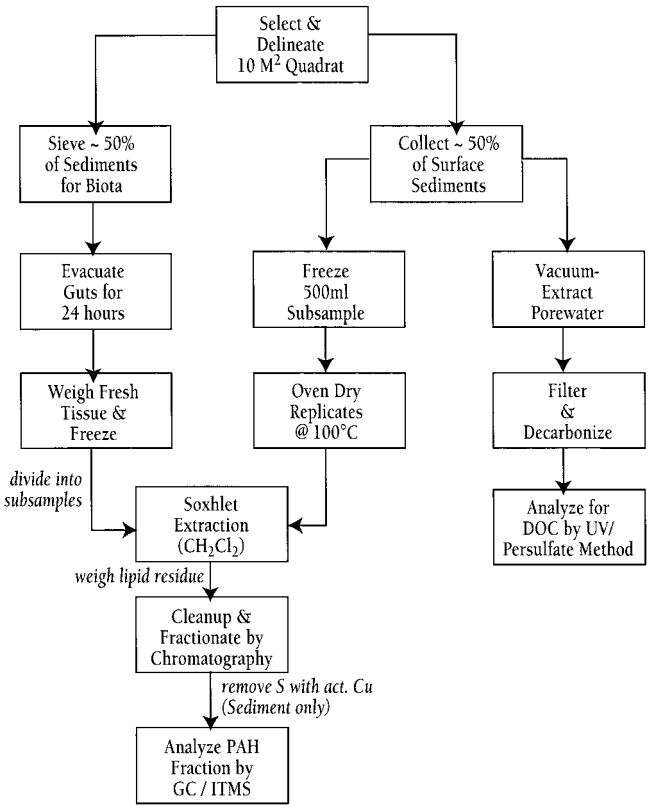

Fig. 2. Collection, processing, and analysis sequences for lipid and sediment samples.

were collected. Organisms of three major groups—*P. amurensis*, *T. japonica*, and polychaete species of the families Capitellidae, Neptyidae, Nereidae, Phyllocidae, and Spionidae—were collected in sufficient quantity for PAH analysis.

The collection, processing, and analysis sequence for sediments and organism tissues is illustrated in Figure 2. Once on site, the quadrat was delineated with a premarked nylon rope. Roughly half of the exposed sediments in the quadrat were collected to a depth of ~5 cm with a stainless steel scoop and bucket, mixing the contents periodically. The remaining sediment was sieved through a No. 10 mesh screen, and retained animals were placed in glass jars filled with ambient water using stainless steel forceps. All viable animals were collected; larger fragments of polychaete worms were also included. Immediately after collection, media were transported to our RFS laboratory where sediments were remixed and homogenized. A subsample (~500 ml) was then placed into a precleaned teflon or glass jar and frozen. Animals were separated by taxa, placed in filtered seawater, and left to stand for a 24-h period. Each specimen was then carefully rinsed with filtered seawater, tap, and finally with milli-Q grade water, placed in glass vials or kiln-fired aluminum foil, weighed, and promptly frozen.

Homogenized subsamples (10–50 g) of thawed wet sediment were weighed in a teflon beaker and oven dried at 100 ± 2°C for 24 h. Subsamples from each location were analyzed in duplicate (7/93 and 4/94) or triplicate (10/93 and 1/94). After weighing, dried sediment was homogenized with an equal amount of kiln-fired Na_2SO_4 in a glass pestle and mortar and extracted with 500 ml of CH_2Cl_2 in a soxhlet apparatus for 8 h minimum. Prior to extraction, perdeuterated PAHs (ULTRA Scientific; Kingston, RI, USA) were added to the sediment/Na_2SO_4 mixture as recovery surrogates. Soxhlet extracts were then reduced using rotary evaporation, redissolved in hexane, rereduced to 1 to 4 ml, and stored in glass vials with teflon-lined screw caps. Additional subsamples (~1 g) of dried sediment were homogenized with a small ceramic mortar and pestle and sent for TOC analysis (difference method) to Huffman Analytical Laboratories (Golden, CO, USA). The remaining frozen sediment aliquots were sent to Toxscan, Inc. (Watsonville, CA, USA) for grain size analysis (pipette method). Particle size distribution was reported for 14 size classes ($-4 \leq \Phi \leq 9$; 0.002 mm $< d_p \leq$ 32 mm), including sand ($d_p >$ 0.062 mm), silt (0.004 mm $< d_p \leq$ 0.062 mm), and clay ($d_p \leq$ 0.004 mm).

Frozen whole animals were thawed, pooled into one to three subsamples depending on expected lipid weight and/or sample availability, wet homogenized with Na_2SO_4, and soxhlet extracted as described previously for sediments. An exception was made for tissues of *T. japonica*, which were shucked and in some cases freeze-dried prior to extraction. Lipid extracts were protected from airborne fallout and allowed to evaporate to constant weight at room temperature. Lipid weight was then determined gravimetrically as the residue remaining after CH_2Cl_2 evaporation.

Sediment porewater was vacuum-extracted from homogenized whole sediments in a dark, cold room using a modified version of the vacuum-syringe extraction technique [24]. Extracted porewater was filtered with Whatman GF/F binder-free glass fiber filters (0.7 μm nominal pore size) under gentle vacuum. The filtrate was then decarbonized in 50-ml glass beakers by adding a few drops of concentrated H_2SO_4 and sparging with filtered, high purity (>99.99%) nitrogen for 10–15 min. Sparged aliquots were analyzed for dissolved organic carbon (DOC) using the persulfate-UV oxidation method [25] with a Xertex-Dohrmann DC-80 analyzer.

Sample cleanup, fractionation, and GC/MS analysis

Both tissue and sediment hexane extracts were cleaned up and fractionated using packed column chromatography, the detailed description of which is given elsewhere [1,23]. The PAHs were eluted from the sorbent (either Florisil or an alumina/silica gel combination) with a mixture of CH_2Cl_2 and hexane in the second fraction (F2) after elution of aliphatic/nonpolar interferences with hexane in the first fraction. Each F2 was then exchanged to hexane, reduced to ≤1 ml using a gentle stream of nitrogen, and analyzed on a Varian 3400 gas chromatograph (GC) coupled to a Saturn II ion trap mass spectrometer (ITMS) using helium as the carrier gas. The analysis conditions were as follows: sample volume, 1 μl; injection mode, splitless; GC column, DB-5MS, 30 m fused silica with 0.025 mm stationary phase film thickness (J&W Scientific); injector program, (1) 100°C for 0.10 min, (2) increase to 280°C at 300°C/min, (3) isothermal at 280°C for 5 min; column program, (1) 100°C for 5 min, (2) increase to 280°C at 4°C/min, and (3) isothermal at 280°C for the remaining 60-min program. The transfer line was held constant at 280°C. The ITMS was operated in the electron ionization mode with a mass detection range of 50–650 amu scanning at 1.0 scans/s and was turned on 4 min after sample injection.

Eighteen unsubstituted PAHs—naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benz[*a*]anthracene, chrysene, benzo[*b*] fluoranthene, benzo[*k*]fluoranthene, benzo[*e*]pyrene, benzo[*a*]pyrene, perylene, indeno[*1,2,3-cd*]pyrene, dibenz[*a,h*] an-

Table 1. Grain size, organic carbon (f_{oc}), and PAHs in Hoffman Marsh sediments[a]

ID[a]	Sampling date	%Sand	%Silt	%Clay	f_{oc}	$TPAH_{dw}$[b] (μg/g)	$TPAH_{oc}$[c] (μg/g)	MP/P[d]
IM-1	07/09/93	55.5	35.6	8.9	0.0095	1.4	150	0.37
MM-1	07/07/93	79.3	16.7	4.0	0.0078	0.65	84	0.36
OM-1	07/06/93	80.3	15.4	4.3	0.0039	0.23	59	0.66
IM-2	10/13/93	n/a	n/a	n/a	0.0072	0.34	47	0.47
MM-2	10/15/93	n/a	n/a	n/a	0.0039	0.051	13	0.53
OM-2	10/27/93	93.0	4.9	2.1	0.0033	0.026	7.9	0.72
IM-3	01/28/94	79.2	13.7	7.1	0.0076	0.62	82	0.43
MM-3	01/24/94	80.7	15.1	4.2	0.0059	0.54	91	0.50
OM-3	01/26/94	n/a	n/a	n/a	0.0044	0.18	41	0.68
IM-4	04/28/94	75.8	19.0	5.2	0.0051	0.49	96	0.51

[a] See Figure 1 for exact sampling locations.
[b] Sum of 18 2–6 ring unsubstituted PAHs on a dry weight basis.
[c] Sum of 18 2–6 ring unsubstituted PAHs on an organic carbon basis.
[d] Ratio of total methyl-phenanthrenes (sum of four isomers) to phenanthrene in sediment.

thracene, and benzo[*g,h,i*]perylene—were identified by GC/ITMS based on injections of an authentic PAH standard mixture (ULTRA Scientific). Average response factors for benzo[*a*]pyrene were substituted for benzo[*e*]pyrene and perylene, the two compounds not present in the standard mixture. Retention times for these isomers were confirmed from parallel work in our laboratory [26]. Instrument sensitivity was closely monitored by periodic injections of a dilute standard mixture. Final PAH levels in media were computed using PC spreadsheet software after correction for recovery of perdeuterated PAH surrogates and relative instrument sensitivity.

Quality control

For the analysis of PAHs, 50-g aliquots of kiln-fired Na_2SO_4 were processed and analyzed as procedural blanks. The number of detections of individual PAH in these blanks was very few and in all of these cases represented less than 1% of the lowest reported PAH concentration in either tissue or sediment. A standard reference marine sediment (HS-6; NRC Canada) and a *Mytilus edulis* homogenate (SRM1974; National Institute of Standards and Technology, Gaithersburg, MD, USA) were also analyzed. The mean percent deviation (MPD) from reported values for 16 of the 18 PAHs was 24 ± 19 for HS-6. The MPD for 11 congeners (of which only 9 were certified) was 23 ± 19 for SRM 1974. Mean recoveries of perdeuterated surrogates in sediment extracts were 45, 82, 92, 88, and 47% for naphthalene-d8, acenaphthene-d10, phenanthrene-d10, chrysene-d10, and perylene-d12, respectively. The corresponding values for tissue extracts were 57, 42, 89, 98, and 80%. The PAHs were included in the computation of BSAFs if mass spectra confirmed their presence (S/N ≥ 5) and surrogate recovery was greater than 50% but less than 150%. Mean coefficients of variation (CVs) for all PAHs (except perylene) were 24% and 28% for sediment replicates and pooled tissue subsamples, respectively. Perylene was not detected in any of the tissue samples and therefore was reported in sediment samples only. Only two PAHs, acenaphthene (65%) and benzo[*a*]pyrene (58%), had CVs greater than 50%. Detection limits for individual PAHs under these conditions were on the order of 0.5 ng/g dry weight and 5 ng/g fresh weight.

For DOC analysis, calibration curves of standard dilutions of potassium hydrogen phthalate were highly linear ($r^2 > 0.99$) in all cases. Procedural blanks, consisting of 100 ml of milli-Q water extracted with the vacuum apparatus, typically contained <2 mg DOC/L; freshly prepared milli-Q water typically measured <1 mg/L. The blank was unsatisfactorily high for the January 1994 IM-3 porewater; hence, DOC was not reported for this sample. Because the remaining DOC levels typically exceeded 20 mg/L, samples were not corrected for blanks.

RESULTS

Sediment grain size, organic carbon, and PAHs

The sediments collected in this study were sandy to silty muds with a relatively low organic carbon content (Table 1). The amount of sediment fines (%fines = %silt + %clay) ranged between 6.0 and 44% wet weight; the mass fraction of sediment organic carbon on a dry weight basis (f_{oc}) varied by a factor of three (0.0033–0.0095). Total PAHs as the sum of 18 unsubstituted congeners (see Materials and Methods) on a dry weight ($TPAH_{dw}$) and on an organic carbon basis ($TPAH_{oc}$) were relatively moderate in concentration (mid ng/g to low μg/g range) and varied by more than a factor of 20. The use of TPAH as a relative measure of PAH contamination is justified because individual congener concentrations (e.g., phenanthrene, fluoranthene, chrysene) were correlated ($p < 0.05$) with both $TPAH_{dw}$ and $TPAH_{oc}$. Furthermore, the less-than-unity ratios of methylphenanthrenes (sum of four isomers) to phenanthrene (MP/P) indicated that PAHs in the marsh were primarily combustion in nature [27,28].

Biota sediment accumulation factors

Compound-specific BSAFs were computed using Equation 1 for *P. amurensis*, *T. japonica*, and polychaetes (Tables 2, 3 and 4). Polychaete BSAFs were further subdivided into a ''large'' (nereids and neptyds) and ''small'' (mostly capitellids) category (Table 4). In cases where sufficient tissue allowed for the analysis of subsamples, mean BSAFs are reported. Although median values were similar, the magnitude of individual BSAFs ranged from 0.0069 to 5.4 g SOC/g lipid, a spread of almost three orders of magnitude (Table 5). Within each organism grouping, polychaetes had the lowest variation (factor of 50) and *T. japonica* the highest (factor of 300).

Intertidal zone and seasonal variations

Three trends in BSAF were apparent, the first of which was the consistent increase in BSAF from Inner to Outer Marsh locations. The rankings of BSAF values for each of the three major organism groups were compared by intertidal zone using

Table 2. Lipid content, total PAHs, and BSAF values for the asian clam (*Potamocorbula amurensis*)

Location	IM-1	MM-1	OM-1	IM-2	MM-2	OM-2	IM-3	OM-3
%lipid	0.48	0.46	0.15	0.29	0.36	0.32	0.68	0.43
$TPAH_{lip}$[b]	36	15	30	9.6	7.2	12	8.2	15
Acenaphthylene	0.424	2.70	—	5.42	—	—	—	—
Acenaphthene	—	0.588	—	—	—	—	—	—
Fluorene	0.569	0.945	3.37	1.71	1.35	2.30	0.196	0.409
Phenanthrene	0.286	0.348	2.01	0.464	1.09	2.79	0.169	0.567
Anthracene	0.176	0.088	1.20	0.288	—	—	0.146	—
Fluoranthene	0.268	0.355	1.03	0.281	1.11	2.20	0.112	0.589
Pyrene	—	—	—	—	0.116	1.50	0.208	0.856
Benz[*a*]anthracene	1.82	—	1.03	—	—	2.48	0.085	0.702
Chrysene	0.261	0.184	0.519	0.263	0.430	0.969	0.133	0.316
Benzo[*b*]fluoranthene	0.219	0.292	0.383	0.362	0.820	1.11	—	0.117
Benzo[*k*]fluoranthene	0.172	0.351	0.481	0.398	0.605	1.15	0.088	0.291
Benzo[*e*]pyrene	0.214	0.274	—	—	0.752	1.09	0.106	0.293
Benzo[*a*]pyrene	—	—	0.168	—	—	—	0.062	0.190
Indeno[*123cd*]pyrene	—	0.166	—	—	—	1.33	0.158	0.355
Dibenz[*ah*]anthracene	—	0.071	—	—	0.555	1.18	—	—
Benzo[*ghi*]perylene	—	—	0.078	—	—	—	0.112	0.310

[a] Percent lipid wet-weight basis.
[b] Sum of 17 2–6 ring unsubstituted PAHs on an organism lipid basis (μg/g lipid).

nonparametric ANOVA (Kruskal–Wallis method). In all cases, a difference at the 0.01 level was observed (Table 6). Individual multiple comparisons were made using Bonferroni's inequality; the overall type I error in these tests was selected as 0.05. The BSAFs for *P. amurensis* from the IM were determined to be different from (lower than) those from the OM ($p < 0.017$). For *T. japonica,* BSAFs in each intertidal zone (i.e., IM, MM, and OM) were different among themselves, with IM specimens having the lowest values and OM specimens the highest. For polychaetes, BSAFs for the IM were lower than those of either the MM and OM.

The second trend was the difference in BSAF by sampling season. The highest BSAFs for *P. amurensis* were measured in the fall event (October) followed by the summer (July) and winter (January) (Table 6). For *T. japonica,* BSAFs for three of the six possible seasonal comparisons were significantly different ($p < 0.0083$) with the fall BSAFs again being the highest. Similarly, polychaete BSAFs for July 1993 were different than those determined for January and April 1994 ($p < 0.017$). In each case, values were lowest in the rainy season (January and April).

The third trend was the decrease in BSAF with increasing PAH hydrophobicity, particularly for the two bivalves. This relationship, along with the intertidal zone and seasonal differences, can be seen in plots of BSAF versus log K_{ow}. Values of K_{ow} were taken from several sources [14,29–31]. The BSAF was negatively correlated with log K_{ow} ($p < 0.05$) for seven of the nine possible intertidal zone data sets (Fig. 3). The two exceptions were for polychaetes in the high- and middle-intertidal zones. In addition, the dependence of BSAF on log K_{ow} (as measured by the regression slopes) increased with decreasing intertidal height for all organism groups. This increase in slope corresponded to decreases in f_{oc} and %FINES. Furthermore, $TPAH_{oc}$ decreased along this transect. The effect of season on the relationship of BSAF and log K_{ow} was consistent among organism groups (Fig. 4), although fewer (4 of 10 possible) regressions were significant ($p < 0.05$).

Differences among taxa

A four-way ranking of BSAFs (categories were *P. amurensis, T. japonica,* large and small polychaetes) again using Kruskal–Wallis ANOVA revealed that a difference in BSAF among organism groups was significant ($\chi^2 = 30.51$; $p < 0.001$); however, individual multiple comparisons using Bonferroni's inequality indicated that BSAFs for small and large polychaetes were not significantly different ($Z = -1.01$; $p > 0.05$). These data were therefore pooled into a single polychaete composite. Repeating this analysis with three categories (*P. amurensis, T. japonica,* and polychaetes) revealed that a significant difference among BSAF values still existed among the organism groups ($\chi^2 = 29.5$; $p < 0.001$). Individual multiple comparisons showed that BSAFs for *P. amurensis* were different than both *T. japonica* and polychaetes ($Z = -5.27$ and -3.57, respectively; $p < 0.017$ for both).

Lipid content and porewater DOC

Lipid content (%lipid) ranged from 0.15 to 2.1% wet weight for the three groups of organisms; polychaete composites generally had the highest lipid content, shucked tissues of *T. japonica* had intermediate levels, and *P. amurensis* had the lowest (Tables 2–4). These levels are consistent with those reported for sandworms and mussels—1.78 and 1.42%, respectively—using a chloroform/methanol extraction procedure [32]. Lipids were generally highest at IM sites and lowest in specimens from the OM. Correspondingly, %lipid was linearly correlated with %fines for *T. japonica* ($r^2 = 0.81$, $p \leq 0.05$) and with f_{oc} for both bivalves ($r^2 = 0.53$ [*P. amurensis*] and 0.64 [*T. japonica*]; $p < 0.05$ for both). No relationships between %fines or f_{oc} and %lipid for polychaetes were noted. The sum of 17 PAHs (excluding perylene) in lipids, or $TPAH_{lip}$, varied by a factor of 10 (3.6–36 mg/kg lipid). No obvious trends were apparent across organism groups; however, $TPAH_{lip}$ were generally highest for bivalves whose %lipid were lowest (i.e., at OM locations). There were no significant correlations between $TPAH_{lip}$ and any of the other measured parameters (including $TPAH_{dw}$ and $TPAH_{oc}$).

Porewater DOC concentrations for our limited sample size ($n = 6$) ranged between 16 mg/L for IM-4 to 93 mg/L for OM-3. The remaining levels were 69, 28, 70, and 67 for IM-2, MM-2, OM-2, and MM-3, respectively. These levels were somewhat higher than those reported in Boston Harbor sediment cores (5.3–23 mg/L) [33] but were comparable in range

Table 3. Lipid content, total PAHs, and BSAF values for the Japanese littleneck clam (*Tapes japonica*)

Location	IM-1	MM-1	OM-1	IM-2	MM-2	OM-2	IM-3	IM-4
%lipid[a]	1.6	0.93	0.32	0.69	0.63	0.25	0.85	1.1
$TPAH_{lip}$[b]	5.6	8.3	27	5.1	5.2	7.7	5.1	3.6
Naphthalene	—	—	—	—	—	—	—	2.09
Acenaphthylene	0.208	1.69	—	0.807	—	—	—	0.102
Acenaphthene	—	0.108	—	—	—	—	—	0.465
Fluorene	0.184	0.377	—	0.695	1.08	1.87	—	0.208
Phenanthrene	0.100	0.160	0.937	0.271	1.15	2.66	0.050	0.085
Anthracene	0.057	0.133	—	—	—	—	—	0.067
Fluoranthene	0.113	0.151	0.462	0.159	0.985	1.78	0.087	0.082
Pyrene	0.054	0.090	—	—	0.197	0.440	0.155	0.083
Benz[*a*]anthracene	0.071	0.076	1.24	—	—	—	0.139	0.047
Chrysene	0.059	0.103	0.313	0.168	0.373	1.03	0.118	0.034
Benzo[*b*]fluoranthene	0.064	0.151	0.216	0.187	0.687	1.27	—	—
Benzo[*k*]fluoranthene	0.046	0.128	0.170	0.132	0.347	0.353	0.067	—
Benzo[*e*]pyrene	0.056	0.184	0.120	—	—	—	0.057	—
Benzo[*a*]pyrene	0.014	0.048	—	0.184	—	—	—	—
Indeno[*123cd*]pyrene	—	0.043	0.102	—	—	—	—	—
Dibenz[*ah*]anthracene	—	0.278	—	0.102	—	0.415	—	0.040
Benzo[*ghi*]perylene	—	—	—	0.135	—	—	—	0.0069

[a] Percent lipid wet-weight basis.
[b] Sum of 17 2–6 ring unsubstituted PAHs on an organism lipid basis (μg/g lipid).

to those reported in a 36-cm core from New Bedford Harbor [34]. No seasonal or intertidal zone patterns were apparent for these data. In addition, DOC was not correlated with %fines or f_{oc}, or any other parameter, including total PAHs in sediments or lipids.

Relationships of BSAF with other parameters

The BSAF was also negatively associated with log K_{ow} (Fig. 5; Eqns. 2–4) at the 0.05 level of significance for each of the three organism groups.

P. amurensis

$$\text{BSAF} = 2.9 - 0.39 \log K_{ow} \quad (n = 84;\ r^2 = 0.15;\ p < 0.001) \tag{2}$$

T. japonica

$$\text{BSAF} = 1.6 - 0.23 \log K_{ow} \quad (n = 79;\ r^2 = 0.14;\ p < 0.001) \tag{3}$$

Polychaetes

$$\text{BSAF} = 0.93 - 0.18 \log K_{ow} \quad (n = 63;\ r^2 = 0.090;\ p < 0.05) \tag{4}$$

The magnitude of the dependence of BSAF on log K_{ow} (i.e., the slope of these equations) decreases from *P. amurensis* to *T. japonica* to polychaetes. The slope of Equation 2 for *P. amurensis* is significantly different than that for polychaetes (Student's *t*-test; $p < 0.05$).

Other parameters, however, explained portions of the variance of BSAF consistently as much or more than did log K_{ow}. Stepwise multiple regression analysis of BSAF against all study parameters (sediment fines, organic carbon and PAH concentrations, porewater dissolved organic carbon, organism lipid and PAH concentrations) showed that (1) for *P. amurensis*, a combination of f_{oc}, log K_{ow}, and %fines explained 55% of the total variation in BSAF, with individual contributions of 32, 18, and 5.3%, respectively; (2) for *T. japonica*, $TPAH_{oc}$, log K_{ow}, and %fines explained 30, 15, and 7%, respectively; and (3) for polychaetes, $TPAH_{dw}$, log K_{ow}, and

Table 4. Lipid content, total PAHs, and BSAF values for polychaetes

Location	OM-1	IM-3	MM-3	OM-3	IM-3	MM-3	IM-4
Group	Large	Large	Large	Large	Small	Small	Small
%lipid[a]	2.0	2.1	2.0	0.96	1.7	1.8	0.66
$TPAH_{lip}$[b]	16	7.6	3.3	6.5	21	20	12
Acenaphthene	—	—	—	—	2.04	—	—
Fluorene	1.13	0.196	—	—	0.286	0.312	—
Phenanthrene	0.652	0.169	0.166	0.488	0.169	0.267	0.149
Anthracene	—	0.146	—	—	0.271	0.214	0.132
Fluoranthene	0.312	0.112	0.047	0.308	0.279	0.266	0.161
Pyrene	—	0.208	0.169	0.803	0.575	0.514	0.342
Benz[*a*]anthracene	1.40	0.085	—	0.109	0.213	0.218	0.122
Chrysene	0.335	0.133	0.072	0.201	0.223	0.228	0.192
Benzo[*b*]fluoranthene	0.160	—	—	—	0.140	—	—
Benzo[*k*]fluoranthene	0.421	0.088	—	—	0.236	0.255	0.162
Benzo[*e*]pyrene	0.221	0.106	—	—	0.391	0.309	—
Benzo[*a*]pyrene	—	0.062	—	—	0.191	—	—
Indeno[*123cd*]pyrene	—	0.158	—	—	0.124	0.355	0.054
Benzo[*ghi*]perylene	—	0.112	—	—	0.267	0.295	0.185

[a] Percent lipid wet-weight basis.
[b] Sum of 17 2–6 ring unsubstituted PAHs on an organism lipid basis (μg/g lipid).

Table 5. Median and range of BSAF values by organism group (g sediment organic C/g lipid)

Parameter	*Potamocorbula amurensis*	*Tapes japonica*	Polychaetes
No. BSAF values	84	79	63
Median	0.30	0.15	0.20
Minimum	0.062	0.0069	0.044
Maximum	5.4	2.1	2.0
Max/min	87	300	46

$TPAH_{lip}$ accounted for 9.5, 9.2, and 8.1%, respectively, of the BSAF variance.

In addition to the solubilities of PAHs in water, or specifically the ratio of their solubilities in octanol and water, both the organic carbon content (f_{oc}) and the nature of the organic carbon matrix appear therefore to influence the values of BSAF obtained in this study. Spearman's rank correlation analysis of BSAFs of individual PAHs also showed that BSAFs decreased significantly with increasing f_{oc}; moreover, BSAFs also decreased significantly with increasing PAH concentrations on an organic carbon basis (Table 7). A heterogeneity of the organic carbon matrix is therefore indicated.

Furthermore, the logarithms of measured values of K_{oc}' [1], the ratio of concentrations in the sediments on an organic carbon basis to concentrations in the porewaters were, except for the small polychaetes, more strongly correlated with BSAFs than were log K_{ow}s (Table 8).

DISCUSSION

The concept of equilibrium partitioning, as developed to date for the establishment of sediment quality criteria for nonionic chemicals, addresses varying bioavailability across sediments by normalizing concentrations on an organic carbon basis and using the octanol–water partition coefficients to establish a relationship between concentrations in water and those in sediments [10]. Contrary to the predictions of this theory, this study has shown that measured values of BSAF varied both seasonally and spatially in an intertidal marsh in San Francisco Bay. Moreover, the variation was considerably greater than the factor of two to three considered to be sufficient to account for unexplained variability.

Because the patterns of variability in BSAFs closely parallel the seasonal and spatial variability in measured values of K_{oc}', which we have attributed to seasonal and spatial variations in the soot content of sediments [1], we also attribute the major portion of the variability in BSAF that is unexplained by equilibrium partitioning [10] to the soot content of the sediments. In the San Francisco Bay area, the local rainy season typically begins in November and extends into April, with little or no rain between May and October. Input of combustion-derived PAH, associated with soot particles, into this highly urbanized estuary from surface runoff would therefore occur during the rainy season only. The PAHs would be expected to maintain their affinity with the highly aromatic soot particles [35] because their chemical activity coefficients would be lower in this medium than in other types of sediment organic carbon matrices (e.g., those derived primarily from ''natural'' or ''biogenic'' sources [18]). Proportionately more of the PAHs in this intertidal environment would therefore be associated with the organic carbon fraction of the sediments during the local rainy season, hence the lower values of the measured BSAFs.

During the dry season, continued resuspension of finer sediment particles in areas of wind and tidal activity would result in their preferential deposition in areas of lowest resuspension, particularly the high intertidal zone (IM) [13]. Moreover, the average diameter of soot particles (~0.004 mm) to which 97% of benzo[*a*]pyrene in air was sorbed was well within the silt/clay particle size regime [36]. The amount of sediment fines

Table 7. Spearman's rank correlation coefficients (r_s) for BSAF versus sediment organic carbon (f_{oc}) and organic carbon normalized total PAHs ($TPAH_{oc}$)

	f_{oc}	$TPAH_{oc}$
Potamocorbula amurensis (n = 8 all)		
Fluorene	−0.635	−0.405
Phenanthrene	−0.922[b]	−0.786[a]
Fluoranthene	−0.874[b]	−0.786[a]
Chrysene	−0.898[b]	−0.762[a]
Benzo[*k*]fluoranthene	−0.826[a]	−0.738[a]
Tapes japonica		
Fluorene (n = 6)	−0.829[a]	−1.00[b]
Phenanthrene (n = 8)	−0.731[a]	−0.810[a]
Fluoranthene (n = 8)	−0.683[a]	−0.857[b]
Chrysene (n = 8)	−0.755[a]	−0.952[b]
Benzo[*k*]fluoranthene (n = 7)	−0.955[b]	−0.929[b]
Polychaetes		
Phenanthrene (n = 7)	−0.473	−0.800[a]
Fluoranthene (n = 7)	−0.582	−0.691
Chrysene (n = 7)	−0.400	−0.364
Benzo[*k*]fluoranthene (n = 5)	−0.616	−0.410

[a] $p < 0.05$.
[b] $p < 0.01$.

Table 6. χ^2 and Z values for intertidal zone and seasonal comparisons of BSAF

Organism group	χ^2	Z-statistic
Intertidal zone		
P. amurensis	16.33[a]	−2.07 (IM-MM); −1.65 (MM-OM); 4.16[c] (IM-OM)
T. japonica	240.0[a]	−2.49[c] (IM-MM); −2.50[c] (MM-OM); −4.91[c] (IM-OM)
Polychaetes	68.78[a]	4.52[c] (IM-MM); −1.51 (MM-OM); −3.37[c] (IM-OM)
Season		
P. amurensis	25.33[a]	−3.08[c] (Jul–Oct); 4.98[c] (Oct–Jan); 2.18[c] (Jul–Jan)
T. japonica	240.0[a]	−3.83[c] (Jul–Oct); 3.37[c] (Oct–Jan); 1.06 (Jul–Jan) 1.10 (Jul–Apr); 3.92[c] (Oct–Apr); −0.146 (Jan–Apr)
Polychaetes	10.15[b]	2.53[c] (Jul–Jan); 1.51 (Jan–Apr); 3.13[c] (Jul–Apr)

[a] $p < 0.001$.
[b] $p < 0.01$.
[c] $p < 0.05$ (overall type I error).

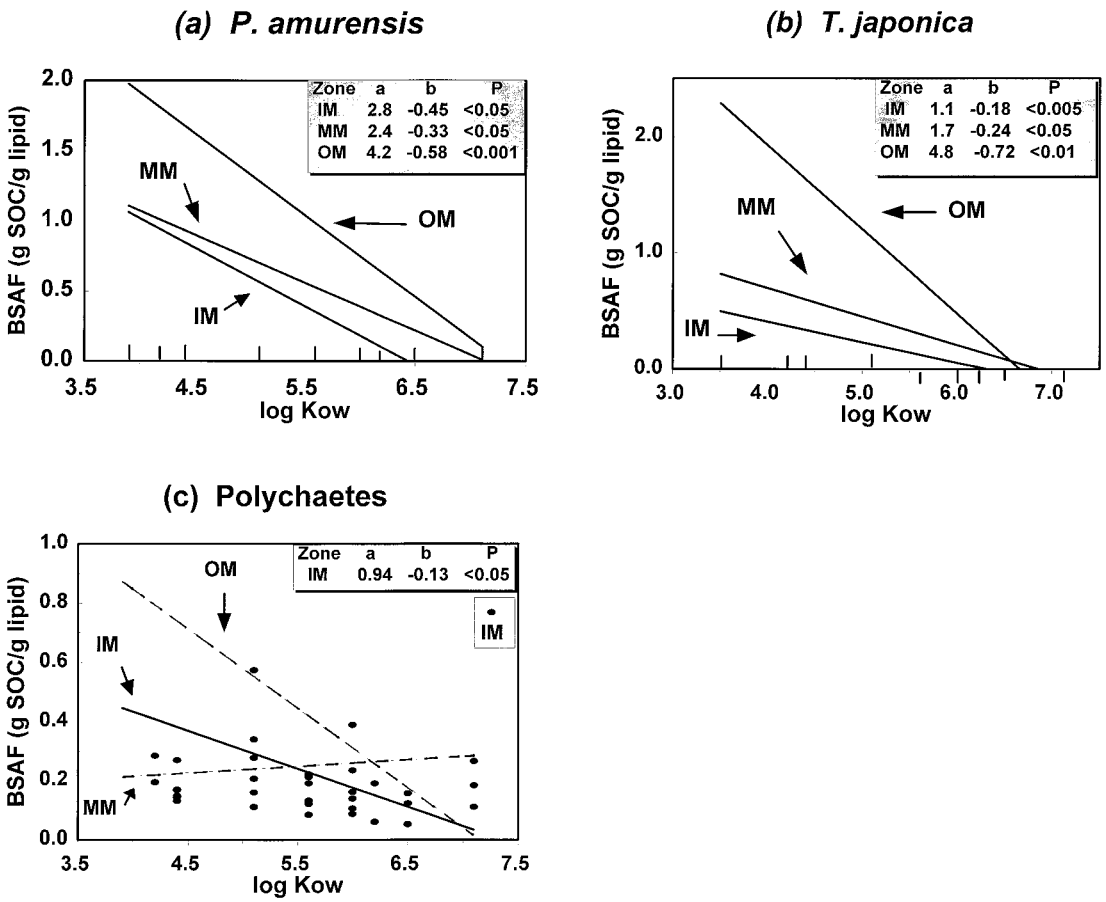

Fig. 3. Relationship between BSAF and log K_{ow} according to intertidal zone for (**a**) *Potamocorbula amurensis,* (**b**) *Tapes japonica,* and (**c**) polychaetes. Regression equations are of the form $y = a + bx$; p = probability of no correlation. Solid lines denote significant correlations ($p < 0.05$); broken lines represent linear ($p > 0.05$) fits for comparison purposes only.

(silt and clay fractions) and organic carbon-normalized sediment PAHs in our study were highest in this zone [1]; we propose that this would account for the observed spatial variations in BSAF.

Earlier conclusions that the nature of the sediment organic matrix did not affect the bioavailability of the PAH fluoranthene to benthic organisms might therefore be reconsidered because only ''natural'' sources of organic carbon were evaluated [18]. Based on our results, the organic carbon matrices of sediments exposed to recent surface runoff or in areas of deposition of fine particles in an urban environment are likely to be more ''soot-like'' with the corresponding effects on partitioning and bioavailability of combustion-source PAHs.

The stronger association of BSAFs with log K_{oc}', equivalent to the logarithm of the ratios of chemical activity coefficients in porewaters and sediments, than with log K_{ow}, equivalent to the ratios of these coefficients in water and octanol, indicates a limitation of octanol as a surrogate for all types of organic carbon matrices in sediments. The theoretical basis of equilibrium partitioning would therefore be more appropriately based on ratios of chemical activity coefficients, expressed as K_{oc}s, rather than on K_{ow}s. For most uses, these have to date been considered to be equivalent or near equivalent in the development of the equilibrium partitioning concept [10]. Phenomena previously unexplained, or apparently inconsistent with the predictions of equilibrium partitioning, such as the observed greater uptake clearance for 2,4,5,2′,4′,5′-hexachlorobiphenyl than for benzo[*a*]pyrene, in spite of a higher K_{ow} [37] are, however, more readily explained by media-associated differences of chemical activity coefficients under equilibrium or near-equilibrium conditions [1]. More specifically, we propose that by making allowances for K_{oc}s not always being equivalent to K_{ow}s, the predictability of BSAFs can be improved.

Several polychaete species have been shown to possess the enzyme systems that transform aromatic hydrocarbons, including PAHs, into polar metabolites [38–40], whereas there is very little evidence that bivalves possess similar capabilities [38,41]. Species that possess these systems show lower BSAFs when PAH concentrations are reported, as in our study, as parent compounds alone rather than as the sum of parent compounds and polar derivatives [42]. The lower BSAFs we have reported for polychaetes than for bivalves may therefore result from metabolism. The PAH profile, the relative amounts among the principal compounds, was similar in polychaetes and sediments. This does not necessarily indicate a lack of metabolism because there is no evidence for selective metabolism of either lower or higher MW compounds [43].

It has been proposed that kinetic limitations may affect PAH partitioning behavior and bioavailability [44]. These may in part explain the decreasing BSAFs with increasing K_{ow}s observed in both bivalve species in our study. Although these are primarily surface detritus feeders, they are also exposed to the sediment/water column interface. Data of Rubinstein et al. [45] analyzed by DiToro et al. [10] show, however, that decreasing BSAFs of a series of PCB congeners were also

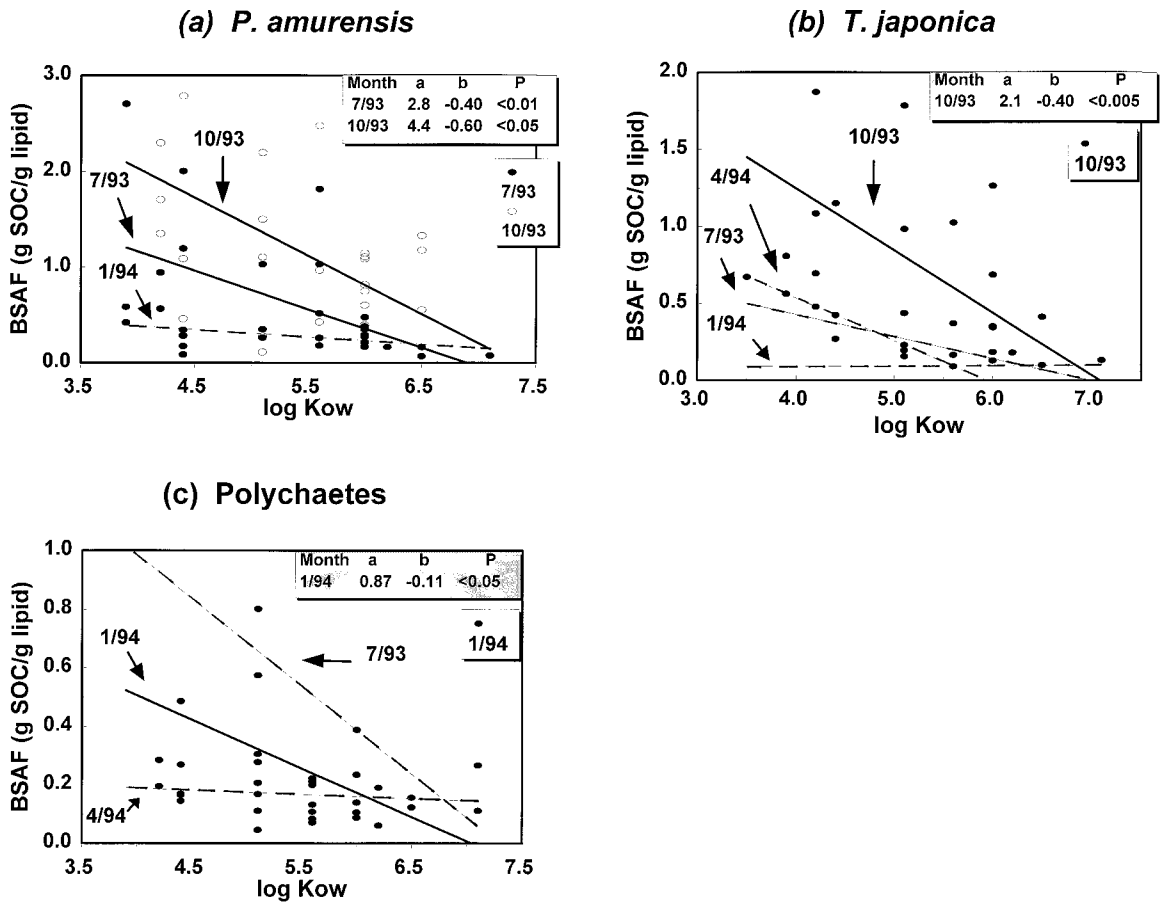

Fig. 4. Relationship between BSAF and log K_{ow} according to season for (**a**) *Potamocorbula amurensis*, (**b**) *Tapes japonica*, and (**c**) polychaetes. Regression equations are of the form $y = a + bx$; p = probability of no correlation. Solid lines denote significant correlations ($p < 0.05$); broken lines represent linear ($p > 0.05$) fits for comparison purposes only.

associated with increasing K_{ow}s when bivalves were exposed to spiked sediments over a period of time considered sufficient for attainment of equilibrium conditions. It appears likely therefore that the observed relationships between BSAFs and log K_{ow}s of the respective congeners result from the relationships among the chemical activity coefficients in bivalve lipid, local sediments, and local water.

The higher lipid content of the organisms in the higher intertidal zone and lower lipid content in areas covered by water for longer periods of time may be associated with the abundance and quality of food in this area; measurements of benthic algal productivity have shown that the high intertidal is three to four times more productive than other areas of San Francisco Bay (J. Gregg and A.J. Horne, unpublished data). We do not attribute differences in lipid content to any kind of contaminant effect. The levels of PAHs measured in this study are well below levels associated with induced tumors in bot-

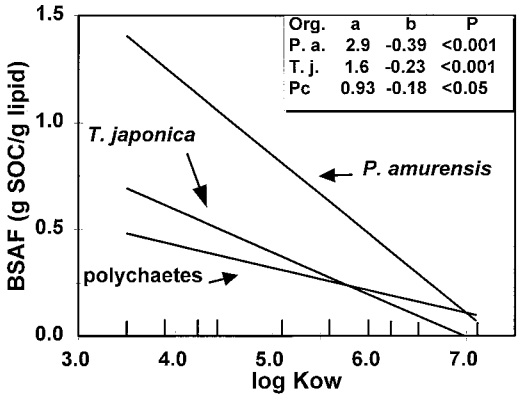

Fig. 5. BSAF and log K_{ow} by organism (*Potamocorbula amurensis* [P.a.], *Tapes japonica* [T.j.], and polychaete composites [Pc]). Regression equations are of the form $y = a + bx$; p = probability of no correlation.

Table 8. Spearman's rank correlation coefficients (r_s) for BSAF versus log PV (PV = K_{ow} or K_{oc}')

Predictor variable (PV)	n	r_s	p
Potamocorbula amurensis			
K_{ow}	26	−0.535	<0.005
K_{oc}'	26	−0.701	<0.005
Tapes japonica			
K_{ow}	17	−0.548	<0.025
K_{oc}'	17	−0.766	<0.005
Large polychaetes			
K_{ow}	13	−0.376	>0.05
K_{oc}'	13	−0.638	<0.025
Small polychaetes			
K_{ow}	14	−0.547	<0.05
K_{oc}'	14	−0.526	<0.05

tom-dwelling fish [46,47] and have not been associated with any documented effects on a population level.

Our suggested modification of the equilibrium partitioning concept, while enhancing the theoretical predictability of bioaccumulation factors, would in practice require that the uncertainty factor in predicting bioaccumulation of PAHs based on log K_{ow} be increased to a factor of about 10 in the direction of lower bioavailability. This is in agreement with an evaluation of equilibrium partitioning for pyrene based on measured sediment toxicity and toxicokinetic parameters [44]. This level of uncertainty, compared with the previously acknowledged unexplained variability of two to three in measures of bioavailability [10], would account for differences in organic carbon matrices in the real world.

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EPA **ECO Update**

Intermittent Bulletin

The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Risk Assessments

Screening-Level Ecological Risk Assessments (SLERAs)

Screening-Level Ecological Risk Assessments are conservative assessments in that they provide a high level of confidence in determining a low probability of adverse risk, and they incorporate uncertainty in a precautionary manner. It must be stressed that SLERAs are not designed nor intended to provide definitive estimates of actual risk, generate cleanup goals and, in general, are not based upon site-specific assumptions. Rather, the purpose of SLERAs is to assess the need, and if required, the level of effort necessary, to conduct a detailed or "baseline" ecological risk assessment for a particular site or facility. Therefore, refinement of contaminants of concern occurs in the baseline risk assessment rather than in the SLERA.

It is also important to note that SLERAs, like baseline ecological risk assessments, should take place with input from Regional Ecological Risk Assessors and/or the Biological Technical Assistance Group as well as in coordination with Natural Resource Trustees.

IN THIS BULLETIN

Introduction

This supplemental Ecological Risk Assessment (ERA) guidance is intended to provide further

clarification and direction regarding Screening Level Ecological Risk Assessments (SLERAs), as described in Step 1 - Preliminary Problem Formulation, and Step 2 - Preliminary Risk Calculation, of the Agency's program guidance: *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments* (U.S. EPA 1997).[1] It also provides an approach for incorporating additional components into the Problem Formulation phase of more detailed (i.e., "baseline") ecological risk assessments, particularly in Step 3.2, which discusses refining contaminants of potential concern (COPCs). The Superfund program guidance, which may be applicable to RCRA Corrective Actions, describes a process that incorporates flexibility in refining COPCs in order to focus and streamline the overall ERA process while still ensuring a consistent approach. This guidance provides more detail on how to incorporate that flexibility.

The Purpose of Screening-Level Ecological Risk Assessments

Screening-Level Ecological Risk Assessments provide a general indication of the *potential* for ecological risk (or lack thereof) and may be conducted for several purposes including: 1) to estimate the likelihood that a particular ecological risk exists, 2) to identify the need for site-specific data collection efforts, or 3) to focus site-specific ecological risk assessments where warranted.

It is important to note that this guidance adopts the presumption that all data used in the SLERA are of adequate quantity and quality, and if data deficiencies are identified, either further data collection will be undertaken or other means employed to more fully characterize exposures (e.g., fate and transport modeling). If, for example, the SLERA indicates that adverse ecological effects are possible at environmental concentrations below standard quantitation limits, a "non-detect" based on those limits cannot be used as the sole basis for a "no risk" decision. Further lines of evidence (e.g., more refined/usable data, modeling results, or other measures) are needed to fully characterize the potential for adverse effects.

This guidance also reaffirms that a screening level assessment, while abbreviated, is nonetheless a complete risk assessment. Therefore, regardless of the findings of the Scientific Management Decision Point (SMDP) occurring after Step 2 (i.e., further assessment or no further assessment required), each SLERA should include documentation supporting the risk characterization and uncertainty analysis.

If the SLERA risk characterization indicates the need for further assessment, Step 3 is begun, and decisions are made regarding additional elements of problem formulation, analysis and decision point criteria. This supplemental guidance addresses how background, frequency and magnitude of detection, and dietary considerations may be used to reduce the COPCs. The use of site-specific information, as provided for in this *ECO Update*, should be discussed with the Regional Ecological Risk Assessors and/or Biological Technical Assistance Group (BTAG) early in the Problem Formulation phase of the baseline ERA.

It is the intent of this supplemental guidance to promote consistency in the screening process, yet allow for flexibility in application and timing of the elements that can help streamline more detailed assessments. Screening-Level Ecological Risk Assessments may be completed in relatively short time frames, whereas baseline ERAs may require much longer periods for planning and implementation, particularly when attempting to address seasonal or other cyclic events. Regional Ecological Risk Assessors can use this flexible approach when introducing components into the Problem Formulation phase based on regional and site-specific needs. This will effectively reduce the COPCs carried through the baseline ERA and the time required for its completion.

The Purpose of Baseline Ecological Risk Assessments

Within the Office of Solid Waste and Emergency Response (OSWER), the Superfund and RCRA Corrective Action cleanup programs generally use baseline ecological risk assessments to: "1) identify and characterize the current and potential threats to the environment from a hazardous substance release, 2) evaluate the ecological impacts of alternative remediation strategies, and 3) establish cleanup levels in the selected remedy that will protect those natural resources at risk." (U.S. EPA 1994e, OSWER Directive # 9285.7-17). The Superfund program guidance outlines an eight-step process that meets the three OSWER objectives for the baseline ERA while further implementing the Agency's policy of writing risk assessments that provide transparency in EPA's decision making process and clarity in communication with the public regarding environmental risk (U.S. EPA 1995, Risk Characterization Policy). In addition, application of the information in this ECO Update should further ensure that, for OSWER cleanups, core assumptions and science policy are consistent and comparable across programs, well grounded in science, and fall within a "zone of reasonableness."

[1] The first three steps of the Superfund ecological risk assessment process are described in the text box on page 3.

Standard Components of ERAs

The following text box highlights the risk assessment components common to both a SLERA and the Problem Formulation phase of a baseline ERA. In addition, the text helps to identify points in the ERA process where additional components may be considered in developing risk estimates.

Components of a SLERA

Although less detailed than a baseline ERA, screening assessments still include all of the following components:

- Screening level Problem Formulation and Ecological Effects Characterization (Step 1)
  - Identification of environmental setting and preliminary contaminants of concern
  - Determination of contaminant fate and transport pathways
  - Description of contaminant mechanisms of ecotoxicity and categories of receptors likely affected
  - Identification of complete exposure pathways and selection of generic assessment endpoints
  - Selection of screening ecotoxicity values
  - Evaluation of uncertainties
- Screening level Exposure Estimate and Risk Calculation (Step 2)
  - Determination of screening-level exposure estimate
  - Calculation of risk estimate
  - Risk characterization and evaluation of uncertainties
- Scientific Management Decision Point indicating either negligible risk or continuation to a baseline risk assessment

Components of a Baseline Ecological Risk Assessment Problem Formulation

Problem Formulation for a baseline ERA (Step 3) includes the following components:

- Refinement of the Contaminants of Potential Concern (COPCs) by examining the assumptions used in Steps 1 and 2
- Further characterization of ecological effects of contaminants
- Reviewing and refining information on contaminant fate and transport, complete exposure pathways, and ecosystems potentially at risk
- Selection of site-specific assessment endpoints
- Development of a conceptual model and associated risk questions
- Scientific Management Decision Point summarizing agreement on contaminants of concern, assessment endpoints, exposure pathways, and risk questions

Refining Contaminants of Concern

Screening is the comparison of site media concentrations with conservative toxicologically based numbers. Contaminants of concern may be refined to help streamline the overall ERA process by considering additional components early in the baseline ERA. After consultation with your Regional Ecological Risk Assessors and/or BTAG, one or more of the following components may be included in Step 3.2 of Problem Formulation. When added, it is important that the resulting Risk Characterization and Uncertainty Analysis fully address the issues listed for each component and describe the rationale underlying the selection of each component.

These components need not be implemented in the order presented in this document, nor do all the components need to be implemented. If, however, any contaminants are identified for exclusion from the baseline ERA through application of any or all of the three supplemental components described herein, it is essential to evaluate bioaccumulation, biomagnification, and bioconcentration of each such contaminant as well.

Supplemental Component 1: Background

Background concentrations of contaminants are those concentrations found in areas surrounding a site, but are unrelated to site releases. Contributions to these contaminant concentrations come from two major sources: first, natural sources (i.e., geologically derived concentrations of chemicals in the environment not influenced by human activity), and second, ambient or anthropogenic sources (i.e., concentrations present due to human activities, such as automobile use or pesticide dispersion in farming areas).

While contaminants of concern may be removed from further assessment through comparison with toxicological benchmarks, comparison with background levels generally cannot be used to remove contaminants of concern owing to the need to fully characterize site risk. Such comparisons, however, can be used effectively to focus the baseline risk assessment, if needed. An example of the application of background comparisons would be at a mining site with high levels of naturally occurring background metals due to local or nearby geological formations.

Consideration of background assumes that background contaminant levels have been properly determined. Until specific guidance on determining background levels is available, consult with your Regional Ecological Risk Assessors and/or BTAG to select an acceptable approach including minimum data requirements.

Issues to be discussed:

1. Potential toxicity of any contaminants identified as below background (particularly when toxicity benchmarks are lacking or when contaminants exceed toxicity benchmarks);
2. Potential for adverse effects caused by interactions between chemicals considered as background and those COPCs to be further investigated; and
3. Enumeration of all criteria by which contaminants are considered either background or site-related.

Supplemental Component 2: Frequency and Magnitude of Detection

Use of this component presumes that the sampling plan comports with *Guidance for Data Useability in Risk Assessment* (U.S. EPA 1992e). In particular, the sampling plan needs to characterize the full range of variability and distribution in the data and also needs to satisfactorily meet the criteria for completeness, comparability, representativeness, precision, and accuracy.

Similar to this supplemental guidance, current EPA human health risk assessment guidance discusses evaluation of COPCs based on frequency of detection and provides conditions under which compounds may be eliminated from further assessment. Owing to the typically small datasets available for ERAs, particularly screening-level assessments, compared to most human health risk assessments, a number of the conditions may not be applicable to ERAs. Nonetheless, given adequate data quality, further reduction of COPCs through application of this component may be determined acceptable following consultation with the Regional Ecological Risk Assessors and/or BTAG. Furthermore, the Project Manager's approval should be obtained before eliminating any chemicals from the risk assessment.

Issues to be discussed:

1. Influence of random and/or biased sampling on the frequency and magnitude of detected values within the distribution of data;
2. Spatial and temporal pattern of contaminants identified as low frequency and/or low magnitude;
3. Comparison of risk-based detection limits with toxicity benchmarks; and
4. Relationship of detected values to toxicity benchmarks.

Supplemental Component 3: Dietary Considerations

A number of chemicals that may be site-related function as nutrients in organisms serving as physiological electrolytes, such as calcium, iron, magnesium, sodium, and potassium. When present at concentrations that allow them to function in this manner, they typically pose little ecological risk. Conversely, nutrients such as selenium, copper, molybdenum, and boron, can transition from essential to toxic at only slightly higher concentrations.

Issues to be discussed:

1. The suite of nutrients relevant to the range of ecological receptors (wildlife vs. plants) considered at the site;
2. The potential for toxic effects resulting from site concentrations relative to the toxicological benchmarks for nutrients;
3. Whether contaminant interactions may result in a nutrient deficiency for organisms of concern; and
4. Whether the nutrient deficiency level and the toxicity benchmark are similar in magnitude.

Additional Considerations

For those COPCs identified by applying any of the components listed above, it is essential to evaluate their potential to bioaccumulate, bioconcentrate, and/or biomagnify prior to eliminating them from further consideration in the risk assessment. Compounds with a high potential to accumulate and persist in the food chain should be carried through the risk assessment.

Issues to be discussed:

1. The likelihood that contaminants identified for removal from the list of COPCs could exert adverse effects on higher trophic level organisms; and

2. A determination that bioaccumulation and/or biomagnification has been satisfactorily addressed through modeling, site-related tissue measurements, or other methods developed in consultation with the Regional Ecological Risk Assessors and/or BTAG.

The Role of Tiers and Sub-Tiers in ERA

The Superfund program guidance describes a tiered approach for conducting ERAs and further describes the potential need for additional sub-tiers or iterations of specific activities at large or complex sites. In addition to refining contaminants of concern, effective use of sub-tiering will help focus the ERA process and improve the quality of risk characterizations.

The Two-Tier Process

A two-tier process for implementing an ERA is outlined in Highlight 3-1 in the Superfund program guidance. The first tier of this process (Steps 1 and 2) is the screening-level ERA; the second tier (Steps 3 through 8) represents a baseline ERA. The two-tier process is a means by which to quickly and efficiently evaluate sites with minimal potential for ecological risk and eliminate them from further evaluation in the baseline ERA. The screening-level ERA also allows contaminants that do not pose a substantial ecological risk to be removed from the list of COPCs prior to conducting the baseline ERA.

Although a decision can be made to proceed with cleanup after any tier of the ERA process, for some sites of relatively small size or where the contamination has a sharply defined boundary, it may be preferable to cleanup the site to the screening values rather than to spend time and resources determining a less conservative cleanup number. For example, a pond receiving a discharge may contain contaminated sediments and removing these sediments (resulting in remediation to conservatively derived levels) may be less costly than the studies necessary to determine the site-specific risk based cleanup levels. Conversely, for many sites, it is preferable to move directly to a baseline ERA after the initial screening, and the guidance routinely provides for this second tier.

Sub-Tiering

A sub-tier may consist of any incremental iteration of the exposure, effects, or risk characterizations being conducted within the ERA and may occur at any point in Steps 3 through 7. It may be focused on a parameter, assumption, or assessment endpoint and may be necessitated through discovery of new information or new results from completed studies. Sub-tiering has the goal of focusing the evaluation of COPCs, so resources can be more effectively applied to the ERA process. The use of sub-tiers is primarily a function of the need to further reduce uncertainties in the baseline ERA, but incremental costs may also limit the amount or extent of additional activities.

To efficiently utilize sub-tiers, it is important to establish agreement early on the planning, execution, and documentation of the work to be performed. This is due, in part, to the time and effort needed to produce documents for the next sub-tier (e.g., conclusions of SLERA and follow-on work plan). In practice, the ecological risk assessor should provide support for effective sub-tiering by anticipating the potential sub-tiering options and facilitating agreement with the risk manager regarding criteria for acceptance of the resulting product. Anticipating results of successive risk calculations and facilitating agreements may take place at any appropriate time within the baseline ERA based on the existing information.

Example: Relationship Between Sub-Tiering and Reduction of COPCs

A screening-level ERA is to be conducted for a site with numerous COPCs. The stakeholders agree that the first evaluation will be to compare the maximum media contaminant levels to the most conservative ecotoxicity screening values, although they expect that this will result in removal of only a few COPCs from the list.

Moving from the screening phase into Problem Formulation, experience predicts that there will be COPCs with no toxicity benchmarks and other COPCs that are analyzed for, but not detected at risk-based detection limits. Therefore, the work plan for the baseline ERA states that contaminants included in the analysis of samples, but not detected, will be removed from the list of COPCs. Next, the plan states that a dietary exposure model will be used for specified and retained COPCs using conservative default assumptions, such as 100% absorption efficiency of all ingested material. The work plan further states that, for specific contaminants, an alternate lower absorption efficiency factor may be applied, if these contaminants are retained and if the lower factor is "pre-approved". This process could then continue as deemed appropriate and effective.

In this way, iterative evaluations (i.e., sub-tiering) can be done in an objective and technically sound manner, confidence may be increased in risk estimates, and bias (or perceived bias) in the risk characterization may be avoided by using input from both the risk assessor and the risk manager.

Documentation of Sub-Tiering

In terms of effectiveness of resource utilization, sub-tiering has its greatest potential benefit at the point in the ERA process before data intensive evaluations are designed. The experience and ability of the risk assessor to anticipate relevant risk questions and associated risk calculations and the ability of the site manager to organize the site documentation contribute to the most effective use of sub-tiering. What is often lacking and thereby a source of controversy, however, is the approach used to document and support the various decisions influencing work plans for each particular tier or sub-tier of the ERA. The rationale for each iteration, the questions to be answered, and intended use of the resulting information should be clearly defined and agreed upon with the Regional Ecological Risk Assessors and/or BTAG.

Analogy: Reduction of COPCs and Sieving Soil Particles

Reducing the list of COPCs within an ERA has a direct analogy to the physical separation of particles in soil particle size distribution analysis. The physical screens allow a known size particle to pass through the sieve (up to the diameter of the screen mesh size). What is not known is the absolute magnitude and size distribution of the material retained by the screen. This is precisely the rationale contained in the Superfund program guidance for the use of screening in the ecological risk assessment process. Upon the completion of a conservative screen, if no materials (contaminants) are retained by the screen, one can confidently state that there is a minimal potential for ecological risk to exist. Alternatively, if materials (contaminants) are retained by the screen, one cannot conclude that an ecological risk "actually" exists; the characteristics of the material retained by the screen are unknown, other than its size is above some specified minimum value. This is the basis for the statement in the Superfund program guidance that screening level values do not constitute technically defensible cleanup goals; those must be derived through the baseline ERA process.

Continuing to draw upon the physical analogy, the next challenge is to devise a means of sorting out desired material from extraneous material. Within the baseline ERA, we wish to focus on the contaminants that may actually pose an ecological risk (commonly referred to as the risk drivers) rather than on those COPC's that either do not actually pose an ecological risk, pose only a minimal ecological risk, or pose an ecological risk that is not related to the site and /or cannot be effectively reduced.

To sort through the "material," larger mesh sieves are used iteratively. This is done until: 1) all of the material has passed through the screen and it is concluded that the mesh size was not too large to allow wanted material to pass through, 2) it can be seen that additional iterations will not be functionally effective and a "different" approach is needed, or 3) the actual material desired is obtained. Correlating these outcomes with the SMDPs at the end of Step 2 of the Superfund program guidance document, the outcomes may be restated as follows: 1) "There is adequate information to conclude that ecological risks are negligible and therefore no need for remediation on the basis of ecological risk," 2) "The information is not adequate to make a decision at this point, and the ecological risk assessment process will continue to Step 3," or 3) "The information indicates a potential for adverse ecological effects, and a more thorough assessment is warranted."

What corresponds to these incrementally increasing mesh sizes within the ERA process? First, it must be recognized that the same things are always occurring in the thought process. Just as the same thought process occurs in Steps 1 and 2 as occurs in Steps 3 to 7, each iteration of the ERA, whether called a tier, a sub-tier, or any other name, includes similar considerations. In each successive tier, however, more information is used and assumptions and calculations are modified appropriately. The key transition in the process is from screening, which is conducted by comparison with benchmarks, to the baseline ERA, where comparisons generally require the use of negotiated values agreed upon with Regional Ecological Risk Assessors and/or BTAGs.

Summary

This supplemental guidance clarifies the two-tier process for conducting ERAs at Superfund sites and RCRA Corrective Action facilities discussed in U.S. EPA 1997. It describes the purpose of each tier (i.e., screening-level and baseline ERAs) and highlights those components common to both. It further provides an approach for refining contaminants of concern and streamlining the ERA process. Readers are referred to the references listed below for further information.

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AN EVALUATION OF CAUSE-EFFECT RELATIONSHIPS BETWEEN DDT (AND METABOLITES) AND SEDIMENT TOXICITY TO BENTHIC INVERTEBRATES

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ABSTRACT

Widely applied, empirical sediment quality benchmarks for DDT and its metabolites (DDx) are intended to predict sediment toxicity but were derived in a manner that does not necessarily reflect cause-effect, concentration-response relationships. An alternative approach to evaluating risks of sediment-associated DDx to benthic invertebrates is thus needed. We review and synthesize information on DDx toxicity to invertebrates based on several lines of evidence, including (1) extensive spiked sediment toxicity studies, (2) toxicity testing and benthic invertebrate community assessments from major DDT-contaminated sites, and (3) extrapolation of aquatic toxicity data to sediment using the equilibrium partitioning approach. These lines of evidence show that the existing sediment quality benchmarks overestimate the toxicity of DDx to invertebrates, apparently reflecting the distribution of sediment DDx concentrations in the underlying databases rather than causal relationships between DDx exposures and biological outcomes. Alternative screening values identified from our review should provide a stronger basis for prioritizing and interpreting site-specific investigations of DDT-contaminated sediments.

INTRODUCTION

- Toxicity to benthic invertebrates is one of several concerns for sediments containing DDT and its major metabolites, DDD and DDE (collectively DDx).
- Existing sediment quality guidelines (SQGs) are problematic:

	SQGs (µg/kg dry weight)	SQGs (µg/goc assuming 1% TOC)
4,4'-DDT	1 - 710	0.1 - 71
4,4'-DDD	1.2 - 60	0.12 - 6
4,4'-DDE	1.4 - 370	0.14 - 37
Total DDx	1.6 - 570	0.16 - 57

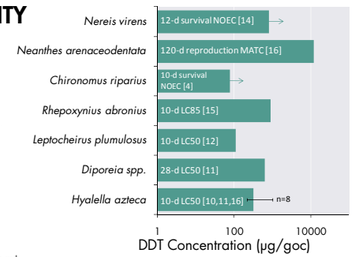
Source: http://rais.ornl.gov/tools/eco_search.php

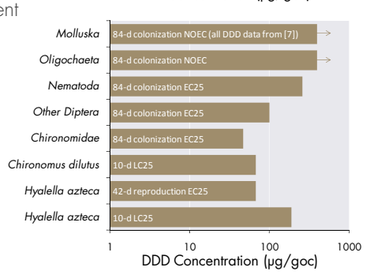
  - Based on associations between chemistry & toxicity for sediments containing many chemicals
  - Association ≠ Causation

- We examine causal evidence:
  - Spiked sediment toxicity studies
  - Biological & chemistry data for major DDx-contaminated sediment sites
  - Extrapolation of aquatic toxicity data to sediment using the equilibrium partitioning approach

SPIKED SEDIMENT TOXICITY TESTS

- Controlled experiments capable of characterizing cause-effect, concentration-response relationships
- Must understand bioavailability to apply to different sediments
  - DDx studies had sufficient equilibration time
  - Results normalized to organic carbon
- Most sensitive: Chironomid colonization
  - EC25 = 47 µg DDD/goc



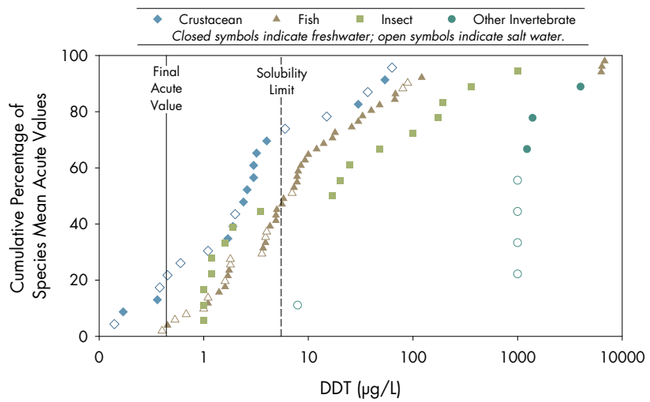


MAJOR DDx SITES

Benthic Invertebrate Total DDx Toxicity Thresholds from Major DDT Manufacturing Sites				
Site	Biological Endpoint	Effect	Total DDx Concentration (µg/goc)	Ref.
United Heckathorn (Richmond CA), Palos Verde (Los Angeles CA), Triana/Tennessee River (Huntsville AL)	Review & synthesis of amphipod toxicity tests and benthic community surveys	Amphipod LC50s Amphipod toxicity threshold Amphipod abundance threshold	1,040 - 2,600 300 100	[18]
United Heckathorn	Benthic invertebrate infaunal index (relative abundance of tolerant/sensitive species)	No effect Slight to intermediate effect Clear effect	1 - 2 **32 - 190** 2,700	[3]
Triana/Tennessee River	Midge (*Chironomus dilutus*) 10-d survival	Clear dose-response threshold	3000	[20]
Triana/Tennessee River	Amphipod (*Hyalella azteca*) 10-d survival	<20% mortality 20-30% mortality >30% mortality	1.2 - 9.8 17 - 3,110 340 - 7,920	[6]
Triana/Tennessee River	Benthic community composition: abundance of sensitive species	High variability; authors note consistency with Swartz et al. [18] threshold	100	[7]
Lake Maggiore, Italy	Benthic community composition; midge (*Chironomus riparius*) 28-d development/emergence; amphipod (*H. azteca*) 28-d survival/growth	No effects associated with DDx	>55	[5]

EQUILIBRIUM PARTITIONING

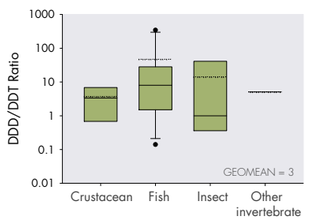
1. Calculate Final Acute Value (FAV) for DDT
   - FAV = 0.44 ug/L ≈ 5th percentile of 96-h LC50s

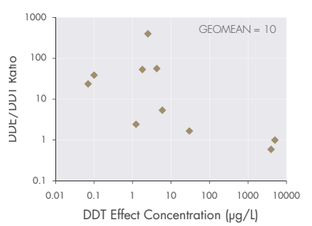


2. Calculate Acute Chronic Ratio (ACR) & Final Chronic Value (FCV)
   - Geometric mean ACR = 29; DDT FCV = 0.015 µg/L

Species	Chemical	Chronic Effect	ACR	Reference
Fathead minnow (*Pimephales promelas*)	DDT	Early life stage survival & growth	65	[9]
Copepod (*Nitocra spinipes*)	DDE	Reproduction	46	[1]
Amphipod (*Hyalella azteca*)	DDT, DDD, DDE	Reproduction	≈8	[10,8]

3. Identify relative toxicity of metabolites based on controlled comparisons





4. Calculate Equilibrium Partitioning Sediment Benchmarks (ESBs)
   - ESB = FCV x Koc x 0.001
   - This works because porewater concentrations are predictive of toxicity.

	FCV (µg/L)	Log Koc	ESB (µg/goc)
DDT	0.015	6.5	51
DDD	0.045	6.1	57
DDE	0.15	6.8	860

5. Calculate Total DDx ESBs based on DDx composition

DDT	DDD	DDE	DDx ESB (µg/goc)
33%	33%	33%	79
10%	15%	75%	180
10%	75%	15%	65

INTEGRATING THE EVIDENCE

- All lines of evidence support a cause-effect screening value for DDx on the order of 50-100 µg/goc.
- Data from major DDx sites suggest severe effects at about 2,000 – 3,000 µg/goc.
- Site-specific factors affect DDx toxicity:
  - Enhanced sorption to black carbon decreases bioavailability (Tomaszewski et al. 2007).
  - DDx is less bioavailable if present in crystalline form (Boese et al. 1997).
  - Total DDx is less toxic if DDE predominates.
  - Benthic community is less sensitive if habitat conditions exclude DDx-sensitive species.

CONCLUSIONS

- An appropriate screening value for potential DDx effects on benthic invertebrates is on the order of 50 to 100 µg/goc.
- Generic SQGs are typically orders of magnitude lower, due to limitations in SQG derivation methods.
- Site-specific effects thresholds may differ, due to differences in sediment characteristics, DDx composition and form, and benthic community characteristics.

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Draft Report for Review Purposes Only

Indicator Development and Framework for Assessing Indirect Effects of Sediment Contaminants

Draft report to the California State Water Resources Control Board

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Table of Contents

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This report is produced as part of a subcontract from SCCWRP to SFEI, and is part of the products provided to the State Water Resources Control Board. The aim of the Sediment Quality Objectives science team (including SFEI and SCCWRP scientists) is to provide the State Water Board with scientific background information that may aid in development of Sediment Quality Objectives for bays and estuaries in California. This report represents the technical viewpoints of the authors, and not necessarily the viewpoint of the State Water Board or those acknowledged for technical assistance.

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[1] Scientific Steering Committee members included Todd Bridges, Ed Long, Peter Landrum, Rob Burgess, Bob Van Doleh, and Tom Gries

Abstract

Contaminated sediments can indirectly affect demersal fish, as well as piscivorous birds, marine mammals and humans. This report describes a general approach to evaluate sediments in bays and estuaries for these indirect (i.e., food web mediated) effects. The work described herein is part of a larger effort to assist the State Water Resources Control Board address technical issues that arise in developing Sediment Quality Objectives.

The approach is based upon a multiple line of evidence framework to determine the probability that sediments in a water body pose substantial risks to human or wildlife health. The three lines of evidence (LOE) utilized in this framework are tissue chemistry of resident prey animals (fish or invertebrates), sediment chemistry, and bioavailability tests. Key attributes of the framework include sequential application of the LOE, a probability-based approach with multiple categories, and evaluation of separate LOE at different spatial scales.

This report also includes technical guidance to support application of the framework. The guidance includes appropriate types of measurements for each LOE (e.g., fish species, ancillary parameters), tissue thresholds for interpreting the results, and a methodology for combining each LOE into a final assessment classification. To demonstrate the approach, the framework is applied to chlorinated organic contaminants in two case studies: Newport Bay and San Francisco Bay. The case studies apply the framework to evaluate PCBs and organochlorine pesticides.

Development of a defensible relationship between sediment and biota concentrations is an important aspect of the framework. Empirical data and mechanistic modeling should be combined to that end. The case studies evaluate use of empirical data and a steady-state food web model to calculate bioaccumulation factors for legacy pesticides in San Francisco Bay and Newport Bay. In all cases empirical field data should be compared to model results. When inconsistencies are observed, this helps to identify potential data gaps or model limitations. Resulting bioaccumulation factors are then combined with effects thresholds to identify sediment concentrations that would be protective of wildlife and human receptors.

Abbreviations and definitions

bioaccumulation – A process in which the chemical concentration in an organism achieves a level that exceeds that in its surrounding environment as a result of chemical uptake through all routes of chemical exposure (e.g. dietary and dermal absorption and transport across the respiratory surface)
bioaccumulation factor (BAF) – The ratio of wet weight contaminant concentration in biota to dry weight contaminant concentration in some other matrix. In this report, unless specified otherwise, the term "bioaccumulation factor" refers to wet weight concentration in fish or invertebrate tissue divided by dry weight concentration in sediment
bioaccumulation rate parameter – An estimate of the ratio of contaminant concentration in biota to contaminant concentration in sediment. In this report, "bioaccumulation rate parameter" is intended as a general term for some estimate of this ratio. The bioaccumulation rate parameter could be the bioaccumulation factor (BAF), the biota-sediment accumulation factor (BSAF), or some similar estimate of the ratio, depending on the data available and statistical findings in a given water body.
bioavailability – The ability of a pollutant to be transferred from an abiotic matrix, such as sediments, to the tissue of a living organism via ingestion, absorption, or other mechanisms
biota-sediment accumulation factor (BSAF) – This is the bioaccumulation factor for tissue vs. sediment, normalized for lipid and organic carbon. BSAF = (tissue contaminant concentration in wet wt. * sediment % organic carbon) / (sediment contaminant concentration in dry wt. * tissue % lipid)
BTAG – the Biological Technical Assistance Group, a multi-agency group responsible for developing toxicity reference values and other ecological risk assessment policy in California
CASQO – The California Sediment Quality Objectives Database, a database compiled for methods development for the SQO program as a whole
CDFG – California Department of Fish and Game
CSF - Cancer Slope Factor for carcinogens
DDTs (sum) –The combination of DDT (dichlorodiphenyltrichloroethane) and its degradation products, DDD (dichlorodiphenyldichlorethane) and DDE (dichlorodiphenyldichloroethylene)
DTSC – California Department of Toxic Substances Control, a Department within Cal/EPA, that protects human health and the environment by regulating hazardous waste, conducting and overseeing cleanups, and developing and promoting pollution prevention.
dw – dry weight
EPA – U. S. Environmental Protection Agency
indirect effects - adverse effects to humans and wildlife as a result of consuming prey items exposed to polluted sediments
K_{OW} – Octanol-water partitioning coefficient
LCL – Lower Confidence Limit (e.g., the lower limit of the95% confidence interval of the mean)
LOAEL – Lowest observed adverse effects level
LOE – Line of evidence
MLOE - Multiple Lines of Evidence

NOAA –National Oceanic and Atmospheric Administration
NOAEL – No observed adverse effects level
OEHHA – The Office of Environmental Health Hazard Assessment, an Office within the California Environmental Portection Agency (Cal/EPA responsible for assessing the risk posed by hazardous substances on human health and the environment and in addition, the development and posting of fish consumption advisories.
PAH – polycyclic aromatic hydrocarbons
PCBs (sum) – polychlorinated biphenyls
Receptor – an ecological entity (e.g., organism) that is exposed to a stressor (e.g., pollutants).
RfD - Reference dose for noncarcinogens
SE – Standard error of the mean
SQO – Sediment Quality Objective
SARWQCB – Santa Ana Regional Water Quality Control Board
SFBRWRCB - San Francisco Bay Regional Water Quality Control Board
SWRCB – State Water Resources Control Board of California
TMDL – Total Maximum Daily Load, a policy document to control pollutants in a water body
toxicity reference value - a level of exposure that is considered to be a threshold indicative of probability of adverse effects to a target organism. TRVs are a key piece of information in development of site-specific effects thresholds.
TRV – see Toxicity reference value
TRV – High - A toxicity reference value above which adverse effects to consumers are expected
TRV – Low - A toxicity reference value below which adverse effects to consumers are not expected
UCL – Upper Confidence Limit (e.g., the upper limit of the 95% confidence interval of the mean)
U. S. EPA ECO-SSL – A set of toxicity reference values developed by EPA, in collaboration with other agencies and stakeholders, for use in screening contaminated soils in risk assessments
ww – wet weight

1. Introduction

A primary technical challenge for natural resource managers of aquatic systems is assessment of sediment quality (Wenning *et al.* 2005). Sediment quality assessments typically evaluate direct effects of sediment pollution to benthic aquatic life. Several methods have been developed to evaluate these direct effects (e.g., Chapman 1996, Di Toro and McGrath 2000, Anderson *et al.* 2001, Vidal and Bay 2005).

Another issue in sediment assessment is evaluation of indirect effects to organisms that do not permanently reside within the sediments (Bridges *et al.* 2005, Chapman and Anderson 2005). DDTs, polychlorinated biphenyls (PCBs), methyl mercury, and many other sediment-associated compounds bioaccumulate[2], resulting in elevated concentrations in the tissues of exposed organisms (Boese *et al.* 1997, Mason and Lawrence 1999, van der Oost *et al.* 2003). These compounds may also biomagnify[2], exhibiting elevated concentrations in higher trophic levels of the food web (Rasmussen *et al.* 1990, Suedel *et al.* 1994). Bioaccumulation and biomagnification are of concern because deleterious effects to wildlife may occur at relatively low tissue concentrations (see for example, Beyer *et al.* 1996). Contaminants in fish can also reach concentrations sufficient to pose potential health risks to human sport and subsistence anglers (U. S. EPA 2000b, Burger *et al.* 2001, Davis *et al.* 2002, Greenfield *et al.* 2005, Schaeffer *et al.* 2006). These toxicological effects are referred to as indirect effects because they are not dependant on direct sediment contact by the wildlife or human receptors[3].

The inclusion of indirect effects in sediment evaluation is an important and challenging issue. The bioavailability[2] of contaminants (i.e., their potential for uptake by sediment dwelling organisms) varies widely among sediments and water bodies (Boese *et al.* 1995, Boese *et al.* 1997, Mason and Lawrence 1999, Kraaij *et al.* 2002, Battelle *et al.* 2005). Standard bulk chemical analysis of sediment does not differentiate between the contaminant fraction that is tightly bound to sediment and that which may be accumulated into tissues of an organism (Kraaij *et al.* 2002, Talley *et al.* 2002, Ghosh *et al.* 2003). Therefore, inclusion of other measures of exposure, along with sediment bioaccumulation tests, may improve the interpretability of chemical measurements (Chapman 1996, Chapman and Anderson 2005).

Multiple lines of evidence (MLOE) are frequently used to assess the direct effects of sediment pollutants. Virtually all of the estuarine ambient monitoring programs in the U. S. rely on some form of sediment quality triad (Chapman *et al.* 1997), evaluating measures of sediment toxicity, chemistry, and benthic community alteration[4]. In addition

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[2] Defined in "Abbreviations and Definitions"

[3] In this report, *"receptor"* is defined as the ecological entity that is exposed to the stressor of concern, following U. S. EPA (1998). Specifically, the report describes a method for evaluating the effects of sediment pollutants (the stressor) on wildlife and humans (the receptors).

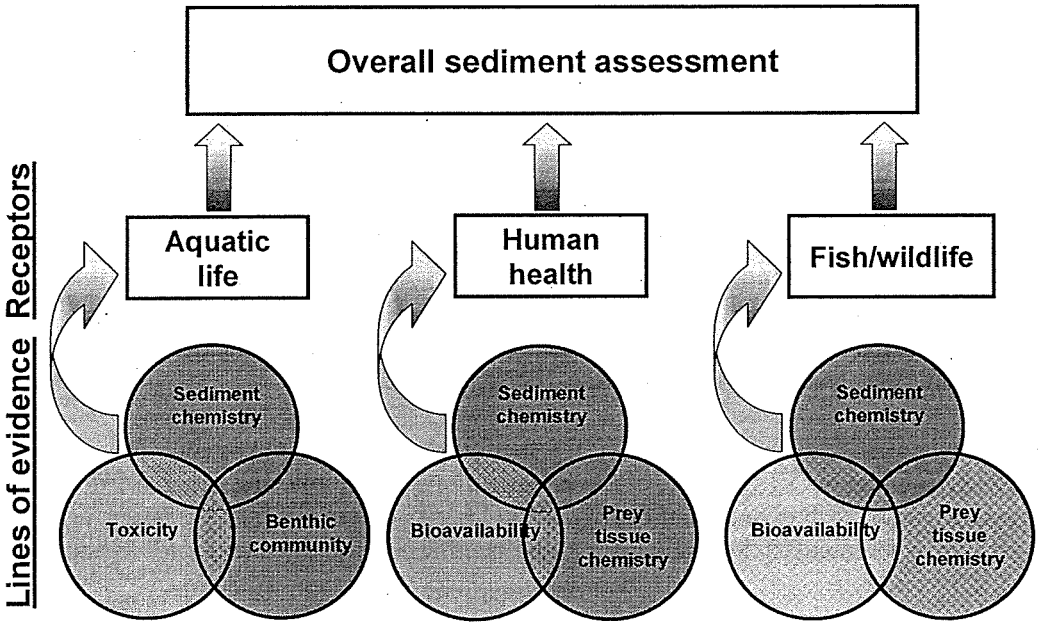
[4] These include the two largest nationwide estuarine monitoring programs, EPA's Environmental Monitoring and Assessment Program and NOAA's National Status and Trends Program, as well numerous regional monitoring programs, including those for the Great Lakes, Puget Sound, San Francisco Bay, Chesapeake Bay, the Southern California Bight, Tampa Bay, and New York/New Jersey Harbor.

to the evaluation of direct effects, the use of MLOE may also facilitate assessment of indirect effects (Figure 1.1).

To evaluate indirect effects, two technical issues must be addressed. First, a robust relationship must be developed between contaminant concentrations in sediments and receptor organisms. To this end, many studies have documented predictable relationships between contaminant concentrations in sediments and associated fish (Endicott and Cook 1994, Wong *et al.* 2001, Zeng and Tran 2002, Burkhard *et al.* 2004), and mechanistic models have been developed to describe these associations (e.g., Thomann *et al.* 1992, Gobas 1993).

A second issue in evaluating indirect effects is that contaminant effects thresholds must be developed based on potential risks to receptor organisms. Effects to human and wildlife receptors may be estimated using standard risk-assessment methods (Sample *et al.* 1996, U. S. EPA 1997, 1998, 2000c, 2006b). Thresholds are often calculated using exhaustive reviews of all available toxicity studies (California DTSC Human and Ecological Risk Division 2000, Zeeman 2004, U. S. EPA 2005, 2006b).

Figure 1.1. Components of multiple line of evidence (MLOE) framework for overall sediment assessments. Separate MLOE assessments could be conducted for direct effects to aquatic life, indirect effects to human health, and indirect effects to wildlife.



1.1. Regulatory background

Current government-established sediment quality guidelines generally don't address contaminant bioaccumulation or consequent effects to higher trophic levels (MacDonald

1994, Canadian Council of Ministers of the Environment 1999). Washington is the only U. S. state with legally enforceable sediment quality standards. In Washington, statewide sediment quality standards are based on direct measures of chemistry, benthic invertebrate toxicity tests, and benthic community composition (SAIC and Avocet Consulting 2002, Michelsen 2003, State of Washington 2005). Although narrative standards to protect fish, wildlife, and human health have resulted in some site-specific numeric thresholds for sediment and tissue (Tom Gries, Washington Department of Ecology, *Pers. comm.*), a standardized framework for evaluating indirect effects is lacking. Informal sediment quality guidelines have been adopted in a number of U. S. states, including New York, New Jersey, Florida, Texas, South Carolina, and Minnesota. However, none of these states prescribe measurement of tissue uptake or bioaccumulation (Ed Long, ERL Environmental, *Pers. comm.*). For example, in the guidelines adopted by the state of New York, potential hazards posed to wildlife and human health are estimated as a function of equilibrium partitioning into pore water, rather than direct measurement or estimation of food web trophic transfer (New York State Department of Environmental Conservation 1999).

Despite the absence of bioaccumulation-based regulatory criteria, human health and wildlife risk assessments are frequently undertaken to establish guidelines for specific water bodies. In the United States, a large body of ecological risk assessment methodology has been established by the Comprehensive Environmental Response, Compensation, and Liability Act (i.e., CERCLA, reviewed in Hamilton and Viscusi 1999). This program, commonly referred to as the Superfund Program, has resulted in a series of guidelines for performing ecological risk assessment (e.g., U. S. EPA 1997, 1998). Risk assessments performed in accordance with CERCLA require a series of steps and studies that are time consuming and costly to perform. Evaluation of individual Superfund sites often requires expenditure of tens of millions of dollars, in a process that can take a decade or more. In other management circumstances, more focused approaches to evaluating risk have been established. The conceptual model may be predetermined, including an a priori understanding of the contaminants of concern, exposure mechanisms, targets, and toxicological effects. It then becomes possible to use a straightforward procedure. For example, the U. S. government has established a streamlined procedure for evaluating exposure risks due to disposal of dredged materials (U. S. EPA and U. S. Army Corps of Engineers 1991, 1998).

For management circumstances when the focus is on a specific set of impacts and receptors, it may not be necessary or appropriate to perform a risk assessment at the level of breadth and expense of the CERCLA program. For example, if one wishes to evaluate the indirect effects of contaminated sediments to humans and wildlife, the approach should focus on key issues and transport pathways, including contaminant bioavailability, food web transfer, and effects thresholds.

1.2. Report objectives and organization

This report presents a standardized framework for evaluating indirect effects of contaminated sediments to humans and wildlife. The framework was developed to

differentiate unimpacted sediments from impacted sediments in bays and estuaries. It should be applicable to a variety of pollutants that may pose a risk due to indirect effects of contaminated sediments. The report also includes technical guidance and two detailed case study examples (Table 1.1).

The intent of the framework is to provide a consistent and cost-effective approach for sediment assessment. It focuses resources on resolving specific uncertainties and policy decisions that are known to be important for evaluating indirect effects of sediments. While most of the questions and needs identified in this approach can be resolved by applying sound scientific principals and appropriate measures, there are questions that can only be decided by the regulatory agencies within a formal and public administrative process. These include, among other topics, the target species and populations, and the degree of risk that is deemed acceptable for these targets. The goal of this report is to describe some potential choices and provide relevant technical background information. The use of a consistent approach should enable natural resource managers and stakeholders to focus on key policy decisions (e.g., how much risk is acceptable), rather than struggling with the assessment approach itself.

The report is organized as outlined in Table 1.1. Section 2 describes the conceptual model, which is the basis for the approach. Section 2 also includes an overview of how the entire process works, including how the individual lines of evidence (LOE) are combined in a sequential approach. Section 3 outlines key technical issues that must be addressed in applying the framework. Technical guidance on these issues is provided in Section 3 and a series of appendices. Sections 4 and 5 demonstrate the approach in two case studies. Section 6 presents conclusions.

Table 1.1. Summary of applicability of work presented to pollutants of concern.

Report Sections	Topic	Legacy Pesticides [a]	PCBs	Mercury	Other Pollutants [b]
2	Framework and application	X	X	X	X
3	Technical issues	X	X		
4	Case study - Newport Bay	X	X		
5	Case study - San Francisco Bay	X			

a. Including DDTs, chlordanes, dieldrin, toxaphene, aldrin, and heptachlor.
b. Including but not limited to selenium, arsenic, PAHs, and dioxins.

The case studies apply the assessment framework described in Sections 2 and 3 to San Francisco Bay and Newport Bay using data from prior studies. The purpose of these case studies is twofold: to illustrate the application of the assessment framework using realistic data, and also to explore many of the technical issues identified in Section 3 in order to develop recommendations for implementation. For both case studies, the evaluation includes corroboration of field-observed bioaccumulation factors with mechanistic food web model results. This entails comparing mechanistic modeling results on contaminant partitioning to actual observed partitioning between sediments and biota and the ecosystem. Consistent findings between mechanistic models and empirical data would support the assumptions and methods of the framework. If the findings are not

consistent, a combination of additional data collection and model revision may be required.

The case studies in this report will focus on nonpolar organic pollutants, including polychlorinated biphenyls (PCBs), and legacy pesticides (DDTs, chlordanes, and dieldrin). The case studies focus on these compounds for several reasons. First, they are known to bioaccumulate in tissues of invertebrates, fishes, and their predators, and have abundant field data available for evaluation (e.g., Suedel *et al.* 1994, Boese *et al.* 1997, Kidd *et al.* 1998, Schiff 2000, Connolly and Glaser 2002, Greenfield *et al.* 2005). Additionally, efforts are currently underway to manage these compounds in bays and estuaries (e.g., SFBRWQCB 2004, SARWQCB 2006). Furthermore, bioaccumulation of PCBs and legacy pesticides is more predictable than some other compounds. Specifically, bioaccumulation of non-polar organics may be predictably related to octanol-water partition coefficient (K_{ow}), biota whole body lipid content, and abundance and types of sediment organic carbon (Clark *et al.* 1988, Thomann 1989, U. S. EPA 2000a, Cornelissen *et al.* 2005). Bioaccumulation is more difficult to predict for trace metals, requiring ancillary data frequently not collected in monitoring programs (Hansen *et al.* 1996, Lee and Jones-Lee 2003). Lastly, general mechanisms of bioavailability and bioaccumulation are likely to be similar between legacy organochlorines and other compounds, including polybrominated diphenyl ethers and dioxins.

2. Conceptual approach and framework

2.1. Problem statement

The goal of the framework is to evaluate two potential risks posed by polluted sediments. The first is the risk of effects to human health via consuming fish and shellfish tissue. The second is the risk of effects to wildlife via consuming fish and shellfish tissue. Narrative benefits to be protected include preservation and enhancement of wildlife, wildlife habitats, and use of waters by humans for recreational or commercial fishing. In short, the framework focuses on protecting two groups of receptors: humans and wildlife.

A key component of the indirect effects framework development process is regular review and feedback by committees of scientific experts, regulatory agencies, and affected stakeholders. Appendix A summarizes feedback received by committees that had input on development of this framework. This report aims to address those issues.

2.2. Conceptual model

Figure 2.1 presents a general conceptual model for evaluating indirect effects. This conceptual model is not designed to depict all processes that influence contaminant fate in bays and estuaries; rather, it represents the most important processes for understanding indirect effects of sediments to wildlife and humans.

The conceptual model is intended to depict the aquatic ecosystem processes that are most important for indirect effects of contaminated sediments. This includes abiotic and biotic components and their linkages. Some degree of contaminant transfer between the sediments and the overlying water column is assumed. The relative concentrations in sediments vs. the water column are determined by a variety of processes. For organic pollutants, these processes include equilibrium partitioning (Di Toro *et al.* 1991, Mackay and Paterson 1991), water and sediment movement and mixing dynamics (Burkhard *et al.* 2003), and nonlinear sorption to carbonaceous particles (Ghosh *et al.* 2003, Cornelissen *et al.* 2005). In practical terms, this combination of processes often makes it difficult to distinguish between sediment vs. water column sources of contamination.

Sediment-dwelling invertebrates are exposed to sediment pollutants primarily via dietary uptake and respiratory exposure to sediment porewater. Invertebrates are also exposed to contaminants in the overlying water column as a result of filter feeding, particle feeding, and respiratory exposure. Food-web trophic transfer (as represented by dietary uptake of invertebrates) is the most significant route of exposure for fish (Figure 2.1). Additionally, fish can encounter pollutants via consumption of sediments (Laffaille *et al.* 2002, Almeida 2003) and respiration of water.

Wildlife (e.g., birds and aquatic mammals) and humans consume contaminated finfish (thick arrows) and invertebrates (thin arrows), resulting in contaminant exposure (Figure 2.1). That is, the conceptual model (and resulting framework) includes organisms that are not in direct contact with the sediments. These organisms are exposed to pollutants

indirectly by food web trophic transfer, and these indirect exposures may lead to indirect effects.

Figure 2.1. Conceptual model for assessment of indirect effects of pollutants in bays and estuaries. Boxes indicate key matrices, including sediments, water, and animal biota. Biota are further categorized as invertebrates, fishes, wildlife and humans. Arrows represent contaminant transfer pathways, with the size and direction of the arrow representing the significance and direction of the transfer mechanism. The vertical dashed line distinguishes between matrices evaluated for potential exposure using empirical data and contaminant fate models (left) , and matrices evaluated for potential effects using toxicological effect models (right).

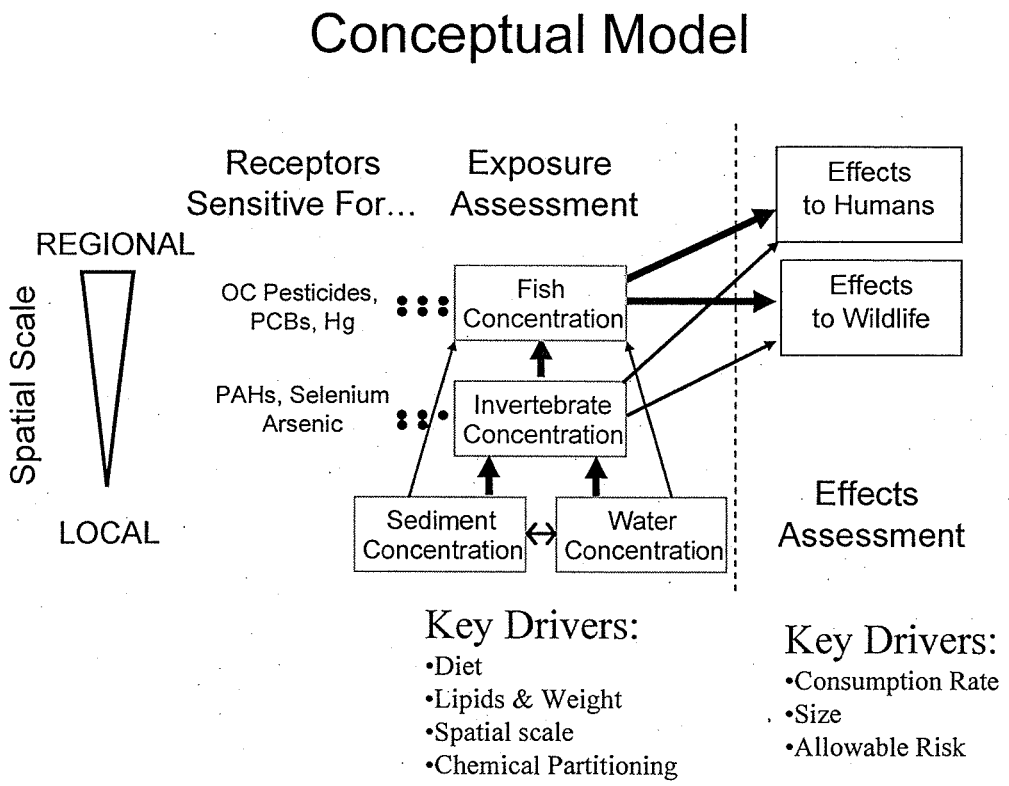


Figure 2.1 also indicates linkages between selected bioaccumulative compounds and the receptors that would be sensitive indicators of exposure to these pollutants. In particular, mercury (Hg), PCBs, and legacy pesticides (e.g., DDTs, dieldrin, and chlordanes) can biomagnify up the food web, resulting in elevated concentrations in fish tissue (Suedel *et al.* 1994, van der Oost *et al.* 2003). In contrast, PAHs are rapidly metabolized by fish (Eisler 1987, van der Oost *et al.* 2003), and arsenic and selenium are not consistently biomagnified from invertebrates to fishes (Suedel *et al.* 1994, Abernathy 2004); elevated concentrations of these compounds are therefore likely to be more evident in sediment-exposed invertebrate tissues than fish tissues.

Because the conceptual model focuses on the most important processes that govern indirect effects of sediment pollution to human and wildlife predators, it is simpler than some sediment risk assessment models (e.g., Bridges *et al.* 2005). The intent is to enable evaluation of individual bays and estuaries when resources are limited.

The relative simplicity of the model and approach is intended to reflect the limited scope and resources available to conduct individual evaluations. The intent is to avoid creating a program that is too burdensome to implement. Nevertheless, in-depth evaluation of scientific uncertainties outside the conceptual model may be useful in specific circumstances. These factors may include: 1. contaminant transfer between deep sediment, surface sediment, and the water column; 2. toxicological effects to aquatic plants or invertebrates; 3. predator tissue residue-based effects evaluation for humans or wildlife[5]; or 4. changes over time in contaminant concentrations and transfer pathways. All of these factors may be potentially important in site-specific evaluations, establishment of water body TMDL targets, or other regulatory objectives (Davis 2004, Bridges *et al.* 2005, Gobas and Arnot 2005, Greenfield and Davis 2005). Nevertheless, the approaches for evaluating these factors are outside the scope of this document.

The individual case study evaluations on San Francisco and Newport Bays each contain site-specific conceptual models (Sections 4 and 5). These models contain additional boxes to represent the water-body specific understanding of food-web trophic transfer pathways and local receptor organisms.

Both exposure and effects information must be used to determine the risk posed by sediment contamination to humans and wildlife. The conceptual model indicates that the exposure and effects assessment would focus on separate matrices. In the indirect effects assessment framework, exposure is evaluated using direct measurements of contaminant concentrations in the local water body. The following sections describes how exposure and effects evaluations are incorporated into the framework.

2.3. Multiple line of evidence framework

Based on the conceptual model, a multiple line of evidence framework may be developed to evaluate pollutant exposure. The framework would be applied separately to human vs. wildlife receptors (Figure 1.1). In this framework, three lines of evidence (LOE) are evaluated:

1. Prey tissue chemistry
2. Sediment chemistry
3. Bioavailability

The prey tissue and sediment chemistry LOE compare pollutant concentrations in field-collected sediments or tissues to thresholds based upon biological effects. The bioavailability LOE compares concentrations in laboratory test organisms or field benthic organisms to concentrations in sediments in order to establish bioavailability of sediment-

[5] That is, the framework focuses on risk evaluations based on pollutant concentrations in the tissues of prey organisms and sediments, rather than evaluations based on pollutant body residues in the predators, themselves.

associated pollutants. When all LOE are integrated, the result is a classification of sediments into four categories of probability of impact: unlikely impact, possible impact, likely impact, or clear impact.

2.3.1. Prey tissue chemistry

The first LOE (prey tissue) evaluates the tissue pollutant concentrations in field-collected fish or invertebrates. Prey tissue concentrations are measured to determine whether humans or wildlife are at risk due to consuming contaminated prey organisms. As indicated in the conceptual model, measurements could focus on fish or invertebrates, depending on the receptor and contaminant. If contaminant concentrations in prey are high enough, consumption of the prey would pose a risk of mortality or sublethal effects. Conversely, if contaminant concentrations in their prey do not pose a risk, humans and wildlife would be protected. The use of direct prey tissue measurements is central to dietary exposure risk evaluations (Lee *et al.* 1994, U. S. EPA 2000b, CH2M HILL 2004, U. S. EPA 2004, Battelle *et al.* 2005, Chapman and Anderson 2005).

For the prey tissue line of evidence, use of field-captured fish is sometimes questioned based on the concern that mobility of fish inhibits the ability to establish a predictive relationship between sediment chemistry and fish tissue chemistry. However, our analyses, and published literature, indicate that significant relationships are often observed (Appendix B).

2.3.2. Sediment chemistry

The second LOE evaluates whether sediment pollutant concentrations are high enough to pose risks to predatory wildlife or humans. If sediment concentrations are not high enough to cause the observed concentrations in prey, other contaminant sources are implicated. These may include external loads or water column concentrations. Pollutant concentrations are measured in sediments from multiple locations in order to identify locations where potential risk is relatively high.

To develop thresholds for this LOE, empirical data are used to determine ratios between pollutant concentrations in sediments versus prey organisms. These ratios will be more defensible if developed separately for individual bays and estuaries, rather than using a generalized bioaccumulation rate parameter (e.g., bioaccumulation factor) for all regions. Appendix C provides evidence that bioaccumulation factors vary significantly among water bodies, requiring local development of the ratio between sediment and prey organism. Mechanistic models (Arnot and Gobas 2004, Gobas and Arnot 2005) may be used to corroborate the empirical ratios. These ratios are then combined with diet-based effects thresholds to develop sediment thresholds.

2.3.3. Bioavailability

The third LOE (bioavailability) evaluates the tendency for a substance to accumulate in the tissue of sediment-exposed organisms. The purpose of this LOE is to confirm that

contaminants in sediments are bioavailable. This would establish the linkage between the boxes in the exposure side of the conceptual model (Figure 2.1).

A number of methods may be used to establish whether sediment compounds are bioavailable. These include laboratory bioaccumulation tests or field measurement of exposure in resident or transplanted organisms. New measurements would only be necessary for contaminants not known to be generally bioavailable. Thus, the first step for this LOE is an evaluation of recent peer-reviewed scientific literature to determine whether the pollutants in question are bioavailable, when associated with sediments in natural waters. If it becomes established that sediment-bound contaminants are biologically available, they become a probable exposure mechanism to all organisms associated with the benthic food web.

2.3.4. Evaluation of effects to receptors

Effects assessments must be made to determine whether the human and wildlife receptors are at risk. Likelihood of effects are estimated by comparing pollutant concentrations in the sediments and prey tissues to calculated risk thresholds. These risk thresholds are developed using risk assessment methodologies. A strength of risk assessment calculations is that they rely on a widely available, standardized, and peer-reviewed methodology. For example, human health risk assessments are based on the U. S. EPA IRIS database of toxicity reference values for humans (U. S. EPA 2006b).

Although effects assessments may be obtained by direct measurement of receptor organisms (i.e., human and wildlife predators), this is not included in the framework. There are several reasons for this. The first reason is that for humans and many sensitive wildlife species, legal and ethical barriers render such measurements illegal or highly impractical on a wide scale. Secondly, requirement of standardized measurements of biological effects to wildlife would be very costly to implement. Finally, it would be extremely difficult to develop guidance and criteria for standardized application of such measurements. When local data on effects to predators are available, they may be used as additional information to guide management decisions. Technical guidance on the use of local effects data is outside the scope of this report.

2.4. Assessment categories for the framework

The framework results in classifying sediments among four categories of impact. As the evaluation is based on exceeding risk-based thresholds, each category indicates progressively greater risk of adverse effects to wildlife or human predators. The four categories are: A. Unlikely impact. B. Possible impact. C. Likely impact. D. Clear impact. They may be described as follows:

A. Unlikely impact. Sediment contamination at the site is not expected to cause adverse impacts to human or wildlife consumers of prey captured within the water body. The probability of impact is low because concentrations in prey tissues and sediments are below protective risk thresholds, or because sediment pollutants are not bioavailable.

B. Possible impact. Sediment contamination at the site may be causing adverse impacts to human or wildlife predators. This classification occurs when sediment-associated pollutants are bioavailable and concentrations in sediments and prey tissues indicate intermediate levels of risk to chronically exposed predators.

C. Likely impact. There is a high degree of confidence that sediment contamination at the site is causing adverse impacts to human or wildlife predators. Prey tissue or sediment pollutant concentrations are high enough to indicate substantial risk of effects to chronically exposed predators. Additionally, sediment-associated pollutants are bioavailable.

D. Clear impact. There is a very high degree of confidence that sediment contamination at the site is causing adverse impacts to human or wildlife predators. Pollutant concentrations in both sediments and prey tissue are high enough to indicate substantial risk of impacts to human or wildlife survival, reproduction, or development. For human carcinogens, the expected rate of increased cancer in the population is deemed unacceptably high.

2.5. Integration of the framework

This section describes how the three LOE are integrated into the overall framework for evaluating sediments (see Figure 2.2 and Table 2.1).

The assessment begins by evaluating the prey tissue LOE. For this LOE, appropriate data on pollutant concentrations in prey tissues from the entire water body are compiled[6] and used to estimate average concentrations. To determine the risk posed by prey tissue contamination, the estimate of average tissue chemistry is compared to a low effects threshold and a high effects threshold. This comparison results in one of three possible classifications: low, moderate, and high prey tissue chemistry. This classification is the first step in the multiple line of evidence exposure assessment (Figure 2.2).

The low and high effects thresholds are developed using standard risk-based approaches[7]. Each threshold is developed using on probability-based assumptions appropriate for that level of risk. The low threshold represents a tissue concentration below which adverse effects to the consumer are unlikely. The high threshold represents a concentration above which adverse effects are probable.

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[6]For the framework described in this report, the term "water body" is intended to be operationally defined, based on the needs of the agencies applying the indirect effects SQO. In the general case, the SQO would be applied for managing an entire bay or estuary, using all available data. However, if there is a regulatory need or basis to conduct evaluations at a scale smaller than the entire water body, the approach should not preclude this. For example, if the water body is divided into separate hydrological units (reaches or basins) for 303d listing purposes, and sufficient data are available on each of these units, the indirect effects SQO could be applied to evaluate each of these units separately.

[7] Further technical guidance is provided in Section 3 of this report.

For protection of humans, the prey tissue LOE would also be evaluated by determining whether the Office of Environmental Health Hazard Assessment (OEHHA - California EPA) has developed water-body specific consumption advisories for any contaminants. If OEHHA has recommended that people reduce their consumption of fish as a result of a specific contaminant or class of contaminants, then it follows that fish from that water body may pose a human health risk that needs to be further considered.

If prey tissue chemistry is in the lowest category, then the sediments in the water body are considered to be protective for wildlife or human consumers of fish, and the assessment is complete. This result is indicated by the decision tree ending at the green light in the top right panel of Figure 2.2. The receptors are considered to be unimpacted with respect to the pollutant of concern. Concentrations below the low threshold represent a stopping point in the assessment. Therefore, the low threshold must be calculated so that prey tissue concentrations below it pose an acceptable risk level.

The framework proceeds to the sediment chemistry LOE if prey tissue chemistry is in the intermediate (yellow light) or high (red light) category. Like the prey tissue chemistry LOE, the sediment chemistry LOE compares field-collected samples to a low threshold and a high threshold (Table 2.1). However, for the sediment chemistry LOE, sediments from individual collection locations are compared separately to the sediment chemistry thresholds. This results in a spatial map that characterizes sediments within the water body, among low, moderate, and high categories (Figure 2.2, middle left panel).

The sediment chemistry thresholds are calculated as the quotient of the prey tissue threshold and a bioaccumulation rate parameter:

$$\text{Sediment Threshold} = \frac{\text{Prey Tissue Threshold}}{\text{Bioaccumulation Rate Parameter}}$$

The bioaccumulation rate parameter should be either a bioaccumulation factor (BAF) [8], a biota sediment accumulation factor (BSAF) [9], or some combination of the two. The bioaccumulation rate parameter is water-body specific. It should be calculated based on measurements collected from the bay or estuary to be evaluated. These technical issues will be discussed in Section III.

In combination, the prey tissue and sediment chemistry LOE result in a provisional classification of each sediment station within the water body. Each station is classified into one of four categories described above in Section 2.4. This classification flows logically from how concentrations compare to the thresholds for each LOE (Table 2.1, Figure 2.2).

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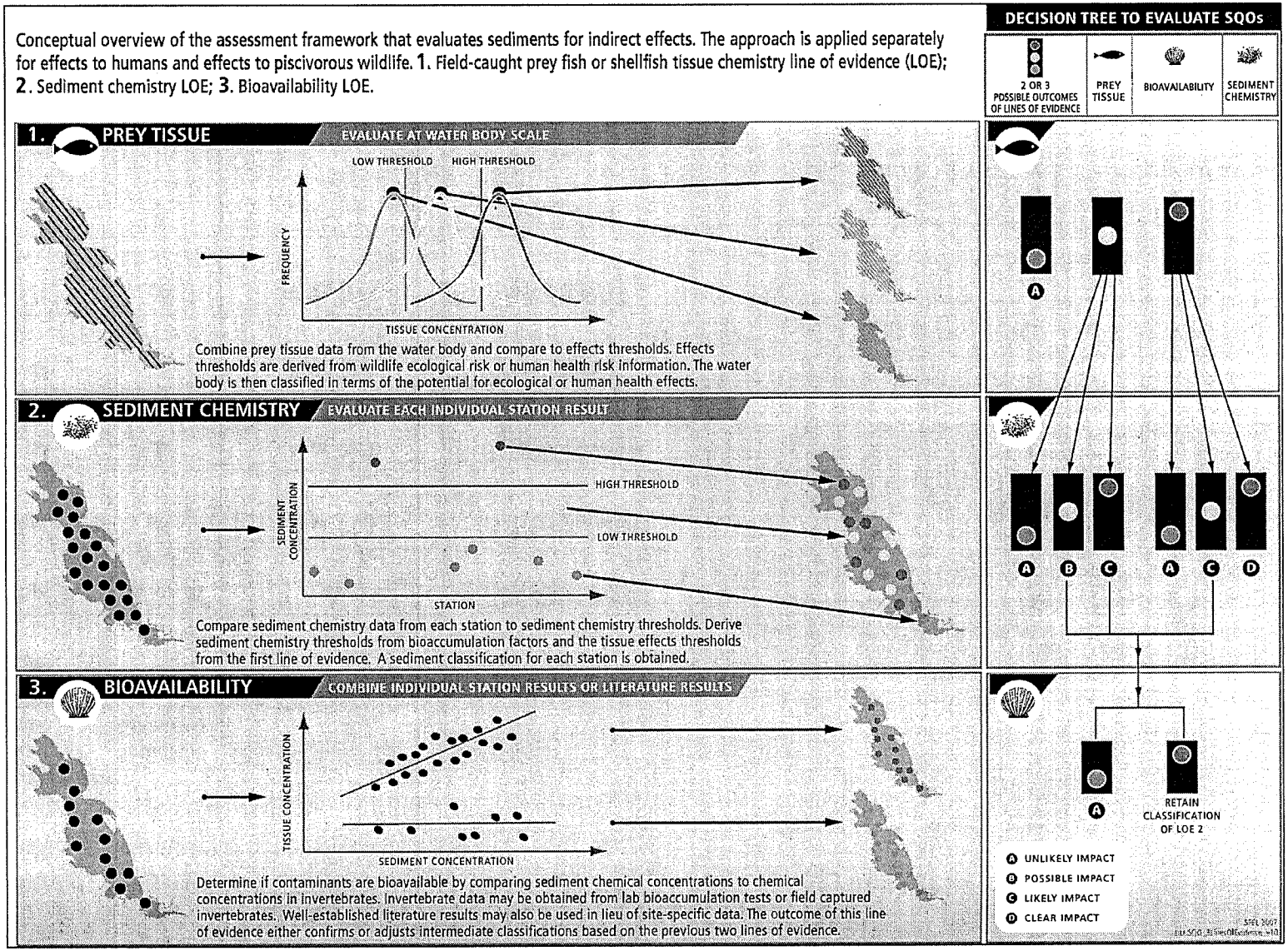
[8] BAF = tissue concentration (wet weight)/ sediment concentration (dry weight), as defined in "Definitions and Abbreviations"

[9] BSAF = tissue concentration (lipid weight)/sediment concentration (organic carbon normalized weight). Also defined in "Definitions and Abbreviations"

Table 2.1. Interpretation of MLOE for indirect effects. Symbols: ▲ = low chemistry threshold not exceeded (fish, sediment) or significant bioavailability not observed (bioavailability test). ◘ = chemistry between low and high thresholds. ● = high chemistry threshold exceeded (fish, sediment) or significant bioavailability observed (bioavailability test). - = evaluation not needed. NA = data not available.

Field Prey Tissue Chemistry	Sediment Chemistry	Bioavailability Test	Conclusion	Interpretation
▲	-	-	A. Unlikely impact	Field prey tissue chemistry indicates no evidence of risk to human or wildlife consumers above the defined level of concern. Therefore, additional assessment need not be done.
◘ or ●	▲ or ◘ or ●	▲	A. Unlikely impact	Although tissues are contaminated at a level of concern, the lack of bioavailability indicates that sediment contaminants are not the source.
◘ or ●	▲	● or NA	A. Unlikely impact	Sediment chemistry indicates that concentrations are below the level of concern. These sediments pose a low risk of impact to human or wildlife consumers (indirect effects).
◘	◘	● or NA	B. Possible impact	Sediment chemistry indicates that concentrations may be above a level of concern. The degree of impact to human or wildlife consumers (indirect effects) from these sediments is uncertain.
◘	●	● or NA	C. Likely impact	Sediment chemistry evaluated at individual sites indicates that concentrations are above a level of concern. Impact to human or wildlife consumers (indirect effects) from these sediments is likely.
●	◘	● or NA	C. Likely impact	Tissue chemistry indicates that adverse effects to wildlife or humans are likely, but sediments are not quite high enough to be certain that they are the major source.
●	●	-	D. Clear impact	Sediment chemistry evaluated at individual sites and prey tissue chemistry from the water body are above a probable risk threshold. This category represents the highest degree of certainty that sediments pose a risk of indirect effects.

Figure 2.2 (following page)


Conceptual overview of the assessment framework that evaluates sediments for indirect effects. The approach is applied separately for effects to humans and effects to piscivorous wildlife. 1. Field-caught prey fish or shellfish tissue chemistry line of evidence (LOE); 2. Sediment chemistry LOE; 3. Bioavailability LOE.
DECISION TREE TO EVALUATE SQOs
2 OR 3 POSSIBLE OUTCOMES OF LINES OF EVIDENCE
PREY TISSUE
BIOAVAILABILITY
SEDIMENT CHEMISTRY
1. PREY TISSUE
EVALUATE AT WATER BODY SCALE
LOW THRESHOLD
HIGH THRESHOLD
FREQUENCY
TISSUE CONCENTRATION
Combine prey tissue data from the water body and compare to effects thresholds. Effects thresholds are derived from wildlife ecological risk or human health risk information. The water body is then classified in terms of the potential for ecological or human health effects.
2. SEDIMENT CHEMISTRY
EVALUATE EACH INDIVIDUAL STATION RESULT
SEDIMENT CONCENTRATION
HIGH THRESHOLD
LOW THRESHOLD
STATION
Compare sediment chemistry data from each station to sediment chemistry thresholds. Derive sediment chemistry thresholds from bioaccumulation factors and the tissue effects thresholds from the first line of evidence. A sediment classification for each station is obtained.
3. BIOAVAILABILITY
COMBINE INDIVIDUAL STATION RESULTS OR LITERATURE RESULTS
TISSUE CONCENTRATION
SEDIMENT CONCENTRATION
Determine if contaminants are bioavailable by comparing sediment chemical concentrations to chemical concentrations in invertebrates. Invertebrate data may be obtained from lab bioaccumulation tests or field captured invertebrates. Well-established literature results may also be used in lieu of site-specific data. The outcome of this line of evidence either confirms or adjusts intermediate classifications based on the previous two lines of evidence.
RETAIN CLASSIFICATION OF LOE 2
A UNLIKELY IMPACT
B POSSIBLE IMPACT
C LIKELY IMPACT
D CLEAR IMPACT

The bioavailability LOE is used to confirm or modify the provisional classification obtained from the first two LOE. Specifically, the bioavailability LOE will either retain the provisional classification or convert it to "A. Unlikely impacted." The bioavailability LOE need not be evaluated if the first two LOE already result in a classification of "A. Unlikely impacted." The bioavailability LOE also does not need to be evaluated if the first two LOE result in the highest risk category: "D. Clearly impacted." That is, if concentrations in prey tissues and sediments are both above thresholds where risk is highly probable, the framework determines that sediments pose substantial risks to human or wildlife predators (Table 2.1).

For many compounds, the bioavailability LOE may be evaluated using existing literature and data[10]. If local bioavailability tests are to be performed, two methods may be used. The first method is to compare contaminant concentrations between collocated sediments and field-captured invertebrates. The second method is to perform a laboratory test in which invertebrates are exposed to the sediments to determine bioavailability. For either method, selection of appropriate invertebrate species, sample design, analytical detection limits, and sufficient sample size are all important considerations. These issues will be further discussed in Section 3.

For the bioavailability LOE, all available data are evaluated at the water body scale. This is similar to the prey tissue LOE, but different from the sediment chemistry LOE, in which data for each individual station are evaluated separately. Unlike the other LOE, the bioavailability LOE is applied using a simple hypothesis test (Figure 2.2, Table 2.1). The null hypothesis is that sediment contaminants are not biologically available to organisms in the water body. The alternate hypothesis is that these contaminants are biologically available.

If the bioavailability LOE conclusively indicates that sediment-associated contaminants are not bioavailable (i.e., no exposure), the water body is placed in the "A. Unlikely impacted" category (Table 2.1). If however, there is any indication of bioavailability, the category resulting from the other two LOE is retained. Also, if bioavailability test results are inconclusive, because the evaluation was not designed or applied properly, the category resulting from the other two LOE is retained. In short, the results of the field tissue and sediment chemistry LOE form the basis for the conclusion about the sediment, unless local data demonstrate that sediment-associated pollutants are not bioavailable (Figure 2.2, Table 2.1).

2.6. Key features and rationale of the framework

From the previous subsection, Table 2.1, and Figure 2.2, several key attributes of the indirect effects framework become apparent:

- **Separate assessment for humans vs. wildlife**
- **Risk-based approach**

[10] Support for the use of literature information in lieu of local data is provided in Section 3

- **Multiple categories**
- **Sequential approach**
- **Application at multiple scales**
- **Separate evaluation of specific contaminant classes**

2.6.1. Separate assessment for humans vs. wildlife

The use of separate evaluations for humans vs. wildlife receptors is standard practice in exposure and effects evaluations (Moore *et al.* 2005). Routes of exposure and effects thresholds can differ greatly between wildlife and humans. Humans and wildlife also have different endpoints. In risk assessments, standard human effects endpoints include disease incidence and other factors that may influence individuals in a population (U. S. EPA 2000c, Moore *et al.* 2005). In contrast, wildlife endpoints are typically limited to severe survival, developmental, or reproductive impacts likely to result in population-level effects (California DTSC Human and Ecological Risk Division 2000, Zeeman 2004, Moore *et al.* 2005). Although wildlife often have higher exposures, their effects thresholds may be higher because they are focused on stopping population effects rather than increased disease incidence.

2.6.2. Risk-based approach

A risk and probability based approach is used to develop and evaluate the fish tissue and sediment thresholds. The use of risk-based calculations in threshold development explicitly considers uncertainty in parameter estimation. Risk assessment parameters are generally estimated using probability distributions. Examples include probability of carcinogenic effects or other forms of toxicity, distribution of consumption rates, estimates of contaminant concentration, or bioaccumulation rate parameters. The approach is not a full-blown CERCLA-level ecological risk assessment, which may be too resource and labor intensive for widespread application. Rather, the approach uses key risk assessment principles, including identification of key local receptors to protect, use of probability theory, development of multiple risk-based thresholds, and a decision framework (U. S. EPA 1997, 1998, Bridges *et al.* 2005, Chapman and Anderson 2005).

2.6.3. Multiple categories

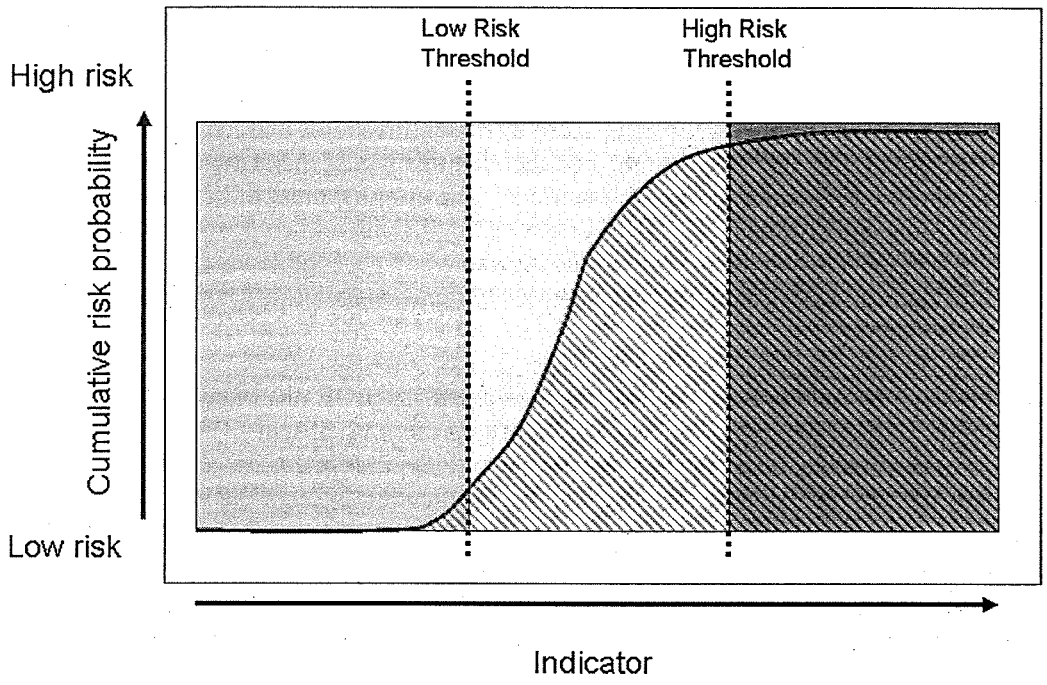
The proposed framework includes multiple categories for probability of indirect effects. The field prey tissue and sediment chemistry LOE are separately compared to three categories. The final classification results in sediments classified into four categories: unlikely impact, possible impact, likely impact, or clear impact.

Multiple categories address the uncertainty inherent in effects determinations. The use of multiple categories also allows straightforward application of a probabilistic risk-assessment framework.

The benefit of using multiple thresholds and categories for the indirect effects framework can be illustrated with a simple conceptual model (Figure 2.3). The blue curve in Figure

2.3 presents cumulative risk probability as a function of a given parameter such as pollutant concentration in sediments or tissues. The overall risk estimate depicted in this curve would be obtained by combining distributions from the parameters used in a risk assessment calculation. Thresholds may then be established across this probability distribution to represent relative probability of adverse effects. The two vertical dotted lines represent two thresholds (i.e., a low and high threshold), which partition the probability distribution into areas of low, intermediate, and high risk. For example, we may take the horizontal axis to represent exposure concentrations in biota, and the vertical axis to represent probability of adverse effects.

Figure 2.3. Conceptual model of the use of multiple thresholds for a numeric indicator used in sediment quality assessment. See text for details.



The use of two thresholds and three risk categories is an intuitive and straightforward way to combine aggregated risk information into an overall assessment of risk probability. Below the low threshold, cumulative probability of adverse effects (i.e., the shaded area under the curve) is relatively low. In the context of sediment quality assessment, an indicator below the low threshold would provide evidence of a lack of impact. Above the high threshold, this probability is relatively high. This would provide evidence indicating probable impact. Between the two thresholds, probability is intermediate; in the context of assessment, this would suggest that there is uncertainty regarding sediment impact.

The conceptual model in Figure 2.3 specifically depicts two risk thresholds resulting in three categories for a LOE. This also makes intuitive sense for partitioning risk in a

probabilistic assessment. For risk-based assessments, it is intuitive to identify the results as either above a benchmark where adverse effects are highly probable (i.e., probable exposure), below a separate benchmark where adverse effects are highly unlikely, or in between these two benchmarks (i.e., possible exposure). For example, wildlife effects threshold data are frequently presented as "no-effects" and "low-effects" levels, and therefore readily amenable to two benchmarks.

There is good precedent for partitioning individual LOE into three categories. The use of three categories is often seen in site-specific risk assessments, and has been recommended in recently published decision-making frameworks for soil and sediment risk assessment (Chapman and Anderson 2005, Hull and Swanson 2006). Three categories are also incorporated into the TrophicTrace bioaccumulation prediction software (Bridges and von Stackelberg 2003), and were used by U. S. EPA in its national screening level assessment of sediments (U. S. EPA 2004).

Despite the precedent of using three categories to partition risk and the conceptual model depicted above, one might ask whether more than two thresholds and three categories should be used for an individual LOE. We feel that this would make the assessment process unnecessarily complex and contentious. The establishment of benchmarks for the prey tissue and sediment chemistry LOE will be difficult and controversial. Policy judgments must be made about appropriate receptors and acceptable levels of risk. Given these difficulties, fewer categories are expected to focus decision-making and require less policy disputation.

In contrast to the field prey tissue and sediment chemistry LOE, the bioavailability LOE is interpreted using two categories: bioavailability present vs. absent. The purpose of this LOE is to verify a key assumption in the MLOE strategy: sediment-associated contaminants are bioavailable for uptake and magnification through the food chain. This question can be addressed using a yes/no type of evaluation. A number of factors limit the development of multiple thresholds for the bioavailability LOE. First of all, extrapolation of laboratory test data to other trophic levels or species (e.g., fish) is problematic due to constraints characteristic of the test (Moore *et al.* 2005). In addition, the relationship of laboratory body burdens to effects on receptors has not been established to the degree that it can be used for regulatory purposes.

2.6.4. Sequential approach

A sequential approach is frequently recommended in risk evaluation guidance. Examples include the Tiered Approach in the U. S. ACE dredged materials testing program (U. S. EPA and U. S. Army Corps of Engineers 1991, 1998, Bridges and von Stackelberg 2003), the multiphase risk assessments in the Federal Superfund program (U. S. EPA 1997), the decision-making framework for evaluating contaminated sediments developed by Chapman and Anderson (2005), and the risk assessment framework developed by Hull and Swanson (2006). The benefit of a sequential approach is focusing resources and effort on pollutants that pose high uncertainty or potential risk. For example, the dredged material testing program initially evaluates contaminant concentrations in the dredged

sediments to determine whether they exceed general regulatory thresholds, before proceeding to more costly bioaccumulation and toxicity testing.

The framework we describe follows a sequential approach of evaluating 1. prey tissue chemistry; 2. sediment chemistry; and 3. bioavailability testing. This approach is appropriate given that the goal of the framework is to protect wildlife and human health from food web mediated exposure to contaminants in local sediments. The field-caught prey tissue LOE is the only indicator that determines whether there are indirect effects to human or wildlife prey. If concentrations in prey tissues are not high enough to pose risks to humans or wildlife, these receptors are protected for that pollutant and there is no further need to evaluate the sediments. If, on the other hand, tissue concentrations indicate that indirect effects may be occurring, the framework proceeds to determining whether the sediments may be a source of the pollutants to the food web.

Determining thresholds for the sediment chemistry LOE involves greater uncertainty and more assumptions. In addition to determining tissue thresholds for humans and wildlife, translators that relate concentrations between sediments and biota must be developed. Collection of detailed site-specific data and modeling are often required (e.g., Connolly and Glaser 1997, Arnot and Gobas 2004, Gobas and Arnot 2005).

Finally, bioavailability tests or field invertebrate collections are performed to confirm that sediment contaminants are bioavailable. These approaches are time-consuming and costly to perform. They are therefore only required in circumstances where there remains significant uncertainty as to whether sediments cause the elevated exposure of local prey. For some pollutants, literature evaluations may establish general bioavailability of the pollutants in question.

2.6.5. Application at multiple scales

As indicated in Figure 2.1, the spatial scale of the transfer pathways is expected to increase with increasing trophic level. Contamination of sediments can be monitored at a local scale (i.e., evaluation of individual sampling stations), since sediment mobility is limited. Effects on benthic invertebrates might be monitored at an intermediate spatial scale. These organisms are generally sessile, and therefore expected to reflect contaminant exposure for local conditions. At the same time, degree of contaminant bioavailability may be similar across a water body as a result of similar contaminant sources and sediment binding properties. Higher trophic levels, including fish, wildlife, and humans, move and forage at multiple locations. Therefore, exposure of these receptors is expected to occur at broader spatial scales. Due to this spatial mobility, probabilistic evaluation of exposure and effects data may be appropriate at larger spatial scales (e.g., Linkov *et al.* 2002, e.g., Gobas and Arnot 2005).

In the framework, the field prey tissue and bioavailability test LOE are evaluated at the water body scale, while the sediment chemistry LOE is evaluated at the individual station scale. There are several reasons that the prey tissue LOE is applied at the scale of a whole water body, rather than individual sediment stations. Due to difference in

sampling methods and completeness, prey samples are usually not associated with the same locations as the sediment collection station. In addition, as discussed in the conceptual model, finfish typically move within the water body and thus integrate their exposure over a wider area than an individual sediment station (Linkov *et al.* 2002, Gobas and Arnot 2005). Finally, the risk to humans and wildlife is expected to occur at a scale better represented by the water body than the individual sediment station.

The sediment chemistry LOE is evaluated at the individual station scale. Characterization of sediments at a station scale would facilitate spatial evaluation of the water body to decide upon appropriate management actions. For example, management strategies for a large contiguous impacted area may be different from strategies for several isolated impacted areas. Although evaluation of sediment chemistry occurs on an individual station scale, development of the sediment chemistry thresholds is based on information from the entire water body. This will be further discussed in Section 3.

There are many reasons why the bioavailability LOE combines individual station results to make a determination across the entire water body. The intent of the bioavailability LOE is to determine whether the sediment-associated contaminant exhibits any measurable bioavailability in the water body. Laboratory bioaccumulation tests have uncertainty related to the duration of exposure, test conditions, degree of replication, and choice of reference stations (Boese *et al.* 1997, Moore *et al.* 2005). These uncertainties may reduce the accuracy and statistical power of the results for sediment samples from a specific site. Similarly, it may not be possible to examine bioavailability in field-collected organisms at all sites, due to lack of abundance. Due to these factors, as well as the high expense of bioaccumulation tests, the bioavailability LOE combines results from stations across the water body.

2.6.6. Separate evaluation of specific contaminant classes

For effects of bioaccumulative contaminants including PCBs and legacy pesticides, the cumulative evaluation of exposure risk due to contaminant mixtures (i.e., combined effects of PCBs, legacy pesticides, and other contaminant categories) is an ongoing area of research and development. However, federal and state guidance documents generally evaluate effects to wildlife and humans on a contaminant-by-contaminant basis (e.g., Brodberg and Pollock 1999, California DTSC Human and Ecological Risk Division 2000, U. S. EPA 2000b, Klasing and Brodberg 2006). That is, PCBs as a group are evaluated separately from DDTs, and so forth. In the absence of U. S. or California – sanctioned methodologies for combining effects of multiple contaminant categories, individual contaminant classes are evaluated separately in the current formulation of the framework.

3. Technical issues in implementation of the framework

There are a number of specific technical issues that arise in evaluating each line of evidence. This section outlines these technical issues as a series of questions. The use of questions emphasizes the thought process that is undertaken in applying the framework. Possible approaches and recommendations are then presented for each question. These recommendations are intended to meet the goal of the framework, as stated in Section 2.1: *to protect humans and wildlife against the risk of contamination as a result of dietary exposure to finfish and shellfish associated with contaminated sediments.* In some cases, Appendices develop more detailed guidance and background information.

3.1. Prey tissue chemistry

Table 3.1 lists technical and policy questions to be addressed in applying the prey tissue chemistry line of evidence (LOE). These questions pertain to two topics. The first topic is estimating tissue concentrations. To estimate tissue concentrations, decisions must be made about the types of data to be used, and statistical and probability issues that arise. The second topic is the selection of the low and high effects thresholds for evaluating this LOE. General guidance is provided in this section, with additional information about parameter selection described in Appendices and U. S. EPA (2000b).

Table 3.1. Questions to address in applying the prey tissue line of evidence.

Question	Section
Questions about estimating tissue chemistry concentration:	
What data are readily available and how should data be assembled?	3.1.1
At what spatial scale should data be compiled and evaluated?	3.1.2
How should consumption advisory information be included in the assessment?	3.1.3
What species and sizes of prey organisms should be included in the evaluation?	3.1.4
How recent should data be collected to be included in the evaluation?	3.1.5
What kinds of tissue preparations should be included in the evaluation?	3.1.6
How should reporting limits be considered in the analyses?	3.1.7
What sample sizes are adequate?	3.1.8
How should tissue contaminant concentrations be estimated from available data?	3.1.9
Questions about development of tissue chemistry thresholds:	
How should the low and high tissue thresholds be calculated?	3.1.10
What toxicity reference values are readily available for wildlife and humans?	3.1.11
How can wildlife body mass and consumption rate be estimated?	3.1.12
What are some standard risk assumptions for calculating human effects thresholds?	3.1.13
Once thresholds are developed, how should they be refined and updated?	3.1.14

3.1.1. What data are readily available and how should data be assembled?

There are many data sources of contaminant concentrations in finfish and shellfish from California estuaries (Table 3.2). It will be helpful to begin by compiling a database of all samples from the water body that contains data for contaminants of concern. Prey tissue chemistry data may be assembled simultaneously with sediment chemistry and bioavailability data. Prey tissue data may be obtained from sources listed in Table 3.2, in addition to other known, locally available sources.

Ancillary information in the database will help to identify appropriate samples for evaluating the different LOE. For the prey tissue LOE, useful parameters will include capture location, sample size, study, date, length, mass, species, and preparation method. Preparation method should be in a separate column, and should include whole body, skin-on fillet, skin-off fillet, or other tissue type (e.g., liver, gonads, etc.).

Table 3.2. Partial list of potential data sources for prey contaminant concentrations in California bays and estuaries.

Data Source	Web Access	Contact
Coastal Fish Contamination Program	Not currently available on the web.	Margy Gassel, OEHHA
Toxic Substances Monitoring Program	http://www.waterboards.ca.gov/programs/smw/	Del Rasmussen, SWRCB
Southern California Bight Survey	http://www.sccwrp.org/regional/98bight/bight98_trawl_report.html	M. James Allen, SCCWRP
Regional Monitoring Program (SF Bay only)	http://www.sfei.org/rmp/rmp_data_access.html	Ben Greenfield, SFEI
State Mussel Watch Program (Shellfish only)	http://www.waterboards.ca.gov/programs/smw/	Del Rasmussen, SWRCB
CASQO Database	http://www.sccwrp.org/data/catalog.html	Steven Bay, SCCWRP
Surface Water Ambient Monitoring Program (SWAMP)	Not currently available on the web.	Jay Davis and Aroon Melwani, SFEI
Environmental Mercury Mapping, Modeling, & Analysis Database	http://emmma.usgs.gov/datasets.aspx	Stephen Wente, USGS
California Coastal Water Quality Monitoring Inventory	http://www.sfei.org/camp/index.html	Thomas Jabusch, SFEI

3.1.2. At what spatial scale should prey tissue chemistry data be compiled and evaluated?

Sections 2.5 and 2.6.5 recommend that the prey tissue chemistry LOE be compiled and evaluated at the "water body" scale. This is because many prey and predator species will be mobile and data associated with individual sediment stations will often be unavailable.

The definition of "water body" may be based on the needs of the agencies applying the indirect effects framework. In most cases, the framework would be applied for managing an entire bay or estuary, using all available data. However, if there is a regulatory need or conceptual basis to conduct evaluations at a scale smaller than the entire water body, the framework could be adapted to this. For example, if the bay is divided into separate hydrological units (reaches or basins) for 303(d) listing purposes, and sufficient data are available on each of these units, the framework could be applied to evaluate each of these units separately.

3.1.3. How should consumption advisory information be included in the assessment?

Fish consumption advisories are an important data source for the prey tissue line of evidence. In State waters, finfish and shellfish consumption advisories are developed by OEHHA. Table 3.3 briefly summarizes current advisories relevant to California bays and estuaries. The most current listings may be found on the OEHHA website at http://www.oehha.ca.gov/fish/so_cal/index.html. If OEHHA has established a fish consumption advisory based on elevated risk of human exposure to a contaminant in a water body, this indicates that human consumption of fish captured from that water body may pose a risk to the consumers. These positive findings by OEHHA are important for determining that contaminant concentrations in prey tissues are high enough to pose a risk to human sport fish consumers.

Table 3.3. Summary of OEHHA fish consumption advisories for bays and estuaries of California.

Water body	Contaminant	Advisory
San Francisco Bay and Delta	Hg and PCBs	Interim advisory recommends limited consumption of some fish species.
Tomales Bay	Hg	Advisory recommend limited consumption of some fish species by all people, and no consumption of sharks by women of childbearing age or children.
Southern California locations between Point Dume and Dana Point	PCBs and DDTs	Advisory recommends limited consumption or no consumption for some fish species and locations.

For a given water body and individual contaminant having OEHHA consumption advisories, the prey tissue line of evidence should not be classified in the lowest risk category. Rather, it should be placed in the intermediate or high-risk categories,

depending on whether calculated tissue concentrations are above or below the high-risk threshold (See Table 2.1 and Figure 2.2).

3.1.4. What species and sizes of prey should be included in the evaluation?

Species for prey tissue monitoring should be local organisms commonly consumed by humans or by wildlife predators local to the water body (U. S. EPA 2000b). Separate species may be targeted for protection of human vs. wildlife predators. Prey organisms should also be selected based on available data and knowledge of behavior and life history. Peer-reviewed literature, local experts, and the FishBase website (www.fishbase.org) may be consulted to aid in selection of appropriate target species. Predictive relationships between sediment and tissue contamination are more likely in species that exhibit limited movement or migrations, reside close to the sediment during their adult life, and prey on animals that live in or on the sediment (Figure 3.1). For example, a sportfish such as striped bass would be appropriate for assessing the potential risk to human health, however due to this species' migratory pattern, tissue residue would demonstrate little correlation with localized sediment contamination.

Figure 3.1 Traits of ideal biota for assessing indirect effects.

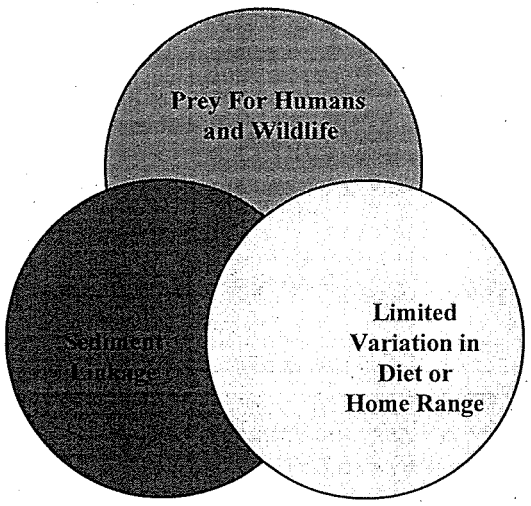


Table 3.4 lists some appropriate fish species for the prey tissue LOE. These species were selected based on life history information[11]. All species were previously captured in sediment quality and related studies assembled in the CASQO database[12]. These species were further ranked for use based on diet and movement. The top ranking (i.e., number

[11] Sources included Cailliet (2000), Tasto (1975), Domeier and Chun (1995), Orsi (1999), and Froese and Pauly (2006). Recommendations were also obtained from M. J. Allen (SCCWRP), G. Cailliet (MLML), Andy Jahn (Port of Oakland), and Kathy Hieb (CDFG).

[12] The California Sediment Quality Objectives (CASQO) database was compiled for methods development for the SQO program.

1) was reserved for species exhibiting exclusively benthic or epibenthic diets and relatively limited movement (Table 3.4).

Table 3.4. Recommended organisms for evaluation in the prey tissue chemistry line of evidence. Species listed in boldface exhibited significant positive associations between sediment and tissue concentrations for some legacy organochlorine pollutants (Appendix B).

Common name	Species name	Primary diet	Movement	Rank
Arrow goby	*Clevelandia ios*	Epibenthic	Resident	1
Bay goby	*Lepidogobius lepidus*	Epibenthic	Sedentary	1
Black perch	*Embiotoca jacksoni*	Epibenthic	Resident	1
California killifish	*Fundulus parvipinnis*	Epibenthic	Resident	1
California tonguefish	*Symphurus atricaudus*	Epibenthic	Sedentary	1
Dwarf surfperch	*Micrometrus minimus*	Epibenthic	Sedentary	1
Pacific staghorn sculpin	*Leptocottus armatus*	Epibenthic	Sedentary	1
Round stingray	*Urobatis halleri*	Benthic	Resident	1
Saddleback sculpin	*Oligocottus rimensis*	Epibenthic	Sedentary	1
Slender sole	*Lyopsetta exilis*	Epibenthic	Resident	1
Speckled sanddab	***Citharichthys stigmaeus***	**Epibenthic**	**Resident**	**1**
Starry flounder	*Platichthys stellatus*	Benthic	Resident	1
Three-spined Stickleback	*Gasterosteus aculeatus*	Epibenthic	Sedentary	1
Walleye surfperch	*Hyperprosopon argenteum*	Epibenthic	Resident	1
White surfperch	*Phanerodon furcatus*	Epibenthic	Resident	1
Barred sand bass	*Paralabrax nebulifer*	Pelagic/epibenthic	Resident	2
California corbina	*Menticirrhus undulatus*	Epibenthic	Transient	2
English sole	***Parophrys vetulus***	**Benthic**	**Transient**	**2**
Longfin sanddab	*Citharichthys xanthostigma*	Pelagic/epibenthic	Resident	2
Pacific sanddab	*Citharichthys sordidus*	Pelagic/epibenthic	Resident	2
Shiner surfperch	***Cymatogaster aggregata***	**Epibenthic**	**Transient**	**2**
Spotted sand bass	*Paralabrax maculatofasciatus*	Pelagic/epibenthic	Resident	2
Striped mullet	*Mugil cephalus*	Epibenthic	Transient	2
California halibut	***Paralichthys californicus***	**Pelagic/epibenthic**	**Transient**	**3**
White croaker	***Genyonemus lineatus***	**Pelagic/epibenthic**	**Transient**	**3**
Yellowfin croaker	*Umbrina roncador*	Pelagic/epibenthic	Transient	3
Bent-nosed clam	***Macoma nasuta***	**Benthic**	**Sedentary**	**1**

Notes to Table 3.4
Primary Diet: Benthic species primarily feed on organisms residing within the sediment; Epibenthic species primarily feed on organisms in contact with the sediment surface; Pelagic/Epibenthic species primarily feed on organisms in the water column or in contact with the sediment surface
Movement: Sedentary species move less than 1 km during adult lifespan; Resident species move between one and 10 km during adult lifespan; Transient species move more than 10 km during adult lifespan
Rank: Based on assumed sediment association and site fidelity for a species. Species that move little and prey on sediment-dwelling species are assigned the highest rank (1). The lowest rank (3) is assigned to species that are transient and feed in both sediment and the water-column.

Five fish species listed in boldface in Table 3.4 had sufficient data to demonstrate a positive statistical relationship between sediment and biota contaminant concentration and are common in coastal waters. These findings, further described in Appendix B,

suggest that these species bioaccumulate contaminants from nearby sediments and are spatially linked to the sites at which they were captured. Shiner surfperch exhibited significant positive relationships with sediments for all contaminants evaluated (Appendix B). This finding suggests that shiner surfperch may be a particularly appropriate species for sediment vs. biota comparisons. Based on the positive association with sediment contaminants, the following fish species are also strong candidates for monitoring for the prey tissue LOE: shiner surfperch, speckled sanddab, California halibut, white croaker, and English sole (Appendix B).

Among candidate invertebrate species, the bent-nosed clam (*Macoma nasuta*), and other *Macoma* species, are good indicators of prey exposure to sediment contamination (Pruell *et al.* 1993, Boese *et al.* 1997, U. S. EPA and U. S. Army Corps of Engineers 1998, MEC Analytical Systems 2003, Werner *et al.* 2004). The utility of *M. nasuta* is further discussed in Section 3.3.

In addition to species, body size is an important consideration in sample selection, particularly for finfish. Lengths may be compared to CDFG legal fishing sizes to determine whether fish samples are appropriate as human prey. Legal fishing size information may be obtained from the CDFG website http://www.dfg.ca.gov/ (California Department of Fish and Game 2006).

For assessment of effects to wildlife receptors, appropriate prey sizes will vary depending on local piscivorous wildlife. Appendix D reviews selected literature on fish prey captured by piscivorous birds. In general, small and medium shorebirds, such as western grebe, elegant tern, and least tern generally consume fish having total lengths less than 10 cm, or body depths less than 20 mm (Elliott 2005). Large birds, including double crested cormorants, osprey and bald eagle, may consume much larger fish, ranging from 100 to over 400 mm (Appendix D).

3.1.5. How recent should data be collected to be included in the evaluation?

It is preferable to use data from recently collected samples to characterize current risk. Many legacy pollutants, including organochlorine pesticides and PCBs, have shown declining concentrations since monitoring began (Greenfield *et al.* 2005, Connor *et al.* 2006, Davis *et al.* 2006a, O'Connor and Lauenstein 2006). More recent use compounds, such as PBDEs, have shown dramatic increases in recent years (de Wit 2002, She *et al.* 2002, Hites 2004).

The timeframe of data to be included may vary among pollutants and water bodies examined. Differences may result from expected or observed recent time trends for the pollutant in question, interannual variability, and availability of recent data. Time permitting, it would be appropriate to evaluate the impact of different data selection procedures on final interpretation.

As a general rule of thumb, it may be appropriate to flag data more than 10 years old and exclude it from further evaluations (i.e. at the time of publication of this report, only data

from 1998 – 2007 would be used). If information indicates temporal trends more recently than 10 years ago, and sufficient data are available, it may be appropriate to limit data to a shorter time period.

3.1.6. What kinds of tissue preparations should be included in the evaluation?

In general, tissue type and preparation can significantly influence contaminant concentrations in fish. Concentrations may vary between whole body vs. fillet tissue (e.g., Goldstein *et al.* 1996, Amrhein *et al.* 1999, Greenfield *et al.* 2001), and among tissue preparation methods (e.g., Reinert *et al.* 1972, Davis *et al.* 2002, Burger *et al.* 2003). These factors should be considered in sample preparation and selection.

Because piscivorous wildlife generally consume the entire fish, finfish sampled for the wildlife line of evidence are best evaluated as whole body samples. For risk to human consumers, all finfish samples should be analyzed as fillet tissue, unless local information indicates that the consumer population regularly consumes additional tissues. U. S. EPA (2000b) recommends analyzing all fish samples as skin-on fillets, with belly flap included for evaluating trace organic contaminant exposure to the general population or sport fishers. If evaluations are focused on Hg, skin-off fillet preparation will be more conservative, as Hg tends to accumulate in muscle tissue (U. S. EPA 2000b).

In some cases it may be necessary to estimate tissue contaminant concentrations in whole body vs. fillet samples. Appendix E provides a methodology for conducting this conversion for lipid-associated contaminants[13] based on estimating lipid concentrations among tissue types.

3.1.7. How should reporting limits be considered in the analyses?

Reporting limits (RL)[14] should be sufficiently low to quantify contamination in biota at concentrations relevant to the tissue LOE thresholds. One of the findings of the Newport Bay case study (Section 4) was that reporting limits for field-caught finfish were often too high to compare PCBs and legacy pesticides to the low tissue thresholds.

To have a good chance of quantifying PCBs and DDTs at environmentally relevant concentrations, tissue chemistry analytical RLs should be 1 ng/g or lower for individual PCB congeners and p,p'-DDTs[15]. Such RLs are readily achievable in analytical labs, provided that sufficient tissue is available for analysis (U. S. EPA 2000b).

In cases where most samples are below reporting limits (BRL), treatment of BRL samples can strongly affect the calculated average. This issue is further explored in the Newport Bay case study (e.g., Table 4.10). One method of dealing with these situations

[13] Including PCBs, legacy pesticides, and other halogenated organic pollutants

[14] A reporting limit (sometimes referred to as the sample quantitation limit) is the lowest concentration at which a numeric value is reported. Below the reporting limit, accurate concentration estimates cannot be made.

[15] Including p,p'-DDD, p,p'-DDE, and p,p'-DDT

is application of the robust method of Helsel and Hirsch (2002) for estimating summary statistics (Chapter 13 in Helsel and Hirsch 2002). Specifically, the observed data above the RL are plotted on a normal scores plot to determine the appropriate transformation, a linear regression model is applied, and estimates of all BRL values are imputed based on this regression. The imputed values are then combined with data above the RL to compute summary statistic estimates (Helsel and Hirsch 2002).

In some cases, it may be necessary to estimate concentrations when almost every analytical result is BRL. In the Newport Bay case study (Section 4), this was the case for heptachlor, dieldrin, aldrin and toxaphene. With a vast majority of BRL values, it is not possible to calculate the mean or confidence intervals of the results.

When all or almost all results are BRL, one option is to use the median of all RLs to estimate average tissue concentrations (Helsel and Hirsch 2002). A more conservative option is to use the maximum RL or the maximum reported value above the RL (whichever is higher); this second option will closer approximate an upper confidence limit estimate of concentration. Ultimately, it may not be possible to confirm whether tissue residues are above or below relevant tissue chemistry thresholds. If this turns out to be the case, supporting documentation should describe these inconclusive results, and additional analyses at lower RL may be advisable.

3.1.8. What sample sizes are adequate and how can data be interpreted given limited sample sizes?

Assessment of prey tissue chemistry should be based on sufficiently large sample sizes. Appendix F describes power analysis conducted using California fish tissue data to determine optimal sample sizes. These analyses suggest that sample sizes below 10 separate samples per water body may result in estimated average concentrations that deviate substantially from the actual average concentrations within the water body. Therefore, when planning additional analyses for the framework evaluation, it would be ideal to obtain at least 10 separate prey samples for each primary receptor.

If sample sizes are below 10, it may be appropriate to use lower and upper confidence intervals for comparison to the thresholds. For example, the average concentration could be replaced by the 95% upper or lower confidence interval of the average, for comparison to the upper and lower tissue threshold, respectively. If results were inconclusive due to low sample sizes, it may not be possible to make a confident determination regarding risk due to prey consumption. In such a situation, it would be appropriate to select the intermediate-risk category for the prey tissue LOE. This would reflect the uncertainty in the assessment and consequently place greater emphasis on the other lines of evidence (see Figure 2.2)

Sample size may also be increased by combining results for multiple sediment-associated prey species. Alternatively, variability of concentration estimates may be reduced by accounting for confounding factors, like tissue percent lipid. The approaches of

combining multiple species and lipid normalization are further evaluated in the Newport Bay case study (Section 4).

3.1.9. How should tissue contaminant concentrations be estimated from available data?

Selection of appropriate parameter estimates is important in probabilistic risk assessments. Prey tissue contaminant concentration is a key local parameter for the prey tissue chemistry LOE. In order to determine whether the prey tissue line of evidence indicates unlikely exposure, possible exposure, or probable exposure, estimates of contaminant concentration must be compared to low and high effects thresholds (Section 3.1.10). A probability-based approach is recommended to estimate the appropriate prey tissue concentration to compare to these thresholds.

Estimates of the central tendency of tissue concentrations include arithmetic and geometric means, and the median. In general, the arithmetic mean will be appropriate to provide an unbiased estimate of the average exposure of consumers to contamination in the prey (Appendix G).

Estimators of the potential range of the entire data set include various upper and lower confidence limits (including 75%, 90%, 95% and 99% upper and lower confidence limits). All of these estimates represent extremes in predator contaminant exposure. Their use in the threshold comparison would likely overestimate or underestimate actual exposure over the lifespan of a predator.

Confidence limits of estimates for the average are distinct from confidence limits for the range of the entire data set. The difference between average and range estimates creates substantial differences in predator exposure estimates. Figure 3.2 presents simulation results to illustrate this point. In Figure 3.2, estimates of the average of the data set (e.g., 95% upper confidence limit of the arithmetic mean[16]) exhibit a much smaller range of uncertainty than estimates of overall range of the data set (e.g., 95% upper confidence limit of the data[17]).

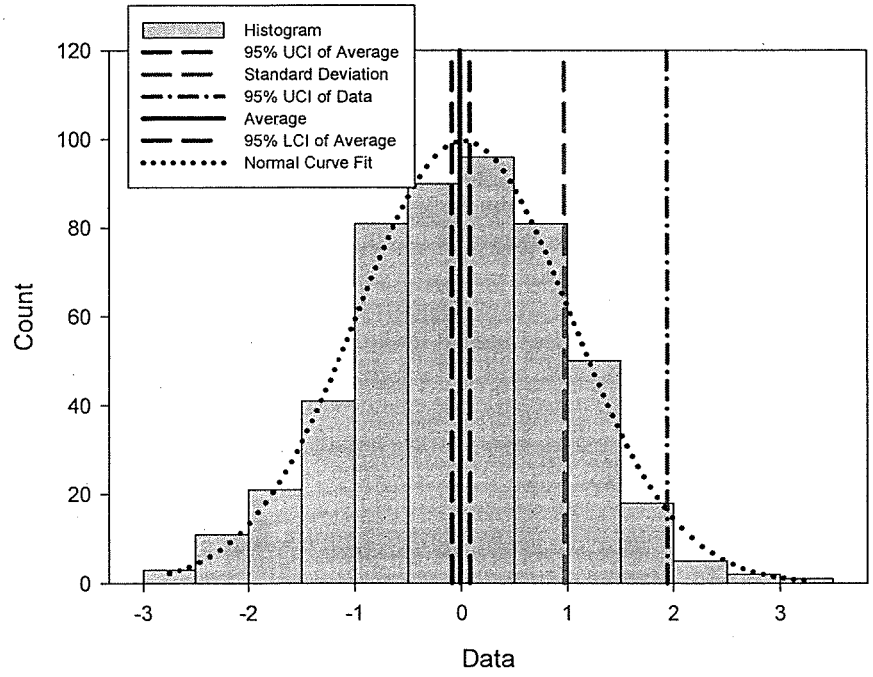
The difference between the two types of confidence interval calculation methods (Figure 3.2) is important in choosing appropriate estimates for the assessment. This is because predator lifetime exposure is best represented by average prey tissue concentrations. The population of receptors (humans) are expected, on average, to consume prey across the entire water body, rather than occurring at only one location. Therefore, we recommend estimations of the average, and calculations of uncertainty regarding the average, for use in the prey tissue LOE.

Selection of the exact tissue concentration estimation method for comparison to thresholds involves judgment about how conservative the approach should be. The high threshold represents a concentration above which adverse effects to the consumer are probable. For comparison to the high threshold, the simple arithmetic average

[16] i.e., 2 times the standard error
[17] i.e., 2 times the standard deviation

concentration may be used. The use of arithmetic average is appropriate for dietary uptake of contaminants due to prey consumption (Appendix G).

Figure 3.2. Potential estimators of a randomly generated simulated data set consisting of 500 data points. The data are simulated from a normal distribution with mean of zero and standard deviation of one. The grey bars represent a histogram of the data set. The different vertical lines represent different estimators of the data. For example, the solid black line represents the arithmetic average.



The estimated concentration for comparison to the low threshold should be conservative. This is because the analysis does not proceed to the next LOE (sediment chemistry) unless estimated tissue concentrations are above the low threshold (Section 2.5; Table 2.1; Figure 2.2)[18]. An appropriate estimate may be the 95% upper confidence interval (UCI) of the average tissue concentration (e.g., mean plus two times the standard error of the mean). When sample sizes are relatively small, use of the 95% UCI will account for difficulty in estimating the population average. When sample sizes are large, the 95% UCI will actually be very similar to the mean concentration (Figure 3.2), and will have negligible impact on the threshold comparison.

3.1.10. How should the low and high tissue thresholds be calculated?

For the indirect effects assessment framework, prey tissue effects thresholds are needed to classify the level of potential risk. Separate tissue thresholds are developed to be

[18] For water bodies having OEHHA fish consumption advisories (Table 3.3), the prey tissue chemistry assessment should automatically be placed into the intermediate or high-risk category, following 3.1.3.

protective of wildlife and human consumers of the prey organisms. Additionally, for each receptor, both low and high prey tissue effects threshold are developed, in order to address uncertainty inherent in effects of dietary exposure (Table 2.1, Figure 2.2).

The effects thresholds are developed based on a combination of policy decisions and scientific information. The State Water Board is the agency responsible for ultimately deciding policy related issues in regard to statewide water quality regulation control plans and policies for surface waters.

Policy decisions include the allowable level of risk (e.g., rate of allowable increased cancer risk for humans) and the specific receptors and target population to be protected. For wildlife, receptors should be species of management importance for the evaluated water body. For humans, the target population needs to be selected, particularly with respect to the rate of fish consumption. Examples of target populations include the general population in the region, sport fishers, or subsistence fishers (U. S. EPA 2000b).

Choices of receptor and allowable risk level strongly influence calculated thresholds for sediments or fish tissue. This report describes some potential choices and provides relevant technical background information, but is not intended to be the final word on policy development. If tissue thresholds are used in a policy framework, there should be many opportunities for stakeholder review and feedback.

Prey tissue effects thresholds can be calculated based on two parameters, which are commonly used in wildlife risk assessments (e.g., Sample *et al.* 1996, California DTSC Human and Ecological Risk Division 2000, von Stackelberg *et al.* 2003, Arcadis G&M Inc. and Matrix Design Group 2004, Zeeman 2004). The first parameter measures the relative toxicity of the contaminant to a particular wildlife species; as toxicity increases, the threshold will decrease. This parameter is referred to as the toxicity reference value (TRV). The second parameter is the expected rate of exposure by the wildlife receptor; as daily exposure rate increases, the prey tissue threshold will decrease. The rate of exposure is equivalent to the prey consumption rate by the predator, relative to the body mass of the predator. All of these relationships are mathematically depicted in following simple equation:

$$\text{Threshold concentration in prey tissue} = \text{TRV} * \frac{\text{MA}}{\text{CR}} \qquad \textit{(Equation 3.1)}$$

Here, TRV equals the toxicity reference value, which is defined as a level of exposure that indicates a probability of adverse effects to the target organism. The more toxic contaminants have lower TRVs. MA and CR are the body mass (MA) and daily prey consumption rate (CR) by the predator. Note that as MA/CR decreases, the mass-specific consumption rate increases, resulting in lower threshold values.

Detailed methodology for deriving thresholds to protect human consumers of fish may be found in U. S. EPA guidance documentation (U. S. EPA 2000d, 2000b). For non-carcinogens, the general approach is the same as for wildlife thresholds:

$$\text{Threshold} = \frac{\text{RfD} * \text{MA}}{\text{CR}} \qquad \textit{(Equation 3.2)}$$

where RfD = oral reference dose (mg/(kg*d)), which is in essence a TRV applied to humans. MA and CR are as above, but for a target human population. In short, the methodology requires good estimates of three parameters (RfD, MA, and CR).

For human exposure to carcinogens, the calculation also incorporates the carcinogenicity of the compound relative to the maximum acceptable risk level:

$$\text{Threshold} = \frac{\text{RF} * \text{MA}}{\text{q1} * \text{CR}} \qquad \textit{(Equation 3.3)}$$

where RF = Risk Factor (the maximum acceptable risk level; dimensionless), q1 = Oral cancer slope factor (a measure of carcinogenicity; kg*d/mg), and the other parameters are as above. The risk factor indicates the expected number of increased cancer occurrences in the human population. The decision about what risk factor to use is a policy decision, rather than a technical decision.

3.1.11. What toxicity reference values are readily available for wildlife and humans?

Toxicity reference values are an important component of threshold determination. Due to the large impact of TRV selection on threshold calculations, TRV development can be controversial. To reduce cost, it may be appropriate for previously calculated TRVs to be used in the framework. This section describes TRVs that have been developed and recommended elsewhere.

For protection of humans, TRVs (i.e., reference doses and cancer slope factors) are readily available from standardized U. S. Federal guidance on contaminant toxicity. In particular, the U. S. EPA's Integrated Risk Information System (IRIS) provides a consistent and updated data source, including reference doses and cancer slope factors for use in human health risk evaluation (U. S. EPA 2006b). For developing human health effects thresholds, cancer and non-cancer effects may be calculated based on these U. S. EPA IRIS cancer slope factors (CSF) and reference doses (RfD). Tissue thresholds may then be based on the CSF or RfD approach, whichever is more protective.

For protection of wildlife receptors, there is no single source for guidance on TRVs equivalent to the U. S. EPA IRIS website. However, a number of TRVs have been developed. Ideally, TRVs should fit three criteria:

1. Ecologically relevant endpoints that are likely to affect populations. These include mortality, growth, reproduction, and development (PRC Environmental

Management 1997, California DTSC Human and Ecological Risk Division 2000, U. S. EPA 2005).
2. A review of all available literature.
3. A consensus approach in development. Specifically, toxicologists from multiple agencies should reach agreement on the TRVs in a team-based effort.

Appendix H presents a set of toxicity reference values that may be considered for inclusion in the assessment framework.

Federal and state agencies in U. S. EPA Region IX have developed a Biological Technical Assistance Group (BTAG), which evaluates various issues in wildlife risk assessment in California[19]. The BTAG has established a formal process for developing and refining TRVs based on additional available data (California DTSC Human and Ecological Risk Division 2000). Where appropriate, TRVs developed by the BTAG are presented in Appendix H. The input of the BTAG would be beneficial in developing TRVs for the assessment framework. Input from regional stakeholders should also be considered when refining the TRV values used in the framework.

Another source of TRVs and related toxicity information is the U. S. EPA Ecological Soil Screening Levels (ECO-SSL). The ECO-SSLs are developed as a collaborative process including Federal and State agency toxicologists, in addition to consultants, industry scientists, and academics (U. S. EPA 2007). Although ECO-SSLs are not currently available for most organic pollutants, Appendix H includes a draft ECO-SSL developed for dieldrin (U. S. EPA 2005). Only a few syntheses of information on indirect effects of pollutants to predatory finfish have been published. Appendix I summarizes the findings of three recent studies (Johnson *et al.* 2002, Meador *et al.* 2002b, Beckvar *et al.* 2006) that recommend values for protection of finfish.

3.1.12. How can wildlife body mass and consumption rate be estimated?

To develop prey tissue thresholds (Equation 5.1), wildlife predator body mass and consumption rate must also be estimated. For protective thresholds in the absence of local data, generic body mass estimates may be used (Table 3.5) (B. Stanton, R. Donohue, and J. Yamamoto, CDFG-OSPR, *Pers. comm.*). Alternatively, if local body mass data are available, they may be used for local wildlife receptors.

Daily food consumption rates may be calculated based on body mass using allometric equations presented in Nagy (2001). Figure 3.3 presents an illustrative example, in which PCB low and high thresholds for small birds are calculated. Appendix J presents prey tissue concentration thresholds (dry weight) calculated using the TRVs from Appendix H, and the body masses and feeding rate equations from Table 3.5.

Note that dry weight concentrations may be estimated from wet weight concentrations as:

[19] EPA Region IX includes California, Nevada, Arizona, Hawaii, and the Pacific Islands. However, the BTAG focuses primarily on toxicology issues in California.

$$CDW = \frac{CWW}{(1 - Moist)} \qquad \textit{(Equation 3.4)}$$

where CDW = contaminant concentration in tissue (dry weight basis), CWW = contaminant concentration in tissue (wet weight basis), and Moist = proportion of total tissue mass that is moisture.

In the absence of local tissue percent moisture, it is common use a factor of five to convert from dry weight to wet weight for finfish prey (Jarvinen and Ankley 1999, Meador *et al.* 2002a). Therefore, if the proportion of tissue moisture is unknown, finfish dry weight concentration may be estimated assuming 80% tissue moisture as CDW = CWW*5.

Table 3.5. Generic size classes (based on mass) to calculate tissue concentration thresholds (B. Stanton, R. Donohue, and J. Yamamoto, CDFG-OSPR, *Pers. comm.*). These masses may be replaced with site-specific information on likely species, when such information are available. IR = ingestion rate.

Birds	Size range	Example species	Body mass to use in generic calculations	Equation to estimate food ingestion rate (IR) for generic calculations
Small	25 - 300 g	Snowy plover Western sandpiper Killdeer Least tern Forster's tern Black skimmer	25 g	Marine Birds (Nagy, 2001) IR (g dry mass/d) = 0.880*(g body mass)$^{0.658}$
Medium	300 – 1000 g	Clapper rail Lesser scaup Surf scoter Western grebe Black-crowned night heron	300 g	
Large	>1000 g	Brown pelican Bald eagle Osprey Double-crested cormorant Great blue heron	1000 g	
Marine Mammals				
Small	20 – 90 kg	Southern sea otter	20 kg	Carnivores (Nagy, 2001) IR (g dry mass/d) = 0.153*(g body mass)$^{0.834}$
Large	>90 kg	Harbor seal California sea lion Steller sea lion	90 kg	

Figure 3.3. Example to illustrate calculation methods for small bird effects threshold following guidance in Sample *et al.* (1996) and Zeeman (2004). dw = dry weight and ww = wet weight.

$$\text{Threshold value concentration in prey tissue} = TRV * \frac{MA}{CR} \qquad \textit{(Equation 3.1)}$$

where TRV = toxicity reference value, MA = predator body mass, and CR = daily consumption rate

For PCBs in small birds (Following Appendix H):

Low TRV = 0.09 mg dw/(kg*day); (California DTSC Human and Ecological Risk Division 2000)
High TRV = 1.27 mg dw/(kg*day); (California DTSC Human and Ecological Risk Division 2000)

MA for small bird = 25 g; (Table 3.5)

CR (in grams dw) = Allometric function of body mass (in grams dw) = $(0.880 * MA^{0.658})$; (Nagy 2001)

Low prey tissue threshold = TRV_{low} * Mass / Consumption rate
= $0.09 * 25 / (0.880 * 25^{0.658})$
= $0.09_{mg/(kg*d)} * 25_{g} / 7.32_{g/d}$
= 0.308 mg/kg
= 0.308 ppm dry weight

High prey tissue threshold = TRV_{high} * Mass / Consumption rate
= $1.27 * 25 / (0.880 * 25^{0.658})$
= $1.27_{mg/(kg*d)} * 25_{g} / 7.32_{g/d}$
= 4.34 mg/kg
= 4.34 ppm dry weight

3.1.13. What are some standard risk assumptions for calculating human effects thresholds?

The framework is intended to protect humans against the risk of contamination as a result of dietary exposure to finfish and shellfish associated with contaminated sediments Following Equations 3.2 and 3.3, two policy decisions must be made to determine appropriate thresholds for this goal. These policy decisions are the human population to be protected, and the allowable risk factor for carcinogens.

In human health risk calculations, the population to be protected is identified based on the daily fish consumption rate (i.e., CF in Equations 3.2 and 3.3). Consumption rates are typically chosen to be representative of the general population in the region, sport fishers, or subsistence fishers (U. S. EPA 2000b). Depending upon this choice, a wide range of potential consumption rates may be appropriate. For example, the U. S. EPA estimates consumption at 17.5 g/d for the general adult population and recreational fishers, and 142.4 g/d for subsistence fishers (U. S. EPA 2000b). The consumption rate for the general population listed in the California Toxics Rule was 6.3 g/d (U. S. EPA 2000d), based on prior U. S. EPA guidance (U. S. EPA 1995b). Empirical estimates of average consumption rate by sport fishers in California waters range between 18 g/d and 32 g/d (Allen *et al.* 1996, SFEI 2000). To develop fish tissue screening values, OEHHA has used consumption rates ranging from 21 g/d (Brodberg and Pollock 1999) to 90 g/d (Klasing and Brodberg 2006). Median consumption rates as high as 540 g/d have been reported for Native American subsistence consumers (Harris and Harper 1997); however, such high rates are unlikely in California (R. Brodberg, OEHHA, *Pers. comm.*). Among 501 recent sport fish consumers surveyed in San Francisco Bay, the highest individual consumption rate reported was 324 g/d (SFEI 2000).

Average lifetime consumption rate can be modified to account for expected variations in lifetime exposure. That is, the total risk will be proportional to the time period spent consuming the local fish averaged over the total lifespan. For example, the draft report of Klasing and Brodberg (2006) assumed that 30 years of a 70 year lifespan would be spent consuming fish at the current contaminant level. The calculated screening values were consequently based on exposure rates of 3/7 of maximum lifetime exposure (Appendix K, Table K.1).

For carcinogens, the allowable risk factor[20] (RF, following Equation 3.3) must also be selected. U.S. EPA has stated that risk factors between 10^{-4} to 10^{-6} are protective of public health under the agencies statutory authorities including the Federal Clean Water Act (Reference. USEPA 1995 *Water Quality Guidance for Great Lakes System Supplementary Information Document.* EPA 820-B95-001 Office of Water , March). As a result this represents the range typically applied in human risk evaluations. A 10^{-4} RF has commonly been used as an action level in Federal Superfund site assessments (i.e., CERCLA). At these sites, when total carcinogenic risk is lower than 10^{-4}, remediation activities may not be warranted (Hamilton and Viscusi 1999). A RF of 10^{-4} was also

[20] The population-level expected rate of increased cancer risk

proposed by OEHHA scientists in a recent draft report developing guidance fish tissue levels and screening values[21] (Klasing and Brodberg 2006).

A RF of 10^{-5} or 10^{-6} has been used in many Water Quality Control Plans, Basin Plans, and TMDLs throughout California. A 10^{-5} RF was also used by OEHHA previously in developing fish consumption advisories in California water bodies (Brodberg and Pollock 1999), and by U. S. EPA in recommending human health screening values (U. S. EPA 1995b, 2000b). A 10^{-6} RF was used in combination with a general population fish consumption rate (6.5 g/d) in the California Toxics Rule legislation (U. S. EPA 2000d).

Another consideration in human health threshold development is changes in tissue concentration as a result of tissue preparation and cooking method. Exposure to organic pollutants may be reduced by cooking the fish (Reinert *et al.* 1972, Puffer and Gossett 1983). This may be accounted for by calculating the population exposure rate as a portion of the raw tissue concentrations. For example, in their draft guidance document, Klasing and Brodberg (2006) reduce exposure rates to 0.7 of raw tissue concentrations in their calculations. For Hg, Burger *et al.* (2003) actually found concentrations to increase with cooking, and recommended a conversion factor of about two for increased Hg concentration in cooked fish. Finally, exposure rates can differ based upon preparation method. For finfish, concentrations of organic pollutants are generally higher in fillets with skin, than skin-off fillets (Davis *et al.* 2002). Consumption of digestive tract tissue or preparation of fish in soups has been reported by some anglers (Allen *et al.* 1996, SFEI 2000), and these methods may cause changes in contaminant exposure.

Appendix K presents a set of calculated tissue thresholds, incorporating various assumptions described above. Where possible, these thresholds are based on assumptions provided in previous national and state guidance documents.

3.1.14. Once thresholds are developed, how should they be refined and updated?

Once developed, thresholds should be reviewed periodically in order to reflect changes in national guidance and the availability of additional data. All relevant TRVs, cancer slope factors, reference doses, and consumption rates should also be reviewed. The U. S. EPA Region IX Biological Technical Assistance Group (BTAG) has established a formal process for developing and refining TRVs based on additional available data (California DTSC Human and Ecological Risk Division 2000). The input of the BTAG should be considered in the review. Scientists at OEHHA-California EPA should also be consulted, particularly regarding human health thresholds and underlying assumptions. Finally, feedback from stakeholders should be solicited and reviewed.

[21] OEHHA defines screening values as, "specific guidance tissue levels used to identify situations where contaminant concentrations in fish are of potential health concern and further action (e.g., additional sampling or developing consumption advice) is recommended." (Klasing and Brodberg 2006)

3.2. Sediment chemistry

Table 3.6 presents some technical and policy questions that must be addressed in applying the sediment chemistry line of evidence. The first three questions pertain to the collection and analysis of sediment chemistry data. Other questions evaluate methods for development and application of the sediment chemistry thresholds. The recommended approach is to develop thresholds based on prey tissue thresholds and a bioaccumulation rate parameter. The bioaccumulation rate parameter is needed to back-calculate sediment chemistry thresholds from tissue thresholds.

Each question in Table 3.6 is discussed in an individual section. Additional guidance on sampling and analysis for sediment chemistry will be provided in other documents prepared for the sediment quality objectives program. Zeeman (2004) also provides a good introduction to development of bioaccumulation rate parameters.

Table 3.6. Questions to address in applying the sediment chemistry line of evidence.

Question	Section
What compounds should be evaluated?	3.2.1
What data are readily available and how should data be assembled?	3.2.2
How should additional sediment chemistry data be collected?	3.2.3
Questions regarding calculation of the bioaccumulation rate parameter and the sediment chemistry threshold:	
How should the sediment chemistry thresholds be calculated?	3.2.4
What prey species should be included in bioaccumulation rate parameter development?	3.2.5
Should the bioaccumulation rate parameter be normalized to lipids or organic carbon?	3.2.6
How should the bioaccumulation rate parameter be estimated from available data?	3.2.7
How can mechanistic models be used to validate the bioaccumulation rate parameter?	3.2.8
How are sediment data compared to the low and high thresholds?	3.2.9

3.2.1. What compounds should be evaluated?

Target compounds should be selected based on results of the prey tissue chemistry LOE. Consistent with application of the assessment framework, separate evaluatations are conducted for human health vs. wildlife risk. If prey tissue chemistry concentrations are below the low tissue threshold, and there is no listing of the water body for fish consumption, then the framework determines that compound to pose "unlikely risk" (see Table 2.1 and Figure 2.2). It is only necessary to evaluate a compound in the sediment chemistry line of evidence if the prey tissue results are above the low tissue threshold or if that compound is listed by OEHHA for a human health consumption advisory (Table 3.3).

3.2.2. What data are readily available and how should data be assembled?

As with the prey tissue chemistry LOE, a sediment chemistry database should be assembled from all readily available sources. Some data sources are listed in Table 3.7. Additional data may also be sought from the U. S. Army Corps of Engineers, local ports responsible for dredging activity, and other local research and management agencies.

An extensive database of historical sediment chemistry has already been prepared, which should aid in database preparation for specific California bays and estuaries (Table 3.7, first row). Data from this California Sediment Quality Objectives (CASQO) database should be included in the survey of available sediment chemistry data sets.

Table 3.7. Partial list of potential data sources for sediment contaminant concentrations in California bays and estuaries.

Data Source	Web Access	Contact
CASQO database	http://www.sccwrp.org/data/catalog.html	Steven Bay, SCCWRP steveb@sccwrp.org
Southern California Bight Survey	http://www.sccwrp.org/data/metadata/1998chem.htm	Steven Bay, SCCWRP steveb@sccwrp.org
Regional Monitoring Program (SF Bay only)	http://www.sfei.org/rmp/rmp_data_access.html	Cristina Grosso, SFEI cristina@sfei.org
U. S. EPA West Coast EMAP	http://www.epa.gov/emap/nca/html/data/index.html	Dan Guzman, U. S. EPA Corvallis
Bay Protection and Toxics Cleanup Program	http://www.swrcb.ca.gov/bptcp/	Rusty Fairey, MLML
California Coastal Water Quality Monitoring Inventory	http://www.sfei.org/camp/index.html	Thomas Jabusch, SFEI thomas@sfei.org
Los Angeles Basin Contaminated Sediments Task Force (CSTF)	http://www.sccwrp.org/data/catalog.html	Steven Bay, SCCWRP
U. S. EPA National Sediment Quality Survey	http://www.epa.gov/waterscience/cs/nsidbase.html	Bob Shippen, U. S. EPA shippen.robert@epa.gov

In addition to sediment chemistry for contaminants of concern, the database should also include ancillary information when available. Key parameters include sample location with GIS coordinates, date collected, sediment grain size, percent organic carbon, percent moisture, detection and reporting limits, and sample collection depth.

As with the prey tissue chemistry LOE, sediment chemistry may be expected to change over long time periods (e.g., Venkatesan *et al.* 1999). To minimize the risk of long-term changes, it may be appropriate to limit analyses to data collected within the last 10 years or a shorter time period.

3.2.3. How should additional sediment chemistry data be collected?

The primary uses of sediment chemistry data in the framework are: 1. development of empirical bioaccumulation rate parameters, and 2. assessment of sediments within the water body. Guidance documents exist on appropriate methods for sediment chemistry data collection and analyses (e.g., U. S. EPA and U. S. Army Corps of Engineers 1991, 1998).

To increase the usefulness of sediment chemistry data for the framework, several specific recommendations should be followed. Samples should generally be collected from the sediment surface (i.e., top 5 to 10 cm), as these are most representative of potential exposure to local prey species. Sample detection limits should be sufficiently low to allow for accurate comparison to the low and high sediment chemistry thresholds. For example, in the case studies (Sections 4 and 5), detection limits at or below 1.0 ng/g dry weight were generally needed to allow comparison to the low sediment chemistry thresholds. To enable development of normalized biota-sediment accumulation factors for organic pollutants, organic carbon concentration should also be collected alongside sediment chemistry.

Sampling design is an important consideration that will depend on the specific objectives of the data collection. If new data are needed for current assessments, a probabilistic survey design may be employed. The U. S. EPA Office of Research and Development[22] has developed extensive guidance on development of probabilistic survey designs, which would be appropriate for sediment surveys.

If available data are insufficient to characterize empirical bioaccumulation rate parameters, it may also be appropriate to perform a special study to characterize sediment vs. prey tissue contaminant concentrations. This would include assessment of multiple stations in a large water body. A gradient design may be employed, emphasizing a range from low to high sediment contaminant concentrations.

3.2.4. How should the sediment chemistry thresholds be calculated?

As discussed in section 2.5, the sediment chemistry thresholds are calculated as a quotient of the prey tissue thresholds and a bioaccumulation rate parameter:

$$\text{Sediment Threshold} = \frac{\text{Prey Tissue Threshold}}{\text{Bioaccumulation Rate Parameter}} \qquad \textit{(Equation 3.5)}$$

As with the prey tissue thresholds, development of the low and high sediment chemistry thresholds for the framework requires judgment regarding how conservative the assumptions should be. To this end, the low and high sediment chemistry thresholds should be based on the low and high prey tissue thresholds, respectively (Table 3.8). Additionally, it would be appropriate to use more conservative assumptions in estimating the bioaccumulation rate parameter for the low sediment threshold (Table 3.8).

[22] http://www.epa.gov/nheerl/arm/designpages/design&analysis.htm

Table 3.8. Summary of assumptions used for calculating low and high sediment thresholds. See also Table 3.9.

Parameter	Low sediment threshold	High sediment threshold
Prey tissue threshold	Low tissue threshold	High tissue threshold
Bioaccumulation rate parameter	More conservative estimate	Less conservative estimate

The bioaccumulation rate should be estimated empirically from measured concentrations in organisms and sediments, as well as ancillary variables known to be influential. Empirical models may be readily used to develop statistical associations between biota and sediment contamination (Mackay and Fraser 2000, U. S. EPA 2000a). These models are typically derived on a site- and species-specific basis (e.g., Froese *et al.* 1998, Wong *et al.* 2001, Burkhard *et al.* 2004). The next questions more specifically address development of the bioaccumulation rate parameter.

3.2.5. What prey species should be included in bioaccumulation rate parameter development?

The database developed for the prey tissue line of evidence may also be used for developing the bioaccumulation rate parameter for the sediment chemistry line of evidence. As with the prey tissue line of evidence, the ideal organisms for parameter development will exhibit three attributes: 1. a trophic linkage to the sediments; 2. strong local site affinity; and 3. consumed by human or wildlife predators.

One approach would be to select a single surrogate species for development of the empirical bioaccumulate rate parameter. This is performed in the San Francisco Bay case study (Section 5), in which shiner surfperch are evaluated as a representative surrogate prey species for parameter development in the water body. If a surrogate species is used, it should have a relatively sedentary life history, resulting in a relatively strong spatial association between tissue and sediment chemistry concentrations within a region and a lipid content that is similar to the prey species used in the assessment. The advantage of this association is that it will allow parameter development for individual sites or local areas within the water body, potentially reducing the uncertainty in parameter calculations. In the case of sessile benthic invertebrates, a spatial association may be assumed. For finfish, Appendix L describes a spatial regression procedure that may be used, data permitting, to identify such an association. The San Francisco Bay case study presents other approaches to demonstrate an association using existing data; these include evaluation of spatial patterns in contaminant concentrations using ANOVA, and use of stable isotope analyses (Section 5.3.1.1).

3.2.6. Should the bioaccumulation rate parameter be normalized to lipids or organic carbon?

For trace organic compounds, the most common methods of estimating the bioaccumulation rate are an uncorrected bioaccumulation factor (BAF) and a normalized biota-sediment accumulation factor (BSAF). The bioaccumulation factor (BAF) is the ratio of a chemical compound's concentration in tissue (C_t in mg/kg) to a compound's concentration in water (C_w in mg/L) or in sediment (C_s in mg/kg dry wt). For sediments:

$$BAF = \frac{C_t}{C_s} \qquad \textit{(Equation 3.6)}$$

Empirically measured BAFs integrate all environmental routes of exposure and take into account the bioavailability of the chemical in the system being studied.

The biota-sediment accumulation factor (BSAF) is the ratio of biota to sediment contamination concentration, corrected for lipid content of the biota and organic carbon content of the sediment (reviewed in Wong *et al.* 2001, Burkhard *et al.* 2004). That is:

$$BSAF = \frac{\left(C_t / f_L\right)}{\left(C_s / f_{OC}\right)} \qquad \textit{(Equation 3.7)}$$

where f_L is the fraction of lipid in tissue and f_{OC} is the fraction of organic carbon in sediment (U. S. EPA 2000a and references cited therein). The use of lipid and organic carbon normalization rests on the principle that many contaminants are predominantly associated with these matrices, producing more reliable relationships between them (Clark *et al.* 1988). Organic carbon normalization has been supported by empirical evaluations of contaminant fractionation among sediment types in multiple datasets (Di Toro *et al.* 1991, Di Toro and De Rosa 1998).

A bioaccumulation rate parameter may also be developed based on a model that is intermediate between the BAF and the BSAF. For example, wet weight tissue concentrations could be compared to concentrations in organic carbon-normalized sediment. Alternatively, lipid weight tissue could be compared to dry-weight sediment. There is less of a precedent for these approaches in the literature, as compared to a simple BAF or BSAF.

Which empirical model is used should ultimately depend on the available data and success of each model type in characterizing the sediment vs. biota relationship. Graphical and statistical analysis of the tissue lipid vs. contaminant relationship can help decide whether to lipid normalize prey tissue data. Similarly, the sediment organic carbon vs. contaminant relationship may be analyzed to determine if sediment data should be organic carbon normalized. Finally, a comparison of results from the models in terms of variance explained may aid in deciding which model to use. Linear regression analyses comparing sediment vs. biota concentrations will aid in this process.

Examples of these evaluations are provided in the Newport Bay case study (Section 4.4.1).

Accurate information on organism lipid content and sediment total organic carbon (TOC) content are needed for deriving a BSAF. In contrast, BAFs require no ancillary data other than biota and sediment contaminant concentrations. Given that these ancillary data are sometimes unavailable, a larger dataset is often available for estimating BAFs, than for BSAFs.

The BSAF has received more support and discussion in the peer-reviewed scientific literature. When thermodynamic equilibrium is reached, the BSAF is expected to range between 1 and 2 (Ankley *et al.* 1992, Moore *et al.* 2005). Equilibrium conditions may occur for short-lived benthic invertebrates, but fish and wildlife often exhibit BSAFs above or below these values, reflecting disequilibrium (van der Oost *et al.* 2003). For fish, the BSAF can vary as a result of food web trophic transfer, lack of equilibrium between the sediments and water column, variation in benthic-pelagic coupling, and metabolic breakdown of contaminants (Burkhard *et al.* 2003, Burkhard *et al.* 2004). Equilibrium conditions occur in some local studies (e.g., Froese *et al.* 1998), but BSAFs often differ from equilibrium, as a result of these factors (Morrison *et al.* 1996, Wong *et al.* 2001, Burkhard *et al.* 2004).

3.2.7. How should the bioaccumulation rate parameter be estimated from available data?

A number of decisions must be made to develop the bioaccumulation rate parameter:

a. Use of individual vs. pooled species data for prey tissue concentrations
b. The spatial scale at which biota vs. sediment samples are compared
c. Treatment of samples below reporting limits
d. How to address uncertainty of the parameter estimates
e. How to average tissue and sediment data (e.g., arithmetic vs. geometric average)

These decisions depend on the available data and require a degree of best professional judgment. The two case studies (Sections 4 and 5) present examples of how available data were evaluated to make appropriate choices for these issues. Beyond that, some general guidance can be provided.

For the optimal rigor, the decisions should be based on graphical and statistical analyses of the available empirical data. Much of the decision-making may be based on which approach minimizes uncertainty of parameter estimates. For example, uncertainty of average predator exposure may be compared between individual vs. pooled prey species data (a, above). Calculation of standard errors and confidence intervals will aid in determining whether pooling multiple prey species may improve estimates of average predator exposure. Appendix L describes an empirical method for identifying the best spatial scale at which to compare biota vs. sediment data (b, above). These analyses are also demonstrated in the case studies.

The impact of samples below reporting limits (BRL) can be significant for compounds with a majority of samples BRL (c, above). As for prey tissue chemistry (Section 3.1.7), BRL sediment chemistry values may be set to ½ the reporting limit (RL) or estimated for averaging by the robust methods of Helsel and Hirsch (2002). If analyses indicate that RL will strongly affect final determination, it may be appropriate to conduct additional sampling, using analytical labs with lower RLs.

As with prey tissue chemistry (section 3.1.9), a probability-based approach is needed to address uncertainty in parameter development (d, above). The bioaccumulation rate parameter is a quotient of prey tissue chemistry and sediment chemistry. Estimates of these individual parameters will depend on averaging method and distribution assumptions. In particular, more vs. less conservative estimates should be used to derive the low and high sediment thresholds, respectively (Table 3.8).

To account for uncertainty in the bioaccumulation rate parameter estimate (d, above), it may be appropriate to use upper and lower confidence intervals of the means in deriving the lower (i.e., more conservative) sediment chemistry threshold. This approach is employed in the case studies (Sections 4 and 5). In the case studies, the arithmetic average prey tissue and sediment chemistry concentrations are used for calculating the less conservative estimate of the bioaccumulation rate parameter, to be used in the high sediment threshold. In contrast, the bioaccumulation rate parameter for the low sediment threshold is calculated using the 95th percentile of the average tissue concentration divided by the 5th percentile of the average sediment concentration (Table 3.9). This results in a much more conservative estimate of the threshold, which is appropriate because the low threshold represents sediment chemistry concentrations below which there is a negligible expected risk of indirect effects.

Table 3.9. Summary of recommended probability approach used for calculating the bioaccumulation rate parameter in the case studies.

Parameter	**Estimates used for high sediment threshold**	**Estimates used for low sediment threshold**
Prey tissue concentration	50th percentile	95th percentile of the average
Sediment concentration	50th percentile	5th percentile of the average

Appendix G provides information on the general attributes of arithmetic vs. geometric averaging (e, above). As with prey tissue chemistry, the arithmetic mean of sediment chemistry best represents the average exposure of prey species to sediment-associated contaminants (Appendix G). The arithmetic mean sediment concentration will generally be higher than the geometric mean. Therefore, it may be appropriate to calculate both arithmetic and geometric averages and uncertainty estimates, and evaluate the impact of using one method vs. the other.

3.2.8. How can mechanistic models be used to validate the bioaccumulation rate parameter?

Mechanistic models use equations to quantify the specific contaminant uptake and loss processes (e.g., respiration, feeding, absorption, and excretion), in order to predict concentrations in biota from a specific ecosystem (Mackay and Fraser 2000). A number of mechanistic models have been developed to represent food web trophic transfer of trace organic compounds (e.g., Connolly 1991, Thomann *et al.* 1992).

A mechanistic model can aid the development and application of the bioaccumulation rate parameter in many ways. First, it may be used to ascertain whether the empirical parameter is a realistic depiction of sediment vs. biota partitioning. Second, the mechanistic model may be used to provide an independent estimate of uncertainty of bioaccumulation rate predictions, using probabilistic application, such as Monte Carlo simulation. Third, the mechanistic model may be used to identify the major sources of this uncertainty, as a means to focus future research efforts. Fourth, it may be used to evaluate the relative role of sediment vs. water column concentrations in influencing predictions. Finally, if mechanistic model results are validated by separate empirical data, the model can be used to conduct scenario testing, evaluating issues such as the potential bioaccumulation for other prey species or related contaminants, and forecasting the possible effects of specific management actions.

The comparison of the empirical vs. mechanistic models can be helpful to determine whether the empirical results indicate local sediments as a primary contaminant source. If the empirical bioaccumulation rate parameter were higher than mechanistic model results, this may indicate that a substantial proportion of the bioaccumulation is due to contaminant uptake from other sources. These may include locations outside of the water body, such as point source or non-point source discharges. Undocumented hotspots of elevated contamination may also be present within the water body. For non-sedentary species such as finfish, deviation between empirical and mechanistic results may also indicate movement and exposure to sources from outside of the embayment.

The case studies (Sections 4 and 5) use graphical evaluations to compare empirical vs. mechanistic results (see Sections 4.4.2.6, 5.3.2.2, and Appendix X). Graphical evaluations are appropriate when the number of compounds is relatively small. When evaluating a large number of compounds having detectable data for sediments and biota, as when evaluating individual PCB congeners, model bias should be estimated quantitatively. Gobas and Arnot (2005) document a simple methodology for calculating model bias.

The empirical vs. mechanistic comparison may also be helpful to determine distributional assumptions for developing the bioaccumulation rate parameter. If empirical data are limited and uncertain, use of arithmetic vs. geometric assumptions will cause substantial differences in the range between the average and the upper confidence interval. The coefficient of variation or other measures of variability in the mechanistic model results can help determine which distributional assumptions are more appropriate.

The model used in the case studies (Sections 4 and 5) is a steady state non-equilibrium model developed to assess transfer of non-polar organic contaminants through food webs (Gobas 1993, Arnot and Gobas 2004). This model simulates organic contaminant transfer from sediments and water through a multi-species food web by combining contaminant kinetics in biota (e.g., uptake and elimination) and food web dynamics (Gobas 1993, U. S. EPA 2000a). It is appropriate for evaluation of non-polar organic contaminants, such as PCBs, legacy pesticides, dioxins, and PBDEs. It is not appropriate for evaluation of metals or organometals, such as Hg, Sn, or Se.

The Gobas model is recommended as a tool in the assessment framework based on its relative ease of use, generally good correspondence with field results, and history of use in significant management applications. The model has been used in a wide range of research and regulatory applications by multiple agencies and scientists (Table 3.10). It can provide a simple estimate of the relative inputs of a particular chemical into an organism from the water column versus directly from the sediment and porewater (see Section 5.4). These estimates may be utilized in conjunction with food web interactions to estimate the direct contributions of sediment contamination to organisms throughout the food web. However, additional models and data would be required to contrast the amount of contaminant exposure due to resuspended sediments within the water column vs. direct surface water runoff or other sources. More complex kinetic models would also be required to incorporate time or age-dependant changes in biota concentrations or BSAFs (e.g., Borgmann and Whittle 1992).

A version of the model has been made available to the general public in the TrophicTrace software program developed by the U. S. Army Corps of Engineers (Bridges and von Stackelberg 2003). This version of the model was developed for dredged material disposal applications, but it is also appropriate for the present sediment assessment framework. TrophicTrace may be downloaded at http://el.erdc.usace.army.mil/trophictrace/ or by contacting Dr. Todd Bridges at Todd.S.Bridges@erdc.usace.army.mil. Technical information regarding use of TrophicTrace is also available through this website.

The model has recently been updated to incorporate new research regarding phytoplankton uptake and elimination, fish and invertebrate ventilation rates, chemical partitioning and mechanisms of gastrointestinal magnification of contaminants (Morrison *et al.* 1996, Arnot and Gobas 2004). The version applied to the work in this report contains these updates. The model equations are summarized in Appendix M. Appendix Y summarizes input data requirements for the model. A spreadsheet copy of the updated version parameterized for PCBs in San Francisco Bay is also available for free download from Frank Gobas at http://www.rem.sfu.ca/toxicology/models/models.htm. The MATLAB programmed version used for this report may be obtained by contacting the report authors.

Table 3.10. Partial list of previous applications that use or recommend the Gobas (1993) contaminant bioaccumulation and trophic transfer model.

Application	Summary	Reference
TrophicTrace[a] and FishRand	Software developed by U. S. ACE for dredged material disposal evaluation, applied at national scale	Bridges and von Stackelberg (2003)
Rhine Channel, Newport Bay	Focused ecological risk assessment	Anchor Environmental (2005)
New York-New Jersey Coast	Spatially explicit evaluation of impacts of dredged material disposal on natural fish populations	Linkov *et al.* (2002), von Stackelberg *et al.* (2002)
Moss Landing Harbor, CA	Ecological risk assessment	von Stackelberg *et al.* (2003)
San Francisco Bay[b]	Used to develop bioaccumulation factors for PCB TMDL	Gobas and Wilcockson (2002), Gobas and Arnot (2005)
U. S. EPA Federal Guidance	Recommended as part of a suite of tools for evaluating contaminated sediments	U. S. EPA (2000a)
U. S. EPA Office of Solid Waste	Formed the theoretical basis for multimedia multireceptor risk assessment model developed for U. S. EPA use in fresh waters	Kroner and Cozzie (1999)
Hudson River	Baseline ecological risk assessment for PCBs in Hudson River	TAMS Consultants Inc. (2000)
Great Lakes	Development of criteria for protection of wildlife under the Great Lakes Water Quality Initiative	U. S. EPA (1995a)
Model Validation	Model results compared favorably to independently derived field data	Arnot and Gobas (2004)
Model Validation	Model results compared favorably to another, more complex kinetic model (Thomann *et al.* 1992)	Burkhard (1998)

a. Model spreadsheet available at http://el.erdc.usace.army.mil/trophictrace/
b. Model spreadsheet available at http://www.rem.sfu.ca/toxicology/models/models.htm

If Monte Carlo simulations are performed to evaluate the uncertainty or variability in model predicted bioaccumulation rates, the probability distributions of model input parameters must be selected. Given that the primary objective of the Monte Carlo simulation is to compare empirical vs. model averages and uncertainties, probability distributions should be based on expected uncertainty in average parameter estimates. That is, standard errors about the mean should be developed and used as uncertainty input parameters. Each parameter should be based on the distribution observed in the source data, be it normal, lognormal, uniform, or some other distribution. For other objectives, other uncertainty estimates may be more appropriate. For example, if the objective is to generate the full range of possible individual estimates for the bioaccumulation rate parameter, then each uncertainty input parameter could be based on the full distribution of data available for that parameter. This would result in much wider error bounds about the model predictions, and would not be representative of the range of average exposures encountered by predators.

3.2.9. How are sediment data compared to the low and high thresholds?

Once sediment thresholds are developed, all sediment data are compared to these thresholds. Final determination of risk posed by sediments is on a station-specific basis, and results in one of four possible risk categories: unlikely risk, possible risk, likely risk, and clear risk (Table 2.1 and Figure 2.2). The spatial pattern of risk categories may be graphically evaluated using a simple map of sediment results (e.g., Figures 4.21 and 4.22).

3.3. Bioavailability

The bioavailability line of evidence (LOE) is used to establish that sediment-bound contaminants are bioavailable. This can be achieved by demonstrating that sediment-residing organisms exhibit elevated tissue contaminant concentrations when exposed to sediments from the bay or estuary being evaluated. This section provides general technical guidance in applying the bioavailability line of evidence.

A number of potential approaches are available for this line of evidence. For compounds having sufficient information in prior published studies, bioavailability may be evaluated using peer reviewed scientific literature. For relatively unstudied compounds, or compounds with inconsistent findings in the literature, local data should be used. Bioavailability tests may be conducted using either laboratory test organisms or field caught organisms. The line of evidence may be evaluated using a sampling design and statistical analysis approach based on linear regression, a t-test, or a straightforward demonstration of bioavailability at some sites in the water body.

In the proposed framework, if contaminants are demonstrated to be not bioavailable, the water body sediments would be placed in the lowest risk category: unlikely impact (Table 2.1, Figure 2.2). Due to the importance of the bioavailability LOE in determination of risk, the sampling design, data collection, and interpretation of results should be carefully considered. Specific concerns include the need for sufficient sample sizes, appropriate

test organisms, sufficiently low detection limits, and sufficient duration of laboratory tests (i.e., at least 28 days) (Table 3.11). These issues are further addressed as a series of questions listed in Table 3.12.

Table 3.11. General recommendations for the bioavailability LOE.

Issue	Recommendation
Sample size	If a statistical approach is applied, sample size should be sufficient to have adequate power to detect bioaccumulation
Test organism	All samples should be the same species; the species chosen should be a sediment dwelling and ingesting organism. If laboratory bioaccumulation tests are used, the clam *Macoma nasuta* is a good candidate test species
Detection limits	Detection limits should be sufficiently low (e.g., 1 ng/g or lower is recommended for PCBs or legacy pesticides)
Test duration	Laboratory bioaccumulation tests should be at least 28 days in duration

Table 3.12. Questions to address in applying the bioavailability line of evidence.

Question	Section
Should local data or the scientific literature be used?	3.3.1
Questions that arise if local data are collected:	
How should test results be interpreted statistically?	3.3.2
Should field-caught organisms or laboratory test studies be employed?	3.3.3
What are appropriate test organisms?	3.3.4
What factors should be considered in sampling design?	3.3.5
What sample sizes are needed?	3.3.6
What detection limits are needed?	3.3.7
What ancillary parameters should be measured?	3.3.8

3.3.1. Should local data or the scientific literature be used?

For contaminants that are well characterized, a simple review of the literature may indicate bioavailability. The focus of the literature review would be to demonstrate that the contaminants being evaluated, when associated with sediments, are generally bioavailable to benthic organisms, and are likely to bioaccumulate in the water body in question.

PCBs and legacy pesticides[23] are examples of contaminants with literature indicating bioavailability to benthic invertebrates across a wide range of conditions. Studies demonstrating associations between sediment and biota tissue concentrations include field studies of resident and transplanted bivalves (Lee *et al.* 1994, Nasci *et al.* 2000), and laboratory studies indicating that contamination in bivalves increases with increased

[23] E.g., DDTs, chlordanes, dieldrin, and toxaphene

sediment contamination (Lee *et al.* 1994, Boese *et al.* 1997, Anderson *et al.* 2001, Battelle *et al.* 2005). In addition, the cases studies in this report (Sections 4.5 and 5.5) indicate that PCBs and DDTs are generally bioavailable. Based on these findings, it may not be necessary to expend limited funds on further field studies or bioaccumulation testing for PCBs or chlorinated legacy pesticides. For these pollutants, if local data are not readily available, a conservative approach would be to assume bioavailability.

When literature results are inconsistent or uncertain, it may be appropriate to collect and analyze local data on bioavailability. The remaining questions address approaches for local data collection.

Prior to performing new studies, local agencies may be queried for readily available bioavailability data. As part of dredged materials testing protocols (U. S. EPA and U. S. Army Corps of Engineers 1991, 1998), local ports and the U. S. Army Corps of Engineers routinely evaluate bioavailability using standard laboratory test protocols. Additional bioavailability test information may be found at contaminated sites in the water body, as part of CERCLA risk assessments (e.g., Lee *et al.* 1994, Battelle *et al.* 2005) or monitoring funded by the state of California (e.g., Hunt *et al.* 1998, Anderson *et al.* 2001). These data may assist in determining whether there is bioavailability of specific sediment-associated compounds in the water body. However, data should be carefully interpreted, with attention to the questions and considerations discussed in the remainder of this section (Table 3.12).

3.3.2. How should test results be interpreted statistically?

Table 3.13 compares two statistical test types for evaluating bioavailability: regression analysis and a t-test. For both test approaches, paired samples of contaminant concentrations in sediment and biota are needed. For the regression model, paired samples would be collected from a number of locations throughout the water body, varying in sediment contaminant concentrations. Then, regression analysis (Draper and Smith 1998) would be performed, comparing concentrations in sediment vs. concentrations in tissue, to determine whether there was a significant slope. One issue with the regression analysis approach is that it would require a high sample size, resulting in increased costs. Power analyses described in Section 3.3.6 indicate that the sample size should be at least 18 to be confident of identifying a relationship using the regression approach with typical bioaccumulation data sets.

For the t-test, samples would be collected from several representative locations and statistically compared to control samples. The control samples would be either sediments from locations outside of the water body that are known to have very low contaminant concentrations or laboratory control sediments that are uncontaminated. A simple t-test for independent samples would then be conducted comparing the tissue samples from the water body to the control tissue samples. If the results of the analysis were significant, the contaminant would be considered to be biologically available.

Table 3.13. Comparison of alternative statistical evaluation approaches for bioaccumulation LOE.

	Regression analysis	**t-test**
Scale of evaluation	Samples collected throughout entire water body	Samples collected throughout entire water body or in highly contaminated area
Control sample	Not required	Control samples collected from uncontaminated location or uncontaminated laboratory test sediments
Minimum sample size	18 samples among different locations in water body	5 samples from within water body and at least 3 control samples
Sampling design	Must sample across a contamination gradient	Water body samples from random locations within water body or in location known to have high contamination

If there are substantial funding constraints, but local data are needed, a qualitative approach could be employed. In this approach, the biota tissue chemistry results would simply be assessed for any incidence of detectable pollutant residues. If any paired samples indicated detectable residues in both sediments and tissues, bioavailability would be considered to be present. This treats the bioavailability LOE as a simple check whether pollutants in sediments are biologically available to any extent. Clearly, this is an oversimplification, as the rate of bioavailability varies among contaminants, sediments, and tissues (Pruell *et al.* 1993, Boese *et al.* 1995, Boese *et al.* 1997, Kraaij *et al.* 2002, Battelle *et al.* 2005). Nevertheless, such an approach would reduce effort and expense.

3.3.3. Should field-caught organisms or laboratory test studies be employed?

There are relative advantages and disadvantages of using field caught organisms vs. laboratory test organisms for the bioavailability line of evidence (Table 3.14). Using field organisms, it can be difficult to obtain sufficient tissue sample mass. Another challenge is finding comparable organisms throughout the water body (Table 3.14). Therefore, scientists familiar with the benthic fauna and collection techniques in the water body should be consulted.

With laboratory bioaccumulation tests, concerns are frequently raised regarding extrapolations between field and laboratory conditions (e.g., equilibrium may not be achieved over the test time frame) (Boese *et al.* 1997, Moore *et al.* 2005). Due to this fact, laboratory test organisms are not reliable for developing bioaccumulation rate parameters for the sediment chemistry LOE. In particular, bioaccumulation rate could be significantly underestimated using laboratory test organisms.

Table 3.14. Comparison of field vs. laboratory test organisms for bioaccumulation test LOE.

	Laboratory test organisms	**Field caught organisms**
Causality	Possible to clearly establish that sediments are causing contaminant uptake	May be possible that contaminants are derived from other sources (e.g., the water column)
Disadvantages	Costly to apply	Difficult to obtain sufficient tissue mass. Difficult to obtain comparable organisms throughout water body
Applicability to other LOE	Not applicable due to differences between laboratory and field caught organisms	May be appropriate as prey tissue line of evidence for predators that consume invertebrates. May assist in development of mechanistic model
Prior knowledge	Substantial experience from Dredge Materials Testing Programs in California and this report	Prior application limited and inconsistent. Filter feeding bivalves used in the State Mussel Watch Program and the San Francisco Bay RMP are not appropriate test organisms.

3.3.4. What are appropriate test organisms?

To be appropriate for bioaccumulation testing, a test organism should have the following attributes:

- A sediment-associated life history, including sediment dwelling and feeding.
- For field organisms, sufficient abundance and distribution to be collected in adequate biomass without unreasonable effort.
- For laboratory organisms, sufficiently developed culturing procedures to allow routine application of 28 day exposure tests.
- Positive demonstration of a tendency to bioaccumulate contaminants at detectable concentrations.

Benthic invertebrates are most appropriate for measuring bioavailability from sediments. Sediment contaminant bioaccumulation tests are usually performed using benthic invertebrates, with preference to those that ingest sediments and where bedded sediment is the primary route of exposure. Polychaetes and bivalves are commonly selected for such studies, as they often possess the appropriate life histories to match these criteria (Lee *et al.* 1993, Lee 1998).

In many cases, historically used bioaccumulation test organisms are not appropriate for the assessment framework because they are water column filter feeders, and therefore do not have a strong dietary association with the sediment. For example, the State Mussel Watch Program (Stephenson *et al.* 1995) and the Regional Monitoring Program For Water Quality in San Francisco Bay (Gunther *et al.* 1999) sample resident and

transplanted filter-feeding bivalves, which are more appropriate to determine water-column contaminant bioavailability, rather than sediment contaminant bioavailability.

The bent-nosed clam (*Macoma nasuta*) is well suited for evaluating the potential for bioaccumulation in estuary waters. *M. nasuta* burrows in and ingests sediments, and is therefore a good indicator of bioavailable sediment-associated contaminants. *Macoma spp.* have been recommended previously for bioaccumulation evaluations based on known tolerance, exposure history, and data availability (e.g., Lee *et al.* 1993, U. S. EPA 2000a). *M. nasuta* has also been used extensively in laboratory bioaccumulation experiments due to life-history factors that cause high sediment exposure (Pruell *et al.* 1993, Boese *et al.* 1997, U. S. EPA and U. S. Army Corps of Engineers 1998, MEC Analytical Systems 2003, Werner *et al.* 2004).

Macoma nasuta is the only test organism with California data demonstrating that it would be an appropriate bioaccumulation test organisms for California estuary waters. The majority of *M. nasuta* samples analyzed in 28 day laboratory bioaccumulation tests had detectable residues of the target organochlorine compound (Table 3.15). There is also good evidence of a statistical association between *M. nasuta* and sediment contaminant concentrations for trace organic contaminants. Table 3.16 presents results of regression analyses pooling data from multiple studies by water body. All studies exhibited statistically significant relationships ($p < 0.05$), and amount of variation explained was often relatively high (Table 3.16). For example, a strong positive relationship was observed between *M. nasuta* and sediment concentrations for total chlordanes in San Francisco Bay (Figure 3.4).

Based on test results with legacy organochlorine pollutants, the polychaete species, *Neanthes virens* and *Nephtys caecoides* do not appear to be suitable for evaluating the bioaccumulation LOE. The sample sizes and proportion of detectable concentrations were relatively low in these species (Table 3.15). The large proportion of below detection samples may prevent confirmation of bioavailability. Similarly, Pruell *et al.* (1993) indicated that *Neanthes virens* has relatively low bioaccumulation rates for PCBs and dioxins, compared to *Macoma nasuta*. Pruell *et al.* (1993) also showed evidence of biotransformation of some organochlorines by *Neanthes virens*, as well as very long times (70 to 120 days) required for these test organisms to reach steady state concentrations.

As additional information becomes available, other species may be identified that are appropriate to evaluate bioaccumulation in a laboratory setting. These may include local species, or laboratory test species not considered in this report.

Table 3.15. Frequency of detection among laboratory bioaccumulation species. All samples were analyzed in 28 day laboratory bioaccumulation tests performed with California sediments. Test results were compiled from the CASQO database.

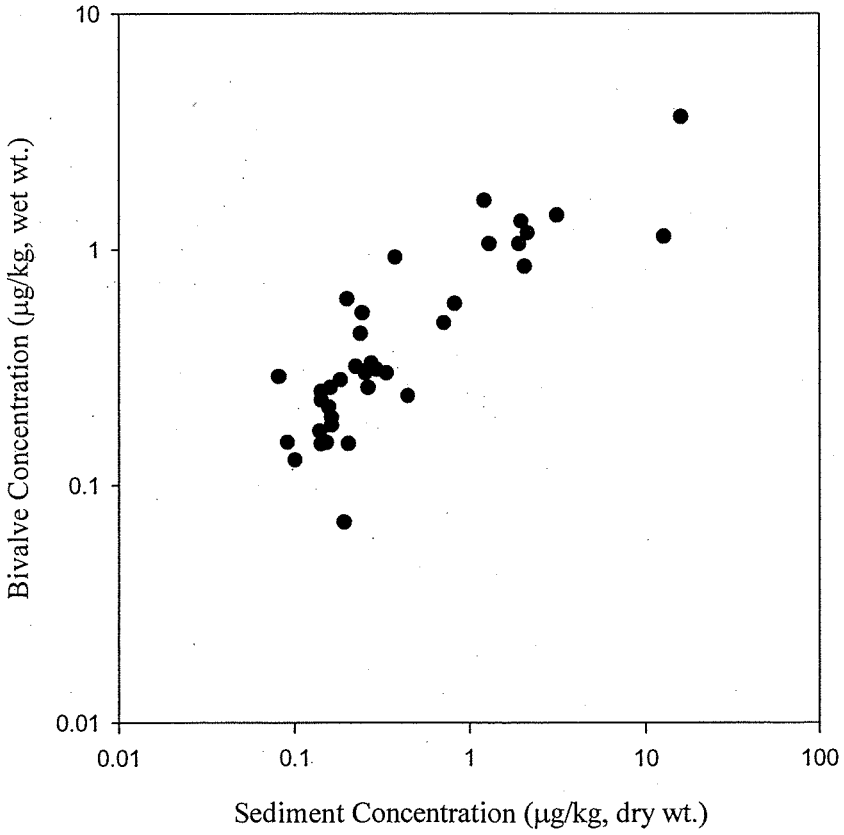
Species	Contaminant	Number of samples	Frequency of detection	Sample type
Macoma nasuta	p,p'-DDE	178	71%	All samples [a]
Macoma nasuta	p,p'-DDE	106	80%	Matched to sediment [b]
Macoma nasuta	alpha-Chlordane	112	50%	All samples
Macoma nasuta	alpha-Chlordane	68	50%	Matched to sediment
Macoma nasuta	Dieldrin	178	39%	All samples
Macoma nasuta	Dieldrin	105	40%	Matched to sediment
Macoma nasuta	PCB 118	67	100%	All samples
Macoma nasuta	PCB 118	39	100%	Matched to sediment
Macoma nasuta	Weighted average [c]		61%	
Neanthes virens	p,p'-DDE	53	58%	All samples
Neanthes virens	p,p'-DDE	27	100%	Matched to sediment
Neanthes virens	alpha-Chlordane	39	0%	All samples
Neanthes virens	alpha-Chlordane	24	0%	Matched to sediment
Neanthes virens	Dieldrin	65	8%	All samples
Neanthes virens	Dieldrin	40	12%	Matched to sediment
Neanthes virens	PCB 118	0		All samples
Neanthes virens	PCB 118	0		Matched to sediment
Neanthes virens	Weighted average [c]		27%	
Nephtys caecoides	p,p'-DDE	40	65%	All samples
Nephtys caecoides	p,p'-DDE	25	76%	Matched to sediment
Nephtys caecoides	alpha-Chlordane	9	0%	All samples
Nephtys caecoides	alpha-Chlordane	7	0%	Matched to sediment
Nephtys caecoides	Dieldrin	40	22%	All samples
Nephtys caecoides	Dieldrin	26	23%	Matched to sediment
Nephtys caecoides	PCB 118	3	33%	All samples
Nephtys caecoides	PCB 118	2	50%	Matched to sediment
Nephtys caecoides	Weighted average [c]		41%	

a. All samples: indicates the combination of all available data in California.
b. Matched to sediment: indicates data where sediment pollutant data were collected at the same location as laboratory test organisms.
c. Weighted average: average of all results for species, weighted by sample size for each compound.

Table 3.16. Results of linear regression analysis of log-transformed sediment concentrations vs. log-transformed *Macoma nasuta* tissue concentrations for different water body and contaminant combinations. All samples were analyzed in 28 day laboratory bioaccumulation tests performed with California sediments. In all cases, N = number of unique spatial locations sampled both for sediment and *M. nasuta* in a given water body. All slopes were positive. See Appendix N for compounds included in sums.

Water body	Contaminant	N (locations)	R^2	p-value
Newport Bay	p,p'-DDE	11	0.74	< 0.001
San Diego Bay	Total HPAHs	14	0.75	< 0.001
San Diego Bay	Total PCBs	14	0.93	< 0.0001
San Francisco Bay	Total chlordanes	37	0.74	< 0.0001
San Francisco Bay	Dieldrin	38	0.49	< 0.0001
San Francisco Bay	Total DDTs	38	0.42	< 0.0001
San Francisco Bay	Total HPAHs	39	0.15	0.014
San Francisco Bay	Total PCBs	37	0.72	< 0.0001

Figure 3.4. Scatter plot total of chlordanes in San Francisco Bay sediments vs. *Macoma nasuta* exposed to those sediments in 28 day laboratory tests. Note log scale.



3.3.5. What factors should be considered in sampling design?

The ability to detect a correlation between sediment and biota concentrations will increase with an increase in the range of measured concentrations. To achieve this, sampling locations may be selected to take advantage of spatial contamination gradients that exist in the water body. For example, samples could be targeted in proximity to contaminant sources, and in less contaminated locations. Depending on the contaminant, sources may include populated areas, industrial areas, and/or large freshwater inlets. Less contaminated locations may include estuary outlets, deep channels, areas with strong tidal mixing, or other locations expected to have a high degree of sediment interaction with uncontaminated waters. If currently available data suggest that sediment contamination is relatively uniform across the water body, it would be difficult to evaluate bioavailability across a contamination gradient. In these circumstances, the t-test approach may be more appropriate than regression analysis (see also Section 3.3.2).

To ensure sufficient sensitivity to detect contaminant bioavailability, laboratory tests should be at least 28 days in duration. For example, Boese *et al.* (1997) indicate a clear time dependence of bioaccumulation, and guidance documents recommend exposure durations of at least 28 days (U. S. EPA and U. S. Army Corps of Engineers 1998, U. S. EPA 2000a).

3.3.6. What sample sizes are needed?

If the regression analysis approach is employed (Section 3.3.2), sample size must be sufficient to detect a positive relationship between sediments and biota, should such a relationship exist. Power analysis may be used to determine sample size required to detect a linear trend for a given statistical power, R^2, and p-value. Sample size required to detect trend varies widely as a function of correlation coefficient between two factors (Figure 3.5). Evaluations using currently available *Macoma* sp. laboratory tests have indicated that strength of relationships (R^2) between *Macoma* and sediment results varied by system and contaminant. However, R^2 was above 0.49 for six of eight regression analyses conducted with DDTs, PCBs, chlordanes and PAHs (Table 3.16). At least 18 samples would be required to achieve a 90% probability of determining a significant trend ($p < 0.05$) when the regression R^2 is 0.5 (Figure 3.5).

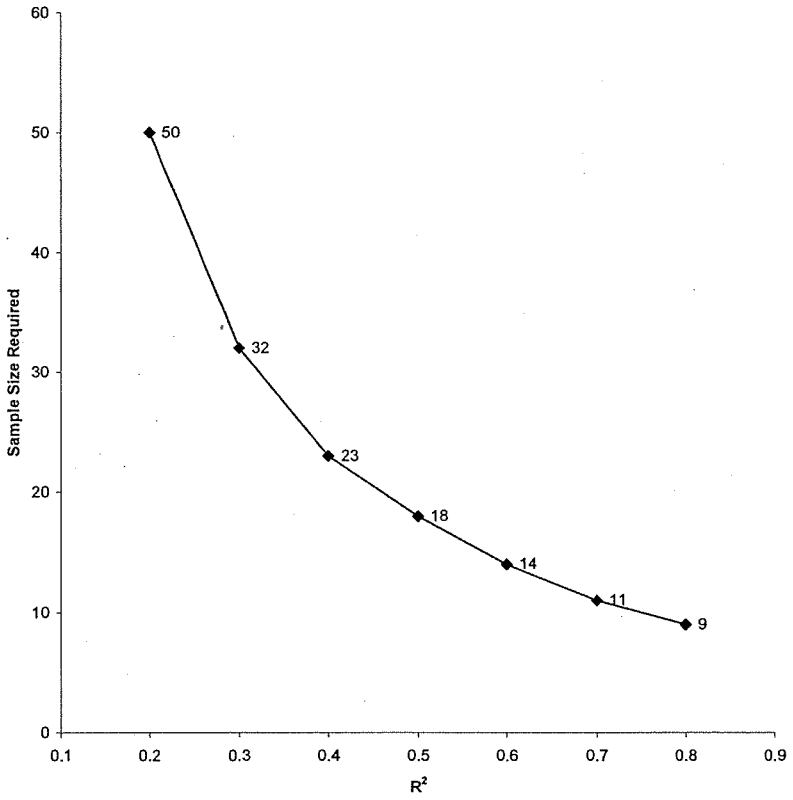
3.3.7. What detection limits are needed?

In the Newport Bay case study (Section 4), detection limits for available bioaccumulation test data[24] were too high to detect residues of PCBs or chlordanes despite the presence of these compounds in field caught organisms. Both case studies included low sediment thresholds of approximately 1-2 ng/g. Assuming chemical equilibrium between sediments and benthic organisms is achieved (McFarland 1998, Zeng and Tran 2002, Moore *et al.* 2005), bioaccumulation test detection limits should be similar. To be safe, it would be appropriate to use detection limits at or below one ng/g to determine bioavailability at relevant scales. Even lower detection limits are desirable given that

[24] Data from MEC Analytical Systems (2003)

laboratory tests may not reach steady state conditions in biota exposed to natural sediments (Boese *et al.* 1997, U. S. EPA and U. S. Army Corps of Engineers 1998, U. S. EPA 2000a).

Figure 3.5. Sample size required to achieve a 90% probability of statistical significance ($p < 0.05$) for varying R^2 in evaluations of correlations between two continuous variables. Results were calculated in SYSTAT 11.



3.3.8. What ancillary parameters should be measured?

In addition to chemical concentration in sediments and organisms, certain ancillary parameters may help in understanding bioavailability. For organic compounds, tissue percent lipid, and sediment percent organic carbon would be useful. As discussed in the Newport Bay case study (Section 4) and elsewhere (e.g., Clark *et al.* 1988, Di Toro *et al.* 1991), organic contaminants tend to be associated with lipid and organic carbon, and variability in contaminant concentration estimates may often be reduced by normalizing for these variables. Percent lipid is sensitive to analysis method and extraction solvent. Consistency across studies would be better achieved by using a gravimetric method for lipid analysis, using dichloromethane as the extraction solvent (Bligh and Dyer 1959, U. S. EPA 2000b).

For laboratory tests, data on mortality, growth rate and body condition of test organisms may be collected and evaluated. In some cases, these parameters may explain unexpected patterns in experimental results, such as variability in bioaccumulation rates (Gunther *et al.* 1999).

4. Application of the assessment framework to evaluate sediments in Newport Bay

4.1. Introduction to case study

This case study describes the application of the assessment framework to PCBs and legacy pesticides in Newport Bay, where a TMDL planning process is underway for these compounds (SARWQCB 2006). The Newport Bay case study includes a review of current data on fish bioaccumulation, and water body biotic and abiotic properties. Data review also includes characterization of contaminant residues in fish and sediments. For the prey tissue line of evidence, tissue thresholds are based on assumptions previously requested by the Regional Water Board responsible for development of the Newport Bay TMDL. The case study also details the approach and rationale for developing specific bioaccumulation rate parameters used to evaluate the sediment chemistry line of evidence.

4.2. Conceptual model for Newport Bay

For Newport Bay, the framework was applied to evaluate bioaccumulation of PCBs, DDTs, and chlordanes. PCBs and legacy pesticides are management concerns in Newport Bay. This is evidenced by 303(d) listing[25] of Upper Newport Bay for these and other compounds and consequent development of a TMDL for Upper and Lower Newport Bay (SWRCB - Santa Ana Region 2006). Following the application framework (Figure 1.1, Section 2.6.1), separate assessments will be conducted on human consumption of sport fish species (e.g., spotted sand bass) and wildlife consumption of forage fish species (e.g., arrow goby and California killifish).

Figure 4.1 presents a simplified food-web conceptual model for Newport Bay. The conceptual model combines literature information on dietary transfer pathways, guidance from local scientists[26], stakeholders, and agency representatives on important human and wildlife receptors[27]. The conceptual model displays the major pathways by which trace organic contaminants are expected to transfer from water and sediments, through the food web to primary producers, invertebrates, fishes, birds, and humans. Primary productivity occurs in the water column (i.e., phytoplankton) and in benthic algae attached to the sediment (Kamer *et al.* 2004a). Primary consumers rely on primary production, as well as organic matter sources resulting from detritus within the sediments. As with all estuarine environments subject to tidal and wind-driven mixing, significant contaminant flux may be expected to occur between the water column and sediments (Davis 2004, Kimmerer 2004). Benthic invertebrates, including crustaceans, mollusks, and polychaetes, feed on the sediments and attached algae. These invertebrates are consumed by multiple resident and transient fish species. In Newport Bay, striped mullet and topsmelt predominantly consume sediment and attached algae (Logothetis *et al.* 2001), and shiner perch augment their epibenthic diet with zooplanktivory (Allen 1980). Many fish species consume

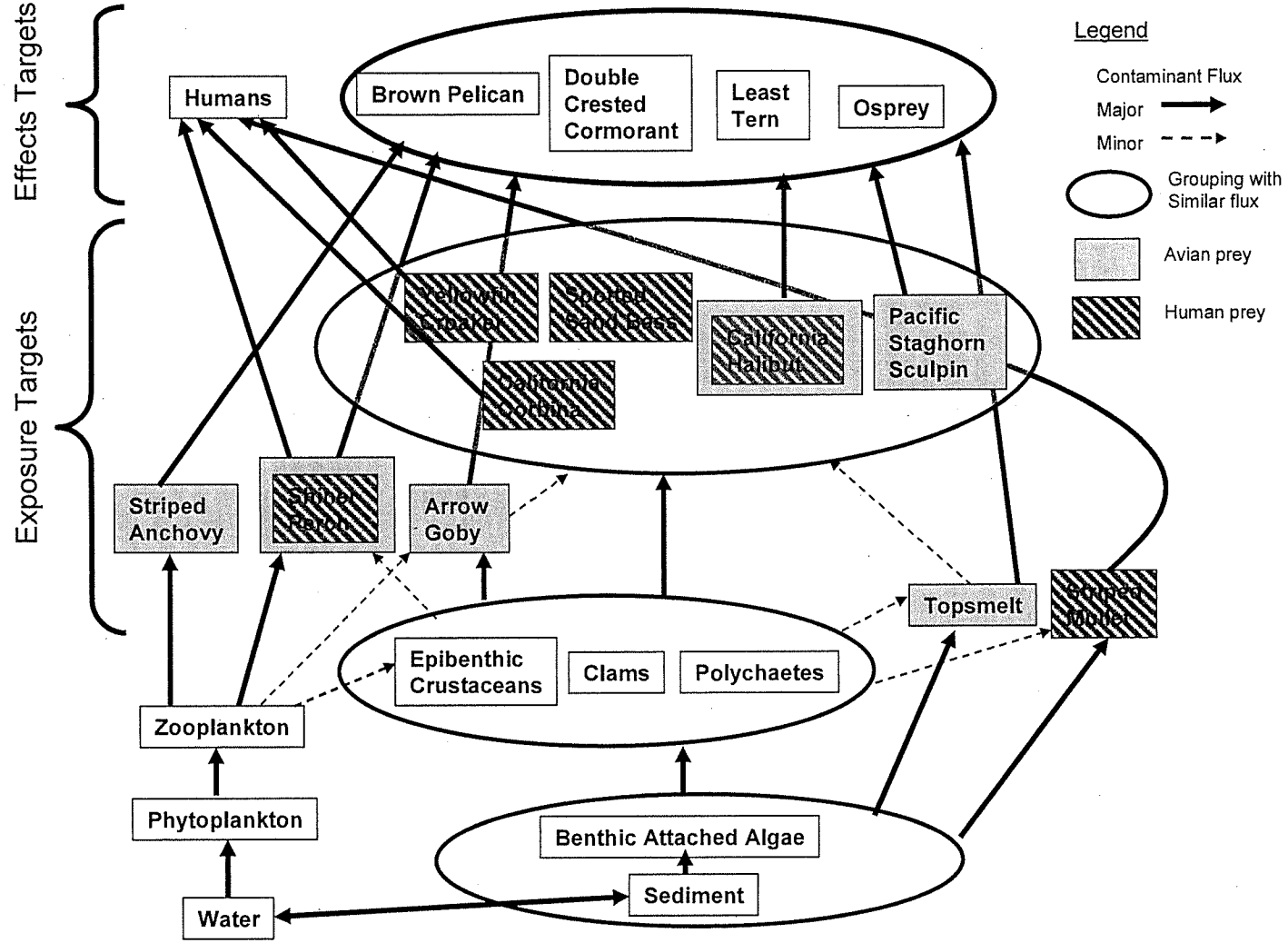
[25] The 303(d) list is a federal listing of water bodies impaired for specific pollutants or pollutant categories.
[26] M. James Allen, SCCWRP and Don Cadien, LACSD, unpublished data and personal communication
[27] Target species were identified by the SQO Bioaccumulation Work Group, the June 22, 2005 Newport Bay TMDL Stakeholders meeting, and SWRQCB – Santa Ana Region staff

mostly invertebrates, with some piscivory on smaller fish, including topsmelt and arrow goby. This information on dietary transfer is used in developing parameters for the mechanistic model on contaminant uptake.

Human sport fishers capture and consume a variety of fish species in Newport Bay, including shiner surfperch, striped mullet, California corbina, spotted sand bass, and yellowfin croaker (Allen *et al.* 2004). Piscivorous birds of concern include brown pelican, least tern, osprey, and double crested cormorant (Terri Reeder, SWRCB – SARWQCB, *Pers. comm.*; Figure 4.1).

This case study focuses on potential effects to wildlife and humans from consuming finfish prey. The case study does not include assessment of exposure due to humans and wildlife directly consuming contaminated benthic invertebrates. This simplification of the risk evaluation has been made in consideration of time constraints. Additionally, data on resident invertebrate tissue concentrations of PCBs and legacy pesticides are very limited in Newport Bay, making it difficult to determine potential exposure. Those data that are available indicate that invertebrate tissue concentrations are generally substantially lower than concentrations in fish (Allen *et al.* 2004, Sutula *et al.* 2005). Therefore, a more protective assessment of exposure and effects will focus on exposure due to consuming contaminated finfish.

Figure 4.1. Conceptual model of Newport Bay for indirect effects case study. Trophic position generally increases moving up the diagram. Arrows represent contaminant flux (dietary transfer for animals). Circled areas represent groupings to simplify understanding of contaminant flux. When an arrow points to a circle, all organisms within that circle receive that flux.



4.3. Prey tissue line of evidence

The first component of the framework (Figure 2.2) is to evaluate local prey tissue concentrations in order to determine the extent of potential risk posed to wildlife and human receptors. As indicated in the conceptual model (Figure 4.1), the receptors in Newport Bay are humans and several bird species. As discussed previously (Sections 2 and 3.1), tissue concentrations are compared to high and low numeric thresholds, to determine the extent of risk posed to these predators.

4.3.1. Methods

4.3.1.1. Database preparation

The case study for Newport Bay compares contaminant concentrations in fish to calculated threshold values. To evaluate fish from Newport Bay, a tissue database was compiled, including concentrations of total PCBs, total DDTs, total chlordanes, dieldrin, and toxaphene. Appendix N lists individual compounds that are included in each contaminant class. The database also includes ancillary information (*e.g.,* length and type of tissue analyzed). Additionally, the database identifies species preyed upon by humans (i.e., legal-sized sport fish) or wildlife (small fish). The database does not include samples from San Diego Creek and coastal samples collected outside of the Newport Bay entrance channel. The primary data source for the database is a two-year study of fish tissue contamination in multiple recreational and forage fish species (Allen *et al.* 2004). A small amount of additional data were obtained from the Coastal Fish Contamination Program, the Toxic Substances Monitoring Program, and the Southern California Bight Survey (Allen *et al.* 2002a).

In Newport Bay and other California waters, legacy pesticides and PCBs have been shown to decline significantly over time (Greenfield *et al.* 2004, Davis *et al.* 2006b), necessitating use of data only from a recent time horizon. For this reason, data collected prior to 1997 were not included in the fish tissue database.

4.3.1.2. Prey species

Following Section 3.1.4, the prey tissue line of evidence (LOE) focused on sediment-associated fish species that are known to be prey for humans or wildlife. Sediment association was defined by expected behavior (i.e., benthic association), or data on prey items (i.e., diets including infaunal or epifaunal animals). Due to the relatively small size of Newport Bay, and salinity conditions similar to coastal waters, some species captured within the Bay may be transient (i.e., spend a portion of their lives along the coast). In particular, the majority of the species targeted by human sport fishers are expected to be transient (Table 4.1). To evaluate the impact on findings of inclusion vs. exclusion of transient species, the comparison to human health thresholds was conducted in three ways: 1. resident species only, 2. all human prey species (both resident and transient), and 3. all species, including both human and wildlife prey (Table 4.1).

Table 4.1. Sample type, sample size (N) and arithmetic mean concentrations for compounds in Newport Bay fish samples. Resident = species resides within Bay; Transient = species likely to move between Bay and coast. NA = not available. For averaging, values below detection were set at ½ detection limits.

Species	Movement[a]	Tissue type	N	Total DDTs (ng/g wet)	Total PCBs (ng/g wet)	Total chlordanes (ng/g wet)
Human prey						
Brown smoothhound shark	Transient	Fillet	1	28	NA	0.5
CA halibut	R/T[b]	Fillet	4	66	2.5	2.3
Diamond turbot	Resident	Fillet	3	66	2.5	1.8
Orangemouth corvina	Transient	Fillet	1	61	NA	1.9
Round stingray	Transient	Fillet	1	0	NA	0.5
Striped mullet	Transient	Fillet	1	416	NA	37.5
Spotted sand bass	Resident	Fillet	6	90	8.4	5.1
Yellowfin croaker	Transient	Fillet	7	82	13.1	3.0
Barred sand bass	Transient	Fillet	2	56	2.5	2.5
CA corbina	Transient	Fillet	3	361	21.6	16.0
Fantail sole	Transient	Fillet	1	36	2.5	2.5
Spotfin croaker	Transient	Fillet	2	68	2.5	4.5
Spotted turbot	Transient	Fillet	3	30	NA	0.8
All human prey		Fillet	35	101	8.8	5.0
Wildlife prey						
Arrow goby	Resident	Whole	5	141	2.5	5.8
Black perch	Resident	Whole	1	117	2.5	12.6
CA halibut	R/T[b]	Whole	2	85	31.0	6.9
CA killifish	Resident	Whole	3	100	32.5	1.1
Diamond turbot	Resident	Whole	1	119	2.5	6.4
Pacific staghorn sculpin	Resident	Whole	4	143	2.5	14.1
Shiner surfperch	Resident	Whole	2	91	2.5	1.9
Cheekspot goby	Resident	Whole	1	195	53.4	11.0
All wildlife prey		Whole	19	124	12.9	7.2
All samples		All	54	109	10.7	5.7

a. M. J. Allen, SCCWRP, *Pers. comm.* b. California halibut < 20 cm reside in the Bay; halibut > 20 cm are likely to move to the coast (M. J. Allen, *Pers. comm.*).

4.3.1.3. Prey sizes and tissue types

Following Section 3.1.4, body size was considered in selecting appropriate samples for the prey tissue LOE. Review of the sport fish data showed that species that would be consumed by humans were generally greater than 15 cm (total length) (Appendix O, Table O.1). Samples less than 15 cm (total length) were not included as potential human prey species. Although minimum legal size requirements are in some cases higher than 15 cm (California Department of Fish and Game 2006), some undersized fish were included in the analysis due to data limitations. Size for wildlife prey was selected based

on published literature on avian predator prey size (Appendix D). Based on this review, only fish of 10 cm total length or less were included in evaluations of risk to wildlife predators. Fish tissue samples evaluated for human health risk were analyzed as skin-off fillets. Samples evaluated for wildlife risk were analyzed as whole body samples (Table 4.1, Appendix O).

4.3.1.4. Calculation of tissue concentrations for comparison to the low and high tissue thresholds

As discussed previously, (Section 3.1.9), the framework accounts for uncertainty in the findings by comparing two thresholds to estimates of the average tissue concentration. Following guidance in Section 3.1.9, a more conservative estimate of the average was used for comparison to the low threshold. Specifically, the mean concentration was compared to the higher (less conservative) tissue threshold and the 95% upper confidence limit of the mean concentration was compared to the lower (more conservative) tissue threshold (Table 4.2). For the Newport Bay prey tissue LOE, both arithmetic and geometric means and 95% UCIs were developed, to determine the impact of averaging method (see Appendix G). For data below detection, ½ the detection limits were used, as reported in Allen *et al.* (2004).

4.3.1.5. *Development and calculation of low and high tissue thresholds*

The assessment framework requires calculation of two risk-based effects thresholds. Specific policy judgments need to be made in developing these thresholds. These include factors such as the allowable level of risk (e.g., cancer risk factor) and the target population to be protected (e.g., human consumption rate or target wildlife; see Sections 3.1.10 and 3.1.13). To evaluate the impact of potential policy decisions on the results of this case study, two sets of human health thresholds were evaluated. The first set of thresholds was established based on feedback from SARWQCB. The second set was established based on prior recommendations by the California Office of Environmental Health Hazard Assessment (OEHHA).

Table 4.2. Description of three categories for prey tissue line of evidence for human and wildlife receptors.

Fish tissue score	General description	Humans[a]	Humans	Wildlife	Wildlife
		Threshold	Interpretation	Threshold	Interpretation
▲ Low exposure	95% upper confidence limit of average fish tissue concentration is below the low toxicity threshold for humans or wildlife	Fish tissue threshold based on protecting the 95 percentile of sport fish consumers such that no more than 1 in 100,000 faces an increased cancer risk	Using a conservative set of assumptions, dietary exposure to fish in water body poses a low risk to fishers who consume their catch and to the general public	Fish tissue threshold based on toxicity reference values representing no-effects levels (TRV-Low) to wildlife (Appendix H)	Using a conservative set of assumptions, dietary exposure is unlikely to pose a risk to the most sensitive wildlife endpoint.
◘ Moderate exposure	Tissue concentration is intermediate between low and high threshold	Both thresholds are relevant	Dietary exposure to fish in a water body may pose a risk to fishers who consume their catch	Both thresholds are relevant	Dietary exposure may pose a risk to the most sensitive wildlife endpoint
● High exposure	Average fish tissue concentration is above the high toxicity threshold for humans or wildlife	Fish tissue threshold based on protecting the average sport fish consumer such that no more than 1 in 100,000 faces an increased cancer risk	Dietary exposure to fish in a water body is likely to pose 1 in 100,000 increased cancer risk for sport fish consumers	Fish tissue threshold based on toxicity reference values representing mid-range adverse effects levels (TRV-High) to wildlife (Appendix H)	Dietary exposure is likely to pose a risk to the most sensitive wildlife endpoint

a. For the Newport Bay case study, two sets of human health effects thresholds are considered including this one, and the threshold resulting from assumptions in Appendix P.

Staff of theSanta Ana Regional Water Quality Control Board provided written guidance regarding the risk threshold calculations in the human health effects case study (Appendix P):

1. The low tissue threshold would be developed based on 10^{-6} rate of increased cancer risk.
2. The high tissue threshold would be developed based on 10^{-5} rate of increased cancer risk.
3. The consumption rate would be estimated based on the Santa Monica Bay Seafood Consumption Study (Allen *et al.* 1996). The median consumption rate from this study is 21 g/d.

Use of these assumptions resulted in low and high thresholds that differ from each other by a factor of ten (Table 4.3).

Table 4.3. Low and high thresholds for organochlorine contaminants in fish tissue, for application of the framework to evaluate human health effects in Newport Bay. Thresholds were calculated using methods in Sections 3.1.10, 3.1.11, and 3.1.13 of this report. All thresholds are in ng/g wet tissue mass.

Source	Threshold	DDT	PCB	Chlordane	Dieldrin
SARWQCB	Low human	9.8	1.7	9.5	0.2
Table 4.2 and Appendix K	Low human	64	11	63	1.4
SARWQCB	High human	98	17	95	2.1
Table 4.2 and Appendix K	High human	118	20	114	2.5

The second set of thresholds in Table 4.3 follows the categories outlined in Table 4.2. These thresholds varied consumption rate rather than allowable cancer risk factor. For this set, the low and high thresholds both incorporated a 10^{-5} increased cancer risk, following U. S. EPA guidance for tissue screening values (U. S. EPA 2000b). The low and high thresholds were distinguished by a varying fish intake rate, following OEHHA recommendations to calculate a threshold based on average intake and a threshold based on upper percentile intake rate from the fish consuming population (OEHHA 2001). The average intake rate was set at the U. S. EPA recommended consumption rate of 17.5 g/d for the general adult population and recreational fishers (U. S. EPA 2000b). The upper percentile consumption rate was set at 32 g/d, which is the 95^{th} percentile consumption rate of all sport fish consumers surveyed in the San Francisco Bay Seafood Consumption Study (SFEI 2000). Use of 10^{-5} or 10^{-4} risk factors for the low and high SARWQCB thresholds, respectively, would increase the values of these thresholds by a factor of ten. Additional information on derivation of these thresholds is available in Appendix K.

For the wildlife effects evaluation, the Regional Board did not make specific requests. These thresholds were calculated using the toxicity reference values and assumptions in Appendix H (Table H.1). Following Table 3.5, body mass estimates for large birds were used for brown pelicans, osprey, and double-crested cormorants. Estimates for small birds were used for least tern. Appendix Table J.1 presents appropriate thresholds for dry

weight prey tissue concentrations, but Newport Bay studies generally provide contaminant concentrations in wet weight (e.g., Allen *et al.* 2002a, 2004). Table 4.4 presents the thresholds converted to wet weight concentrations assuming 80% moisture in prey fish tissue (Jarvinen and Ankley 1999, Meador *et al.* 2002b)[28].

Table 4.4. Low and high prey tissue thresholds for application of the framework to evaluate effects to birds in Newport Bay. Ingestion rates are calculated following Table 3.5 and Nagy (2001). Tissue thresholds are calculated following Section 3.1.10, using toxicity reference values from Appendix H, and converting prey tissue from dry to wet weight, assuming 80% prey tissue moisture.

Species	Body mass (kg)	Daily ingestion rate (kg dw food/day)	Ingestion rate (kg dw food/ (kg body weight*day))	DDT low (ppb ww fish tissue)	DDT high (ppb wet weight)	PCB low (ppb wet weight)	PCB high (ppb wet weight)	Chlordane low (ppb ww)	Chlordane high (ppb ww)	Dieldrin low (ppb ww)	Dieldrin high (ppb ww)
Brown pelican	3.20[a]	0.178	0.056	32	2,156	324	4,562	502	25,142	255	3,772
Double crested cormorant	2.45[b]	0.149	0.061	30	1,966	296	4,164	458	22,948	232	3,442
Least tern	0.045[c]	0.011	0.239	8	502	76	1,062	116	5,848	59	878
Osprey	1.5[a]	0.108	0.072	25	1,664	249	3,520	388	19,402	197	2,910

a. Dunning (1993). b. Hatch and Weseloh (1999). c. Thompson *et al.* (1997), Zeeman (2004)

Many assumptions were made in application of the prey tissue LOE for Newport Bay. These assumptions and their likely impacts are summarized in Table 4.5.

4.3.2. Results

4.3.2.1. DDTs

In general, results indicated that total DDTs were intermediate between the low and high prey tissue thresholds. This finding was consistent among both human sport fish consumers and piscivorous birds (Table 4.6). For the high effects threshold comparison, both arithmetic and geometric average concentrations were below the threshold for humans (98 ng/g wet weight; Table 4.3) and birds (DDT high threshold ranging from 502 to 2156; Table 4.4). For the low threshold comparison, arithmetic and geometric 95% UCI concentrations were above the low effects thresholds for humans (DDT low threshold = 9.8 ng/g; Table 4.3) and birds (DDT low thresholds ranging from 8 to 32; Table 4.4).

[28] Wet weight thresholds = dry weight thresholds*(0.20)

Table 4.5. Summary of assumptions, their basis, and effects in prey tissue LOE evaluation for the Newport Bay case study.

Decision or assumption	Basis	Effect
Evaluate two alternative groups of species for prey tissue line of evidence, human health evaluation: 1. all routinely monitored species, and 2. only species with sediment association and permanent Bay residence	Evaluate sensitivity of assessment results to sport fish species selection	Slight effect on calculated tissue chemistry residues. No effect on comparison to thresholds
Pooling fish tissue concentrations for multiple prey species	Limited sample size for looking at individual species. Conceptual model of exposure risk and available data indicate that receptors (humans/wildlife) consume multiple prey	Simplifies tissue concentration calculation. Not examining exposure separately for individual prey species
Only including samples with sizes likely to be consumed by receptor. < 10 cm for wildlife; > 15 cm for humans	Not appropriate to use surrogate prey species unlikely to be consumed	No effect for human health evaluation since program targets legal sized fish species. Possible reduction in sample size and calculated tissue concentration residues for wildlife evaluation
Focusing on finfish prey, rather than shellfish	Use of finfish prey likely to be more protective for main target contaminants of concern (legacy pesticides and PCBs). Limited data availability for prey shellfish. Time and budgetary constraints preclude evaluating all possible prey	Uncertain
For human health evaluation, fish consumption rate based on sport fish consumers	Santa Ana Regional Board staff requested that thresholds be protective of recreational anglers (following Allen *et al.* 1996).	Thresholds are higher than if a subsistence fisher consumption rate were used, slightly lower if a general population consumption rate were used.
Carcinogen risk thresholds based on 10^{-5} allowable increased risk for high threshold and 10^{-6} allowable increased risk for low threshold.	Santa Ana Regional Board staff specifically requested these risk assumptions for development of low and high tissue thresholds (Appendix P).	Low threshold is 10-fold lower than if 10^{-5} carcinogen risk were used.
Calculate tissue chemistry average based on arithmetic mean	Arithmetic averages better reflect the average overall exposure due to consuming multiple fish	Risk determination generally the same as when geometric average is used
Calculate tissue chemistry 95% UCI based on arithmetic estimate of standard error, rather than standard deviation	Use of standard error better reflects the average overall exposure due to consuming multiple fish	95% UCI estimated exposure less conservative than if standard deviation were used
Only using samples prepared according to receptors' consumption patterns	Humans generally do not consume whole fish, and wildlife predators do. Contaminant concentration estimates will be related to tissue preparation	Sample size unchanged because most samples were prepared appropriately for target predator. Preparation method may cause increase or decrease in estimated tissue contaminant concentration

The finding of total DDTs intermediate between the two thresholds was robust to a number of assumptions in the averaging and threshold calculation method (Table 4.6). For the wildlife evaluation, concentrations were greater than the low threshold, regardless of averaging method or probability assumption (Table 4.6). Average concentrations were below the high threshold for all calculated high thresholds (as presented in Table 4.4) and averaging methods (Table 4.6). For the human evaluation, 95% UCI concentrations were greater than the low threshold regardless of whether the threshold was based on policy

assumptions requested by the Regional Board (i.e., 9.8 ng/g; Table 4.3, Appendix P) or assumptions described in Appendix K (Table K.1). Tissue concentrations were also greater than the 9.8 ng/g low thresholds, regardless of the averaging method (arithmetic vs. geometric), probability assumption (average vs. 95% UCI of the average), or data set (resident fish only vs. all locally captured sport fish). When all human prey data were combined, the arithmetic average concentration (112 ng/g ww) was greater than the 98 ng/g high effects threshold.

Table 4.6. Estimated total DDTs in Newport Bay fish samples calculated using different averaging methods and different sample inclusion criteria. Values highlighted in grey exceed the relevant threshold. N = sample size. 95% UCI = average + two standard errors. Geo mean = geometric mean (calculated on log scale). All values except sample size are ng/g wet weight.

DDT results[a]	Resident wildlife prey[b]	Relevant threshold[b]	Resident human prey[c]	All human prey[c]	All[b]	Relevant threshold
N	23		6	21	78	
Mean	122	High (>502)	62	112[d]	94	High (98)
95% UCI	144	Low (8 – 32)	92	164	112	Low (9.8)
Geo mean	111	High (>502)	50	71	68	High (98)
Geo 95% UCI	134	Low (8 – 32)	94	108	82	Low (9.8)
Median	116		63	68	67	

a. Values are different from Table 4.1 because Table 4.6 excludes samples collected prior to 1997. b. Compared to bird thresholds presented in Table 4.4. c. Compared to human thresholds presented in Table 4.3. d. Exceeds 98 ng/g but not alternate high threshold from Table 4.3 (118 ng/g).

Based on these results and the use of the framework described in Section 2 (see Table 4.2), consumption of resident sediment-associated fish may pose a possible risk due to DDT exposure. This risk may occur for human sport fish consumers and also predatory birds in Newport Bay. Following the assessment framework (Table 2.1), evaluation of the additional lines of evidence (sediment chemistry and bioaccumulation tests) is warranted.

4.3.2.2. PCBs

In general, total PCBs were below both effects thresholds for wildlife, and intermediate between the low and high effects thresholds for humans (Table 4.7). Both the arithmetic and geometric 95% UCI estimate of average wildlife prey concentrations were well below the most conservative wildlife effects threshold (76 ppb, for least tern, Table 4.4). In contrast, average concentrations were above the conservative human health thresholds (Table 4.3), regardless of averaging method (arithmetic vs. geometric) or species sampled (all human prey species vs. resident species only) (Table 4.7).

For human sport fish consumers, assumptions made in threshold calculation affected whether thresholds would be exceeded. If the PCB lower threshold described in

Appendix K (11 ng/g) had been used instead of the threshold requested by the SARWQCB (1.7 ng/d), this threshold would not be exceeded by the arithmetic mean, geometric mean, or geometric 95% UCI estimate of concentration. However, the 11 ng/g threshold would still be exceeded by the arithmetic 95% UCI concentration estimate (14.6 ng/g for resident human prey; Table 4.7).

The PCB average concentrations demonstrate a statistical artifact for data having skewed distributions; specifically, geometric average estimates are generally lower than arithmetic average estimates (Table 4.7). The arithmetic average is appropriate for categorizing prey tissue results within the assessment framework (see Appendix G). This is because it accounts for the potential for relatively high exposure due to occasional consumption of highly elevated samples (i.e., outliers) (Helsel and Hirsch 2002). The relatively large difference between average and 95% UCI estimates for PCBs (Table 4.7) results from the small sample sizes and skewed distributions of these data, with many samples below detection limits (Allen *et al.* 2004). This is a concern given that in this case study, detection limits (5 ng/g for each individual PCB congener) (Allen *et al.* 2004) are relatively close to and sometimes even exceed some of the human health effects thresholds (Table 4.3). The issue of treatment of samples below detection limits will be evaluated further in the sediment chemistry line of evidence evaluation.

Table 4.7. Estimated total PCBs in Newport Bay fish samples calculated using different averaging methods and different sample inclusion criteria. Values highlighted in grey exceed the relevant threshold. N = sample size. 95% UCI = average + two standard errors. Geo mean = geometric mean. All values except sample size are ng/g wet weight. Note that below detection were set to ½ the detection limit.

PCB results[a]	Resident wildlife prey[b]	Relevant threshold	Resident human prey[c]	All human prey[c]	All	Relevant threshold
N	23		6	18	78	
Mean	12.7	High (>1062)	7.4	7.7	12.9	High (17)
95% UCI	23.0	Low (76 - 324)	14.6	14.2	18.9	Low (1.7)
Geo mean	4.3	High (>1062)	4.6	4.1	0.7	High (17)
Geo 95% UCI	7.2	Low (76 - 324)	10.4[d]	6.3[d]	6.7[d]	Low (1.7)
Median	2.5		2.5	2.5	2.5	

a. Values are different from Table 4.1 because Table 4.7 excludes samples collected prior to 1997. b. Compared to bird thresholds presented in Table 4.4. c. Compared to human thresholds presented in Table 4.3. d. Exceeds 1.7 ng/g but not alternate low threshold from Table 4.3 (11 ng/g).

4.3.2.3. Chlordanes

Results for total chlordanes indicated concentrations below both relevant thresholds for wildlife and human health (Table 4.8). This finding was consistent regardless of averaging method, sample selection method, or use of locally requested thresholds vs. thresholds from Appendix K (see Table 4.3). Based on the results of the prey tissue line of evidence, using the assessment framework presented in Section 2 (e.g., Table 2.1 and

Figure 2.2), risk to humans and wildlife due to sediment exposure is unlikely. Specifically, fish tissue chemistry indicates no evidence of risk to human or wildlife consumers above the defined level of concern. Therefore, following the assessment framework, it is not necessary to proceed to the other lines of evidence for chlordanes. In general, chlordanes have declined significantly in California waters over the past two decades (Greenfield *et al.* 2004, Greenfield *et al.* 2005, Davis *et al.* 2006b). This is likely a result of biodegradation and other chemical properties leading to relatively rapid ecosystem loss of chlordanes (Leatherbarrow *et al.* 2006).

Table 4.8. Estimated total chlordanes in Newport Bay fish samples calculated using different averaging methods and different sample inclusion criteria. No values exceed the relevant threshold. N = sample size. 95 % UCI = average + two standard errors. Geo mean = geometric mean (calculated on log scale). All values except sample size are ng/g wet weight. Values below detection were set to ½ the detection limit.

Chlordane results[a]	Resident wildlife prey[b]	Relevant threshold	Resident human prey[c]	All human prey[c]	All	Relevant threshold
N	23		6	21	78	
Mean	6.0	High (>5848)	3.5	4.9	4.4	High (95)
95% UCI	8.5	Low (116 - 502)	4.7	7.2	5.5	Low (9.5)
Geo mean	3.5	High (>5848)	3.2	3.2	3.0	High (95)
Geo 95% UCI	5.7	Low (116 - 502)	4.4	4.8	3.6	Low (9.5)
Median	2.5		2.5	2.5	2.5	

a. Values are different from Table 4.1 because Table 4.8 excludes samples collected prior to 1997. b. Compared to bird thresholds presented in Table 4.4. c. Compared to human thresholds presented in Table 4.3.

4.3.2.4. Dieldrin

Evaluations for dieldrin are difficult to interpret because the detection limits of currently available data are above human health thresholds. For the most recent data (Allen *et al.* 2004), detection limits were 5 ng/g and all samples evaluated were below detection. Nevertheless, risk thresholds based on sport fisher consumption rates and 10^{-5} to 10^{-6} allowable cancer risk assumptions range from 0.2 to 2.5 ng/g (Table 4.3, Appendix K). Therefore, it is not possible to determine conclusively whether dieldrin in fish tissue poses a risk to human consumers above the case study thresholds. Since the fish tissue line of evidence is inconclusive, the appropriate course of action would be to proceed to other components of the framework (sediment chemistry and bioaccumulation test organism evaluation).

For wildlife piscivores, both the low and high-risk thresholds are well above the 5 ng/g detection limits, ranging from 59 (least tern low threshold) to 3,772 (brown pelican high threshold) (Table 4.4). Given that all samples were below detection, fish tissue chemistry indicates low risk and it is not necessary to evaluate other lines of evidence for wildlife piscivores in Newport Bay.

4.3.3. Summary of findings for the prey tissue LOE

Fish tissue concentrations were generally intermediate between the low and high effects thresholds for human health for DDTs and PCBs (Tables 4.6 and 4.7), and below both thresholds for chlordanes (Table 4.8). Tissue concentrations were also intermediate between the low and high wildlife thresholds for DDTs (Table 4.6), but were below the low threshold for PCBs and chlordanes (Tables 4.7 and 4.8). As expected, estimated contaminant concentrations in fish in Newport Bay were higher using arithmetic average than geometric average. Nevertheless, use of the arithmetic vs. geometric averaging methods often did not affect results in comparison to thresholds. An exception was PCBs in humans, for which changes in the general combination of assumptions resulted in changes between the low and intermediate categorization.

4.4. Sediment chemistry line of evidence

As described in the assessment framework (Table 2.1 and Figure 2.2), the sediment chemistry line of evidence (LOE) need only be applied for those compounds for which the prey tissue LOE was above the low threshold or inconclusive. For Newport Bay, this includes DDTs (human health and wildlife), PCBs (human health only), and dieldrin (human health results inconclusive). The development of sediment thresholds from tissue thresholds and a bioaccumulation rate parameter is a challenging technical issue. Therefore, much of this section describes approaches for sediment threshold development, followed by comparison of the thresholds to available sediment chemistry data.

4.4.1. Development of empirical bioaccumulation factor

4.4.1.1. Fish tissue data and lipid normalization

A number of criteria were used in selecting appropriate fish tissue samples for bioaccumulation rate parameter[29] development; these included appropriate body size of samples, use of species resident in Newport Bay, and appropriate tissue preparation methods. Table 4.9 summarizes key assumptions and their probable impact on bioaccumulation rate parameter development. Fish sample selection criteria were similar to criteria for the prey tissue line of evidence (Table 4.5). The final data set selected for fish tissue concentration estimation consisted entirely of fish analyzed as part of the survey conducted by Allen *et al.* (2004). The data set contained 18 human prey fish samples and 23 wildlife prey fish samples (Appendix O).

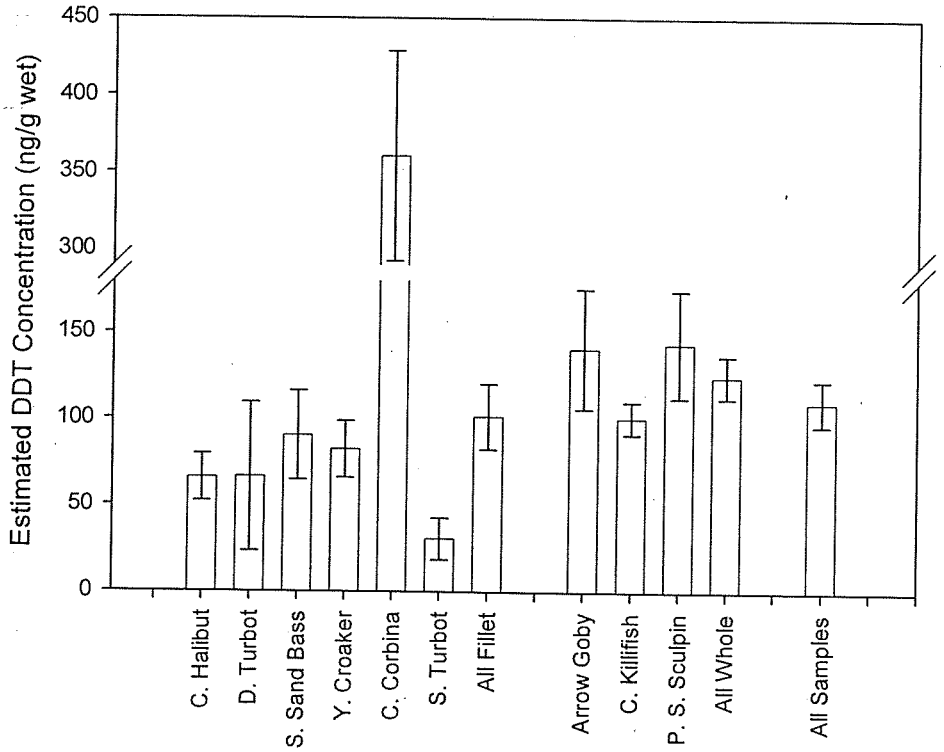
[29] As defined in "abbreviations and definitions"

Table 4.9. Summary of assumptions, their basis, and effects on developing BAF for sediment chemistry LOE, Newport Bay case study.

Decision or assumption	Basis	Effect
Compare results of BAF development based on arithmetic vs. geometric averaging for fish tissue and sediments	Results are generally lognormally distributed, suggesting geometric mean is more appropriate estimate of central tendency. Nevertheless, mechanistic model error estimates are more consistent with arithmetic averaging.	Range of predicted bioaccumulation factors would be wider for geometric averaging.
Lipid normalizing fish tissue concentrations	Lipid data are available and generally correlated with contaminant concentrations.	Will reduce variability of bioaccumulation estimate, by accounting for variation due to lipids. This is particularly true when pooling multiple species due to interspecific variation in lipid content.
Not normalizing sediment concentrations for organic carbon	Although sediment contaminant data are correlated with sediment organic carbon, a large proportion of the sites lack organic carbon data.	Greater variability of bioaccumulation estimate.
Pooling fish tissue concentrations for multiple prey species	Limited sample size for looking at individual species. Conceptual model of exposure risk indicates that receptors (humans/wildlife) consume multiple prey. Variability in average prey concentration estimate is reduced by pooling species.	Reduced variability of bioaccumulation estimate. Simplifies bioaccumulation calculation. Not examining bioaccumulation for individual prey species.
Convert sediment and fish values reported below detection to the detection limits	For PCBs, many fish samples are below detection and should be included in calculations. Model results indicate that actual total congener concentrations in fish may be well above detection. Similar treatment of sediment and fish data will minimize bias in BAF calculation.	Uncertain effect on BAF calculation results.
Develop BAF using only fish samples expected to be permanent residents of Newport Bay	Accounts for concern that fish may have migrated from other locations.	Considerably reduced sample size for human prey sample calculations. Little effect on wildlife prey sample calculations.
Include fish > 15 cm for BAF calculation for humans	Balance between need to use samples representative of human exposure, and need to use available local data.	Makes it possible to develop BAF for human sport fishers using available data.
Include only target sized (< 10 cm) fish for BAF calculation for wildlife	Sufficient samples available within target size range.	Risk evaluations are based on appropriate prey species sizes.
Only accept compounds in sediment for which N > 10	Confidence of average estimate very low when N < 10.	Not possible to calculate empirical bioaccumulation factor for dieldrin, mirex, 20 PCB congeners, and other compounds.
Only accept compounds in sediment for which > 10% of samples are above detection limits	Confidence of average estimate very low when a majority of samples are below detection. Nevertheless, due to relatively high detection limits and limited data, it was necessary to accept samples having a majority below detection.	Not possible to calculate empirical bioaccumulation factor for 7 PCB congeners. Remaining BAFs will be uncertain.
Calculate bioaccumulation factor of sum of compounds, rather than for each individual compound	Individual compounds often lacking in sufficient results above detection (particularly for fish). Pooling of compounds reduces impact of compounds having most results below detection. Effects comparisons are on a summed contaminant basis.	Calculation of average and variation in bioaccumulation factor more straightforward. Not possible to look at dioxin-like PCB effects.
Use of 2 * standard error rather than standard deviation to calculate 95% confidence interval of estimates	The objective is to calculate the central tendency and uncertainty about that central tendency for bioaccumulation in the water body. This is because the population of receptor species (humans and piscivorous birds) are expected, on average, to consume prey across the water body, rather than occurring at only one location.	The calculated uncertainty in estimated bioaccumulation factor will be lower using standard error, rather than standard deviation.
Only using samples prepared according to receptors' consumption patterns	Humans generally do not consume whole fish, and avian predators do. Contaminant concentration estimates will be related to tissue preparation.	Sample size unchanged. Preparation method may cause increase or decrease in estimated tissue contaminant concentration.

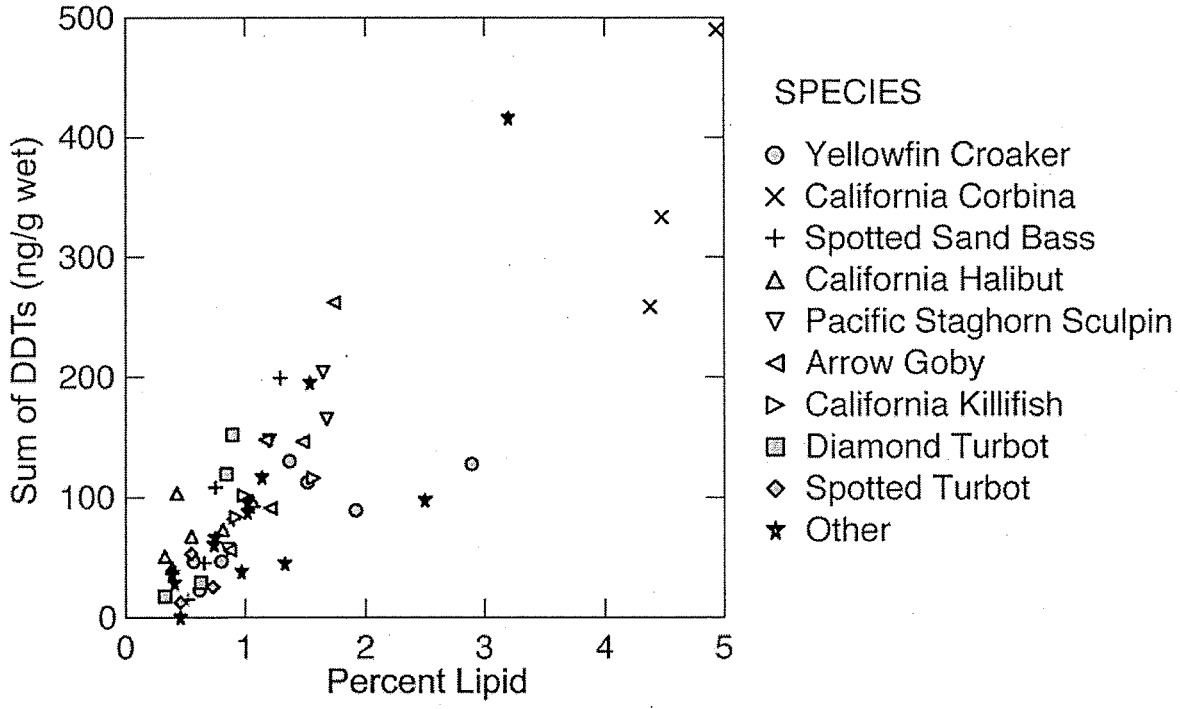
In the case of Newport Bay, the sample size of potential fish species was limited, with individual species ranging in number of samples analyzed from one to seven samples (Table 4.1). The limited sample size interferes with the ability to detect spatial patterns in tissue PCBs or DDTs. Therefore, it is not possible to develop meaningful spatially explicit statistical models of tissue vs. sediment chemistry relationships (i.e., Appendix L). As discussed in Section 3.1.8 and Appendix F, the ability to estimate average concentrations will also be reduced when sample sizes are small. This can be seen in Figure 4.2, which plots the average concentration and standard error of the mean for DDTs in all species in Newport Bay having at least three samples (Table 4.1). Figure 4.2 indicates that several fish species have relatively wide average estimates. In particular, standard errors of the mean are wide for diamond turbot, spotted sand bass, California corbina, arrow goby, and pacific staghorn sculpin. Generally, the standard error of the mean estimates for these species is larger than the standard error taking the combined average of all fillet samples, all whole body samples, or all samples (Figure 4.2). This indicates that, despite differences among species in exposure, the ability to estimate typical predator exposure in Newport Bay will not improve by focusing on specific individual species. Rather, a compilation of multiple species will be more appropriate to estimate expected exposure at a given sediment concentration.

Figure 4.2. Average DDT concentration (ng/g wet weight) by species. Error bars represent one standard error of the mean for each species. All fillet = all samples evaluated as fillet tissue. All whole = all samples evaluated as whole body samples. Note break in y-axis.



Another issue in bioaccumulation rate parameter development is whether tissue lipid and sediment organic carbon normalization are appropriate (Section 3.2.6). In some circumstances, calculation of the BSAF will improve the estimate of the relationship over using a standard BAF. For Newport Bay, tissue contaminant concentrations are significantly positively correlated with fish tissue lipid for total DDTs (Pearson's $r = 0.85$; $p < 0.001$) and total chlordanes ($r = 0.63$; $p < 0.001$), but not PCBs ($r = 0.22$; $p > 0.15$). A positive association is observed, with fish species high in lipid (e.g., California corbina, arrow goby, and staghorn sculpin) having elevated DDTs (Figure 4.3).

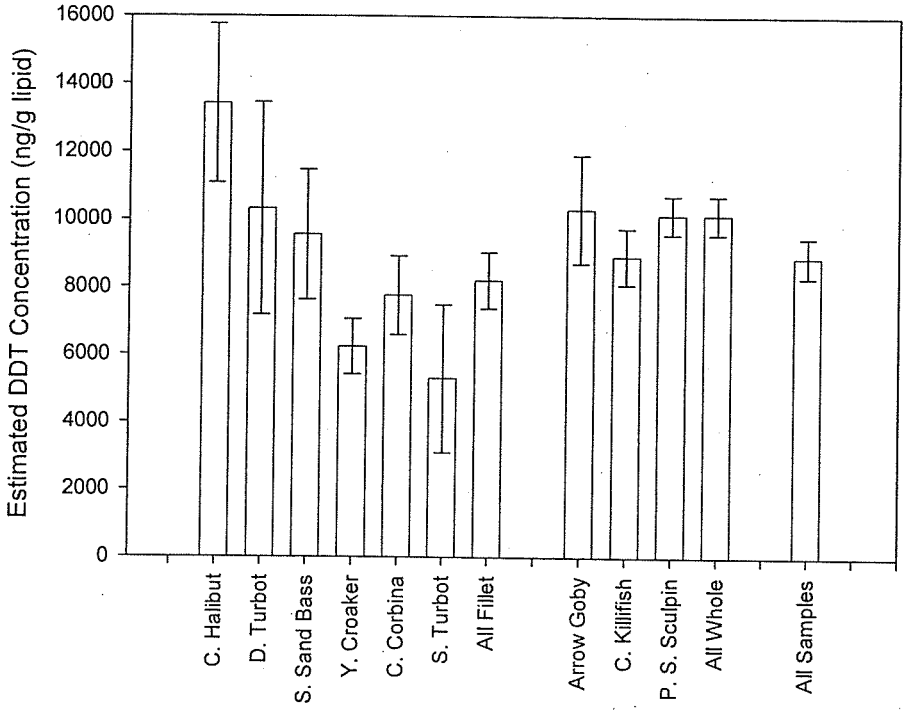
Figure 4.3. Fish tissue total DDT concentration (ng/g wet) vs. percent lipid for all fish species analyzed in Newport Bay.



The significant lipid vs. contaminant relationship in Newport Bay fish suggests that lipid normalization[30] will improve overall ability to estimate average contaminant concentration. In fact, lipid normalized total DDTs (Figure 4.4) do exhibit a lower ratio of standard error to mean, when compared to wet weight data (Figure 4.2). For example, the ratio of standard error to mean for total DDTs in fillet samples is 19% (i.e., 19.1/101.1) for wet weight, but only 10% (829/8197) for lipid weight. In order to reduce variance in the estimate of the bioaccumulation rate parameter, lipid weight data will be used.

[30] i.e., dividing wet weight tissue concentration by total percent lipid

Figure 4.4. Average DDT concentration (ng/g lipid weight) by species. Error bars represent one standard error of the mean for each species. Sample sizes in Table 4.1.



4.4.1.2. Sediment chemistry data and organic carbon normalization

Trace organic contaminant concentrations in sediments are generally correlated with organic carbon content (e.g., Figure 4.5). Significant correlations between total organic carbon and contaminant concentrations are apparent for p,p'-DDE (Pearson's $r = 0.50$; $p < 0.001$; $N = 56$) and total chlordanes ($r = 0.60$; $p < 0.001$; $N = 55$), but not for PCBs ($r = 0.35$; $p = 0.13$; $N = 44$).

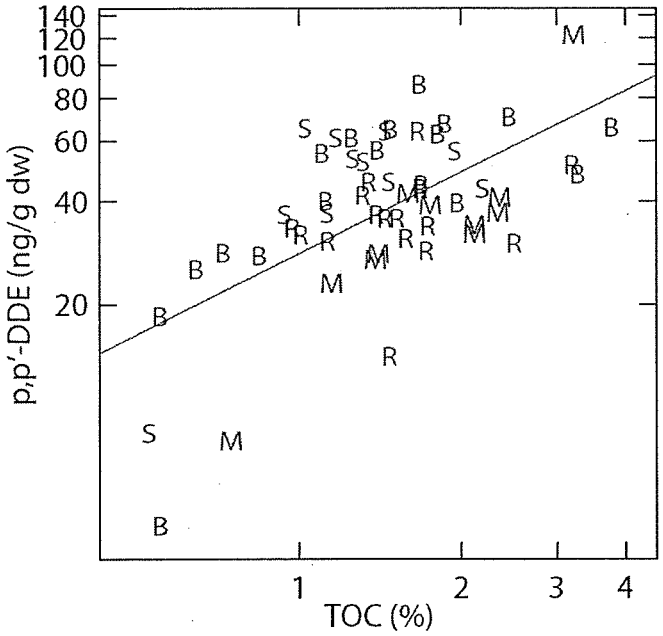
Of the studies that did collect contaminant data, the Bay Protection and Toxic Cleanup Program contains data from the early 1990's. As with the fish data, it is preferable to focus on relatively recent sediment data, given that legacy pesticides often have declining trends over the past decade (Greenfield *et al.* 2005, Connor *et al.* 2006, Davis *et al.* 2006a, O'Connor and Lauenstein 2006). Careful examination of Figure 4.5 indicates that samples collected in the Bay Protection and Toxic Cleanup Program (BPTCP; indicated in Figure by a 'B') tend to have higher total DDTs at a given organic carbon content. This suggests that concentrations were elevated in sediments in the early 1990s, as compared to more recently. BPTCP samples will therefore be excluded from BAF calculations.

The significant association between organic carbon and contaminant content suggests that organic carbon normalization would be appropriate. However, after exclusion of BPTCP

samples, much of the remaining data do not include organic carbon data. Therefore, in order to include all available data, sediment data will not be organic carbon normalized. Thus, for the Newport Bay case study, a modified BAF will be calculated as follows:

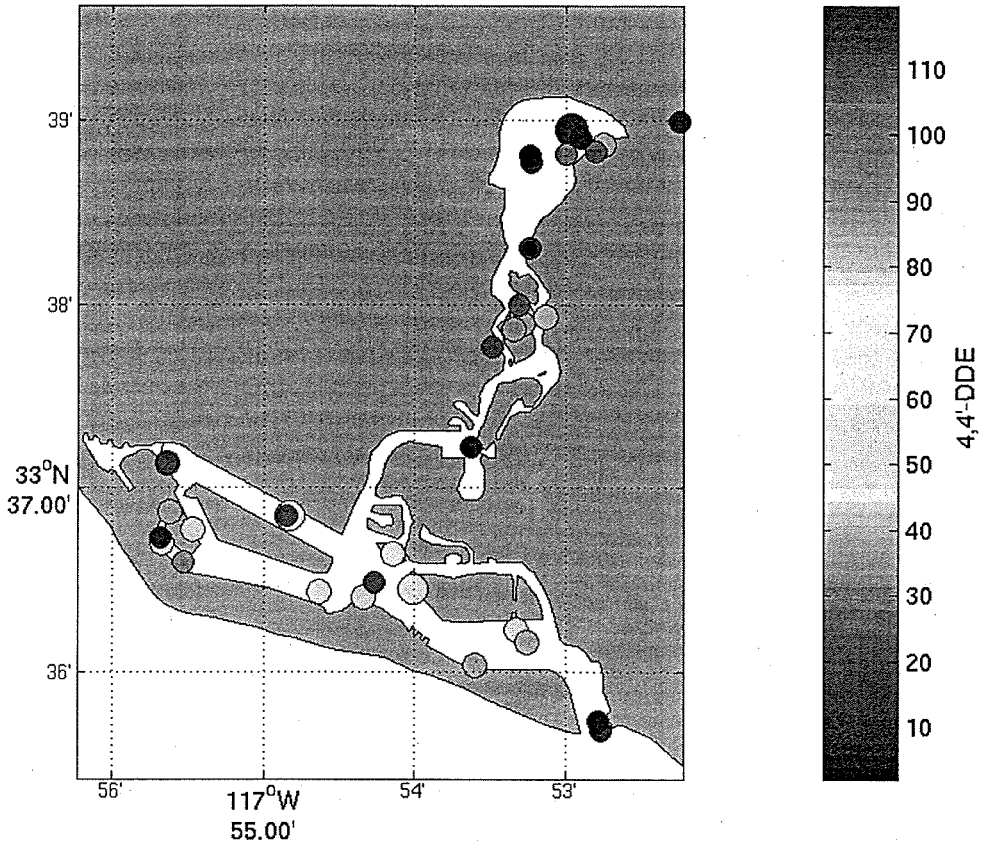
$$\text{BAF} = \frac{\text{Fish Tissue Concentration (ng/g lipid weight)}}{\text{Sediment Concentration (ng/g dry weight)}}$$

Figure 4.5. Percent total organic carbon (TOC) vs. p,p'-DDE concentration (ng/g dry weight) in Newport Bay sediments. Each letter represents a sediment collection location, with letters indicating collection study. B = Bay Protection and Toxic Cleanup Program; S = SCCWRP Southern California Bight 1998 Study (Noblet *et al.* 2002); R = Rhine Channel Study (Bay and Brown 2003); M = Masters and Inman (2000). The smoother line represents the best-fit linear regression model on log-transformed data. Note log scale.



The following sediment data were used in BAF development: Masters and Inman (2000), the Rhine Channel Study (Bay and Brown 2003), the Newport Bay Sediment Toxicity Study (Bay *et al.* 2004), and the Southern California Bight 1998 Survey (Noblet *et al.* 2002). Combining these studies resulted in relatively good spatial coverage across Upper and Lower Newport Bay (Figures 4.6 and 4.7). Concentrations varied spatially, with DDT concentrations highest at the mouth of San Diego Creek and in Lower Newport Bay (Figure 4.6). The elevated concentrations at the mouth of San Diego Creek likely result from upstream loading (Masters and Inman 2000). PCB concentrations were elevated in Inner Lower Bay, particularly in the highly industrialized Rhine Channel (Bay and Brown 2003), and were lower in Upper Newport Bay (Figure 4.7).

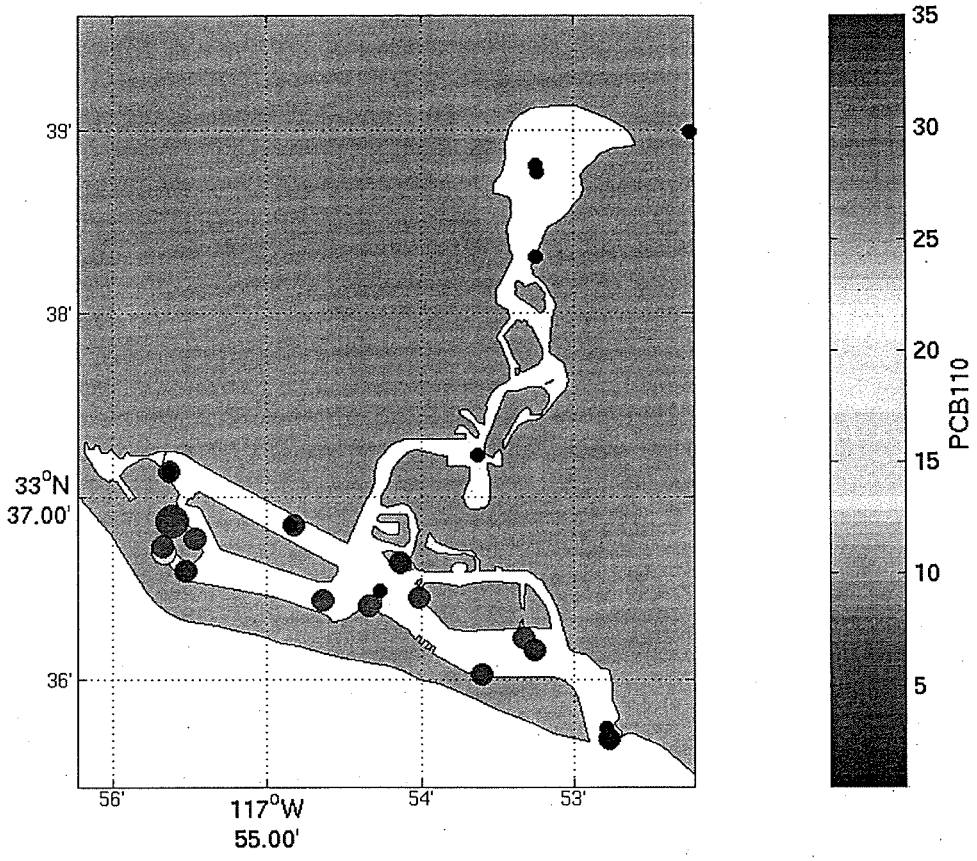
Figure 4.6. Spatial map of sediment contaminant data for p,p'-DDE in Newport Bay. Vertical bar indicates concentration of individual sediment samples (ng/g dry weight). Size and color of dots is proportional to concentration.



In order to calculate the empirical BAF, average and uncertainty estimates must be developed for sediments and fish tissue. However, as discussed elsewhere in this report[31], the method of averaging and treatment of samples that are below detection can strongly affect average estimates (Helsel and Hirsch 2002). For many compounds, a majority of Newport Bay sediment samples were below detection (Table 4.10; Appendix Q, Tables Q.1 and Q.3). The geometric mean was often far below the arithmetic mean, due to the relative greater influence of small values on the calculation (Table 4.10). In situations were a majority of samples were below detection (e.g., o,p'-DDT's), treatment of detection limits affected calculated averages. In particular, average calculations were lower when values below detection were replaced with the laboratory reported detection limits. Average calculations were higher when half of the laboratory reported limits of quantification were used (Table 4.10). For the case study BAF calculations, detection limit values were used for samples below quantification (Table 4.9).

[31] See Sections 3.1.7, 3.1.9, 3.2.7, and Appendix G.

Figure 4.7. Spatial map of sediment contaminant data for PCB 110 in Newport Bay. Vertical bar indicates concentration of individual sediment samples (ng/g dry weight). Size and color of dots is proportional to concentration.



4.4.1.3. Effect of assumptions on BAF

A number of data treatment assumptions will affect sediment and tissue data and consequent BAF calculations. These include averaging methods, sample selection, and treatment of samples below detection (Table 4.9). Without a sufficient number of detectable samples, concentration estimates become highly uncertain (Table 4.10). Appendix Q (Table Q.1) lists concentration estimates for 61 PCBs and legacy pesticides for which data have been collected in recent studies. Of these compounds, 24 have a vast majority (>90%) of measurements below detection. An additional seven compounds have very few (< 10) measurements total throughout Newport Bay (Table Q.1). In calculating totals for BAF estimation, these compounds were removed from the data set, with the remaining 30 compounds included (Table 4.9).

For the Newport Bay case study, averaging method and tissue sample selection were both evaluated to determine their impact on the BAF calculation. Average fish tissue and

sediment concentrations for total PCBs, DDTs, and chlordanes[32] were calculated using both arithmetic and geometric averaging methods. Tissue sample averages were calculated for all fish samples, only human prey, and only wildlife prey, following the designations in Table 4.1.

Table 4.10. Different estimates of concentrations of selected contaminants in Newport Bay sediments. Mean = arithmetic mean. Geometric mean = mean of log_{10}-transformed values, converted to arithmetic units. Median = central value. DL = samples below detection were replaced with limit of detection. ½ QL = samples below detection were replaced with 0.5*(limit of quantification). % ND = total percent of samples below detection.

Average Type:	Mean	Mean	Geometric mean	Geometric mean	Median	N	% ND
How treat NDs:	DL	1/2 QL	DL	1/2 QL	DL		
o,p'-DDD	0.51	0.98	0.18	0.78	0.07	45	80%
o,p'-DDE	1.25	1.46	0.27	0.78	0.46	45	56%
o,p'-DDT	0.27	0.76	0.10	0.70	0.04	45	84%
p,p'-DDD	6.69	6.81	1.85	3.61	6.97	45	27%
p,p'-DDE	29.02	29.02	20.54	20.54	26.46	45	0%
p,p'-DDT	3.27	3.54	0.46	1.47	0.06	45	58%
Total DDTs	41.02	42.56	23.39	27.87	34.05	45	0%
PCB118	2.01	2.25	0.26	0.97	0.04	34	71%
alpha-Chlordane	1.49	1.69	0.28	1.19	1.57	24	46%

4.4.1.4. Empirical BAF results

Empirical bioaccumulation factors were calculated based on tissue and sediment concentrations for total DDTs, total PCBs, and total chlordanes (Appendix N). Individual compound concentrations were averaged replacing ND concentrations with the method detection limit (e.g., Table 4.10, second and fourth columns).

Bioaccumulation factors tended to be higher and substantially more uncertain using geometric averaging methods, than arithmetic methods (Table 4.11). The uncertainty mostly resulted from the high variability in geometric estimates of sediment chemistry (Table 4.11, sediment rows). Uncertainty was very high in geometric estimates of sediment chemistry due to two factors. First of all, with so many individual chemicals below reporting limits, the uncertainty of concentration estimates became very high on a log-scale. This uncertainty propogated as individual chemical results were combined to obtain sediment chemistry average estimates. Bioaccumulation factors were generally similar for different prey groupings, with the exception that geometric average

[32] As the fish tissue line of evidence indicated low risk due to chlordane exposure, the chlordane results are presented for illustrative purposes only.

bioaccumulation factors were substantially lower for chlordanes in wildlife prey than human prey (Table 4.11).

Clearly, the selection of arithmetic vs. geometric averaging and error estimation will substantially influence the final thresholds selected. Results from the mechanistic model uncertainty evaluation will be used to help determine whether the arithmetic or geometric approach is more appropriate to represent the water body BAFs.

Table 4.11. Summary statistics and calculations for bioaccumulation factors. Results using both arithmetic and geometric averaging methods are presented. 95%iles are presented as upper confidence limits for tissue and lower confidence limits for sediments, based on standard error of the mean for individual compounds. For each row, the BAF is the quotient of the fish and sediment concentration. Note that all fish tissue concentrations are presented as ng/g lipid weight, sediment concentrations are ng/g dry weight and BAFs are on a (tissue lipid weight/sediment dry weight) basis.

	Arithmetic					Geometric				
Grouping	Mean	SE	95%ile	Average BAF	UCI BAF	Mean	SE_a	95%ile	Average BAF	UCI BAF
PCBs										
All fish	1,104	223	1,550	38	66	747	1.13	949	154	4,104
Human prey	997	140	1,277	34	54	909	1.10	1,091	187	4,716
Wildlife prey	1,188	385	1,959	41	84	640	1.22	951	132	4,112
All sediments	29	2.9	23			4.9	4.58	0.2		
Chlordanes										
All fish	618	64	745	168	302	409	1.19	582	646	5,722
Human prey	830	49	928	226	377	806	1.06	907	1,275	8,910
Wildlife prey	451	94	639	123	259	240	1.30	408	380	4,012
All sediments	3.7	0.6	2.5			0.6	2.49	0.1		
DDTs										
All fish	8,754	474	9,701	213	287	8,203	1.06	9,237	351	1,526
Human prey	7,828	745	9,318	191	276	7,232	1.10	8,808	309	1,456
Wildlife prey	9,479	581	10,641	231	315	9,053	1.07	10,358	387	1,712
All sediments	41	3.6	34			23	1.97	6.1		

a. Presented in logarithmic (base 10) units

4.4.2. Development of bioaccumulation factor using mechanistic model

It is beneficial to corroborate empirical estimates of bioaccumulation by independently applying a mechanistic model to predict bioaccumulation (Section 3.2.8). This will help to determine whether a variety of factors interfere with the ability to estimate bioaccumulation rates. For example, limitations in data availability may increase uncertainty in bioaccumulation estimates. Additionally, a number of processes may complicate efforts to predict bioaccumulation rates. At a given site, a portion of an organism's contaminant body burden may result from uptake from other sources, such as the overlying water column. For highly mobile organisms, variation in home range can

affect the relative impact of contamination at a specific site (Linkov *et al.* 2002). Variations in food web structure can also cause differences in contaminant bioaccumulation (Gobas and Wilcockson 2002). An uncalibrated model provides an independent estimate of the BAF; if model and empirical results are similar, this supports the empirical BAF approach.

Use of the mechanistic model in the case studies served several objectives. The primary objective was to ascertain whether the empirical bioaccumulation factor was a reasonable estimate of sediment vs. biota partitioning. If the empirical bioaccumulation factor were higher than mechanistic model results, this may support the hypothesis that a substantial proportion of the bioaccumulation was due to contaminant uptake from other sources. Other objectives included characterizing the uncertainty of BAF predictions and identifying possible areas for future research. The remainder of this section summarizes the input data for using the mechanistic model, and compares model output to empirical results. Additional information regarding model input parameters and their basis can be found in Appendices R – V and Appendix Y.

4.4.2.1. Model description

The case studies used a steady state non-equilibrium model developed to assess transfer of non-polar organic contaminants through food webs (Gobas 1993). This model has been updated to incorporate new research regarding phytoplankton uptake and elimination, fish and invertebrate ventilation rates, chemical partitioning and mechanisms of gastrointestinal magnification of contaminants (Morrison *et al.* 1996, Arnot and Gobas 2004). The model, and applications where it has been used previously, are further described in Section 3.2.8 and Table 3.10. Appendix M presents a listing of all model parameters and equations and Appendix Y indicates data needs for the model. Readers interested in further information on model assumptions and structure should also refer to Arnot and Gobas (2004) and to Gobas and Arnot (2005).

The food web bioaccumulation model was converted from a Microsoft Excel spreadsheet (provided by Jon Arnot and Dr. Frank Gobas) to a set of MATLAB routines (www.mathworks.com) in an effort to increase the transparency of model parameterizations. The current model is capable of incorporating an arbitrary number of species and contaminants, a feature that allows the model to be easily adapted for application to any appropriate region of interest.

4.4.2.2. Parameter development for Newport Bay

Estimation of model input parameters is an important part of any modeling exercise. For each case study, local data were compiled and used to generate parameter distributions and ranges. Based on previous sensitivity analyses in San Francisco Bay and other ecosystems, the model is likely to be relatively sensitive to particular environmental, biological and chemical parameters (Arnot and Gobas 2004). These include contaminant concentrations in water and sediments, organic carbon content in water and sediments, sediment-water partitioning coefficients, organism dietary composition, body weight, and

lipid content (Gobas and Arnot 2005). The model has previously been parameterized to calculate uptake of PCBs in selected San Francisco Bay fish and wildlife (Gobas and Wilcockson 2002, Arnot and Gobas 2004).

In general, all available model input data for Newport Bay were assembled with preferential use of site-specific data over data from similar sites. Appendix R describes data sources and compilation methods for use in the mechanistic model. Appendix S presents input parameters for octanol-water partitioning coefficient, which strongly influences contaminant bioaccumulation.

Table 4.12 summarizes input data for physical features of Newport Bay, which were estimated as described in Appendix R. Table 4.13 summarizes input data for biota diet, and biology, which were compiled based on a review of published and unpublished studies (Appendices T, U, and V). Estimates of food web structure are based on total mass proportion of prey items consumed, and reflect the proposed conceptual model for Newport Bay (Figure 4.1). Dietary information for fish represents a review of published and unpublished sources on dietary studies (Appendix T). Table 4.14 presents additional input parameters, and their distribution assumptions for Monte Carlo simulations (described further below).

To facilitate interconversion to the most appropriate tissue type for the contaminant exposure evaluation, concentrations were extrapolated between whole body and fillet samples, assuming lipid equivalence:

Whole Body Conc. = Fillet Conc. * (Whole Body Lipid % / Fillet Lipid, %)

When lipid concentrations were not available in both tissue types from the same species, a regression relationship was used to extrapolate whole body vs. fillet lipid content (Appendix E).

Table 4.12. Physical input parameters for mechanistic food-web contaminant uptake model application to Newport Bay. Appendix R describes source data used to estimate these parameters.

Constant	Value	Distribution	Standard deviation	Description
Cox	7.6	Normal	1	Dissolved oxygen concentration (mg O_2/L)
T	17.5	Normal	0.8	Mean water temperature
salinity	27.2	Normal	2	Water salinity (PSU)
ocsed	0.0145	Lognormal	0.0254 [a]	Organic carbon proportion in sediment (kg/kg)
vss	8.58E-05	Lognormal	0.0256 [a]	Concentration of suspended solids (kg/L)
xpoc	3.77E-06	Lognormal	0.0256 [a]	POC concentration in water (kg/L)
xdoc	1.19E-06	Lognormal	0.0392 [a]	DOC concentration in water (kg/L)

a. This is the log-scale standard deviation, and is applied to log-transformed data, which is then back-transformed to develop input parameters for Monte Carlo simulation.

Table 4.13. Biological input parameters for applying the mechanistic food-web contaminant model to Newport Bay (see also Appendices T, U, and V). % Respiration PW = percent of respiration that is pore water. For benthic plants and invertebrates, scientific names are for representative species. For percent lipid, **bold values** were estimated following the regression relationship presented in Appendix E.

			Prey items (% Composition) [a]													
Common name	Scientific name	% Respiration PW	Sediment	Benthic algae	Phytoplankton	Zooplankton	Amphipods	Annelids	Mollusks	Hydrozoa	Echiuroidea	Topsmelt	Arrow goby	% Lipid in whole body	% Lipid in fillet	Mass (g) [a b]
Phytoplankton		0	-	-	-	-	-	-	-	-	-	-	-	0.12 [b]	-	-
Benthic algae	*Ulva* sp., *Enteromorpha* sp.	5	-	-	-	-	-	-	-	-	-	-	-	0.5 [a]	-	10.0
Zooplankton		0	0	0	100	0	0	0	0	0	0	0	0	0.75 [bc]	-	0.000071
Amphipod	*Grandidierella japonica*	5	20	40	20	20	0	0	0	0	0	0	0	0.75 [bc]	-	0.001
Annelid	*Pseudopolydora japonica kempi*	5	40	30	15	15	0	0	0	0	0	0	0	1.0 [bc]	-	0.0005
Mollusk	*Cerithidea californica*	5	20	80	0	0	0	0	0	0	0	0	0	1.5 [bc]	-	0.1
Hydrozoa	*Tubularia crocea*	0	0	0	100	0	0	0	0	0	0	0	0	2.5 [bc]	-	0.06
Echinoderm	*Leptosynapta* sp.	5	100	0	0	0	0	0	0	0	0	0	0	0.75 [bc]	-	0.08
CA halibut	*Paralichthys californicus*	5	0	0	0	0	8	0	2	0	0	45	45	0.94 [d]	0.4	1463.3
Yellowfin croaker	*Umbrina roncador*	5	5	0	0	0	25	45	10	10	0	2.5	2.5	**3.6** [d]	1.7	385.0
Topsmelt	*Atherinops affinis*	0	23	60	0	5	12	0	0	0	0	0	0	1.6 [d]	**0.7**	20.0
Striped mullet	*Mugil cephalus*	5	30	55	0	0	5	5	5	0	0	0	0	**9.4** [e]	4.4	1229.6
Arrow goby	*Clevelandia ios*	10	0	0	0	35	10	55	0	0	0	0	0	1.3 [d]	**0.6**	0.5
CA killifish	*Fundulus parvipinnis*	0	0	0	0	10	20	25	45	0	0	0	0	1.5 [d]	**0.7**	7.0
Shiner surfperch	*Cymatogaster aggregata*	0	10	0	0	60	14	14	2	0	0	0	0	1.0 [d]	0.5	8.5
Staghorn sculpin	*Leptocottus armatus*	5	0	0	0	0	75	5	5	0	0	7.5	7.5	1.4 [d]	**0.6**	1.8
Spotted sand bass	*Paralabrax maculatofasciatus*	5	0	0	0	0	25	0	35	0	20	10	10	**1.9** [d]	0.9	599.0
CA corbina	*Menticirrhus undulatus*	5	0	0	0	0	25	25	25	0	0	12.5	12.5	**9.8** [d]	4.6	668.3

a. Sources in Appendices T, U and V. b. Gobas and Arnot (2005). c. Don Cadien, LACSD, *Pers. comm.* d. Allen *et al.* (2004). e. TSMP data (Rasmussen 1995)

Table 4.14. Specification of parameter distribution and variation for Monte Carlo simulations.

Parameter	Value	Distribution	Variation	Description
lipid	Table 4.13	Normal	SD = 0.2*μ [a b]	% Lipid error as proportion of average value
NLOM phytoplankton	6%	Normal	SD = 0.2% [b]	% Non-lipid organic matter
NLOM benthic algae	15%	Normal	SD = 1.5% [c]	% Non-lipid organic matter
NLOM animals	20%	Normal	SD = 1% [b]	% Non-lipid organic matter
mp benthic algae	5%	Uniform	Range = 0 – 10% [d]	% Transpiration uptake that is pore water
mp animals	0 – 10%	Uniform	Range = 0.8*μ – 1.2*μ [b]	% Respiration uptake that is pore water
Wb	Table 4.13	Normal	SD = 0.1*μ [b]	Body mass (kg)
K_{ow}	Appendix S	Lognormal	SD = 0.1 [b e f]	Octanol-water partitioning coefficient
cw	Appendix Table Q.2	Lognormal	SD = 0.1 [e g]	Contaminant concentration in water (µg/L)
cs	Appendix Table Q.1	Lognormal	Appendix Table Q.1	Contaminant concentration in sediment (ng/g dry)

a. Rasmussen (1995). b. Gobas and Arnot (2005). c. c.f., Appendix V. d. estimated. e. This is the log-scale standard deviation, and is applied to log-transformed data, which is then back-transformed to develop input parameters for Monte Carlo simulation. f. Mackay *et al.* (2000). g. Average of standard errors in Appendix Q, Table Q.2.

4.4.2.3. Calculation of total BAFs

Many of the chemical compounds included in the food web models of Newport Bay and San Francisco Bay comprise specific chemical groups (i.e., PCBs, chlordanes, or DDTs). Thus, in addition to calculating compound-specific BAF values, it is possible to estimate the BAF for a given chemical group, referred to as the total BAF (BAFt). The calculation of total BAFs from compound-specific values is as follows:

$$BAFt = \frac{\sum_i Cb_i}{\sum_i Cs_i} = \frac{\sum_i (Cs_i \times BAF_i)}{Cs_T}$$

where Cb_i is the model predicted compound-specific contaminant concentration in biota, Cs_i is the compound-specific contaminant concentration in sediment, BAF_i is the model predicted compound-specific BAF, and Cs_T is the sum of all compound-specific contaminant concentrations in sediment.

4.4.2.4. Monte Carlo uncertainty analysis

A Monte Carlo methodology was used to determine the uncertainty in model predictions of BAF resulting from the inherent variability and error associated with model input parameters. This methodology samples model input parameters from statistical distributions rather than point estimates. The methodology repeatedly samples the parameter distributions, executes the food web model, and stores the results. A distribution of model results is generated which represents the variability of model predictions due to the variability and error in model input parameters.

Monte Carlo distributions may be used to represent the multiple sources of variability inherent in model parameters, including geospatial, temporal, and behavioral variability and differences in sampling protocols. However, as discussed in the conceptual model for the indirect assessment framework (Section 2), it is expected that avian piscivores and human sport fish consumers integrate spatially and temporally across the water body. Based on this assumption, the input parameters for the Monte Carlo simulation are intended to represent average conditions that fish consumers may encounter. Therefore, input parameter distributions in this exercise represent uncertainty about the average, due to uncertain and variable input data. In particular, model input standard deviations were typically estimated based on standard error of the mean, rather than standard deviation of the entire data set (following Section 3.1.9).

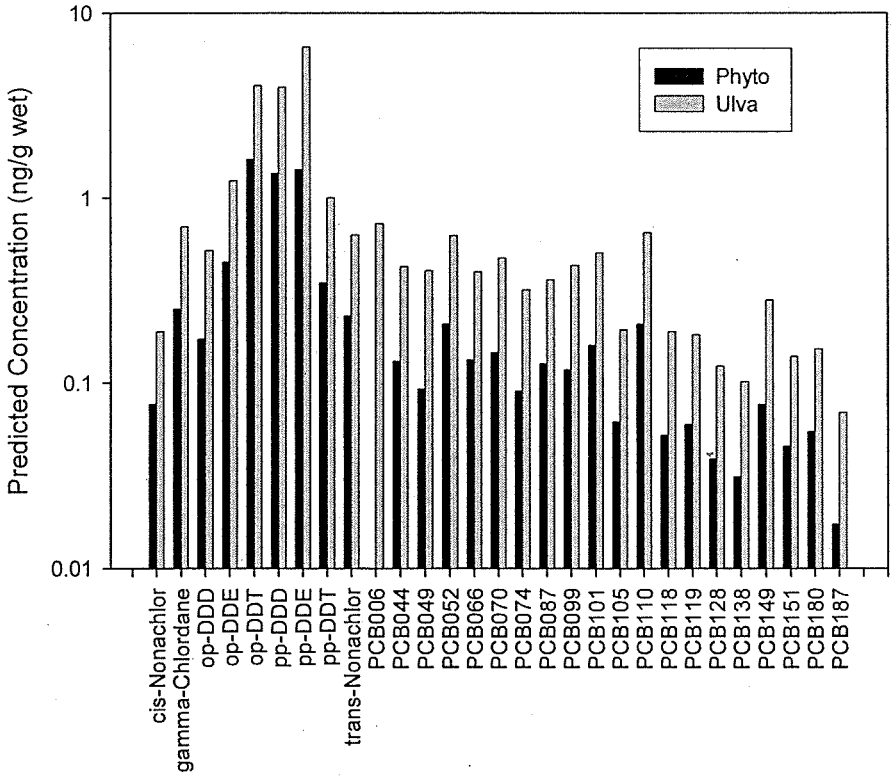
The Monte Carlo Simulation (MCS) analysis module of the food web model uses MATLAB's intrinsic random number generator to generate distributions of model input parameters. The parameter distributions are generated at model initialization and the model is run iteratively. The module is capable of generating normal, lognormal, triangular, and uniform distributions. Inputs to the MCS module are the parameter mean, standard deviation, distribution type (i.e., normal, lognormal, triangular, or uniform), and

the number of samples. Tables 4.12 and 4.14 indicate these values for MCS inputs. The MCS was performed for 5000 model runs.

4.4.2.5. Results of model simulations

General behavior of the model has been described in previous applications (e.g., Burkhard 1998, Arnot and Gobas 2004). Findings from the current application highlight key model assumptions, and their effects on the current case study. The model generates as output the BAF and biota whole body concentrations for individual organic compounds. These results are generated for all compounds and organisms modeled, including primary producers (Figure 4.8), invertebrates (e.g., Figure 4.9) and fish. Individual compound concentrations are summed within a compound category to obtain total PCB, DDT, and chlordanes in all organisms. These total concentrations may then be divided by original input sediment concentrations to obtain BAFs (Figures 4.10 – 4.14). Monte Carlo simulation results were approximately normally distributed about the mean for total DDTs and PCBs (e.g., Figure 4.13), and log-normally distributed about the mean for total chlordanes (e.g., Figure 4.14).

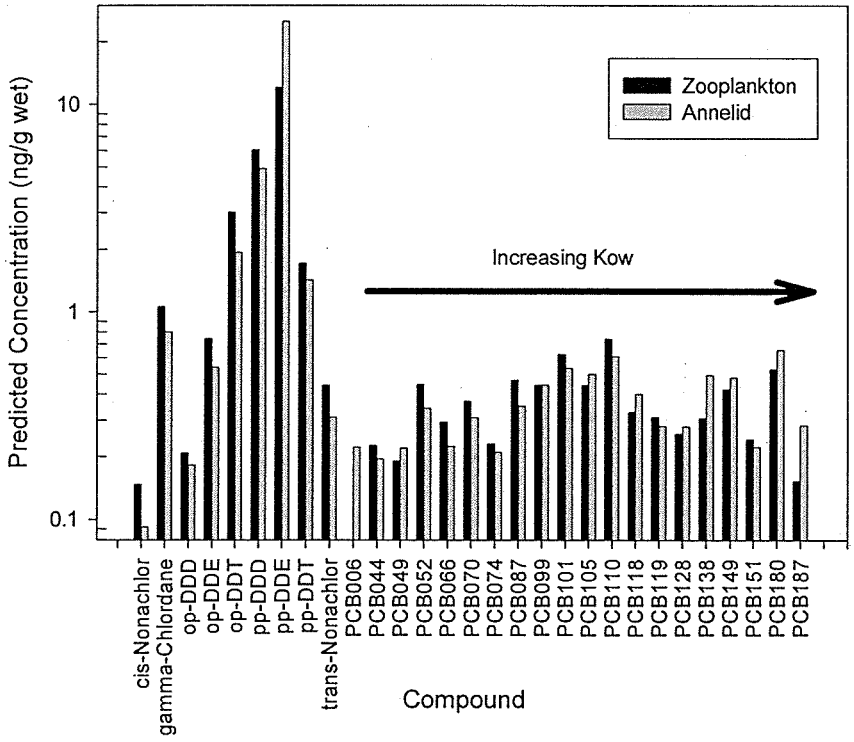
Figure 4.8. Concentrations of organic compounds predicted in phytoplankton (black bars) and benthic algae (e.g., *Ulva* sp.; grey bars) in the Newport Bay simulation. Note log scale y-axis.



In general, the model predicted progressively higher bioaccumulation rates for higher trophic levels and fattier organisms, consistent with known trophic fractionation behavior of organochlorine compounds (e.g., Clark *et al.* 1988, Kidd *et al.* 1995). Total PCBs, DDTs, and chlordanes were generally predicted to be highest in fishes, intermediate in benthic invertebrates, and lowest in aquatic plants (Figures 4.10, 4.11, and 4.12). Among fish species, predicted concentrations were relatively high in striped mullet, California corbina, and yellowfin croaker, which also had relatively high tissue lipid contents (Table 4.13; Figures 4.10 – 4.12). Predicted concentrations were relatively low in topsmelt (Figures 4.10 – 4.12). The low model predicted concentrations likely results from topsmelt's high dietary reliance on algae (Table 4.13 and Appendix T) (Logothetis *et al.* 2001).

Variability in Monte Carlo simulation output increased with increasing mean results (Figures 4.10 – 4.12) and was small enough to allow model predictions within approximately 50% to 150% of average values. The coefficient of variation (CV)[33] for all species was higher for total chlordanes (average CV = 36%; range = 18% to 40%; N = 18 taxonomic groups; e.g., Figure 4.14) than for PCBs or DDTs (average CV = 17%; range = 11% to 20% for both compound classes; e.g., Figure 4.13).

Figure 4.9. Concentrations of organic compounds predicted in zooplankton and benthic annelidae (e.g., *Pseudopolydora japonica kempi)* in the Newport Bay simulation. Note that y-axis is log scale and ranges from 0.08 to 30 ng/g.



[33] Coefficient of variation = Standard deviation/Mean

Figure 4.10. Model predicted bioaccumulation factor (BAF) in Newport Bay organisms for total PCBs. Results are from a Monte Carlo simulation with a sample size of 5000. Grey bars represent the mean BAF, and error bars represent the standard deviation.

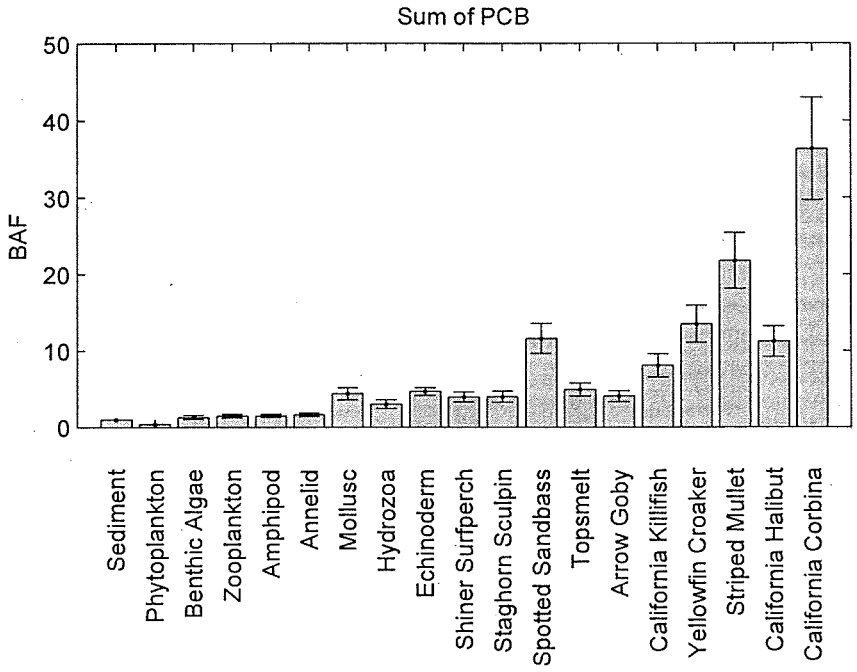


Figure 4.11. Model predicted bioaccumulation factor (BAF) in Newport Bay organisms for sum of six DDTs. Results are from a Monte Carlo simulation with a sample size of 5000. Grey bars represent the mean BAF, and error bars represent the standard deviation.

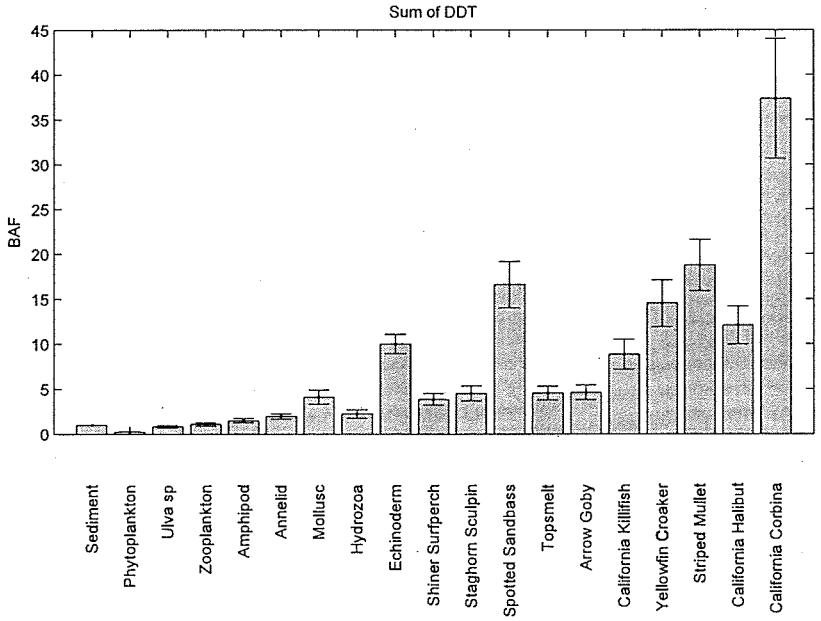


Figure 4.12. Model predicted bioaccumulation factor (BAF) in Newport Bay organisms for sum of three chlordanes (cis-Nonachlor, trans-Nonachlor, and gamma-Chlordane). Results are from a Monte Carlo simulation with a sample size of 5000. Grey bars represent the mean BAF, and error bars represent the standard deviation.

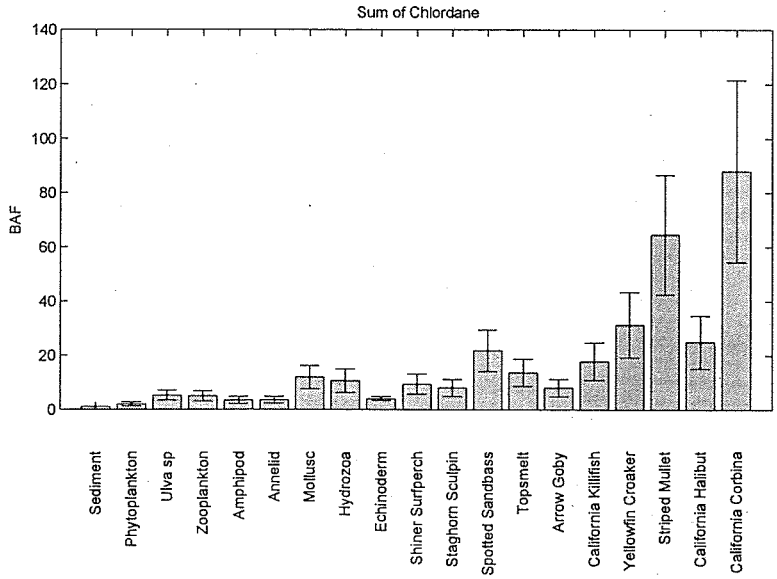


Figure 4.13. Monte Carlo simulation output for application of the model to estimate BAF for total PCBs in striped mullet.

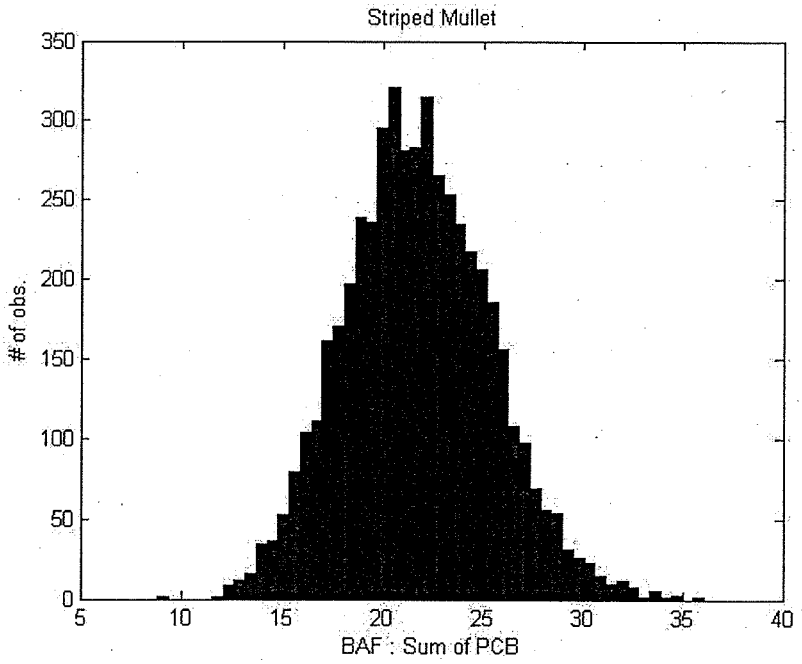
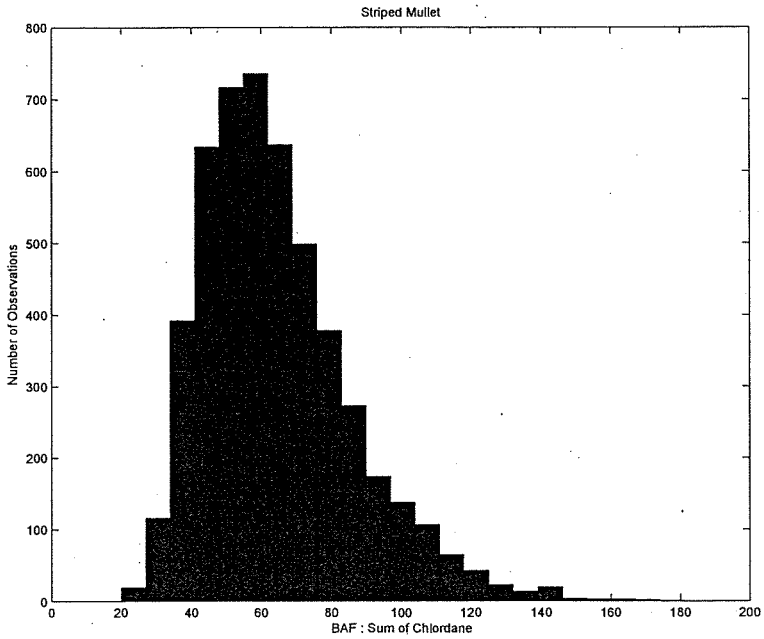


Figure 4.14. Monte Carlo simulation output for application of the model to estimate BAF for sum of three chlordanes in striped mullet.



The model output assisted in determining whether to use arithmetic or geometric averaging to develop average and 95% UCI for empirical BAF predictions. As described in Section 4.4.1.4, empirical data indicated higher averages and substantially greater variance using geometric averaging methods, as opposed to arithmetic methods (Table 4.11). In general, the model Monte Carlo simulation output was more consistent with the variance of the arithmetic averaging method for PCBs and DDTs. In particular, model output was normally distributed and the coefficient of variation was less than 20% of the mean, suggesting that 95% UCI would be around 150% of the mean or less. This would not be consistent with the BAFs developed using geometric methods, which exhibited 95% UCI estimates that were 500% to 2000% of the mean (Table 4.11, last two columns).

Newport Bay differs from many estuaries in having a significant proportion of primary production due to benthic algae (Kamer *et al.* 2004a). Simulation results indicated consistently higher predicted contaminant concentrations for benthic algae, than for phytoplankton (Figure 4.8). Although substantial efforts were made to develop model parameters appropriate for benthic algae (e.g., Appendices R and V), local data are lacking. Input parameter uncertainties include tissue lipid content, non-lipid organic matter (NLOM), porewater exposure and contaminant uptake kinetics. Phytoplankton have been modeled previously (Gobas 1993, Burkhard 1998, Arnot and Gobas 2004, Gobas and Arnot 2005), and literature estimates of tissue lipid content and NLOM are lower for phytoplankton than benthic algae (Tables 4.13 and 4.14, Appendix V).

Manipulation of these parameters indicates that the higher predicted contaminant concentrations for benthic algae resulted from a combination of higher tissue lipid, slower growth rate and consequent growth dilution, and higher exposure to sediment porewater and associated compounds (results not shown). Confirmation of the higher concentrations in benthic algae would require field characterization of contaminants in these taxa.

Model results for benthic annelids vs. pelagic zooplankton indicated relatively similar exposure for the two taxa (Figure 4.9). Nevertheless, differences occurred as a function of K_{OW}. Close examination of Figure 4.9 indicates that for PCBs, modeled concentrations of less chlorinated congeners with lower K_{OW} were higher for zooplankton, whereas modeled concentrations of more chlorinated congeners (higher K_{OW}'s) were higher for annelids. Compound K_{OW} affects model results by affecting calculated sediment-porewater partitioning, chemical specific gut-transfer efficiency, and contaminant respiratory uptake rate (equations outlined in Appendix M). The net result of these processes is higher exposure of sediment dwelling organisms, consistent with the expectation that high K_{OW} compounds have greater sediment association (e.g., Clark *et al.* 1988).

4.4.2.6. Comparison of model vs. empirical results

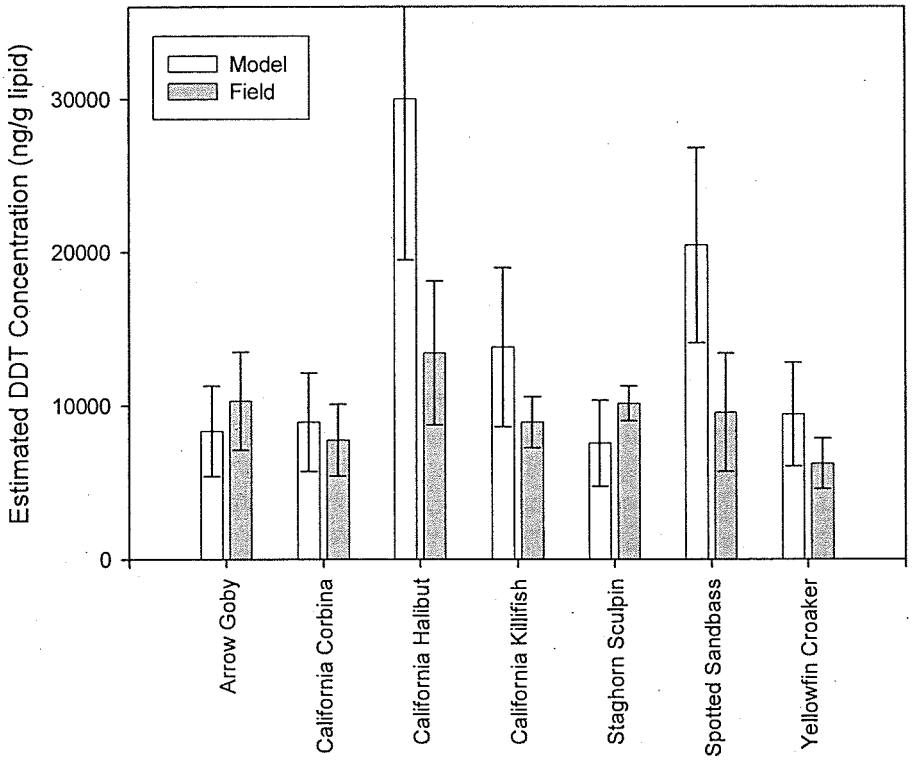
For the Newport Bay case study, the primary use of the model was to corroborate empirical observations of bioaccumulation in the ecosystem. Because model predicted bioaccumulation factors are independent of field measured bioaccumulation factors, consistency between these results would suggest that the empirical BAFs are appropriate. This, in turn, would support the use of the empirical BAFs to develop sediment thresholds. If model vs. field results were inconsistent, or field data were insufficient to facilitate comparison, this would suggest either incorrect model assumptions or additional data needs.

For this study, comparison of model vs. field tissue concentrations focused on total concentrations in the target compound classes (i.e., total PCBs, DDTs, or chlordanes). The model calculates whole body concentrations, while field data for some species were muscle fillet samples (Table 4.1). To account for possible concentration differences between whole body and muscle samples, some results are presented on a lipid weight basis, assuming that lipid normalized concentrations will be similar among tissue types (Bayen *et al.* 2005). When comparisons were conducted on a wet weight basis, interconversion between whole body and fillet types was conducted assuming lipid equivalent concentrations, and based on the tissue lipid estimates in Table 4.13 (following Appendix E).

Evaluation of lipid weight total DDTs indicated reasonably good overall correspondence between model and field results (Figure 4.15). However, there was some indication of model bias. Best estimate model results were higher than empirical results for all species except staghorn sculpin and arrow goby. Model results were about two times field results for California halibut and spotted sand bass, suggesting that some model assumptions

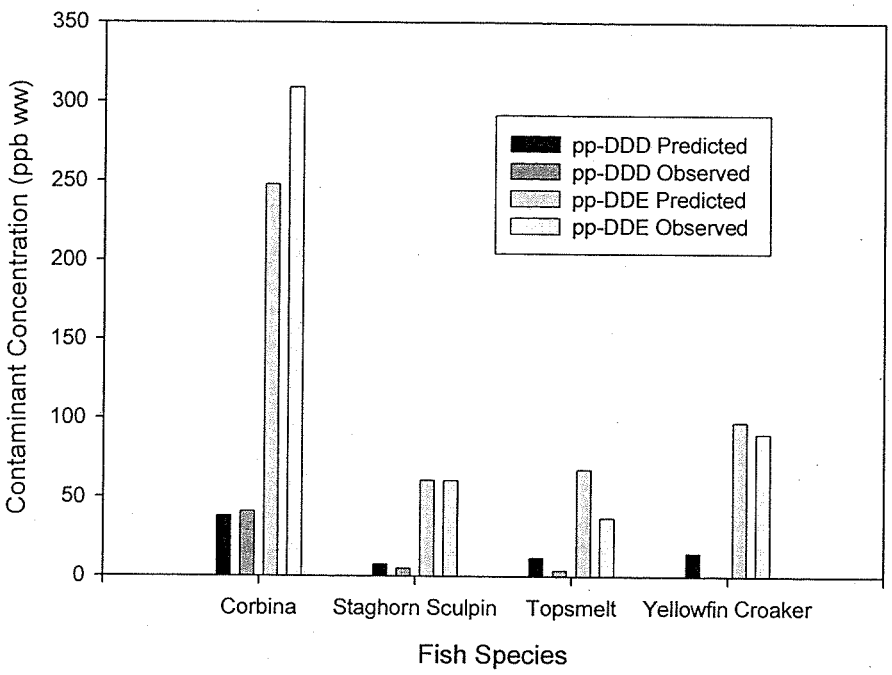
may have been incorrect (Figure 4.15). When the DDT congeners p,p'-DDE and p,p'-DDD were evaluated on a wet weight basis for four fish species, there was good correspondence between model output and field empirical data (Figure 4.16).

Figure 4.15. Comparison of mean model output (white bars) and empirical field data (grey bars) for fish tissue DDTs in Newport Bay. Results are presented on a lipid weight basis (ng/g lipid). Error bars represent model variation (two * standard deviation) and field mean estimates (two * standard error).



There are many possible explanations for the discrepancy between model and empirical results for California halibut and spotted sand bass. These include limited field sample availability and spatial coverage (Table 4.1) (see also Allen *et al.* 2004) or migration of the captured fish from less contaminated offshore locations. One possible explanation for model results higher than field concentrations is that the model does not include metabolic breakdown of DDTs. Although metabolic dechlorination of DDTs is known to occur in finfish (van der Oost *et al.* 2003) at higher rates than PCB breakdown, actual rates are not well characterized (van der Linde *et al.* 2001, Bayen *et al.* 2005). Another possible explanation for the discrepancy between model and field results is that dietary specifications for the model may have been incorrect. Many of the dietary studies for Newport Bay were not recent (Appendix T), and indicated high piscivory by California halibut and spotted sand bass, in addition to sand bass dietary reliance on echinoderms. Changing input parameters to increase reliance on benthic amphipods, mollusks, and annelids brought model results closer to field observations (data not shown).

Figure 4.16. Comparison of mean model output and empirical field data for fish tissue concentrations of p,p'-DDD and p,p'-DDE in Newport Bay. Results are presented on a wet weight basis. California corbina and yellowfin croaker results are for muscle fillets. Staghorn sculpin and topsmelt results are for whole body samples.



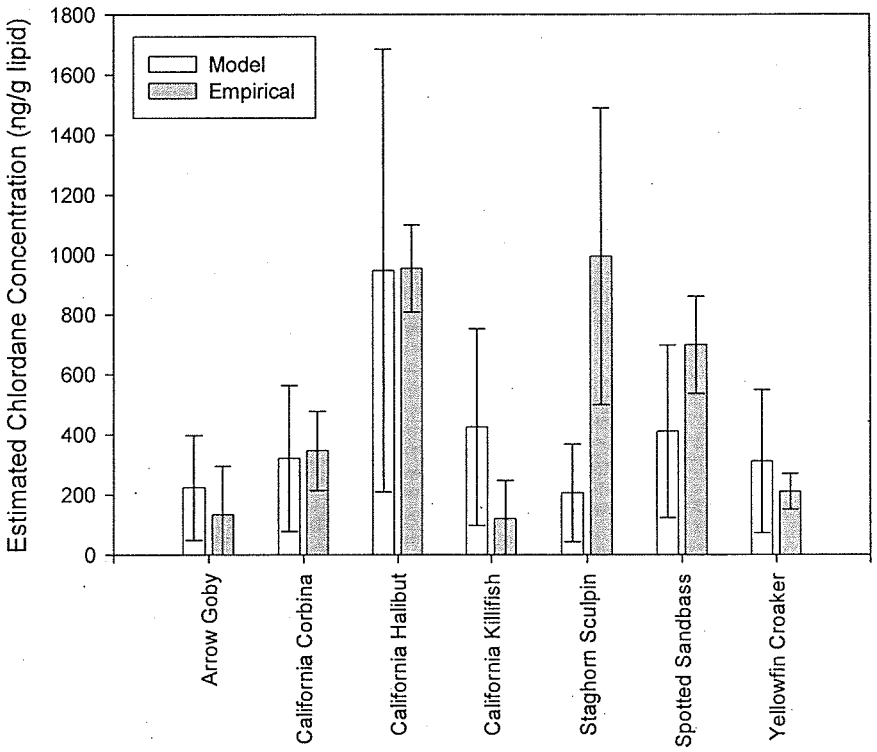
Although data are lacking on Newport Bay resident invertebrate contaminant chemistry, Sutula *et al.* (2005) did determine total DDTs in four amphipod and four bivalve samples collected from Upper Newport Bay. Comparing mechanistic model predictions with the field observations of Sutula *et al.* (2005) indicated that results corresponded only within an order of magnitude [34]. Model predicted amphipod BAF for total DDTs (1.5) was higher than the observed geometric average BAF (0.6) but similar to the observed maximum of 1.6. Model predicted bivalve BAF (4.1) was an order of magnitude higher than the observed BAF (geometric average = 0.3; maximum = 1.2). Given the small sample size and limited spatial scale of the field data set, it is difficult to generate broad conclusions based on these comparisons. The lack of correspondence for bivalves may result from DDT depuration within tissues, reduced bioavailability of sediment-bound DDT at the collection site, or spatial variation in pesticide concentrations and bioavailability (Masters and Inman 2000).

Correspondence between model predicted and field-observed chlordane concentrations was good for four of seven species examined (arrow goby, California corbina, California halibut, and yellowfin croaker; Figure 4.17). Model predictions were lower than field

[34] Dry weight BAF values reported in Sutula *et al.* (2005) are multiplied by 0.25 estimated proportion dry matter in total sample to achieve BAF in g wet wt. tissue/g dry wt. sediment.

observations for staghorn sculpin and spotted sand bass, and higher than field observations for California killifish.

Figure 4.17. Comparison of mean model output (white bars) and empirical field data (grey bars) for fish tissue total chlordanes in Newport Bay. Results are presented on a lipid weight basis (ng/g lipid). Error bars represent model variation (two * standard deviation) and field mean estimates (two * standard error).



For total PCBs, it was not possible to develop confident estimates of field concentrations because the vast majority of congeners were below method detection limits and the detection limits were relatively high (5 ng/g) (Allen *et al.* 2004). For example, if a field sample were below detection for all 20 modeled PCB compounds, the actual concentration could be between 0 ng/g and 100 ng/g, the latter value calculated as total number of compounds (20) multiplied by the detection limit for each compound (5 ng/g). Figure 4.18 shows the wide potential range of concentrations that would be consistent with the observed field data. Model calculated total PCBs were close to the low field concentration estimate for California killifish and yellowfin croaker, and were intermediate between the two field concentration estimates for the other five species compared (Figure 4.18).

Model results indicate that the PCB concentration estimates in Allen (2004), which are used in the fish tissue line of evidence (Table 4.6), are likely to be considerably less than actual PCB fish tissue concentrations in Newport Bay. This is because the model

predicted PCBs below the 5 ng/g detection limit, but above 0 ng/g for all congeners. As a result, model predicted total PCBs were between 19 and 108 ng/g, although measurable concentrations using 5 ng/g detection limits would often be zero (Table 4.15). Detection limits in other monitoring studies are often at 0.2 ng/g for individual congeners (e.g., Davis *et al.* 2000, Greenfield *et al.* 2003). Modeled fish tissue concentrations were generally above these achievable detection limits (Figure 4.19). Given that human health screening values for total PCBs can be as low as 2.5 to 20 ng/g (Appendix K, Table K.1), future fish tissue PCB analyses for human health risk characterization should attempt to achieve these lower detection limits.

Figure 4.18. Comparison of mean model output (white bars) and empirical field data (grey and blue bars) for fish tissue total PCBs in Newport Bay (ng/g lipid). Results are presented on a lipid weight basis. Field results are presented with two different treatments of values below detection limits. For the low estimate, all compounds below detection limits were treated as zero. For the high estimate, all compounds below detection limits were treated as equal to the detection limit (5 ng/g wet). Error bars represent model variation (two * standard deviation) and field mean estimates (two * standard error).

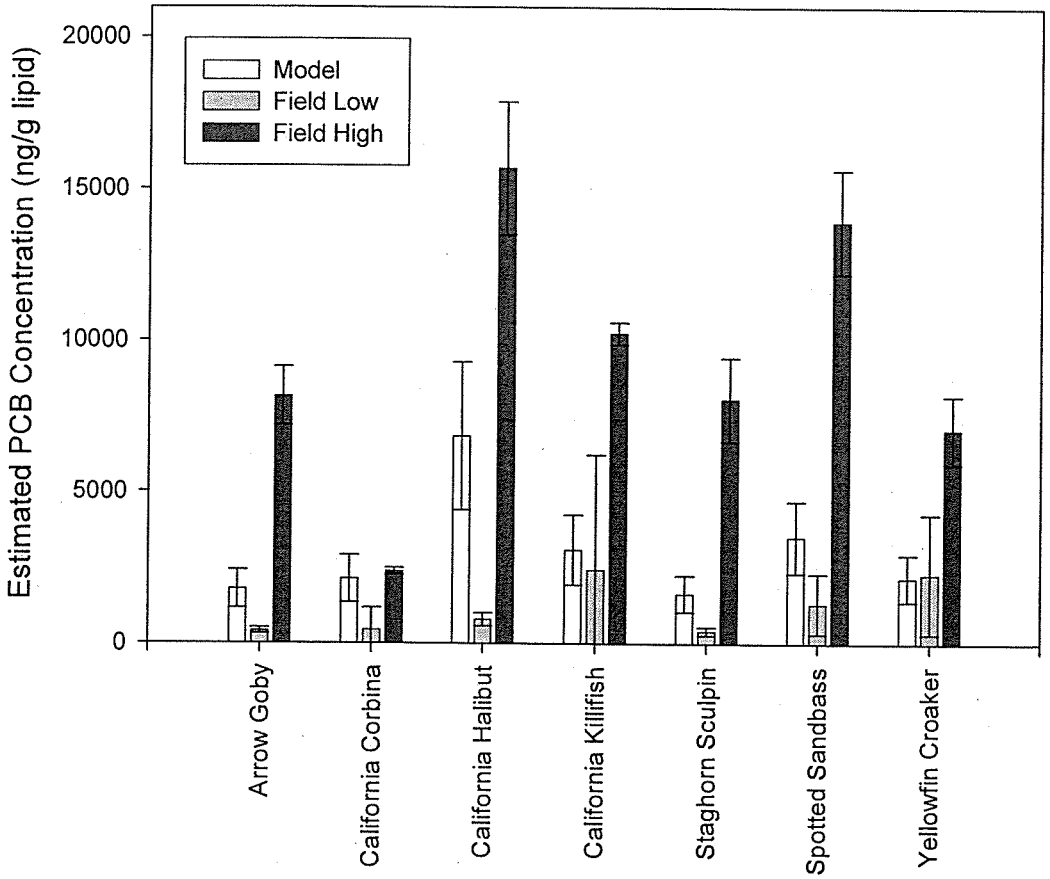


Table 4.15. Summary of results of model simulation evaluating individual PCB congeners as compared to detection limits in currently available studies. WB = whole body. % < DL = percent of the 20 modeled congeners that were present in Newport Bay sediments (Appendix N) but below tissue detection limits of 5.0 ng/g. Exposure estimate = the sum of model predicted PCB concentrations, including those residues that are below detection limits. Estimate with high DLs = sum of only those PCB congeners predicted to be > 5.0 ng/g.

Species	Tissue type	% < DL	Exposure estimate (ng/g wet)	Estimate with high DLs (ng/g wet)
Shiner surfperch	WB	100	19	0
Pacific staghorn sculpin	WB	100	19	0
Spotted sand bass	Fillet [a]	100	27	0
Topsmelt	WB	100	24	0
Arrow goby	WB	100	19	0
California killifish	WB	100	40	0
Yellowfin croaker	Fillet [a]	100	31	0
Striped mullet	WB	45	108	78
California halibut	WB	100	55	0
California halibut	Fillet [a]	100	36	0
California corbina	Fillet [a]	65	85	44

a. Fillet concentration estimated as lipid weight normalized concentration using model output whole ^{body} concentrations and the whole body vs. fillet lipid ratios in Table 4.13.

Overall, model simulation results were reasonably consistent with field observed BAFs for organochlorine contaminants in Newport Bay fish. DDTs and chlordanes corresponded well for many fish species, and modeled PCB concentrations were within the range of the relatively uncertain field values. In particular, field-observed tissue concentrations were not substantially greater than model predictions, based on Newport Bay sediment and water column concentrations. This result supports the conceptual interpretation that tissue concentrations in Newport Bay fish result from dietary and respiratory exposure to sediment and water column contaminants within the Bay. Monte Carlo simulation results indicated that uncertainty of predicted BAFs was more consistent with arithmetic (rather then geometric) techniques of estimating empirical uncertainty.

4.4.3. Sediment thresholds comparison

4.4.3.1. Approach - evaluation of multiple scenarios

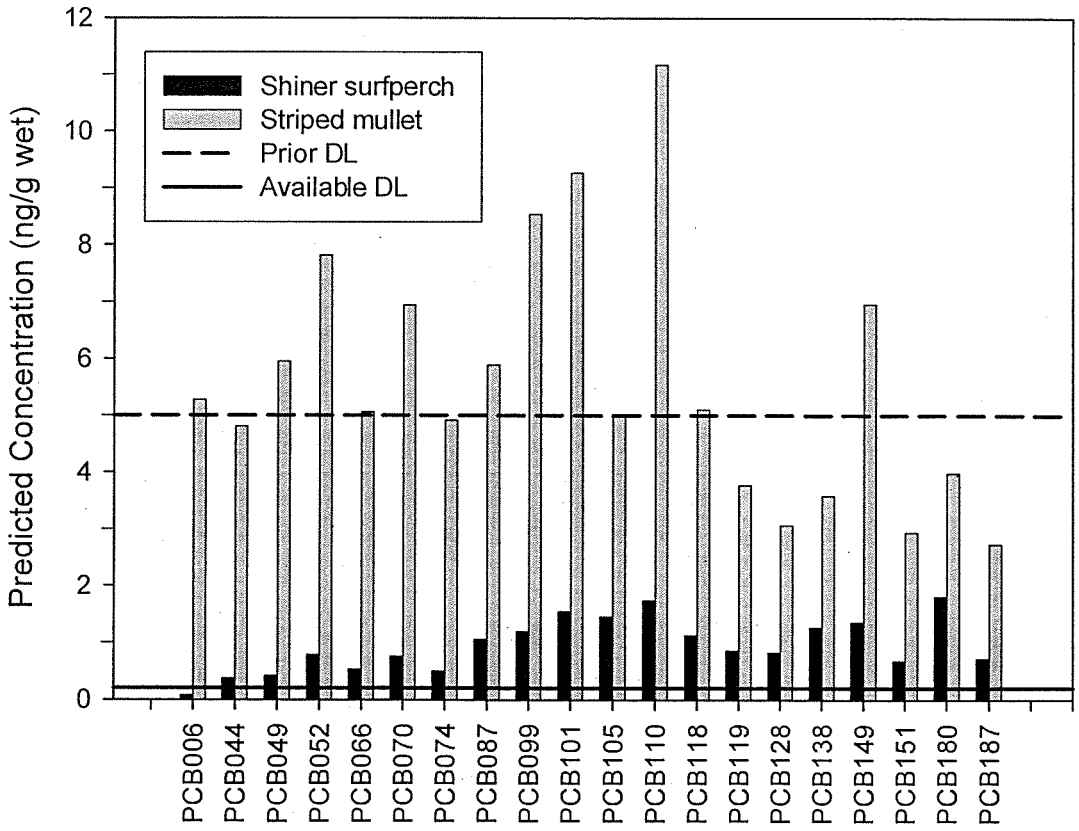
Given that the mechanistic model findings supported use of the empirical BAFs, the analysis proceeds to development of sediment thresholds and final sediment evaluation. However, a number of decisions affect the development of sediment thresholds (Tables 4.9 and 4.16). These include tissue percent lipid, assumptions that affect prey tissue thresholds (including consumption rate and allowable increased carcinogenic risk), and

BAF averaging method (arithmetic vs. geometric). The sediments selected for evaluation will also affect the results. In this section, multiple scenarios are performed to evaluate the effects of different assumptions on the final sediment assessment.

Because the empirical BAFs were developed based on lipid weight tissue concentrations and the effects thresholds are based on wet weight tissue concentrations, it is necessary to back-calculate to wet weight tissue concentrations in order to develop sediment thresholds:

$$\text{Sediment threshold (ng/g dw)} = \frac{\text{Prey tissue threshold (ng/g wet)}}{\text{BAF(g dw/g lipid)} \times \text{\% Lipid (g lipid/g wet)}}$$

Figure 4.19. Model output for 20 PCB congeners for shiner surfperch and striped mullet in Newport Bay. Horizontal lines represent the detection limit from recent studies (dashed line) and detection limits that are readily available in contract analytical labs (solid line).



Prey tissue thresholds for the Newport Bay case study were presented in Tables 4.3 and 4.4. Average and 95% upper confidence interval BAFs were presented in Table 4.11. Tissue lipid content must be estimated, and is influenced by assumptions such as prey

types and proportions. Appendix W describes the procedures used and results obtained for estimating prey tissue lipid content for the case study.

Table 4.16. Summary of assumptions, their basis, and effects on developing sediment thresholds for the sediment chemistry LOE, Newport Bay case study. This represents the baseline scenarios (Scenarios A and AA). The impact of key decisions is also evaluated using alternative scenarios (Tables 4.17 and 4.18). See also Table 4.9.

Decision or assumption	Basis	Effect
Use SARWQCB Staff requested human target population and cancer risk	See Table 4.5	Higher incidence of exceeding thresholds than other sets of assumptions (Tables 4.19 – 4.21)
Evaluate wildlife risk using least tern	The assessment framework should be protective of the most sensitive wildlife species, which is calculated to be least tern	Slightly higher incidence of exceeding low threshold than other assumptions. No effect on high threshold (Tables 4.19 - 4.21)
Use empirical arithmetic average BAFs (rather than geometric averaging) to develop sediment thresholds	Mechanistic model results and uncertainty more consistent with arithmetic averaging method	Lower incidence of exceeding thresholds for arithmetic calculations than geometric calculations (Tables 4.19 - 4.21)
Base the high threshold on an average (50%ile) estimate of bioaccumulation factor	Less conservative threshold protects the average consumer (see Section 3.2.7)	
Base the low threshold on an 95% upper confidence limit estimate of average bioaccumulation factor	More conservative threshold protects the average consumer, including a protection factor to account for uncertainty in the estimate of bioaccumulation (see Section 3.2.7)	Greater than 95% probability that the bioaccumulation estimate is equal to or greater than the actual average bioaccumulation in Newport Bay
Calculate prey lipid based on average of primary prey species	Actual prey consumption preferences are not known for Newport Bay (further discussed in Appendix W)	For humans, higher incidence of exceeding thresholds than other sets of assumptions (Tables 4.19 - 4.21). For wildlife, unknown effect.

To ascertain the impact of different assumptions on final sediment assessment, a number of scenarios were compared. These are summarized in Tables 4.17 and 4.18, and described below:

Baseline Scenarios A and AA Baseline scenarios were developed for both human and wildlife receptors (Tables 4.17 and 4.18). For the human receptor (Scenario A), prey tissue concentration thresholds were developed based on local agency requests (Appendix P). This included a variable 10^{-5} to 10^{-6} allowable increased cancer risk, and consumption rates based on Allen *et al.* (1996). The resulting tissue thresholds are summarized in Table 4.3. For the wildlife receptor (Scenario AA), prey tissue concentration thresholds were developed based on protecting least tern, which is the most sensitive wildlife endpoint[35] (Table 4.4). For both scenarios, BAFs were developed using

[35] Here sensitivity is defined based on having the lowest effects threshold, using the approach outlined in Section 3.1.

arithmetic average and upper confidence limits (following Table 4.11). Tissue percent lipid was calculated as the average of major human or wildlife prey species (Appendix W). All sediment samples collected in Newport Bay since 1997 were compared to the thresholds in the baseline scenario. Fifty-seven samples collected in four studies were evaluated (Table Q.3 in Appendix Q).

Table 4.17. Eight scenarios to evaluate effects of varying assumptions on sediment threshold development for human receptors in Newport Bay. A = arithmetic averaging; G = geometric averaging.

Scenario	Description	Prey species to estimate tissue lipid	Cancer risk factor	Fish consumption rate (g/d)	Statistical method for BAF	Sediment data included
A	Baseline scenario	All human prey	10^{-6} to 10^{-5}	21	A	All
B	Lipid estimate based on resident human prey	Resident species	10^{-6} to 10^{-5}	21	A	All
C	Lipid estimate based on catch rates in Allen *et al.* (1996)	Weighted by capture rate	10^{-6} to 10^{-5}	21	A	All
D	Subsistence fisher consumption rate (U. S. EPA 2000b)	All human prey	10^{-6} to 10^{-5}	142	A	All
E	10-fold increase in allowable cancer risk factor	All human prey	10^{-5} to 10^{-4}	21	A	All
F	Risk assumptions from Table K.1	All human prey	10^{-5}	17.5 to 32	A	All
G	Determine BAF using geometric average and error estimation	All human prey	10^{-6} to 10^{-5}	21	G	All
H	Excluding Rhine Channel data	All human prey	10^{-6} to 10^{-5}	21	A	Exclude Rhine Channel

Alternative scenarios

Varying prey species for tissue lipid estimation (Scenarios B and C). These scenarios determine the sensitivity of results to prey tissue estimated lipid content (Table 4.17). Source data and calculations for these scenarios are described in Appendix W.

In the baseline scenario for effects to human consumers, prey tissue percent lipid was calculated based on all human prey species. In these alternate scenarios, prey tissue lipid content were based on estimated dietary proportions from the Santa Monica Bay seafood consumption study (Allen *et al.* 1996) (Scenario B) or based on using resident Newport Bay fish species only (Scenario C).

Varying risk assessment policy decisions (Scenarios D, E, F, BB, and DD). The framework (and risk assessments in general) is highly sensitive to policy decisions about target population and allowable risk (Appendix K, Table K.1). Five scenarios were run to explore this issue (Tables 4.17 and 4.18). These scenarios evaluate different combinations of assumptions and their final effect on the case study results. In Scenario D, tissue thresholds are calculated based on expected consumption rates for human subsistence fishers (142 g/d) (U. S. EPA 2000b), which is more conservative than the baseline Scenario A. Scenario E modifies the allowable increased cancer risk by an order of magnitude, changing it from 10^{-5} for the lower threshold and 10^{-6} for the higher threshold to 10^{-4} and 10^{-5}, respectively. In Scenario F, thresholds are calculated based on the assumptions outlined in Table 4.2 and Appendix K (Table K.1, grey rows). These include a 10^{-5} allowable increased cancer risk and average and UCI sport fisher consumption rate estimates. This scenario results in a less conservative set of assumptions, particularly for the low threshold. Scenarios BB and DD (Table 4.18) develop thresholds based on protection of brown pelican. Because of its larger size and consequent lower specific consumption rate, brown pelican prey tissue thresholds are four times higher than least tern thresholds (Table 4.4).

Table 4.18. Five scenarios to evaluate effects of varying assumptions on sediment threshold development for wildlife receptors in Newport Bay. A = arithmetic averaging; G = geometric averaging.

Scenario	Description	Wildlife species	Statistical method for BAF	Sediment data included
AA	Baseline scenario	Least tern	A	All
BB	Larger bird species	Brown pelican	A	All
CC	Geometric method to calculate BAF	Least tern	G	All
DD	Larger bird species and geometric method	Brown pelican	G	All
EE	Exclude Rhine Channel data	Least tern	A	Exclude Rhine Channel

Calculating BAF using geometric calculations (Scenarios G, CC, and DD). BAF results differed substantially between arithmetic and geometric approaches. Using geometric methods, average and UCI BAFs were substantially higher than arithmetic methods (Table 4.11). Scenarios G, CC, and DD evaluate the impact of the geometric approach on sediment thresholds and on the consequent number of samples that exceed the thresholds. These scenarios are for humans, least terns, and brown pelicans, respectively.

Inclusion vs. exclusion of Rhine Channel sediments (Scenarios H and EE). Newport Bay exhibits spatially variable contaminant concentrations (Figures 4.6 and 4.7). For example, the highly industrialized Rhine Channel has been identified as a hot spot of contaminant concentrations and toxicity (Bay and Brown 2003, Bay *et al.* 2004, Anchor Environmental 2005). Sediment sampling locations across the Bay preferentially included the Rhine Channel, with a site-specific evaluation of Rhine Channel (Bay and Brown 2003) comprising 15 of the 57 Newport Bay sediment samples (Appendix Q, Table Q.3). To evaluate the impact of this contaminant hotspot on overall assessment

results, Scenarios H and EE were run following baseline assumptions for humans and least tern, but excluding the 15 Rhine Channel Study samples from evaluation.

4.4.3.2. Results

In the baseline scenarios, the low sediment chemistry thresholds for human health effects ranged between 1.3 and 1.9 ng/g dry weight (Tables 4.19, 4.20, and 4.21). Because detection limits for sediment compounds are around 1 ng/g in many studies (e.g., Masters and Inman 2000, Bay and Brown 2003, Bay *et al.* 2004), almost any detectable contaminant residue in sediment would exceed the low threshold. This finding supports the use of a sequential approach (Figure 2.2). The tissue chemistry LOE evaluates whether wildlife or human consumers are actually at risk due to consumption of contaminated prey in the water body. In cases where the prey tissue line of evidence indicates low risk to predators, the results of the sediment chemistry LOE would not affect the outcome of the analysis. For the Newport Bay case study, total PCBs for wildlife and total chlordanes for both humans and wildlife have already been placed in the low-risk category, based on the tissue chemistry line of evidence.

In the baseline scenarios, high sediment chemistry thresholds for human effects ranged between 22.4 and 27.3 ng/g dry weight (Scenario A in Tables 4.19 – 4.21). These thresholds are approximately ten times the low thresholds. Wildlife thresholds were generally well above the comparable thresholds for human effects. For example, the PCB low and high sediment thresholds for least tern were 76 and 2177 ng/g, approximately 50 to 100 times the human thresholds (Table 4.20). The one case where wildlife thresholds were similar to human thresholds was the low sediment threshold for DDT effects to least tern (Table 4.19; 2.1 ng/g for wildlife vs. 1.9 ng/g for humans). As discussed in Appendix H, these thresholds are based on the Navy/BTAG TRVs (California DTSC Human and Ecological Risk Division 2000), which in turn are based on field monitoring of impacts of DDT discharged off the southern California coast to brown pelicans (Anderson *et al.* 1975, Anderson *et al.* 1977).

As observed for the prey tissue line of evidence, results for the sediment chemistry LOE varied by contaminant. For DDTs, despite the variety of scenarios examined, the majority of sediments were consistently above the low sediment threshold for both human and wildlife receptors (Table 4.19). Following the assessment framework (Table 2.1; Figure 2.2), this would result in categorization of most sediments into two categories: possible impact and likely impact. For PCBs and chlordanes, results were more variable for both the low and high thresholds (Tables 4.20 and 4.21). However, the high thresholds for avian predators were not exceeded for any contaminant class (Tables 4.19, 4.20, and 4.21).

Table 4.19. Sediment threshold development assumptions and results for DDTs. Percentage of sediments above thresholds are based on actual data from Newport Bay (N = 57; see Appendix Q, Table Q.3). Proportion of baseline = how the scenario threshold compares to the relevant baseline threshold. E.g., 0.1 indicates that the new threshold is 10% of the baseline threshold.

Scenario	High tissue threshold (ng/g wet)	Low tissue threshold (ng/g wet)	Average BAF (g lipid/g dry) [a]	95% UCI BAF (g lipid/g dry) [a]	Tissue % lipid	Low sediment threshold (ng/g dry)	High sediment threshold (ng/g dry)	% sediments above low	% sediments above high	Proportion of baseline
A. Human - baseline scenario	98	9.8	191	276	1.88%	1.9	27.3	100%	63%	
B. Human resident prey only	98	9.8	191	276	0.65%	5.5	78.9	93%	11%	2.9
C. Human seafood study prop.	98	9.8	191	276	1.15%	3.1	44.6	96%	51%	1.6
D. Human subsistence consumers	14	1.4	191	276	1.88%	0.3	3.9	100%	93%	0.14
E. Human 10*risk	980	98	191	276	1.88%	18.9	272.9	75%	0%	10.0
F. Human risk Table K.1	118	64	191	276	1.88%	12.3	32.9	86%	61%	6.5
G. Human geometric averaging	98	9.8	309	1456	1.88%	0.4	16.9	100%	77%	0.2
H. Human exclude Rhine Channel	98	9.8	191	276	1.88%	1.9	27.3	100%	50%	1.0
AA. Least tern - baseline	502	8	231	315	1.19%	2.1	182.6	100%	0%	
BB. Brown pelican	2156	32	231	315	1.19%	8.5	784.3	93%	0%	4.0
CC. Least tern geometric	502	8	387	1712	1.19%	0.4	109.0	100%	4%	0.18
DD. Brown pelican geo.	2156	32	387	1712	1.19%	1.6	468.2	100%	0%	0.74
EE. Least tern exclude Rhine Channel	502	8	231	315	1.19%	2.1	182.6	100%	0%	1.0

a. From Table 4.11

In the scenario comparisons, both risk assessment assumptions and averaging method (arithmetic vs. geometric) substantially influenced sediment thresholds. Changing to geometric averaging methods (Scenario G), increasing allowable cancer risk tenfold (Scenario E), and switching to subsistence fisher consumption rates (Scenario D) each caused an order of magnitude or greater difference in sediment thresholds for DDTs, PCBs and chlordanes (Tables 4.19, 4.20, and 4.21). These changes often resulted in a different categorization of the majority of sediments. For example, when allowable increased cancer risk was switched from 10^{-5} to 10^{-4} (Scenario E), the percent of sediment samples exceeding the high human health threshold for DDTs changed from 63% to 0% (Table 4.19). Changes were particularly strong using geometric averaging for PCBs (Table 4.20). Evaluating human risk (Scenario G) and least tern risk (Scenario CC), thresholds were reduced 50 to 100 fold using geometric methods. However, because these thresholds were generally well above or well below observed contaminant

concentrations, there was little change in the number of sediments that exceeded the thresholds (Table 4.20).

Table 4.20. Sediment threshold development assumptions and results for PCBs. Percentage of sediments above thresholds are based on actual data from Newport Bay (N = 46; see Appendix Q, Table Q.3). Proportion of baseline = how the scenario threshold compares to the relevant baseline threshold.

Scenario	High tissue threshold (ng/g wet)	Low tissue threshold (ng/g wet)	Average BAF (g lipid/g dry) a	95% UCI BAF (g lipid/g dry) a	Tissue % lipid	Low sediment threshold (ng/g dry)	High sediment threshold (ng/g dry)	% sediments above low	% sediments above high	Proportion of baseline
A. Human - baseline scenario	17	1.7	34	54	1.88%	1.7	26.6	63%	37%	
B. Human resident prey only	17	1.7	34	54	0.65%	4.8	76.9	61%	26%	2.9
C. Human seafood study prop.	17	1.7	34	54	1.15%	2.7	43.5	63%	33%	1.6
D. Human subsistence cons.	2.5	0.25	34	54	1.88%	0.2	3.9	63%	61%	0.15
E. Human 10*risk	170	17	34	54	1.88%	16.7	266.0	39%	0%	10.0
F. Human risk Table K.1	20	10.9	34	54	1.88%	10.7	31.3	50%	35%	6.4
G. Human geometric averaging	17	1.7	187	4716	1.88%	0.02	4.8	63%	61%	0.01
H. Human exclude Rhine Ch.	17	1.7	34	54	1.88%	1.7	26.6	45%	10%	1.0
AA. Least tern - baseline scen.	1062	76	41	84	1.19%	76.0	2176.7	26%	0%	
BB. Brown pelican	4562	324	41	84	1.19%	324.1	9350.3	0%	0%	4.3
CC. Least tern geometric avg.	1062	76	132	4112	1.19%	1.6	676.1	63%	0%	0.02
DD. Brown pelican geo. avg.	4562	324	132	4112	1.19%	6.6	2904.3	61%	0%	0.09
EE. Least tern exclude Rhine	1062	76	41	84	1.19%	76.0	2176.7	0%	0%	1.0

a. From Table 4.11.

Changing the method of calculating prey lipid (Scenarios B and C) had less of an effect on the thresholds. Nevertheless, for DDTs, switching to the percent lipid for resident prey (Scenario B) resulted in the percentage of sediments above the high threshold declining from 63% to 11% (Table 4.19). This finding again indicates the high sensitivity of these evaluations to the chosen combinations of assumptions.

Exclusion of the Rhine Channel sediments (Scenario H) did affect the human health assessment for PCBs and chlordanes. With Rhine Channel sediments included, 63% and 37% of samples exceeded the low and high PCB thresholds, respectively. When these sediments were excluded from the analyses, the number of sediments above the thresholds declined to 45% and 10%, respectively (Table 4.20). For chlordanes (Table 4.21), the opposite effect occurred. Because all of the Rhine Channel sediments were below detection for chlordanes (Appendix Q, Table Q.3), excluding them from the

analysis actually caused the number of samples above the low threshold for humans to increase from 54% to 91%.

Table 4.21. Sediment threshold development assumptions and results for chlordanes. Percentage of sediments above thresholds are based on actual data from Newport Bay (N = 37; see Appendix Q, Table Q.3). Proportion of baseline = how the scenario threshold compares to the relevant baseline threshold.

Scenario	High tissue threshold (ng/g wet)	Low tissue threshold (ng/g wet)	Average BAF (g lipid/g dry) a	95% UCI BAF (g lipid/g dry) a	Tissue % lipid	Low sediment threshold (ng/g dry)	High sediment threshold (ng/g dry)	% sediments above low	% sediments above high	Proportion of baseline
A. Human - baseline scenario	95	9.5	226	377	1.88%	1.3	22.4	54%	5%	
B. Human resident prey only	95	9.5	226	377	0.65%	3.9	64.7	46%	0%	2.9
C. Human seafood study prop.	95	9.5	226	377	1.15%	2.2	36.6	54%	5%	1.6
D. Human subsistence cons.	14	1.4	226	377	1.88%	0.2	3.3	57%	51%	0.15
E. Human 10*risk	950	95	226	377	1.88%	13.4	223.6	27%	0%	10.0
F. Human risk Table K.1	114	63	226	377	1.88%	8.9	26.8	30%	5%	6.6
G. Human geometric avg.	95	9.5	1275	8910	1.88%	0.06	4.0	57%	46%	0.04
H. Human exclude Rhine Ch.	95	9.5	226	377	1.88%	1.3	22.4	91%	9%	1.0
AA. Least tern - baseline	5848	116	123	259	1.19%	37.6	3995	5%	0%	
BB. Brown pelican	25142	502	123	259	1.19%	163	17177	0%	0%	4.3
CC. Least tern geo. avg.	5848	116	380	4012	1.19%	2.4	1293	54%	0%	0.06
DD. Brown pelican geo. avg.	25142	502	380	4012	1.19%	10.5	5560	30%	0%	0.28
EE. Least tern exclude Rhine	5848	116	123	259	1.19%	37.6	3995	9%	0%	1.0

a. From Table 4.11.

4.4.4. Summary for sediment chemistry LOE

Based on thresholds calculated for Newport Bay, sediments were found to occur in all three risk categories, with more frequent exceedances for DDTs than PCBs or chlordanes. However, thresholds were very sensitive to a variety of assumptions and judgments. These assumptions included allowable risk, consumption rate, data statistical distribution and prey lipid. Table 4.22 categorizes the scenario results in terms of relative impact on overall conclusions for the sediment chemistry LOE.

For both the human and wildlife scenario evaluations, averaging method (arithmetic vs. geometric) strongly impacted outcome of the evaluation (Table 4.22, Scenarios G and CC). Calculation of geometric averages and standard deviations increased the uncertainty of estimates, resulting in much lower thresholds (Tables 4.19 – 4.21). For PCBs and chlordanes, these changes resulted in substantial increases in the number of

sediment samples exceeding one or both of the sediment chemistry thresholds (Tables 4.20 and 4.21).

Table 4.22. Summary of scenario evaluation. The relative impact on the outcome is summarized for each contaminant, based on proportion of baseline BAF and threshold comparison results (Tables 4.19 – 4.21). L = relatively low impact on outcome. M = moderate impact. H = relatively high impact on outcome.

Scenario	Description	Assumptions Changed	Impact of change on DDT results	Impact of change on PCB results	Impact of change on Chlordane results	Overall impact
A	Baseline scenario					
B	Lipid estimate based on resident human prey	Prey biology	M	M	L	M
C	Lipid estimate based on catch rates in Allen *et al.* (1996)	Prey biology	L	L	L	L
D	Subsistence fisher consumption rate (U. S. EPA 2000b)	Target population	H	H	H	H
E	10-fold increase in allowable cancer risk factor	Allowable risk	H	H	H	H
F	Risk assumptions from Table K.1	Allowable risk and target population	M	M	H	M
G	Determine BAF using geometric average and error estimation	Statistical assumptions	M	H	H	H
H	Excluding Rhine Channel data	Study spatial area	L	M	H	M
AA	Baseline scenario					
BB	Larger bird species	Target population	L	H	L	M
CC	Geometric method to calculate BAF	Statistical assumptions	L	H	H	H
DD	Larger bird species and geometric method	Allowable risk and statistical assumptions	L	H	M	M
EE	Exclude Rhine Channel data	Study spatial area	L	H	L	M

For humans, Scenarios D and E caused the greatest effects on outcome. These scenarios changed standard risk assessment parameters. Scenario D changed assumptions regarding the definition of the target population to protect and Scenario E changed the acceptable level of increased carcinogenic risk. Both of these parameters are strongly

influenced by policy decisions, which often vary among water bodies based on agency judgments (Hamilton and Viscusi 1999, U. S. EPA 2000b). These findings support the need for substantial efforts to engage stakeholders in framework application, to adequately resolve these policy issues (U. S. EPA 1997, 1998, Bridges *et al.* 2005).

4.5. Bioavailability line of evidence

The bioavailability line of evidence is recommended as a final step when there are questions raised as to bioavailability of sediment-associated contaminants. In particular, this line of evidence is applied when the first two lines of evidence result in intermediate risk categories (following Figure 2.2). In the Newport Bay case study, some of the sediment chemistry results combined with prey tissue chemistry did indicate intermediate levels of apparent risk (i.e., categories B and C in Figure 2.2). Based on the framework (Table 2.1, Figure 2.2), it would then be appropriate to proceed to the bioavailability line of evidence for this case study.

The purpose of the bioavailability line of evidence is to confirm that the contaminants in question are bioavailable from local sediments to biota. Literature evidence strongly indicates bioavailability of DDTs and PCBs in marine and estuarine sediments in California and elsewhere (Ferraro *et al.* 1990, Boese *et al.* 1997, Brown *et al.* 1998, Zeng and Tran 2002, Kuzyk *et al.* 2005b). Based on the framework, use of this literature information may be sufficient to assume bioavailability (following Section 3.3.1). Nevertheless, to demonstrate the method, bioaccumulation data were assembled and evaluated specifically for Newport Bay.

4.5.1. Methods

Data used to investigate bioavailability of contaminants in Newport Bay sediments were obtained from a study funded by the U. S. Army Corps of Engineers (MEC Analytical Systems 2003). The objective of this sampling and analysis program was to characterize the sediments to be dredged from Upper Newport Bay as part of a proposed restoration project. Bioaccumulation was investigated in laboratory tests on *Macoma nasuta* (clam) and *Nereis virens* (polychaete). The tests used sediments collected from eleven stations in Upper Newport Bay. Evaluation of bioaccumulation potential was not performed at Lower Newport Bay.

According to the study report (MEC Analytical Systems 2003), bioaccumulation testing followed guidance outlined in the Ocean Testing Manual (U. S. EPA and U. S. Army Corps of Engineers 1991) and U. S. EPA (1994). Homogenized sediment of known field concentration were administered to "contaminant-free" test organisms in a laboratory environment, with test conditions monitored throughout the experiments. Tissue analyses were subsequently performed after 28 days to determine the availability of sediment contaminants taken up by the test organisms. These 28 day Tier III bioaccumulation tests are generally considered to be sufficient to assess the potential for bioaccumulation, though equilibrium may not be reached in the 28 day time period (McFarland 1998, U. S. EPA 2000a).

The analyses were used to demonstrate whether a consistent relationship between biota contaminant concentration and sediment contaminant concentration could be shown. All tissue values for *Nereis virens* were below detection. Therefore, only *Macoma nasuta* could be evaluated for the bioavailability of contaminants. The arithmetic mean final p,p'-DDE concentration in *M. nasuta* from each station was compared to the administered homogenized-sediment concentration. Individual sample concentrations that were below detection limits were treated as half the detection limit. For p,p'-DDE, analysis methods employed in this study were sufficient to detect concentrations above 2 μg/kg. Methods to evaluate levels of PCBs, chlordanes, and dieldrin were only sufficient to detect concentrations above 5 μg/kg.

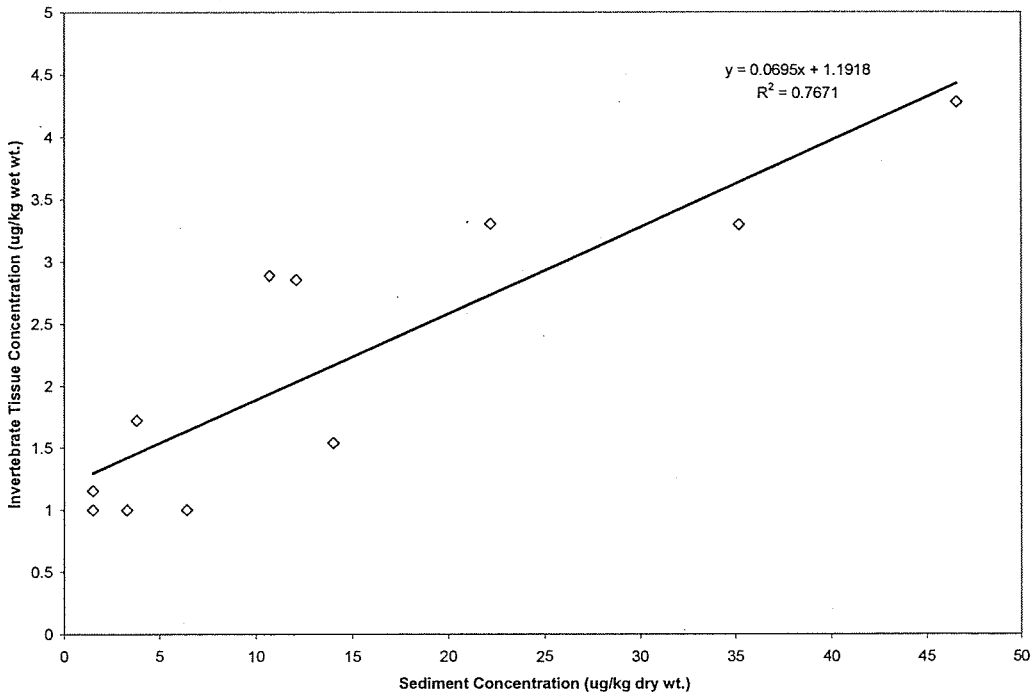
Linear regression analysis was applied to compare sediment concentration to *M. nasuta* tissue values (following Section 3.3.2). Data sufficiently met assumptions of normality, equal variance, and independence. A significant positive relationship in this comparison would indicate that a compound is bioaccumulating and is therefore bioavailable to *M. nasuta*.

4.5.2. Results

A statistically significant linear relationship ($R^2 = 0.77$, $p = 0.0004$) was observed in p,p'-DDE (Figure 4.20). The lowest sediment and tissue concentrations shown at the bottom-left of the plot represent the non-detectable results. The regression indicated a positive relationship between sediment and tissue p,p'-DDE concentration; as sediment concentration increases, there was a general linear increase in tissue concentration. The results were still significant when the values below detection were removed from the analysis.

Concentrations in all samples were below detection for PCBs, chlordanes, and dieldrin. However, the detection limits for these compounds were relatively high. The detection limits are 5 ng/g, which are higher than some of the sediment and prey tissue thresholds (Tables 4.3, 4.19 - 4.21). Therefore, bioaccumulation test results were inconclusive for these compounds.

Figure 4.20. Linear regression of sediment p,p'-DDE concentration vs. *Macoma nasuta* p,p'-DDE concentration.



4.6. Final assessment of Newport Bay sediments

To illustrate the framework, a "final assessment" of Newport Bay sediments may be conducted based on the baseline scenario assumptions laid out throughout this section (Tables 4.3, 4.5, 4.9, and 4.16 - 4.18). The intent of this assessment is for illustrative purposes only. The results of the prey tissue and sediment chemistry lines of evidence were particularly sensitive to many scientific and policy assumptions, and should be viewed in this light.

When results for all lines of evidence were combined following Table 2.1, the assessment framework indicated unlikely impact of sediment chlordanes to local wildlife or humans, and unlikely impact of PCBs to local wildlife (Table 4.23). This designation resulted from the prey tissue line of evidence being below the low threshold. The assessment framework indicated possible impact of DDTs to birds, because the prey tissue and sediment chemistry lines of evidence were both intermediate between the low and high thresholds. For impacts of DDTs and PCBs to humans, the assessment framework varied for individual stations among unlikely impact, possible impact, and likely impact. No sediments were categorized as having clear impact (the highest risk category). This is because average prey tissue concentrations were always below the high threshold (following Table 2.1). In this case study, the bioaccumulation test results did not change the initial findings reached with the prey tissue and sediment chemistry lines of evidence.

This is because the test either indicated bioavailability (for DDTs) or was inconclusive due to high detection limits (for PCBs and chlordanes).

Evaluating impacts to humans, distinct spatial patterns were observed in sediment assessment results. PCBs exhibited the higher risk category (likely impact) in sediments within and adjacent to the Rhine Channel, the "possible impact" category along the channels in lower Newport Bay, and the "unlikely impact" category throughout upper Newport Bay (Figure 4.21). DDT results varied between two impact categories, with "possible impact" and "likely impact" samples located throughout upper and lower Newport Bay (Figure 4.22).

In closing, it should be stated that the assessment framework is not designed to indicate whether remediation is needed; this is a management decision that must be influenced by policy and economic considerations beyond the scope of the framework. Nor does the assessment framework evaluate among remediation alternatives, such as dredging, capping, best management practices or monitored natural recovery. Nevertheless, the spatial pattern of sediment categories does indicate that for PCBs, relatively contaminated areas are all in the same location. This finding may have implications for the relative costs and benefits of specific remediation alternatives, if remediation is determined to be an appropriate course of action.

Figure 4.21. Final characterization of Newport Bay sediments for PCBs following the baseline scenario assumptions for risk to humans. Green squares = unlikely impact. Yellow diamonds = possible impact. Red circles = likely impact.

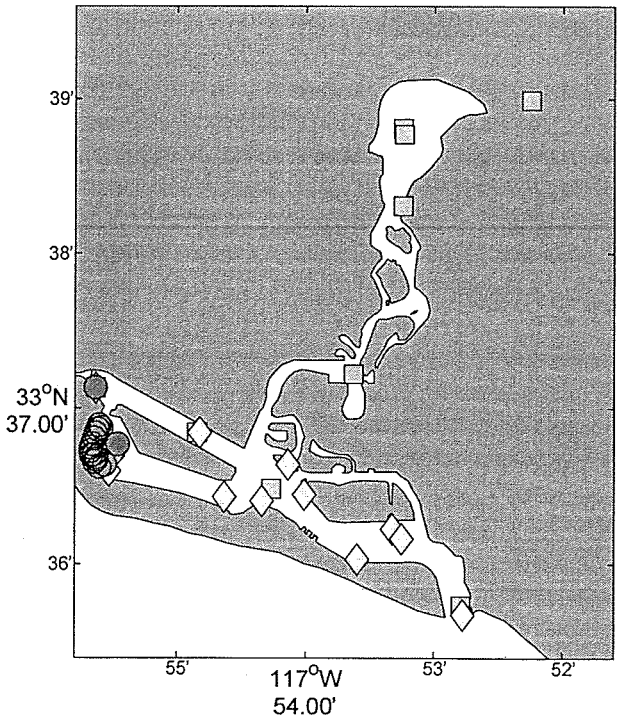


Figure 4.22. Final characterization of Newport Bay sediments for DDTs following the baseline scenario assumptions for risk to humans. Yellow diamonds = possible impact. Red circles = likely impact.

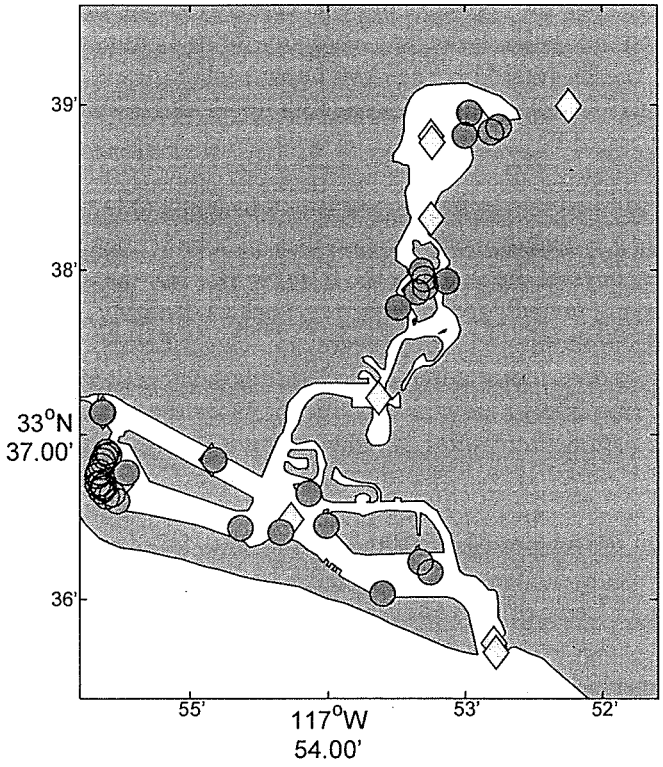


Table 4.23. Summary of results from individual lines of evidence and final characterization of Newport Bay sediments.

Compound	Receptor	Field prey tissue chemistry	Sediment chemistry			Bioavailability	Final determination[a]
			Below low	Between	Above high		
DDT	Humans	Between thresholds	0/57	21/57	36/57	Bioavailable	B. Possible impact (21 samples); C. Likely impact (36 samples)
DDT	Birds	Between thresholds	0/57	57/57	0/57	Bioavailable	B. Possible impact
PCBs	Humans	Between thresholds	17/46	12/46	17/46	Inconclusive	A. Unlikely impact (17 samples); B. Possible impact (12 samples); C. Likely impact (17 samples)
PCBs	Birds	Below low threshold	34/46	12/46	0/46	Inconclusive	A. Unlikely impact
Chlordanes	Humans	Below low threshold	17/37	18/37	2/37	Inconclusive	A. Unlikely impact
Chlordanes	Birds	Below low threshold	35/37	2/37	0/37	Inconclusive	A. Unlikely impact

a. Following Section 2.4

5. Application of the assessment framework to evaluate sediments in San Francisco Bay

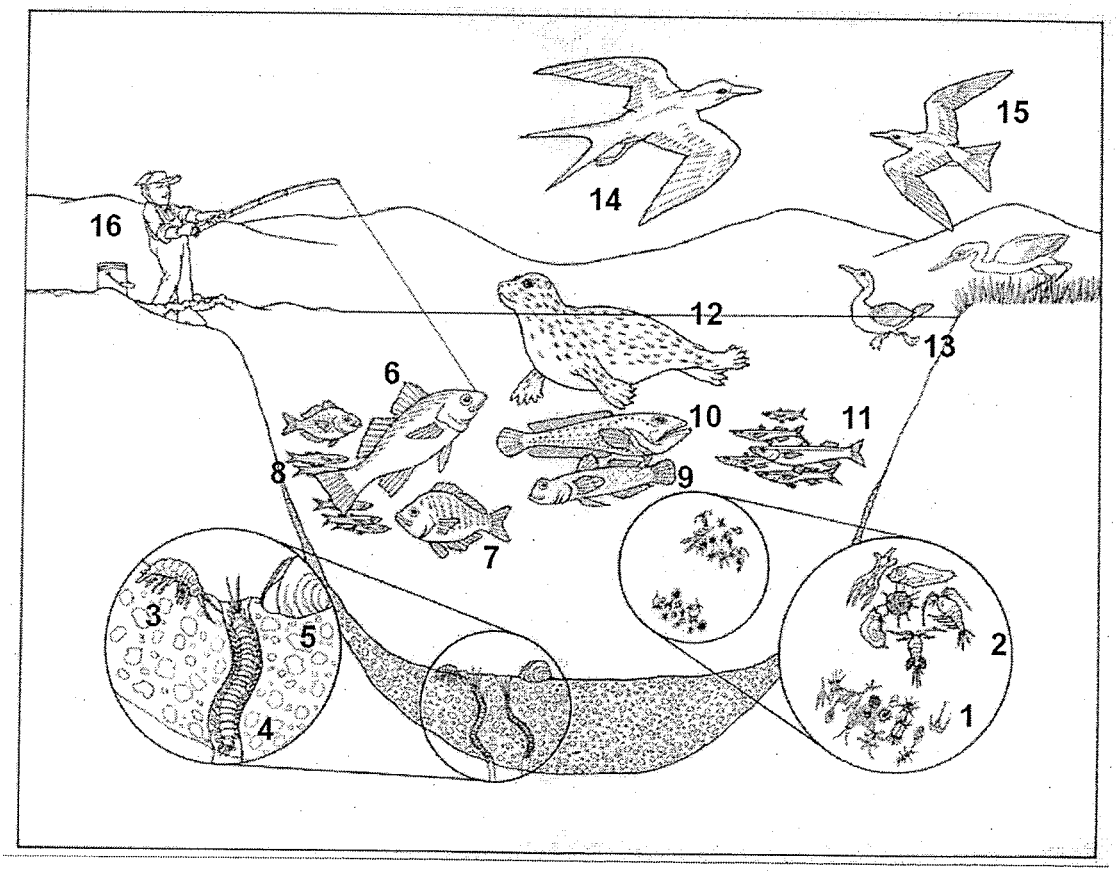
For the San Francisco Bay case study, the assessment framework will be applied to evaluate human health risks from three legacy pesticides: DDTs, chlordanes, and dieldrin. The case study demonstrates application of the framework in a water body having a large historical data set. Due to the extent and breadth of the data set, it is possible to demonstrate several techniques. Using empirical data, a representative fish species is selected for bioaccumulation factor development. The bioaccumulation factor is developed using spatially explicit information on the distribution and expected movement range of the fish. The empirical results are compared to the mechanistic model for pesticides. The model has previously been applied to San Francisco Bay for PCBs (Gobas and Wilcockson 2002, Gobas and Arnot 2005). The mechanistic model is also used to simulate the relative influence of water column vs. sediment contaminants on bioaccumulation. Finally, 28 day laboratory test results are used to illustrate another example of bioavailability of sediment associated DDTs.

5.1. Conceptual model for San Francisco Bay

San Francisco Bay is the largest estuary on the Pacific coast of the Americas, extending from the confluence of the Sacramento and San Joaquin Rivers to the Golden Gate, where the Bay meets the Pacific Ocean. The pesticides DDTs, chlordanes, and dieldrin remain in the San Francisco Estuary as a result of agricultural and residential applications from the early to late 20th century (Connor *et al.* 2006). The Bay is listed as impaired by legacy pesticides on California's 303(d) list because of an interim fish consumption advisory developed by OEHHA. The advisory was based on a 1994 fish tissue study (SFBRWQCB 1995), which indicated that legacy pesticides, PCBs, mercury, and dioxins were present at levels of potential concern. Sources of pesticides entering the Bay include runoff from the Central Valley, runoff from local watersheds, municipal and industrial discharges, atmospheric deposition, erosion of sediments buried beneath the active sediment layer, and dredging and disposal of deep sediments (Connor *et al.* 2006). The Regional Monitoring Program for Water Quality (RMP), a comprehensive contaminant and water quality monitoring program, has collected annual data on legacy pesticide concentrations in San Francisco Bay water, sediments, and bivalves since 1993 (SFEI 2005). The RMP also monitors concentrations of legacy pesticides in sport fish every three years, with the most recent sampling event in 2003 (Connor *et al.* 2006).

A conceptual model for the San Francisco Bay food webs has been described elsewhere (Gobas and Arnot 2005, Davis *et al.* 2006a) and is summarized in Figure 5.1.

Figure 5.1. Conceptual model for the San Francisco Bay food web. Legacy pollutants enter the food web via accumulation by phytoplankton (1) at the base of the food web. Concentrations then increase with each step up the food web, reaching maximum concentrations and posing the greatest health risks in species that consume Bay fish. Phytoplankton (1) are consumed by small animals including zooplankton (2) and invertebrates such as amphipods (3), worms (4), or clams (5). Invertebrates in the sediment also accumulate contaminants directly from sediment through ingestion of particles and from contact with sediment porewater. Fish consume the zooplankton and invertebrates and receive a higher dose of bioaccumulative compounds. Humans (16) and wildlife species consume the fish and receive higher doses. Wildlife consume smaller fish species such as yellowfin goby (9), plainfin midshipmen (10), and anchovy (11). Humans consume larger species such as white croaker (6), shiner surfperch (7), and jacksmelt (8). Wildlife species of concern for bioaccumulation and effects include harbor seals (12), cormorants (13), Forster's terns (14), and the endangered least tern (15). Figure and caption reprinted from Davis *et al.* (2006a).



5.2. Field prey tissue line of evidence

5.2.1. Methods

5.2.1.1. Database preparation

Legacy pesticides in San Francisco Bay have been shown to change significantly over time (Gunther *et al.* 1999, Greenfield *et al.* 2005, Connor *et al.* 2006, O'Connor and

Lauenstein 2006), necessitating use of data from a limited time horizon. For the prey tissue line of evidence, data were compiled from the RMP 2000 and 2003 fish sampling program[36]. All samples in this program are species consumed by local fishers (SFEI 2000), and are prepared for contaminant analysis by filleting, using preparation methods appropriate for human consumption (Davis *et al.* 1999, Greenfield *et al.* 2003).

5.2.1.2. Prey species

The Regional Monitoring Program samples seven species that are known to be consumed by human sport fishers (Davis *et al.* 1999, SFEI 2000) (Table 5.1). Four of these species (shiner surfperch, white croaker, California halibut, and leopard shark) exhibit life history traits that make them appropriate for the prey tissue line of evidence, including dietary benthic association and residence within the Bay for the majority of their life history (see Section 3.1.4 and Appendix B.). For example, contaminant and stable isotope analyses indicate that leopard shark captured within San Francisco Bay generally reside within a specific subembayment, rather than migrating between the Bay and coast (Greenfield *et al.* 2002, Greenfield *et al.* 2003). Total pesticide residues were calculated for these four species combined (N = 87 samples total) and separately calculated for all seven species combined (N = 135 samples total; Table 5.1).

Table 5.1. Potential fish species for tissue concentration evaluation in San Francisco Bay. Species in boldface fit criteria outlined in Section 3.1.4. Bay Resident = species likely resides within Bay throughout most of its lifespan.

Species	Bay resident?	Tissue type	Diet
California halibut	**Yes**	**Skin-off fillet**	**Pelagic/epibenthic**
Jacksmelt	Yes	Skin-on fillet	Pelagic
Leopard shark	**Yes**	**Skin-off fillet**	**Epibenthic**
Shiner surfperch	**Yes**	**Skin-on fillet**	**Epibenthic**
Striped bass	No	Skin-off fillet	Pelagic
White croaker	**Yes**	**Skin-on fillet**	**Pelagic/epibenthic**
White sturgeon	No	Skin-off fillet	Epibenthic

5.2.1.3. Prey sizes and tissue type

The RMP focuses on capturing fish that are legal sized for human consumption (Davis *et al.* 1999, Greenfield *et al.* 2003). Therefore, all fish evaluated for the prey tissue chemistry line of evidence were legal sized in accordance with state regulations (California Department of Fish and Game 2006). Samples evaluated for human health risk were analyzed as either skin-on or skin-off fillet samples, following the common preparation methods for particular species (Davis *et al.* 1999, Greenfield *et al.* 2003).

[36] Data collected for the RMP in 1994 and 1997 and other programs prior to 2000 were excluded due to concerns about trends over time.

5.2.1.4. Prey tissue thresholds

The San Francisco Bay Regional Water Quality Control Board did not have specific guidance regarding appropriate tissue thresholds for the case study (Fred Hetzel, SF Bay Regional Water Board, *Pers. comm.*). In the absence of local guidance to deviate from general thresholds, the case study evaluations were conducted following general assumptions and background information in Section 3.1 and Appendix K (Table K.1, grey rows).

For the case study, the thresholds were set to protect sport fish consumers at a 10^{-5} cancer risk factor. The high tissue threshold was based on the U. S. EPA (2000b) recommended average consumption rate for sport fish consumers of 17.5 g/d, which should protect the average sport fish consumer in San Francisco Bay (SFEI 2000). The low tissue threshold is based on protecting the 95 percentile of sport fish consumers (32 g/d; SFEI 2000, OEHHA 2001). The resulting categories for the prey tissue line of evidence are outlined in Table 5.2.

Table 5.2. Description of three categories for prey tissue line of evidence for human receptors.

Fish Tissue Score	General description	Humans	Humans
		Threshold	Interpretation
▲ Low exposure	95% upper confidence limit of average fish tissue concentration is below the low toxicity threshold	Fish tissue threshold based on protecting the 95 percentile of sport fish consumers such that no more than 1 in 100,000 faces an increased cancer risk	Using a conservative set of assumptions, dietary exposure to fish in the water body poses a low risk to the general public and to fishers who consume their catch
◘ Moderate exposure	Tissue concentration is intermediate between low and high threshold	Both thresholds are relevant	Dietary exposure to fish in a water body may pose a risk to fishers who consume their catch
● High exposure	Average fish tissue concentration is above the high toxicity threshold	Fish tissue threshold based on protecting the average sport fish consumer such that no more than 1 in 100,000 faces an increased cancer risk	Dietary exposure to fish in a water body is likely to pose an increased cancer risk of more than 1 in 100,000 for sport fish consumers

5.2.1.5. Calculation of average and 95% upper confidence interval concentrations

Arithmetic mean and 95% UCI of average concentrations were calculated for prey tissue chemistry. Geometric mean and 95% UCI were also calculated, following Appendix G.

General assumptions in the prey tissue line of evidence evaluation are summarized in Table 5.3.

Table 5.3. Summary of assumptions, their basis, and effects in prey tissue LOE evaluation for the San Francisco Bay case study.

Decision or assumption	Basis	Effect
Evaluate two alternative groups of species for prey tissue line of evidence: 1. all routinely monitored species, and 2. only species with sediment association and permanent Bay residence	Evaluate sensitivity of assessment results to sport fish species selection	Slight effect on calculated tissue chemistry residues. No effect on comparison to thresholds
Pooling fish tissue concentrations for multiple prey species	Limited sample size for looking at individual species. Conceptual model of exposure risk and available data indicate that receptors (humans) consume multiple prey	Simplifies tissue concentration calculation. Not examining exposure separately for individual prey species
Focusing on finfish prey, rather than shellfish	Use of finfish prey is likely to be more protective for main contaminants of concern (legacy pesticides and PCBs). Limited data availability for prey shellfish. Time and budgetary constraints preclude evaluating all possible prey species	Uncertain
Only including samples with sizes likely to be consumed by receptor. i.e., legal capture sizes for humans	Not appropriate to use surrogate prey species unlikely to be consumed	No effect for human health evaluation since program targets legal sized fish species
For human health evaluation, fish consumption rate based on average and 95th percentile of sport fish consumer consumption rates	San Francisco Bay Regional Board staff did not request specific risk assumptions. Following risk assumptions outlined in Section 3.1.9 and Appendix K, based on local consumption rate data (SFEI, 2000)	Thresholds are higher than if a subsistence fisher consumption rate were used, and lower than if a general population consumption rate were used (see Table K.1).
Carcinogen risk thresholds based on 10^{-5} allowable increased risk	San Francisco Bay Regional Board staff did not request specific risk assumptions. Following risk assumptions outlined in Appendix K, Table K.1, grey rows	Thresholds are 10-fold higher than if 10^{-6} carcinogen risk were used; 10-fold lower than if 10^{-4} carcinogen risk were used
Calculate tissue chemistry average based on arithmetic mean	Arithmetic averages better reflect the average overall exposure due to consuming multiple fish (Appendix G)	Risk determination unchanged given other sets of assumptions (Tables 5.4 and 5.5)
Calculate tissue chemistry 95% UCI based on 2 times the arithmetic estimate of standard error, rather than standard deviation	Use of standard error better reflects the average overall exposure due to consuming multiple fish (Section 3.1.9)	95% UCI estimated exposure less conservative than if standard deviation were used
Only using samples prepared according to predators' consumption pattern	Humans generally do not consume whole fish, and wildlife predators do. Contaminant concentration estimates will be related to tissue preparation	Sample size unchanged because most samples were prepared appropriately for predator consumption. Preparation method may cause increase or decrease in estimated tissue contaminant concentration

5.2.2. Results

Results indicated relatively low concentrations for total DDTs, total chlordanes, and dieldrin. For evaluation of potential human health effects, fish tissue concentrations were below thresholds when all seven human prey species were examined (Table 5.4). Concentrations were also below thresholds for just the four species selected for benthic association and residence within the Bay (Table 5.5). However, variability of predicted

average concentrations was greater for the four species (SE = 2.8) than when all seven species were combined (SE = 2.1), because of the smaller sample size.

Based on the assessment framework, these results indicate that prey tissue concentrations of legacy pesticides pose a low risk to human sport fishers of San Francisco Bay. Therefore, evaluation of the remaining lines of evidence wouldn't be necessary (Table 2.1, Figure 2.2). Results were slightly higher for the four species with strong sediment association (Table 5.5) than all species (Table 5.4); this discrepancy likely resulted from the relatively high tissue lipid content of two of the sediment-associated species, shiner surfperch and white croaker (Davis *et al.* 2002).

Table 5.4. Estimated total organochlorine pesticide concentrations in San Francisco Bay fish samples that are potential prey for human sport fish consumers. Concentrations were calculated for seven fish species (Table 5.1). Data are for all RMP sport fish samples from 2000 and 2003 (N = 135). 95 % UCI = average + two standard errors. Geo mean = geometric mean (calculated on log scale). Note: no values exceeded relevant thresholds.

Value	DDTs (ng/g wet)	Chlordanes (ng/g wet)	Dieldrin (ng/g wet)	Relevant Threshold
Low threshold[a]	64	63	1.4	
High threshold[a]	118	114	2.5	
Mean	31.8	5.3	0.7	High
Standard Error	2.1	0.6	0.1	
95% UCI	36.0	6.4	0.9	Low
Geo Mean	20.4	2.8	0.5	High
Standard Error[b]	0.0	0.0	0.0	
95% UCI	25.2	3.6	0.6	Low
Median	26.4	2.2	0.0	

a. From Table K.1, grey rows. b. presented in log-scale units, rounded to nearest decimal

Tissue concentrations of total DDTs in San Francisco Bay fish (arithmetic average for all fish = 32 ng/g) were considerably lower than Newport Bay (94 ng/g), indicating lower overall exposure to humans and other piscivores. It is worth noting that the 95% UCI of average DDTs in San Francisco Bay fish would be well above the 9.8 ng/g human health risk threshold used in the Newport Bay case study, highlighting the impact of policy choices (allowable risk and target population to protect) on the outcome of this assessment framework[37].

Although the average concentrations were well below the thresholds, concentrations in individual samples sometimes exceeded the low threshold for DDTs (64 ng/g; Figure 5.2) and other legacy pesticides. The human health tissue thresholds used in this case study would be protective of the average and 95th percentile of sport fish consumers, assuming

[37] As presented in Section 4, the choice of low tissue DDT thresholds (9.8 vs. 63 ng/g wet) would not impact the outcome of the Newport Bay case study as the 95% UCI of the average concentration is above both thresholds.

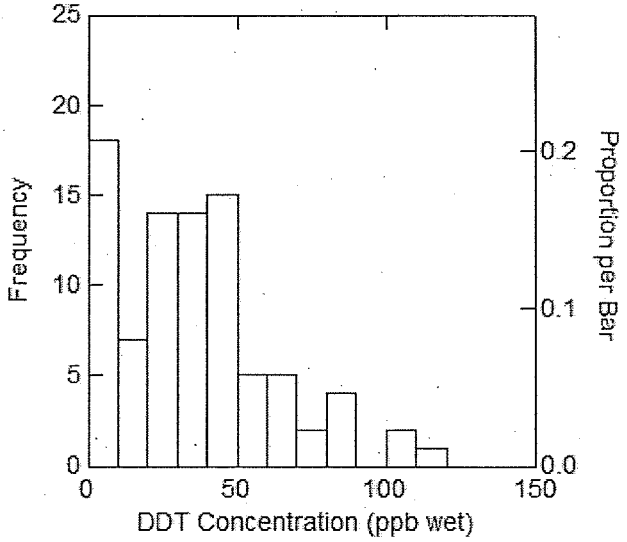
that their consumption samples across the distribution of sport fish present in the Bay. It follows that focusing on the results of individual samples that exceed the threshold would not be appropriate for the framework.

Table 5.5. Estimated total organochlorine pesticide concentrations in San Francisco Bay fish samples calculated using different averaging methods and different sample inclusion criteria. Data are for RMP species sampled in 2000 and 2003 that have life history attributes appropriate for sediment quality assessment (N = 87; Table 5.1). 95 % UCI = average + two standard errors. Geo mean = geometric mean (calculated on log scale). As in Table 5.4, no values exceeded relevant thresholds.

Value	DDTs (ng/g wet)	Chlordanes (ng/g wet)	Dieldrin (ng/g wet)	Relevant Threshold
Low threshold[a]	64	63	1.4	
High threshold[a]	118	114	2.5	
Mean	34.9	6.7	0.9	High
Standard Error	2.8	0.7	0.1	
95% UCI	40.5	8.1	1.1	Low
Geo Mean	20.3	4.0	0.6	High
Standard Error[b]	0.1	0.0	0.0	
95% UCI	27.5	3.8	0.7	Low
Median	33.6	5.5	0.0	

a. From Table K.1, grey rows. b. presented in log-scale units, rounded to nearest decimal

Figure 5.2. Frequency histogram of total DDTs in four sport fish species sampled in San Francisco Bay. Results include California halibut, shiner surfperch, white croaker, and leopard shark sampled in 2000 and 2003 (N = 87).



5.3. Sediment chemistry line of evidence

The evaluation of the prey tissue line of evidence indicated that 95% upper confidence of the average sport fish tissue concentrations were below the low threshold for sport fish consumers. Based on the assessment framework (Table 2.1; Figure 2.2), the evaluation would be complete for those legacy pesticide compounds, with the final determination that indirect effects of sediments are unlikely to pose a risk to humans. It is therefore not necessary to proceed to the sediment chemistry line of evidence.

The purpose of the case studies is to demonstrate technical issues that arise in applying the framework. To this end, we will proceed with developing the remaining lines of evidence, even though sequential application of the framework renders them unnecessary in this case. To conserve time and space, the remaining lines of evidence will be illustrated for total DDTs only. As in the previous case study, several steps are undertaken in the sediment chemistry line of evidence: development of empirical BAFs, comparing them to mechanistic model results, development of sediment thresholds, and comparing actual sediment concentrations to these thresholds.

5.3.1. Development of empirical bioaccumulation factor

In contrast to Newport Bay, San Francisco Bay has sufficient data to make highly specific choices regarding data selection and analysis. In particular, San Francisco Bay has a very large number of samples analyzed for sediment and tissue concentration. Sediment data include samples from the RMP (SFEI 2005), as well as the NOAA - EMAP (Environmental Monitoring and Assessment Program), conducted in 2000 and 2001 (U. S. EPA 2006a) (Figure 5.3). Both of these studies employed a random sampling design. Data from these studies were used in BAF development.

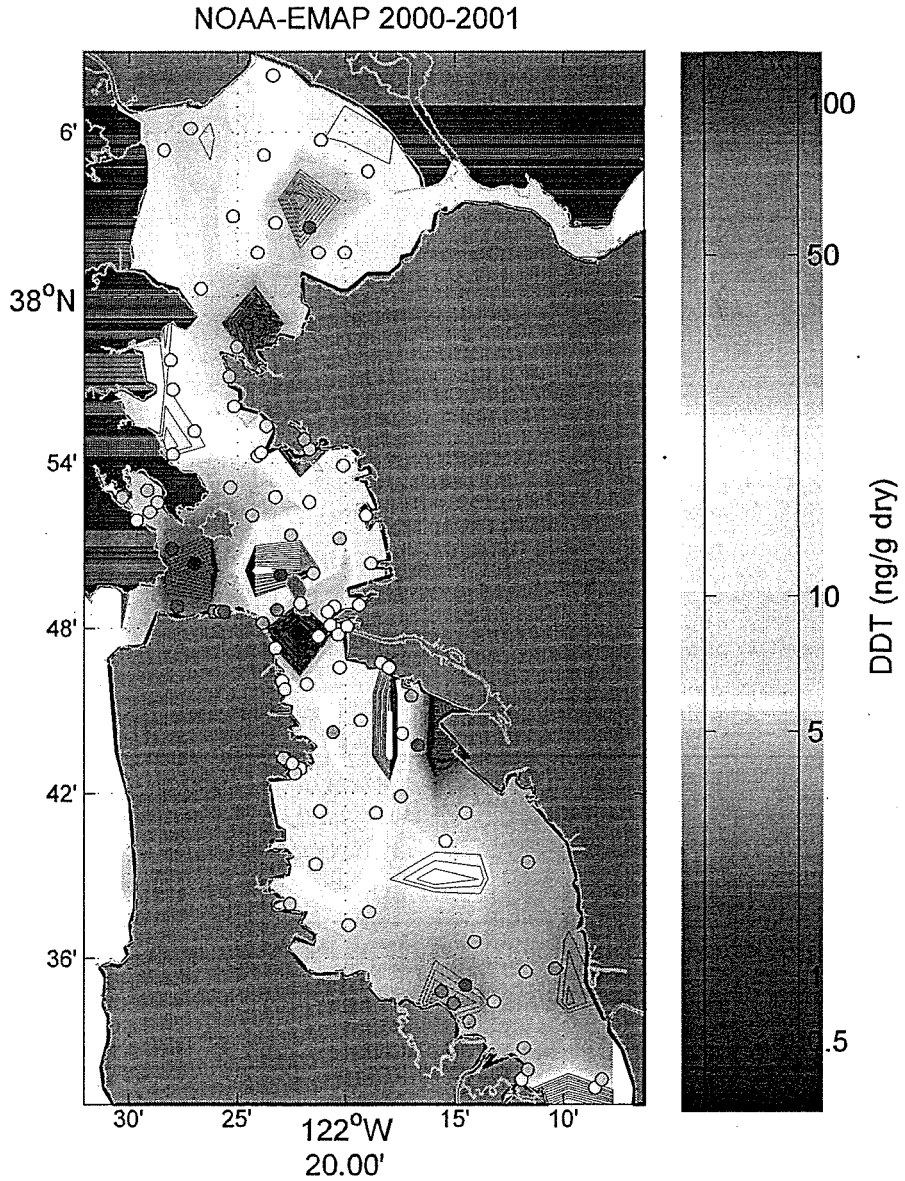
5.3.1.1. Selection and use of a surrogate prey species to develop a BAF

Fish data included large sample sizes ($n > 30$) for each of the multiple fish species(e.g., Greenfield *et al.* 2003). Given this fact, it is possible to develop empirical BAFs using a representative fish species as a surrogate for overall sediment exposure to prey fish (Section 3.1.8; Appendix F). The ideal surrogate species would have a benthic dietary association, a high probability of residing within the Bay throughout its life history, and be eaten as prey by the receptor (Section 3.1.4). As discussed elsewhere (Section 3.1.4, Table 5.1, and Appendix B), shiner surfperch, white croaker, and California halibut all have benthic dietary associations. Furthermore, they are all consumed by sport and subsistence fishers in the region (SFEI 2000).

White croaker, California halibut, and shiner surfperch were examined using a spatial regression analysis described in Appendix B to determine which had tissue contaminant residues significantly associated with nearby sediments. The results indicated that shiner surfperch had the strongest spatial associations between tissue and sediment concentrations. For shiner surfperch, R^2 ranged between 0.25 and 0.44 for PCBs, chlordanes, DDTs, and dieldrin, with p values less than 0.005 in all cases (e.g., Figure

B.2). Furthermore, shiner surfperch exhibited spatially distinct contamination (Figure 5.4) and stable isotope signatures[38] (Figure 5.5) in specific sampling locations. This combination of distinct contaminant and stable isotope signatures among sampling locations further support the contention that shiner surfperch do not move among different locations in San Francisco Bay.

Figure 5.3 Contour plot of total DDTs in San Francisco Bay sediments, based on results of the NOAA-EMAP study.



[38] Stable nitrogen and carbon isotopes are biochemical tracers that often exhibit spatial variation according to food source and geochemical baseline (Peterson and Fry 1987). They may be used to infer fish migration or site fidelity (e.g., Hesslein *et al.* 1991).

Figure 5.4. Total PCBs in shiner surfperch sampled in six San Francisco Bay locations in 2000. Each data point represents a composite of 20 individual fish. These results indicated highly significant differences among sampling locations (ANOVA $p < 0.01$). Reprinted from Greenfield *et al.* (2003).

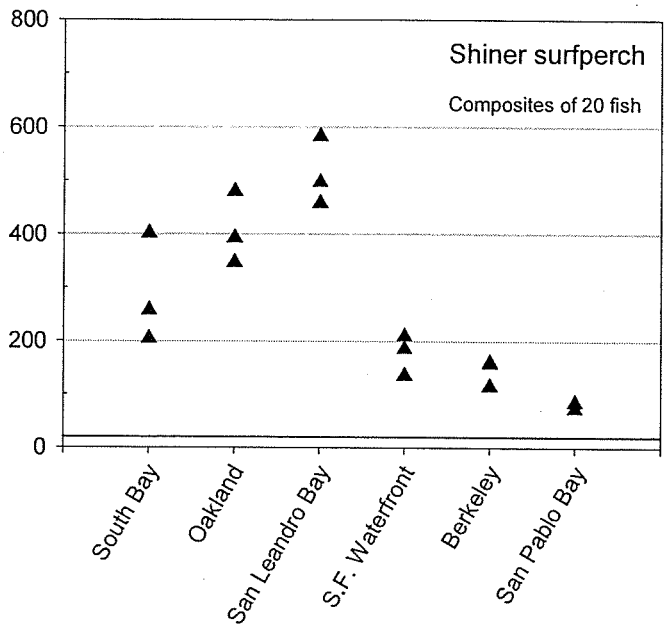
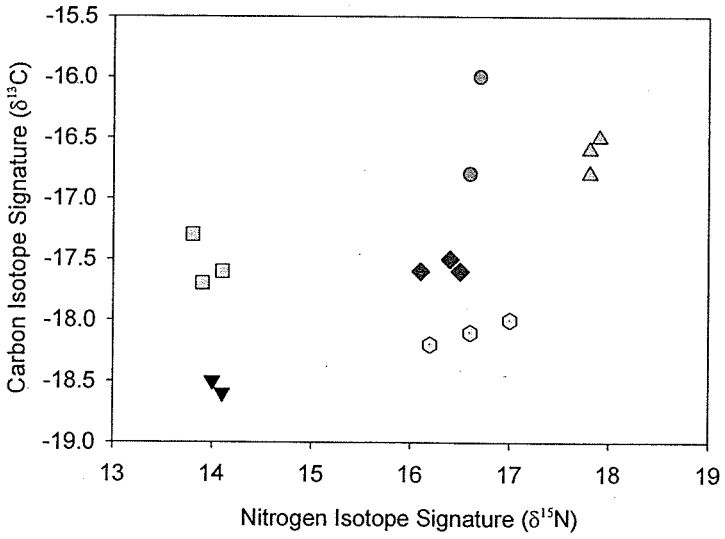
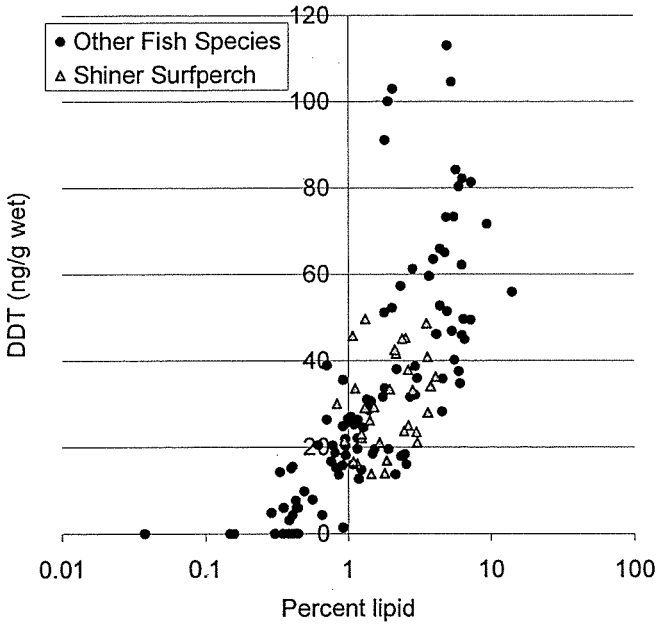


Figure 5.5. Stable carbon and nitrogen isotope signatures in shiner surfperch sampled in six San Francisco Bay locations in 2000. Isotope analyses followed standard protocols (Peterson and Fry 1987). Each data point represents a composite of 20 individual fish. Sampling locations are Berkeley Marina (black triangles), Oakland Inner Harbor (red circles), San Francisco Waterfront (green squares), San Leandro Bay (grey triangles), San Pablo Bay (blue diamonds), and South Bay (yellow hexagons).



Another consideration in selecting a surrogate species is that they should represent the average contaminant exposure to the receptor (in this case, humans). Figure 5.6 illustrates that observed concentrations of DDTs and other organochlorine compounds in shiner surf perch are centered about the average concentration among Bay sport fish in general. In summary, shiner surfperch have several desirable traits for a surrogate species: 1. a spatial association with the benthic food web of a particular location; 2. DDT exposure that is representative of finfish in the water body; and 3. a reasonably large data set which includes co-occurring sediment chemistry data. Therefore, shiner surfperch were used in the case study to estimate a representative empirical BAF for DDTs in San Francisco Bay.

Figure 5.6. DDT tissue concentrations vs. tissue percent lipid for shiner surfperch (pink triangles) and 6 other species routinely monitored in San Francisco Bay (Greenfield *et al.* 2003) (blue circles).



5.3.1.2. BAF development

Empirical BAFs were generated by comparing shiner surfperch contaminant concentrations to concentrations in surrounding sediments. Linear regression was conducted to determine the optimal spatial scale for averaging sediments around the fish (following Appendix L). The results of this analysis indicated the strongest relationships when surfperch were compared to sediments in a 1 km radius disk surrounding each fish sampling location. Lipid and organic carbon normalizations did not improve BAF calculations (data not shown). Therefore, BAF was calculated as:

BAF = wet weight tissue concentration/dry weight sediment concentration

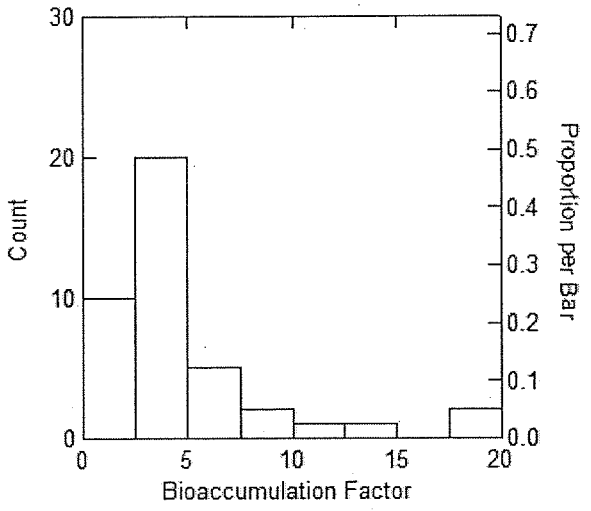
The results for multiple fish samples were combined to generate a distribution of BAFs. Table 5.6 summarizes the assumptions and decisions that went into the BAF calculation, their rationale, and probable impact on results. As illustrated in the Newport Bay case study, changes in many of the assumptions in Table 5.6 could substantially impact the sediment evaluation.

Table 5.6. Summary of assumptions, their basis, and effects on developing a bioaccumulation factor for the sediment chemistry LOE, San Francisco Bay DDT case study.

Decision or Assumption	Basis	Effect
Develop BAF based on shiner surfperch	Sufficient sample size to use a single representative species to develop BAF. Shiner surfperch has appropriate life history attributes. These include a small movement range, benthic dietary association, human sport fish consumption, and spatial association with sediments	Reduces variability and simplifies bioaccumulation calculation by focusing on a single species
Develop BAF based on arithmetic average concentrations in fish vs. average concentrations in sediment samples in a disk within 1-km radius of each fish site	Spatial scale-dependant regression model indicates a significant relationship between fish and nearby sediments, with the strongest relationship at 1-km scale	Reduces variability in bioaccumulation calculation
Using wet weight tissue concentrations	Lipid normalization does not improve the BAF calculation	Limited effect on results
Not normalizing sediment concentrations for organic carbon	Organic carbon normalization does not improve the BAF calculation	Limited effect on results
Only accept compounds in sediment for which > 25% of samples are above detection limits	Confidence of average estimate is very low when vast majority of samples are below detection	Not calculating BAF for o,p'-DDE, heptachlor, or heptachlor epoxide
Calculate bioaccumulation factor for the sum of compounds, rather than for each individual compound	Effects comparisons are on a summed contaminant basis	Calculation of average and variation in bioaccumulation factor more straightforward
Use of 2 * standard error rather than standard deviation to calculate 95% confidence interval of estimates	The objective is to calculate the central tendency and uncertainty about that central tendency for bioaccumulation in the water body (Section 3.1.9)	The calculated uncertainty in estimated bioaccumulation factor will be lower using standard error, rather than standard deviation
Only using samples prepared according to predators' consumption pattern	Humans generally do not consume whole fish and contaminant concentration estimates will be related to tissue preparation method	Sample size unchanged because most samples were prepared appropriately for predator consumption. Preparation method may cause increase or decrease in estimated tissue contaminant concentration

BAFs for total DDTs exhibited a skewed distribution, with most values below 5, and a small number of values ranging up to around 20 (Figure 5.7). The resulting arithmetic mean empirical BAF was 5.0. Although individual BAF results varied from 0.6 to 20, the standard error of the mean BAF was only 0.7. Using this standard error, the resulting 95% upper confidence interval of the mean BAF was 6.4.

Figure 5.7. Histogram of bioaccumulation factors determined for shiner surfperch vs. surrounding sediments (surrounding circle 1 km radius) in San Francisco Bay.



5.3.2. Comparison of empirical bioaccumulation to mechanistic model

The mechanistic model is used to corroborate the empirical relationships between sediment and biota exposure. To this end, several fish and bivalves species are compared.

5.3.2.1. Model description and parameter development

The mechanistic model is described in Section 3.2.8, with model equations summarized in Appendix M. It has previously been parameterized to calculate uptake of PCBs in several fish and wildlife species in San Francisco Bay as part of the PCB TMDL (Total Maximum Daily Load) studies (Gobas and Wilcockson 2002, Gobas and Arnot 2005). This included a detailed food web study to parameterize and validate the model for PCB uptake (Roberts *et al.* 2002). Parameter development for the present case study included developing chemical property estimates for the legacy pesticides: DDTs, chlordanes, and dieldrin (Table 5.7). These parameters included chemical K_{OW} and total concentrations in water and sediment. Data sources for K_{OW} are summarized in Appendix S. Concentrations in water and sediment were calculated using data from the RMP and NOAA-EMAP program (for sediment data only, U. S. EPA 2006a). RMP water and sediment data were combined from 1998 to 2002 collection dates and EMAP sediment data were collected in 2000 and 2001. For water and sediment data, the arithmetic mean was calculated for each individual site (N = 67 sites for water; N = 91 sites for sediment). The geometric mean was then calculated for all sites combined as the model input parameter (Table 5.7). Values below detection were replaced with one-half of the detection limit. Heptachlor, heptachlor epoxide, and o,p'-DDE had > 75% of values below detection limits, and were not included in the analysis (Table 5.6).

Monte Carlo uncertainty analysis was conducted to estimate uncertainty of model predictions. Analyses used parameters and distributions presented in Gobas and Arnot (2005). For pesticide properties (Table 5.7), Monte Carlo simulations assumed lognormal distributions, with standard deviations (log scale) of 0.1 for K_{OW} and concentration in water, and 0.146 (log scale) for concentration in sediment.

Table 5.7. Model input parameters for legacy pesticides. $K_{ow}TS$ = octanol water partitioning coefficient (log scale) corrected for temperature and salinity conditions of San Francisco Bay. Water concentration is total, rather than dissolved.

Compound	K_{ow}	$K_{ow}TS$	Concentration in sediment (ng/g dw)	Concentration in water (µg/L)
o,p'-DDD	5.49	5.66	0.138	2.63×10^{-5}
o,p'-DDT	5.85	6.03	0.088	8.10×10^{-6}
p,p'-DDD	6.48	6.65	0.902	9.62×10^{-5}
p,p'-DDE	7.08	7.24	0.783	8.61×10^{-5}
p,p'-DDT	6.54	6.72	0.307	2.34×10^{-5}
cis-Nonachlor	5.85	6.04	0.138	1.39×10^{-5}
dieldrin	5.63	5.81	0.242	4.27×10^{-5}
gamma-Chlordane	6.42	6.60	0.070	1.11×10^{-5}
hexachlorobenzene	5.67	5.79	0.061	3.62×10^{-5}
mirex	7.15	7.37	0.122	1.68×10^{-6}
trans-Nonachlor	5.85	6.04	0.070	9.86×10^{-6}

5.3.2.2. Results

Comparison of model results with field data indicated general correspondence for individual legacy pesticide compounds (Figure 5.8). In three fish species (white croaker, shiner surfperch, and jacksmelt), and two bivalve mollusk species, the model predicted tissue concentrations were generally similar to Bay wide observed concentrations for DDTs, chlordanes, and dieldrin (Figure 5.9; additional figures in Appendix X). This is similar to evaluations for 40 PCB congeners performed previously in San Francisco Bay (Gobas and Arnot 2005) and the Great Lakes (Arnot and Gobas 2004). This suggests that empirically derived BAFs may be used for San Francisco Bay fish and sediments (Figure 5.9). This finding is also consistent with the interpretation that the biota ultimately receive a proportion of their pesticide exposure from sediments in San Francisco Bay, as included in the model.

For chlordanes, dieldrin, and ortho-para DDT congeners, there was no systematic model bias; that is, the model predicted concentrations were not systematically higher than empirical concentrations, or vice versa. In many cases, the model predicted biota concentration were below field detection limits (yellow bars; Figure 5.9, Appendix X), and were therefore consistent with field results.

Figure 5.8. Comparison of model predicted vs. observed field data for individual DDT compounds. Results are for bivalve mollusks (e.g., *Mytilus* spp.), shiner surfperch, jacksmelt, and white croaker. Error bars indicate the standard deviation of field observations and the uncertainty of model results as estimated by a Monte Carlo analysis of model variables.

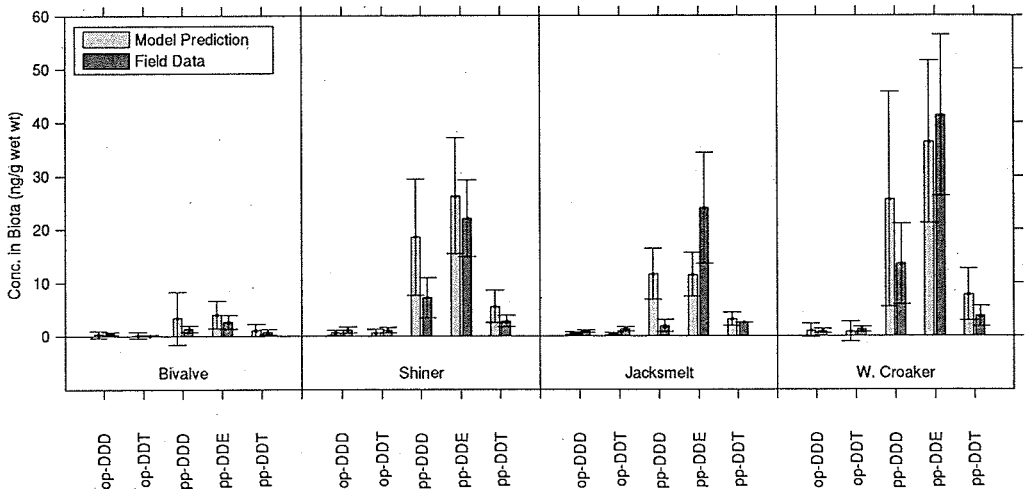
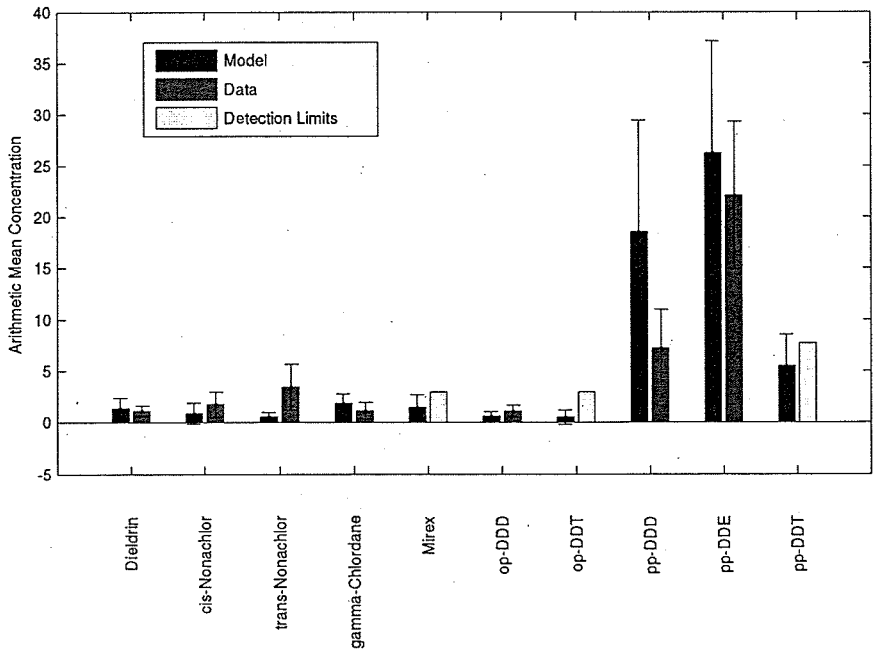


Figure 5.9. Comparison of model predicted vs. observed field data for individual pesticide compounds in shiner surfperch. Error bars indicate the standard deviation of field observations and the uncertainty of model results as estimated by a Monte Carlo analysis of model variables. Yellow bars indicate detection limits in cases where greater than 90% of field data were below detection limits.



For para-para DDD (p,p'-DDD) and para-para DDE (p,p'-DDE), the model predicted concentrations were sometimes higher than field-observed concentrations. For example, model predicted concentrations were about 15 ng/g higher than field concentrations for p,p'-DDD in shiner surfperch (Figure 5.9). In the Newport Bay case study, model estimated total DDTs were also higher than field results for a number of fish species (Figure 4.15). This model bias for DDTs may result from a number of factors. First, model input data on K_{OW} may not accurately represent in-situ partitioning in San Francisco Bay due to incorrect specification (Pontolillo and Eganhouse 2001) or chemical disequilibrium (Zeng and Tran 2002, Burkhard *et al.* 2003). Additionally, the lack of contaminant metabolism in the model may not account for biodegradation that occurs in finfish or invertebrates (van der Linde *et al.* 2001, van der Oost *et al.* 2003, Bayen *et al.* 2005). In any case, given the sometimes elevated model results as compared to empirical results, it can be expected that BAF estimates made using the Gobas mechanistic model would be relatively conservative for total DDTs.

5.3.2.3. Sediment threshold comparison

Sediment thresholds for total DDTs may be calculated as the quotient of the human health tissue thresholds (Table 5.4), and the wet weight determined BAFs of 5.0 (arithmetic mean) and 6.4 (95% upper confidence interval of arithmetic mean). Assumptions and decisions made for sediment threshold development include BAF assumptions in Table 5.6, and policy-related assumptions about target population (human sport fish consumers) and level of allowable risk (Table 5.8). Following the framework, the high threshold is combined with the average BAF, and the low threshold is combined with the UCI BAF, resulting in two sediment thresholds (Table 5.9). As was explored in the Newport Bay case study, a substantial array of decisions and assumptions must be made prior to developing the sediment thresholds for use in the assessment framework (Tables 5.3 and 5.10). These include both technical and policy decisions and can significantly impact assessment results (as described in Section 4.4.3.2).

Table 5.8. Summary of risk assessment assumptions, their basis, and effects on developing sediment thresholds for the sediment chemistry LOE, San Francisco Bay case study. See also Table 5.6.

Decision or Assumption	Basis	Effect
Use risk assumptions in Table 5.2 to set low and high tissue thresholds (average and 95%ile sport fish consumer; 10^{-5} increased cancer risk)	San Francisco Bay Regional Board staff did not request specific risk assumptions. Following general risk assumptions outlined in Section 3.1 and Appendix K of report, based on local consumption rate data (SFEI, 2000)	As demonstrated in the Newport Bay case study (Section 4), the risk assumptions are policy decisions and can substantially impact the incidence of exceeding thresholds
Base the high threshold on an arithmetic average (50%ile) estimate of bioaccumulation factor	Less conservative threshold protects the average consumer (Table 5.2)	Higher incidence of exceeding thresholds than using geometric mean or median estimated BAF
Base the low threshold on an 95% upper confidence limit estimate of bioaccumulation factor	More conservative threshold protects the average consumer, including a protection factor to account for uncertainty in the estimate of bioaccumulation	Greater than 95% probability that the bioaccumulation estimate is equal to or greater than the actual average bioaccumulation for surfperch in San Francisco Bay

Table 5.9. Development of low and high sediment thresholds for total DDTs. Thresholds were developed based on human health effects of consuming contaminated sport fish.

Threshold type	Risk	Tissue threshold	BAF	Sediment threshold
High	Higher	118	5.0	118/5 = 23.6
Low	Lower	64	6.4	64/6.4 = 10.0

When individual sediment samples from San Francisco Bay were evaluated in comparison to the two thresholds for total DDTs, the majority (82%) of samples were below both thresholds (Table 5.10). This finding is not surprising given that the thresholds were developed to be protective of sport fish consumers, and average sport fish tissue concentrations were below their respective thresholds, as well. Based on the prey tissue LOE, the assessment framework conclusion would remain "unlikely impact" for all sediment stations. Clearly, the framework focus on specific beneficial uses of the water body (in this case, human sport fish consumption) strongly affects the interpretation of individual results.

Table 5.10. Comparison of San Francisco Bay sediment samples to the low and high thresholds developed for total DDTs. Samples evaluated included data from RMP, EMAP, and all studies assembled in the CASQO database.

Sediment category	Concentration	Number Samples	Percent
Below low threshold	< 10.0	577	82%
Between thresholds	10.0 - 23.6	58	8%
Above high threshold	>23.6	72	10%

5.4. Relative contribution of water and sediment to food web

The relative importance of various sources in biota exposure to contaminants is an important issue (Burkhard *et al.* 2003). As indicated in the assessment framework conceptual model (Section 2.2, Figure 2.1), exchange between water and sediments makes it difficult to distinguish between these two contaminant reservoirs. The relative importance of sediments vs. the water column for contaminant uptake can be explored using mechanistic models. In San Francisco Bay (Davis 2004) and in other water bodies (Burkhard *et al.* 2003), there may be significant contaminant flux between sediments and the water column in a dynamic process, making it impossible to treat them as independent sources of bioavailable contaminants. Nevertheless, a model that separately quantifies the biological uptake of contaminants from sediments and the water column provides a first order approximation of the relative importance of these two matrices to biota exposure.

5.4.1. Methods

Manipulations were performed on the mechanistic food web model used and described elsewhere in the report (Section 3.2.8 and Appendix M) and other sources (Gobas 1993, Arnot and Gobas 2004, Gobas and Arnot 2005). The mechanistic food web model is used to manipulate the relative contribution of water vs. sediment to total contaminant uptake by biota. This is achieved by running model simulations in which the sediment vs. water column sources are separately "turned off," by setting concentrations to zero. The resulting change in modeled biota concentrations are then determined. This exercise provides an estimate of the total proportion of exposure resulting from water column vs. sediment.

Two scenarios were evaluated. In the first scenario, the relative contributions of water and sediment to contaminant levels in biota were varied in 10% increments (Table 5.11). This scenario was performed using initial parameter estimates for p,p'-DDE from San Francisco Bay (Table 5.7), and results were evaluated for shiner surfperch and jacksmelt. The food web model was run using these concentrations as inputs, to determine sensitivity of the model BAF calculation to relative sediment vs. water column inputs. Model calculated BAFs were then compared to the ratio of model input concentration in sediment (C_s) vs. concentration in water (C_w) (i.e., C_s/C_w).

In the second scenario, sediment and water column concentrations were separately manipulated from their original values to zero. The relative contribution of sediment vs. water column to biota exposure and resulting BAF calculation was then determined as:

$$\text{Contribution of water column (\%)} = \frac{\text{Biota concentration when sediment} = 0}{\text{Biota concentration with original sediment and water}}$$

$$\text{Contribution of sediment (\%)} = \frac{\text{Biota concentration when water} = 0}{\text{Biota concentration with original sediment and water}}$$

5.4.2. Results

The model predicted p,p'-DDE bioaccumulation factor (i.e., concentration in biota/concentration in sediment) was inversely related to C_s/C_w (Figure 5.10). When concentration in sediment (ng/g) was greater than 10,000 times concentration in water (µg/L), the model predicted BAF was around 15 for jacksmelt and 30 for shiner surfperch. As C_s/C_w dropped below 10,000, the BAF began to increase sharply. At C_s/C_w of 1600, BAF reached almost 80 and 160 for shiner surfperch and jacksmelt (Figure 5.10). The reason for the model's sensitivity to C_s/C_w is that the model explicitly considers both sediment and water column exposure. Biota in the model may be exposed to sediment contaminant due to direct dietary uptake, as well as respiratory exposure to porewater contaminants, which are modeled assuming equilibrium with sediments. Biota may also be exposed to water column contaminants due to respiratory exposure, or transpiration in phytoplankton (Appendix M). At relatively low sediment concentrations, water column contaminants drive biota exposure, resulting in very high BAFs.

Table 5.11. p,p'-DDE concentrations in sediment (C_s) and water (C_w) used to investigate the relative contributions of each source to modeled p,p'-DDE levels in Newport Bay biota.

	(C_s) (ng/g dry wt)		(C_w) (µg/L)	C_s/C_w
Mean	0.78	Mean	8.61×10^{-5}	9,094
Mean-10%	0.70	Mean+10%	9.47×10^{-5}	7,441
Mean-20%	0.63	Mean+20%	1.03×10^{-4}	6,063
Mean-30%	0.55	Mean+30%	1.12×10^{-4}	4,897
Mean-40%	0.47	Mean+40%	1.21×10^{-4}	3,897
Mean-50%	0.39	Mean+50%	1.29×10^{-4}	3,031
Mean-60%	0.31	Mean+60%	1.38×10^{-4}	2,274
Mean-70%	0.23	Mean+70%	1.46×10^{-4}	1,605
Mean+10%	0.86	Mean-10%	7.75×10^{-5}	11,115
Mean+20%	0.94	Mean-20%	6.89×10^{-5}	13,641
Mean+30%	1.02	Mean-30%	6.03×10^{-5}	16,889
Mean+40%	1.10	Mean-40%	5.17×10^{-5}	21,220
Mean+50%	1.17	Mean-50%	4.31×10^{-5}	27,282
Mean+60%	1.25	Mean-60%	3.44×10^{-5}	36,376
Mean+70%	1.33	Mean-70%	2.58×10^{-5}	51,533

Based on Figure 5.10, one might expect that BAFs would be elevated due to the water column contribution. This expectation is supported when using the model to examine the relative contribution of p,p'-DDE in sediments vs. water column. For all fish species and benthic invertebrate species, water column exposure resulted in 50% to 100% of modeled body burden (Figure 5.11). Results were particularly pronounced for fish, for which water column sources resulted in at least 75% of body burden. Water column sources were most important for those species that prey predominantly on plankton, including *Mysis* sp., *Crangon* sp., and jacksmelt, while sediment exposure was greatest for the sediment dwelling and consuming annelid, *Neanthes succinea*. This emphasizes the importance of selecting species with sediment life history associations when developing empirical BAFs (Section 3.1.4).

Results were somewhat similar when evaluating PCB 118, in that water column vs. sediment exposure varied according to the life history of modeled species (Figure 5.12). However, in comparison to p,p'-DDE results, PCB 118 exhibited a higher overall sediment contribution to biota exposure in general. This difference results from the much higher ratio of sediment vs. water column concentration for PCB 118 (approximately 130,000), as compared to p,p'-DDE (9,000), in San Francisco Bay. In general, PCBs exhibited higher sediment to water column ratios than DDTs at a given K_{ow} (Figure 5.13). This discrepancy between the two compound classes suggests that K_{ow} is not the only factor driving sediment vs. water column concentrations in San Francisco Bay. Other potential factors may include external loading of DDTs to the water column or nonlinear sorption of PCBs to carbonaceous particles (Maruya *et al.* 1996, Ghosh *et al.* 2003, Cornelissen *et al.* 2005).

Figure 5.10. Model predicted bioaccumulation factor (BAF) for p,p'-DDE in jacksmelt and shiner surfperch as a function of C_s/C_w (from Table 5.11).

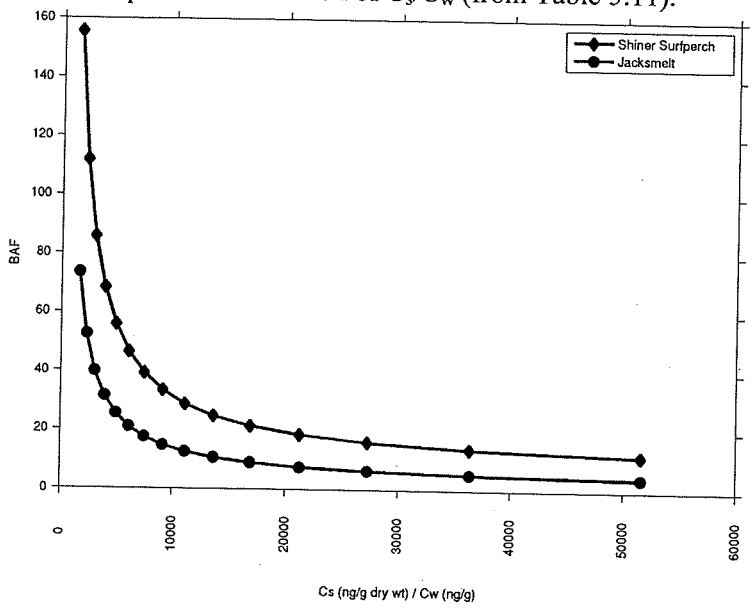


Figure 5.11. Relative contribution of water and sediment to p,p'-DDE bioaccumulation. Results are based on mechanistic model application to multiple species using parameters developed for San Francisco Bay.

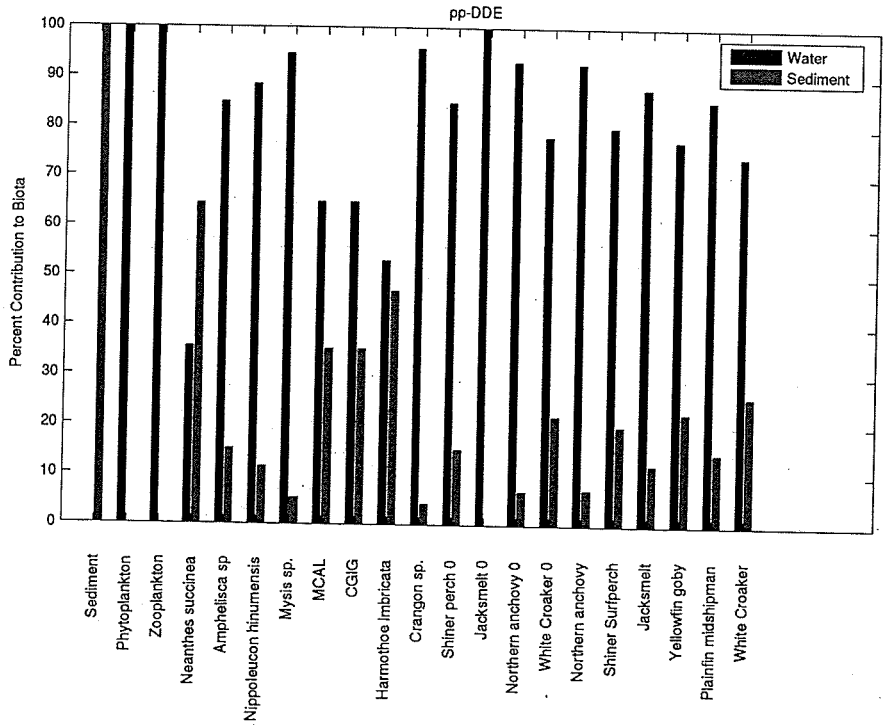
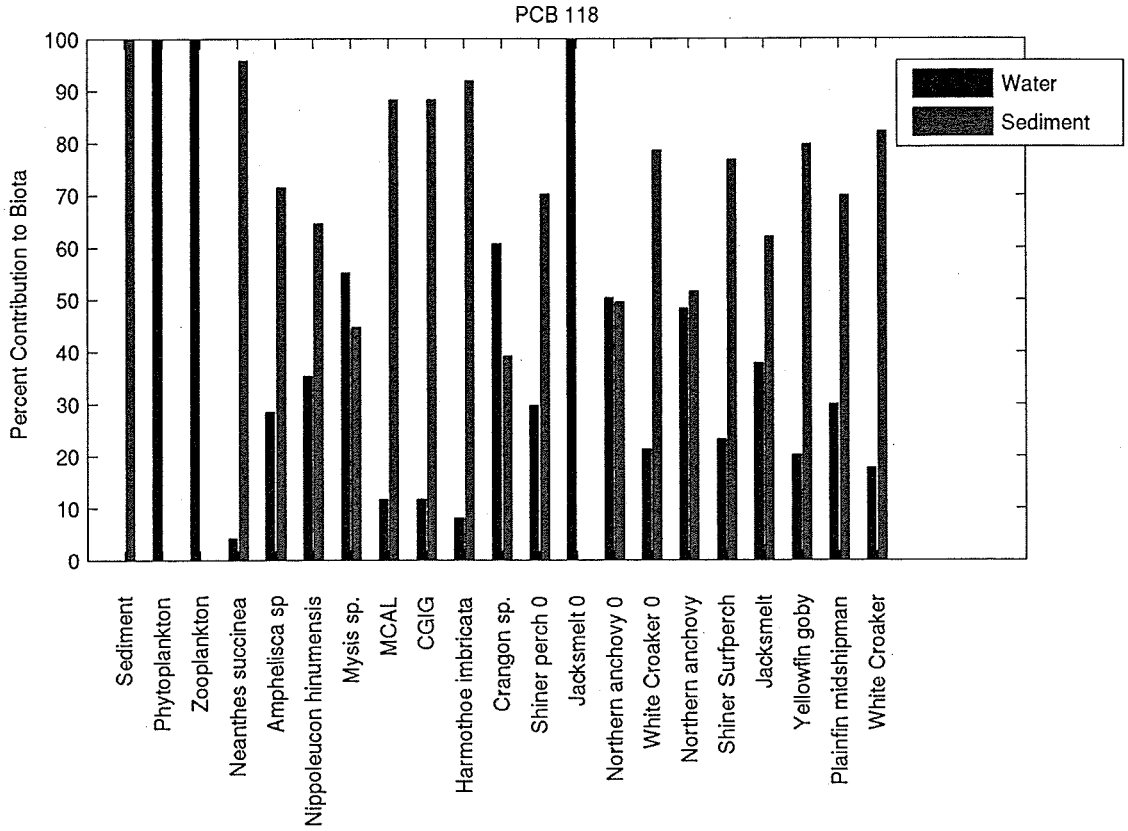


Figure 5.12. Relative contribution of water and sediment to bioaccumulation of PCB 118. Results are based on mechanistic model application to multiple species using parameters developed for San Francisco Bay.



It would be possible to use mechanistic model results to modify the empirically developed BAFs to focus on the direct contributions of sediments to food-web contaminant exposure. For example, the BAFs developed for shiner surfperch (Table 5.8) could be multiplied by 0.20, based on the model estimation of 20% of total p,p'-DDE exposure to adult surfperch resulting from sediments (Figure 5.11). Although results would differ among DDT compounds, the vast majority of biota DDTs are in the form of p,p'-DDE, allowing the assumption that sediment partitioning for total DDTs will be similar to p,p'-DDE. Multiplying the BAFs by 0.20 would result in new BAFs of 1.0 and 1.3. This would produce considerably different threshold calculations and would result in a higher percentage of samples below the low threshold (92% as compared to 82%; Table 5.12 vs. Table 5.10). However, these modified BAFs would probably underestimate sediment exposure to biota due to indirect effects. This is because actual water column residues are likely to be substantially derived from resuspended sediments, which currently constitute a primary reservoir of PCBs and legacy pesticides (Venkatesan *et al.* 1999, Davis 2004). Additional contaminant fate modeling would be required to establish the relative proportion of water column concentrations derived from resuspended sediments vs. external loading or atmospheric deposition.

Figure 5.13. Relationship between literature-based K_{ow} and C_s/C_w for PCBs (triangles) and DDTs (circles) in San Francisco Bay. PCBs are from left to right: PCB 18, PCB 66, PCB 118, PCB 153, and PCB 194. DDTs are from left to right: o,p'-DDD, o,p'-DDT, p,p'-DDD, p,p'-DDT, and p,p'-DDE. Based on Bay-wide average concentration estimates (Table 5.7, and Gobas and Arnot (2005)). Note log scale y-axis.

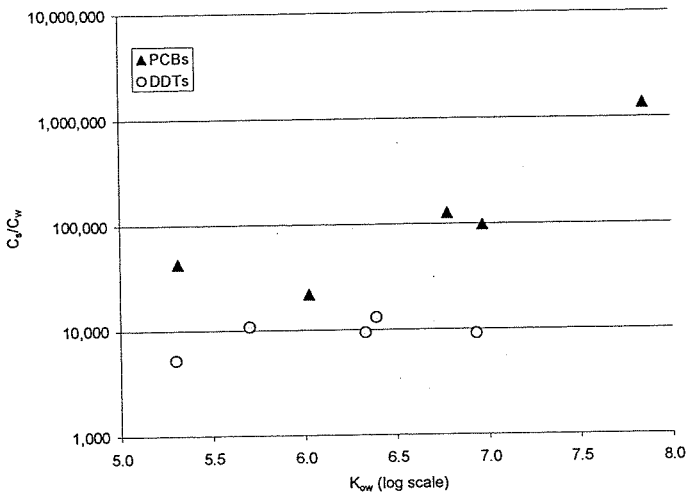


Table 5.12. Comparison of San Francisco Bay sediment samples to low and high thresholds developed for total DDTs, and calibrated based on modeled percent contribution of sediments to p,p'-DDE concentrations. Samples evaluated included data from RMP, EMAP, and all studies assembled in the CASQO database.

Sediment category	Concentration	Number Samples	Percent
Below low threshold	< 50	651	92%
Between thresholds	50 – 118	10	1%
Above high threshold	>118	46	7%

5.5. Bioavailability line of evidence

The bioavailability line of evidence is recommended as a final step when the previous LOE suggest that sediments may be impacted by indirect effects due to contamination. This LOE is used to demonstrate whether sediment-associated contaminants are available for biological uptake (Section 3.3). A relatively straightforward approach for the bioavailability LOE is to demonstrate a positive association between sediment contaminant concentration and biota contaminant concentration. To demonstrate this approach, bioaccumulation data were assembled and evaluated for DDTs and other legacy organochlorine compounds in San Francisco Bay.

As discussed in Section 5.2.2, the prey tissue LOE fell into the lowest risk category. Based on the assessment framework, this would result in an assignment of "Unlikely Impacted," with no need to proceed to the sediment chemistry or bioavailability LOE for the San Francisco Bay case study. The purpose of this Section is simply to illustrate the potential use of the bioavailability LOE, as well as likely results for legacy organochlorine compounds.

5.5.1. Methods

Data used to investigate bioavailability of contaminants in San Francisco Bay sediments were obtained from the California Sediment Quality Objectives database. Data included 28 day laboratory bioaccumulation tests conducted at Hunters Point, Oakland Harbor and Richmond Harbor, as well as control samples from other locations in the Bay associated with these studies (Lee *et al.* 1994, Battelle *et al.* 2005).

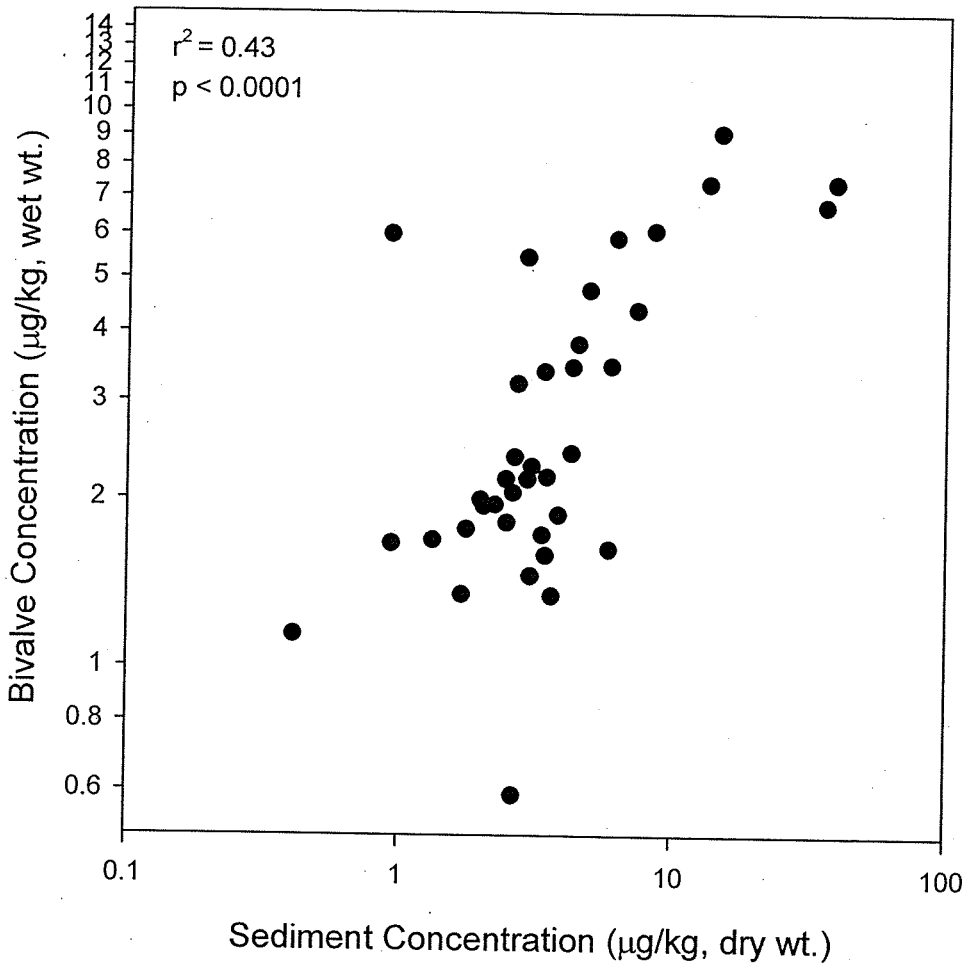
Studies were performed using standard protocols for 28 day laboratory bioaccumulation tests (U. S. EPA and U. S. Army Corps of Engineers 1991, 1998). Homogenized sediments of known field concentrations were administered to *Macoma nasuta* test organisms in a laboratory environment, with laboratory test conditions monitored throughout the experiments. Tissue analyses were subsequently performed after 28 days to determine the availability of sediment contaminants taken up by the test organisms.

Comparison between sediments and *M. nasuta* were performed for total DDTs, total chlordanes, total PCBs, dieldrin, and high molecular weight PAHs (as defined in Appendix N). Linear regression analysis was applied to compare sediment vs. corresponding *M. nasuta* tissue concentrations (Draper and Smith 1998). A significant positive relationship in the comparison was interpreted to indicate that the contaminants were bioavailable to *M. nasuta*.

5.5.2. Results

A statistically significant positive linear relationship was observed for total DDTs (Figure 5.14; $R^2 = 0.43$, $p < 0.0001$, $N = 38$). Significant positive relationships were also observed for total chlordanes ($R^2 = 0.74$), dieldrin ($R^2 = 0.49$), and total PCBs ($R^2 = 0.72$; $p < 0.0001$ in all cases), and a weak positive trend was observed for high molecular weight PAHs ($R^2 = 0.15$, $p = 0.014$). These results indicated bioavailability of DDT, chlordanes, dieldrin, and total PCBs in San Francisco Bay sediments. Results for PAHs were inconclusive, and suggest that other approaches may be warranted. For example, control (unexposed) bivalves could be analyzed in comparison to exposed bivalves, to determine whether exposure results in increased tissue concentrations (Section 3.3). Bioaccumulation of PAHs may vary widely as a function of PAH sources, sediment properties, non-linear sorption to carbon particles, and taxa-specific metabolism rates (Meador *et al.* 1995, Cornelissen *et al.* 2005). This suggests a need for assessment of bioaccumulation in local invertebrate prey organisms to apply the framework to PAHs.

Figure 5.14. Linear regression of sediment vs. *Macoma nasuta* total DDTs in 28 day laboratory bioaccumulation test.



6. Conclusions and recommendations:

6.1. Overview of the framework

The assessment framework aims to streamline and standardize the evaluation of indirect effects of sediment pollution. It is based on a simplified conceptual model of biota exposure to sediment pollution (Figure 2.1). It incorporates general risk assessment principles, including the use of site-specific receptors, probability-based evaluation of exposure and effects, and a sequential decision process. Exposure is evaluated based on local data using a weight of evidence approach. The exposure lines of evidence are intended to be reasonably straightforward to collect and analyze, though interpretation of the results presents some challenges. The effects evaluation predominantly relies on readily available published risk information, such as federal and state-recommended toxicity reference values for dietary exposure to pollutants.

A primary goal of the framework is to simplify risk assessment for dietary exposure to sediment pollution. Federal CERCLA risk assessments managed by the Superfund program are time consuming and costly to perform. For example, the Remedial Investigation and Feasibility study underway for the Lower Passaic River in New Jersey is expected to cost at least 10 million dollars, with a Record of Decision target date of 2014 (Hyatt 2007). The framework in our report is intended to be appropriate for a statewide program to characterize many water bodes. The aim is to reduce confusion and expense by: 1. focusing on clearly defined objectives (protection of human and wildlife from contaminated prey); and 2. using a simplified decision framework that targets pollutants of concern.

6.2. Case study findings and interpretations

The goal of the case studies was to illustrate some of the technical issues and policy decisions that arise in applying the framework. A number of important findings were revealed. First of all, bioaccumulation rates varied among the water bodies. This finding supported the collection and use of local exposure data. Second, empirical bioaccumulation rates were generally corroborated by an independent mechanistic model. This suggests that many of the confounding factors frequently discussed (e.g., migration, waterborne exposure, spatial and temporal heterogeneity, and dietary variation) do not fundamentally undermine the development of a site-specific bioaccumulation rate parameter for sediment-associated finfish. Third, evaluation of prey tissue chemistry sometimes revealed that the narrative objectives (i.e., reduction of risk to human or wildlife piscivores) were achieved. This finding supports the use of sequential application, beginning with prey tissue chemistry, to effectively focus effort and resources on those pollutants that pose the greatest risks. For example, since prey fish tissue concentrations were below the low pesticides thresholds in San Francisco Bay, efforts could be focused on evaluating or preventing other pollutants that pose potential risks.

The case study results indicated that policy decisions, such as target population and acceptable level of risk, sometimes affected the outcome of the assessment. Results were particularly sensitive to the allowable level of risk to humans. For example, in the Newport Bay case study, potential human health impacts of sediment PCBs would fall into the intermediate or low risk category, depending upon specific assumptions about allowable cancer risk. One goal of the framework is to focus the policy discussion towards developing low and high effects thresholds. The use of two thresholds may aid in developing compromises between multiple interested parties.

The case studies also revealed a number of technical uncertainties and data limitations. Mechanistic model simulations indicated that a significant portion of biota exposure to DDTs stemmed from dissolved and particulate compounds in the water column. In general, modeled BAFs were highly sensitive to water column concentrations, highlighting the potential uncertainty regarding the ultimate source of pollutants. Although it is likely that a large portion of the water column concentrations were linked to direct sediment resuspension, direct loading from the watershed and upstream rivers should also be considered.

In the Newport Bay case study, certain biota exhibited twofold differences between the empirical and mechanistic model results. These highlight the common limitations on input data needed for the mechanistic model, such as food web structure, and compound-specific metabolic transformation rates. The benthic algae specific to Newport Bay were also difficult to parameterize.

Empirical contaminant data in Newport Bay biota were sometimes rather poor. Surprisingly, this had a relatively limited impact on the outcome of the assessment. For example, the framework would characterize sediment impact due to PCB exposure as a combination of unlikely impact, possible impact, and likely impact across a wide range of assumptions and policy judgments (Table 4.20).

The technical approach and findings varied among the two case studies, according to differences in available data. San Francisco Bay may be viewed as a "best case scenario," in that extensive data are available on sediment chemistry, tissue chemistry, ancillary parameters, and biota life history. This enabled the use of a single surrogate species and a spatial averaging approach in BAF development.

Newport Bay had more limited data than San Francisco Bay, with relatively small sample sizes and chemistry data often marred by high detection limits. The data limitations were addressed to some extent by pooling multiple prey species and developing a modified BAF (based on lipid weight prey tissue and dry weight sediments). Probabilistic approaches, such as generation of average and upper confidence interval parameter estimates, enabled the framework to be applied even given the limited and uncertain data. Nevertheless, the data limitations likely reduced the overall certainty of the assessment. This uncertainty was illustrated by the sensitivity of the Newport Bay case study results to data distribution assumptions (i.e., arithmetic vs. geometric calculations). The data

uncertainty may have also caused a greater occurrence of intermediate risk classifications (i.e., the "possibly impacted" and "likely impacted" categories).

6.3. Avenues for future study

Thus far, application of the framework has been limited to assessment of the legacy organic contaminants: PCBs and organochlorine pesticides. This leaves open a wide array of topics that merit evaluation. The wildlife toxicity reference values and the bioavailability technical guidance may benefit from further review and revision. Potential avenues for future research include evaluation of contaminant mixtures, assessment of additional compounds, and effects to finfish.

Improved understanding of the bioavailability line of evidence

The guidance in this report predominantly focuses on use of standard laboratory test species in 28 day evaluations. Alternative options would result in a more flexible approach. Evaluation of bioavailability is an area of active research. Areas of development include semipermeable membrane devices, solid-phase microextraction fibers, in-vitro assays using gut extracts, and field deployment of caged biota (van der Oost *et al.* 2003, Moore *et al.* 2005). Data may also be available on additional appropriate test organisms. Another avenue for exploration is the development of approaches that can be readily translated to field bioaccumulation rates, to provide corroborative information for the prey tissue chemistry LOE.

Development of new and revised toxicity reference values

Most of the wildlife TRVs used in the case studies were developed about a decade ago. It is likely that new toxicity studies have been performed, resulting in improved TRVs. Additional TRVs should also be developed for recent use compounds, such as brominated flame retardants. The U. S. EPA Region IX BTAG has a procedure for developing or refining TRVs (California DTSC Human and Ecological Risk Division 2000). This procedure may be employed to update the TRVs for framework application to estuarine and bay sediments.

Evaluation of indirect effects of contaminant mixtures

The case studies and technical guidance in this report focus on evaluation of individual contaminants. Future research and development could consider applying the framework to evaluate effects of contaminant mixtures. In particular, additive mixture models could be employed to evaluate cumulative risks of carcinogenic or non-carcinogenic effects. Toxicity equivalency factors (TEF) models may also be employed, including dioxin TEFs for dioxin-like compounds (Van den Berg *et al.* 1998), and benzo[a]pyrene TEFs for PAHs (Nisbet and Lagoy 1992).

Indirect effects to finfish

So far, the focus of the framework has been on evaluating risks to birds, aquatic mammals, and humans. However, finfish also prey on benthic organisms, and may be indirectly exposed to contaminated sediments. Finfish tissue-residue based toxicity reference values are readily available for some compounds (e.g., Appendix I). For these compounds, it would be relatively straightforward to apply the framework to assess risks to fish.

Application of the framework to additional compounds

A wide range of compounds may be present at elevated concentrations in sediments due to anthropogenic sources. These include Hg, Se, and other metals, dioxins, PAHs, and multiple previously unmonitored compounds. Current use pesticides, such as organophosphates and pyrethroids, are of particular management concern in estuaries with agricultural or residential development. Each class of compounds is likely to present specific technical challenges for assessment of indirect effects. For example, the acid-volatile sulfide model has been proposed to predict bioavailability of metals (Hansen *et al.* 1996), but success has been limited (Lee *et al.* 2000, Meador *et al.* 2005).

Two approaches could be employed to evaluate additional compounds. The first approach is to undertake additional case studies, focusing on water bodies and pollutants that present specific challenges. For example, the effects of contaminant mixtures in the Sacramento-San Joaquin Rivers Delta could be evaluated. An alternative approach would be to conduct a general screening-level risk assessment to identify priority compounds for more careful consideration. This would entail application of the framework using simplified assumptions and representative parameter estimates. The objective would be to identify those compounds that pose the greatest potential risks to consumers of sediment-exposed prey organisms. This information would form the basis for future research and framework development.

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8. Appendices

Appendix A. Key elements of a framework for evaluating indirect effects of sediment pollution.

A number of issues have been raised by committees involved in the California SQO development process. These committees include the Sediment Quality Advisory Committee, representing stakeholders that will be impacted by the program, the Agency Coordinating Committee, including staff of agencies that will apply the program, and the Scientific Steering Committee, composed of scientific experts in the field. Despite the diverse representation of these committees, feedback has consistently included the following:

- Before determining that sediments are impacted by pollutants, there should be some evidence that the sediments may be responsible for the impacts to wildlife or human health.
- In order to have benefit, the approach should be able to differentiate among individual sediments.
- The method should be flexible enough to be applicable for the range of conditions and bioaccumulative contaminants that may occur in California bays and estuaries. This may include incorporation of local data, including local fish consumption advisories, when available.
- The approach should explicitly incorporate uncertainty into the analysis. Uncertainty may be addressed using probability-based methods, and by having multiple categories reflecting different degrees of certainty regarding whether objectives are met or exceeded.
- Target receptors should be appropriate for the beneficial uses they are intended to protect. For example, receptors to represent wildlife prey may be different from receptors to represent human fish consumption.
- The issue of spatial scale should be considered, and the scale at which the framework is applied must be appropriate for the scale at which beneficial uses may be impacted. Fish, wildlife, and humans obtain food from a variety of sources and locations and site fidelity will vary among species. Fish captured at a certain location may not necessarily have been foraging in that exact location prior to capture. Comparisons should be made between foraging area of the receptor and areas of elevated sediment concentration. Whenever possible, receptors with limited movement and high site fidelity should be chosen.
- Recommendations should be made for circumstances when data availability or sample sizes are limited.
- California state agencies responsible for characterizing sediments will have limited funding and resources. Given these constraints, the assessment framework must be reasonably practical to apply. The conceptual framework and underlying methods should not be more complex than necessary.

Appendix B. Relationship between sediment and finfish contaminant concentrations.

Introduction

This Appendix provides technical support for the use of finfish tissue chemistry in the framework, by addressing two issues. First of all, it supports the use of finfish tissue concentrations as part of an indirect effects evaluation framework for sediments. Specifically, there is a statistically significant association between pollution in sediments and in fish captured near these sediments.

This Appendix also identifies finfish species that may be particularly useful for the prey tissue LOE based on their association with sediment chemistry. To accomplish this, empirical data are compared between finfish and sediment pollutant concentrations to identify candidate finfish species. When significant relationships are observed, this suggests that those fish species exhibit relatively strong affinity to sediments and pollutant exposure at local scales. Similar support for the use of invertebrate chemistry in the bioavailability LOE is provided in Section 3.3.

Methods

Results are based on reviews of published literature, and analyses of data available from the California Sediment Quality Objectives (CASQO) database[39].

To identify good candidate species for the prey tissue LOE, sediment-biota relationships were evaluated for PCBs, organochlorine pesticides, and other pollutants as available. Linear regressions were conducted on log-transformed data to improve normality and variance homoskedasticity of residuals (Draper and Smith 1998). Analyses focused on DDTs, total PCBs, total chlordanes, and dieldrin. Evaluations were conducted for those species having at least five separate fish tissue collections for a given water body in the CASQO database. Separate evaluations were conducted for the three water bodies having sufficient data: San Francisco Bay, San Pedro Bay, and San Diego Bay. These analyses used previously published organic pollutant data. The analyses were conducted comparing fish tissue concentrations to surrounding sediment concentrations at 1 km radius increments at spatial scales from 1 to 5 km. Appendix L describes the statistical procedure to characterize the spatial scale at which sediment pollutant concentrations were best related to those in fish tissue.

Results of literature review

There are many examples of spatial correlation between fish tissue and sediment chemistry in peer-reviewed literature and technical reports. Such relationships are frequently observed for PCBs and other chlorinated organic compounds (Table B.1). In southern California, statistically significant relationships have been shown for trace organic contaminants in sanddabs and other flatfishes (e.g., Schiff 2000, Allen *et al.* 2004), as well as white croaker (Table B.1). Laboratory results corroborate these field

[39] As described in Section 3.2.2

observations. For example, Ankley *et al.* (1992) observed elevated PCB concentrations in fathead minnows exposed to contaminated sediments in the laboratory. The study also observed BSAFs in the laboratory that were similar to field observed BSAFs. At least one study detected significant relationships for the trace metals, mercury and lead (Meador *et al.* 2005). Finally, a substantial literature demonstrates spatial associations between sediment PAH concentrations, and toxic effects in benthic fish. Effects include hepatic lesions, reproductive impairment, and DNA damage (Spies *et al.* 1990, Stehr *et al.* 1997, Johnson *et al.* 2002). Species exhibiting these relationships generally have benthic dietary associations. Relationships are generally stronger when spatial contamination gradients are stronger.

Table B.1. Selected literature sources indicating significant relationships between sediment and fish

Species	Contaminants with significant sediment association	Source
Shiner surfperch	DDTs	Lee (1994)
Sanddab guild, California halibut	PCBs, DDTs	Allen *et al.* (2002a, 2002b)
White croaker	PCBs, DDTs, chlordanes	This study, Connolly and Glaser (1997)
Shorthorn sculpin	PCBs	Kuzyk *et al.* (2005a, 2005b)
White croaker, English sole	Hg, lead	Meador *et al.* (2005)
White croaker, four-horn sculpin, flathead sole, English sole, starry flounder, hornyhead turbot, barred sand bass, and black croaker	PCBs, DDTs, chlordanes, dieldrin, PAHs, hexachlorobenzene	Brown *et al.* (1998)
White sucker, carp, sea bass, and other species	PCBs, dioxins, DDTs, chlordanes	Burkhard *et al.* (2005), Wong *et al.* (2001)
Longjaw mudsucker	PCBs, DDTs	Hwang *et al.* (2006)
Forage fish (Pacific staghorn sculpin, yellowfin goby, and chameleon goby)	PCBs	Battelle *et al.*(2005)

These findings indicate a common association between sediment and fish contamination. This supports the judicious use of data from field-captured fish for risk evaluation in the indirect effects assessment.

Results of empirical data analysis

Six species met the data criteria for empirical analysis: California halibut, English sole, shiner surfperch, speckled sanddab, staghorn sculpin, and white croaker. Each of these species exhibited a statistically significant correlation between fish tissue chemistry and sediment chemistry for at least one contaminant evaluated (Table B.2). For example, a strong association in total chlordanes was observed between sediment and white croaker collected in San Pedro Bay (Figure B.1). These results indicate that there is a spatial association between fish and sediment contamination for all species. This finding further

suggests that these species may have bioaccumulated contaminants from nearby sediments.

Table B.2. Empirical model results of linear regressions relating contaminant concentrations in sediment to biota tissue. **Bold** indicates a significant positive relationship ($p<0.05$). N = number of locations.

Water body	Species	Contaminant	N	R^2	p-value	Slope direction	Spatial scale
San Diego Bay	**California halibut**	**Total DDTs**	**11**	**0.63**	**0.003**	**positive**	**4 km**
San Diego Bay	**California halibut**	**Total PCBs**	**11**	**0.86**	**<0.0001**	**positive**	**4 km**
San Francisco Bay	**California halibut**	**Chlordanes**	**7**	**0.78**	**0.009**	**positive**	**2 km**
San Francisco Bay	California halibut	Dieldrin	23	0.15	0.07	positive	2 km
San Francisco Bay	California halibut	Total DDTs	18	0.18	0.08	positive	1 km
San Francisco Bay	California halibut	Total PCBs	17	0.19	0.07	positive	1 km
San Pedro Bay	**California halibut**	**Total DDTs**	**6**	**0.75**	**0.03**	**positive**	**3 km**
San Pedro Bay	California halibut	Total PCBs	6	0.04	0.7	negative	3 km
San Francisco Bay	English sole	Dieldrin	11	0.09	0.35	positive	5 km
San Francisco Bay	**English sole**	**Total DDTs**	**12**	**0.41**	**0.03**	**positive**	**5 km**
San Francisco Bay	English sole	Total PCBs	11	0.15	0.24	positive	2 km
San Francisco Bay	**Shiner surfperch**	**Chlordanes**	**36**	**0.25**	**0.002**	**positive**	**1 km**
San Francisco Bay	**Shiner surfperch**	**Dieldrin**	**41**	**0.33**	**0.0001**	**positive**	**1 km**
San Francisco Bay	**Shiner surfperch**	**Total DDTs**	**41**	**0.44**	**<0.0001**	**positive**	**1 km**
San Francisco Bay	**Shiner surfperch**	**Total PCBs**	**39**	**0.33**	**0.0001**	**positive**	**1 km**
San Francisco Bay	**Speckled sanddab**	**Dieldrin**	**5**	**0.99**	**0.0001**	**positive**	**2 km**
San Francisco Bay	**Speckled sanddab**	**Total DDTs**	**5**	**0.99**	**0.0001**	**positive**	**2 km**
San Francisco Bay	Staghorn sculpin	Dieldrin	25	0.50	0.0002	negative	1 km
San Francisco Bay	Staghorn sculpin	Total DDTs	25	0.04	0.4	positive	1 km
San Francisco Bay	**Staghorn sculpin**	**Total PCBs**	**25**	**0.73**	**<0.0001**	**positive**	**1 km**
San Francisco Bay	**White croaker**	**Chlordanes**	**33**	**0.17**	**0.02**	**positive**	**5 km**
San Francisco Bay	**White croaker**	**Dieldrin**	**17**	**0.36**	**0.01**	**positive**	**1 km**
San Francisco Bay	White croaker	Total DDTs	28	0.04	0.33	negative	2 km
San Francisco Bay	**White croaker**	**Total PCBs**	**15**	**0.36**	**0.02**	**positive**	**1 km**
San Pedro Bay	**White croaker**	**Chlordanes**	**10**	**0.80**	**0.0005**	**positive**	**2 km**
San Pedro Bay	White croaker	Dieldrin	10	0.11	0.36	positive	4 km
San Pedro Bay	White croaker	Total DDTs	10	0.17	0.24	positive	2 km
San Pedro Bay	White croaker	Total PCBs	10	0.04	0.59	negative	3 km

Because the prey tissue line of evidence evaluates exposure risk at the water body scale, it only requires confidence that the sampled species bioaccumulate contaminants from sediments within the entire water body. Since all species evaluated exhibited some evidence of affinity at relatively small spatial scales (< 5 km), the results in Table B.2 suggest that all species evaluated would be appropriate candidate species for using the prey tissue chemistry line of evidence at the water body scale.

Shiner surfperch exhibited significant relationships in all pollutants tested. For example, in San Francisco Bay, PCBs in shiner surfperch were significantly related to PCBs in sediments (Figure B.2), though the amount of variation explained was limited. The small spatial scale (1-2 km) at which significant relationships were observed, suggests that

surfperch are resident species with a benthic association. This contradicts previous reports (Shaw 1974, Emmett 1991) that shiner surfperch is a transient species. Bane (1970) indicated that the diet of shiner surfperch was primarily derived from benthic sources (i.e., including small crustaceans, sand/mud, and plant material). Evidence (this study and others) therefore suggests that shiner surfperch would be a highly appropriate species for the indirect effects framework.

Figure B.1. Linear regression of sediment and white croaker total chlordane concentration in San Pedro Bay. Appropriate spatial scale was determined following the method in Appendix L. Note log scale. Compounds included in totals are in Appendix N.

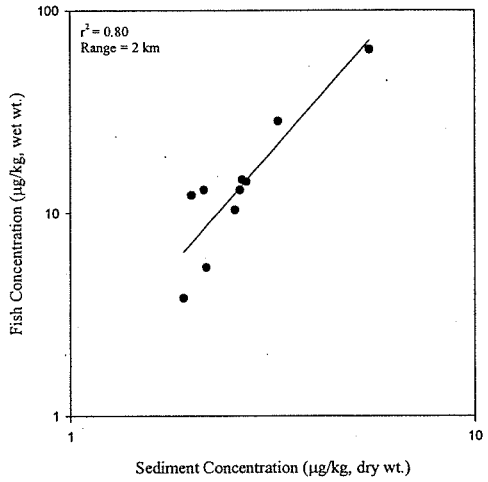
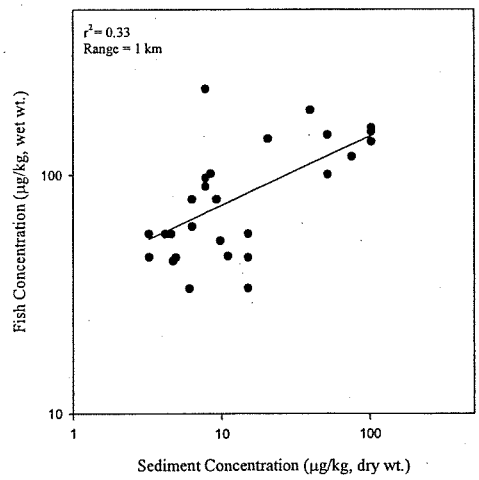


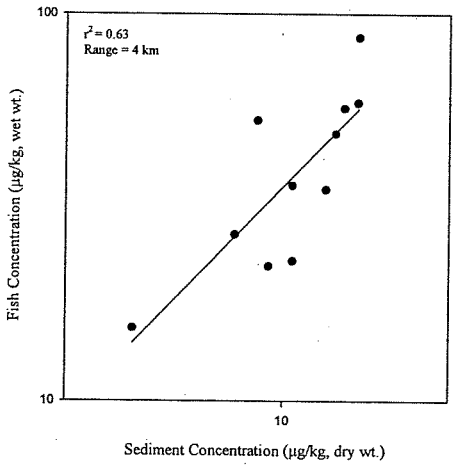
Figure B.2. Linear regression of sediment and shiner surfperch total PCBs (following Appendix L) in San Francisco Bay. Note log scale.



A number of species did not show significant relationships for some pollutants evaluated (Table B.2). One possible explanation is that we did not lipid-normalize the tissue data or organic carbon normalize the sediment data. For example, we did not observe significant relationships for PCBs or DDTs in San Pedro Bay white croaker. In contrast, Connolly and Glaser (1997), using the same data set, found an association between lipid weight PCBs and DDTs in white croaker vs. organic carbon normalized concentrations in sediment. The issues of lipid and organic carbon normalization are explored in the case studies (Sections 4 and 5).

Another possible explanation for the absence of significant spatial association between fish and sediments is that fish foraging ranges may be large enough to obscure spatial patterns in contaminant exposure at the scales examined (< 5 km). However this does not necessarily preclude using these fish species in the prey tissue line of evidence. For white croaker, results were inconsistent among contaminants and locations, with some contaminant-location combinations showing non-significant relationships (Table B.2), but with a very strong relationship for chlordanes in San Pedro Bay (Figure B.1). For white croaker, there is evidence of residence in a bay or estuary for the majority of the year, with some emigration to the coastal ocean during winter months (Fleming 1999). Flatfishes are known to vary their range depending on stage of development and time of year. For example, juvenile California halibut (less than 8 inches in length) are thought to remain relatively localized in bays and estuaries (Frey 1971). However, adult halibut generally migrate to deeper waters and may exhibit average movements of 13 km (Domeier and Chun 1995). In general, adults flatfishes limit movements to seasonal onshore-offshore migrations, but are generally resident within a given season (reviewed in Connolly and Glaser 1997). Despite potential for offshore movement, California halibut exhibited a positive association with sediment DDTs (Figure B.3).

Figure B.3. Linear regression of sediment and California halibut total DDT concentration in San Diego Bay. Note log scale.



Appendix C. The need to develop water-body specific bioaccumulation factors.

Introduction

The indirect effects assessment framework recommends that separate bioaccumulation factors be developed for individual water bodies, rather than using generic bioaccumulation factors (e.g., Arnot and Gobas 2003). This section critically evaluates the assumption that exposure parameters for sediment assessment should be developed separately for individual bays and estuaries. The sediment chemistry (Section 3.2) and case study (Sections 4 and 5) portions of this report provide more detailed guidance on how to develop these water body-specific parameters.

The theoretical basis for developing water-body specific bioaccumulation factors is that drivers of bioaccumulation will vary among water bodies. Differences among water bodies in biota vs. sediment relationships may result from multiple factors. These factors include differences in diet, food web structure, tissue lipid content, sediment organic carbon, and sediment vs. water column disequilibrium (Burkhard *et al.* 2003). There is substantial variation in BSAFs among locations, as observed in syntheses undertaken at national and global scales (Wong *et al.* 2001, Burkhard *et al.* 2005). The analyses in this section demonstrate the same phenomenon using California data.

Methods

This section used the CASQO database and empirical methods, as described in Appendix B. Analyses were conducted on finfish, and also on 28 day laboratory bioaccumulation test data for *Macoma nasuta* (bent-nosed clam) exposed to PCBs and DDTs in sediments collected from multiple water bodies.

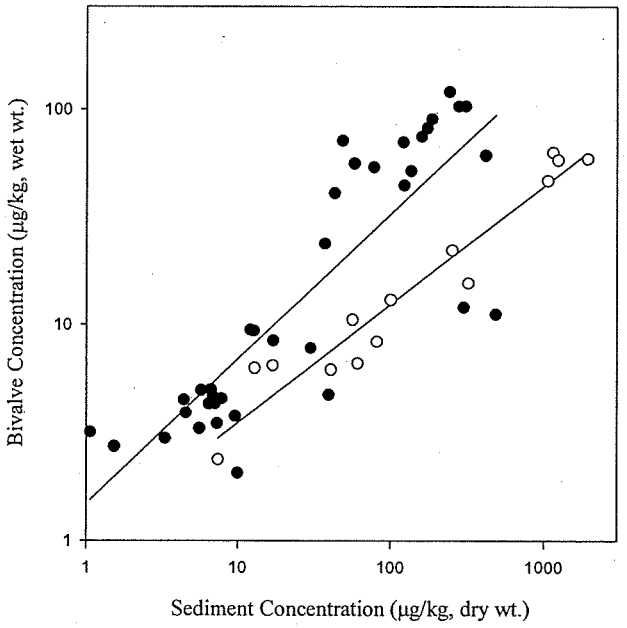
Data from were evaluated using general linear models. The focus of these analyses was determining whether statistically significant differences were observed between water bodies in sediment vs. biota pollutant relationships. The statistical approach was regression analysis using categorical (dummy) variables for individual water bodies. This is a generalization of the Analysis of Covariance model, in which general linear models are constructed and differences in slope or elevation of a linear relationship are evaluated for statistical significance (i.e., $p < 0.05$) of added parameters. This is a commonly used and well described statistical approach (e.g., Packard and Boardman 1988, Draper and Smith 1998, Tremblay *et al.* 1998, Greenfield *et al.* 2005).

The empirical comparisons among water bodies were complimented with mechanistic food web model simulations. Mechanistic model simulations were performed using the methods and parameters described in application guidance (Section 3.2.8) and the Newport Bay and San Francisco Bay case studies (Sections 4.4 and 5.3.2). Briefly, a steady state non-equilibrium food web model (Gobas 1993, Morrison *et al.* 1996, Arnot and Gobas 2004) was applied using local parameters for each water body. The model was run for selected contaminants for each water body to evaluate how model-predicted bioaccumulation factor varied between water bodies.

Results of empirical analysis

Analysis of bioaccumulation test data from multiple water bodies indicates that contaminant bioavailability varied significantly among water bodies. *Macoma nasuta* PCB tissue concentrations were significantly higher at a given sediment PCB concentration when the tests were conducted using San Francisco Bay sediments than San Diego Bay sediments (Figure C.1). Regression analysis of log-log data indicated a significant difference in the model constant (y-intercept) when a categorical variable was applied to distinguish among the data from the two bays (bay variable p-value < 0.001; N = 51). When the variable to distinguish among bays was added, the R^2 increased from 0.65 to 0.75. At the arithmetic mean sediment concentration of the data set (190 ng/g dry weight), the model predicted that laboratory *Macoma* concentrations in San Francisco Bay were 2.6 times the concentrations predicted in San Diego Bay (47 vs. 18 ng/g). Similar results were also observed comparing p,p'-DDE concentrations in paired samples from Newport Bay vs. San Francisco Bay. Both water bodies showed significant relationships between sediment and *Macoma* p,p'-DDE concentrations ($p < 0.001$), but *Macoma* concentrations were significantly higher in San Francisco Bay (for addition of categorical variable to distinguish among bays, $p < 0.0001$).

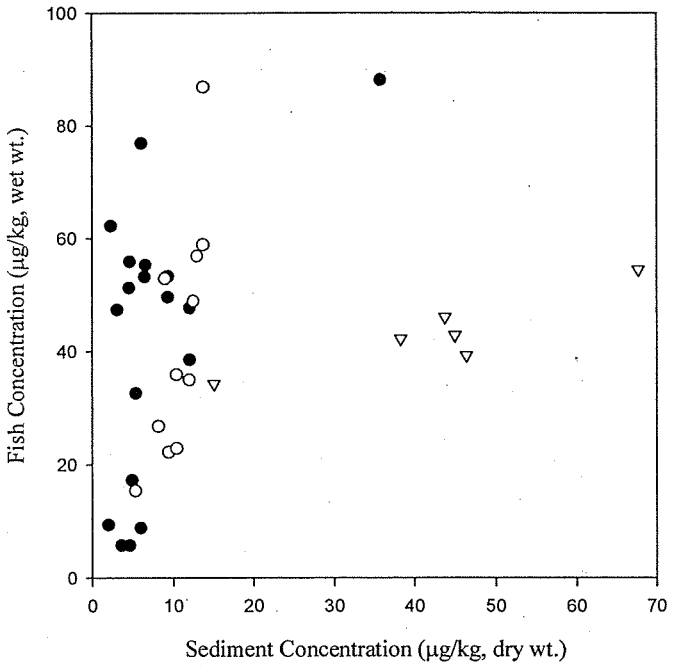
Figure C.1. Relationship between PCBs in sediments vs. *Macoma nasuta* exposed in 28 day laboratory tests. Black dots (•) = San Francisco Bay. White dots (○) = San Diego Bay. Lines represent best linear regression fit on log-log regression. Note log scale of axes.



Available field captured fish data also indicated differences in bioavailability among water bodies. California halibut vs. sediment DDTs were compared among San

Francisco Bay, San Diego Bay, and San Pedro Bay (Figure C.2). Evaluated individually, regression relationships differed among water bodies, with San Pedro Bay ($R^2 = 0.75$; $p = 0.03$; $N = 6$) and San Diego Bay ($R^2 = 0.63$; $p = 0.003$; $N = 11$) exhibiting positive relationships, while San Francisco Bay ($R^2 = 0.18$; $p = 0.08$; $N = 18$) exhibited only a weak positive trend. Figure C.2 indicates that the slope of these relationships also varied among water bodies, with fish increasing much more rapidly at a given sediment concentration in San Diego Bay, than San Pedro Bay.

Figure C.2. Relationship between total DDTs in sediments vs. California Halibut captured from different embayments. Black dots (●) = San Francisco Bay. White dots (○) = San Diego Bay. Triangles (Δ) = San Pedro Bay.



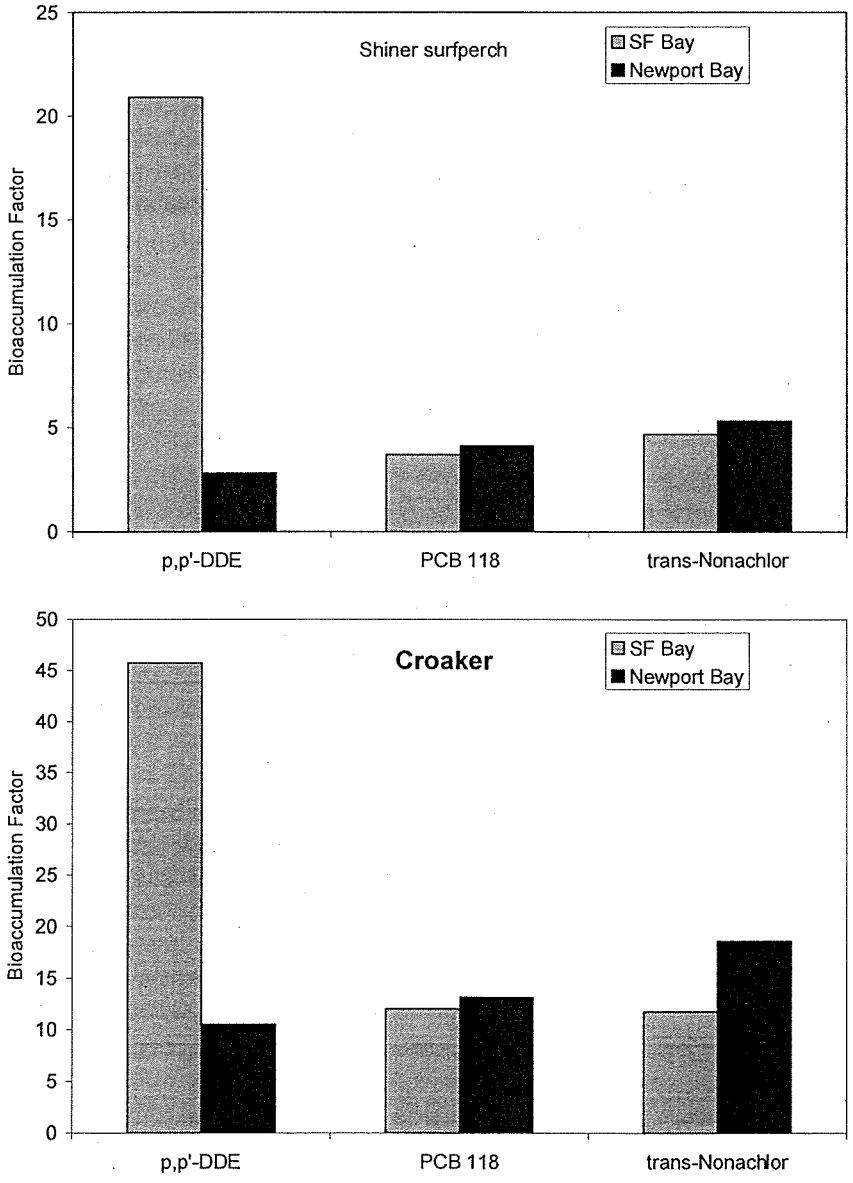
Results of mechanistic modeling

When the mechanistic model was applied using parameters from the San Francisco vs. Newport Bay case studies, differences in bioaccumulation factor were observed. Differences were most apparent for p,p'-DDE (Figure C.3). These model results suggest that BAFs may differ among the same or similar fish species, as a result of chemical or biological differences among water bodies.

Taken in combination, the empirical data analyses and mechanistic model results suggest that contaminant bioavailability may vary greatly among different bays and estuaries. Therefore, attempts to predict bioaccumulation factors using generalized equations are likely to introduce uncertainty or inappropriate sediment chemistry thresholds.

If predictive translators between sediments and biota were to be developed on a statewide basis, it would be advisable to conduct a special study examining the range of conditions among multiple water bodies of interest, by collecting new data on sediment vs. biota concentrations for widely distributed fish species.

Figure C.3. Results of mechanistic food web model predicting bioaccumulation factor (BAF) for p,p'-DDE, PCB 118, and trans-Nonachlor in similar fish from San Francisco Bay (blue) vs. Newport Bay (black). All results based on application of the food web model developed by Arnot and Gobas (2004), as described in Sections 4 and 5 of this report. Top panel: shiner surfperch (juvenile). Bottom panel: white croaker (SF Bay) vs. yellowfin croaker (Newport Bay).



Appendix D. Literature review on body length of fish consumed by piscivorous birds.

Table D.1. Results of a literature review on body length of fish consumed by piscivorous birds. The review targeted California species and locations, where possible. For large birds (Table 3.5), maximum body length of prey consumed was 473 mm. For medium sized birds, maximum prey body length was 165 mm. For least tern, fish up to 107 mm were captured (Atwood and Kelly 1984, Elliott 2005).

Predator species	Predator type [a]	Prey fish length average (mm)	Prey fish length range (mm)	Location	Reference
Bald eagle	Large bird		230-380	Shasta Co., CA	Jenkins and Jackman (1994)
Bald eagle	Large bird	274	127 - 473	Arizona	Haywood and Ohmart (1986)
Double-crested cormorant	Large bird		45 - 300	Maine and Florida	Scattergood (1950)
Osprey	Large bird		130 - 430	Minnesota	Dunstan (1974)
Western grebe	Medium bird		< 100	British Columbia	Forbes and Sealy (1990)
Western grebe	Medium bird		27 - 88	Clear Lake, CA	Lawrence (1950)
Elegant tern	Medium bird		40 - 165	San Diego, CA	Schaffner (1986)
Black skimmer	Small bird		21-55	Florida	Leavitt (1957)
Black skimmer	Small bird		< 73	Louisiana	Arthur (1927)
Least tern	Small bird		< 100	California Coast	Atwood and Kelly (1984)
Least tern	Small bird	52	17 – 107	Alameda, San Francisco Bay, CA	Elliot (2005)

a. Following Table 3.5.

Appendix E. Method for extrapolating between whole body and muscle fillet organochlorine contaminant content in fish.

In the case of lipophilic contaminants, approximate lipid equivalence among tissue types may be assumed. This assumption should be appropriate for PCBs, DDTs, and other legacy organochlorine pesticides. It may also be appropriate for dioxins, PBDEs, and other compounds having a high K_{OW}. To facilitate interconversion to the most appropriate tissue type for contaminant exposure evaluation, concentrations may be extrapolated between whole body and fillet samples, assuming lipid equivalence:

$$\text{Whole Body Concentration} = \text{Fillet Concentration} * \frac{\text{Whole Body \% Lipid}}{\text{Fillet \% Lipid}}$$

When lipid concentrations are not available in both tissue types from the same species, an empirical relationship may be used to extrapolate whole body vs. fillet (skin off) lipid content, based on published literature. The equation for this relationship is:

$$\text{(Whole Body \% Lipid)} = 2.1372\ \text{(Fillet \% Lipid)}^{1.0025}$$

This empirical model is based on published literature on the ratio between whole body and muscle (skin off fillet) lipid (Table E.1, following page). For development of this model, each separate species was treated as an individual data point, regardless of the number of trials in an individual study. The comparison results indicated a strong relationship ($R^2 = 0.93$; N = 11; Figure E.1).

Figure E.1. Relationship between whole body and muscle fillet lipid content in fish. Data from Table E.1 are graphically depicted and results of a regression on log-transformed data are presented. Note log scale, both axes.

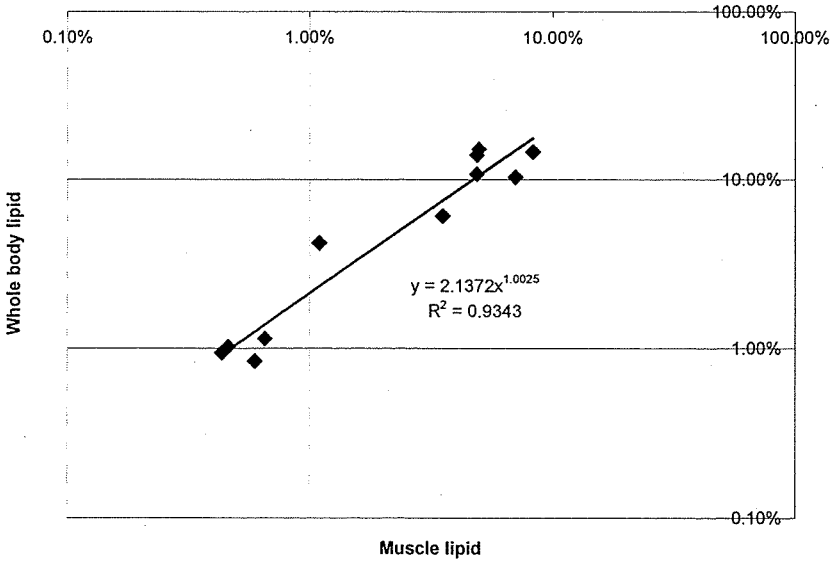


Table E.1. Summary of published data on whole-body vs. fillet tissue lipid contents in fish.

Common name	Scientific name	Body size (g)	Whole body lipid	Muscle lipid	Ratio	Notes	Reference
Turbot	*Psetta maxima*	656	4.20%	1.10%	26%	pre-treatment	C. Regost, J. Arzel, M. Cardinal, J. Robin, M. Laroche, S.J. Kaushik. 2001. Dietary lipid level, hepatic lipogenesis and flesh quality in turbot (*Psetta maxima*). Aquaculture 193 291–309.
Triploid brown trout	*Salmo trutta*	1488	14.60%	8.30%	57%	pre-treatment	C. Regost, J. Arzel, M. Cardinal, M. Laroche, S.J. Kaushik. 2001. Fat deposition and flesh quality in seawater reared, triploid brown trout *Salmo trutta* as affected by dietary fat levels and starvation. Aquaculture 193 325–345
European sea bass	*Dicentrarchus labrax* L.	92	14.00%	4.90%	35%	average of all final treatments	Domenico Lanari, Bianca Maria Poli, Rodolfo Ballestrazzi, Paola Lupi, Edo D'Agaro, Massimo Mecatti. 1999. The effects of dietary fat and NFE levels on growing European sea bass *Dicentrarchus labrax* L.). Growth rate, body and fillet composition, carcass traits and nutrient retention efficiency. Aquaculture 179 351–364
Atlantic salmon	*Salmo salar*	400.5	10.80%	4.90%	45%	average of all final treatments	B.S. Dosanjh, D.A. Higgs, D.J.McKenzie, D.J. Randall, J.G. Eales, N. Rowshandeli, M. Rowshandeli1 and G. Deacon. 1998. Influence of dietary blends of menhaden oil and canola oil on growth, muscle lipid composition, and thyroidal status of Atlantic salmon (*Salmo salar*) in sea water. Fish Physiology and Biochemistry 19: 123–134
Lake trout	*Salvelinus namaycush*		15.20%	5.00%	33%	dorsal muscle	Hoffman, AD, Jenson, CT, Lien, GJ, and McKim, JM. 1999. Individual tissue weight to total body weight relationships and total, polar, and nonpolar lipids in tissues of hatchery lake trout. Trans. Am. Fish. Soc. 128 1:178-181.
Sand lance	*Ammodytes hexapterus*		10.35%	7.05%	68%	averages	Kaneniwa, Masaki, Sato, Hajimu, Okamoto, Hiroaki, Kunimoto, Masahiko. 1997. Comparison of lipid components between two species of sand lance, *Ammodytes hexapterus* and *Ammodytes personatus*, in Northern Hokkaido. Fish. Sci. 63 4: 600-604.
Sand lance	*Ammodytes personatus*		6.05%	3.55%	59%	averages	Kaneniwa, Masaki, Sato, Hajimu, Okamoto, Hiroaki, Kunimoto, Masahiko. 1997. Comparison of lipid components between two species of sand lance, *Ammodytes hexapterus* and *Ammodytes personatus*, in Northern Hokkaido. Fish. Sci. 63 4: 600-604.

California halibut	*Paralichthys californicus*		0.94%	0.43%	46%	muscle tissue are adult; whole body forage fish	Allen *et al.* (2004)
Shiner surfperch	*Cymatogaster aggregata*		1.02%	0.46%	45%	muscle tissue are adult; whole body forage fish	Allen *et al.* (2004)
Black perch			1.14%	0.65%	57%	muscle tissue are adult; whole body forage fish	Allen *et al.* (2004)
Diamond turbot			0.84%	0.59%	71%	muscle tissue are adult; whole body forage fish	Allen *et al.* (2004)
average St. Dev. Min. Max.					49% 14% 26% 71%		

Appendix F. Power analyses to determine appropriate sample sizes for prey tissue line of evidence.

For the prey tissue line of evidence (LOE), field collections and chemical analyses can be costly. Nevertheless, accurate estimate of chemical concentrations in prey tissue is a key data need for the framework. To address this, power analyses were performed to determine appropriate sample sizes to estimate concentrations in prey tissue.

Methods

Power analyses were performed using representative data on finfish tissue concentrations of legacy contaminants. Analyses determined the upper and lower 95% confidence intervals of average tissue concentration estimates, based on the average and standard deviation of the representative data, across a range of potential sample sizes. To evaluate a range of potential conditions, two data sets were modeled. Prior to analysis, both data sets were square-root transformed to achieve normal distributions.

The first data set evaluated was 1994 data for total DDTs in white croaker in San Francisco Bay (N = 25 composite samples), obtained by Fairey *et al.* (1997). Error bars were estimated based on the average square root concentration (7.63 in square root units; 58 in linear units), the standard deviation of this average (1.77 in square root units), and the sample size. This analysis likely represents a low-end estimate of total variance (leading to relatively high power), because the data represent a single species in a single water body.

The second data set evaluated included all fish tissue data from the CASQO database for total PCBs in California fish (N = 378). Error bars were estimated based on the average square root concentration (9.08 in square root units; 82 in linear units) the standard deviation of this average (3.87 in square root units), and the sample size. This analysis likely represents a high-end estimate of total variance (leading to relatively low power), because the data represent multiple species in multiple water bodies.

Results and interpretation

The first power analysis was based on parameters estimated for total DDTs in white croaker analyzed in 1994 in San Francisco Bay. Based on this analysis, sample sizes below 10 resulted in upper and lower confidence estimates of averages between 70% and 130% of the actual average concentration (Figure F.1). In the second analysis, parameters were estimated using PCB concentrations in all fish in California waters. The analysis indicated that sample sizes below 10 resulted in upper and lower confidence estimates of averages between 50% and 170% of the average concentrations (Figure F.2). It should be noted that this latter simulation is likely to overestimate actual variability in concentrations in individual bays or estuaries, because of the additional variation between estuaries.

Based on these results, sample sizes should be at least 10 to make assessments for the fish tissue line of evidence for a particular water body.

Figure F.1. Results of power analysis on ability to estimate average tissue concentrations (wet weight), for DDTs in white croaker collected in 1994. Bar represents average concentration, and error bars represent upper and lower 95% confidence intervals of the average.

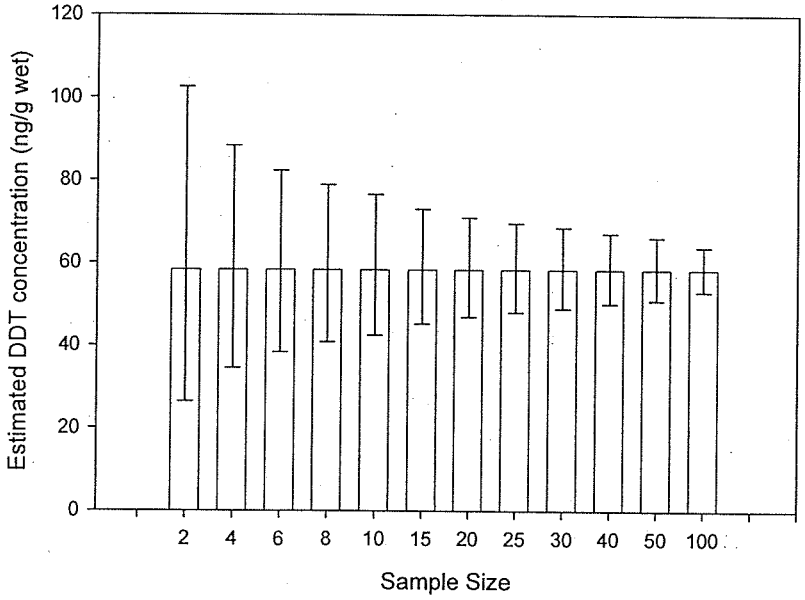
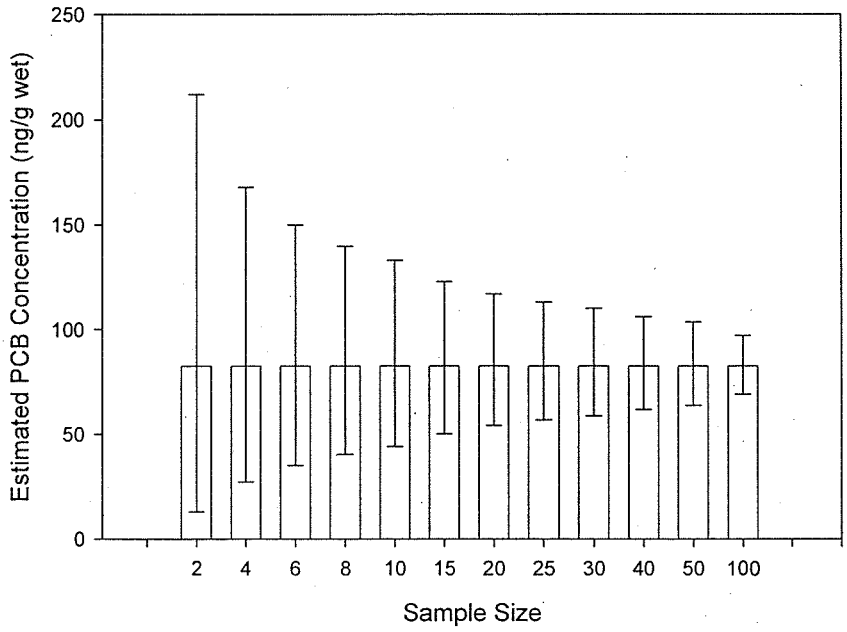


Figure F.2. Results of power analysis on ability to estimate average fish tissue concentrations (wet weight), for total PCBs in California fish. Bar represents average concentration, and error bars represent upper and lower 95% confidence intervals of average.



Appendix G. Background information on averaging methods

Typical methods of averaging data include arithmetic mean, geometric mean, and median. For the purposes of estimating contaminant concentration and exposure, each method has relative advantages and disadvantages (Helsel and Hirsch 2002). Contaminant concentrations are typically lognormally distributed, rather than normally distributed, suggesting that geometric mean better represents the central tendency of the distribution (MacLeod *et al.* 2002). Medians are less sensitive to outliers, such as concentrations below detection limits.

Arithmetic mean is most appropriate to represent mass transfer processes such as overall contaminant exposure of predators due to consuming contaminated prey. This is because arithmetic mean will best represent the importance of uncommon occurrences of high magnitude, such as occasional consumption of highly contaminated prey items (Helsel and Hirsch 2002). Arithmetic mean will be higher, and thus more conservative for estimating contaminant exposure. In general, averages and upper confidence intervals should be estimated using arithmetic and geometric mean, in order to estimate the skewness of the data-set[40]. If the different averaging procedures yield differing results, it would be more appropriate to use the arithmetic mean because it accounts for the relative importance of rare but highly contaminated samples.

In addition to calculating the arithmetic mean (μ), the 95% upper confidence interval (UCI) of the mean should be calculated as:

$$95\%\ UCI = \mu + 2^{*}\sigma$$

$$\text{where } \sigma = \text{standard error of mean} = \frac{\text{standard deviation}}{\sqrt[2]{n}}$$

and n = total number of samples.

The geometric mean is calculated as:

$$10^{\left[\frac{\sum_{i}^{n} Log_{10}(x_i)}{n}\right]}$$

where x_i = each individual sample measurement, and n = the total number of samples.

However, in many cases, some sample concentrations will be below detection limits (ND). It is not possible to determine geometric mean concentrations, estimating ND values as zero (as log(0) does not exist). To handle ND values, values may be set to ½ of

[40] For data skewed towards lower values (i.e., long tail of higher concentrations), the geometric mean will be lower than the arithmetic mean.

the detection limit or values may be estimated for averaging by the robust methods of Helsel and Hirsch (2002).

To estimate geometric mean in instances with ND values set at zero, it is also possible to obtain an estimate of geometric mean by adding 1 to each value, obtaining the geometric mean, subtracting 1 from the geometric mean, and exponentiating. That is, geometric mean estimate equals:

$$10^{\left[\frac{\sum_{i}^{n} Log_{10}(x_i+1)}{n}\right]} - 1$$

where x_i = each individual sample measurement, and n = the total number of samples.

Geometric standard error of the mean (σ) is simply the standard error of all log transformed values.

The 95% upper confidence interval (UCI) of the geometric mean may be calculated as:

$$10^{\left[\frac{\sum_{i}^{n} Log_{10}(x_i+1)}{n}\right]} + 2*\sigma$$

Appendix H. Toxicity reference values (TRVs), reference doses (RfD), and cancer slope factors (CSF) for birds, mammals, and humans.

Table H.1. A compilation of toxicity reference values (TRVs), reference doses (RfD), and cancer slope factors (CSF) for birds, mammals, and humans. Prey tissue chemistry thresholds in the framework could be based on these or other appropriate TRVs, as decided by risk assessors.

Contaminant	Mammal Low TRV mg dw/(kg ww*day)	Mammal High TRV mg dw/(kg ww*day)	Ref.	Avian Low TRV mg dw/(kg ww*day)	Avian High TRV mg dw/(kg ww*day)	Ref.	Human Noncarcinogen RfD (mg/kg*d)	Human Carcinogen CSF (kg*d/mg) More protective	Ref.
Sum 6 DDTs	0.8	16	1	0.009	0.6	1 d	5.00E-04	0.34	5
PCBs a	g	1.28	1	0.09	1.27	1	2.00E-05	2	5
Aldrin	0.1	1	1	NA	NA		3.00E-05	1.7	5
Heptachlor	0.13	6.8	1	NA	NA		5.00E-04	4.5	5
Total Chlordanes b	0.15	7.7	2, 6	0.14	7.0	3 i	5.00E-04	0.35	5 e
Dieldrin	0.015	1.80	4 h	0.0709	1.05	4 h	5.00E-05	16	5
Toxaphene	NA	NA		NA	NA		NA	1.1	5
Mercury	0.027	0.27	1 c	0.039	0.18	1	1.00E-04	NA	5 f

Notes to Table H.1:

a. Total congeners or aroclors
b. Sum of cis and trans chlordane, cis and trans Nonachlor and oxychlordane
c. BTAG value for large mammals (mink)
d. Avian high TRV for total DDTs based on p,p'-DDE, which is the major congener
e. Note that OEHHA CSF for chlordanes was different from U. S. EPA IRIS, and was set at 1.3 (Klasing and Brodberg 2006).
f. The mercury RfD is the value to protect the fetus from neurodevelopmental effects. It is applicable to women of childbearing age to protect the fetus.
g. PCB Mammal Low TRV is a tissue concentration reference value (500 mg/kg), rather than a dose-based TRV (mg/(kg*day)) (Chapman 2003).
h. The TRV-High is geometric mean of all avian or mammalian values for survival, growth, and reproductive LOAEL dose values, reported in U. S. EPA (2005).

NA = Not available due to insufficient data

1. California DTSC Human and Ecological Risk Division (California DTSC Human and Ecological Risk Division 2000)
2. Khasawinah and Grutsch (1989)
3. Stickel *et al.* (1983)
4. U. S. EPA (2005)
5. U. S. EPA (2000b) EPA IRIS Database. http://www.epa.gov/iris/index.html
6. World Health Organization (1984) (primary citation Keplinger *et al.* 1968)

Description of toxicity reference values in Table H.1

Table H.1 presents a set of wildlife and human TRVs that may be considered for use in the assessment framework. These TRVs may be input into Equations 3.1, 3.2, and 3.3 (from Section 3.1.10), following the example in Figure 3.3. For avian and mammalian targets, Table H.1 includes low (no observable adverse effects level) and high (mid-range of lowest observable adverse effects) toxicity reference values (TRVs). This corresponds with the assessment framework's use of two risk thresholds. In particular, for wildlife targets, the low TRV may be used to derive low prey tissue thresholds and the high TRV may be used to derive high prey tissue thresholds. Example thresholds for generic bird and mammal targets are presented in Appendix J.

The human TRVs in Table H.1 include reference doses for non-carcinogenic effects, and cancer slope factors for carcinogenic effects. All of these values were taken from the U. S. EPA's IRIS database (U. S. EPA 2006b),. Example thresholds for human targets are presented and discussed in Appendix K.

Whenever possible, the TRVs in Table H.1 were developed using a multi-agency consensus approach. Two sets of such values were available for inclusion. These are the Navy/BTAG TRVs (California DTSC Human and Ecological Risk Division 2000) and the U. S. EPA ECO-SSL values (U. S. EPA 2005). Navy/BTAG TRVs have been used in recent risk assessments in California estuaries (von Stackelberg *et al.* 2003, Arcadis G&M Inc. and Matrix Design Group 2004, Zeeman 2004). The derivation and basis for these TRVs are presented elsewhere (PRC Environmental Management 1997, California DTSC Human and Ecological Risk Division 2000). For dieldrin, the Navy/BTAG TRVs are not available; U. S. EPA ECO-SSL values are presented instead (U. S. EPA 2005) (Table H.1).

Where warranted, TRVs for specific compound classes are discussed further in the remainder of this section.

DDTs

Of the TRVs presented, the lowest is the avian TRV-Low for DDTs. This TRV is based on a field study of brown pelicans in California (Anderson *et al.* 1975, Anderson *et al.* 1977). It was recommended by the U. S. EPA as part of the Great Lakes Water Quality Initiative (U. S. EPA 1995a), and by BTAG for risk assessment in California (California DTSC Human and Ecological Risk Division 2000). It has also been used by the U. S. Army Corps of Engineers for a risk assessment in Moss Landing Harbor, California (von Stackelberg *et al.* 2003), by the U. S. Fish and Wildlife Service for risk assessment in San Diego Bay (Zeeman 2004), and in a risk assessment for the former Oakland Army Base (Arcadis G&M Inc. and Matrix Design Group 2004). In Anderson *et al.* (1975), concentrations of DDD, DDE, and DDT from 1969 to 1974 were associated with adverse impacts to eggshell thickness and reproductive success. A dose-response relationship was observed, and adverse effects were still observed in the least contaminated population. An uncertainty factor of 3 was used to convert this LOAEL to an NOAEL

value, resulting in a final TRV of 0.009 mg/(kg body weight*day) (Anderson *et al.* 1975, Anderson *et al.* 1977, U. S. EPA 1995a).

Dieldrin

For dieldrin, the U. S. EPA ECO-SSL would be appropriate for a TRV-Low (values are presented for birds and mammals in Table H.1). The ECO-SSL TRV is based on NOAELs, and may therefore be too conservative for a TRV-High. To obtain a TRV-High for dieldrin, the synthesis of appropriate studies conducted to generate the ECO-SSL may be used (U. S. EPA 2005). Specifically, the geometric mean of all avian and mammalian LOAELs for survival, growth, and reproductive effects would be an appropriate TRV-High. Based on this calculation, the TRV-High for dieldrin in mammals is 1.80 mg/kg/d (N = 34 studies) and the TRV–High for dieldrin in birds is 1.05 mg/kg/d (N = 37) (Table H.1).

PCBs

Table H.1 does not include a TRV – Low for mammals. Instead, footnote g of Table H.1 references use of a prey tissue concentration of 0.5 mg/kg. The Navy/BTAG TRVs for PCBs in mammals are based on rodents, which are not the most sensitive indicator, and are also not aquatic animals. Of aquatic mammals evaluated, mink is the most sensitive indicator for PCBs (Jones *et al.* 2001). A recent review conducted in the Great Lakes Region has summarized the literature on NOAELs and LOAELs in mink (Chapman 2003). Based on this review, a PCB concentration TRV of 0.5 mg/kg in prey tissue would be an appropriate TRV-Low (Chapman 2003). Note that this is a prey tissue concentration TRV (units of mg/kg), rather than a dose-based TRV (units of mg/[kg*d]).

Chlordanes

Chlordane TRV development presents a challenge, because there have been few studies of dietary uptake in animals, and no consensus evaluations. For total chlordanes in birds, Table H.1 includes TRVs based on Stickel *et al.* (1983). This set of values is based on dietary uptake of chlordanes by red-winged blackbird, and was recommended for TRV calculation by Sample *et al.* (1996). It was also recommended by staff members of the CA Department of Toxic Substances Control (M. Anderson, DTSC, *Pers. comm.*), and has been used in a number of ecological risk assessments in California (von Stackelberg *et al.* 2003, Arcadis G&M Inc. and Matrix Design Group 2004).

Chlordane calculations for birds were as follows. Stickel et. al (1983) reports a mortality NOAEL of 10 mg chlordane/kg food and a mortality LOAEL of 50 mg chlordane/kg food. The study exposure duration was 84 days, which may be interpreted as chronic or subchronic exposure (Sample *et al.* 1996). Food intake rate is 9 g/day based on allometric calculations for omnivorous birds and a reported body mass of 64 g (Nagy 2001). A TRV-High was calculated based on the LOAEL, and assuming that 84 days represents chronic exposure, as:

(50 mg chlordane/kg food)*(0.009 kg food intake/day)/0.064 kg body mass
= 7.0 mg/(kg body weight*day)

A TRV-Low was calculated based on the NOAEL, assuming that exposure was subchronic and reducing by a factor of 10 as a conservative estimate of chronic exposure from the subchronic exposure (Sample *et al.* 1996):

(10 mg chlordane/kg food)*(0.009 kg food intake/day)/
(0.064 kg body mass * 10 correction factor)
= 0.14 mg/(kg body weight*day)

For total chlordanes in mammals, Table H.1 includes a TRV - Low based on Khasawinah and Grutsch (1989) and a TRV – High based on World Health Organization (1984) (primary citation Keplinger *et al.* 1968). These values are based on dietary uptake of chlordanes by mice. Khasawinah and Grutsch (1989) was recommended for TRV calculation by staff members of CA Department of Toxic Substances Control, and has been used in a number of military base ecological risk assessments in California (M. Anderson, DTSC, *Pers. comm.*). Keplinger *et al.* (1968) were recommended for TRV calculation by Sample *et al.* (1996), and subsequently used by von Stackelberg (2003).

The chronic LOAEL in Keplinger *et al.* (1968) is 50 mg chlordane/kg food. Food intake rate is 4.6 g/day based on allometric calculations for rodents and a reported body mass of 30 g (Nagy 2001). A TRV-High based on this LOAEL is:

(50 mg chlordane/kg food)*(0.0046 kg food intake/day)/0.030 kg body mass
= 7.7 mg/(kg body weight*day)

Khasawinah and Grutsch (1989) report a chronic NOAEL of 1 mg chlordane/kg food. Food intake rate is 4.6 g/day based on allometric calculations for mice and a reported body mass of 30 g (Nagy 2001). A TRV-Low based on this NOAEL is:

(1 mg chlordane/kg food)*(0.0046 kg food intake/day)/0.030 kg body mass
= 0.15 mg/(kg body weight*day)

Based on evaluation of the CASQO database and other studies, exceedance of these chlordane thresholds (both low and high) is highly unlikely for California fishes collected in the recent past.

Heptachlor, aldrin and toxaphene

The BTAG has previously developed mammalian TRVs for the pesticides, heptachlor and aldrin (Table H.1). However, avian TRVs have not been developed for these pesticides by BTAG or by Sample *et al.* (1996), and published dose-response studies on these compounds are lacking. The BTAG has not developed mammalian or avian TRVs for toxaphene. Heptachlor and aldrin are not currently present in detectable concentrations in fish in California waters, and toxaphene concentrations are rarely

detected (Davis *et al.* 1999, Greenfield *et al.* 2003, Allen *et al.* 2004, Greenfield *et al.* 2004). Therefore, development of additional TRVs for these contaminants is a relatively low priority.

Appendix I. Potential thresholds for protection of fish

Fishes represent another receptor that may be adversely impacted due to indirect effects from sediment contamination. Finfish may be impacted by dietary exposure to contaminated prey residing in contaminated sediments, as well as consumption of and direct contact with contaminated sediments.

Table I.1 lists thresholds from several recent syntheses of biological effects to fish. Each of these syntheses was a careful well-documented development of an effects threshold based on appropriate studies. Each synthesis conducted a broad review of available literature, and included a screening step to remove inappropriate studies. These studies were also discussed and favorably reviewed by members of Region IX BTAG (Laurie Sullivan, NOAA-National Ocean Service, *Pers. comm.*), although no formal statement has been made.

Table I.1. Literature recommended values for protection of finfish. All studies represent syntheses of the latest available literature at the time of publication. ww fish = wet weight fish tissue concentration; lw fish = lipid weight fish tissue concentration; dw = dry weight sediment concentration

Group	Mercury	DDT	PCB	PAH	Units and matrix	Endpoint	Source
Juvenile and adult fish	0.2	0.6			ppm ww fish	growth, reproduction, development, behavior	a
Early life-stage fish		0.7			ppm ww fish	growth, reproduction, development, behavior	a
Juvenile salmonids			2.4		ppm lw fish tissue	growth, development, mortality, disease, biochemical	b
English sole				1.0	ppm dw sediment	growth, reproduction, tumor development	c

a. Beckvar *et al.* (2006)
b. Meador *et al.* (2002b)
c. Johnson *et al.* (2002)

Appendix J. Selected prey tissue thresholds for protection of generic wildlife consumers of finfish and shellfish.

Table J.1. Selected prey tissue thresholds for protection of generic wildlife consumers of finfish and shellfish. Wildlife categories are based on generic estuary wildlife in California, following Table 3.5. Thresholds are calculated based on information in H.1, following methodologies in the text (see Equation 3.1 and Figure 3.3). Note that all thresholds are tissue dry weight thresholds (ppm). MA = Body Mass. NA = Not available due to insufficient data

Group	Body Mass (kg) [a]	Allometric Equation [a,b]	Daily Ingestion Rate (kg dw food/day)	Ingestion Rate (kg dw food/(kg body weight*day))	DDT Low (ppm dry weight fish tissue)	DDT High (ppm dry weight)	PCB Low (ppm dry weight)	PCB High (ppm dry weight)	Chlordane Low (ppm dw)	Chlordane High (ppm dw)	Dieldrin Low (ppm dw)	Dieldrin High (ppm dw)	Heptachlor Low (ppm dw)	Heptachlor High (ppm dw)	Aldrin Low (ppm dw)	Aldrin High (ppm dw)	Mercury Low (ppm dw)	Mercury High (ppm dw)
Small Birds	0.025	$0.880(MA\ g)^{0.658}$	0.007	0.293	0.03	2.05	0.31	4.34	0.48	23.9	0.24	3.59	NA	NA	NA	NA	0.13	0.62
Medium Birds	0.3	$0.880(MA\ g)^{0.658}$	0.038	0.125	0.07	4.80	0.72	10.2	1.12	56.0	0.57	8.39	NA	NA	NA	NA	0.31	1.44
Large Birds	1	$0.880(MA\ g)^{0.658}$	0.083	0.083	0.11	7.24	1.09	15.3	1.69	84.5	0.86	12.7	NA	NA	NA	NA	0.47	2.17
Small Mammals	20	$0.153(MA\ g)^{0.834}$	0.591	0.030	27.1	541	0.5 [c]	43.3	5.07	260	0.51	60.9	4.40	230	3.38	33.8	0.91	9.13
Large Mammals	90	$0.153(MA\ g)^{0.834}$	2.073	0.023	34.7	695	0.5 [c]	55.6	6.51	334	0.65	78.2	5.64	295	4.34	43.4	1.17	11.7

a. From Table 3.5.
b. Note that units are in grams.
c. Following Appendix H, the PCB Mammal Low TRV is a tissue concentration reference value (500 mg/kg), rather than a dose-based TRV (Chapman 2003).

Appendix K. Selected prey tissue thresholds for protection of human consumers of finfish and shellfish.

Table K.1 illustrates some potential tissue thresholds for protection of human consumers of fish. These thresholds are calculated based on a range of assumptions recently presented by California and national regulatory agencies for protection of consumers of wild-caught fish (Brodberg and Pollock 1999, U. S. EPA 2000b, 2000d, OEHHA 2001, Klasing and Brodberg 2006). All thresholds were calculated according to U. S. EPA guidance (U. S. EPA 2000b), summarized in Section 3.1.10, 3.1.11, and 3.1.13). Reference doses and cancer slope factors were taken from Appendix Table H.1, based on U. S. EPA (2006b). Body masses were set at 70 kg. The risk factor for carcinogens varied between 10^{-4} and 10^{-6}, and consumption rates varied from subsistence fishers, to sport fishers, to the general population. The risk calculation that uses a less conservative combination of assumptions is in the top row of the table, and each following row is increasingly more conservative.

Table K.1 includes the following sets of thresholds and assumptions:

- Risk calculations presented in a draft guidance document prepared by OEHHA scientists for developing guidance tissue levels in California waters (Klasing and Brodberg 2006).
- The risk calculation following U. S. EPA recommendations for screening values to protect sport fishers and the general public (U. S. EPA 2000b).
- A risk calculation presented by OEHHA as a screening value to protect human consumers of sport fish in California waters (Brodberg and Pollock 1999), and used in calculating many OEHHA consumption advisories (R. Brodberg, OEHHA, *Pers. comm.*).
- A risk calculation based on an estimated 95 percentile consumption rate by sport fish consumers in San Francisco Bay (SFEI 2000)[41].
- The risk calculation that was legislated in the California Toxics Rule (U. S. EPA 2000d).
- The risk calculation following U. S. EPA recommendations for screening values to protect subsistence fishers (U. S. EPA 2000b).

The case studies in the report (Sections 4 and 5) use the U. S. EPA screening values and the calculation based on 95 percentile sport fish consumer as the bases for the high and low tissue thresholds. These are highlighted in grey in Table K.1. These selections should not be viewed as specific recommendations for statewide sediment quality

[41] U. S. EPA does not provide recommendations for an upper percentile intake rate for recreational sport fishers or the general public. As discussed in OEHHA (2001), the San Francisco Bay Seafood Consumption Study is the most recent peer-reviewed study to calculate confidence distributions for sport fish consumers in California. This study calculates consumption rates based on 12 month recall or 4 week recall. The 12 month recall is more likely prone to errors, and was not adjusted for avidity bias (a sampling bias introduced by positive correlation between consumption rate and frequency of fishing). Therefore, the 4 week recall rate was used to calculate the 95 percentile sport fish consumer consumption rate. In short, this set of thresholds was based on a consumption rate of 32 g/d, which is the 95th percentile consumption rate of all sport fish consumers surveyed (4 week recall), adjusted for avidity bias (Table 5.6) (SFEI 2000).

evaluation guidance. As discussed elsewhere in the report[42], threshold development involves policy judgments, and must occur in a policy arena with sufficient opportunity for stakeholder review and feedback.

[42] See the Acknowledgements and Disclaimer section, as well as Sections 3.1.10 and 3.1.13.

Table K.1. Some fish tissue thresholds for protection of human consumers of finfish and shellfish in California bays and estuaries. Bold values are the lower threshold among the cancer risk and non-cancer risk (reference dose) approaches. Grey rows are used to develop high and low fish tissue and sediment thresholds in report case studies (Sections 4 and 5). All scenarios assume a consumer body mass of 70 kg (U. S. EPA 2000b).

Description of combination of assumptions	Risk factor	Risk factor reference	Consumption rate (kg/day)	Consumption rate reference	Cooking reduction	Lifespan exposure	Cancer DDT (ppb wet weight)	Non-cancer DDT (ppb wet weight)	Cancer PCB (ppb wet weight)	Non-cancer PCB (ppb wet)	Cancer chlordane (ppb ww)	Non-cancer chlordane (ppb ww)	Cancer dieldrin (ppb ww)	Non-cancer dieldrin (ppb ww)	Cancer heptachlor (ppb ww)	Non-cancer heptachlor (ppb ww)	Cancer aldrin (ppb ww)	Non-cancer aldrin (ppb ww)	Cancer mercury (ppm ww)	Non-cancer mercury (ppm ww)
OEHHA draft screening values (SV) (2006)	10^{-4}	a	0.0900	a	0.7	0.43	760	**560**	130	**20**	**200**	560	**16**	56	NA	NA	NA	NA	NA	**0.23**
U. S. EPA SV sport fishers & general pop.	10^{-5}	b	0.0175	c	1	1	**118**	2000	**20**	80	**114**	2000	**2.5**	200	**8.9**	2000	**23.5**	120	NA	**0.40**
OEHHA screening values (1999)	10^{-5}	b	0.0210	d	1	1	**98**	1667	**17**	67	**95**	1667	**2.1**	167	**7.4**	1667	**19.6**	100	NA	**0.33**
Sport fisher 95%ile consumption rate	10^{-5}	b	0.0320	e	1	1	**64**	1094	**11**	44	**63**	1094	**1.4**	109	**4.9**	1094	**12.9**	66	NA	**0.22**
California Toxics Rule value	10^{-6}	f	0.0065	f	1	1	**32**	5385	**5.4**	215	**31**	5385	**0.7**	538	**2.4**	5385	**6.3**	323	NA	**1.08**
U. S. EPA SV for subsistance fishers	10^{-5}	b	0.1424	g	1	1	**14**	246	**2.5**	10	**14**	246	**0.3**	25	**1.1**	246	**2.9**	15	NA	**0.05**

a. Klasing and Brodberg (2006)
b. Used for OEHHA and U. S. EPA screening values (Brodberg and Pollock 1999, U. S. EPA 2000b)
c. Consumption rate for sport fishers and general population (U. S. EPA 2000b)
d. Santa Monica Bay Seafood Consumption Study (SCCWRP and MBC 1994, Allen *et al.* 1996). Recommended by OEHHA (OEHHA 2001)
e. 95th percentile consumption rate of sport fish consumers surveyed in San Francisco Bay (4 week recall) (SFEI 2000)
f. U. S. EPA (2000d)
g. Subsistence fisher consumption rate (U. S. EPA 2000b)

Appendix L. Description of procedure to determine spatial association between sediment and biota contaminant concentrations.

A statistical procedure was developed to address difficulty evaluating animal home range, and need for overlapping fish and sediment samples (CH2M HILL 2004). In this procedure, fish or other mobile organisms are averaged at each discrete sampling location. The average concentration is then compared to sediment samples located in a circle centered at the spatial coordinates of that sampling location (Figure L.1). Sediment results are averaged over the entire pooled spatial area, and the average result is paired with the average of organism tissue concentrations at that location. Regression analysis is conducted to evaluate the paired results. Following standard practice in linear regression analysis, residuals should be checked for normality and variance homoskedasticity, and biota and/or sediment concentrations transformed if necessary (Draper and Smith 1998). Frequently, log-transformation of both sediment and biota data is necessary. The procedure may be applied using lipid normalized or wet weight tissue data and organic carbon or dry weight sediment data. The procedure does not work on data sets having less than five separate biota sampling locations, as a lot of spurious correlations occur. The size of the circle may be varied at increasing radial distance (e.g., 0 – 10 km at 1 km increments) to evaluate overall correlation between biota and sediment concentrations, and identify the spatial scale at which the correlation is greatest (Figure L.2).

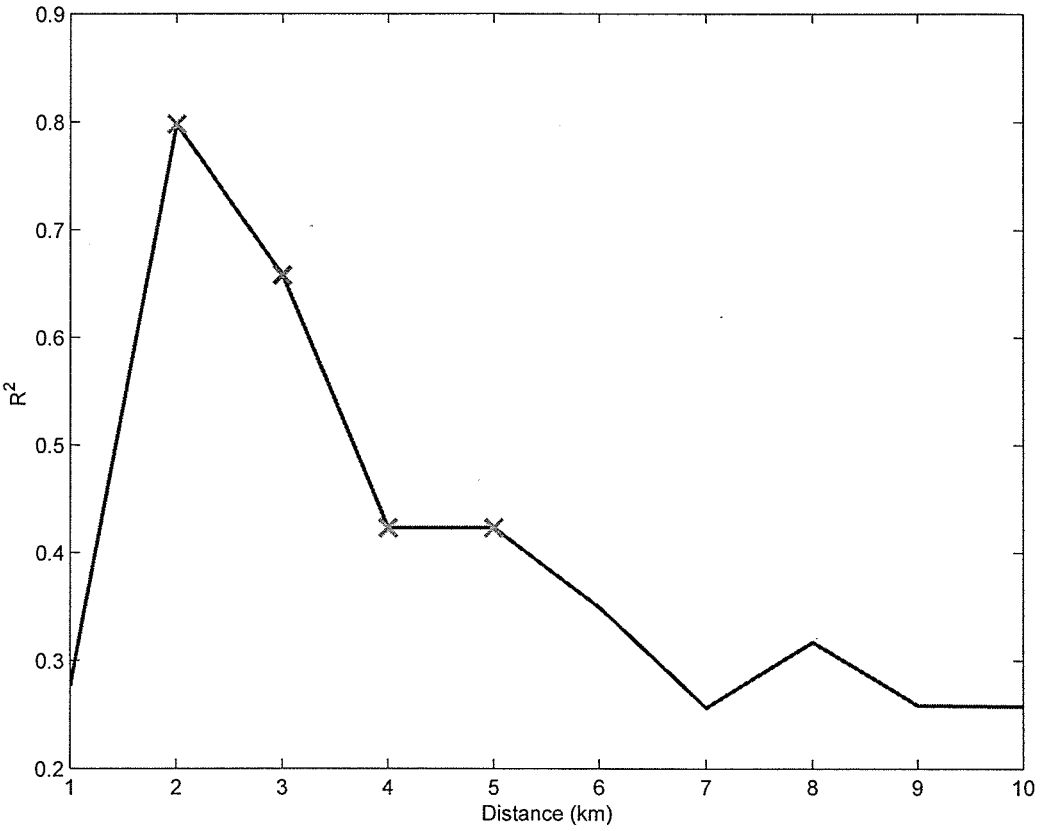
This method can be used for several purposes:

- Identify the spatial scale of biota exposure to sediments, when fish may be expected to range across a large area.
- Identify species with relatively strong spatial association to sediment contamination, based on significant sediment vs. biota concentrations. Such species can be appropriate for developing empirical BAFs for the sediment chemistry line of evidence.
- Develop a data set for determining empirical BAFs when biota and sediment sampling were not at identical locations, as is frequently the case when combining sediment and fish chemistry databases.

Conceivably, this method could also be used to develop linear regression models for predicting BAFs across a range of sediment concentrations. In theory, this provides the advantage of allowing BAF to vary according to the statistically determined slope and y-intercept of a particular empirical data set (Packard and Boardman 1988, Hebert and Keenleyside 1995). In practice, however, these models tend to have high predictive uncertainty and not be very useful. This results from limited available data, the log-transformations, and high variability of the data set. Though such a use could certainly be explored to develop bioaccumulation models in specific circumstances, it was determined to be inappropriate for the Newport Bay and San Francisco Bay case studies.

Figure L.1. Graphical depiction of comparison of fish vs. sediment data in a circle at increasing radial distance from the fish. Red dots represent fish samples, and blue dots represent sediment samples.

Figure L.2. Example of model output from spatial association procedure. Results are presented for chlordanes (wet weight) in white croaker in San Pedro Bay (N = 10). Red X's indicate significant relationships ($p < 0.05$) at the given spatial scale. Note that this is the same relationship as presented in Figure B.1 and Table B.2.



Appendix M. Summary of parameters and equations for Arnot and Gobas mechanistic food web model.

This model is used to corroborate empirical field results in this report. All model equations and assumptions have been presented elsewhere (Gobas 1993, Arnot and Gobas 2004, Gobas and Arnot 2005).

Abiotic Input Parameters

Cox = dissolved oxygen concentration (mg O_2/L)
T = mean water temperature
Kow = octanol water partitioning coefficient
KowTS = octanol water partitioning coefficient (corrected for temperature and salinity)
cwater = contaminant concentration in waters
csed = contaminant concentration in sediments
salinity = water salinity (PSU)
MCS = molar concentration of seawater at 35 ppt (mol/L)
SPC = Setschenow proportionality constant (L/cm^3)
ocsed = organic carbon proportion in sediment (kg/kg)
vss = concentration of suspended solids (kg/L)
xpoc = POC concentration in H_2O (kg/L)
xdoc = DOC concentration in H_2O (kg/L)
dpoc = disequilibrium factor for POC partitioning
ddoc = disequilibrium factor for DOC partitioning
alphapoc = proportionality constant describing phase partitioning of POC and DOC
alphadoc = proportionality constant describing phase partitioning of POC and DOC

Biotic Input Parameters

A, B = constants for phytoplankton aqueous uptake rate
Wb = body weight
assimEff(i) = assimilation efficiency for lipid(i = 1) nlom (i = 2) and water (i = 3)
EdA = Constant A in dietary uptake efficiency equation
EdB = Constant B in dietary uptake efficiency equation
lipid = tissue lipid content
nlom = tissue non-lipid organic matter content
beta = lipid-equivalency conversion factor for bioconcentration factor for non-lipid organic matter
betap = plant lipid-equivalency conversion factor for bioconcentration factor (for nlom)
wc = tissue water content
mo = proportion of respiration or transpiration due to overlying water column
mp = proportion of respiration or transpiration due to porewater
kM = metabolic rate constant for contaminant in biota (set to zero in this study)
scav = filter feeding particle scavenging efficiency
preyprop = proportion of diet due to individual prey (calculated from prey proportion matrix)

vld = proportion of diet that is lipid (calculated from prey proportion matrix)
vnd = proportion of diet that is non-lipid organic matter (calculated from prey proportion matrix)
vwd= proportion of diet that is water (calculated from prey proportion matrix)
kGconst = growth rate equation constant
scav = scavenging efficiency of particles absorbed from water by filter feeders (percent)

Table M.1. Constant values set for model simulations (following Arnot and Gobas 2004, Gobas and Arnot 2005).

Constant	Value	Description
MCS	0.5	Molar concentration of seawater at 35 ppt (mol/L)
SPC	0.0018	Setschenow proportionality constant (L/cm^3)
A	6.00E-05	Constant for phytoplankton aqueous uptake rate
B	5.5	Constant for phytoplankton aqueous uptake rate
alphapoc	0.35	Proportionality constant describing phase partitioning of POC
alphadoc	0.08	Proportionality constant describing phase partitioning of DOC
dpoc	1	Disequilibrium factor for POC partitioning
ddoc	1	Disequilibrium factor for DOC partitioning
EdA	8.50E-08	Constant A in dietary uptake efficiency equation
EdB	2	Constant B in dietary uptake efficiency equation
scav	100%	Scavenging efficiency of particles absorbed from water by filter feeders (percent)

Model Variables (calculated for each contaminant and organism combination)

k1 = aqueous uptake rate constant
k2 = elimination rate constant
kG = growth rate
Gv = gill ventilation rate
Gd = feeding rate
Gf = fecal egestion rate
vlg = lipid fraction of gut
vng = nlom fraction of gut
vwg = water fraction of gut
kgb = gut-biota partition coefficient
ke = fecal egestion rate constant (1/d)
kd = dietary uptake rate constant
Ew = contaminant-specific gill chemical uptake efficiency
Ed = contaminant-specific dietary chemical transfer efficiency (also called gut uptake efficiency)
phi = freely dissolved contaminant fraction in overlying water column
cpw = contaminant concentration in porewater
kbw = biota-water partition coefficient (i.e., bioconcentration factor)
cbiota = contaminant concentration in biota

cprey = contaminant concentration in prey diet

Model Calculations

Ew = 1/(1.85+1.55/KowTS)

Ed = 1/(EdA*Kow + EdB)

phi = 1/(1 + xpoc*dpoc*alphapoc*KowTS + xdoc*ddoc*alphapoc*alphadoc*KowTS)

cpw = csed/(ocsed*0.35*KowTS)

Calculations for phytoplankton and benthic algae

k1 = 1/(A + B/KowTS)

kbw = (lipid*KowTS+ nlom*betap*KowTS + wc)

k2 = k1/kbw

cbiota=k1*(mo*phi*cwater + mp*cpw)/ (k2 + kG + kM)

Calculations for invertebrates and fishes

Gv = (1400*Wb^0.65)/Cox

k1 = Ew*Gv/Wb

kbw = (lipid*KowTS + nlom*beta*KowTS + wc)

k2 = k1/kbw

Gd = 0.022 * (Wb^0.85) * exp(0.06*T) [For non filter feeders]

Gd = Gv*vss*scav [For filter feeders]

kd = Ed*Gd/Wb

kG = kGconst * Wb^-0.2

Gf=Gd*((1-assimEff(1))*vld+ (1-assimEff(2))*vnd+ (1-assimEff(3))*vwd)

vlg= (1-assimEff(1))*vld/ ((1-assimEff(1))*vld+ (1-assimEff(2))*vnd+ (1-assimEff(3))*vwd)

vng= (1-assimEff(2))*vnd/ ((1-assimEff(1))*vld+ (1-assimEff(2))*vnd+ (1-assimEff(3))*vwd)

vwg= (1-assimEff(3))*vwd/ ((1-assimEff(1))*vld+ (1-assimEff(2))*vnd+ (1-assimEff(3))*vwd)

kgb=((vlg*Kow + vng*beta*Kow + vwg)/ (lipid*Kow + nlom*beta*Kow + wc))

ke = Gf*Ed*kgb/Wb

cprey = preyprop*cbiota

cbiota=(k1*(mo*phi*cwater + mp*cpw)+ kd*cprey) / (k2 + ke + kG + kM)

Appendix N. Individual molecules for inclusion in total organochlorine compounds.

Summation procedures for sediment and tissue data followed the procedures developed by the SQO Science Team.

Total DDTs

The following compounds comprised total DDTs for all analyses: o,p'-DDD, o,p'-DDE, o,p'-DDT, p,p'-DDD, p,p'-DDE, and p,p'-DDT

Total chlordanes

The following five chlordanes comprised total chlordanes for all analyses: alpha-Chlordane, gamma-Chlordane, Oxychlordane, trans-Chlordane, and trans-Nonachlor.

Total PCBs

Total PCBs were calculated by summing individual congeners. Individual congeners measured often vary among studies. In order to reduce inconsistencies and artifacts associated with compounds always found below detection limits, it was necessary to use two different groups of summed compounds for analyses in this project. This report does not conduct explicit comparisons between these groups of samples summed in different fashions.

The first group of congeners were included in total PCBs for analyses involving the laboratory bioaccumulation test organisms *Macoma nasuta*, *Neanthes virens* and *Nephtys caecoides* (including sediment vs. biota comparisons): PCB 008, PCB 018, PCB 028, PCB 044, PCB 052, PCB 066, PCB 101, PCB 105, PCB 118, PCB 128, PCB 138, PCB 153, PCB 180, PCB 187, and PCB 195. These congeners were selected to be consistent the summation procedure developed for the CASQO database, from which laboratory bioaccumulation test data were obtained.

The second group of congeners were included in total PCBs for the fish tissue and sediment chemistry lines of evidence in the Newport Bay case study: PCB 006, PCB 044, PCB 049, PCB 052, PCB 066, PCB 070, PCB 074, PCB 087, PCB 099, PCB 101, PCB 105, PCB 110, PCB 118, PCB 119, PCB 128, PCB 138, PCB 149, PCB 151, PCB 180, and PCB 187. These congeners were selected for the case study because they were present at detectable concentrations in Newport Bay sediments in at least 10% of samples and analyzed for at least 10 samples (see also Appendix Q, Table Q.1).

Total high molecular weight PAHs (HPAH)

The following compounds comprised total HPAHs: benz(a)anthracene, benzo(a)pyrene, benzo(e)pyrene, chrysene, dibenz(a,h)anthracene, fluoranthene, perylene, and pyrene. Note that in this report, total HPAHs are only evaluated in laboratory bioaccumulation test organism vs. sediment comparisons (Sections 4.5 and 5.5).

Appendix O. Fish data used for developing empirical bioaccumulation factors (BAFs). All data are from Allen *et al.* (2004).

Table O.1. Data used to evaluate human sport fish consumption in Newport Bay.

Sample ID	Species	Length	Receptor	Tissue preparation	Percent lipid	Total PCBs	Total chlordanes	Total DDTs	Total PCBs	Total chlordanes	Total DDTs
		mm			%	ng/g wet	ng/g wet	ng/g wet	ng/g lipid	ng/g lipid	ng/g lipid
BPNBOLS01-ORGSKOF-3	Black perch	182	Humans	Fillet skin off	0.63	5.0	5.0	42.2	794	794	6698
BPNBOLW00-01-ORGSKOF-1	Black perch	169	Humans	Fillet skin off	0.77	5.0	5.0	42.1	650	650	5472
CHNBOLS01-ORGSKOF-1	California halibut	183	Humans	Fillet skin off	0.58	5.0	5.0	71.2	860	860	12250
CHNBOLW00-01-ORGSKOF-1	California halibut	152	Humans	Fillet skin off	0.51	5.0	5.0	66.9	979	979	13105
CHNBOUW00-01-ORGSKOF-2	California halibut	153	Humans	Fillet skin off	0.61	5.0	5.0	66.4	826	826	10968
DTNBOLS01-ORGSKOF-1	Diamond turbot	187	Humans	Fillet skin off	0.63	5.0	5.0	24.8	788	788	3909
DTNBOLW00-01-ORGSKOF-1	Diamond turbot	175	Humans	Fillet skin off	0.56	5.0	5.0	43.6	895	895	7804
DTNBOLW00-01-ORGSKOF-2	Diamond turbot	207	Humans	Fillet skin off	0.63	5.0	5.0	29.2	799	799	4665
DTNBOUS01-ORGSKOF-1	Diamond turbot	177	Humans	Fillet skin off	0.47	5.0	5.0	34.2	1058	1058	7234
DTNBOUS01-ORGSKOF-2	Diamond turbot	195	Humans	Fillet skin off	0.68	5.0	5.0	42.5	740	740	6290
DTNBOUW00-01-ORGSKOF-1	Diamond turbot	158	Humans	Fillet skin off	0.40	5.0	5.0	29.8	1241	1241	7398
DTNBOUW00-01-ORGSKOF-2	Diamond turbot	193	Humans	Fillet skin off	0.86	5.0	5.0	65.7	584	584	7672
DTNBOUW00-01-ORGSKOF-3	Diamond turbot	175	Humans	Fillet skin off	0.41	5.0	5.0	22.3	1234	1234	5502
SSBNBOLS01-ORGSKOF-1	Spotted sand bass	244	Humans	Fillet skin off	0.66	5.0	5.0	45.3	759	759	6874
SSBNBOLS01-ORGSKOF-2	Spotted sand bass	284	Humans	Fillet skin off	1.07	10.4	5.4	92.2	964	502	8577
SSBNBOLS01-ORGSKOF-3	Spotted sand bass	277	Humans	Fillet skin off	0.75	24.2	5.3	108.2	3252	711	14515
SSBNBOLW00-01-ORGSKOF-1	Spotted sand bass	259	Humans	Fillet skin off	0.52	5.0	5.0	15.0	963	963	2889
SSBNBOUW00-01-ORGSKOF-1	Spotted sand bass	271	Humans	Fillet skin off	0.90	5.0	5.0	81.6	556	556	9075

Table O.2. Data used to evaluate wildlife predators in Newport Bay.

Sample ID	Species	Length	Receptor	Tissue preparation	Percent lipid	Total PCBs	Total chlordanes	Total DDTs	Total PCBs	Total Chlordanes	Total DDTs
		Mm			%	ng/g wet	ng/g wet	ng/g wet	ng/g lipid	ng/g lipid	ng/g lipid
AGNBILS02-ORGWHOL-1	Arrow goby	22	Wildlife	Whole	0.87	5.0	0.2	56.0	573	27	6415
AGNBIUS02-ORGWHOL-1	Arrow goby	29	Wildlife	Whole	1.74	5.0	7.9	262.0	287	453	15032
AGNBIUW02-ORGWHOL-1	Arrow goby	22	Wildlife	Whole	1.17	5.0	0.6	147.6	427	51	12594
AGNBOUS02-ORGWHOL-1	Arrow goby	25	Wildlife	Whole	1.48	5.0	1.4	146.2	339	95	9898
AGNBOUW02-ORGWHOL-1	Arrow goby	23	Wildlife	Whole	1.21	5.0	0.5	91.0	413	38	7508
BPNBOLS02-ORGWHOL-1	Black perch	80	Wildlife	Whole	1.14	5.0	12.6	116.6	440	1109	10264
CHNBOLW02-ORGWHOL-1	California halibut	96	Wildlife	Whole	1.06	5.0	12.2	97.0	471	1149	9134
KLNBOUS02-ORGWHOL-1	California killifish	66	Wildlife	Whole	0.92	5.0	0.8	83.6	546	86	9127
KLNBUS02-ORGWHOL-1	California killifish	67	Wildlife	Whole	0.99	5.0	2.4	101.6	505	242	10263
KNBILW02-ORGWHOL-1	California killifish	47	Wildlife	Whole	1.57	97.6	0.5	116.0	6217	31	7389
CGNBILS02-ORGWHOL-1	Cheekspot goby	25	Wildlife	Whole	1.54	53.4	11.0	195.0	3474	716	12687
DTNBILS02-ORGWHOL-1	Diamond turbot	77	Wildlife	Whole	0.84	5.0	6.4	119.4	598	766	14282
PSNB0LW02-ORGWHOL-1	Pacific staghorn sculpin	55	Wildlife	Whole	0.85	5.0	3.0	57.0	592	355	6746
PSNBILS02-ORGWHOL-1	Pacific staghorn sculpin	67	Wildlife	Whole	1.65	5.0	21.2	204.2	302	1283	12353
PSNBOLS02-ORGWHOL-1	Pacific staghorn sculpin	77	Wildlife	Whole	1.20	5.0	17.6	147.2	418	1472	12308
PSNBOUS02-ORGWHOL-1	Pacific staghorn sculpin	64	Wildlife	Whole	1.68	5.0	14.6	165.0	297	869	9816
SPNBOLS02-ORGWHOL-2	Shiner surfperch	46	Wildlife	Whole	1.02	5.0	1.3	95.2	489	127	9306
SPNBOUW00-01-ORGSKOF-1	Shiner surfperch	80	Wildlife	Whole	1.02	5.0	5.0	86.8	490	490	8512
TSNBILS02-ORGWHOL-1	Topsmelt	28	Wildlife	Whole	1.48	33.6	2.8	127.6	2267	189	8610
TSNBOLS02-ORGWHOL-1	Topsmelt	33	Wildlife	Whole	1.77	5.0	1.8	160.0	282	101	9019

TSNBUS02-ORGWHOL-1	Topsmelt	27	Wildlife	Whole	1.70	5.0	6.4	114.2	294	377	6726
TSNBILS02-ORGWHOL-2	Topsmelt	NA	Wildlife	Whole	1.47	101.2	1.1	95.0	6903	75	6480
TSNBILS02-ORGWHOL-3	Topsmelt	NA	Wildlife	Whole	1.41	10.0	4.0	50.0	709	283	3544

Appendix P. Letter from SARWQCB staff requesting specific management assumptions for Newport Bay case study (submitted electronically).

California Regional Water Quality Control Board
Santa Ana Region

Alan C. Lloyd, Ph.D.
Agency Secretary

3737 Main Street, Suite 500, Riverside, California 92501-3348
Phone (951) 782-4130 — FAX (951) 781-6288 — TTY (951) 782-3221
http://www.waterboards.ca.gov/santaana

Arnold Schwarzenegger
Governor

September 1, 2005

Mr. Ben Greenfield
San Francisco Estuary Institute
7770 Pardee Lane
Oakland, CA 94621

Re: Human Health Risk Parameters to Use in Newport Bay Model

Dear Ben:

The following values should be used in the Newport Bay modeling effort evaluating human health risk:

Risk Threshold:

We request that you evaluate three thresholds reflecting "no problem (green)," "caution (yellow)," and "problem likely (red)." These thresholds correspond to the $<10^{-6}$ risk, 10^{-5} to 10^{-6} risk, and the $>10^{-5}$ risk, respectively.

Consumption Rate:

We request that you use the consumption rates that were reported in the Santa Monica Bay Seafood Consumption Study (Allen, 1994).

Please give me a call at (951) 782-4468, or Kathy Rose at (951) 321-4585, if you have any questions.

Sincerely,

Wanda Mais-Smith
Chief, Coastal Waters Planning Section

Appendix Q. Summary statistics for water and sediment contaminants in Newport Bay.

Table Q.1. Summary statistics for individual sediment compounds monitored in Newport Bay. All sample values below quantification were set at the detection limits. Samples in grey were accepted for bioaccumulation factor calculation based on sufficient sample size and incidence of samples above detection. For data sources, see Appendix R.

Compound	Class	Geometric mean (ng/g dw)	Log SE	Arithmetic mean (ng/g dw)	Arithmetic SE (ng/g dw)	Median (ng/g dw)	Number samples (N)	Below detection (%)	N > 10	Below detection < 90%	Accepted
o,p'-DDD	DDT	0.18	0.10	0.51	0.15	0.07	45	80%	Yes	Yes	Yes
o,p'-DDE	DDT	0.27	0.12	1.25	0.42	0.46	45	56%	Yes	Yes	Yes
o,p'-DDT	DDT	0.10	0.10	0.27	0.07	0.04	45	84%	Yes	Yes	Yes
p,p'-DDD	DDT	1.85	0.16	6.69	0.96	6.97	45	27%	Yes	Yes	Yes
p,p'-DDE	DDT	20.54	0.06	29.02	3.35	26.46	45	0%	Yes	Yes	Yes
p,p'-DDT	DDT	0.46	0.15	3.27	0.86	0.06	45	58%	Yes	Yes	Yes
Dieldrin		0.03	0.00	0.03	0.00	0.03	16	100%	Yes	No	No
Mirex		0.08	0.00	0.08	0.00	0.08	3	100%	No	No	No
alpha-Chlordane	Chlordane	0.28	0.23	1.49	0.33	1.57	24	46%	Yes	Yes	Yes
cis-Nonachlor	Chlordane	0.05	0.00	0.05	0.00	0.05	10	100%	Yes	No	No
gamma-Chlordane	Chlordane	0.23	0.24	1.42	0.31	1.44	24	46%	Yes	Yes	Yes
trans-Nonachlor	Chlordane	0.13	0.22	0.77	0.40	0.05	13	77%	Yes	Yes	Yes
PCB006	PCB	1.35	0.09	1.83	0.63	1.00	11	82%	Yes	Yes	Yes
PCB018	PCB	0.05	0.16	0.33	0.08	0.01	34	100%	Yes	No	No
PCB028	PCB	0.16	0.12	0.59	0.17	0.05	34	91%	Yes	No	No
PCB031	PCB	1.37	0.57	3.40	1.65	5.00	3	33%	No	Yes	No
PCB033	PCB	0.31	0.40	0.75	0.62	0.13	3	67%	No	Yes	No
PCB037	PCB	0.23	0.11	0.75	0.27	0.08	34	94%	Yes	No	No
PCB044	PCB	0.21	0.14	1.27	0.54	0.05	34	82%	Yes	Yes	Yes
PCB049	PCB	0.38	0.12	1.53	0.54	0.09	34	79%	Yes	Yes	Yes
PCB052	PCB	0.24	0.15	1.45	0.51	0.04	34	79%	Yes	Yes	Yes
PCB066	PCB	0.15	0.20	2.75	1.46	0.05	23	78%	Yes	Yes	Yes
PCB070	PCB	0.26	0.15	1.78	0.73	0.05	34	79%	Yes	Yes	Yes
PCB074	PCB	0.22	0.13	1.15	0.47	0.05	34	85%	Yes	Yes	Yes
PCB077	PCB	0.16	0.09	0.35	0.08	0.07	34	97%	Yes	No	No
PCB081	PCB	0.02	0.21	0.33	0.08	0.00	34	100%	Yes	No	No
PCB087	PCB	0.13	0.15	0.98	0.45	0.03	34	88%	Yes	Yes	Yes
PCB095	PCB	1.29	0.86	6.74	4.08	6.10	3	33%	No	Yes	No
PCB097	PCB	1.83	0.69	6.63	4.22	5.30	3	33%	No	Yes	No
PCB099	PCB	0.39	0.13	1.76	0.60	0.09	34	79%	Yes	Yes	Yes
PCB101	PCB	0.31	0.15	2.16	0.83	0.06	34	68%	Yes	Yes	Yes
PCB105	PCB	0.21	0.15	1.16	0.43	0.04	34	82%	Yes	Yes	Yes
PCB110	PCB	0.36	0.19	3.23	1.10	0.33	34	53%	Yes	Yes	Yes

PCB114	PCB	0.02	0.21	0.33	0.08	0.00	34	100%	Yes	No	No
PCB118	PCB	0.26	0.16	2.01	0.78	0.04	34	71%	Yes	Yes	Yes
PCB119	PCB	0.12	0.19	1.21	0.32	0.02	34	68%	Yes	Yes	Yes
PCB123	PCB	0.02	0.22	0.42	0.14	0.00	34	97%	Yes	No	No
PCB126	PCB	0.12	0.12	0.38	0.08	0.04	34	97%	Yes	No	No
PCB128	PCB	0.12	0.13	0.47	0.10	0.03	34	88%	Yes	Yes	Yes
PCB138	PCB	0.24	0.16	2.09	0.79	0.04	34	65%	Yes	Yes	Yes
PCB141	PCB	0.07	0.84	1.07	1.06	0.01	3	67%	No	Yes	No
PCB149	PCB	0.33	0.11	1.21	0.40	0.11	34	85%	Yes	Yes	Yes
PCB151	PCB	0.09	0.15	0.46	0.10	0.02	34	88%	Yes	Yes	Yes
PCB153	PCB	8.88	0.23	11.07	4.01	14.30	3	0%	No	Yes	No
PCB153/168	PCB	0.48	0.09	1.13	0.38	0.22	31	71%	Yes	Yes	Yes
PCB156	PCB	0.02	0.21	0.33	0.08	0.00	34	100%	Yes	No	No
PCB157	PCB	0.02	0.23	0.60	0.26	0.00	34	91%	Yes	No	No
PCB158	PCB	0.21	0.10	0.48	0.11	0.08	34	91%	Yes	No	No
PCB167	PCB	0.03	0.21	0.40	0.09	0.00	34	94%	Yes	No	No
PCB168/132	PCB	1.03	0.31	1.80	1.30	0.50	3	67%	No	Yes	No
PCB169	PCB	0.03	0.24	1.05	0.56	0.00	34	94%	Yes	No	No
PCB170	PCB	0.03	0.20	0.45	0.13	0.01	34	91%	Yes	No	No
PCB177	PCB	0.23	0.09	0.46	0.09	0.10	34	91%	Yes	No	No
PCB180	PCB	0.15	0.13	0.74	0.27	0.05	34	85%	Yes	Yes	Yes
PCB183	PCB	0.09	0.13	0.36	0.08	0.03	34	94%	Yes	No	No
PCB187	PCB	0.20	0.11	0.65	0.21	0.07	34	88%	Yes	Yes	Yes
PCB189	PCB	0.02	0.21	0.33	0.08	0.00	34	100%	Yes	No	No
PCB194	PCB	0.13	0.12	0.42	0.10	0.05	34	94%	Yes	No	No
PCB200	PCB	1.25	0.00	1.25	0.00	1.25	23	100%	Yes	No	No
PCB201	PCB	0.03	0.20	0.43	0.11	0.01	34	91%	Yes	No	No
PCB206	PCB	0.12	0.11	0.37	0.09	0.04	34	97%	Yes	No	No

Table Q.2. Summary statistics for individual water compounds monitored in Newport Bay. Results are for total constituent (i.e., dissolved + particulate). All sample values below detection were set at ½ detection limits. ND/N = proportion of samples below detection/total sample size (N = 3 in all cases). All data were collected by SCCWRP scientific staff in 2005 (Appendix R).

Compound	ND/N	Arithmetic mean (µg/L)	Arithmetic SE (µg/L)	Geometric mean (µg/L)	Log SE
alpha-Chlordane	0/3	4.56E-05	4.05E-06	4.53E-05	0.040
cis-Nonachlor	1/3	1.59E-05	3.05E-06	1.52E-05	0.093
Dieldrin	3/3	1.00E-05	0.00E+00	1.00E-05	0.000
gamma-Chlordane	0/3	3.41E-05	3.31E-06	3.38E-05	0.041
Oxychlordane	3/3	1.00E-05	0.00E+00	1.00E-05	0.000
trans-Nonachlor	0/3	3.11E-05	4.07E-06	3.05E-05	0.060
o p-DDD	0/3	3.47E-05	4.30E-06	3.41E-05	0.058
o,p'-DDE	0/3	6.00E-05	6.81E-06	5.93E-05	0.047
o,p'-DDT	0/3	1.98E-04	2.73E-05	1.94E-04	0.060
p,p'-DDD	0/3	1.71E-04	2.44E-05	1.67E-04	0.067
p,p'-DDE	0/3	3.63E-04	4.57E-05	3.58E-04	0.052
p,p'-DDT	0/3	4.96E-05	2.30E-05	4.06E-05	0.187
PCB18	1/3	1.51E-05	3.50E-06	1.43E-05	0.099
PCB28	0/3	3.09E-05	5.93E-06	2.96E-05	0.094
PCB37	0/3	2.54E-05	6.49E-06	2.37E-05	0.117
PCB44	0/3	1.99E-05	4.28E-06	1.90E-05	0.089
PCB49	1/3	1.59E-05	2.95E-06	1.53E-05	0.092
PCB52	0/3	2.59E-05	5.27E-06	2.48E-05	0.092
PCB66	1/3	2.03E-05	5.73E-06	1.85E-05	0.140
PCB70	1/3	2.14E-05	6.28E-06	1.93E-05	0.149
PCB74	1/3	1.68E-05	3.43E-06	1.60E-05	0.102
PCB77	1/3	2.00E-05	5.47E-06	1.82E-05	0.136
PCB81	1/3	1.62E-05	3.70E-06	1.53E-05	0.103
PCB87	1/3	1.98E-05	5.10E-06	1.82E-05	0.133
PCB99	1/3	1.89E-05	4.91E-06	1.74E-05	0.127
PCB101	1/3	2.40E-05	8.64E-06	2.07E-05	0.174
PCB105	1/3	1.85E-05	5.38E-06	1.70E-05	0.131
PCB110	0/3	2.74E-05	7.78E-06	2.51E-05	0.132
PCB114	3/3	1.00E-05	0.00E+00	1.00E-05	0.000
PCB118	1/3	1.52E-05	2.64E-06	1.47E-05	0.084
PCB119	1/3	1.49E-05	3.09E-06	1.43E-05	0.091
PCB123	1/3	2.26E-05	8.75E-06	1.94E-05	0.172
PCB 126	1/3	1.35E-05	1.80E-06	1.32E-05	0.062
PCB128	2/3	1.51E-05	5.10E-06	1.36E-05	0.134
PCB138	1/3	1.83E-05	4.14E-06	1.71E-05	0.117
PCB149	1/3	1.77E-05	4.01E-06	1.67E-05	0.113
PCB 151	1/3	1.31E-05	1.64E-06	1.28E-05	0.057
PCB153/168	1/3	1.50E-05	2.63E-06	1.45E-05	0.083
PCB156	1/3	1.47E-05	4.72E-06	1.34E-05	0.128
PCB157	1/3	1.48E-05	2.73E-06	1.42E-05	0.084
PCB158	1/3	1.60E-05	3.00E-06	1.53E-05	0.093
PCB167	3/3	1.00E-05	0.00E+00	1.00E-05	0.000

PCB169	1/3	2.12E-05	9.59E-06	1.75E-05	0.184
PCB170	1/3	2.18E-05	6.19E-06	1.96E-05	0.149
PCB177	2/3	1.39E-05	3.93E-06	1.30E-05	0.113
PCB180	0/3	2.41E-05	6.51E-06	2.22E-05	0.131
PCB183	2/3	1.30E-05	3.02E-06	1.24E-05	0.093
PCB187	2/3	1.31E-05	3.13E-06	1.25E-05	0.096
PCB189	3/3	1.00E-05	0.00E+00	1.00E-05	0.000
PCB194	2/3	1.34E-05	3.40E-06	1.26E-05	0.102
PCB200	2/3	1.70E-05	6.98E-06	1.46E-05	0.163
PCB201	2/3	1.68E-05	6.76E-06	1.45E-05	0.160
PCB206	3/3	1.00E-05	0.00E+00	1.00E-05	0.000

Table Q.3. Total PCBs, total DDTs, and total chlordanes in Newport Bay Sediments sampled since 1997. All values are ng/g dry weight. RL = study reporting limit for compound. NA = reporting limit not available. Values listed as 0.0 are below reporting limits.

Study ref.	Station ID	Latitude	Longitude	Sample ID	Total DDT	DDT RL	Total chlordanes	Chlordane RL	Total PCBs	PCB RL
a	1	33.649	-117.883	1	159.0	2.0	55.0	NA		
a	2	33.648	-117.882	2	13.0	2.0	14.0	NA		
a	3	33.647	-117.880	3	38.0	2.0	19.0	NA		
a	4	33.648	-117.879	4	63.0	2.0	14.0	NA		
a	5	33.631	-117.889	5	48.0	2.0	15.0	NA		
a	6	33.632	-117.888	6	49.0	2.0	19.0	NA		
a	7	33.633	-117.889	7	37.0	2.0	0.0	NA		
a	8	33.632	-117.888	8	59.0	2.0	19.0	NA		
a	9	33.647	-117.883	9	46.0	2.0	54.0	NA		
a	10	33.632	-117.886	10	59.0	2.0	19.0	NA		
a	11	33.630	-117.891	11	43.0	2.0	20.0	NA		
b	NB1	33.596	-117.880	Sep-00	3.8	1.0			0.0	1.0
b	NB1	33.596	-117.880	May-01	2.5	1.0			0.0	1.0
b	NB2	33.608	-117.904	Sep-00	25.7	1.0			0.0	1.0
b	NB2	33.608	-117.904	May-01	26.3	1.0			0.0	1.0
b	NB3	33.612	-117.928	Sep-00	15.1	1.0			30.9	1.0
b	NB3	33.612	-117.928	May-01	9.3	1.0			23.7	1.0
b	NB4	33.619	-117.927	Sep-00	26.1	1.0			3.1	1.0
b	NB4	33.619	-117.927	May-01	56.0	1.0			0.0	1.0
b	NB5	33.614	-117.914	Sep-00	23.9	1.0			0.0	1.0
b	NB5	33.614	-117.914	May-01	16.1	1.0			0.0	1.0
b	NB6	33.620	-117.894	Sep-00	11.4	1.0			0.0	1.0
b	NB6	33.620	-117.894	May-01	12.7	1.0			0.0	1.0
b	NB7	33.647	-117.887	Sep-00	2.8	1.0			0.0	1.0
b	NB8	33.646	-117.887	Sep-00	25.4	1.0			0.0	1.0
b	NB9	33.638	-117.887	Sep-00	3.7	1.0			0.0	1.0
b	NB10	33.650	-117.871	Sep-00	17.0	1.0			0.0	1.0

b	NB7	33.647	-117.887	May-01	8.8	1.0			0.0	1.0
b	NB8	33.646	-117.887	May-01	12.9	1.0			0.0	1.0
b	NB9	33.638	-117.887	May-01	20.8	1.0			0.0	1.0
b	NB10	33.650	-117.871	May-01	19.7	1.0			0.0	1.0
c	RC1	33.615	-117.926	1	28.0	1.0	0.0	1.0	46.1	1.0
c	RC2	33.615	-117.927	2	54.5	1.0	0.0	1.0	174.1	1.0
c	RC3	33.614	-117.927	3	44.1	1.0	0.0	1.0	157.1	1.0
c	RC4	33.614	-117.927	4	51.1	1.0	0.0	1.0	194.1	1.0
c	RC5	33.613	-117.927	5	45.4	1.0	0.0	1.0	101.6	1.0
c	RC6	33.613	-117.928	6	43.1	1.0	0.0	1.0	103.1	1.0
c	RC7	33.613	-117.928	7	75.7	1.0	0.0	1.0	88.4	1.0
c	RC8	33.612	-117.928	8	59.0	1.0	0.0	1.0	116.2	1.0
c	RC9	33.612	-117.927	9	57.9	1.0	0.0	1.0	106.5	1.0
c	RC10	33.611	-117.927	10	47.7	1.0	0.0	1.0	68.0	1.0
c	RC11	33.611	-117.927	11	56.8	1.0	0.0	1.0	95.9	1.0
c	RC12	33.611	-117.927	12	44.7	1.0	0.0	1.0	92.0	1.0
c	RC13	33.610	-117.927	13	49.5	1.0	0.0	1.0	193.5	1.0
c	RC14	33.611	-117.926	14	95.9	1.0	0.0	1.0	125.1	1.0
c	RC15	33.610	-117.926	15	40.3	1.0	0.0	1.0	15.4	1.0
d	2136	33.619	-117.927	2136	85.1	0.2	11.5	0.1	47.5	0.2
d	2137	33.613	-117.924	2137	53.9	0.2	3.7	0.1	33.8	0.2
d	2138	33.614	-117.914	2138	92.4	0.2	5.5	0.1	13.1	0.2
d	2141	33.611	-117.902	2141	63.6	0.2	4.0	0.1	9.7	0.2
d	2142	33.607	-117.911	2142	85.3	0.2	5.3	0.1	12.1	0.2
d	2143	33.607	-117.906	2143	75.0	0.2	4.4	0.1	11.0	0.2
d	2144	33.607	-117.900	2144	117.9	0.2	6.3	0.1	12.8	0.2
d	2145	33.604	-117.889	2145	75.8	0.2	4.5	0.1	10.1	0.2
d	2146	33.603	-117.888	2146	52.4	0.2	3.2	0.1	10.3	0.2
d	2147	33.601	-117.893	2147	53.9	0.2	3.5	0.1	10.2	0.2
d	2148	33.595	-117.880	2148	12.2	0.2	0.8	0.1	8.6	0.2

a. Masters and Inman (2000) b. Bay *et al.* (2004) c. Bay and Brown (2003) d. Noblet *et al.* (2002)

Appendix R. General description of data sources used for Newport Bay case study.

Sediment contaminant concentrations – Data from the CASQO database were combined with data from Masters and Inman (2000). The CASQO database included data from the Rhine Channel Study (Bay and Brown 2003), the Newport Bay Sediment Toxicity Study (Bay *et al.* 2004), and the Southern California Bight 1998 Survey (Noblet *et al.* 2002). Although data were available from the Bay Protection and Toxics Cleanup Program (BPTCP) (Hunt *et al.* 1998), these data were collected in 1994 and do not represent current conditions. Therefore, BPTCP data were not included. Evaluation of these source data sets indicated that a substantial proportion of the measured values for many PCBs and pesticides were below quantification limits (BQL), necessitating decision-making regarding the imputed values when results were below quantification limits (QL). To evaluate the best model fit, BQL values were imputed using both values at ½ the QL and at the detection limit (DL). These imputation methods resulted in substantially different results for geometric means. Both geometric means were evaluated to determine which exhibited better model fits.

For comparison of model calculated biota contaminant predictions vs. empirical results, only certain contaminants had sufficient source data for sediment contamination. Rejection of data resulted when there were not at least 10 occurrences of sediment contaminant measurements or when more than 90% of results were BQL.

Water contaminant concentrations In 2005, SCCWRP collected water column contaminant data for this study using an in-situ pump. Study methods were similar to those in Bay *et al.* (2004). Samples were collected from three locations: Sheriff Station, Nautical Museum, and Carr's Deck. A fourth station (Lido Island) was rejected because water flow volume could not be estimated accurately. Following Gobas and Arnot (2005), particulate and dissolved phase results were combined to determine total concentrations.

Sediment organic carbon – Estimates of sediment organic carbon were based on data from the CASQO database (see **Sediment contaminant concentrations**, above), Masters and Inman (2000), and BPTCP data (Hunt *et al.* 1998).

Water total suspended solids (TSS), particulate organic carbon (POC), and dissolved organic carbon (DOC) – TSS, POC, and DOC were provided by Martha Sutula (SCCWRP, *unpublished data*). Data were collected at three Newport Bay sites, at two depths for each site (surface and bottom). Results represent the average across these sites, and include six sample collection dates in 2004 (1/15, 2/10, 3/23, 6/8, 9/15, and 11/11).

Water dissolved oxygen, temperature, and salinity – Temperature and salinity data were assembled from Table 5 of Kennison *et al.* (2003). Dissolved oxygen data was combined from this source and from Figure 4.1, Figure 4.2, and Table 4.1 of Kamer and Stein (2003).

Contaminant octanol-water partitioning coefficients (K_{OW}) – Contaminant K_{OW}'s are presented in Appendix S. K_{OW}'s for PCBs were obtained from Gobas and Arnot (2005). For those PCBs not evaluated in Gobas and Arnot, K_{OW}'s were the median of results combined from five published sources: Li *et al.* (2003), Mackay *et al.* (2000), Beyer *et al.* (2002), Hansen *et al.* (1999), and Hawker and Connell (1988). K_{OW}'s for pesticides were taken from Shen and Wania (2005), or Leatherbarrow *et al.* (2006), which compiled K_{OW}'s from Mackay *et al.* (2000).

K_{OW}'s were temperature and salinity corrected to be appropriate for the water body average temperature and salinity. Methods followed Gobas and Arnot (2005), and references cited therein. Specifically, the Kow values were corrected for temperatures using the following equation (Li *et al.* 2003):

$$\log K_{ow}E_T = \log K_{ow}D_T - \frac{\Delta U_{OW}}{\ln(10)\cdot R}\cdot\left(\frac{1}{E_T}-\frac{1}{D_T}\right)$$

where E_T is the environmental temperature (Kelvin) and D_T is the data collection temperature (Kelvin), ΔUow is the internal energy of octanol-water phase transfer and R is the gas law constant (0.0083145 kJ/mol K). Empirically-derived ΔUow are currently unavailable for pesticides, and were therefore estimated to be -25 kJ/mol (Shen and Wania 2005).

Salinity corrections followed Xie *et al.* (1997):

$$Kow = Kow \times 10^{(SPC\cdot Vh\cdot MCS\cdot SAL/35)}$$

where SPC is the Setschenow proportionality constant (0.0018 L/cm^3), Vh is the LeBas molar volume (cm^3/mol) of the chemical (calculated following Tucker and Nelken 1982), MCS is the molar concentration of seawater at 35 practical salinity units (0.5) and SAL is the salinity for the system of interest (psu).

Attached algae - According to dietary studies, a significant proportion of prey for some Newport Bay animal species are benthic attached algae (e.g., Logothetis *et al.* 2001). These attached algae are important primary producers in the system, growing densely in intertidal areas, particularly in the upper Bay (Kamer *et al.* 2004a). They include *Ulva expansa, Ulva lactusa, Ulva foliosa, Enteromorpha intestinalis*, as well as other *Ulva* spp. and *Enteromorpha* spp. (Krista Kamer, MLML, *Pers. comm.*). The plant-water two compartment model developed for uptake/elimination kinetics and described by Arnot and Gobas (2004), and summarized in Appendix G, was parameterized for phytoplankton. For the Newport Bay case study, the same model is applied to attached benthic algae. Although the specific parameters have not been calibrated for attached algae, the general model assumptions have been supported by laboratory studies (e.g., Gobas *et al.* 1991).

Algae growth rate, lipid content, porewater exposure - Tissue lipid content and non-lipid organic matter have not been measured for Newport Bay benthic algae or

phytoplankton. For phytoplankton, parameters were taken from Gobas and Arnot (2005). For benthic attached algae, these parameters were estimated by reviewing literature values for *Ulva* spp. and *Enteromorpha* spp. (Appendix V). Growth rates were based on *Enteromorpha intestinalis* laboratory mesocosm studies from Newport Bay (Kamer *et al.* 2004a, Kamer *et al.* 2004b) and a study of similar salinity conditions (Martins *et al.* 1999). These studies exhibited an order of magnitude difference in daily growth rates, warranting a wide range in the probabilistic estimation of growth rate, including the use of a lognormal distribution for probabilistic simulations. Combining results from both sets of studies, the geometric mean growth rate was 4.7% / d, with a standard deviation (log scale) of 0.25 (Table V.2). When plants are submerged (i.e., during high tides), tissue buoyancy causes the plants to float and have no porewater exposure (K. Kamer, *Pers. comm.*). Thus, the nominal value for porewater transpiration exposure (i.e., Mp in Appendix M) was set at zero.

Respiratory exposure to porewater – The proportion of respiratory exposure to porewater was varied between 0% and 10%. Values for benthic invertebrates were obtained from Gobas and Arnot (2005). Values for local fish were based on life history information. Arrow goby, which has a strong benthic association due to dwelling within sediment burrows (M. James Allen, SCCWRP, *Pers. comm.*), was assigned 10%. Other fish with close benthic associations were assigned 5% exposure. Fish with a weaker benthic association were assigned 0%.

Invertebrate diet, body mass, and lipid content – A search for published literature on mass or lipid content in local invertebrates was largely unsuccessful. Invertebrate species selection, body mass estimates, lipid content and dietary proportions were based on local expert guidance and unpublished data (Don Cadien, LACSD, *Pers. comm.*), and on Gobas and Arnot (2005).

Fish body mass and lipid content – Fish tissue lipid content was taken from Allen *et al.* (2004), and confirmed using published literature sources as well as data from the Regional Monitoring Program for Water Quality in San Francisco Bay (Appendix U). The method for extrapolating lipid content between whole body and muscle tissue is presented in Appendix E. Body mass was combined from a variety of published and unpublished sources (Appendix U), and averaged to represent a typical organism from Newport Bay. Body mass is a relatively insensitive parameter (Gobas and Arnot 2005).

Fish diets - Fish dietary proportions were determined from gut content analysis presented in published journal articles and grey literature. The model calculates uptake using a mass-balance approach; therefore, model input data were generated based on relative mass or volume of prey consumed, rather than number captured or frequency of occurrence. For most species, these included data from Newport Bay, as referenced in Appendix T. For staghorn sculpin and California corbina, no dietary data were found specific to Newport Bay, so estimated diets were based on published studies from other sites (Appendix T).

Contaminant dietary assimilation efficiencies – All assimilation efficiencies followed Gobas and Arnot (2005). Assimilation efficiencies for invertebrates were 72% for lipids, 75% for NLOM, and 55% for water. Assimilation efficiencies for fish were 90% for lipids, 50% for NLOM, and 55% for water.

Animal growth rate equation constants (kG) – Following Gobas and Arnot (2005), growth rate equation constants were set at 3.5×10^{-4} for invertebrates and 7×10^{-4} for fishes.

Appendix S. Octanol-water partitioning coefficients (K_{ow}) used in model simulations for Newport and San Francisco Bays.

Note: TS = corrected for local temperature and salinity for Newport Bay or San Francisco Bay.

Compound	LeBas molar vol. (cm^3/mol)	Log K_{ow}		Log K_{ow} T Newport	Log K_{ow} TS Newport	Log K_{ow} T SF	Log K_{ow} TS SF
Pesticides							
cis-Nonachlor	361.4	5.70	a	5.81	6.06	5.85	6.04
Dieldrin	332.2	5.48	b	5.59	5.82	5.63	5.80
gamma-Chlordane	340.5	6.27	b	6.38	6.62	6.42	6.60
HCB	221.4	5.52	b	5.63	5.79	5.67	5.81
Mirex	420.2	7.00	c	7.11	7.40	7.15	7.37
op-DDD	312.6	5.34	a	5.45	5.67	5.49	5.64
op-DDE	305.2	5.63	a	5.74	5.95	5.78	5.93
op-DDT	333.5	5.70	a	5.81	6.04	5.85	6.01
pp-DDD	312.6	6.33	b	6.44	6.66	6.48	6.63
pp-DDE	305.2	6.93	b	7.04	7.25	7.08	7.23
pp-DDT	333.5	6.39	b	6.50	6.73	6.54	6.70
trans-Nonachlor	361.4	5.70	a	5.81	6.06	5.85	6.04
PCB Congeners							
PCB006	226.4	5.04	e	5.13	5.29		
PCB018	247.4	5.32	d	5.28	5.45		
PCB028	247.4	5.74	d	5.71	5.88		
PCB037	247.4	5.80	e	5.92	6.09		
PCB044	268.4	5.84	d	5.80	5.99		
PCB049	268.4	5.97	d	5.93	6.12		
PCB052	268.4	6.00	d	5.96	6.15		
PCB066	268.4	6.02	d	5.98	6.17		
PCB070	268.4	6.12	d	6.08	6.27		
PCB074	268.4	6.13	d	6.09	6.27		
PCB077	268.4	6.33	e	6.47	6.65		
PCB081	268.4	6.25	e	6.38	6.56		
PCB087	289.4	6.37	d	6.33	6.53		
PCB099	289.4	6.38	d	6.34	6.54		
PCB101	289.4	6.41	d	6.37	6.57		
PCB105	289.4	6.91	d	6.87	7.07		
PCB110	289.4	6.34	d	6.30	6.50		
PCB118	289.4	6.78	d	6.74	6.94		
PCB119	289.4	6.46	e	6.58	6.78		
PCB123	310.4	6.60	e	6.73	6.93		
PCB126	310.4	6.75	e	6.88	7.08		
PCB128	310.4	6.82	d	6.78	6.99		
PCB138	310.4	7.30	d	7.26	7.47		
PCB149	310.4	6.65	d	6.61	6.82		
PCB151	310.4	6.63	d	6.58	6.80		
PCB156	310.4	7.04	d	7.00	7.21		
PCB157	310.4	7.01	e	7.15	7.36		
PCB158	310.4	6.90	d	6.86	7.07		
PCB169	310.4	7.30	e	7.43	7.65		

PCB170	331.4	7.21	d	7.17	7.40
PCB177	331.4	7.04	d	6.99	7.22
PCB180	331.4	7.25	d	7.21	7.44
PCB183	331.4	7.15	d	7.10	7.33
PCB187	331.4	7.12	d	7.08	7.31
PCB194	352.4	7.85	d	7.81	8.05
PCB200	352.4	7.18	e	7.32	7.56
PCB201	352.4	7.54	d	7.49	7.74

a. Mackay *et al.* (2000); Leatherbarrow *et al.* (2006). b. Shen and Wania (2005). c. Fisk *et al.* (1999). d. Gobas and Arnot (2005). Note that K_{ow}'s here are calculated at 15° C. e. See K_{ow} section of Appendix R.

Appendix T. Summary of published information and grey literature on diets of selected Newport Bay fish species.

Results include all available studies from within Newport Bay, in addition to appropriate literature off the Southern California coast. All results indicate % prey mass in diet, except where ** indicates % frequency of occurrence. All studies were based on gut-content analyses. Grey results indicate averages and ranges for species having at least two studies with percent mass data.

			Prey type (% mass)											
Species	Sample size	Length (mm)	Sediment	Phytoplankton	Zooplankton	Crustaceans	Annelids	Mollusk	Benthic algae	Hydrozoa	Echinoderm	Whole fish	Fish eggs	Reference
California halibut	5	NA				0		0				100		a
California halibut	429	159-1055				1		10				89		b
Juv. California halibut	20	136				21		1				78		c
Juv. California halibut	21	154				0		0				100		c
Juv. California halibut	52	200-299				17		0				83		b
California halibut	Average					7.8		2.2				90.0		
California halibut	Range					0 - 21		0 - 10				78 - 100		
Yellowfin croaker	20	157	6			25	45	10		12		2		c
Yellowfin croaker	5	NA	0			5	26	60		0		9		
Yellowfin croaker	Average		3.0			15.0	35.5	35.0		6.0		5.5		
Yellowfin croaker	Range		0 - 6			5 - 25	26 - 45	10 - 60		0 - 12		2 - 9		
Topsmelt	30	96	50	0	0	0	0	0	50					c
Topsmelt	20	124	40	0	0	45	0	0	15					c
Topsmelt	19	<50	0	0	20	0	2	0	78					a
Topsmelt	57	>50	0	1	0	1	0	1	97					a
Topsmelt	Average		22.5	0.3	5.0	11.5	0.5	0.3	60.0					
Topsmelt	Range		0 - 50	0 - 1	0 - 20	0 - 45	0 - 2	0 - 1	15 - 97					
Striped mullet	11	166	65		0	10	10	5	10					c
Striped mullet	14	< 100	0		1	0	0	0	99					a
Striped mullet	Average		32.5		0.5	5.0	5.0	2.5	54.5					
Striped mullet	Range		0 - 65		0 - 1	0 - 10	0 - 10	0 - 5	10 - 99					
Arrow goby	58	10 - 45			37	9	54							a

California killifish	64	10 - 70		8	20	24	48					a
California killifish**	NA	NA	0	0	100	0	0	0		0		d
Shiner surfperch	25	NA (age 0)	0	86	13	1	0				0	a
Shiner surfperch	138	NA	32	55	3	2	6				2	e
Shiner surfperch	15	56-104	0	43	25	32	0				0	f
Shiner surfperch	Average		10.7	61.3	13.7	11.7	2.0				0.7	
Shiner surfperch	Range		0 - 32	43 - 86	3 - 25	1 - 32	0 - 6				0 - 2	
Staghorn sculpin**	NA	NA			100	0	0			0		g
Spotted sand bass	3	NA			0	51	47	0	0	2	0	a
Spotted sand bass	443	240			41	6	43	0	0	10	0	h
Spotted sand bass	92	250			28	0	17	2	13	38	2	i
Spotted sand bass	Average				23.0	19.0	35.7	0.7	4.3	16.7	0.7	
Spotted sand bass	Range				0 - 41	0 - 51	17 - 47	0 - 2	0 - 13	2 - 38	0 - 2	
California corbina	500	32-505			20	20	60			0		j
California corbina	NA	NA			50	0	0			50		k
California corbina	NA	NA			0	53	14			33		l
California corbina	Average				23.3	24.3	24.7			27.7		
California corbina	Range				0 - 50	0 - 53	0 - 60			0 - 50		

** Percent frequency of occurrence.

a. Allen (1980). b. Wertz and Domeier (1997). c. Marine Biological Consultants Inc. and SCCWRP (1980). d. Fritz (1975). e. Odenweller (1975). f. Allen (1983). g. Tasto (1975). h. Allen *et al.* (1995). i. Mendoza-Carranza and Rosales-Casian (2000). j. O'Brien and Valle (2000). k. Cruz-Escalona *et al* (2000). l. Bocanegra-Castillo *et al.* (2000).

Appendix U. Summary of published information and grey literature on body mass, tissue percent lipid, and tissue percent water content for Newport Bay fish species.

For evaluation of percent lipid, tissue type was reported as WB = whole body, or F = muscle fillet tissue. Grey results indicate averages and ranges for species having at least two studies with appropriate data.

Species	Length (mm)	Mass (kg)	Tissue type	Lipid (%)	Water (%)	Reference
Arrow goby	16 - 36		WB	1.3		a
Arrow goby	23	0.0002				b
Arrow goby	10 - 45	0.0002				c
Arrow goby		0.001				d
Arrow goby		0.001		1.3		
Arrow goby		0.0002 - 0.001		1.3		
California corbina	312	0.478	F	3.0	76.3	e
California corbina	288 - 386	0.668	F	4.6		a
California corbina		0.573		3.8		
California corbina		0.478 – 0.668		3.0 – 4.6		
California halibut	354	0.486	F	0.3	77.3	e
California halibut	550 - 980		F, Skin off	0.4		f
California halibut	374	1.058	F	0.4		a
California halibut	138 - 422		F	0.5		a
California halibut	89 - 144		WB	0.9		a
California halibut		0.003				c
California halibut	63	0.033				b
California halibut	74	0.050				g
California halibut	152	0.064				a
California halibut		4.500				d
California halibut		0.885		0.5		
California halibut		0.003 - 4.5		0.3 – 0.9		
California killifish	38 - 76		WB	1.5		a
California killifish	57	0.003	WB	1.7	78.0	e
California killifish	10 - 70	0.001				c
California killifish	64	0.013				b
California killifish		0.006		1.6		
California killifish		0.001 - 0.013		1.5 - 1.7		
Shiner surfperch	116	0.036	F	0.3	79.7	e
Shiner surfperch	80	0.015	WB	0.5		a
Shiner surfperch	73 - 83		WB	0.5		a
Shiner surfperch	45 - 60		WB	1.0		a
Shiner surfperch	58	0.003	WB	1.6	80.1	e

Shiner surfperch	90-150		WB	2.6		f
Shiner surfperch	NA (age 0)	0.003				c
Shiner surfperch	56	0.007				b
Shiner surfperch		0.013		1.1		
Shiner surfperch		0.003 - 0.015		0.3 – 2.6		
Spotted sand bass	233-300		F	0.8		a
Spotted sand bass	259	0.456	F	0.9		a
Spotted sand bass	284	0.742	F	1.1		a
Spotted sand bass	325	0.561	F	1.2	77.1	e
Spotted sand bass		0.586		1.0		
Spotted sand bass		0.456 - 0.742		0.8 - 1.2		
Staghorn sculpin	239	0.027	F	0.3	81.0	e
Staghorn sculpin	45-95		WB	1.3		a
Staghorn sculpin	75	0.006	WB	1.5	81.1	e
Staghorn sculpin	90 – 150		WB	1.6		h
Staghorn sculpin		0.002				c
Staghorn sculpin		0.012		1.2		
Staghorn sculpin		0.002 – 0.027		0.3 – 1.6		
Striped mullet			WB	0.5		i
Striped mullet	250	0.344	F	1.2		j
Striped mullet	86	0.008	WB	3.5	75.6	e
Striped mullet	393	1.126	F	4.4	74.2	e
Striped mullet	550	1.735	F	4.5		k
Striped mullet	NA (age 0)	0.009				c
Striped mullet	478	2.450				b
Striped mullet		0.945		2.8		
Striped mullet		0.008 - 2.45		0.5 - 4.5		
Topsmelt	18-44		WB	1.6		a
Topsmelt	NA (age 0)	0.002				c
Topsmelt		0.033				b
Topsmelt		0.017		1.6		
Topsmelt		0.002 - 0.033		1.6		
Yellowfin croaker	272	0.351	F	1.5	76.4	e
Yellowfin croaker	212 - 306			1.7		a
Yellowfin croaker		0.044				c
Yellowfin croaker	225	0.340				b
Yellowfin croaker	274	0.430				a
Yellowfin croaker		0.291		1.6		
Yellowfin croaker		0.044 - 0.43		1.5 - 1.7		

a. Allen *et al.* (2004). b. Marine Biological Consultants Inc. (1997). c. Allen (1980). d. von Stackelberg *et al.* (2003). e. Rasmussen (1995). f. Davis *et al.* (1999); Greenfield *et al.* (2003). g. Allen (1990). h. Jay A. Davis, unpublished data. i. Maruya and Lee (1998). j. Kannan *et al.* (1998). k. Sakurai *et al.* (2000)

Appendix V. Literature review and results summary on model input parameters for Newport Bay attached algae.

Table V.1. Parameter values from individual studies. NLOM = non lipid organic matter.

Species	Sample size	Growth rate (d^{-1})	Porewater exposure	Lipid (%)	Water (%)	NLOM (%)	Reference
Enteromorpha intestinalis	30	0.0164					Kamer *et al.* (2004a, 2004b)
Enteromorpha intestinalis		0.1369					Martins *et al.* (1999)
Ulva fenestrate	20	0.0500					Bjornsater and Wheeler (1990)
Ulva pertusa	2			2.01%			Vaskovsky *et al.* (1996)
Ulva lactuca				0.84%	83.80%	15.36%	Wahbeh (1997)
Enteromorpha compressa				0.65%	90.20%	9.15%	Wahbeh (1997)
Enteromorpha sp.	3			0.23%	83.90%	15.87%	Zemke-White and Clements (1999)
Ulva rigida	3			0.17%	83.79%	16.04%	Zemke-White and Clements (1999)
Ulva fenestrate				0.50%			Khotimchenko and Yakovleva (2004)
Ulva sp./*Enteromorpha* sp.			0%	0.05%			Don Cadien, LACSD, *Pers. comm.*
Enteromorpha intestinalis and *Ulva* sp.			0%				Krista Kamer, Moss Landing Marine Labs, *Pers. comm.*

Table V.2. Selected parameter values, distribution type and summary statistics for Monte Carlo simulations.

Parameter	Value	Distribution Type	Endpoints
Percent water column exp.	0	Uniform	minimum = 0; maximum = 5%
Tissue lipid content	0.40%	Normal	μ = 0.4%; SD = 0.3%; SE = 0.1%
Tissue % NLOM	15.5%	Normal	μ = 15.5%; SD = 3%; SE = 1.7%
Growth Rate	4.7%	Lognormal	GeoMean = 4.7% (Log= -1.32); Log SD = 0.25

Appendix W. Method for estimating overall prey tissue lipid content encountered in Newport Bay case study.

There are a number of approaches one could take to average percent lipid across all of the fish species consumed by a predator. For estimation of the sediment threshold in the Newport Bay case study (Section 4.4.3), sensitivity to two factors is considered: 1. whether the prey species are likely to be resident of Newport Bay; and 2. the relative importance of the prey species, based on sport fish consumption studies.

The simplest approach to determining average percent lipid is to simply average the percent lipid of major prey species of human or wildlife piscivores. Table W.1 lists major prey species, type of tissues and percent lipid for human and wildlife predators. Averaging across all prey species evenly, the arithmetic mean of percent lipid is 1.88 for human prey, and 1.19 for wildlife prey. Human prey concentrations higher due to expected consumption of the fatty California corbina and striped mullet (Table W.1).

Table W.1. Major prey species, type of tissues and percent lipid consumed by human and wildlife predators. Survey results are obtained from Allen *et al.* (1996).

Prey Species	Receptor	Tissue Type	% Lipid [a]	Resident?	Survey Analogous Species[b]	Survey Number Captured[b]	Survey % Captured
Barred sand bass	Human	Fillet	1.0	N	Barred sand bass	388	32%
California corbina	Human	Fillet	4.6	N	None	0	0%
California halibut	Human	Fillet	0.4	Y	California halibut	62	5%
Spotted sand bass	Human	Fillet	0.9	Y	Kelp bass	327	27%
Striped mullet	Human	Fillet	4.4	N	None	0	0%
Yellowfin croaker	Human	Fillet	1.7	N	White croaker	339	28%
Black perch	Human	Fillet	0.7	N	Black perch	26	2%
Jacksmelt	Human	Fillet	1.3	N	Jacksmelt	74	6%
Total	Human					1216	
Arrow goby	Wildlife	Whole	1.3	Y			
Black perch	Wildlife	Whole	1.1	Y			
California killifish	Wildlife	Whole	1.5	Y			
California halibut	Wildlife	Whole	0.9	Y			
Cheekspot goby	Wildlife	Whole	1.5	Y			
Diamond turbot	Wildlife	Whole	0.8	Y			
Staghorn sculpin	Wildlife	Whole	1.4	Y			
Shiner surfperch	Wildlife	Whole	1.0	Y			

a. From Table 4.13 and Appendix U of this report, and Allen *et al.* (2004). b. Santa Monica Bay Seafood Consumption Survey (Allen *et al.* 1996).

Percent lipid may also be estimated using weighted averages based on relative consumption rates of different types of prey. In the case of Newport Bay, although

relative consumption rates are not known, they may be estimated for human sport fishers based on results of the Santa Monica Bay Seafood Consumption Survey (Allen *et al.* 1996). This survey indicates local consumption practices (OEHHA 2001), and includes total number of each fish species taken across the entire survey. Proportionate number captured in the survey may be combined with percent lipid in fillet tissue from the same or analogous species in Newport Bay (Table W.1) to derive an estimate of tissue percent lipid, weighted by popularity of the prey item. This weighted average estimate of tissue percent lipid (1.15%) is lower than the simple average of all species targeted by sport fish consumers (1.88%) because the fattier species were not captured in Santa Monica Bay. The absence of striped mullet and California corbina from this average, when these species are known to be recreational sport fish (www.fishbase.org), highlights the substantial uncertainty in actual human sport fish consumption estimates in Newport Bay. Weighted averages were not conducted for wildlife predators because local information on relative prey preferences are lacking.

An alternative approach to prey attribute estimation is to focus on resident species only. The rationale for this approach is that non-resident species may receive some contaminant burden from other locations, and therefore not be representative of Newport Bay sediment exposure. All wildlife prey are considered to be resident species, but among human prey, only California halibut and spotted sand bass are resident (M. J. Allen, SCCWRP, *Pers. comm.*). The average tissue percent lipid of these two species (0.65%) is substantially lower than other averages (Table W.2).

Table W.2. Summary of estimated tissue percent lipids for human and wildlife prey using different selection and averaging methods.

Treatment	Tissue % lipid
All wildlife prey	1.19
All human prey	1.88
Human prey resident only	0.65
Human prey Santa Monica Survey	1.15

Appendix X. Comparison of model vs. empirical tissue concentrations of pesticide pollutants in San Francisco Bay

Figure X.1. Results of model vs. data comparison for white croaker (*Genyonemus lineatus*). Results are derived from the Gobas and Arnot food-web model (model) vs. empirical data (data). For all figures, error bars represent one standard deviation of the mean. When the empirical data are represented by a yellow bar, this indicates the detection limit, as greater than 90% of empirical results were below detection for that compound.

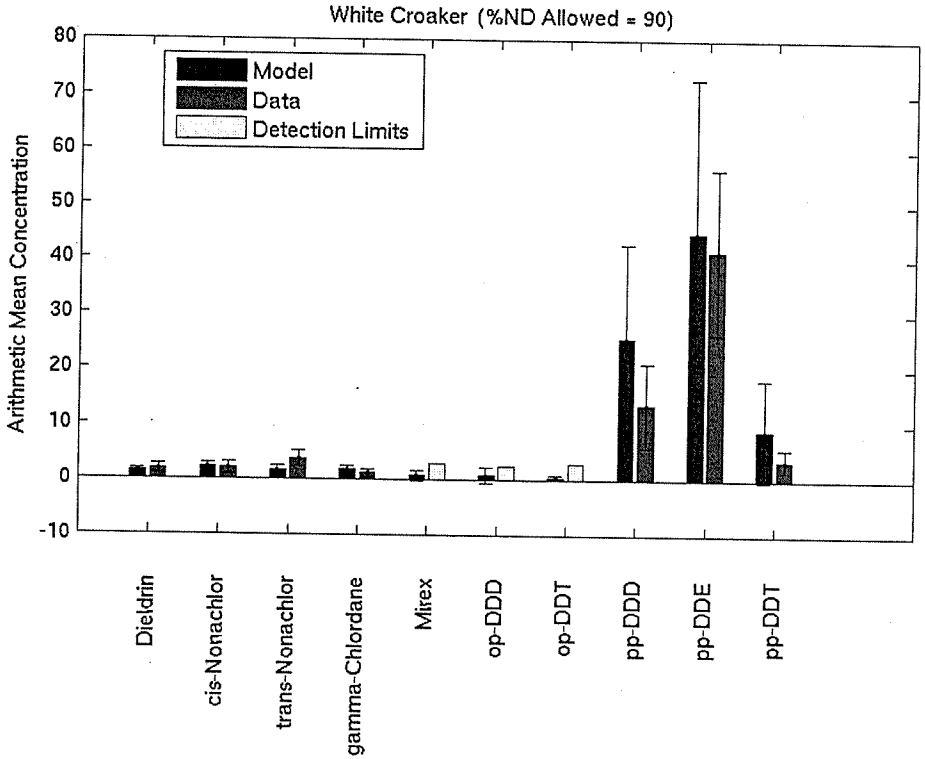


Figure X.2. Results of model vs. data comparison for jacksmelt (*Atherinopsis californiensis*). See caption for Figure X.1.

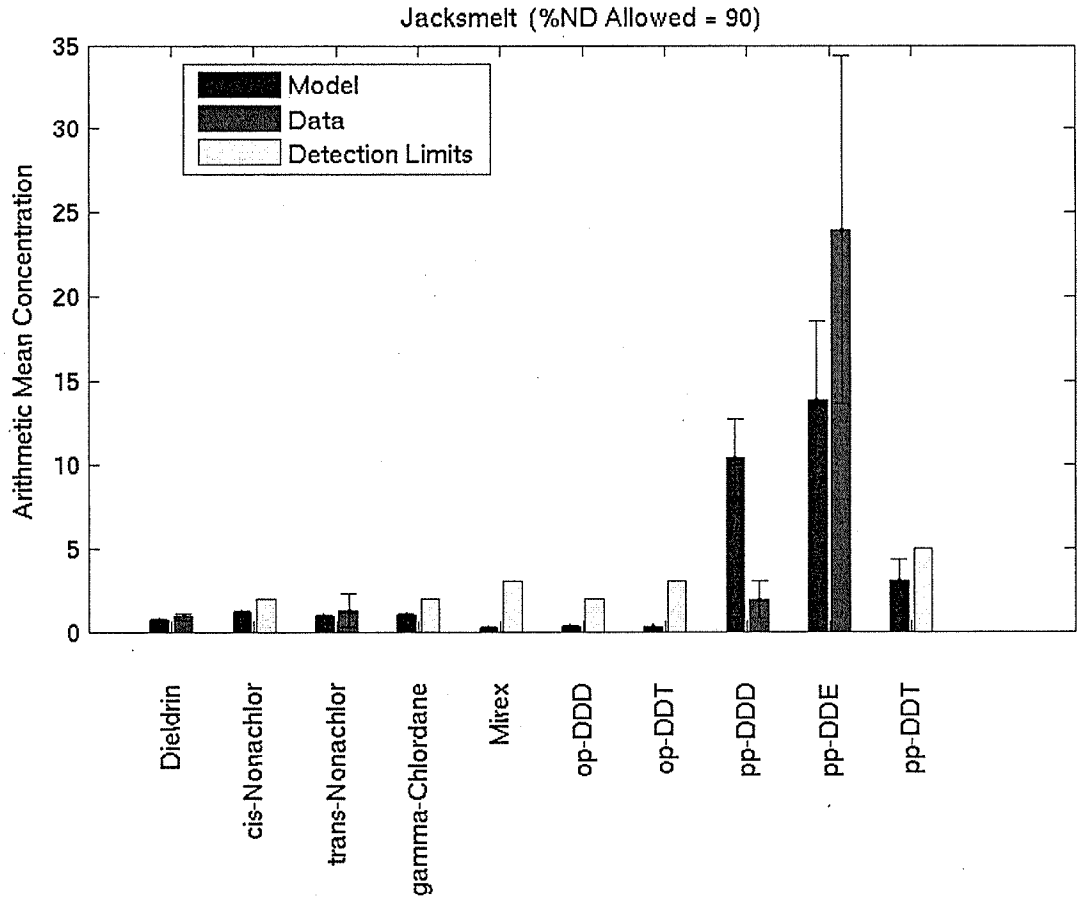


Figure X.3. Results of model vs. data comparison for oysters (*Crassostrea gigas*). See caption for Figure X.1.

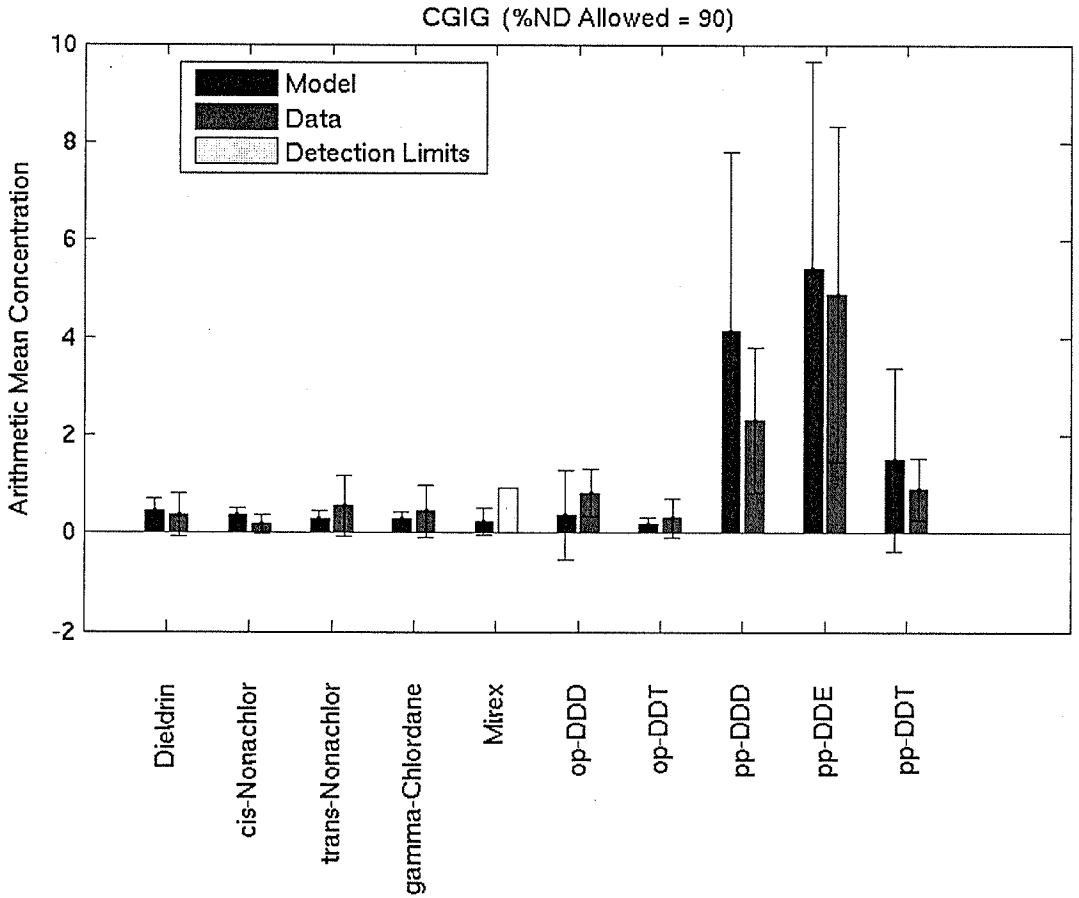
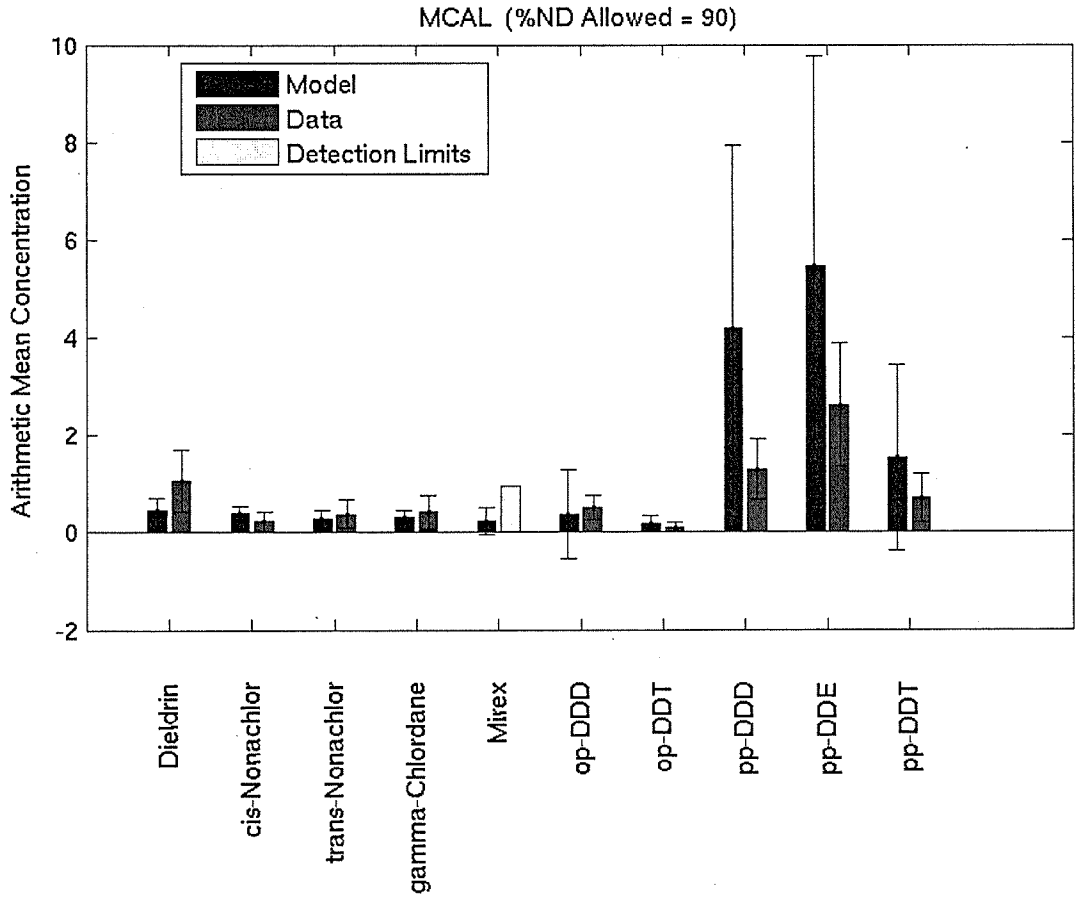


Figure X.4. Results of model vs. data comparison for California mussel (*Mytilus californianus*). See caption for Figure X.1.



Appendix Y. Summary of input data for application of Arnot and Gobas (2004) mechanistic food web model used in this report.

Physical data

1. Mean water and air temperature
2. Salinity
3. Dissolved organic carbon content - water column
4. Particulate organic carbon content - water column
5. Concentration of suspended solids – water column (TSS)
6. Percentage of organic carbon - sediment

Contaminant data

7. Concentrations of target contaminants in water and sediments. This is preferably sampled according to similar temporal and spatial scales as the biota.
8. Concentrations of target contaminants in each organism (for model validation or calibration). This is preferably whole body wet weight analysis. If specific tissues are analyzed (i.e. muscle fillet) then it is preferable to measure tissue-specific lipid content as well as the whole body lipid content, and convert following Appendix E.

Biological data

9. Develop a simplified representation of the local food web to model. This should include about 10 species, typically generalizing primary producers and invertebrates across broad taxonomic categories.
10. Develop a matrix of percent diet for each species in the food web. Depending on data availability, this can be the result of gut surveys, stable isotope analysis or general feeding behaviors (e.g., species X predominantly feeds on species Y and Z which are represented by organisms with the same trophic guilds in the model).
11. Whole body percent lipid for each organism.
12. Whole body wet weight for each organism.
13. A narrative description of the exposure of the organism's gills or respiratory organs to sediments and pore water, as compared to the overlying water column (used to estimate percent of respired water that is pore water).

Optional local input data for model (can be estimated if not available)

Physical data

14. Density of organic carbon in sediment.
15. Dissolved oxygen concentration in the water column.

Contaminant data

16. Freely dissolved water contaminant concentration
17. Pore water contaminant concentration

6/12/99

Sediment Quality Guidelines developed for the National Status and Trends Program

Background and Intended Uses

Through its National Status and Trends (NS&T) Program, NOAA generates considerable amounts of chemical data on sediments. Without national criteria or other widely-applicable numerical tools, NOAA scientists found it difficult to estimate the possible toxicological significance of chemical concentrations in sediments. Thus, numerical sediment quality guidelines (SQGs) were developed as informal, interpretive tools for the NS&T Program.

The SQGs were initially intended for use by NOAA scientists in ranking areas that warranted further detailed study on the actual occurrence of adverse effects such as toxicity. Also, they were intended for use in ranking chemicals that might be of potential concern. In many regional surveys of sediment toxicity performed throughout North America, NOAA has used the guidelines to compare the degree of contamination among sub-regions, and to identify chemicals elevated in concentration above the guidelines that were also associated with measures of adverse effects.

The SQGs were not promulgated as regulatory criteria or standards. They were not intended as cleanup or remediation targets, nor as discharge attainment targets. Nor were they intended as pass-fail criteria for dredged material disposal decisions or any other regulatory purpose. Rather, they were intended as informal (non-regulatory) guidelines for use in interpreting chemical data from analyses of sediments.

Derivation

SQGs were needed relatively quickly for use in interpreting data from the ongoing NS&T Program studies; thus, existing data were used in their derivation, rather than data from tedious and expensive laboratory tests or modeling approaches. SQGs were needed that could be applied nationwide in the NS&T Program; therefore, data from studies performed throughout North America were assembled and compiled into a database to ensure broad applicability of the guidelines. Because guidelines were needed that were based on measures of biological effects associated with toxicants, data were compiled that included both chemical measures and biological effects. SQGs based on a weight of evidence from numerous studies were expected to be more useful nationwide than values based upon only limited amounts of data. SQGs were needed for a variety of different substances commonly measured in the NS&T Program; accordingly, guidelines were developed for as many chemicals as the data would warrant. SQGs were needed that would estimate the “safe” concentrations, i.e.,

concentrations below which effects were not likely. Also, guidelines were needed above which adverse effects were more likely. Therefore, two values were derived for each substance.

SQGs were derived initially using a database compiled from studies performed in both saltwater and freshwater and published in NOAA Technical Memorandum NOS OMA 52 (Long and Morgan 1990). A larger database compiled from many studies performed by numerous investigators in only saltwater was used to revise and update the SQGs (Long et al. 1995). Data from freshwater studies and/or of marginal quality used in 1990 were removed from the database in 1995, and a considerable amount of higher quality data were added to the database. Data from each study were arranged in order of ascending concentrations. Study endpoints in which adverse effects were reported were identified. From the ascending data tables, the 10th percentile and the 50th percentile (median) of the effects database were identified for each substance. The 10th percentile values were named the "Effects Range-Low" (ERL), indicative of concentrations below which adverse effects rarely occur. The 50th percentiles were named the "Effects Range-Median" (ERM) values, representative of concentrations above which effects frequently occur.

An example of the derivation method is shown in **Figure 1** in which the data for phenanthrene are arranged in ascending order. Green symbols indicate study endpoints in which no adverse effects were observed, such as in reference area samples. Red symbols indicate those study endpoints at which an adverse effect was observed. In the case of phenanthrene, there were 53 study endpoints indicating adverse effects. The 10th percentile of this data distribution was the 6th value, equivalent to 240 ppb phenanthrene. The 50th percentile was the 27th value, equivalent to 1500 ppb. As was apparent in the data for phenanthrene, the percentages of study endpoints indicating toxicity increased with increasing concentrations of most chemicals. The measures of reliability discussed below were calculated from the data available within the three concentration ranges defined by the ERL and ERM.

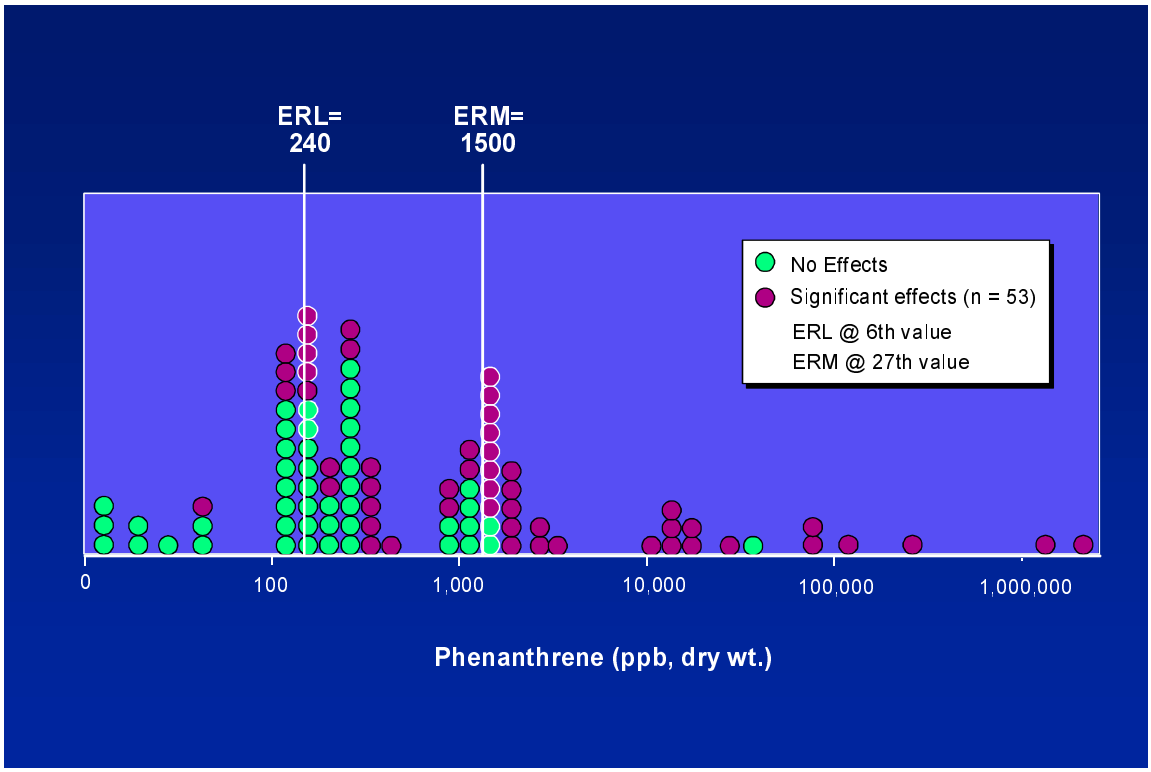


Figure 1. An example of the database used to derive the SQGs. Data for phenanthrene in which no adverse effects were observed are indicated by green symbols and those in which toxicity or some other measure of adverse effects were observed are indicated by red symbols. ERL= Effects Range-Low; ERM= Effects Range-Median.

The sediment quality guidelines

Based on the database assembled by Long et al. (1995), ERL and ERM values were calculated for 9 trace metals, 13 individual PAHs, 3 classes of PAHs, and 3 classes of chlorinated organic hydrocarbons **(Tables 1 and 2)**. There were insufficient amounts of reliable data available to perform similar calculations for other substances, including a few previously reported by Long and Morgan (1990).

The amount and quality of data used to derive the SQGs differed among the substances. Therefore, to provide a measure of the reliability of the SQGs, the percentages of study endpoints indicating adverse effects were calculated for the chemical ranges defined by the ERLs and ERMs **(Tables 1 and 2)**. Because the ERLs were intended to represent concentrations below which effects were rarely observed, low percentages of studies were expected to indicate effects within the ranges below the ERLs. Indeed, for all trace metals the percent of studies indicating adverse effects was less than 10% when concentrations were below the ERL values. For most organics, the incidence of effects was less than 25% when concentrations were below the ERLs.

Table 1. ERL and ERM guideline values for trace metals (ppm, dry wt.) and percent incidence of biological effects in concentration ranges defined by the two values (from Long et al., 1995). ERL= Effects Range-Low; ERM= Effects Range-Median.

	Guidelines		Percent incidence of effects*		
Chemical	ERL	ERM	<ERL	ERL - ERM	>ERM
Arsenic	8.2	70	5.0	11.1	63.0
Cadmium	1.2	9.6	6.6	36.6	65.7
Chromium	81	370	2.9	21.1	95.0
Copper	34	270	9.4	29.1	83.7
Lead	46.7	218	8.0	35.8	90.2
Mercury	0.15	0.71	8.3	23.5	42.3
Nickel	20.9	51.6	1.9	16.7	16.9
Silver	1.0	3.7	2.6	32.3	92.8
Zinc	150	410	6.1	47.0	69.8

*Number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range.

The incidence of effects increased to 20% to 30% for most trace metals and 40% to 60% for most organics when concentrations exceeded ERL values but were lower than the ERM values. When concentrations exceeded the ERM values, the incidence of adverse effects increased to 60% to 90% for most trace metals and 80% to 100% for most organics. However, the reliabilities of the ERMs for nickel, mercury, DDE, total DDTs, and total PCBs were much lower than those for other substances. Therefore, the probabilities that the ERM values for these substances would accurately predict adverse effects are much lower than those for most chemicals.

Table 2. ERL and ERM guideline values for organic compounds (ppb, dry wt.) and percent incidence of biological effects in concentration ranges defined by the two values (from Long et al. 1995). ERL= Effects Range-Low; ERM= Effects Range-Median.

	Guidelines		Percent incidence of effects*		
Chemical	ERL	ERM	<ERL	ERL--ERM	>ERM
Acenaphthene	16	500	20.0	32.4	84.2
Acenaphthylene	44	640	14.3	17.9	100
Anthracene	85.3	1100	25.0	44.2	85.2
Fluorene	19	540	27.3	36.5	86.7
2-methyl naphthalene	70	670	12.5	73.3	100
Naphthalene	160	2100	16.0	41.0	88.9
Phenanthrene	240	1500	18.5	46.2	90.3
Sum LPAH	552	3160	13.0	48.1	100
Benz(a)anthracene	261	1600	21.1	43.8	92.6
Benzo(a)pyrene	430	1600	10.3	63.0	80.0
Chrysene	384	2800	19.0	45.0	88.5
Dibenzo (a,h) anthracene	63.4	260	11.5	54.5	66.7
Fluoranthene	600	5100	20.6	63.6	92.3
Pyrene	665	2600	17.2	53.1	87.5
Sum HPAH	1700	9600	10.5	40.0	81.2
Sum of total PAH	4022	44792	14.3	36.1	85.0
p,p'-DDE	2.2	27	5.0	50.0	50.0
Sum total DDTs	1.58	46.1	20.0	75.0	53.6
Total PCBs	22.7	180	18.5	40.8	51.0

*Number of data entries within each concentration range in which biological effects were observed divided by the total number of entries within each range.

Interpretation

Two guideline values were generated for each chemical: the ERL and the ERM. It is important to understand that these values were not derived as toxicity thresholds. That is, there is no assurance that there will be a total lack of toxicity when chemical concentrations are less than the ERL values. Similarly, there is no assurance that samples in which ERM values are exceeded will be toxic. Toxicity, or a lack thereof, must be confirmed with empirical data from toxicity tests.

The ERL values were not intended as concentrations that are always predictive of toxicity. Rather, they were intended and should be used primarily as estimates of the concentrations below which toxicity is least likely. As shown in Tables 1 and 2, the incidence of effects was usually higher when concentrations exceeded the ERLs than when concentrations were below the ERLs. However, the ERM values are better indicators of concentrations associated with effects than the ERLs.

Uses

The guidelines are commonly used in North America both to rank and prioritize sites of concern and chemicals of concern. That is, samples or study areas in which many chemicals exceed the ERM values and exceed them by a large

degree may be considered as more contaminated than those in which none of the SQGs are exceeded. Samples in which ERL concentrations are exceeded, but no ERM values are exceeded, might be given intermediate ranks. Similarly, chemicals at concentrations well above the ERM values might be given a higher priority than those at concentrations below the ERLs. Chemicals at intermediate concentrations may qualify as a moderate priority. However, caution should be used when prioritizing sites or chemicals where only the concentrations of nickel, mercury, DDE, total DDTs, or total PCBs are elevated.

In studies performed by NOAA of toxicity and contamination of sediments in specific estuaries and bays, the SQGs have been used to rank and prioritize both sites and chemicals of potential concern. In these studies the chemical data were compared with the SQGs to identify spatial patterns in contamination, to estimate the spatial scales in contamination, and to rank sampling sites. The data also were compared with the SQGs to (1) identify which chemicals, if any, exceeded the ERL and ERM values, (2) tally the number of samples in which the SQGs were exceeded, (3) calculate the degrees to which the SQGs were exceeded, and (4) to identify which chemicals were most associated with measures of toxicity. For each regional assessment of bioeffects, the SQGs were used along with the results of toxicity tests to estimate the relative quality of sediments throughout the study area.

Field validation of predictive ability

To provide quantitative information on how well the SQGs correctly predict toxicity in actual field conditions, an analysis was conducted (Long et al. 1998a) with existing data compiled from many regional assessments conducted by NOAA and EPA. Matching chemistry and toxicity data from 1,068 samples from the Atlantic, Gulf of Mexico, and Pacific coasts were compiled into a database. Data were available from acute amphipod survival tests for all 1,068 samples; data from one or two additional tests in which sublethal responses were recorded were available for 437 samples. Several analyses were conducted with the data to investigate the predictive ability of the SQGs.

In the first analysis, the percentages of samples that were highly toxic were determined when individual ERM values were equaled or exceeded. That is, samples were identified in which the ERM value was equaled/exceeded for a particular substance. The percentages of those samples that were highly toxic in either the amphipod survival tests alone or in a battery of 2 to 4 tests (including those with amphipods) were then determined. Statistical analyses were used to classify samples as either non-toxic ($p>0.05$), marginally toxic ($p<0.05$), or highly toxic ($p<0.05$ and sample means exceed minimum significant differences) relative to controls in the laboratory tests. The predictive abilities of 28 sets of ERLs/ERMs were determined.

For most substances, 40% to 60% of samples in which chemical concentrations exceeded individual ERMs were highly toxic in the amphipod tests **(Figure 2)**.

For example, among the samples in which copper concentrations exceeded the ERM value (n=25), 52% were highly toxic in the amphipod survival tests. More than 75% of samples were highly toxic in which the ERMs for lead, 2-methylnaphthalene, and acenaphthylene were exceeded. For most substances, an increase in predictive ability of approximately 20% to 30% occurred when the data from the sublethal tests were included along with the amphipod data. Therefore, for most substances, 80% to 90% of samples were highly toxic in at least one of the tests performed when concentrations exceeded individual ERMs.

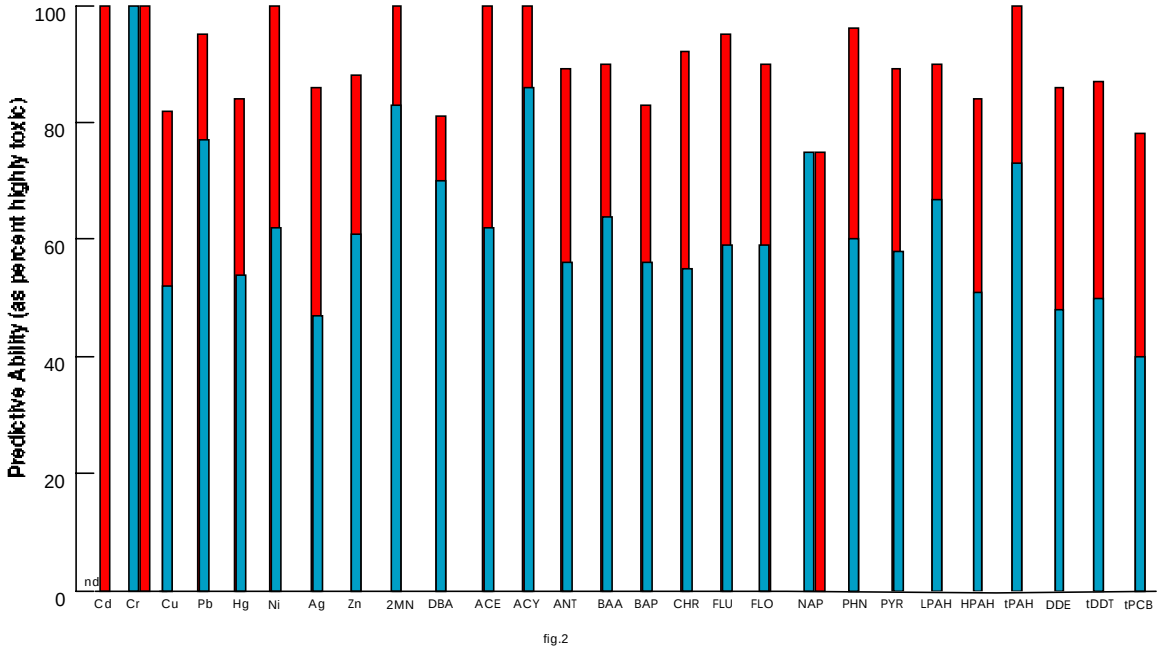


Figure 2. Percent of sediment samples in field validation database in which highly significant toxicity was observed in amphipod survival tests alone (blue bars) and in any of 2 to 4 tests performed (red bars) when chemical concentrations equaled or exceeded individual ERM values (from Long et al., 1998a). ERL= Effects Range-Low; ERM= Effects Range-Median.

These data suggest that individual ERM values were reasonably predictive of toxicity. Given that the ERMs were derived as median values (not toxicity thresholds) in the effects database, predictive abilities of roughly 50% might be expected. Indeed, in the amphipod tests, 40% to 60% of samples were highly toxic when individual ERMs were exceeded. However, chemicals often occur in complex mixtures in environmental samples and toxicity in these tests could not be necessarily attributable to the substance which occurred at concentrations greater than the ERM values.

Therefore, a second series of analyses was conducted to estimate the effects of additivity of toxicants upon measures of toxicity. In these analyses the

percentages of samples were calculated for several categories of samples: (1) no SQGs exceeded, (2) only ERLs exceeded, no ERMs equaled/exceeded, (3) increasing numbers of ERMs exceeded.

Table 3. Percentages of samples in which no significant toxicity, marginal toxicity, and highly significant toxicity was observed in amphipod survival tests (from Long et al., 1998a). ERL= Effects Range-Low; ERM= Effects Range-Median.

Chemical category	Number of samples	Percent not toxic	Percent marginally toxic	Percent highly toxic
no ERLs exceeded	329	68	21	11
1 or more ERLs exceeded	448	63	20	18
1 or more ERMs exceeded	291	48	13	39
1 to 5 ERMs exceeded	225	53	15	32
6 to 10 ERMs exceeded	46	37	11	52
11 to 20 ERMs exceeded	20	10	05	85

Only 11% of the 329 samples were highly toxic in the amphipod tests when none of the ERLs were exceeded **(Table 3)**. In this category, 21% of the samples were marginally toxic and 68% were not significantly toxic in this category. These data suggest that the ERLs were reasonably predictive of non-toxic conditions.

Given that the ERLs were calculated as the 10th percentiles of effects data, roughly equivalent predictive abilities (i.e., about 10%) were expected in this field validation study. The data, however, indicated that 18% of samples in which one or more ERLs (but, no ERMs) were exceeded were highly toxic. The incidence of toxicity increased with increases in the numbers of ERLs exceeded, peaking at 67% when 15 to 19 ERLs were exceeded (Long et al. 1998a; data not shown).

Given that the ERMs were derived as 50th percentile values in the effects databases, roughly equivalent predictive abilities (i.e., about 50%) were expected. There were 291 samples in which at least 1 ERM was exceeded by any amount **(Table 3)**. Among these samples, 13% were marginally toxic and 39% were highly toxic. As the numbers of chemicals exceeding the ERMs increased, there was an increase in the percentages of samples that were highly toxic, peaking at 85% when 11 to 20 ERMs were exceeded.

Mean ERM quotients

Chemicals often occur in saltwater sediments as complex mixtures. To provide a tool useful in assessing the potential toxicological significance of the presence of mixtures, mean ERM quotients were calculated for all 1068 samples used in the field validation study (Long et al. 1998a). These indices were derived as the average of the 25 quotients obtained by dividing the individual chemical concentrations by their respective ERM values. The percentages of samples

that were not toxic, marginally toxic, and highly toxic were determined within ranges in the quotients. The data suggested a relatively consistent dose-response relationship: as the mean ERM quotients increased, the incidence of highly toxic responses increased (Long et al. 1998a). As more experience is gained with this tool, it may be useful in assessing the potential significance of chemical mixtures in sediment samples.

Probabilities of toxicity

The data from the study of predictive ability were compiled for both the sets of ERL and ERM values (from Long et al. 1995) and the comparable TEL (Threshold Effects Levels) and PEL (Probable Effects Levels) values from MacDonald et al. (1996) to provide a synopsis of the likelihood of significant toxicity in amphipod survival tests (Long and MacDonald 1998). This is an attempt to estimate the likelihood that samples with certain chemical characteristics would be toxic.

Table 4 lists the chemical characteristics that equate to different probabilities of amphipod toxicity based on the data from Long et al. (1998a). Data used to derive **Table 4** were compiled from Long et al. (1998a) in which there were 1086 samples and merged with more recent data from Biscayne Bay (FL) (n=226) and Pearl Harbor (HI) n=219), giving a total data set of 1513 samples. These samples were collected in various studies performed on the Atlantic, Pacific and Gulf of Mexico coasts.

The percent incidence of highly toxic responses and the average survival of the amphipods in all samples within each cateogry are shown in **Table 4**. Four chemical indices calibrated to the SQGs are shown for each of four categories. In category 1, sediments least likely to be toxic were actually toxic in only 8-9% of the samples. Average amphipod survival in these samples was 92-93%, indicating that survival, on average, was not decreased appreciably from what would be expected in clean reference sediments. As the numbers of SQGs exceeded increases and as the mean SQG quotients increase, the incidence of toxicity increases and the average survival rate decreases.

Samples with chemical characteristics equivalent to Category 2 have the most uncertainty as to toxicity. Average survival approximates the critical threshold of 80% of controls, whereas in the other categories, average survival is clearly greater than or less than 80%. In category 3, about 50% of samples were toxic and average survival was about 60-70%. In category 4, about 73-83% of samples were toxic and average survival dropped to about 40%, indicating high probabilities of toxic conditions.

These data may be useful in determining the need for additional testing and analyses of sediments. For example, the probability of incorrectly classifying a site as non-toxic when all chemical concentrations are below all SQGs and either of the mean SQG quotients is less than 0.1 is about 10% and the probability of a site being toxic is about 75% or greater when chemical data match the

characteristics of Category 4 conditions in **Table 4**. However, in sediments classified as Category 2, toxicity or the lack thereof is more uncertain.

Table 4. Percent incidence of highly toxic samples and average percent amphipod survival in marine sediment samples classified according to numerical sediment quality guidelines.

Chemical characteristics relative to sediment guidelines	Percent highly toxic* samples		Average, control-adjusted amphipod survival	
	National** database (n=1068)	Combined summary (n=1513)	National** database (n=1068)	Combined summary (n=1513)
Category 1:				
• mean ERM quotients <0.1	11	9	93	93
• mean PEL quotients <0.1	10	8	93	93
• no ERLs exceeded	11	9	92	92
• no TELs exceeded	9	8	92	92
Category 2:				
• mean ERM quotients 0.11 - 0.5	30	21	81	86
• mean PEL quotients 0.11 - 1.5	25	21	84	86
• 1-5 ERMs exceeded	32	32	79	79
• 1-5 PELs exceeded	24	18	83	88
Category 3:				
• mean ERM quotients 0.51-1.5	46	49	74	70
• mean PEL quotients 1.51 - 2.3	50	49	66	68
• 6-10 ERMs exceeded	52	57	63	59
• 6-20 PELs exceeded	47	48	71	70
Category 4:				
• mean ERM quotients >1.5	75	76	43	41
• mean PEL quotients >2.3	77	73	47	46
• >10 ERMs exceeded	85	80	41	41
• >20 PELs exceeded	88	83	38	37

* mean survival significantly different from controls and <80% of controls
** data from Long et al., 1998

The ERLs and mean ERM quotients for saltwater were more efficient at correctly predicting non-toxicity (100% and 93% correct, respectively) than SEM:AVS ratios (80% correct) based on analyses of data compiled to field-validate the SEM:AVS criteria (Long et al., 1998b). Also, the ERMs and mean ERM quotients were slightly more predictive of toxic conditions (33% and 42% correct, respectively) than the SEM:AVS ratios (26% correct). These data suggest that the predictive abilities of SQGs based on bulk trace metals data are not improved with SEM-to-AVS normalizations (Long et al., 1998b).

Limitations

The SQGs should be used with caution and common sense. There are no SQGs available for many substances that can be highly toxic in sediments. The abilities of the SQGs to correctly predict toxicity of co-varying substances for which there are no SQGs are unknown. The SQGs were derived in units of dry weight sediments; therefore, they do not account for the potential effects of geochemical factors in sediments that may influence contaminant bioavailability. The SQGs were not intended for use in predicting effects in wildlife or humans through bioaccumulation pathways. The SQGs were neither calculated nor intended as toxicological thresholds; therefore, there is no certainty that they will always correctly predict either non-toxicity or toxicity. The SQGs were derived with data from soft sedimentary deposits; they should not be applied to assessments of upland soils, gravel, coarse sand, tar, slag, or metal ore.

The SQGs are best applied when accompanied by measures of effects such as laboratory toxicity tests and/or benthic community analyses and/or bioaccumulation tests, which lead to the preparation of a weight of evidence. Furthermore, they are best applied in a comprehensive assessment framework involving the establishment of clear study objectives, *a priori* methods for data analyses, and well-understood decision points regarding the uses of the data.

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Human and Ecological Risk Assessment: Vol. 4, No. 5, pp. 1019-1039 (1998)

See p. 1032

PERSPECTIVE:

Recommended Uses of Empirically Derived, Sediment Quality Guidelines for Marine and Estuarine Ecosystems

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ABSTRACT

Sediment quality guidelines (SQGs), based upon empirical analyses of matching chemical and biological data, have been developed for many potentially toxic substances. The predictive abilities and recommended applications of two sets of guidelines, ERLs/ERMs and TELs/PELs, are discussed in this paper. The SQGs were intended as informal (*i.e.*, non-regulatory) benchmarks to aid in the interpretation of chemical data. Low-range values (*i.e.*, ERLs or TELs) were intended as concentrations below which adverse effects upon sediment-dwelling fauna would be expected only infrequently. In contrast, the ERMs and PELs represent chemical concentrations above which adverse effects are likely to occur. Evaluations of the reliability and predictive ability of the SQGs indicate they can be used effectively to assess the quality of soft, aqueous, sedimentary deposits. Specifically, the SQGs can be used to classify sediment samples with regard to their potential for toxicity, to identify contaminants of concern, and to prioritize areas of concern based on the frequency and degree to which guidelines are exceeded. Toxicity and bioaccumulation tests, toxicity identification evaluations, and benthic community assessments provide complimentary information for assessing sediment quality.

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INTRODUCTION

Numerical sediment quality guidelines (SQGs) have been developed using a variety of approaches, typically involving statistical comparisons of chemical concentrations and measures of adverse biological effects upon exposure to sediments (see USEPA, 1992a; Adams *et al.*, 1992; Chapman, 1989; Ingersoll *et al.*, 1997 for reviews). For certain substances, equilibrium-partitioning models have been used to develop proposed sediment quality criteria (SQC) (USEPA, 1992b; 1994a, 1994b; Ankley *et al.*, 1997; Swartz *et al.*, 1995). Such SQGs and proposed SQCs were developed to provide effects-based, interpretive tools for assessing the quality of sediments. All values were intended to represent either the chemical concentrations below which adverse biological effects were not expected or levels above which effects, such as acute toxicity, would be expected. Thus far, none of these values have been promulgated and implemented for standards or criteria in the U.S.

All SQGs developed thus far, except for the standards set for marine sediments by the state of Washington (WDOE, 1995) and the five criteria proposed by USEPA (1994a), were intended to provide informal interpretive tools or benchmarks. That is, they were developed to provide a basis for evaluating the risks posed to sediment-dwelling organisms by sediment-associated contaminants. While some agencies have developed their own SQGs, others have elected to adopt guidelines prepared by others. Collectively, we are aware of at least 25 federal and state agencies or programs in North America that have either developed SQGs or adopted those prepared by other organizations.

One set of guidelines — Effects Range-Low (ERL) and Effects Range-Median (ERM) values — was originally calculated to provide a means for interpreting monitoring data collected under the National Status and Trends Program (NSTP) of the National Oceanic and Atmospheric Administration (NOAA) (Long and Morgan, 1990; Long *et al.*, 1995). An anologous set of saltwater values — Threshold Effects Levels (TEL) and Probable Effects Levels (PEL) — was calculated for the state of Florida (MacDonald *et al.*, 1996).

Many scientists and administrators in North America have used available SQGs to rank and/or prioritize chemicals of potential concern in specific regions or hazardous waste sites and/or to rank and prioritize areas for further investigation and/or management action. Other uses of SQGs have included: planning monitoring programs; designing spiked sediment bioassays; interpreting historical data; evaluating the need for further studies; assessing the quality of prospective dredge materials; and conducting remedial investigations and ecological risk assessments of waste sites. Often the ways in which the guidelines have been used were consistent with their original intent. However, we have observed that SQGs have also been used in other ways, occasionally resulting in controversies over their proper use. The objectives of this paper are to: (1) provide a synopsis of the reliability and predictive ability of the two sets of SQGs and (2) provide recommendations on the uses of these SQGs for assessing sediment quality, primarily based upon their ability to correctly classifying sediments as either toxic or non-toxic.

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CALCULATIONS OF EMPIRICALLY DERIVED SQGS

This paper focuses upon two sets of saltwater SQGs; ERLs and ERMs (Long *et al.*, 1995) and TELs and PELs (MacDonald *et al.*, 1996). Both sets of SQGs were derived using statistical analyses of matching chemistry and biological data compiled from numerous field, laboratory, and modeling studies performed throughout North America. Sufficient amounts of data were compiled to identify with a weight of evidence the chemical concentrations that were rarely, occasionally, and frequently associated with measures of toxicity. Data were derived from analyses of sediments with different physical and geochemical characteristics and from numerous locations to provide a basis for establishing SQGs that should be widely applicable throughout North America. The majority of the data used to derive the guidelines were from field studies in which mixtures of substances occurred in the samples, thus maximizing the applicability of the SQGs to most real-world situations (*i.e.*, those involving mixtures of contaminants). Data for a variety of toxicological endpoints were gathered from published studies also to broaden the applicability of the resulting SQGs.

ERL and ERM values were derived using only chemical concentrations associated with adverse effects (Long *et al.*, 1995), whereas calculations of the TEL and PEL values incorporated concentrations associated with both effects and no observed effects (MacDonald *et al.*, 1996). The ERL and TEL values represented concentrations below which effects were rarely observed, and, therefore, estimated the low end of the effects range. In contrast, the ERM and PEL values represented chemical concentrations toward the middle of the effects range and above which effects would be likely to occur. However, the ERM and PEL values are not intended to represent effects thresholds above which adverse effects would always be observed (such as with apparent effects thresholds — Barrick *et al.*, 1988). By deriving a lower-range and mid-range values for each substance, we attempted to minimize the incidence of both false negatives and false positives.

RELIABILITY AND PREDICTIVE ABILITY OF EMPIRICALLY DERIVED SQGS

We believe that information on the reliability and predictive ability of numerical SQGs is essential to determining their appropriate uses. A panel of environmental scientists concluded that "field validation is an essential element of the SQGs development process because it provides a basis for assessing their reliability in sediment quality assessments" (Ingersoll *et al.*, 1997). In this context, the term "reliability" is defined as the ability of the SQGs to correctly classify sediments as toxic or non-toxic based upon the data used to derive the guidelines. The degree to which the SQGs correctly classify sediments as either toxic and non-toxic in independent data sets (*i.e.*, not used in the guidelines derivation) is referred to here as "predictive ability". All approaches to the derivation of numerical values have specific strengths and weaknesses (USEPA, 1992a; Ingersoll *et al.*, 1997) that will influence their reliability and predictive

ability. Regardless of how they are derived, SQGs, if they are to be useful, must be shown to correctly classify samples as either non-toxic or toxic under a wide variety of ambient conditions.

Reliability of SQGs.

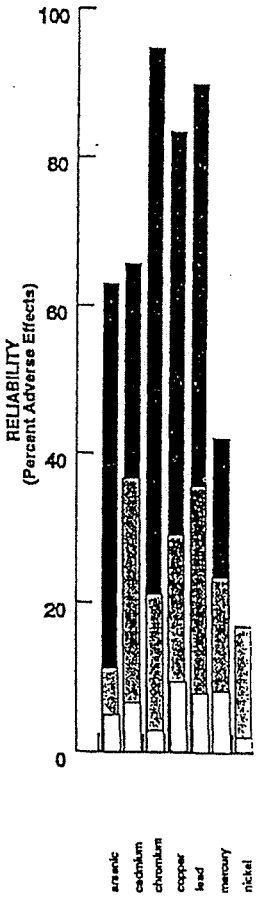
The reliability of both the marine ERL and ERM values (reported by Long *et al.*, 1995) and of marine TEL and PEL values (reported by MacDonald *et al.*, 1996) are summarized in Figures 1 and 2. The percentage of study endpoints showing adverse effects at concentrations less than the marine ERLs ranged from 2% for nickel to 27% for fluorene (Figure 1). For most substances the incidence of effects was less than 15% when concentrations were less than the ERL. The percentages of studies indicating effects with concentrations less than the TEL values were slightly lower than those indicated with the ERL values (Figure 2). For most substances, the incidence of effects ranged from 0% (2-methylnaphthalene) to 16% (total PCBs). An exception was total DDTs; 48% of the studies showed adverse effects at concentrations below the TEL concentrations, and the incidence of effects decreased to 26% at concentrations between the TEL and PEL.

The incidence of study endpoints indicating adverse effects generally increased to 30 to 50% when chemical concentrations were between the ERL and ERM values (Figure 1). For most substances, the incidence of effects increased to 15 to 25% when concentrations exceeded the TELs, but were below the PELs (Figure 2).

Among studies in which chemical concentrations exceeded the ERM values by any amount, the incidence of effects generally ranged from 75 to 100%, indicating relatively high reliability for most substances (Figure 1). When concentrations exceeded the PEL values, adverse effects generally were indicated in 55 to 80% of studies (Figure 2). However, the ERMs and PELs for several substances — notably, chlordane, lindane, nickel, and mercury — were less reliable than those for other substances. Also, the data for total DDT showed relatively high variability.

The percentages of studies indicating adverse effects generally were slightly lower for the TEL and PEL values than for the comparable ERL and ERM values. These small differences were probably attributable to differences in the derivation methods. That is, calculations of TELs and PELs included data from endpoints in which no adverse effects were observed, whereas the derivation of ERLs and ERMs involved the use of effects data only

The saltwater SQGs are comparable to those derived with similar empirical methods, but different data bases, for freshwater sediments (Persaud *et al.*, 1992; Long *et al.*, 1995; Ingersoll *et al.*, 1996). The estimates of the reliability of saltwater SQGs also are similar to those obtained with freshwater SQGs (Smith *et al.*, 1996; Ingersoll *et al.*, 1996). The overall similarity in both the numerical values and the reliability of saltwater and freshwater SQGs suggests that the underlying empirical methods provide a consistent basis for determining chemical concentrations that are likely to be associated with adverse biological effects.



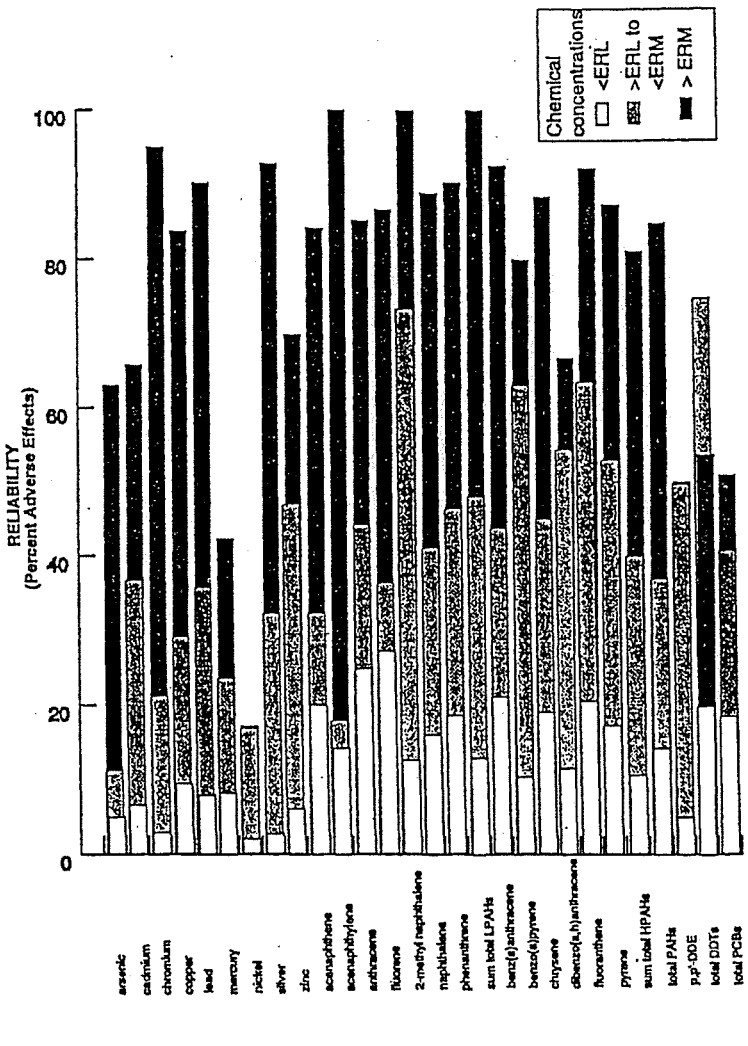


Figure 1. Reliability of marine ERL and ERM values as percentages of study end-points indicating adverse biological effects at concentrations less than the ERLs, equal to or greater than the ERLs but less than the ERMs, and equal to or greater than the ERMs. (From Long *et al.*, 1995.)

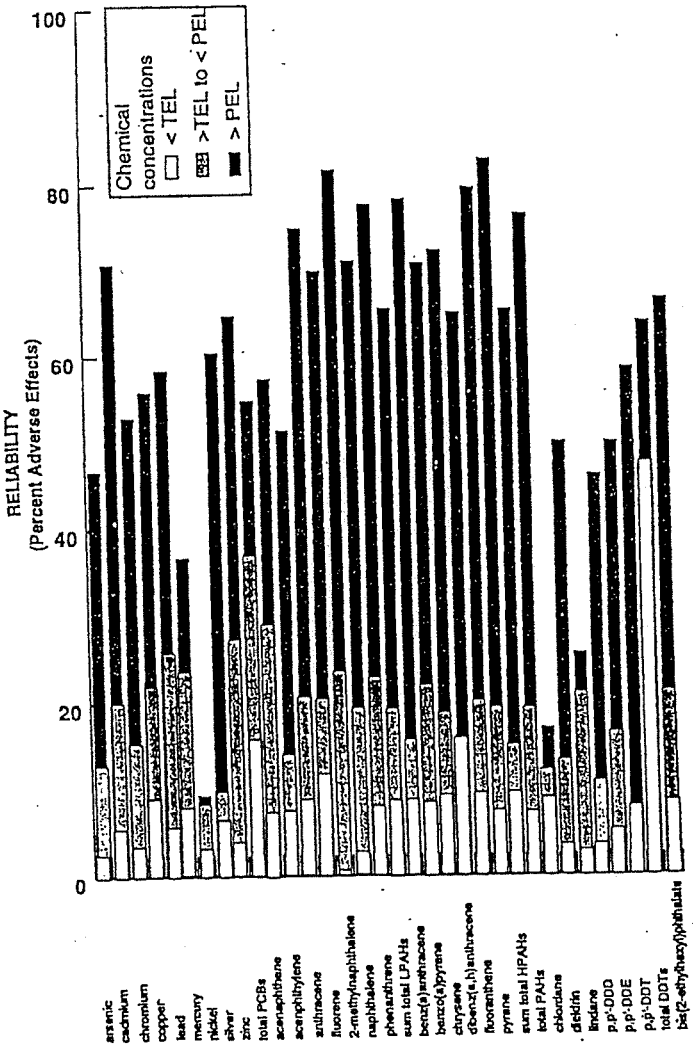


Figure 2. Reliability of marine TEL and PEL values as percentages of study end-points indicating adverse biological effects at concentrations less than the TELs, equal to or greater than the TELs but less than the PELs, and equal to or greater than the PELs (From MacDonald *et al.*, 1996.)

Predictive Ability of SQ

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Predictive Ability of SQGs.

Following the analyses of reliability, additional evaluations were conducted to estimate the predictive ability of saltwater SQGs with a large independent data set not used in the guidelines derivation (Long *et al.*, 1998a). Data were compiled from studies performed along portions of the Atlantic, Gulf, and Pacific coasts. The data set was composed of samples in which amphipod survival tests were performed (n = 1068) and in which one to three sublethal bioassays were performed in addition to the amphipod tests (n = 437). Statistical analyses were used to classify samples as either non-toxic ($p > 0.05$), marginally toxic ($p < 0.05$), or highly toxic ($p < 0.05$ and sample means exceed minimum significant differences) relative to controls in the laboratory tests. The predictive abilities of 28 sets of ERLs/ERMs and 33 sets of TELs/PELs were determined.

For most substances, 40 to 60% of samples in which chemical concentrations exceeded individual ERMs or PELs were highly toxic in the amphipod tests (Figures 3 and 4). For example, among the samples in which copper concentrations exceeded the ERM value (n = 25) or the PEL value (n = 179), 52 and 48%, respectively, were highly toxic in the amphipod survival tests. More than 75% of samples were highly toxic in which the ERMs for lead, 2-methylnaphthalene, and acenaphthylene were exceeded. The predictive abilities of the PELs for p,p'-DDE, total PCBs, and lindane were lowest; 33, 39, and 15%, respectively, in amphipod tests. For most substances, an increase in predictive ability of about 20 to 30% occurred when the data from the sublethal tests were included along with the amphipod data. Therefore, for most substances, 80 to 90% of samples were highly toxic in at least one of the tests performed when concentrations exceeded individual ERMs or PELs. For most chemicals, the ERMs and PELs were similar in predictive ability.

To account for the presence of mixtures of chemicals in different concentrations that may have additive toxicity effects, mean ERM and mean PEL quotients were derived as the average of the ratios between the chemical concentrations in the samples and the respective ERM or PEL values (Carr *et al.*, 1996; Long *et al.*, 1998a). The mean ERM quotients corresponded very well with the percentages of the ERMs that were exceeded and with the incidence of toxicity in amphipod tests (Figures 5 and 6). The mean ERM quotients began to exceed 1.0 among samples in which 12 (50%) or more of the 25 ERMs were exceeded (Figure 5). Also, the incidence of highly toxic samples increased to >71% among samples with mean ERM quotients of 1.0 or greater (Long *et al.*, 1998a). The associations between the number of PELs exceeded, the mean PEL quotients, and the incidence of toxicity were similar to those observed for the ERMs (Figure 6); the incidence of toxicity in amphipod tests exceeded 68% among samples with mean PEL quotients of 1.6 or greater (Long *et al.*, 1998a). These data suggest that toxicity increased coincidentally with increases in both the number of guidelines exceeded and with the chemical concentrations of the mixtures of substances in bulk sediments.

MacDonald *et al.*, 1996.)

Figure 3. Predictive ability of 28 ERMs as percentages of samples indicating highly significant toxicity, *i.e.*, $p < 0.05$ and mean survival <80% of controls (data from Long *et al.*, 1998a). Dark bars indicate results from amphipod survival tests performed with either *Ampelisca abdita* or *Rhepoxynius abronius*. Open bars indicate results from any of two to four tests performed, including amphipod tests (see Table 1 for types of other tests). Chemical abbreviations as follows: Cd = cadmium; Cr = chromium; Cu = copper; Pb = lead; Hg = mercury; Ni = nickel; Ag = silver; Zn = zinc; 2MN = 2-methylnaphthalene; DBA = dibenz*(a,h)*anthracene; ACE = acenaphthene; ACY = acenaphthylene; ANT = anthracene; BAA = benz*(a)*anthracene; BAP = benzo*(a)*pyrene; CHR = chrysene; FLU = fluoranthene; FLO = fluorene; NAP = naphthalene; PHN = phenanthrene; PYR = pyrene; LPAH = sum of low molecular weight PAH; HPAH = sum of high molecular weight PAH; tPAH = sum of PAHs; DDE = *p,p'*-DDE; tDDT = sum of six DDT isomers; tPCB = sum of 20 PCB congeners.

PAH; tPAH = sum of PAHs, DDE = *p,p*-DDE, tDDT = sum of six DDT isomers, tPCB = sum of 26 PCB congeners.

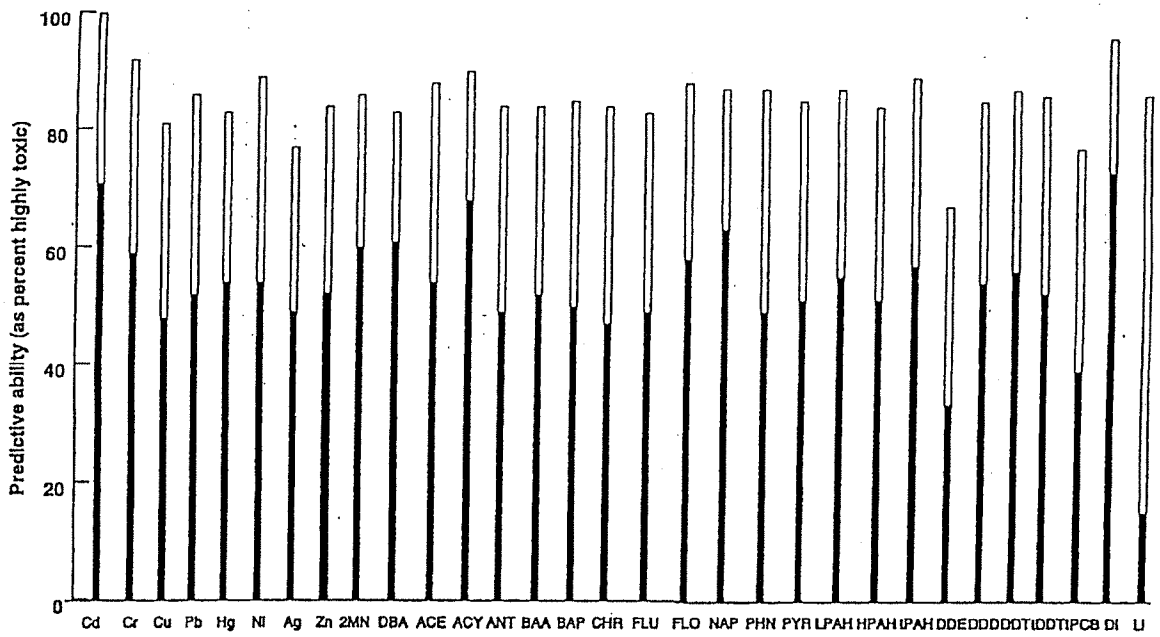

Figure 4. Predictive ability of 32 PELs as percentages of samples indicating highly significant toxicity, *i.e.*, $p < 0.05$ and mean survival <80% of controls (data from Long *et al.*, 1998a). Dark bars indicate results from amphipod survival tests performed with either *Ampelisca abdita* or *Rhepoxynius abronius*. Open bars indicate results from any of two to four tests performed, including amphipod tests (see Table 1 for types of other tests). Chemical abbreviations as in Figure 3, plus: DDD = *p,p'*-DDD; DDT = *p,p'*-DDT; Di = dieldrin; and Li = lindane.

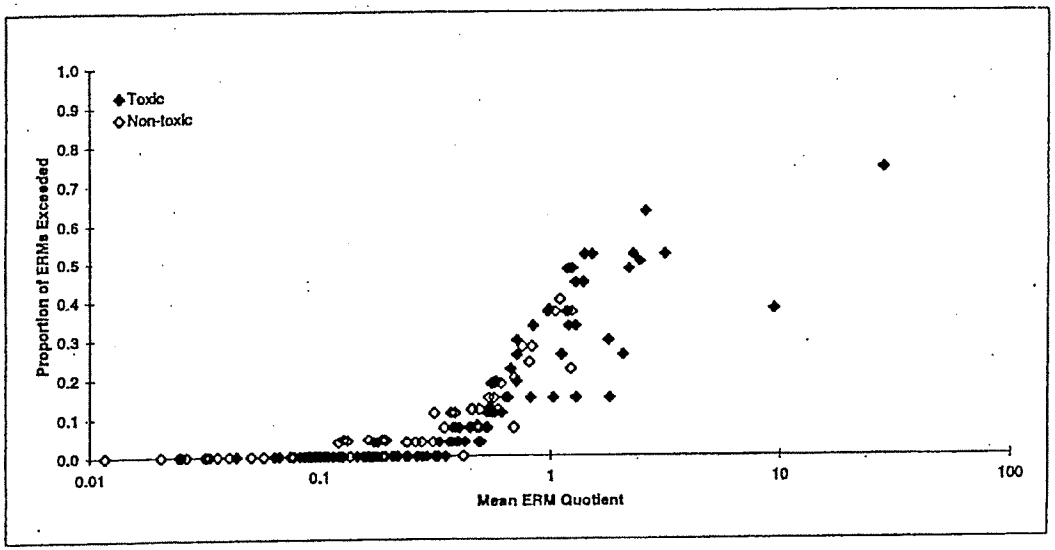

Figure 5. Relationship between the proportion of marine ERMs exceeded and mean ERM quotients. Samples that were toxic in amphipod (*Ampelisca abdita*) survival tests ($p < 0.05$) shown as closed symbols; non-toxic samples ($p > 0.05$) shown as open symbols (n = 212).

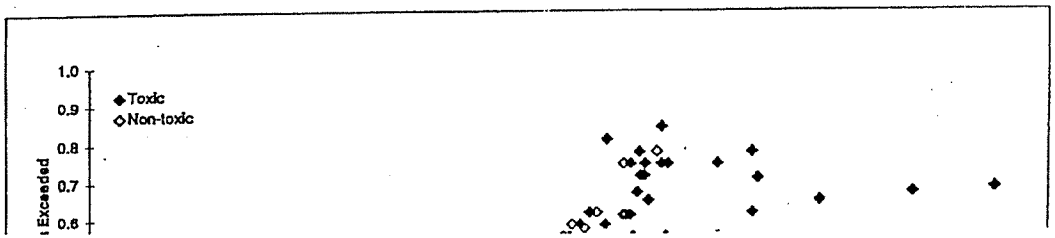

Figure 6. Relationship between the proportion of marine PELs exceeded and mean PEL quotients (n = 212). Samples that were toxic in amphipod (*Ampelisca abdita*) survival tests ($p < 0.05$) shown as closed symbols; non-toxic samples ($p > 0.05$) shown as open symbols.

Estimating the Probabilities of Toxicity with SQGs

Among the samples in which chemical concentrations were < all ERLs or < all TELs, 11 and 9%, respectively, were highly toxic in amphipod survival tests (Table 1). The percentages of samples that were highly toxic when mean ERM quotients and mean PEL quotients were <0.1 were similar: 12 and 10%, respectively (Long *et al.*, 1998a). Therefore, sediment samples in which no ERLs or TELs are exceeded and/or in which the mean SQG quotients are less than 0.1 have the lowest probabilties of highly significant toxicity in amphipod survival tests (9 to 12%).

Chemical characteristics that correspond to the lowest probabilities (9 to 12%), medium-low (24 to 32%), medium-high (46 to 53%), and highest probabilities (74 to 88%) of toxicity in amphipod survival tests are listed in Table 2. Data are shown for both the numbers of chemicals exceeding SQGs and for mean SQG quotients. We recommend these data be used to estimate the likelihood of observing acute toxicity in newly collected marine and saltwater sediments.

The ERLs and mean ERM quotients for saltwater were more efficient at correctly predicting non-toxicity (100 and 93% correct, respectively) than SEM:AVS ratios (80% correct) based upon analyses of data compiled to field-validate the SEM:AVS criteria (Long *et al.*, 1998b). Also, the ERMs and mean ERM quotients were slightly more predictive of toxic conditions (33 and 42% correct, respectively) than the SEM:AVS ratios (26% correct). These data suggest that the predictive ability of SQGs based upon bulk trace metals concentrations is not improved with SEM-to-AVS normalizations (Long *et al.*, 1998b).

The large data set evaluated suggests that some variability exists in the relationships between bulk sediment chemical concentrations and measures of toxicity. However, this variability is equivalent to that observed with the predictive ability of the SEM:AVS ratios (Hansen *et al.*, 1996) and may have been, at least partly, attributable to the uneven distribution of the chemistry data (*i.e.*, too few data points in some concentration ranges). Also, large differences in the physical and geochemical properties of the sediments may have contributed to this variability. Therefore, although the SGQs are not perfect (100%) predictors of non-toxicity and toxicity, it appears that they provide reasonable estimates of the likelihood of toxicity. For example, about 50% of samples were either marginally or highly toxic in acute amphipod survival tests when one or more saltwater ERMs or PELs were equalled or exceeded. Also, about 86% of samples with one or more ERMs or PELs exceeded were either marginally or highly toxic in at least one of a battery of two to four tests performed (Table 1).

The probabilties of observing toxicity generally increased with increases in both the numbers of SQGs exceeded and in mean ERM and PEL quotients (Table 2). Therefore, these data suggest that chemical concentrations normalized to dry wt. can be used to estimate the probability of toxicity. Consequently, we believe that empirically derived SQGs can be used to evaluate the potential

Table 1. Summary of predictive ability[a] of marine ERL/ERM and TEL/PEL values in amphipod survival tests only and in any of 2-4 tests performed (expressed as % of samples indicating toxicity).

Toxicity tests categorized as either marginally toxic (p<0.05 and mean response >80% of controls) or highly toxic (p<0.05 and mean response <80% of controls) and total of both. Amphipod tests performed with Ampelisca abdita and Rhepoxynius abronius. Other tests included sea urchin fertilization (Arbacia punctulata, Strongylocentrotus purpuratus) in pore-water and mollusc embryo development (Mulinia lateralis or Haliotis rufescens in pore-water) and microbial bioluminescence (Microtox) in solvent extracts.

Table 1. Summary of predictive ability* of marine ERL/ERM and TEL/PEL values in amphipod survival tests only and in any of 2-4 tests performed (expressed as % of samples indicating toxicity).
Toxicity tests categorized as either marginally toxic (p<0.05 and mean response >80% of controls) or highly toxic (p<0.05 and mean response <80% of controls) and total of both. Amphipod tests performed with Ampelisca abdita and Rhepoxynius abronius. Other tests included sea urchin fertilization (Arbacia punctulata, Strongylocentrotus purpuratus) in pore-water and mollusc embryo development (Mulinia lateralis in elutriates or Haliotis rufescens in pore-water) and microbial bioluminescence (Microtox) in solvent extracts.

Chemical concentrations	Amphipod tests only				Any test performed			
	n	marginally toxic	highly toxic	Total toxicity	n	marginally toxic	highly toxic	Total toxicity
< all ERLs	329	21	11	32	39	13	41	54
> 1 or more ERLs	448	20	18	38	173	16	64	80
> 1 or more ERMs	291	13	39	52	225	8	78	86
< all TELs	233	26	9	35	26	15	23	38
> 1 or more TELs	450	19	16	35	116	17	60	77
> 1 or more PELs	385	13	35	48	295	9	77	86

*Data from Long et al., 1998a

Table 2. Chemical characteristics and probabilities of highly significant toxicity in amphipod survival tests among four categories of sediment samples.

Site Categories	Chemical characteristics	Probability (%) of toxicity in amphipod survival tests*
Highest Priority Sites:		
	• mean ERM quotients >1.5	74% (INJURY / LOST SERVICES)
	• mean PEL quotients >2.3	76%
	• >10 ERMs exceeded	85%
	• >21 PELs exceeded	88%
Medium-high Priority Sites:		
	• mean ERM quotients 0.51-1.5	46%
	• mean PEL quotients 1.51-2.3	50%
	• 6-10 ERMs exceeded	52%
	• 6-20 PELs exceeded	53%
Medium-low Priority Sites:		
	• mean ERM quotients 0.11 to 0.5	30%
	• mean PEL quotients 0.11 to 1.5	25%
	• 1-5 ERMs exceeded	32%
	• 1-5 PELs exceeded	24%
Lowest Priority Sites:		
	• mean ERM quotients <0.1	12%
	• mean PEL quotients <0.1	10%
	• no ERLs exceeded	11%
	• no TELs exceeded	9%

* Data from Long et al., 1998a

toxicological significance of bulk sediment chemical concentrations. However, because of the variability observed in the relationships between chemical concentrations and toxicity among samples with intermediate concentrations (*e.g.*, ERLs exceeded but no ERMs exceeded or mean ERM quotients of 0.1 to 1.0), adverse effects should be verified in samples with intermediate probabilities of toxicity by conducting toxicity tests and/or other biological analyses.

RECOMMENDED APPLICATIONS OF EMPIRICALLY DERIVED SQGS

Both sets of guidelines were intended to be used as informal (*i.e.*, non-regulatory) tools to assist in the interpretation of chemical data for sediments. Primarily, both sets of values were intended to be used to evaluate and rank both sites and chemicals of potential concern.

Evaluating Sites of Concern

Recently, a panel of environmental chemists and toxicologists concluded "that there is sufficient certainty associated with SQGs to recommend their use in sediment ecological risk assessments" (Ingersoll *et al.*, 1997). Consistent

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with this observation, we recommend that SQGs can be used to evaluate and categorize the relative quality of sediments on both an individual-sample basis as well as within regions within which multiple samples were collected. In addition, they can be used to categorize sites in terms of the relative probabilities of observing toxicity. Data from regional, ambient monitoring programs can be evaluated to identify which sites, if any, have chemical concentrations that exceed the SQGs and the percentage of sites that exceeded them. If ambient monitoring is performed with stratified-random sampling designs, the surficial extent of toxicity (expressed as area and percent of total area) can be calculated. Data from age-dated cores or time-series sampling can be used to identify temporal trends in sediment quality by using the SQGs. The guidelines can be used to compare and rank the average quality of sediments among basins, waterways or other mid- to large-scale geographic regions. The chemical concentrations in sediments influenced by point sources can be compared to the SQGs and to the chemical characteristics of the effluents to determine if source control measures should be considered for protecting and/or restoring sediment quality.

Data from investigations of hazardous waste sites can be compared to the SQGs to aid in ranking and prioritizing sites and to determine the need for further information to support management decisions. The SQGs can be used in ecological risk assessments at these sites to estimate the potential for adverse biological damage. Ecological risk assessments of sediments are most comprehensive when all three components of the sediment quality triad are included in the approach (Chapman, 1996). Tiered analyses of chemical data from areas to be dredged may be aided by use of the SQGs, especially when accompanied by measures of bioavailability and toxicity.

In all of these applications, sediments in which none of the chemicals equal or exceed any low-range SQGs (ERLs or TELs) should be classified as having the lowest potential for adverse effects on sediment-dwelling organisms, and, therefore, as being the least contaminated (Table 2). Also, if these chemical concentrations are within the expected bounds for background sediments based upon ratios to grain size, aluminum, lithium or other geochemical factors (Schropp *et al.*, 1991), there would be further evidence that the samples are among the least contaminated. However, as indicated by the false negative rates of about 9 to 12% in acute toxicity tests for the TELs and ERLs, there remains some possibility of toxicity due to the presence of substances not quantified in chemical analyses and/or for which there are no applicable SQGs. Furthermore, the probabilities of toxicity in any of a battery of sublethal tests remains about 30 to 50% among samples with these characteristics. The absence of toxicity can be absolutely assured only by performing confirmatory toxicity tests and/or other biological analyses. Sampling areas in which all chemical concentrations are lower than SQGs and in which toxicity is not observed could qualify as reference areas (Long and Wilson, 1997).

The reliability of the ERMs and PELs for nickel, mercury, and DDT were relatively low when compared with the other SQGs. However, the predictive

abilities of these values were similar to those for other substances. Nevertheless, we recommend that sediments in which only these SQGs are exceeded should not be viewed as high priority unless confirmed as such with additional studies, such as multiple toxicity tests, toxicity identification evaluations or sediment-spiking experiments. Studies performed on data sets selected to focus specifically upon toxicity attributable to concentrations of total DDTs in sediments suggest that the reliability of an SQG of 7.12 ppm is much higher — over 95% (MacDonald, 1997).

The ERL and TEL values were not intended to be used as tools for predicting adverse biological effects. Therefore, samples in which chemical concentrations exceed low-range guidelines, but none of the mid-range values, should be viewed as medium-low or medium-high in priority (Table 2). Often, chemical concentrations between the ERLs and ERMs and between the TELs and PELs represented ranges in which there was a transition from principally non-toxic conditions to mainly toxic conditions. Actual toxicity may be highly influenced in these concentration ranges by a number of geochemical factors and by the sensitivity of the toxicity tests.

Toxicity cannot be necessarily expected in sediments in which only a single ERM or PEL was exceeded because these SQGs were not intended as toxicity thresholds or absolute predictors of toxicity. Those samples in which, say, one to five ERMs or PELs are exceeded should be viewed as medium-low priority because there is a probability of about 24 to 32% that the samples would be highly toxic in acute amphipod tests. Samples in which 11 or more ERMs or 21 or more PELs are exceeded have 85 to 88% probabilities of being highly toxic to amphipods and, therefore, should be considered as highest priority. We have established that the number of ERMs and PELs exceeded corresponds very well with the mean ERM and PEL quotients, therefore, comparisons among samples and among areas may be based upon either or both of these sets of cumulative indices and used to estimate the probabilities of observing toxicity.

Evaluations of Chemicals of Potential Concern

The SQGs can be used effectively to identify, rank, and prioritize chemicals of potential toxicological concern by, first comparing the chemical concentrations in test samples to the SQGs, and then comparing the numbers of SQGs exceeded and the mean SQG quotients to the probabilities of observing toxicity (Table 2). Those substances that occur at concentrations below the low-range SQGs and remain low in concentrations in both toxic and non-toxic samples should be identified as relatively low-priority chemicals. Those substances whose concentrations exceed the low-range SQGs, but do not equal or exceed the mid-range SQGs should be ranked as chemicals of low-to-moderate concern. Substances that equal the mid-range SQGs or exceed them by a moderate amount (*e.g.*, factors of 1.1 to 9.9) should be viewed as chemicals of high concern. Chemicals that most frequently exceed the mid-range SQGs and by the greatest amount (*e.g.*, factor ≥10) should be viewed as chemicals of

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highest concern, especially if they exceed all SQGs in samples that are highly toxic.

If results of toxicity tests are available, those substances that show coincident patterns of high concentrations in the most toxic samples should be identified as chemicals of highest concern. These patterns of concordance can be demonstrated with correlation statistics and regression plots (Carr *et al.*, 1996). Some chemicals may appear ostensibly as high-priority substances (*i.e.*, they occur in concentrations above the ERLs and/or equal to the ERMs), but, unless it can be shown that their concentrations are coincident with toxicity or other adverse effects, they should not be ranked as chemicals of highest concern.

In evaluations of chemicals of concern, users of the SQGs must take into account the relative reliability and predictive ability of individual guidelines. The reliability of the ERMs and PELs for nickel and mercury, for example, were relatively low compared to those for most other substances (Long et al., 1995; MacDonald et al., 1996). In contrast, the probability of observing toxicity in samples with concentrations of, for example, PAHs that exceeded the SQGs would be relatively high. The probabilities of observing toxicity in samples that exceed only the SQGs for nickel would be much lower.

Regardless of the degree to which chemical concentrations exceed any SQGs, there is not 100% certainty that samples in which SQGs are exceeded will actually be toxic. Toxicity and other adverse effects in sediments are functions of the total concentration and bioavailability of the sediment-associated chemicals. Chemical form or speciation (where applicable), sensitivity and appropriateness of the biological assay, and exposure times also are important factors that influence toxicity. It is important to understand that the SQGs do not provide a direct means of determining which substances are responsible for causing toxicity in sediments. However, SQGs can assist in identifying the chemicals that are most likely to contribute to toxicity, particularly when used in conjunction with other tools. Toxicity identification evaluations and sediment spiking experiments can be performed to determine which chemical(s) actually caused or contributed to toxicity. In this application, the SQGs provide a basis for developing a short list of potentially toxic substances and therefore a means of focusing additional confirmatory studies.

A variety of SQGs can be used to rank and prioritize chemicals in samples. The choice of whether to use the ERL/TEL or ERM/PEL values or others should be predicated upon the probability of toxicity (*i.e.*, level of risk) that is considered tolerable or acceptable. If a relatively low level of risk to sediment-dwelling organisms is tolerable, ERL/TEL values should be used. However, they should be used with an understanding that concentrations exceeding these values by minor amounts are likely to be associated with relatively low probabilities of toxicity. If a higher level of risk is tolerable, then the ERM/PEL values should be used to identify chemicals of potential concern. We suggest that users of the SQGs primarily focus upon the mid-range (ERM and PEL) SQGs as benchmarks for effects and the low-range (ERL and TEL) values as benchmarks for no-effects. Because the probabilities of effects are much

higher when concentrations exceed the ERMs and PELs than when they exceed only the ERLs and TELs, the former would be expected to be more indicative of adverse effects than the latter.

Despite the demonstrated reliability and predictive ability of the SQGs, there is still some uncertainty associated with their use. There are no SQGs for many substances that can be toxic in sediments. In addition, the SQGs were intended to be applicable to acute toxicity endpoints; their applicability to toxicological risks to wildlife and humans through bioaccumulation pathways is unknown. Although data assembled to evaluate predictive ability often indicated that many toxicants occurred together and co-varied in concentrations, the SQGs should not be assumed to be applicable to chemicals other than those for which the SQGs were derived.

It is important to apply the guidelines only to soft, aqueous, sedimentary deposits that include fine-grained particles. The SQGs are not intended to be used for assessing upland soils, or atypical sediments such as gravel, cobble, slag, paint chips, or tar. Applications of the SQGs in such circumstances may result in erroneous conclusions.

CONCLUSIONS

The reliability and predictive ability of two sets of SQGs — the ERLs/ERMs and the TELs/PELs — were summarized to support recommendations on the applications of these sediment assessment tools. The results of these evaluations showed that the ERLs and TELs provide reliable and predictive tools for identifying the concentrations of chemicals in sediments that are unlikely to be associated with adverse biological effects. That is, the incidence of highly significant toxicity in amphipod survival tests is about 10% in sediments in which none of the guidelines are exceeded. The ERM and PEL values are the chemical concentrations above which there is a relatively high likelihood of toxicity (predictive ability of 86%) in at least one of a battery of tests conducted with sensitive, sediment-dwelling organisms. Mean ERM — and PEL— quotients provide additional tools for assessing the quality of sediments in which there are complex mixtures of substances. The evaluations of reliability and predictive ability suggest that the guidelines provide a scientifically defensible basis for assessing the quality of soft sediments in marine and estuarine ecosystems.

The SQGs provide an effective basis for ranking or prioritizing areas of concern with respect to sediment contamination. Because the probability of observing toxicity in amphipod survival tests is in the order of 74 to 88% in sediments in which multiple SQGs are exceeded (>10 ERMs or >21 PELs) or in which mean SQG quotients are high (*i.e.*, >1.5 for ERMs or >2.3 for PELs); a high level of priority should be assigned to sediments with such chemical characteristics. Sediments should be assigned the lowest priority when none of the low-range SQGs are exceeded or when mean SQG quotients are lowest (*i.e.*, <0.1 for both ERMs and PELs). Areas with intermediate chemical concentrations can be assigned priorities in accordance with the probabilities of

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observing toxicity or other adverse effects based upon the numbers of chemicals exceeding SQGs or the calculated mean SQG quotients. However, the actual toxicity of sediments with intermediate chemical concentrations may be influenced by a variety of factors, such as contaminant bioavailability, presence of un-measured substances, presence of substances for which there are no SQGs, and other confounding variables. Therefore, we recommend that other assessment tools, such as toxicity tests, bioaccumulation analyses, and benthic invertebrate community analyses, be used to further evaluate the quality of these sediments.

The SQGs also provide a means of identifying chemicals of potential concern in marine and estuarine sediments. Those substances that occur at concentrations below the low-range SQGs, either in individual samples or in multiple samples within a study area, should be classified as lowest priority. Those chemicals that occur at concentrations exceeding background concentrations, both the ERMs and PELs, and other relevant SQGs or criteria should be considered as relatively high priority, particularly if the concentrations of these substances are significantly correlated with measures of effects. However, care should be exercised when using the SQGs to identify the contaminants that are actually causing toxicity in sediments with complex mixtures of chemicals. In these cases, we recommend the use of spiked sediment bioassays, and/or toxicity identification evaluations to confirm which chemicals actually warrant the greatest concern.

Although empirically derived SQGs have been shown to be both reliable and predictive of toxicity, we recommend that sediment management decisions be based upon a weight of evidence that reflects the significance and expense of the decisions. Therefore, the SQGs should be used in conjunction with other tools within an integrated framework for assessing sediment quality.

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PREDICTING TOXICITY IN MARINE SEDIMENTS WITH NUMERICAL SEDIMENT QUALITY GUIDELINES

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Abstract—Matching synoptically collected chemical and laboratory bioassay data (n = 1,068) were compiled from analyses of surficial sediment samples collected during 1990 to 1993 to evaluate the predictive ability of sediment quality guidelines (SQGs), specifically, effects range—low (ERL), effects range—median (ERM), threshold effects level (TEL), and probable effects level (PEL) values. Data were acquired from surveys of sediment quality performed in estuaries along the Atlantic, Pacific, and Gulf of Mexico coasts. Samples were classified as either nontoxic ($p > 0.05$ relative to controls), marginally toxic ($p < 0.05$ only), or highly toxic ($p < 0.05$ and response greater than minimum significant difference relative to controls). This analysis indicated that, when not exceeded, the ERLs and TELs were highly predictive of nontoxicity. The percentages of samples that were highly toxic generally increased with increasing numbers of guidelines (particularly the ERMs and PELs) that were exceeded. Also, the incidence of toxicity increased with increases in concentrations of mixtures of chemicals normalized to (divided by) the SQGs. The ERMs and PELs indicated high predictive ability in samples in which many substances exceeded these concentrations. Suggestions are provided on the uses of these estimates of the predictive ability of sediment guidelines.

Keywords—Sediment quality guidelines Predictive ability Laboratory toxicity tests

INTRODUCTION

Using similar empirical approaches, sediment quality guidelines (SQGs) were prepared for salt water [1–3] and freshwater [4,5] as informal (nonregulatory) benchmarks to aid in the interpretation of sediment chemistry data. For marine sediments, effects range—low (ERL) and effects range—median (ERM) concentrations for 9 trace metals, 3 chlorinated organics, and 13 polynuclear aromatic hydrocarbons (PAHs) were identified [1]. Threshold effects level (TEL) and probable effects level (PEL) concentrations for 9 trace metals, 8 chlorinated organics, 1 phthalate, and 13 PAHs were published [2]. These guidelines were not based upon experiments in which causality was determined. Rather, both sets of marine guidelines were based upon empirical analyses of data compiled from numerous field and laboratory studies performed in many estuaries and bays of North America. These studies included chemistry data and a variety of different types of biological data for numerous taxa derived from either bioassays of field-collected samples, laboratory toxicity tests of clean sediments spiked with specific toxicants, benthic community analyses, or equilibrium-partitioning models.

The objectives of the ERL and TEL values and of the ERM and PEL values were comparable. The ERLs and TELs were intended to represent chemical concentrations toward the low end of the effects ranges, that is, below which adverse biological effects were rarely observed. The ERMs and PELs were intended to represent concentrations toward the middle of the effects ranges and above which effects were more frequently observed. As estimates of reliability, the incidence of adverse effects within concentration ranges defined by these SQGs were determined using data with which they were derived [1,2]. Generally, adverse effects occurred in less than 10% of studies in which concentrations were below the respective ERL or TEL values and were observed in more than 75% or 50% of studies in which concentrations exceeded the ERMs or PELs, respectively.

Since they were published, the guidelines [1,2] have been used as interpretive tools in many sediment assessments throughout North America and elsewhere. Generally, the ERLs and TELs have been used to identify relatively uncontaminated samples that pose a limited risk of toxicity. The ERMs and PELs have been used to identify those samples and areas in which chemical concentrations were sufficiently elevated to warrant further evaluation. Because these guidelines were based upon analyses of large databases, mostly composed of field-collected data in which mixtures of toxicants were encountered, it was assumed [1,2] that the guidelines would provide relatively accurate tools for classifying newly collected samples as potentially toxic or nontoxic. Thus far, however, the accuracy of the two sets of guidelines in predicting nontoxic and toxic conditions correctly has not been evaluated. Therefore, because of the widespread use of these guidelines, we concluded there was a need for analyses of their predictive ability with data independent of those with which the SQGs were derived.

The objectives of this paper are to quantify the frequency with which ERL/ERM and TEL/PEL guidelines correctly classify samples as either nontoxic or toxic; to quantify the incidence of toxicity among samples in which different numbers of SQGs were exceeded; to determine the incidence of toxicity

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Table 1. Sources of data and the toxicity tests performed in each study

Survey area	Year sampled	No. samples	Bioassays performed: Amphipod survival	Clam embryo survival	Clam embryo development	Microbial bioluminescence	Urchin egg fertilization	Urchin embryo development	Abalone embryo development
Hudson–Raritan estuary	1991	38	X	X	X	X			
Newark Bay	1993	20	X						
Long Island Sound	1991	63	X	X	X	X			
Boston Harbor	1993	30	X			X	X		
Tampa Bay phase 1	1992	16	X			X	X		
Tampa Bay phase 2	1993	45	X				X		
San Diego Bay	1993	121	X					X	
San Pedro Bay	1992	45	X						X
Charleston Harbor	1993	79	X			X	X		
EMAP—Estuaries[a]	1990–1992	611	X						
Total		1,068							

[a] EMAP = Environmental Monitoring and Assessment Program; data from mysid tests not included.

to the SQGs; and to compare the relative predictive ability of the two sets of guidelines. The design followed that of a previous study in freshwater [5] in which type I and type II errors were determined for ERL/ERM and TEL/PEL values. Type I errors (false positives) are those in which toxicity was expected (based upon high chemical concentrations), but was not observed. Type II errors (false negatives) are those in which no toxicity was expected (low chemical concentrations), but was actually observed.

METHODS

Matching, synoptically collected, sediment chemistry and bioassay data for 1,068 samples were compiled from studies performed by the National Oceanic and Atmospheric Administration (NOAA) and U.S. Environmental Protection Agency (U.S. EPA) (Table 1). Regional sediment quality assessments were conducted as a part of NOAA's National Status and Trends Program (NS&TP) and included those performed in (all in the USA) the Hudson–Raritan estuary in New York and New Jersey [6], Newark Bay in New Jersey [6], the bays adjoining Long Island Sound in New York and Connecticut [7], Boston Harbor in Massachusetts [8], Tampa Bay in Florida [9], San Diego Bay [10] and San Pedro Bay [11] in southern California, and Charleston Harbor in South Carolina (unpublished). The U.S. EPA data were generated in Environmental Monitoring and Assessment Program (EMAP) studies of the Virginian and Louisianian estuarine provinces [12–14].

All of these data were generated during surveys performed to quantify the spatial extent, patterns, and severity of adverse biological effects attributable to toxic substances. Samples from the upper 2 to 3 cm of the sediments were collected with grab samplers throughout each survey area to characterize surficial sediment contamination and toxicity. Data for these analyses were selected because they were generated with similar protocols, included matching chemistry and toxicity results, indicated a range in toxicity responses, and represented conditions from all three coastlines.

Sample collection and handling methods, toxicity testing methods, chemical analytical protocols, and raw data are included in the respective technical reports. All analytical laboratories followed the performance-based protocols of the NS&TP and EMAP—Estuaries to ensure comparability among

rials [16,17] for the amphipod survival tests, U.S. National Biological Service [18] for the urchin tests, and U.S. EPA [19] and Schiewe et al. [20] for the Microtox® tests (AZUR Environmental, Carlsbad, CA, USA). All bioassay data were expressed as percent of negative, laboratory controls (not reference samples) to account for variability among studies and laboratories in organism viability.

We considered several different approaches to the classification of samples as either nontoxic or toxic. In an interlaboratory comparison of performance, results of amphipod survival tests were classified as either nontoxic (mean survival 96–96.5%), marginally toxic (mean survival 76.5–83%), clearly toxic (mean survival < 76%), highly toxic (mean survival < 20%) [21]. Swartz et al. [22] classified results of amphipod survival tests as either not toxic (<13% mortality), uncertain (13–24% mortality), or toxic (>24% mortality). Statistical tests were recommended [16] to determine if differences in results of tests of field-collected samples and controls are statistically significant. An alternative approach [23], based upon results of power analyses of amphipod survival tests, recommended the use of minimum significant differences (MSDs) from controls as criteria for classifying samples as toxic.

We chose to use a combination of these approaches to classify samples. Following standardized procedures [16], samples in which test results were not statistically different from negative controls (i.e., $p > 0.05$) were classified as nontoxic and samples in which results were significantly different from controls were classified as toxic. However, to further distinguish differences in degrees of toxicity, sample classifications followed the recommendations of Thursby et al [23]. Samples in which test results were significant relative to controls, but were less than MSDs were labeled as marginally toxic and those in which results were both significant and greater than MSDs were labeled as highly toxic. The highly toxic label does not imply that toxicity was severe; rather, it was used to identify those results for which statistical certainty was greatest. The MSD values calculated and published for *Ampelisca abdita* [23] were used for all amphipod test results. The MSD values for Microtox tests [24], *Arbacia punctulata* fertilization tests [25], and and all other tests were determined empirically with power analyses of the frequency distributions of data from each test.

laboratory replicates is very small [23]. However, these samples could not be classified as nontoxic because they were significantly different from controls. Therefore, we chose to classify them separately as neither nontoxic nor highly toxic. Because of the uncertainty associated with marginally toxic results, this evaluation focuses mainly upon the nontoxic and highly toxic categories.

Following the completion of an electronic database, several analyses were performed to determine the predictive ability of the guidelines. In these analyses, the guidelines for nickel were excluded because of the low degree of reliability determined for these values [1,2]. Also, the sums of low- and high-molecular-weight PAHs and total PAHs were excluded to avoid redundancy with the data for individual compounds. In summations of total polychlorinated biphenyls (PCBs), total dichlorodiphenyltrichloroethanes (DDTs), and total PAHs, concentrations of individual compounds were treated as zeroes when they were below method detection limits (MDLs). The MDLs achieved differed slightly among laboratories; therefore, the use of zeroes minimized inconsistencies in data treatments. In any case the use of either one half of the MDL or zeroes had no effect upon classification of samples relative to the SQGs.

Three data analyses were performed. First, the predictive abilities of individual SQGs were determined. Second, the incidence of toxicity was determined among samples in which none of the substances equaled or exceeded the ERL concentrations; in which one or increasing numbers of substances exceeded ERL concentrations, but none exceeded any ERM; and in which one or increasing numbers of substances exceeded ERM concentrations. The same approach was used to evaluate the predictive ability of the TEL/PELs. We scored samples as exceeding SQGs when a chemical concentration either equaled the value or exceeded it by any amount.

In the third analysis, the incidence of toxicity over ranges in mean SQG quotients [5,25] was determined. The concentrations of individual chemicals were divided by their respective ERMs or PELs and the means of these concentration-to-SQG quotients were determined. The means of these quotients were determined to account for differences among studies in the numbers of chemicals for which analyses were performed. Predictive ability was calculated with samples classified as either nontoxic or highly toxic, excluding the marginally toxic results.

Similar to the criteria used to determine guideline reliability [1], we considered the guidelines to be predictive if the incidence of toxicity was less than 25% when all concentrations were less than the ERLs or TELs and greater than 75% when at least one concentration exceeded an ERM or PEL. Therefore, our target level for both false negatives and false positives was ≤25%.

Data are reported for the results of amphipod survival tests alone and for any one of the battery of two to four tests performed. In the latter analyses, samples were classified as marginally or highly toxic if one or more of the bioassays met the criteria for these classifications.

RESULTS

The database

Data were compiled from 1,068 samples analyzed during EMAP and NOAA studies conducted during 1990 to 1993. Roughly one third of the data were obtained from the NOAA from 20 to 121 (Table 1). The EMAP data from the Atlantic and Gulf coasts constituted the remaining two thirds of the database (n = 611).

Amphipod survival was determined for all samples; one to three additional tests were performed on all samples except those collected in the EMAP and Newark Bay studies (n = 437). The data from bioassays performed with mysids by the EMAP were not used because these tests failed to indicate toxicity. Amphipod survival was determined with *A. abdita* in Atlantic and Gulf coast surveys and with *Rhepoxynius abronius* in California surveys. Other tests included bivalve (*Mulinia lateralis*) embryo survival and development with exposures to elutriates; microbial bioluminescence (Microtox) in exposures to organic solvent extracts; and pore-water tests of echinoderm (*A. punctulata*) fertilization in Gulf and Atlantic coast areas, echinoderm (purple urchin, *Strongylocentrotus purpuratus*) embryo development in San Diego Bay, and embryological development of red abalone (*Haliotis rufescens*) embryos in San Pedro Bay. Insufficient numbers of samples were tested in any of these nonamphipod tests to warrant analyses alone; therefore, the results of these tests were combined.

The chemical data from each survey indicated that samples contained mixtures of contaminants, including trace metals, PAHs, and chlorinated hydrocarbons. The numbers of samples analyzed for each chemical ranged from 399 to 1,060 (Table 2). Analyte concentrations exceeded the MDL in a majority of the samples. The concentrations of most trace metals ranged over two to three orders of magnitude, and those of most organic compounds ranged over four to six orders of magnitude. Concentrations of the PAHs were most often less than the MDL.

None of the samples exceeded the ERM value for arsenic and <1.0% exceeded the ERMs for cadmium and chromium (Table 2). Relatively small proportions of the samples had chemical concentrations that exceeded ERM values, indicating that the data were not skewed toward waste sites with unusually high concentrations. Undoubtedly, some samples contained chemicals that were not quantified or for which there were no SQGs.

Among the different tests performed, 15 to 91% of the samples were at least marginally toxic (Table 3). Bioassay results showed a wide range of response, often from 0 to >100% of mean control responses. In the amphipod tests 36 to 52% of the samples were toxic whereas in the tests of pore water 56 to 91% of samples were toxic.

The frequency distributions of the data from most of the tests were similar, that is, responses in most samples were >80% of control responses (Table 3). Many of the EMAP samples were marginally toxic in amphipod tests. The data from embryological tests with the purple urchin (*S. purpuratus*) and red abalone (*H. rufescens*) indicated similar frequency distributions, both suggesting higher sensitivities to the samples than found in the amphipods. Empirically derived MSDs for each bioassay were very similar, ranging from 80 to 87%.

Incidence of toxicity

Concentrations greater than individual SQGs. Table 4 summarizes the percentages of samples that were not toxic, were marginally toxic, and were highly toxic in the amphipod tests alone and in any of the two to four tests performed when the concentrations of substances equaled or exceeded individ-

Table 2. Ranges in chemical concentrations, numbers of samples in which concentrations were less than or greater than method detection limits (MDLs), and percentages of samples in which effects range—median (ERM) values were exceeded

Chemical[a]	Units	No. samples	% > ERM[b]	No. > MDL	Range in detected concentrations		Range in concn. below detection limits[c]		No. < MDL
					Lowest	Highest	Lowest	Highest	
Arsenic	ppm	920	0.0	913	0.1	41	1.2	1.7	7
Cadmium	ppm	987	0.2	987	0.03	19.8	0.01	0.05	0
Chromium	ppm	1,058	0.5	1,045	1	1,220	1.2	18	13
Copper	ppm	1,057	2.4	1,031	0.7	1,770	0.2	1	26
Lead	ppm	1,052	3.4	1,038	1.4	510	0.3	1.3	14
Mercury	ppm	994	12.7	994	0.01	15	0.001	0.01	0
Nickel	ppm	1,042	2.1	1,006	0.3	136	0.2	1.7	36
Silver	ppm	919	4.4	866	0.01	10.1	0.01	0.7	53
Zinc	ppm	1,060	5.3	1,060	1	1,880	NA	NA	0
2-Methylnaphthalene	ppb	921	1.0	591	0.40	15,557	0.20	10	330
Dibenz[*a,h*]anthracene	ppb	399	11.8	363	0.40	4,534	0.70	10	36
Acenaphthene	ppb	977	3.3	394	0.10	56,338	0.10	80	583
Acenaphthylene	ppb	807	2.8	254	0.40	12,915	0.02	100	553
Anthracene	ppb	997	4.8	521	0.20	89,366	0.03	90	476
Benz[*a*]anthracene	ppb	996	7.2	652	0.30	59,298	0.02	130	344
Benzo[*a*]pyrene	ppb	980	10.0	631	0.20	54,862	0.02	170	349
Chrysene	ppb	997	5.5	688	0.20	60,331	0.10	130	309
Fluoranthene	ppb	1,000	4.2	755	0.30	108,236	0.20	110	245
Fluorene	ppb	945	3.2	530	0.10	54,209	0.10	90	415
Naphthalene	ppb	900	0.9	456	0.70	17,414	0.40	70	444
Phenanthrene	ppb	1,054	5.1	779	0.40	194,343	0.40	90	275
Pyrene	ppb	1,029	8.1	819	0.40	143,132	0.10	120	210
Total LMW PAHs	ppb	956	5.0	956	0.2	552,124	NA	NA	0
Total HMW PAHs	ppb	925	8.2	925	2	461,675	NA	NA	0
Total PAHs	ppb	1,003	1.1	1,003	0.2	1,013,799	NA	NA	0
p,p'-DDE	ppb	789	12.0	741	0.004	2,900	0.03	0.3	48
p,p'-DDD	ppb	742	No ERM	666	0.004	784	0.1	1	76
p,p'-DDT	ppb	656	No ERM	543	0.004	3,517	0.02	1	113
Total DDTs	ppb	813	13.2	813	0.01	4,631	NA	NA	0
Total PCBs	ppb	830	23.4	830	0.1	16,675	NA	NA	0
Dieldrin	ppb	615	No ERM	490	0.002	21.2	0.03	0.5	125
Lindane	ppb	533	No ERM	306	0.01	157	0.05	1	227

[a] LMW = low-molecular-weight, PAH = polynuclear aromatic hydrocarbon, HMW = high-molecular-weight, DDE = dichlorodiphenyldichloroethylene, DDT = dichlorodiphenyltrichloroethane, PCB = polychlorinated biphenyl.
[b] Percent of samples with detectable concentrations.
[c] NA = not applicable for summed concentrations.

results occurred in amphipod tests in 40 to 65% of the samples. The percentages of samples that were highly toxic in amphipod tests ranged from 40% for the ERM value for total PCB to 100% for the cadmium and chromium ERMs. The target percent of false positives (≤25% not toxic) was observed for 13 of the ERMs. The ERMs for six substances correctly classified ≥75% of samples as highly toxic in amphipod tests. Marginally toxic samples contributed relatively little (0–20%) to over-

Table 3. Frequency distribution of toxicity responses (expressed as percent of the total number of samples tested within categories of toxicologic responses), incidence of toxicity, and minimum significant differences (MSDs) for each test

Test medium/species[a]	Endpoint	Duration	*n*	% Control response					% Samples toxic[b]	MSD value
				<20%	20–39.99%	40–59.99%	60–80%	>80%		
Solid phase										
Ampelisca abdita—NOAA	Survival	10 d	289	6.6	4.8	4.5	11.8	72.3	36.3	80
A. abdita—EMAP	Survival	10 d	611	1.4	1.0	2.3	12.1	83.1	38.3	80
Rhepoxynius abronius	Survival	10 d	166	6.0	8.4	6.0	18.7	60.8	51.8	80
Solvent extract										
Photobacterium phosphoreum	Bioluminescence	15 min	224	17.4	12.1	9.8	12.9	47.8	44.6	80
Elutriate										
Mulinea lateralis	Survival	48 h	100	1.0	8.0	12.0	11.0	68.0	29.0	80
M. lateralis	Normal development	48 h	100	7.0	3.0	0.0	1.0	89.0	15.0	80
Porewater										
Arbacia punctulata	Fertilization	1 h	168	24.4	5.9	5.4	5.4	58.9	56.0	87
Strongylocentrotus purpuratus	Normal development	1 h	52	86.5	0.0	3.8	1.9	7.7	90.4	85
Haliotis rufescens	Normal development	48 h	45	71.1	4.4	4.4	6.7	13.3	91.1	85

[a] NOAA = National Oceanic and Atmospheric Administration, EMAP = Environmental Monitoring and Assessment Program.
[b] Marginally + highly toxic ($p < 0.05$, *t* tests).

Table 4. Incidence of toxicity in either amphipod tests alone or any of the two to four tests performed among samples in which individual effects range—median (ERM) values were exceeded

Chemical[a]	Amphipod tests (n = 1,068)					Any test performed[b] (n = 437)				
	No.	% Not toxic	% Marginally toxic	% Highly toxic	% Total toxic	No.	% Not toxic	% Marginally toxic	% Highly toxic	% Total toxic
Metals										
Cadmium	2	0	0	100	100	0	NA	NA	NA	NA
Chromium	5	0	0	100	100	2	0	0	100	100
Copper	25	48	0	52	52	22	18	0	82	82
Lead	35	17	6	77	83	20	5	0	95	95
Mercury	126	34	12	54	66	81	10	6	84	90
Nickel	21	24	14	62	76	5	0	0	100	100
Silver	38	34	18	47	65	22	0	14	86	100
Zinc	56	34	5	61	66	32	13	0	88	88
PAHs										
2-Methylnaphthalene	6	0	17	83	100	4	0	0	100	100
Dibenz[*a*,*h*]anthracene	43	28	2	70	72	31	19	0	81	81
Acenaphthene	13	23	15	62	77	7	0	0	100	100
Acenaphthylene	7	0	14	86	100	6	0	0	100	100
Anthracene	25	24	20	56	76	19	11	0	89	89
Benz[*a*]anthracene	47	23	13	64	77	30	10	0	90	90
Benzo[*a*]pyrene	63	37	8	56	64	46	17	0	83	83
Chrysene	38	32	13	55	68	26	8	0	92	92
Fluoranthene	32	28	13	59	72	21	5	0	95	95
Fluorene	17	29	12	59	71	10	10	0	90	90
Naphthalene	4	25	0	75	75	4	0	25	75	100
Phenanthrene	40	25	15	60	75	25	4	0	96	96
Pyrene	66	33	9	58	67	46	11	0	89	89
Sum LMW PAHs	48	21	13	67	80	31	3	6	90	96
Sum HMW PAHs	76	39	9	51	60	56	16	0	84	84
Sum total PAHs	11	9	18	73	91	6	0	0	100	100
Chlorinated hydrocarbons										
p,p′-DDE	89	45	7	48	55	70	9	6	86	92
Total DDTs	107	38	11	50	61	82	5	9	87	96
Total PCBs	194	49	11	40	51	162	17	6	78	84

[a] PAH = polynuclear aromatic hydrocarbon, LMW = low-molecular weight, HMW = high-molecular-weight, DDE = dichlorodiphenyldichloroethylene, DDT = dichlorodiphenyltrichloroethane, PCB = polychlorinated biphenyl.
[b] Excludes Environmental Monitoring and Assessment Program and Newark Bay samples; NA = not applicable.

all predictive ability. However, based upon sums of the marginally toxic and highly toxic responses, the number of ERMs that correctly predicted toxicity in ≥75% of samples increased from 6 to 13.

Relative to results of the amphipod tests, predictive ability increased considerably when the results were considered for all of the tests performed: ≥75% for all substances that exceeded the ERM concentrations (Table 4). The target percent of false positives (≤25%) was observed for all ERMs and was ≤10% for 18 substances. As with the amphipod data, the marginally toxic results in all tests performed contributed relatively little to overall predictive ability; that is, the samples often were either nontoxic or highly toxic.

Predictive ability observed with the individual PELs was slightly lower than that of equivalent ERMs (Table 5). The percentages of samples exceeding PELs that were highly toxic in amphipod tests ranged from 15% (lindane) to 73% (dieldrin). For 25 of the 31 PELs, highly toxic conditions in amphipod tests occurred in 40 to 65% of the samples. Predictive ability of ≥75% was observed for none of the PELs with only highly toxic responses and with three PELs (cadmium, acenaphthylene, and dieldrin) with marginally plus highly toxic responses combined. The target percent of false positives (≤25%) was observed for the same three PELs. When the results of any of the tests performed were considered, the percent of false positives for the PELs was ≤25% for all except one substance (p,p′-dichlorodiphenyldichloroethylene [p,p′-DDE]) and was ≤10.0% for 15 PELs. For most substances, marginally toxic results contributed 5 to 10% to overall predictive ability in both the amphipod tests alone and in all tests considered. Predictive ability of ≥75% (with highly toxic responses) was observed in any of the tests performed for all PELs except that for p,p′-DDE.

Concentrations above and below all ERL or TEL concentrations. Among the 329 samples in which none of the chemical concentrations exceeded any ERL values, 68% were not toxic, 21% were marginally toxic, and 11% were highly toxic in the amphipod tests (Table 6). Among samples in which multiple bioassays were performed, 46% were not toxic in all tests and 41% were highly toxic in at least one test when all chemical concentrations were less than the ERLs.

Of the samples tested with amphipods, 448 were found in which one or more of the 24 concentrations were greater than or equal to the ERL, but none of the concentrations were greater than or equal to the ERM values; 63% were nontoxic, 20% were marginally toxic, and 18% were highly toxic. A total of 64% of 173 samples was highly toxic in any test performed when one or more ERLs was exceeded and no ERMs were exceeded. The percent of false positives for one or more ERLs exceeded was 63% for amphipod tests alone and 20% for all tests performed.

Generally, the incidence of toxicity increased with the number of chemicals greater than or equal to the ERL concentrations; however, this pattern was variable and inconsistent (Ta-

Table 5. Incidence of toxicity in either amphipod tests alone or any of the two to four tests performed among samples in which individual probable effects levels (PELs) were exceeded

Chemical[a]	Amphipod tests (n = 1,068)					Any test performed[b] (n = 437)				
	No.	% Not toxic	% Marginally toxic	% Highly toxic	% Total toxic	No.	% Not toxic	% Marginally toxic	% Highly toxic	% Total toxic
Metals										
Cadmium	21	19	10	71	81	6	0	0	100	100
Chromium	41	34	7	59	66	24	8	0	92	92
Copper	179	41	11	48	59	146	13	6	81	87
Lead	122	37	11	52	63	85	8	6	86	92
Mercury	127	35	12	54	66	82	11	6	83	89
Nickel	74	34	12	54	66	37	5	5	89	94
Silver	109	41	10	49	59	82	12	11	77	88
Zinc	126	38	10	52	62	87	14	2	84	86
PAHs										
2-Methylnaphthalene	47	28	13	60	73	22	5	9	86	95
Dibenz[*a*,*h*]anthracene	80	36	3	61	64	65	15	2	83	85
Acenaphthene	84	38	8	54	62	56	5	7	88	95
Acenaphthylene	47	23	9	68	77	40	3	8	90	98
Anthracene	131	44	7	49	56	100	11	5	84	89
Benz[*a*]anthracene	116	39	9	52	61	93	12	4	84	88
Benzo[*a*]pyrene	126	41	9	50	59	100	12	3	85	88
Chrysene	116	43	9	47	56	93	12	4	84	88
Fluoranthene	103	42	10	49	59	80	13	5	83	88
Fluorene	74	30	12	58	70	51	6	6	88	94
Naphthalene	38	26	11	63	74	23	0	13	87	100
Phenanthrene	106	40	11	49	60	77	8	5	87	92
Pyrene	117	40	9	51	60	94	11	4	85	89
Sum LMW PAHs	117	36	9	55	64	79	8	5	87	92
Sum HMW PAHs	114	42	7	51	58	90	12	3	84	87
Sum total PAHs	56	32	11	57	68	38	11	0	89	89
Chlorinated hydrocarbons										
p,p'-DDE	3	67	0	33	33	3	33	0	67	67
p,p'-DDD	144	35	11	54	65	115	8	7	85	92
p,p'-DDT	97	33	11	56	67	68	6	7	87	94
Total DDTs	101	36	12	52	64	78	5	9	86	95
Total PCBs	191	50	10	39	49	159	17	6	77	83
Dieldrin	41	20	7	73	80	25	4	0	96	96
Lindane	54	81	4	15	19	50	14	0	86	86

[a] PAH = polynuclear aromatic hydrocarbon, LMW = low-molecular-weight, HMW = high-molecular-weight, DDE = dichlorodiphenyldichloroethylene, DDD = dichlorodiphenyldichloroethane, DDT = dichlorodiphenyltrichloroethane, PCB = polychlorinated biphenyl.
[b] Excludes Environmental Monitoring and Assessment Program and Newark Bay samples.

ble 6). Because of the relatively small numbers of samples in which many ERLs were exceeded, the incidence of toxicity also was calculated for several combined ERL categories. In the amphipod tests (n = 777), the incidence of highly toxic responses was 9% with only 1 ERL exceeded, 13% with 1 to 4 ERLs exceeded, 22% with 5 to 9 ERLs exceeded, and peaked at 67% with 15 to 19 ERLs exceeded.

The proportion of samples that was highly toxic in any test performed was 67% when only one ERL was exceeded (Table 6). The incidence of highly toxic samples increased quickly with the number of ERLs that were exceeded, reaching ≥89% when 10 to 14 concentrations were greater than or equal to the ERLs. With several exceptions (notably one sample in which 22 ERLs were exceeded), generally the proportions of samples that were marginally toxic decreased with increases in the number of concentrations greater than or equal to the ERLs.

Among the 233 samples in which all concentrations were less than the TELs; 65% were not toxic, 26% were marginally toxic, and 9% were highly toxic in amphipod tests (Table 7). A total of 62% of samples (n = 26) were not toxic in all tests performed when all concentrations were less than the TELs. The incidence of toxicity did not increase consistently in either amphipod tests alone or in any tests with increases in the number of TELs exceeded. Sample sizes in which multiple bioassays were performed were relatively small and, partly as a consequence, results were highly variable.

Concentrations above and below all ERM and PEL concentrations. Among the 1,068 samples included in this analysis, 777 and 683 had chemical concentrations less than all ERMs and less than all PELs, respectively (Tables 8 and 9). In amphipod tests, 15 and 13%, respectively, of these samples were highly toxic (false negatives). The incidence of highly toxic responses when one or more concentrations was greater than or equal to the ERM or greater than or equal to the PEL was 39 and 35%, respectively, in amphipod tests and 78 and 77%, respectively, in any test performed. With both the marginally and highly toxic responses combined, the incidence of toxicity in samples with concentrations greater than or equal to one or more ERMs or PELs increased slightly to 52 and 48%, respectively, in the amphipod tests and 86.2 and 86.1%, respectively, in any test.

In both the amphipod tests and any tests performed, the incidence of highly toxic responses generally increased and the incidence of marginally toxic responses markedly decreased with increases in the numbers of ERMs or PELs that were exceeded (Tables 8 and 9). The incidence of highly toxic responses in amphipod tests increased from 23% with only 1

Table 6. Incidence of toxicity in either amphipod tests alone or in any test performed among samples with concentrations of 0 to 24 substances greater than or equal to the effects range—low (ERL) values, but all less than the effects range—median (ERM) values

	Amphipod survival only (n = 777)				Any test performed[a] (n = 212)			
No. ERL values exceeded	No. samples	% Not toxic	% Marginally toxic	% Highly toxic	No. samples	% Not toxic	% Marginally toxic	% Highly toxic
0	329	68	21	11	39	46	13	41
1	143	68	23	9	15	13	20	67
2	66	71	15	14	13	46	8	46
3	37	62	22	16	12	42	17	42
4	43	63	16	21	21	33	14	52
5	30	60	17	23	13	38	8	54
6	33	64	12	24	24	21	13	67
7	20	55	35	10	15	27	20	53
8	15	53	27	20	12	8	33	58
9	8	50	13	38	6	0	33	67
10	9	89	0	11	6	0	0	100
11	12	42	25	33	7	0	14	86
12	9	78	11	11	8	0	13	88
13	2	0	0	100	2	0	0	100
14	4	25	50	25	3	0	33	67
15	2	50	50	0	2	0	50	50
17	1	0	0	100	1	0	0	100
18	2	0	0	100	2	0	0	100
19	1	0	0	100	1	0	0	100
20	4	50	0	50	3	0	0	100
21	4	25	25	50	4	0	0	100
22	1	0	100	0	1	0	100	0
23	1	0	0	100	1	0	0	100
24	1	0	0	100	1	0	0	100
1 or more	448	62.7	19.6	17.6	173	20.2	15.6	64.2
1 to 4	289	67.1	20.1	12.8	61	32.8	14.8	52.5
5 to 9	106	58.5	19.8	21.7	70	21.4	18.6	60.0
10 to 14	36	58.3	16.7	25.0	26	0.0	11.5	88.5
15 to 19	6	16.7	16.7	66.7	6	0.0	16.7	77.8
20 to 24	11	27.3	18.2	54.5	10	0.0	10.0	90.0

[a] Excludes Environmental Monitoring and Assessment Program and Newark Bay data.

ERM exceeded to 32% with 1 to 5 ERMs exceeded, to 52% with 6 to 10 ERMs exceeded, and peaked at 85% with ≥11 ERMs exceeded (Table 8). The lowest percent false positives (10%) occurred among samples with 11 to 20 ERMs exceeded. In samples in which multiple bioassays were performed, incidence of highly toxic responses increased from 70% with only 1 ERM exceeded, to 89% with 6 to 10 ERMs exceeded, and peaked at 100% with ≥11 ERMs exceeded. Results were variable among samples with greater than or equal to eight ERMs exceeded because of the small sample sizes.

The predictive ability of the PELs was somewhat lower than that of the ERMs, but, nevertheless, indicated a similar pattern of increasing incidence of highly toxic responses with increasing numbers of PELs exceeded (Table 9). In the amphipod tests, the incidence of highly toxic responses was 14% with 1 PEL exceeded, 24% with 1 to 5 PELs exceeded, 40% with 6 to 10 PELs exceeded, 50% with 11 to 20 PELs exceeded, and 88% with ≥21 PELs exceeded. The lowest percent false positives (17%) occurred among samples with ≥21 PELs exceeded. The proportion of samples showing highly toxic results was much higher when all bioassays were considered, averaging 80% with 6 to 10 PELs exceeded and peaking at 100% with ≥21 PELs exceeded. Percent false positives in any of the tests performed was <25% when one or more PEL was exceeded.

Over ranges in mean SQG quotients. In the preceeding analyses, the methods did not account for the degree to which the chemical concentrations exceeded the different SQGs. That is, samples in which chemical concentrations exceeded SOGs by very different amounts were scored the same. Given similar sediment characteristics and toxicant bioavailability, the probability of toxicity could increase with increasing concentrations. Therefore, to account for both the actual concentrations of individual substances and the combinations of chemicals occurring as mixtures, the predictive abilities of the mean SQG quotients were determined.

The relationships between the incidence of highly toxic responses in the amphipod tests and mean SQG quotients are illustrated in Figures 1 and 2. To clarify these relationships, the chemical concentrations are shown as medians of 39 SQG quotient intervals, each consisting of at least 25 samples. These relationships were considerably more variable when marginally toxic responses were included; therefore, the plots are shown only for highly toxic responses. The incidence of highly toxic responses was most variable and ranged from 0 to 40% among samples with the lowest mean ERM quotients (0.001–0.02) and PEL quotients (0.006–0.05). A gradual, albeit variable, pattern of increasing incidence of toxicity beginning at mean ERM and PEL quotients of 0.04 and 0.07, respectively, was evident. Among samples with mean ERM or PEL quotients ≥1.0 or ≥1.6, respectively, 60 to 80% were highly toxic in the amphipod tests. Percent false positives decreased to <25% with mean ERM or PEL quotients >1.2 or >2.3, respectively.

Some of the samples with the lowest mean ERM and PEL quotients were highly toxic, as indicated in the left tails of the distributions (Figs. 1 and 2). These samples shared very few of the same characteristics. They were scattered among many

Table 7. Incidence of toxicity in either amphipod tests alone or in any test performed among samples with concentrations of 0 to 27 substances greater than or equal to the threshold effects level (TEL) values, but all less than the probable effects level (PEL) values

No. TEL values exceeded	Amphipod survival only (n = 683)				Any test performed[a] (n = 142)			
	No. samples	% Not toxic	% Marginally toxic	% Highly toxic	No. samples	% Not toxic	% Marginally toxic	% Highly toxic
0	233	65	26	9	26	62	15	23
1	102	74	15	12	9	22	11	67
2	67	67	24	9	5	40	20	40
3	62	69	21	10	10	40	30	30
4	46	65	11	24	5	0	0	100
5	28	61	25	14	7	29	14	57
6	15	53	33	13	4	50	0	50
7	10	70	20	10	5	20	20	60
8	15	73	7	20	6	17	0	83
9	5	60	20	20	3	0	0	100
10	12	67	8	25	6	33	17	50
11	11	27	27	45	6	17	17	67
12	15	67	0	33	11	27	0	73
13	10	90	0	10	5	40	0	60
14	7	71	29	0	4	50	25	25
15	3	33	33	33	2	50	50	0
16	4	75	25	0	1	0	100	0
17	2	50	50	0	1	0	0	100
18	4	75	0	25	2	0	50	50
19	8	63	25	13	3	33	0	67
20	5	40	20	40	4	0	25	75
21	4	0	75	25	4	0	25	75
22	3	33	33	33	3	33	33	33
23	9	33	44	22	7	0	43	57
24	1	0	0	100	1	0	0	100
25	1	0	0	100	1	0	0	100
27	1	0	100	0	1	0	100	0
1 or more	450	65.1	19.1	15.8	116	23.3	17.2	59.5
1 to 5	305	68.9	18.4	12.8	36	27.8	16.7	55.6
6 to 9	45	64.4	20.0	15.6	18	22.2	5.6	72.2
10 to 14	55	63.6	10.9	25.5	32	31.3	9.4	59.4
15 to 19	21	61.9	23.8	14.3	9	22.2	33.3	44.4
20 to 27	24	25.0	41.7	33.3	21	4.8	33.3	61.9

[a] Excludes Environmental Monitoring and Assessment Program and Newark Bay data.

Table 8. Incidence of toxicity in either amphipod tests alone or in any of two to four tests performed among samples with concentrations of 0 to 20 substances greater than or equal to the effects range—median (ERM) concentrations

No. ERM values exceeded	Amphipod survival only (n = 1,068)				Any test performed[a] (n = 437)			
	No. samples	% Not toxic	% Marginally toxic	% Highly toxic	No. samples	% Not toxic	% Marginally toxic	% Highly toxic
0	777	65	20	15	212	25	15	60
1	95	59	18	23	69	17	13	70
2	66	52	12	36	62	11	11	77
3	34	56	21	24	30	17	7	77
4	19	32	5	63	10	20	0	80
5	11	45	0	55	9	11	0	89
6	11	55	9	36	10	20	0	80
7	10	40	0	60	8	13	0	88
8	4	25	25	50	4	0	0	100
9	11	9	9	82	7	0	0	100
10	10	50	20	30	6	17	0	83
11	4	0	25	75	1	0	0	100
12	6	33	0	67	3	0	0	100
13	4	0	0	100	3	0	0	100
14	3	0	0	100	1	0	0	100
15	1	0	0	100	1	0	0	100
17	1	0	0	100	0	0	0	0
20	1	0	0	100	1	0	0	100
1 or more	291	47.8	13.4	38.8	225	13.8	8.0	78.2
1 to 5	225	53.3	14.7	32.0	180	15.0	10.0	75.0
6 to 10	46	37.0	10.9	52.2	35	11.4	0.0	88.6
11 to 20	20	10.0	5.0	85.0	10	0.0	0.0	100.0

[a] Excludes Environmental Monitoring and Assessment Program and Newark Bay data.

Table 9. Incidence of toxicity in either amphipod tests alone or in any of two to four tests performed among samples with concentrations of 0 to 20 substances greater than or equal to the probable effects level (PEL) concentrations

	Amphipod survival only (n = 1,148)				Any test performed[a] (n = 517)			
No. PEL values exceeded	No. samples	% Not toxic	% Marginally toxic	% Highly toxic	No. samples	% Not toxic	% Marginally toxic	% Highly toxic
0	683	65	22	13	142	30	17	53
1	106	73	13	14	79	15	10	75
2	49	45	18	37	42	19	14	67
3	36	53	14	33	25	12	16	72
4	17	47	29	24	11	18	36	45
5	16	56	13	31	13	15	0	85
6	10	30	20	50	7	0	0	100
7	4	25	25	50	3	0	33	67
8	18	56	6	39	16	31	0	69
9	11	55	0	45	7	0	0	100
10	9	67	11	22	7	29	0	71
11	19	53	16	32	17	12	0	88
12	8	38	13	50	7	14	0	86
13	13	54	8	38	11	18	0	82
14	8	38	13	50	8	0	13	88
15	11	36	9	55	10	20	0	80
16	8	25	13	63	7	0	14	86
17	7	43	0	57	5	0	0	100
18	10	40	0	60	8	0	13	88
19	5	0	20	80	2	0	0	100
20	3	0	33	67	3	0	0	100
21	2	0	0	100	1	0	0	100
22	5	0	0	100	1	0	0	100
23	5	20	0	80	1	0	0	100
24	4	25	0	75	3	0	0	100
26	1	0	0	100	1	0	0	100
1 or more	385	51.7	13.0	35.3	295	13.9	8.8	77.3
1 to 5	224	60.3	15.6	24.1	170	15.9	12.9	71.2
6 to 10	52	50.0	9.6	40.4	40	17.5	2.5	80.0
11 to 20	92	39.1	10.9	50.0	78	9.0	3.8	87.2
21 to 26	17	11.8	0.0	88.2	7	0.0	0.0	100.0

[a] Excludes Environmental Monitoring and Assessment Program and Newark Bay data.

of the different NOAA and EMAP study areas. These samples often, but not always, had relatively low organic carbon content (<1.0%) and percent fine-grained materials (<50%) and detectable concentrations of butyl tins, chlorinated pesticides, alkyl-substituted PAHs, ammonia, or other substances not accounted for with the SQGs.

DISCUSSION AND CONCLUSIONS

Sediment quality guidelines [1,2] were based upon empirical analyses of data compiled from many different studies. The SQGs were intended to provide informal (nonregulatory), effects-based benchmarks to aid in the interpretation of sed-

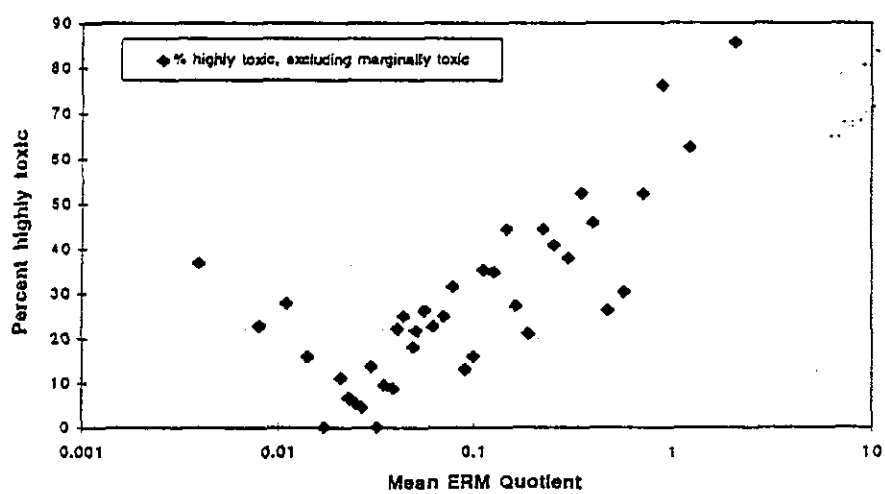

Fig. 1. The relationship between the incidence of toxicity in amphipod survival tests and mean effects range—median (ERM) quotients (plotted as the medians of 39 quotient intervals, each consisting of 25 samples).

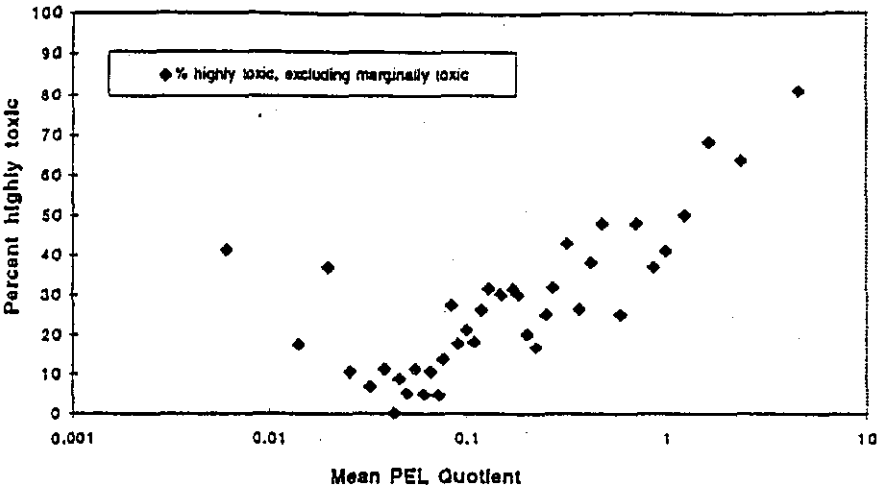

Fig. 2. The relationship between the incidence of toxicity in amphipod survival tests and mean probable effects level (PEL) quotients (plotted as the medians of 39 quotient intervals, each consisting of 25 samples).

iment chemistry data. The ERL and TEL values were intended to represent chemical concentrations below which the probability of toxicity and other effects was minimal. In contrast, the ERM and PEL values were intended to represent mid-range concentrations above which adverse effects were more likely, although not always expected. Intermediate frequencies of effects were expected at chemical concentrations between the ERLs and ERMs and between the TELs and PELs. In this analysis of independent data sets, we attempted to determine if the incidence of toxicity in selected, acute laboratory bioassays would follow the same pattern as observed with multiple measures of effects in the databases used to derive the guidelines.

The majority of the data compiled to develop the guidelines was generated in field studies in which different chemical mixtures were encountered. In these field studies causality could not be determined. The intent of this study was to also use data from surveys of numerous saltwater areas to determine the frequency with which the guidelines correctly predicted nontoxic and toxic conditions.

Unlike SQGs based upon the apparent effects threshold approach [26], the ERL/ERMs and TEL/PELs were not intended to represent concentrations above which adverse effects were always observed. Because the ERLs and TELs were intended to represent conservative concentrations below which toxicity was not frequently expected, we estimated the frequency of false negatives as the incidence of toxicity among samples in which all concentrations were lower than these values. Earlier [1,2], as a measure of reliability, we reported that the frequency of false negatives among the data sets used to derive the guidelines was ≤25% for most chemicals and ≤10% for many chemicals. Specifically, at concentrations below the individual ERL and TEL values for nine trace metals, the incidence of effects ranged from 1.9 to 9.4% and from 2.7 to 9.0%, respectively. For organic compounds, the incidence of effects was more variable, ranging from 5.0 to 27.3% for 19 ERLs and from 0.0 to 47.6% for 25 TELs when concentrations were below these levels.

The same criterion (≤25% false positives) previously used for estimates of reliability was used as the target for estimates of predictive ability in this analysis. Based upon the highly toxic responses, the ERLs and TELs indicated 11 and 9% false negatives (toxicity observed when not expected), respectively, in the tests of amphipod survival, thus bettering the target of ≤25%. The incidence of false negatives also was relatively low (41 and 23% for the ERLs and TELs, respectively) in any one of the two to four tests performed. Based again upon the highly toxic responses, the incidences of false negatives in amphipod tests were, as expected, slightly higher (15 and 13%, respectively) for the ERMs and PELs than for the ERLs and TELs. Therefore, the probabilities of highly toxic responses in amphipod survival tests are relatively low (≤16%) among samples in which all chemical concentrations are lower than both sets of SQGs. However, the incidences of false negatives among any of the tests performed were 60 and 53% (highly toxic responses) for the ERMs and PELs, respectively. These data suggest that there remains a moderate probability of toxicity among samples with all chemical concentrations less than the ERMs or less than the PELs if a battery of relatively sensitive, sublethal bioassays is considered.

In the amphipod tests, the incidences of highly toxic responses and total toxic responses were 18 to 20% and 16 to 19%, respectively, when one or more chemicals exceeded the ERLs and/or TELs. These results agreed well with the original intent of the ERLs and TELs as indicators of the lower end of the possible effects range. These results also agreed very well with the estimates of reliability (calculated with the database used to derive the SQGs) for most ERLs and TELs (30–50% effects) [1,2]. However, when predictive ability was estimated with data from more sensitive sublethal tests, toxicity was observed much more frequently than in the amphipod tests alone.

The ERMs and PELs were derived as mid-range points within the distributions of effects data for each chemical. The ERMs were calculated as the medians (50th percentiles) of chemical concentrations associated with measures of adverse effects. The derivation of the PELs incorporated both the no-effects data along with effects data into the calculations of mid-range concentrations. Neither set of guidelines was intended as a toxicity threshold above which effects were always expected. The incidence of highly significant toxicity in the amphipod survival tests among samples that exceeded individual ERMs and PELs generally agreed with the intent of these values (i.e., as mid-range values). That is, 40 to 65% of

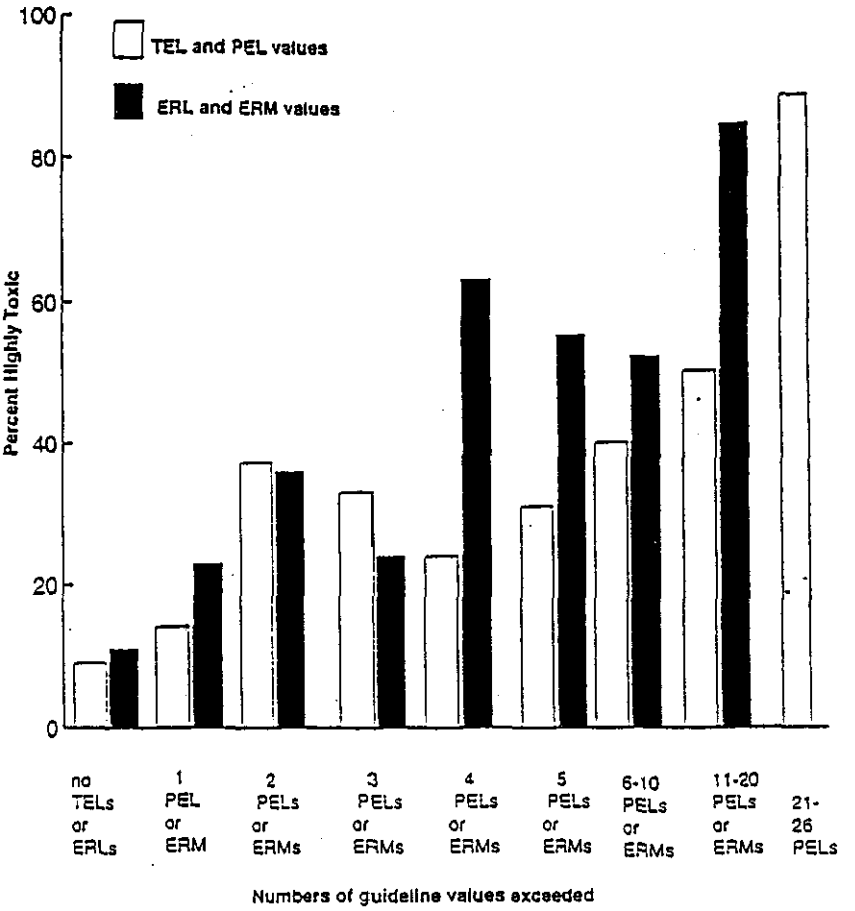

Fig. 3. Summary of the predictive ability of threshold effects level/probable effects level (TEL/PEL) values and effects range—low/effects range—median (ERL/ERM) values in amphipod survival tests (as percent highly toxic among the total numbers of samples).

the samples were highly toxic in amphipod tests at concentrations above most of these individual values. Also, the incidence of total toxicity (marginally + highly toxic) was 52 and 48% when the concentrations of one or more chemicals exceeded ERMs and PELs, respectively. When results from any one of a battery of bioassays were considered, the percentages of samples that were highly toxic increased remarkably to ≥85% for 19 of the ERMs and for 19 of the PELs and to 77 to 78% when one or more ERMs and/or PELs were exceeded.

In all analyses performed on the predictive ability of the SQGs, the percentages of samples demonstrating toxicity were lowest when either no chemicals or the least number of chemicals exceeded the lower range guidelines and increased with increases in the numbers of mid-range guidelines that were exceeded (Fig. 3). Results were variable at intermediate concentrations, but, nevertheless, the data indicated an overall pattern of increasing incidence of toxicity with increasing numbers of ERMs and PELs exceeded. Percent false positives in amphipod tests (no toxicity observed when toxicity was expected) dropped to <25% among samples in which 11 to 20 ERMs (n = 20) and 21 to 26 PELs (n = 17) were exceeded.

Because the two sets of SQGs were derived with slightly different procedures, one objective of this evaluation was to compare their predictive ability. The results indicated that the two sets of SQGs were very similar in predicting toxicity (Fig. 3). The percentages of false negatives for the ERLs and TELs were 11 and 9%, respectively, in the amphipod tests. The incidences of highly toxic responses in amphipod tests were slightly higher for the PELs than for the ERMs among samples in which two or three chemicals exceeded the guideline concentrations. Otherwise, the incidence of toxicity often was higher when chemical concentrations exceeded the ERMs as compared to when the concentrations exceeded the PELs.

Based upon these data, users of the SQGs can identify the probability that their samples would be toxic by comparing the chemical concentrations in their samples to the appropriate SQGs and then to the incidence of toxicity shown in this paper. For example, highly toxic responses would be expected in amphipod survival tests in only approximately 9 to 11% of the samples when all chemical concentrations are below the TELs or ERLs (Fig. 3). Among samples in which only one ERL or TEL value is exceeded and no other chemicals exceeded any other ERL/ERMs or TEL/PELs, toxicity in amphipod tests would be expected in only 9 and 12% of the samples, respectively.

The probability of toxicity in amphipod survival tests is not very high (23 and 14%, respectively) among samples in which only one ERM or only one PEL value is exceeded (Fig. 3). However, the probabilities of toxicity increase with the number of ERMs and PELs exceeded. Based upon the results of this evaluation (n = 1,068), users can expect toxicity in a large majority of samples, that is, in >85% of the samples in amphipod tests (n = 20, n = 17) and in 100% of samples in any one of a battery of sensitive bioassays (n = 9 or 6) when 11 or more ERMs or 21 or more PELs are exceeded. Therefore,

Table 10. Incidence of toxicity in amphipod tests only within three ranges in mean sediment quality guideline quotients

	No. samples	% Not toxic	% Marginally toxic	% Highly toxic
Mean effects range—median quotients				
<0.1	653	67.8	20.5	11.6
0.11 to 1.0	364	51.6	16.5	31.9
>1.0	51	23.5	5.9	70.6
Mean probable effects level quotients				
<0.1	481	67.6	22.0	10.4
0.11 to 1.0	474	58.6	17.1	24.3
>1.0	113	35.4	8.8	55.8

the probability of incorrectly classifying samples as toxic would be 15 and 0%, respectively, in these highly contaminated samples.

The data from the analyses of the mean SQG quotients suggest that the probability of observing toxicity was a function of not only the number of guidelines exceeded but the degree to which they were exceeded. Therefore, the probabilities of highly toxic responses would be relatively low (<12% in amphipod tests) among samples with mean SQG quotients <0.1 (Table 10). The probabilities of toxicity increase to 32 and 24%, respectively, with mean ERM and PEL quotients of 0.11 to 1.0 and increase again to 71 and 56%, respectively, with quotients >1.0.

Despite the selection of high-quality data sets from NS&TP and EMAP—Estuaries studies, the analyses of predictive ability had a number of limitations or potential sources of error. Different results may have been obtained if other data had been used in this evaluation of predictive ability.

The core bioassay upon which these analyses focused was the amphipod survival test. This bioassay has become the most widely applied sediment toxicity test in North America and provides important information for many research, monitoring, and management programs. Amphipod survival tests have been used in both the derivation and field validation of various guidelines [22,26]. However, because different taxa have different sensitivities to toxicants, the use of a battery of toxicity tests is widely accepted and highly recommended in sediment quality assessments [27]. Furthermore, the use of multiple tests increases the number of surrogates of sediment-dwelling taxa. Considerable gains in predictive ability were attained by the addition of data from other tests to those from the amphipod tests. Because only one, two, or three (not, say, 10) tests accompanied the amphipod bioassays, we attribute the gains in predictive ability not to the number of tests performed, but, rather, to the greater sensitivity of the tests to the chemicals in the sediments.

Tests of invertebrate gametes and embryos exposed to pore waters and bioluminescent bacteria exposed to solvent extracts have been used widely in U.S. estuaries [24] and generally are more sensitive than are test with amphipods to the same samples. The large differences in sensitivity between the amphipod survival tests and the other tests performed is reflected in the data that were analyzed. The probabilities of observing toxicity in the more sensitive sublethal tests would be much higher than in the amphipod tests. Users are advised to consider the data from both categories of bioassays when using the guidelines, especially because highly sensitive tests such as those teria [28] have shown strong associations with chemical concentrations.

Sediment quality guidelines were not available for many substances that were measured in the samples. Some substances may have occurred at concentrations above toxicologic thresholds. Other substances that were not measured probably occurred in many or all samples. Also, some samples may have had high concentrations of ammonia and hydrogen sulfide that covaried with anthropogenic substances and contributed to toxicity. Together, the effects of these substances may have contributed to the false negatives observed. However, our nationwide experience indicates that toxicants often covary with each other to a large degree [7,25] and the quantified substances for which SQGs were available should have served as reasonable surrogates for the covariates. Furthermore, our experience in assessments of surficial sediments suggests that ammonia and sulfides occur in either pore water or overlying water in test chambers at toxicologically significant concentrations in <10% of the samples. Nevertheless, the contribution of all potentially toxic substances in the samples could not be accounted for.

Although standardized and widely accepted methods and protocols were used, some interlaboratory and interstudy differences in methods may have occurred. Some variability in results may have been attributable to merging data from different studies and geographic areas. For example, data were compiled from tests performed with two species of amphipods to increase the sample size and to include data from all three coastlines. Differences in sensitivity between these two amphipod species may have contributed to variability in the results. Also, variability may have been increased by merging data from different species of urchins and molluscs along with data from the Microtox tests into one category.

Most of the samples were not collected within hazardous waste sites and most were not highly contaminated (zero to five SQGs exceeded). The relatively small numbers of highly contaminated samples appeared to contribute to variability in results. Additional data from highly contaminated sites would be useful in further clarification of predictive ability.

Despite these potential limitations of this study, the predictive ability estimated with these data often matched their previously reported reliability. Also, the results of this analysis agreed relatively well with the the estimates of reliability reported [5] for freshwater sediment effects concentrations. The results of this analysis [5] determined type I (false positive) and type II (false negative) errors for freshwater ERL/ERM and TEL/PEL values based upon data from individual samples from numerous studies. For most substances, the errors ranged from 5 to 30%. The paired sets of values, however, differed somewhat in absolute concentrations and error rates.

The toxicity/chemistry relationships observed in this study may not apply in all situations, especially in sediments in which contaminants are found in forms such as copper slag [29] or coal pitch in organically enriched mud [30]. The guidelines are most useful when applied to fine-grained, sedimentary deposits such as those sampled during the NOAA and EMAP—Estuaries studies.

In conclusion, the results of these analyses indicate the following: the probabilities of highly toxic responses occurring in amphipod survival tests among samples in which all chemical concentrations are less than ERLs and/or TELs are 9 to 11%; the probabilities of highly toxic responses occurring in

quotients are <0.1 are 10 to 12%; the probabilities of highly toxic responses occurring when one or more ERLs or TELs are exceeded and no ERMs or PELs are exceeded are 16 to 18% in amphipod tests alone and 60 to 64% in any one of a battery of sensitive tests performed; the probabilities of either marginally or highly toxic responses occurring are 48 to 52% in amphipod tests and 86% in any one of a battery of sensitive tests performed when concentrations exceed one or more ERMs or PELs; consistent with their original intent, the ERMs and PELs are considerably better at predicting toxicity than are the ERLs and TELs. Furthermore, the probabilities of toxicity occurring generally increase with increasing numbers of chemicals that exceed the ERM and PEL concentrations; the probabilities of toxicity occurring generally increase with increasing mean SQG quotients; and the incidence of false negatives is slightly lower for the TELs than for the ERLs, but the incidence of false positives is generally higher for the PELs than for the ERMs; however, there is good overall agreement in the predictive ability of the TEL/PELs and the ERL/ERMs.

Based upon these analyses of predictive ability and previous analyses of reliability, it appears that the SQGs provide reasonably accurate estimates of chemical concentrations that are either nontoxic or toxic in laboratory bioassays. However, we urge that all SQGs should be used with caution, because, as observed in this analysis, they are not perfect predictors of toxicity. Especially among samples with intermediate chemical concentrations, the SQGs are most useful when accompanied by data from in situ biological analyses, other toxicologic assays, other interpretive tools such as metals : aluminum ratios, and other guidelines derived either from empirical approaches and/or cause–effects studies.

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Development and evaluation of sediment quality guidelines for Florida coastal waters

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The weight-of-evidence approach to the development of sediment quality guidelines (SQGs) was modified to support the derivation of biological effects-based SQGs for Florida coastal waters. Numerical SQGs were derived for 34 substances, including nine trace metals, 13 individual polycyclic aromatic hydrocarbons (PAHs), three groups of PAHs, total polychlorinated biphenyls (PCBs), seven pesticides and one phthalate ester. For each substance, a threshold effects level (TEL) and a probable effects level (PEL) was calculated. These two values defined three ranges of chemical concentrations, including those that were (1) rarely, (2) occasionally or (3) frequently associated with adverse effects. The SQGs were then evaluated to determine their degree of agreement with other guidelines (an indicator of comparability) and the percent incidence of adverse effects within each concentration range (an indicator of reliability). The guidelines also were used to classify (using a dichotomous system: toxic, with one or more exceedances of the PELs or non-toxic, with no exceedances of the TELs) sediment samples collected from various locations in Florida and the Gulf of Mexico. The accuracy of these predictions was then evaluated using the results of the biological tests that were performed on the same sediment samples. The resultant SQGs were demonstrated to provide practical, reliable and predictive tools for assessing sediment quality in Florida and elsewhere in the southeastern portion of the United States.

Keywords: sediment quality guidelines; contaminants; biological effects; marine; estuarine.

Introduction

Sediment chemistry data indicate that Florida coastal sediments in several areas are contaminated (Long and Morgan 1990, Delfino *et al.* 1991, FDEP 1994). For example, sediments in Tampa Bay, Pensacola Bay and Biscayne Bay are contaminated with trace metals, polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs) and certain pesticides. Additionally, Choctawhatchee Bay and St Andrews Bay sediments are contaminated with metals, PAHs and pesticides and elevated levels of PCBs have been

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detected in St Andrews Bay, Apalachicola Bay, Charlotte Harbor, Naples Bay, Rookery Bay and the St Johns River (Long and Morgan 1990).

While sediment chemistry data are essential for evaluating sediment quality, they do not provide a comprehensive basis for classifying or managing contaminated sediments (Long *et al.* 1995). Interpretive tools are also required to relate sediment chemistry data to the potential for adverse biological effects. Various toxicity and bioaccumulation tests can be performed to evaluate the biological significance of sediment-associated contaminants (Burton 1992). In addition, numerical sediment quality guidelines (SQGs) can be used to help interpret the significance of chemical concentrations in sediments, identify contaminants of concern and prioritize areas for regulation or restoration (Long and Morgan 1990, Di Toro *et al.* 1991, Persaud *et al.* 1992, MacDonald *et al.* 1992).

A variety of approaches have been developed to support the derivation of numerical SQGs in the United States and Canada (see Chapman 1989, Adams *et al.* 1992, MacDonald *et al.* 1992, USEPA 1992, MacDonald, 1994 for reviews). The procedures used for deriving the SQGs and the philosophy behind their development are approach specific. Consequently, each approach has certain advantages and limitations that affect the applicability of the resultant guidelines. Presently there is little agreement as to which approach provides the most reliable guidelines, therefore, each of the major approaches was evaluated to determine which would best address Florida's unique requirements for SQGs (MacDonald 1994). The results of this evaluation indicated that the weight-of-evidence approach, developed to provide informal guidelines for evaluating data collected under the National Status and Trends Program of the National Oceanic and Atmospheric Administration (NOAA; Long and Morgan 1990, Long 1992), would provide scientifically defensible and broadly applicable guidelines for assessing sediment quality. The evaluation by MacDonald (1994) also indicated that some modifications to the original approach could potentially increase the applicability of the procedure for deriving SQGs for Florida's coastal waters.

The objectives of this study were to (1) describe the modifications made to the weight-of-evidence approach to support the development of SQGs for Florida coastal waters, (2) present the SQGs that were derived and (3) evaluate the applicability of these SQGs for Florida coastal waters. The comparability of the SQGs was assessed by comparing them with those derived using other data or other methods. The reliability of the guidelines was evaluated by determining the percent incidence of adverse effects within each of three ranges of contaminant concentrations. The predictability of the SQGs was assessed using several independent data sets from Florida and the Gulf of Mexico, which contain both sediment chemistry and biological effects data.

Methods

The weight-of-evidence approach to the development of numerical sediment quality guidelines has been described in detail elsewhere (Long and Morgan 1990, Long 1992, Long and MacDonald 1992, Long *et al.* 1995), so only an overview of the approach and the modifications that were adopted in Florida will be presented here. The derivation of numerical guidelines using the weight-of-evidence approach consists of three main steps. First, all of the available information which described associations between contaminant concentrations and adverse biological effects in sediments was collected and evaluated. These data were collated in a biological effects database for sediments (BEDS;

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MacDonald *et al.* 1994). Next, two SQGs, a threshold effects level (TEL) and a probable effects level (PEL), were derived for each chemical substance. These guidelines were intended to define three ranges of contaminant concentrations that were rarely, occasionally and frequently associated with adverse biological effects. Finally, the SQGs were evaluated to determine their applicability to Florida coastal waters.

Development of a biological effects database for sediments

The first step in the derivation of SQGs involved the development of a biological effects database for sediments (BEDS) to compile matching chemical and biological data from numerous studies conducted throughout North America. Over 350 reports were reviewed and critically evaluated for this purpose. These reports provided information from equilibrium-partitioning models, laboratory spiked-sediment toxicity tests and field studies used to investigate the toxicity and/or benthic community composition of marine and estuarine sediments. Each record in the database included the citation, type of test and/or biological effects observed or predicted approach that was used, study area, test duration (if applicable and reported), species tested or the benthic community considered, TOC concentrations (if reported) and the concentration(s) of each chemical (expressed on a dry weight basis). More detailed descriptions of the BEDS are provided in Long (1992), Long and MacDonald (1992), CCME (1994), MacDonald (1994), Long *et al.* (1995).

Derivation of sediment quality guidelines

The BEDS was used as the sole source of information about the potential effects of sediment-associated contaminants. Data for each of the chemicals of concern in Florida coastal waters were retrieved from the database, incorporated into data tables and sorted in ascending order of the chemical's concentration. Each ascending data table summarized all available information that related the concentrations of the chemical to effects on aquatic organisms (MacDonald *et al.* 1994). The information in these tables was then sorted into two data sets, including (1) an 'effects data set', which included all of the 'effects' data entries (i.e. those for which an adverse biological effect was observed in association with at least a 2-fold elevation in the chemical concentration above reference conditions; Long *et al.* 1995) and (2) a 'no effects data set', which included all of the 'no effects' data entries (i.e. those for which either no adverse biological effect was observed or an effect was observed but the chemical concentration was not elevated relative to reference conditions; less than a 2-fold elevation). Both a TEL and a PEL were derived for each substance that had at least 20 data entries in both the 'effects' and 'no effects' category.

In this study, the original derivation procedures were modified to develop a TEL and a PEL for each analyte. Originally, the 10th (ER-L) and 50th (ER-M) percentile values in the effects data set were used to establish sediment quality guidelines (Long and Morgan 1990, Long 1992, Long and MacDonald 1992, Long *et al.* 1995). This method was similar to the procedure used by Klapow and Lewis (1979) to establish marine water quality standards in California. These authors reasoned that the use of percentiles of aquatic toxicity data effectively minimized the influence of single (potentially outlier) data points on the resultant assessment values (e.g. Barrick *et al.* 1988). The original procedure did not utilize the information in the no effects data set, however. Nonetheless, data on the concentrations of contaminants that are not associated with

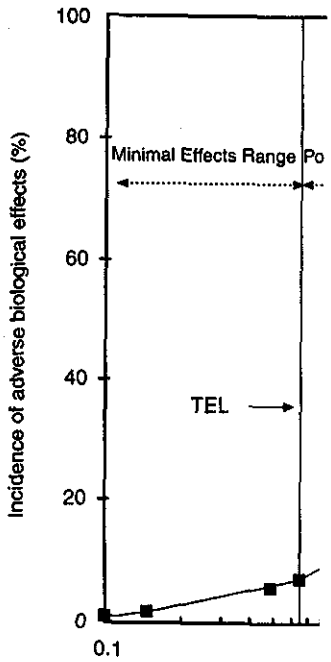
adverse effects may provide additional information for defining the relationships between contaminant exposure and biological effects and was, therefore, used in this investigation.

In the present study, two SQGs were derived for each analyte using the information in both the effects and the no effects data sets, with the distributions of these data sets determined using percentiles (Byrkit 1975). For each analyte, a TEL was derived by calculating the geometric mean of the 15th percentile of the effects data set and the 50th percentile of the no effects data set. Similarly, a PEL was developed for each chemical by determining the geometric mean of the 50th percentile of the effects data set and the 85th percentile of the no effects data set. These arithmetic procedures were established by testing a variety of options using data for cadmium, copper, fluoranthene and phenanthrene and, subsequently, evaluating the resultant guidelines relative to their narrative objective. In this respect, the TEL was intended to estimate the concentration of a chemical below which adverse effects only rarely occurred (i.e. a minimal effects range; Fig. 1). Similarly, the PEL was intended to provide an estimate of the concentration above which adverse effects frequently occurred (i.e. probable effects range). Therefore, the TEL and PEL were intended to define three concentration ranges for a chemical, including those that were rarely, occasionally and frequently associated with adverse effects (Fig. 1). The extent to which the tested options satisfied these narrative objectives was determined by calculating the percent incidence of adverse effects below the TEL and above the PEL (Long *et al.* 1995).

The arithmetic procedures used to derive the SQGs in Florida were similar to those that have been used in other applications. For example, the Canadian Council of Ministers of the Environment (CCME, 1992) calculated the geometric mean of the lowest-observed-effect concentration (LOEC) and the no-observed-effect concentration (NOEC) to establish effects-based soil quality criteria. Similarly, Rand and Petrocelli (1985) calculated maximum acceptable toxicant concentrations (MATCs) from LOECs and NOECs from aquatic toxicity tests. The Canadian Council of Resource and Environment Ministers (CCREM, 1987) established a protocol that utilizes an analogous method for deriving water quality guidelines for the protection of agricultural water uses. In each of these cases, the geometric mean, rather than the arithmetic mean, was calculated due to uncertainty regarding the distributions of the data sets (i.e., the data were not expected to be normally distributed; Sokal and Rohlf 1981). The arithmetic procedures used in this study recently were adopted for deriving national SQGs in Canada (CCME 1994).

Evaluation of sediment quality guidelines

The SQGs developed in this study were evaluated using three procedures. The comparability of the SQGs was evaluated by comparing the TELs and PELs with similar assessment tools that have been derived using different approaches or procedures. The reliability of the SQGs was evaluated by calculating the percent incidence of adverse effects within ranges of contaminant concentrations defined by the TELs and PELs, using the information contained in the BEDS (MacDonald 1994, Long *et al.* 1995). Finally, the predictability of the SQGs was assessed using independent sediment chemistry and biological effects data sets from areas throughout the southeastern portion of the United States (MacDonald 1994).



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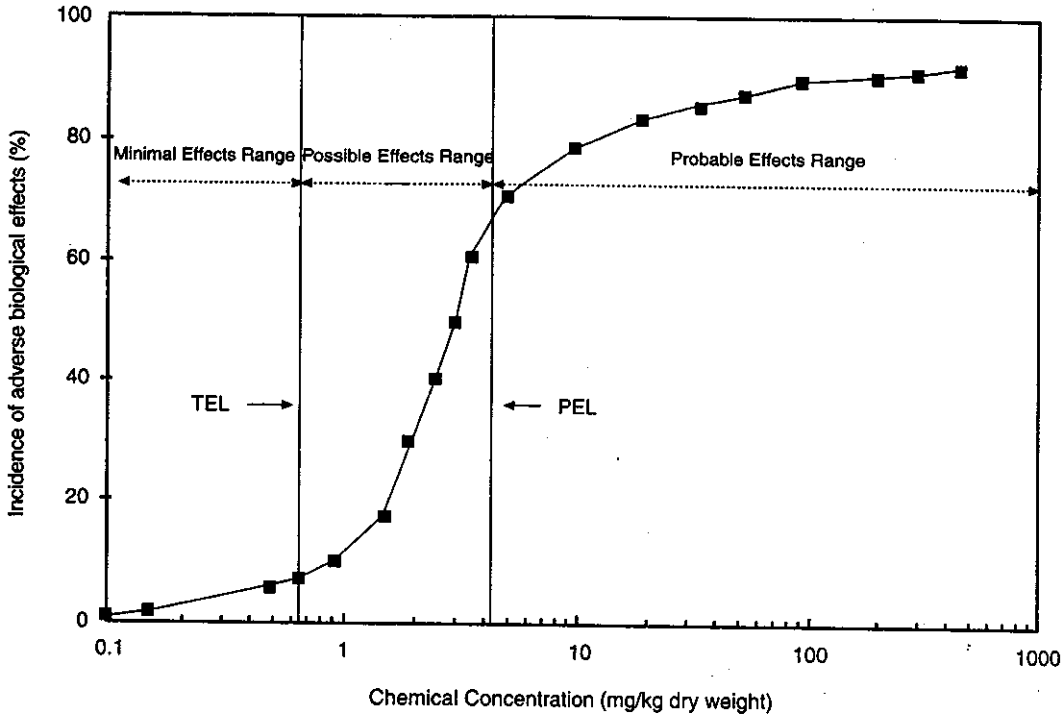

Fig. 1. Conceptual representation of the ranges of contaminant concentrations defined by SQGs and the potential for observing adverse effects within these ranges.

Comparability. The SQGs were compared to a variety of sediment quality criteria, guidelines, objectives and standards that were developed for use in other applications (Tables 1 and 2). Guidelines developed using different approaches or different procedures were considered to be comparable if they agreed within a factor of three of the SQGs developed in this study (Lorenzato *et al.* 1991).

We interpret the threshold effects levels (TELs) as the concentrations below which adverse biological effects rarely occur. Hence, the TELs are considered to provide a high level of protection for aquatic organisms. A total of four sets of similar guidelines were identified for comparison with the TELs derived in this study, including (1) the NOAA effects range-low values (ER-Ls; Long *et al.* 1995), (2) the screening levels (PSDDA-SL) developed for the Puget Sound Dredged Disposal Analysis Program (USACOE 1988), (3) the USEPA chronic sediment quality criteria (SQC-chronic; Lyman *et al.* 1987, Pavlou 1987, Hansen *et al.* 1993a,b,c,d,e) and (4) the sediment quality objectives SQOs) developed for Burrard Inlet (Swain and Nijman 1991).

We interpret the probable effects level (PELs) as the concentrations above which adverse biological effects frequently occur. Hence, the PELs are considered to provide a lower level of protection for aquatic organisms. The four sets of guidelines that were compared to the PELs included (1) the NOAA effects range-median values (ER-Ms; Long *et al.* 1995), (2) the lowest Puget Sound apparent effects threshold values (LAETs; Barrick *et al.* 1988), (3) the USEPA acute sediment quality criteria (SQC-

Table 1. A comparison of the TELs to other sediment quality guidelines for coastal and marine waters

Substance	TEL[a]	ER-L[b]	PSDDA-SL[c]	SQC-Chronic[d]	SQO[e]	Number of SQGs[f] comparable to TEL
Metals (SQGs in mg kg^{-1})						
Arsenic	7.24	8.2*	70	8.2*	20	2
Cadmium	0.68	1.2*	0.96*	7.7	1*	3
Chromium	52.3	81*	NG[g]	NG	60*	2
Copper	18.7	34*	81	34*	100	2
Lead	30.2	46.7*	66*	33*	30*	4
Mercury	0.13	0.15*	0.21*	0.01	0.15*	3
Nickel	15.9	20.9*	NG	NG	45*	2
Silver	0.73	1*	1.2*	NG	NG	2
Zinc	124	150*	160*	190*	150*	4
Polychlorinated biphenyls (PCBs; SQGs in µg kg^{-1})						
Total PCBs	21.6	22.7*	130	NG	30*	2
Pesticides (SQGs in µg kg^{-1})						
Chlordane	2.26	0.5	NG	0.3	NG	0
Dieldrin	0.72	0.02	NG	200	NG	0
p,p′-DDD	1.22	2*	NG	NG	NG	1
p,p′-DDE	2.07	2.2*	NG	NG	NG	1
p,p′-DDT	1.19	1*	NG	1.6*	NG	2
Total DDT	3.89	1.58*	NG	1.6*	NG	2
Lindane (gamma-BHC)	0.32	NG	NG	3.1	NG	0
Phthalates (SQGs in µg kg^{-1})						
Bis(2-ethylhexyl)phthalate	182	NG	3100	NG	NG	0
Polycyclic aromatic hydrocarbons (PAHs; SQGs in µg kg^{-1})						
Acenaphthene	6.71	16*	63			

Pesticides (SQGs in $\mu g\,kg^{-1}$)						
Chlordane	2.26	0.5	NG	0.3	NG	0
Dieldrin	0.72	0.02	NG	200	NG	0
p,p′-DDD	1.22	2*	NG	NG	NG	1
p,p′-DDE	2.07	2.2*	NG	NG	NG	1
p,p′-DDT	1.19	1*	NG	1.6*	NG	2
Total DDT	3.89	1.58*	NG	1.6*	NG	2
Lindane (gamma-BHC)	0.32	NG	NG	3.1	NG	0
Phthalates (SQGs in $\mu g\,kg^{-1}$)						
Bis(2-ethylhexyl)phthalate	182	NG	3100	NG	NG	0
Polycyclic aromatic hydrocarbons (PAHs; SQGs in $\mu g\,kg^{-1}$)						
Acenaphthene	6.71	16*	63	2400	50	1
Acenaphthylene	5.87	44	64	NG	60	0
Anthracene	46.9	85.3*	130*	190	10	2
Fluorene	21.2	19*	64	59	50*	2
Naphthalene	34.6	160	210	500	200	0
2-Methylnaphthalene	20.2	70	67	NG	NG	0
Phenanthrene	86.7	240*	320	2400	15	1
Total low molecular weight PAHs	312	552*	610*	NG	NG	2
Benz(a)anthracene	74.8	261	450	1600	130*	1
Benzo(a)pyrene	88.8	430	680	18 000	160*	1
Chrysene	108	384	670	1200	140*	1
Dibenz(a,h)anthracene	6.22	63.4	120	12 000	60	0
Fluoranthene	113	600	630	1600	170*	1
Pyrene	153	665	430*	850	260*	2
Total high molecular weight PAHs	655	1700*	1800*	NG	NG	2
Total PAHs	1684	4022*	NG	NG	NG	1

[a]TEL, threshold effect level (this study).
[b]ER-L, effects range low (Long and Morgan 1990; Long *et al.* 1995).
[c]PSDDA-SL, screening level used in the Puget Sound Dredged Disposal Analysis Program (USACOE 1988).
[d]SQC-chronic, chronic sediment quality criterion (assuming 1% TOC; EqPA; Lyman *et al.* 1987, Pavlou 1987, Hansen *et al.* 1993a,b,c,d,e).
[e]SQO, sediment quality objective (Swain and Nijman 1991).
[f]SQG, sediment quality guideline.
[g]NG, no guideline available.
*Indicates that the SQGs are within a factor of three of the TEL.

Table 2. A comparison of the PELs to other sediment quality guidelines for coastal and marine waters

Substance	PEL[a]	ER-M[b]	LAET[c]	SQC[d]-acute	SLC[e]	Number of SQGs[f] comparable to PEL
Metals (SQGs in mg kg^{-1})						
Arsenic	41.6	70*	57*	16	NG[g]	2
Cadmium	4.21	9.6*	5.1*	24	NG	2
Chromium	160	370*	260*	NG	NG	2
Copper	108	270*	390	54	NG	1
Lead	112	218*	450	840	NG	1
Mercury	0.7	0.71*	0.41*	0.15	NG	2
Nickel	42.8	51.6*	>140	NG	NG	1
Silver	1.77	3.7*	>0.56	NG	NG	1
Zinc	271	410*	410*	560*	NG	3
Polychlorinated biphenyls (PCBs; SQGs in µg kg^{-1})						
Total PCBs	189	180*	130*	NG	36.6	2
Pesticides (SQGs in µg kg^{-1})						
Chlordane	4.79	6*	NG	NG	NG	1
Dieldrin	4.3	8*	NG	NG	NG	1
p,p′-DDD	7.81	20*	16*	NG	NG	2
p,p′-DDE	374	27*	9	NG	NG	0
p,p′-DDT	4.77	7*	34	NG	NG	1
Total DDT	51.7	46.1*	NG	210	505	1
Lindane (gamma-BHC)	0.99	NG	NG	NG	NG	0
Phthalates (SQGs in µg kg^{-1})						
Bis(2-ethylhexyl)phthalate	2647	NG	1900*	NG	NG	1
Polycyclic aromatic hydrocarbons (PAHs; SQGs in µg kg^{-1})						
Acenaphthene	88.9	500	500	NG	NG	0

Pesticides (SQGs in $\mu g\,kg^{-1}$)						
Chlordane	4.79	6*	NG	NG	NG	1
Dieldrin	4.3	8*	NG	NG	NG	1
p,p′-DDD	7.81	20*	16*	NG	NG	2
p,p′-DDE	374	27*	9	NG	NG	0
p,p′-DDT	4.77	7*	34	NG	NG	1
Total DDT	51.7	46.1*	NG	210	505	1
Lindane (gamma-BHC)	0.99	NG	NG	NG	NG	0
Phthalates (SQGs in $\mu g\,kg^{-1}$)						
Bis(2-ethylhexyl)phthalate	2647	NG	1900*	NG	NG	1
Polycyclic aromatic hydrocarbons (PAHs; SQGs in $\mu g\,kg^{-1}$)						
Acenaphthene	88.9	500	500	NG	NG	0
Acenaphthylene	128	640	>560	NG	47.4*	1
Anthracene	245	1100	960	NG	163*	1
Fluorene	144	540	540	NG	101*	1
Naphthalene	391	2100	2100	10 500	414*	1
2-Methylnaphthalene	201	670	670	NG	NG	0
Phenanthrene	544	1500*	1500*	14 000	368	2
Total low molecular weight PAHs	1442	3160*	5200	NG	NG	1
Benz(a)anthracene	693	1600*	1300*	55 000	261	2
Benzo(a)pyrene	763	1600*	1600*	450 000	397	2
Chrysene	846	2800	1400*	115 000	384	1
Dibenz(a,h)anthracene	135	260*	230*	NG	NG	2
Fluoranthene	1494	5100	1700*	9000	644*	2
Pyrene	1398	2600*	2600*	49 500	665	2
Total high molecular weight PAHs	6676	9600*	12 000*	NG	NG	2
Total PAHs	16 770	44 792*	NG	NG	NG	1

[a]PEL, probable effect level (this study).
[b]ER-M, effects range median (Long *et al.* 1995, Long and Morgan 1990).
[c]LAET, lowest apparent effects threshold (PTI, 1988).
[d]SQC-acute, acute sediment quality criterion (assuming 1% TOC; EqPA; Lyman *et al.* 1987, Pavlou 1987).
[e]SLC, national screening level concentration (Neff *et al.* 1987).
[f]SQG, sediment quality guideline.
[g]NG, no guideline available.
*Indicates that the SQGs are within a factor of three of the PEL.

acute; Lyman *et al.* 1987, Pavlou 1987) and (4) the national screening level concentrations (SLCs; Neff *et al.* 1987).

Reliability. The reliability of the SQGs developed in this study was evaluated using the information contained in the ascending data tables. To facilitate this evaluation, a scoring system was devised to integrate information on three distinct attributes of the SQGs, including (1) the incidence of adverse biological effects within the minimal effects range, (2) the incidence of adverse biological effects within the probable effects range and (3) the degree of concordance between the concentrations of sediment-associated contaminants and the incidence of adverse biological effects. Good concordance between these two variables is indicated by marked increases in the incidence of effects over the three ranges of contaminant concentrations (Long *et al.* 1995).

First, a TEL score (TS) was determined for each analyte to quantify incidence of adverse biological effects within the minimal effects range. Specifically, the number of effects data entries and the total number of data entries that were contained within the minimal effects range were determined for each substance. Subsequently, the percent incidence of adverse effects was calculated for each substance by dividing the number of effects data entries by the total number of data entries within the minimal effects range and multiplying this value by 100. A TS of 2, 1 or 0 was assigned if the incidence of adverse biological effects within the minimal effects range was <10%, 10–25%, or >25%, respectively (MacDonald 1994).

Next, a PEL score (PS) was determined for each substance for which SQGs were derived. Consistent with the procedures that were used to determine the TS, the percent incidence of adverse biological effects within the probable effects range was calculated for each substance. A PS of 2, 1 or 0 was subsequently assigned if the incidence of adverse biological effects within the probable effects range was >65%, 50–65% or <50%, respectively (MacDonald 1994).

A concordance score (CS) was determined to assess the degree of agreement between contaminant concentrations and the incidence of adverse biological effects. The CS was calculated by first determining the incidence of adverse biological effects within the possible effects range (i.e. between the TEL and PEL). Next, the percent incidence of adverse biological effects within each of the three ranges of contaminant concentrations were compared. Long *et al.* (1995) indicated that there should be a consistent and marked increase in the incidence of effects within the three concentration ranges. Therefore, the presence of at least a 2-fold increase in the incidence of effects between adjacent ranges of concentrations was used as an indicator of concordance. A CS of 2 was assigned if the percent incidence of adverse biological effects was a factor of 2 or more higher in the probable effects range than in the possible effects range and in the possible effects range compared to the minimal effects range. A CS of 1 was assigned if the factor of 2 difference in the incidence of effects was apparent between only two ranges. A CS of 0 was assigned if there was no apparent concordance between chemical concentrations and the incidence of adverse effects.

Finally, the overall reliability of the guidelines for each substance was evaluated by calculating a total reliability score (TRS; MacDonald 1994). The TRS was determined by calculating the sum of TS, PS and CS for each substance. The guidelines for a substance were considered to have a high degree of reliability if they had a TRS of 6 (i.e. the maximum score). A moderate degree of reliability was assigned when

intermediate scores were obtained for one or two of the parameters or when a low score was obtained for one parameter but high scores were assigned for the other two (i.e. with a TRS of 4 or 5). SQGs with a TRS that was <4 were considered to have a low degree of reliability.

Predictability. The predictability of the TELs and PELs was evaluated separately using independent data from field surveys conducted at sites in Tampa Bay, Pensacola Bay, coastal Gulf of Mexico and offshore Gulf of Mexico. First, the concentration of each substance in each sediment sample was compared to the SQGs. Sediment samples with concentrations of one or more substances that exceeded their respective PELs were predicted to be toxic. Sediment samples were predicted to be non-toxic if the concentrations of all measured substances were below the TELs. Sediment samples with concentrations of one or more contaminants above the TEL but below the PEL (i.e. within the possible effects range) were neither predicted to be toxic nor non-toxic and were not used to evaluate the predictability of the SQGs.

The accuracy of the predictions was then evaluated by comparing them with the results of the biological investigations. The predictability of the PEL was calculated as the ratio of the number of samples that were correctly predicted to be toxic and the number of samples that were originally predicted to be toxic (expressed as a percentage). Similarly, the predictability of the TEL was calculated as the ratio of the number of samples that were correctly predicted to be non-toxic and the number of samples that were originally predicted to be non-toxic (expressed as a percentage). In this assessment, toxic samples were defined as those in which one or more of the measured bioassay end-points were significantly different from control or reference samples.

Results

Numerical SQGs were derived for a total of 34 substances, including nine trace metals, total PCBs, 13 individual PAHs, three groups of PAHs, seven pesticides and bis(2-ethylhexyl)phthalate. The TELs are listed in Table 1 and the PELs are listed in Table 2. Fewer than 20 effects data records were available for a number of additional substances (e.g. tributyltin, pentachlorophenol, dioxins, furans and a suite of pesticides) that are also contaminants of concern in Florida coastal sediments (MacDonald 1994). Therefore, SQGs could not be derived for these substances.

Comparability

The evaluation of the comparability of the SQGs was impaired by the lack of guidelines for certain substances. For example, guidelines from three or more approaches were available for only 19 of the substances for which TELs have been developed and for only 18 of the substances for which PELs had been developed. An adequate number of guidelines were not available for chromium, nickel, silver, bis(2-ethylhexyl)phthalate, several PAHs and most of the pesticides. Nonetheless, the results of this evaluation indicate that many of the SQGs compare favourably to guidelines that were derived for other applications.

The TELs for 17 of the 34 substances were within a factor of three for two or more other guidelines (Table 1). The best agreement was observed for metals and the poorest

agreement was observed for high molecular weight (HMW) PAHs. The TELs were usually lower than values developed using other guidelines, indicating that the TELs could be more protective. The PELs for 14 of the 34 substances were within a factor of three for two or more of the other guidelines listed in Table 2. Once again, greatest agreement among the various guidelines was observed for metals. Relatively poor agreement was observed among the guidelines for pesticides and low molecular weight (LMW) PAHs. As was the case for the TELs, the PELs were generally lower than values developed using guidelines based on other procedures.

Reliability

Using information in the ascending data tables, the reliability of the TELs for 30 substances was found to be relatively high (TS = 2), as indicated by the low incidence of effects (<10%) within the minimal effects range (Tables 3 and 4). This group included the TELs for nine metals, 14 individual PAHs or groups of PAHs, six pesticides and bis(2-ethylhexyl)phthalate. Moderate reliability (TS = 1) was indicated for the TELs for fluorene, dibenz(a,h)anthracene and total PCBs. Low reliability (TS = 0) was indicated for only one substance (total DDT).

The reliability of the PELs generally was lower than that of the TELs. The PELs for 16 substances had a relatively high degree of reliability (PS = 2), as indicated by a high incidence of adverse biological effects (>65%) within the probable effects ranges (Tables 3 and 4). Of the highly reliable PELs, 14 were for individual PAHs or groups of PAHs. The PELs for cadmium and bis(2-ethylhexyl)phthalate were also considered to be highly reliable. A moderate degree of reliability (PS = 1) was indicated for five of the nine metals; a low degree of reliability (PS = 0) was indicated for arsenic, mercury and nickel. The PELs for pesticides and total PCBs had either a moderate or low level of reliability.

A high degree of concordance between contaminant concentrations and the incidence of adverse biological effects was observed for the majority of the SQGs (Tables 3 and 4). The incidence of adverse effects consistently and markedly increased with increasing concentrations for all trace metals except mercury, nickel and silver. Two-fold increases in the incidence of effects between the minimal effects range and possible effects range and the possible effects range and probable effects range were also observed for ten of the 16 individual PAHs and groups of PAHs (Table 4). The concordance scores for three pesticide guidelines were high (CS = 2 for dieldrin, p,p′-DDD and p,p′-DDE), while those for four other pesticides and total PCBs were lower (CS = 0 or 1).

Overall, total reliability scores of 4 or more were calculated for the majority of the guidelines, indicating high or moderate reliability (Table 4). A high degree of reliability (TRS = 6) was indicated for one trace metal (cadmium), ten individual PAHs or groups of PAHs and bis(2-ethylhexyl)phthalate. The SQGs for 16 other substances were moderately reliable (TRS = 4 or 5), including those for six trace metals, five individual PAHs, total HMW-PAHs, dieldrin, p,p′-DDE, p,p′-DDD and p,p′-DDT. The reliability of the guidelines for mercury, nickel, total PCBs, chlordane, lindane and total DDT was lower (TRS < 4).

Predictability

The predictability of the SQGs was evaluated using four independent data sets from the southeastern portion of the United States. In Tampa Bay, Florida, matching sediment

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y, Florida, matching sediment

chemistry and biological effects data were available for 61 sediment samples (Long *et al.* 1994). The analytes in these samples included metals, PAHs, PCBs and a suite of pesticides. Based on a comparison of the sediment chemistry data with the PELs, 46 of these samples were predicted to be toxic (Table 5). A suite of toxicity tests was also conducted on these samples, including a 10 day amphipod (*Ampelisca abdita*) survival test (using bulk sediments) and a 1 hr sea urchin (*Arabacia punctulata*) fertilization test (using undiluted pore water). Based on the results of these toxicity tests, 40 samples were observed to be toxic (i.e. toxicity was observed in one or more tests). Therefore, the predictability of the PELs was 87% (40 out of 46 samples). Only two of the 61 sites were predicted to be non-toxic; the results of the biological tests conducted on these two samples revealed that neither sample was toxic. Thirteen samples were not classified, as the concentrations of all analytes were below the PEL but the concentrations of one or more substances exceeded the TEL (i.e. within the possible effects range).

In Pensacola Bay, data for 20 samples were available for assessing the predictability of the guidelines (E.R. Long, unpublished data). The concentrations of metals, PAHs and a suite of pesticides were measured in each of these samples. Sediment toxicity was assessed using a 10 day amphipod (*A. abdita*) survival test, a sea urchin (*A. punctulata*) fertilization (1 h) and embryo development test (48 h) and a microbial bioluminescence test (Microtox). Based on comparisons of the metals and organics data with the PELs, 12 samples were predicted to be toxic (Table 6). Of these, 11 of the samples were observed to be toxic. Therefore, the predictability of the PELs was 92%. Using the TELs, two samples were predicted to be non-toxic; both of these samples were observed to be toxic. It should be noted that these samples contained elevated levels of one or more substances that could not be identified using a range of analytical techniques and which could have caused or contributed to the toxicity.

As part of the Environmental Monitoring and Assessment Program (EMAP), administered by the US Environmental Protection Agency, matching sediment chemistry and biological effects data were collected from eight areas in the Gulf of Mexico region in 1991 (USEPA, unpublished data). The areas sampled in this survey included Galveston Bay (TX), Matagorda Bay (TX), Mississippi River (LA), Mississippi Sound (LA), Mobile Bay AL), Pensacola Bay (FL), Florida Panhandle (FL) and West Central Florida (FL). Sediment chemistry data were collected on metals, PAHs, PCBs, a suite of pesticides and several additional substances. Sediment toxicity was assessed using acute toxicity (lethality) tests with the amphipod, *A. abdita* (10 days) and the mysid, *Mysidopsis bahia* (4 days). As no statistical evaluation of the toxicity test results were reported, a 20% difference between the survival of test organisms in Gulf of Mexico sediments versus control sediments was assumed to indicate toxicity in this evaluation (USEPA/USACOE 1991, Schimmel *et al.* 1994). Of the 47 samples collected in this survey, three were predicted to be toxic and 16 were predicted to be non-toxic (Table 7). The results of the two toxicity tests indicated that none of the samples that were predicted to be toxic were observed to be toxic (predictability = 0%). In contrast, 15 of the 16 samples that were predicted to be non-toxic were in fact non-toxic (predictability = 94%).

As part of the Minerals Management Services (MMS) Gulf of Mexico Offshore Operators Monitoring Experiment (GOOMEX), sediment chemistry and toxicity data were collected in the vicinity of petroleum exploration and production platforms in the Gulf of Mexico (R.S. Carr, D.C. Chapman, B.J. Prestley, J.M. Biedenbach, L. Robertson

Table 3. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments

Substance	% effects in the minimal effects range (< = TEL[a])	% effects of the possible effects range (>TEL to <PEL[a])	% effects in the probable effects range (> = PEL)
Metals			
Arsenic	2.7	12.9	46.8
Cadmium	5.6	20.1	70.8
Chromium	3.5	15.4	52.9
Copper	9.0	21.9	55.9
Lead	5.8	25.8	58.4
Mercury	7.8	23.6	36.7
Nickel	3.3	8.4	9.4
Silver	6.6	9.8	60.5
Zinc	3.8	27.2	64.8
Polychlorinated biphenyls (PCBs)			
Total PCBs	15.7	36.9	54.9
Polycyclic aromatic hydrocarbons (PAHs)			
Acenaphthene	7.5	29.1	57.4
Acenaphthylene	7.4	13.9	51.4
Anthracene	8.7	20.5	75.0
Fluorene	11.7	20.5	70.0
2-methylnaphthalene	0.0	23.4	81.5
Naphthalene	2.6	19.3	71.2
Phenanthrene	8.0	22.8	77.8
Total low molecular weight PAHs	8.7	19.4	65.6
Benz(a)anthracene	8.7	15.7	78.4

Polychlorinated biphenyls (PCBs)			
Total PCBs	15.7	36.9	54.9
Polycyclic aromatic hydrocarbons (PAHs)			
Acenaphthene	7.5	29.1	57.4
Acenaphthylene	7.4	13.9	51.4
Anthracene	8.7	20.5	75.0
Fluorene	11.7	20.5	70.0
2-methylnaphthalene	0.0	23.4	81.5
Naphthalene	2.6	19.3	71.2
Phenanthrene	8.0	22.8	77.8
Total low molecular weight PAHs	8.7	19.4	65.6
Benz(a)anthracene	8.7	15.7	78.4
Benzo(a)pyrene	8.5	22.1	70.9
Chrysene	9.2	18.8	72.4
Dibenz(a,h)anthracene	15.8	11.6	65.1
Fluoranthene	9.5	20.2	79.7
Pyrene	7.4	19.3	83.0
Total high molecular weight PAHs	9.5	15.0	65.5
Total PAHs	7.3	19.3	76.7
Pesticides			
Chlordane	9.0	12.1	17.0
Dieldrin	3.5	13.2	50.0
Lindane (gamma-BHC)	2.9	21.1	25.6
p,p'-DDD	3.6	10.9	46.2
p,p'-DDE	5.3	16.5	50.0
p,p'-DDT	7.9	4.8	58.6
Total DDT	47.6	25.6	64.0
Phthalates			
Bis(2-ethylhexyl)phthalate	8.5	21.2	66.7

In the ascending data tables for each chemical, entries were identified as effects data entries if an adverse biological effect was reported and concordance was apparent between the observed biological response and the measured chemical concentration.
% effects = (number of effects data entries in the range/total number of data entries in the range) × 100.
[a]TEL, threshold effect level; PEL, probable effect level.

Table 4. An evaluation of the overall reliability of the sediment quality assessment guidelines for priority substances in Florida coastal waters

Substance	TEL[a] score (TS)	PEL[a] score (PS)	Concordance score (CS)	Total reliability score (TRS)	Overall[b] reliability
Metals					
Arsenic	2	0	2	4	M
Cadmium	2	2	2	6	H
Chromium	2	1	2	5	M
Copper	2	1	2	5	M
Lead	2	1	2	5	M
Mercury	2	0	1	3	L
Nickel	2	0	1	3	L
Silver	2	1	1	4	M
Zinc	2	1	2	5	M
Polychlorinated biphenyls (PCBs)					
Total PCBs	1	1	1	3	L
Pesticides					
Chlordane	2	0	0	2	L
Dieldrin	2	1	2	5	M
p,p′-DDD	2	0	2	4	M
p,p′-DDE	2	1	2	5	M
p,p′-DDT	2	1	1	4	M
Total DDT	0	1	0	1	L
Lindane (gamma-BHC)	2	0	1	3	L
Phthalates					
Bis(2-ethylhexyl)phthalate	2	2	2	6	H
Polycyclic aromatic hydrocarbons (PAHs)					
Acenaphthene	2	1	1	4	M
Acenaphthylene	2	1	1	4	M
Antracene	2	2	2	6	H
Fluorene	1	2	1	4	M
Naphthalene	2	2	2	6	H
2-Methylnaphthalene	2	2	2	6	H
Phenanthrene	2	2	2	6	H
Total low molecular weight PAHs	2	2	2	6	H
Benz(a)anthracene	2	2	1	5	M
Benzo(a)pyrene	2	2	2	6	H
Chrysene	2	2	2	6	H
Dibenz(a,h)anthracene	1	2	1	4	M
Fluoranthene	2	2	2	6	H
Pyrene	2	2	2	6	H
Total high molecular weight PAHs	2	2	1	5	M
Total PAHs	2	2	2	6	H

[a]TEL, threshold effect level; PEL, probable effect level.
[b]H, high (TRS = 6); M, moderate (TRS = 4–5); L, low (TRS < 4).

Table 5. *Predictability of the TELs[a] and PELs[a] in Tampa Bay sediments (Long et al. 1994)*

Category	Number of samples per category	Observed number of toxic samples:	Predictability (using one to two

Total reliability score (TRS)	Overall[b] reliability
4	M
6	H
5	M
5	M
5	M
3	L
3	L
4	M
5	M
3	L
2	L
5	M
4	M
5	M
4	M
1	L
3	L
6	H
4	M
4	M
6	H
4	M
6	H
6	H
6	H
6	H
6	H
6	H
6	H
5	M
6	H
6	H
6	H
4	M
6	H
6	H
5	M
6	H

Table 5. Predictability of the TELs[a] and PELs[a] in Tampa Bay sediments (Long *et al.* 1994)

Category	Number of samples per category	Observed number of toxic samples: amphipod test (%)	Observed number of toxic samples: sea urchin test (%)	Predictability (using one to two tests; % correct)
Actual toxicity	61	8 of 61 (13)	50 of 61 (82)	–
Not toxic (<TELs)	2	0 (0)	0 (0)	100 (2 of 2)
Possibly toxic (>TELs; <PELs)	13	1 (8)	10 (77)	–
Toxic (one PEL exceeded)	22	0 (0)	16 (73)	73 (16 of 22)
Toxic (two to five PELs exceeded)	3	2 (67)	3 (100)	100 (3 of 3)
Toxic (six to nine PELs exceeded)	5	1 (20)	5 (100)	100 (5 of 5)
Toxic (>10 PELs exceeded)	16	4 (25)	16 (100)	100 (16 of 16)
Toxic (one or more PELs exceeded)	46	7 (15)	40 (87)	87 (40 of 46)
Toxic (two or more PELs exceeded)	24	7 (29)	24 (100)	100 (24 of 24)

[a]TEL, threshold effect level; PEL, probable effect level.

Table 6. Predictability of the TELs[a] and PELs[a] in Pensacola Bay sediments (E.R. Long, unpublished data)

Category	Number of samples per category	Number of toxic samples: amphipod test (%)	Number of toxic samples: sea urchin test (%)	Number of toxic samples: Microtox test (%)	Predictability (using one to three tests; % correct)
Actual toxicity	–	0 of 20 (0)	13 of 20 (65)	15 of 20 (75)	–
Not toxic (<TELs)	2	0 (0)	1 (50)	2 (100)	0 (0 of 2)
Possibly toxic (>TELs; <PELs)	6	0 (0)	3 (50)	4 (67)	–
Toxic (one PEL exceeded)	2	0 (0)	2 (100)	2 (100)	100 (2 of 2)
Toxic (two to five PELs exceeded)	5	0 (0)	3 (60)	4 (80)	80 (4 of 5)
Toxic (six to nine PELs exceeded)	2	0 (0)	2 (100)	2 (100)	100 (2 of 2)
Toxic (>10 PELs exceeded)	3	0 (0)	2 (67)	3 (100)	100 (3 of 3)
Toxic (one or more PELs exceeded)	12	0 (0)	9 (75)	11 (92)	92 (11 of 12)
Toxic (two or more PELs exceeded)	10	0 (0)	7 (70)	9 (90)	90 (9 of 10)

[a]TEL, threshold effect level; PEL, probable effect level.

Table 7. Predictability of the TELs[a] and PELs[a] in coastal Gulf of Mexico sediments (USEPA, unpublished data)

Category	Number of samples per category	Number of toxic samples: amphipod test (%)	Number of toxic samples: mysid test (%)	Predictability (using one to two tests; % correct)
Actual toxicity	47	7 of 47 (15)	0 of 47 (0)	–
Not toxic (<TELs)	16	1 (6)	0 (0)	94 (15 of 16)
Possibly toxic (>TELs; <PELs)	28	6 (27)	0 (0)	–
Toxic (one PEL exceeded)	3	0 (0)	0 (0)	0 (0 of 3)
Toxic (two to five PELs exceeded)	0	–	–	–
Toxic (six to nine PELs exceeded)	0	–	–	–
Toxic (>10 PELs exceeded)	0	–	–	–
Toxic (one or more PELs exceeded)	3	0 (0)	0 (0)	0 (0 of 3)
Toxic (two or more PELs exceeded)	0	–	–	–

[a]TEL, threshold effect level; PEL, probable effect level.

and S.P. Boothe submitted). Five platforms were sampled during the first cruise and three platforms were sampled during the second cruise. Twenty-five stations were sampled per platform per cruise, with the stations arranged in a radial array around each platform. The sediment chemistry data consisted of a suite of trace metals and petroleum hydrocarbons; however, no chlorinated organic compounds were measured. Both sea urchin (*A. punctulata*) fertilization and embryological development tests were conducted with sediment pore water from the 125 samples from the first cruise, but only the embryological development test was used for the samples collected during the second cruise, as the latter test proved to be more sensitive for assessing the effects of the contaminants present at these platforms.

In the GOOMEX study, 16 of the 200 samples tested were toxic (Kennicutt 1993). Based on comparisons of the sediment chemistry data to the guidelines, 27 of the 200 samples were predicted to be toxic and 50 were predicted to be non-toxic (Table 8). Thirty-seven percent (ten out of 27) of the samples predicted to be toxic (i.e. one or more PELs exceeded) were observed to be toxic. The predictability increased to 58% (seven out of 12) when two or more PELs were exceeded in sediment samples. All of the non-toxic samples with contaminant concentrations that exceeded one or more PELs were predicted to be toxic based on the concentrations of zinc or lead. Ninety-six percent of the 50 samples predicted to be non-toxic were observed to be non-toxic using these very sensitive toxicity tests (Carr and Chapman 1992).

Considering all of the data collected in the Gulf of Mexico region, the results of biological investigations indicated that 59 of the 88 samples that were predicted to be toxic actually were toxic (Table 9). Hence, an overall predictability of 67% was calculated for the PELs. By comparison, 66 of the 70 samples that were predicted to be non-toxic actually were non-toxic to all of the organisms tested. The predictability of the TELs was, therefore, calculated to be 94%. When data from coastal areas of the Gulf of Mexico only were considered, the predictability of the TELs and PELs were 85 and 84%, respectively. It should be noted that slightly more than one-half of the samples collected in these surveys had concentrations of one or more contaminants that fell within the possible effects range; therefore, it was not possible to predict whether or not these samples would be toxic. On average, approximately one-fifth of these samples were observed to be toxic, based on the results of a battery of tests.

Table 8. Predictability of the TELs[a] and PELs[b] in offshore Gulf of Mexico sediments using the sea urchin embryological development test (Carr *et al.* 1995)

Category	Number of samples per category	Number of toxic samples (%)	Predictability (%)
Actual toxicity	200	16 of 200 (11)	–
Not toxic (<TELs)	50	2 (4)	96 (48 of 50)
Possibly toxic (>TELs; <PELs)	123	11 (9)	–
Toxic (one PEL exceeded)	15	3 (20)	20 (3 of 15)
Toxic (two to five PELs exceeded)	12	7 (58)	58 (7 of 12)
Toxic (six to nine PELs exceeded)	0	–	–
Toxic (>10 PELs exceeded)	0	–	–
Toxic (one or more PELs exceeded)	27	10 (37)	37 (10 of 27)
Toxic (two or more PELs exceeded)	12	7 (58)	58 (7 of 12)

[a]TEL, threshold effect level; PEL, probable effect level.

[a]TEL, threshold effect level; PEL, probable effect level.

Table 9. Evaluation of the predictability of the TELs[a] and PELs[a] in southeastern United States sediments

Location	Number of samples correctly predicted as not toxic (%)	Number of samples correctly predicted as toxic (%)
Coastal Gulf of Mexico		
Tampa Bay	2 of 2 (100)	40 of 46 (87)
Pansacola Bay	0 of 2 (0)	11 of 12 (92)
Coastal Estuaries	15 of 16 (94)	0 of 3 (0)
Total coastal Gulf of Mexico	17 of 20 (85)	51 of 61 (84)
Offshore Gulf of Mexico	48 of 50 (96)	10 of 27 (37)
Gulf of Mexico (total coastal plus offshore)	65 of 70 (93)	61 of 88 (69)

[a]TEL, threshold effect level; PEL, probable effect level.

Discussion

The modified weight-of-evidence approach developed here is characterized by a number of attributes that make it attractive for deriving SQGs for Florida coastal waters. Unlike many other approaches to the development of SQGs, the weight-of-evidence approach does not attempt to establish absolute sediment quality assessment values. Instead, the approach delineates ranges of contaminant concentrations that are probably, possibly and not likely to be associated with adverse biological effects. This approach recognizes the uncertainty associated with the prediction of biological effects under a variety of field conditions and relies upon the evidence assembled from numerous independent studies.

One of the more important attributes of the weight-of-evidence approach is its overall practicality. Guidelines for 34 potentially toxic substances were calculated relatively quickly and inexpensively using available data. In addition, by considering matching sediment chemistry and biological effects data from studies conducted in the field, the influence of mixtures of chemicals in sediments is incorporated in the resultant SQGs. This feature increases the degree of environmental realism and, thus, applicability of the guidelines. Furthermore, the information in the BEDS is highly relevant to the guidelines derivation process because it applies to a wide range of biological organisms and end points, incorporates a large number of direct measurements on organisms that are normally associated with bedded sediments and includes many data from various studies conducted in the southeastern United States (including Florida). These attributes are likely to give the SQGs derived using the modified weight-of-evidence approach broad applicability in the southeast, increasing the probability that the guidelines would be appropriate for implementation in Florida.

In addition to the other advantages of the approach, the arithmetic procedures used in this study for calculating SQGs considered the information in both the effects and no effects data sets. Hence, the resultant guidelines were more likely to satisfy narrative objectives. And, in contrast to the apparent effects threshold approach (Barrick *et al.* 1988), the weight-of-evidence procedure does not rely heavily on individual data points. Therefore, outliers do not excessively influence values in the overall guidelines derivation process.

Despite the benefits associated with this approach, a number of limitations were also evident which could restrict application of these guidelines. First, the weight-of-

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evidence approach does not fully support the quantitative evaluation of cause and effect relationships between contaminant concentrations and biological responses. Although information from spiked-sediment toxicity tests and equilibrium partitioning models is included in the BEDS, the weight-of-evidence approach is still largely based on associations between contaminant concentrations and biological responses. Various factors other than concentrations of the contaminant under consideration could have influenced the actual response observed in any given investigation, including (but not limited to) the additive and synergistic effects of co-occurring contaminants. While the assembly of extensive information from numerous estuarine and marine sites across North America into a single database reduces this limitation, the level of uncertainty associated with the resultant SQGs is still not quantified. We are currently investigating the use of toxic units models to reduce uncertainty (i.e. R.C. Swartz, D.W. Schults, R.J. Ozretich, J.O. Lamberson, F.A. Cole, T.H. DeWitt, M.S. Redmond and S.P. Ferraro submitted).

Application of the recommended approach may also be restricted by other limitations on the available information. Presently, few data exist on the chronic responses of marine and estuarine organisms to contaminated sediments. Furthermore, few data are available for some potentially important contaminants of sediments in Florida, including tributyltin, pentachlorophenol, dioxins and furans and various pesticides. These data gaps impair our ability to evaluate the overall applicability of the approach to Florida.

Sediment-associated contaminants can accumulate in the tissues of aquatic organisms and thus have the potential to adversely affect human and non-human consumers of aquatic biota. However, the guidelines developed in this study do not address either the potential for bioaccumulation or the associated adverse effects of bioaccumulation on higher trophic levels.

One shortcoming of the weight-of-evidence approach is associated with the limitations on the data that describe the potential bioavailability of sediment-associated contaminants (Di Toro *et al.* 1990). Large differences in toxicity of sediment-associated contaminants have been reported for relatively small ranges in concentration for total organic carbon (TOC) and/or acid volatile sulphide (AVS) (Swartz *et al.* 1990, Di Toro *et al.* 1991, Adams *et al.* 1992). However, data on sediment grain size, levels of TOC and concentrations of AVS were not provided in most of the reports reviewed in this study. Thus, it was not possible to express the guidelines in terms of the factors that may influence the bioavailability of these contaminants. While reliance on ranges of concentrations instead of absolute values and consideration of the no effects data set reduces this limitation, sediment quality guidelines are less defensible if they do not account for the factors that control bioavailability (e.g. Di Toro *et al.* 1991). For this reason, the SQGs derived in this study were evaluated to determine their comparability, reliability and predictability. Sediment quality guidelines derived using other approaches and being considered for use in national or regional programs should also be thoroughly evaluated before being implemented.

The results of this evaluation indicate that the SQGs can be used with a high or moderate degree of confidence to assess sediment quality. The SQGs for approximately half of the substances were comparable to other guidelines that have been developed using different approaches or different procedures. Additionally, the SQGs for 28 substances had a moderate or high degree of reliability, as indicated by the data contained in the BEDS. Furthermore, the SQGs for 34 substances, when used

collectively, provided predictive tools for correctly classifying marine and estuarine sediments with respect to adverse biological effects. Overall, the predictability of the dry weight-normalized PELs and TELs was >67 and >94%, respectively, indicating that the potential limitations identified previously do not seriously compromise the applicability of the SQGs. Interestingly, the predictability of the PELs was higher (>80%) for sediment samples that had complex mixtures of contaminants than it was for sediment samples that were contaminated by one or two substances only (predictability = 37%). This increased ability to predict toxicity in sediments with complex mixtures is likely because the database, upon which the guidelines are based, primarily contains information from sites with sediments that contained many chemical substances. Therefore, the guidelines are particularly relevant for assessing sediment quality in areas with multiple contaminant inputs.

A critical consideration in sediment risk assessment is the potential for incorrectly classifying sediments. Based on the results of this assessment, it is apparent that there is an approximately 33% probability of incorrectly classifying non-toxic samples as toxic using the PELs (i.e. false positives). In contrast, there was a relatively low probability of incorrectly classifying toxic samples as non-toxic using the TELs (i.e false negatives). Therefore, the guidelines are considered to be conservative tools for assessing contaminated sediments (i.e. they err on the side of environmental protection; see C.G. Ingersoll, P.S. Haverland, E.L. Brunson, T.J. Canfield, F.J. Dwyer, C.E. Henke, N.E. Kemble, D.R. Mount and R.C. Fox (submitted) for a more comprehensive evaluation of type I and type II errors using SQGs). Together, the evaluations of comparability, reliability and predictability indicate that the SQGs are likely to be appropriate for use in a variety of applications and sites in Florida and, perhaps, elsewhere.

The GOOMEX study, conducted in the vicinity of offshore oil and gas exploration and production platforms in the Gulf of Mexico, provided a unique environment for testing the predictability of the SQGs. The primary contaminants of concern were metals (Cd, Cu, Hg, Pb and Zn) and the sampling design provided a gradient in contaminant concentrations along transects extending from the point source discharge. This situation was unique compared with most urban coastal environments where complex mixtures of organic and inorganic contaminants are usually present due to point and non-point sources of pollutants. Compared with the two studies in coastal areas which used the sea urchin pore water tests, the GOOMEX study had a much lower concordance between predicted and observed toxicity. This may be due to the fact that most of the data used to develop the SQGs was obtained from studies conducted near urbanized coastal areas, where complex mixtures of contaminants are more prevalent. The additive and synergistic effects of these co-occurring chemicals are of necessity incorporated into the SQGs. In this unique study in the Gulf of Mexico, where the numbers and classes of chemicals present were limited compared to urbanized coastal areas, the PELs may be overprotective due to the limited additive or synergistic effects of co-occurring contaminants. Another explanation might be that the particular forms of the metals were insoluble or ligands were present which minimized their bioavailability. The TELs, however, provided accurate predictions of non-toxicity in the GOOMEX study.

The PELs accurately predicted sediment toxicity in two out of three studies used to assess the predictability of the SQGs in coastal areas of the Gulf of Mexico. No

concordance was observed between predicted and observed toxicity in the EMAP study; this disparity might be explained by differences in the types of toxicity tests that were used among studies. The EMAP study utilized whole sediment toxicity tests with adult crustaceans, whereas the GOOMEX study used sea urchin pore water toxicity tests. The sea urchin fertilization and embryo development tests with pore water provide estimates of chronic toxicity and uses, as an end-point, responses of a sensitive life stage of a sensitive species. In contrast, the whole sediment assessments used in the EMAP study involved estimates of acute toxicity to adult crustaceans. The PELs appear to be more predictive of chronic, sublethal effects than the more obvious acute, lethal effects. Likewise, the TELs appear to be protective of chronic, sublethal effects, as demonstrated by the high degree of concordance between the TEL predictions and the observed toxicity with the sea urchin tests. These results emphasize the fact that the organisms used in various toxicity tests differ with respect to their ability to estimate effects of sediment-associated contaminants. Therefore, comprehensive sediment quality assessments should employ a battery of biological tests, at least one of which should be sensitive enough to detect chronic, sublethal effects in sensitive species.

The results of this investigation indicate that the SQGs generally provide reliable and predictive tools for assessing coastal sediment quality in Florida and elsewhere in the southeast. However, the SQGs should not be used as stand-alone sediment quality criteria. The applicability of these guidelines in other coastal areas of the United States has not been fully evaluated. For this reason, the predictability of the guidelines derived using the original (Long and Morgan 1990) and modified (this study) approaches will be compared using independent data sets from throughout the United States and Canada. The results of this comparison, which will be published in a subsequent manuscript, will provide additional guidance in the use and applicability of the respective SQGs.

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Disclaimer

The methods and guidelines presented in this report do not necessarily represent the policy of the National Biological Survey, the Florida Department of Environmental Protection or the National Oceanic and Atmospheric Administration.

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Review of

Sediment Quality Objectives for Enclosed Bays and Estuaries of California

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This is a review of the report

Draft Staff Report
Water Quality Control Plan for Enclosed Bays and Estuaries
Part 1. Sediment Quality
State Water Resources Control Board
California Environmental Protection Agency
September 27, 2007

The review is in two parts. The first responds directly to the questions posed in Attachment 2. The second is an Appendix that presents a more complete discussion of the issues and a preliminary analysis of the sediment toxicity data to illustrate the application of mechanistic criteria.

1. Are benthic invertebrates important ecologically relevant receptors to protect from direct exposure to toxic pollutants in sediments within the bays and estuaries of California?

Yes, and the rationale for protecting benthic invertebrates are presented very well in the report.

2. Are multiple lines of evidence appropriate to assess the potential risk to benthic invertebrates from toxic pollutants in sediments within the bays and estuaries of California?

Clearly multiple lines of evidence are required to assess the potential risk to benthic invertebrates from toxic pollutants in sediments. This is the case both within the bays and estuaries of California and for other sites, e.g. streams, rivers and lakes. The report presents the rationale and appropriate citations to the literature supporting this position.

3. Individual lines of Evidence
    a. Are proposed sediment toxicity indicators appropriate for assessing both the potential risk of exposure from toxic pollutants and the biological effects in benthic invertebrates within the bays and estuaries of California?

The analysis of the available toxicity tests and the methodology presented in the report for converting toxicity tests for use in judging the level of toxicity appears to be sound. I find the rejection of the Ampelisca abdita test a little strange since the test is employed widely, but a rationale is presented.

b. Are proposed sediment chemistry indicators appropriate for assessing both the potential risk of exposure from toxic pollutants to benthic invertebrates within the bays and estuaries of California?

The sediment chemistry indicator developed in the report is incomplete. As the report states, there are two general methods available for assessing the potential for toxicity in sediments: empirical and mechanistic. The report embraces the empirical method and dismisses the mechanistic method in a few sentences. In Section 5.5.3.2 "What chemistry indicators should be used?" the reasons are given

> "Mechanistic SQGs based on equilibrium partitioning were not included for several reasons. Data for some of the key parameters needed to apply the mechanistic guidelines (e.g. sediment acid volatile sulfides and simultaneously extracted metals) were not available. In addition chemistry data were not available for all the potential toxicants in the samples, which limited the predictive ability of the guidelines for organics. Previous analyses using Southern California data showed that these limitations significantly affected mechanistic SQG performance; application of a partial suite of mechanistic SQGs for organics resulted in poor predictive ability (Vidal and Bay 2005)."

However *both* empirical and mechanistic methods are incomplete. Neither method can predict with more than a modest degree of certainty the outcome of a toxicity test on a sediment from the field that is contaminated with many, and possibly unknown and unmeasured contaminants. Fig. 1 presents the results of the analysis from "Comparative Sediment Quality Guideline Performance For Predicting Sediment Toxicity In Southern California, USA" by Vidal and Bay 2005.

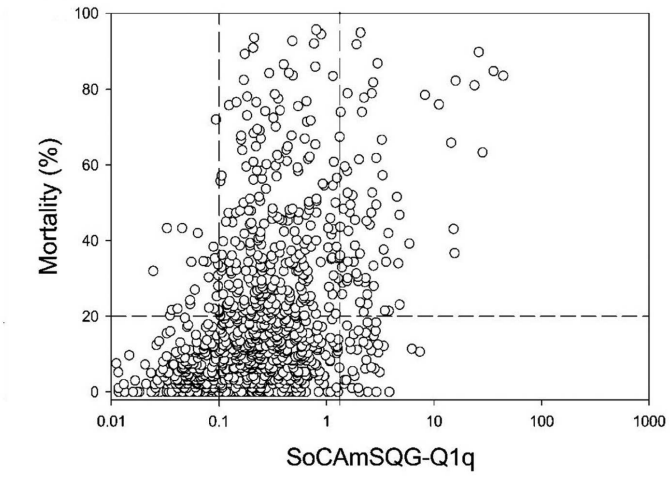

Figure 1

The empirical criteria predicts the lack of toxicity reasonably well (SoCAmSQG-Q1 < 0,1) but fails to discriminate between toxic and non toxic sediments at the same value of SoCAmSQG-

Q1q for the bulk of the data in the range of 0.1 to 1.0. The mechanistic criteria as evaluated by Vidal and Bay appeared to have no predictive ability at all in this data set.

I have prepared an appendix attached to this review that discusses these issues in more detail. It illustrates the applicability of mechanistic criteria to the available data to demonstrate their utility, even if the necessary data for a complete and rigorous application are not available. As demonstrated in the appendix, the role of mechanistic criteria is not to predict toxicity. For the reasons given above and as presented in more detail in the appendix, the role of mechanistic criteria is to determine if the observed toxicity can be explained by known modes of bioavailability and toxic mechanisms.

The results can be used to judge whether the chemical cause of the toxicity for particular sediment is likely to be metals, PAHs and other narcotics, or the pesticides that have been measured. The alternative is that none of these classes of chemicals appear to be the cause of the observed toxicity and the situation is quite uncertain. If the later is the case, then the result of the best professional judgment assessment of the situation would change to be very uncertain, regardless of the level of chemical contamination. Also, in my opinion, more information about the toxic sediment should be collected so that a more secure decision can be made.

Therefore, both mechanistic and empirical criteria should be used to judge the extent of toxicity that is likely due to chemicals, and if the chemical data are consistent with known measures of bioavailability and modes of chemical toxicity. Ignoring mechanistic criteria is not employing the best available science to support regulatory judgments. Mechanistic criteria have been developed and validated from very large datasets. A comprehensive review with citations to the primary literature is available (Di Toro et al., 2005). They are based on quantitative mechanistic models that have been published over the years in the peer reviewed literature, are highly cited, and have been tested by numerous independent investigators. They provide a framework for understanding chemical causes of sediment toxicity, and can be used to discriminate between two important cases: (1) we understand the chemical cause of the observed toxicity; (2) we do not, at our present level of understanding. Empirical criteria cannot provide this important additional information.

c. Are proposed benthic community indicators appropriate for assessing the biological effects through benthic community condition within the bays and estuaries of California?

The report presents the rationale and methodology for selecting the benthic community indicators and they appear to be sound.

4. Is the integration framework appropriate for determining if a station meets the narrative objective?

The integration framework – the quantification of best professional judgment (BPJ) – is to be commended. It produces a specific outcome for the data to be evaluated. The test of the method by experts on a small dataset is a nice demonstration of its utility in quantifying BPJ and making it applicable to specific sediment.

I would suggest one further test. Evaluate the entire dataset for which the necessary triad information is available. What proportion of the tested sediments is in which level of concern? There are a number of arbitrary cutoff levels in the framework, and it is important to know if these choices trigger many highly toxic sediments. A criterion that is too restrictive and triggers too many false positives is not a useful regulatory tool.

5. Is the implementation of the narrative SGO appropriate given the limitations of the individual tools and potential uncertainty associated with sediment quality assessment?

I would strongly recommend the inclusion of the results of an analysis of the data using mechanistic criteria for the purposes of determining the probable cause(s) of toxicity, or whither the cause is unknown. An example application is included in the appendix to this review.

1. Are there any additional scientific issues that are part of the scientific basis of the proposed rule not described above?

I would recommend that a report be prepared that documents the calculations that lead to the LRM in the report so that the analysis can be reproduced, including the analysis leading to Table 2 from Direct Effects Calculation

In order to apply mechanistic criteria without the approximations used in the appendix, certain data are required. Although the historical data may not include the appropriate measurements, all future data collection should include at least: SEM and AVS for a proper assessment of metal toxicity; a complete suite of PAHs including alkylated PAHs and sediment organic carbon to evaluate PAH toxicity. Not requiring such data is not consistent with using the best science.

Section 5.7.4. The staff recommendation is to apply the narrative SQGs to NPDES permits as receiving water limits. Unless mechanistic criteria can successfully identify the chemical causes of the toxicity it is not possible to establish receiving water limits. As discussed above it is the universally agreed that empirical criteria cannot be used to identify the chemical causes of toxicity.

2. Taken as a whole is the scientific portion of the proposed rule based upon sound scientific knowledge methods and practices?

With the exception of the exclusion of mechanistic criteria for judging the possible chemical causes of toxicity – and this is a glaring problem – the implementation is based on sound scientific knowledge methods and practices.

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Appendix 1

Empirical and Mechanistic Criteria

To put my review in context, I will quote from the paper "Comparative Sediment Quality Guideline Performance For Predicting Sediment Toxicity In Southern California, USA" (Vidal and Bay, 2005), cited in the report (p76), which examines these issues. First, the nature of the two methods:

> "Sediment quality guidelines can be classified in two main categories based on the approach used to derive their values: empirical and mechanistic. Empirical SQG approaches are based on the statistical analysis of large databases of synoptic sediment chemistry and toxicity data to identify chemical concentrations associated with various levels of biological effects. Examples of this type of SQG include the effects range–low and effects range–median (ERM) values, which are concentrations corresponding to the 10th and 50th percentiles of the distribution observed in toxic samples, respectively [2]. Variations in chemical speciation and bioavailability are not directly addressed in empirical SQGs; such effects are indirectly incorporated into these guidelines through the use of a database containing samples from diverse locations and sediment types. Empirical SQGs have two major practical advantages: they can be calculated for a large number of contaminants, and only routine chemical analysis data are needed for their application. "
>
> "The second principal type of SQG approach includes values based on mechanistic models that incorporate factors that affect the bioavailability of chemicals in the sediment. Mechanistic SQGs may incorporate the effects of sediment organic carbon or sulfides (for metals) on the equilibrium partitioning of contaminants and also use laboratory dose–response models to account for the effects of multiple contaminants [3–5]. Sediment quality guidelines based on equilibrium partitioning (EqP) for organics have been developed for selected pesticides and organics [6–8]. The EqP for organics theory assumes that nonionic chemicals in sediment partition between the organic carbon present in the sediment as well as in the interstitial (pore) water and the benthic organisms living on the sediment. At equilibrium, if a concentration is known in one of the phases (e.g., sediment), then the other ones can be predicted [6]. By accounting for variations in bioavailability and mixture effects, mechanistic SQGs have a greater ability relative to empirical SQGs to determine the specific contaminants responsible for toxicity. Mechanistic SQGs often require more extensive chemical data, and published values are not available for many contaminants, relative to empirical SQGs."

This is a correct characterization of the current understanding of the nature and appropriate use of the two methods. The report embraces the empirical methods and dismisses the mechanistic methods in a few sentences.

> "5.5.3.2 What chemistry indicators should be used? … Mechanistic SQGs based on equilibrium partitioning were not included for several reasons. Data for some of the key parameters needed to apply the mechanistic guidelines (e.g. sediment acid volatile sulfides and simultaneously extracted metals) were not available. In addition chemistry data were not available for all the potential toxicants in the samples, which limited the predictive ability of the guidelines for organics. Previous analyses using Southern California data showed that these limitations significantly affected mechanistic SQG performance; application of a partial suite of mechanistic SQGs for organics resulted in poor predictive ability (Vidal and Bay 2005)."

I regard this dismissal as premature and potentially dangerous. There has been much discussion in the literature and at meetings about the appropriate uses of empirical and mechanistic guides (Wenning and Ingersoll, 2005). The empirical guidelines suggested in this report are based on fitting a logistic probability model to large sets of amphipod mortality data sets collected in California. An equation is developed for each measured potential toxicant in the sediment. Then these probabilities are combined to make predictions of results of these The limitations of such a procedure are well known. To quote from Vidal and Bay, 2005

> "The results of these analyses showed that exceedances of individual empirical chemical guidelines are unreliable indicators of toxicity and do not necessarily indicate the cause of toxicity. For example, the mean SQGQ1q and mean ERMq had similar nontoxicity efficiency and specificity values, yet the mean SQGQ1q uses only nine chemicals in comparison to the 24 used for the mean ERMq. The presence of many contaminants in a sediment sample and the high degree of correlation among them indicates that most empirical SQG values should not be used in isolation but rather be used in combination to provide an overall indication of the potential for adverse effects (e.g., likely to be toxic or nontoxic). The exceedance of an individual empirical SQG value is not an indication that a chemical is toxic to organisms. Other studies have also suggested caution in the use of individual chemical SQG values when assessing sediment quality [14,16]. "

The Regression Model

The California regression model is based on the log logistic equation (page 13 of Appendix A and page 2 of Direct Effects Calculation)

$$p = \exp(b_0 + b_1 \log_{10}(c))/(1+ \exp(b_0 + b_1 \log_{10}(c)) \quad (1)$$

It can be shown that this equation is equivalent to the more intuitive formulation

$$p = 1/(1 + (EC50/c)^{\beta}) \quad (2)$$

where

$$\beta = b_1 /\ln(10) \quad (3)$$

$$EC50 = \exp(-b_0/ \beta) \quad (4)$$

The EC50 is the concentration at which a 50% mortality is predicted and β is the usual slope parameter.

The example in the Direct Effects Calculation can be used to check these equations.
For cadmium: c = 0.15 mg/kg, b_0 = 0.2894, b_1 = 3.1764 and p = 0.09. Using the above equations: β = 1.38, EC50 = 0.81 mg/kg and p = 0.09 as before. Note that the EC50 is approximately 1 mg Cd/kg by visual inspection of Fig. 2 in Direct Effects Calculation, which is consistent with EC50 = 0.81 mg/kg calculated above.The parameters for the other chemicals are listed below

Table 1
(Table 2 from Direct Effects Calculation and EC50 and β)

	units	b_0	b_1	β	EC50
Cd	mg/kg	0.2894	3.1764	1.38	0.81
Cu	mg/kg	-5.5931	2.5885	1.12	144.79
Pb	mg/kg	-4.7228	2.8404	1.23	46.00
Hg	mg/kg	-0.0618	2.6837	1.17	1.05
Zn	mg/kg	-5.1337	2.4205	1.05	132.11
HPAH	ug/kg	-8.1922	1.9995	0.87	12506.17
LPAH	ug/kg	-6.8071	1.8827	0.82	4126.72
Alpha Chlordane	ug/kg	-3.408	4.457	1.94	5.82
Dieldrin	ug/kg	-1.8344	2.589	1.12	5.11
Trans Nonachlor	ug/kg	-4.259	5.3135	2.31	6.33
Total PCBs	ug/kg	-4.4144	1.4837	0.64	944.64
4-4-DDT	ug/kg	-3.5531	3.2621	1.42	12.28

The Basis for the Model

The model parameters (b_0 and b_1, or equivalently EC50 and β) are based on regression fits to the toxicity and chemical data set assembled for this purpose. The report, appendices, and supplementary information do not contain the data and procedures from which these parameters were derived. In an attempt to understand the procedure in more detail, I have attempted to reproduce the fitting procedure. The Access database StatewideSQO_11_17_06.mdb is available on the web. I retrieved the Eohaustorius estuarius (EE) mortality data and the corresponding chemistry. It was not clear what data was used in generating the report values and I did not have the time completely understand this very large database. I restricted the retrieval to "SP" (survival percentage) and "SD_RESULT" (not replicates etc.) which seemed reasonable choices. One of my recommendations is that a report be prepared that documents the calculations that lead to the LRM in the report so that the analysis can be reproduced. Nevertheless the results of this analysis are very instructive.

This analysis will focus on cadmium as an illustration. The Cd data are presented below in Fig. 1.

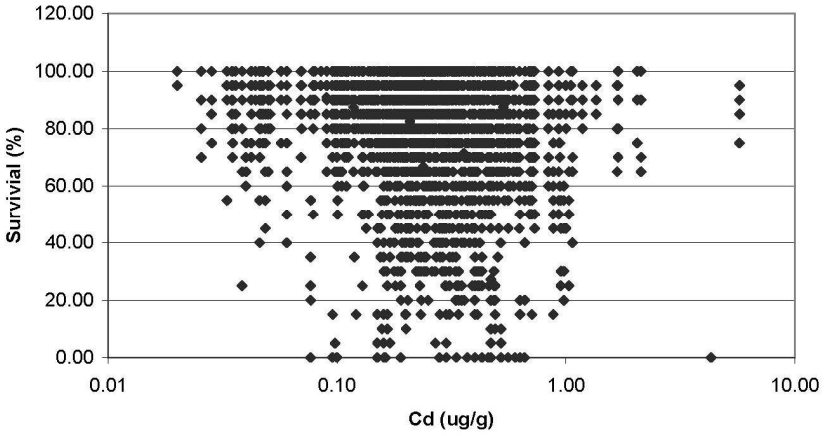


Figure 1

The other metals and PAH data are shown in figures 11 and 12 in the Figure appendix. The data all share a common feature. At low concentrations there is mostly >80% survival indicating no toxicity. At higher concentrations, some samples are not toxic (>80% survival) and others are highly toxic (0% survival). Note that these two extremes can occur *at the same cadmium concentration!* This is the central problem in understanding the toxicity of chemicals in field collected sediments with multiple contaminants. The difficulty is that it is not clear that Cd is causing toxicity in any of these sediments since bioavailability is not accounted for in empirical criteria. It is mechanistic criteria that strive to causally relate a chemical concentration to a toxic response.

This idea behind logistic regression models is to see if it is more probable that as the Cd concentration increases, the survival percentage increases. Fig. 3 presents the results of a fit of the logistic regression equation (2) to the data. The logistic equation using the parameters in Table 1 is also shown. A fit to the data produces an almost flat relationship, indicating that there is virtually no relationship between percent survival and Cd concentration. Yet the logistic equation using the Table 1 parameters seem to indicate a strong relationship.

The reason is, I think, that the data are prescreened before the logistic equation is fit. The procedure is described in Field et al., 2002.

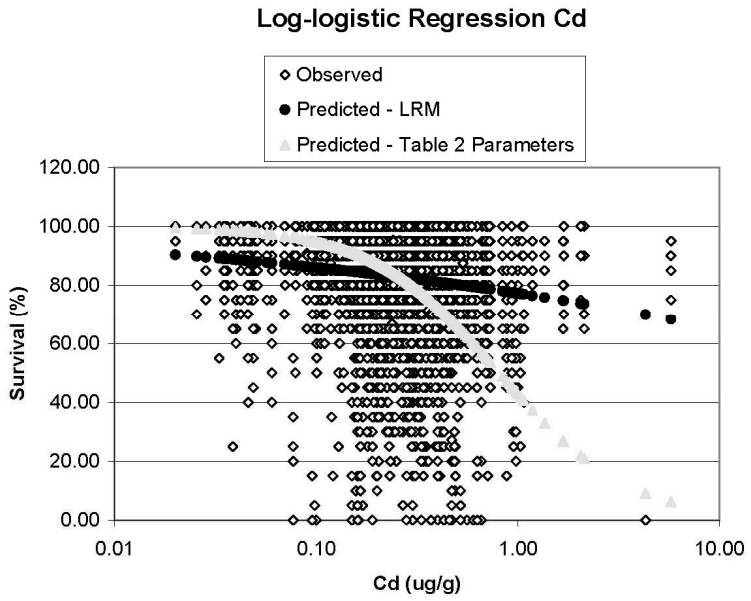


Figure 2

"The presence of multiple contaminants, many of which may be present at very low concentrations, frequently complicates evaluating the relationship between the concentration of an individual contaminant and toxicity in field-collected sediments. Consequently, the data for samples that were identified as toxic in this investigation were further screened before being used to develop the logistic models for each individual contaminant [5]. This screening process excluded toxic samples in which the selected contaminant was unlikely to contribute substantially to the observed toxicity. Following the general screening approach used by Ingersoll et al. [12] and similar to that used by others [1,7,13], the concentration of the selected chemical in each toxic sample was compared with the mean of the concentration of that substance in the nontoxic samples collected in the same study and geographic area. If the concentration of a chemical in an individual toxic sample was less than or equal to the mean concentration of that chemical in the nontoxic samples from that study area, it was considered unlikely that the observed

toxicity could be attributed to that chemical. Therefore, these toxic samples were not included in the screened data set used for developing the logistic model for that chemical. All nontoxic samples were included in these analyses."

An example of the importance of pre-screening the data is shown in Fig. 4 from Field et al, 1999. Before screening, there is virtually no relationship between probability of toxicity and phenanthrene concentration. After prescreening, there is a very nice relationship. Thus the role of pre-screening is critical to the development of LRMs.

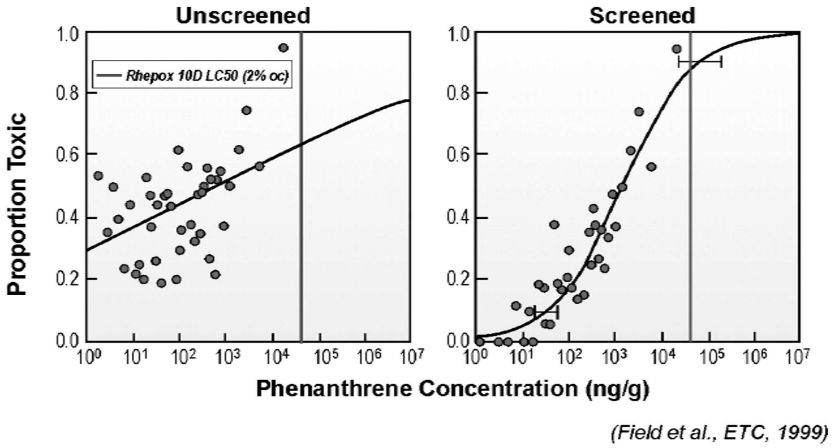


Figure 3

Following this procedure, the median concentration of all nontoxic (survival >80%) samples was found (Cd = 0.26 mg/kg). Then all toxic samples (survival >80%) for which Cd < 0.26 mg/kg were removed. The result is shown in Fig. 5. Since the samples that exhibited toxicity at low Cd concentrations (the samples in the lower left quadrant) have been removed, there is now a relationship between toxicity and Cd concentration. A fit of equation (2) to the screened data is now closer to the result using the Table 1 parameters. Since the methodology used to derive the results in the report are not available, it is not possible to understand why there is still a discrepancy. Nevertheless, it is clear that the pre-screening of the data is a critical part of the analysis.

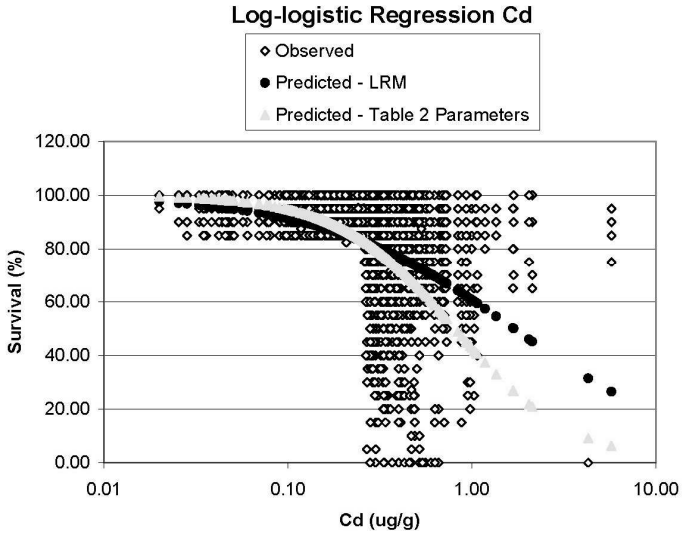


Figure 4

Critique of the Logistic Regression Model (LRM)

Consider the situation when the logistic regression model (LRM) is applied to a new sediment sample. The probability of survival that is computed from the Cd concentration uses the curve derived from the data in Fig. 5. But applying that curve presupposes that the new data comes from the prescreened data set, i.e. it is known a priori that whatever toxicity the sample might exhibit is not due to Cd if the Cd concentration is low. But there is no way of actually knowing that is the case for the new sample at hand. It is, rather, an assumption upon which the method is based. Also note that this result is not peculiar to cadmium. All the toxicity-chemistry data share the same general pattern, and all are pre-screened to produce the LRM.

Another interesting feature of the LRM is that the EC50s for the metals, which are derived from the screened dataset, are comparable to the median concentrations of the metals in the entire dataset. Fig. 6 presents the ratio of the EC50 (Table 1) to the median concentrations computed from the entire data set and also for the non-toxic samples. The ratio ranges from 1 to 4, indicating that the EC50 used in the LRM is a measure of the general level of contamination of the sediments in the dataset. Also the β's are roughly the same. This suggests that for the metals at least, the LRMs are modeling the extent of contamination. They predict low toxicity if the level of metal concentration is well below the median concentration in the datasets.

This is not an unreasonable way to predict *lack* of toxicity for relatively clean, i.e. uncontaminated, sediments. However, it is not much of a guide for predicting the actual toxicity if the level of contamination is larger. The reason the logistic model "fits the data" is that the troublesome data – those showing toxicity at low concentrations – are removed by the pre-screening procedure.

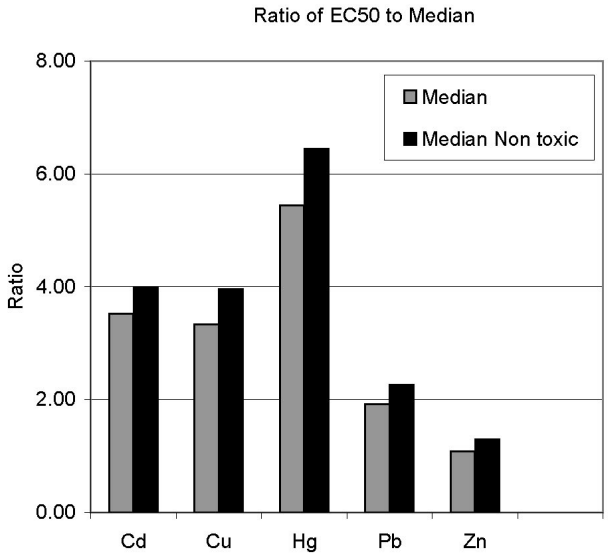


Figure 5

Use of Mechanistic Models

Mechanistic models attempt to relate toxicity to the bioavailable fraction of the chemicals in sediments. The most well developed of there are for mixtures of metals (Ankley et al., 1996, USEPA, 2005) and mixtures of PAHs (Di Toro & McGrath, 2000, USEPA, 2003). They use the Equilibrium Partition Model (Di Toro et al., 1990) as the general framework and apply toxicity mixture and partitioning models to predict the toxicity of single chemicals and chemical mixtures. The models have been validated using spiked sediments (Berry et al., 1996) for which the toxic chemical(s) are not in doubt. Additionally field datasets have been employed that are heavily contaminated with either metals (Hansen et al., 1996) or PAHs (Di Toro & McGrath, 2000) for which the chemicals causing the toxicity can be reasonably assumed to be known.

It has been found that for the large dataset employed for establishing the empirical criteria in this report, the mechanistic criteria do not appear to be as predictive as the empirical criteria. Fig. 7 presents the results of the analysis from Vidal and Bay 2005.

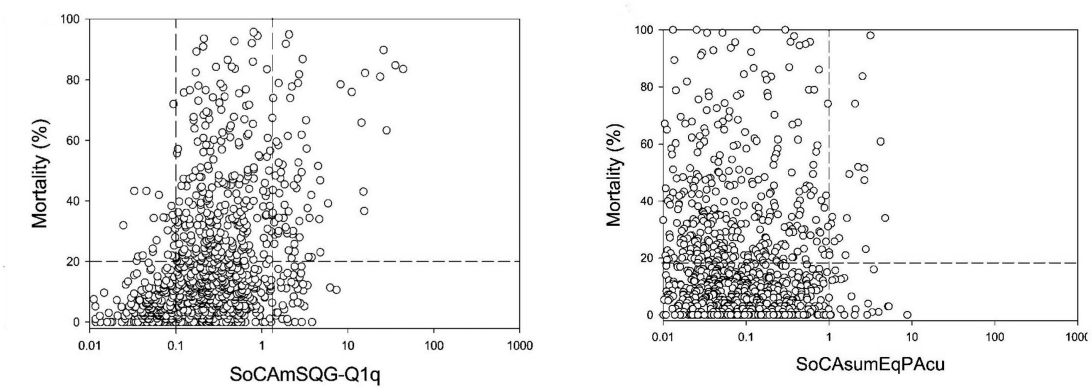


Figure 6 from Vidal and Bay 2005

The SoCAmSQG-Q1q model has very much the same pattern of predictive power as the individual datasets (Fig. 5). For a low level of contamination there is only a control level of mortality. At higher levels of contamination there are both toxic and non-toxic sediments at the same level of contamination (the x-axis). By contrast the EqP comparison shows no discrimination.

There are a number of possible reasons for the failure of the EqP based predictions Certainly one important problem is the lack of the appropriate measure of the critical metal binding parameter acid volatile sulfides (AVS) (Di Toro et al., 1990, 1992) in the majority of sediments in the dataset. The second is the lack of measurements for all the significant PAHs that may be present (McGrath & Di Toro 2000). Finally, and the most vexing problem, is the lack of measurements for other compounds that may be causing toxicity. Nevertheless, the EqP models can be very useful in understanding the possible causes of toxicity.

SEM-AVS Model of Metal Toxicity

For metal toxicity, it has been shown that if the molar sum of the metal concentrations that is simultaneously extracted (∑SEM) with the AVS is less than the AVS concentration, i.e. ∑ SEM – AVS < 0 no toxicity is expected. This has been demonstrated using acute and chronic laboratory spiked and field deployed spiked sediments (Di Toro et al., 2005). SEM data are not available but the molar sum of the total extracted metals (Total Metal = Cd + Cu + Ni + Pb + Zn) are available and inferences can be drawn from these concentrations. Fig.8 presents the data.

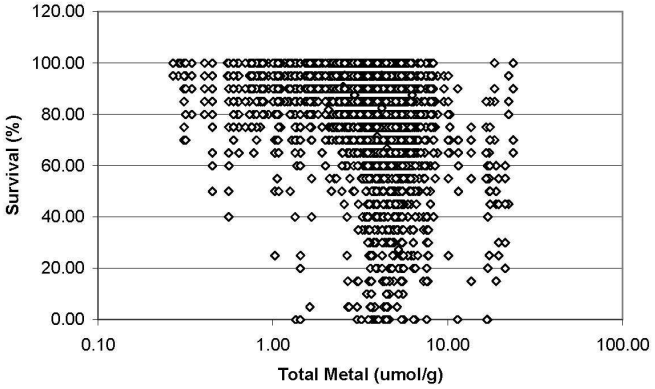


Figure 7

The pattern of the data is not much different from either Cd (fig 2) or the other metals (Figs. 11-12). However the difference is that this distribution can be interpreted in terms concentration of AVS in sediments. For example, little mortality is seen for total metal concentrations < 2 umol/g. If the AVS in all the sediment samples were at least 2 umol/g, not a large amount of AVS for muddy sediments, then the lack of toxicity due to metals would be expected. If AVS concentrations were available for all the data, then metal toxicity could be unambiguously ruled out for those sediment for which Total Metal – AVS < 0, since this would guarantee that $\sum$ SEM – AVS < 0.

There is a small amount of AVS data in the database for which Total Metal – AVS can be calculated and compared to observed mortality. These are shown in Fig. 9. Most of the toxic sediments have AVS concentrations greater than Total Metal, i.e. Total Metal –AVS < 0. Since Total metal > $\sum$ SEM, the data would plot further toward the negative values if $\sum$ SEM were available. This would indicate that in these sediments AVS is greater than $\sum$ SEM and it is unlikely that metals are causing toxicity in this subset of the database. The point is that a judgment can be made about the likely cause of toxicity in these sediments that is not possible using the empirical criteria.

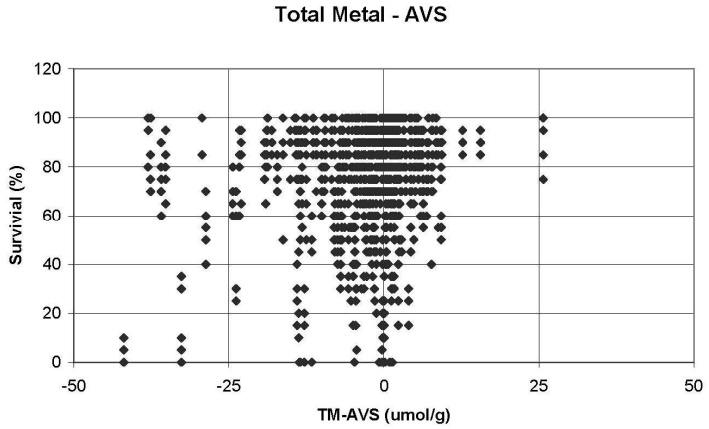


Figure 8

Target Lipid Model of PAH Mixture Toxicity

An EqP model has been developed for mixtures of PAHs is sediments, based on the Target Lipid Model of Narcosis Toxicity (Di Toro et al., 2000). The Criteria corresponding to chronic effects and 10 day Rhepoxynius abronius survival are listed in Table 2. The average and standard deviation of criteria for low (LPAH) and high (HPAH) molecular weight PAH sums as well as total PAH are listed. The toxicity of a mixture is found by summing the toxic units – the ratio of the concentrations to the criteria in Table 2 -- comparing the results to one toxic unit for 50% effect. To a good approximation, the same result is obtained be evaluated by summing the organic carbon normalized molar concentrations of PAHs and comparing the sum to the average criteria. The reason is that the organic carbon normalized sediment criteria for the individual PAHs do not vary very much. For example, the criteria vary from 16.18 to 21.96 umol/gOC for the R. abronius LC50s. An explanation based on the equations for toxic units is available (Di Toro & McGrath, 2000).

Table 2

PAH Sediment Criteria for Chronic Effects and 10 day Rhepoxynius abronius Survival

Chemical	CAS number	MW (g/mol)	Log Kow	Chronic EC50 (umol/gOC)	R. abronius LC50 (umol/gOC)
Acenaphthylene	208968	152.2	3.22	5.03	16.18
Naphthalene	91203	128.19	3.36	5.09	16.38
1-Methylnaphthalene	90120	142.2	3.84	5.31	17.08
2-Methylnaphthalene	91576	142.2	3.86	5.32	17.11

Acenaphthene	83329	154.21	4.01	5.39	17.34
Fluorene	86737	166.2	4.21	5.48	17.64
2,6-Dimethylnaphthalene	581420	156.23	4.37	5.56	17.89
Anthracene	120127	178.2	4.53	5.64	18.15
Phenanthrene	85018	178.2	4.57	5.66	18.21
2,3,5-Trimethylnaphthalene	2245387	170.26	4.86	5.8	18.68
LPAH				5.43(0.25)	17.5(0.80)
Pyrene	129000	202.26	4.92	5.83	18.78
1-Methylphenanthrene	832699	192.26	5.04	5.89	18.97
Fluoranthene	206440	202.26	5.08	5.92	19.04
Benzo[a]anthracene	56553	228.29	5.67	6.23	20.05
Chrysene	218019	228.29	5.71	6.25	20.12
Benzo[a]pyrene	50328	252.31	6.11	6.47	20.84
Perylene	198550	252.31	6.14	6.49	20.89
Benzo[e]pyrene	192972	252.32	6.14	6.49	20.89
Benzo[b]fluoranthene	205992	252.32	6.27	6.56	21.13
Benzo[k]fluoranthene	207089	252.32	6.29	6.58	21.17
Benzo[ghi]perylene	191242	276.34	6.51	6.7	21.58
Dibenz[a,h]anthracene	53703	278.35	6.71	6.82	21.96
Indeno[1,2,3-cd]pyrene	193395	276.34	6.72	6.83	21.98
HPAH				6.39(0.34)	20.6(1.1)
TPAH				5.97 (0.57)	19.2(1.84)

The total PAH data in units of umol/gOC is presented in Fig. 10. It is computed from the low (LPAH) and high (HPAH) molecular weight PAH data using average molecular weights for these classes, and the organic carbon concentration of the sediment, which is in the database.

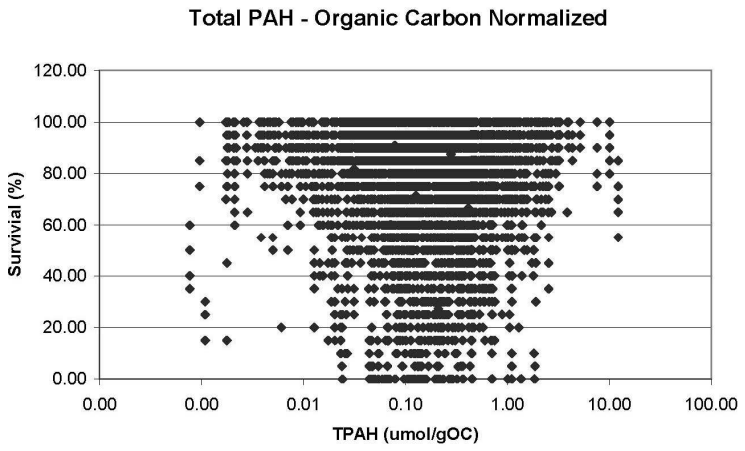


Figure 9

The data has the same shape as the previous chemicals, but as with the metals data, the appropriate toxicity threshold is known. For the 10 day Rhepoxynius abronius survival the average LC50 is 19.2 umol/gOC (TPAH in Table 2). No sediment in Fig. 10 appears to exceed this threshold so it appears that PAHs are not the cause of the toxicity in any of these samples.

There is a significant problem, however, with applying this logic to these data. The criteria apply to the sum of *all* PAHs. But the available data are for only the PAHs in bold face type in Table 2. In particular the alkylated PAHs, which are primarily associated with petroleum contamination, can be a large component of the TPAH and these are not being adequately measured. For these data there is only one representative component 2-methylnaphthalene. Thus it is possible that the total PAH concentration in the sediments could be larger.

The conclusion of this analysis is either that PAHs are not the cause of toxicity in these sediments, or there is large fraction of PAHs that are not being measured, that are contributing to toxicity.

Summary of Empirical and Mechanistic Model Applications

The purpose of this appendix is to examine the utility of empirical and mechanistic models in the evaluation of toxicity of sediment samples. The empirical models estimate the probability of observing toxicity based on the level of contamination. When the sediments have low levels of most contaminants, they predict that the sediment will not be toxic. This conclusion is almost forced by the pre-screening procedure. As levels increase the prediction is that toxicity becomes more likely. But it should be clear from the above analysis that the *cause(s)* of the toxicity cannot be judged from empirical criteria. They are simply responding to the increasing level of overall contamination. The higher the overall level of contamination, the more like it is that toxicity will be found.

The mechanistic criteria can make predictions about which classes of chemicals are possibly involved in the observed toxicity. If the AVS exceeds the total metal concentration, metal toxicity is almost surely not present. If the organic carbon molar sum of the PAHs in the sediment, including the alkylated compounds, is less than the appropriate LC50 for the species being tested, e.g. 19.2 umol/gOC for *Rhepoxynius abronius* survival, then PAHs are almost surely not the cause of toxicity.

If neither metals, nor PAHs are the causes of toxicity, and similar screening calculations can be made for other measured constituents, this information can be included in the next step in the investigation. At least, we know we either know or do not know the causes of toxicity. If the causes are known, we can proceed with confidence. If the cause is unknown, than a completely different approach is warranted. This is crucial information to making judgments about whether sediments are toxic due to chemical contamination, and whether the information at hand is consistent with known chemical modes of toxicity in sediments.

Figure Appendix

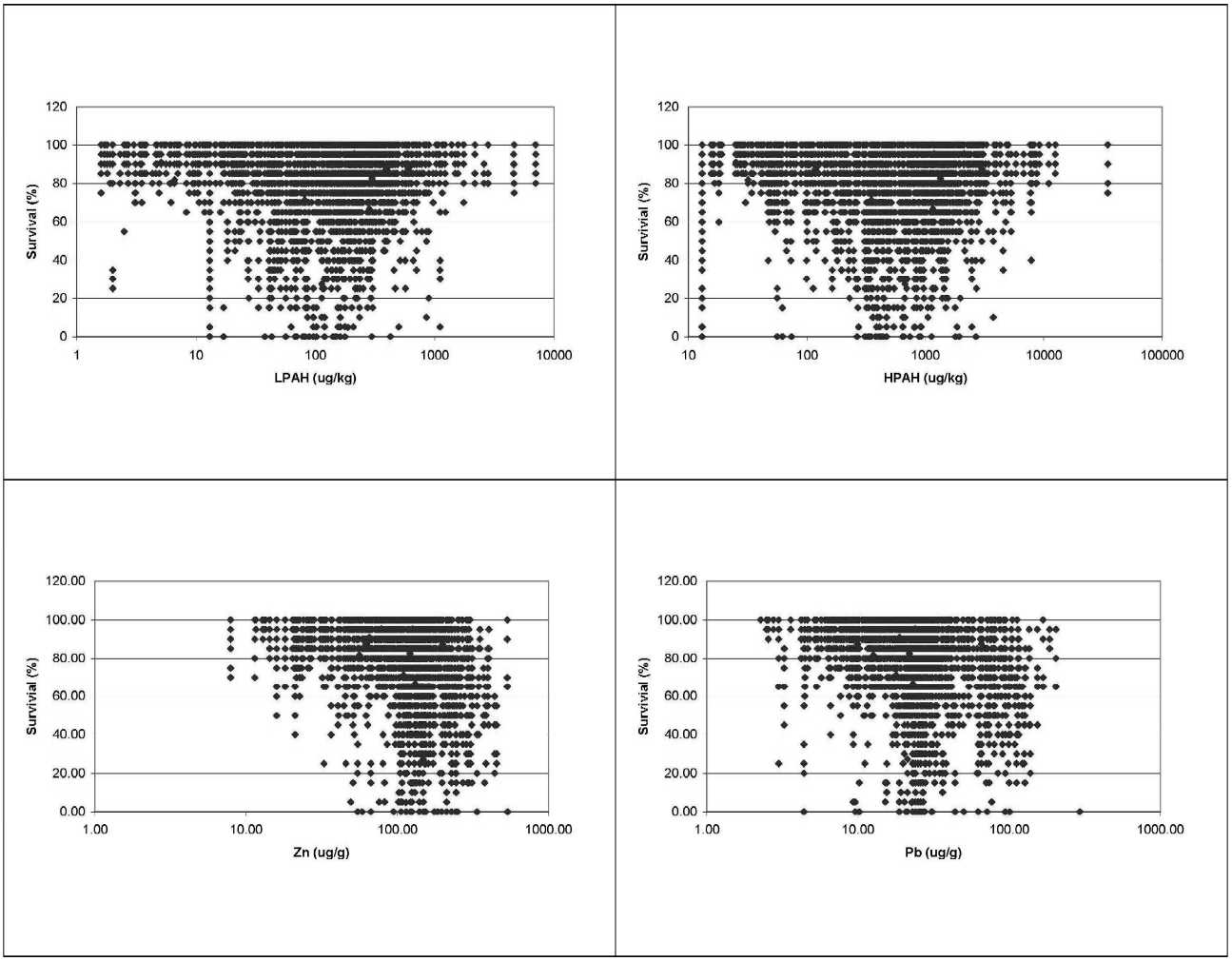
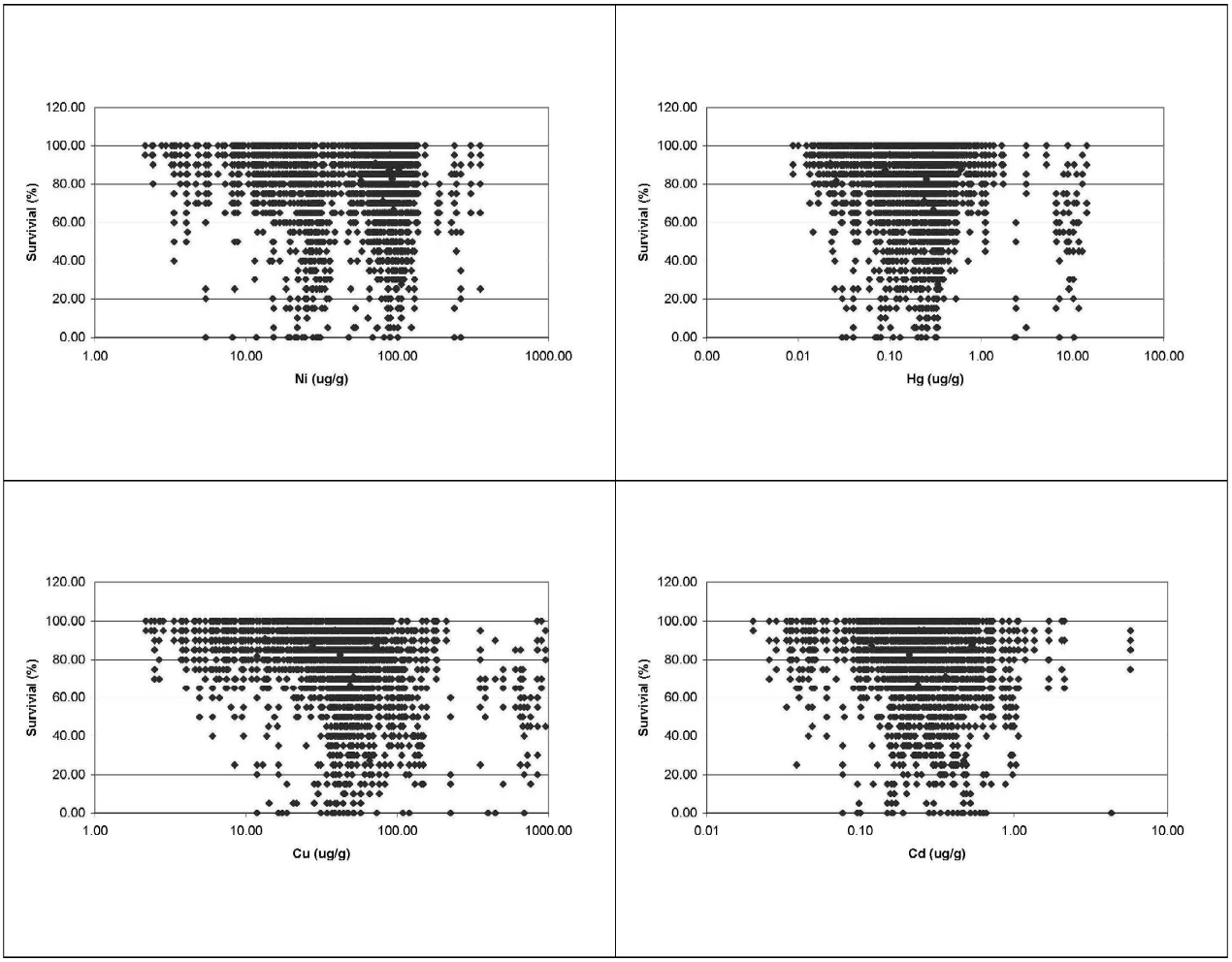


Figure 11


Survivial (%)
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100.00
80.00
60.00
40.00
20.00
0.00
1.00
10.00
100.00
1000.00
Ni (ug/g)
Survivial (%)
0.00
0.01
0.10
1.00
10.00
100.00
Hg (ug/g)
Survivial (%)
1.00
10.00
100.00
1000.00
Cu (ug/g)
Survivial (%)
0.01
0.10
1.00
10.00
Cd (ug/g)

Figure 12

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E N T R I X

EVALUATION OF APPARENT EFFECTS THRESHOLD AND EFFECTS RANGE-MEDIAN APPROACHES FOR DETERMINING SEDIMENT QUALITY GUIDELINES

Prepared For:

Montrose Chemical Corporation of California
Zeneca Holdings, Inc.
Stauffer Management Company
Atkemix Thirty-Seven, Inc.
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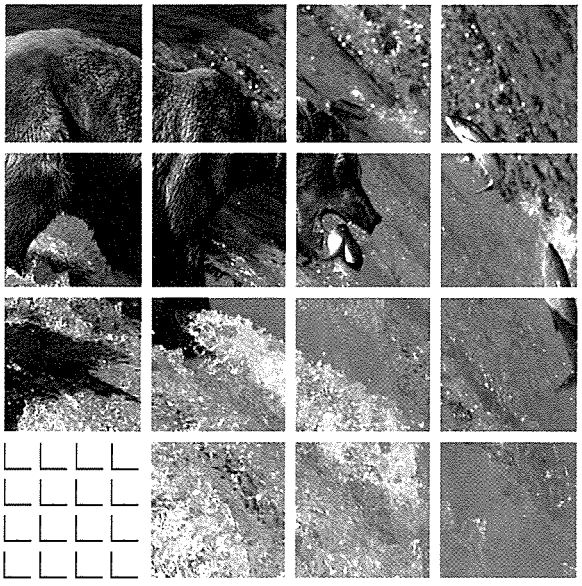

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Project No. 173201 (Task 0008)

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EXECUTIVE SUMMARY

This report summarizes an evaluation of two methods that have been used to establish biological effects thresholds of chemical contamination in sediments; namely, the Apparent Effects Threshold (AET) and the Effects Range-Median (ERM) methods. The work summarized here was initiated by conclusions made in the report entitled *Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments* (MacDonald, 1997). In that report, threshold concentrations of DDTs and PCBs were derived using the ERM method and then used for predicting toxic and non-toxic responses in Southern California Bight sediments. The implication of findings in the MacDonald report was that derived threshold concentrations could be used to establish sediment quality guidelines that could, in turn, be used to set target remediation levels for selected contaminants, or to establish the basis for injury.

In light of these findings, ENTRIX undertook a study to evaluate the two methods used to establish threshold concentrations for sediment contaminants. This study focused on evaluating the strength of relationships between concentrations of various contaminants in sediments and common measures of toxicity, and evaluating the statistical basis of the AET and ERM methods for establishing threshold concentrations in selected marine sediment data.

There were three overall objectives of this study: 1) to determine the general applicability of AET and ERM methods for evaluating sediment quality: 2) to document limitations in applying AET and ERM methods for evaluating specific contaminants of interest (DDT and PCBs) in the Southern California Bight sediments; and 3) to critique the two methods for their ability to provide ecologically and statistically defensible sediment toxicity guidelines. A summary of major activities and findings of the present study are given below.

- ENTRIX conducted an extensive search for relevant data for use in meeting the project objectives. The search included contacts in Canada, Florida, and State of California agencies,international scientific journals, and professionals in the field of sediment toxicology. A total of 40 data files containing matching (i.e., co-occurring or synoptic) sediment chemistry and toxicity data from marine and freshwater areas were retrieved and electronically stored. We found only ten (10) of these files contained outcomes of statistical comparisons between biological responses in test sediments and controls and, hence, were suitable for evaluation of the AET and ERM methods.

  ENTRIX examined these data sets and associated report documents to ensure that the information was technically defensible and accurate using quality Assurance/ quality control procedures. Approximately 15,000 records of co-occurring measurements were examined and included in the database for analysis.

- We found serious discrepancies in data used in the MacDonald (1997) Southern California Bight report , calling into question the validity of findings and conclusions

related to Los Angeles Harbor sediments as presented in the MacDonald (1997) report. Detailed evaluation of the data used in the MacDonald (1997) report in developing DDT and PCB thresholds is warranted.

- Based on our examination and analysis of the literature , we found that the AET and ERM methods have inherent limitations. We confirmed conclusions previously reached by other investigators which are that neither method can 1) establish cause-and-effect relationships between chemical concentration and biological effects; 2) account for factors important in determining bioavailability of chemicals, and; 3) account for biological effects due to unmeasured concentrations of other chemicals or chemical mixtures.
- An underlying assumption in successfully applying either the ERM or AET method for setting injury levels or sediment clean-up goals is being able to find ecologically and statistically significant relationships between individual biological effects and contaminant concentrations. We could not confirm that any such relationships existed in the MacDonald 1997 report after extensive graphical and statistical analyses.
- For San Diego Harbor sediments (Fairey *et al., 1996)*, we determined that use of either AET- or ERM-based toxicity threshold concentrations of total DDT or total PCB for setting injury or sediment clean-up levels would not be justified since these thresholds failed to distinguish between toxic and non-toxic effects for three toxicological endpoints. Hence, implementing sediment quality guidelines based on these thresholds would not guarantee achieving the intended ecological benefits to natural resources

TABLE OF CONTENTS

List of Tables

List of Figures

List of Appendices

1.0 INTRODUCTION

Over the past 10-15 years, concerns have arisen over the biological hazards associated with chemical contaminants that have accumulated in both marine and freshwater sediments. These concerns have motivated development of several methods aimed at deriving sediment quality criteria for providing long-term management of contaminated sediments. Two such methods are the Apparent Effects Threshold (AET) and the Effects Range-Median (ERM) methods. The ERM method is based on a modified version of the National Status and Trends Program (NSTP) method (Long and Morgan 1990; Long et al. 1995). ERM values were also used to determine sediment threshold concentrations of DDTs and PCBs in Southern California Bight sediments and to determine if DDT (and various DDT metabolites) and PCBs occur in Southern California Bight sediments at concentrations sufficient to adversely affect sediment-dwelling (benthic) species occurring in the area (MacDonald 1997). The AET method was first developed by Tetra Tech (1986) for assessing adverse biological effects associated with 64 organic and inorganic substances in sediments of Puget Sound WA.

The methods are statistically based, in that they rely on comparing measured biological effect responses in test sediments (either field-collected or chemically spiked) with the same responses in control sediments. A statistical test is then used to determine whether or not these responses are significantly different from one another. The outcomes of these tests are used as partial decision criteria to classify each sediment sample as being either an *effect* (i.e., response is significantly different from the control) or *no-effect* (i.e., response is not significantly different from the control). Once each sample is properly classified, the methods employ quite different techniques in assessing the relationship between biological effect classification and concentration of a contaminant. For the AET method, *effect* and *no-effect* samples are arrayed against the concentration of each chemical of interest and the threshold is chosen as that concentration above which an *effect* is always observed. For the ERM method, a quantile plot of chemical concentration for the *effect* samples is constructed and the threshold (i.e., the effects range-median or ERM) is designated to be the 50^{th} percentile or median of the distribution. A quantile plot is similar in appearance to a cumulative distribution plot; for example, if chemical concentration for the *effect* samples is normally distributed, then the quantile plot should appear S-shaped. Both methods require matching or co-occurrence data (sediment chemistry and toxicity or effects data that have been measured at the same location at the same time) from field and laboratory studies. Biological responses may include mortality in various sediment bioassays and benthic infaunal community structure measurements. These responses are matched with concentration values on a dry-weight basis (Calabrese and Baldwin 1993).

Both methods share an assumption that there is an inherent association between the concentration of a contaminant and a synoptically measured biological effect, implying a cause-and-effect relationship. Furthermore, the methods imply that the magnitude of an

adverse effect would decrease if contaminant levels were to be reduced to less than a threshold concentration. The AET method is generally considered to be site-specific, meaning that derived threshold values for contaminants are not strictly applicable to areas outside of where they were developed. On the other hand, the ERM method employs geographically broad-based co-occurrence data for deriving contaminant thresholds and, therefore, values have typically been applied to sites that are far-removed from one another. Specific assumptions inherent to the ERM method that were originally made by its developers are listed below.

- *"An underlying assumption of the approach is that, if enough data are accumulated, a pattern of increasing incidence of biological effects should emerge with increasing contaminant concentrations"* (Long et al. 1995).
- *The approach assumes that "associations between effects and chemical concentrations would be more credible if based upon data from many different studies than if based upon data from only one approach or experiment"* (Long 1992).
- *"The approach assumes that the use of a large database accounts for interactions between chemicals in complex mixtures, for effects of unknown or unmeasured chemicals or materials, for factors other than measured chemical concentrations that may affect species responses, and that indicators of bioavailability and effects can be determined based on field and/or laboratory data"* (Chapman 1989).

2.0 OBJECTIVES AND DESCRIPTION OF ACTIVITIES

The objectives of this investigation were 1) to determine the general applicability of AET and ERM methods for evaluating sediment quality, 2) to document limitations in applying AET and ERM methods for evaluating specific chemicals in sediments, and 3) to critique the two methods for their ability to provide ecologically and statistically defensible sediment toxicity thresholds. In order to address these objectives, this study consisted of five (5) tasks which provided a means of describing specific advantages and disadvantages of using the methods, the limitations in applying the methods to specific chemicals associated with sediments, and a critical examination of sediment quality predictions. The five tasks are: 1) literature review and database searches; 2) information analysis and database management; 3) statistical analyses; 4) interpretation of literature findings and statistical analyses; and 5) report preparation.

2.1 Literature Review and Database Searches

A literature review was performed and database searches conducted by contacting agencies in Canada, Florida, and California as well as professionals in the field of sediment toxicology. The database searches focused on obtaining relevant toxicological and chemical concentration information for marine sediments both within and outside of Southern California and for freshwater areas. One of the primary sources for information obtained for Southern California was found in the report entitled *Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments* (MacDonald 1997). These data included co-occurrence measurements for Sum DDT, DDE, and DDD, total DDT, Aroclor 1254, and total PCBs.

Other data for Southern California were obtained by performing a chain-of-citation search of MacDonald's (1997) report. The two most frequently cited documents in this report were (Fairey et al. 1996; Sapudar et al. 1994) and these authors were contacted directly. This resulted in the retrieval of two documents that contained co-occurrence data on sediments collected in Southern California. The study by Fairey et al (1996) was found to be suitable for evaluations intended for the present investigation and was also available electronically. The report by Sapudar et al (1994) contained relatively few co-occurrence data and was not available electronically and therefore not used in the current study. However, both studies were used to perform QA/QC audits on the MacDonald (1997) report.

The report by Fairey et al. (1996) was the only document found other than that by MacDonald (1997) that could be immediately used as a source of co-occurrence data for Southern California. A total of 350 stations were sampled from San Diego Harbor as part of an ongoing Bay Protection and Toxic Cleanup Program designed to assess the relative degree of chemical contamination and associated biological effects in California's bays and harbors. The study involved chemical analysis of sediments, benthic community analysis, and toxicity

testing of sediments and sediment pore water. Co-occurrence data were available and used for Sum DDT, DDE, and DDD, and total DDT, total PCBs, lead, mercury, and zinc.

Co-occurrence data from outside of the Southern California region were obtained from MacDonald (1994). This report contained extensive co-occurrence data that did not duplicate data found in MacDonald (1997). The 1994 report contained co-occurrence data for total DDT, total PCBs, lead, mercury, and zinc for the southeastern US as well as data for other marine and estuarine areas from throughout the US and Canada. These data were not available electronically and were, therefore, manually entered into the project database.

Freshwater sediment data were also obtained from Environment Canada which has developed an extensive co-occurrence database. Data were obtained for Sum DDT, DDE, and DDD (Environment Canada 1998), total PCB (Environment Canada 1997a), lead (Environment Canada 1997b), mercury (Environment Canada 1997c), and zinc (Environment Canada 1997d) for areas throughout the US and Canada. These data were not available electronically and were manually entered into the project database.

2.2 Information Analysis and Database Management

After receiving each document, it was necessary to review the report, extract those data relevant to this project, and manually transfer these data into a useable electronic format suitable for database management and statistical analysis. Data already available in electronic format (i.e., Fairey et al. 1996) were incorporated directly into the project database. A total of 40 data files containing matching sediment chemistry and toxicity measurements from marine and freshwater areas were retrieved and electronically stored for this work. We found only 10 of these files contained outcomes of statistical comparisons between biological responses in test sediments and controls and, hence, were suitable for evaluation of the AET and ERM methods.

A major component of this task was to ensure that data collected and used in our study were both technically defensible and accurate. The following activities were implemented as part of this task: 1) development of a centralized data management system to facilitate the transfer, update, and maintenance of data collected during the project; 2) standardization of scales of measurement of collected data; 3) implementation of a data tracking system; and 4) implementation of database QA/QC audits.

The project database consists of information obtained from four sources: MacDonald (1997), MacDonald (1994), Fairey et al. (1996), and Environment Canada (1997a-d; 1998). Only data from these sources were used to evaluate the AET and ERM methods in our study evaluation. At this time, the database consists of approximately 15,000 records of co-occurring measurements. Definitions of the nine (9) variables in the database are given below.

1. Chemical concentrations were reported in units of mg/kg dry-weight sediment for metals and µg/kg dry-weight sediment for organic constituents;
2. Code indicating whether or not the data were used to calculate a threshold value;
3. Description of biological endpoint;
4. Value of endpoint measurement;
5. Taxon associated with the endpoint;
6. Taxonomic Code;
7. Life stage of taxon (i.e., adult, subadult, juvenile, larvae, embryo, gamete, variable);
8. Total organic carbon (TOC, %);
9. Code for source of sediment (marine or freshwater).

Endpoint and taxonomic codes are defined in Tables 1 and 2. Data were placed in a Microsoft Excel (Version 6.0) spreadsheet and were updated as new information was received or when revisions were required. Individual data files comprising the database were given descriptive names.

Quality Assurance/Quality Control (QA/QC) audits were performed on data used in this study. In particular, reports by MacDonald (1994, 1997) were audited to ensure that the data matched raw data from cited documents. To accomplish this, the most frequently cited documents in the MacDonald (1997) report were identified and obtained (Fairey et al. 1996; Sapudar et al. 1994; Bay et al. 1994). Several reports cited by MacDonald (1994) were also obtained (Espy, Huston & Associates, 1983a,b; 1985a,b). Data that were manually entered into the project database were also audited in their entirety to ensure accuracy.

2.3 Statistical Analyses

The two objectives of these analyses were to determine the strength of association of different toxicity and biological community endpoint measurements with concentrations of different contaminants and to evaluate statistical limitations in developing AET- and ERM-based marine sediment quality thresholds. For the first objective, linear (Pearson) correlations were determined for endpoint responses and co-occurring measurements of the concentration of lead, mercury, zinc, total PCB, total DDT, Sum DDT, Sum DDD, and Sum DDE.

Table 1. Biological endpoints and associated codes used in the project database.

Endpoint	Code	Endpoint	Code
Mortality, %	M	Infaunal Index	I
Fertilization, %	F	% Emergence, %	E
Avoidance, %	A	Microtox, EC_{50}	P
Normal Development, %	N	Reproduction	RP
Biomass, g	B	Fish Lesions	Y
Growth, mg/day	G	Abnormal Development, %	C
Gonad growth, g/day	O	Length Increase, mm	H
Reburial, %	R	Diversity	SD
Density, No./0.1 m^2	D	Population Growth	PG
Species Richness	S	Burrowing Time, ET_{50}	T
Growth, mm/d	L	Sexual Maturity, %	Q
Biotic Integrity (AIBI)	J	Species, No./0.1 m^2	DS
Deformities, %	K	No. of Taxa	V

Table 2. Taxon and associated taxonomic codes used in the project database.

Taxon	Code	Taxon	Code
Amphipod	A	Arthropod	R
Benthic Invertebrate	B	Sea Urchin	U
Crustacean	C	Fish	F
Echinoderm	E	Microtox	T
Mollusca	M	Sponges	SP
Sand Worm	N	Nemerteans	W
Shrimp	S	Protista	G
Polychaete	P		

In addition, for marine sediment measurements encompassing areas both within and outside of Southern California, separate t-tests were conducted to determine whether or not areas were significantly different from one another on the basis of both contaminant concentration and toxicity response. Similarly, if more than one data source was available for an area, tests were also conducted to determine whether or not data sources were significantly different with respect to the measured variables. For all statistical tests, the nominal significance level was $\alpha = 0.05$. In order to reasonably limit the number of analyses made for marine sediment data, evaluations were restricted to data subsets containing ≥30 total observations in any (area × endpoint × taxonomic code) combination for each chemical. A similar rule was also applied to the freshwater sediment data, whereby the number of analyses were restricted to data subsets containing ≥30 total observations in any (endpoint × taxonomic code) combination.

For the second objective, AET and ERM determinations and related analyses were restricted to data from San Diego Harbor (Fairey et al 1996) that had co-occurring measurements of sediment toxicity (amphipod mortality, sea urchin development, and sea urchin fertilization laboratory bioassays) and concentrations of total DDT and total PCB. Endpoint data for urchin development and fertilization were further restricted to bioassay results using 100% pore water from sediment samples. No attempt was made here to derive AET or ERM sediment criteria for the remaining contaminants. For these data, toxicity responses were determined using sediment collected from a number of stations within the harbor. Laboratory control toxicity was also determined and the test and control responses were statistically compared. A sample sediment was first tentatively considered to be toxic if the response was significantly different from the control. For a sample found to be significantly different, a final determination of toxicity was made on the basis of whether or not toxicity was less than 80% of the control. Using these approaches, the authors stated that "there was no absolute value below which all samples could be considered toxic, although survival below a range of 72-80% was generally considered toxic". For each endpoint, a sample was designated as either an *effect*, meaning that the endpoint response indicated toxicity, or *no-effect*, meaning that the endpoint response did not indicate toxicity. Associated with these endpoint responses and effects designations were synoptic measurements of concentrations of the contaminants.

The first analyses conducted using these data were to determine if statistically significant differences existed between endpoint-specific *effect* and *no-effect* stations with respect to each of the two contaminants. The comparisons between stations grouped in this way were made using separate t-tests and a nominal significance level of $\alpha=0.05$.

Determinations of AET values were then made for total DDT and total PCB using each of the three endpoint responses by arraying the *effect* and *no-effect* stations against the concentration of a contaminant. The concentration above which only *effect* stations were located in the array was then determined and this value was designated as the AET for that endpoint and contaminant. ERM values were also found by determining the concentration of

a contaminant corresponding to the median (i.e., 50th percentile) of the *effect* stations (Long et al 1995). Once this value was determined, it was used to determine the effects incidence which is defined as the ratio of [*effect* stations > ERM] to the combined total of [*effect* + *no-effect* stations > ERM].

Prior to any analyses, data were appropriately transformed to achieve normally or near-normally distributed values in the variables being evaluated. Original bulk sediment concentrations of contaminants, C, were transformed to ln(C, weight/g total Organic Carbon) and endpoint measurements consisting of proportionate counts (e.g., mortality, development, fertilization) were arcsin-square root transformed. For some endpoint measurements, no transformation was required; for example, benthic invertebrate species diversity for freshwater sediments. Checks on the success of data transformations were made by examining distribution and probability plots of the transformed variables.

3.0 RESULTS AND DISCUSSION

3.1 QA/QC DATA AUDITS

The QA/QC audit indicated disparities existed between original data reported by Fairey et al. (1996) and the same data reported by MacDonald (1997). All values of total PCB and total DDT used by MacDonald failed to correspond (as they should have) to the original values of Fairey. The QA/QC audit also indicated that some of the original Sapudar et al. (1994) data cited in MacDonald (1997) did not match. Roughly 50% of all data reported in MacDonald (1997) were inconsistent with the original Sapudar data. Thus, the results presented in the MacDonald (1997) report were in error and the conclusions incorrect. We analyzed the corrected data sets and used these data in our study.

3.2 ADVANTAGES OF THE METHODS

There are a number of strengths or advantages of using an AET or ERM approach to evaluate sediment quality which are inherent to most approaches utilizing co-occurrence data.

1. *The ERM approach is supported by a comprehensive co-occurrence database.* Co-occurrence databases have been recently used in a number of investigations to assess sediment quality. An extensive co-occurrence database on the biological effects of sediment associated chemicals called biological effects database for sediments (BEDS) and similar databases have been used, based on existing data (MacDonald 1994; 1997; Long et al. 1995; Smith et al. 1996a). This large database can be interpreted and used as a tool for evaluating the potential for adverse biological effects at various chemical concentrations and can provide a degree of confidence on the resultant guidelines (MacDonald 1994).
2. *In general, both approaches are practical to develop.* The approaches are considered to be practical because data can be used to generate numerical thresholds for a variety of chemicals, many of which typically accumulate in sediments. Generation of thresholds can be accomplished relatively quickly. The influence of chemical mixtures is incorporated into the data when field data are used. They apply to a wide range of benthic species and endpoints, and incorporate direct measurements taken *in situ*. As such, these attributes are considered to give the approaches broad applicability.
3. *The approaches consider both effects and no-effects data to generate thresholds.* In contrast to the AET method, the ERM approach does not rely heavily on single data points, hence outliers carry less weight in calculating guidelines. By including both effects and no-effects data, detailed tables can be constructed to evaluate the potential biological significance of co-occurrence data (MacDonald 1994).
4. *The database used for deriving ERM values provides a basis for evaluating thresholds.* The co-occurrence database is arranged in ascending order of chemical concentration in a tabular format. This format permits expansion of the database, calculation of the

distribution of *effects* and *no-effects* entries surrounding the guideline, and determination of the reliability (or degree of confidence) of each threshold (MacDonald 1994). These thresholds could then be used within a risk assessment framework for assessing contaminated sediments.

5. *The ERM method has undergone extensive peer review.* The method has been published in the peer reviewed literature (e.g., Long 1992; Long et al. 1995; Smith et al. 1996a). It has received support from a wide variety of user groups and has been adopted by Canada (Smith et al. 1996a) as part of sediment guideline derivation processes. According to MacDonald (1994), the favorable reviews that have been received emphasize the importance and utility of the method in deriving numerical guidelines.
6. *The methods implicitly consider bioavailability and chemical mixture effects.* Since co-occurrence field data are used to generate guidelines, various factors such as organic carbon that may influence bioavailability as well as the effects of mixtures of chemicals are implicitly considered.

3.3 DISADVANTAGES OF THE METHODS

There are a number of inherent limitations or disadvantages in using the AET or ERM methods for evaluating sediment quality. Those listed below apply specifically to the ERM method and, more generally, to the AET method.

1. *The ERM method does not establish cause-and-effect relationships.* The method assumes associations between chemical concentrations and biological effects rather than supporting development of cause-and-effect relationships (MacDonald 1994; Calabrese and Baldwin 1993). Any number of variables such as unmeasured chemicals, ammonia, hydrogen sulfide, and dissolved oxygen content in sediments could affect sediment toxicity and species responses (Chapman 1989; MacDonald 1994).
2. *The ERM method does not account for factors affecting chemical bioavailability.* The method may not account for other factors affecting chemical bioavailability such as grain size and acid volatile sulfide (AVS) content of sediment-sorbed materials (e.g., Nebeker et al. 1989; DiToro et al. 1990). These data are infrequently reported in the open literature and often precludes their use in deriving threshold values. This results in the development of thresholds that may be both under- and over-protective of benthic species (MacDonald 1994).
3. *The method attempts to establish absolute sediment guidelines.* The ERM method typically delineates ranges of chemical concentrations that are probably, possibly, and not likely to be associated with adverse biological effects (MacDonald 1994; Long et al. 1995; Smith et al. 1996a). The method typically recognizes the uncertainty associated with the prediction of adverse effects from sediment-associated chemical measurements and such recognition is believed to enhance defensibility of the guidelines (CCME 1995). However, delineation of ranges was all but ignored by MacDonald (1997) and hence, the

inherent uncertainty surrounding thresholds for the Southern California Bight was ignored, substantially reducing what limited utility they may possess.

4. *Values developed using the method may not reflect chronic responses.* Chronic data are often sparse or absent for a given chemical and hence, the guidelines generated will not reflect chronic responses of benthic species (Chapman 1989; MacDonald 1994).

5. *Guidelines developed by the method may not be applicable for sediments with atypical levels of TOC or other factors.* Care should be exercised when applying the guidelines at a given site because they do not account for factors affecting bioavailability. For example, if sediments in an area contain relatively high or low TOC content, then the thresholds generated may not be applicable (MacDonald 1994). This is because the ameliorating effects of TOC may reduce the bioavailability of organic chemicals thus rendering them over-protective.

6. *The method does not explicitly consider effects as a result of bioaccumulation* The method does not account for potential bioaccumulation of chemicals in benthic organism tissues nor does it account for potential adverse effects on species that may ingest these benthic species (MacDonald 1994; Smith et al. 1996a). However, it has been stated by proponents of the method that because the expression of adverse effects includes effects due to bioaccumulation, then the method indirectly considers bioaccumulative effects (Smith et al. 1996a).

7. *The method requires relatively large co-occurrence databases.* In particular, the ERM method requires at least 20 co-occurring observations associated with an *effect* response (e.g., toxicity significantly different than a control) and an additional 20 co-occurrences of a *no effect* response (CCME 1995). In addition, the concentration of each chemical of interest should vary by at least 10-fold among sampling sites. Finally, statistical procedures used should be presented in detail and test methodology must follow standard protocols (CCME 1995).

8. *The method does not account for effects due to mixtures of chemicals, only a single chemical.* The reliance of the method on co-occurrence data precludes the possibility of separating individual chemical effects from samples where multiple chemicals exist (Adams et al. 1992; Chapman 1989). This limitation is highly relevant to the Southern California Bight, as several chemicals including DDTs and PCBs are present in sediments.

3.4 CORRELATION AND ANALYSES OF VARIANCE

The AET and ERM methods assume that there is a strong association between endpoint response and contaminant concentration. In particular, the ERM method claims that if enough data are examined, a direct relationship should be evident between adverse effect and increasing concentration. In order to examine this assumption, various endpoints and contaminants were selected (refer to selection rule above) for correlation analysis. In those instances where data were from different sources or encompassed areas within and outside of

Southern California, analyses of variance were also conducted to determine whether sources or areas were significantly different from one another. Where significant differences were found, an examination of the overall pattern of relationship between effect and concentration was conducted. No models were derived and tested (via regression analysis) for their adequacy in predicting biological response from co-occurring chemical concentration data. Hence, conventional R^2 values (i.e., the coefficient of determination reflecting the amount of total variation in the response explained by a model) are not reported. The reason that no modeling analyses were conducted is that a particular biological response in natural sediments is a function of many biotic and abiotic factors including concentrations of chemical contaminants. Therefore, it would be inappropriate and misleading to model (i.e., attempt to predict) the magnitude of a biological response using only the corresponding magnitude in concentration of a single chemical.

Linear (Pearson) correlation coefficients between transformed endpoint measurements and chemical concentrations that were obtained for marine and freshwater sediment data are presented in Table 3. Cells in the table having entries $n<30$ indicate that there were insufficient data for analysis according to the criteria defined above. Notice in the table that because of insufficient data, some contaminants of interest were not analyzed; namely, zinc, Sum DDT, and total DDT in freshwater sediments and Sum DDD and Sum DDE for marine sediments. In the following sections, results of analyses that were conducted are discussed in greater detail. All figures associated with these analyses are given in Appendix B.

Lead in Marine Sediments. Comparisons between reference data for lead content in Southern California area sediments having co-occurring amphipod mortality measurements are presented in Figures 1 and 2. It was found that the data reported by MacDonald (1994) had significantly greater concentrations of lead ($P=0.00382$) than did data reported by Fairey et al (1996). However, it was also found that amphipod mortality was significantly greater for the Fairey et al (1996) data ($P=0.00022$). Scatter plots of the Fairey et al (1996) and MacDonald (1994) data are given in Figures 3 and 4 which show the associations between mortality and sedimentary lead content. For the Fairey et al (1996) data in Figure 3, the linear correlation between variables is $r=-0.154$ and for the MacDonald (1994) data in Figure 4, $r=0.278$. In neither case, is there a strong association between mortality and lead concentration.

Comparisons were also made using MacDonald (1994) data for sedimentary lead content in areas within and outside of Southern California having co-occurring measurements of amphipod mortality (Figures 5 and 6). No statistically significant differences were found among the areas for lead content or mortality ($P=0.07646$ and $P=0.15921$, respectively). A scatter plot of mortality against lead content for the combined data is shown in Figure 7 which yielded a linear correlation coefficient of $r=0.226$.

Table 3. Linear (Pearson) correlations between different endpoint measurements and concentrations of chemicals for marine and freshwater sediments. All coefficients were determined using transformed variable values (see text).

Sediment Type	Sediment Location	Endpoint	Chemical Constituent							
			Lead	Mercury	Zinc	Total PCB	Total DDT	Sum DDT	Sum DDD	Sum DDE
Marine	Southern CA	Amphipod Mortality	-0.154 (a) 0.278 (b)	-0.061 (b)	-0.153 (b)	0.574 (a) -0.060 (b)	0.090 (a) -0.084 (b)	-0.196 (b)	n<30	n<30
Marine	Southern CA	Sea Urchin Development	0.226 (b)	0.123 (b)	0.171 (b)	0.052 (b)	n<30	-0.061 (b)	n<30	n<30
Marine	Southern CA	Sea Urchin Fertilization	-0.147 (b)	-0.064	-0.143 (b)	-0.043 (b)	-0.328 (a, b)	-0.288 (b)	n<30	n<30
Marine	Outside Southern CA	Amphipod Mortality	0.226 (a)	0.263 (a)	0.266 (a)	n<30	n<30	n<30	n<30	n<30
	Outside Southern CA	Sea Urchin Development	n<30	0.254 (a)	0.189 (a)	n<30	n<30	n<30	n<30	n<30
Freshwater	Unknown	Benthic Invertebrate Species Diversity	0.126	-0.263	ND	n<30	n<30	n<30	-0.052	0.032
Freshwater	Unknown	Benthic Invertebrate Density	0.041	0.052	n<30	0.042	n<30	n<30	-0.183	-0.187
Freshwater	Unknown	Mollusca Density	-0.348	0.011	n<30	n<30	n<30	n<30	n<30	n<30
Freshwater	Unknown	Amphipod Density	n<30	-0.045	n<30	n<30	n<30	n<30	n<30	n<30

(a) MacDonald 1994
(b) Fairey et al 1996
ND Not Determined

Two additional scatter plots are presented in Figures 8 and 9 showing the association between lead content in Southern California area sediments and sea urchin development and fertilization toxicity, respectively. For the development endpoint, the correlation with lead content is r=0.226 and for fertilization ρ=-0.147. Neither of these coefficients indicate a strong association between toxicity endpoints and sedimentary lead content.

Lead in Freshwater Sediments. Scatter plots of benthic invertebrate (infaunal) density, species diversity, and mollusca density against the concentration of lead are presented in Figures 10-12. Provided that sedimentary lead content exerts a singular influence on the values of these variables, then we would expect to observe a monotonically decreasing association in each of these cases. No such trends are evident in Figures 10 and 11 for density or diversity (ρ=0.041 and ρ=0.126, respectively). For mollusca density, the trend is correct (ρ=-0.348), but it is clearly not monotonic.

Mercury in Marine Sediments. Comparisons between areas within (Fairey et al 1996) and outside (MacDonald 1994) of Southern California for sedimentary mercury having co-occurring amphipod mortality measurements are presented in Figures 13 and 14. These comparisons revealed that both the concentration of mercury and mortality are significantly lower for sediments outside of the Southern California area (P=0.00331 and P=<0.00001, respectively). Scatter plots of mortality against mercury content are presented in Figures 15 and 16. No meaningful linear association is apparent between the variables (r=-0.061 and r=0.263, respectively).

Comparisons of areas were also conducted for sedimentary mercury having co-occurring measurements of sea urchin development. These comparisons are presented in Figures 17 and 18. Mercury content was found to be significantly lower in sediments outside of Southern California (P=0.01176). In contrast, areas did not differ significantly in development toxicity (P=0.07656), although this conclusion must be viewed as tentative given the level of the P-value. Scatter plots of mercury content against development toxicity are presented in Figures 19 and 20 which show no meaningful evidence of a linear association between the variables (r=0.254 and r=0.123, respectively).

One additional scatter plot of sea urchin fertilization against mercury content for Southern California area sediments is presented in Figure 21. There is no apparent linear association between the variables (r=-0.064).

Mercury in Freshwater Sediments. Associations between sedimentary mercury and benthic invertebrate (infaunal) density (r=0.052), species diversity (r=-0.263), amphipod density (r=-0.045), and mollusca density (r=0.011) are shown in Figures 22-25, respectively. It is clear from these figures that no linear association can be claimed between the different endpoint measurements and concentration of mercury in sediments.

Zinc in Marine Sediments. Comparisons between areas within (Fairey et al 1996) and outside (MacDonald 1994) of Southern California for sedimentary zinc having co-occurring amphipod mortality measurements are presented in Figures 26 and 27. These comparisons revealed that the concentration of zinc was not significantly different for these sediments (P=0.05582) but that mortality was very significantly lower in sediments outside of the southern California area (P=<0.00001). Scatter plots of mortality against zinc content are presented in Figures 28 and 29. There is no linear association apparent between the variables, evidenced by the correlation for areas within (r=-0.153) and outside (r=0.266) of Southern California.

Comparisons of areas within (Fairey et al 1996) and outside (MacDonald 1994) of Southern California for sedimentary zinc having co-occurring sea urchin development measurements are presented in Figures 30 and 31. It was found that areas differed significantly in zinc content (P=0.00504) and that sediments outside of Southern California contained lesser concentrations of zinc. However, it was also found that areas probably did not differ significantly in development (P=0.07492). Scatter plots of zinc content against development are presented in Figures 32 and 33. In neither case, is there a meaningful linear association between the variables; the correlation between variables for sediments within the Southern California area is r=0.171 and r=0.189 for sediments outside of Southern California.

One additional scatter plot is presented in Figure 34 showing the association between zinc content and sea urchin fertilization toxicity in Southern California area sediments. Again, no apparent linear relationship between the variables is evident (r=-0.143).

Total PCB in Marine Sediments. Comparisons between the Fairey et al (1996) and MacDonald (1994) data for total PCB content having co-occurring amphipod mortality measurements in Southern California area sediments are presented in Figures 35 and 36. It was found that concentrations of total PCB were not significantly different in the two data sets (P=0.42892) but that the Fairey et al (1996) data exhibited significantly greater mortality (P=<0.00001). A scatter plot of total PCB against mortality for the MacDonald (1994) data is presented in Figure 37 which yielded a linear correlation coefficient of r=0.574. A similar scatter plot of these data in the original (non-transformed) space is shown in Figure 38 which suggests that mortality is an increasing exponential function of the concentration of sedimentary total PCB concentration. There is an outlying observation in these data occurring at a concentration of approximately 5 μg total PCB/g TOC and associated mortality of 95%. If that one observation were to be neglected, for example, on the basis of poor laboratory testing procedures for either toxicity testing or chemical concentration determination, then the linear correlation between the variables would be improved considerably. It would, however, not be ecologically reasonable to attempt to model mortality solely as a (exponential) function of concentration. There were undoubtedly other relevant environmental variables that also affected mortality which were not measured and, therefore, could not be included in the model. Neglecting these other unknown variables would lead to a misleading model that only total PCB content in these sediments determines

amphipod mortality. A scatter plot of the Fairey et al (1996) data is presented in Figure 39 which shows that, in contrast to the MacDonald data, there is little linear association between amphipod mortality and total PCB concentration (r=-0.060).

Comparisons between the Fairey et al (1996) and MacDonald (1994) data for total PCB content having co-occurring sea urchin fertilization measurements in Southern California area sediments are presented in Figures 40 and 41. It was found that these data sources did not differ significantly for either total PCB content or urchin fertilization (P=0.18614 and P=0.80104, respectively). A scatter plot for the combined data (Figure 42) shows that there is no apparent linear association between the variables as evidenced by a correlation coefficient of r=-0.043.

One additional scatter plot of sea urchin development toxicity against total PCB content in Southern California area sediments is presented in Figure 43. The linear correlation between these variables is r=0.052, indicating very little association among values.

Total PCB in Freshwater Sediments. A scatter plot of benthic invertebrate density against sedimentary PCB content is presented in Figure 44. The linear correlation between these variables is r=0.042, indicating very little association among values.

Sum DDT in Marine Sediments. Scatter plots of the toxicity endpoints amphipod mortality, sea urchin development, and sea urchin fertilization against Sum DDT content in Southern California area sediments (Fairey et al 1996) are given in Figures 45-47. As shown in Figures 45 and 46, there is no apparent linear association between endpoints and Sum DDT content (r=-0.196 and r=-0.061, respectively). Fertilization toxicity (Figure 47) does show a trend in accordance with expectation (that is, lower fertilization with higher concentration), having a linear correlation coefficient of r=-0.288. However, this apparent trend is largely influenced by several toxicity measurements at concentration values above approximately 2.7 μg Sum DDT/g TOC.

Sum DDD in Freshwater Sediments. Scatter plots of benthic invertebrate density and species diversity against Sum DDD content are presented in Figures 48 and 49. For both endpoint measurements, there is little linear association with concentration (r=-0.183 and r=-0.052, respectively).

Sum DDE in Freshwater Sediments. Scatter plots of benthic invertebrate density and species diversity against Sum DDE content are presented in Figures 50 and 51. For both endpoint measurements, there is little linear association with concentration (r=-0.187 and r=0.032, respectively).

Total DDT in Marine Sediments. Comparisons between the Fairey et al (1996) and MacDonald (1994) data for total DDT content having co-occurring amphipod mortality measurements in Southern California area sediments are presented in Figures 52 and 53. It

was found that these data sources did not differ significantly for total DDT content (P=0.29759). However, data reported by Fairey et al (1996) data exhibited significantly greater mortality (P=0.02490). Scatter plots of these data are shown in Figures 54 and 55. The Fairey et al (1996) data (Figure 54) yielded a correlation coefficient of r=-0.084 while the data reported by MacDonald (1994) yielded r=0.090. In neither case, is there a strong association between mortality and total DDT concentration.

Comparisons between the Fairey et al (1996) and MacDonald (1994) data for total DDT content having co-occurring sea urchin fertilization measurements in Southern California area sediments are presented in Figures 56 and 57. It was found that these data sources did not differ significantly for total DDT content or development toxicity (P=0.80104 and P=0.63995, respectively). A scatter plot of the combined data is shown in Figure 58. While the overall trend in association appears to be in accordance with expectation (i.e., lower fertilization at higher concentration), the association is largely influenced by a single toxicity observation at approximately 67 µg total DDT/g TOC. In addition, no strong linear correlation between the variables is evident (r=-0.323).

In general, the analyses reported above fail to demonstrate the existence of any meaningful correlation between selected endpoint responses and contaminant concentrations in either marine or freshwater sediments. Furthermore, there were instances in which statistically significant differences were found in the magnitude of an endpoint response among areas or among data sources within an area, yet no significant differences were found with respect to the co-occurring concentration of a contaminant. Such inconsistencies immediately call into question the validity of assuming an inherent direct relationship between the magnitude of contamination and the magnitude adverse biological effects using field observations. While this assumption is typically valid for carefully controlled laboratory dose-response tests using a single contaminant, it is rarely satisfied from field observation where samples are often taken across multiple gradients in critical environmental variables other than contaminant levels. Furthermore, the magnitude of field-based responses for a single endpoint is often a function of a complex mixture of contaminants and perhaps other variables, and it is not ecologically or statistically reasonable to ignore such complexity. The combination of effects due to environmental gradients and complex contaminant mixtures precludes simply applying the assumption of cause-and-effect (dose-response) to field observations. Neglecting such effects in the establishment of sediment quality guidelines or clean-up levels for contaminants would, therefore, not be justifiable.

3.5 AET AND ERM DETERMINATIONS: Examples of Misleading Estimates

In our analyses of the data, it is demonstrated that AET- and ERM-based thresholds derived for establishing sediment quality criteria using selected field observations lead to values that have no toxicological or statistical meaning. We used PCB and DDT data reported by Fairey *et al.* (1996) to illustrate that the methods for determining AET and ERM values can lead to sediment quality thresholds which do not achieve their intended toxicological and

ecological relevance. We emphasize that the results presented below follow from the fact that no relationships between toxicity responses and contaminant levels exist in these data and therefore lead to inaccurate estimates of sediment quality criteria.

Comparisons between *effect* and *no-effect* station groups for total DDT and total PCB using the endpoints amphipod mortality, sea urchin development, and sea urchin fertilization are presented in Figures 59-64. Mean concentrations of both contaminants for each endpoint and also the results of t-tests that were conducted to assess whether or not these means were significantly different from one another are presented in Table 4. It can be seen that only the single test comparing total PCB sediment content for sea urchin development was statistically significant (P=0.0043). Results of all other tests were not statistically significant, leading to the conclusion that *effect* stations cannot be distinguished from *no-effect* stations on the basis of the concentration of total DDT or total PCB. In the single instance where *effect* and *no-effect* stations were significantly different, the mean concentration of total PCB in the *no-effect* group was actually greater than in the *effect* group. This result is, of course, contrary to what would otherwise be expected if DDT content in sediment were exerting a negative impact on sea urchin developmental toxicity. In general, these results do not support using the concentration of total DDT or total PCB taken alone, to explain the different toxicity designations in the endpoints considered. These results also demonstrate the inability of being able to predict sediment toxicity response with any acceptable degree of statistical confidence on the basis of arbitrarily chosen levels of contaminant concentration.

Despite these results, both AET and ERM values were determined for each contaminant using each of the biological endpoint measurements. These determinations were made in order to demonstrate differences in potential sediment quality criteria that could result using these two approaches. However, it should be clear from the results presented above that neither method is capable of yielding a criterion having any meaningful statistical or toxicological relevance for contaminated sediments.

AET for Total DDT. AET determinations for total DDT were made using amphipod mortality, sea urchin development, and sea urchin fertilization. These determinations are reflected in Figures 65-67. For amphipod mortality, there were eight stations exhibiting a consistent *effect* greater than the threshold concentration of approximately 2.2 µg/g TOC (Figure 65a). The relationship between mortality and concentration is shown in Figure 65b which yielded a linear correlation of r=0.160. Considering only the eight *effect* stations, the correlation is r=0.630 which is not statistically significant (P=0.0944). It was not possible to determine an AET using urchin development since there were no *effect* stations observed at concentrations greater than that observed for the *no-effect* stations (Figure 66a). The relationship between development and concentration is shown in Figure 66b (r=0.094). For urchin fertilization, there were only two stations exhibiting a consistent *effect* above a threshold concentration of approximately 4.3 µg/g TOC (Figure 67a). The overall linear relationship between fertilization and concentration is shown in Figure 67b which yielded a correlation of r=-0.322.

Table 4. Comparisons of mean concentrations of contaminants for *effect* and *no effect* station groups using different biological endpoints measurements. The P-values result from separate t-Tests conducted for each endpoint × contaminant combination where unequal variances were assumed. Data used in determining these values were from San Diego Harbor marine sediments (Fairey et al 1996).

Biological Endpoint	Mean Total DDT (µg/g TOC)		P-Value (df) Total DDT	Mean Total PCB (µg/g TOC)		P-Value (df) Total PCB
	Effect	No Effect		Effect	No Effect	
Amphipod Mortality	0.75	0.76	0.8922 (76.3)	4.24	5.70	0.0862 (87.0)
Sea Urchin Development	0.74	0.81	0.6706 (24.5)	4.32	7.44	0.0043 (39.1)
Sea Urchin Fertilization	0.82	0.64	0.0849 (84.4)	4.58	5.39	0.3346 (81.5)

AET for Total PCB. AET determinations for total PCB were made using the same three endpoint toxicity response measurements employed for total DDT and are reflected in Figures 68-70. For mortality, there were only two stations exhibiting a consistent *effect* above the threshold of approximately 36.3 µg/g TOC (Figure 68a). The linear relationship between mortality and concentration is presented in Figure 68b which has a corresponding correlation of r=0.029. For sea urchin development, there were four *effect* stations above the threshold concentration of approximately 24.0 µg/g TOC (Figure 69a). Overall, the relationship between this endpoint and concentration appears to be contrary to expectation, having a linear correlation of r=0.296 (Figure 69b). In contrast, the correlation between variables considering just the four *effect* stations above the threshold is r=-0.877. However, the magnitude of this correlation is heavily influenced by the fact that three out of these four *effect* stations had development values of zero. For the sea urchin fertilization endpoint, there were three stations exhibiting a consistent *effect* above the threshold of approximately 25.6 µg/g TOC (Figure 70a). The overall correlation between fertilization and concentration is r=-0.046 while the correlation between the variables considering just the three stations above the threshold is r=-0.862 (Figure 70b).

ERM Determinations. ERM values were determined for total DDT and total PCB using each of the toxicity endpoint measures and the quantile plots reflecting these determinations are shown in Figures 71-76. In addition, associated *effect* incidence values were also determined for each combination of endpoint and contaminant.

A comparison of the various AET and ERM values is given in Table 5. It is immediately apparent that the ERMs are considerably less than the corresponding AETs; on average, ERMs are approximately 83% less than the corresponding AETs. The lesser ERM values are not surprising considering that they are defined to be the median (i.e., 50th percentile) concentrations associated with the *effect* stations whereas the AETs are more equivalent to a much higher quantile (e.g., 90th percentile) of the same *effect* stations. However, in neither case, do the values represent toxicologically meaningful values since they do not provide a statistically significant distinction between *effect* and *no-effect* toxicity responses with respect to concentration of contaminants. Hence, it is not possible to conclude for either contaminant that a concentration above the ERM or AET would consistently exhibit toxicity. Similarly, it is not possible to conclude that concentrations below the ERM or AET would consistently exhibit no toxicity. Results of these analyses show that toxicity response categories are not distinguishable on the basis of contaminant concentration.

The magnitude of an effects incidence value (Table 5) has been used to judge reliability of a derived threshold in predicting an adverse biological response such as sediment toxicity. MacDonald (1994) arbitrarily chose a value ≥80% to indicate acceptable reliability and used this cutoff in deciding whether or not to designate the associated ERM as a sediment quality guideline. Based on analyses of the Fairey *et al.* (1996) data, it is evident that magnitude of effects incidence has virtually no predictive value unless there is a statistically significant difference in contaminant concentration among the *effect* and *no-effect* groups.

Table 5. Comparison of Apparent Effects Threshold (AET) and Effects Range - Median (ERM) for Total DDT and Total PCB using different biological endpoints. Data used in deriving these values were from San Diego Harbor marine sediments (Fairey et al 1996).

Contaminant/Threshold	Biological Endpoint		
	Amphipod Mortality	Sea Urchin Development	Sea Urchin Fertilization
Total DDT			
AET (µg/g TOC)	2.2	(a)	4.3
ERM (µg/g TOC)	0.6	0.7	0.7
Effects Incidence[b] (%)	46	80	70
Total PCB			
AET (µg/g TOC)	36.3	24.0	25.6
ERM (µg/g TOC)	3.8	4.1	4.3
Effects Incidence (%)	42	69	100

(a) Not Defined

(b) The Effects Incidence is variously referred to as the "Incidence of Adverse Effects" (Long et al 1995) or as "Predictability" (MacDonald 1994) for ERM-based thresholds and is determined as follows:

$$\text{Effects Incidence, \%} = \left[\frac{\text{No. Effect Stations} > \text{ERM}}{\text{Total Stations} > \text{ERM}}\right] \times 100$$

4.0 CONCLUSIONS

- The MacDonald (1997) report stated that the thresholds derived for DDTs and PCBs for Southern California Bight sediments "may be used as a scientific basis for establishing target clean-up levels for DDTs and PCBs in bed sediments..." The report further stated that DDT and PCB concentrations were in excess of the derived thresholds and hence, were "sufficient to cause injury to sediment-dwelling organisms". These conclusions may not be rigorously supported if the statistical limitations identified here apply equally to development of thresholds for the Southern California Bight. A detailed evaluation of all data used in the MacDonald (1997) report in developing these DDT and PCB thresholds is warranted. However, it must be noted that proponents of the ERM method (identified below) have stated that derived thresholds should never be used in the manner proposed by MacDonald (1997).

- Based on the statistical evaluations and rationales described herein, the AET and ERM methods have severe limitations in establishing sediment quality guidelines. Methods that rely on a co-occurrence database can be used to examine associations between levels of contamination and biological effects but do not support claims of cause-and-effect. As a result, these methods might only be appropriate as screening tools to identify sediments in areas that may require more detailed evaluation. The evaluations presented in this report suggest that neither method can support the specification of site-specific sediment cleanup goals or estimate injury in sediments containing PCB, DDT, lead, mercury, or zinc.

- Proponents of the ERM method recognized certain disadvantages that limit its use in establishing sediment quality guidelines. They indicated that 1) guidelines generated in this manner should not be used alone in deriving site-specific cleanup goals and 2) other sources of information and data should be obtained before developing site-specific sediment quality objectives. Guidelines developed under this methodology are designed to be used as generic or informal screening tools for assessing sediment quality applied over broad geographic areas (Long et al. 1995; CCME 1995; Smith et al. 1996b). These authors emphasized that the guidelines should be used with other sources of information such as background concentrations of chemicals, further biological assessment (e.g., toxicity test using field-collected sediments, benthic diversity and abundance analyses), and site management considerations before reaching any conclusions regarding sediment quality (Long et al. 1995; CCME 1995; Smith et al. 1996a,b).

- The limitations of such approaches were recently noted by O'Connor *et al.* (1998) who stated that "...ERM exceedance should only be taken to indicate that further analysis is in order. They should never be taken, by themselves, to mean that sediment is exerting a toxic effect upon the environment or that there would be any benefit to decreasing its

chemical content". These statements are clearly supported and substantiated by findings in the present report.

- MacDonald (1994) and Long et al. (1995) previously concluded that the guidelines developed for total DDT, p,p'-DDE, total PCB, and mercury were not "reliable". Reliability refers to the ability of the guidelines to correctly classify toxic and non-toxic sediments in the area of interest. MacDonald (1994) and Long et al. (1995) reached this conclusion based on sediment quality guidelines that were generated to be protective of marine and estuarine sediments on Florida's coasts and of sediments in all areas of the US and Canada, respectively. Our demonstration of deriving misleading (i.e. unreliable) AET and ERM values supports this conclusion.

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Appendix A

Figures Associated With Statistical Analyses and Threshold Determinations

Figure 1

Pb in CA Sediments

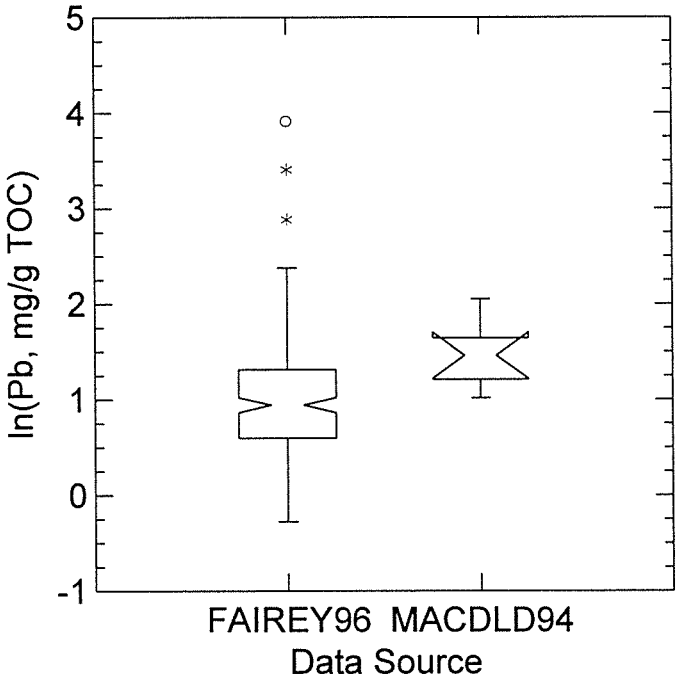


Figure 2

Pb Toxicity of CA Sediments

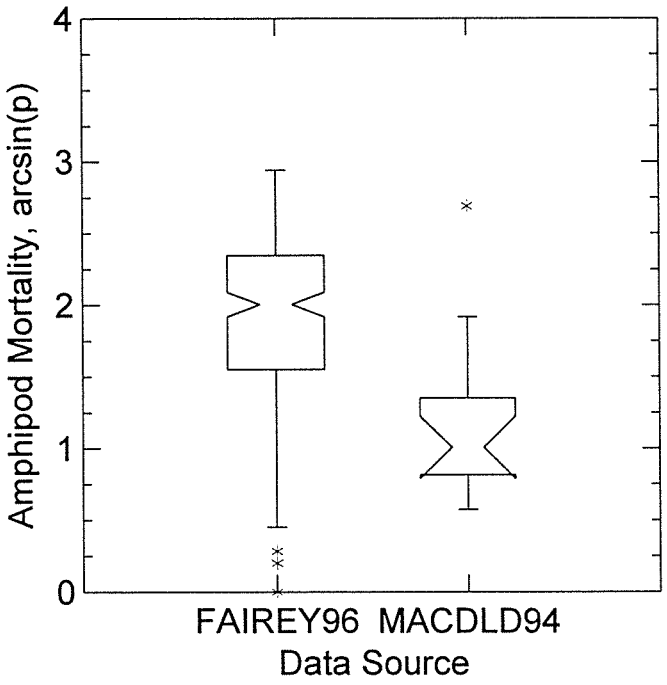


Figure 3

Mortality vs Pb for CA Sediments

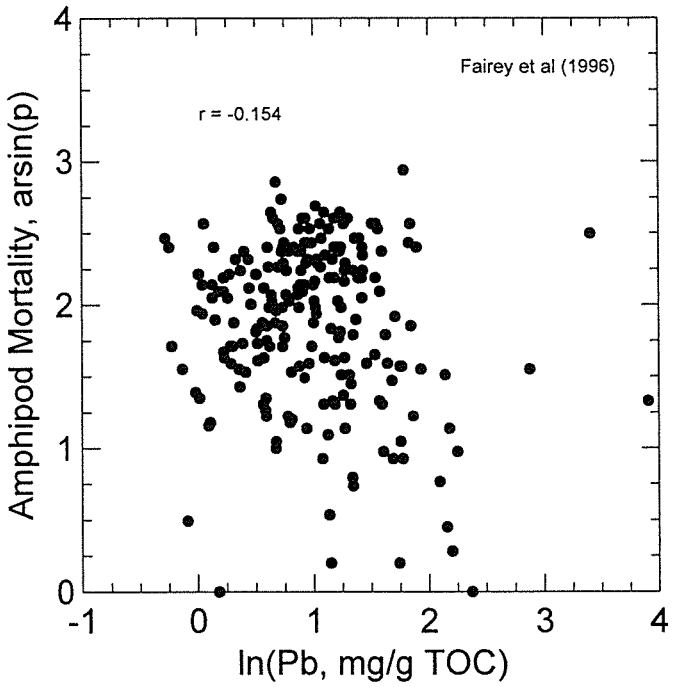


Figure 4

Mortality vs Pb for CA Sediments

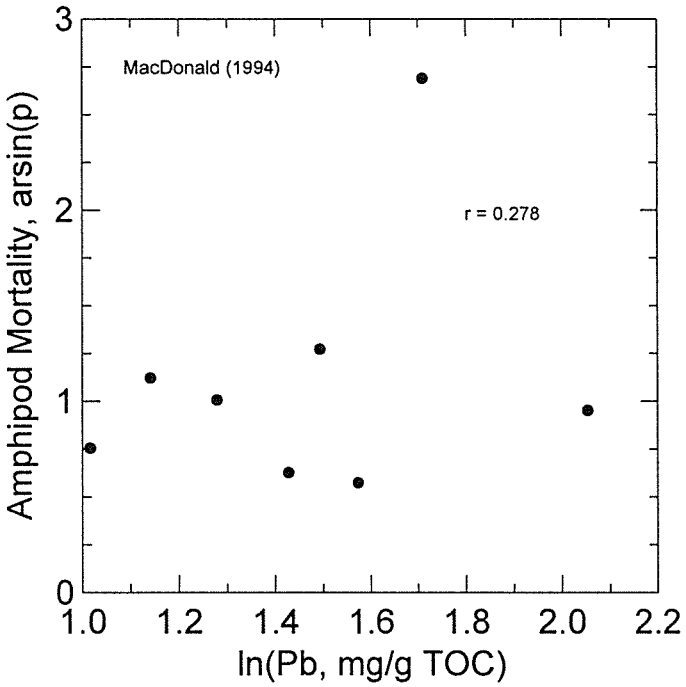


Figure 5

Pb in Marine Sediments

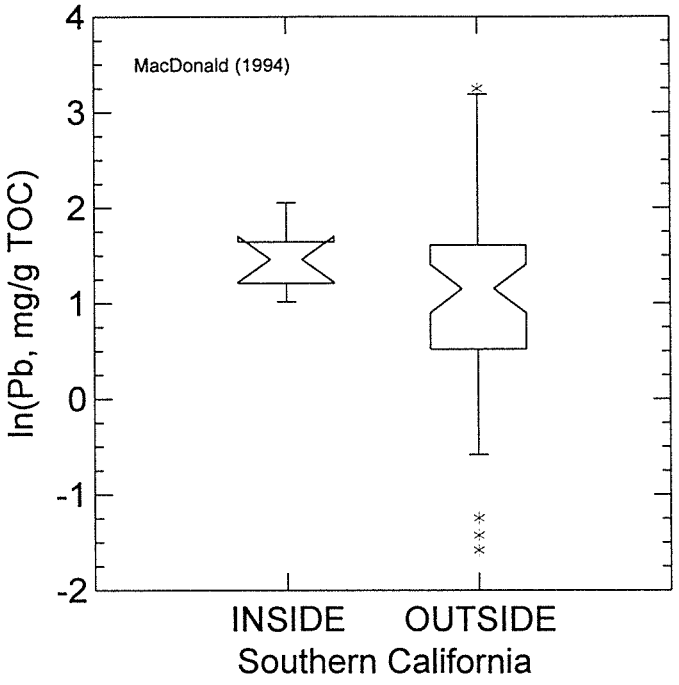


Figure 6

Pb Toxicity of Marine Sediments

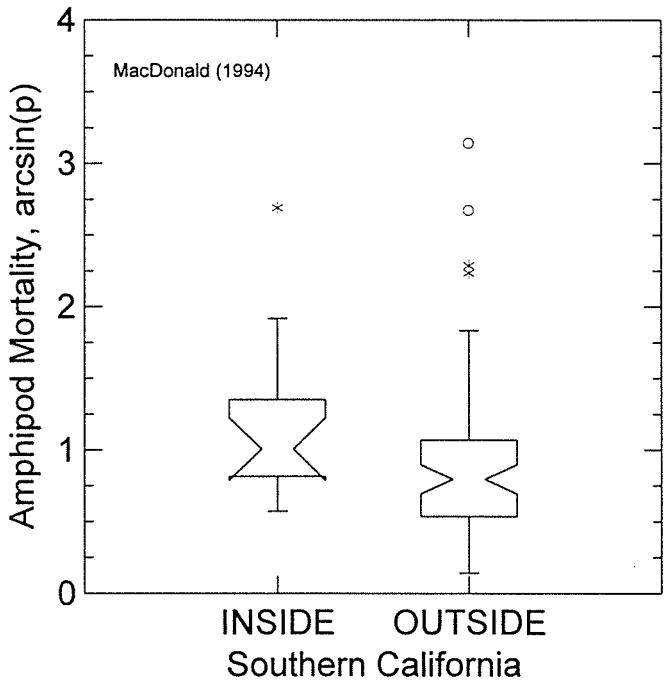


Figure 7

Mortality vs Pb in Marine Sediments

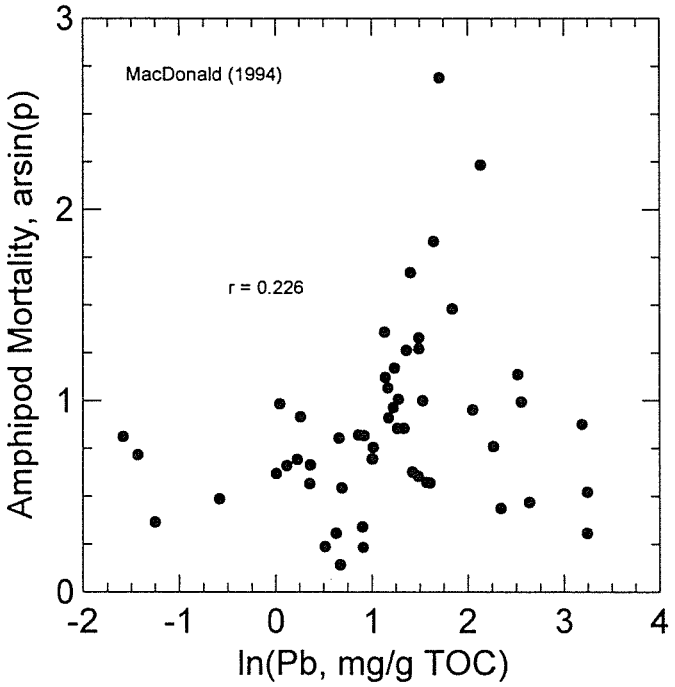


Figure 8

Development vs Pb for CA Sediments

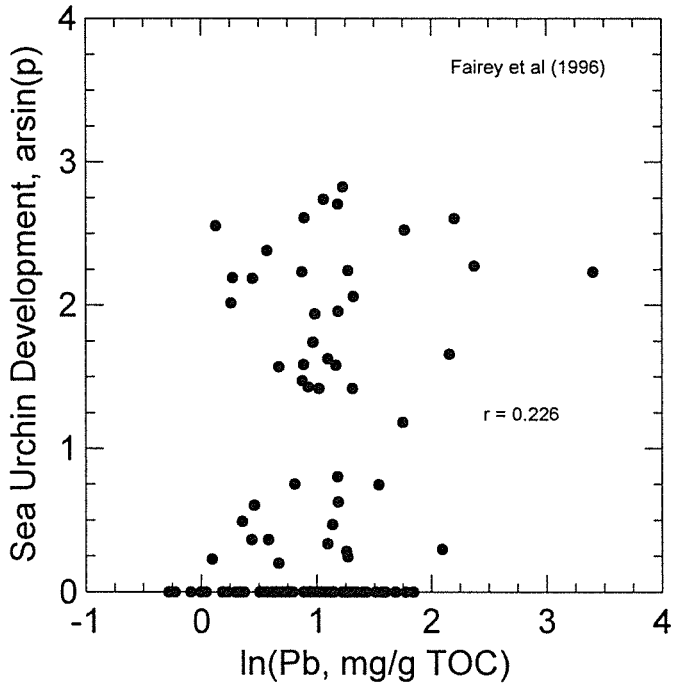


Figure 9

Fertilization vs Pb for CA Sediments

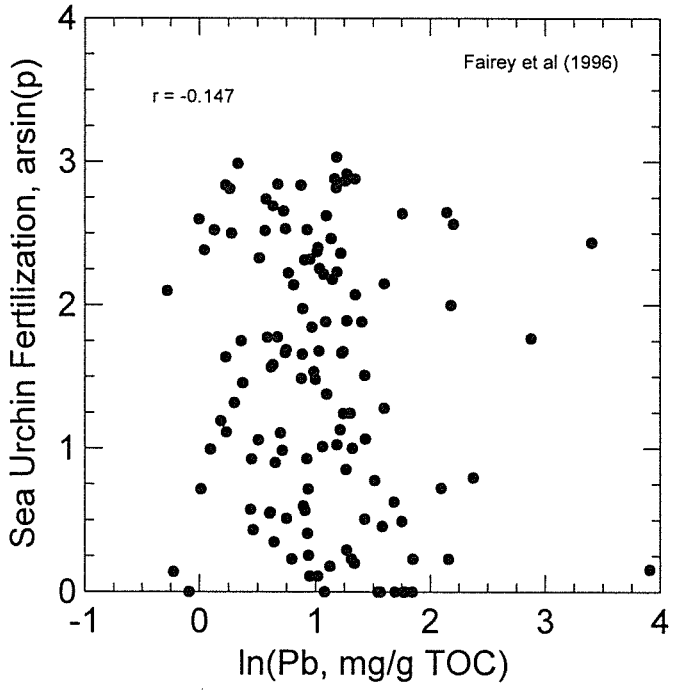


Figure 10

Density vs Pb for FW Sediments

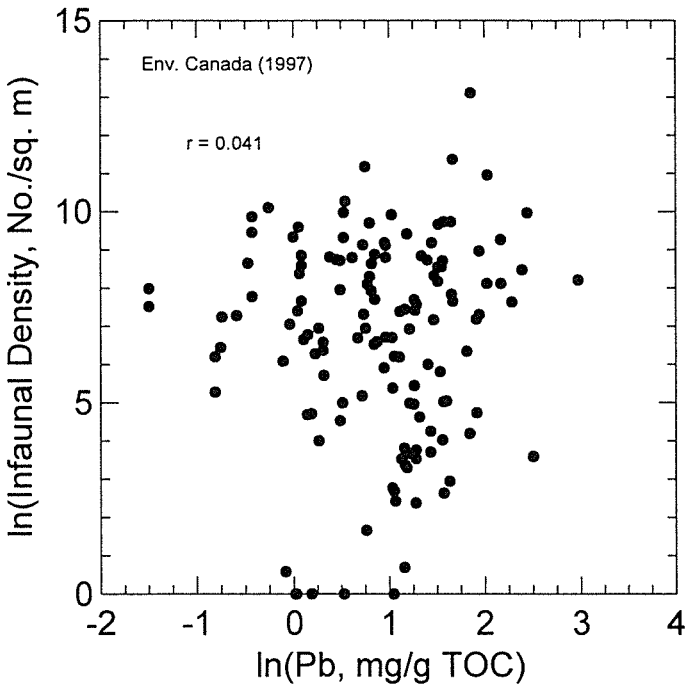


Figure 11

Diversity vs Pb for FW Sediments

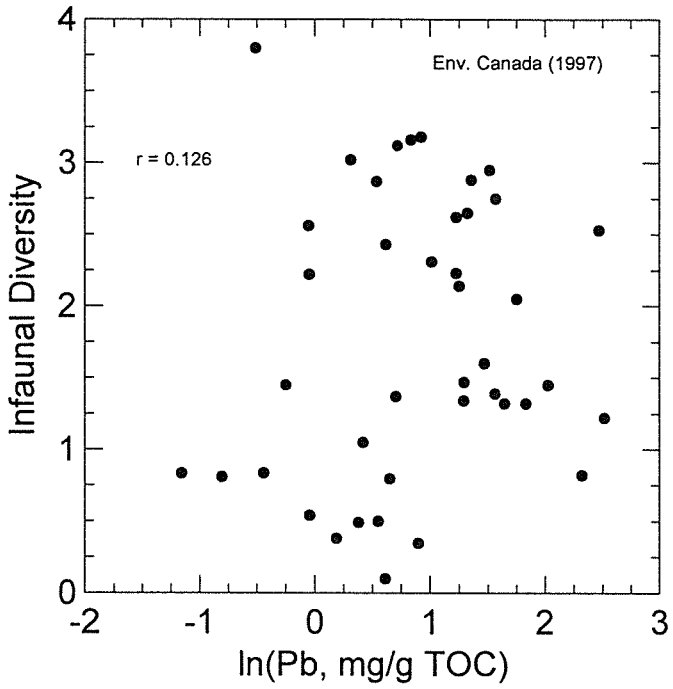


Figure 12

Density vs Pb for FW Sediments

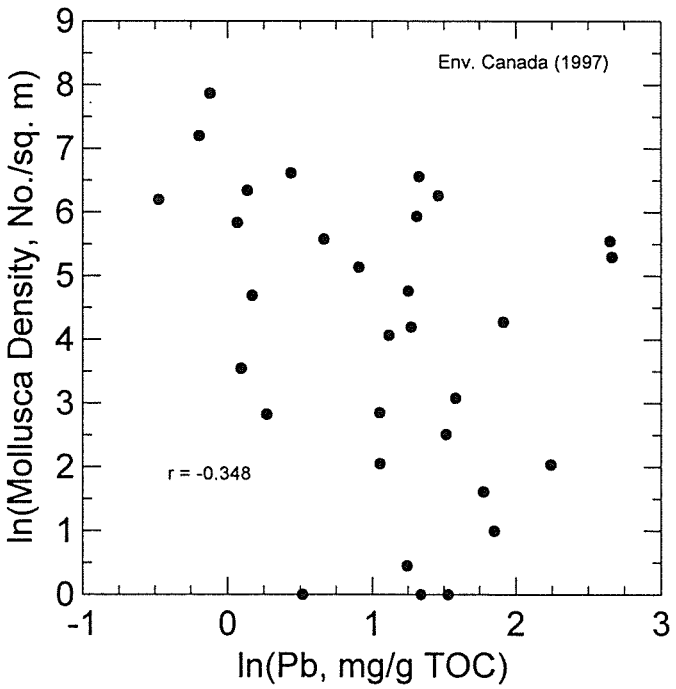


Figure 13

Hg in Marine Sediments

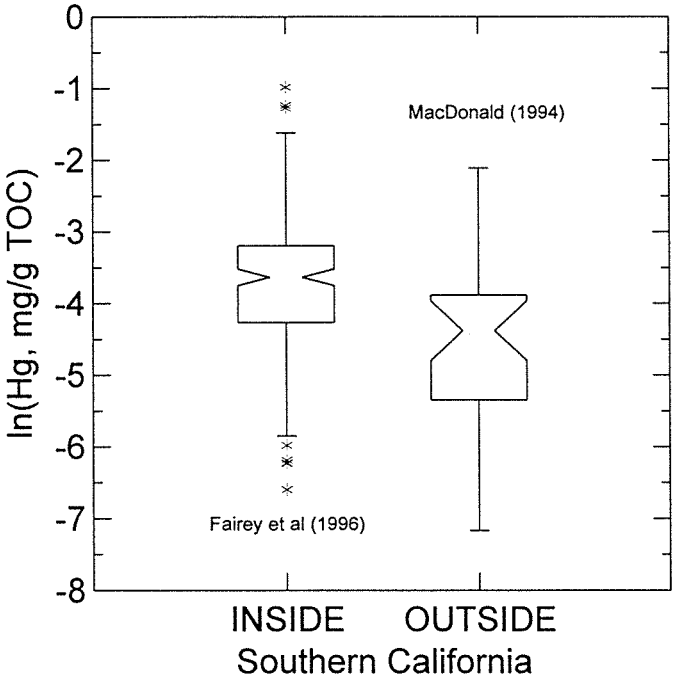


Figure 14

Hg Toxicity in Marine Sediments

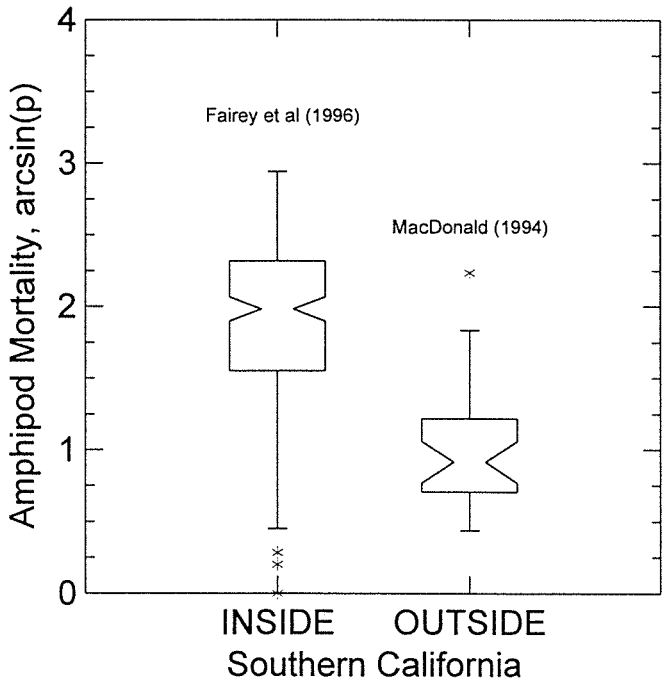


Figure 15

Hg Toxicity in CA Sediments

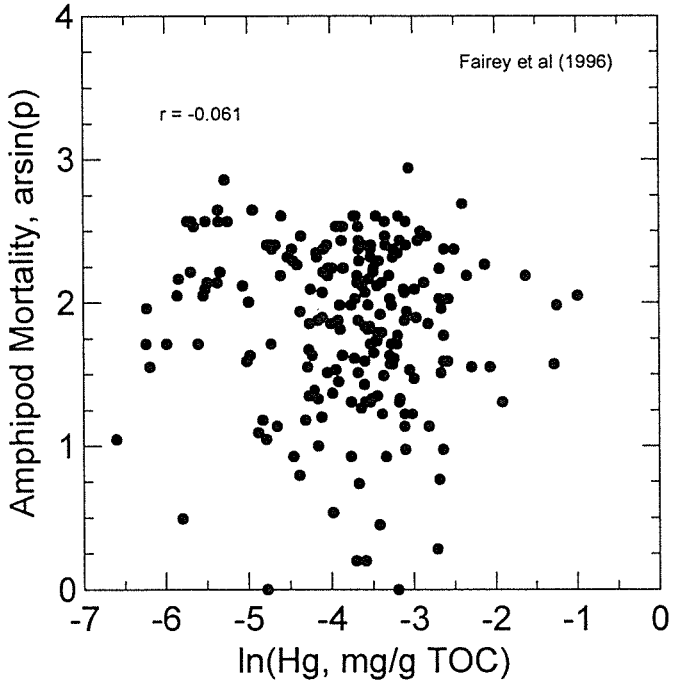


Figure 16

Hg Toxicity in NonCA Sediments

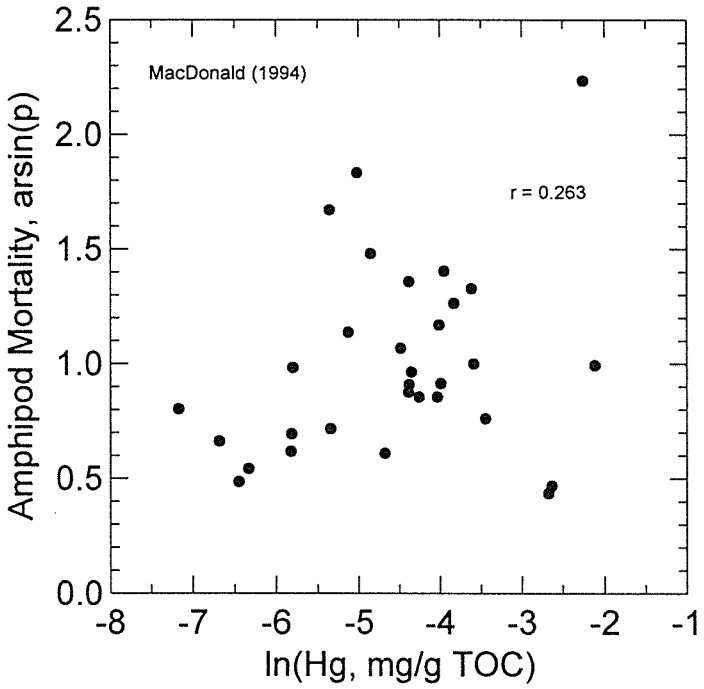


Figure 17

Hg in Marine Sediments

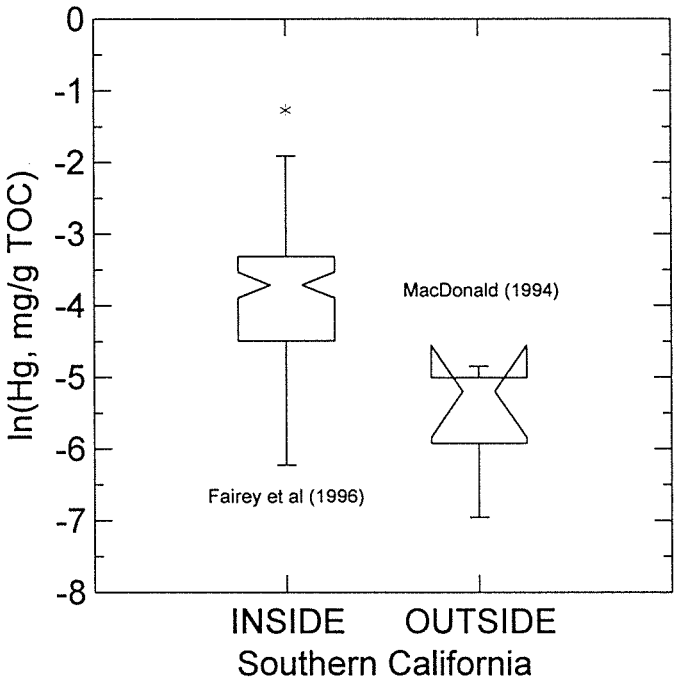


Figure 18

Hg Toxicity in Marine Sediments

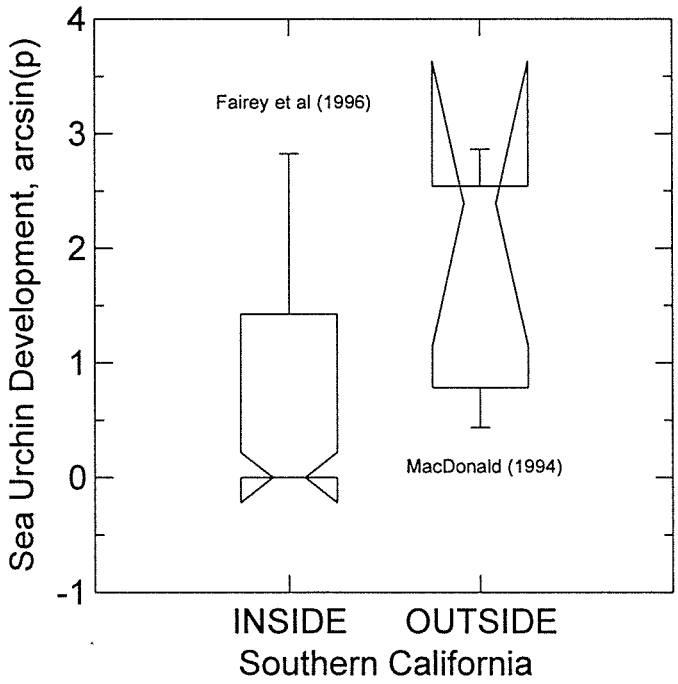


Figure 19

Hg Toxicity in NonCA Sediments

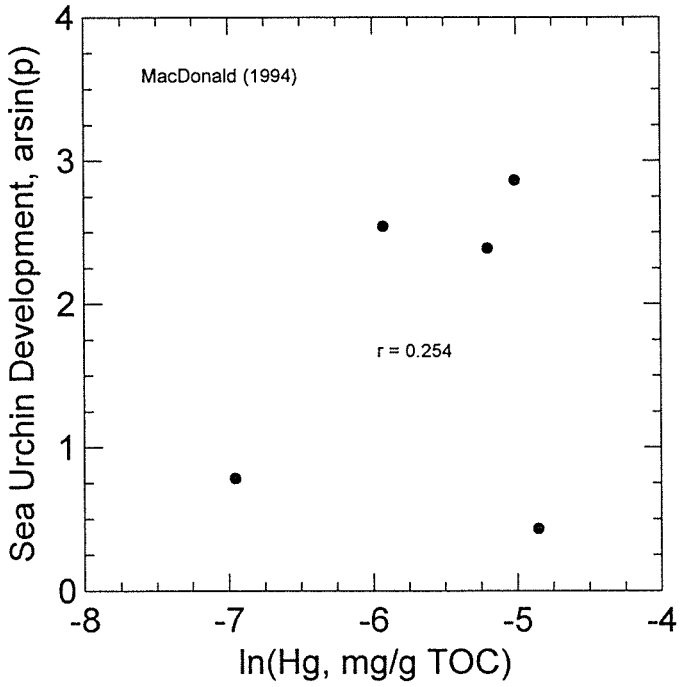


Figure 20

Development vs Hg for CA Sediments

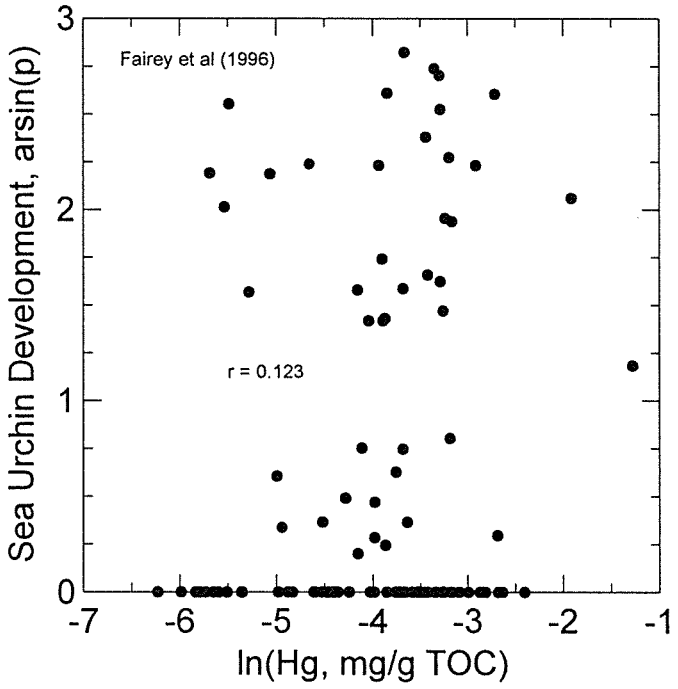


Figure 21

Fertilization vs Hg for CA Sediments

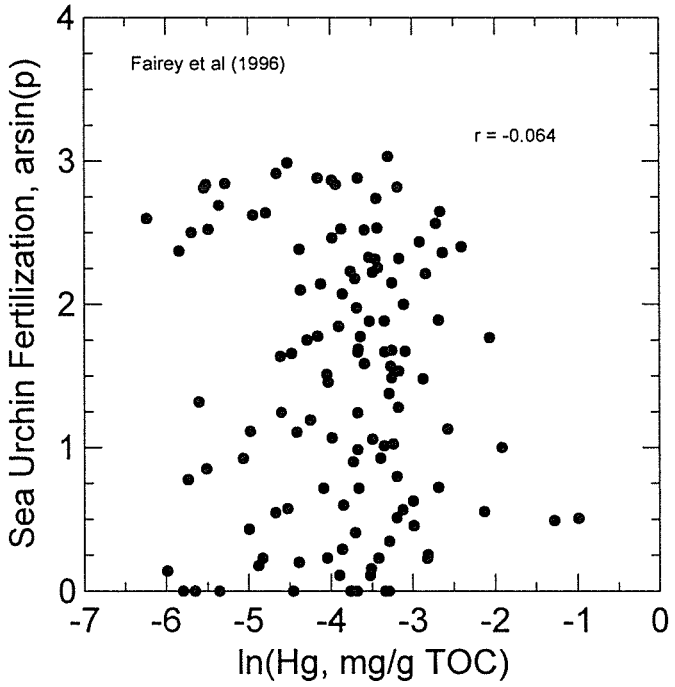


Figure 22

Density vs Hg for FW Sediments

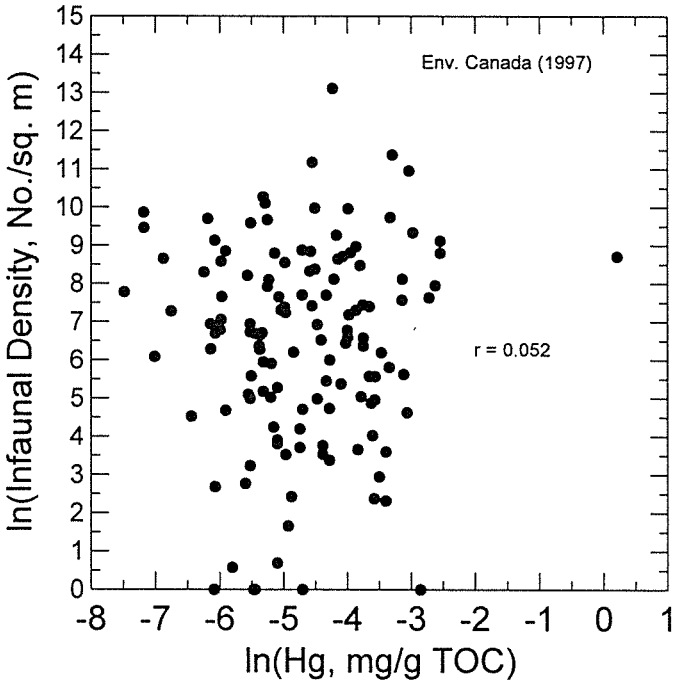


Figure 23

Diversity vs Hg for FW Sediments

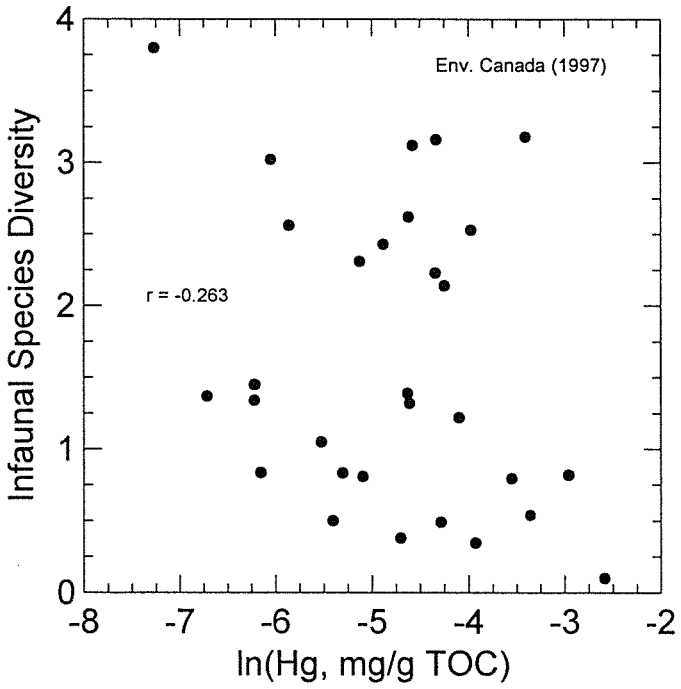


Figure 24

Density vs Hg for FW Sediments

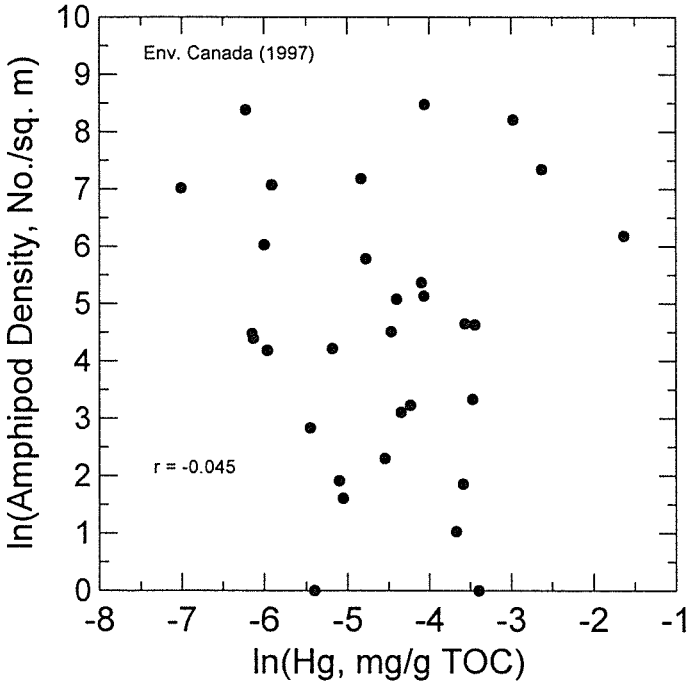


Figure 25

Density vs Hg for FW Sediments

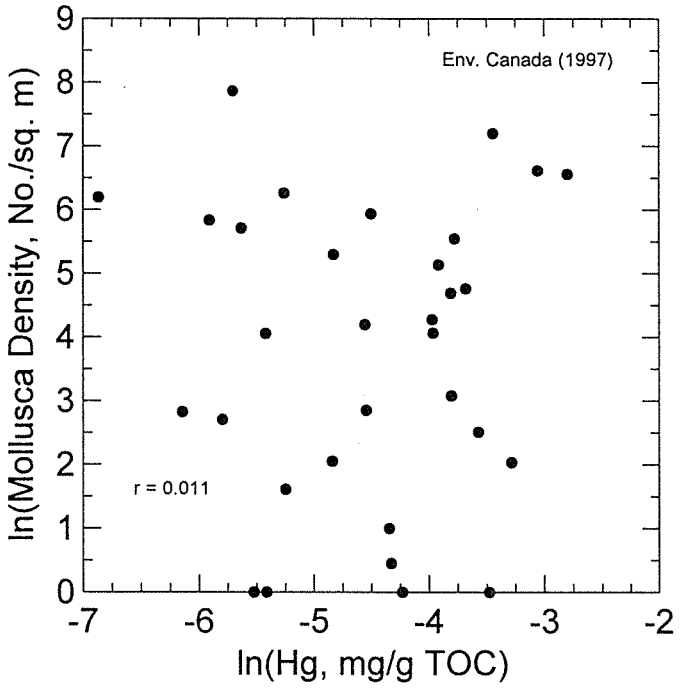


Figure 26

Zn in Marine Sediments

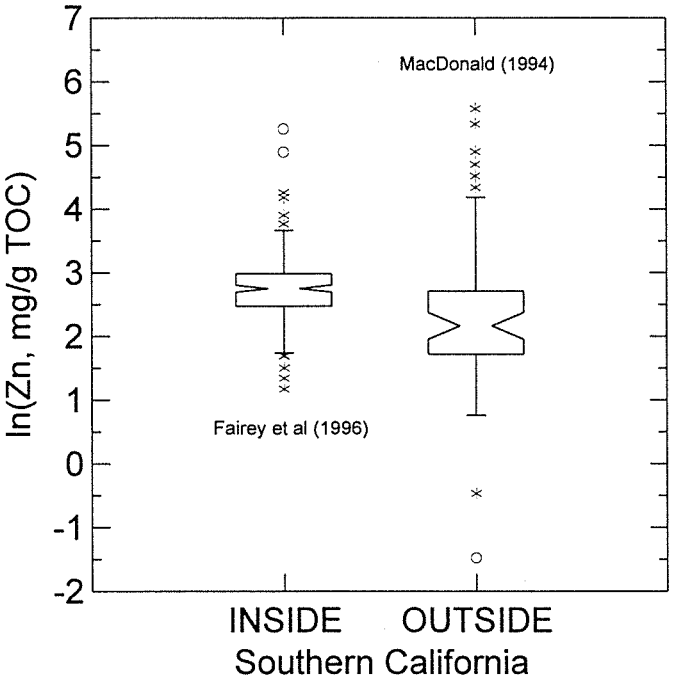


Figure 27

Zn Toxicity in CA Sediments

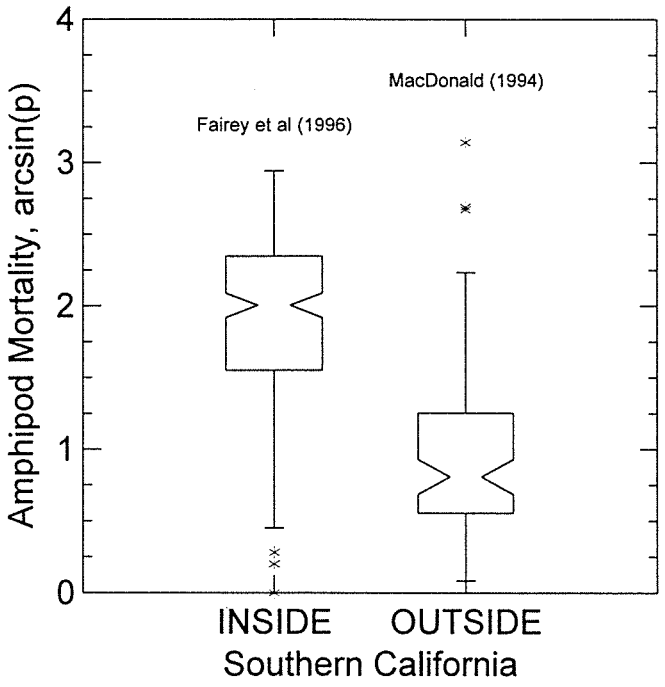


Figure 28

Zn Toxicity in CA Sediments

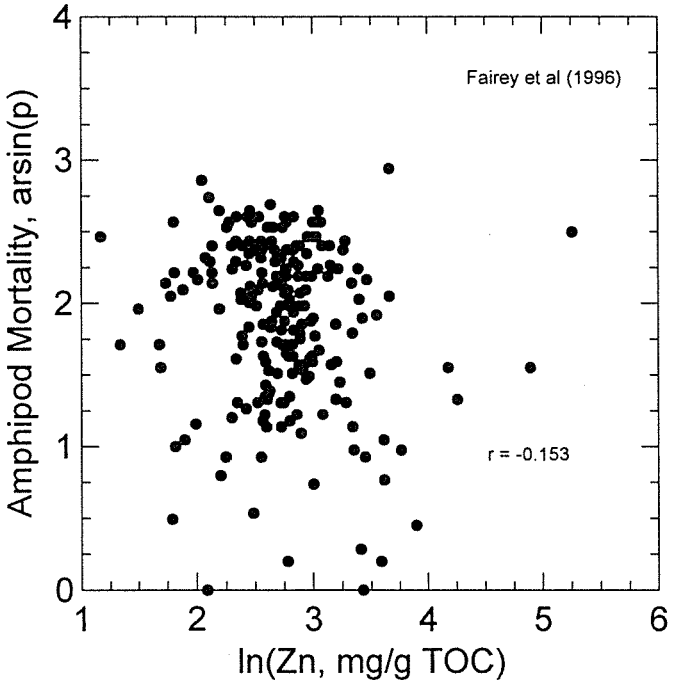


Figure 29

Zn Toxicity in NonCA Sediments

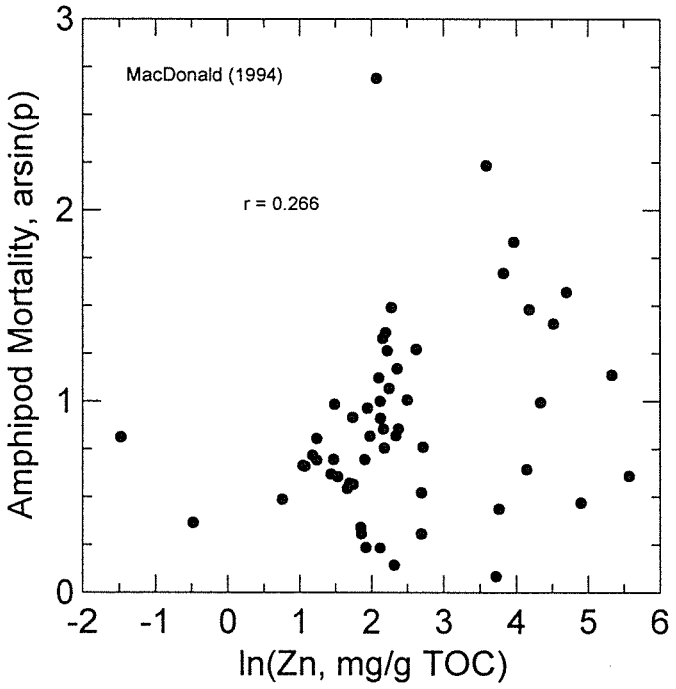


Figure 30

Zn in Marine Sediments

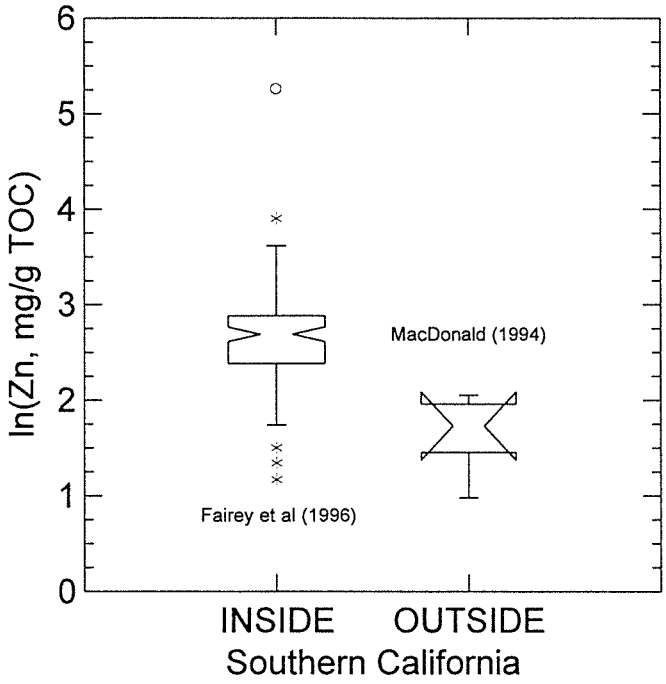


Figure 31

Zn Toxicity in Marine Sediments

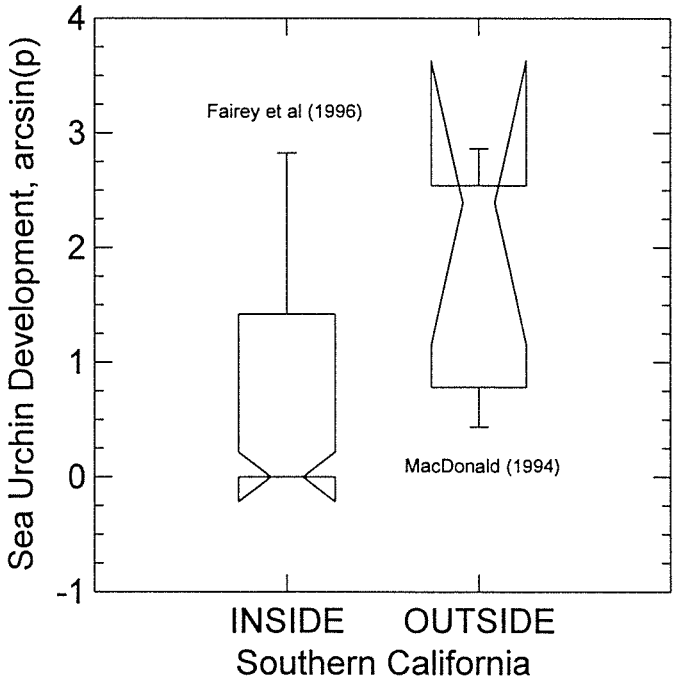


Figure 32

Development vs Zn for CA Sediments

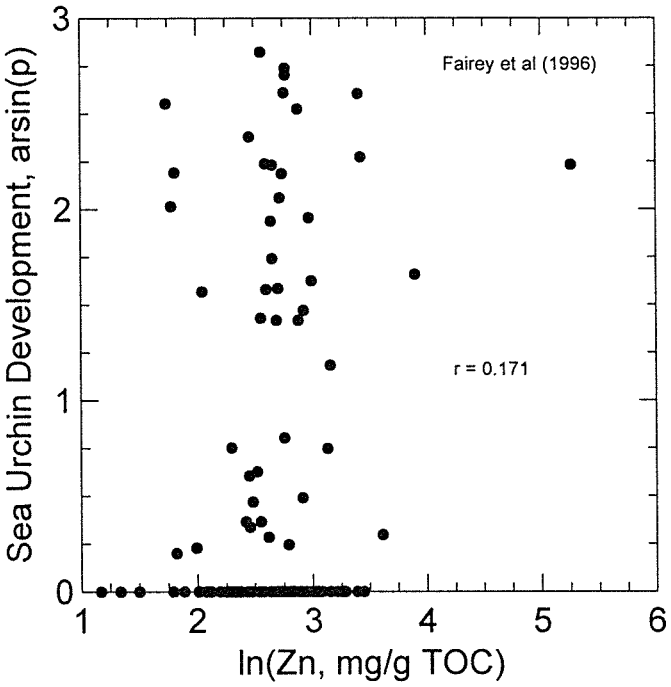


Figure 33

Development vs Zn for NonCA Sediments

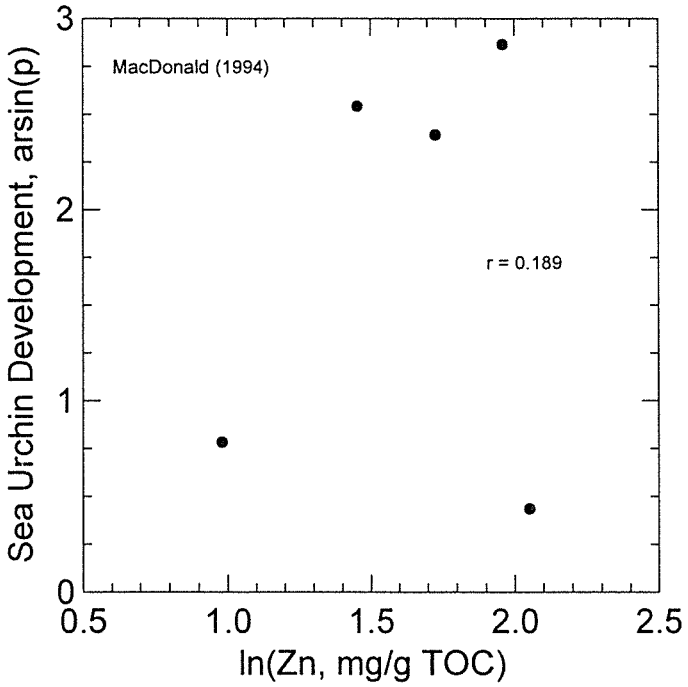


Figure 34

Fertilization vs Zn for CA Sediments

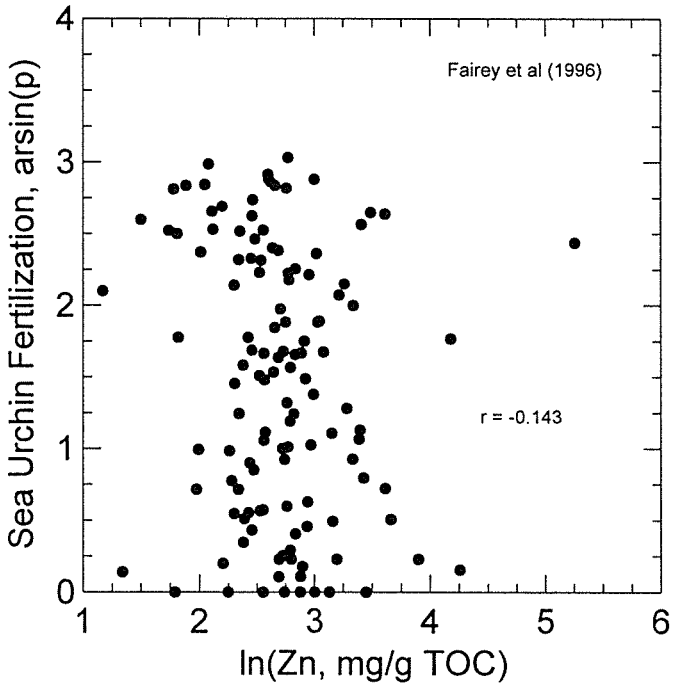


Figure 35

PCB in CA Sediments

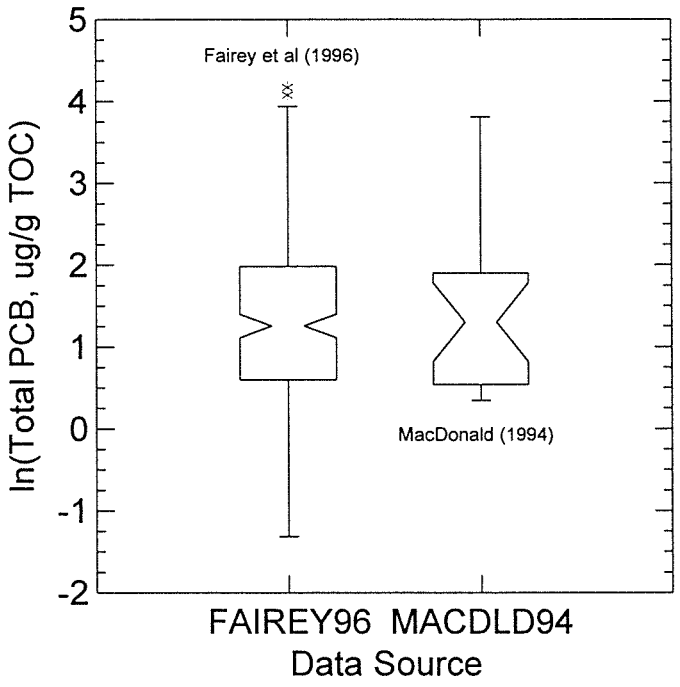


Figure 36

PCB Toxicity in CA Sediments

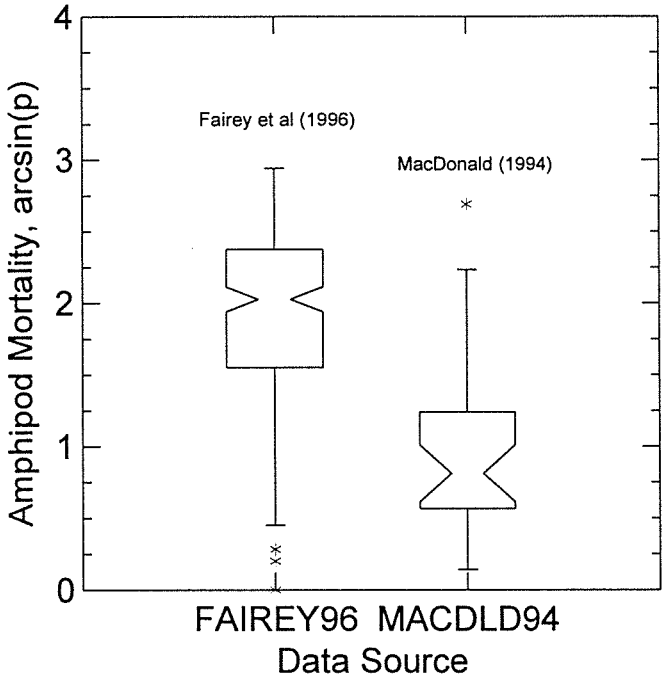


Figure 37

PCB Toxicity in CA Sediments

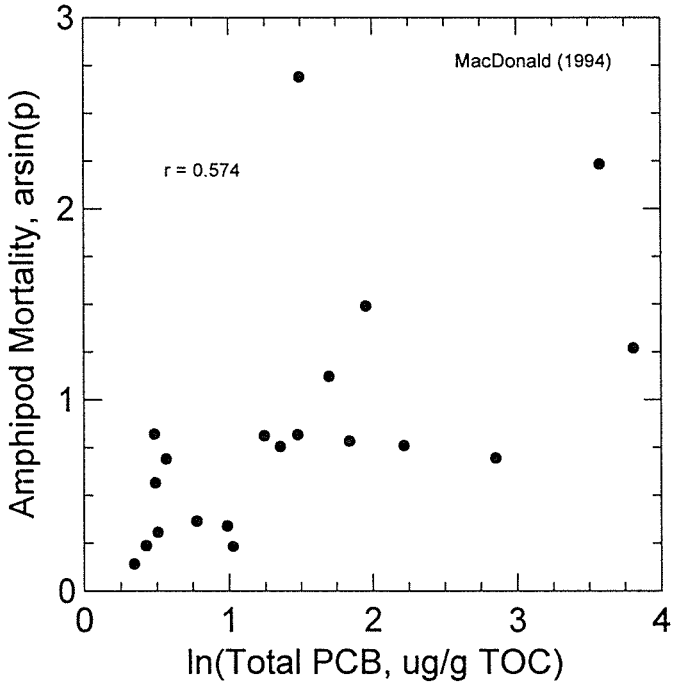


Figure 38

PCB Toxicity in CA Sediments

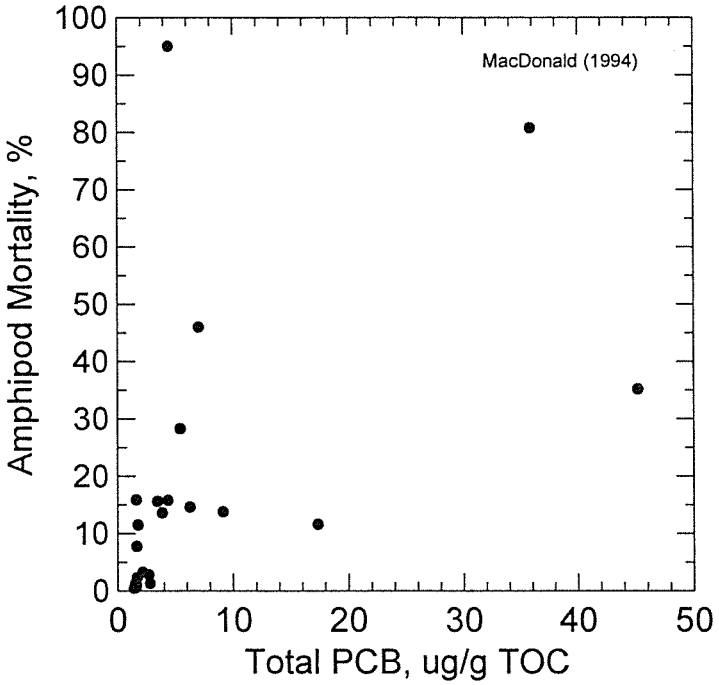


Figure 39

PCB Toxicity in CA Sediments

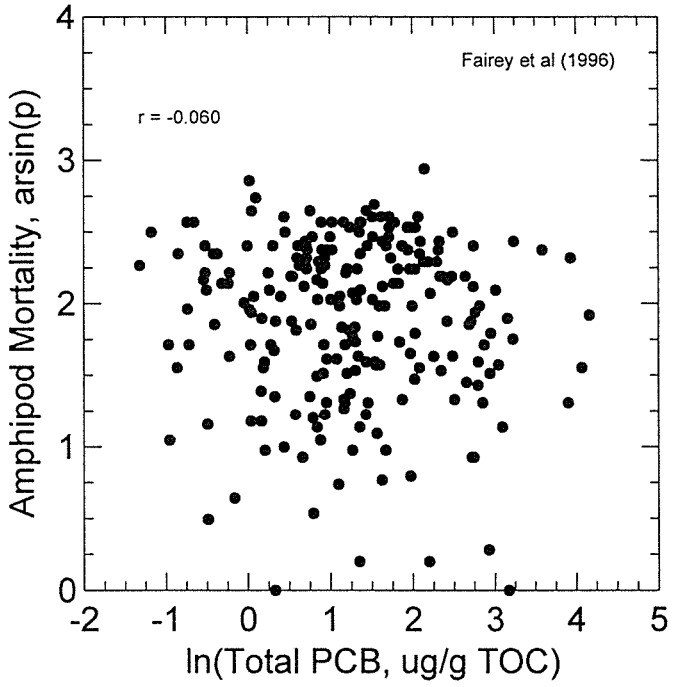


Figure 40

PCB in CA Sediments

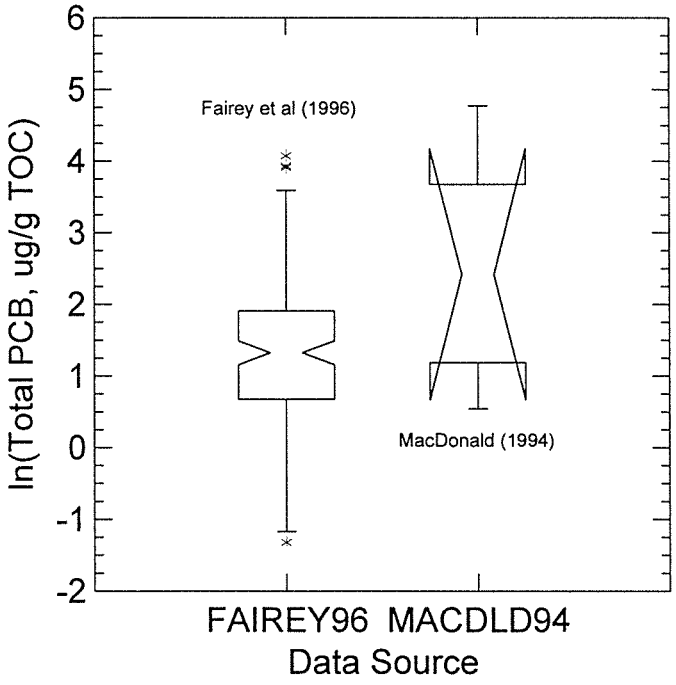


Figure 41

PCB Toxicity in CA Sediments

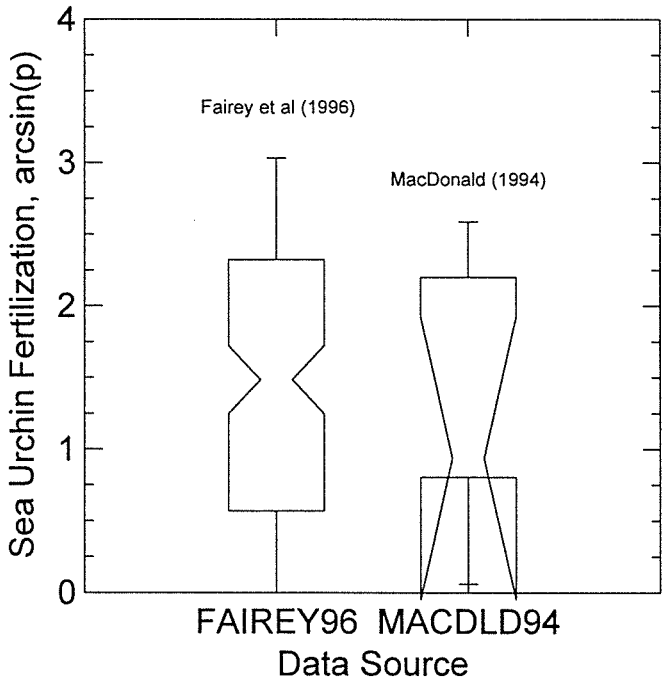


Figure 42

Fertilization vs PCB for CA Sediments

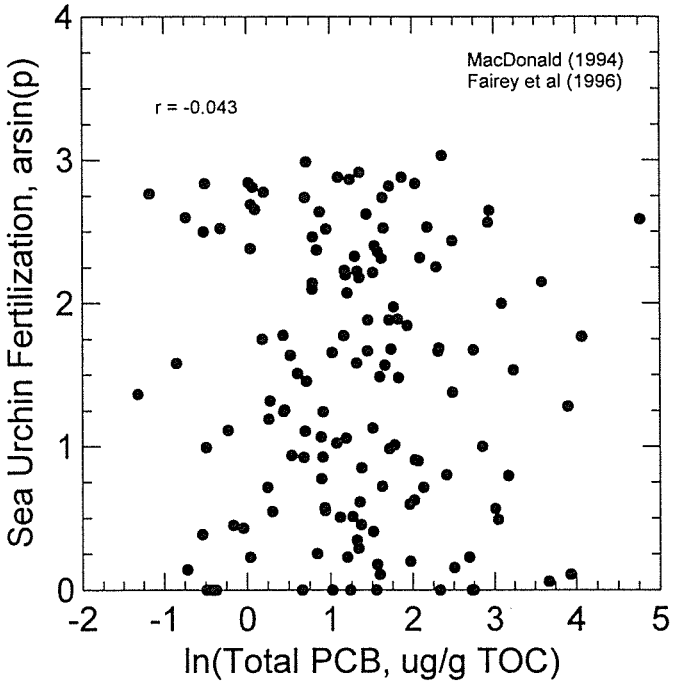


Figure 43

Development vs PCB for CA Sediments

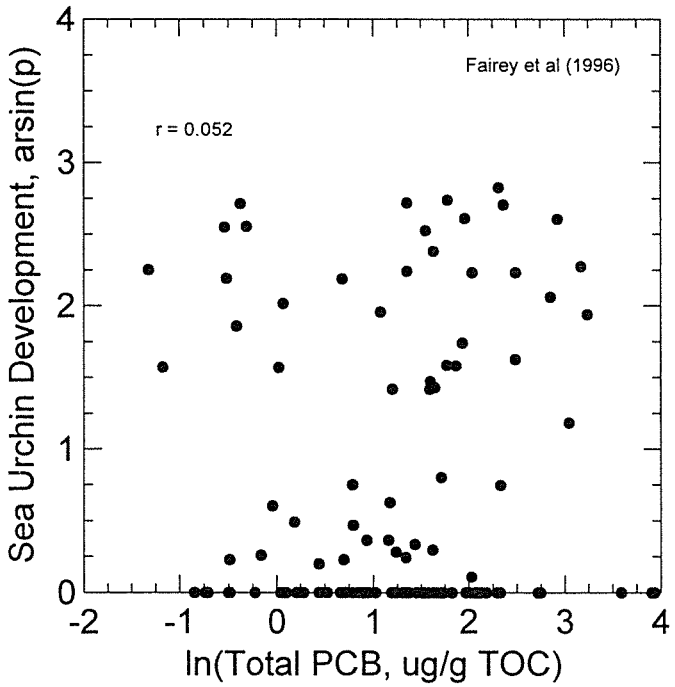


Figure 44

Density vs PCB for FW Sediments

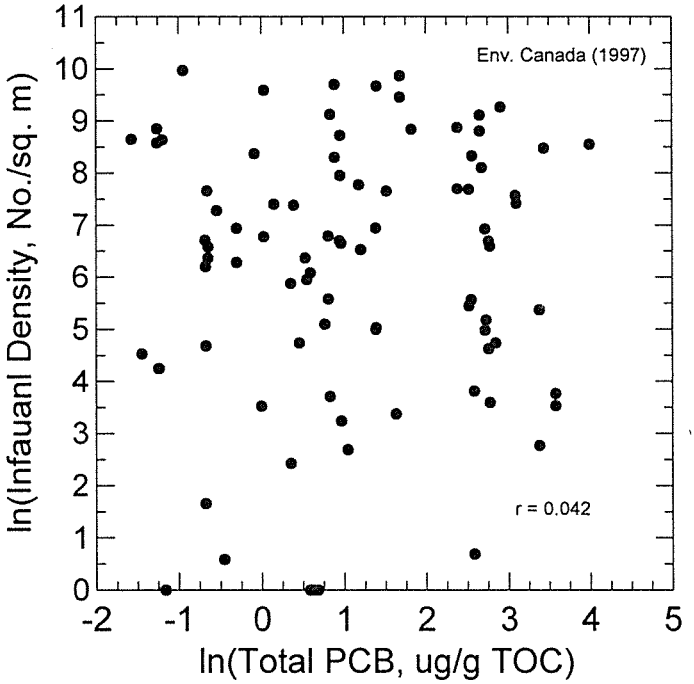


Figure 45

Mortality vs SDDT for CA Sediments

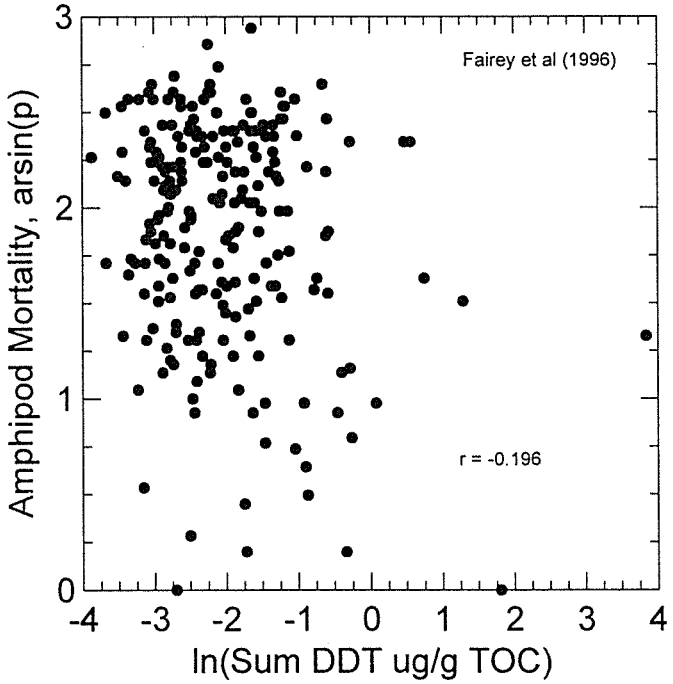


Figure 46

Development vs SDDT for CA Sediments

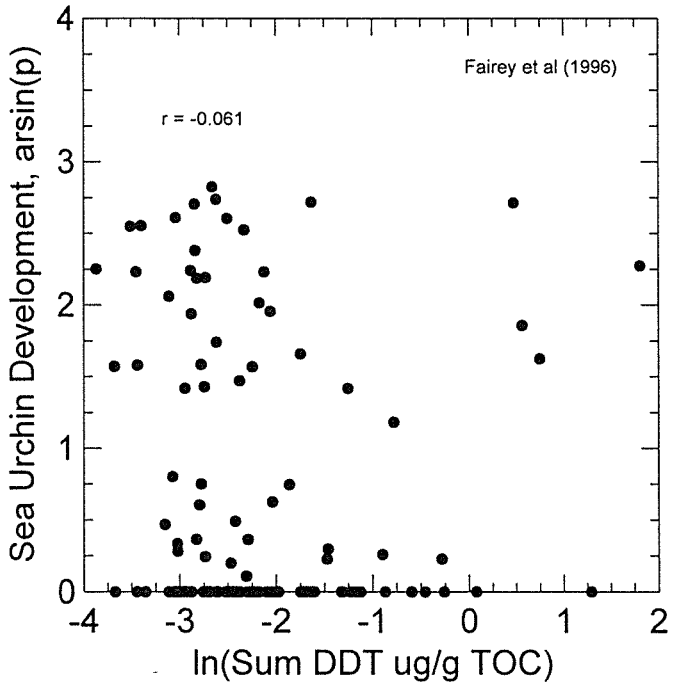


Figure 47

Fertilization vs SDDT for CA Sediments

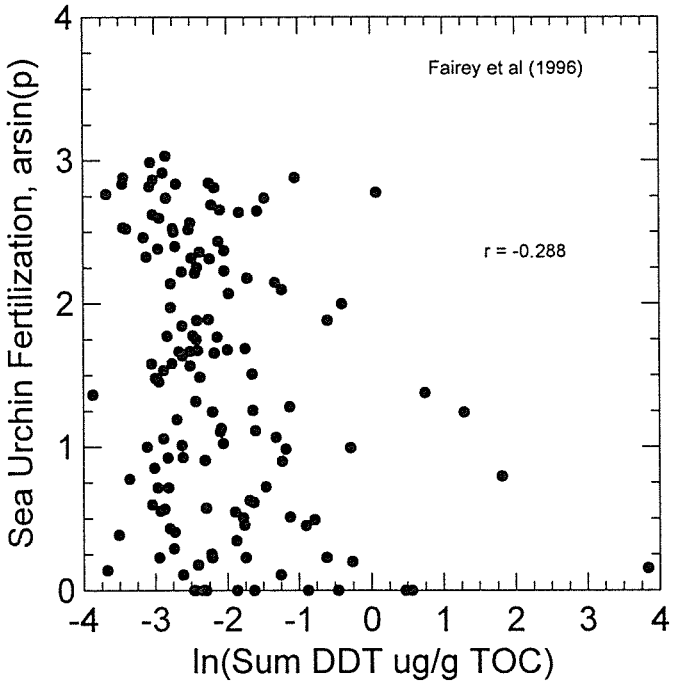


Figure 48

Density vs SDDD for FW Sediments

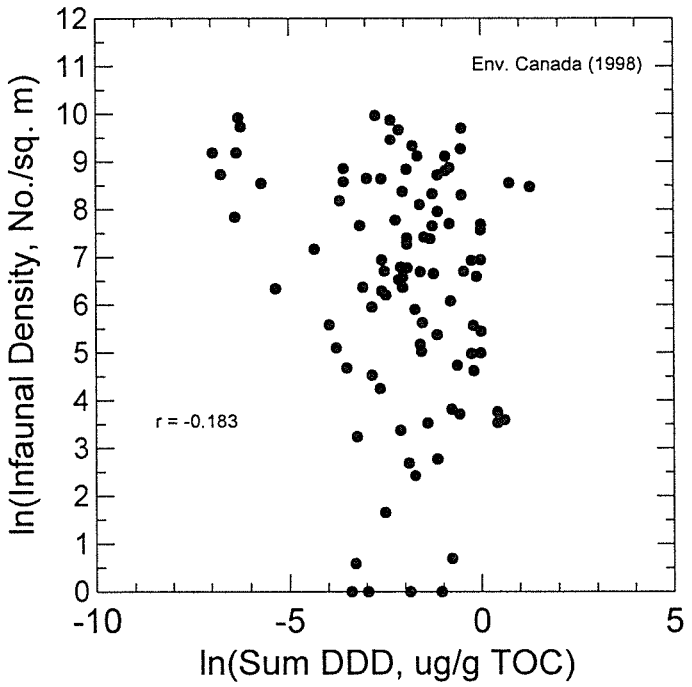


Figure 49

Diversity vs SDDD for FW Sediments

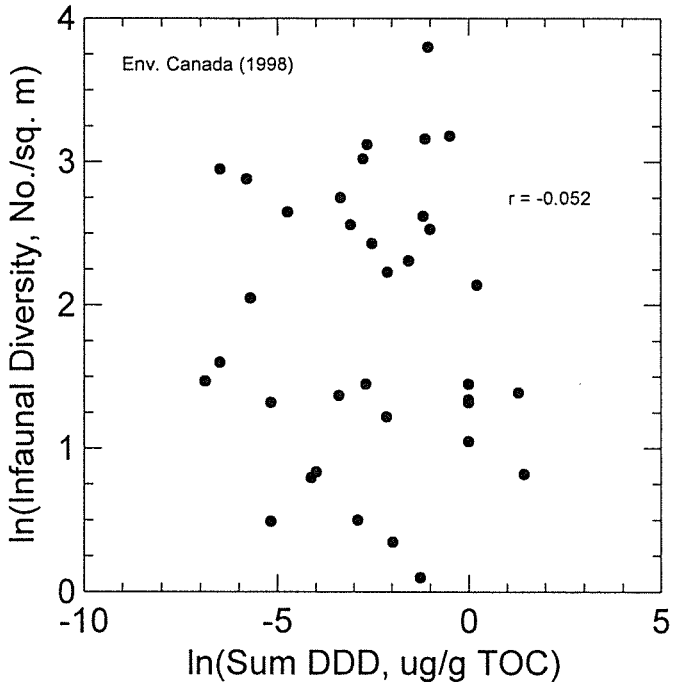


Figure 50

Density vs SDDE for FW Sediments

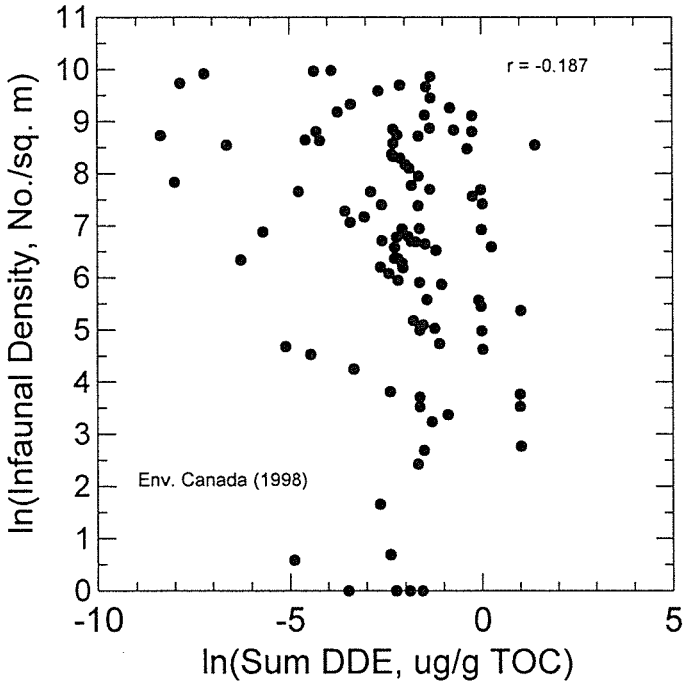


Figure 51

Diversity vs SDDE for FW Sediments

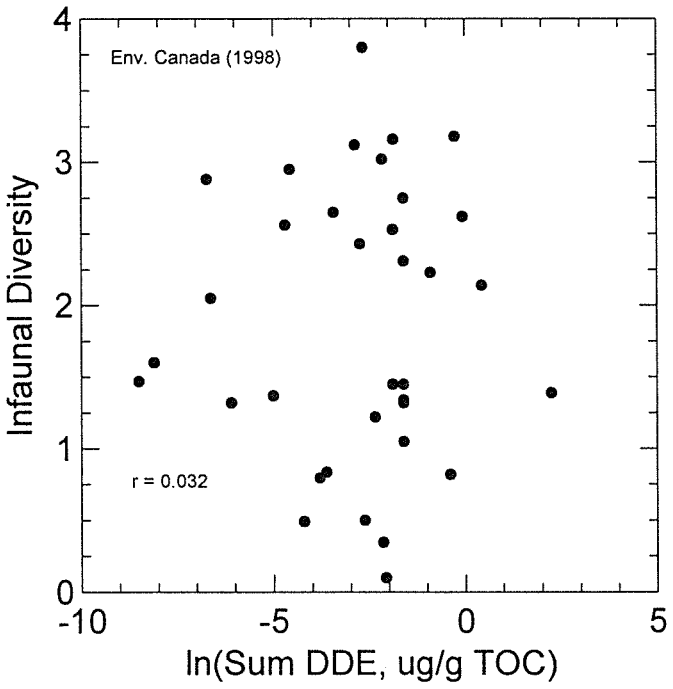


Figure 52

Total DDT in CA Sediments

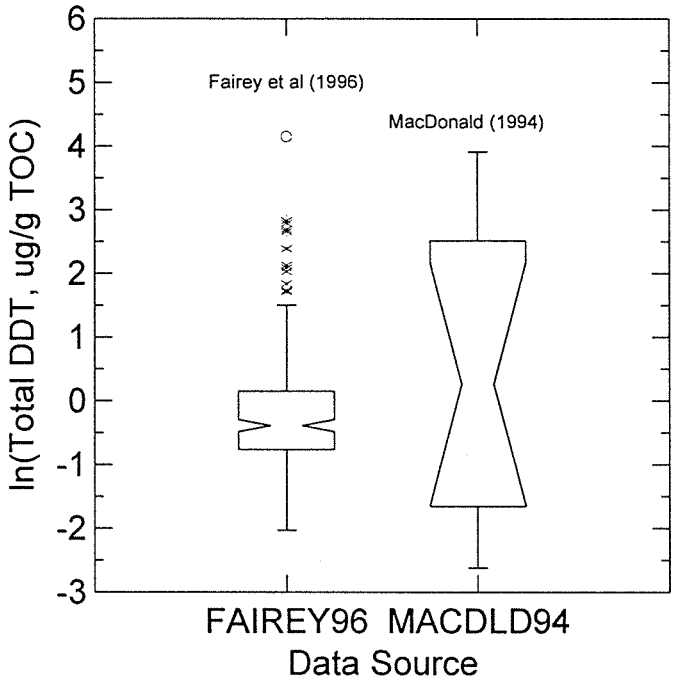


Figure 53

Total DDT Toxicity in CA Sediments

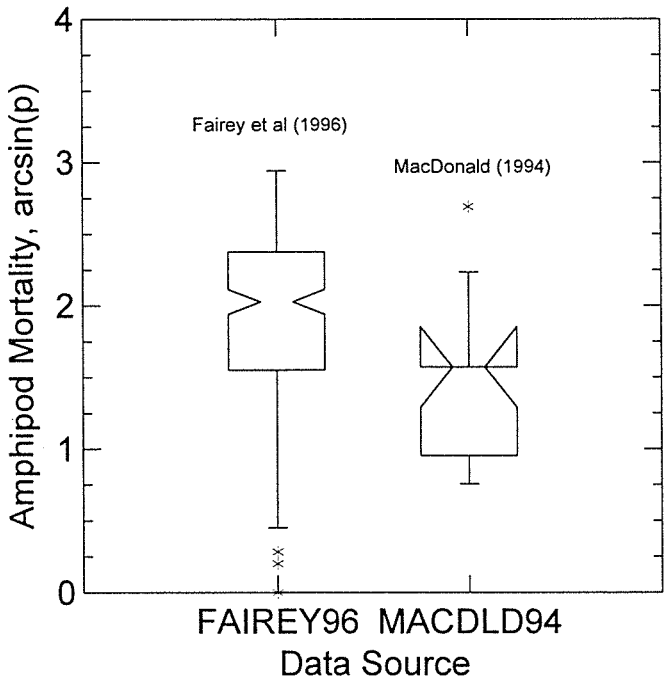


Figure 54

Mortality vs TDDT for CA Sediments

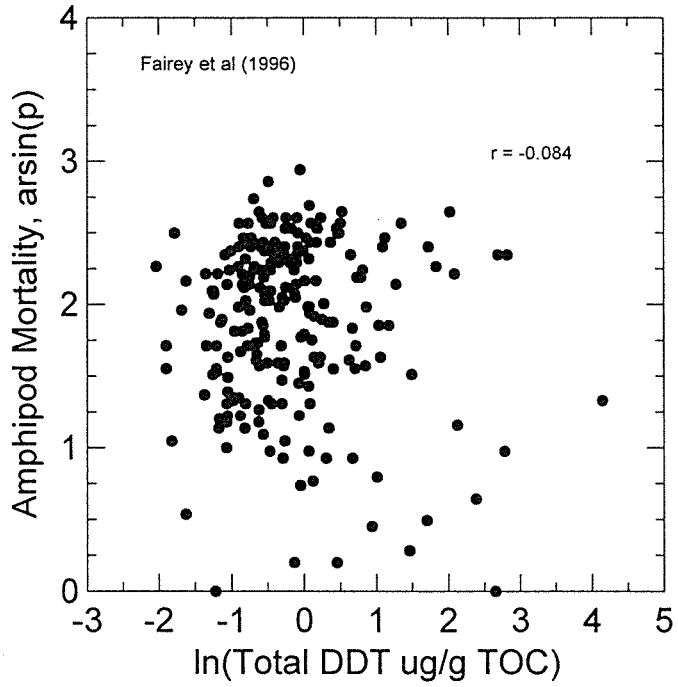


Figure 55

Mortality vs TDDT for CA Sediments

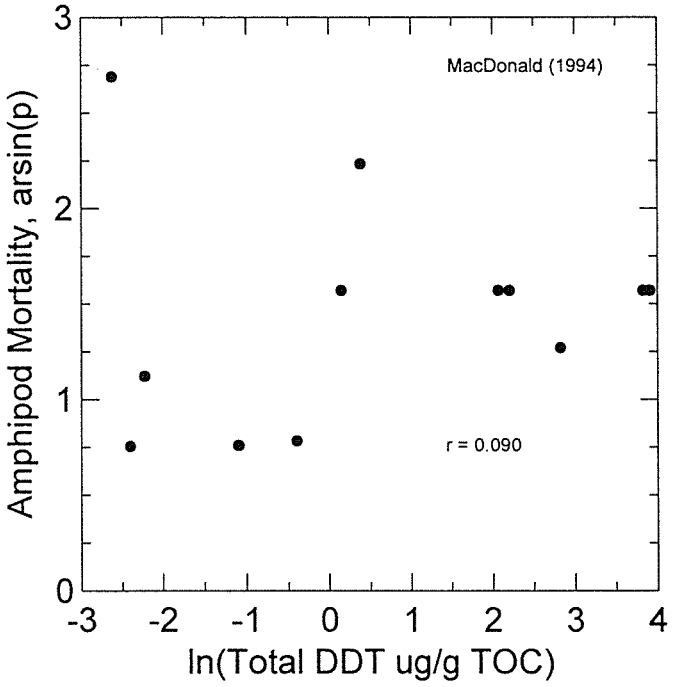


Figure 56

Total DDT in CA Sediments

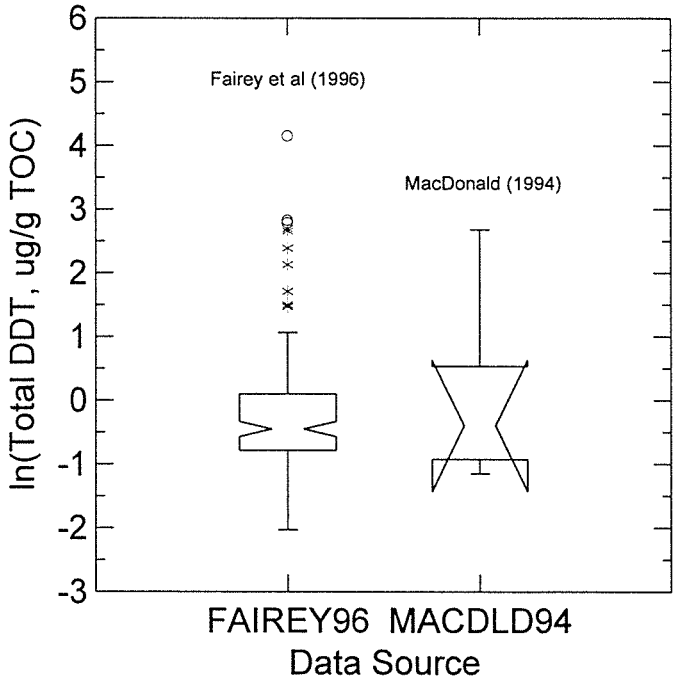


Figure 57

Total DDT Toxicity in CA Sediments

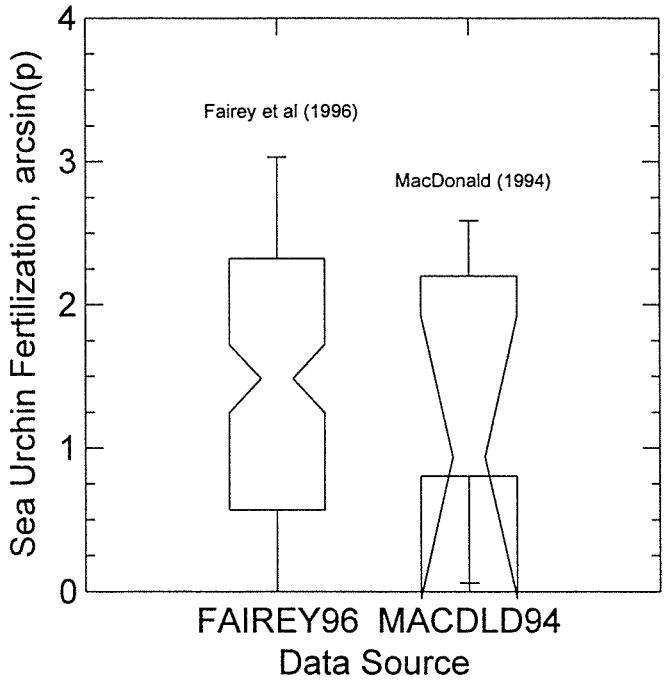


Figure 58

Fertilization vs TDDT for CA Sediments

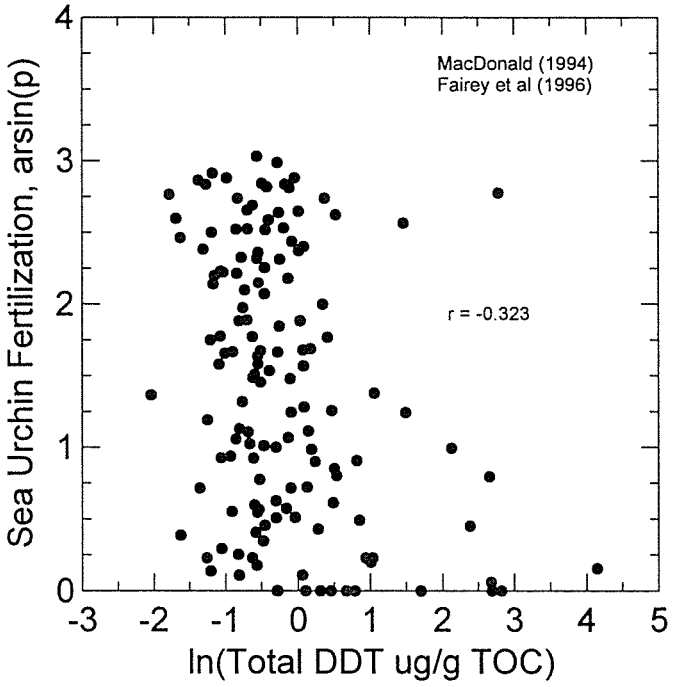


Figure 59

Mortality Effects and Total DDT

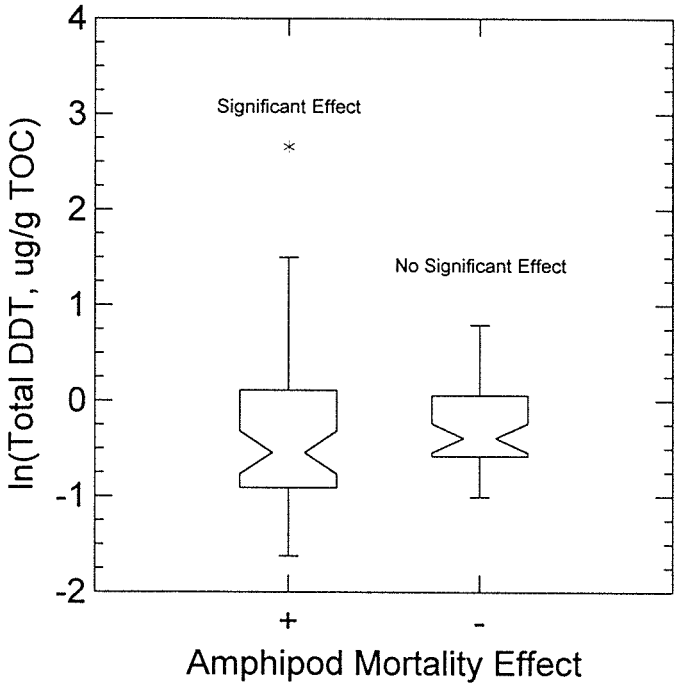


Figure 60

Development Effects and Total DDT

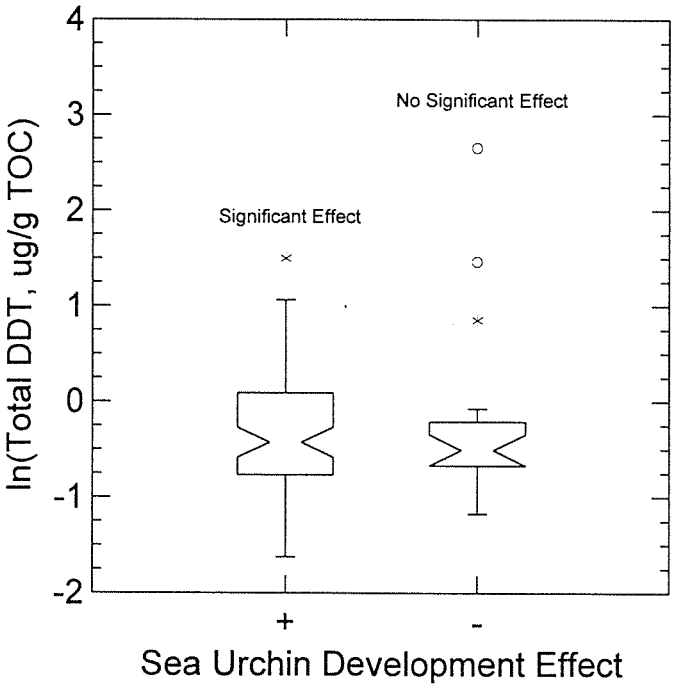


Figure 61

Fertilization Effects and Total DDT

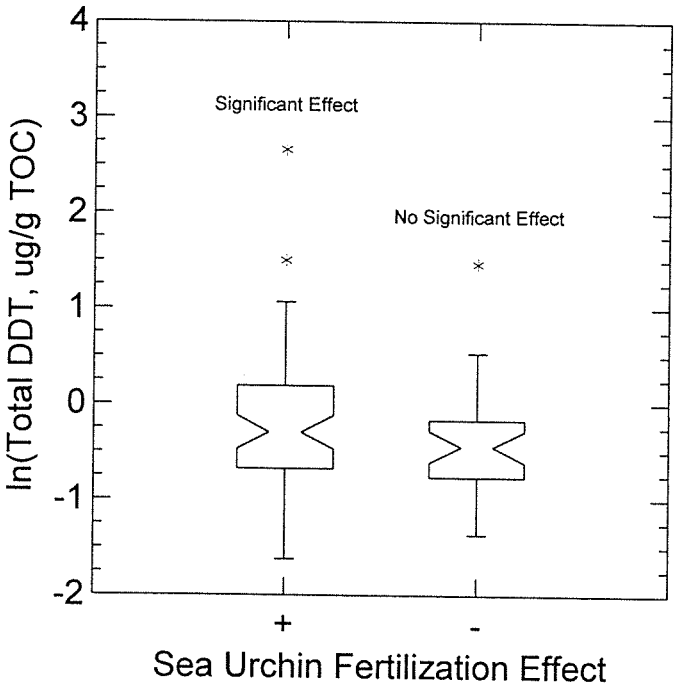


Figure 62

Mortality Effects and Total PCB

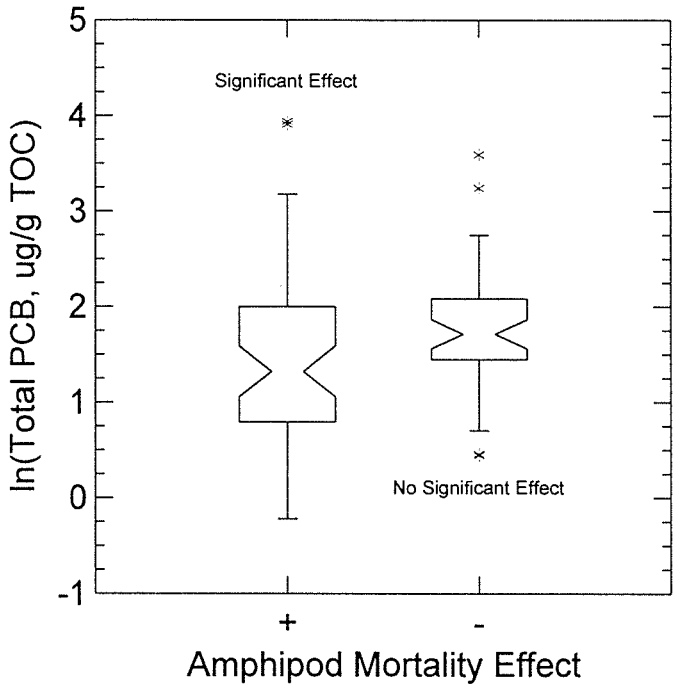


Figure 63

Development Effects and Total PCB

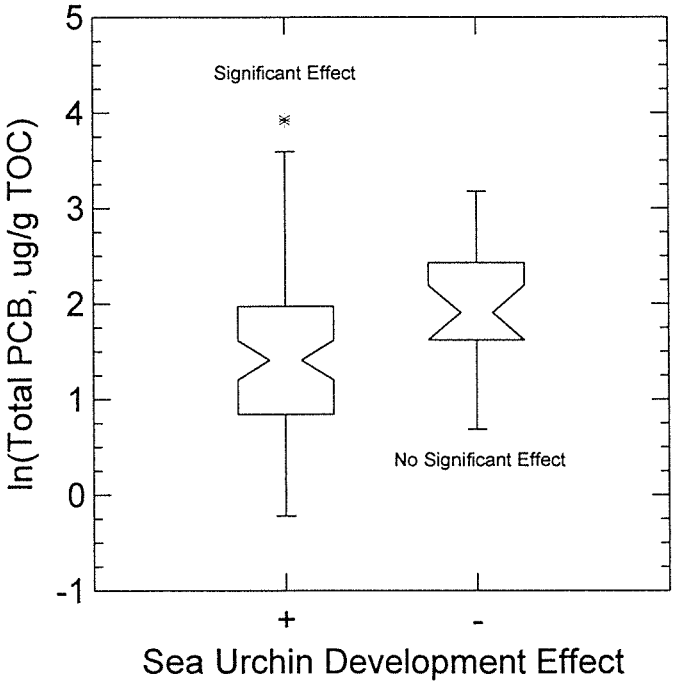


Figure 64

Fertilization Effects and Total PCB

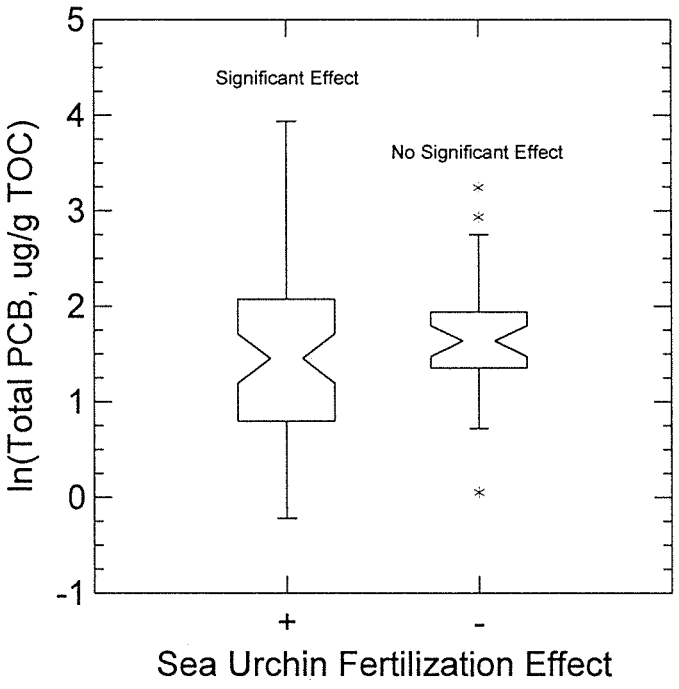


Figure 65a

Total DDT AET For Mortality

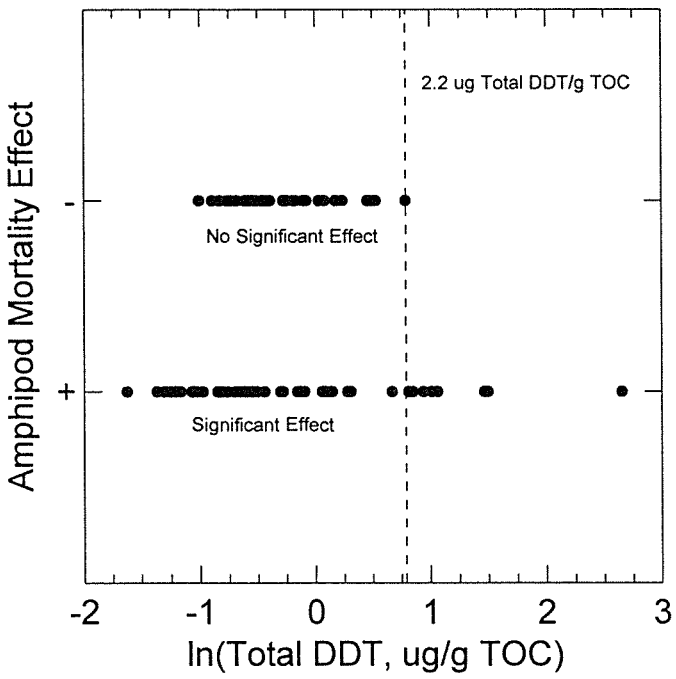


Figure 65b

Mortality vs Total DDT

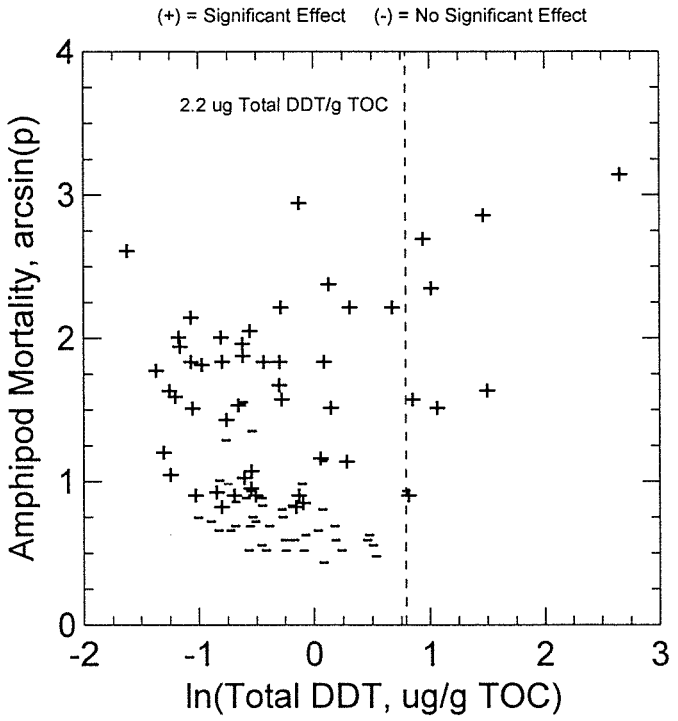


Figure 66a

Total DDT AET For Development

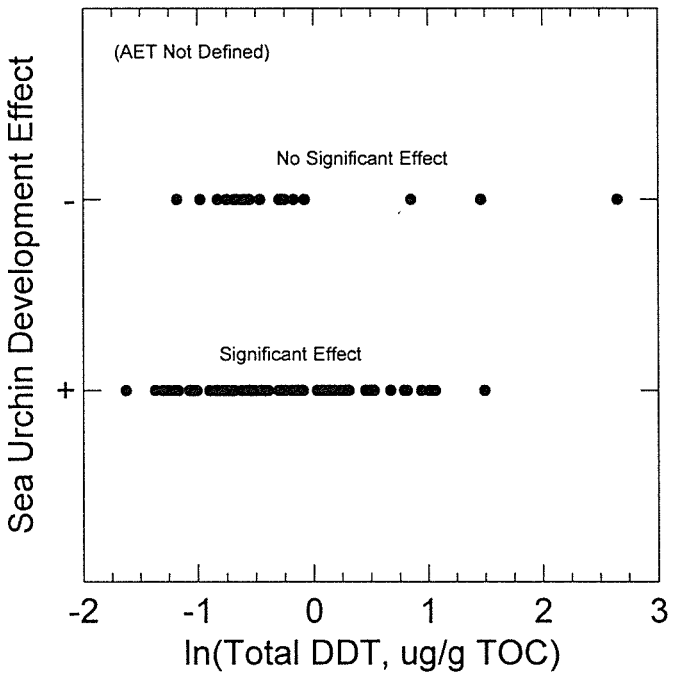


Figure 66b

Development vs Total DDT

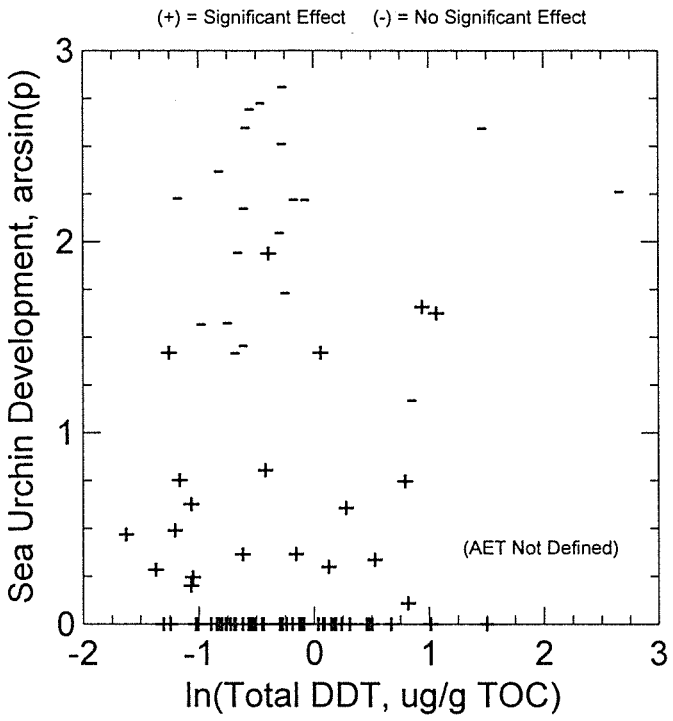


Figure 67a

Total DDT AET For Fertilization

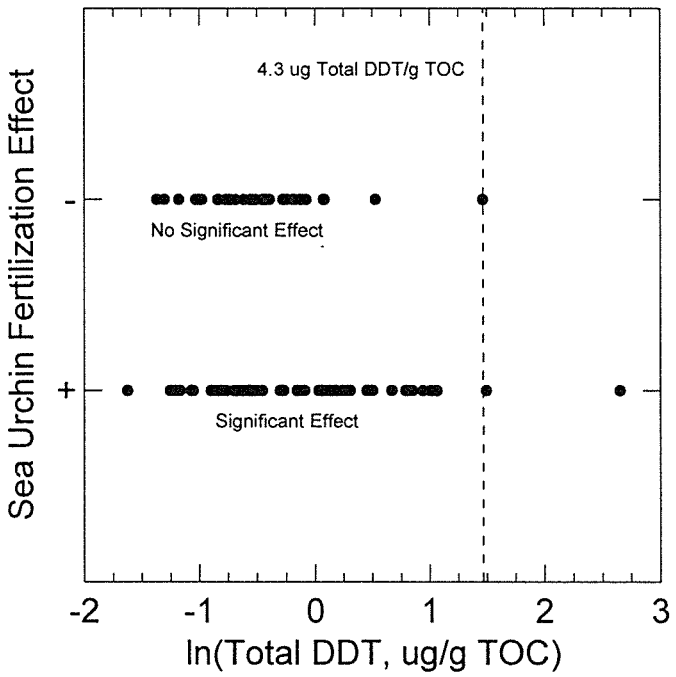


Figure 67b

Fertilization vs Total DDT

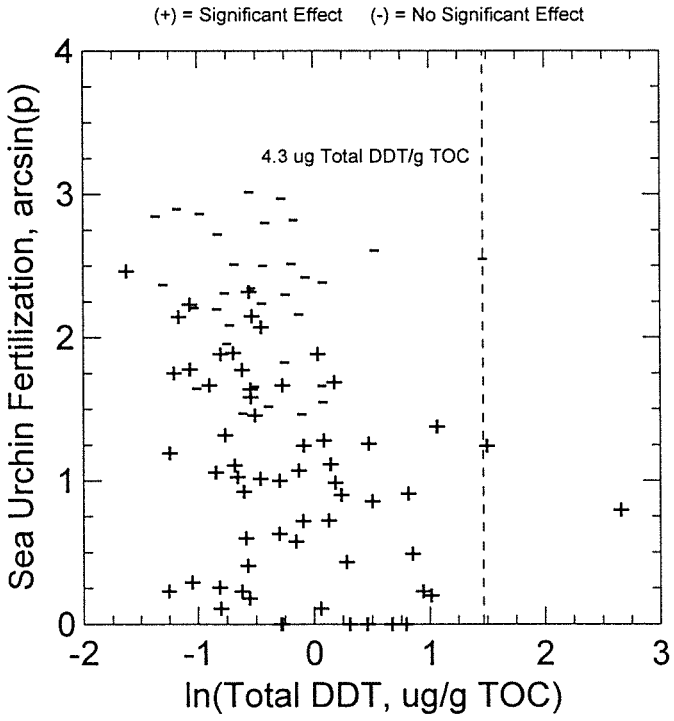


Figure 68a

Total PCB AET For Mortality

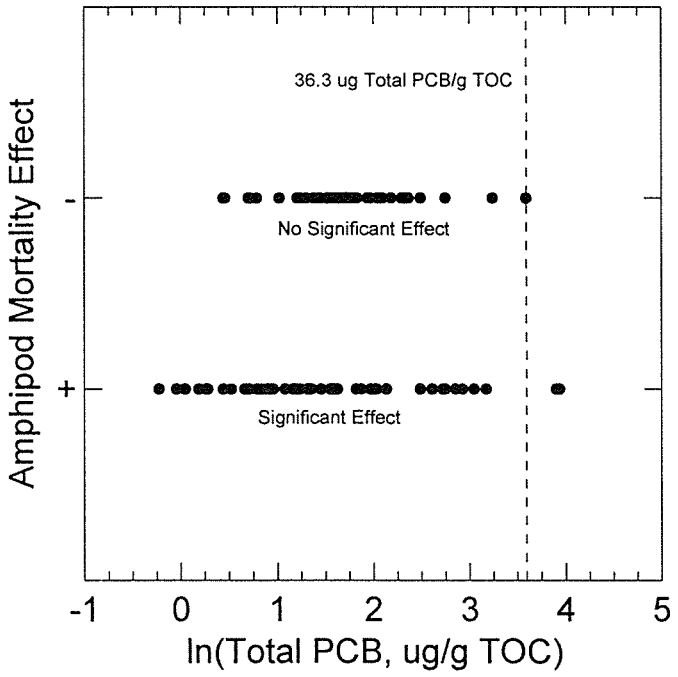


Figure 68b

Mortality vs Total PCB

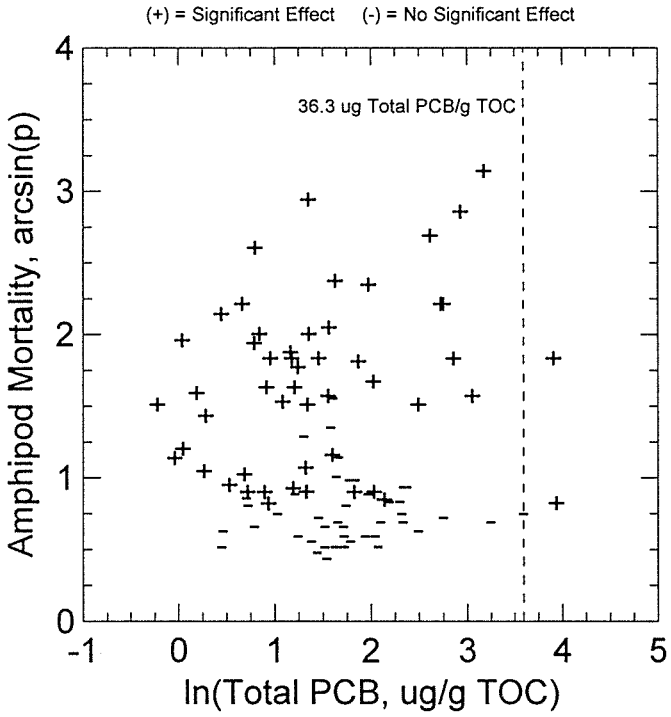


Figure 69a

Total PCB AET For Development

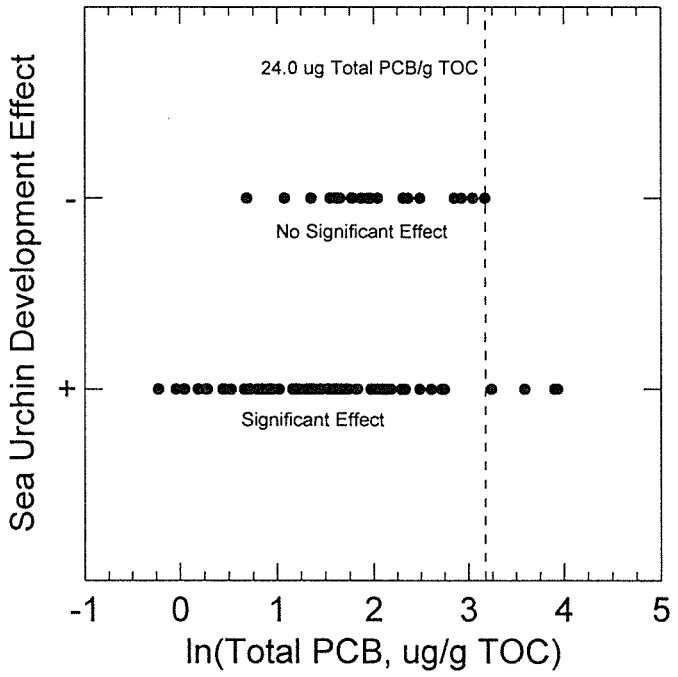


Figure 69b

Development vs Total PCB

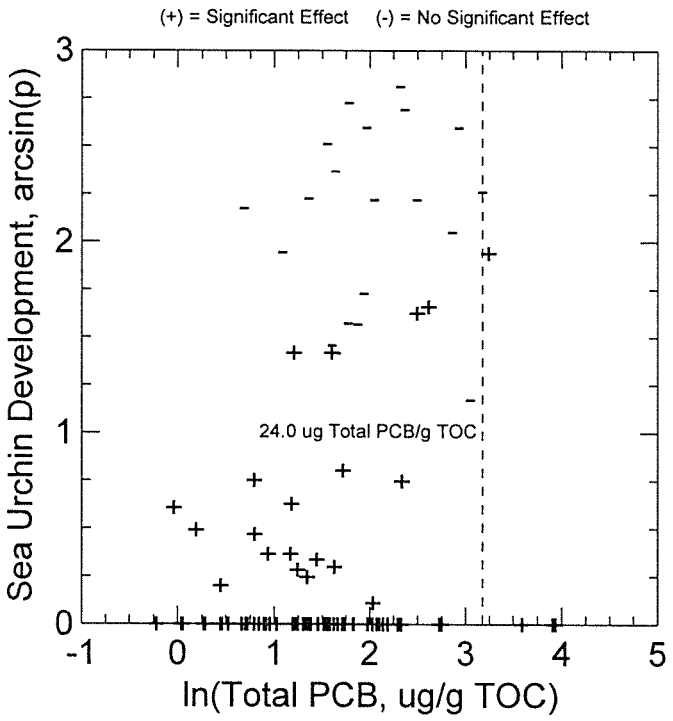


Figure 70a

Total PCB AET For Fertilization

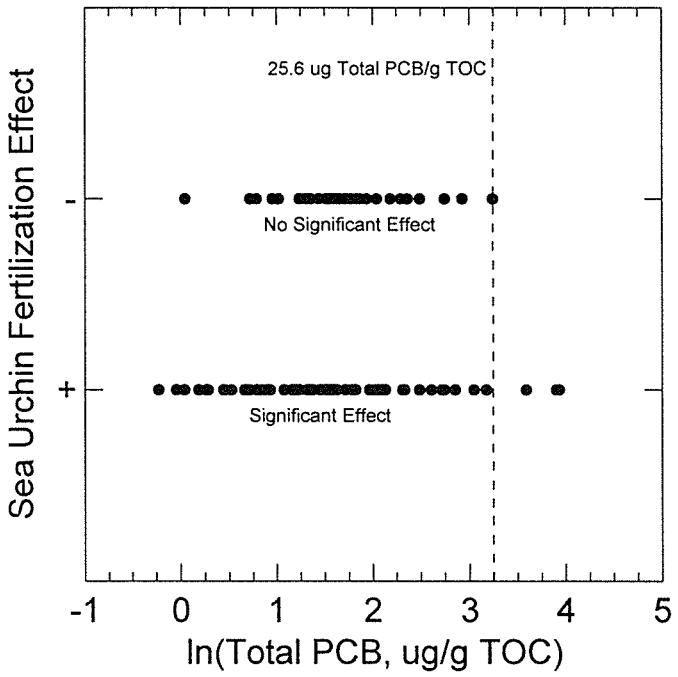


Figure 70b

Fertilization vs Total PCB

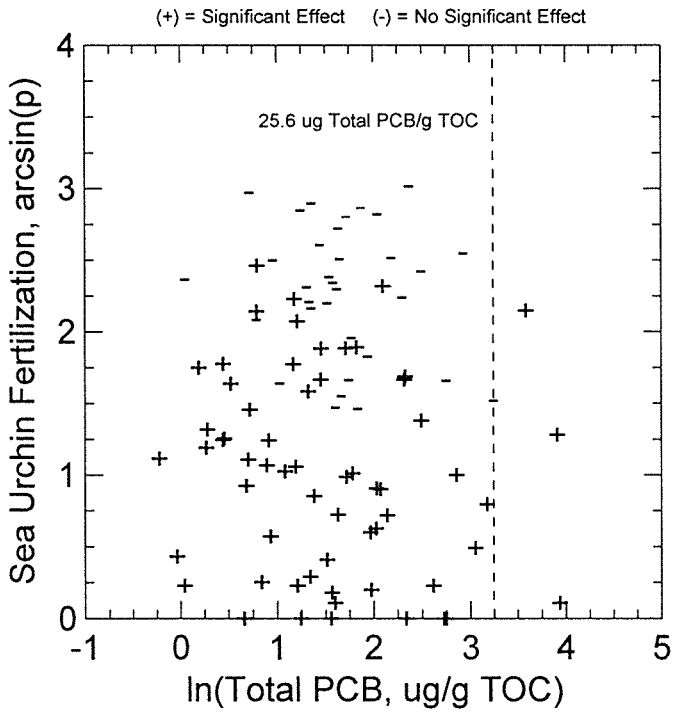


Figure 71

Total DDT ERM For Mortality

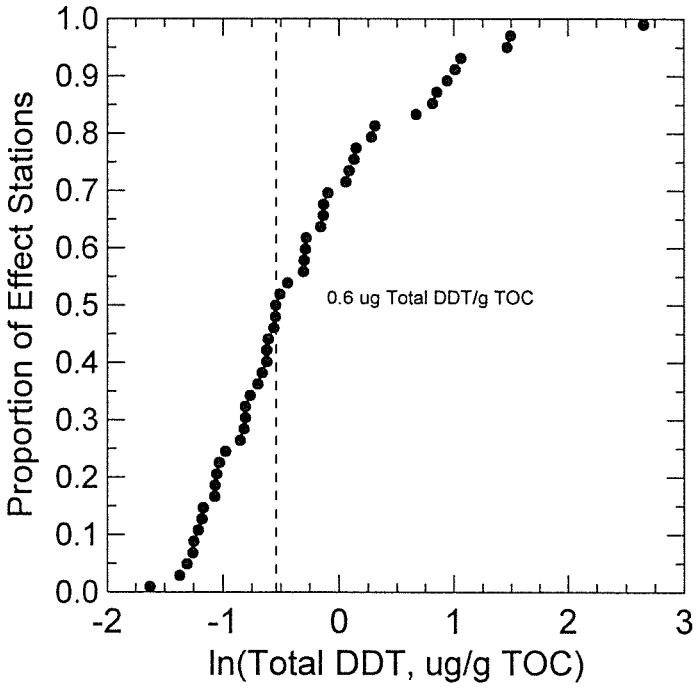


Figure 72

Total DDT ERM For Development

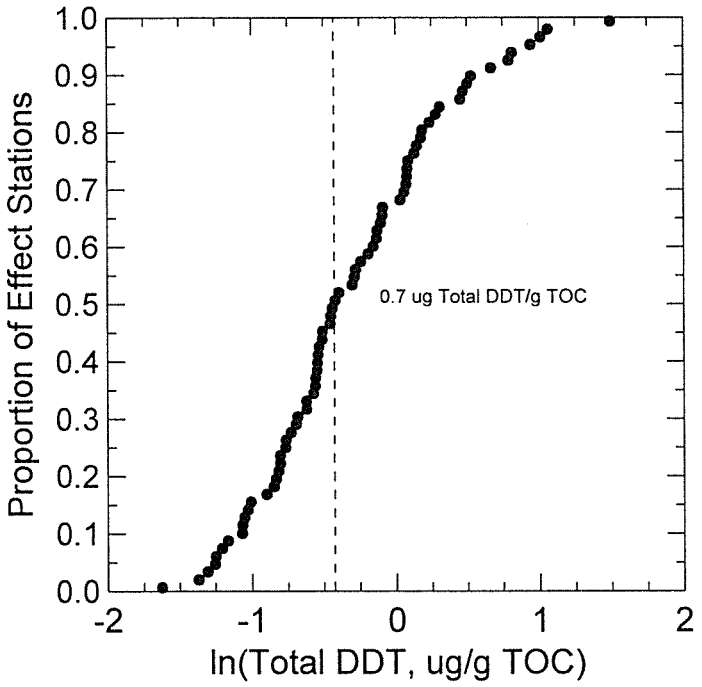


Figure 73

Total DDT ERM For Fertilization

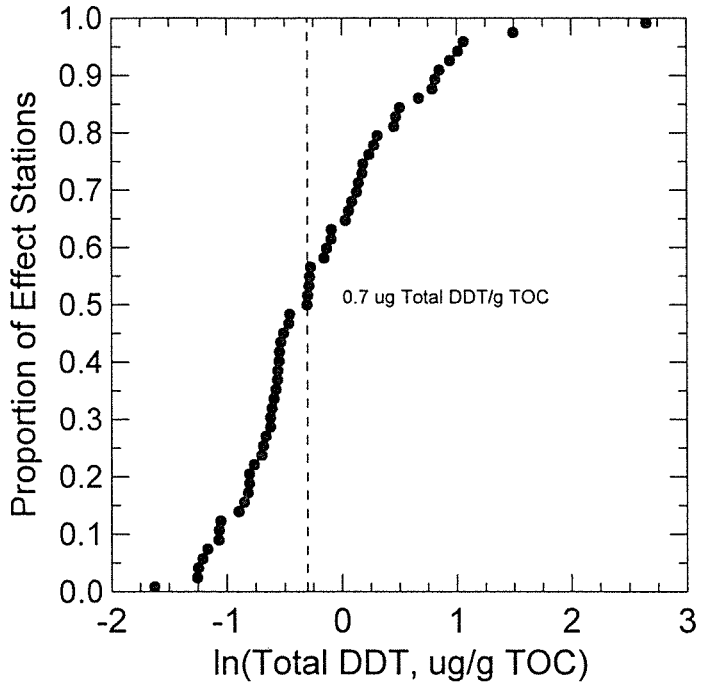


Figure 74

Total PCB ERM For Mortality

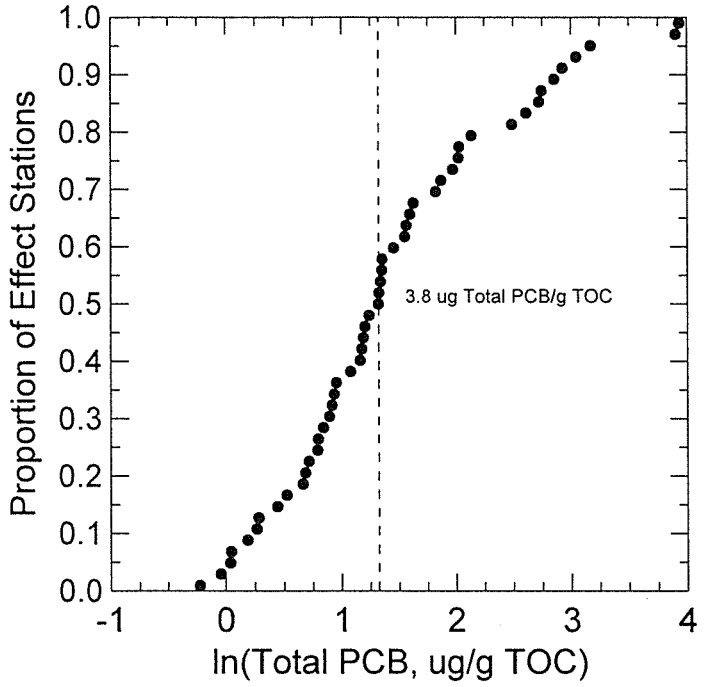


Figure 75

Total PCB ERM For Development

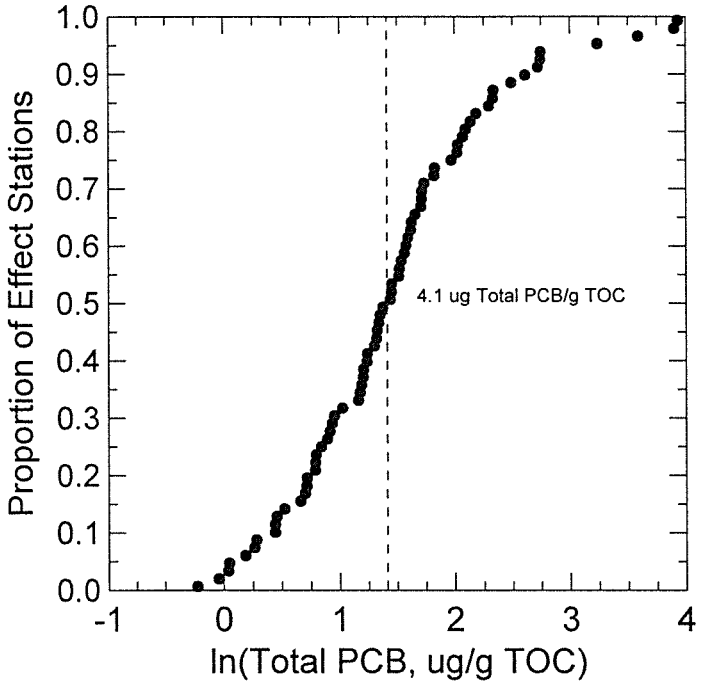
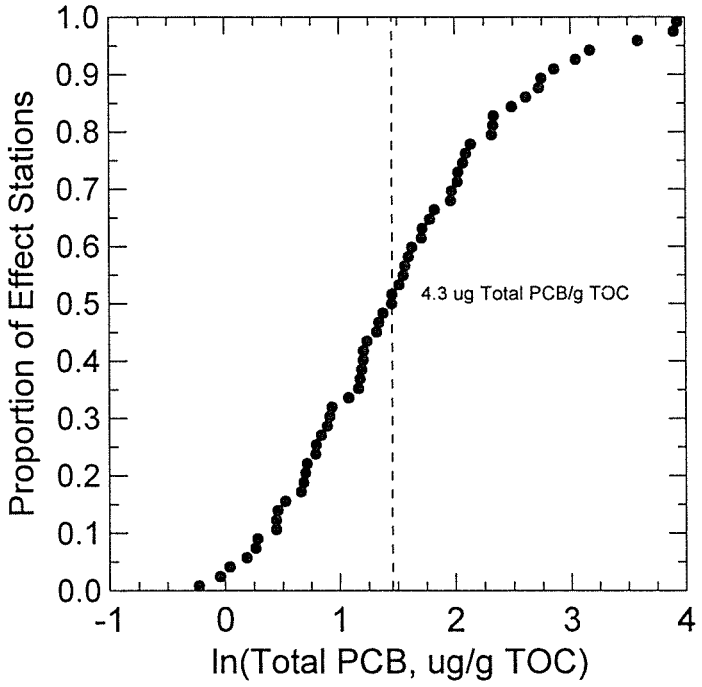


Figure 76

Total PCB ERM For Fertilization



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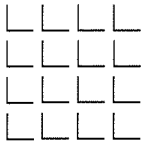

Comments Submitted to:

State Water Resources Control Board
1001 I Street, 24th Floor
Sacramento, California 95814

COMMENTS ON STATE WATER RESOURCES CONTROL BOARD'S PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE - JANUARY 2011

ATTACHMENTS, VOLUME 3
(TAB 36 – TAB 37)

Submitted by:

LATHAM & WATKINS LLP

Counsel for:

Montrose Chemical Corporation of California

Also on Behalf of:

American Council of Engineering Companies California
Building Industry Legal Defense Foundation
California Building Industry Association
California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Southern California Contractors Association

Submittal Date:

March 15, 2011

STATE WATER RESOURCES CONTROL BOARD'S PROPOSED AMENDMENTS TO THE WATER QUALITY CONTROL PLAN FOR ENCLOSED BAYS AND ESTUARIES – PART 1 SEDIMENT QUALITY FOR THE PROTECTION OF FISH AND WILDLIFE - JANUARY 2011

ATTACHMENTS TO MARCH 15, 2011 LETTER SUBMITTED BY

American Council of Engineering Companies California
Building Industry Legal Defense Foundation
California Building Industry Association
California Business Properties Association
California Chamber of Commerce
Construction Industry Coalition on Water Quality
Montrose Chemical Corporation of California
Southern California Contractors Association

Tab	Date	Description
References included in March 15, 2011 letter.		
1.	00/00/96	Chapman, P. M. (1996), A Test of sediment effects concentrations: DDT and PCB in the Southern California Bight. Environmental Toxicology and Chemistry, 15: 1197–1198
2.	02/17/11	Memorandum from Susan Kane-Driscoll, Ph.D., Exponent to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re Site-Specific Bioavailability Has Not Been Considered in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters
3.	09/30/09	Expert Report of Donald Roberts, Ph.D. from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
4.	09/00/09	Expert Report of Amir Attaran, Ph.D., LL.B from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
5.	10/00/09	Expert Report of Seymore Grufferman, M.D., Dr. P.H. from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
6.	n/a	Expert Report of Marion J. Fedoruk from Garza v. Allied Chemi. Corp., Case No. C-4885-99-F(10), 332d Judicial Dist., Hidalgo Co., Texas
7.	10/11/10	Digest and Transcript of Chris Beegan Deposition - In Re Tentative Cleanup and Abatement Order No. R9-2011-0001, Cal. Reg. Water Quality Control Bd., San Diego Region
8.	02/18/11	Memorandum from D. Frederick Bodishbaugh, Ph.D. and Charles Menzie, Ph.D., Exponent to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re Potential for Misuse of California Sediment Quality Objectives in the TMDL Process for Dominguez Channel and Greater Los Angeles and Long Beach Harbor Waters
9.	00/00/95	Long, E.R., MacDonald, D.D., Smith, S.L. and Calder, F.D. (1995), Incidence of Adverse Biological Effects Within Ranges of Chemical Concentrations in Marine and Estuarin Sediments. Environmental Management 19(1): 81-97
10.	00/00/98	Connor, T. P. O., Daskalakis, K. D., Hyland, J. L., Paul, J. F. and Summers, J. K. (1998), Comparisons of sediment toxicity with predictions based on chemical guidelines. Environmental Toxicology and Chemistry, 17: 468–471
11.	02/18/11	Memorandum from John Slocomb, Ph.D. and Paul Mehrle, Ph.D., Cardno Entrix to California Regional Water Quality Control Board, Los Angeles Region; U.S. Environmental Protection Agency, Region 9 re The Effects Range Low (ERL) Value For Numeric Target of Water Body-Pollutant Combinations in Marine Sediments of the Dominguez Channel Estuary and Greater Los Angeles and Long Beach Waters
12.	02/22/11	Letter from David Sunding, The Brattle Group to California Regional Water Quality Control Board, Los Angeles Region; United States Environmental Protection Agency, Region 9 Comments on the cost consideration of, "Dominguez Channel and Greater Los Angeles and Long Beach Harbor Water Toxic Pollutants Total Maximum Daily Loads Draft"

Tab	Date	Description
References included in attached expert reports.		
13.	00/00/00	Morrison, D.E., Robertson, B.K., and Alexander, M. (2000), Bioavailability to Earthworms of Aged DDT, DDE, DDD, and Dieldrin in Soil. Environ. Sci. Technol., 34: 709-713
14.	00/00/95	Alexander, M. (1995), How Toxic are Toxic Chemicals in Soil? Environ. Sci. Technol., 29: 2713–2717
15.	11/00/03	U.S. EPA (2003), Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: PAH mixtures. EPA-600-R-02-013. Office of Research and Development, Washington, D.C.
16.	03/00/08	U.S. EPA (2008), Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Compendium of Tier 2 Values for Nonionic Organics. EPA-600-R-02-016. Office of Research and Development, Washington, D.C.
17.	01/00/05	U.S. EPA (2005), Procedures for the Derivation of Equilibrium Partitioning Sediment Benchmarks (ESBs) for the Protection of Benthic Organisms: Metal Mixtures (Cadmium, Copper, Lead, Nickel, Silver, and Zinc). EPA-600-R-02-011. Office of Research and Development Environmental Protection, Washington, D.C.
18.	00/00/07	Hawthorne, S.B., Azzolina, N.A., Neuhauser, E.F. and Kreitinger, J.P. (2007), Predicting Bioavailablity of Sediment Polycyclic Aromatic Hydrocarbons to Hyalella Azteca Using Equilibrium Partitioning, Supercritical Fluid Extraction, and Pore Water Concentrations. Environ. Sci. Technol., 41: 6297-6304
19.	00/00/09	Kane Driscoll, S.B., Amos, B.C., McArdle, M.E., Menzie, C.A. and Coleman, A. (2009), Predicting Sediment Toxicity at Former Manufactured Gas Plants Using Equilibrium Partitioning Benchmarks for PAH Mixtures. Soil & Sediment Contamination 18(3): 307–319
20.	07/12/10	McArdle, M.E., Kane Driscoll, S.B. and Booth, P.N. (2010), An Ecological Risk-Based Cleanup Strategy for Contaminated Sediments in a Freshwater Brook. International Journal of Soil, Sediment and Water 3(2): 1–24.
21.	00/00/07	ASTM (American Society for Testing and Materials) (2007), Standard Test Method for Determination of Parent and Alkyl Polycyclic Aromatics in Sediment Pore Water Using Solid-Phase Microextraction and Gas Chromatography/Mass Spectrometry in Selected Ion Monitoring Mode. D 7363
22.	00/00/99	Middelburg, J.J., Nieuwenhuize, J. and Van Breugel, P. (1999), Black Carbon in Marine Sediments. Marine Chemical 65: 245-252
23.	00/00/07	Tomaszewski, J.E., Werner, D. and Luthy, R.G. (2007), Activated Carbon Amendment as a Treatment for Residual DDT in Sediment from a Superfund Site in San Francisco Bay, Richmond, California, USA. Environmental Toxicology & Chemistry 10: 2143-2150
24.	00/00/04	Rust, A.J., Burgess, R.M., McElroy, A.E., Cantwell, M.G. and Brownawell, B.J. (2004), Influence of Soot Carbon on the Bioaccumulation of Sediment-Bound Polycyclic Aromatic Hydrocarbons by Marine Benthic Invertebrates: An Interspecies Comparison. Environmental Toxicology & Chemistry 23: 2594–2603
25.	00/00/01	Bucheli, T.D. and Gustafsson, O. (2001), Ubiquitous Observations of Enhanced Solid Affinities for Aromatic Organochlorines in Field Situations: Are in Situ Dissolved Exposures Overestimated by Existing Partitioning Models? Environmental Toxicology & Chemistry 20: 1450-1456
26.	00/00/97	Maruya, K.A., Risebrough, R.W. and Horne, A.J. (1997), The Bioaccumulation of Polynuclear Aromatic Hydrocarbons by Benthic Invertebrates in an Intertidal Marsh. Environmental Toxicology & Chemistry 16: 1087-1097
27.	00/00/01	U.S. EPA, Office of Solid Waste and Emergency Response (2001), Eco Update. The Role of Screening-Level Risk Assessments and Refining Contaminants of Concern in Baseline Ecological Risk Assessments. Publication 9345.-014. EPA 540/F-01/014
28.	00/00/10	Fuchsman P., Perruchon E., Bizzotto E., Dillard J. and Henning, M. (2010), An Evaluation of Cause-Effect Relationships Between DDT (and Metabolites) and Sediment Toxicity to Benthic Invertebrates. Presentation at the Society of Environmental Toxicology and Chemistry North America 31st Annual Meeting, Portland, OR, November 7-11, 2010

Tab	Date	Description
29.	00/00/07	San Francisco Estuary Institute (2007), Indicator Development and Framework for Assessing Indirect Effects of Sediment Contaminants. Draft Report. SFEI Publication # 524
30.	06/12/99	NOAA (1999), Sediment Quality Guidelines developed for the National Status and Trends Program
31.	00/00/98	Long, E.R. and MacDonald, D.D. (1998), Recommended Uses of Empirically Derived Sediment Quality Guidelines for Marine and Estuarine Ecosystems. Human and Ecological Risk Assessment 4(5): 1019-1039
32.	00/00/98	Long, E.R, Field, L.J. and D.D. MacDonald (1998), Predicting Toxicity in Marine Sediments with Numerical Sediment Quality Guidelines. Environmental Toxicology and Chemistry 17(4): 714-727
33.	00/00/96	MacDonald, D.D., Carr, R.S., Calder, F.D., Long, E.R. and Ingersoll, C.R. (1996), Development and Evaluation of Sediment Quality Guidelines for Florida Coastal Waters. Ecotoxicology 5: 253-278
34.	01/03/08	Di Toro, D.M. (2008), Review of Sediment Quality Objectives for Enclosed Bays and Estuaries of California
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36.	09/00/96	Fairey, R., J. Hunt, C. Wilson, M. Stephenson, M. Pluckett and E. Long (1996), Chemistry, Toxicity and Benthic Community Conditions in Sediments of the San Diego Bay Region. Final Report, California State Water Resources Control Board
37.	04/00/97	MacDonald, D. (1997), Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments. Prepared for National Oceanic and Atmospheric Administration

CHEMISTRY, TOXICITY AND BENTHIC COMMUNITY CONDITIONS IN SEDIMENTS OF THE SAN DIEGO BAY REGION

FINAL REPORT

September, 1996

California State Water Resources Control Board

National Oceanic and Atmospheric Administration

California Department of Fish and Game
Marine Pollution Studies Laboratory

Moss Landing Marine Laboratories

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EXECUTIVE SUMMARY

The following report describes and evaluates chemical and biological data collected from San Diego Bay and its historical tributaries between October, 1992 and May, 1994. The study was conducted as part of the ongoing Bay Protection and Toxic Cleanup Program, a legislatively mandated program designed to assess the degree of chemical pollution and associated biological effects in California's bays and harbors. The workplan for this study resulted from a cooperative agreement between the State Water Resources Control Board and the National Oceanic and Atmospheric Administration (NOAA). Monitoring and reporting aspects of the study were conducted by the Environmental Services Division, of the California Department of Fish and Game, and its subcontractors.

The study objectives were:

1. Determine presence or absence of adverse biological effects in representative areas of the San Diego Bay Region;

2. Determine relative degree or severity of adverse effects, and distinguish more severely impacted sediments from less severely impacted sediments;

3. Determine relative spatial extent of toxicant-associated effects in the San Diego Bay Region;

4. Determine relationships between toxicants and measures of effects in the San Diego Bay Region.

The research involved chemical analysis of sediments, benthic community analysis and toxicity testing of sediments and sediment pore water. Chemical analyses and bioassays were performed using aliquots of homogenized sediment samples collected synoptically at each station. Analysis of the benthic community structure was made on a subset of the total number of stations sampled.

Three hundred and fifty stations were sampled between October, 1992 and May, 1994. Areas sampled included San Diego Bay, Mission Bay, the San Diego River Estuary and the Tijuana River Estuary and are collectively termed "the San Diego Bay Region" in the following document. Two types of sampling designs were utilized: direct point sampling and stratified random sampling.

Chemical pollution was demonstrated by using comparisons to established sediment quality guidelines. Two sets of guidelines were used: the Effects Range-Low (ERL)/Effects Range-Median (ERM) guidelines developed by NOAA (Long and Morgan, 1990; Long *et al.*, 1995) and the Threshold Effects Level (TEL)/Probable Effects Level (PEL) guidelines used in Florida (McDonald, 1993; McDonald, 1994). Copper, mercury, zinc, total chlordane, total PCBs and the PAHs were most often found to exceed critical ERM or PEL values

and were considered the major chemicals or chemical groups of concern in the San Diego Bay Region. ERM and PEL summary quotients were used to develop chemical indices for addressing the pollution of sediments with multiple chemicals. An ERM summary quotient >0.85 or a PEL summary quotient >1.29 was indicative of stations where multiple chemicals were significantly elevated. Stations with any chemical concentration >4 times its respective ERM or >5.9 times its respective PEL were considered to exhibit elevated chemistry. Summary quotients and magnitude of sediment quality guideline exceedances were used as additional information to help prioritize stations of concern for Regional Water Quality Control Board staff.

Identification of degraded and undegraded habitat (as determined by macrobenthic community structure) was conducted using a cumulative, weight-of-evidence approach. Analyses were performed to identify relationships between community structure within and between each station or site (*e.g.,* diversity/evenness indices, analyses of habitat and species composition, construction of dissimilarity matrices for pattern testing, assessment of indicator species, and development of a benthic index, cluster analyses, and ordination analyses).

Analyses of the 75 stations sampled for benthic community structure identified 23 undegraded stations, 43 degraded and 9 transitional stations. All sampled stations with an ERM summary quotient >0.85 were found to have degraded communities. All sampled stations with P450 Reporter Gene System responses above 60 μg/g BaPEq. were similarly found to have degraded benthic communities.

The statistical significance of toxicity test results was determined using two approaches: the reference envelope approach and laboratory control comparison approach used by the United States Environmental Protection Agency- Environmental Monitoring and Assessment Program and NOAA- National Status and Trends programs. The reference envelope approach indicated that toxicity for the *Rhepoxynius* (amphipod) sediment test was significant when survival was less than 48% in samples tested. No reference envelope was calculated for the urchin fertilization or development tests due to high variability in pore water data from reference stations.

The laboratory control comparison approach was used to compare test sediment samples against laboratory controls for determination of statistically significant differences in test organism response. Criteria for toxicity in this approach were 1) survival less than 80% of the control value and 2) significant difference between test samples and controls, as determined using a t-test. Using this approach, there was no absolute value below which all samples could be considered toxic, although survival below a range of 72-80% was generally considered toxic.

Using the EMAP definition of toxicity, 56% of the total area sampled was toxic to *Rhepoxynius*. For the *Strongylocentrotus* larval development test, percent of total area toxic was 29%, 54%, and 72% respectively for 25%, 50%, and undiluted pore water concentrations. Samples representing 14%, 27%, or 36% of the study area were toxic to both *Strongylocentrotus* in pore water (25%, 50%, or undiluted, respectively) and *Rhepoxynius* in solid phase sediment.

Linear regression analyses failed to reveal strong correlations between amphipod survival and chemical concentration. It is suspected instead of a linear response to chemical pollutants, most organisms are tolerant of pollutants until a threshold is exceeded. Comparisons to established sediment quality guideline thresholds demonstrate an increased incidence of toxicity for San Diego Bay Region samples with chemical concentrations exceeding the ERM or PEL values. It is further suspected toxicity in urban bays is caused by exposure to complex mixtures of chemicals. Comparisons to ERM summary quotients (multiple chemical indicators) demonstrate that the highest incidence of toxicity (>78%) is found in samples with elevated ERM summary quotients (>0.85).

Statistical analyses of the P450 Reporter Gene System responses versus the PAHs in sediment extracts demonstrated that this biological response indicator was significantly correlated ($r^2 = 0.86$) with sediment PAH (total and high molecular weight) concentration.

Stations requiring further investigation were prioritized based on existing evidence. Each station receiving a high, moderate or low priority ranking meets one or more of the criteria under evaluation for determining hot spot status in the Bay Protection and Toxic Cleanup Program. Those meeting all criteria were given the highest priority for further action. A ranking scheme was developed to evaluate stations of lower priority.

Seven stations (representing four sites) were given a high priority ranking, 43 stations were given a moderate priority ranking, and 57 stations were given a low priority ranking. The seven stations receiving the high priority ranking were in the Seventh Street channel area, two naval shipyard areas near the Coronado Bridge, and the Downtown Anchorage area west of the airport. The majority of stations given moderate rankings were associated with commercial areas and naval shipyard areas in the vicinity of the Coronado Bridge. Low priority stations were interspersed throughout the San Diego Bay Region.

A review of historical data supports the conclusions of the current research. Recommendations are made for complementary investigations which could provide additional evidence for further characterizing stations of concern.

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF APPENDICES

LIST OF ABBREVIATIONS

AA	Atomic Absorption
ASTM	American Society for Testing Materials
AVS	Acid Volatile Sulfide
BPTCP	Bay Protection and Toxic Cleanup Program
CDF	Cumulative Distribution Frequencies
CDFG	California Department of Fish and Game
CH	Chlorinated Hydrocarbon
COC	Chain of Custody
COR	Chain of Records
EDTA	Ethylenediaminetetraacetic Acid
EMAP	Environmental Monitoring and Assessment Program
ERL	Effects Range Low
ERM	Effects Range Median
ERMQ	Effects Range Median Summary Quotient
EqP	Equilibrium Partitioning Coefficient
FAAS	Flame Atomic Absorption Spectroscopy
GC/ECD	Gas Chromatograph Electron Capture Detection
GFAAS	Graphite Furance Atomic Absorption Spectroscopy
HCl	Hydrochloric Acid
HDPE	High-density Polyethylene
HMW PAH	High Molecular Weight Polynuclear Aromatic Hydrocarbons
HNO_3	Nitric Acid
HPLC/SEC	High Performance Liquid Chromatography Size Exclusion
H_2S	Hydrogen Sulfide
IDORG	Identification and Organizational Number
KCL	Potassium Chloride
LC_{50}	Lethal Concentration (to 50 percent of test organisms)
LMW PAH	Low Molecular Weight Polynuclear Aromatic Hydrocarbons
MDL	Method Detection Limit
MDS	Multi-Dimensional Scaling
MLML	Moss Landing Marine Laboratories
MPSL	Marine Pollution Studies Laboratory
NH_3	Ammonia
NOAA	National Oceanic and Atmospheric Administration
NOEC	No Observed Effect Concentration
NS&T	National Status and Trends Program
P450	Cytochrome P450 Enzyme System
PAH	Polynuclear Aromatic Hydrocarbons
PCB	Polychlorinated Biphenyl
PEL	Probable Effects Level
PELQ	Probable Effects Level Summary Quotient
PPE	Porous Polyethylene
PVC	Polyvinyl Chloride
QA	Quality Assurance
QAPP	Quality Assurance Project Plan
QC	Quality Control
REF	Reference
RGS	P450 Reporter Gene System
RWQCB	Regional Water Quality Control Board
SCCWRP	Southern Calif. Coastal Waters Research Project

SPARC	Scientific Planning and Review Committee
SQC	Sediment Quality Criteria
SWRCB	State Water Resources Control Board
T	Temperature
TBT	Tributyltin
TFE	Tefzel Teflon®
TEL	Threshold Effects Level
TIE	Toxicity Identification Evaluation
TOC	Total Organic Carbon
TOF	Trace Organics Facility
UCSC	University of California Santa Cruz
USEPA	U.S. Environmental Protection Agency
WCS	Whole Core Squeezing

Units

liter = 1 l
milliliter = 1 ml
microliter = 1 μl
gram = 1 g
milligram = 1 mg
microgram = 1 μg
nanogram = 1 ng
kilogram = 1 kg
1 part per thousand (ppt) = 1 mg/g
1 part per million (ppm) = 1 mg/kg, 1 μg/g
1 part per billion (ppb) = 1 μg/kg, 1 ng/g

Purpose

In 1992, the State Water Resources Control Board (SWRCB) and the National Oceanic and Atmospheric Administration (NOAA) entered into a three-year cooperative agreement to assess potential adverse biological effects from sediments in coastal bays and harbors of Southern California (SWRCB and NOAA, 1991, 1992, 1993). The study area for the three-year cooperative agreement extended south of the Palos Verdes Peninsula to the USA/Mexico border. The majority of work focused on selected coastal bays, harbors and lagoons where depth ranged from approximately 60 meters to the upper limit of the tidal range. In the first phase of the study, data were collected, analyzed, and reported from the Los Angeles/Long Beach areas (SWRCB and NOAA, 1994).

This report presents results from data collected in the San Diego Bay area during the second and third years of the cooperative agreement. The study was performed in San Diego Bay, Mission Bay, San Diego River Estuary, and Tijuana River Estuary in southern California (Figure 1).

The purposes of the present study were:

1. Determine presence or absence of statistically significant toxicity effects in representative areas of the San Diego Bay Region;

2. Determine relative degree or severity of observed effects, and distinguish more severely impacted sediments from less severely impacted sediments;

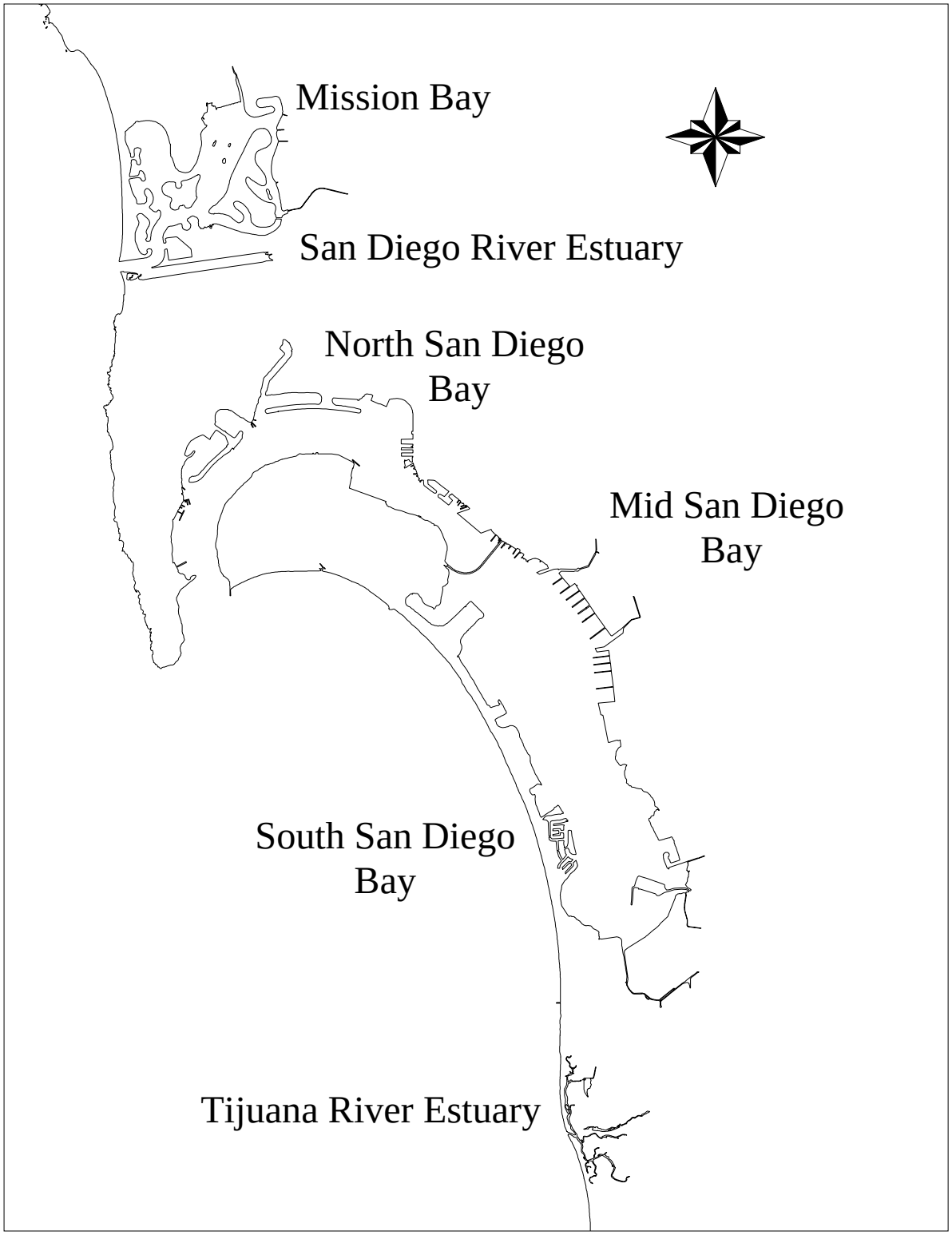
3. Determine relative areal extent of significant toxicity in the San Diego Bay Region;

4. Determine relationships between pollutants and measures of effects in these bays.

Programmatic Background and Needs

Due to the long history of human activity in San Diego Bay and its surrounding waters, there is a need to assess any environmentally detrimental effects which have been associated with those activities. The cooperative agreement between NOAA and SWRCB was designed to investigate these environmental effects by evaluating the biological and chemical state of San Diego Bay sediments. The methods used to assess environmental impacts include sediment and interstitial water bioassays, sediment chemistry analysis, and benthic community analysis. The study areas included San Diego Bay, Mission Bay, Tijuana River Estuary, and the San Diego River. Although these water bodies are separated physically, and are quite different in character, for simplicity they will often be referred to collectively as the "San Diego Bay Region" in this report (Figure 1). The SWRCB and NOAA have common programmatic needs for this research, however, some differences exist. NOAA is mandated by Congress to conduct a

Figure 1
San Diego Bay Region Study Area



program of research and monitoring on marine pollution. Much of this research is conducted through the National Status and Trends (NS&T) Program and the Coastal Ocean Program. The NS&T Program performs intensive regional studies on the magnitude and extent of toxicant-associated bioeffects in selected coastal embayments and estuaries. Areas chosen for these regional studies were those in which pollutant concentrations indicate the greatest potential for biological effect. These biological studies augment regular chemical monitoring activities of the NS&T Program, and provide a means for estimating the extent of toxicity associated with measured concentrations of sediment pollutants.

The California Water Code, Division 7, Chapter 5.6, Section 13390 mandates the State Water Resources Control Board and the Regional Water Quality Control Boards to provide the maximum protection of existing and future beneficial uses of bays and estuarine waters and to plan for remedial actions at those identified toxic hot spots where the beneficial uses are being threatened by toxic pollutants.

A cooperative agreement between NOAA and SWRCB has been implemented through the Bay Protection and Toxic Cleanup Program (BPTCP). Sediment characterization approaches currently used by the BPTCP range from chemical or toxicity monitoring only, to monitoring designs which attempt to generally correlate the presence of pollutants with toxicity or benthic community degradation. Studies were designed, managed, and coordinated by the SWRCB's Bays and Estuaries Unit as a cooperative effort with NOAA's Bioeffects Assessment Branch, and the California Department of Fish and Game's (CDFG) Marine Pollution Studies Laboratory. Funding was provided by the SWRCB and NOAA's Coastal Ocean Program.

Research for the San Diego Bay Region involved toxicity testing and chemical analysis of sediments and sediment pore water. Toxicity tests and chemical analysis were performed using aliquots of homogenized sediment samples collected synoptically from each station, resulting in paired data. Analyses of benthic community structure and P450 enzyme induction were also made on a subset of the total number of stations sampled.

Field and laboratory work was accomplished under interagency agreement with, and under the direction of, the CDFG. Sample collections were performed by staff of the San Jose State University Foundation at the Moss Landing Marine Laboratories, Moss Landing, CA (MLML). Trace metals analyses were performed by CDFG personnel at the trace metal facility at Moss Landing Marine Laboratories. Synthetic organic pesticides, polycyclic aromatic hydrocarbons (PAHs), and polychlorinated biphenyls (PCBs) were analyzed at the UCSC trace organics analytical facility at Long Marine Laboratory in Santa Cruz, California. MLML staff also performed total organic carbon (TOC) and grain size analyses, as well as benthic community analyses. Toxicity testing was conducted by the University of California at Santa Cruz (UCSC) staff at the CDFG toxicity testing laboratory at Granite Canyon,

California. P450 Reporter Gene System analyses were conducted by Columbia Analytical Services in Carlsbad, CA.

Study Area

San Diego Bay

San Diego Bay is the southern-most embayment on the west coast of the United States. It is located within the Southern California Bight and is the largest embayment along the 1450 kilometer stretch of coastline between San Francisco and Central Baja California. Located 16 kilometers northwest of the Mexico border, it is considered one of the finest natural harbors in the world. This reputation is due mainly to its deep entrance and protection from weather it provides ships. San Diego Bay lies entirely in the county of San Diego, extending from the entrance at Point Loma southward to the mouth of the Otay River.

San Diego Bay is a natural, nearly-enclosed, crescent-shaped estuary that encompasses approximately 52 square kilometers. It is approximately 24 kilometers (km) in length and varies from 0.4 km to 5.8 km in width. Depths in the Bay vary from 18 meters near the mouth to less than 1 meter in the southern part of the bay, with the average depth for the entire bay being slightly more than 12 meters. The Bay is much deeper and narrower than it was historically, due mainly to dredging of channels and filling of nearshore areas.

San Diego Bay opens to the Pacific Ocean and is classified as an estuarine system due to its fresh water dilution. The diversion of the San Diego River to Mission Bay by the U.S. Army Corps of Engineers in 1857 was the first major reduction of freshwater input into the bay (Smith, 1977). Sweetwater River and the Otay River were also main sources of freshwater for San Diego Bay, although these sources have been greatly reduced over the years as a result of dam construction, extensive ground water use, and limited rainfall in recent years. Freshwater input is now limited to periodic surface drainage from the metropolitan area and intermittent flow from several rivers and creeks during periods of rainfall. Because of the dry Mediterranean-like climate that characterizes San Diego Bay, average annual rainfall in the Bay is usually between 10 and 13 inches, the majority of which falls between November and February.

Tides in San Diego Bay demonstrate marked variation between the heights of two high tides and two low tides that occur daily, classifying them as diurnal. The range between mean higher high water (MHHW) and mean lower low water (MLLW) is 1.6 meters and the extreme range of tides within the Bay is approximately 2.9 meters (Browning and Speth, 1973). Tidal currents are strongest in the northern part of the Bay where surface velocities reach 2.9 knots on ebb tide and 2.2 knots on flood tide (U.S. Army Corps of Engineers, 1973). Tidal currents are reduced considerably in the shallower central and south bay areas. Average tidal flushing for San Diego Bay is about 30% of the

entire Bay water volume exchanged per tidal cycle (12.5 hours). This volume of water is referred to as the tidal prism and in San Diego Bay represents approximately 74,000,000 cubic meters. Tidal flushing rates differ drastically between the Bay entrance and South Bay. Complete tidal flushing for the South Bay requires seven to fourteen days, whereas, the entrance of the Bay may only require one to two days. It has been estimated over the last century, tidal flushing in San Diego Bay has been reduced by 30% due to channel dredging and landfill projects (Browning and Speth, 1973).

San Diego Bay is a sedimentary environment with the bay floor and bay margins characterized by sand, silt and clay deposits (Peeling, 1974). Sand deposits are found near the Bay's mouth and along western margins, while finer silt and clay deposits are located on the eastern margins and at the southern end of the Bay.

An early navigation chart issued by the U.S. Coastal Survey in 1859 shows an undredged Bay fifteen miles long with a channel varying in depth from 22.2 meters decreasing to 3.6 meters. This natural channel stretched for 13 kilometers from the tip of Point Loma to the South Bay. Salt marshes existed at the mouths of seven creeks and river tributaries.

The early residents of the San Diego Bay area were Native Americans, who hunted and fished in the Bay; Spanish, Mexican, and American ranchers, who traded hides and tallow; and the early Yankee whalers who established camps in North Bay. These groups appeared to have little impact on the water quality in the Bay. By 1830 there were 16 American whaling vessels operating out of San Diego Bay. The whaling industry reached its peak in 1871-72 when 55,000 gallons of oil and 200 tons of whalebone were shipped from Point Loma. Americans participating in the New Town land boom of the 1880's settled in the central San Diego Bay area, site of the present downtown San Diego. This settlement soon represented a considerable increase in the population of the area as well as a dramatic threat to water quality in the Bay.

The Cuyamaca Dam and a flume were completed in 1888, diverting freshwater from eastern mountains into what is now Chollas Reservoir. Forty miles of sewers coupled with a sewage reservoir and outfall located in San Diego Bay off Market street were also completed in 1888. This sewage system marked the beginning of the decline in water quality for the Bay. Conditions within the Bay continued to decline because of the increase in population (30,000 in 1901) and acceptance of the Bay as a major harbor for the U.S. Navy and civilian commerce.

During the next four decades communications and aviation stations were added and docking facilities expanded. Naval facilities expanded greatly during World War II as business and industry boomed. In 1940, the population had increased to 200,000 causing a failure of the overloaded sewage collection and treatment facilities. In 1943, raw or minimally treated sewage was being

discharged into the Bay from 15 outfalls. After World War II and the Korean War, San Diego Bay was subject to the dumping of more than 50 million gallons of sewage and industrial waste per day (San Diego Interagency Water Quality Panel, 1989).

In 1950, the population of the San Diego metropolitan area had increased to over 400,000. In an attempt to curtail the flow of raw sewage into the Bay, San Diego and several neighboring communities combined their sewage outfalls into one system. Unfortunately, this new system was constantly operating on overload and discharging directly into the Bay. Simultaneously, the Bay received untreated industrial discharge from five fish canneries, a large rendering operation, a kelp processing plant, four aircraft manufacturing plants, several shipyards, and the Pacific coast's largest naval base, naval air station, and submarine base (San Diego Interagency Water Quality Panel, 1989). The California Regional Water Quality Control Board was established in 1950 (following the passage of the Dickey Act in 1949). Through extensive water sampling it was concluded that the entire Bay had become contaminated, due to heavy loading of domestic and industrial wastes. Dissolved oxygen concentrations in the Bay had declined to about half normal levels and turbidity in the water resulted in a visibility of less than 1 meter. Bait and game fish had virtually disappeared from the Bay. Coliform bacteria were routinely isolated from the Bay at significant levels. In 1955, the State Board of Public Health and the San Diego Department of Public Health declared much of the Bay contaminated, and posted quarantine and warning signs along 10 miles of shoreline. By 1963, sludge deposits from the treatment plant outfall were two meters deep, extended 200 meters seaward, and along 9000 meters of the shoreline.

A report in the early 1950's from the Regional Board and the San Diego Sewerage Survey report indicated sewage discharge into the Bay was becoming a major problem which had to be corrected. In 1960, San Diego voters approved a bond ($42.5 million) which allowed construction to begin on the Metropolitan Sewerage System. In August of 1963, a massive collection, treatment, and ocean disposal system began operation and by February, 1964, domestic sewage disposal had been eliminated from San Diego Bay. Following the completion of the new sewage treatment plant, dissolved oxygen concentrations rose to an average of more than 5 parts per million, visibility increased to 2 meters, and coliform bacteria counts dropped within the federal safety standards. Plankton blooms were scarce and sludge deposits of more than 30 cm were seldom reported. The sewage system currently processes 170 million gallons of waste per day (City of San Diego, 1995)

Routine sampling, beginning in the 1970's, revealed new information regarding the presence of industrial wastes in the Bay. Regulatory standards were developed for the protection of humans and wildlife based on new sampling systems and more refined analytical techniques. The conventional engineering and bacteriological data gathered earlier did not adequately address

the issue of toxic waste in the Bay. During the late 1980's, the press regarded San Diego Bay as being heavily contaminated, particularly for PCBs. Although conditions in the Bay are similar to other urban influenced embayments in the United States, San Diego Bay has serious problems with chemical pollution. A number of toxic hotspots in the Bay have been identified on lists of water quality impairment such as Clean Water Act Section 303(d), Section 319, Section 304(l) and Section 131.11.

Mission Bay

Mission Bay is located 9 kilometers north of Point Loma and encompasses an area of 1860 hectares. It has two main tributaries, Tecolote creek and Rose creek (Dexter, 1983). Originally named False Bay because its entrance was near San Diego Bay and occasionally fooled ship captains, it is now considered a recreational small-craft harbor (United States Coast Pilot, 1994). Prior to the development of Mission Bay park in 1946, Mission Bay was a natural estuary of over 2020 hectares of salt marshes, tidal channels, and a shallow central bay. Between 1946 and 1962 major dredging within the Bay and modifications to the San Diego River flood control channel gave way to its present-day configuration. Today it is a highly modified lagoon which receives freshwater input only during infrequent, heavy rains. The major additions of freshwater into Mission Bay occur at Rose Inlet, in the northeastern portion of the Bay, and Tecolote Creek, in the southeast. Because of this limited amount of freshwater, the salinities throughout the Bay do not change markedly. Mean tidal range is 1.2 meters and the mean diurnal range is 1.7 meters at the Bay entrance (Levin, 1983).

As a result of circulation patterns within Mission Bay, a variety of sediments are found. In the mouth of the Bay and near the main channel, water movement is sufficient to maintain a sandy bottom. In other parts of the Bay, such as Sail Bay and sites located further east, sediments are muddy with a high silt and clay content (Dexter, 1983).

Tecolote and Rose creeks carry urban pollutants such as oil, grease, fertilizers, and high sediment loads into the back bay. Furthermore, sewer lines back up occasionally into the back bay. The lack of water circulation in the back bay allows these pollutants to accumulate and has resulted in quarantines for several months at a time (Marcus, 1989).

Tijuana River Estuary

The Tijuana River Estuary is located 16 kilometers southeast of Point Loma. Although the estuary is situated entirely within the boundaries of San Diego County, three-fourths of its watershed is in Mexico. It is a wetland dominated estuary with no major embayment, however, a series of channels allows for a relatively narrow ocean connection (Herron, 1972). In the classification scheme developed by Prichard (1967), Tijuana Estuary is considered an intermittent coastal plain estuary due to the large

freshwater input during the winter wet season. During most years, the river mouth has been open and tidal flushing has prevailed. The intertidal area supports salt marsh vegetation (*Salicornia virginica*, *Spartina foliosa*), whereas mudflats and sandflats occupy only a small fraction of the estuary (Zedler *et al.*, 1992).

The Tijuana River Estuary has been altered substantially by natural and human disturbances. In the early 1900's, sewage disposal practices led to dredging of the east-west channel in order to connect an adjacent waste collecting lagoon with the estuary. Dikes were then created to subdivide the lagoon into three wastewater receiving ponds, however, these dikes were later removed to increase tidal flow. Gravel extraction for street and dike construction created isolated ponds within the estuary. Long-term dumping and filling altered most of the peripheral topography, while extensive damage to the southern half of the estuary from military, agricultural, and horse-raising activities is evident (Marcus, 1989).

Wastewater flow from Tijuana has been a serious threat to water quality in the estuary. In 1988, approximately 30 million gallons of sewage per day were produced while only 17 million gallons were collected. The remaining 13 million gallons emptied directly into the Tijuana River and estuary (Seamans, 1988). Breaks in the Tijuana sewer line, which carried collected sewage to an ocean outfall, were also common.

Recent U.S. projects have reduced the threat of sewage pollution. An interceptor on the Tijuana River, completed in early October 1991, diverts approximately 15 million gallons of sewage a day to the San Diego wastewater facility (Zedler, 1992). A sewage treatment plant is planned for the U.S. side of the border, and a new ocean outfall is under evaluation.

Sampling Design

Two basic sampling designs were used to meet both SWRCB's and NOAA's goals. A directed point sampling design was required to address SWRCB's need to identify specific toxic hot spots. A stratified random sampling design was required to address NOAA's need to evaluate spatial extent of pollution. This has resulted in a data set of 350 samples collected between October, 1992 and May, 1994. Of the 350 total samples, 229 were collected from directed point sampled stations and 121 were collected from randomly sampled stations.

When directed point sampling design was required, a two step process was used. Areas of interest were identified, by regional and state water board staff, for sampling during an initial "screening phase". Station locations (latitude & longitude) were predetermined by agreement with the SWRCB, NOAA, Regional Water Quality Control Boards, and DFG personnel. Changing of the site location during sediment collection was allowed only under the following conditions:

1. Lack of access to predetermined site,
2. Inadequate or unusable sediment (i.e. rocks or gravel)
3. Unsafe conditions
4. Agreement of appropriate staff

This phase of work was intended to give a broad assessment of toxicity throughout the San Diego Bay area using multiple test species and toxicity endpoints. Fifty-six stations were sampled during the period between October, 1992 and January, 1993. Chemical analysis was performed on selected samples in which toxicity results prompted further analysis. Stations which met certain criteria during the screening phase, or during the random sampling phase, were then selected for a second round of sampling, termed the "confirmation phase". During this phase sampling was replicated and chemical analysis of samples was more extensive. In addition, benthic community analysis was performed on all confirmation stations sampled during the summer of 1993. Evidence from this two step process is used to establish a higher level of certainty for stations which may later be identified as "toxic hot spots".

Stratified random sampling began in March, 1993 and continued through August, 1993, with a total of 121 stations sampled. The San Diego Bay Region was stratified into areas of similar physical characteristics or uses, such as transit channels, anchorages, marinas, commercial shipping or military uses, and designated as 95 blocks of known size (Figures 2a & 2b). Station coordinates were chosen randomly within the boundaries of each sampling block by USEPA Environmental Monitoring and Assessment Program (USEPA-EMAP) personnel using a computer program developed for that purpose. Eight alternate locations were chosen for each block, a maximum of two of which were actually sampled (Weisberg *et al.*, 1993). This stratified random design "forces"

Figure 2a
Sampling Blocks for Random Stations
San Diego Bay

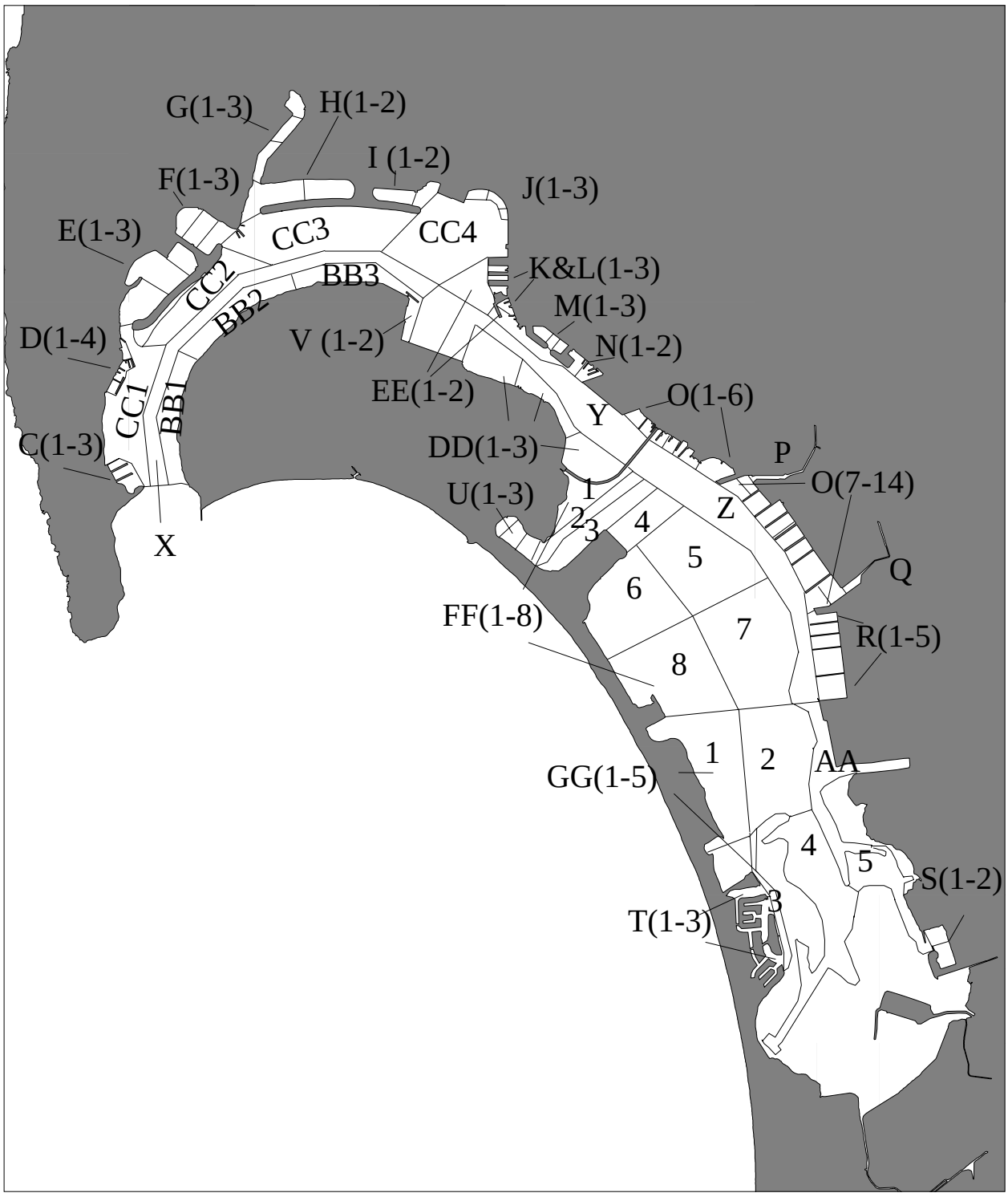
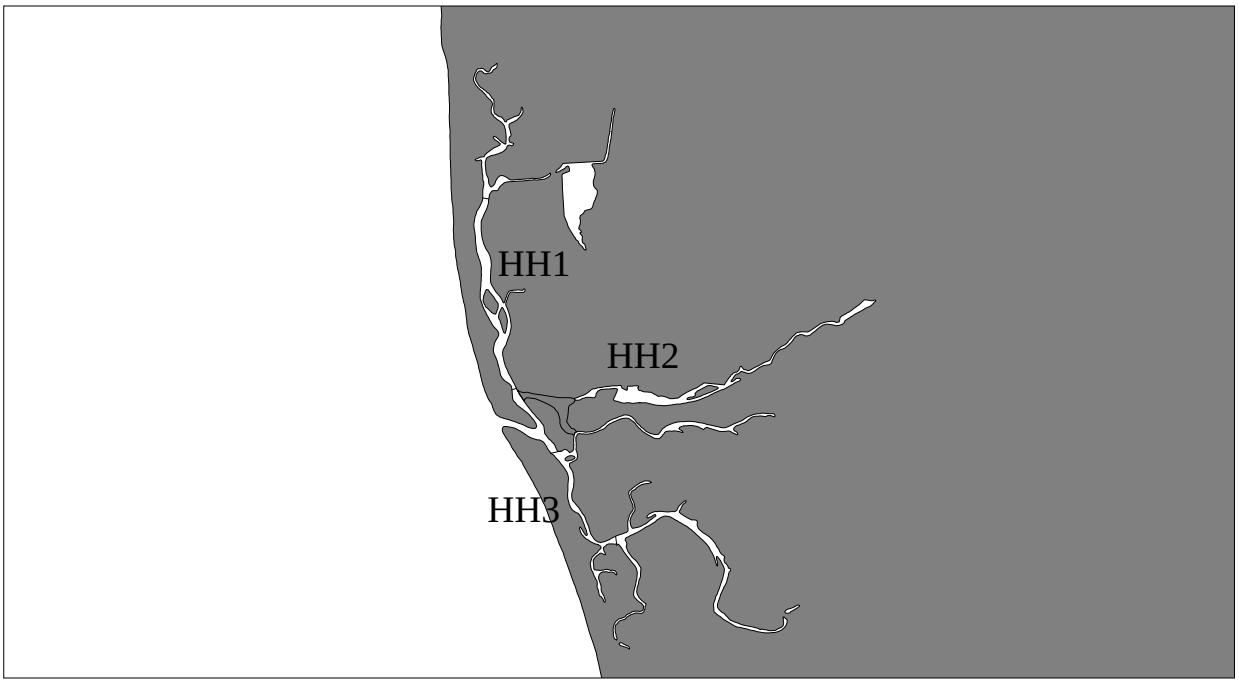


Figure 2b
Sampling Blocks for Random Stations
Mission Bay and San Diego River Estuary

Tijuana River Estuary



random samples to cover all areas of the Bay, whereas a pure random design most likely would miss some areas and oversample others. In the field, sampling was attempted at each designated location (x1-x8), beginning with x1, until a sample was retrieved which met sample acceptability criteria. For example, in block FF2, Station number 93124 was sampled at the random location x1 while in block FF3, Station #93172 was sampled at random location x4 because the grain size was too coarse at locations x1, x2 and x3. Of the 121 stations sampled, ≈15% could not be sampled at the random x1 location, due to the location being inaccessible by boat because of obstructions, vessel moorings, piers or shallow depths. Similarly, ≈3% were not sampled because the grain size was too coarse at the x1 location. Samples were collected successfully at alternate locations (x2, x3, x4, ...) for all stations where x1 was not sampled. This sampling design allows data from random stations to be used for calculation of areal extent of toxicity in the San Diego Bay Region. Chemical analyses were only performed on a limited number of random station samples.

From the combined sampling designs, a total of 350 samples were collected from 183 station locations in the San Diego Bay Region (Figure 3(a-d)). Station locations which were sampled more than once were always resampled at the original location using navigational equipment and lineups. Bioassay tests, grain size and total organic carbon analyses were performed on all 350 samples. Trace metal analysis was performed on 217 samples. Trace synthetic organic analysis was performed on 229 samples. Benthic community analysis was performed on 75 samples.

Sample Collection and Processing

Summary of Methods

Specific techniques used for collecting and processing samples are described in this section. Because collection of sediments influences the results of all subsequent laboratory and data analyses, it was important that samples be collected in a consistent and conventionally acceptable manner. Field and laboratory technicians were trained to conduct a wide variety of activities using standardized protocols to ensure comparability in sample collection among crews and across geographic areas. Sampling protocols in the field followed the accepted procedures of EMAP, NS&T, and ASTM and included methods to avoid cross-contamination; methods to avoid contamination by the sampling activities, crew, and vessel; collection of representative samples of the target surficial sediments; careful temperature control, homogenization and subsampling; and chain of custody procedures.

Cleaning Procedures

All sampling equipment (*i.e.*, containers, container liners, scoops, water collection bottles) was made from non-contaminating materials and was precleaned and packaged protectively prior to entering the field. Sample collection gear and samples were handled only by personnel wearing non-contaminating

Figure 3a
Sampling Locations
North San Diego Bay

Figure 3b
Sampling Locations
Mid San Diego Bay

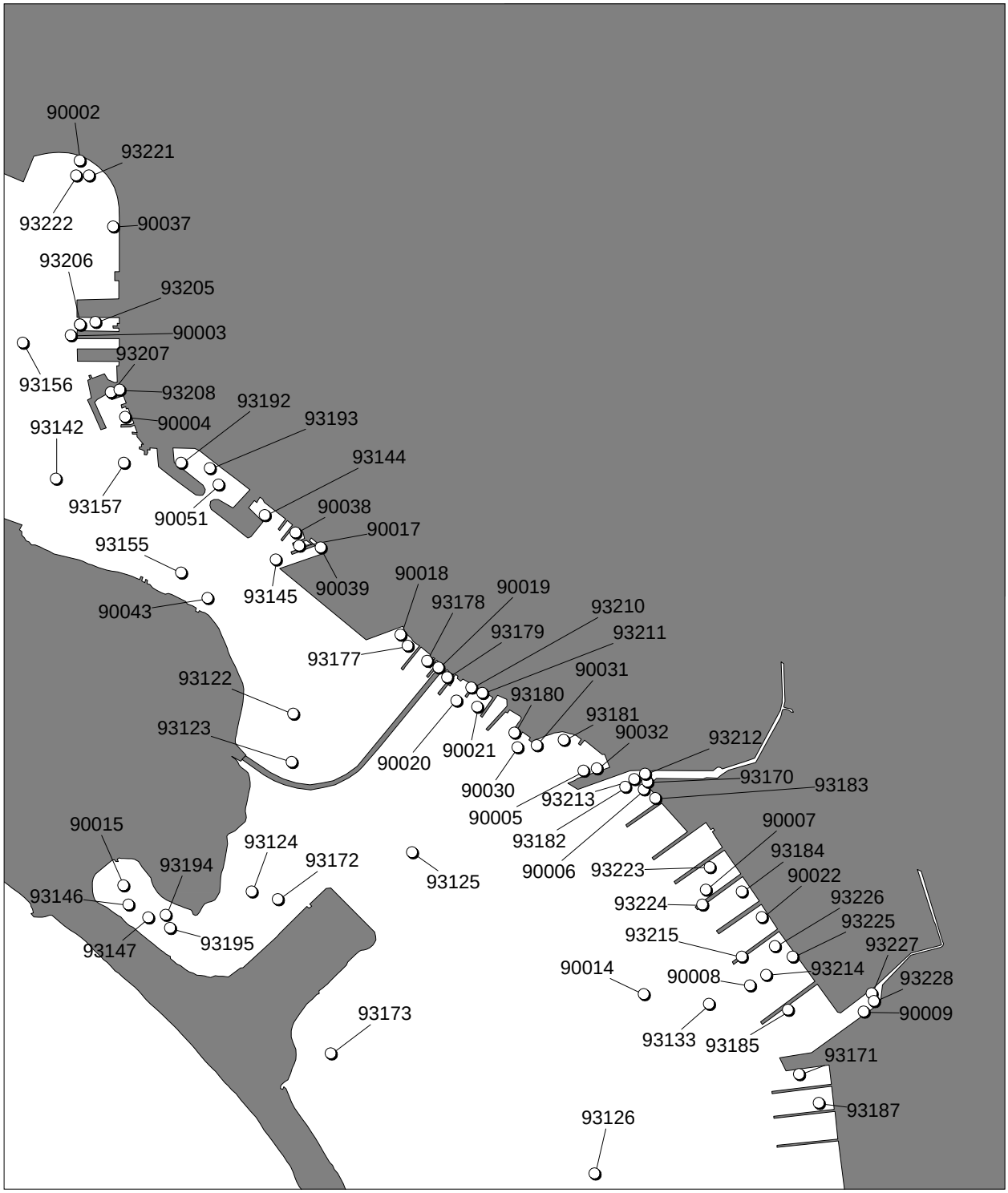


Figure 3c
Sampling Locations
South San Diego Bay

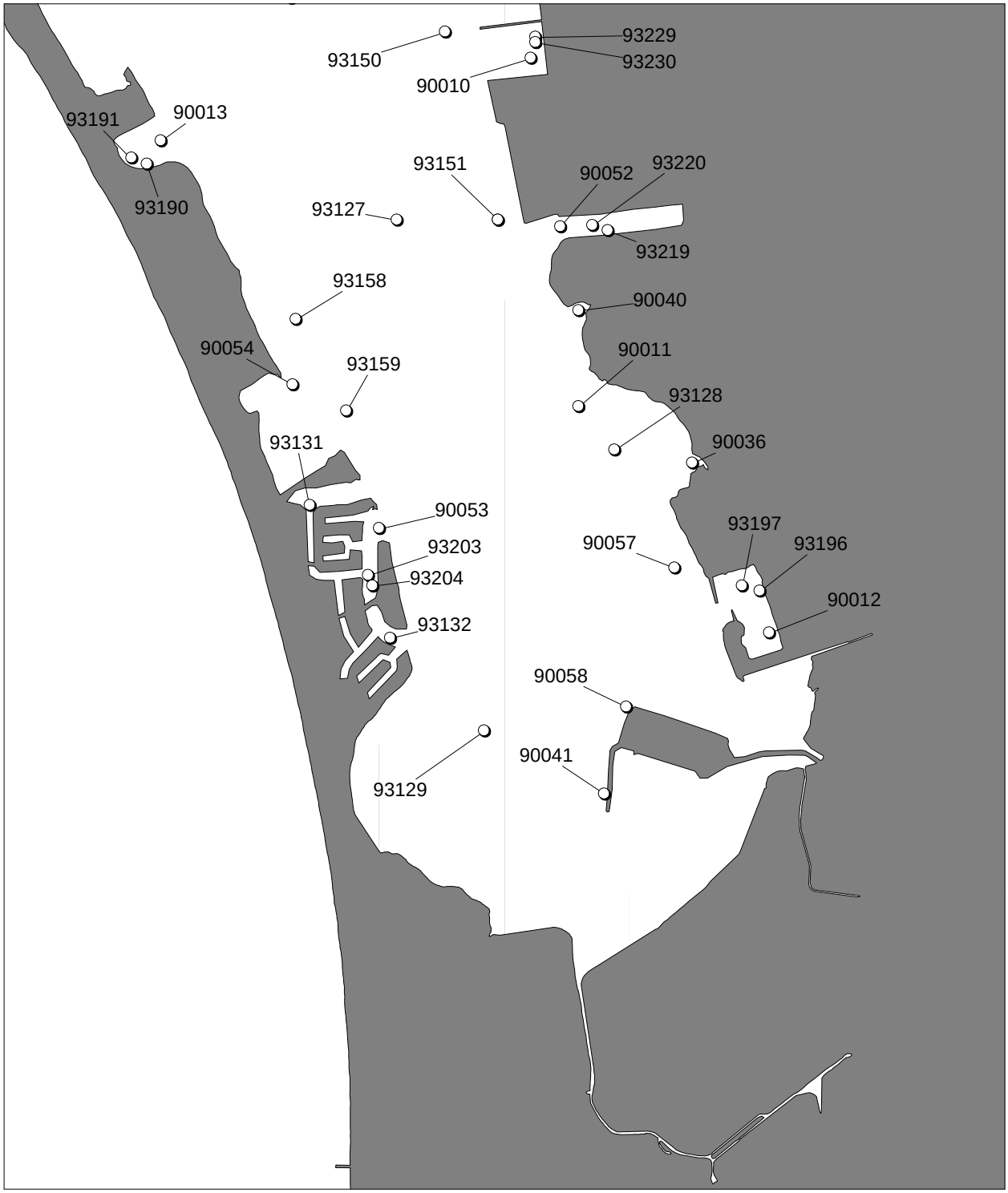


Figure 3d
Sampling Locations
Mission Bay and San Diego River Estuary

Tijuana River Estuary

polyethylene gloves. All sample collection equipment (excluding the sediment grab) was cleaned by using the following sequential process:

> Two-day soak and wash in Micro® detergent, three tap-water rinses, three deionized water rinses, a three-day soak in 10% HCl, three ASTM Type II Milli-Q® water rinses, air dry, three petroleum ether rinses, and air dry.

All cleaning after the Micro® detergent step was performed in a positive pressure "clean" room to prevent airborne contaminants from contacting sample collection equipment. Air supplied to the clean room was filtered.

The sediment grab was cleaned prior to entering the field, and between sampling stations, by utilizing the following sequential steps: a vigorous Micro® detergent wash and scrub, a sea-water rinse, a 10% HCl rinse, and a methanol rinse. The sediment grab was scrubbed with seawater between successive deployments at the same station to remove adhering sediments from contact surfaces possibly originating below the sampled layer.

Sample storage containers were cleaned in accordance with the type of analysis to be performed upon its contents. All containers were cleaned in a positive pressure "clean" room with filtered air to prevent airborne contaminants from contacting sample storage containers.

Plastic containers (HDPE or TFE) for trace metal analysis media (sediment, archive sediment, pore water, and subsurface water) were cleaned by: a two-day Micro® detergent soak, three tap-water rinses, three deionized water rinses, a three-day soak in 10% HCl or HNO_3, three Type II Milli-Q® water rinses, and air dry.

Glass containers for total organic carbon, grain size or synthetic organic analysis media (sediment, archive sediment, pore water, and subsurface water) and additional teflon sheeting cap-liners were cleaned by: a two-day Micro® detergent soak, three tap-water rinses, three deionized water rinses, a three-day soak in 10% HCl or HNO_3, three Type II Milli-Q® water rinses, air dry, three petroleum ether rinses, and air dry.

Sediment Sample Collection

All sampling locations (latitude & longitude), whether altered in the field or predetermined, were verified using a Magellan NAV 5000 Global Positioning System, and recorded in the field logbook. The primary method of sediment collection was by use of a $0.1m^2$ Young-modified Van Veen grab aboard a sampling vessel. Modifications include a non-contaminating Kynar coating which covered the grab's sample box and jaws. After the filled grab sampler was secured on the boat gunnel, the sediment sample was inspected carefully. The following acceptability criteria were met prior to taking sediment samples. If a sample did not meet all the criteria, it was rejected and another sample was collected.

1. Grab sampler was not over-filled (*i.e.*, the sediment surface was not pressed against the top of the grab).
2. Overlying water was present, indicating minimal leakage.
3. Overlying water was not excessively turbid, indicating minimal sample disturbance.
4. Sediment surface was relatively flat, indicating minimal sample disturbance.
5. Sediment sample was not washed out due to an obstruction in the sampler jaws.
6. Desired penetration depth was achieved (*i.e.*, 10 cm).
7. Sample was muddy (>30% fines), not sandy or gravelly.
8. Sample did not include excessive shell, organic or man-made debris.

It was critical that sample contamination be avoided during sample collection. All sampling equipment (*i.e.*, siphon hoses, scoops, containers) was made of non-contaminating material and was cleaned appropriately before use. Samples were not touched with un-gloved fingers. In addition, potential airborne contamination (*e.g.*, from engine exhaust, cigarette smoke) was avoided. Before sub-samples from the grab sampler were taken, the overlying water was removed by slightly opening the sampler, being careful to minimize disturbance or loss of fine-grained surficial sediment. Once overlying water was removed, the top 2 cm of surficial sediment was sub-sampled from the grab. Subsamples were taken using a precleaned flat bottom scoop. This device allowed a relatively large sub-sample to be taken from a consistent depth. When subsampling surficial sediments, unrepresentative material (e.g., large stones or vegetative material) was removed from the sample in the field. Small rocks and other small foreign material remained in the sample. Determination of overall sample quality was determined by the chief scientist in the field. Such removals were noted on the field data sheet. For the sediment sample, the top 2 cm was removed from the grab and placed in a pre-labeled polycarbonate container. Between grabs or cores, the sediment sample in the container was covered with a teflon sheet, and the container covered with a lid and kept cool. When a sufficient amount of sediment was collected, the sample was covered with a teflon sheet assuring no air bubbles. A second, larger teflon sheet was placed over the top of the container to ensure an air tight seal, and nitrogen was vented into the container to purge it of oxygen. If water depth did not permit boat entrance to a site (*e.g.*, <1 meter), divers sampled that site using sediment cores (diver cores). Cores consisted of a 10 cm diameter polycarbonate tube, 30 cm in length, including plastic end caps to aid in transport. Divers entered a study site from one end and sampled in one direction, so as to not disturb the sediment with feet or fins. Cores were taken to a depth of at least 15 cm. Sediment was extruded out of the top end of the core to the prescribed depth of 2-cm, removed with a polycarbonate spatula and deposited into a cleaned polycarbonate tub. Additional samples were taken with the same seawater rinsed core tube until the required total sample volume was attained. Diver core samples were treated the

same as grab samples, with teflon sheets covering the sample and nitrogen purging. All sample acceptability criteria were met as with the grab sampler.

Replicate benthic samples (n=5) were obtained at predetermined sites from separate deployments of the sampler. Three of the replicates were positioned according to the BPTCP sampling protocol (*e.g.*, located by previously assigned lat/long coordinates), while the other two replicates were chosen within the location range of the previous three samples. The coring device was 10 cm in diameter and 14 cm in height, enclosing a 0.0075 m^2 area. Corers were placed into sediment with minimum disruption of the surface sediments, capturing essentially all surface-active fauna as well as species living deeper in the sediment. Corers were pushed about 12 cm into the sediment and retrieved by digging along one side, removing the corer and placing the intact sediment core into a pvc screening device. Sediment cores were sieved through a 0.5 mm screen and residues (*e.g.*, organisms and remaining sediments) were rinsed into pre-labeled storage bags and preserved with a 10% formalin solution. After 3 to 4 days, samples were rinsed and transferred into 70% isopropyl alcohol and stored for future taxonomy and enumeration.

Transport of Samples

Six-liter sample containers were packed (three to an ice chest) with enough ice to keep them cool for 48 hours. Each container was sealed in precleaned, large plastic bags closed with a cable tie to prevent contact with other samples or ice or water. Ice chests were driven back to the laboratory by the sampling crew or flown by air freight within 24 hours of collection.

Homogenization and Aliquoting of Samples

Samples remained in ice chests (on ice, in double-wrapped plastic bags) until the containers were brought back to the laboratory for homogenization. All sample identification information (station numbers, etc.) was recorded on Chain of Custody (COC) and Chain of Record (COR) forms prior to homogenizing and aliquoting. A single container was placed on plastic sheeting while also remaining in original plastic bags. The sample was stirred with a polycarbonate stirring rod until mud appeared homogeneous.

All prelabeled jars were filled using a clean teflon or polycarbonate scoop and stored in freezer/refrigerator (according to media/analysis) until analysis. The sediment sample was aliquoted into appropriate containers for trace metal analysis, organic analysis, pore water extraction, and bioassay testing. Samples were placed in boxes sorted by analysis type and leg number. Sample containers for sediment bioassays were placed in a refrigerator (4^{o}C) while sample containers for sediment chemistry (metals, organics, TOC and grain size) were stored in a freezer (-20^{o}C).

Procedures for the Extraction of Pore Water

The BPTCP primarily used whole core squeezing to extract pore water. The whole core squeezing method, developed by Bender *et*

al. (1987), utilizes low pressure mechanical force to squeeze pore water from interstitial spaces. The following squeezing technique was a modification of the original Bender design with some adaptations based on the work of Fairey (1992), Carr *et al.* (1989), and Long and Buchman (1989). The squeezer's major features consist of an aluminum support framework, 10 cm i.d. acrylic core tubes with sampling ports and a pressure regulated pneumatic ram with air supply valves. Acrylic subcore tubes were filled with approximately 1 liter of homogenized sediment and pressure was applied to the top piston by adjusting the air supply to the pneumatic ram. At no time during squeezing did air pressure exceed 200 psi. A porous prefilter (PPE or TFE) was inserted in the top piston and used to screen large (> 70 microns) sediment particles. Further filtration was accomplished with disposable TFE filters of 5 microns and 0.45 microns in-line with sample effluent. Sample effluent of the required volume was collected in TFE containers under refrigeration. Pore water was subsampled in the volumes and specific containers required for archiving, chemical or toxicological analysis. To avoid contamination, all sample containers, filters and squeezer surfaces in contact with the sample were plastics (acrylic, PVC, and TFE) and cleaned with previously discussed clean techniques.

Chain of Records & Custody

Chain-of-records documents were maintained for each station. Each form was a record of all sub-samples taken from each sample. IDORG (a unique identification number for only that sample), station numbers and station names, leg number (sample collection trip batch number), and date collected were included on each sheet. A Chain-of-Custody form accompanied every sample so that each person releasing or receiving a subsample signed and dated the form.

Authorization/Instructions to Process Samples

Standardized forms entitled "Authorization/Instructions to Process Samples" accompanied the receipt of any samples by any participating laboratory. These forms were completed by DFG personnel, or its authorized designee, and were signed and accepted by both the DFG authorized staff and the staff accepting samples on behalf of the particular laboratory. The forms contain all pertinent information necessary for the laboratory to process the samples, such as the exact type and number of tests to run, number of laboratory replicates, dilutions, exact eligible cost, deliverable products (including hard and soft copy specifications and formats), filenames for soft copy files, expected date of submission of deliverable products to DFG, and other information specific to the lab/analyses being performed.

Trace Metals Analysis of Sediments

Summary of Methods

Trace Metals analyses were conducted at the California Department of Fish and Game's (CDFG) Trace Metals Facility at Moss Landing, CA. Table 1 indicates the trace metals analyzed and lists method detection limits for sediments. These methods were modifications

of those described by Evans and Hanson (1993) as well as those developed by the CDFG (California Department of Fish and Game, 1990). Samples were selected for chemical analyses by SWRCB staff based on results from toxicity tests.

Analytes and Detection Limits

Table 1 - Trace Metal Detection Limits in Sediments (μg/g, dry weight).

Aluminum	1	Antimony	0.1
Arsenic	0.1	Cadmium	0.01
Chromium	0.1	Copper	0.1
Iron	0.1	Lead	0.1
Manganese	0.05	Mercury	0.03
Nickel	0.1	Selenium	0.2
Silver	0.01	Tin	0.02
Tributyltin	0.013	Zinc	0.05

Sediment Digestion Procedures

One gram aliquot of sediment was placed in a pre-weighed Teflon vessel, and one ml concentrated 4:1 nitric:perchloric acid mixture was added. The vessel was capped and heated in a vented oven at 130^0 C for four hours. Three ml Hydrofluoric acid were added to vessel, recapped and returned to oven overnight. Twenty ml of 2.5% boric acid were added to vessel and placed in oven for an additional 8 hours. Weights of vessel and solution were recorded, and solution transfered to 30 ml polyethylene bottles.

Atomic Absorption Methods

Samples were analyzed by furnace AA on a Perkin-Elmer Zeeman 3030 Atomic Absorption Spectrophotometer, with an AS60 auto sampler, or a flame AA Perkin Elmer Model 2280. Samples, blanks, matrix modifiers, and standards were prepared using clean techniques inside a clean laboratory. ASTM Type II water and ultra clean chemicals were used for all standard preparations. All elements were analyzed with platforms for stabilization of temperatures. Matrix modifiers were used when components of the matrix interferes with adsorption. The matrix modifier was used for Sn, Sb and Pb. Continuing calibration check standards (CLC) were analyzed with each furnace sheet, and calibration curves were run with three concentrations after every 10 samples. Blanks and standard reference materials, MESS1, PACS, BCSS1 or 1646 were analyzed with each set of samples for sediments.

Trace Organic Analysis of Sediments (PCBs, Pesticides, and PAHs)

Summary of Methods

Analytical sets of 12 samples were scheduled such that extraction and analysis will occur within a 40 day window. The methods employed by the UCSC-TOF were modifications of those described by Sloan *et al*. (1993). Tables 2 and 3 indicate the pesticides, PCBs, and PAHs currently analyzed and list method detection limits for sediments on a dry weight basis.

Analytes and Detection Limits

Table 2. Organochlorine Pesticides Analyzed and Their Detection Limits in Sediment, ng/g dry weight.

Aldrin	0.5
cis-Chlordane	0.5
trans-Chlordane	0.5
alpha-Chlordene	0.5
gamma-Chlordene	0.5
Chlorpyrifos	1.0
Dacthal	0.2
o,p'-DDD	1.0
p,p'-DDD	0.4
o,p'-DDE	1.0
p,p'-DDE	1.0
p,p'-DDMS	3.0
p,p'-DDMU	2.0
o,p'-DDT	1.0
p,p'-DDT	1.0
p,p'-Dichlorobenzophenone	3.0
Dieldrin	0.5
Endosulfan I	0.5
Endosulfan II	1.0
Endosulfan sulfate	2.0
Endrin	2.0
Ethion	2.0
alpha-HCH	0.2
beta-HCH	1.0
gamma-HCH	0.2
delta-HCH	0.5
Heptachlor	0.5
Heptachlor Epoxide	0.5
Hexachlorobenzene	0.2
Methoxychlor	1.5
Mirex	0.5
cis-Nonachlor	0.5
trans-Nonachlor	0.5
Oxadiazon	2.0
Oxychlordane	0.5
Toxaphene	10

Table 3. PCB Congeners and PAHs Analyzed and Their Detection Limits in Sediment, ng/g dry weight.

NIST Congeners:

PCB Congener 8	PCB Congener 128
PCB Congener 18	PCB Congener 138
PCB Congener 28	PCB Congener 153
PCB Congener 44	PCB Congener 170
PCB Congener 52	PCB Congener 180
PCB Congener 66	PCB Congener 187
PCB Congener 87	PCB Congener 195
PCB Congener 101	PCB Congener 206

Table 3 (cont.). PCB Congeners and PAHs Analyzed and Their Detection Limits in Sediment, ng/g dry weight.

PCB Congener 105	PCB Congener 209
PCB Congener 118	

Additional Congeners:

PCB Congener 5	PCB Congener 137
PCB Congener 15	PCB Congener 149
PCB Congener 27	PCB Congener 151
PCB Congener 29	PCB Congener 156
PCB Congener 31	PCB Congener 157
PCB Congener 49	PCB Congener 158
PCB Congener 70	PCB Congener 174
PCB Congener 74	PCB Congener 177
PCB Congener 95	PCB Congener 183
PCB Congener 97	PCB Congener 189
PCB Congener 99	PCB Congener 194
PCB Congener 110	PCB Congener 201
PCB Congener 132	PCB Congener 203

All individual PCB Congener detection limits were 1 ng/g dry weight.

Aroclors:

Aroclor 5460	50

Polycyclic Aromatic Hydrocarbons

Naphthalene	5
2-Methylnaphthalene	5
1-Methylnaphthalene	5
Biphenyl	5
2,6-Dimethylnaphthalene	5
Acenaphthylene	5
Acenaphthene	5
2,3,5-Trimethylnaphthalene	5
Fluorene	5
Phenanthrene	5
Anthracene	5
1-Methylphenanthrene	5
Fluoranthrene	5
Pyrene	5
Benz[a]anthracene	5
Chrysene	5
Benzo[b]fluoranthrene	5
Benzo[k]fluoranthrene	5
Benzo[e]pyrene	5
Benzo[a]pyrene	5
Perylene	5
Indo[1,2,3-cd]pyrene	5
Dibenz[a,h]anthracene	5
Benzo[ghi]perylene	5

Extraction and Analysis
Samples were removed from the freezer and allowed to thaw. A 10 gram sample of sediment was removed for chemical analysis and an independent 10 gram aliquot was removed for dry weight determinations. The dry weight sample was placed into a pre-weighed aluminum pan and dried at 110◦C for 24 hours. The dried sample was reweighed to determine the sample's percent moisture. The analytical sample was extracted 3 times with methylene chloride in a 250-mL amber Boston round bottle on a modified rock tumbler. Prior to rolling, sodium sulfate, copper, and extraction surrogates were added to the bottle. Sodium sulfate dehydrates the sample allowing for efficient sediment extraction. Copper, which was activated with hydrochloric acid, complexes free sulfur in the sediment.

After combining the three extraction aliquots, the extract was divided into two portions, one for chlorinated hydrocarbon (CH) analysis and the other for polycyclic aromatic hydrocarbon (PAH) analysis.

The CH portion was eluted through a silica/alumina column, separating the analytes into two fractions. Fraction 1 (F1) was eluted with 1% methylene chloride in pentane and contains > 90% of p,p'-DDE and < 10% of p,p'-DDT. Fraction 2 (F2) analytes were eluted with 100% methylene chloride. The two fractions were exchanged into hexane and concentrated to 500 µL using a combination of rotary evaporation, controlled boiling on tube heaters, and dry nitrogen blow downs.

F1 and F2 fractions were analyzed on Hewlett-Packard 5890 Series gas chromatographs utilizing capillary columns and electron capture detection (GC/ECD). A single 2 µl splitless injection was directed onto two 60m x 0.25mm i.d. columns of different polarity (DB-17 & DB-5; J&W Scientific) using a glass Y-splitter to provide a two dimensional confirmation of each analyte. Analytes were quantified using internal standard methodologies. The extract's PAH portion was eluted through a silica/alumina column with methylene chloride. It then underwent additional cleanup using size-exclusion high performance liquid chromatography (HPLC/SEC). The collected PAH fraction was exchanged into hexane and concentrated to 250 µL in the same manner as the CH fractions.

Total Organic Carbon Analysis of Sediments

Summary of Methods
Samples were received in the frozen state and allowed to thaw at room temperature. Source samples were gently stirred and sub-samples were removed with a stainless steel spatula and placed in labeled 20 ml polyethylene scintillation vials. Approximately 5 grams equivalent dry weight of the wet sample was sub-sampled.

Sub-samples were treated with two, 5 ml additions of 0.5 N, reagent grade HCl to remove inorganic carbon (CO^{-3}), agitated, and centrifuged to a clear supernate. Some samples were retreated with HCl to remove residual inorganic carbon. The evolution of gas during HCl treatment indicates the direct presence of inorganic carbon (CO^{-3}). After HCl treatment and decanting, samples were washed with approximately 15 ml of deionized-distilled water, agitated, centrifuged to a clear supernate, and decanted. Two sample washings were required to remove weight determination and analysis interferences.

Prepared samples were placed in a 60◦ C convection oven and allowed to come to complete dryness (approx. 48 hrs.). Visual inspection of the dried sample before homogenization was used to ensure complete removal of carbonate containing materials, (shell fragments). Two 61 mm (1/4") stainless steel solid balls were added to the dried sample, capped and agitated in a commercially available ball mill for three minutes to homogenize the dried sample.

A modification of the high temperature combustion method, utilizing a Weatstone bridge current differential was used in a commercially available instrument, (Control Equipment Co., 440 Elemental Analyzer) to determine carbon and nitrogen concentrations. The manufactures suggested procedures were followed. The methods are comparable to the validation study of USEPA method MARPCPN I. Two to three aliquotes of 5-10 mg of dried prepared sub-sample were used to determine carbon and nitrogen weight percent values. Calibration of the instrument was with known standards using Acetanilide or L-Cystine. Detection limits are 0.2 ug/mg, carbon and 0.01 ug/mg nitrogen dry weight.

The above methods and protocols are modifications of several published papers, reference procedures and analytical experimentation experience (Franson, 1981; Froelich, 1980; Hedges and Stern, 1983; MARPCPN I, 1992).

Quality Control/Quality Assurance

Quality control was tested by the analysis of National Research Council of Canada Marine Sediment Reference Material, BCSS-1 at the beginning and end of each sample analysis set (20-30 individual machine analyses). All analyzed values were within suggested criteria of $\pm$ 0.09% carbon (2.19% Average). Nitrogen was not reported on the standard data report, but was accepted at $\pm$ 0.008% nitrogen (0.195% Average) from the EPA study. Quality assurance was monitored by re-calibration of the instrument every twenty samples and by the analysis of a standard as a unknown and comparing known theoretical percentages with resultant analyzed percentages. Acceptable limits of standard unknowns were less than $\pm$ 2%. Duplicate or triplicate sample analysis variance (standard deviation/mean) greater than 7% is not accepted. Samples were re-homogenized and re-analyzed until the variance between individual runs fell below the acceptable limit of 7.0%.

Grain Size Analysis of Sediments

Summary of Methods
The procedure used combined wet and dry sieve techniques to determine particle size of sediment samples. Methods follow those of Folk (1974).

Sample Splitting and Preparation
Samples were thawed and thoroughly homogenized by stirring with a spatula. Spatulas were rinsed of all adhering sediment between samples. Size of the subsample for analysis was determined by the sand/silt ratio of the sample. During splitting, the sand/silt ratio was estimated and an appropriate sample weight was calculated. Subsamples were placed in clean, pre-weighed beakers. Debris was removed and any adhering sediment was washed into the beaker.

Wet Sieve Analysis (separation of coarse and fine fraction)
Beakers were placed in a drying oven and sediments were dried at less than 55◦C until completely dry (approximately three days). Beakers were removed from drying oven and allowed to equilibrate to room temperature for a least a half-hour. Each beaker and its contents were weighed to the nearest .01 g. This weight minus the empty beaker weight was the total sample weight. Sediments in beakers were disaggregated using 100 ml of a dispersant solution in water (such as 50g Calgon/L water) and the sample was stirred until completely mixed and all lumps disappear. The amount and concentration of dispersant used was recorded on the data sheet for each sample. Sample beakers were placed in an ultrasonic cleaner for 15 minutes for disaggregation. Sediment dispersant slurry was poured into a 63 μm (ASTM #230, 4 phi) stainless steel or brass sieve in a large glass funnel suspended over a 1L hydrometer cylinder by a ring stand. All fine sediments were washed through the sieve with water. Fine sediments were captured in a 1L hydrometer cylinder. Coarse sediments remaining in sieve were collected and returned to the original sample beaker for quantification.

Dry Sieve Analysis (coarse fraction)
The coarse fraction was placed into a preweighed beaker, dried at 55-65◦C, allowed to acclimate, and then weighed to 0.01 g. This weight, minus the empty beaker weight, was the coarse fraction weight. The coarse fraction was poured into the top sieve of a stack of ASTM sieves having the following sizes: No. 10 (2.0 mm), 18 (1.0 mm), 45 (0.354 mm), 60 (0.25 mm), 80 (0.177 mm), 120 (0.125 mm), and 170 (0.088 mm). The stack was placed on a mechanical shaker and shaken at medium intensity for 15 minutes. After shaking, each sieve was inverted onto a large piece of paper and tapped 5 times to free stuck particles. The sieve fractions were added cumulatively to a weighing dish, and the cumulative weight after each addition determined to 0.01g. The sample was returned to its original beaker, and saved until sample computations were completed and checked for errors.

Analytical Procedures
Fractional weights and percentages for various particle size fractions were calculated. If only wet sieve analysis was used, weight of fine fraction was computed by subtracting coarse fraction from total sample weight, and percent fine composition was calculated using fine fraction and total sample weights. If dry sieve was employed as well, fractional weights and percentages for the sieve were calculated using custom software on a Macintosh computer. Calibration factors were stored in the computer.

Benthic Community Analysis

Summary of Methods
Each catalogued sample was processed individually in the laboratory to obtain an accurate assessment of species diversity and abundance. All macroinvertebrates were sorted from residues under a dissecting microscope, identified to lowest possible taxon, and counted. Laboratory processing of benthic cores consists of both rough and fine sorting. Initial sorting separates animals into large taxonomic groups such as polychaetes, crustaceans, mollusks and other (*e.g.*, phoronids). Bound laboratory logbooks were maintained and used to record number of samples processed by each technician, as well as results of any sample resorts, if necessary. Sorters were required to sign and date a Milestone Progress Checksheet for each replicate sample processed. Specimens of similar taxonomic groups were placed in vials and labelled internally and externally with project, date collected, site/station information, and IDORG. Samples were selected for benthic community analysis by SWRCB staff based on results from toxicity tests.

In-house senior taxonomists and outside specialists processed and verified the accuracy of species identification and enumeration. An archived voucher specimen collection was established at this time.

Toxicity Testing

Summary of Methods
All toxicity tests were conducted at the California Department of Fish and Game's Marine Pollution Studies Laboratory (MPSL) at Granite Canyon. Toxicity tests were conducted by personnel from the Institute of Marine Sciences, University of California, Santa Cruz.

Pore Water Samples
Once at MPSL, frozen pore water samples were stored in the dark, at -12^{0}C, until required for testing. Experiments performed by the U.S. National Biological Survey have shown no effects of freezing porewater upon the results of toxicity tests (Carr *et al.*, 1995). Samples were thawed on the day of a test, and pH,

temperature, salinity, and dissolved oxygen were measured in all samples to verify water quality criteria were within the limits defined for test protocol. Pore water samples with salinities outside specified ranges for each protocol were adjusted to within the acceptable range. Salinities were increased by the addition of hypersaline brine, 60 to 80 parts per thousand (ppt), drawn from partially frozen seawater. Dilution water consisted of Granite Canyon seawater (32 to 34 ppt). Water quality parameters were measured at the beginning and end of each test. Dissolved oxygen concentrations and pH were measured using an Orion EA940 expandable ion analyzer. Salinity was measured with a refractometer. Temperature of each sample was measured with a mercury thermometer.

Measurement of Ammonia and Hydrogen Sulfide

Total ammonia concentrations were measured using an Orion Model 95-12 Ammonia Electrode. The concentration of unionized ammonia was derived from the concentration of total ammonia using the following equation (from Whitfield 1974, 1978):

$$[NH_3] = [\text{total ammonia}] \times ((1 + \text{antilog}(pK_a^\circ - pH))^{-1}),$$

where pK_a° is the stoichiometric acidic hydrolysis constant for the test temperature and salinity. Values for pK_a°were experimentally derived by Khoo *et al*. (1977). The method detection limit for total ammonia was 0.1 mg/L.

Total sulfide concentrations were measured using an Orion Model 94-16 Silver/Sulfide Electrode, except that samples tested after February, 1994, were measured on a spectrophotometer using a colorimetric method (Phillips et al. in press). The concentration of hydrogen sulfide was derived from the concentration of total sulfide by using the following equation (ASCE 1989):

$$[H_2S] = [S^{2-}] \times (1 - ((1 + \text{antilog}(pK_a^\circ - pH))^{-1})),$$

where temperature and salinity dependent pK_a° values were taken from Savenko (1977). The method detection limit for total sulfide was 0.1 mg/L for the electrode method, and 0.01 mg/L for the colorimetric method. Values and corresponding detection limits for unionized ammonia and hydrogen sulfide were an order of magnitude lower than those for total ammonia and total sulfide, respectively.

Subsurface Water Samples

The subsurface water toxicity tests are water column toxicity tests (abalone development, mussel development, etc..) performed on water collected with the modified Van Veen grab. A water sample bottle on the frame of the grab and a stopper is pulled as the jaws of the grab close for a sediment sample. The water sample is consequently collected approximately 0.5 meters above the bottom. Subsurface water samples were held in the dark at 4^0C until testing. Toxicity tests were initiated within 14 days of

the sample collection date. Water quality parameters, including ammonia and sulfide concentrations, were measured in one replicate test container from each sample in the overlying water as described above. Measurements were taken at the beginning and end of all tests.

Sediment Samples
Bedded sediment samples were held at 4^0C until required for testing. All *Rhepoxynius abronius* and *Neanthes arenaceodentata* solid phase sediment tests were initiated within 14 days of the sample collection date. All sediment samples were processed according to procedures described in ASTM (1992). Water quality parameters, including ammonia and sulfide concentrations, were measured in one replicate test container from each sample in the overlying water as described above. Measurements were taken at the beginning and end of all *Rhepoxynius* and *Neanthes* tests, and during overlying water renewals in the *Neanthes* tests.

Sea Urchin Larval Development Test
The sea urchin (*Strongylocentrotus purpuratus*) larval development test was conducted on all pore water samples. Details of the test protocol were given in Dinnel (1992). A brief description of the method follows.

Sea urchins were collected from the Monterey County coast near Granite Canyon, and held at MPSL at ambient seawater temperature and salinity (approx. 32±2 ppt) until testing. Adult sea urchins were held in complete darkness to preserve gonadal condition. On the day of a test, urchins were induced to spawn in air by injection with 0.5M KCl. Eggs and sperm collected from the urchins were mixed in seawater at a 500 to 1 sperm to egg ratio, and embryos were distributed to test containers within 1 hour of fertilization. Test containers were polyethylene-capped, sea-water leached, 20ml glass scintillation vials containing 5 mls of pore water. Each test container was inoculated with approximately 150 embryos (30/ml). All pore water samples were tested at three concentrations: 100, 50 and 25% pore water, each having three replicates. Pore water samples were diluted when necessary with one micron-filtered Granite Canyon seawater. Laboratory controls were included with each set of samples tested. Controls include a dilution water control consisting of Granite Canyon seawater, a brine control with all samples that require brine adjustment, and in some tests a frozen seawater control consisting of Granite Canyon seawater that has been frozen along with the pore water samples. Tests were conducted at ambient seawater salinity (usually 33±2 ppt). A positive control reference test was conducted concurrently with each pore water test using a dilution series of copper chloride as a reference toxicant.

After an exposure of 72 or 96 hours (no difference in results was detectable between these periods), larvae were fixed in 5% buffered formalin. Approximately 100 larvae in each container were examined under an inverted light microscope at 100x to determine the proportion of normally developed larvae as

described by Dinnel (1992). Visual clues used to identify embryos as normal included development of skeletal rods (spicules) that extend beyond half the length of the larvae and normal development of a three part gut. Slow growing embryos were considered abnormal.

Percent normal development was calculated as:

$$\frac{\text{(Number of normally developed larvae) X 100}}{\text{(Total number of observed larvae + number of abnormal larvae)}}$$

Sea Urchin Fertilization Test
The sea urchin (*Strongylocentrotus purpuratus*) fertilization test was conducted on pore water samples. Details of the test protocol were described in Dinnel *et al*. (1987).

Sea urchins were from the same stock described for the sea urchin larval development test. On the day of a test, urchins were induced to spawn in air by injection with 0.5M KCl. Sperm were exposed in test containers for sixty minutes before approximately 1000 eggs were added. After twenty minutes of fertilization, the test was fixed in a 5% buffered formalin solution. A constant sperm to egg ratio of 500 to 1 was used in all tests. This ratio maintained fertilization in the 70-90% range required by the test protocol. Fertilization was determined by the presence or absence of a fertilization membrane (raised chorion completely surrounding the egg). Test containers were polyethylene-capped, sea-water leached, 20ml glass scintillation vials containing 5 mls of pore water. All pore water samples were tested at three concentrations: 100, 50 and 25% pore water, each having three replicates. Pore water samples were diluted with one micron-filtered Granite Canyon seawater. Laboratory controls were included with each set of samples tested. Controls included a dilution water control consisting of Granite Canyon seawater, a brine control with all samples that require brine adjustment, and in some tests a frozen seawater control consisting of Granite Canyon seawater that has been frozen along with the pore water samples. Tests were conducted at ambient seawater salinity (usually 33±2 ppt). A positive control reference test was conducted concurrently with each pore water test using a dilution series of copper chloride as a reference toxicant. All eggs in each container were examined under an inverted light microscope at 100x, and counted as either fertilized or unfertilized.

Percent fertilization was calculated as:

$$\frac{\text{(Number of fertilized eggs) x 100}}{\text{(Number of fertilized eggs + number of unfertilized eggs)}}$$

Sea Urchin Cytogenetics Test
Analysis of cytogenetic abnormalities using sea urchin embryos followed methods described in Hose (1985). Sea urchin embryos were exposed to pore water for 48 hours then preserved in 5% buffered formalin. Embryos were placed on a clean glass

microscope slide and excess formalin removed with tissue paper. Embryos were then treated with a few drops of aceto-orcein stain (19 parts aceto-orcein:one part propionic acid) for approximately 1 to 3 minutes, and a cover slip was then applied to the darkly stained embryos. Excess stain was removed by blotting, and embryos were compressed into a monolayer by application of direct pressure. Embryo monolayer preparations were observed under oil immersion using either an Olympus BH2 or Tiyoda light microscope at 100x magnification. Cytogenetic abnormalities were observed in mitotic cells in anaphase and telophase. Possible aberrations observed followed those described in Hose (1985), including: stray or lagging chromosomes, accentric or attached chromosome fragments, and translocated or side-arm bridges . Because a majority of the embryos exposed to the 100 and 50% pore water concentrations displayed gross developmental abnormalities, mitotic aberrations were generally assessed using embryos exposed to 25% pore water.

Red Abalone Larval Development Test

The red abalone (*Haliotis rufescens*) larval development test was conducted on all subsurface water samples. Details of the test protocol were described in Anderson *et al*. (1990). The following was a brief description of the method. Adult male and female abalone were induced to spawn separately using a dilute solution of hydrogen peroxide in sea water. Fertilized eggs were distributed to the test containers within 1 hour of fertilization. Test containers were polyethylene-capped, seawater leached scintillation vials containing 10 mls of sample water. Each of five replicate test containers were inoculated with 100 embryos (10/ml).

Positive control reference tests using zinc sulfate as a reference toxicant were conducted concurrently with each batch of samples. A negative sea water control consisting of one micron-filtered Granite Canyon seawater was tested along with sub-surface water samples and zinc concentrations. After 48 hours of exposure, developing larvae were fixed in 5% buffered formalin. Approximately 100 larvae in each container were examined under an inverted light microscope at 100x to determine the proportion of veliger larvae with normal shells as described in Anderson et al. (1990).

Percent normal development was calculated as:

$$\frac{\text{(Number of normally developed larvae)} \times 100}{\text{Total number of observed larvae}}$$

Amphipod Tests

Solid-phase sediment sample toxicity was assessed using the 10-day amphipod survival toxicity test protocol for *Rhepoxynius abronius* (ASTM 1993).

All test organisms were obtained from Northwest Aquatic Sciences in Yaquina Bay, Oregon. Amphipods were separated into groups of approximately 100 each, placed in polyethylene boxes containing

Yaquina Bay collection site sediment, and then shipped on ice via overnight courier. Upon arrival at Granite Canyon, the amphipods were acclimated slowly (<2 ppt per day) to 28 ppt sea water ($T = 15^0C$). Once acclimated to 28 ppt, the animals were held for an additional 48 hours prior to inoculation into the test containers.

Test containers were one liter glass beakers or jars containing two cm of sediment and filled to the 700 ml line with seawater adjusted to 28 ppt using spring water or distilled well water. Test sediments were not sieved for indigenous organisms prior to testing although at the conclusion of the test, the presence of predators was noted and recorded on the data sheet. Test sediment and overlying water were allowed to equilibrate for 24 hours, after which 20 amphipods were placed in each beaker along with 28 ppt seawater to fill test containers to the one liter line. Test chambers were aerated gently and illuminated continuously at ambient laboratory light levels.

Five laboratory replicates of each sample were tested for ten days. A negative sediment control consisting of five lab replicates of Yaquina Bay home sediment was included with each sediment test. After ten days, the sediments were sieved through a 0.5 mm Nytex screen to recover the test animals, and the number of survivors was recorded for each replicate.

Positive control reference tests were conducted concurrently with each sediment test using cadmium chloride as a reference toxicant. For these tests, amphipod survival was recorded in three replicates of four cadmium concentrations after a 96 hour water-only exposure. A negative seawater control consisting of one micron-filtered Granite Canyon sea water, diluted to 28 ppt was compared to all cadmium concentrations.

Amphipod survival for each replicate was calculated as:

$$\frac{\text{(Number of surviving amphipods) X 100}}{\text{(Initial number of amphipods)}}$$

Polychaete Tests

A subset of sediment samples was tested using *Neanthes arenaceodentata*. The protocol follows procedures described by Johns *et al.* (1990). Newly emergent juvenile *Neanthes* (2 to 3 weeks old) were obtained from Dr. Donald Reish in Long Beach, California. Worms were shipped in seawater in plastic bags at ambient temperature via overnight mail. Upon arrival at MPSL, worms were allowed to acclimate gradually to 28 ppt with <2 ppt daily incremental salinity adjustments. Once acclimated, the worms were maintained for at least 48 hours, and no longer than 10 days, before the start of a test.

The test setup was similar to the amphipod test. Test containers were one liter glass beakers or jars, each containing 2 cm of sediment and filled to the 700 ml line with 28 ppt seawater. Seawater was adjusted to the appropriate salinity using spring

water or distilled well water. After test sediment and overlying water were allowed to equilibrate for 24 hours, 5 worms were placed in each of 5 replicate beakers per sample, and 28 ppt seawater was added up to the one liter line. Test chambers were aerated and illuminated continuously during the 20-day test period. Worms were fed TetraMin® every 2 days, and water was renewed every 3 days. At the end of 20 days, samples were sieved through 0.5mm Nitex® screens, and the number of surviving worms recorded. Surviving worms were placed in pre-weighed foil in a drying oven until they reached a constant weight. Worms were weighed to the nearest 0.1mg.

Worm survival for each replicate was calculated as:

$$\frac{\text{(Number of surviving worms)} \times 100}{\text{Initial number of worms}}$$

Mean weight/worm for each replicate was calculated as:

$$\frac{\text{(Total weight) - (foil weight)}}{\text{Number of surviving worms}}$$

Positive control reference tests were conducted using cadmium chloride as a reference toxicant. Worm survival for 10 worms was recorded in three replicates of four cadmium concentrations in seawater after 96 hours of exposure. A negative seawater control consisting of one micron-filtered Granite Canyon seawater was compared to all cadmium concentrations. A negative sediment control consisting of Yaquina Bay amphipod home sediment was also included in each test.

Mussel Development Test

The bay mussel (*Mytilus edulis*) larval development test was conducted on pore water and sub-surface water samples for which salinity was in the range of 0-26 parts per thousand (ppt). Details of the test protocol are given in ASTM (1992). A brief description of the method follows.

Mussels were shipped via overnight courier and held at MPSL at ambient temperature (11-13°C) and salinity (32-34 ppt) until testing. On the day of a test, adult mussels were transferred to 25°C water to induce spawning through heat stress. Sperm and eggs were mixed in 25 ppt water to give a final sperm-to-egg ratio of 15 to 1. After approximately 20 minutes, fertilized eggs were rinsed on a 25 μm screen to remove excess sperm. Embryos were distributed to the test containers after approximately 90% of the embryos exhibited first cell cleavage (approximately 1 hour).

Test containers were polyethylene-capped, sea water-leached, 20 ml glass scintillation vials containing 10 mls of test solution. Each test container was inoculated with approximately 250 embryos (25/ml). Pore water samples were tested at 25 ± 2 ppt. Low salinity samples were adjusted to 25 ppt using frozen seawater brine. Controls consisted of one micron-filtered Granite Canyon sea water adjusted to 25 ppt, and a separate brine control consisting of sea water brine adjusted to 25 ppt with distilled water. A positive control reference test was conducted concurrently with each test using a dilution series of cadmium chloride as a reference toxicant.
After a 48-hour exposure period, larvae were fixed in 5% buffered formalin. All larvae in each container were examined under an inverted light microscope at 100x to determine the proportion of normally developed larvae as described in ASTM (1992). The percentage normally developed larvae was calculated as:

$$\frac{\text{Observed number of live normal larvae} \times 100}{\text{Mean number of live embryos inoculated at start of test}}$$

Statistical Analysis of Toxicity Test Data
A total of three hundred fifty solid-phase sediment samples were tested for toxicity to amphipods (*Rhepoxynius abronius)* as part of this study. A subset of 154 samples of solid-phase sediment samples were tested with the polychaete *Neanthes arenaceodentata*. Two hundred twenty-five pore water samples were tested using the purple sea urchin *(Strongylocentrotus purpuratus*) fertilization test; 196 samples were tested using the sea urchin larval development test; and 65 subsurface water (water column) samples were tested with the red abalone *(Haliotis rufescens)* larval development test. The bivalve mollusc *(Mytilus edulis)* larval development test was used to test eight sub-surface water and three pore water samples that had salinities below the threshold (26 ppt) selected for use of the sea urchin test.

There were three primary objectives for the toxicity testing portion of this study:
(1) Investigate the areal extent of toxicity in the San Diego Bay region by estimating the percent area considered toxic, based on toxicity test data for each individual protocol; (2) Identify those sites which were most toxic to assist in prioritization and designation of "toxic hot spots"; and (3) Evaluate the performance of each toxicity test protocol.

The first objective (investigating the spatial extent of toxicity) was primarily for use of the National Oceanic and Atmospheric Administration (NOAA)- National Status and Trends Program. The second objective (identifying and prioritizing individual sites as "toxic hot spots") was primarily for the California State Water Resources Control Board.

The different objectives required different sampling designs and different statistical approaches. The first objective, determination of the areal extent of toxicity, was accomplished through a process this report will refer to as the "EMAP approach": statistical procedures that compared samples from

randomly selected stations against the test controls. In this approach, classification of a particular test sample as "toxic" was determined by a two step statistical approach comparing test samples to laboratory controls, as described below.

To accomplish the second objective, distinguishing the most toxic stations in the region to assist in the designation and prioritization of "toxic hot spots", a relatively new statistical method was employed, termed the "reference envelope approach". This approach compared organism response (e.g. % survival) from an individual test sample with mean organism response from a group of reference sites presumed to represent optimal ambient conditions in the San Diego Bay region. Optimal ambient conditions are defined as indicative of conditions that can be found within the study area at sites that have relatively low pollutant concentrations and relatively undisturbed benthic communities. This method was intended to refine the definition of sample toxicity in order to identify a subset of toxic sites that were of greatest concern. This method is also described in detail below.

It should be noted that the EMAP approach and the reference envelope approach are distinctly different, yet complementary, statistical methods for determining toxicity. The intent of using two approaches is to identify non-toxic, significantly toxic and highly toxic locations based on multiple analyses of the data, for ranking toxicity results in a tiered approach.

EMAP Approach for Determining Spatial Extent of Toxicity

The "San Diego Bay Region" incorporates three non-connecting water bodies: San Diego Bay, Mission Bay and Tijuana Slough. Ideally these water bodies should be treated as discrete areas and analyzed separately to determine percent area toxic for each. However, the number of samples from Mission Bay and Tijuana Slough were 13 and 6, respectively, and these were considered too few to accurately represent toxicity in a frequency distribution.

Consequently, data from all three water bodies were combined in this report to determine the percentage of total area that was toxic.

In this analysis, sample toxicity was determined using procedures described by Schimmel *et al.* (1991); a method used in the EPA Environmental Monitoring Assessment Program (EMAP) and in similar NOAA studies nationwide (e.g., Long *et al.*, 1994). Using the EMAP approach, samples were defined as toxic if the following two criteria were met: (1) there was a significant difference in mean organism response (e.g. percent survival) between a sample and the control as determined using a t-test, and (2) mean organism response in the toxicity test was less than 80% of the laboratory control value. The t-test generates a t statistic by dividing the difference between control and test sample response by an expression of the variance between laboratory replicates. If the variation between control and test sample is sufficiently greater than the variation among laboratory replicates, the t-test

indicates a significant difference in response. A "separate variance" t-test was used to adjust the degrees of freedom to account for variance heterogeneity among samples (SYSTAT, 1992).

The second criterion, that sample response must be less than 80% of the control value to be considered toxic, is useful in eliminating those samples that were statistically different from controls only because of a very small variance among laboratory replicates. For example, a sample that had 90 ± 2 % *Rhepoxynius* survival would be significantly different from a control with survival of 96 ± 2 %, and would therefore be considered toxic based on a simple t-test even though the biological significance of this response would be negligible. By adding the second criterion, any sample with percent survival exceeding 80% of the controls would be considered non-toxic. The 80% level was established by examination of numerous amphipod toxicity data sets (Thursby and Schlekat, 1993). These researchers found that samples with survival less than 80% relative to controls were significantly different from controls about 90% of the time. Preliminary analyses of *Rhepoxynius* test data from the BPTCP indicate a similar level of statistical sensitivity. Based on this observation, the 80% criterion has been adopted previously (Schimmel *et al.*, 1991; USEPA/USACOE, 1991). Samples identified as toxic according to these criteria were used to estimate the percent of total area toxic within the San Diego Bay region.

Using Cumulative Distribution Frequencies to Characterize Spatial Extent

The stratified random sampling design, allowed 121 of the total 350 samples collected in this study, to be used to estimate the areal extent of toxicity. Samples collected using directed sampling (non-random sampling directed to areas of particular characteristics) were not included in this analysis since they may have been biased toward increased contamination. Directed non-random sampling was designed to address the State and Regional Water Quality Boards objective to identify and prioritize potential toxic hot spots. Samples were collected from randomly selected stations within 95 non-overlapping mapped blocks of known area in the San Diego Bay region (Figure 2). Total area sampled, calculated as the sum of all 95 block areas, was 40.9 km^2. The estimate of spatial toxicity was determined from cumulative distribution frequencies (CDFs) that relate toxicity response to percent of total sampled area. CDF calculations follow procedures used by both EMAP and NS&T.

CDFs were determined using calculated areas of each block normalized to the number of samples per block. Block areas were calculated using a planimeter on NOAA National Ocean Service navigation chart (means of three trials), calibrated to the scale of the charts. Because no more than two samples were collected per block, numbers of toxic samples per block ranged from 0 to 2, representing 0%, 50% or 100% of a given block area. By combining the blocks with their toxicity designations in a cumulative manner, the CDFs indicate the percentage of total area sampled that was toxic. Sample toxicity was determined from comparisons

with laboratory controls as described above in the EMAP approach; each sample with a mean significantly different from, and less than 80% of, the laboratory control mean was considered toxic. Calculations used to derive percent areas determined to be toxic are shown on worksheets in Appendix F. CDFs were generated from toxicity tests using *Rhepoxynius* survival (solid phase) and *Strongylocentrotus* larval development (pore water). There were insufficient data from randomly selected sites to generate CDFs for *Haliotis*, *Mytilus* and *Neanthes* tests.

The Reference Envelope Approach for Determining Toxicity

The second objective of this study was to assist in the identification of "toxic hotspots", where adverse biological impacts are observed in areas with localized concentrations of pollutants. Identification of problem sites was an essential step in prioritizing efforts to improve sediment and water quality through regulation and remediation programs. While it was possible large areas of San Diego Bay may be degraded to some extent, logistical constraints required efforts be focused on localized areas that were significantly more toxic than optimal ambient conditions that exist in the greater portion of the bay. In this study, a "reference envelope" statistical approach was employed (Smith,1995) to identify samples that exhibit significantly greater toxicity than expected in San Diego Bay as a whole.

The reference envelope approach uses data from "reference sites" to characterize the response expected from sites in the absence of localized pollution. Using data from the reference site population, a tolerance limit was calculated for comparison with data from test sites. Samples with toxicity values greater than the tolerance limit were considered toxic relative to the optimal ambient condition of the Bay.

This relative standard established using reference sites was conceptually different from what might be termed the absolute standard of test organism response in laboratory controls. Rather than comparing sample data to control data using t-tests, with laboratory replication used to characterize the variance component (as in the "EMAP approach" described above), the reference envelope approach compared sample data against a percentile of the reference population of data values, using variation among reference sites as the variance component. The reference envelope variance component, therefore, included variation among laboratory replicates, among field replicates, among sites, and among sampling events.

The reference stations were assumed to be a random sample from an underlying population of reference locations that serve as a standard for what we considered relatively non-impacted conditions. The toxicity measured at different reference locations will vary due to the different local conditions that can affect the toxicity results. In order to determine whether sediments from a test location were toxic, bioassay results for the test location were compared with bioassay results from the population of reference locations.

Assuming the bioassay results from the population of reference locations are normally distributed, an estimate of the probability that the test sediment is from the underlying reference station distribution can be made. For example, if the result for a test sediment was at the first percentile of the underlying reference location distribution (in the direction of toxicity), then there would be about a 1% chance that the test sediment was from the distribution of reference locations. The toxicity level at the first percentile of the reference distribution is not known because there were only limited samples from the underlying distribution and only an estimate could be made of where the first percentile lies. If an estimate of the first percentile value was made a large number of times, using different random samples from the reference distribution, a (non-central t) distribution of estimates, with the distribution mode at the actual first percentile would be obtained (Figure 4). In Figure 4, it can be seen from the distribution of estimates that about one half of the time the estimate from the sample was above the actual first percentile. Ideally, identification of an estimated toxicity value would cover the actual first percentile for a large percentage of the estimates (say 95% of the time). Such a value can be obtained from the left tail of the distribution of estimates where 5% of the estimates are less than the chosen value. The definition of p is the percentile of interest, and alpha is the acceptable error probability associated with an estimate of the pth percentile. Thus, in this example, p=1 and alpha = .05.

The toxicity level can be computed that will cover the pth percentile 1 minus alpha proportion of the time as the lower bound (L) of a tolerance interval (Vardeman 1992) as follows.

$$L = X_r - [\, g_{a,p,n} * S_r \,]$$

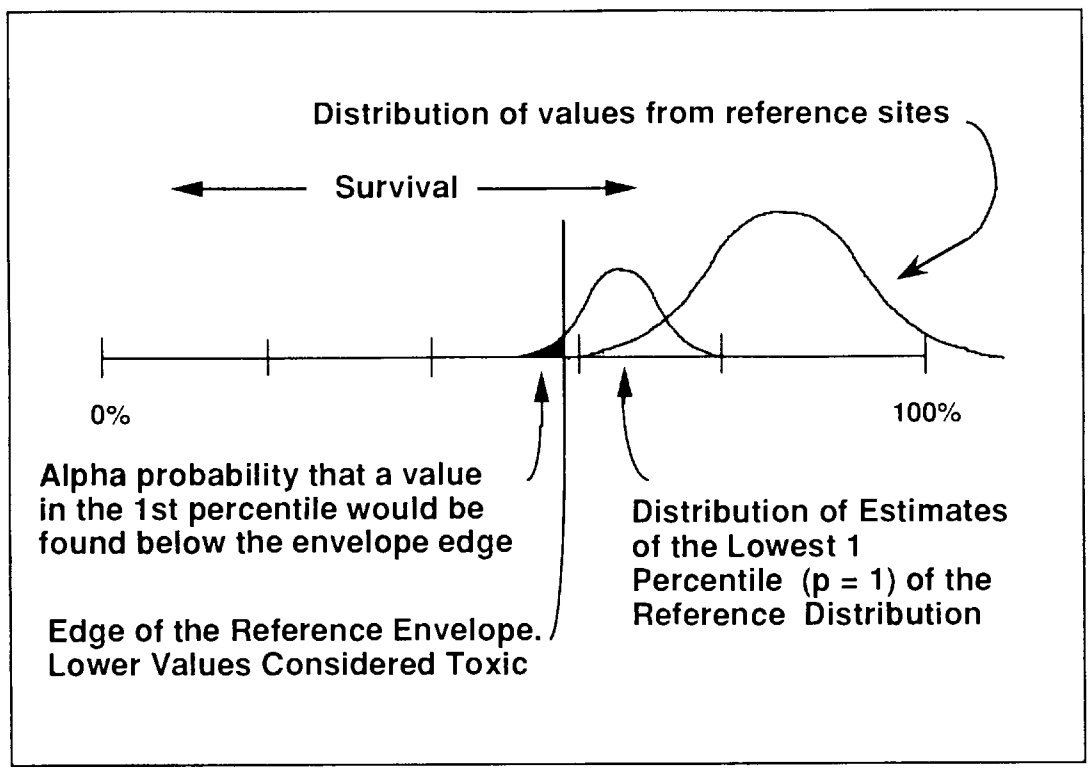
where X_r is the mean of the sample of reference stations, S_r is the standard deviation of the toxicity results among the reference stations, and n is the number of reference stations. The g values, for the given alpha, p, and n values, can be obtained from tables in Hahn and Meeker (1991) or Gilbert (1987). S contains the within- and between-location variability expected among reference locations. If the reference stations are sampled at different times, then S will also incorporate between-time variability. The "edge of the reference envelope" (L) represents a cutoff toxicity level used to distinguish toxic from non-toxic sediments. The value used for p will depend on the level of certainty needed for a particular regulatory situation. In this study a p value equal to 1% was chosen, to distinguish only the most toxic samples, that is, samples having a 95% certainty of being in the most toxic 1%.

Reference Station Selection for Reference Envelope

Reference stations were selected to represent optimal ambient conditions available in San Diego Bay, based on available chemistry and benthic community data. Toxicity data were not

used in the selection process. Stations were selected if both of the following criteria were met: 1) the benthic communities appeared relatively undisturbed (based on indices described in the benthic community analysis section), and 2) sediment chemical concentrations were below Effects Range Median (ERM) levels (Long *et al.*, 1995) and Probable Effects levels (PELs) (McDonald, 1994). Among all stations, both randomly and non-randomly

Figure 4. Schematic illustration of the method for determining the lower tolerance interval bound (edge of the reference envelope) to determine sample toxicity relative to a percentile of the reference site distribution.



selected, a total of 75 samples were analyzed for toxicity, chemistry and benthic ecology in this study. After screening these 75 samples, eleven stations in the San Diego Bay region were selected as reference stations (Table 4). It should be noted these stations were not selected prior to the initiation of the study, but were selected after all of the analyses for the study were completed.

P450 Reporter Gene System

Summary of Methods

A subset of thirty sediment samples was sent to Columbia Analytical Services (CAS) in Kelso, Washington for extraction with methylene chloride. Extracts of 20 g sediment samples were evaporated to 1 ml and placed in small vials for shipment to the Carlsbad, CA laboratory of CAS where 2 μl samples were applied in triplicate to genetically engineered human liver cancer cells (101L cells) developed by Dr. Robert Tukey of the University of California, at San Diego. A previous study partially funded by the State Board (Anderson *et al.*, 1995) had demonstrated that low levels of dioxin, coplanar PCBs and selected PAHs could be detected by the P450-RGS response to the extracts. When this small volume of solvent (with extracted contaminants) is applied to approximately one million cells in 2 ml of medium, induction of the CYP1A1 gene leads to production of the detoxification enzyme, P450, and the luminescent enzyme, luciferase. When the cells are lysed (after 16 hours) and the centrifugate tested with luciferin, the amount of light measured in a luminometer is a function of the concentration and potency of the contaminants on the sediments. When the contents of a single well (containing ≈ one million cells) are centrifuged and placed in the luminometer the resulting measure is in Relative Light Units (RLU). The RLUs of the solvent blank are set to unity and by dividing all RLU readings for the reference toxicant and samples by the RLUs of the blank, the data are converted to Fold Induction (or times background). To make the data more relevant to environmental samples, the data are converted to Equivalents of Benzo(a)pyrene (BaPEq), a ubiquitous PAH compound of environmental concern (U.S. EPA, 1995). To convert mean fold induction to BaPEq in μg/g dry weight, the fold induction values are divided by sixty, which (based on a dose response curve) is the response of the assay to 1μg/ml of Benzo(a)pyrene (BaP). The μg of BaP per volume of extract (e.g. 10 μl) is adjusted to an initial volume of 1 ml and this product divided by the dry grams of sample contained in the 1 ml extract. This method can be used to calculate Equivilants for PAHs, from benz(a)anthracene to benzo(g,h,i)perylene (Table 4), as well as dioxins/furans and coplanar PCBs. Both sediments and tissues (marine mussel) from San Diego Bay have been analyzed for the presence of P450 inducing compounds in previous studies (Anderson *et al.* 1996, in press a). The detailed methods and results of P450-RGS testing with standards and sediment extracts are described in Postlind *et al.* (1994), and Anderson *et al.* (1995). In 1996, three publications will be available describing the specific test methods (ASTM, Standard Methods,and CRC Press).

TABLE 4
REFERENCE STATIONS SELECTED FOR REFERENCE ENVELOPE ANALYSIS

Station #	Station Name	IDORG #	Leg	% Fines	TOC	ERMQ	PELQ	BENTHICS	Amphipod Surv.	Urchin Devo.(25%)
93112.0	MISSION BAY A8 (x1)-REP 1	856	21	30.12	0.81	0.065	0.116	UNDEGRADED	96 ± 5	20.2 ± 1
93112.0	MISSION BAY A8 (x1)-REP 2	857	21	37.28	0.94	0.082	0.134	UNDEGRADED	98 ± 3	89 ± 4
93112.0	MISSION BAY A8 (x1)-REP 3	858	21	43.56	0.91	0.089	0.145	UNDEGRADED	94 ± 5	53.6 ± 49
93202.0	EAST BASIN I1 (x5)	842	21	46.28	1.11	0.238	0.362	UNDEGRADED	83 ± 6	67.2 ± 17
90013.0	37 SWARTZ (MARINA)	815	20	88.21	1.37	0.217	0.347	UNDEGRADED	81 ± 8	73.8 ± 10
93190.0	MARINA II1 (x1)	816	20	93.97	1.22	0.219	0.356	UNDEGRADED	87 ± 12	59.4 ± 9
90053.0	35 SWARTZ (CORONADO CAYS)	843	21	91.85	1.47	0.180	0.292	UNDEGRADED	75 ± 11	29 ± 25
93108.0	MISSION BAY A4 (x1)-REP 2	860	21	64.60	1.87	0.104	0.166	UNDEGRADED	69 ± 14	78.5 ± 16
93195.0	GLORIETTA BAY U1 (x2)	823	20	48.24	0.95	0.239	0.369	UNDEGRADED	81 ± 9	0 ± 0
93194.0	GLORIETTA BAY U1 (x1)	822	20	55.80	1.14	0.232	0.371	UNDEGRADED	89 ± 7	46.3 ± 7
93231.0	CARRIER BASE V2 (x6)	1000	23	57.66	1.57	0.252	0.404	UNDEGRADED	74 ± 12	0 ± 0

None of the above samples exhibited any chemical exceedance of an ERM or PEL.
None of the above samples exhibited elevated ammonia or hydrogen sulfide during toxicity testing.
Amphipod Survival value is the mean and standard deviation from 5 laboratory replicates.
Urchin Development values are the mean and standard deviation of 5 replicates in 25% porewater.
ERM and PEL summary quotients are discussed in Appendix B and the report text.

Quality Assurance/Quality Control

Summary of Methods

Summaries of quality assurance and quality control procedures are described under separate cover in the Bay Protection and Toxic Cleanup Program Quality Assurance Project Plan (QAPP). This document describes procedures within the program which ensure data quality and integrity. Quality assurance procedures follow those of the NS&T Program to ensure comparability with other NOAA survey areas nationwide. In addition, individual laboratories prepare quality assurance evaluations of each discrete set of samples analyzed and authorized by task order. These documents were submitted to the California Department of Fish and Game for review, then forwarded to the State Water Resources Control Board for further review.

RESULTS

Tabulated data for all chemical, benthic, toxicological and P450-RGS analyses are presented in Appendices B, C, D and E. The summary data presented in the following results sections were used to demonstrate significant findings from the analysis of the full data set in Appendices B, C, and D.

Distribution of Chemical Pollutants

Chemical Specific Screening Values

There have been several recent studies associating pollutant concentrations with biological responses (Long and Morgan, 1990; MacDonald, 1992). These studies provide guidance for evaluating the degree to which sediment chemical pollutants levels are responsible for effects observed in a toxicity test. Reported values are based on individual chemical pollutants within sediments. Therefore, their application may be confounded when dealing with: biological effects which could be attributed to a synergistic effect of low levels of multiple chemicals, unrecognized chemicals, or physical parameters in the sediment which were not measured.

The National Status and Trends Program has used chemical and toxicological evidence from a number of modeling, field and laboratory studies to determine the ranges of chemical concentrations which are rarely, sometimes, or usually associated with toxicity (Long and Morgan, 1992). Evaluation of available data (Long *et al.*, 1995) has led to identification of three ranges in concentration for each chemical:

1) Minimal Effects Range: The range in concentration over which toxic effects are rarely observed:

2) Possible Effects Range: The range in concentrations over which toxic effects are occasionally observed;

3) Probable-Effects Range: The range in chemical concentrations over which toxic effects are frequently or always observed.

Two slightly different methods were used to determine these chemical ranges. One method developed by NOAA (Long and Morgan, 1990; Long *et al.*, 1995) used chemical data which were associated with a toxic biological effect. These data were used to determine the lower 10th percentile of ranked data where the chemical level was associated with an effect (Effects Range-Low, or ERL). Sediment samples in which all chemical concentrations were below the 25 ERL values were not expected to be toxic. The Effects Range-Median (ERM) reflects the 50th percentile of ranked data and represents the level above which effects are expected to occur. Effects are expected to occur occasionally when chemical concentrations fall between the ERL and ERM. The probability of toxicity was expected to increase with the number and degree of exceedances of the ERM values.

Another method identifies three ranges using chemical concentration data associated with both toxic biological effects and no observed effects (MacDonald, 1992; MacDonald, 1994; MacDonald *et al., In Press*). The ranges are identified as TEL (Threshold Effects Level) and the PEL (Probable Effects Level). TEL values were derived by taking the geometric mean of the 50th percentile of the "no effects" data and the 15th percentile of the "effects" data. The PEL values were derived by taking the geometric mean of the 85th percentile of the "no effects" data and the 50th percentile of the "effects" data. Although different percentiles were used for these two methods, they are in close agreement, usually within a factor of 2. Values reported for both methods are shown in Table 5. Neither of these methods is advocated over the use of the other in this report. Instead, both are used in the following analysis to create a weight of evidence which should help explain toxicity observed from some sediments.

A cautionary note should be included; the degree of confidence which MacDonald (1994) and Long *et al. (*1995) had in their respective guidelines varied considerably among the different chemicals. For example, they express low confidence in the values derived for nickel, mercury, DDTs, chlordane, dieldrin, and endrin. When more data becomes available regarding these chemicals and their potential effects, the guidelines may be revised, probably upward for some substances.

Primary Chemicals of Concern

Figure 5 presents a summary of the chemicals and chemical groups which exceeded ERM or PEL values at the 217 stations where complete chemical analysis was performed. Copper, mercury, zinc, total chlordane, total PCBs and the PAHs were most often found to exceed ERM or PEL values and are considered the six major chemicals or chemical groups of concern in the San Diego Bay

Table 5- Comparison of Sediment Screening Levels
Developed by NOAA and the State of Florida

SUBSTANCE	State of Florida (1) TEL	State of Florida (1) PEL	NOAA (2) ERL	NOAA (2) ERM
Organics (ug/kg- dry weight)				
Total PCBs	21.550	188.79	22.70	180.0
PAHs				
Acenaphthene	6.710	88.90	16.00	500.0
Acenaphthylene	5.870	127.89	44.00	640.0
Anthracene	46.850	245.00	85.30	1100.0
Fluorene	21.170	144.35	19.00	540.0
2-methylnaphthalene	20.210	201.28	70.00	670.0
Naphthalene	34.570	390.64	160.00	2100.0
Phenanthrene	86.680	543.53	240.00	1500.0
Total LMW-PAHs	311.700	1442.00	552.00	3160.0
Benz(a)anthracene	74.830	692.53	261.00	1600.0
Benzo(a)pyrene	88.810	763.22	430.00	1600.0
Chrysene	107.710	845.98	384.00	2800.0
Dibenz(a,h)anthracene	6.220	134.61	63.40	260.0
Fluoranthene	112.820	1493.54	600.00	5100.0
Pyrene	152.660	1397.60	665.00	2600.0
Total HMW-PAHs	655.340	6676.14	1700.00	9600.0
Total PAHs	1684.060	16770.54	4022.00	44792.0
Pesticides				
p,p'-DDE	2.070	374.17	2.20	27.0
p,p'-DDT	1.190	4.77		
Total DDT	3.890	51.70	1.58	46.1
Lindane	0.320	0.99		
Chlordane	2.260	4.79	0.50	6.0
Dieldrin	0.715	4.30	0.02	8.0
Endrin			0.02	45.0
Metals (mg/kg- dry weight)				
Arsenic	7.240	41.60	8.20	70.0
Antimony			2.00	2.5
Cadmium	0.676	4.21	1.20	9.6
Chromium	52.300	160.40	81.00	370.0
Copper	18.700	108.20	34.00	270.0
Lead	30.240	112.18	46.70	218.0
Mercury	0.130	0.70	0.15	0.7
Nickel	15.900	42.80	20.90	51.6
Silver	0.733	1.77	1.00	3.7
Zinc	124.000	271.00	150.00	410.0

(1) D.D. MacDonald, 1994

(2) Long et al., 1995

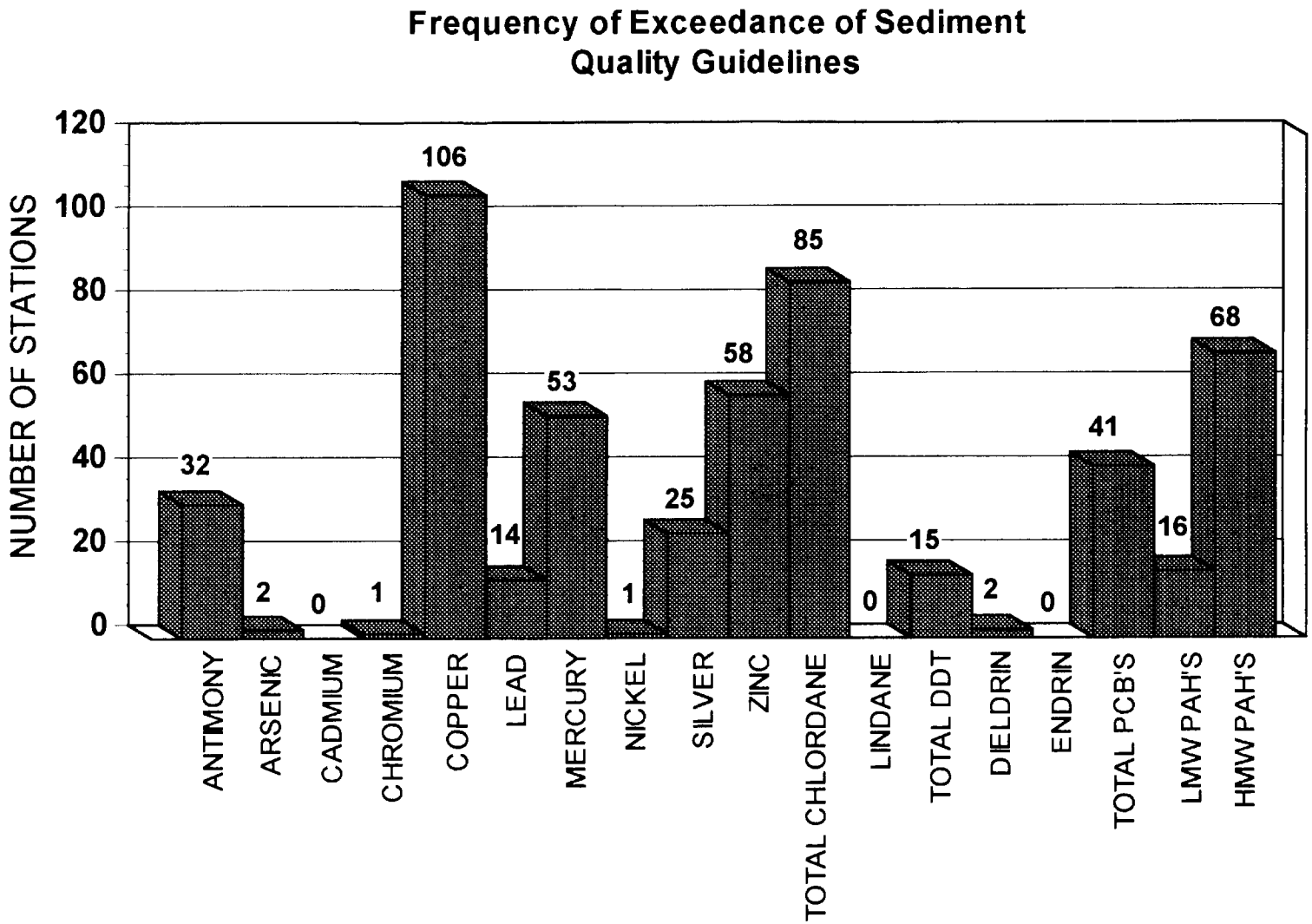


Figure 5. Number of stations which exceeded either the PEL or ERM values.

Region. MacDonald (1994) and Long *et al. (*1995) express relatively high confidence in the ERM and PEL values derived for copper, zinc, total PCBs and PAHs. Figures 6-12 map the geographical distribution of the six chemicals of concern throughout the San Diego Bay Region. Three ranges of chemical concentration are given for each chemical: (1) below the TEL, (2) between the TEL and PEL and (3) above the PEL to the maximum concentration determined.

Copper is a broad spectrum biocide which may be associated with acute and chronic toxicity, reduction in growth, and a wide variety of sublethal effects (Spear and Pierce, 1979). Elevated copper concentrations above the PEL (>108.2 mg/kg) or ERM (>270 mg/kg) were found throughout San Diego Bay (Figure 6(a-d)), with small boat harbors, commercial shipping berths and military berths most often impacted. Considering the historical use of copper based anti-fouling paint in the area, this distribution pattern is expected.

Zinc demonstrates a similar pattern of distribution, although actual exceedances of PEL levels (>271 mg/kg) or ERM levels (>410 mg/kg) only occur in the central portion of the bay, along the naval shipyard waterfront (Figure 7(a-d).

Mercury, particularly methylmercury, is highly toxic to aquatic biota. Although there is variability in sensitivity of different organisms to the substance, bioaccumulation of mercury in aquatic species has significant implications with respect to human health. PEL exceedances (> 0.696 mg/kg) and ERM exceedances (>0.71 mg/kg) of mercury were found in several small boat areas, near commercial shipping operations and predominately near naval shipyard areas (Figure 8(a-d)).

Polycyclic (polynuclear) aromatic hydrocarbons (PAHs) are base/neutral organic compounds with a fused ring structure of two or more benzene rings. They are components of crude and refined petroleum products and are also products of incomplete combustion of organic materials. Exposure to PAHs may result in a wide range of carcinogenic, teratogenic and mutagenic effects to terrestrial and aquatic organisms (Eisler, 1987). Due to their similar modes of toxic action, individual PAHs are often grouped into low and high molecular weight compounds, for concise reporting purposes. Individual PAHs used for the summations of low and high molecular weight PAHs in this report are given in Appendix B -Section VII. PAH pollution, as shown for high molecular weight PAHs in Figure 9(a-d), exceeds the PEL (>6676.14 μg/kg) or ERM (>9600 μg/kg) near commercial shipping operations and naval shipyard areas, as well as the submarine facility near the mouth of the harbor. The pattern for PEL (>1442 μg/kg) or ERM (>3160 μg/kg) exceedances of low molecular weight PAHs is similar to high molecular weight PAHs (Fig. 10(a-d)).

A significant concern is polychlorinated biphenyls (PCBs) levels found in sediments throughout San Diego Bay. PCBs are base/neutral compounds which are formed by direct chlorination of

Figure 6a
Copper Concentrations in Sediment
North San Diego Bay

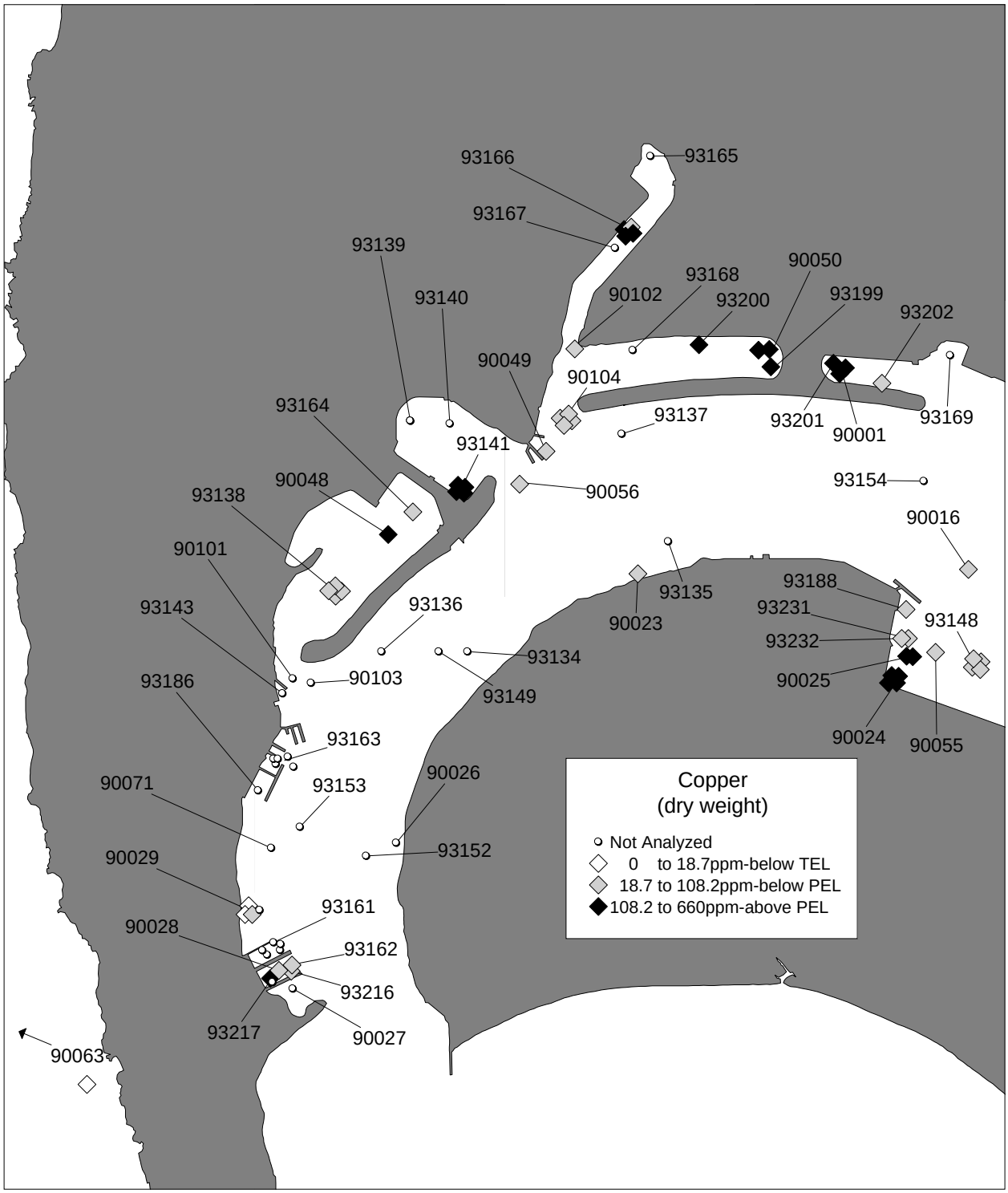


Figure 6b
Copper Concentrations in Sediment
Mid San Diego Bay

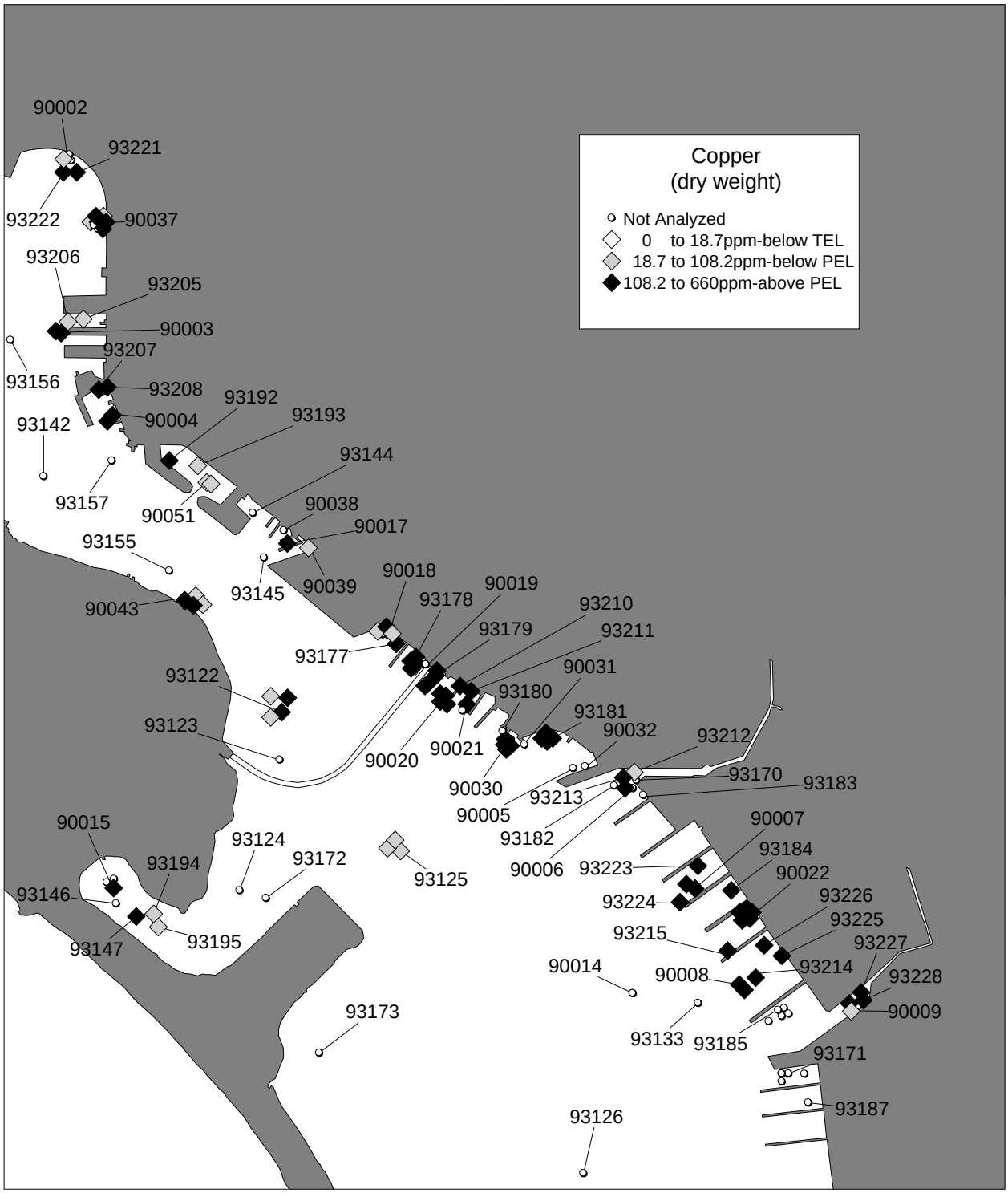


Figure 6c
Copper Concentrations in Sediment
South San Diego Bay

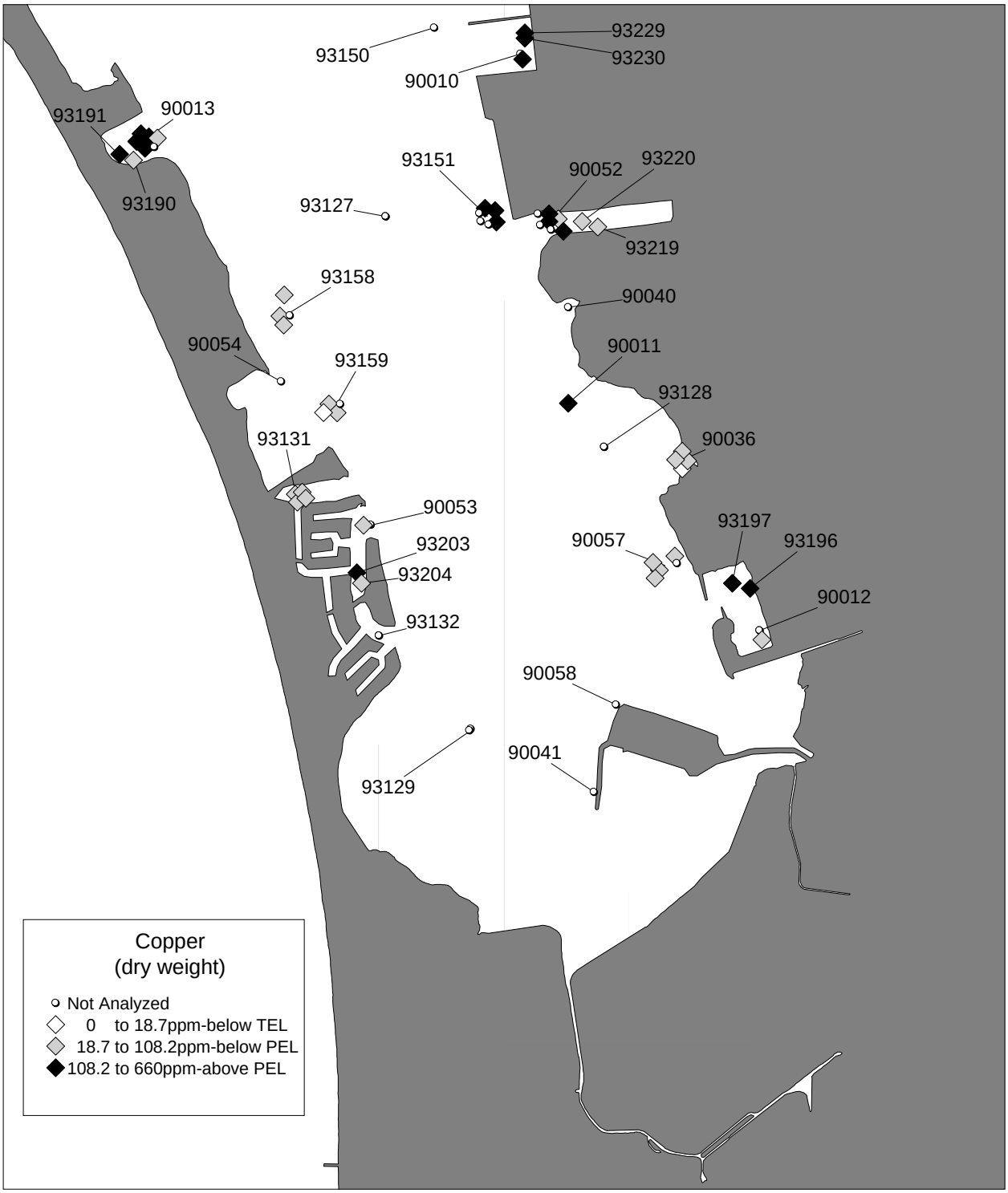
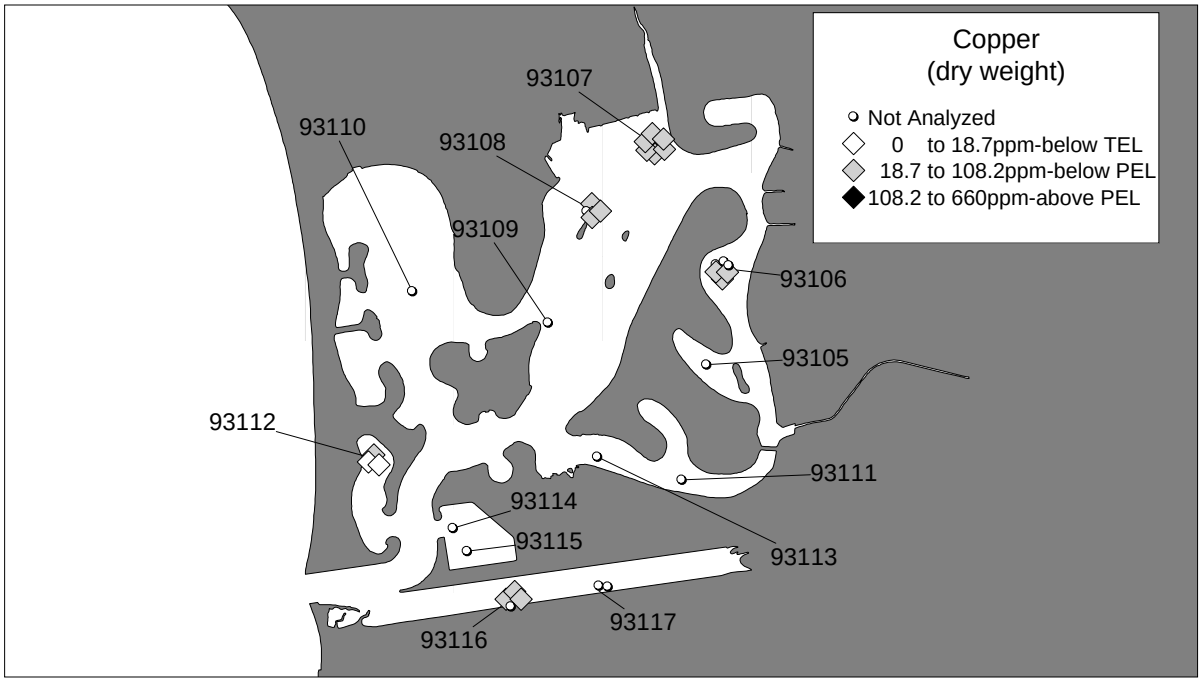


Figure 6d
Copper Concentrations in Sediment
Mission Bay and San Diego River Estuary



Tijuana River Estuary

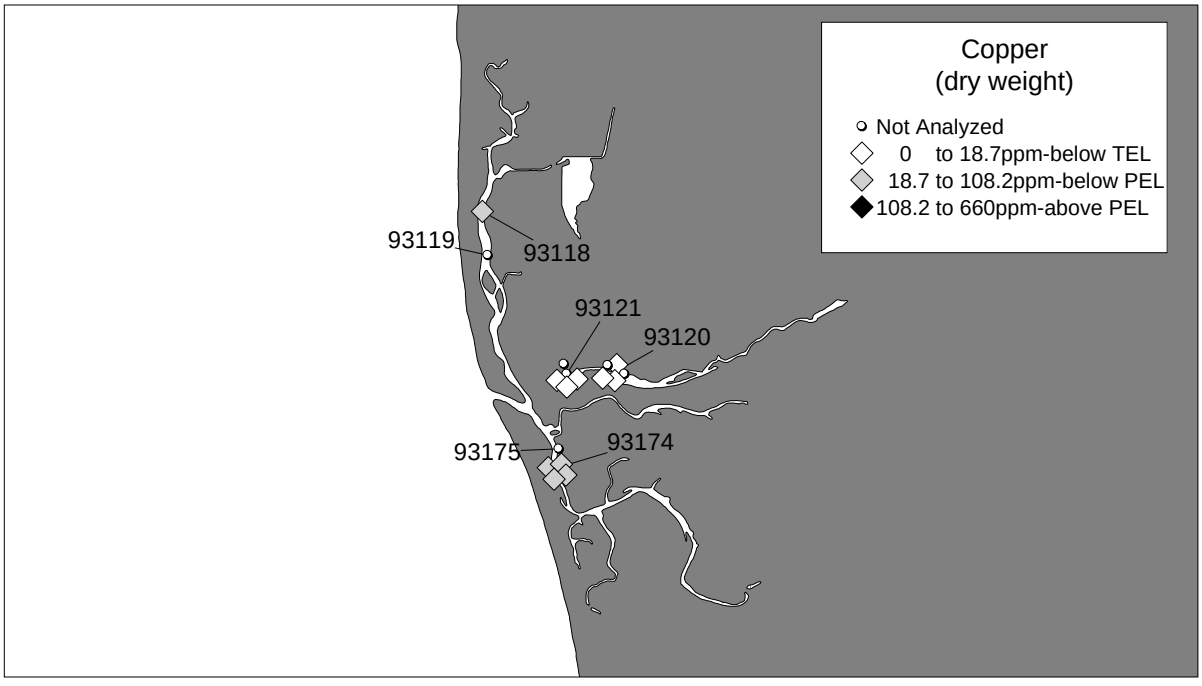


Figure 7a
Zinc Concentrations in Sediment
North San Diego Bay

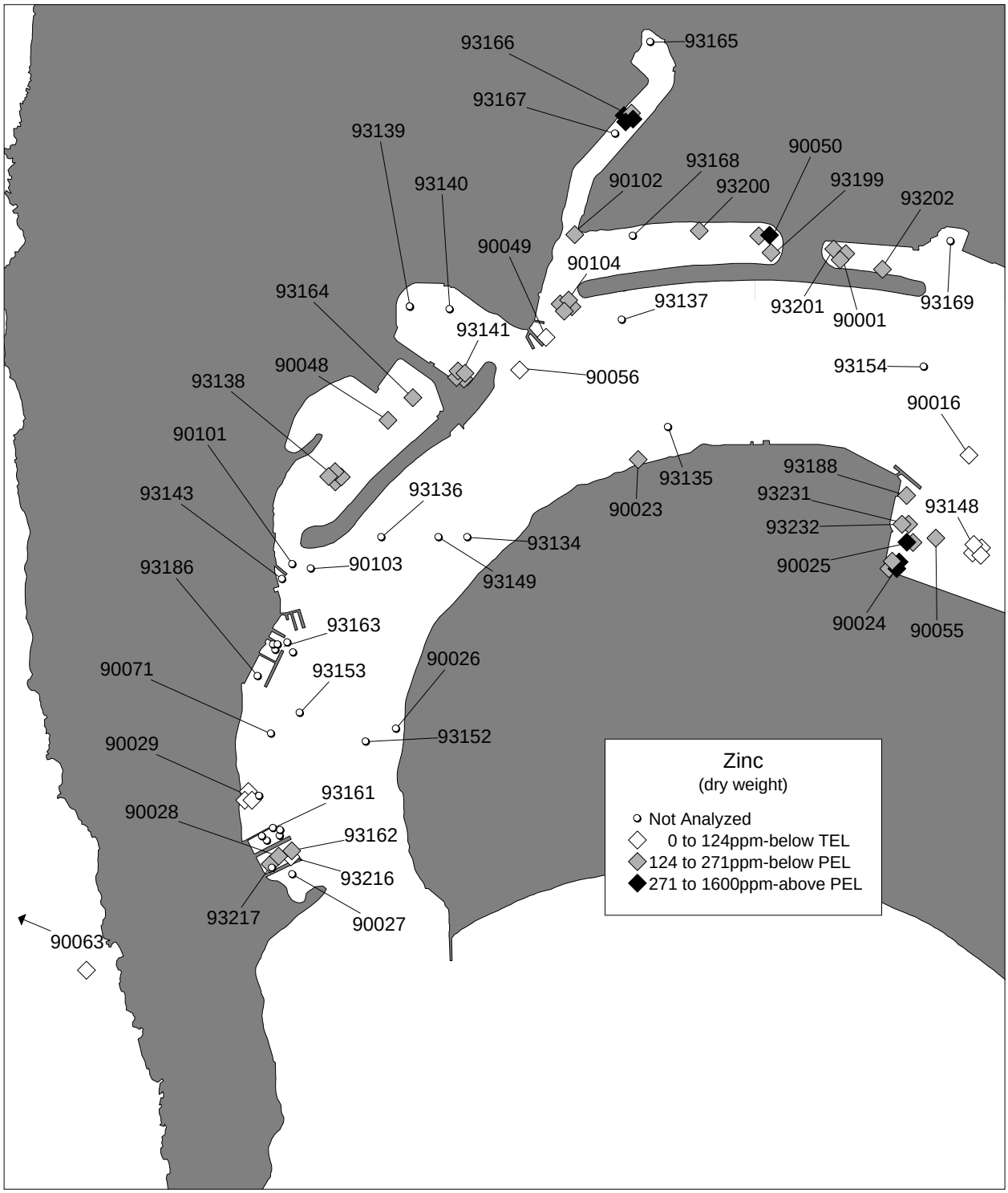


Figure 7b
Zinc Concentrations in Sediment
Mid San Diego Bay

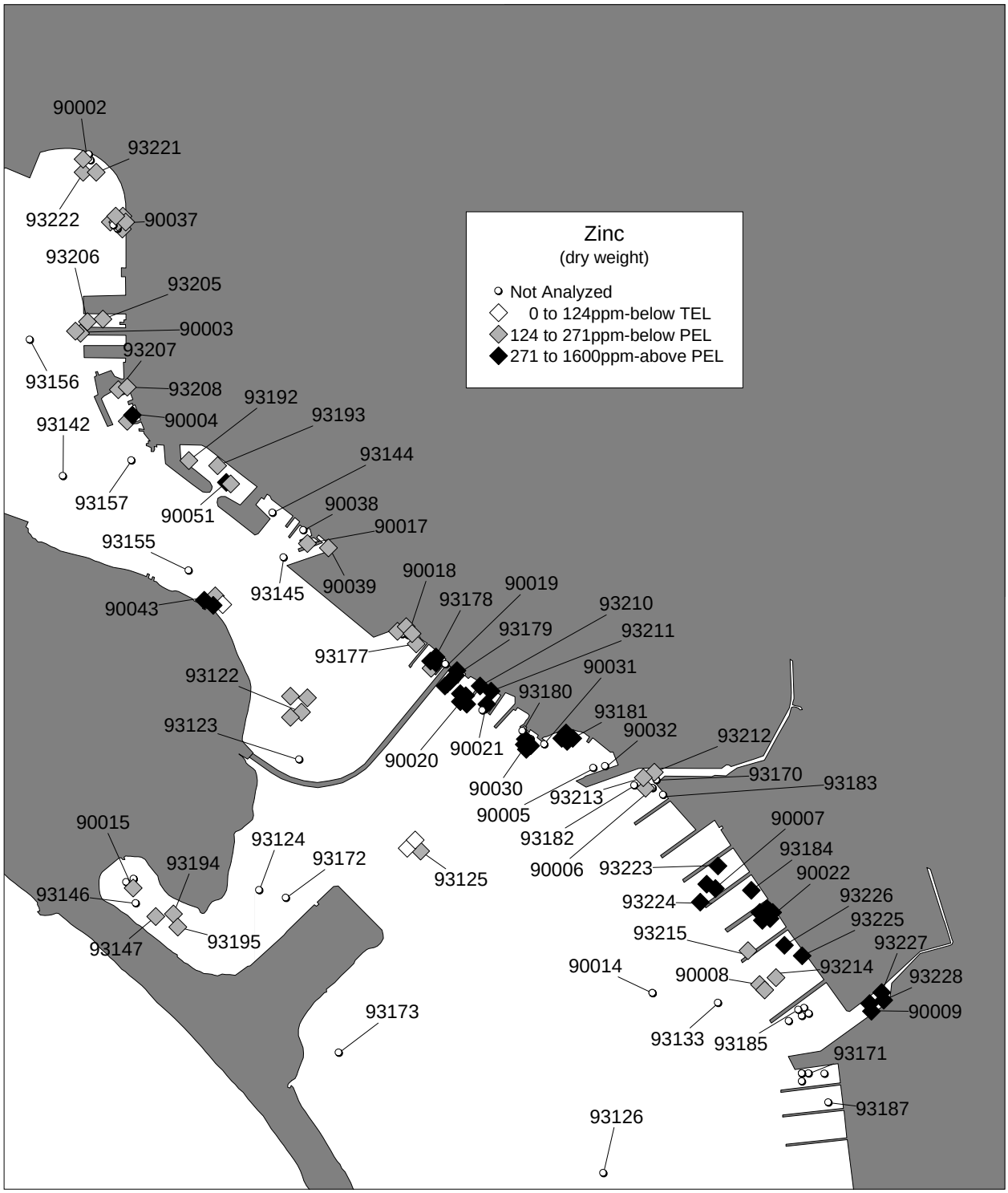


Figure 7c
Zinc Concentrations in Sediment
South San Diego Bay

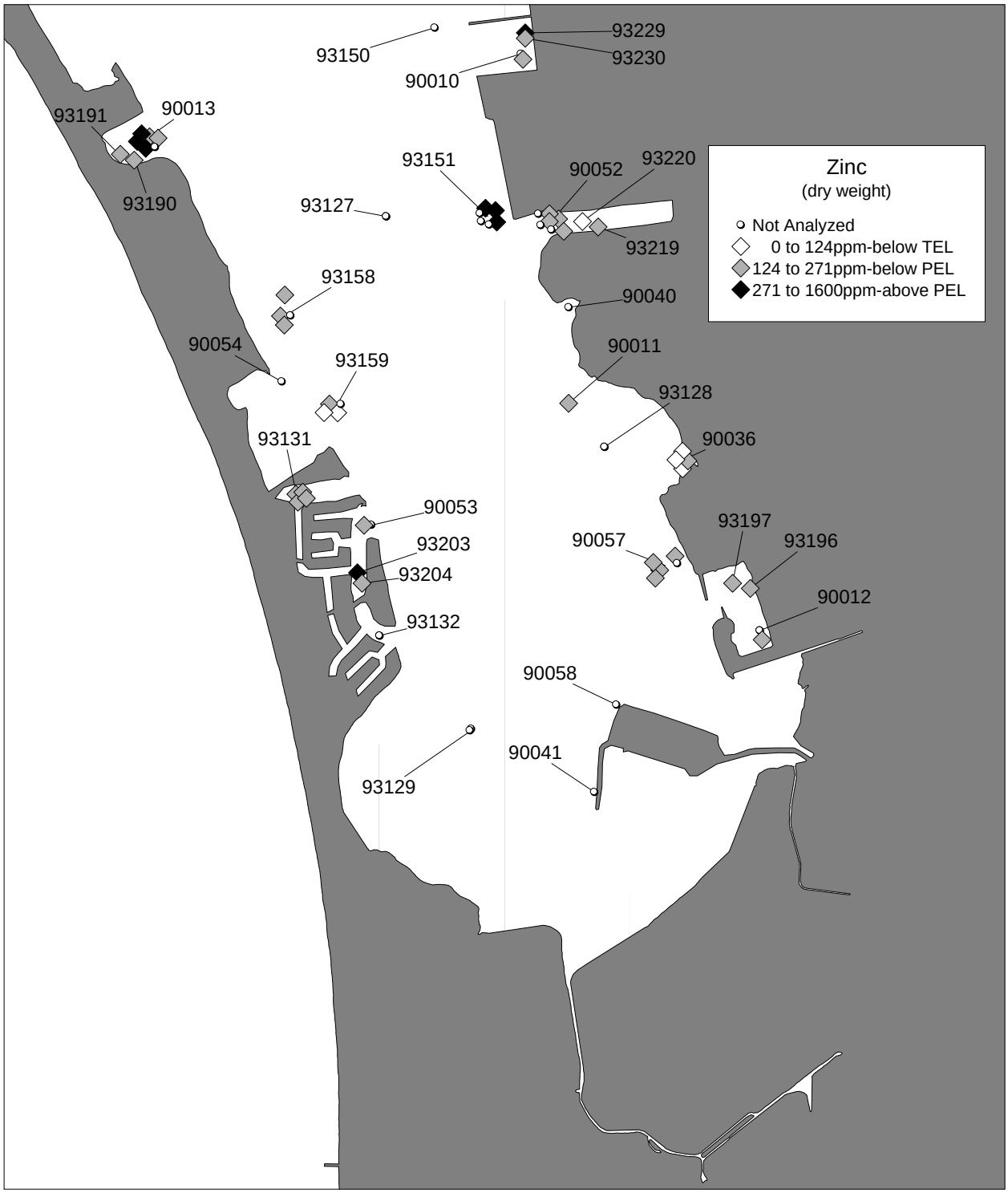
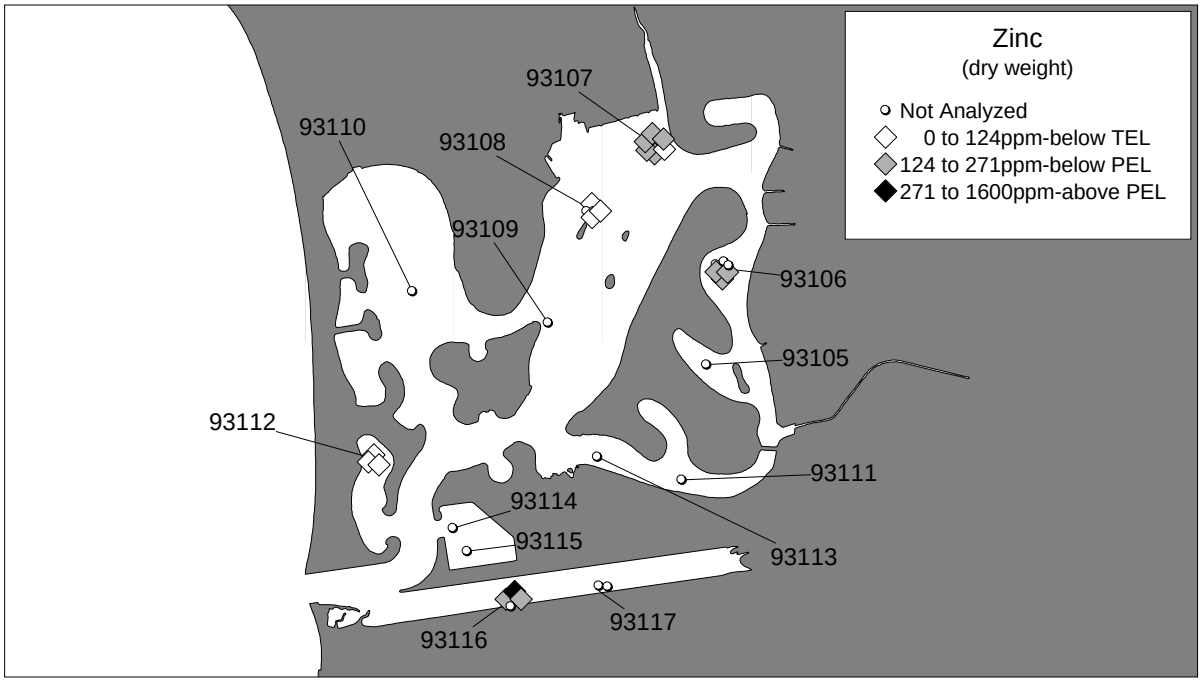


Figure 7d
Zinc Concentrations in Sediment
Mission Bay & San Diego River Estuary



Tijuana River Estuary

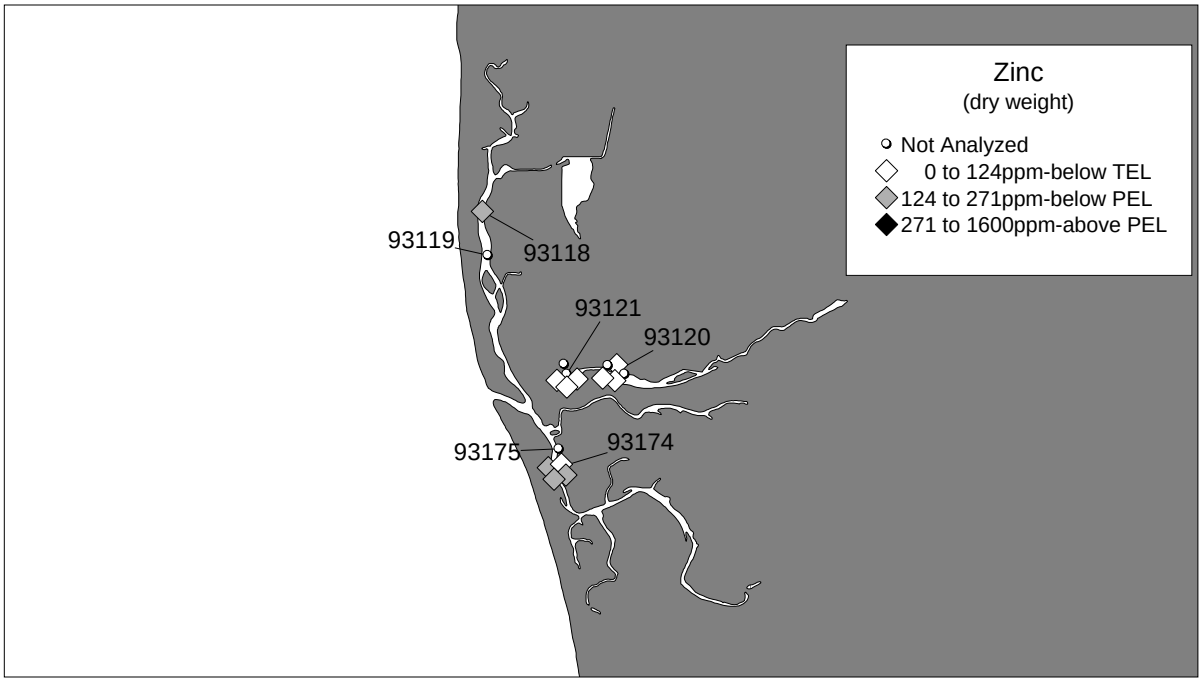


Figure 8a
Mercury Concentrations in Sediment
North San Diego Bay

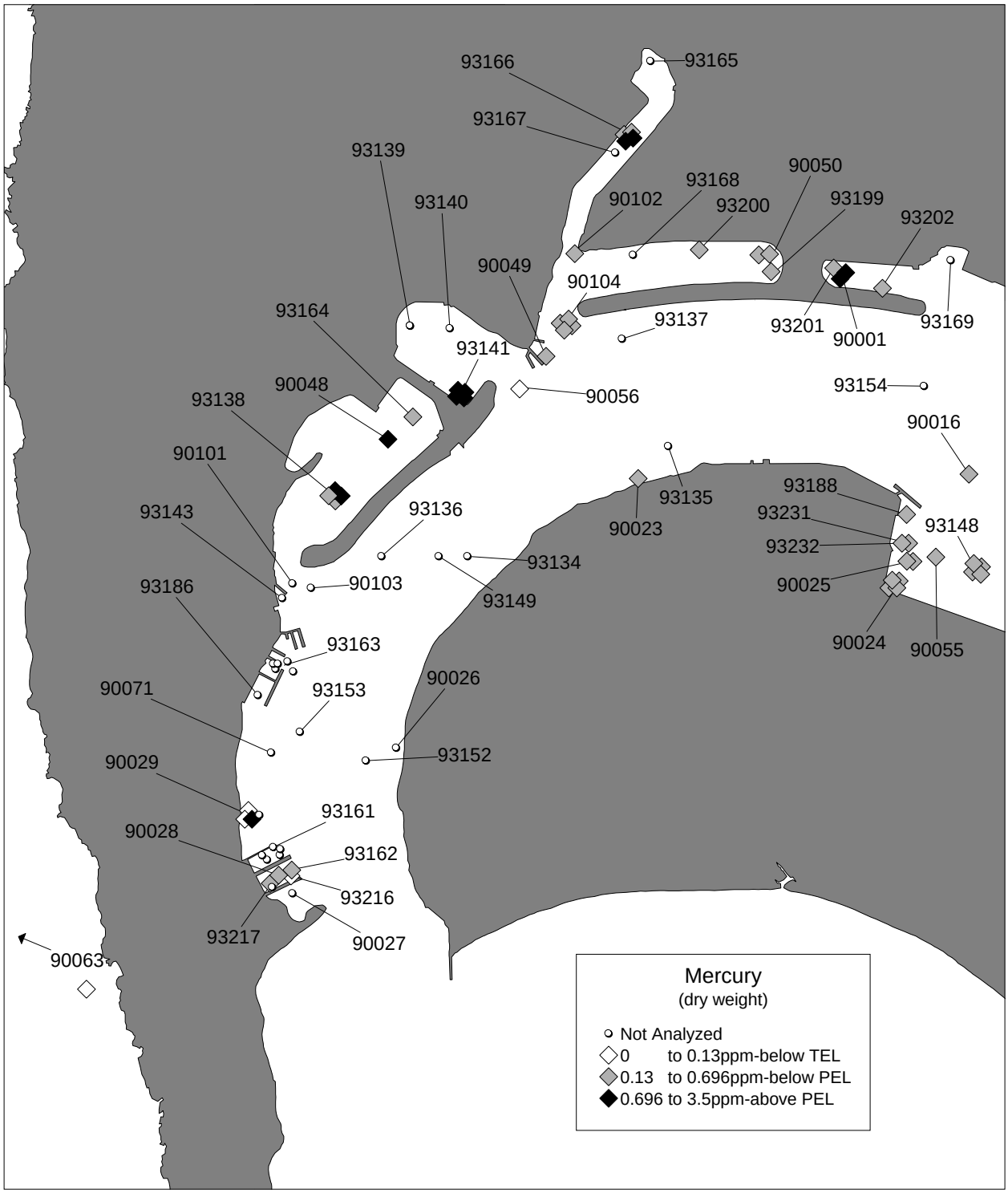


Figure 8b
Mercury Concentrations in Sediment
Mid San Diego Bay

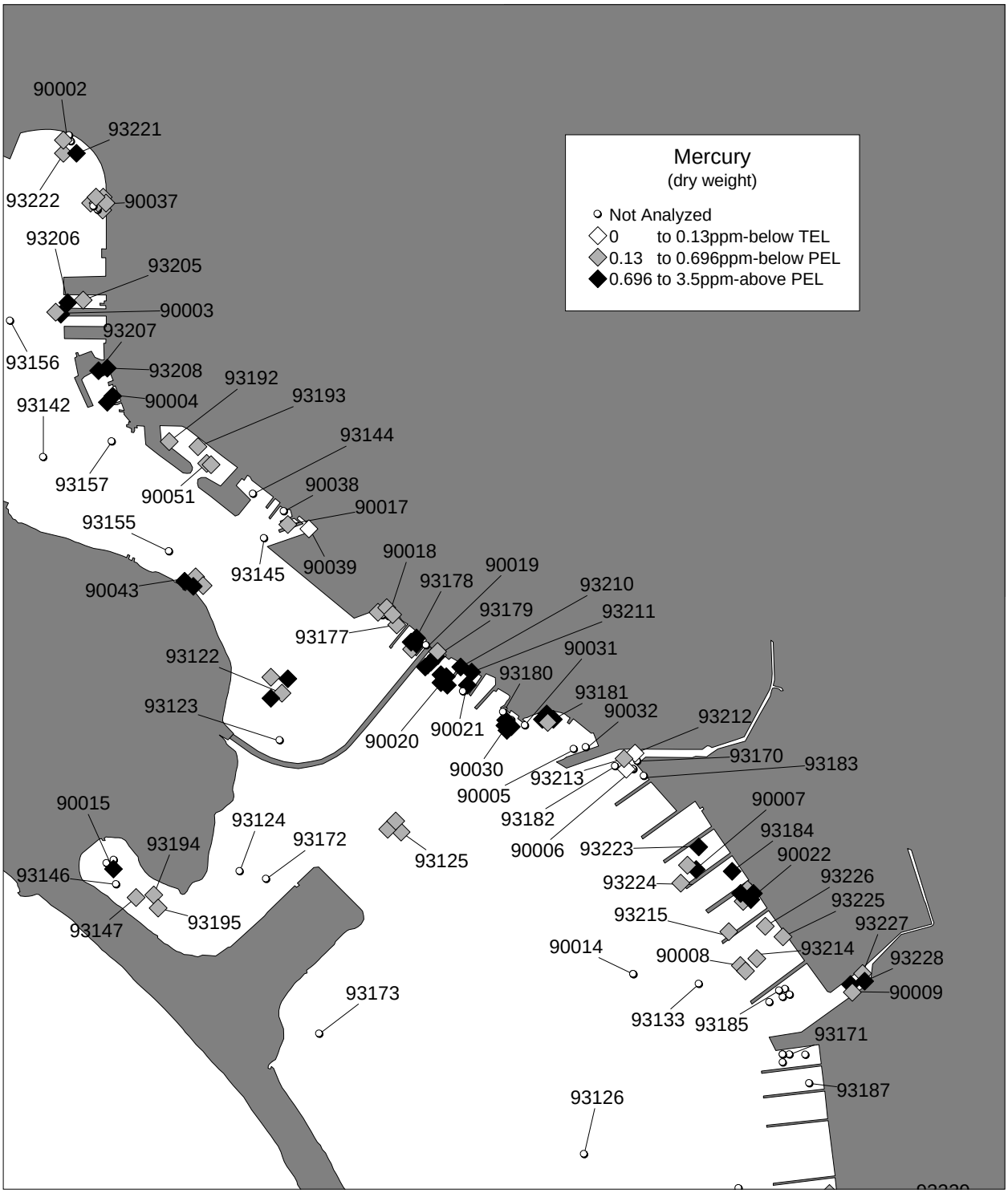


Figure 8c
Mercury Concentrations in Sediment
South San Diego Bay

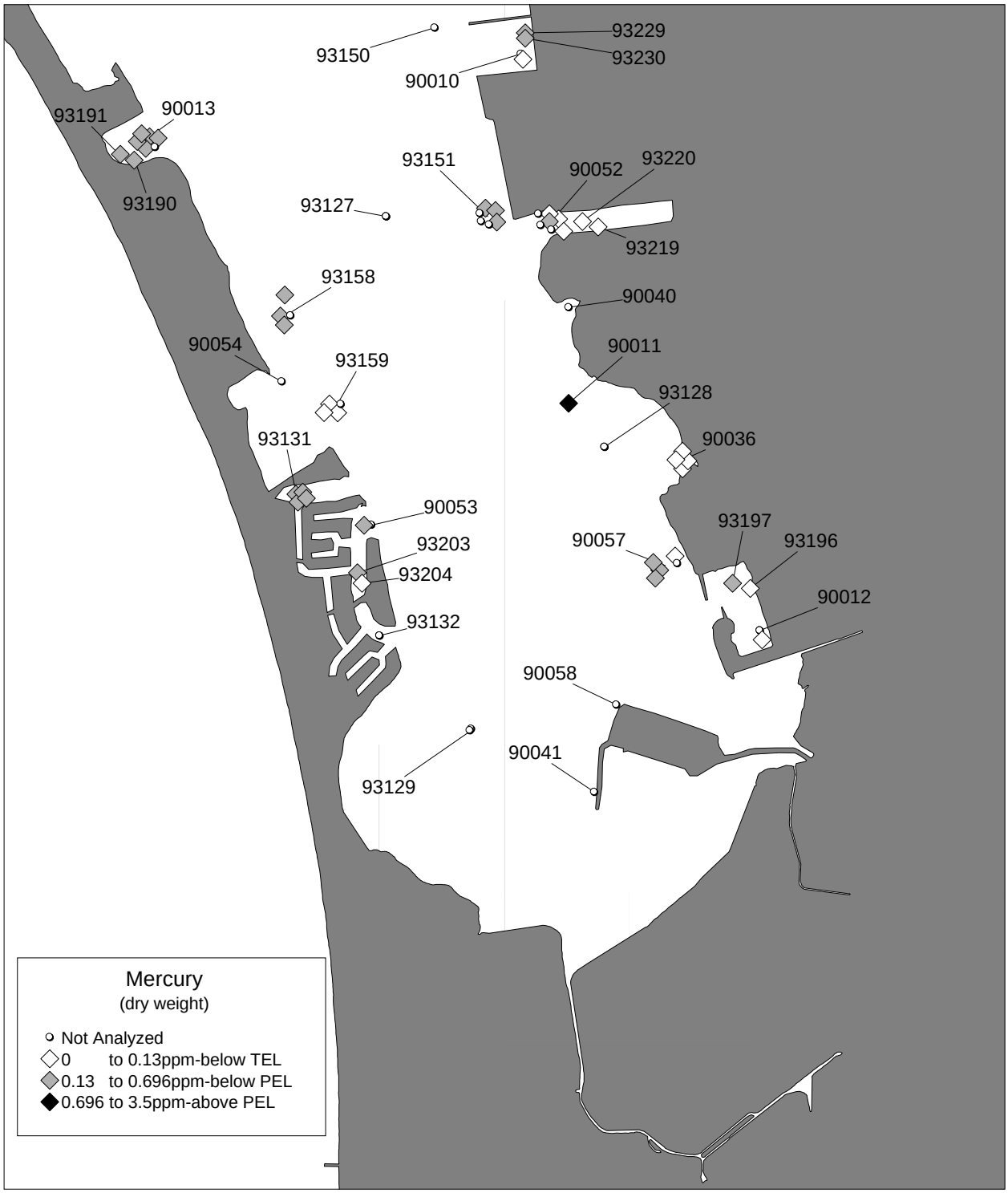
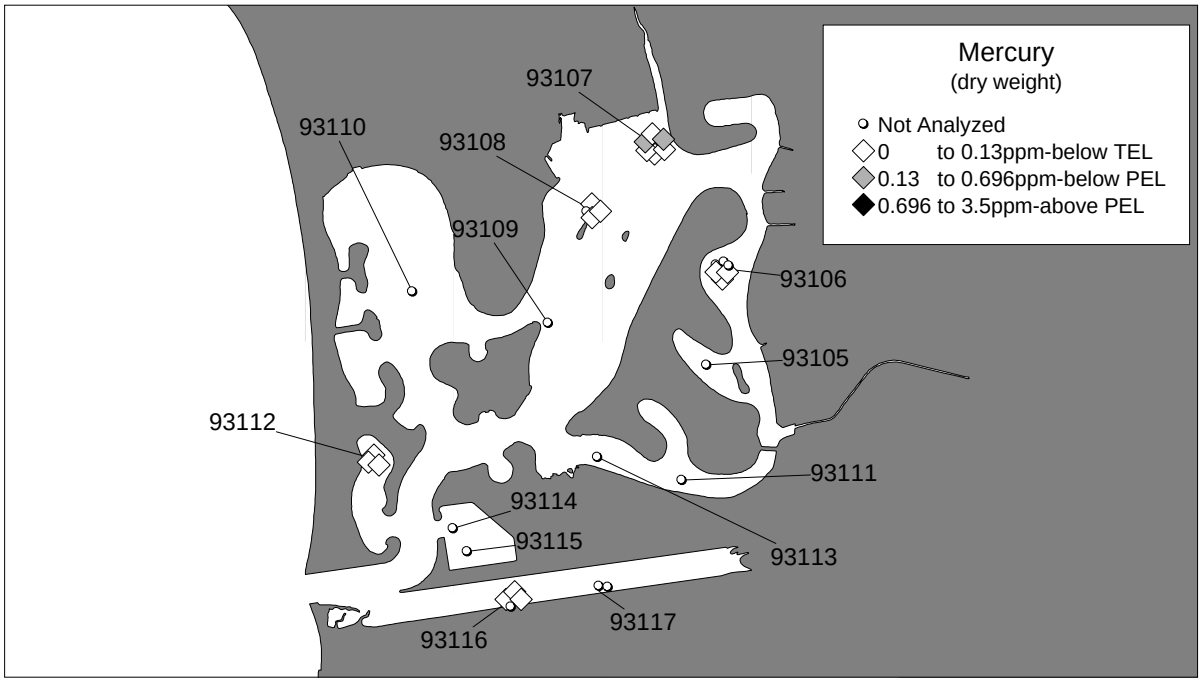


Figure 8d
Mercury Concentrations in Sediment
Mission Bay & San Diego River Estuary



Tijuana River Estuary

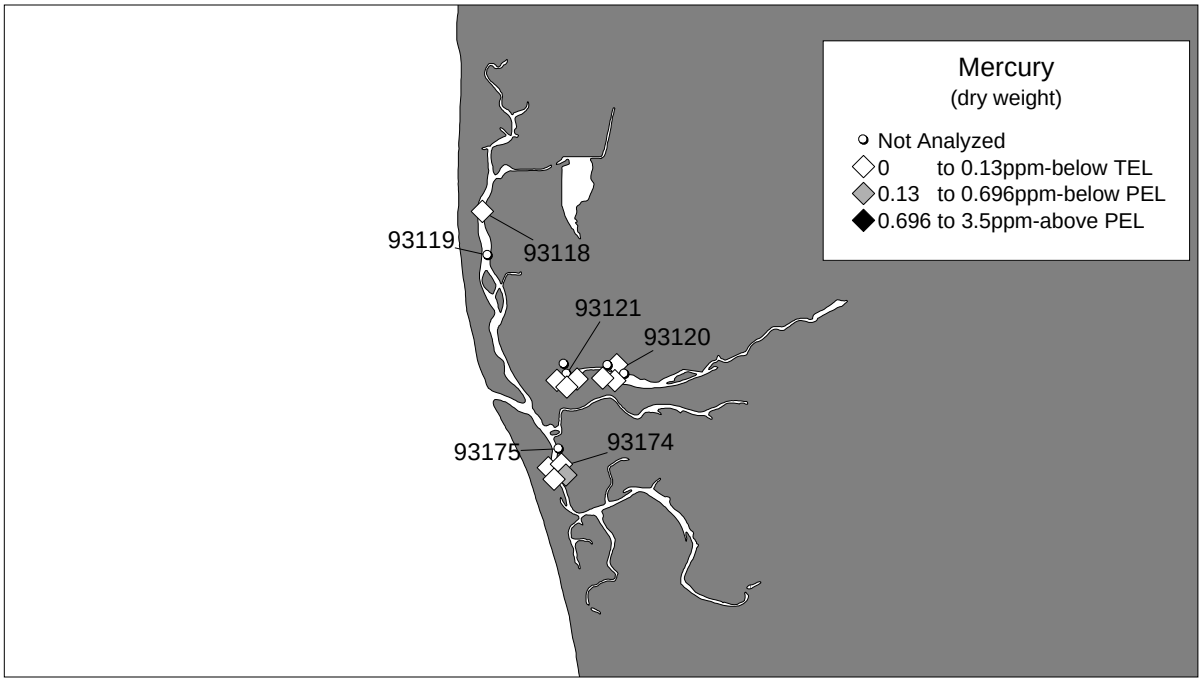


Figure 9a
High Molecular Weight PAH Concentrations in Sediment
North San Diego Bay

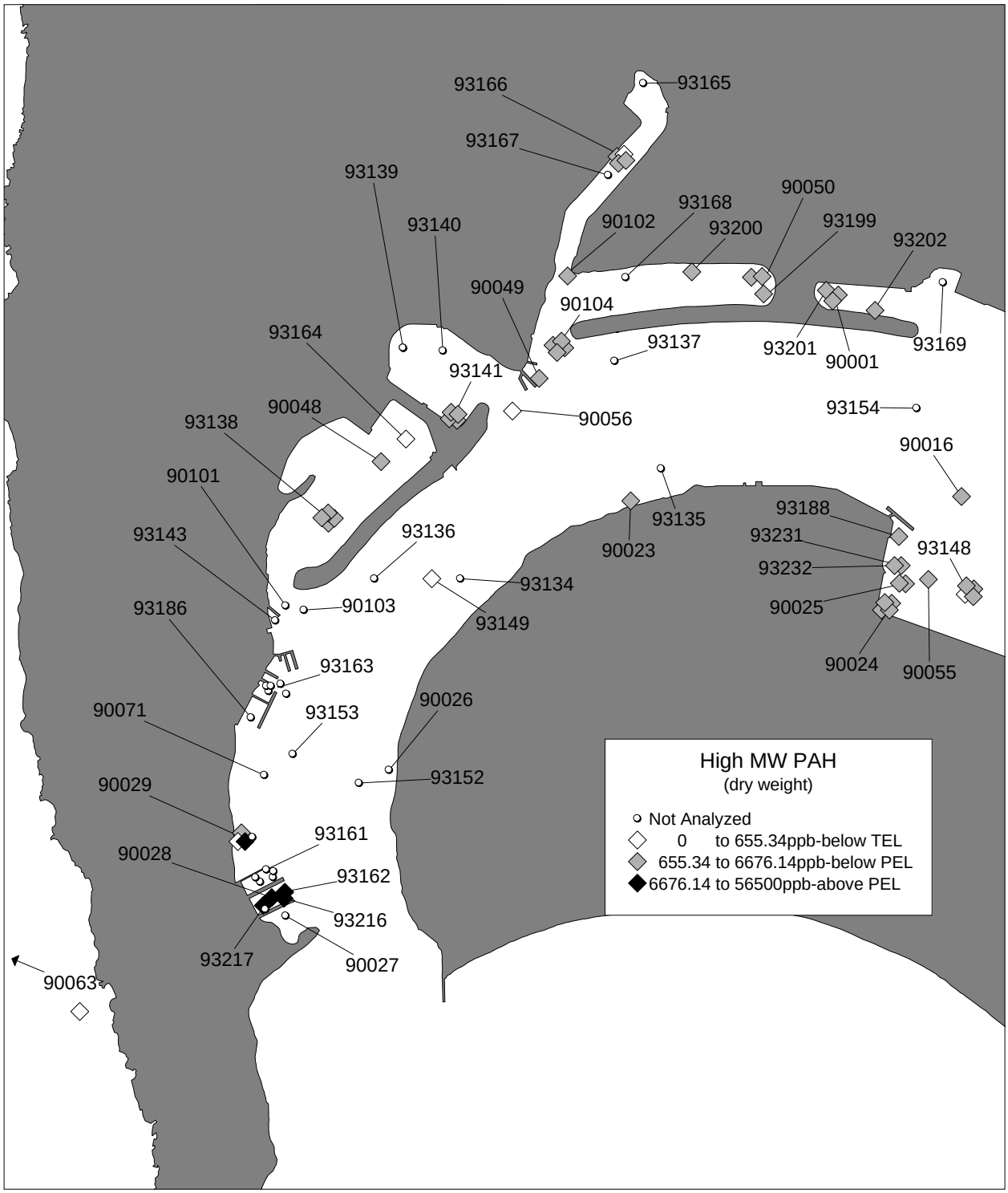


Figure 9b
High Molecular Weight PAH Concentrations in Sediment
Mid San Diego Bay

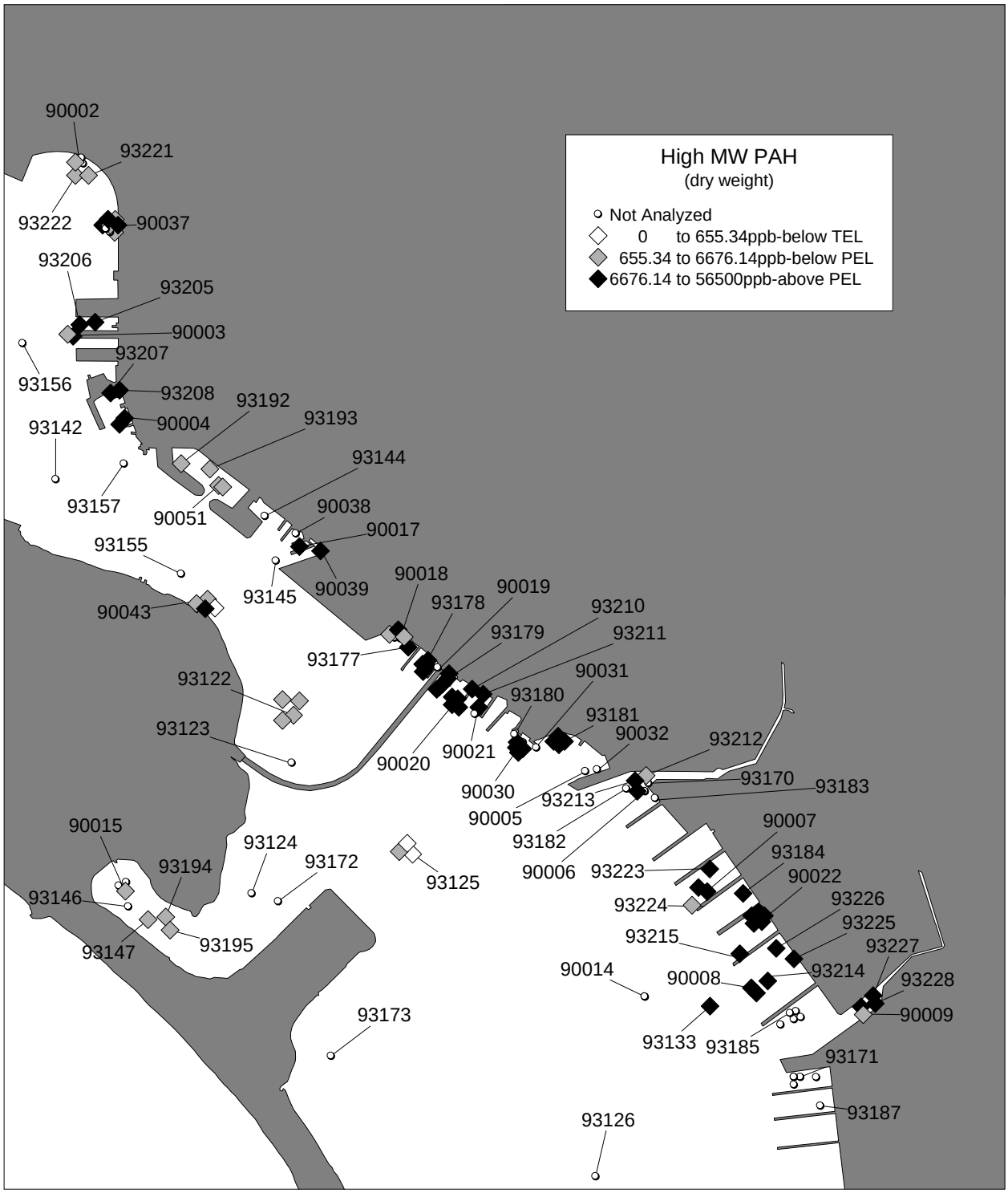


Figure 9c
High Molecular Weight PAH Concentrations in Sediment
South San Diego Bay

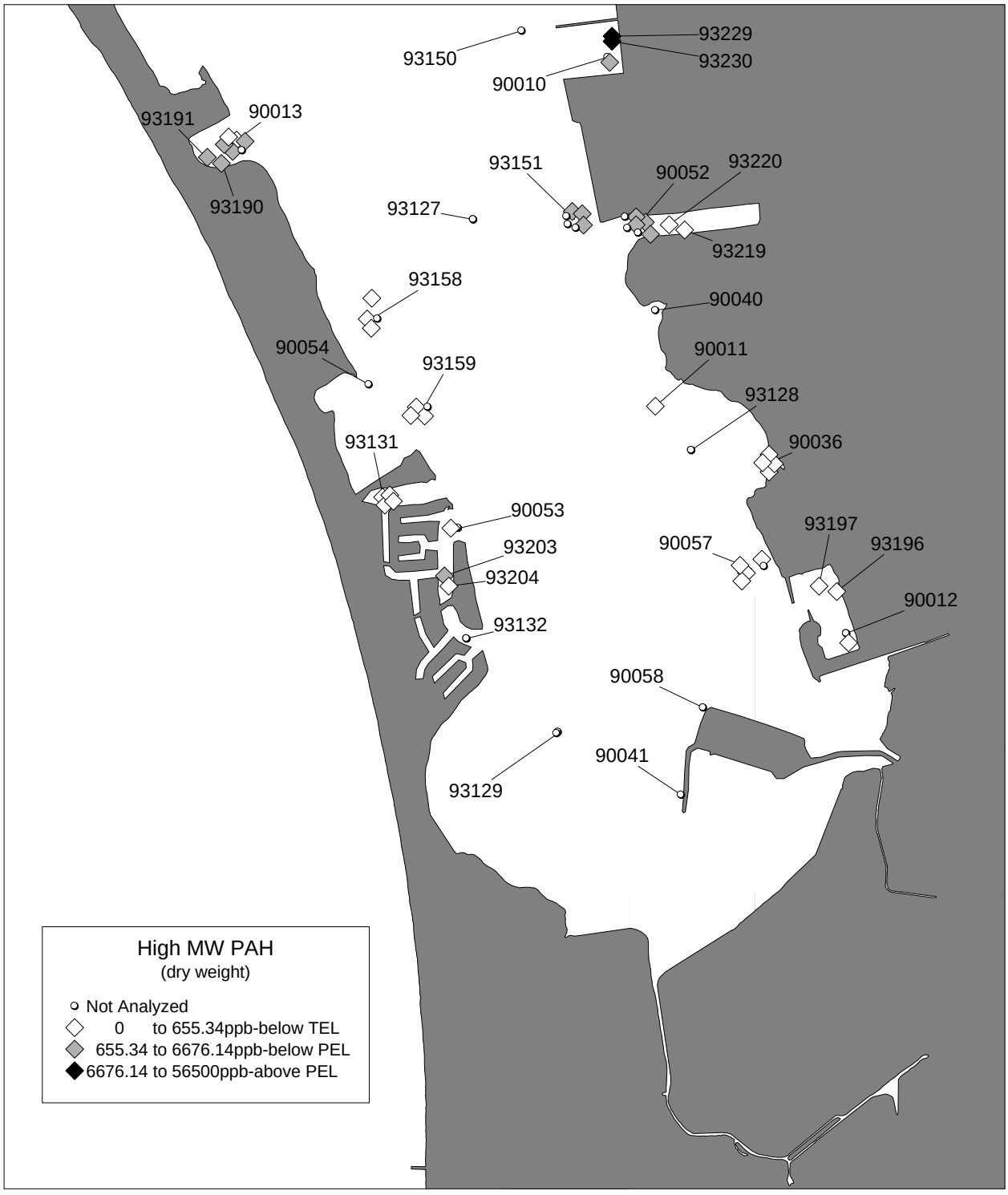
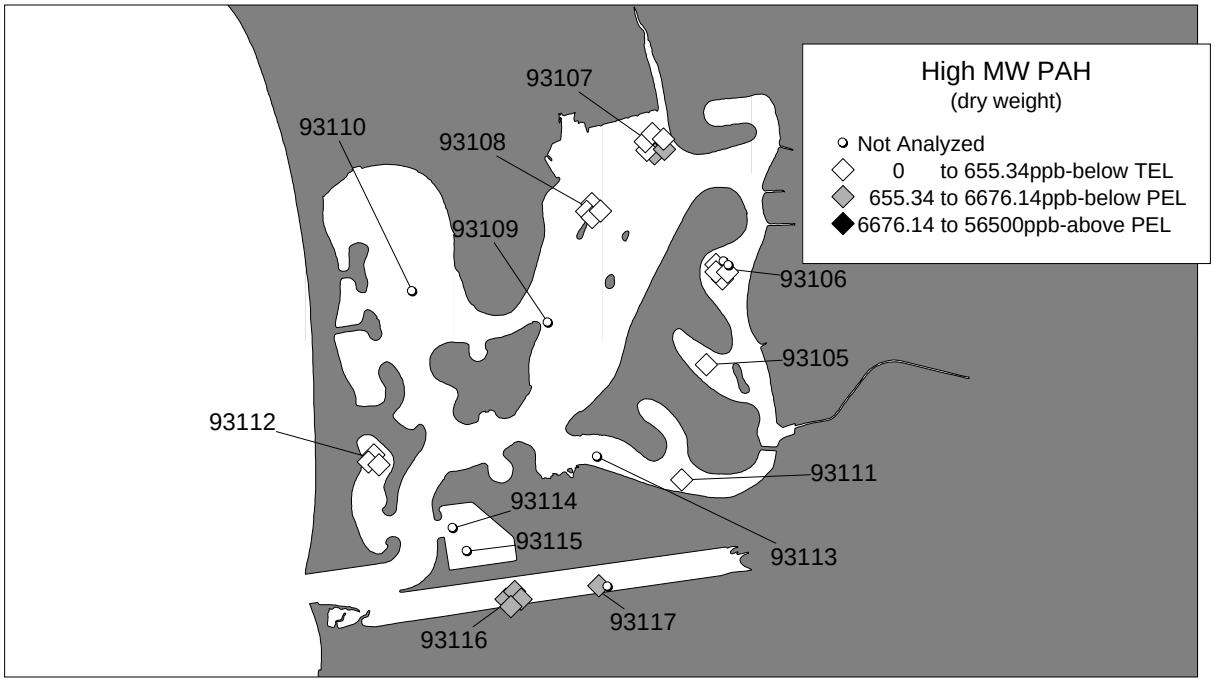


Figure 9d
High Molecular Weight PAH Concentrations in Sediment
Mission Bay and San Diego River Estuary



Tijuana River Estuary

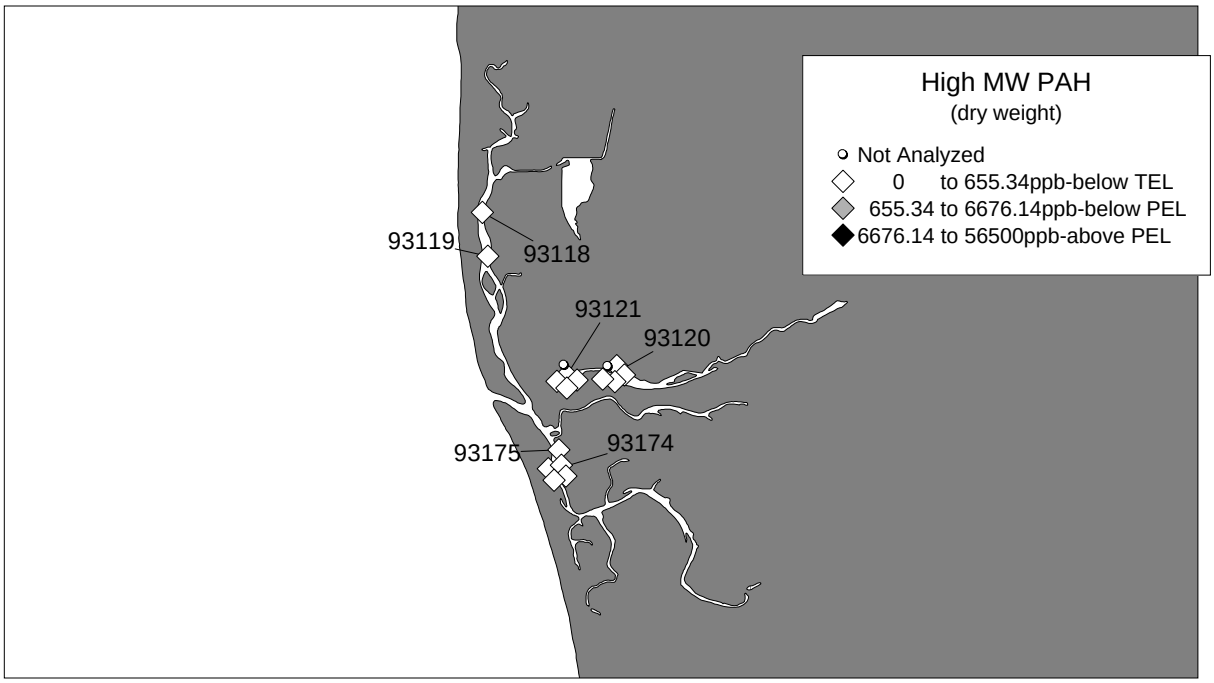


Figure 10a
Low Molecular Weight PAH Concentrations in Sediment
North San Diego Bay

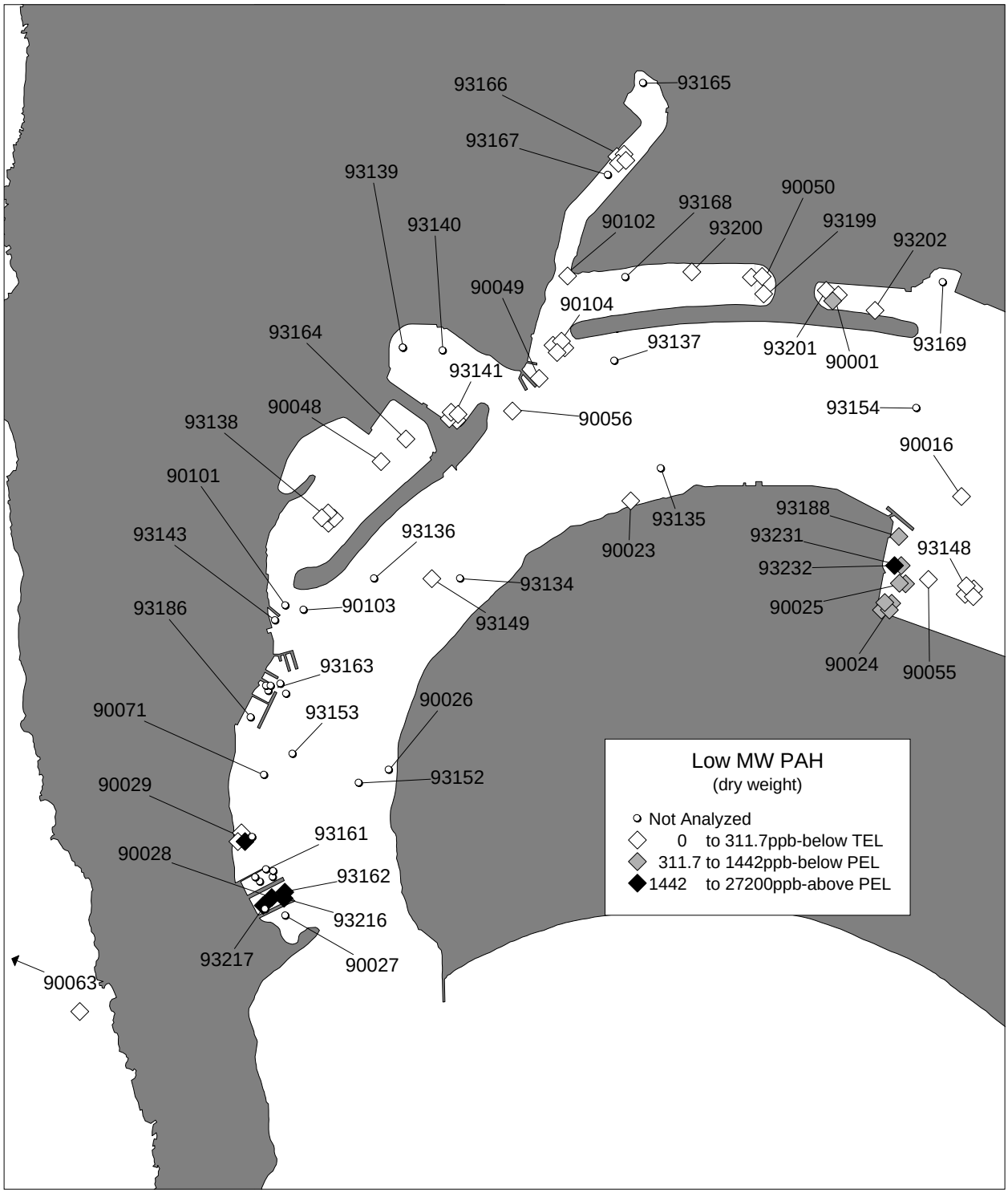


Figure 10b
Low Molecular Weight PAH Concentrations in Sediment
Mid San Diego Bay

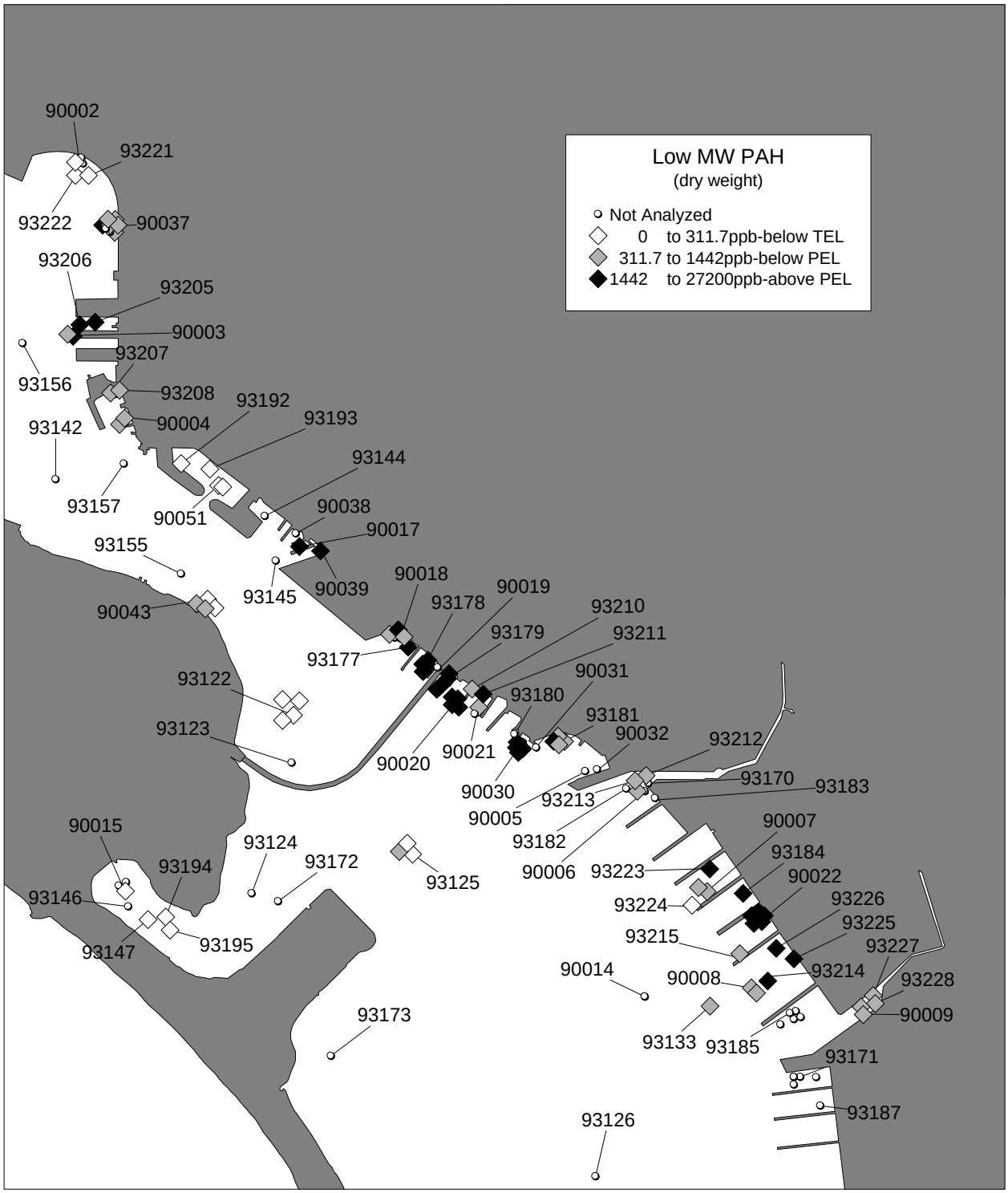


Figure 10c
Low Molecular Weight PAH Concentrations in Sediment
South San Diego Bay

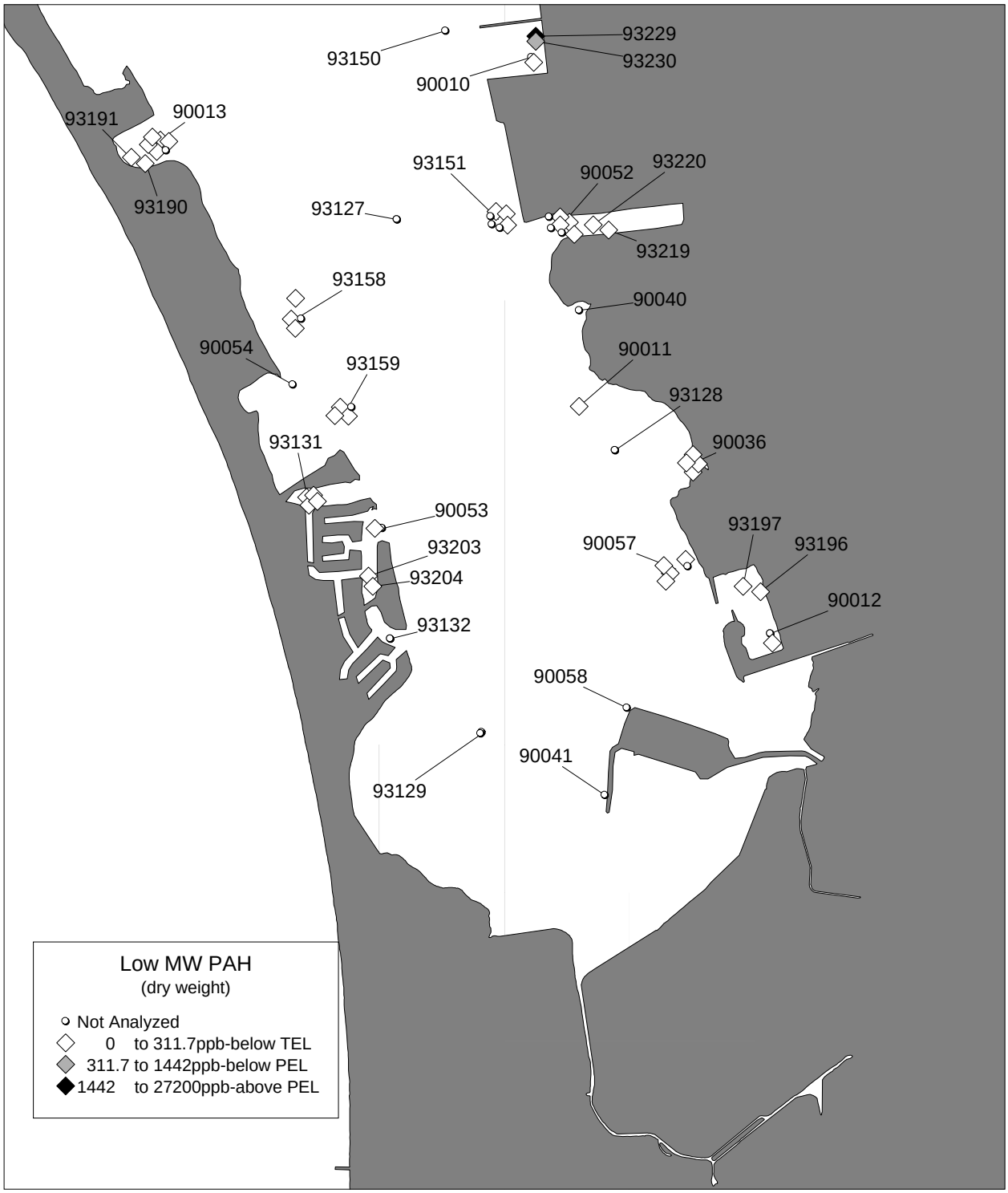
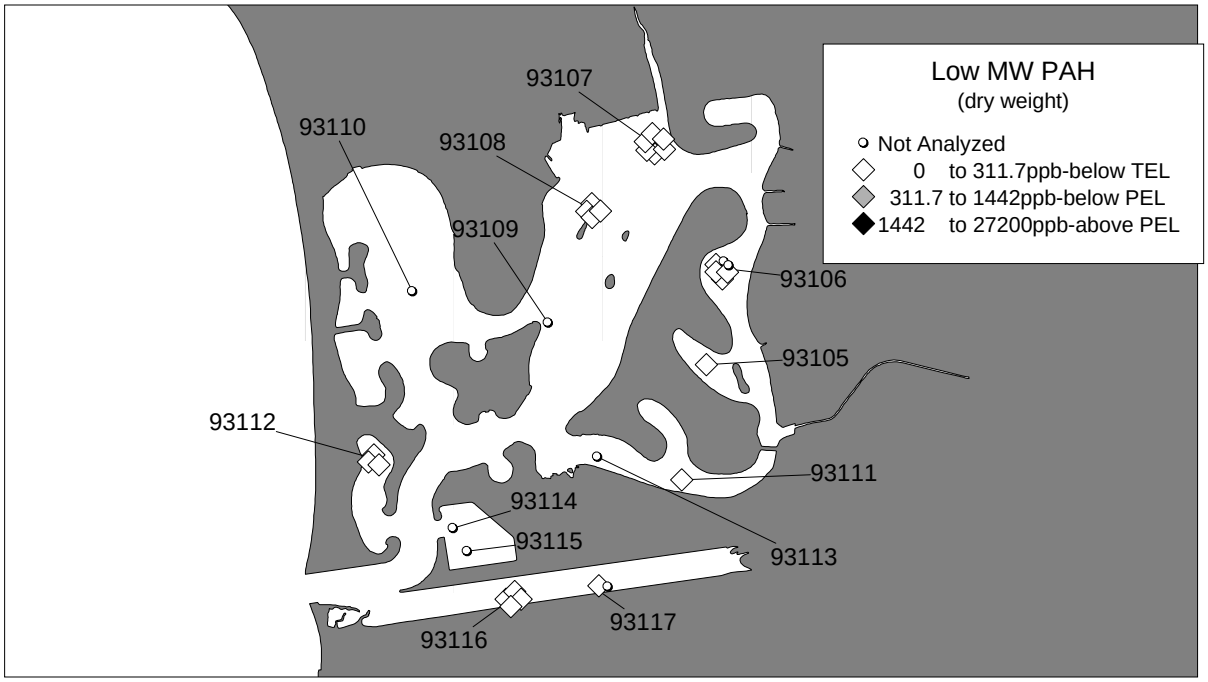
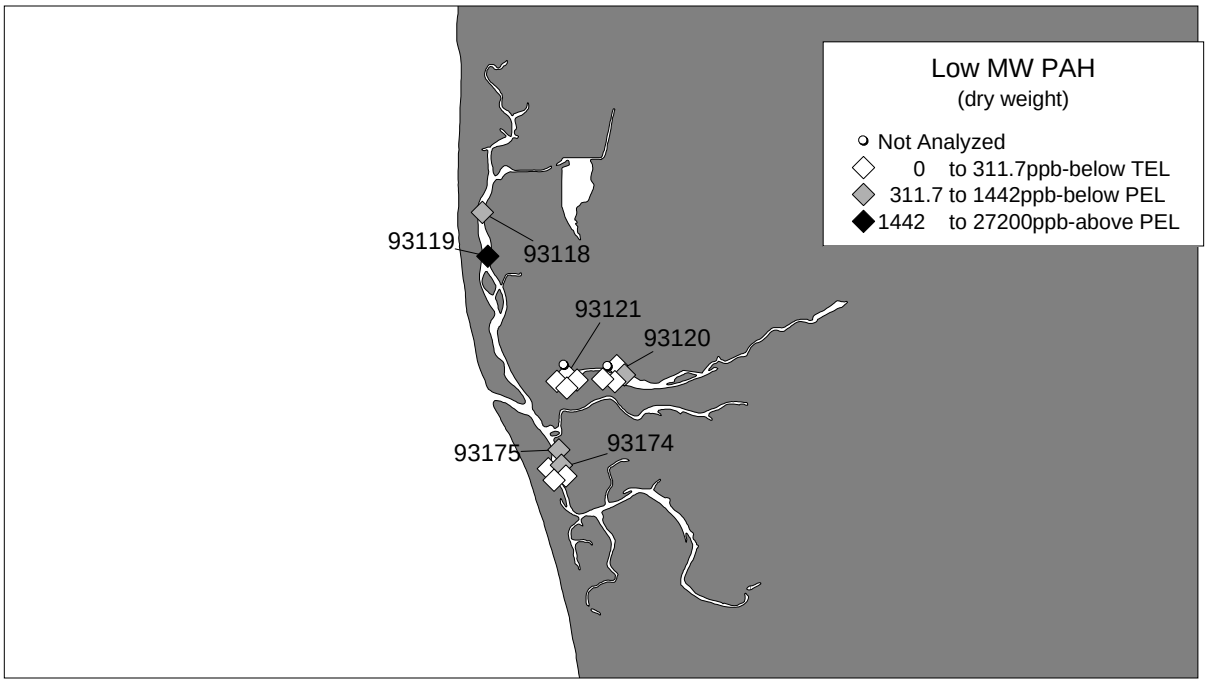


Figure 10d
Low Molecular Weight PAH Concentrations in Sediment
Mission Bay and San Diego River Estuary



Tijuana River Estuary



biphenyl. There are 209 numerically designated individual compounds, called congeners (*i.e.*, PCB #101), based on the possible chlorine substitution patterns. Mixtures of various PCB congeners have been manufactured in the U.S. since 1929 (Phillips, 1987) and are used commercially under the trade name Aroclor. Each PCB mixture has a number designation (*i.e.*, Aroclor 1254) with the last two numbers indicating the percentage of chlorine in the mixture. PCB mixtures were used extensively in the U.S. prior to 1979 for industrial applications which required fluids with thermal stability, fire and oxidation resistance and solubility in organic compounds (Hodges, 1977). PCBs have proven to be extremely persistent in the environment and have demonstrated a variety of adverse carcinogenic and non-carcinogenic effects (USEPA, 1993c). These substances have a high potential to accumulate in the tissues of aquatic organisms and can represent significant hazards to consumers of aquatic species (Moore and Walker, 1991). Total PCB (the sum of 18 congeners, Appendix B - Section VII) pollution is most prominent in sediments along the naval shipyard waterfront (Figure 11(a-d)), although several locations along the downtown waterfront and small boat harbors also show total PCB values in excess of the PEL (>188.79 μg/kg) and ERM (>180 μg/kg).

Chlordane is a multipurpose insecticide which has been used extensively in home and agricultural applications for the control of termites and other insects. Although use of this compound ended in the mid-70s, its persistence in sediments of the region is apparent. Total chlordane is the summation of major constituents of technical grade chlordane and its metabolite (Appendix B - Section VII). Chlordane pollution is extensive along the north shore of San Diego Bay, the San Diego River, and the most northerly station in Mission Bay (Figure 12(a-d)). Areas which receive storm runoff, such as Chollas Creek, Seventh St. Channel, and urban storm drains appear to be the most heavily contaminated (PEL (>4.79 μg/kg) or ERM (>6 μg/kg)).

ERM and PEL Summary Quotients

In this report, comparisons of the data to effects-based numerical guidelines were made to assess how sediment pollution in the San Diego Bay Region compares to sediment pollution on a national scale. Additionally, these guidelines were used to identify chemicals of concern for sediment quality management within the San Diego Bay Region. Rankings and comparisons were made in this report using summary ERM-quotients (ERMQ) and PEL-quotients (PELQ). Summary quotients are summations of chemical concentrations for chemicals listed in Table 5, divided by their respective ERM or PEL value, and then divided by total number of chemicals used. In samples where levels of measured chemicals were below the analytical method detection limit (MDL), a value of one-half the MDL was used for summations. Methods and analytes used for summations and averaging are given in Appendix B-Section VII. This was a simple approach for addressing overall chemical pollution where there were multiple pollutants at a station, and was in addition to the standard chemical by chemical

Figure 11a
Total PCB Concentrations in Sediment
North San Diego Bay

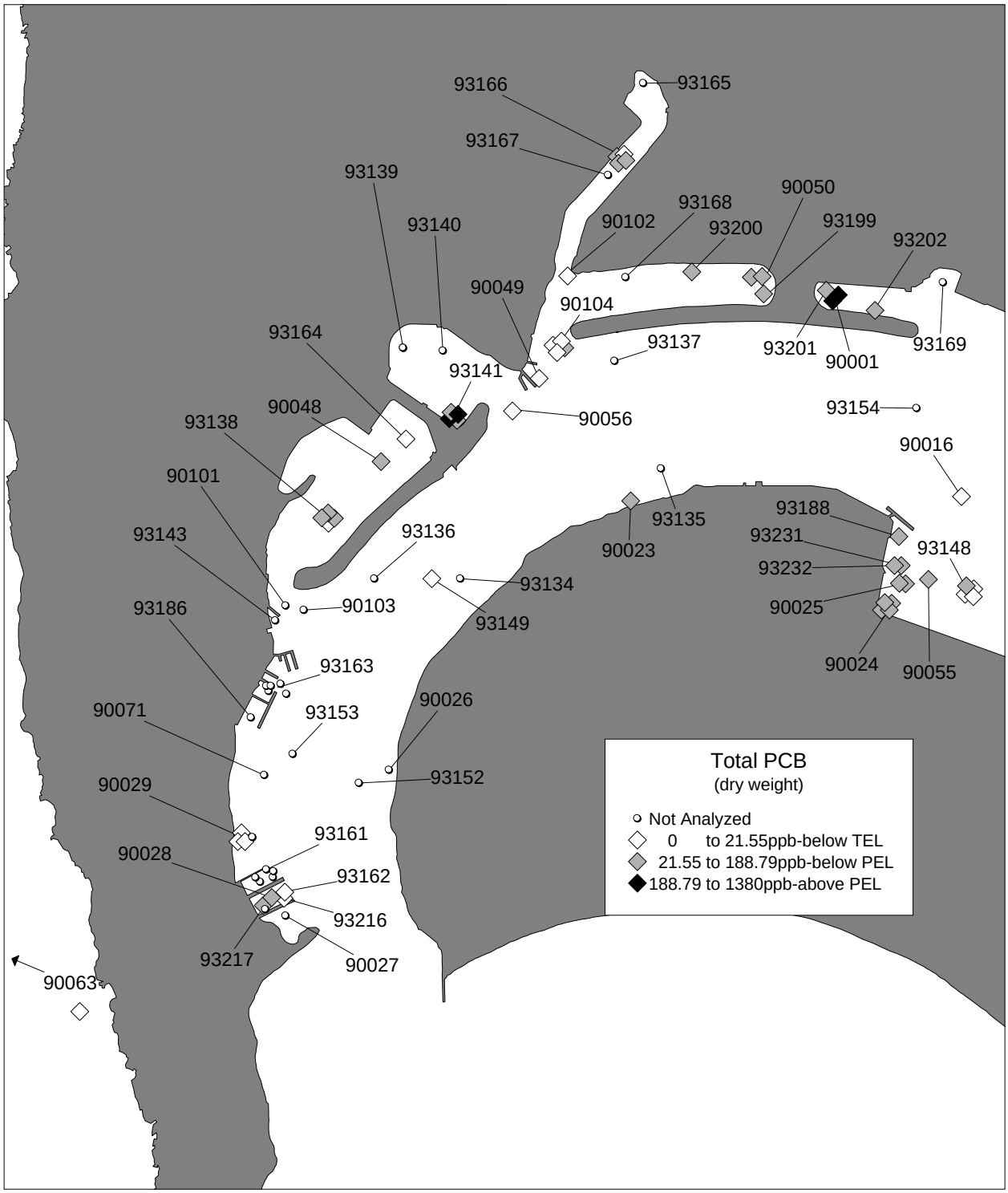


Figure 11b
Total PCB Concentrations in Sediment
Mid San Diego Bay

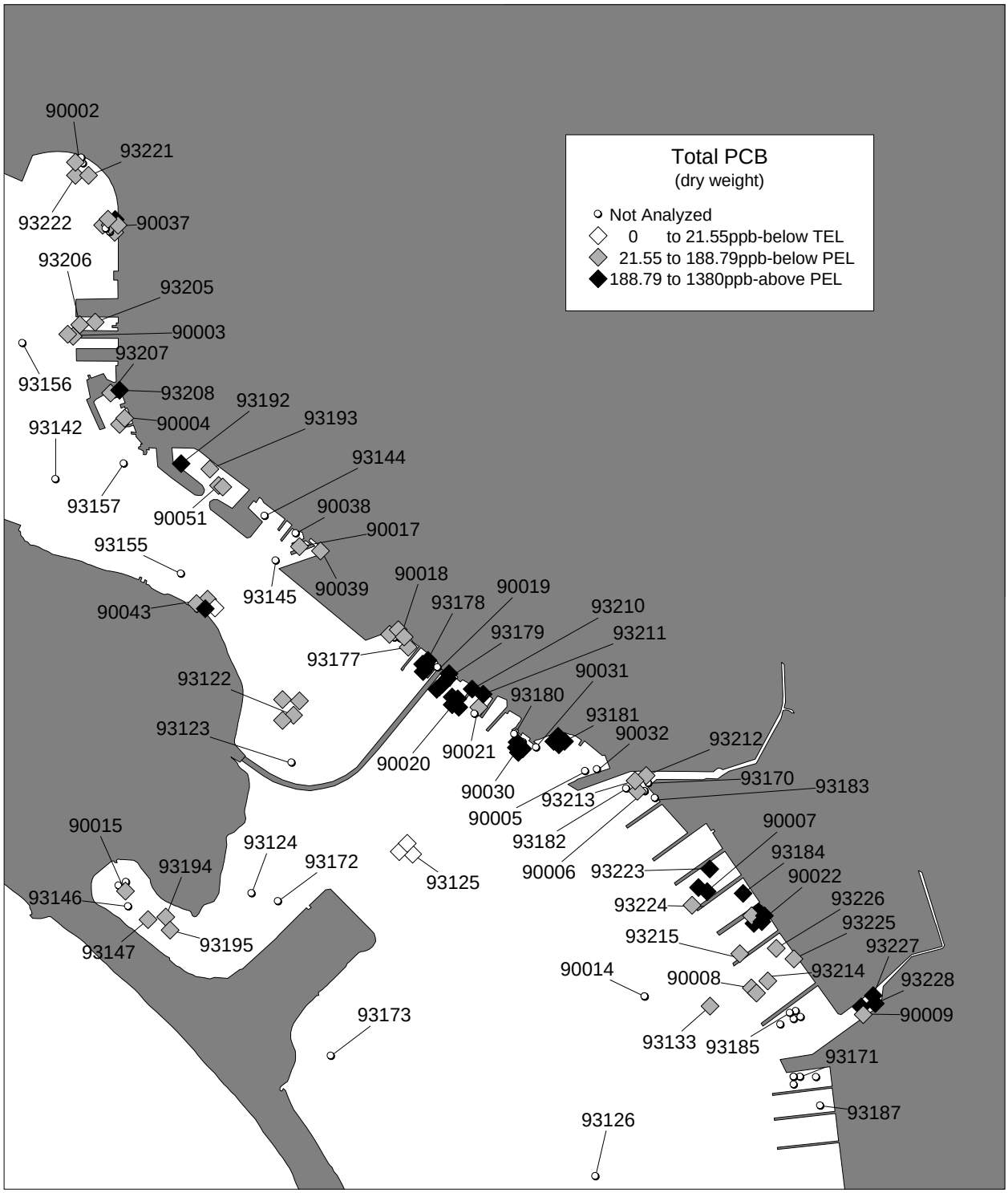


Figure 11c
Total PCB Concentrations in Sediment
South San Diego Bay

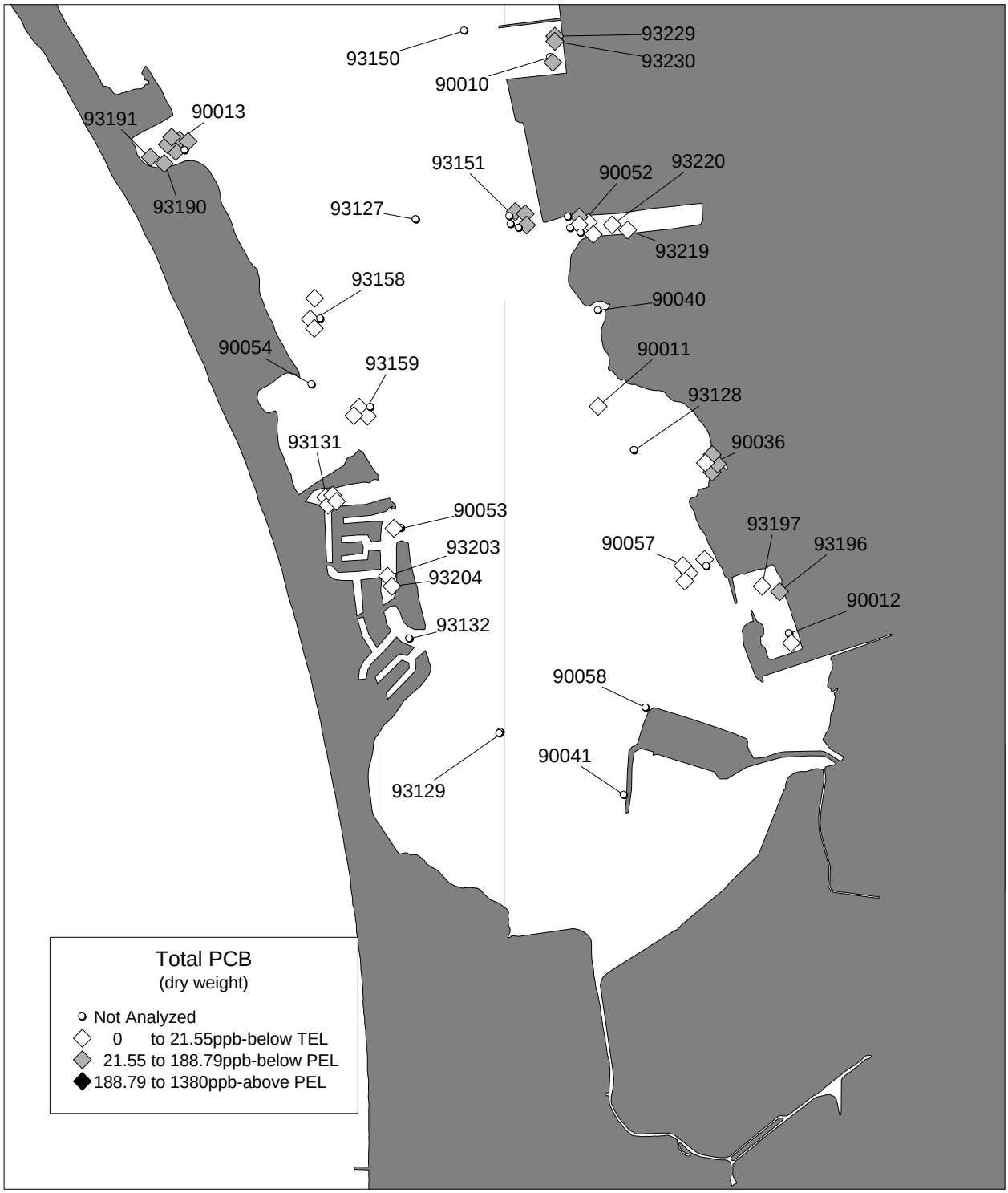
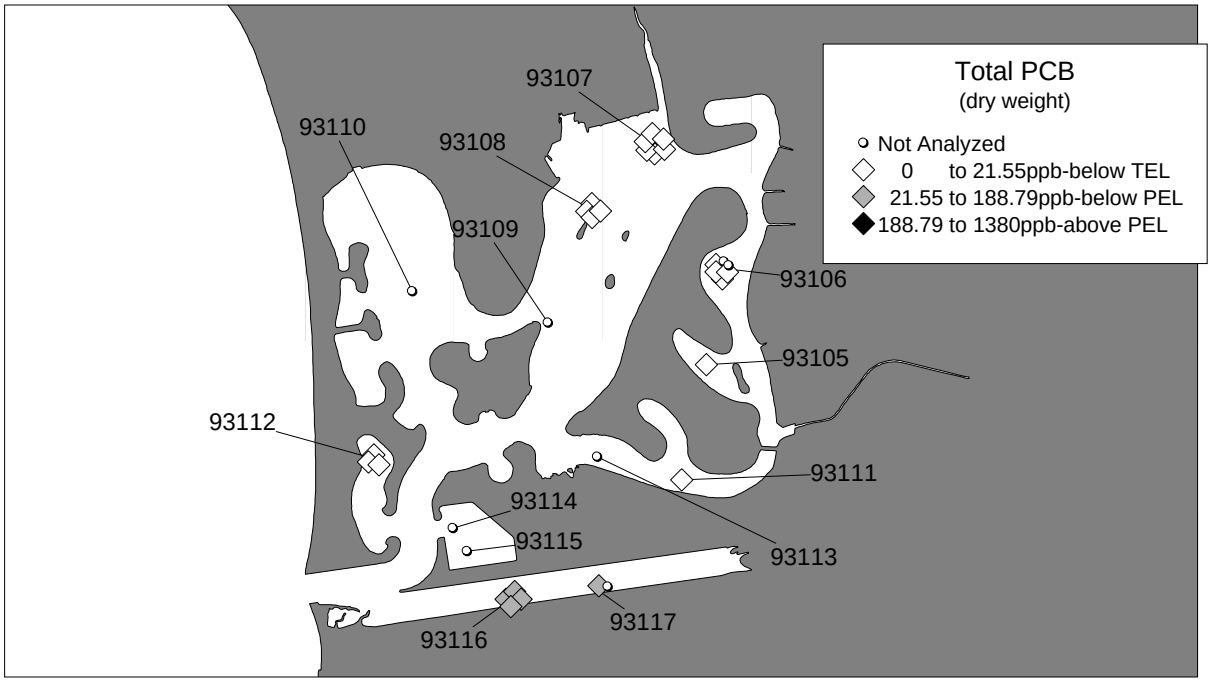


Figure 11d
Total PCB Concentrations in Sediment
Mission Bay and San Diego River Estuary



Tijuana River Estuary

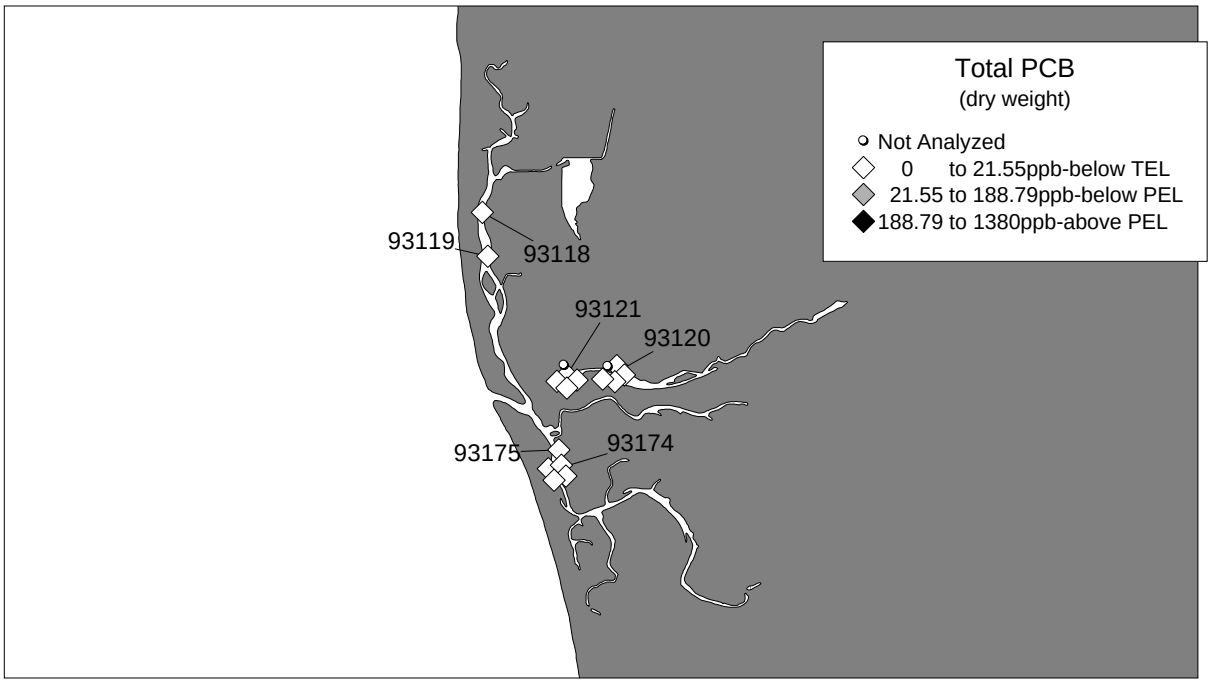


Figure 12a
Total Chlordane Concentrations in Sediment
North San Diego Bay

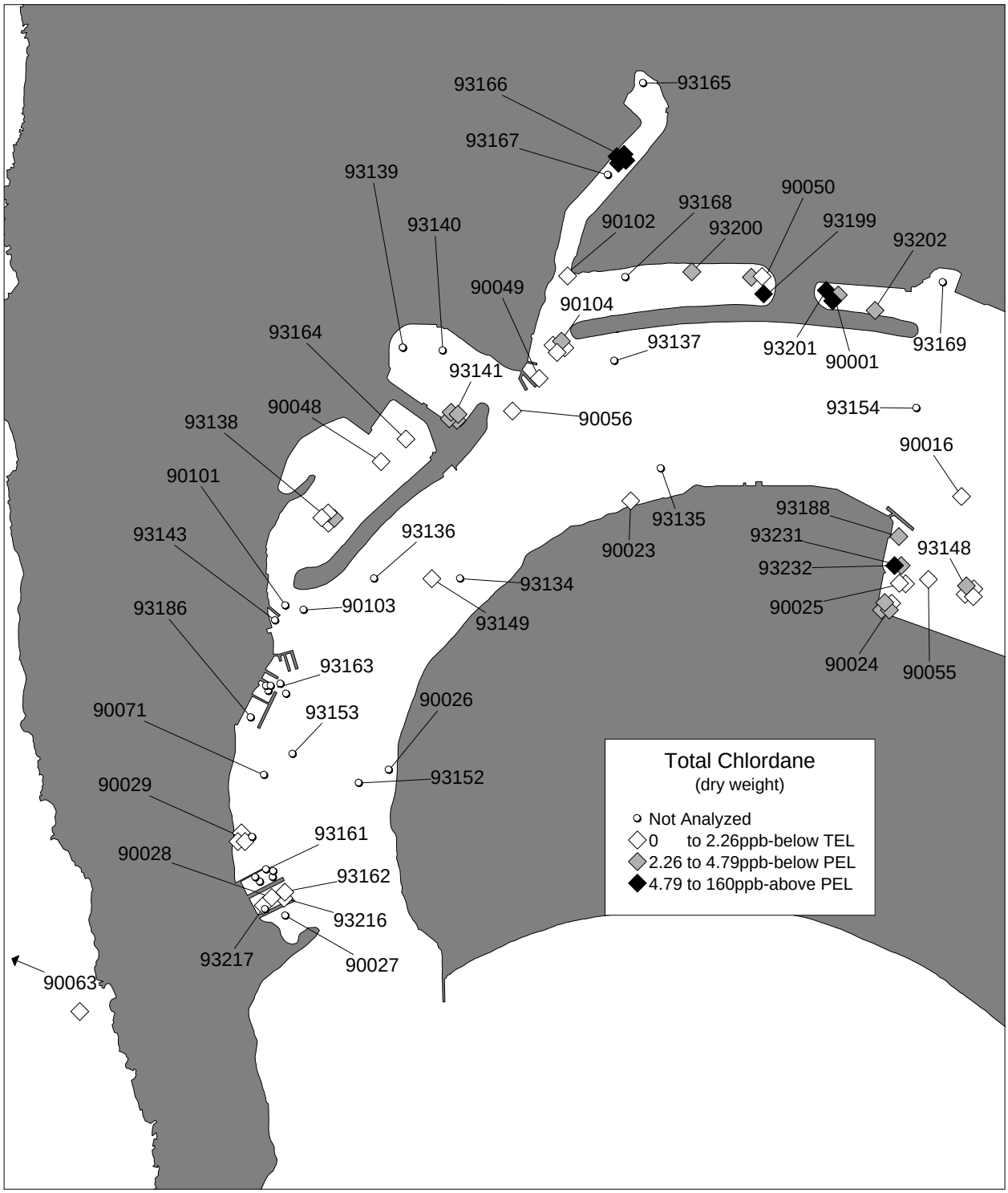


Figure 12b
Total Chlodane Concentrations in Sediment
Mid San Diego Bay

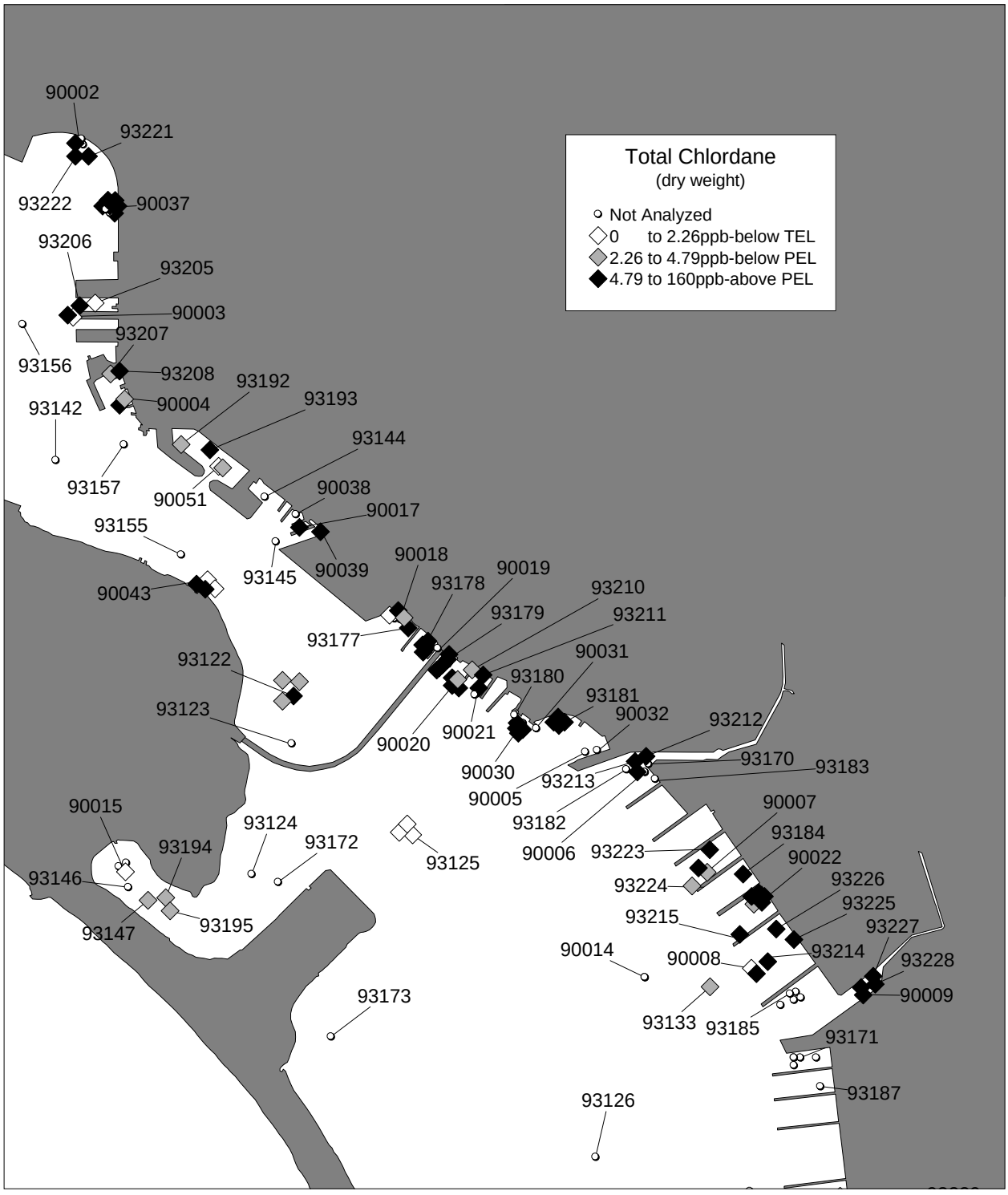


Figure 12c
Total Chlordane Concentrations in Sediment
South San Diego Bay

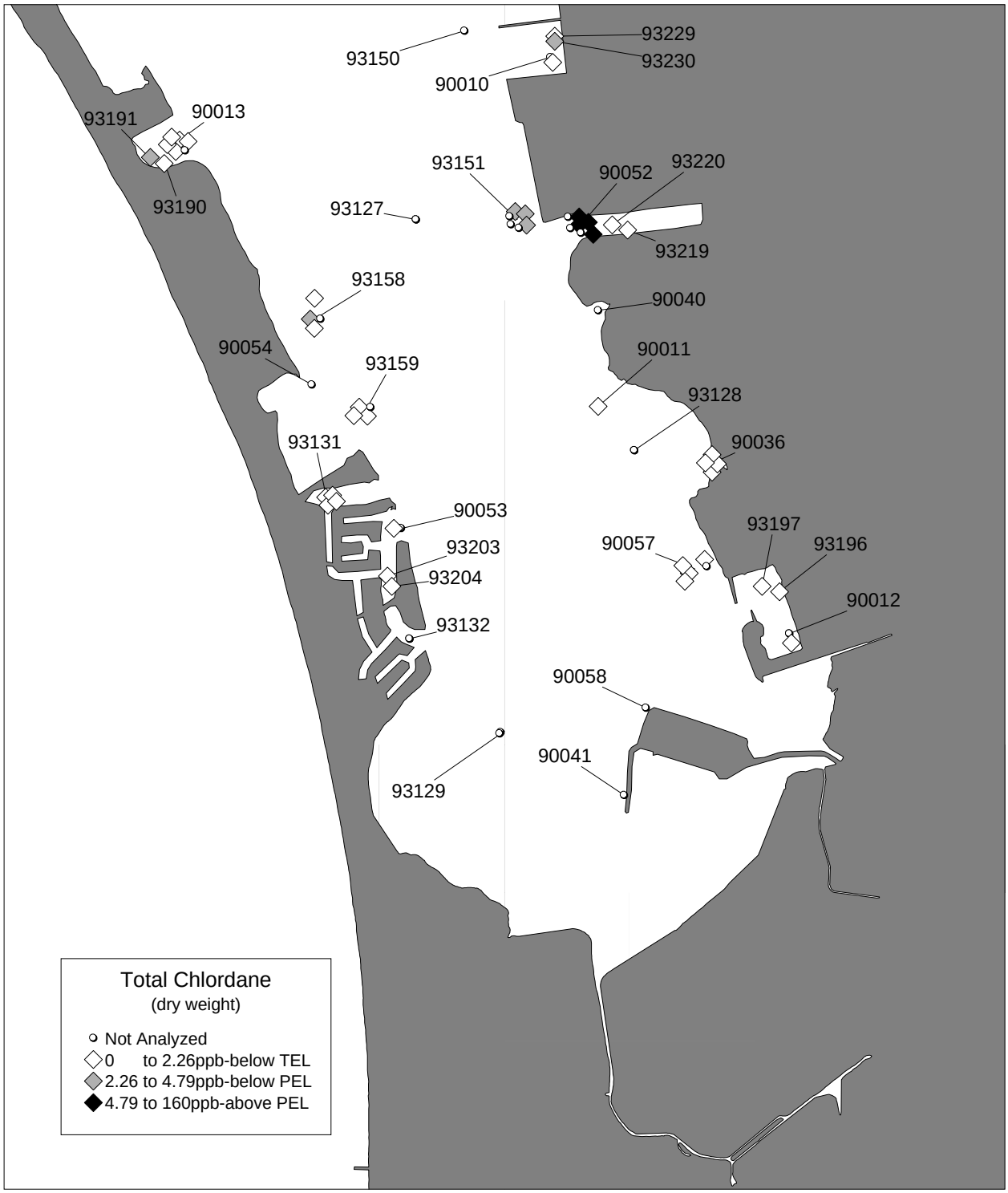
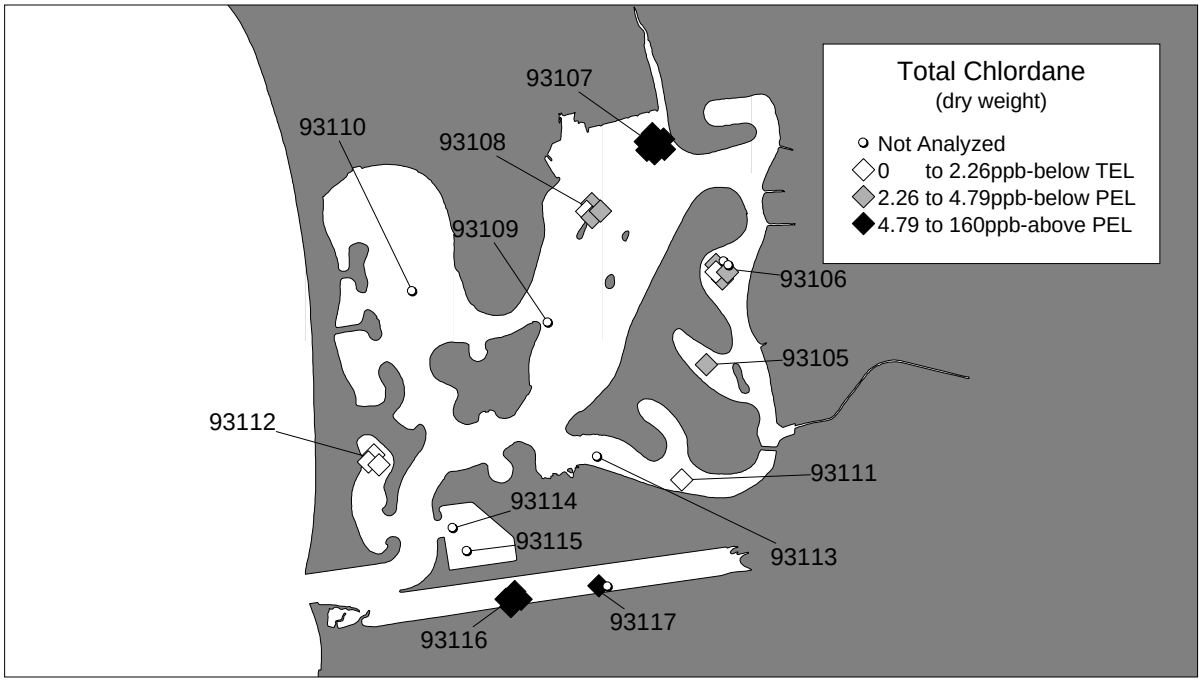
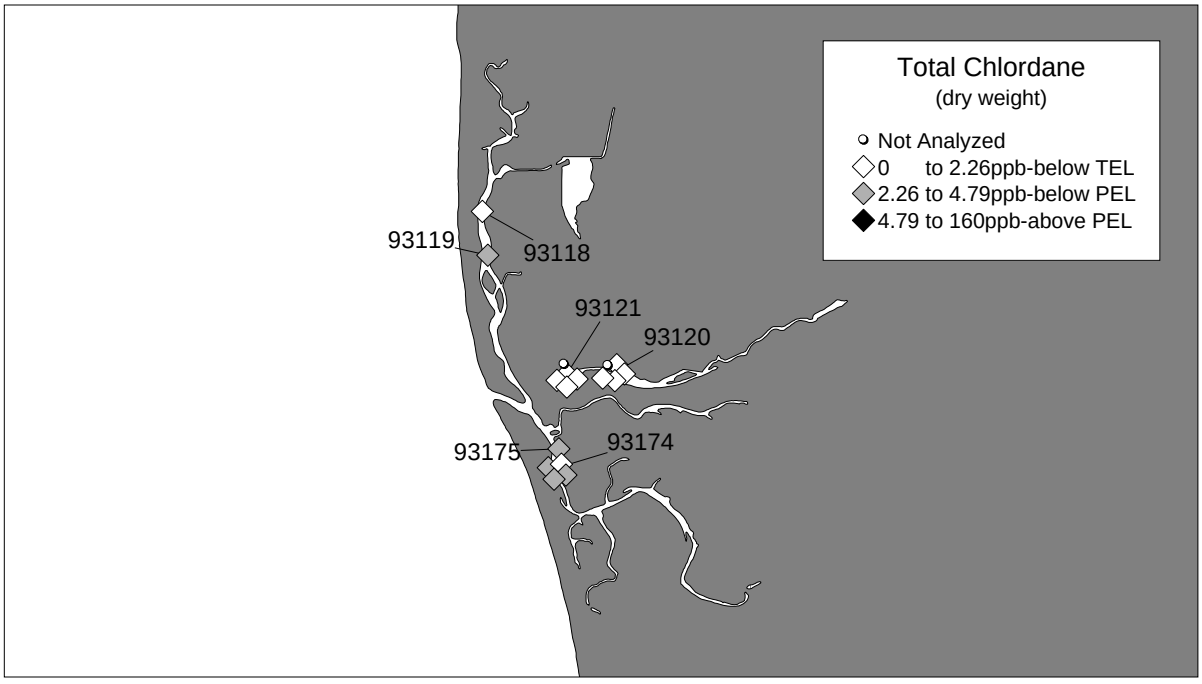


Figure 12d
Total Chlordane Concentrations in Sediment
Mission Bay and San Diego River Estuary



Tijuana River Estuary



approach discussed earlier. This approach considered not only the presence of guideline exceedances, but the number and degree of multiple exceedances.

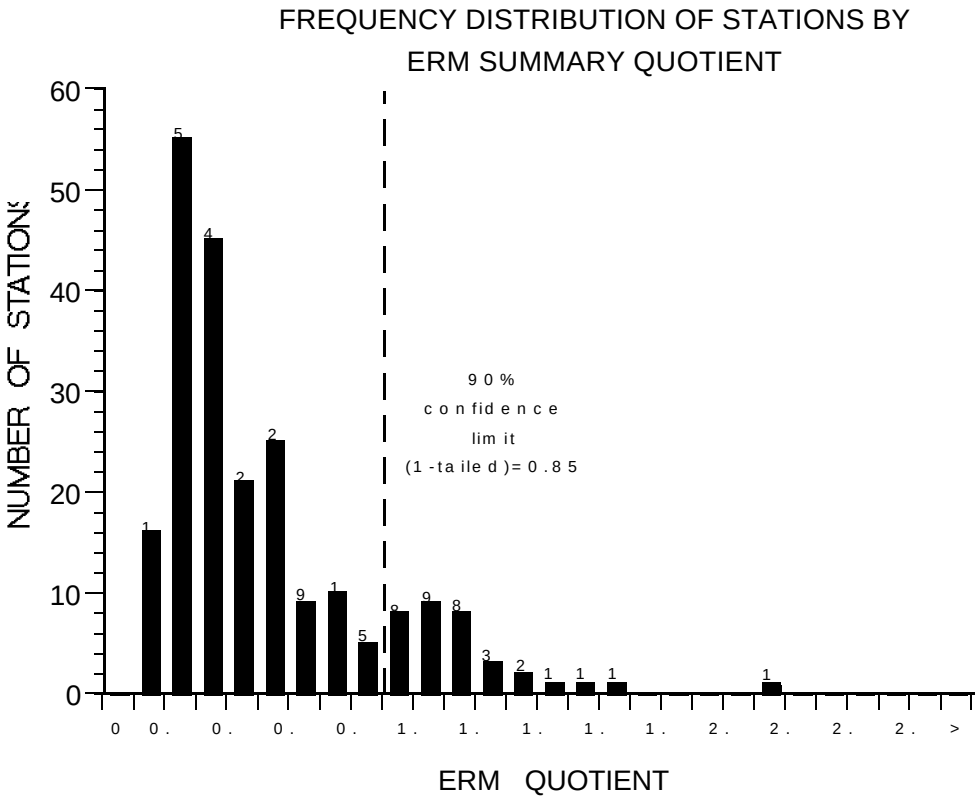
Based upon analyses of the national NS&T and EMAP database, the incidence of toxicity has been shown to increase with increasing summary ERM and PEL quotients (Long, Field and MacDonald, in prep). Synergistic effects are possible, but not implied by the quotient summations, therefore, this method should be recognized only as a ranking scheme meant to better focus management efforts on interpretation of ambient sediment chemistry data.

Interpretations using ERM and PEL summary quotients were limited to statistical analysis within this dataset because the approach has not been formally presented in other reports, therefore, outside comparisons are unavailable at this time. The 90% confidence interval from a 1-tailed t-distribution was chosen as an arbitrary threshold level for evaluating the data set. For the 220 stations on which chemical analysis was performed, stations with an ERMQ>0.85 or a PELQ>1.29 were found to fall above this confidence interval (Figure 13). Although these values of 0.85 and 1.29 cannot be considered threshold levels with proven ecological significance, they can be used for within bay comparative purposes. Forty-one stations exhibited ERM or PEL quotient levels exceeding the confidence interval cutoffs. Of these forty-one stations, twelve received benthic community analysis, all which were determined to have degraded communities in the analysis discussed later (Figure 14). All 41 stations were tested for *Rhepoxynius* toxicity, of which 29% demonstrated significant toxicity, at the 48% limit established by the reference envelope method discussed later. This difference in biological response to pollutants, between benthic community structure and bioassays, may be explained by long term exposure to pollutants in the benthic community relative to short term (10 day) pollutant exposure in bioassay tests. Use of the ERM and PEL quotients appear to give a worthwhile representation of overall chemical pollution and are used later in this report for station rankings and characterizations.

Distribution of Benthic Community Degradation

Data Analyses and Interpretation

The identification of benthic degraded and undegraded habitat (as determined by macrobenthic community structure) was conducted using a cumulative, weight-of-evidence approach. Tests were employed without prior knowledge or integration of results from laboratory exposures or chemical analyses. Analyses were performed to identify relationships between community structure within and between each station or site. This included diversity/evenness indices, analyses of habitat and species composition, construction of dissimilarity matrices for pattern testing, assessment of indicator species and development of a benthic index, cluster and ordination (multidimensional scaling) analyses. Initially, a triangular correlation matrix was produced



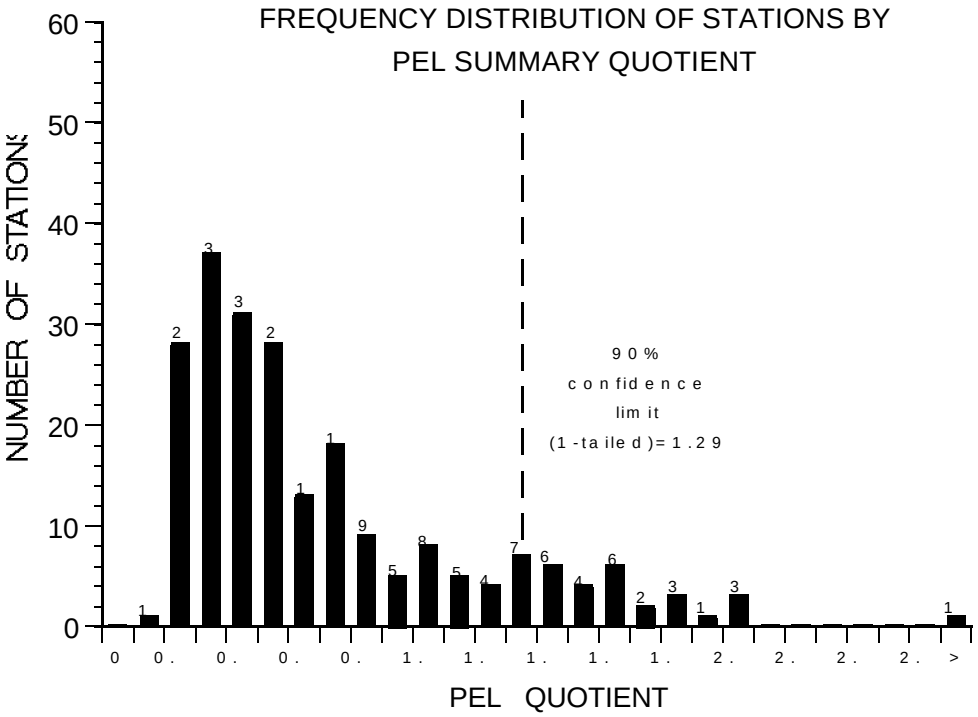


Figure 13. Histogram of the number of stations by ERM or PEL summary quotient group. Vertical dashed line indicates 90% confidence limit of the mean.

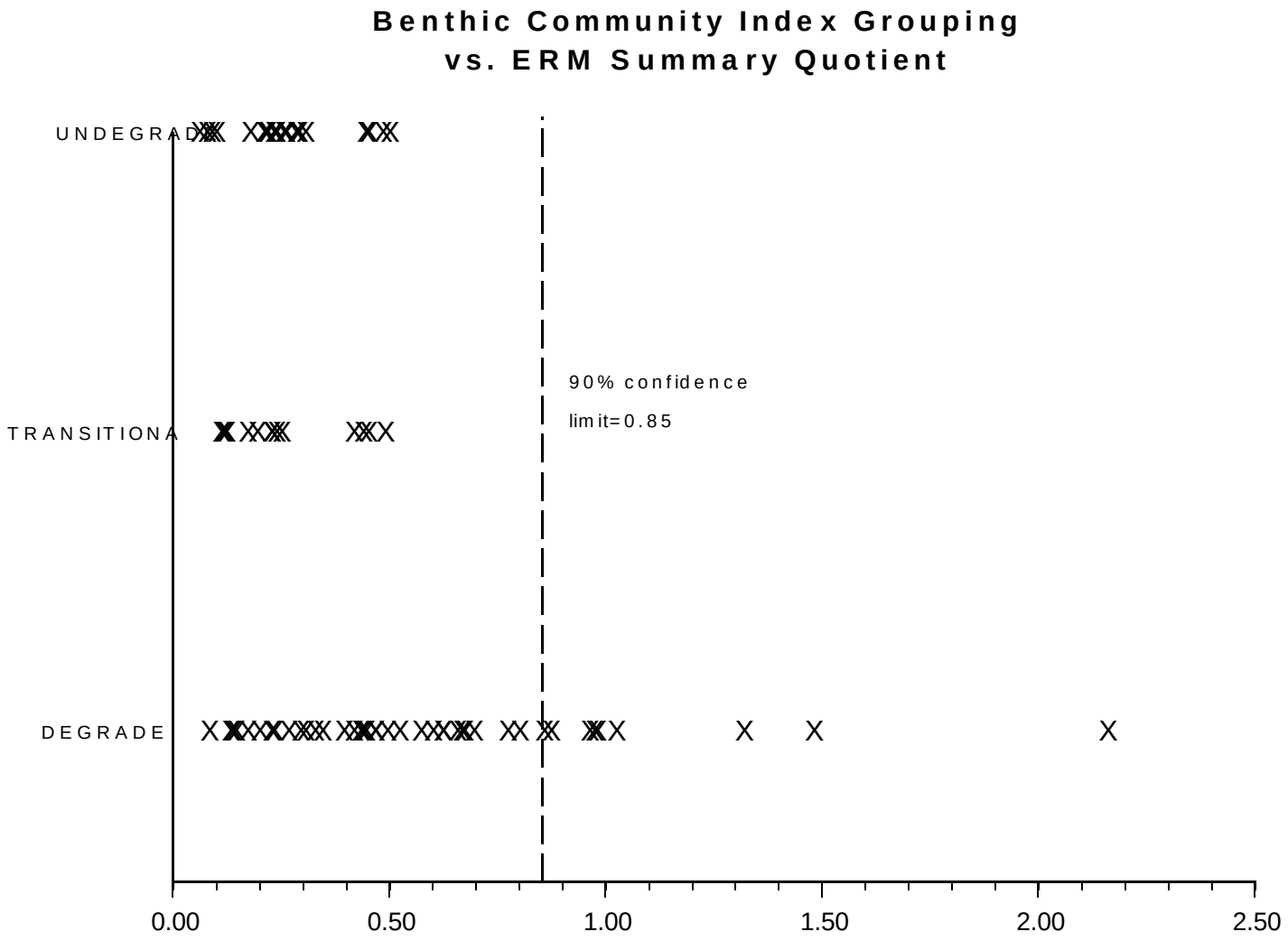


Figure 14. Benthic index grouping vs. ERM summary quotient value. Each data point represents one station (n=75).

from species density data from each site using the Systat® statistical program. From this matrix several tests for association of variables were performed. The tests employed are common in marine and estuarine benthic community analyses and are well-documented in the literature (Field *et al.,* 1982; Pearson *et al* 1983; Swartz *et al.,* 1985; Gray, 1989; Clark and Ainsworth, 1993). Classification analysis was employed to demonstrate site-related community patterns such as species dominance. Cluster analysis is a multivariate procedure for detecting natural groupings in data, and, for our purposes, data were grouped by average similarities in total composition and species abundance (Krebs, 1989). The average-linkage method calculates similarity between a pair of cluster groups as the average similarity among entities in the two groups. Species information is used to compute similarity index values. Grouped stations were clustered at a conservative distance limit of 50-60% similarity, however, this level was purely arbitrary. Because classification analyses have the tendency to force data into artificially distinct groups, another method (*e.g.*, multi-dimensional scaling) was used to confirm the validity of group clusters and site similarity. Ordination analysis was useful because it enables one to see multidimensional gradients in data rather than just groupings (Smith, personal communication).

Multi-dimensional scaling (MDS) is used extensively in the analyses of benthic communities, particularly in estuarine and marine pollution studies. MDS is a procedure for fitting a set of points in space such that the distance between points correspond to a given set of dissimilarities. This technique is more flexible than principal co-ordinate analyses when handling the large number of zero counts generally characteristic of species-samples matrices. Nonmetric MDS analyses were performed using Systat®. For a detailed account of MDS statistical procedures, see Clarke and Ainsworth (1993) and Warwick and Clarke (1993). Inferences from the resultant ordination are also presented. It is important to note that, as with cluster analyses, MDS results are not definitive and must be used in conjunction with additional ecological information. MDS results are based on total species number and numbers of individuals. Inferences from the resultant ordination are also presented.

After classification and ordination patterns were determined, the raw data were reevaluated to assess which species may have influenced the observed patterns. Indicator species were then selected on the basis of a literature review (*i.e.,* distribution, life history strategies and habitat preference), by recommendations from other experienced benthic taxonomists, and review of the raw data. Initially, community analyses were conducted as a per "site" comparison. Later, it was decided analyses also be expanded to a per "station" comparison to produce a more definitive data set for the reference pool. The extended analysis of station variability was performed using the benthic index.

Benthic assemblages have many attributes which make them reliable and sensitive indicators of the ecological condition in estuarine environments. The following procedure summarizes the construction and application of the benthic index used to reliably discriminate between degraded and undegraded conditions at sites in the San Diego Bay Region. Although there are problems with trying to simplify complex biological communities, we attempted to develop a quantitative method which creates a partition between degraded and undegraded areas. Polluted sites can not be conclusively identified using results from benthic community analyses alone, but these analyses impartially describe "environmentally stressed" areas. This benthic index is based on species (indicators), and group (general taxa) information. The index also evaluates community parameters, such as species richness, and abundance or presence of pollution indicators, which identify the extremes of the community characteristics. Sites are ranked according to these extremes and are represented by a single value. In general, decreasing numbers of species, increasing numbers of individuals, and decreasing diversity values are common responses observed near polluted areas. These trends are incorporated into the index. One of the important restrictions with the existing method is it evaluates this limited San Diego Bay benthic data set when dividing groups for categorization. Construction and subsequent validation of this simplified benthic index are loosely based on criteria developed by several agencies, including USEPA-EMAP and SCCWRP. However, the benthic index developed by USEPA-EMAP (Weisberg *et al.*, 1993) included several environmental variables in its construction (*e.g.* dissolved O_2), while the index for San Diego Bay data used only biological parameters. Briefly, the following major steps were followed in constructing and validating this benthic index:

1. Degraded and undegraded (*i.e.,* reference condition) stations were identified on the basis of measured environmental and biological variables.
2. A list of "candidate" parameters was developed using species abundance data. The list included metrics having ecological relevance (*e.g.,* species diversity indices, etc.) which were used to discriminate between degraded and reference areas.
3. A value for each candidate parameter (*i.e.,* diversity, abundance, taxonomic composition) was calculated for each station (*e.g.,* total species per station, total individuals per station, total crustaceans species per station, total number of polychaete individuals, total amphipods per station, etc.).
4. Range of values per metric was determined (lowest to highest value).
5. Quartiles from that range were determined.
6. Ranking within quartiles were assigned: upper quartile=2, lower quartile=0, middle quartile=1. These calculations were applied to the metrics from step 3.

7. The index was defined by values of 0, 1, or 2. A value of 0 defines the degraded (detectable stress) stations(s), and 2 identifies environmentally undegraded stations(s). Stations with an index value of 1 are considered transitional communities, which are neither degraded nor reference stations. Transitional stations have species or other parameters which indicate both degraded and undegraded habitats. These stations are investigated further to determine the cause of ambiguity of the transitional status.

8. Relative abundance of indicator species (both degraded and undegraded habitat indicators) per station is assessed.

A primary concern regarding the benthic index is how well it fulfills the objective of discriminating among degraded and undegraded estuarine conditions. This simplified version forms the basis for ongoing iterative procedures involved in construction of an index. This index will include a variety of indicator values (Bascom *et al.*, 1978; Kerans *et al.*, 1994; EcoAnalysis *et al.*, 1995) for future applications of the assessment of benthic community structure. The following sections report results of benthic community analyses based solely on composition and abundance of macrobenthic species from sediment cores throughout San Diego Bay and its vicinity. Environmental parameters (*e.g.*, total organic carbon levels and sediment grain size range) and other factors capable of influencing benthic composition were examined, but not evaluated in conjunction with the data presented here. Those data are examined later in sections which address correlative analyses.

In this study, bioeffects are required to be demonstrated in relation to properly selected reference sites and to occur in association with significant pollutant levels. The following evidence for undegraded (possible reference) and degraded (possible contaminated) sites was based on benthic community "quality" at each site and station. Benthic community structure was evaluated as an indicator of environmentally degraded or undegraded areas and not as a pollution or contamination indicator. Benthic reference sites were determined predominantly by analyses of specific indicator species and groups (*e.g.*, amphipods). These species are generally not found in polluted or disturbed areas.

The intention of this section is to clearly describe the condition of macrobenthic communities from sampling areas. Definitions of degraded, transitional, and undegraded used in this section are adopted from several papers (Bascom *et al.*, 1978; Pearson and Rosenberg, 1978; Schindler, 1987; Swartz *et al.*, 1985; Underwood and Peterson, 1988). Although the boundaries set in Bascom *et al.* (1978) were based on food supply and not on toxicants, the same general principles apply to this study. In benthic analyses, the term "degraded" does not refer to a

community response to significant levels of toxic chemicals. Degraded areas are those which contain significant numbers of opportunistic species, in the absence of non-opportunistic species, and have relatively low species diversity. Correlations are later used to determine if community profiles are influenced by chemistry or by natural environmental disturbances. Sites and stations which are categorized as "undegraded" have high species diversity, high proportional abundance of amphipods and other crustaceans, while noting there are a few exceptions to this rule (*e.g., Grandidierella japonica*, etc.). Undegraded areas generally contain species which are known to be sensitive to pollutants. Transitional sites and stations are those which are not confidently partitioned into the other two categories. These areas may solicit further study. Overall, an integration of data from laboratory exposures, chemical analyses, and benthic community assessments provide strong complementary evidence of the degree of pollution-induced degradation in aquatic communities. The following data analyses were conducted on a per site basis using sample replicates (n=5) at each sampling location (Table 6). An analysis also was performed using per station data (n=1) and is presented later in this section. Tests included classification and ordination analyses, diversity measurements, construction of a benthic index, and assessment of indicator species. One cautionary note is each of the benthic community and population condition tests are subject to effects of not only the pollutants measured in this study, but many other confounding natural factors, such as depth, salinity, sediment texture, and/or predation.

Abundance and Diversity

There were 7,232 individuals, representing 198 macrobenthic species, collected from 375 benthic cores during sampling legs 20 through 23 of the San Diego Bay confirmation phase (Table 7). Mean number of species was calculated from 5 replicates per site (Table 8). Polychaetes comprised the majority of specimens in samples. Great numbers of mollusks in sites within West Basin, Downtown Piers, and Glorietta Bay were due to the bivalve *Musculista senhousei* which was collected as large aggregates. Echinoderms were found at only 6 of the 25 sites, and were significantly ($p>0.01$) greater at the Mission Bay A3 site (640.0±216.6) and the Mission Bay A8 site (213.3±53.3) compared to all other sites. Holothurians comprised the majority of echinoderms found at these sites, although ophiuroids were also present. Colonial species were not present. Diversity ranged from 9 to 46 benthic species per site in collected samples. Significant differences in species diversity were not as distinct as with other indices and no trends were obvious. Results shown in Table 9 indicate most communities in this study were relatively diverse and even. Simpson's diversity index (D') which emphasizes more common species, and Shannon-Weaver (H') which puts statistical weight on rare species, showed differences in the range of diversity values. Chula Vista Yacht Basin was the only site which showed a moderately high level of dominance as

Table 6. Benthic samples from the San Diego Bay region.

Site-Station Name	Replicate Number	Station No.	IDORG No.
10 Swartz (West Basin)	1	90050.0	837
10 Swartz (West Basin)	2	93199.0	838
10 Swartz (West Basin)	3	93200.0	839
10 Swartz (West Basin)	4	^	837.1
10 Swartz (West Basin)	5	^	837.2
11 Swartz (East Basin)	1	90001.0	840
11 Swartz (East Basin)	2	93201.0	841
11 Swartz (East Basin)	3	93202.0	842
11 Swartz (East Basin)	4	^	840.1
11 Swartz (East Basin)	5	^	840.2
12 Swartz (Downtown Anch)	1	90002.0	878
12 Swartz (Downtown Anch)	2	93221.0	879
12 Swartz (Downtown Anch)	3	93222.0	880
12 Swartz (Downtown Anch)	4	^	878.1
12 Swartz (Downtown Anch)	5	^	878.2
14 Swartz (Downtown Piers)	1	90003.0	846
14 Swartz (Downtown Piers)	2	93205.0	847
14 Swartz (Downtown Piers)	3	93206.0	848
14 Swartz (Downtown Piers)	4	^	846.1
14 Swartz (Downtown Piers)	5	^	846.2
15 Swartz (G St Pier Marina)	1	90004.0	849
15 Swartz (G St Pier Marina)	2	93207.0	850
15 Swartz (G St Pier Marina)	3	93208.0	851
15 Swartz (G St Pier Marina)	4	^	849.1
15 Swartz (G St Pier Marina)	5	^	849.2
16 Swartz (Intercont. Marina)	1	90051.0	818
16 Swartz (Intercont. Marina)	2	93192.0	819
16 Swartz (Intercont. Marina)	3	93193.0	820
16 Swartz (Intercont. Marina)	4	^	818.1
16 Swartz (Intercont. Marina)	5	^	818.2
23 Swartz (Naval Base 07)	1	90006.0	865
23 Swartz (Naval Base 07)	2	93212.0	866
23 Swartz (Naval Base 07)	3	93213.0	867
23 Swartz (Naval Base 07)	4	^	865.1
23 Swartz (Naval Base 07)	5	^	865.2
25 Swartz (Naval base/ SY 010)	1	90007.0	887
25 Swartz (Naval base/ SY 010)	2	93223.0	888
25 Swartz (Naval base/ SY 010)	3	93224.0	889
25 Swartz (Naval base/ SY 010)	4	^	887.1
25 Swartz (Naval base/ SY 010)	5	^	887.2
27 Swartz (Naval Base /SH 013)	1	90008.0	890
27 Swartz (Naval Base /SH 013)	2	93225.0	891
27 Swartz (Naval Base /SH 013)	3	93226.0	892
27 Swartz (Naval Base /SH 013)	4	^	890.1
27 Swartz (Naval Base /SH 013)	5	^	890.2
28 Swartz (7th St Channel Q1)	1	90009.0	893
28 Swartz (7th St Channel Q1)	2	93227.0	894
28 Swartz (7th St Channel Q1)	3	93228.0	895
28 Swartz (7th St Channel Q1)	4	^	893.1
28 Swartz (7th St Channel Q1)	5	^	893.2
31 Swartz (Marine Terminal R3)	1	90010.0	896
31 Swartz (Marine Terminal R3)	2	93229.0	897
31 Swartz (Marine Terminal R3)	3	93230.0	898
31 Swartz (Marine Terminal R3)	4	^	896.1
31 Swartz (Marine Terminal R3)	5	^	896.2
32 Swartz (Sweetwater Ch)	1	90052.0	875
32 Swartz (Sweetwater Ch)	2	93219.0	876
32 Swartz (Sweetwater Ch)	3	93220.0	877
32 Swartz (Sweetwater Ch)	4	^	875.1
32 Swartz (Sweetwater Ch)	5	^	875.2
34 Swartz (CV Yacht Basin)	1	90012.0	824
34 Swartz (CV Yacht Basin)	2	93196.0	825
34 Swartz (CV Yacht Basin)	3	93197.0	826
34 Swartz (CV Yacht Basin)	4	^	824.1
34 Swartz (CV Yacht Basin)	5	^	824.2
35 Swartz (Coronado Cays)	1	90053.0	843
35 Swartz (Coronado Cays)	2	93203.0	844
35 Swartz (Coronado Cays)	3	93204.0	845
35 Swartz (Coronado Cays)	4	^	843.1
35 Swartz (Coronado Cays)	5	^	843.2
37 Swartz (Marina)	1	90013.0	815
37 Swartz (Marina)	2	93190.0	816
37 Swartz (Marina)	3	93191.0	817
37 Swartz (Marina)	4	^	815.1
37 Swartz (Marina)	5	^	815.2
41 Swartz (Glorietta Bay)	1	90015.0	821
41 Swartz (Glorietta Bay)	2	93194.0	822
41 Swartz (Glorietta Bay)	3	93195.0	823
41 Swartz (Glorietta Bay)	4	^	821.1
41 Swartz (Glorietta Bay)	5	^	821.2
K Swartz (Naval Base 04)	1	90021.0	862
K Swartz (Naval Base 04)	2	93210.0	863
K Swartz (Naval Base 04)	3	93211.0	864
K Swartz (Naval Base 04)	4	^	862.1
K Swartz (Naval Base 04)	5	^	862.2
Mission Bay A4	1	93108.0	859
Mission Bay A4	2	93108.0	860
Mission Bay A4	3	93108.0	861
Mission Bay A4	4	93108.0	859.1
Mission Bay A4	5	93108.0	859.2
Mission Bay A8	1	93112.0	856
Mission Bay A8	2	93112.0	857
Mission Bay A8	3	93112.0	858
Mission Bay A8	4	93112.0	856.1
Mission Bay A8	5	93112.0	856.2
Mission Bay A3	1	93107.0	853
Mission Bay A3	2	93107.0	854
Mission Bay A3	3	93107.0	855
Mission Bay A3	4	93107.0	853.1
Mission Bay A3	5	93107.0	853.2
NSB-M1 (Sub Base C2)	1	90028.0	871
NSB-M1 (Sub Base C2)	2	93216.0	872
NSB-M1 (Sub Base C2)	3	93217.0	873
NSB-M1 (Sub Base C2)	4	^	871.1
NSB-M1 (Sub Base C2)	5	^	871.2
P Swartz (Naval Base 012)	1	90022.0	868
P Swartz (Naval Base 012)	2	93214.0	869
P Swartz (Naval Base 012)	3	93215.0	870
P Swartz (Naval Base 012)	4	^	868.1
P Swartz (Naval Base 012)	5	^	868.2
San Diego River B1	1	93116.0	881
San Diego River B1	2	93116.0	882
San Diego River B1	3	93116.0	883
San Diego River B1	4	93116.0	881.1
San Diego River B1	5	93116.0	881.2
SDNI- N5 (Carrier Base V2)	1	90025.0	899
SDNI- N5 (Carrier Base V2)	2	93231.0	1000
SDNI- N5 (Carrier Base V2)	3	93232.0	1001
SDNI- N5 (Carrier Base V2)	4	^	899.1
SDNI- N5 (Carrier Base V2)	5	^	899.2
Stormdrain EM (Grape St.)	1	90037.0	827
Stormdrain EM (Grape St.)	2	90037.0	828
Stormdrain EM (Grape St.)	3	90037.0	829
Stormdrain EM (Grape St.)	4	90037.0	827.1
Stormdrain EM (Grape St.)	5	90037.0	827.2
Long Beach Outer Harbor	1	40018.3	884
Long Beach Outer Harbor	2	40018.3	885
Long Beach Outer Harbor	3	40018.3	886
Long Beach Outer Harbor	4	40018.3	884.1
Long Beach Outer Harbor	5	40018.3	884.2
Lower Main Channel	1	40004.2	830
Lower Main Channel	2	40004.2	831
Lower Main Channel	3	40004.2	832
Lower Main Channel	4	40004.2	830.1
Lower Main Channel	5	40004.2	830.2
Off Cabrillo Beach	1	40010.0	1006
Off Cabrillo Beach	2	40010.0	1007
Off Cabrillo Beach	3	40010.0	1008
Off Cabrillo Beach	4	40010.0	1006.1
Off Cabrillo Beach	5	40010.0	1006.2
Palos Verdes (Swartz 6)	1	40031.2	1002
Palos Verdes (Swartz 6)	2	40031.2	1003
Palos Verdes (Swartz 6)	3	40031.2	1004
Palos Verdes (Swartz 6)	4	40031.2	1002.1
Palos Verdes (Swartz 6)	5	40031.2	1002.2
West Basin Entrance	1	40009.1	834
West Basin Entrance	2	40009.1	835
West Basin Entrance	3	40009.1	836
West Basin Entrance	4	40009.1	834.1
West Basin Entrance	5	40009.1	834.2

Table 7. Species list of macroinvertebrates from the San Diego Bay region benthic samples

Acmira catherinae	Gastropoda	*Fabricinuda limicola*	Polychaeta	*Orchomene pacifica*	Gammaridea
Acmira horikoshii	Gastropoda	*Glycera americana*	Polychaeta	*Orchomene sp.*	Gammaridea
Acuminodeutopus heteruropus	Amphipoda	*Glycera nana*	Polychaeta	*Paracerceis sculpta*	Isopoda
Aglaja sp.	Gastropoda	*Gnathia crenulatifrons*	Isopoda	*Paradexamine sp.*	Amphipod
Alpheus californiensis	Decapoda	*Goniada brunnea*	Polychaeta	*Paramage scutata*	Polychaeta
Amaeana occidentalis	Polychaeta	*Goniada sp(p).*	Polychaeta	*Paranthura elegans*	Isopoda
Ampelisca brevisimulata	Gammaridea	*Grandidierella japonica*	Gammaridea	*Paraprionospio pinnata*	Polychaeta
Ampelisca cristata	Gammaridea	*Harmothoe hirsuta*	Polychaeta	*Parasterope barnesi*	Ostracoda
Ampelisca hancocki	Gammaridea	*Harmothoe imbricata*	Polychaeta	*Parougia caeca*	Polychaeta
Ampharete labrops	Polychaeta	*Heptacarpus cf taylori*	Decapoda	*Parvilucina tenuisculpta*	Bivalvia
Amphicteis scaphobranchiata	Polychaeta	*Heptacarpus sp. A*	Decapoda	*Pectinaria californiensis*	Polychaeta
Amphideutopus oculatus	Amphipoda	*Hesperonoe sp(p).*	Polychaeta	*Pennatulacea*	Anthozoa
Amphilochidae	Gammaridea	*Heterophoxus oculatus*	Gammaridea	*Pherusa capulata*	Polychaeta
Ampithoe sp.	Gammaridea	unidentified holothuroid	Holothuroidea	*Pherusa sp(p).*	Polychaeta
unid. anemone	Anthozoa	*Hyale frequens*	Gammaridea	*Pholoe glabra*	Polychaeta
Aphelochaeta monilaris	Polychaeta	*Hydroides pacificus*	Polychaeta	unidentified phoronida	Phoronida
Aphelochaeta multifilis	Polychaeta	*insect larva*	Arthropoda	*Photis sp.*	Gammaridea
Aphelochaeta sp(p).	Polychaeta	*Laevicardium substriatum*	Bivalvia	*Pista alata*	Polychaeta
Apistobranchus sp(p).	Polychaeta	*Laonice cirrata*	Polychaeta	*Pista sp(p).*	Polychaeta
Aporprionospio pygmaea	Polychaeta	*Leitoscoloplos pugettensis*	Polychaeta	*Pleustidae*	Gammaridea
Armandia brevis	Polychaeta	*Lembos sp.*	Gammaridea	*Podarkeopsis glabra*	Polychaeta
Asteropella slatteryi	Ostracoda	*Leptochelia dubia*	Tanaidacea	*Podarkeopsis perkinsi*	Polychaeta
Autolytus sp(p).	Polychaeta	*Leptognathia sp.*	Tanaidacea	*Podocerus cristatus*	Gammaridea
unidentified bivalve	Bivavia	*Levinsenia gracilis*	Polychaeta	*Poecilochaetus johnsoni*	Polychaeta
Brania brevipharyngea	Polyehaeta	*Listriella goleta*	Gammaridea	*Polydora cornuta*	Polychaeta
Bulla sp.	Gastropoda	*Lophopanopeus bellus diegensis*	Decapoda	*Polydora nuchalis*	Polychaeta
Campylaspis rubromaculata	Cumacea	Lumbrineridae, unident.	Polychaeta	*Polydora socialis*	Polychaeta
Capitella capitata complex	Polychaeta	*Lyonsia californica*	Bivalvia	*Polyophthalmus pictus*	Polychaeta
Caprella californica	Caprellida	*Lysippe labiata*	Polychaeta	*Pontogeneia rostrata*	Gammaridea
Caulleriella sp(p).	Polychaeta	*Macoma cf yoldiformis*	Bivalvia	*Praxillella pacifica*	Polychaeta
Chaetozone corona	Polychaeta	*Macoma nausta*	Bivalvia	*Prionospio heterobranchia*	Polychaeta
Chone mollis	Polychaeta	*Macoma sp.*	Bivalvia	*Prionospio lighti*	Polychaeta
Cirratulidae, unident.	Polychaeta	*Mactra californica*	Bivalvia	*Prionospio sp(p).*	Polychaeta
Cirratulus sp(p).	Polychaeta	*Malmgreniella macginitiei*	Polychaeta	*Prionospio steenstrupi*	Polychaeta
Cirriformia luxuriosa	Polychaeta	*Marphysa disjuncta*	Polychaeta	*Pseudopolydora paucibranchiata*	Polychaeta
Collisela depicta	Gastropoda	*Mayerella banksia*	Amphipoda	*Rhynchospio glutaea*	Polychaeta
Compsomyax subdiaphana	Bivalvia	*Mediomastus californiensis*	Polychaeta	*Rudilemboides stenopropodus*	Amphipoda
Cooperella subdiaphana	Bivalvia	*Megalomma pigmentum*	Polychaeta	*Scleroplax granulata*	Decapoda
Corophium acherusicum	Gammaridea	*Melinna oculata*	Polychaeta	*Scolelepis quinquedentata*	Polychaeta
Corophium heteroceratum	Gammaridea	*Metasychis disparidentata*	Polychaeta	*Scoletoma erecta*	Polychaeta
Cossura candida	Polychaeta	*Microjassa litotes*	Gammaridea	*Scoletoma tetraura*	Polychaeta
Crepidula fornicata	Gastropoda	*Monoculodes hartmanae*	Gammaridea	*Scoloplos acmeceps*	Polychaeta
Crucibulum spinosum	Gastropoda	*Monticellina dorsobranchialis*	Polychaeta	*Scyphoproctus sp(p).*	Polychaeta
Cryptomya californica	Bivalvia	*Monticellina sp. C*	Polychaeta	*Serolis carinata*	Isopoda
Cylichnella inculta	Gastropoda	*Monticellina tesselata*	Polychaeta	*Sigambra tentaculata*	Polychaeta
Cylichnella sp.	Gastropoda	*Munnogonium californiensis*	Isopoda	*Siliqua lucida*	Bivalvia
Diastylis sp.	Cumacea	*Musculista senhousei*	Bivalvia	unidentified spionid	Polychaeta
Diopatra sp(p).	Polychaeta	*Myriochele sp. M*	Polychaeta	*Spiophanes berkeleyorum*	Polychaeta
Diopatra tridentata	Polychaeta	*Mysella sp.*	Bivalvia	*Spiophanes missionensis*	Polychaeta
Diplocirrus sp(p).	Polychaeta	unidentified mysid	Mysidacea	*Sthenelais tertiaglabra*	Polychaeta
Dorvillea longicornis	Polychaeta	*Nassarius perpinguis*	Gastropoda	*Sthenelanella uniformis*	Polychaeta
Drilonereis falcata minor	Polychaeta	*Neanthes acuminata*	Polychaeta	*Streblosoma sp. B*	Polychaeta
Elasmopus rapax	Amphipoda	*Neastacilla californica*	Isopoda	*Streblospio benedicti*	Polychaeta
Eranno lagunae	Polychaeta	*nemertean*	Nemertea	*Sulcoretusa xystrum*	Gastropoda
Eteone californica	Polychaeta	*Neotrypaea californiensis*	Decapoda	*Synchelidium rectipalmum*	Gammaridea
Eteone sp(p).	Polychaeta	*Nephtys caecoides*	Polychaeta	*Synchelidium sp.*	Gammaridea
Euchone limnicola	Polychaeta	*Nephtys cornuta*	Polychaeta	*Tagelus subteres*	Bivalvia
Euclymeninae spp. indet.	Polychaeta	Nereididae, unident.	Polychaeta	*Tellina modesta*	Bivalvia
Eudorella pacifica	Cumacea	*Nereis procera*	Polychaeta	*Tenonia priops*	Polychaeta
Euphilomedes carcharodonta	Ostracoda	*Notomastus tenuis*	Polychaeta	Terebellidae, unident.	Polychaeta
Euphilomedes producta	Ostracoda	*Nuculana taphria*	Bivalvia	*Terebellides californica*	Polychaeta
Eupolymnia sp(p).	Polychaeta	*Odontosyllis phosphorea*	Polychaeta	*Theora fragilis*	Bivalvia
Exogone lourei	Polychaeta	*Odostomia sp.*	Gastropoda	*Trachycardium quadragenarium*	Bivalvia
Exogone molesta	Polychaeta	*oligochaeta*	Oligochaeta	*Turbonilla sp.*	Gastropoda
Exogone sp(p)	Polychaeta	*Olivella baetica*	Gastropoda	*Urocaris infraspinis*	Decapoda
Exogone uniformis	Polychaeta	unidentified ophiuroid	Ophioroidea	*Zeuxo normani*	Tanaidacea

Table 8. Mean densities and standard error of higher taxonomic groups at each site in San Diego Bay. Total area sampled (0.04m2) at each site was from 5 replicate cores.

SITES	ID ORG #	Polychaetes mean #/m2 ± SE	Mollusks mean #/m2 ± SE	Crustaceans mean #/m2 ± SE	Echinoderms mean #/m2 ± SE
10 Swartz (West Basin)	837	4,986.5 ± 481.8	2,213.3 ± 1,211.5	5,199.9 ± 792.2	
11 Swartz (East Basin)	840	5,599.9 ± 1,654.0	640.0 ± 319.4	7,946.5 ± 2,605.1	26.7 ± 26.7
14 Swartz (Downtown Piers)	846	4,213.2 ± 822.5	2,453.3 ± 1,865.5	1,146.6 ± 449.4	
15 Swartz (G St Pier Marina)	849	4,106.6 ± 694.6	1,040.0 ± 508.4	1,120.0 ± 123.6	
16 Swartz (Intercont. Marina)	818	3,893.2 ± 824.5	1,146.6 ± 629.1	2,853.3 ± 905.9	
23 Swartz (Naval Base 07)	865	5,119.9 ± 1,427.1	106.7 ± 77.7	373.3 ± 154.3	
25 Swartz (Naval base/ SY 010)	887	2,639.9 ± 932.7	53.3 ± 53.3	53.3 ± 32.7	
27 Swartz (Naval Base /SH 013)	890	2,373.3 ± 268.0	133.3 ± 73.0	293.3 ± 165.5	
28 Swartz (7th St Channel Q1)	893	2,000.0 ± 944.7	80.0 ± 53.3	26.7 ± 26.7	
31 Swartz (Marine Terminal R3)	896	4,373.2 ± 1,827.4	746.6 ± 225.5	853.3 ± 466.8	
32 Swartz (Sweetwater Ch)	875	5,066.5 ± 1,224.2	213.3 ± 181.8	1,013.3 ± 459.2	
34 Swartz (CV Yacht Basin)	824	10,426.4 ± 2,264.4	373.3 ± 154.3	800.0 ± 332.0	
35 Swartz (Coronado Cays)	843	4,986.5 ± 1,506.5	320.0 ± 171.8	3,199.9 ± 370.0	26.7 ± 26.7
37 Swartz (Marina)	815	4,399.9 ± 1,141.5	426.7 ± 160.0	1,626.6 ± 351.2	
41 Swartz (Glorietta Bay)	821	10,106.4 ± 532.3	5,066.5 ± 2,724.3	1,493.3 ± 816.9	
K Swartz (Naval Base 04)	862	2,799.9 ± 480.7	906.6 ± 208.3	853.3 ± 293.9	
NSB-M1 (Sub Base C2)	871	4,266.6 ± 668.0	1,013.3 ± 149.7	1,146.6 ± 200.4	53.3 ± 53.3
P Swartz (Naval Base 012)	868	4,799.9 ± 808.8	533.3 ± 279.7	533.3 ± 245.8	
SDNI- N5 (Carrier Base V2)	899	7,733.1 ± 2,003.5	1,946.6 ± 512.2	2,000.0 ± 511.2	
12 Swartz (Downtown Anch)	878	3,893.2 ± 760.6	1,333.3 ± 865.1	2,159.9 ± 586.3	
Mission Bay A3	853	1,600.0 ± 152.0	1,440.0 ± 330.4	533.3 ± 242.2	640.0 ± 216.6
Mission Bay A4	859	2,186.6 ± 422.9	213.3 ± 149.7	933.3 ± 467.6	53.3 ± 32.7
Mission Bay A8	856	11,573.0 ± 761.7	320.0 ± 90.4	3,599.9 ± 1,096.2	213.3 ± 53.3
San Diego River B1	881	2,426.6 ± 1,062.0	26.7 ± 26.7	800.0 ± 173.8	
Stormdrain EM (Grape St.)	827	4,239.9 ± 534.0	53.3 ± 53.3	3,813.2 ± 1,345.6	

Table 9. Macrobenthic community variables at sites in San Diego bay. Biological parameters derived from 5 replicate samples per site Physical measurements are from an average of the 3 stations.

SITES	depth (m)	silt:clay (%)	TOC	Total no. of species	Mean no. indiv./m2	Simpson's diversity D	inverse (1/D) diversity	V' evennness	Shannon-W diversity H'	J' evenness	habitat
32 Swartz (Sweetwater Ch)	6	64.49	0.97	31	6,426.5	0.161	6.211	0.005	3.514	0.709	E-sandy
11 Swartz (East Basin)	3	52.71	1.33	35	14,586.3	0.124	8.065	0.004	3.719	0.725	S,Sb
16 Swartz (Intercont. Marina)	4	59.68	1.04	32	8,106.5	0.086	11.628	0.003	4.037	0.807	S,Sb
37 Swartz (Marina)	3	92.77	1.45	29	6,586.5	0.101	9.901	0.003	3.833	0.789	S,Sb
Stormdrain EM (Grape St.)	8	82.47	1.97	33	8,239.8	0.071	14.085	0.002	4.152	0.823	E,Sb
10 Swartz (West Basin)	3	75.36	1.46	34	12,399.7	0.094	10.638	0.003	3.910	0.769	S,Sb
14 Swartz (Downtown Piers)	11	54.59	1.30	37	7,919.8	0.088	11.364	0.002	4.112	0.789	E
15 Swartz (G St Pier Marina)	5	77.25	4.08	33	6,586.5	0.074	13.514	0.002	4.194	0.831	E
41 Swartz (Glorietta Bay)	5	50.00	1.05	28	16,879.6	0.163	6.135	0.006	3.296	0.686	S,Sb
K Swartz (Naval Base 04)	5	62.79	2.23	21	4,586.6	0.129	7.752	0.006	3.481	0.793	E,N
SDNI- N5 (Carrier Base V2)	7	65.80	1.81	46	11,839.7	0.075	13.333	0.002	4.342	0.786	E,N
12 Swartz (Downtown Anch)	5	73.73	1.83	31	7,439.8	0.094	10.638	0.003	3.985	0.804	E
23 Swartz (Naval Base 07)	8	55.09	1.74	29	5,706.5	0.124	8.065	0.004	3.621	0.745	E,N
25 Swartz (Naval base/ SY 010)	9	71.89	1.92	20	2,799.9	0.141	7.092	0.007	3.324	0.769	E,N
27 Swartz (Naval Base /SH 013)	10	71.16	1.90	21	2,826.6	0.111	9.009	0.005	3.631	0.827	E,N
31 Swartz (Marine Terminal R3)	6	61.51	1.58	32	6,079.8	0.142	7.042	0.004	3.634	0.727	E
34 Swartz (CV Yacht Basin)	3	90.40	1.39	33	11,866.4	0.368	2.717	0.011	2.474	0.490	S
35 Swartz (Coronado Cays)	3	82.29	1.39	30	8,613.1	0.103	9.709	0.003	3.847	0.784	S,N
NSB-M1 (Sub Base C2)	10	62.67	1.64	43	6,746.5	0.101	9.901	0.002	4.087	0.753	E,N
P Swartz (Naval Base 012)	10	69.63	2.07	28	5,866.5	0.108	9.259	0.004	3.744	0.779	E,N
Mission Bay A4 REF	2	65.70	1.63	37	3,599.9	0.069	14.493	0.002	4.460	0.856	M
28 Swartz (7th St Channel Q1)	7	45.97	1.73	15	2,159.9	0.178	5.618	0.012	3.156	0.808	nd
Mission Bay A8 REF	5	36.99	0.89	44	15,866.3	0.085	11.765	0.002	4.109	0.753	M
Mission Bay A3 REF	3	93.21	2.98	27	5,653.2	0.130	7.692	0.005	3.516	0.739	M
San Diego River B1 REF	1	76.19	2.31	9	3,466.6	0.332	3.012	0.037	2.081	0.656	M
					Value range=	0-1	1-s	0-1	<5, max=log S	0-1	

E=exposed, S=sheltered, Sb=small boats, N=navy, C=channel, M=Mission Bay

shown by the evenness index (J'=0.490). This was due to an abundance of *Mediomastus californiensis* and *Leitoscoloplos pugettensis* polychaetes. Compared to all other sites, Chula Vista had a significantly lower density of crustaceans. The Mission Bay A4 site had moderately high species diversity but comparatively low species abundance.

Cluster and Ordination Analyses

Cluster analyses produced the dendrogram (Figure 15) of station affinities, based on mean root-root transformed abundance of the 198 macrobenthic species, using Pearson's correlation of similarity and group-average sorting. A root-root transformation, reduced the weighting of abundant species (Field *et al.*, 1982). The similarity level, although arbitrary, was designated somewhat conservatively near 50%. The resulting classification of assemblages reflect general patterns of benthic species composition, domination, and evenness (*e.g.*, sites along the 0.00 line would be identical in species composition and abundance). Six major groups were delineated from the hierarchical clusters, which were defined by an overall dominant species. Group I, which included only a single site (32 Swartz, Sweetwater Channel) was co-dominated by the tube-building tanaid *Zuexo normandi* and polychaete worm *Leitoscoloplos pugettensis*. Groups IV, V and VI were all dominated by the polychaete worm species *L. pugettensis*, *Prionospio heterobranchia*, and co-dominants *P. heterobranchia* and oligochaetes, respectively. Amphipods (*Acuminodeutopus heteruropus*) were the most abundant group in cluster II. The seemingly ubiquitous bivalve *Musculista senhousei* was the numerically important species in Group III. When plotted, these biologically-based clusters provide a qualitative assessment of the pattern of physical data and visually demonstrate the relationship of one site to another. To put the relationship of samples into a more general perspective, the level of similarity found between San Diego Bay site samples and those from Los Angeles Harbor was between 5-10% (Figure 16), revealing the benthos of these northerly areas should not be used comparatively, due to differences in habitats and biotic response. Although tidally influenced, the species composition of the San Diego River B1 site was also found to be highly dissimilar to other San Diego Bay samples, presumably due to habitat differences.

In addition to conventional methods, non-metric multi-dimensional scaling (MDS) using a weighted Spearman rank correlation coefficient dissimilarity matrix was used to determine similarity in species composition between stations. Non-metric MDS can handle large numbers of zeros, missing data, and unequal replication. MDS seeks a representation of individuals in a space of low dimensionality where the distances between individuals in ordination space optimally represent their dissimilarities in variable space (Kenkel and Orloci, 1986). Typically, transformed biotic and abiotic data are initially analyzed separately, then combined to assess common MDS spatial patterns. The resulting ordination for biotic variables is demonstrated here.

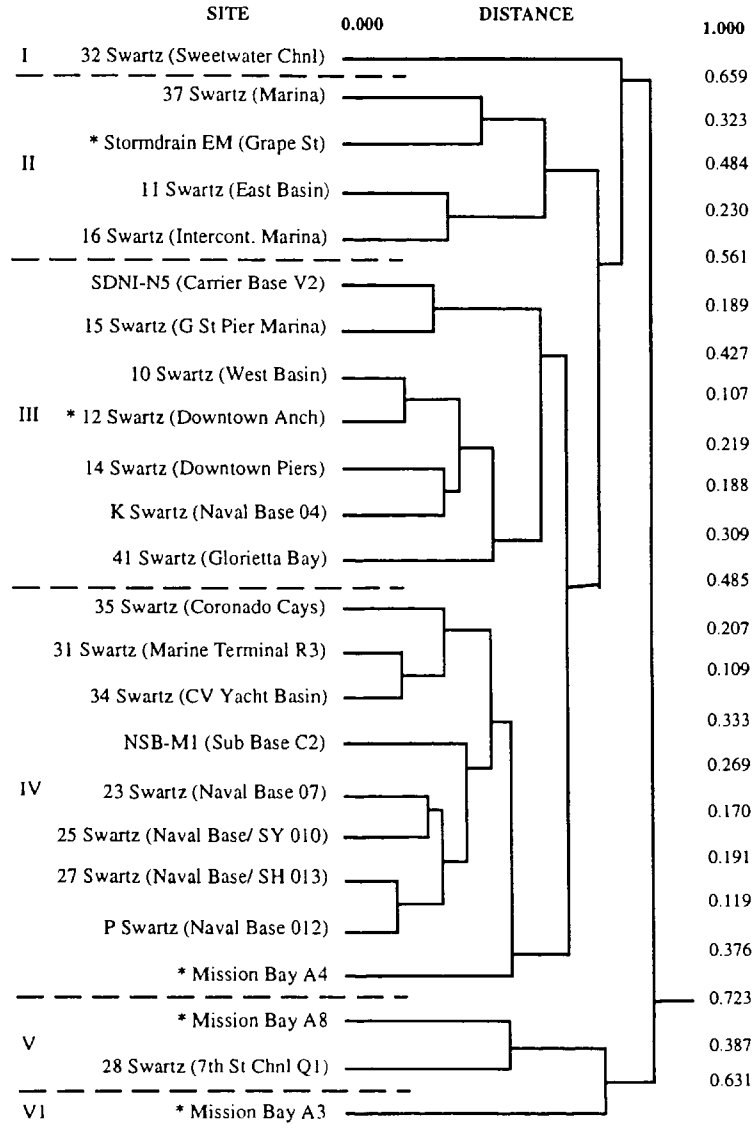


Figure 15. Numerical classification of mean abundance data of 198 macrobenthic species. Clusters are derived from Pearson correlation matrix data and group-average sorting. Six major clusters are shown, each dominated by 1-2 species.

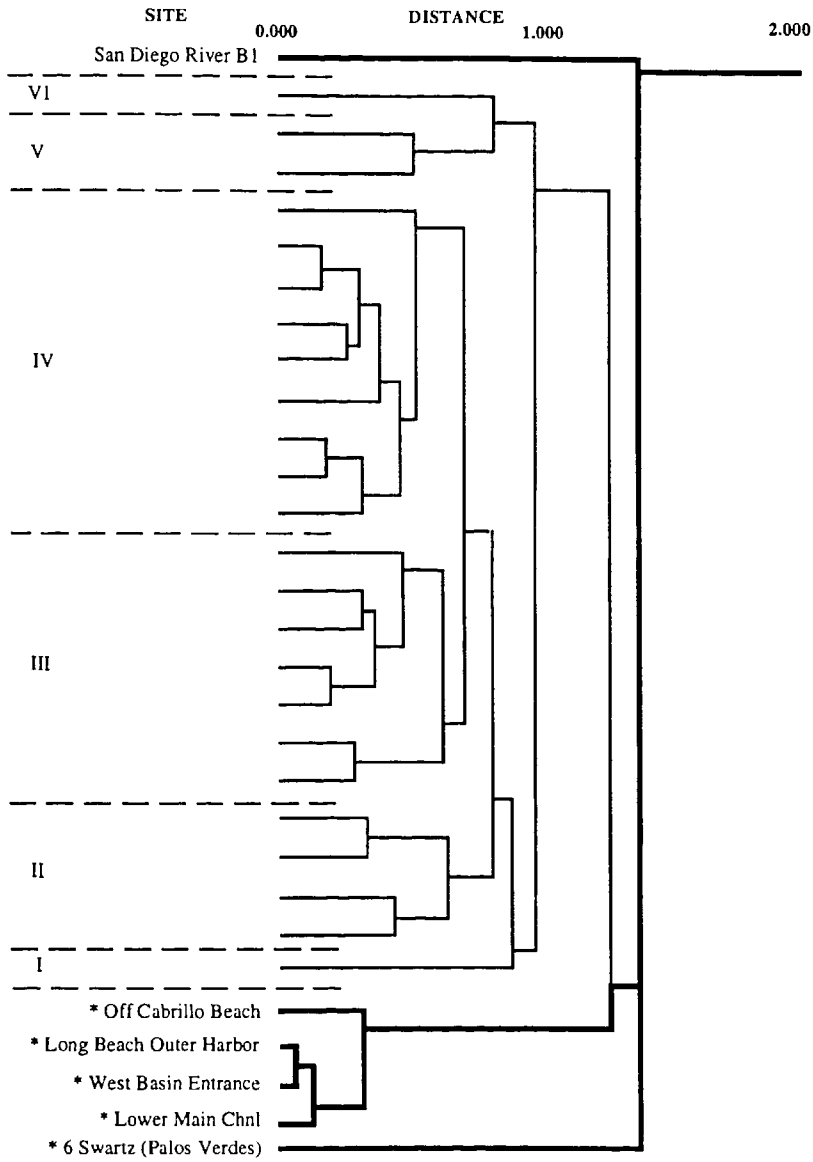


Figure 16. Numerical classification of mean abundance data from San Diego Bay and vicinity and Los Angeles Harbor.

PLOT	VARIABLE/SITE	CORRELATION CLUSTER NO.	PLOT	VARIABLE/SITE	CORRELATION CLUSTER NO.
A	West Basin, Swartz 10	III	N	Marina, Swartz 37	II
B	East Basin, Swartz 11	II	O	Glorietta Bay, Swartz 41	III
C	Downtown Piers, Swartz 14	III	P	Naval Base 04, Swartz K	III
D	G St Pier Marina, Swartz 15	III	Q	Sub Base C2, NSB-M1	IV
E	Intercont. Marina, Swartz 16	II	R	Naval Base 012, Swartz P	IV
F	Naval Base 07, Swartz 23	IV	S	Carrier Base V2, SDNI-N5	III
G	Naval Base/ SY 010, Swartz 25	IV	T	San Diego River B1	nd
H	Naval Base/ SH 013, Swartz 27	IV	U	Stormdrain EM, Grape St	II
I	7th Channel Q1, Swartz 28	V	V	Downtown Anch, Swartz 12	III
J	Marine Terminal R3, Swartz 31	IV	W	Mission Bay A3	VI
K	Sweetwater Channel, Swartz 32	I	X	Mission Bay A4	IV
L	CV Yacht Basin, Swartz 34	IV	Y	Mission Bay A8	V
M	Coronado Cays, Swartz 35	IV			

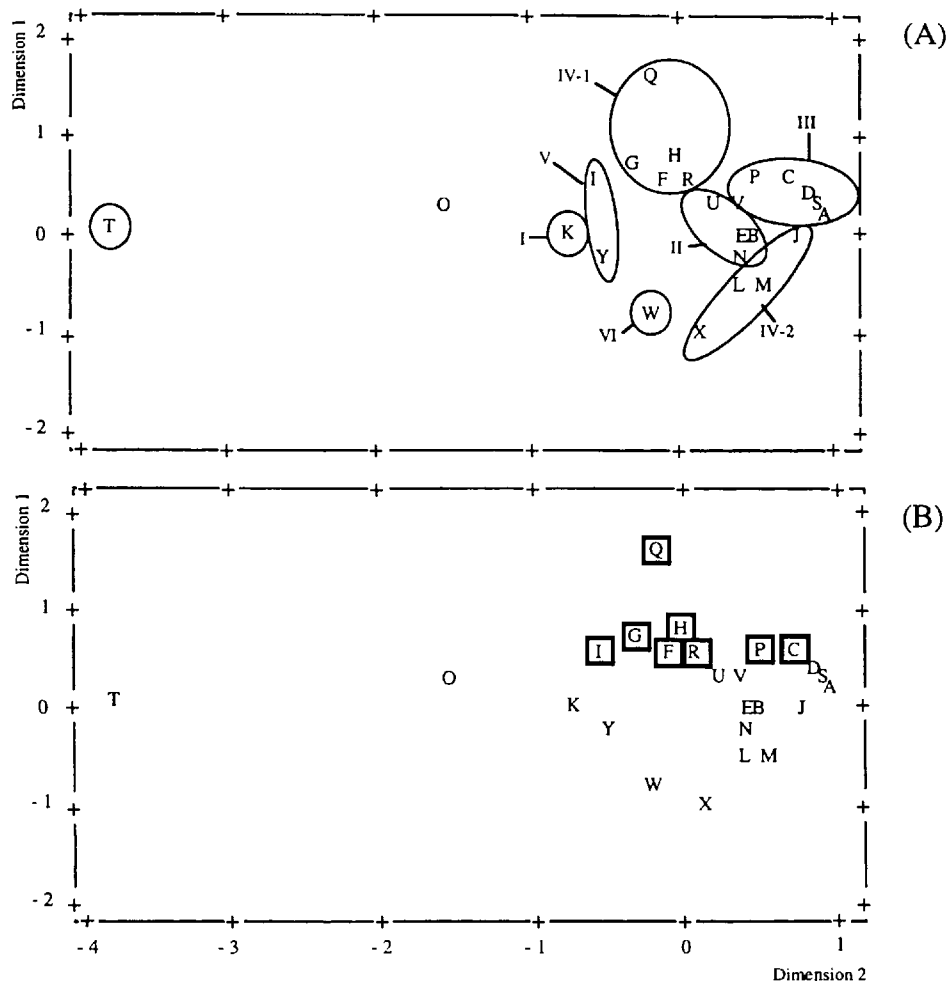


Figure 17. Multidimensional scaling (ms) ordination of site samples from San Diego Bay based on the abundance matrix of 198 macrobenthic species. (A) Clusters deliniated and numbered in Figure 15 dendogram are shown here as circled groups. (B) Qualitative assessment of the relation of chemistries >ERM levels (site codes surrounded by boxes) to ms biotic configuration.

displays the 2-dimensional representation resulting from multidimensional scaling, using the same matrix data applied to classification analysis. Letters surrounded by each circle represent the partitioned cluster groups delineated in the cluster hierarchy. The configuration was not altered when the outlier (T) was removed. The x- and y-axes represent scores for the first and second ordination axes. These scores are based on species diversity data and abundance and composition data.

When sites with chemistry values which exceeded ERM levels were assessed on the MDS plot in a qualitative, cursory manner as shown in Figure 17b (shown with squares), the sites clustered together. When interpreted along the axis gradient, these data suggested dimension 1 likely defined the pollution gradient, where the top quadrant within the plot identified the most contaminated sites (*i.e.*, Q or H). This is assuming the plot configuration is affected by toxic pollution alone and not by any organic enrichment. The y-axis may represent responses to a salinity gradient or change in sediment grain size. These analyses are especially revealing when environmental variables (*e.g*, TOC, grain size, water depth, total PAHs, individual metals, etc.) and biota are scaled together to determine which variables influence the configuration. However, even in the absence of these parallel plots, patterns are apparent from the correlations illustrated in other sections of this report.

Indicator Species

Despite the numerous studies performed in San Diego Bay, there have been no analyses of the fauna as bioindicators (SCCWRP-Diener, personal communication). Indicator species are assessed to determine which species are responsible for the separation of groups in classification and ordination analyses (Field *et al.*, 1982). Indicator species used in this study were selected on the basis of overall abundance in the San Diego Bay data set, literature review which determined distribution, known life histories and habitat preference, and discussions with ecologists experienced with Southern California marine biota and marine habitats. Species indicative of control or reference sites were derived from frequency of occurrence data. The presence or absence of specific polychaetes in sediments provided one valuable indication of the condition or health (Pocklington and Wells, 1992) of the benthic communities in San Diego Bay. The presence of *Capitella capitata* or *Streblospio benedicti*, in the absence of other species, is widely accepted as pollution indicators. Sensitive species like *Harmothoe imbricata* are represented at sites Carrier Base V2 and Mission Bay A8, and are typically found in uncontaminated areas. Additionally, *Nereidae* are accepted as indicators of early successional phases of environmental recovery (Pearson and Rosenberg, 1978) and are evident at site Carrier Base V2. *Mediomastus* polychaetes are found throughout the bay and have been considered to be identifiers of environmentally stressed areas. However, this species was found at the majority of sites. Another common species found in 16 out of 25 station samples was *Diplocirrus* sp.

which had not been found in previous studies in San Diego Bay (SCCWRP, personal communication). *Dipolocirrus* sp. was significantly ($p>0.05$) abundant at the Mission Bay A8 site. This unusual species is thought to have been introduced from the arctic region (G. Ruff, personal communication).

The benthic index discussed later was used to rank and calculate site partitions using the following indicator species: *Capitella capitata* (polychaete), *Armandia brevis* (polychaete), *Dorvillea longicornis* (polychaete), *Heterophoxus oculatus* (gammarid amphipod), and *Diastylis* sp. (cumacean). The polychaete worm *C. capitata* is widely accepted as a pollution indicator. *Diastylis* sp. ("sand-licker") feeds on nutrients adhered to sand grains and its presence indicates a relatively clean sample. Although it can tolerate moderately contaminated sediments, *H. oculatus* is a burrower and is considered an indicator of clean sediment.

One of the limitations in benthic community assessment is that patterns are more apparent where there is a strong gradient of pollutants, or when samples are selected from areas with distinctively low and high pollutant signals. There are limitations to what can be surmised from analyses of abundance of specific species, and selection of indicator species are highly site specific (Swartz *et al*., 1985). However, these species, combined with information from ordination and other supplemental analyses, make it apparent that these are important as ecologically relevant data. Many species used to assess environmental quality are used because they respond quickly to changes in environmental conditions. (Pocklington and Wells, 1992). Therefore, a station designated in the initial phases of sample collection as a having reference conditions, based on toxicity test or chemical analysis results, could be removed from the reference station list based on subsequent benthic community analyses.

Benthic Index

Benthic communities, and occasionally single benthic species, have been used to elucidate the severity of human disturbance to nearshore marine and estuarine environments. It is possible to develop a comparable disturbance classification for species and use a simple numerical infaunal index with these species. Distinct pollution gradients are rare in most embayments because of confounding environmental gradients and historical changes. Still, an index has the best potential to quantitatively assess benthic community responses to disturbance. Some benthic indices are based on *a priori* information and are developed using test sites representing the extremes within a range of environmental conditions which adversely affect benthos. In contrast, the index developed and used in this study was based solely on information which characterized the benthic community, such as specific indicator species and community parameters (species richness, abundance, presence of pollution indicator species, etc.). This elementary index approach may be best for this study because San Diego Bay encompasses a variety of habitats, each of which may

require a very specific set of index variables (SCCWRP-Diener, personal communication). Note that identification of degraded and undegraded sites here resulted from evaluation of a limited data set, without site comparison to an existing known reference. The index was used within this limited data set to designate the partition between degraded, undegraded and transitional areas.

Site and Station Application of Benthic Index

Table 10 shows the results of benthic index application to data from sampling sites in legs 20-23. Sites (25 sites with 5 replicates each) were ranked and partitioned into 9 degraded, 3 undegraded and 13 transitional sites using 8 biotic parameters. Due to spatial differences in sampling of the benthic replicates at the 25 sites, the benthic index was also applied to individual stations (n=75). When benthic community structure was evaluated "by site", 5 replicates were used. Replicates 1, 2 and 3 were sampled at numbered stations locations (Table 6) where associated toxicity and chemistry data could be directly compared. When later analyses were expanded to a "by station" evaluation, the 4th and 5th replicates were not included in the per station assessment. These replicates were randomly sampled within the "site" for benthic community analysis only and did not receive synoptic chemistry and toxicity analysis. While the results did not alter the degraded and undegraded determination of sites assessed "by site", it did separate stations within the initial "transitional" status into one of the three categories (*e.g.*, degraded, transitional or undegraded). Station analyses heavily emphasized benthic index, amphipod abundance, species diversity and crustacean numbers.

As part of analytical procedure, the BPTCP Scientific Planning and Review Committee (SPARC) recommended additional emphasis on the use of amphipod abundance and overall species diversity as indicators of degraded and undegraded areas. These parameters were assessed and incorporated into the "station evaluation" versions of the benthic index. Species number and abundance of amphipods were calculated from the proportions of total species and total individuals, respectively. The resultant categorization of stations into one of the three partitions (*e.g.*, degraded, transitional, undegraded) did not change, so the assessment of amphipods further supported the partition derived from previous analyses. The density of all amphipods was significantly more abundant at the following stations: West Basin (90050, 93199, 93200), East Basin (90001, 93201), Downtown Anchorage (93221, 93222), Coronado Cays (90053, 93203), Sweetwater Channel (93220), Mission Bay A8 (93112), Carrier Base V2 (90025) and Grape St. Stormdrain (90037). No amphipods were found at stations 14 Downtown Piers (90003), Naval Base O7 (93212), Naval Base/SY O10 (93223, 93224), Naval Base/SH O13 (93225, 93226), 7th St. Channel Q1 (90009, 93227, 93228), Marine Terminal R3 (93229), K Swartz Naval Base O4 (93210), Sub Base C2 (93216, 93217), and Naval Base O12 (93215). Stations with abundant amphipods but dominated by *Grandidierella japonica* were evaluated with caution, because *G. japonica* has been found to be tolerant of high

Table 10. Results of Benthic Index application on San Diego Bay data. Benthic community condition based on mean abundance of 5 replicate samples per site. Community status indicates allocation of a station to an Index partition: 2-undegraded sites, 1=transitional sites, 0=degraded sites.

SITES (5 replicates)	Community Status	SITES (5 replicates)	Community Status
10 Swartz (West Basin)	1	35 Swartz (Coronado Cays)	1
11 Swartz (East Basin)	2	37 Swartz (Marina)	2
12 Swartz (Downtown Anch)	1	41 Swartz (Glorietta Bay)	1
14 Swartz (Downtown Piers)	0	K Swartz (Naval Base 04)	1
15 Swartz (G St Pier Marina)	1	Mission Bay A3	1
16 Swartz (Intercont. Marina)	1	Mission Bay A4	1
23 Swartz (Naval Base 07)	0	Mission Bay A8	2
25 Swartz (Naval base/ SY 010)	0	NSB-M1 (Sub Base C2)	0
27 Swartz (Naval Base /SH 013)	0	P Swartz (Naval Base 012)	0
28 Swartz (7th St Channel Q1)	0	San Diego River B1	0
31 Swartz (Marine Terminal R3)	1	SDNI- N5 (Carrier Base V2)	1
32 Swartz (Sweetwater Ch)	1	Stormdrain EM (Grape St.)	1
34 Swartz (CV Yacht Basin)	0		

sediment toxicity (Slattery and Swartz, personal communication). Final benthic community evaluation of 75 stations (Table 11) resulted in the designation of 23 undegraded, 43 degraded and 9 transitional stations. A map of the distribution of degraded, transitional and undegraded stations is shown in Figure 18(a-d). Degraded stations were found at the submarine base in North San Diego Bay. Commercial shipping, storm drainages and the naval shipyard waterfronts all had degraded communities in the Mid San Diego Bay. In South San Diego Bay, industrial and small boat locations exhibited benthic community degradation. In Mission Bay the stations near Rose Inlet and in the San Diego River were found to be degraded.

Chemically clean sites, as determined by ERM and PEL summary quotients and lack of ERM and PEL guideline exceedances, were reexamined to expand the undegraded list from possible "borderline" transitional stations. Stations 93194 and 93231 appropriately fit this category (Table 4) and were used as undegraded stations in the construction of the reference envelope for toxicity determination, discussed earlier.

As shown earlier in Figure 14, the relationship between benthic community conditions and elevated chemical conditions (as determined by using ERM and PEL Summary Quotients) was quite dramatic. Benthic communities were always found to be degraded when chemical levels were elevated (ERMQ>0.85), where both analyses were performed at a station.

Distribution Of Toxicity

The results of all toxicity tests conducted as part of this study are presented in tables in Appendix D. These tables show means and standard deviations for each toxicity test response (e.g. percent survival of amphipods; percent normal development of larval sea urchins) for three to five replicates of each sample tested. Associated ammonia and hydrogen sulfide concentrations are also presented in Appendix D.

Toxicity Testing Quality Assurance/Quality Control Evaluation

All toxicity test data produced for this report were evaluated for acceptability using the Quality Assurance guidelines described in the BPTCP Quality Assurance Project Plan (QAPP; Stephenson *et al.*, 1994). Toxicity data reported here met all test acceptability standards for each protocol, with the following exceptions. Of the solid phase tests with amphipods, two samples (Station 93120- IDORG# 702 and Station 93107- IDORG# 721) were tested with only one laboratory replicate, due to a lack of sufficient sample volume. Survival in those two samples was 90% and 85%, respectively, indicating a lack of toxicity. All amphipod samples tested in Leg 15 (Appendix D) have the following QA qualification. The test protocol requires five replicates of a control sample to be tested concurrently with test samples. In some early sampling legs of this study, 15 laboratory replicates of the control sediment were tested, to

Table 11. Benthic Index results showing the recalculation of San Diego Bay data based on individual stations. Replicates 4 and 5 in the site evaluation were not included (see text). Community status indicates allocation of a station to an Index partition: 2=undegraded stations, 1=transitional stations, 0=degraded stations.

IDORG	Station #	Station name	Community Status	IDORG	Station #	Station name	Community Status	IDORG	Station #	Station name	Community Status
837	90050	10 Sw (West Basin)	2	892	93226	27 Sw (Naval Base /SH 013)	0	854	93107	Mission Bay A3	0
838	93199	10 Sw (West Basin)	2	893	90009	28 Sw (7th St Channel Q1)	0	855	93107	Mission Bay A3	1
839	93200	10 Sw (West Basin)	2	894	93227	28 Sw (7th St Channel Q1)	0	859	93108	Mission Bay A4	1
840	90001	11 Sw (East Basin)	2	895	93228	28 Sw (7th St Channel Q1)	0	860	93108	Mission Bay A4	2
841	93201	11 Sw (East Basin)	2	896	90010	31 Sw (Marine Terminal R3)	0	861	93108	Mission Bay A4	1
842	93202	11 Sw (East Basin)	2	897	93229	31 Sw (Marine Terminal R3)	0	856	93112	Mission Bay A8	2
878	90002	12 Sw (Downtown Anch)	0	898	93230	31 Sw (Marine Terminal R3)	0	857	93112	Mission Bay A8	2
879	93221	12 Sw (Downtown Anch)	2	875	90052	32 Sw (Sweetwater Ch)	1	858	93112	Mission Bay A8	2
880	93222	12 Sw (Downtown Anch)	2	876	93219	32 Sw (Sweetwater Ch)	1	871	90028	NSB-M1 (Sub Base C2)	0
846	90003	14 Sw (Downtown Piers)	0	877	93220	32 Sw (Sweetwater Ch)	0	872	93216	NSB-M1 (Sub Base C2)	0
847	93205	14 Sw (Downtown Piers)	0	824	90012	34 Sw (CV Yacht Basin)	0	873	93217	NSB-M1 (Sub Base C2)	0
848	93206	14 Sw (Downtown Piers)	0	825	93196	34 Sw (CV Yacht Basin)	0	868	90022	P Sw (Naval Base 012)	0
849	90004	15 Sw (G St Pier Marina)	0	826	93197	34 Sw (CV Yacht Basin)	0	869	93214	P Sw (Naval Base 012)	0
850	93207	15 Sw (G St Pier Marina)	0	843	90053	35 Sw (Coronado Cays)	2	870	93215	P Sw (Naval Base 012)	0
851	93208	15 Sw (G St Pier Marina)	0	844	93203	35 Sw (Coronado Cays)	2	881	93116	San Diego River B1	0
818	90051	16 Sw (Intercont. Marina)	1	845	93204	35 Sw (Coronado Cays)	0	882	93116	San Diego River B1	0
819	93192	16 Sw (Intercont. Marina)	1	815	90013	37 Sw (Marina)	2	883	93116	San Diego River B1	0
820	93193	16 Sw (Intercont. Marina)	1	816	93190	37 Sw (Marina)	2	899	90025	SDNI- N5 (Carrier Base V2)	2
865	90006	23 Sw (Naval Base 07)	0	817	93191	37 Sw (Marina)	2	1000	93231	SDNI- N5 (Carrier Base V2)	2
866	93212	23 Sw (Naval Base 07)	0	821	90015	41 Sw (Glorietta Bay)	1	1001	93232	SDNI- N5 (Carrier Base V2)	2
867	93213	23 Sw (Naval Base 07)	0	822	93194	41 Sw (Glorietta Bay)	2	827	90037	Stormdrain EM (Grape St.)	0
887	90007	25 Sw (Naval base/ SY 010)	0	823	93195	41 Sw (Glorietta Bay)	2	828	90037	Stormdrain EM (Grape St.)	0
888	93223	25 Sw (Naval base/ SY 010)	0	862	90021	K Sw (Naval Base 04)	0	829	90037	Stormdrain EM (Grape St.)	2
889	93224	25 Sw (Naval base/ SY 010)	0	863	93210	K Sw (Naval Base 04)	0				
890	90008	27 Sw (Naval Base /SH 013)	0	864	93211	K Sw (Naval Base 04)	0				
891	93225	27 Sw (Naval Base /SH 013)	0	853	93107	Mission Bay A3	0				

Figure 18a
Benthic Community Analyses
North San Diego Bay

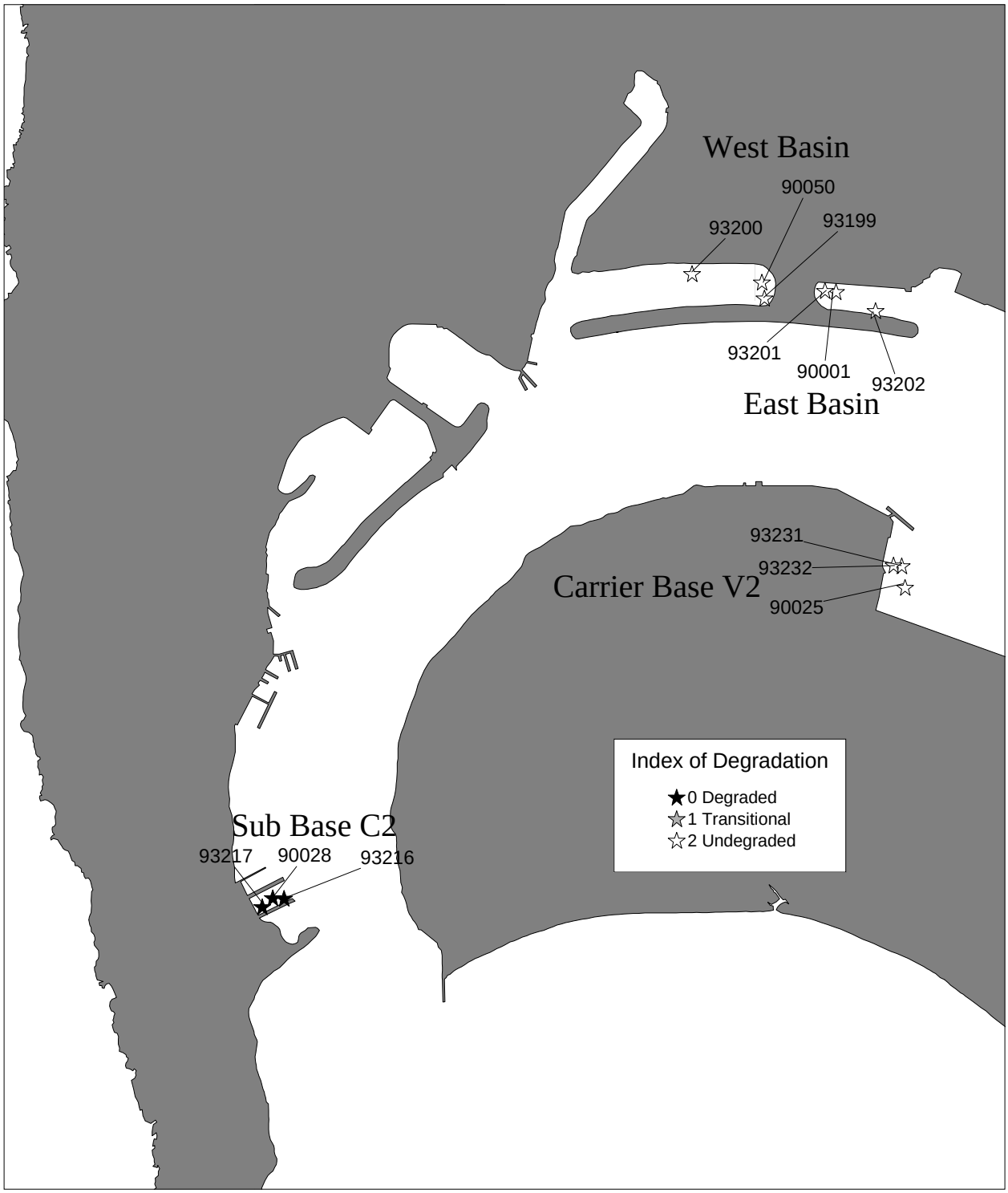


Figure 18a
Benthic Community Analyses
North San Diego Bay

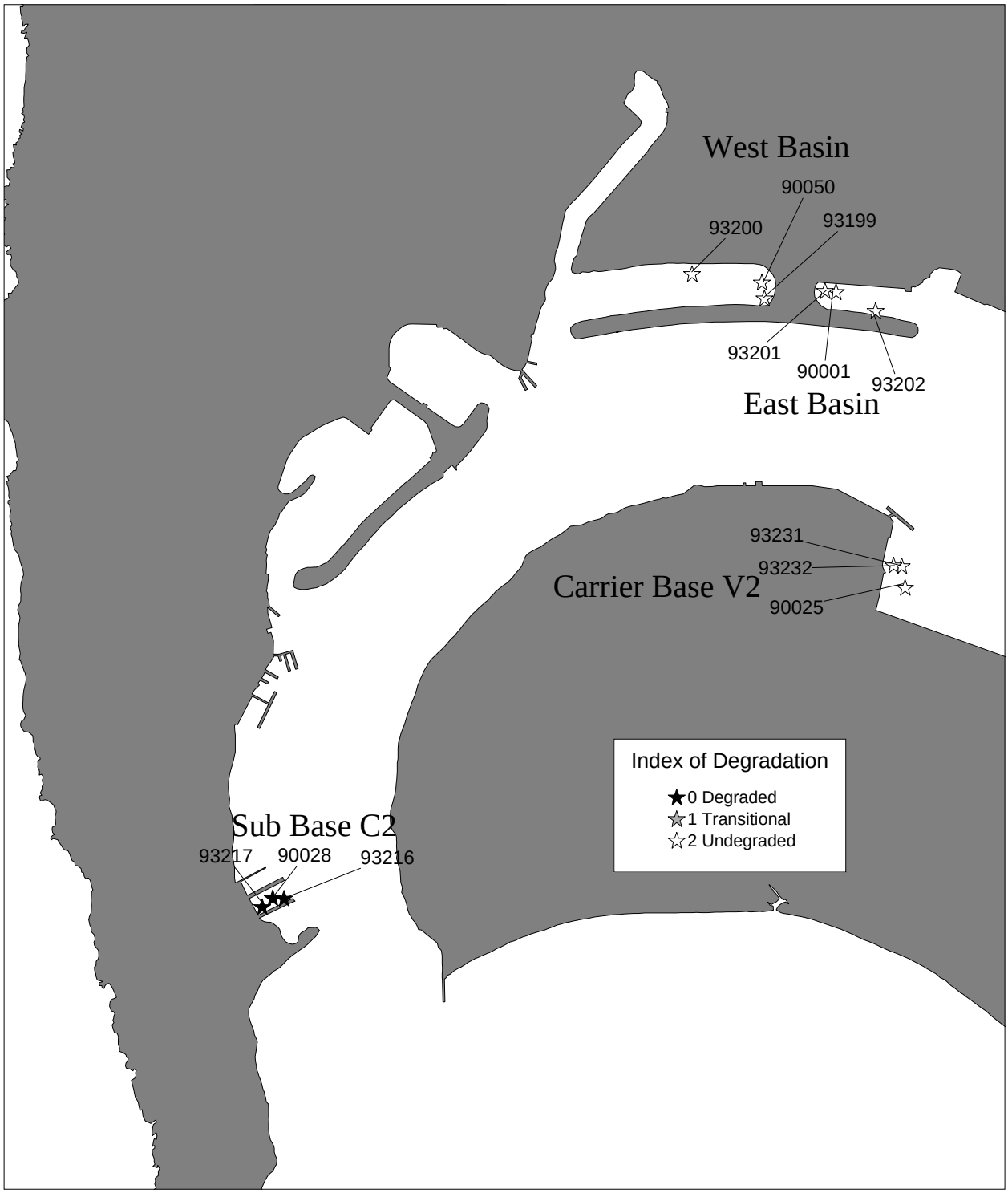


Figure 18b
Benthic Community Analyses
Mid San Diego Bay

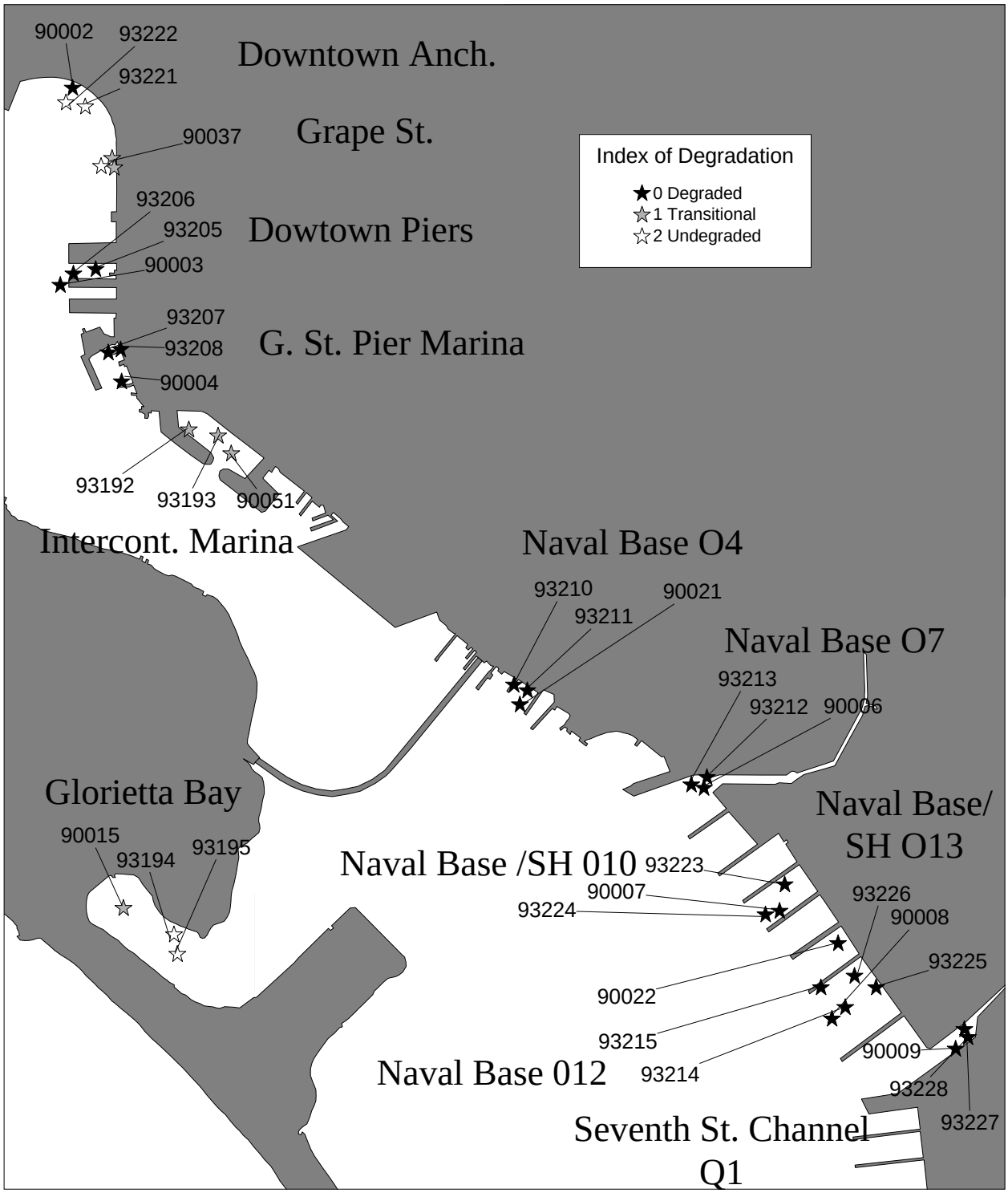


Figure 18c
Benthic Community Analyses
South San Diego Bay

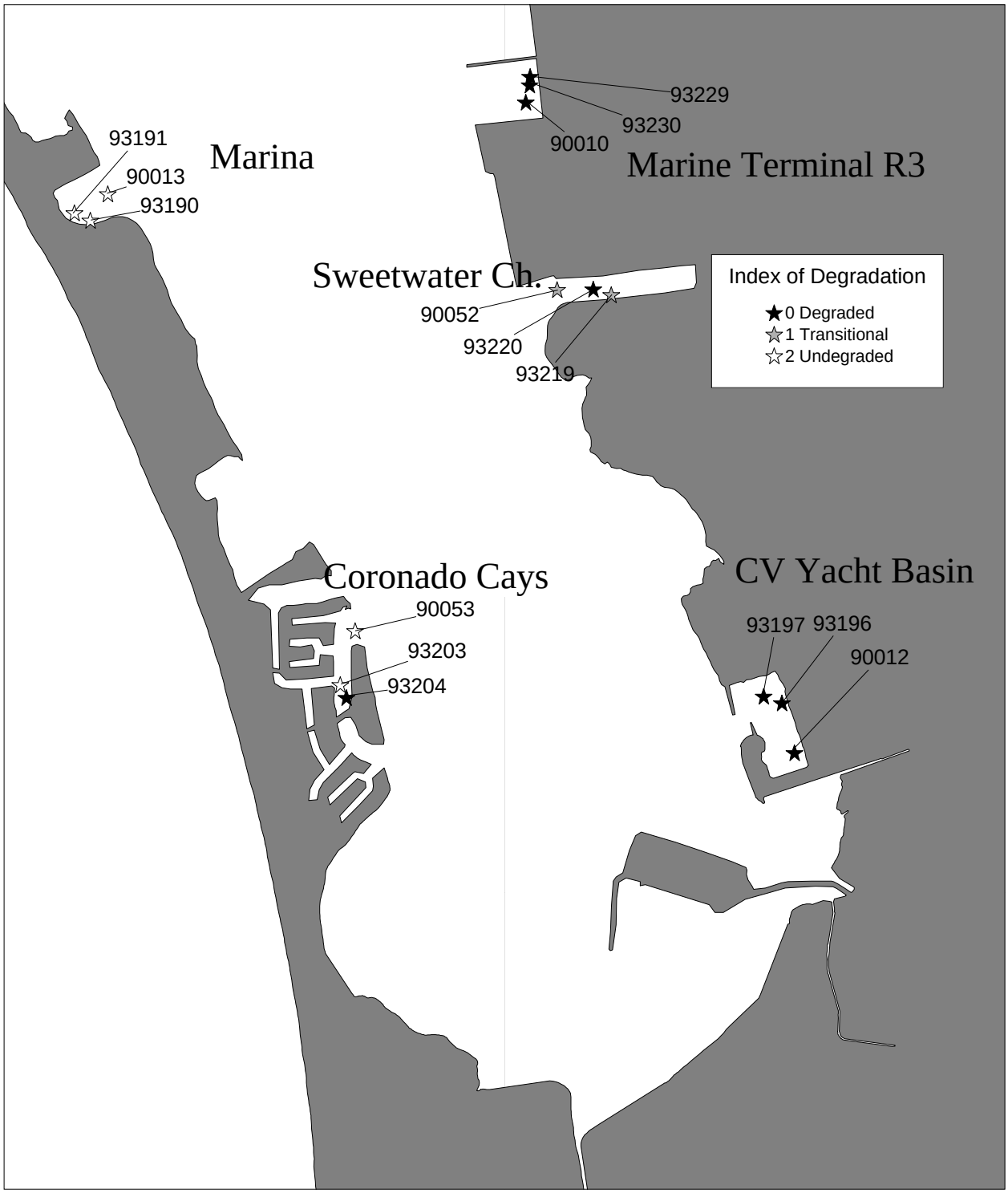
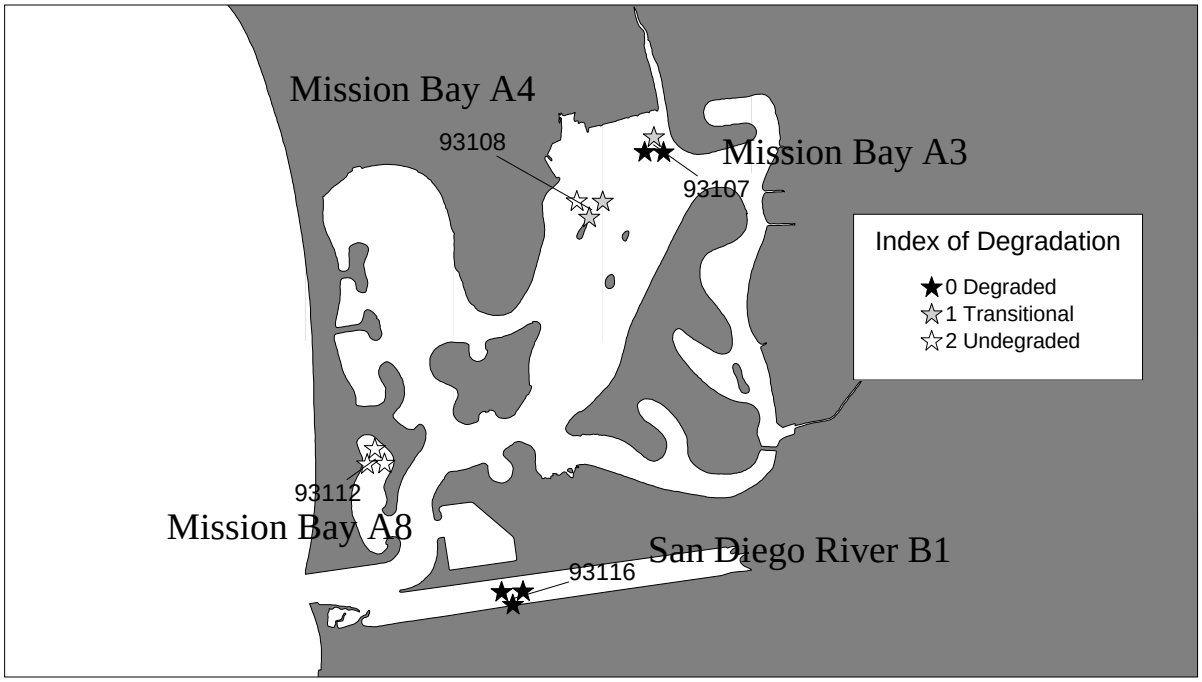


Figure 18d
Benthic Community Analyses
Mission Bay and San Diego River Estuary



allow use of alternative statistical procedures. Of the fifteen control replicates in Leg 15, two had 75% survival, which is below the 80% criterion given in the protocol. In tests using the *Neanthes arenaceodentata* (hereafter *Neanthes*) protocol on solid phase sediments, all samples tested in Leg 21 used sediment that was held in the laboratory three days beyond the fourteen-day specified holding time. These QA exceptions in solid phase tests have been judged by the toxicity project officers to not adversely affect interpretation of toxicity results. These and lesser departures from acceptable standards are recorded in the Quality Assurance Evaluative Reports accompanying each dataset for this study. Quality Assurance Evaluative Reports for toxicity testing are available for review from the SWRCB. Minor departures not mentioned above included elevated dissolved oxygen measurements in overlying water and other variations in water quality measurement that were considered to have little probability of affecting the outcome of the respective toxicity test.

There were no deviations from quality assurance criteria, other than minor deviations in measurement of water quality parameters as cited above, in any of the abalone, mussel, or sea urchin larval development tests in pore water or water column samples (subsurface water).

Sea urchin fertilization tests were conducted on over 300 pore water samples. Many of these were retested because of poor response in brine controls. Bay *et al.* (1993) discussed commonly observed problems using the *Strongylocentrotus purpuratus* (hereafter *Strongylocentrotus*) fertilization test in samples requiring salinity adjustment with hypersaline brine. Through numerous repeated tests, acceptable brine control results were produced for all but one sample. However, as described in BPTCP QA reports to the SWRCB, an additional control for the storage effects of frozen pore water samples in Teflon bottles was included in later tests. These additional controls, which were not required by the original QAPP, indicated that toxicity may be associated with frozen sample storage in Teflon bottles. Because all pore water samples for fertilization tests were stored frozen in Teflon bottles, we have no assurance the data from any of these fertilization tests is truly indicative of sample toxicity. Any toxicity observed in the fertilization tests may be wholly or partially due to storage effects. For this reason, we retested all samples from legs 15-23 with the sea urchin larval development test, unless those samples had already been tested with the development test. The urchin larval development test has been unaffected by storage artifacts, as indicated by response in frozen storage bottle controls. While sea urchin fertilization data are reported in Appendix D, they were not used in any further data analysis for this report. The use of fertilization data, for determination of toxicity, was therefore not considered prudent considering the possibility of false positive results related to sample storage.

Areal Extent of Toxicity Based on the EMAP Approach

The Cumulative Distribution Frequency (CDF) analyses indicated that 56% of the total area sampled was toxic to *Rhepoxynius abronius* (hereafter *Rhepoxynius*) (Table 12, Figure 19). The sea urchin larval development test of undiluted (100%), 50%, and 25% pore water indicated 74%, 54%, and 29% percent of the total study area was toxic, respectively (Table 12, Figure 20). A number of samples were toxic to both sea urchins and amphipods. Samples representing 36%, 27%, or 14% of the study area were toxic to *Rhepoxynius* in solid phase sediment and to sea urchin larvae in 100%, 50%, or 25% pore water, respectively. The percentage of area toxic was based on comparisons with laboratory controls using the EMAP statistical approach described in the methods section. These analyses utilized data from random stations within the stratified sampling blocks, and did not include data from stations utilizing the non-random, directed sampling design (Figure 21a-d, Figure 22a-d).

The curves on the CDF plots indicate the magnitude of toxicity throughout the Region. Each point on the CDF plot represents a single sample. The distribution of the amphipod data (Figure 19) show there were few samples with survival less than 40%, a greater number of samples with survival between 40% and 80%, and about half of all samples with survival greater than 80%. NOAA surveys of Tampa Bay, Florida and EMAP surveys of the Mid-Atlantic coast region (Virginian Province) produced CDF curves for amphipod mortality data further right on the scale and much steeper than the San Diego Bay Region plot, and had more than 90% of samples with greater than 90% survival in both regions (Long *et al*., 1994; Schimmel *et al*., 1991).

The CDF plot of San Diego Bay Region sea urchin larval development test data (Figure 20) shows a cluster of samples with 0% normal larval development, a smaller number of samples with intermediate response, and a cluster of samples with percent normal development roughly equal to that observed in controls. The 25% pore water dilutions had a majority of samples resulting in percent normal larval development roughly equal to controls. As pore water concentration increased to 50% and 100% pore water, the distribution of samples shifted toward the more toxic end of the scale, and the 100% pore water tests had a majority of samples resulting in 0% normal larval development. A similar pattern was observed in sea urchin fertilization tests of pore water from Tampa Bay, Florida (NOAA, 1994). As with the amphipod data, the San Diego distribution is shifted further to the left, indicating higher overall toxicity observed from San Diego Bay Region samples.

Toxicity Based on Reference Envelope Approach

Using the *Rhepoxynius* data and a p-value of 1%, a lower reference envelope tolerance bound of 48% survival was calculated, indicating that samples with survival values below 48% are significantly more toxic than samples representative of less

Table 12. Percent of total area sampled determined to be toxic with each toxicity test protocol. Sample toxicity is based on the EMAP statistical approach using two criteria for any given sample: significant difference from the control using a separate variance t-test and an alpha of 0.05 and a sample mean value less than 80% of the control value. Calculations for cumulative distribution frequency (CDFs) used to compute the percent of area toxic are explained in text and presented in Appendix F. Total study area was 47 square kilometers.

Toxicity Test and Pore Water Dilution	**Percent of Total Area Determined to be Toxic**
Rhepoxynius abronius Survival in Solid Phase	56%
Strongylocentrotus purpuratus Development in:	
100% (undiluted) Pore Water	74%
50% Pore Water	54%
25% Pore Water	29%

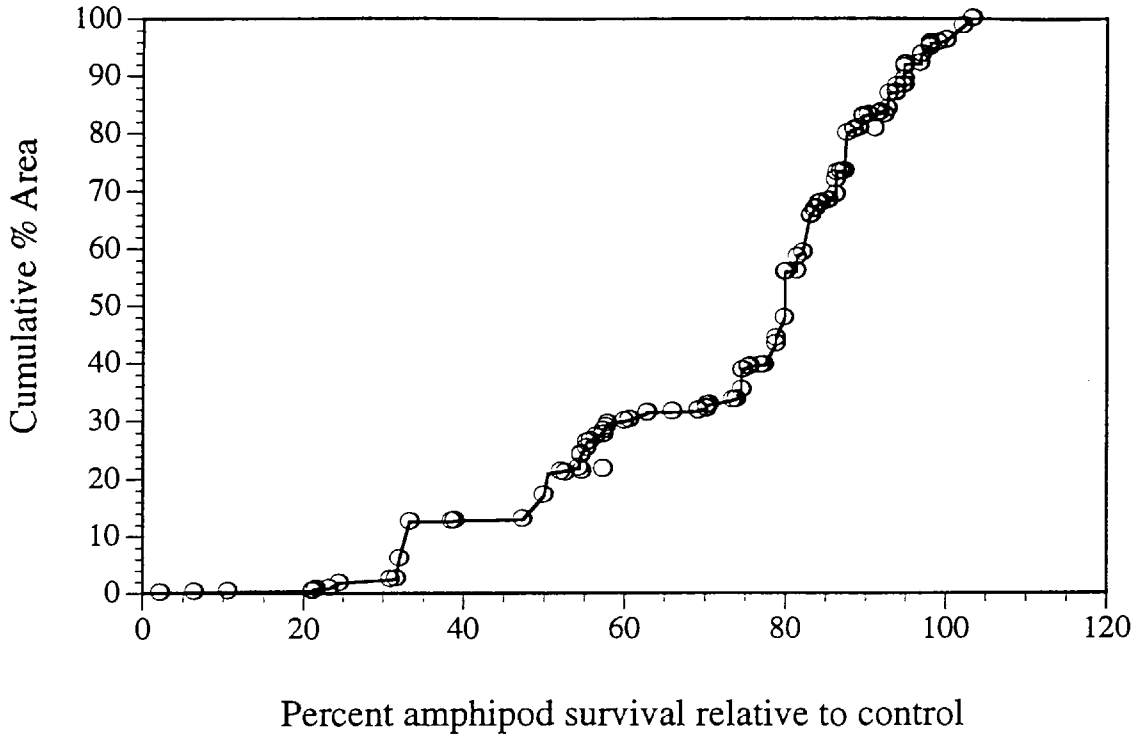


Figure 19. Cumulative distribution frequency of percent *Rhepoxynius* survival against percent of total area sampled. Data points correspond to individual samples.

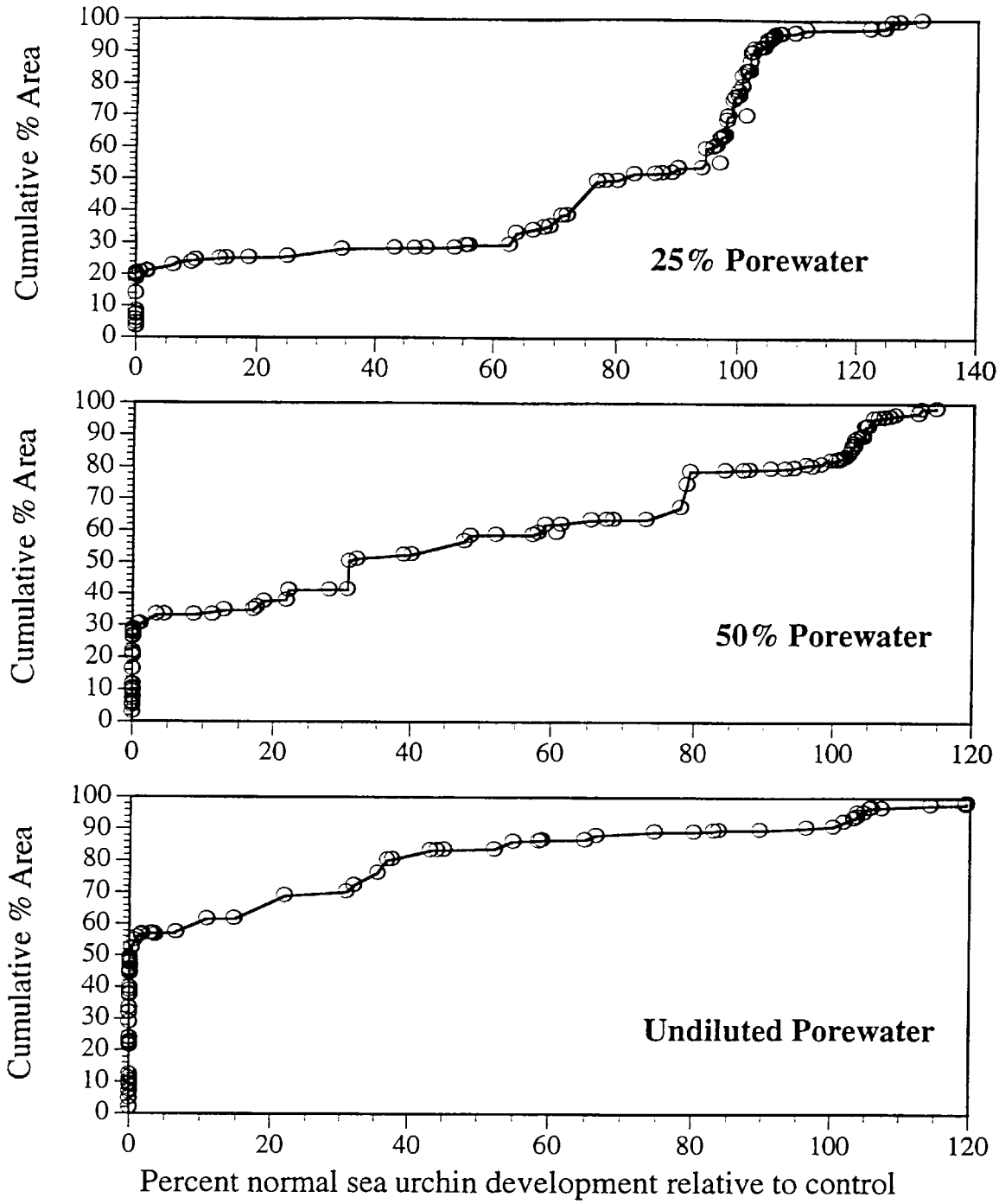


Figure 20. Cumulative distribution frequency of percent normal sea urchin larval development in 25%, 50%, and undiluted porewater against percent of total area sampled. Data points correspond to individual samples.

Figure 21a
Amphipod Toxicity Using Lab Controls
for Randomly Sampled Stations
North San Diego Bay

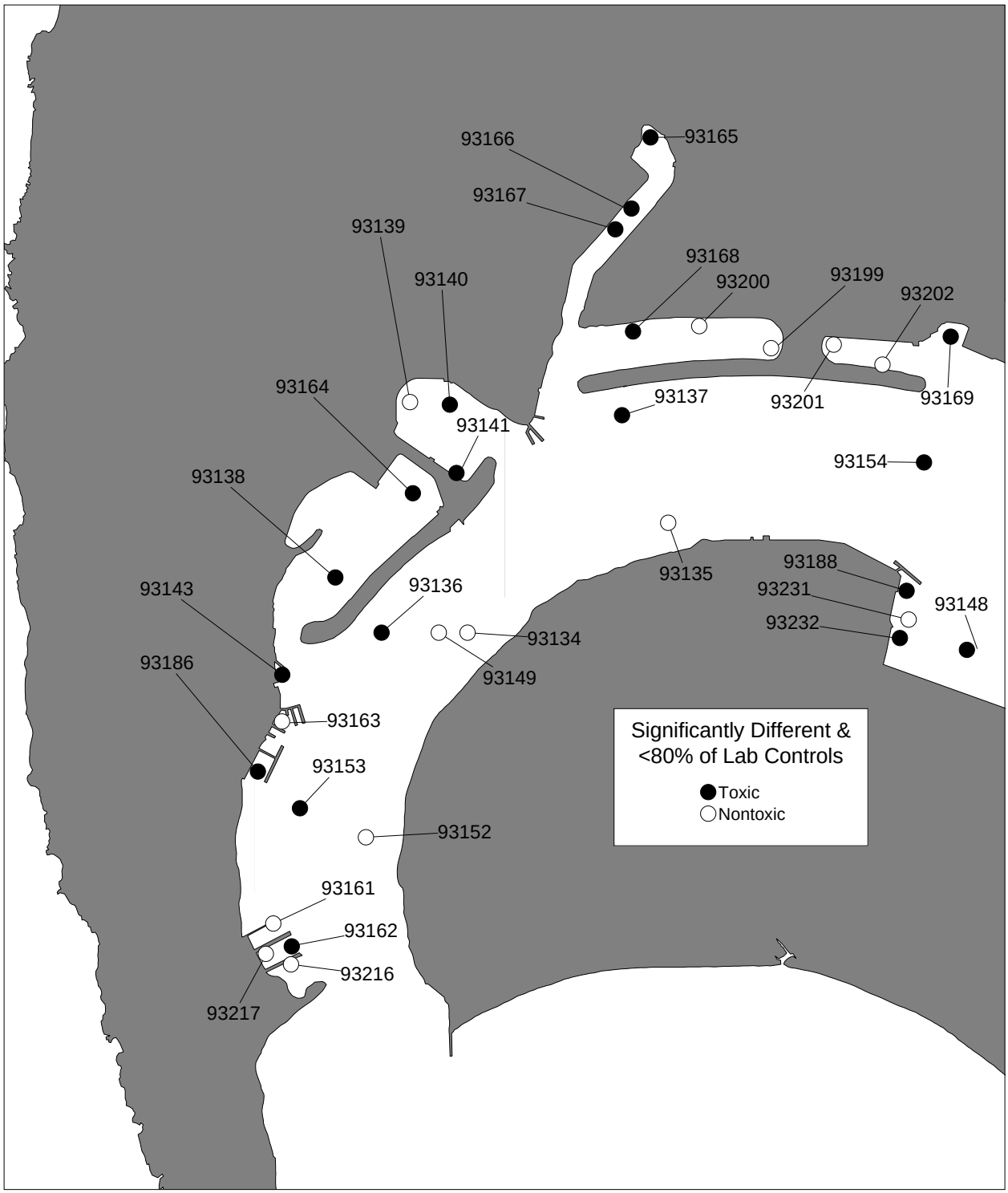


Figure 21b
Amphipod Toxicity Using Lab Controls for Randomly Sampled Stations Mid San Diego Bay

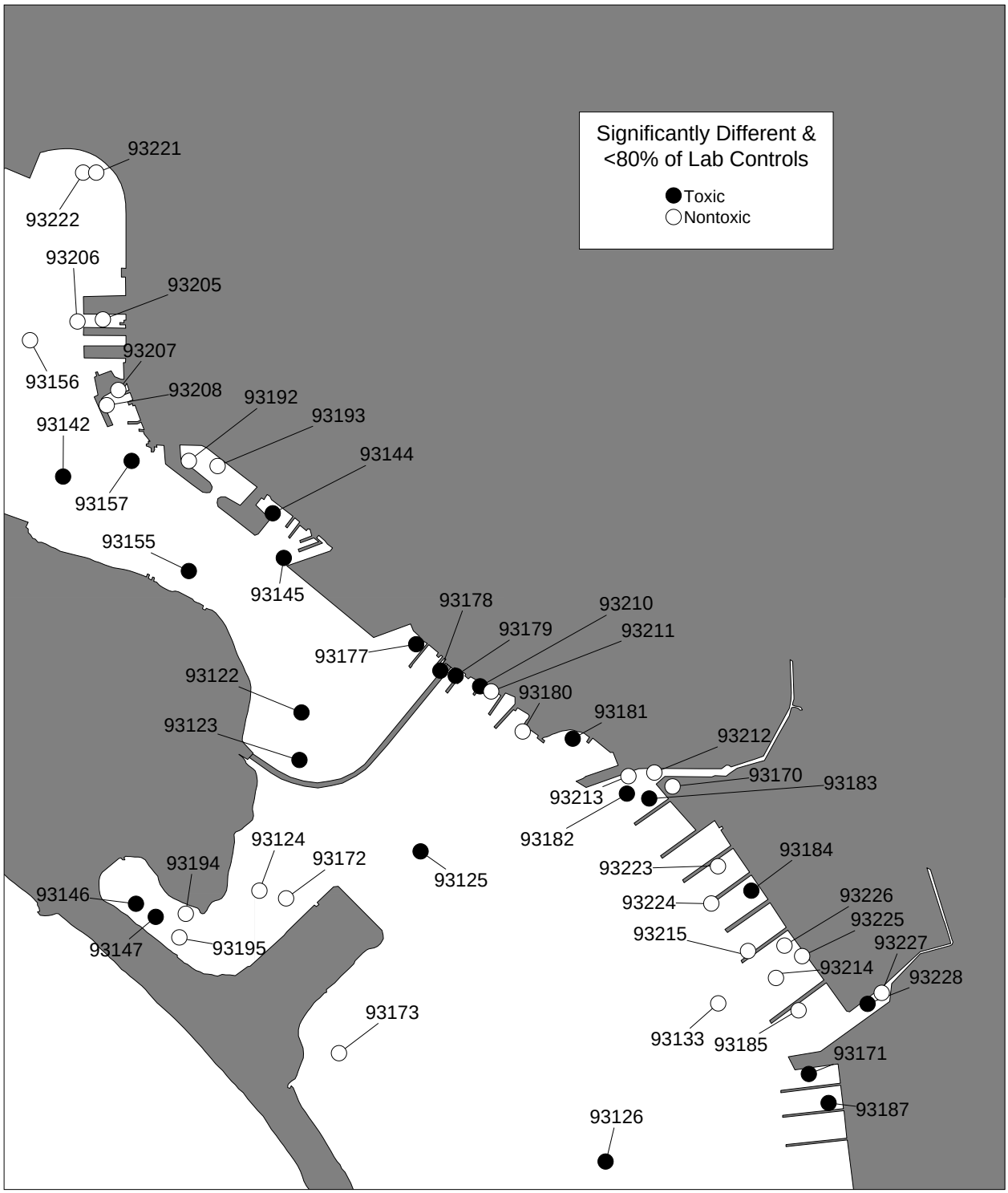


Figure 21c
Amphipod Toxicity Using Lab Controls for Randomly Sampled Stations South San Diego Bay

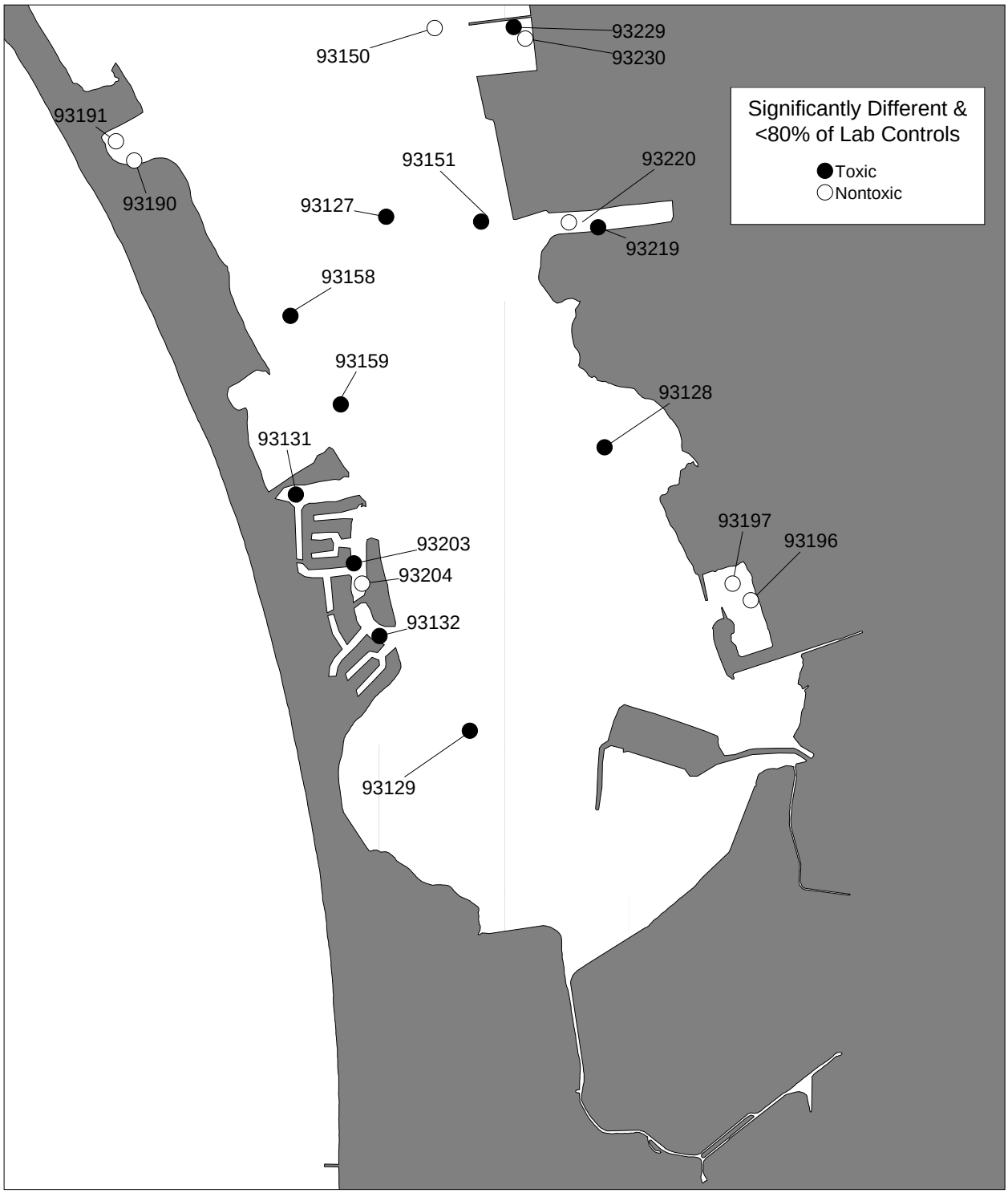
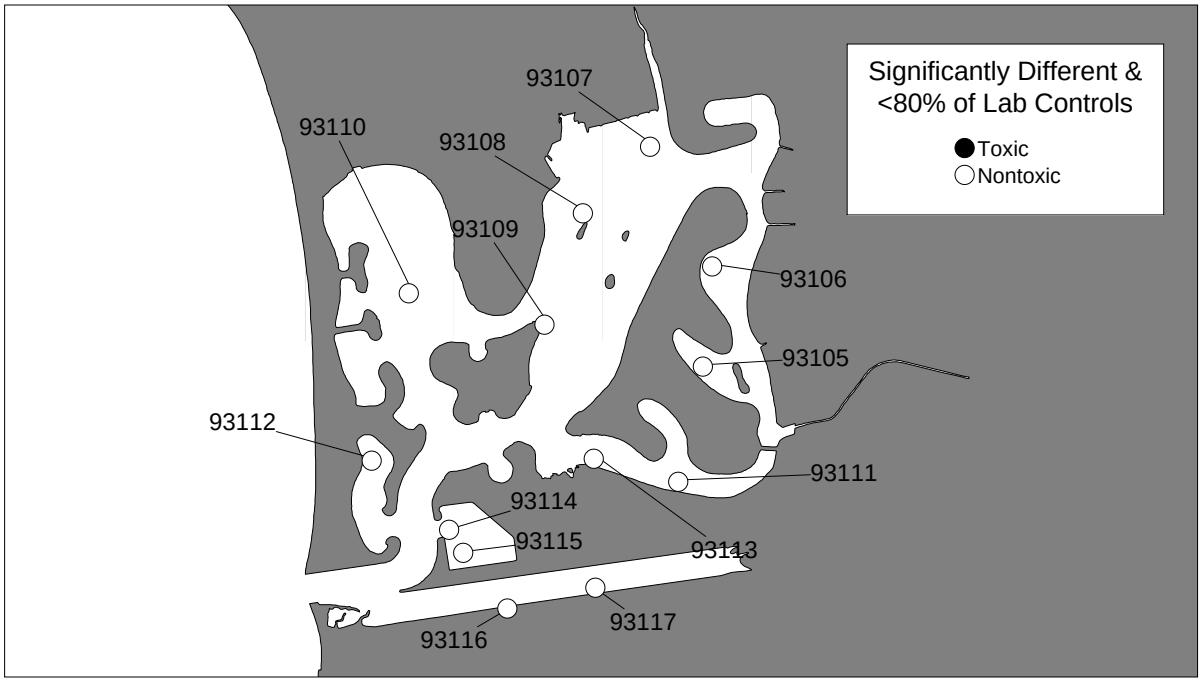


Figure 21d
Amphipod Toxicity Using Lab Controls for Randomly Sampled Stations Mission Bay and San Diego River Estuary



Tijuana River Estuary

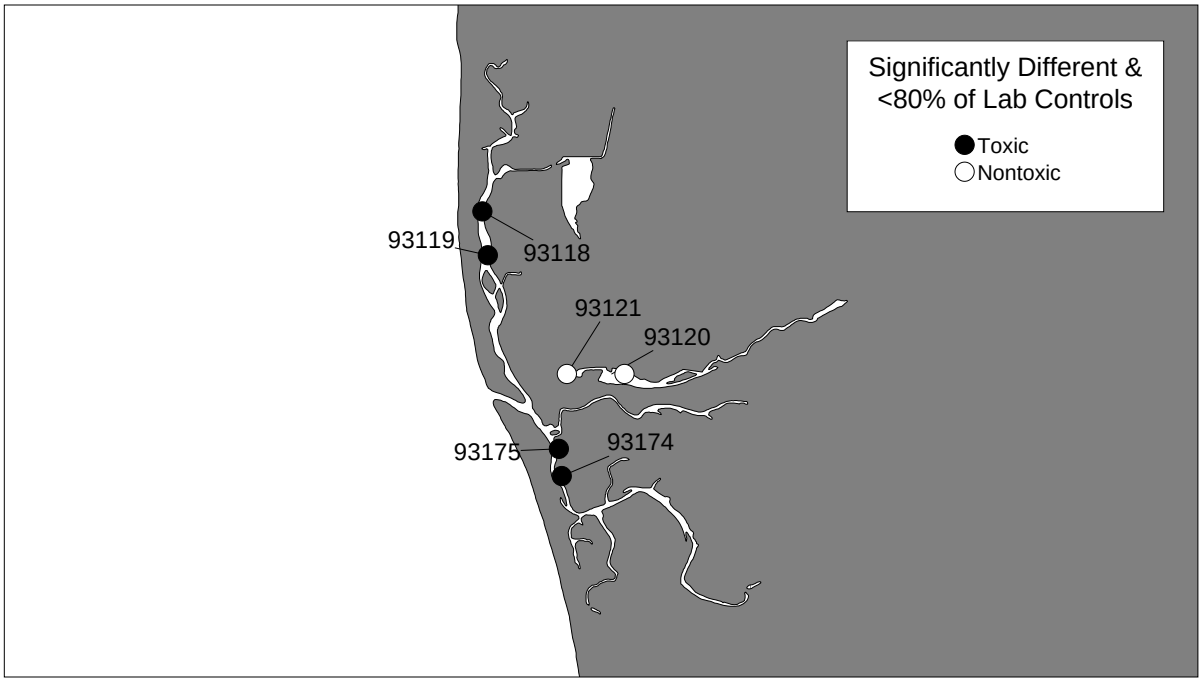


Figure 22a
Urchin Development Toxicity Using Lab Controls
for Randomly Sampled Stations
North San Diego Bay

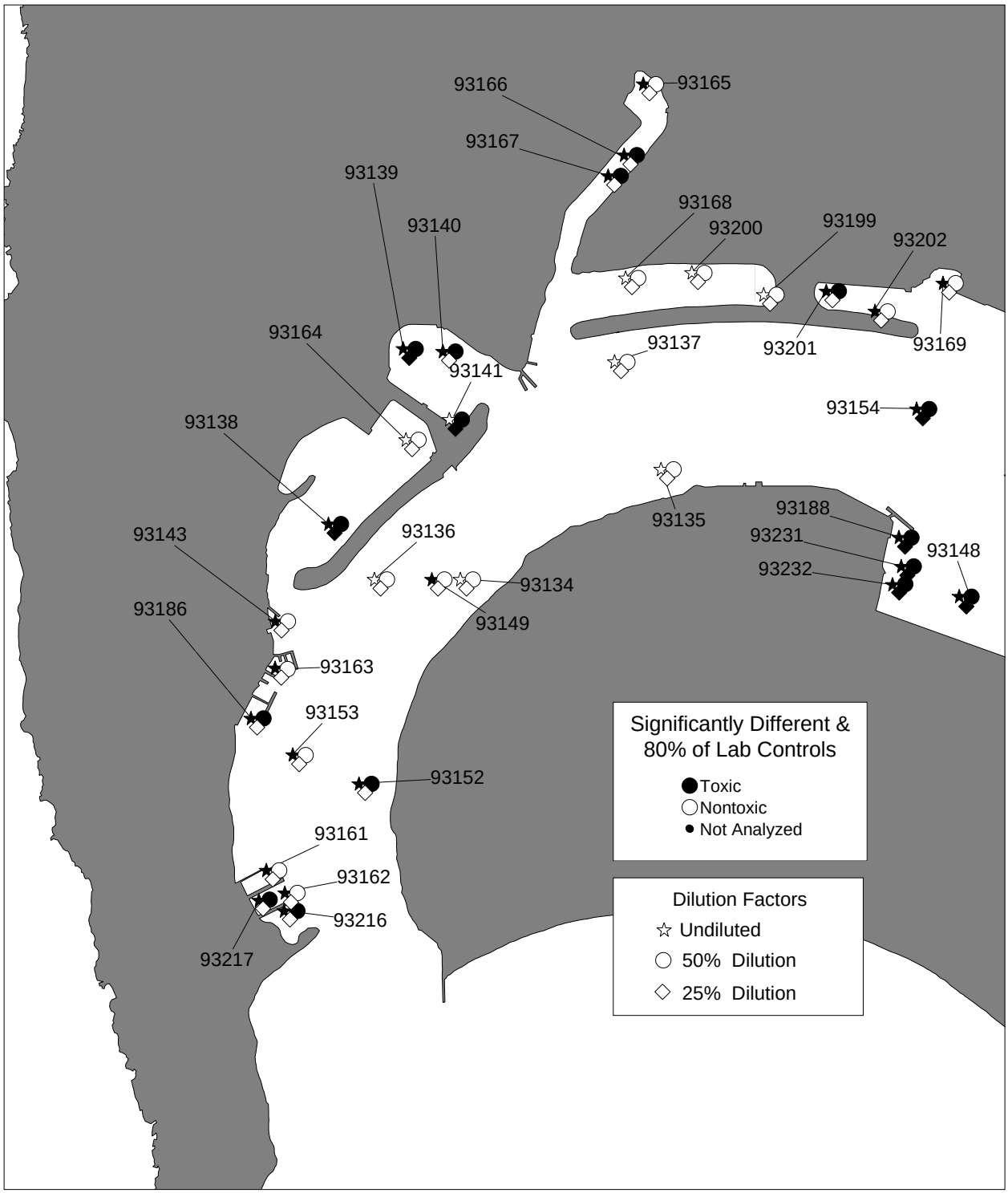


Figure 22b
Urchin Development Toxicity Using Lab Controls for Randomly Sampled Stations Mid San Diego Bay

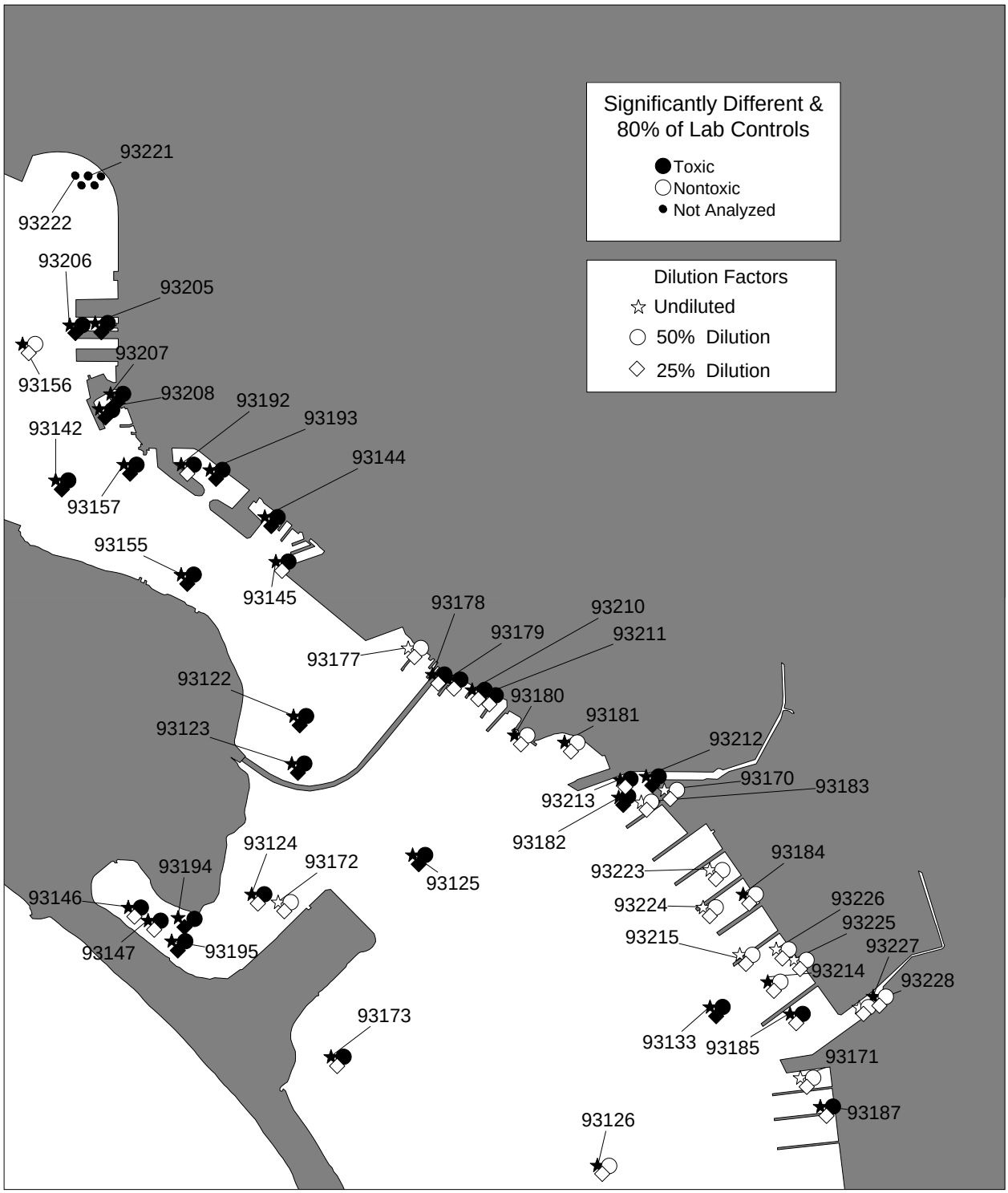


Figure 22c
Urchin Development Toxicity Using Lab Controls for Randomly Sampled Stations South San Diego Bay

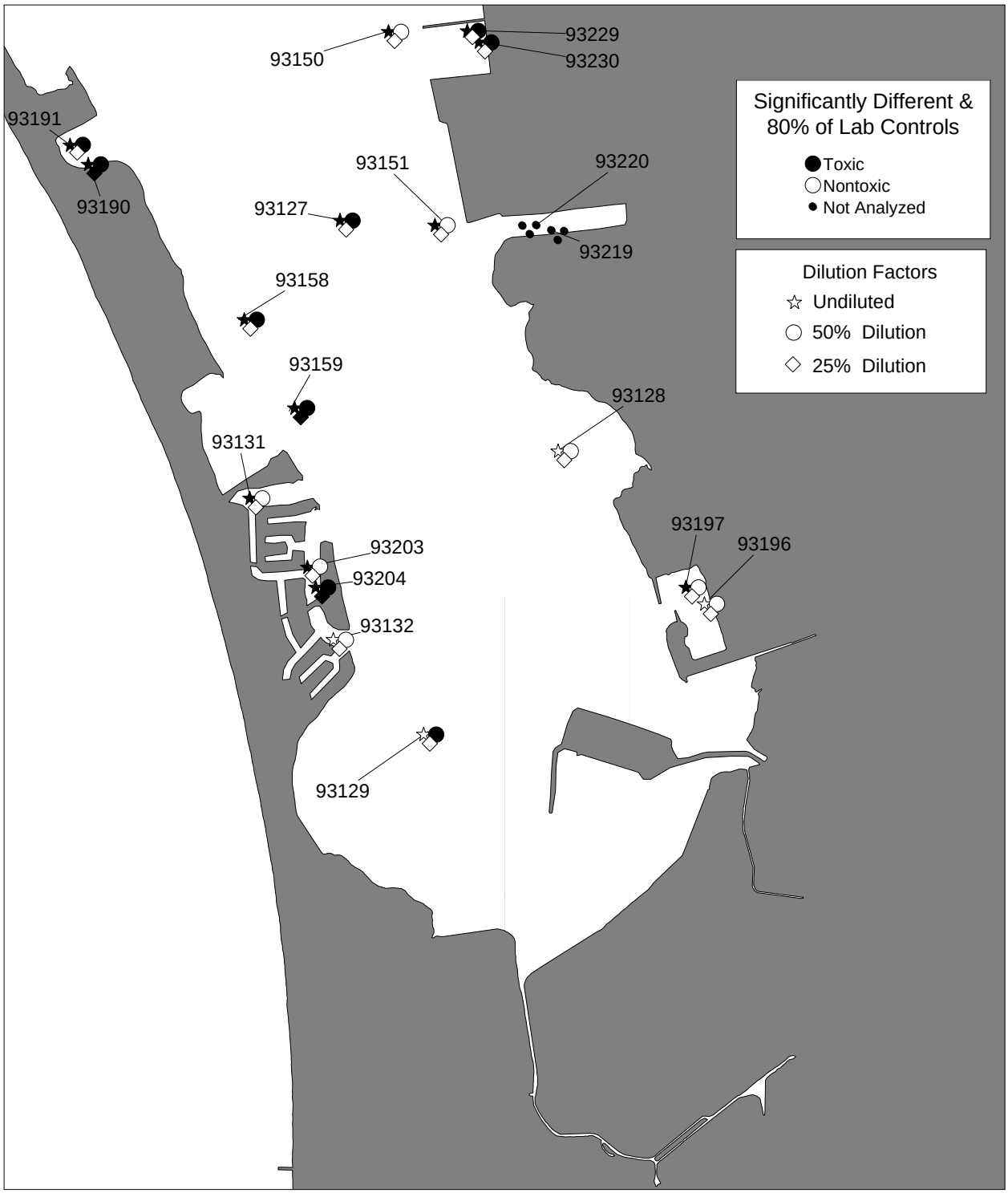
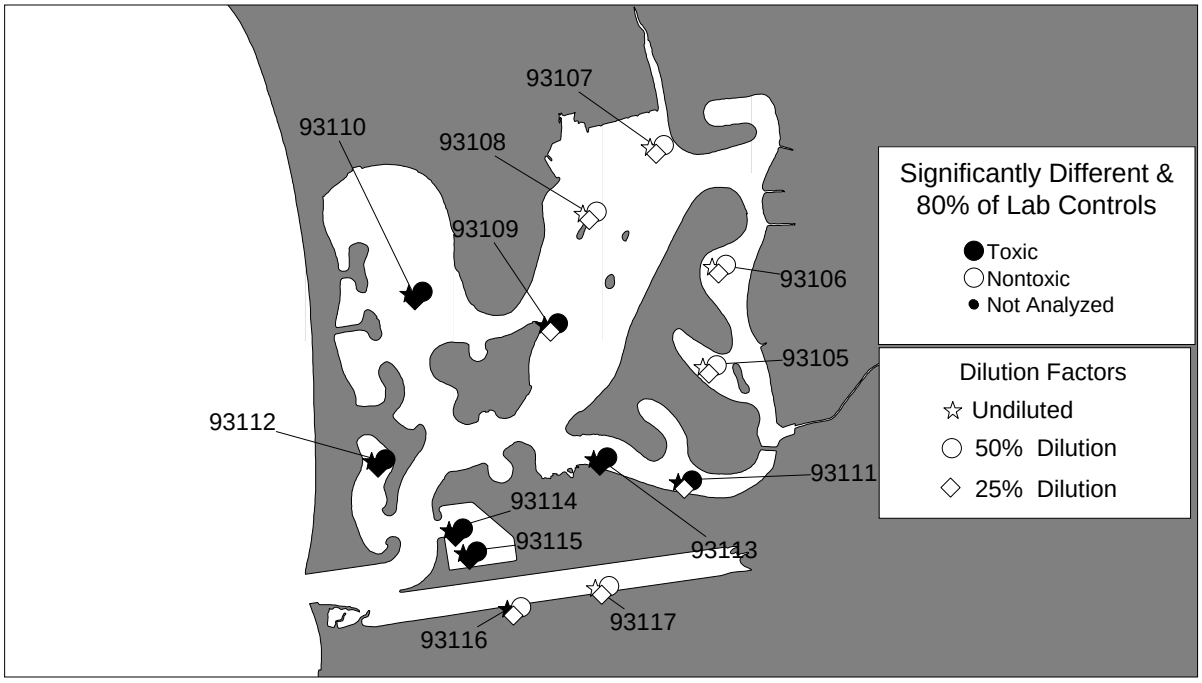
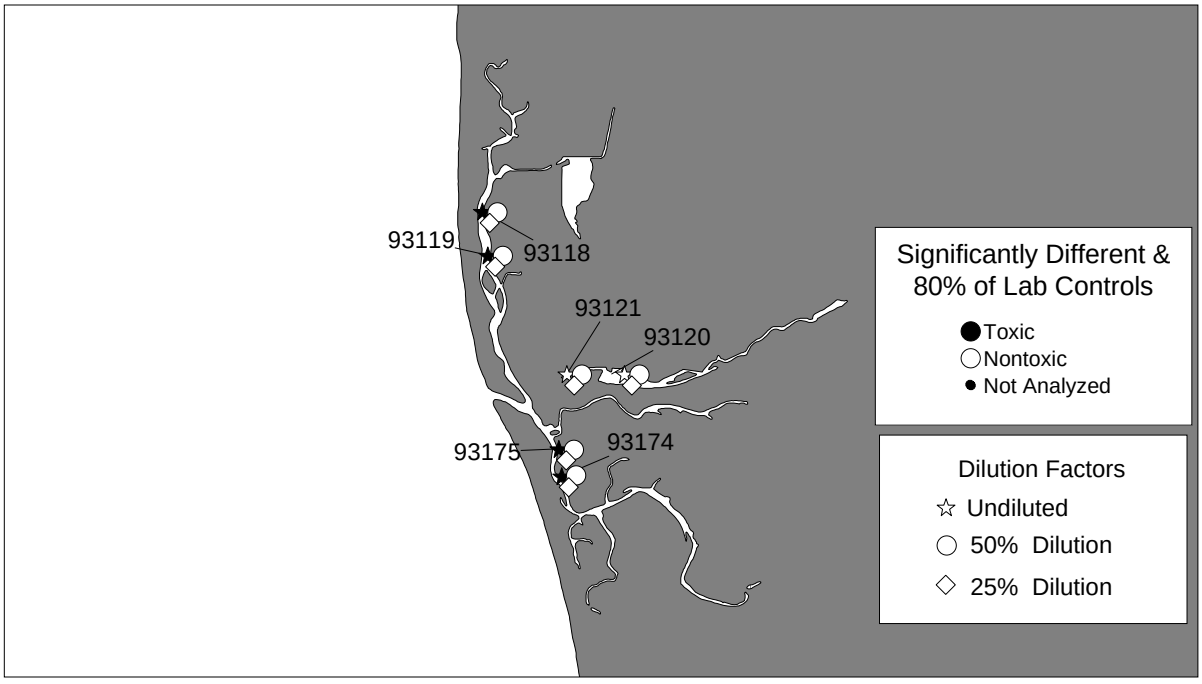


Figure 22d
Urchin Development Toxicity Using Lab Controls
for Randomly Sampled Stations
Mission Bay and San Diego River Estuary



Tijuana River Estuary



contaminated ambient conditions in the San Diego Bay Region. There is a 95% probability that samples with survival values less than 48% are more toxic than the most toxic 1% of samples from the reference site population. Of 350 samples tested with the *Rhepoxynius* test (from both random and non-randomly selected stations), 61 samples were found to be toxic using the reference envelope analysis (Figure 23a-d). Toxicity based on the reference envelope approach is used later in this report for prioritizing stations of concern.

Strongylocentrotus pore water data from reference stations produced a lower mean value and greater variability than was found for the amphipod solid phase data (Table 4). The variability in pore water data from sea urchin larval development tests produced a reference site distribution extending across the range from 0 to 100% normal development. A p-value of 1% (see Methods Section) produced a tolerance bound (reference envelope edge) which was below zero, indicating no distinctions could be made between reference and toxic stations. The high degree of variability in the pore water results from the reference sites may be related to the sensitivity of this test to measured or unmeasured toxicants, and/or may reflect artifacts related to pore water extraction and handling. Potential artifacts and sources of variability related to pore water testing are discussed below.

Comparison of Toxicity Test Protocols

Solid phase toxicity tests using the amphipod *Rhepoxynius* provided a wide range of response, from 0 to 98% survival. Amphipod survival ranged from 68-98 % for the eleven reference stations, suggesting that relatively high *Rhepoxynius* survival is a consistent feature of sites with relatively low chemical concentrations and undegraded benthic communities. The *Rhepoxynius* test identified multiple toxic samples, which indicated adequate sensitivity. Of the two solid phase protocols used in this study, the *Rhepoxynius* test provided the best test performance in terms of convenience, consistency, and sensitivity.

Solid phase toxicity tests which used the polychaete *Neanthes* were less sensitive than the *Rhepoxynius* test, and usually indicated no toxicity in samples that were toxic to test organisms using other protocols. In all instances where a sediment sample was toxic to *Neanthes* (survival or growth - relative to controls), it was also toxic to *Rhepoxynius*, whereas many samples that were toxic to *Rhepoxynius* were not toxic to *Neanthes* test. Because the *Neanthes* test demonstrated considerably less sensitivity than the *Rhepoxynius* test, the *Neanthes* test was not recommended for continued use in this program.

Two pore water tests, using *Strongylocentrotus* fertilization and larval development protocols, were performed on three concentrations of pore water samples to evaluate their usefulness

Figure 23a
Amphipod Toxicity Using Reference Envelope
for All Stations
North San Diego Bay

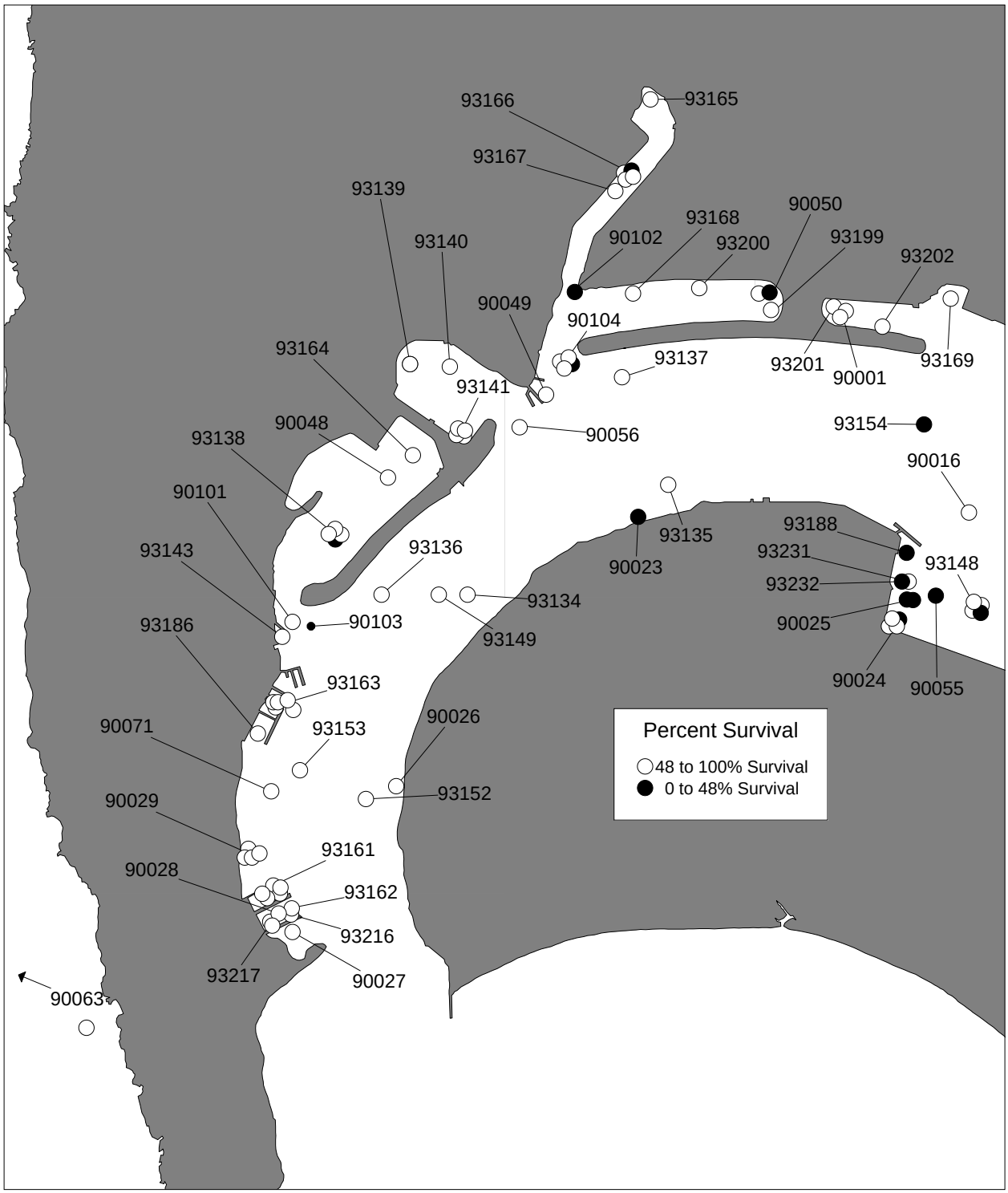


Figure 23b
Amphipod Toxicity Using Reference Envelope for All Stations
Mid San Diego Bay

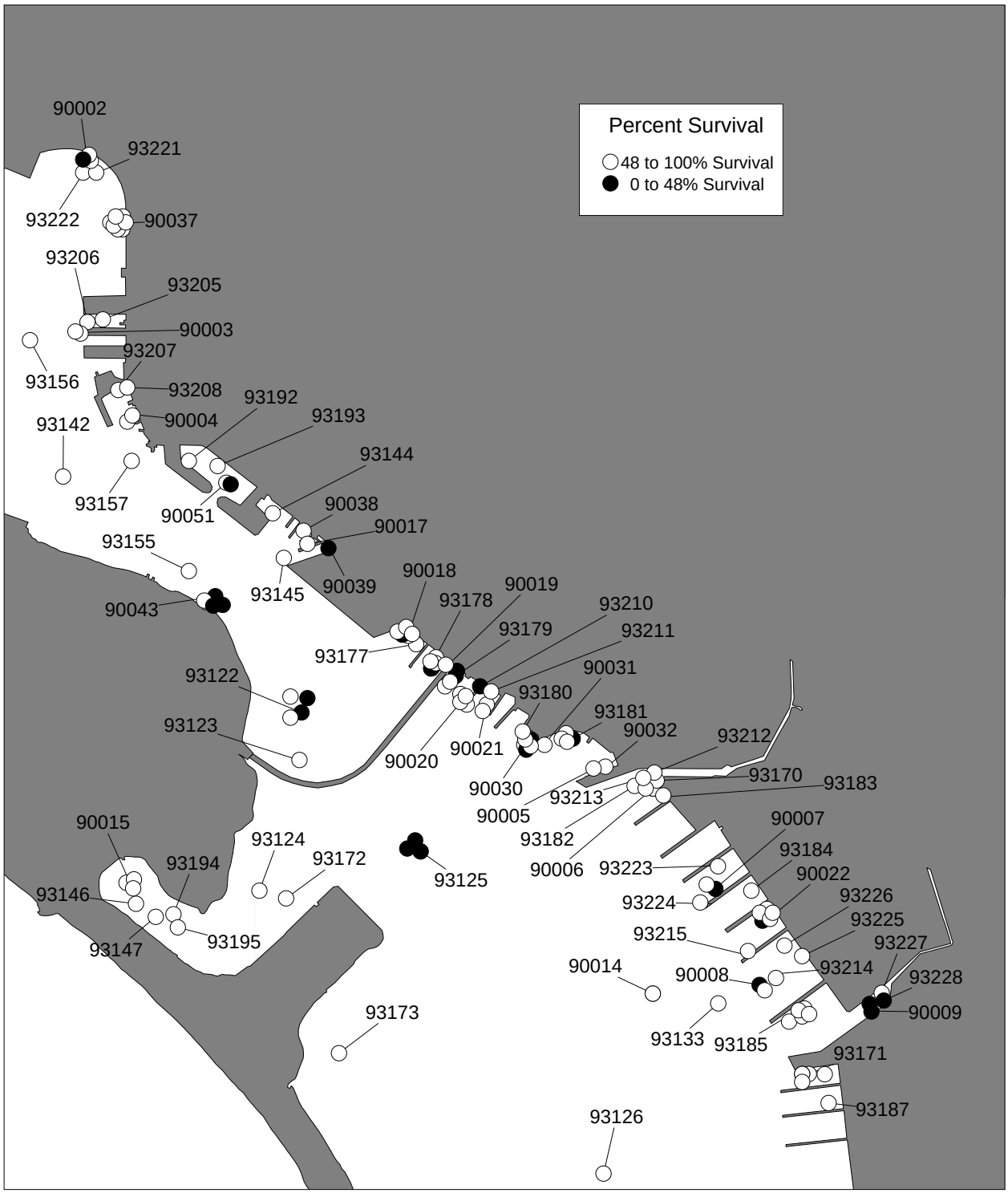


Figure 23c
Amphipod Toxicity Using Reference Envelope
for All Stations
South San Diego Bay

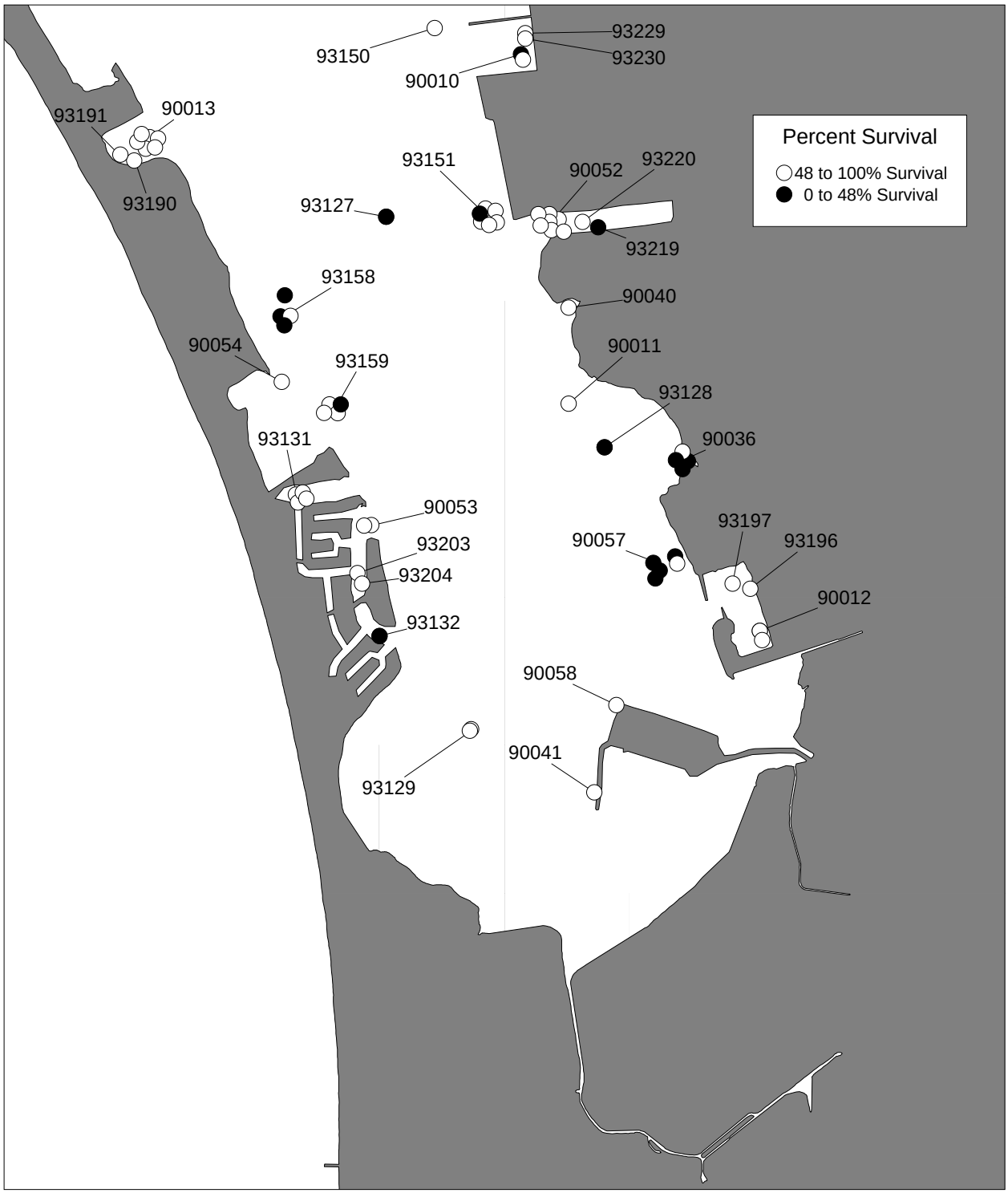
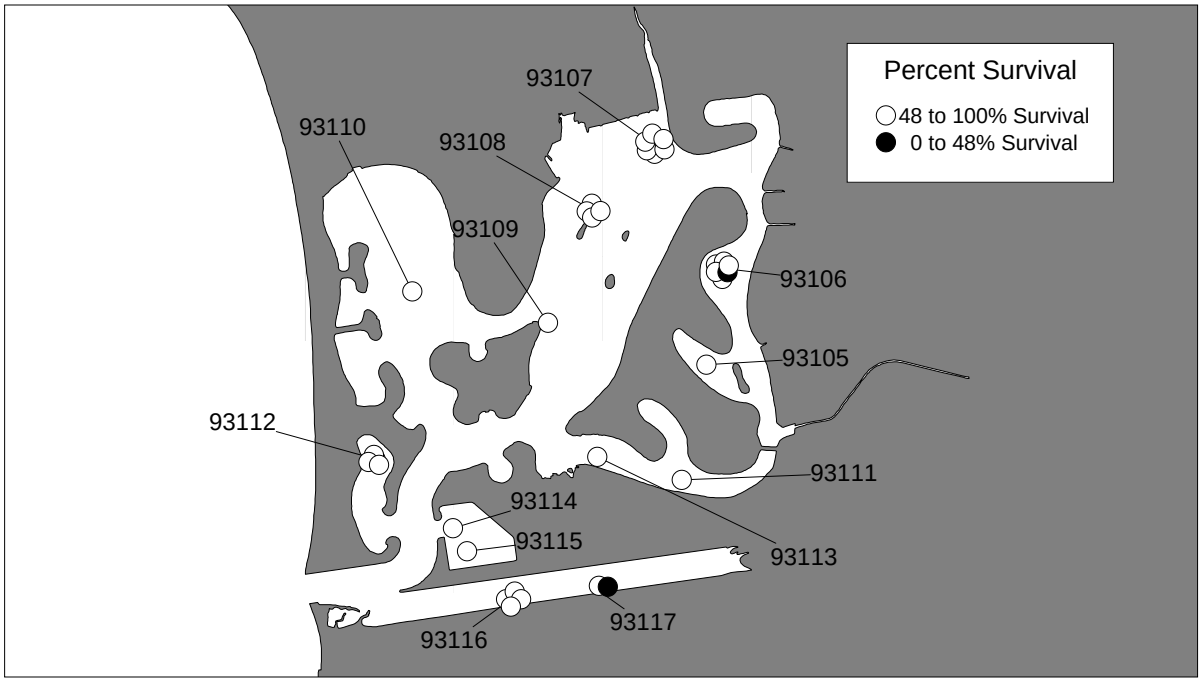
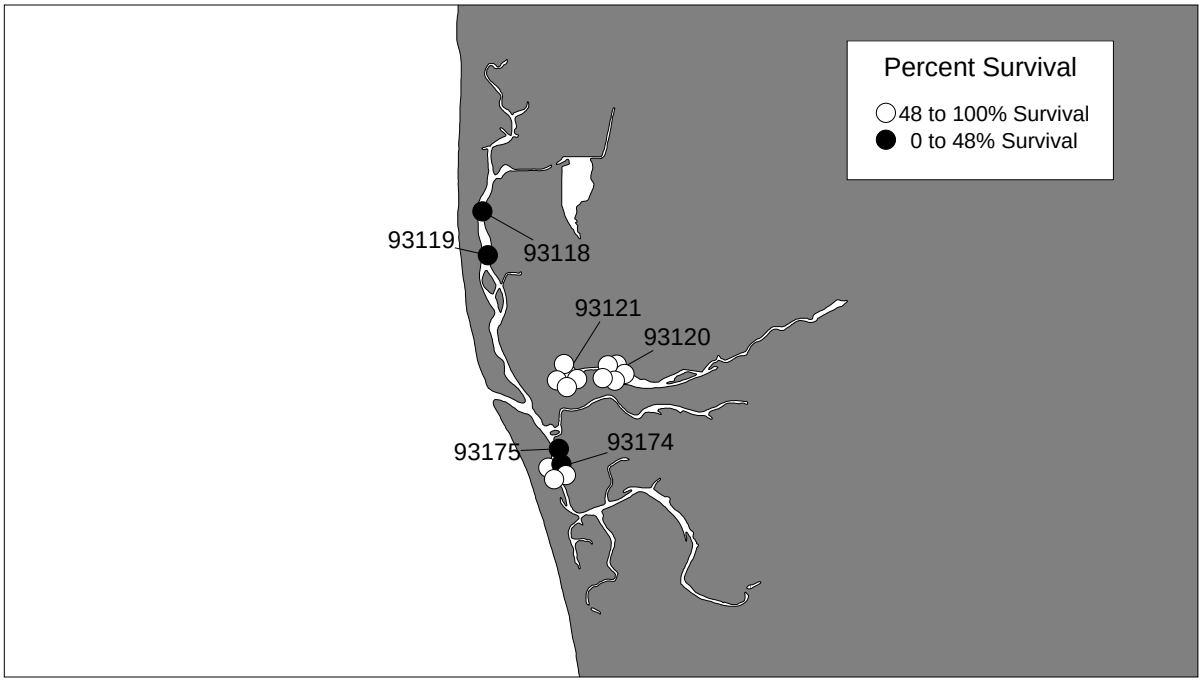


Figure 23d
Amphipod Toxicity Using Reference Envelope
for All Stations
Mission Bay and San Diego River Estuary



Tijuana River Estuary



as components of the BPTCP. Results indicated these tests were extremely sensitive to pollutants and/or other pore water constituents in the study area, particularly at the 100% porewater concentration. It is reasonable to expect that pore water sea urchin tests, which measure sublethal effects on sensitive early life stages, would be more sensitive than the amphipod solid phase tests, which measure adult mortality. It is also likely that all three protocols respond differently to different contaminants. The high sensitivity of the sea urchin protocols has been observed in other studies assessing pore water toxicity (Burgess *et al.*, 1993; Carr and Chapman, 1992; Long *et al.*, 1990).

Rhepoxynius solid phase test results agreed with *Strongylocentrotus* development (100% and 50%) pore water results in 61 of 117 concurrently tested samples (52%). For the 25% pore water dilution, results agreed in 48% of samples. The three dilutions for the *Strongylocentrotus* tests agreed with each other 56% of the time. In all but two cases, *Strongylocentrotus* results differed from each other because samples were less toxic as pore water was increasingly diluted. In one case the 50% pore water was toxic when the 100% and 25% were not, and in another case, the 50% and 25% were toxic when the 100% was not.

Carr and Chapman (1992) noted that sensitive toxicity test protocols are necessary to adequately characterize the toxicity of potentially contaminated sediments. Pore water tests provide the following advantages: allow the use of a variety of sensitive sublethal toxicity test protocols which have not yet been developed for solid phase tests; eliminate interference from physical factors such as sediment grain size; and allow test organisms to be directly exposed to the aqueous sediment fraction, the probable primary route of pollutant exposure to organisms (Adams *et al.*, 1985; DiToro, 1990). In addition, pore water is currently the only sediment matrix suitable for toxicity identification evaluations that may be useful in identifying toxicants responsible for observed sediment toxicity.

Despite the need to evaluate pore water toxicity, logistical issues of pore water extraction and handling are still a focus of current research (Carr *et al.*, 1995). Among the samples associated with high toxicity in the sea urchin pore water tests were a number from the selected reference stations. These stations had non-degraded benthic communities, relatively low concentrations of pollutants, and ammonia concentrations below levels expected to have an observable effect. The wide range in pore water toxicity at the reference stations was unexpected, and prevented identification of toxic sites using the reference envelope approach. Pore water properties and sampling manipulations that may have affected pore water test results are discussed later.

Samples of water collected one meter above the sediment surface were tested for toxicity at a number of stations. These subsurface water samples were tested as one of the suite of

screening bioassays conducted on suspected areas of water quality impairment. Sixty-five subsurface water samples were tested with the red abalone (*Haliotis rufescens*) larval shell development protocol. Of these, eleven samples were significantly toxic, indicating degradation of the water column in 17% of the stations tested. Water column testing has not been a consistent component of the BPTCP, and will probably be reserved for special investigations. The abalone test appears appropriate for this application.

The bivalve (*Mytilus sp.)* larval shell development test was used to test eight subsurface water samples and three pore water samples. This test was used only in cases where salinity was less than 30 or 26 parts per thousand, the low end of salinity ranges for abalone and sea urchin larval development tests, respectively. Because seawater salinities in the San Diego Bay region were usually in the acceptable range for abalone and sea urchins, the bivalve test was used sparingly. None of the subsurface water samples tested with mussels were significantly toxic, and one of three pore water samples tested with mussels was significantly toxic. This protocol is well established as a sensitive test method, and has the advantage of a relatively wide salinity range. In situations where the salinity range precludes the use of abalone or sea urchins, the bivalve test is an acceptable alternative.

The presence of mitotic aberrations in anaphase cells (cytogentic abnormalities) of *Strongylocentrotus* were determined in some samples. Cells undergoing mitosis were analyzed for chromosomal abnormalities. This porewater test is appropriate for identifying samples containing genotoxic compounds, which may affect reproductive capacity in a wide variety of organisms. Though the test is useful for specific applications, it proved time-consuming for assessing large numbers of samples. Most porewater samples that demonstrated increased aberration rates also were significantly toxic in larval development tests. Since the larval development test was considerably easier to quantify and was being used routinely as part of the study, the mitotic aberration endpoint was discontinued for logistical reasons. It would be useful in specific applications where the effects of genotoxic compounds must be assessed.

Evaluation of Utilization of Pore Water as a Test Medium for the BPTCP

The diffusive flux of dissolved chemicals through the sediment water interface into the overlying water column is a major component of sediment diagenesis and chemical cycles. Bioassay testing of the filtered pore water is an attempt to address exposure of animals living in the sediment matrix, or near the sediment/water interface, to chemicals not associated with the particulate phase. Equilibrium-partitioning theory predicts pore water is the controlling exposure medium in the toxicity of sediments to infaunal organisms (Adams *et al.*, 1985; DiToro, 1990). To accurately interpret pore water test results, it is

important to determine how manipulations of pore water during extraction and handling may have affected observed toxicity. The BPTCP utilized a low pressure (<200psi) squeezing extraction technique with filtration to 0.45 um, and subsequent freezing of pore water samples, prior to testing. There has been some debate regarding appropriate pore water extraction methods and sample manipulations for the purposes of toxicity testing (Carr et al., 1995; Schults et al., 1992). Squeezing techniques allow pore water to be selectively filtered, thus eliminating particulates.

Suspected artifacts from the squeezing technique may include chemical disequilibria through physical disruption of weakly charged ion/particulate associations or lysing of cell walls with resultant changes in concentration of dissolved and particulate organic carbon or other organic components. There is also concern that filtration has a profound effect on observed toxicity. Pore size and filter material can cause variability in measured chemical concentrations (Schults, *et al.*, 1992). Many scientists are now using centrifugation to obtain pore water from sediment for toxicity testing, because this method may be less subject to toxicity artifacts than squeezing (Lange *et al*., 1992; Giesy *et al.*, 1990).

Toxicity has been observed to decrease in bedded sediments which are tested after freezing and thawing, with observed changes assumed to be related to the release of soluble organic carbon through disruption of natural lattices, clay aggregates and organic matter (Schuytema *et al.*, 1989). Although solids are removed from pore water samples, there remain some soluble organic carbon concerns due to disruption of colloidal aggregations in the pore water, however centrifugation of pore water samples prior to freezing helps minimize this effect (Carr and Chapman, 1995). There are other unresolved concerns related to the toxicity testing of sediment pore waters which require additional study. These include sediment sample handling and storage conditions prior to testing, oxygen contamination, storage time of pore water samples prior to testing (Lange *et al.*, 1992) and sorption kinetics in toxicity test containers and extraction devices (Pittinger, 1988).

Dose responses from the three pore water dilutions demonstrate decreasing toxicity with increasing pore water dilution, confirming that some factor associated with pore water was causing toxicity. However, considering the uncertainty of introduced artifacts during sample manipulations, the ability to discriminate more severely impacted sediments from less severely impacted sediments (a primary goal of the BPTCP) is clearly compromised. As a result of this uncertainty, toxicity testing using pore water as the test medium was suspended in August, 1993, pending further method evaluation. Pore water extraction methods and pore water sample handling have been under evaluation by the BPTCP since that time, with preliminary results indicating that centrifugation and refrigerated (not frozen) sample storage may be the preferable methods when testing this matrix. Recent method comparison research of Carr and Chapman (1995) supports

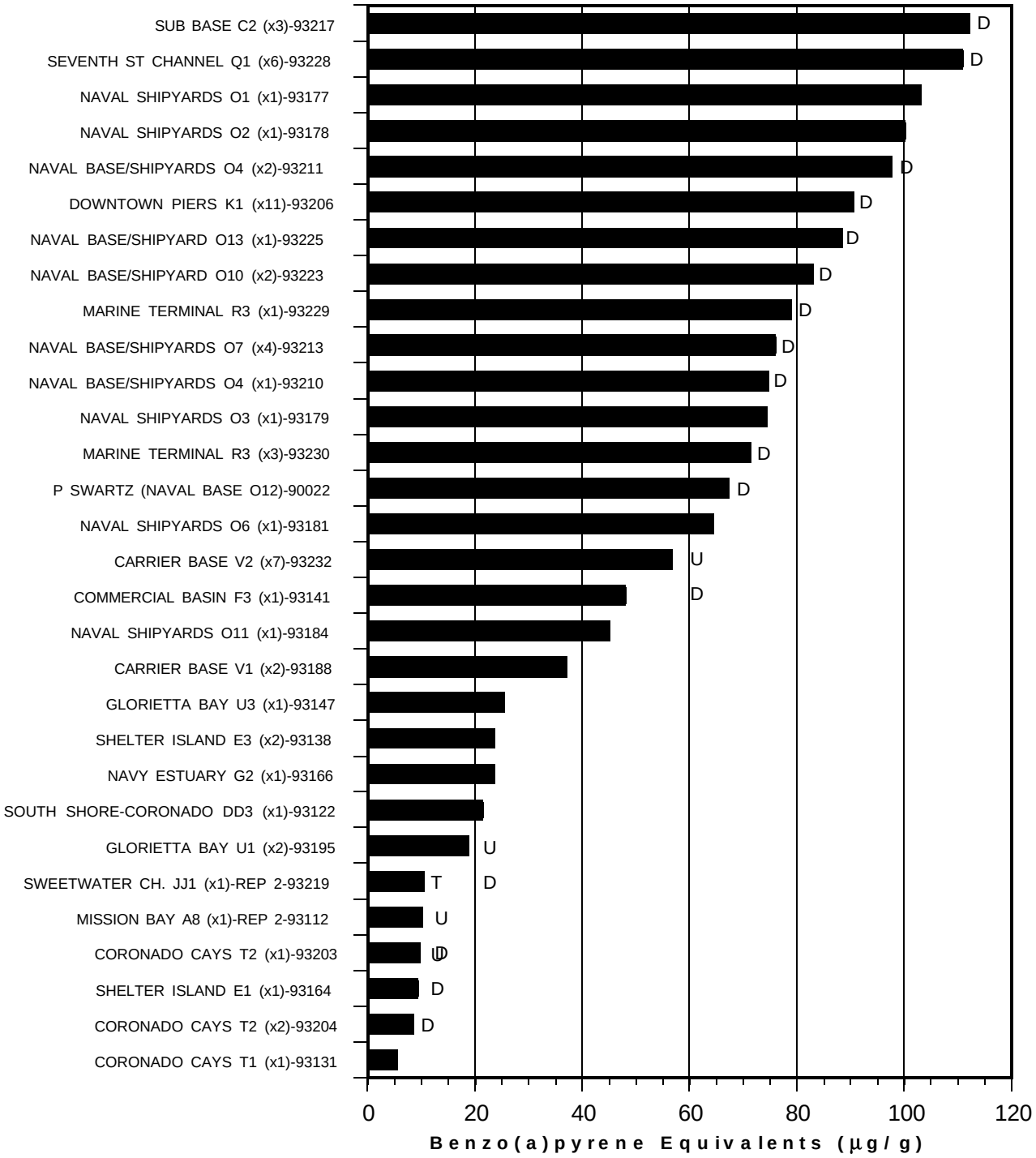
the use of squeezing technique yet concludes that in situations where hydrophobic organic compounds are a concern (as they are in this program), centrifugation is the method of choice for maximizing the sensitivity of the toxicity test. Sample storage and holding times were critical for all methods evaluated and require further investigation (Schults *et al.*, 1992).
As pore water test methods, test organism selection, and the interpretation of results continue to evolve, they will be evaluated for use by the BPTCP. Because test sensitivity is necessary for accurate sediment characterization, the *Strongylocentrotus* pore water larval development toxicity test protocol should continue to be included in BPTCP. At present, pore water toxicity data by themselves are difficult to interpret. If pore water toxicity tests are used in conjunction with solid phase toxicity tests, chemical measurements and benthic community evaluations, they can provide useful additional information when using a weight of evidence approach toward site characterization.

Distribution of P450 Reporter Gene System Response

Induction of the CYP1A1 gene on the human chromosome is produced by such compounds as dioxins, furans, dioxin-like PCB congeners (coplanar), and several high molecular weight polycyclic aromatic hydrocarbons. This induction and resulting production of the detoxifying enzyme, P450, infers that these xenobiotics are present at levels that are potentially toxic, carcinogenic, or mutagenic to organisms. The P450 Reporter Gene System (RGS) assay can measure the response of human (101L) cells to organic extracts when a firefly plasmid at the CYP1A1 site produces the enzyme luciferase. A luminometer is used to quantify the luciferase as a function of concentration and potency of the organics in the extract. Solvent extracts (using standard extraction methods EPA 3510, 3450 or 3550) of water, aquatic sediments, soils and tissues can be tested in the assay system, with a measured response in 16 hours (Anderson *et al.,* 1996).

Findings of the P450 Reporter Gene System (RGS) assay of sediment extracts from 30 stations are summarized in Figure 24, where the RGS responses (in 101L cells) are expressed as μg/g (ppm) of benzo(a)pyrene equivalents (BaPEq). The Mission Bay A8 (93112) station, Coronado Cays T2 (93203, 93204) stations, Shelter Island E1 & E3 (93138, 63164) and the Sweetwater Channel stations produced baseline responses in the range of 5.3 to 10.4 μg/g BaPEq. Figure 24 shows that all Naval Shipyard stations, the Commercial Basin station, the Marine Terminal and Downtown piers, as well as Seventh Street and the Sub Base stations all produced strong RGS responses. These responses suggest that benthic fish and invertebrates living in contact with these sediments have a high probability of P450 enzyme levels above background, which could result in chronic toxicity, and/or damage to tissues and reproductive potential.
http://www.norcalsetac.org/meetings.htm
Examination of the relationship between RGS response to sediment extracts and total PAHs concentration in sediments demonstrates

Figure 24. P450 Responses to Extracts of Sediments From San Diego Bay



P450-RGS response (expressed as benzo(a)pyrene equivalents) and benthic community index. Stations with degraded benthic communities are shown with a "D" label. Undegraded are shown with "UD," and transitional stations are shown with "T." Benthic community analysis was not performed on unlabeled stations.

a strong correlation (r^2= 0.86) between the two measures (Figure 25). This is expected, because samples significantly contaminated with PAHs and/or other compounds (coplanar PCBs) have been shown to produce induction of the CYP1A1 gene and the RGS response (Anderson *et al.*, 1995).

Figures 9a-d show stations with high molecular weight PAHs at the PEL (6676 ng/g) and above in black. Examination of these data demonstrated that RGS responses above 60 μg/g BaPEq were always associated with total PAHs at levels above the PEL. This comparison with the PEL suggested that sediment samples with RGS responses above 60 μg/g BaPEq also had a high probability of demonstrating a toxic biological effect, based on sediment quality guidelines. Interestingly, stations identified by RGS to contain significant amounts of inducing organic compounds (> 60μg/g BaPEq) were also found to have degraded benthic communities, at all stations where both analyses were performed. Toxicity test results did not demonstrate a similar strong association with the RGS response.

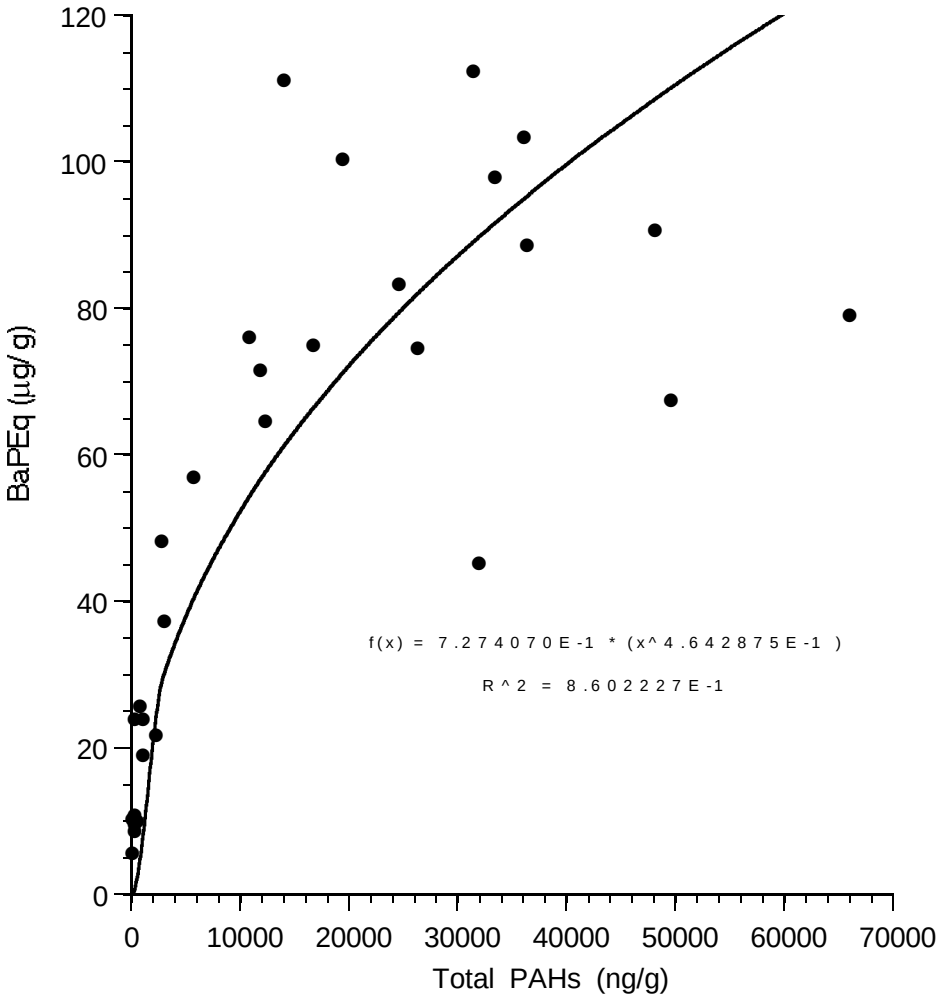
The P450 Reporter Gene System proved to be effective for rapidly (16 hr test) and inexpensively assessing the magnitude of PAHs at selected stations in the San Diego Bay Region. It further proved useful by demonstrating a RGS response threshold above which benthic community degradation was expected. This method may be appropriate as a screening test at additional locations when benthic community degradation and contamination from multiple PAHs, coplanar PCBs, dioxins and furans is suspected. The bioeffects branch of NOAA has utilized this assay in investigations of coastal studies in southern California, Charleston Harbor, S.C., Sabine Lake and Galveston Bay, Texas, and Biscane Bay Florida. In concert with other chemical and biological measures, this method provides additional convincing evidence for the assessment of overall pollution at sites of chemical concern.

Determination of Relationships Between Toxicity and Chemistry

Linear regression was used to describe the relationship between toxicity and chemical concentrations. The dependent variable values are assumed to be normally distributed around the predicted values on the regression line. If this assumption has been met, then a significance test evaluating the null hypothesis (slope of the regression equation is equal to zero), is performed. In addition to a significant probability ($p < 0.05$), the coefficient of determination (r^2) is also an indication of regression strength. The coefficient of determination value represents the proportion of total variance of the dependent variable which can be explained by the independent variable, with a r^2 value of greater than 0.60 being significant. Regression is preferable to non-parametric tests because there is greater power to detect significant relationships with this method (Zar, 1984).

Linear regressions were used to assess the relationship between *Rhepoxynius* (amphipod) mean survival and chemical concentration.

Figure 25. Total PAHs vs P450-RGS Response Expressed as Benzo(a)pyrene Equivalents



Systat® v.5.04 was used for all analyses. The arcsine (square root) transformation is utilized to equalize variance over the entire range of proportions. Chemistry data were checked for normality and transformed using Log(x+1), when necessary (Zar, 1984). Examination of residuals reveal homogeneity of variances exists when these transformations are performed and therefore, the statistical assumptions of a regression can be met. The coefficient of determination (r^2) was reported only when the linear regression was significant ($p<0.05$).

Regressions using amphipod data and chemical concentrations for all stations were analyzed. Testing the degree of dependence of amphipod survival on individual chemical concentrations yielded several regressions which are significant, however, there were no r^2 values greater than 0.072 (Table 13).

To investigate dependence of amphipods on chemistry within specific areas of the Bay, all stations were grouped into one of six specific areas (Appendix B). Groupings were performed to combine stations with similar physical characteristics or uses. These six groups were military use areas (Navy), commercial basins for shipping and industrial activities, small boat harbors and marinas, Mission Bay, rivers (San Diego and Tijuana), and "other" stations, which generally were in open areas removed from San Diego Bay shorelines. The area into which each station was grouped is reported in Appendix B. These regressions were used to test the degree of relationship between amphipod survival and specific areas in the San Diego Bay Region.

Regressions using the navy station group were significant for some chemical groups although no regression had an r^2 value greater than 0.272 (Table 14). In commercial basins, low and high molecular weight PAHs, several metals and one PCB compound were significant, but all had low r^2 values (Table 15). In the small boat harbor group, several PAH and PCB compounds and one pesticide were significant, however, no r^2 values were greater than 0.167 (Table 16). In river stations low molecular weight PAHs were strongly correlated with amphipod survival (Table 17), producing the most significant regressions of the statistical analysis. These regression results from the river stations were somewhat misleading, however, because PAH levels were low relative to most stations in San Diego Bay and to ERM guidelines. For regressions using the "other" station designations, several metals and PCB compounds and one PAH, were significant (Table 18) yet, r^2 values were never better than 0.265. When testing the six station groups, there were no significant regressions for chemistry or amphipods within the Mission Bay group. This was expected because of the low chemical concentrations, therefore no table is shown.

Ammonia, hydrogen sulfide and grain size are suspected non-anthropogenic contributors to toxicity, and have been discussed previously by Ankley *et al.*(1990), Knesovich *et al.* (In Press), and DeWitt *et al.* (1988). To investigate whether these natural

Table 13. Linear regression of amphipod survival dependence on chemistry concentrations for all stations (chemistry with * and all PCB and PAH compounds were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant).

Metal	n	p	r^2	Pesticide	n	p	r^2	PCB	n	p	r^2	PAH	n	p	r^2
Aluminum	217	0.000	0.047	ALDRIN	229	ns		PCB8	229	0.008	0.031	ACY	198	ns	
Antimony	217	0.015	0.027	CCHLOR*	229	ns		PCB15	78	ns		ACE	229	ns	
Arsenic	217	ns		TCHLOR*	198	ns		PCB18	229	0.001	0.049	ANT	229	ns	
Cadmium*	217	0.000	0.06	ACDEN	217	ns		PCB27	78	ns		BAA	229	ns	
Chromium	217	ns		GCDEN	186	ns		PCB31	78	0.018	0.072	BAP	229	ns	
Copper	217	ns		CLPYR	165	0.011	0.039	PCB44	229	ns		BBF	198	ns	
Iron*	217	ns		Total CHLR	229	ns		PCB49	78	ns		BKF	198	ns	
Manganese	217	ns		DACTH	186	0.000	0.049	PCB52	229	ns		BGP	198	ns	
Nickel	217	ns		OPDDD	229	0.000	0.060	PCB66	229	ns		BEP	229	ns	
Silver	217	0.023	0.024	PPDDD	229	0.000	0.057	PCB70	78	ns		BPH	229	ns	
Selenium	217	ns		OPDDE	229	ns		PCB74	78	ns		CHR	229	ns	
Tin	217	0.000	0.049	PPDDE	229	ns		PCB87	109	ns		DBA	229	ns	
Zinc	217	ns		OPDDT	229	ns		PCB95	78	ns		DMN	229	0.012	0.028
				Total DDT	229	ns		PCB97	78	ns		FLA	229	ns	
				DICLB	186	ns		PCB99	78	ns		FLU	229	ns	
				DIELDRIN	229	ns		PCB101	229	ns		IND	198	ns	
				HCHG	229	ns		PCB105	229	ns		MNP1	229	ns	
				HEPTACHL	229	0.000	0.068	PCB110	78	ns		MNP2	229	ns	
				HCB	229	ns		PCB118	229	ns		MPH1	229	ns	
				METHOXY	217	0.04	0.020	PCB128	229	ns		NPH	198	ns	
				MIREX	229	ns		PCB132	78	ns		PHN	229	ns	
				CNONA	186	ns		PCB138	229	ns		PER	229	ns	
				TNONA	217	ns		PCB149	78	ns		PYR	229	ns	
				TBT	217	ns		PCB153	229	ns		LMW PAH	229	ns	
								PCB156	78	ns		HMW PAH	229	ns	
								PCB157	78	ns		Total PAH	229	ns	
								PCB158	78	ns					
								PCB170	229	ns					
								PCB174	78	ns					
								PCB177	78	ns					
								PCB180	229	ns					
								PCB183	78	ns					
								PCB187	78	ns					
								PCB194	78	ns					
								PCB195	229	ns					
								PCB201	78	ns					
								PCB203	78	ns					
								PCB206	229	ns					
								PCB209	229	ns					
								Total PCB	229	ns					

Table 14. Linear regression of amphipod survival dependence on chemistry concentrations in navy stations (all chemistry data were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant). All PAH compound regressions were not significant and therefore not shown.

Metal	n	p	r^2	Pesticide	n	p	r^2	PCB	n	p	r^2
Aluminum	65	0.024	0.078	ALDRIN	65	ns		PCB 15	25	ns	
Antimony	65	ns		CCHLOR	65	ns		PCB 18	65	0.024	0.078
Arsenic	65	ns		OPDDD	65	ns		PCB 27	25	ns	
Cadmium	65	0.021	0.082	PPDDD	65	ns		PCB 31	25	0.007	0.272
Chromium	65	ns		TCHLOR	57	ns		PCB 44	65	ns	
Copper	65	ns		OPDDE	65	ns		PCB 49	25	ns	
Iron	65	ns		PPDDE	65	ns		PCB 52	65	ns	
Lead	65	0.014	0.092	OPDDT	65	ns		PCB 66	65	0.026	0.077
Manganese	65	ns		PPDDT	65	0.011	0.098	PCB 70	25	0.017	0.222
Mercury	65	0.022	0.081	Total DDT	65	ns		PCB 74	25	0.013	0.240
Silver	65	ns		ACDEN	65	ns		PCB 87	33	ns	
Nickel	65	ns		Total CHLR	65	ns		PCB 97	25	ns	
Selenium	65	ns		DIELDRIN	65	ns		PCB 95	25	ns	
Tin	65	0.000	0.215	HCHG	65	ns		PCB 99	25	ns	
Zinc	65	ns		HEPTACH	65	0.001	0.168	PCB 101	65	ns	
				HCB	65	ns		PCB 105	65	0.020	0.084
				METHOXY	65	ns		PCB 110	25	ns	
				CNONA	57	ns		PCB 118	65	ns	
				TNONA	65	ns		PCB 128	65	0.029	0.073
				TBT	65	ns		PCB 132	25	ns	
								PCB 138	65	ns	
								PCB 149	25	ns	
								PCB 153	65	ns	
								PCB 156	25	ns	
								PCB 158	25	ns	
								PCB 170	65	ns	
								PCB 174	25	ns	
								PCB 177	25	ns	
								PCB 180	65	ns	
								PCB 183	25	ns	
								PCB 187	25	ns	
								PCB 194	25	ns	
								PCB 195	65	ns	
								PCB 201	25	ns	
								PCB 203	25	ns	
								PCB 206	65	ns	
								PCB 209	65	ns	
								TTLPCB	65	ns	

Table 15. Linear regression of amphipod survival dependence on chemistry concentrations in commercial basin stations (all chemistry data were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant). All pesticide compound regressions were not significant and therefore not shown.

Metal	n	p	r^2	PAHs	n	p	r^2	PCBs	n	p	r^2
Aluminum	44	0.000	0.266	ACY	37	0.024	0.137	PCB 8	44	ns	
Antimony	44	ns		ACE	44	0.016	0.130	PCB 15	19	ns	
Arsenic	44	0.007	0.163	ANT	44	0.001	0.216	PCB 18	44	ns	
Cadmium	44	0.006	0.168	BAA	44	0.018	0.127	PCB 31	19	ns	
Chromium	44	0.026	0.112	BAP	44	0.010	0.146	PCB 44	44	ns	
Copper	44	ns		BBF	37	0.008	0.187	PCB 49	19	ns	
Iron	44	ns		BKF	37	0.009	0.180	PCB52	44	ns	
Lead	44	ns		BGP	37	0.009	0.180	PCB 66	44	ns	
Manganese	44	ns		BEP	44	0.020	0.123	PCB 70	19	ns	
Mercury	44	ns		BPH	44	ns		PCB 74	19	ns	
Nickel	44	ns		CHR	44	0.016	0.130	PCB 87	26	ns	
Silver	44	ns		DBA	44	0.014	0.135	PCB 95	19	ns	
Selenium	44	ns		DMN	44	ns		PCB 99	19	ns	
Tin	44	ns		FLA	44	0.025	0.114	PCB 101	44	ns	
Zinc	44	ns		FLU	44	0.008	0.158	PCB 105	44	ns	
				IND	37	0.005	0.207	PCB 110	19	ns	
				MNP1	44	ns		PCB118	44	ns	
				MNP2	44	0.013	0.137	PCB 128	44	ns	
				MPH1	44	0.039	0.097	PCB 132	19	ns	
				NPH	37	0.004	0.218	PCB 138	44	ns	
				PHN	44	0.023	0.116	PCB 149	19	ns	
				PER	44	0.019	0.124	PCB 153	44	ns	
				PYR	44	0.025	0.114	PCB 156	19	ns	
				TMN	37	ns		PCB 157	19	ns	
				HMW PAH	44	0.008	0.156	PCB 170	44	ns	
				LMW PAH	44	0.007	0.158	PCB 174	19	ns	
				Total PAH	44	0.006	0.168	PCB 177	19	ns	
								PCB 180	44	ns	
								PCB 183	19	ns	
								PCB 194	19	ns	
								PCB 195	44	ns	
								PCB 201	19	ns	
								PCB 203	19	ns	
								PCB 206	44	ns	
								PCB 209	44	0.000	0.091
								Total PCB	44	ns	

Table 16. Linear regression of amphipod survival dependence on chemistry concentrations in small boat stations (all chemistry data were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant). All metal concentration regressions were not significant and therefore not shown.

PAHs	n	p	r^2	PCBs	n	p	r^2	Pesticide	n	p	r^2
ACY	39	ns		PCB 5	22	ns		CCHLOR	44	ns	
ACE	44	ns		PCB 18	44	ns		TCHLOR	39	ns	
ANT	44	ns		PCB 31	22	ns		Total CHLR	44	ns	
BAA	44	ns		PCB 44	44	ns		OPDDD	44	ns	
BAP	44	ns		PCB 49	22	ns		PPDDD	44	ns	
BBF	39	ns		PCB 52	44	ns		OPDDE	44	ns	
BKF	39	ns		PCB 66	44	ns		PPDDE	44	ns	
BGP	39	0.015	0.150	PCB 70	22	ns		OPDDT	44	ns	
BEP	44	0.038	0.099	PCB 74	22	ns		PPDDT	44	ns	
CHR	44	ns		PCB 87	27	ns		Total DDT	44	ns	
DBA	44	0.043	0.094	PCB 95	22	ns		CNONA	39	ns	
FLA	44	0.009	0.153	PCB 97	22	ns		TNONA	44	0.047	0.091
FLU	44	0.034	0.102	PCB 101	44	ns		TBT	44	ns	
IND	39	0.035	0.114	PCB 105	44	ns					
MNP2	44	ns		PCB 110	22	ns					
MPH1	44	ns		PCB 118	44	ns					
NPH	39	ns		PCB 128	44	ns					
PHN	44	0.040	0.097	PCB 132	22	ns					
PER	44	ns		PCB 138	44	0.036	0.100				
PYR	44	0.006	0.167	PCB 149	22	ns					
LMW PAH	44	0.050	0.089	PCB 153	44	0.041	0.096				
HMW PAH	44	0.030	0.108	PCB 156	22	ns					
Total PAH	44	0.030	0.108	PCB 157	22	ns					
				PCB 170	44	ns					
				PCB 174	22	ns					
				PCB 177	22	ns					
				PCB 180	44	ns					
				PCB 183	22	ns					
				PCB 187	22	ns					
				PCB 194	22	ns					
				PCN 195	44	ns					
				PCB 201	22	ns					
				PCB 203	22	ns					
				PCB 206	44	ns					
				Total PCB	44	0.049	0.089				

Table 17. Linear regression of amphipod survival dependence on chemistry concentrations in river stations (all chemistry data were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant). All metal, pesticide, and PCB compound regressions were not significant and therefore not shown.

PAHs	n	p	r2
ACY	18	ns	
ACE	20	0.028	0.240
ANT	20	ns	
BAA	20	ns	
BAP	20	ns	
BBF	18	ns	
BKF	18	ns	
BGP	18	ns	
BEP	20	ns	
BPH	20	0.000	0.646
CHR	20	ns	
DBA	20	ns	
DMN	20	0.000	0.672
FLA	20	ns	
FLU	20	0.000	0.692
IND	18	ns	
MNP1	20	0.000	0.669
MNP2	20	0.000	0.634
MPH1	20	0.000	0.714
NPH	18	ns	
PHN	20	0.005	0.358
PER	20	ns	
PYR	20	ns	
TMN	18	0.000	0.591
LMW PAH	20	0.000	0.607
HMW PAH	20	ns	
Total PAH	20	ns	

Table 18. Linear regression of amphipod survival dependence on chemistry concentrations in "other" stations (all chemistry data were Log (x+1) transformed, r^2 is presented when $p<0.05$, ns=nonsignificant). All pesticide compound regressions were not significant and therefore not shown.

Metal	n	p	r^2	PAHs	n	p	r^2	PCBs	n	p	r^2
Aluminum	35	ns		ACY	28	ns		PCB 5	37	ns	
Antimony	35	0.002	0.255	ACE	37	ns		PCB 18	37	ns	
Arsenic	35	ns		ANT	37	ns		PCB 44	37	ns	
Cadmium	35	ns		BAA	37	ns		PCB 52	37	ns	
Chromium	35	0.017	0.161	BAP	37	ns		PCB 66	37	ns	
Copper	35	0.023	0.147	BBF	28	ns		PCB 87	9	ns	
Iron	35	0.009	0.188	BKF	28	ns		PCB 101	37	0.033	0.124
Lead	35	0.019	0.155	BGP	28	ns		PCB 105	37	ns	
Manganese	35	ns		BEP	37	ns		PCB 118	37	0.033	0.124
Mercury	35	ns		BPH	37	ns		PCB 128	37	ns	
Nickel	35	ns		CHR	37	ns		PCB 138	37	ns	
Silver	35	0.003	0.232	DBA	37	ns		PCB 153	37	0.017	0.151
Selenium	35	ns		DMN	37	ns		PCB 170	37	ns	
Tin	35	0.046	0.159	FLA	37	ns		PCB 180	37	ns	
Zinc	35	0.003	0.232	FLU	37	ns		PCB 195	37	ns	
				IND	28	ns		PCB 206	37	ns	
				MNP1	37	ns		PCB 209	37	ns	
				MNP2	37	ns		Total PCB	37	0.049	0.106
				MPH1	37	ns					
				NPH	28	0.005	0.265				
				LPHN	37	ns					
				PER	37	ns					
				PYR	37	ns					
				TMN	28	ns					
				LMW PAH	37	ns					
				HMW PAH	37	ns					
				Total PAH	37	ns					

factors influenced the effects of anthropogenic chemicals in test sediments from the San Diego Bay Region, data were adjusted to exclude tests where unionized ammonia was greater than O.4 mg/L in overlying water and/or hydrogen sulfide was greater than 0.06 mg/L. The 0.4 mg/L ammonia threshold value is based on the NOEC value for the EPA test protocols for marine amphipods (USEPA, 1994) and the 0.06 mg/L hydrogen sulfide threshold value is based on data presented by Knesovich *et al*. (In Press). A general trend is seen by DeWitt *et al.* (1988), in which survival decreases with increasing fines. However, because this trend was not apparent in the San Diego Bay Region and no clear cutoff has been conclusively demonstrated, data were not adjusted to exclude samples with a high percentage of fines. NH_3 and H_2S adjusted amphipod data were compared to the thirty two chemicals or chemical groups, for which PEL values have been derived, and to ERM and PEL summary quotients. Regressions were significant for cadmium, chromium, copper, nickel, silver, zinc, DDT, dieldrin, acenapthene, and the ERM and PEL summary quotients (Table 19). By eliminating high ammonia concentrations (>0.4 mg/L) and high hydrogen sulfide concentrations (0.06 mg/L), regressions do improve slightly, however r^2 values are generally low. It is prudent though to recognize that these natural factors may confound interpretation of toxicity results and that caution should be exercised when elevated ammonia or hydrogen is noted.

In summary, simple linear regressions provide few clues to understanding the relationship between amphipod survival in the toxicity tests and measured single chemical concentrations. When viewing scatter plots, it remains difficult to convincingly argue that there is, or should be, a linear toxic response to increasing chemical concentrations in natural settings. In industrialized settings such as San Diego Bay, where multiple pollutants are common, co-variation and possible synergistic effects within a group of multiple pollutants further confound the separation of effects to single pollutants. A single multiple regression or a variable selection technique may statistically better describe the relationship between toxicity and multiple chemicals, but these were not performed in this analysis.

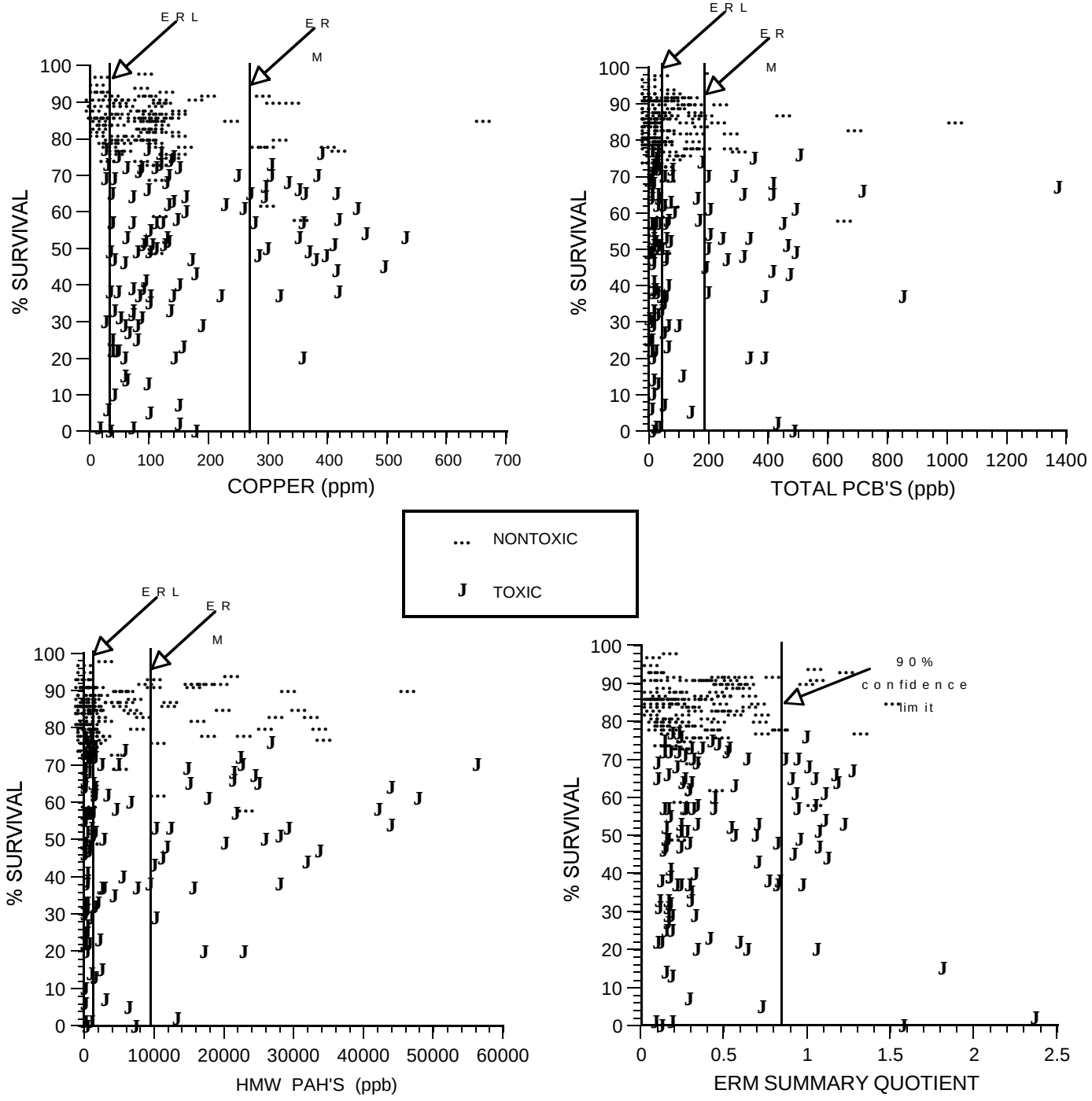
Figure 26 is typical of chemical vs. toxicity scatter plots seen throughout the region, with considerable scatter at low chemical concentrations and a gradual decrease in survival at elevated chemical concentrations. Because regressions did not generally support a linear toxic response to chemical pollutants, it is suspected that most organisms are tolerant of pollutants until a threshold is exceeded. This threshold effect appears well demonstrated in the San Diego Bay Region's benthic communities setting, as illustrated in Figure 14.

Although it was less evident for acute toxicity tests, where high amphipod survival was observed even at elevated chemical levels (Figure 26), a distinct response pattern still emerges. When the EMAP approach for determination of toxicity (significantly different from controls and less than 80% of controls) was used, 28 of 39 (72%) sediment samples were toxic when copper

Table 19. Linear regression of amphipod survival dependence on chemical analytes for which PEL levels have been developed. Amphipod data has overlying unionized ammonia values >0.4 ppm and hydrogen sulfide values >0.06 ppb removed (all chemical data are Log (x+1) transformed. r^2 is presented when $p<0.05$, ns= nonsignificant).

ANALYTE	n	p	r2
Metal			
Arsenic	193	ns	
Cadmium	193	0.000	0.074
Chromium	193	0.028	0.025
Copper	193	0.014	0.031
Lead	176	ns	
Nickel	193	0.003	0.044
Mercury	193	ns	
Silver	193	0.008	0.036
Zinc	193	0.001	0.057
Pesticide			
Total Chlordane	193	ns	
PPDDE	193	ns	
PPDDT	193	0.000	0.068
Total DDT	193	0.008	0.036
Dieldrin	193	0.023	0.027
Lindane	193	ns	
PAH			
ACY	170	ns	0.031
ACE	193	ns	
ANT	193	ns	
BAA	193	ns	
BAP	193	ns	
CHR	193	ns	
DBA	193	ns	
FLA	193	ns	
FLU	193	ns	
MNP2	193	ns	
NPH	170	ns	
PHN	193	ns	
PYR	193	ns	
LMW PAH	193	ns	
HMW PAH	193	ns	
Total PAH	193	ns	
PCB			
Total PCB	193	ns	
Summary Quotients			
PELQ	184	0.050	0.020
ERMQ	184	0.014	0.033

Figure 26. Amphipod Survival vs ERM Summmary Quotient or Chemical Level



concentrations exceeded the ERM value whereas only about 7 of 28 samples (25%) were toxic when copper concentrations were below the ERL value. This was also seen with total PCBs with 73% of the samples being toxic when PCB concentrations exceeded the ERM value and only 53% toxic below the ERL. Because it is suspected that toxicity in urban bays is caused by exposure to complex mixtures of chemicals comparisons to ERM summary quotients (multiple chemical indicators) were made. The highest incidence of toxicity (>78%) is found in samples with elevated ERM summary quotients (>0.85), supporting the theory that the effects of elevated levels of multiple pollutants may elucidate the toxic response. This pattern of increased incidence of toxicity when chemical concentrations exceed established sediment quality guidelines or the summary quotient 90% confidence interval seems to support the threshold response theory for amphipod bioassays in the San Diego Bay Region.

Guideline thresholds are quantitatively estimated from large national or statewide data sets, as described earlier, but the applicability of calculated values may be limited in specific water bodies. Use of unique guidelines for the San Diego Bay Region, which account for local physical, chemical and biological conditions, would be optimal when evaluating data. However, without substantial additional data, chemical specific thresholds for the San Diego Bay region cannot be accurately determined. Currently the most useful tools for addressing the relationship between toxicity and chemical concentration appears to be threshold approaches, such as the ERM/ERL and TEL/PEL guidelines.

Station Specific Sediment Quality Assessments

One of the primary goals of the BPTCP is to establish state guidelines under which contaminated or toxic stations can be designated "toxic hot spots". These guidelines are currently being developed based on data collected throughout the state. Although final guidelines are contingent upon further data analysis, the "toxic hot spot" definition currently utilized by the BPTCP, requires that one or more of the following criteria must be met:

1. The water or sediment exhibits toxicity associated with toxic pollutants, based on toxicity tests acceptable to the SWRCB or the RWQCB. To determine whether toxicity exists, recurrent measurements (at least two separate sampling dates) should demonstrate an effect.

2. Significant degradation in biological populations and/or benthic communities associated with presence of elevated levels of toxic pollutants.

3. The site exceeds water or sediment quality objectives for toxic pollutants which are contained in appropriate water quality control plans, or exceeds water quality criteria promulgated by the U.S. Environmental Protection Agency.

4. The tissue toxic pollutant levels of organisms collected from the site exceed levels established by the United States Food and Drug Administration (FDA) for protection of human health, or the National Academy of Sciences (NAS) for the protection of human health or wildlife.

Because tissue residues were not analyzed in this study, criteria are limited to the first three. Satisfying any one of these criteria can designate a site a "toxic hot spot". Satisfying more than one criterion and the severity demonstrated within each criterion determines the weighting for which qualitative rankings can be made. In this report, stations were not designated as "toxic hot spots", because this designation is still under evaluation and development by the BPTCP. Instead, stations were prioritized for further evaluation for hot spot status. This priority was classified as high, moderate, low, or no action and may be used by State and Regional Water Board staff to direct further investigations at these stations. Each station receiving a high to low priority ranking meets one or more of the first three criteria established above. Those meeting all three criteria were designated as the highest priority for further action.

Stations were evaluated for repeat toxicity (criterion 1) using the reference envelope method, the most conservative measure developed. Only those stations which demonstrated amphipod survival less than 48% in repeated tests, without confounding ammonia, hydrogen sulfide or grain size effects, were considered to exhibit repeat toxicity hits. Because only one critical value could be determined for any of the dilutions of the pore water bioassays, pore water toxicity results were not evaluated for repeat toxicity when prioritizing stations.

Stations with repeat toxicity and elevated chemistry and/or degraded benthic communities, were assigned a moderate or high priority. Stations with repeat toxicity, but lacking elevated chemistry or degraded benthic communities, were assigned a low priority (Tables 20 and 21- REPEAT TOXICITY HITS).

Stations with only a single toxicity hit were also considered a moderate or high priority, when associated with elevated chemistry and/or degraded benthic communities. Stations with a single toxicity hit, but lacking elevated chemistry or degraded benthic communities, were assigned a low priority. (Tables 20 and 21- SINGLE TOXICITY HITS).

Nineteen stations demonstrated repeat or single toxicity hits but were given a "no action" recommendation at this time (Tables 20 and 21). These stations had measured hydrogen sulfide or ammonia concentrations which confounded interpretation of the bioassay test results. Chemistry levels were low, or not analyzed, and the benthic community was undegraded or transitional, where sampled. These results provided little or no evidence that these stations should be prioritized for hot spot status. A toxicity identification evaluation (TIE) should be considered for these

TABLE 20

FUTURE INVESTIGATION PRIORITY LIST FOR THE SAN DIEGO BAY REGION

Stations With Synpotic Chemical, Toxicological and Benthic Community Analyses

STANUM	STATION	IDORG	LEG	H2S	NH3	% AMPHI. SURVIVAL	>4X ERM OR >5.9X PEL	ERMQ	PELQ	BENTHICS	COMMENTS	PRIORITY
	REPEAT TOXICITY											
90009.0	28 SWARTZ (7TH ST CHANNEL Q1)	893	23	nd	0.016	5.00	Chlordane	0.732	0.990	DEGRADED	TOXICITY, ELEVATED CHEM, BENTHIC HIT	HIGH
93228.0	SEVENTH ST CHANNEL Q1 (x6)	895	23	nd	0.010	2.00	Chlordane	**2.373**	**3.082**	DEGRADED	TOXICITY, ELEVATED CHEM, BENTHIC HIT	HIGH
90025.0	SDNI-N5 (CARRIER BASE V2)	899	23	nd	**0.643**	37.00		0.294	0.454	UNDEGRADED	NH3>0.4	NO ACTION
93232.0	CARRIER BASE V2 (x7)	1001	23	nd	**0.773**	35.00		0.300	0.481	UNDEGRADED	NH3>0.4	NO ACTION
	SINGLE TOXICITY											
90002.0	12 SWARTZ(DOWNTOWN ANCH)-REP 1	878	22	nd	**1.836**	15.00	Chlordane	**1.818**	**2.444**	DEGRADED	TOXICITY (NH3>0.4), ELEVATED CHEM, BENTHIC HIT	HIGH
93210.0	NAVAL BASE/SHIPYARDS O4 (x1)	863	22	0.0023	**0.775**	37.00		**0.975**	1.157	DEGRADED	TOXICITY (NH3>0.4), ELEVATED CHEM, BENTHIC HIT	HIGH
90051.0	16 SWARTZ (INTERCONT. MARINA)	818	20	0.0010	**3.340**	1.00		0.190	0.301	TRANSITIONAL	NH3>0.4	LOW
93219.0	SWEETWATER CH JJ1 (x1)-REP 2	876	22	nd	0.319	31.00		0.115	0.188	TRANSITIONAL		LOW
	DEGRADED BENTHICS											
90007.0	25 SWARTZ (NAVAL BASE/SY O10)	887	23	nd	0.014	86.00		0.702	1.025	DEGRADED		LOW
93223.0	NAVAL BASE/SHIPYARD O10 (x2)	888	23	nd	0.016	79.00		0.847	**1.308**	DEGRADED	ELEVATED CHEM	MODERATE
93224.0	NAVAL BASE/SHIPYARD O10(x6)	889	23	nd	0.010	90.00	Zinc	0.623	0.994	DEGRADED	ELEVATED CHEM	MODERATE
93211.0	NAVAL BASE/SHIPYARDS O4 (x2)	864	22	nd	0.158	86.00	Antimony, Copper, PCB	**1.509**	**1.945**	DEGRADED	ELEVATED CHEM	MODERATE
90021.0	K SWARTZ (NAVAL BASE O4)	862	22	nd	0.060	93.00		0.626	0.961	DEGRADED		LOW
90006.0	23 SWARTZ (NAVAL BASE O7)	865	22	nd	0.054	92.00	Chlordane	**1.056**	**1.487**	DEGRADED	ELEVATED CHEM	MODERATE
93212.0	NAVAL BASE/SHIPYARDS 07 (x1)	866	22	nd	0.026	91.00	Chlordane	0.589	0.847	DEGRADED	ELEVATED CHEM	MODERATE
93213.0	NAVAL BASE/SHIPYARDS O7 (x4)	867	22	nd	0.010	94.00	Chlordane	**1.230**	**1.730**	DEGRADED	ELEVATED CHEM	MODERATE
93227.0	SEVENTH ST CHANNEL Q1 (x5)	894	23	nd	0.076	79.00	Chlordane	0.837	1.175	DEGRADED	ELEVATED CHEM	MODERATE
93206.0	DOWNTOWN PIERS K1 (x11)	848	21	nd	0.048	95.00	PAHs	**1.042**	**1.956**	DEGRADED	ELEVATED CHEM	MODERATE
90004.0	15 SWARTZ (G ST. PIER MARINA)	849	21	nd	0.220	77.00		0.494	0.736	DEGRADED		LOW
93207.0	G ST PIER MARINA L1 (x4)	850	21	nd	0.173	89.00		0.454	0.674	DEGRADED		LOW
90022.0	P SWARTZ (NAVAL BASE O12)	868	22	nd	0.061	91.00	PAHs	**1.001**	**1.522**	DEGRADED	ELEVATED CHEM	MODERATE
93214.0	NAVAL BASE/SHIPYARDS O12 (x3)	869	22	nd	0.031	93.00		0.465	0.710	DEGRADED		LOW
93215.0	NAVAL BASE/SHIPYARDS O12 (x4)	870	22	nd	0.017	88.00		0.361	0.578	DEGRADED		LOW
90008.0	27 SWARTZ (NAVAL BASE/SH O13)	890	23	nd	0.008	92.00		0.419	0.665	DEGRADED		LOW
93225.0	NAVAL BASE/SHIPYARD O13 (x1)	891	23	0.0213	0.013	81.00		0.719	1.130	DEGRADED		LOW
93226.0	NAVAL BASE/SHIPYARD O13 (x3)	892	23	nd	0.019	91.00		0.642	1.033	DEGRADED		LOW
90010.0	31 SWARTZ (MARINE TERMINAL R3)	896	23	nd	0.077	86.00		0.145	0.254	DEGRADED		LOW
93229.0	MARINE TERMINAL R3 (x1)	897	23	nd	0.109	70.00	PAHs	**0.876**	**1.504**	DEGRADED	ELEVATED CHEM	MODERATE
93230.0	MARINE TERMINAL R3 (x3)	898	23	nd	0.056	63.00		0.449	0.737	DEGRADED		LOW
93116.0	SAN DIEGO RIVER B1 (x4)-REP 1	881	22	nd	0.216	92.00		0.282	0.381	DEGRADED		LOW
93116.0	SAN DIEGO RIVER B1 (x4)-REP 2	882	22	nd	0.098	92.00	Chlordane	0.540	0.770	DEGRADED	ELEVATED CHEM	MODERATE
93116.0	SAN DIEGO RIVER B1 (x4)-REP 3	883	22	nd	0.162	78.00	Chlordane	0.728	1.026	DEGRADED	ELEVATED CHEM	MODERATE
90028.0	NSB-M1 (SUB BASE C2)	871	22	nd	0.078	84.00	PAHs	0.577	1.038	DEGRADED	ELEVATED CHEM	MODERATE
93216.0	SUB BASE C2 (x1)	872	22	nd	0.079	93.00		0.201	0.351	DEGRADED		LOW
93217.0	SUB BASE C2 (x3)	873	22	nd	0.074	81.00		0.472	0.818	DEGRADED		LOW
90012.0	34 SWARTZ (C.V. YACHT BASIN)	824	20	0.0002	0.334	57.00		0.135	0.243	DEGRADED		LOW
93196.0	CHULA V. YACHT BASIN S1 (x1)	825	20	0.0003	0.260	78.00		0.236	0.426	DEGRADED		LOW
93197.0	CHULA V. YACHT BASIN S1 (x3)	826	20	0.0003	0.165	79.00		0.177	0.308	DEGRADED		LOW
90003.0	14 SWARTZ (DOWNTOWN PIERS)	846	21	nd	0.084	70.00		0.314	0.483	DEGRADED		LOW
93205.0	DOWNTOWN PIERS K1 (x9)	847	21	nd	0.167	84.00	PAHs	0.329	0.552	DEGRADED	ELEVATED CHEM	MODERATE
93107.0	MISSION BAY A3 (x1)-REP 1	853	21	nd	0.075	57.00		0.311	0.429	DEGRADED		LOW
93107.0	MISSION BAY A3 (x1)-REP 2	854	21	nd	0.046	77.00		0.364	0.483	DEGRADED		LOW
93204.0	CORONADO CAYS T2 (x2)	845	21	nd	0.062	82.00		0.140	0.234	DEGRADED		LOW
93220.0	SWEETWATER CH. JJ1 (x8)-REP 3	877	22	nd	0.129	81.00		0.088	0.150	DEGRADED		LOW
93208.0	G ST. PIER MARINA L1 (x5)	851	21	nd	0.064	83.00		0.728	1.047	DEGRADED		LOW
	CHEMISTRY-Individual Chemicals											
93107.0	MISSION BAY A3 (x1)-REP 3	855	21	nd	0.145	73.00	Chlordane	0.535	0.724	TRANSITIONAL	ELEVATED CHEM	MODERATE
93221.0	DOWNTOWN ANCH. J1 (x1)-REP 2	879	22	nd	0.143	83.00	Chlordane	0.564	0.803	UNDEGRADED	ELEVATED CHEM	LOW
STANUM	**STATION**	**IDORG**	**LEG**	**H2S**	**NH3**	**% AMPHI. SURVIVAL**	**>4X ERM OR >5.9X PEL**	**ERMQ**	**PELQ**	**BENTHICS**	**COMMENTS**	**PRIORITY**
	REPEAT TOXICITY HITS											
90009.0	28 SWARTZ	158	7	not analyzed	0.002	0.00	Chlordane, DDT	**1.570**	**1.839**	not analyzed	ELEVATED CHEM, SITE DEGRADED IN LEG 23	HIGH
93179.0	NAVAL SHIPYARDS O3 (x1)	797	19	not analyzed	**0.539**	20.00		**1.056**	**1.534**	not analyzed	ELEVATED CHEM, ADJACENT SITE DEGRADED	HIGH
93179.0	NAVAL SHIPYARDS O3 (x1)-REP 1	1122	27	0.0003	0.059	44.00		**1.119**	**1.525**	not analyzed	ELEVATED CHEM, ADJACENT SITE DEGRADED	HIGH
90043.0	CORONADO WHARF	192	12	not analyzed	**0.884**	29.00		0.169	0.249	not analyzed	NH3>0.4	NO ACTION
90043.0	CORONADO WHARF-REP 1	1156	28	0.0016	**0.423**	33.00		0.113	0.187	not analyzed	NH3>0.4	NO ACTION
90043.0	CORONADO WHARF-REP 2	1157	28	0.0030	0.224	43.00		0.703	0.996	not analyzed		LOW
90030.0	BF SCHROEDER SITE F	179	12	not analyzed	0.066	47.00	PAHs	**1.067**	**1.788**	not analyzed	ELEVATED CHEM	MODERATE
90030.0	BF SCHROEDER SITE F	749	16	not analyzed	0.204	43.00		not analyzed	not analyzed	not analyzed		LOW

TABLE 21

FUTURE INVESTIGATION PRIORITY LIST FOR THE SAN DIEGO BAY REGION

Stations Without Synpotic Chemical, Toxicological and Benthic Community Analyses

STANUM	STATION	IDORG	LEG	H2S	NH3	% AMPHI. SURVIVAL	>4X ERM OR >5.9X PEL	ERMQ	PELQ	BENTHICS	COMMENTS	PRIORITY
	REPEAT TOXICITY											
90009.0	28 SWARTZ	158	7	not analyzed	0.002	0.00	Chlordane, DDT	**1.570**	**1.839**	not analyzed	ELEVATED CHEM, SITE DEGRADED IN LEG 23	HIGH
93179.0	NAVAL SHIPYARDS O3 (x1)	797	19	not analyzed	**0.539**	20.00		**1.056**	**1.534**	not analyzed	ELEVATED CHEM, ADJACENT SITE DEGRADED	HIGH
93179.0	NAVAL SHIPYARDS O3 (x1)-REP 1	1122	27	0.0003	0.059	44.00		**1.119**	**1.525**	not analyzed	ELEVATED CHEM, ADJACENT SITE DEGRADED	HIGH
90043.0	CORONADO WHARF	192	12	not analyzed	**0.684**	29.00		0.169	0.249	not analyzed	NH3>0.4	NO ACTION
90043.0	CORONADO WHARF-REP 1	1156	28	0.0016	**0.423**	33.00		0.113	0.187	not analyzed	NH3>0.4	NO ACTION
90043.0	CORONADO WHARF-REP 2	1157	28	0.0030	0.224	43.00		0.703	0.996	not analyzed		LOW
90030.0	BF SCHROEDER SITE F	179	12	not analyzed	0.066	47.00	PAHs	**1.067**	**1.768**	not analyzed	ELEVATED CHEM	MODERATE
90030.0	BF SCHROEDER SITE F	749	16	not analyzed	0.204	43.00		not analyzed	not analyzed	not analyzed		LOW
93122.0	SOUTH SHORE-CORONADO DD3 (x1)	725	16	not analyzed	**0.740**	23.00		0.416	0.617	not analyzed	NH3>0.4	NO ACTION
93122.0	S.S.- CORONADO DD3 (x1) REP 1	1013	24	nd	**0.463**	33.00		0.306	0.471	not analyzed	NH3>0.4	NO ACTION
90036.0	STORM DRAIN- ROHR CHANNEL	185	5	not analyzed	**0.894**	27.00		0.162	0.253	not analyzed	NH3>0.4	NO ACTION
90036.0	STORMDRAIN EA (ROHR CH.) REP 3	1024	24	nd	0.119	1.00		0.090	0.144	not analyzed		LOW
90036.0	STORMDRAIN EA (ROHR CH.) REP 1	1022	24	**0.0679**	0.136	0.00		0.131	0.174	not analyzed	H2S> 0.06	NO ACTION
93125.0	SILVER STRAND FF4 (x4) REP 1	1016	24	nd	**0.514**	38.00		0.121	0.199	not analyzed	NH3>0.4	NO ACTION
93125.0	SILVER STRAND FF4 (x4) REP 2	1017	24	nd	**0.720**	22.00		0.102	0.171	not analyzed	NH3>0.4	NO ACTION
93125.0	SILVER STRAND FF4 (x4) REP 3	1018	24	**1.2744**	**0.484**	22.00		0.125	0.210	not analyzed	H2S> 0.06, NH3>0.4	NO ACTION
93158.0	SOUTH BAY GG1 (x1) REP 1	1035	24	nd	0.043	33.00		0.163	0.265	not analyzed		LOW
93158.0	SOUTH BAY GG1 (x1) REP 2	1036	24	nd	0.108	39.00		0.175	0.277	not analyzed		LOW
93158.0	SOUTH BAY GG1 (x1) REP 3	1037	24	nd	0.072	46.00		0.141	0.233	not analyzed		LOW
90024.0	SDNI-N1	173	7	not analyzed	**0.684**	40.00		0.323	0.506	not analyzed	NH3>0.4, SITE UNDEGRADED IN LEG 23	NO ACTION
90025.0	SDNI-N5	174	7	not analyzed	**0.925**	7.00		0.295	0.489	not analyzed	NH3>0.4, SITE UNDEGRADED IN LEG 23	NO ACTION
93188.0	CARRIER BASE V1 (x2)	806	19	not analyzed	**2.593**	37.00		0.220	0.349	not analyzed	NH3>0.4 SITE UNDEGRADED IN LEG 23	NO ACTION
90057.0	5 SDG&E	206	12	not analyzed	0.011	25.00		0.147	0.249	not analyzed		LOW
90057.0	5 SDG&E REP 1	1019	24	nd	0.046	41.00		0.176	0.290	not analyzed		LOW
90057.0	5 SDG&E REP 2	1020	24	0.0132	0.011	39.00		0.172	0.283	not analyzed		LOW
90057.0	5 SDG&E REP 3	1021	24	nd	0.032	31.00		0.169	0.281	not analyzed		LOW
	SINGLE TOXICITY											
90007.0	25 SWARTZ	156	7	not analyzed	0.004	37.00	Mercury	0.820	1.088	not analyzed	ELEVATED CHEM, SITE DEGRADED IN LEG 23	MODERATE
90008.0	27 SWARTZ	157	7	not analyzed	0.010	29.00		0.333	0.564	not analyzed	SITE DEGRADED IN LEG 23	MODERATE
90022.0	P SWARTZ	171	7	not analyzed	0.008	38.00		0.771	1.207	not analyzed	SITE DEGRADED IN LEG 22	MODERATE
93181.0	NAVAL SHIPYARDS O6 (x1)	799	19	not analyzed	0.042	45.00		**0.920**	**1.382**	not analyzed	ELEVATED CHEM	MODERATE
90010.0	31 SWARTZ	159	6	not analyzed	**1.291**	39.00		not analyzed	not analyzed	not analyzed	NH3>0.4, SITE DEGRADED IN LEG 23	LOW
90039.0	CL	188	12	not analyzed	0.090	38.00	Chlordane, DDT	0.835	1.156	not analyzed	ELEVATED CHEM	MODERATE
93178.0	NAVAL SHIPYARDS O2 (x1)	796	19	not analyzed	0.350	20.00		0.647	0.936	not analyzed		LOW
93166.0	NAVY ESTUARY G2 (x1)	779	18	not analyzed	**1.129**	20.00		0.336	0.501	not analyzed	NH3>0.4	NO ACTION
93118.0	TIJUANA R. ESTUARY HH1 (x2)	713	15	0.0005	0.187	30.00	DDE	not analyzed	not analyzed	not analyzed	ELEVATED CHEM	MODERATE
90018.0	D DE LAPPE	748	16	not analyzed	0.039	19.00		not analyzed	not analyzed	not analyzed		LOW
90023.0	NM SANDBAG	172	7	not analyzed	0.378	32.00		0.173	0.302	not analyzed		LOW
90050.0	10 SWARTZ	199	7	not analyzed	0.004	47.00		0.240	0.416	not analyzed		LOW
90055.0	43 SWARTZ	204	7	not analyzed	0.075	37.00		0.238	0.372	not analyzed		LOW
90102.0	HARBOR BRIDGE 71A	256	7	not analyzed	0.113	14.00		0.149	0.243	not analyzed		LOW
90104.0	WEST BASIN ENTRANCE (71C) REF	275	12	not analyzed	**1.046**	13.00		0.192	0.314	not analyzed	NH3>0.4	NO ACTION
93106.0	MISSION BAY A2 (x1)-REP 2	1102	27	0.0007	0.106	25.00		0.190	0.275	not analyzed		LOW
93117.0	SAN DIEGO RIVER B2 (x2)	1029	24	0.0125	0.110	0.00		not analyzed	not analyzed	not analyzed		LOW
93119.0	TIJUANA R. ESTUARY HH1 (x1)	714	15	0.0015	0.224	22.00	DDE, DDT	0.599	0.726	not analyzed	ELEVATED CHEM	MODERATE
93127.0	SOUTH BAY GG2 (x1)	1028	24	nd	0.096	47.00		not analyzed	not analyzed	not analyzed		LOW
93128.0	SOUTHBAY GG5 (x1)	1033	24	nd	0.031	27.00		not analyzed	not analyzed	not analyzed		LOW
93132.0	CORONADO CAYS T3 (x1)	1025	24	nd	0.004	47.00		not analyzed	not analyzed	not analyzed		LOW
93138.0	SHELTER ISLAND E3 (x2)	741	16	not analyzed	0.020	29.00		0.185	0.282	not analyzed		LOW
93148.0	CHANNEL-CORONADO Y1 (x2)	751	16	not analyzed	**0.525**	47.00		0.151	0.225	not analyzed	NH3>0.4	NO ACTION
93154.0	NORTH SHORE-MOUTH CC4 (x1)	763	17	not analyzed	**0.836**	31.00		not analyzed	not analyzed	not analyzed	NH3>0.4	NO ACTION
93159.0	SOUTH BAY GG3 (x1)	768	17	not analyzed	**0.675**	21.00		not analyzed	not analyzed	not analyzed	NH3>0.4	NO ACTION
93174.0	TIJUANA R. ESTUARY HH3 (x2)	787	18	not analyzed	0.282	6.00		not analyzed	not analyzed	not analyzed		LOW
93175.0	TIJUANA R. ESTUARY HH3 (x3)	788	18	not analyzed	0.141	10.00	DDE, DDT	not analyzed	not analyzed	not analyzed	ELEVATED CHEM	MODERATE

sites to confirm the source of toxicity as non-anthropogenic. Stations were evaluated for benthic community condition using the benthic index discussed earlier (Table 11). Stations determined to be degraded, with elevated chemistry and/or toxicity, were assigned a moderate or high priority. Stations determined to be degraded, but which did not demonstrate elevated chemistry or toxicity, were assigned a low priority. Transitional and undegraded stations were not considered a priority unless chemical or toxicity results initially prioritized the stations. (Table 20- DEGRADED BENTHICS)

Stations were evaluated for elevated chemistry (criterion 3) using an ERM Summary Quotient >0.85 or a PEL Summary Quotient >1.29. In the earlier discussion of ERM and PEL summary quotients, it was determined these values are statistically above the 90% confidence interval of summary quotients from all stations analyzed. These quotients were used to identify stations where multiple pollutants were near or above established ERM and PEL guidelines (Table 22-CHEMISTRY-Summary Quotients). As shown in Figure 14, 100% of the stations analyzed for benthics were found to be degraded when chemical analysis demonstrated an ERMQ above 0.85. Although the eighteen stations in Table 22 (CHEMISTRY-Summary Quotients) did not have benthic community analysis performed, it is likely these stations will demonstrate degraded benthic communities, when analyzed. In consideration of this concern, all stations with elevated chemistry, based on ERM summary quotients above 0.85, were assigned a moderate priority ranking.

In situations where high summary quotient values were not found, but where any single chemical concentration exceeded four times (4x) its associated ERM or 5.9 times (5.9x) its associated PEL, the station was also considered to exhibit elevated chemistry. The 4x and 5.9x cutoffs were not statistically determined using the 90% confidence interval as they were with the summary quotients. Values for individual chemical quotients were not normally distributed and transformations did not improve distributions, so statistical determination of confidence limits was not appropriate. Instead, a qualitative examination of the data set indicated that only in the top 10th percentile of chemical measurements do values exceed four times their respective ERM or 5.9 times their respective PEL (Tables 20 and 22- CHEMISTRY-Individual Chemicals). These cutoffs were used to help identify stations where any single chemical was extremely elevated. Stations with elevated individual chemical quotients and evidence of benthic community degradation were assigned a moderate ranking. Stations which exhibited elevated chemistry, but showed no biological effects, were assigned a low priority.

Stations which satisfied all three of the criteria were considered a triad hit and are given the highest priority ranking. These stations demonstrated toxicity in the bioassay tests, benthic community degradation and elevated chemistry. Four stations (representing three sites) fell in this category: the Seventh Street Channel (90009-leg 23 and 93228), 12 Swartz

TABLE 22

FUTURE INVESTIGATION PRIORITY LIST FOR THE SAN DIEGO BAY REGION

Stations Without Synoptic Chemical, Toxicological and Benthic Community Analyses

STANUM	STATION	IDORG	LEG	H2S	NH3	% AMPHI. SURVIVAL	>4X ERM OR >5.9X PEL	ERMQ	PELQ	BENTHICS	COMMENTS	PRIORITY
	CHEMISTRY-Summary Quotients											
90020.0	G DE LAPPE	169	12	not analyzed	0.020	49.00		**0.964**	1.255	not analyzed	ELEVATED CHEM	MODERATE
90020.0	G DE LAPPE-REP 1	1104	27	0.0006	0.086	65.00		**1.051**	**1.411**	not analyzed	ELEVATED CHEM	MODERATE
90020.0	G DE LAPPE-REP 2	1105	27	0.0007	0.087	59.00		**1.043**	**1.401**	not analyzed	ELEVATED CHEM	MODERATE
90020.0	G DE LAPPE-REP 3	1106	27	0.0009	0.049	57.00		**0.947**	**1.293**	not analyzed	ELEVATED CHEM	MODERATE
90030.0	BF SCHROEDER SITE F-REP 1	1144	28	0.0012	0.192	70.00		**0.948**	**1.419**	not analyzed	ELEVATED CHEM	MODERATE
90030.0	BF SCHROEDER SITE F-REP 2	1145	28	0.0025	**0.616**	76.00	PAHs	**1.000**	**1.537**	not analyzed	ELEVATED CHEM	MODERATE
90030.0	BF SCHROEDER SITE F-REP 3	1146	28	0.0013	0.017	68.00		**1.007**	**1.438**	not analyzed	ELEVATED CHEM	MODERATE
93178.0	NAVAL SHIPYARDS O2 (x1)-REP 1	1119	27	0.0022	0.185	61.00		**0.934**	**1.294**	not analyzed	ELEVATED CHEM	MODERATE
93178.0	NAVAL SHIPYARDS O2 (x1)-REP 2	1120	27	nd	0.145	66.00	PCBs	**1.170**	**1.618**	not analyzed	ELEVATED CHEM	MODERATE
93178.0	NAVAL SHIPYARDS O2 (x1)-REP 3	1121	27	0.0007	0.168	67.00	PCBs	**1.269**	**1.651**	not analyzed	ELEVATED CHEM	MODERATE
90022.0	P SWARTZ-REP 1	1107	27	0.0003	0.061	58.00	PAHs	**1.042**	**1.549**	not analyzed	ELEVATED CHEM	MODERATE
90022.0	P SWARTZ-REP 2	1108	27	0.0008	0.073	61.00	PAHs	**1.109**	**1.770**	not analyzed	ELEVATED CHEM	MODERATE
90022.0	P SWARTZ-REP 3	1109	27	0.0008	0.038	54.00	PAHs	**1.107**	**1.724**	not analyzed	ELEVATED CHEM	MODERATE
93179.0	NAVAL SHIPYARDS O3 (x1)-REP 2	1123	27	nd	0.049	51.00		**1.071**	**1.462**	not analyzed	ELEVATED CHEM	MODERATE
93179.0	NAVAL SHIPYARDS O3 (x1)-REP 3	1124	27	nd	0.115	78.00	Antimony	**1.330**	**1.658**	not analyzed	ELEVATED CHEM	MODERATE
93184.0	NAVAL SHIPYARDS O11 (x1)	802	19	not analyzed	0.070	53.00	DDT	**1.226**	**1.774**	not analyzed	ELEVATED CHEM	MODERATE
90017.0	C DELAPPE	166	6	not analyzed	**0.840**	64.00	PAHs	**1.183**	**1.943**	not analyzed	ELEVATED CHEM	MODERATE
93181.0	NAVAL SHIPYARDS O6 (x1)-REP 3	1112	27	0.003	0.037	65.00		**0.904**	**1.362**	not analyzed	ELEVATED CHEM	MODERATE
	CHEMISTRY-Individual Chemicals											
93162.0	SUB BASE C3 (x1)	775	18	not analyzed	**0.585**	53.00	PAHs	0.347	0.596	not analyzed	ELEVATED CHEM	LOW
90037.0	STORMDRAIN EM(GRAPE ST.)-REP 3	1161	29	0.0012	0.290	85.00	Chlordane	0.656	0.934	not analyzed	ELEVATED CHEM	LOW
93141.0	COMMERCIAL BASIN F3 (x1)-REP 3	1170	29	0.0004	0.057	70.00	Mercury	0.650	0.905	not analyzed	ELEVATED CHEM	LOW
93116.0	SAN DIEGO RIVER B1 (x4)	711	15	**0.0893**	0.137	88.00	Chlordane	0.659	0.913	not analyzed	ELEVATED CHEM, SITE DEGRADED IN LEG 22	MODERATE
93120.0	TIJUANA R. ESTUARY HH2 (x1)	715	15	0.0002	0.087	85.00	DDE	0.321	0.358	not analyzed	ELEVATED CHEM	LOW
93121.0	TIJUANA R. ESTUARY HH2 (x5)	716	15	0.0016	0.010	85.00	DDE	0.287	0.314	not analyzed	ELEVATED CHEM	LOW
93174.0	TIJUANA R. EST. HH3 (x2)-REP 3	1152	28	0.0044	0.084	80.00	DDE	0.325	0.395	not analyzed	ELEVATED CHEM	LOW
93177.0	NAVAL SHIPYARDS O1 (x1)	795	19	not analyzed	0.023	50.00	PAHs	0.694	1.204	not analyzed	ELEVATED CHEM	LOW

Downtown Anchorage (90002) and Naval Base/Shipyards O4 (93210). Three stations were given a high priority ranking although not all conditions of the triad were met (Seventh Street Channel (90009-leg 7) and Naval Shipyards O3 (93179- legs 19 & 27)). These stations demonstrated repeated toxicity and elevated chemistry but no benthic analyses were performed. However, benthic data for stations analyzed in the same proximity, or later sampling of the station, led to the concern that these sites would have been found degraded, if analyzed. In addition, chemical summary quotients at these three stations were at levels which suggest probable benthic community degradation, as discussed earlier. These concerns warranted upgrading these three stations from a moderate priority to a high priority. Forty three stations were given moderate priorities and 57 were given low priorities, based on the methods of prioritization previously discussed. Prioritized stations are mapped in Figure 27(a-d).

Stations were prioritized to assist SWRCB and RWQCB staff in meeting sediment quality management objectives for San Diego Bay. These recommendations were based on scientific evaluation of data collected between 1992 and 1994. They are intended to focus future efforts toward scientifically and economically responsible characterization of locations which have a high probability of causing adverse effects to aquatic life. This report should be evaluated in conjunction with all available information and additional research when management and policy decisions are made by SWRCB and RWQCB staff.

Possible Sources of Pollutants at Prioritized Stations

A brief description is given, where additional information was available, of factors which may have contributed to elevated chemical levels, toxicity, or benthic community degradation at the prioritized stations. Descriptions are given in order of geographic distribution, proceeding from north (Mission Bay) to south (Tijuana River Estuary).

In Mission Bay only one location was given the moderate priority ranking (station 93116). This station was located in the San Diego River flood control channel and demonstrated high total chlordane concentrations (36.1 ppb). Chlordane is not expected to undergo significant hydrolysis, oxidation, or direct photolysis in water, thus it may persist in soils for extended periods of time (Howard, 1991). Cohen *et al.* (1990) conducted a study on chlordane in soil samples near golf courses and found unusually high concentrations of chlordane (4.75-4310 ppb). Station 93116 is located directly down river from a golf course, therefore, runoff from this facility could be a chlordane source. Station 93107, in the mouth of Rose Inlet (northern Mission Bay), received a moderate priority listing, based on high chlordane concentrations. Its location is also near a golf course.

One site in North San Diego Bay (Point Loma area) received a moderate priority recommendation; stations 90028 (Submarine Base). This station had degraded benthic communities, high

Figure 27a
Future Investigation Priority List
North San Diego Bay

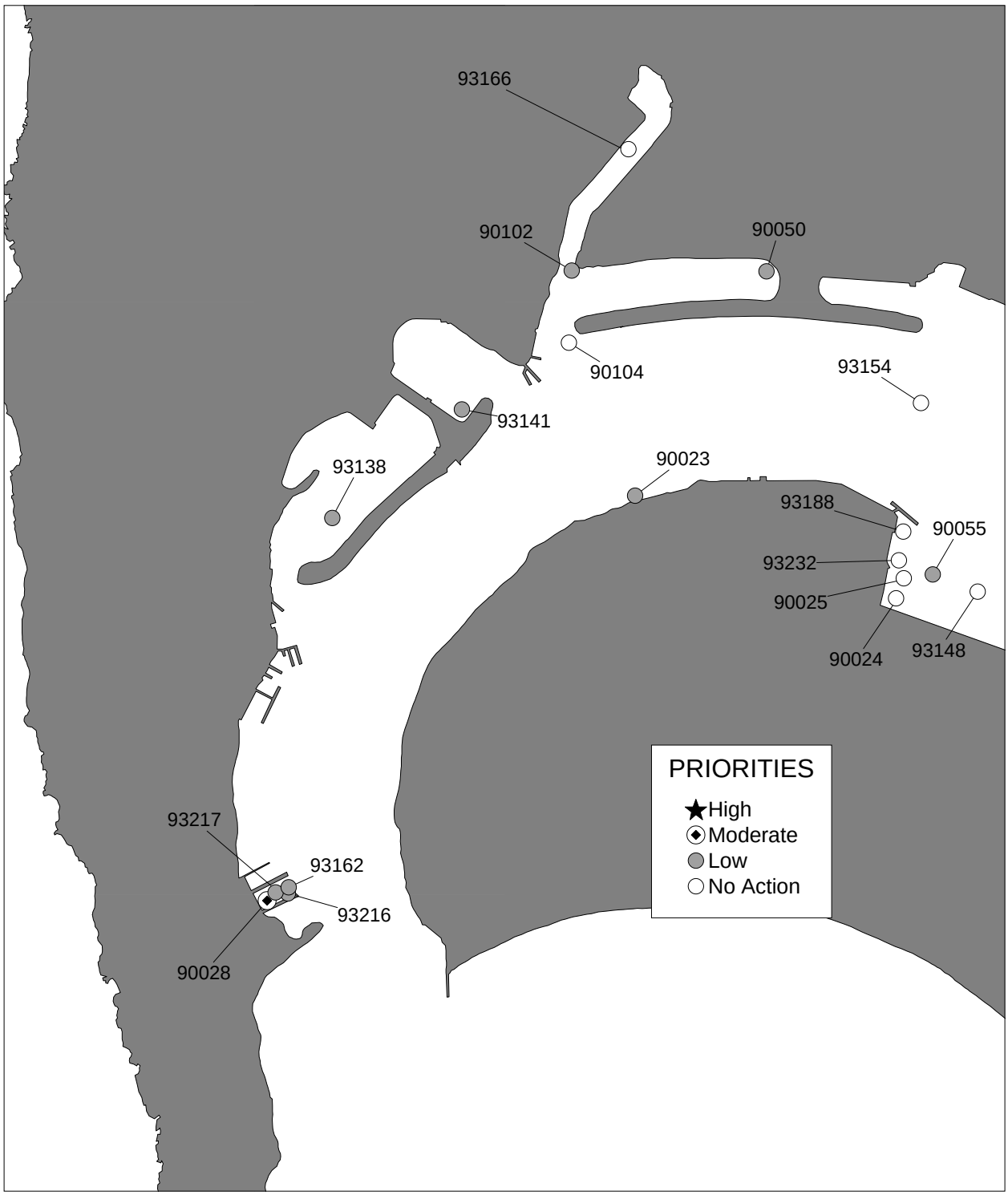


Figure 27b
Future Investigation Priority List
Mid San Diego Bay

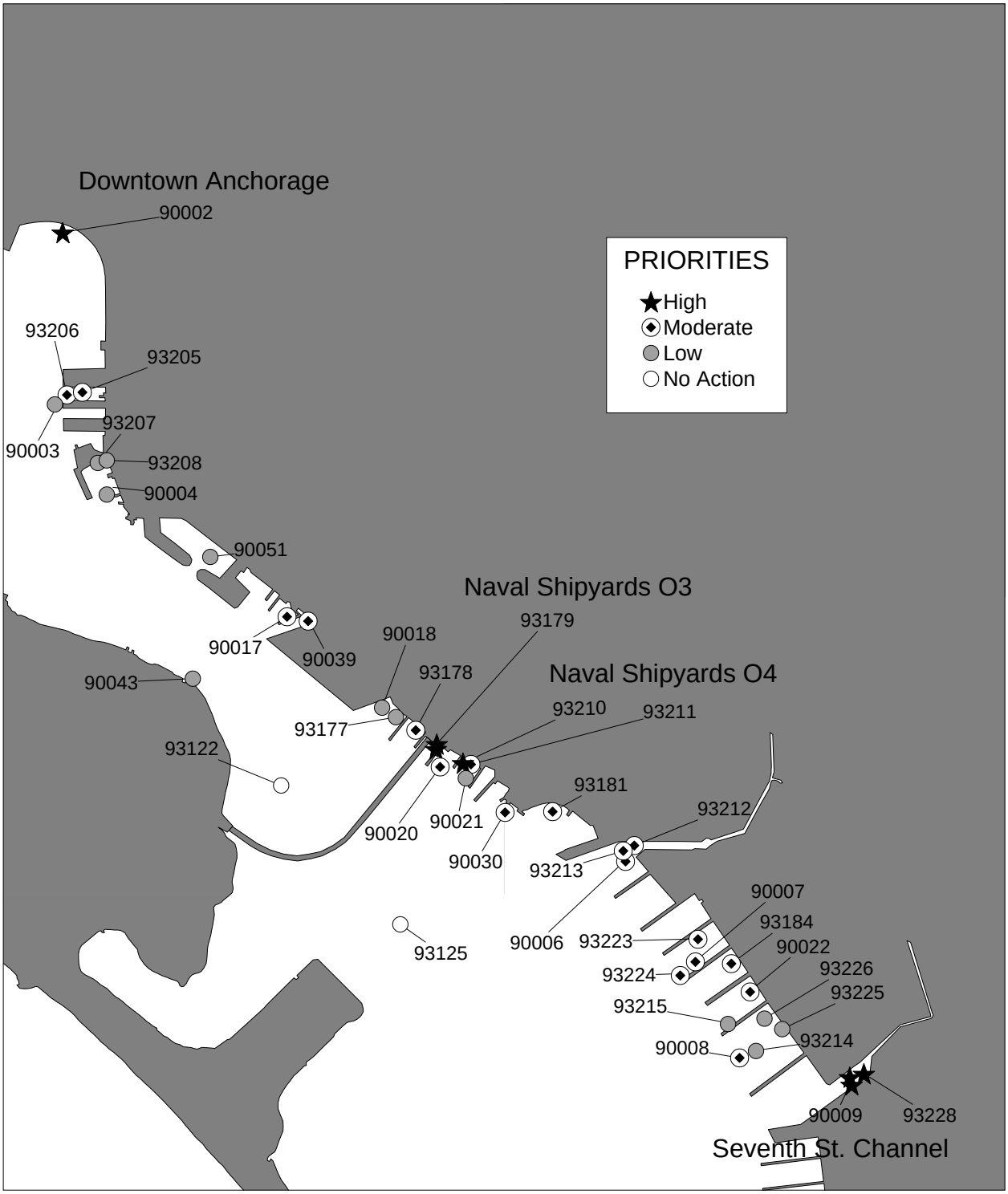


Figure 27c
Future Investigation Priority List
South San Diego Bay

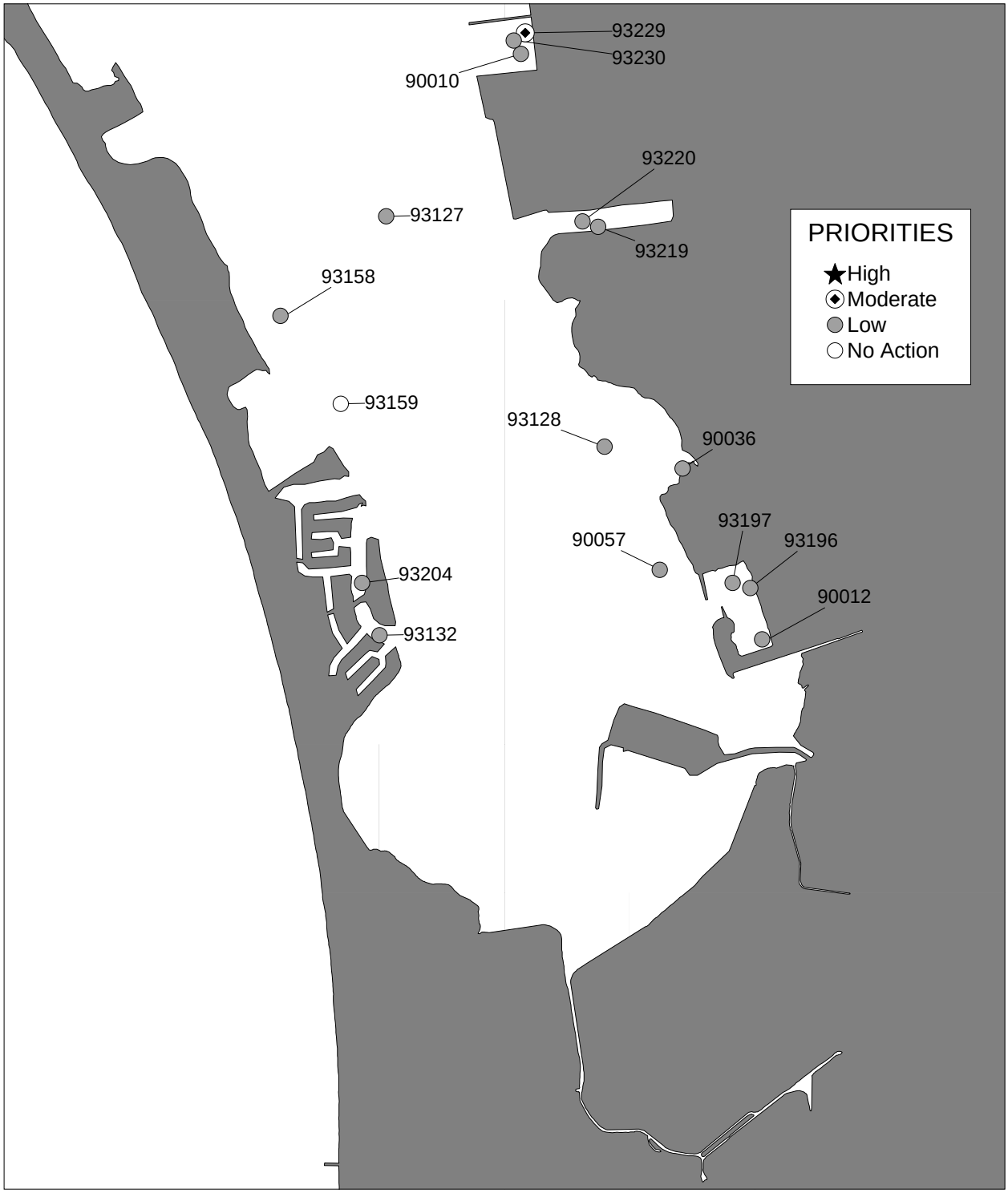
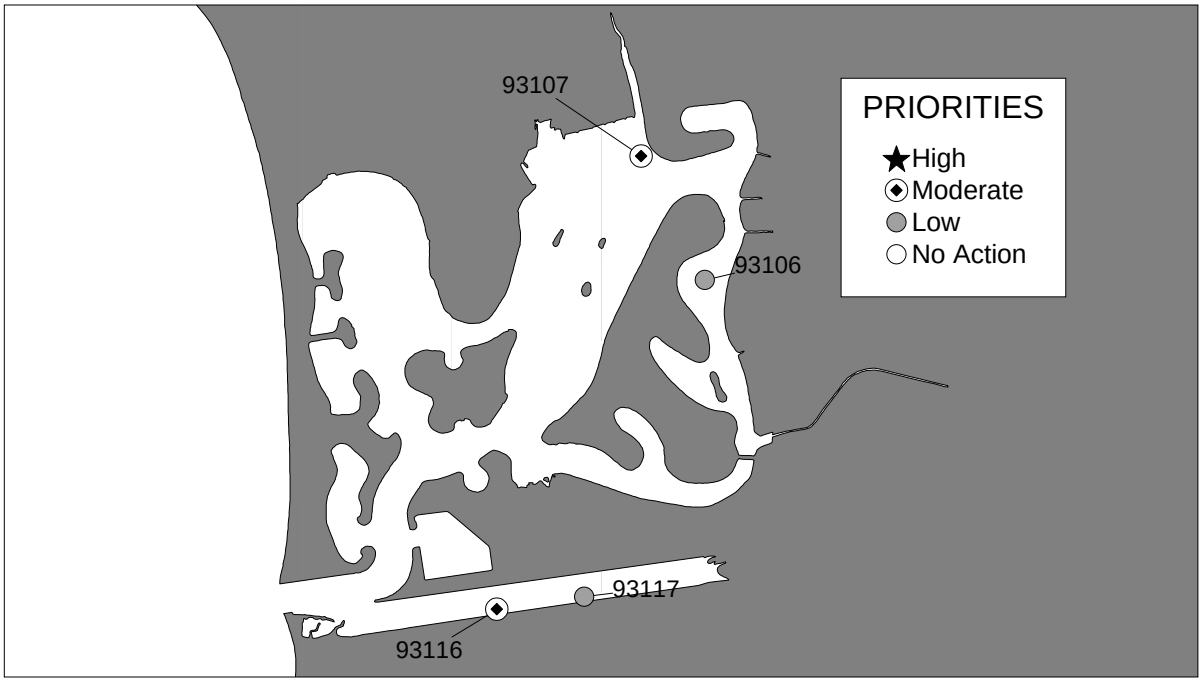


Figure 27d
Future Investigation Priority List
Mission Bay and San Diego River Estuary



Tijuana River Estuary

concentrations of low and high molecular weight PAHs, and moderate levels of metals. Historically the Naval Complex at Point Loma has received plating waste, sewage, and sludge containing high concentrations of metals and chlorinated hydrocarbons (Johnston *et al.*, 1989). Although it is difficult to identify the source of high concentrations of PAHs at these stations, Lung (1983) suggests ground water gradients promote groundwater flow towards San Diego Bay, thus potentially allowing PAHs in the nearby soil to migrate to the Bay. A number sites investigated by the Navy (Eakes and Smith, 1986), which were previously used for waste oil and drum disposal, are located onshore adjacent to and immediately north of stations 93216, 93217 and 90028. Migration of pollutants from these onshore sites is likely. Minor spills during fueling operations at the submarine base are also possible.

Station 90002 (Downtown Anchorage), located in the northern end of mid San Diego Bay, was one of the stations which received a high priority recommendation. High concentrations of metals and chlordane were present, as well as a degraded benthic community. This station also had a low survival for *Rhepoxynius* in solid phase toxicity tests. Perhaps the most obvious explanation for these data would be the presence of a large storm drain and numerous smaller storm drains, which empty into the Bay near this station. These storm drains drain parking lots, light industrial and commercial areas (Conway and Gilb, 1990). Another possible source for observed toxicity and chemistry is runoff from nearby San Diego International Airport. Results from the State Mussel Watch Program 1987-1993 indicate elevated levels of both metals and pesticides in mussel tissue and sediments in this area. Elevated levels of metals could have originated from anti-fouling paints on private boats anchored near the station (90002). The area around this station becomes a modified eddy during ebb tide and may serve to recirculate pollutants, creating a pollutant sink and preventing chemicals from being flushed out of the area (Peeling, 1974).

Located just south of station 90002, stations 93205 and 93206 (Downtown Piers) were given moderate priority ratings based on high chlordane and PAHs concentrations, and degraded benthic communities. Located between the B street pier and the Broadway pier, elevated levels of pollutants can most likely be attributed to sources similar to those described above. Commercial shipping is likely an additional contributor to the observed PAH signal in this area.

Two stations, 90017 and 90039 (located immediately north of the 10th avenue marine terminal), were assigned moderate priority rankings based on high concentrations of chlordane, metals, and PAHs at each of these stations. Campbell Industries operate five ship repair piers and four dry-docking facilities in this area. Sandblasting, painting, and other ship repair activities are probably the cause of the elevated levels of copper, zinc and mercury. High concentrations of metals have historically been detected at this site (Barry, 1972). The 10th avenue Marine

Terminal berths 1 and 2 are also located in this area (station 90039). Ships are loaded and unloaded at this site and supplied with fuel from four steel storage tanks located near the berths. Increased levels of PAHs and metals detected in this area may be related to the cargo transfer facility.

In addition to the ship repair facilities and cargo transfer areas, there is a large storm drain system which is directly south of the 10th and Imperial Trolley station. The system drains approximately eleven square kilometers of residential (including Balboa Park) and industrial areas before emptying into the Bay. The elevated levels of chlordane and PAHs at both of the sites could have additional sources from within this drainage system.

Immediately south of the Coronado Bridge was station 93179 (Naval Shipyards-O3) which was designated as a high priority site for future investigations. To the north and south of this site are numerous stations assigned a moderate prioritization. The predominant activity in this area is ship building and repair (NASSCO, Continental Maritime, Southwest Marine), thus indicating the probable source of high levels of metals, PCBs and PAHs found at stations sampled in this area. A stormdrain, which drains an industrial area and empties into the Bay immediately adjacent to the bridge, is the likely chlordane source to the area. Runoff from the bridge itself could also be viewed as a potential source of PAHs and metals in the Bay. The California State Mussel Watch Program (1995) has sampled extensively in this area of San Diego Bay and found chemistry values for mussels and sediment to be comparable to the current study. This area has also been extensively sampled in other studies resulting in similar conclusions (de Lappe, 1989; Martin, 1985; Anderson, 1989). Toxicity, chemical pollution and benthic community degradation are extensive in this area and warrant further site characterizations.

Stations 93212, 93213, and 90006 (Naval Shipyards-O7) were located near the 28th Street pier and were each given a moderate priority ranking. Chollas Creek empties into the Bay near this site, carrying with it runoff from a large urban area. This creek is believed to carry high concentrations of PAHs into the Bay (McCain *et al.*, 1992) and is the likely source of high chlordane levels at the site.

Numerous low, moderate and high priority sites were located in the Naval Station between the 28th Street pier and 7th Street channel. This area demonstrated toxicity, high metal and chemistry concentrations and degraded benthic communities. The area is predominantly used for ship repair, outfitting, and conversion. Sand blasting, painting, and the changing of zinc electrolysis plates are some of the specific activities conducted in this area and are likely the main sources of metals found in the sediments.

Station 93227 was located in the 7th Street Channel at the southern end of the San Diego Naval Station. This site was given

the high priority ranking based on high metal, chlordane and PAH concentrations, as well as toxicity and degraded benthic communities. Repeated sampling of this site resulted in similar findings. Paleta Creek runs directly into 7th Street channel with numerous drains located in the immediate area emptying into the creek and bay. Also, a large stormdrain is present which drains a residential area east of Interstate 5 and the Naval station adjacent to the channel.

The Navy has used 7th Street channel and the surrounding area for a variety of activities. Excess materials (solid waste, ships stores, and waste hydraulic fluids) from decommissioned ships were disposed of in the ship repair basins. Overflow from salvage yards, lube and hydraulic oil wastes, and paint sludge from nearby Naval repair facilities were often taken to the area's wet docks for disposal. In the late 1970's trucks and heavy equipment returning form Vietnam were routinely decontaminated by spraying with diesel fuel and dunking (by crane) into Paleta Creek. It is estimated that approximately 75,000 to 360,000 gallons of petroleum based material were disposed of at this site during its period of operation (1945-1973).

The 7th Street channel is located near a Navy salvage yard which has stormdrains emptying directly into the channel. In 1976, soil samples retrieved from the area contained PCB concentrations high enough to result in the upper eight inches of soil being removed as contaminated waste and the entire area paved. Although the Navy has attempted to deal with this historic pollution in the area, further investigations were requested by a Naval initial assessment team in 1986 (Eakes and Smith, 1986). Furthermore, the California State Mussel Watch program has stations located in the area and concluded 7th Street channel had some of the highest chemical concentrations in San Diego Bay (State Mussel Watch Program, 1995).

The Marine terminal site (stations 90010, 93230 and 93229) demonstrated elevated copper and PAH levels and a degraded benthic community. Moderate and low priorities were assigned to these stations even though a portion of this area is currently undergoing cleanup activities. Due to the large amount ore spillage at the PACO copper loading facility, this area should continue to be monitored after cleanup activities are completed.

The southern portion of San Diego Bay, from 7th Street channel to the Otay River, did not receive any moderate or high priority rankings. Although this result could give the impression south San Diego Bay is in not polluted, it is important to note some stations still demonstrated high metals concentrations. The Sweetwater channel area (station 93220), and other sites in the South San Diego Bay had high concentrations of copper, most likely reflecting the input from the copper ore loading facility (Martin, 1985). Three stations in the Chula Vista area and one in Coronado Cays received low priority rankings due to elevated levels of metals and degraded benthic communities. Each of these stations were located within marinas where numerous private boats

are berthed. Increased levels of metals detected in this area are probably from anti-fouling paint scrapings or zinc electrolysis blocks used on virtually all boats. Few studies have concentrated sampling in the South San Diego Bay, presumably due to reduced shipping activity and population.

Stations from the Tijuana River Estuary demonstrated elevated concentrations of DDT and DDE, as well as toxicity to amphipods. This resulted in a number of stations receiving moderate and low prioritizations. The presumed sources of this pesticide were wastewater discharges from Mexico, into the Tijuana River (California State Coastal Conservancy, 1989).

Comparison of Pollution with Other Water Bodies

Numerous studies comparing San Diego Bay with other bays and harbors have been conducted (NOAA, 1991; Grovenhoug *et al.*, 1987; Goldberg *et al.*, 1978). In one such study, Robertson (1989) analyzed sediments for a number of organic pollutants at approximately 200 sites around the coasts of the United States. Results ranked San Diego Bay seventh highest in the country for total concentrations of PCBs. Interestingly, San Diego Bay did not rank high in comparison to the rest of the country for any other organic pollutant, although results from the current study clearly showed elevated concentrations (relative to ERMs and PELs) of total PAHs, chlordane, and certain trace metals throughout the Bay.

In a similar study, Johnston (1990) evaluated 367 waste disposal sites at 58 Navy and Marine Corps bases located throughout the country. Each of the bases, or areas of activity, were located in the coastal zone and were reviewed to characterize the pollutants, disposal methods, and potential impact to the surrounding aquatic environment. Four sites were chosen in San Diego Bay: Naval Station San Diego (located immediately south of the seventh street channel), Naval Amphibious Base (near Glorietta Bay), Naval Training Center, and Naval Complex Point Loma. Although these sites were not ranked or compared with sites in other parts of the country, the types of contamination listed were somewhat similar for each of the sites described. Paint, oil, and solvent contamination was reported at all of the sites in addition to some site specific forms of contamination(*i.e.* sandblasting grit disposal area at the Naval Amphibious Base and drum disposal area at the Naval Complex Point Loma).

San Diego Bay has also been compared to other bodies of water on a regional scale. In a SCCWRP project funded by the State Board, Anderson and Gossett (1987) analyzed PAHs in sediments collected at stations between Santa Monica Bay and San Diego Bay and found the Seventh Street (Paleta Creek) and Chollas Creek stations to contain the highest levels of these hydrocarbons. In a follow-up State Board/SCCWRP study Anderson *et al.* (1988) compared ten coastal sites in southern California for concentrations of trace metals, PAHs, chlorinated hydrocarbons and toxicity. Samples from San Diego Bay were shown to have the highest concentrations of

metals, PAHs, and hydrocarbons of all stations sampled, and were the most toxic in two out of three toxicity tests used. Anderson *et al.* (1988) identified the Seventh Street Channel station as the most polluted area in the San Diego Bay Region. This conclusion is corroborated by the current study which also found sampling stations in the Seventh Street Channel to be the most polluted and most toxic stations in the region. Flegal and Sanudo-Wilhelmy (1993) showed total dissolved trace metal (Ag, Cd, Co, Cu, Ni, and Pb) concentrations in San Diego Bay are comparable to levels of trace element pollution in south San Francisco Bay. Specifically, copper was found in elevated concentrations in both bays. The current study found copper to be the predominant trace element pollutant in San Diego Bay. Flegal and Sanudo-Wilhelmy concluded that unlike south San Francisco Bay, elevated trace metal concentrations in San Diego Bay could not be directly linked to point-source inputs, because all wastewater discharges to San Diego Bay were terminated in 1964. Copper based anti-fouling paints and urban runoff are currently the most likely sources of copper. Elevated concentrations of copper in San Diego Bay have also been reported in other studies (Zirino *et al.*, 1978).

It is also important to analyze available site specific data within San Diego Bay from previous studies. In the current study, commercial and naval shipyards located near the Coronado Bridge consistently demonstrated high concentrations of pollutants, a high incidence of toxicity, and benthic community degradation. Shipbuilding activity, in addition to storm drains and creeks, appear to be the primary sources of organic and trace metal pollutants in these areas (Conway and Gilb, 1990). Secondary sources of contamination may include runoff from the Coronado Bridge (San Diego Interagency Water Quality Panel, 1989) and polluted fill in the area (Peter Michael, San Diego Regional Water Quality Control Board, personal communication). This is supported by the conclusions of McCain (1992) who found several major sources of pollutants in the central portion of San Diego Bay.

Specific organic pollutants such as PCBs have been historically identified in certain parts of the bay. In one of the earliest studies of PCBs in San Diego Bay, Young and Heesen (1977) identified PCBs in mussel tissues. The highest measured concentrations occurred in Commercial Basin (Shelter Island). Subsequent studies have also shown elevated levels of PCBs in the Shelter Island area, as well as near Harbor Island and numerous other spots throughout the Bay (Stephenson *et al.*, 1980; Martin, 1985). Similar results were obtained from sediment samples in the current study in which high concentrations of PCBs were reported from areas near the Coronado Bridge, west Commercial Basin and East Basin near Harbor Island. The Regional Water Quality Control Board has identified a 60 inch storm drain as the main source of PCBs into the East Basin site. Cleanup and Abatement Orders, regarding PCBs, have been issued to boatyards in and around Shelter Island and Harbor Island (San Diego Interagency Water Quality Panel, 1994).

Tributyltin (TBT), an organic based biocide, was widely used as an antifoulant on ships and small craft until 1988 (Richard and Lillebo, 1988). Although TBT is highly efficient at killing fouling organisms it is also acutely toxic to non-target organisms, making it a continuing concern in the San Diego Bay Region. Toxic effects have been observed in concentrations as low as 1 ng/L (Henderson, 1988). Long term monitoring of U.S. harbors indicates that among naval bases, San Diego has relatively low concentrations of TBT (Kram *et al.*, 1989; Seligman *et al.*, 1990). These studies focused on comparisons between U.S. Naval facilities (i.e. Pearl harbor, Norfolk harbor) where use of TBT anti-fouling paints is not restricted on vessels over 25 meters in length (Organotin Antifouling Paint Control Act, 1988). Because San Diego Bay is a multi-use port, where smaller non-naval vessels must conform to the 1988 legislation, TBT values are expectedly lower than harbors which solely contain large naval vessels. In the current study, TBT values were highest in naval and commercial basin areas, similar to the findings of Seligman *et al.* (1990). Although both studies found elevated levels of TBT in commercial and naval sites, data from the current study indicates an overall decline in TBT sediment concentrations at these locations. This is most likely a reflection of restrictive legislation on TBT use in antifouling paints. Given the historical use of antifouling paints in San Diego Bay, continued monitoring is recommended, although results from the current study were encouraging.

Limitations

The two step sampling design of this study relied on an initial "screening phase" to give a broad assessment of toxicity in the San Diego Bay Region. Subsequent toxicity test, chemical analysis and benthic community analysis were performed only on selected stations ($\approx$ 40% of the screened stations) which demonstrated toxicity during the screening phase, or were considered candidates as reference stations. The remaining stations, from the screening phase, did not receive additional testing or analysis. Therefore, statistical analyses, comparisons to chemical specific screening values, identification of undegraded and degraded habitats, and prioritized rankings could not be performed on all stations sampled. Currently these stations fall under a no action recommendation, but it should be understood that for these stations a weight-of-evidence evaluation was not performed, due to the absence of chemical and/or benthic community data.

In determination of toxicity for the reference envelope approach, values must be chosen for alpha and the percentile (p) to calculate the edge of the reference envelope (L) using the following equation:

$$L = X_r - [\, g_{a,p,n} * S_r \,]$$

The values of alpha and p are chosen to express the degree of certainty desired when classifying a sample as toxic. In this study values of alpha=.05 and p=1 were used to distinguish the most toxic samples which have a 95% certainty of being in the most toxic 1% (Figure 4). This calculation resulted in a determination of toxicity for the *Rhepoxynius* test when samples had a mean survival of less than 48%. If the value of p was chosen to equal 10% (*i.e.*, a 95% certainty of being in the most toxic 10%) the determination of toxicity (edge of the reference envelope) would have been at 63% survival. Obviously, a choice of p=10% would broaden the range of samples which would be classified as "toxic". It must be recognized the 48% level used in this study was chosen as a conservative guideline to identify only the most toxic stations for setting priorities for future work. The 48% survival cutoff used in this study should be recognized as a statistical determination which may or may not reflect the certainty desired by SWRCB and RWQCB staff for sediment quality management purposes.

There is a necessary caution to the ecological applicability of data collected from studies such as reported here. Although measures of toxicity and chemical concentration are used extensively in this study, they can only be used as indicators of possible adverse effects to indigenous communities. Benthic community assessment is the only tool used in this study which can demonstrate actual effects to resident biological communities. In combination, these three measures provide a strong weight of evidence for the conditions found at a particular sampling location. However, it is recommended these lines of evidence be supported with an ecological risk assessment during subsequent investigations of stations of concern.

CONCLUSIONS

The major conclusions of this study were:

1. Two sets of sediment quality guidelines were useful in demonstrating chemical pollution: The ERL/ERM thresholds developed by NOAA (Long and Morgan, 1990; Long *et al.*, 1995) and the TEL/PEL thresholds used in Florida (MacDonald, 1993; MacDonald, 1994). Copper, mercury, zinc, total chlordane, total PCBs, and PAHs were most often found to exceed critical ERM or PEL values. These were considered the major chemicals or chemical groups of concern in the San Diego Bay Region. ERM and PEL summary quotients were developed as chemical indices for evaluating pollution of sediments with multiple chemicals. An ERM summary quotient >0.85 or a PEL summary quotient >1.29 was indicative of sites where multiple chemicals were significantly elevated. Stations with any chemical concentration >4 times its respective ERM or >5.9 times its respective PEL were considered to exhibit elevated chemistry.

2. The identification of degraded and undegraded habitat was determined by macrobenthic community structure, using a cumulative, weight-of-evidence approach. Analyses of the 75 stations sampled for benthic community structure identified 23 undegraded stations, 43 degraded and 9 transitional stations. All sampled stations with an ERM quotient>0.85 were found to have degraded communities. All sampled stations with P450 responses above 60 μg/g BaPEq. were found to have degraded benthic communities.

3. Exceedances of toxicity thresholds were determined using two approaches: the reference envelope approach and laboratory control comparison approach. The reference envelope approach was the more conservative of the two, indicating toxicity for the *Rhepoxynius* (amphipod) sediment test was significant when survival was less than 48%, in samples tested. No reference envelope was determined for the *Strongylocentrotus* (urchin) fertilization or development tests. High variability in pore water data from reference stations produced a lower confidence boundary for the reference envelope below 0% survival. This indicates no significant distinction in toxicity could be made between reference stations and other stations for these pore water tests.

4. Using the EMAP definition of toxicity, 56% of the total area sampled in the San Diego Bay Region was toxic to *Rhepoxynius*. For *Strongylocentrotus* development test, percent of total area toxic was 29%, 54%, and 72% respectively for 25%, 50%, and undiluted pore water concentrations. Samples representing 36%, 27%, or 14% of the study area were toxic to both *Rhepoxynius* in solid phase sediment and to *Strongylocentrotus* larvae in 100%, 50%, or 25% pore water, respectively. Spatial extent of toxicity was not determined using the reference envelope definition of toxicity.

5. Linear regression analyses failed to reveal strong correlations between amphipod survival and chemical concentration. It is suspected instead of a linear response to chemical pollutants, most organisms are tolerant of pollutants until a threshold is exceeded. Comparisons to established sediment quality guideline thresholds demonstrate an increased incidence of toxicity for San Diego Bay Region samples with chemical concentrations exceeding the ERM or PEL values. It is further suspected toxicity in urban bays is caused by exposure to complex mixtures of chemicals. Comparisons to ERM summary quotients (multiple chemical indicators) demonstrate that the highest incidence of toxicity (>78%) is found in samples with elevated ERM summary quotients (>0.85).

Statistical analyses of the P450 Reporter Gene System responses versus the PAHs in sediment extracts demonstrated that this biological response indicator was significantly correlated (r^2 = 0.86) with sediment PAH (total and high molecular weight) concentrations.

6. Stations requiring further investigation were prioritized based on combined evidence from toxicity, chemical and benthic community data. Prioritizations were developed to help direct future investigations by State and Regional Water Board staff at these stations. Each station receiving a high, moderate, or low priority ranking meets one or more of the criteria under evaluation for determining hot spot status in the Bay Protection and Toxic Cleanup Program. Those meeting all criteria were given the highest priority for further action.

Seven stations (representing four sites) were given a high priority ranking, 43 stations were given a moderate priority ranking, and 57 stations were given a low priority ranking. The seven stations receiving the high priority ranking were in the Seventh Street channel area, two naval shipyard areas near the Coronado Bridge, and the Downtown Anchorage area west of the airport. The majority of stations given moderate rankings were associated with commercial areas and naval shipyard areas in the vicinity of the Coronado Bridge. Low priority stations were interspersed throughout the San Diego Bay Region.

7. A review of historical data supports the conclusions of the current research. Possible sources for pollution at prioritized stations are given. Recommendations are made for complementary investigations which could provide additional evidence for further characterizing stations of concern.

RECOMMENDATIONS

Given the supporting evidence of previous studies, the patterns of chemical pollution and bioeffects observed during this assessment of the San Diego Bay Region are convincing. There are additional avenues of investigation though which would complement the results of this study. The results also should be confirmed with further studies before any adverse ecological impacts can be conclusively demonstrated.

Due to the large number of elevated chemicals at the majority of the prioritized sampling stations, toxic biological responses can only be associated with overall chemical pollution, rather than a particular chemical. However, stations on the priority list, where the number of ERM or PEL exceedances is low and the exceedance for a particular chemical is high, are excellent candidates for toxicity identification evaluations (TIE). The ability to distinguish between causative factors of toxicity is enhanced when multiple chemicals are not involved. Stations Naval Base O7(x1), 12 Swartz (Downtown Anchorage), and the San Diego River, where high chlordane concentrations are found, are well suited for TIE manipulations which would attempt to test this organic pesticide as the causative toxicity agent. The Naval Base/Shipyard O10(x6) station, which only demonstrates ERM or PEL exceedances for trace metals, is well suited for manipulations which could remove metal toxicity (*e.g.*, EDTA additions).

Several chemicals of concern identified in the San Diego Bay region have been shown to bioconcentrate and biomagnify in the tissues of marine species. A tissue contamination study for lipophilic compounds such as PCBs, chlordane, and possibly methylmercury is recommended to address human health concerns due to consumption of impacted resident species. This line of investigation seems necessary considering tissue contamination is the only BPTCP criterion not investigated during this study.

Although specific stations are identified as having a high probability of causing adverse effects, no attempt can be made to define the boundaries of the impacted area. Sampling specifically designed to quantify areal extent of an impacted area must be addressed during intensive site characterizations.

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APPENDIX A

DATA BASE DESCRIPTION

for the

SWRCB/NOAA COOPERATIVE PROJECT
SAN DIEGO BAY

A Report prepared for the

California State Water Resources Control Board
Bays and Estuaries Unit
Bay Protection and Toxic Cleanup Program

by the

California Department of Fish and Game
Marine Pollution Studies Laboratories
7711 Sandholdt Road
Moss Landing, CA 95039

September, 1996

I. OVERVIEW OF THE BAY PROTECTION PROGRAM

The California State Water Resources Control Board (SWRCB) has contracted the California Department of Fish and Game (CDFG) to coordinate the scientific aspects of the Bay Protection and Toxic Cleanup Program (BPTCP), a SWRCB program mandated by the California Legislature. The BPTCP is a comprehensive, long-term effort to regulate toxic pollutants in California's enclosed bays and estuaries. The program consists of both short-term and long-term activities. The short-term activities include the identification and priority ranking of toxic hot spots, development and implementation of regional monitoring programs designed to identify toxic hot spots, development of narrative sediment quality objectives, development and implementation of cleanup plans, revision of waste discharge requirements as needed to alleviate impacts of toxic pollutants, and development of a comprehensive database containing information pertinent to describing and managing toxic hot spots. The long-term activities include development of numeric sediment quality objectives; development and implementation of strategies to prevent the formation of new toxic hot spots and to reduce the severity of effects from existing toxic hot spots; revision of water quality control plans, cleanup plans, and monitoring programs; and maintenance of the comprehensive database.

Actual field and laboratory work is performed under contract by the California Department of Fish and Game (CDFG). The CDFG subcontracts the toxicity testing to Dr. Ron Tjeerdema at the University of California at Santa Cruz (UCSC) and the laboratory testing is performed at the CDFG toxicity testing laboratory at Granite Canyon, south of Carmel. The CDFG contracts the majority of the sample collection activities to Dr. John Oliver of San Jose State University at the Moss Landing Marine Laboratories (MLML) in Moss Landing. Dr. Oliver also is subcontracted to perform the TOC and grain size analyses, as well as to perform the benthic community analyses. CDFG personnel perform the trace metals analyses at the trace metals facility at Moss Landing Marine Laboratories in Moss Landing. The synthetic organic pesticides, PAHs and PCBs are contracted by CDFG to Dr. Ron Tjeerdema at the UCSC trace organics facility at Long Marine Laboratory in Santa Cruz. MLML currently maintains the Bay Protection and Toxic Cleanup Database for the SWRCB. Described below is a description of that database system.

II. DESCRIPTION OF COMPUTER FILES

The sample collection/field information, chemical, and toxicity data are stored on hard copy, computer disks and on a 486DX PC at Moss Landing Marine Laboratories. Access is limited to Russell Fairey. Contact Russell Fairey at (408) 633-6035 for copies of data. The data are stored in a dBase 4 program and can be exported to a variety of formats. There are three backups of this database stored in two different laboratories. The data are entered into 1 of 2 files. REG9CHEM.DBF file contains all the collection and chemical data. REG9TOX.DBF file contains all the collection and toxicity test data. A hardcopy printout of the dBase database structure is attached, showing precise characteristics of each field.

The REG9CHEM.DBF file is the chemistry data file which contains the following fields (the number at the start of each field is the field number):

1. STANUM. This numeric field is 7 characters wide with 1 decimal place and contains the CDFG station numbers that are used statewide. The format is YXXXX.Z where Y is the Regional Water Quality Control Board Region number and XXXX is the number that corresponds to a given location or site and Z is the number of the station within that site. An example is West Basin in San Diego Harbor where the STANUM is 90050.0. The 9 indicates Region 9. The 0050 indicates that it is Site 50 and the .0 is the replicate (if any) at the station within Site 50.

2. STATION. This character field is 30 characters wide and contains the exact name of the station.

3. IDORG. This numeric field is 8 characters wide and contains the unique i.d. organizational number for the sample. For each station collected on a unique date, an idorg sample number is assigned. This should be the field that links the collection, toxicity, chemical, and other data bases.

4. DATE. This date field is 8 characters long and is the date that each sample was collected in the field. It is listed as MM/DD/YY.

5. LEG. This numeric field is 6 characters wide and is the leg number of the project in which the sample was collected.

6. LATITUDE. This character field is 12 characters wide and contains the latitude of the center of the station sampled. The format is a character field as follows: XX,YY,ZZ, where XX is in degrees, YY is in minutes, and ZZ is in seconds or hundreds.

7. LONGITUDE. This character field is 14 characters wide and contains the longitude of the center of the station sampled. The format is a character field as follows: XX,YY,ZZ, where XXX is in degrees, YY is in minutes, and ZZ is in seconds or hundreds.

8. GISLAT. This numeric field is 12 characters wide with 8 decimal places and contains the latitude of the station sampled in Geographical Information System format. The format is a numeric field as follows: XX.YYYYYYYY, where XX is in degrees and YYYYYYYY is a decimal fraction of the preceding degree.

9. GISLONG. This character field is 14 characters wide with 8 decimal places and contains the longitude of the station sampled. The format is a character field as follows: XXXX.YYYYYYYY where XXXX is in degrees and YYYYYYYY is a decimal fraction of the preceding degree.

10. HUND_SECS. This character is 1 character wide and contains the designation "h" if the latitude and longitude are

given in degrees, minutes and hundredths of a minute. The designation "s" is given when latitude and longitude are given in degrees, minutes and seconds.

11. DEPTH. This character field is 4 characters wide and contains the depth at which the sediment sample was collected, in meters to the nearest one half meter.

12. METADATA. This is an index directing the user to tables or files of ancillary data pertinent to associated test. Character field, width 12.

TRACE METALS IN SEDIMENT are presented in fields 13 through 32. All sediment trace metal results are reported on a dry weight basis in parts per million (ppm).

A. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed.
B. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected.

Sediment trace metals are numeric fields of varying character width, and including the following elements, listed by field number, then field name as it appears in the database, then numeric character width and number of decimal places:

13. TMMOIST. 6.2
14. ALUMINUM. 9.2
15. ANTIMONY. 7.3
16. ARSENIC. 6.3
17. CADMIUM. 7.4
18. CHROMIUM. 8.3
19. COPPER. 7.2
20. IRON. 7.1
21. LEAD. 6.3
22. MANGANESE. 7.2
23. MERCURY. 7.4
24. NICKEL. 7.3
25. SILVER. 7.4
26. SELENIUM. 6.3
27. TIN. 8.4
28. ZINC. 9.4
29. ASBATCH. 5.1
30. SEBATCH. 5.1
31. TMBATCH. The Batch number that the sample was digested in, numeric character width 5 and 1 decimal places.
32. TMDATAQC. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 3. Data qualifier codes are as follows:

A. When the sample meets or exceeds the control criteria requirements, the value is reported as "-4".
B. When the sample has minor exceedances of control criteria

but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, QA evaluations should be consulted before using the data.

C. When QA samples have major exceedances of control criteria requirements and the data are not usable for most assessments and reporting purposes, the value is reported as "-6".
D. When the sample has minor exceedances of control criteriaand is unlikely to affect assessments, the value is reported as -3.

SYNTHETIC ORGANICS are presented in fields 33 through 147. All synthetic organic results are reported on a dry weight basis in parts per billion (ppb or ng/g).

A. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed.
B. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected.

Synthetic organics are reported on a dry weight basis in parts per billion (ppb or ng/g) and are numeric fields of varying character width, and include the following compounds, listed by field number, then field name as it appears in database (and followed by the compound name if not obvious), and then finally, the numeric character width and number of decimal places is given:

33. SOWEIGHT. This numeric field is 6 characters wide with 2 decimal places and contains the weight of the sample extracted for analysis.
34. SOMOIST. This numeric field is 6 characters wide with 2 decimal places and contains the percent moisture of the sample extracted.
35. ALDRIN. 9.3
36. CCHLOR. cis-Chlordane. 9.3
37. TCHLOR. trans-Chlordane. 9.3
38. ACDEN. alpha-Chlordene. 9.3
39. GCDEN. gamma-Chlordene. 9.3
40. CLPYR. Chlorpyrifos. 8.2
41. DACTH. Dacthal. 9.3
42. OPDDD. o,p'-DDD. 8.2
43. PPDDD. p,p'-DDD. 9.3
44. OPDDE. o,p'-DDE. 8.2
45. PPDDE. p,p'-DDE. 8.2
46. PPDDMS. p,p'-DDMS. 8.2
47. PPDDMU. p,p'-DDMU. 8.2
48. OPDDT. o,p'-DDT. 8.2
49. PPDDT. p,p'-DDT. 8.2
50. DICLB. p,p'-Dichlorobenzophenone. 8.2
51. DIELDRIN. 9.3
52. ENDO_I. Endosulfan I. 9.3
53. ENDO_II. Endosulfan II. 8.2
54. ESO4. Endosulfan sulfate. 8.2

55. ENDRIN. 8.2
56. ETHION. 8.2
57. HCHA. alpha HCH 9.3
58. HCHB. beta HCH 8.2
59. HCHG. gamma HCH (Lindane) 9.3
60. HCHD. delta HCH 9.3
61. HEPTACHLOR. 9.3
62. HE. Heptachlor Epoxide. 9.3
63. HCB. Hexachlorobenzene. 9.3
64. METHOXY. Methoxychlor. 8.2
65. MIREX. 9.3
66. CNONA. cis-Nonachlor. 9.3
67. TNONA. trans-nonachlor. 9.3
68. OXAD. Oxadiazon. 8.2
69. OCDAN. Oxychlordane. 9.3
70. TOXAPH. Toxaphene. 7.2
71. PESBATCH.The batch number that the sample was extracted in, numeric character width 6 and 2 decimal places.
72. TBT. tributyltin. 8.4
73. TBTBATCH.The batch number that the sample was extracted in, numeric character width 5 and 1 decimal place.
74. PCB5. 9.3
75. PCB8. 9.3
76. PCB15. 9.3
77. PCB18. 9.3
78. PCB27. 9.3
79. PCB28. 9.3
80. PCB29. 9.3
81. PCB31. 9.3
82. PCB44. 9.3
83. PCB49. 9.3
84. PCB52. 9.3
85. PCB66. 9.3
86. PCB70. 9.3
87. PCB74. 9.3
88. PCB87. 9.3
89. PCB95. 9.3
90. PCB97. 9.3
91. PCB99. 9.3
92. PCB101. 9.3
93. PCB105. 9.3
94. PCB11O. 9.3
95. PCB118. 9.3
96. PCB128. 9.3
97. PCB132. 9.3
98. PCB137. 9.3
99. PCB138. 9.3
100. PCB149. 9.3
101. PCB151. 9.3
102. PCB153. 9.3
103. PCB156. 9.3
104. PCB157. 9.3
105. PCB158. 9.3
106. PCB170. 9.3
107. PCB174. 9.3

108. PCB177. 9.3
109. PCB180. 9.3
110. PCB183. 9.3
111. PCB187. 9.3
112. PCB189. 9.3
113. PCB194. 9.3
114. PCB195. 9.3
115. PCB201. 9.3
116. PCB203. 9.3
117. PCB206. 9.3
118. PCB209. 9.3
119. PCBBATCH. The batch number that the sample was extracted in, numeric character width 6 and 2 decimal place.
120. ARO5460. 9.3
121. ACY. Acenaphthylene. 8.2
122. ACE. Acenaphthene. 8.2
123. ANT. Anthracene. 8.2
124. BAA. Benz[a]anthracene. 8.2
125. BAP. Benzo[a]pyrene. 8.2
126. BBF. Benzo[b]fluoranthrene. 8.2
127. BKF. Benzo[k]fluoranthrene. 8.2
128. BGP. Benzo[ghi]perylene. 8.2
129. BEP. Benzo[e]pyrene. 8.2
130. BPH. Biphenyl. 8.2
131. CHR. Chrysene. 8.2
132. DBA. Dibenz[a,h]anthracene. 8.2
133. DMN. 2,6-Dimethylnaphthalene. 8.2
134. FLA. Fluoranthrene. 8.2
135. FLU. Fluorene. 8.2
136. IND. Indo[1,2,3-cd]pyrene. 8.2
137. MNP1. 1-Methylnaphthalene. 8.2
138. MNP2. 2-Methylnaphthalene. 8.2
139. MPH1. 1-Methylphenanthrene. 8.2
140. NPH. Naphthalene. 8.2
141. PHN. Phenanthrene. 8.2
142. PER. Perylene. 8.2
143. PYR. Pyrene. 8.2
144. TMN. 2,3,4-Trimethylnaphthalene. 8.2
145. PAHBATCH. The batch number that the sample was extracted in, numeric character width 6 and 2 decimal places.
146. SOBATCH. The batch number that the sample was extracted in, numeric character width 6 and 2 decimal places.
147. SODATAQA. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 3. Data qualifier codes are as follows:

A. When the sample meets or exceeds the control criteria requirements, the value is reported as "-4".

B. When the sample has minor exceedances of control criteria but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, the QA evaluations should be consulted before using the data.

C. When QA samples have major exceedances of control criteria requirements and the data are not usable for most assessments and

reporting purposes, the value is reported as "-6".
D. When the sample has minor exceedances of control criteria and is unlikely to affect assessments, the value is reported as -3.

SEDIMENT PARTICULATE SIZE ANALYSES DATA. Field 148, with a field name of "FINES", represents the sediment particulate size ("grain size") analyses data for each station. The grain size results are reported as percent fines.

148. FINES. Sediment grain size (percent fines) for each station. Numeric field, width 5 and 2 decimal places.
A. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed.
B. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected.
149. FINEBATCH. The batch number that the sample was analyzed in, numeric field character width 4.
150. FINEDATAQC. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 3. Data qualifier codes are as follows:
A. When the sample meets or exceeds the control criteria requirements, the value is reported as "-4".
B. When the sample has minor exceedances of control criteria but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, QA evaluations should be consulted before using the data.
C. When QA samples have major exceedances of control criteria requirements and the data are not usable for most assessments and reporting purposes, the value is reported as "-6".
D. When the sample has minor exceedances of control criteria and is unlikely to affect assessments, the value is reported as -3.

SEDIMENT TOTAL ORGANIC CARBON (TOC) ANALYSES DATA. Field 151 presents the levels of total organic carbon detected in the sediment samples at each station. All TOC results are reported as percent of dry weight.

151. TOC. Total Organic Carbon (TOC) levels (percent of dry weight) in sediment, for each station. Numeric field, width 6 and 2 decimal places.
A. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed.
B. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected.
152. TOCBATCH. The batch number that the sample was analyzed in, numeric field character width 4.
153. TOCDATAQC. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 3. Data qualifier codes are as follows:
A. When the sample meets or exceeds the control criteria

requirements, the value is reported as "-4".

B. When the sample has minor exceedances of control criteria but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, the QA evaluations should be consulted before using the data.

C. When QA samples have major exceedances of control criteria requirements and the data are not usable for most assessments and reporting purposes, the value is reported as "-6".

D. When the sample has minor exceedances of control criteria and is unlikely to affect assessments, the value is reported as -3.

The REG9TOX.DBF file is the toxicity data file which contains the following fields (the number at the start of each field is the field number:

1. STANUM. This numeric field is 7 characters wide with 1 decimal place and contains the CDFG station numbers that are used statewide. The format is YXXXX.Z where Y is the Regional Water Quality Control Board Region number and XXXX is the number that corresponds to a given location or site and Z is the number of the station within that site. An example is West Basin in San Diego Harbor where the STANUM is 90050.0. The 9 indicates Region 9. The 0050 indicates that it is Site 50 and the .0 is the replicate (if any) at the station within Site 50.
2. STATION. This character field is 30 characters wide and contains the exact name of the station.
3. IDORG. This numeric field is 8 characters wide with 1 decimal place and contains the unique i.d. organizational number for the sample. For each station collected on a unique date, an idorg sample number is assigned. This should be the field that links the collection, toxicity, chemical, and other data bases.
4. DATE. This date field is 8 characters long and is the date that each sample was collected in the field. It is listed as MM/DD/YY.
5. LEG. This numeric field is 6 characters wide and is the leg number of the project in which the sample was collected.
6. TYPE. This character field is 7 characters wide and describes whether the sample was a field sample, replicate or control.
7. METADATA. This is an index directing the user to tables or files of ancillary data pertinent to associated test. Character field, width 12.
8. CTRL. This character field is 5 characters wide and describes the type of control being used.
9. LATITUDE. This character field is 12 characters wide and contains the latitude of the center of the station sampled. The format is a character field as follows: XX,YY,ZZ, where XX is in degrees, YY is in minutes, and ZZ is in seconds or hundreds.
10. LONGITUDE. This character field is 14 characters wide and contains the longitude of the center of the station sampled. The format is a character field as follows: XX,YY,ZZ, where XXX is in degrees, YY is in minutes, and ZZ is in seconds or hundreds.
11. GISLAT. This numeric field is 12 characters wide with 8 decimal places and contains the latitude of the station sampled

in Geographical Information System format. The format is a numeric field as follows: XX.YYYYYYYY, where XX is in degrees and YYYYYYYY is a decimal fraction of the preceding degree.

12. GISLONG. This character field is 14 characters wide with 8 decimal places and contains the longitude of the station sampled. The format is a character field as follows: XXXX.YYYYYYYY where XXXX is in degrees and YYYYYYYY is a decimal fraction of the preceding degree.

AMPHIPOD SURVIVAL TOXICITY TEST DATA. The following are descriptions of the field headings for the amphipod (Rhepoxynius abronius (RA), presented in fields 13 through 24.

13. RA_MN. Station mean percent survival. Numeric field, width 6 and 2 decimal places.
14. RA_SD. Station standard deviation of percent survival. Numeric field, width 6 and 2 decimal places.
15. RA_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
16. RASITE_MN. Station mean percent survival for replicate of three, when appropriate. Numeric field, width 6 and 2 decimal places.
17. RASITE_SD. Station standard deviation of percent survival for replicate of three, when appropriate. Numeric field, width 6 and 2 decimal places.
18. RASITE_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
19. RA_OTNH3. Total ammonia concentration (mg/L in water) in overlying water (water above bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
20. RA_OUNH3. Unionized ammonia concentration (mg/L in water) in overlying water (water above bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
21. RA_OH2S. Hydrogen sulfide concentration (mg/L in water) in overlying water (water above bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is

missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 4 decimal places.

22. RA_ITNH3. Total ammonia concentration (mg/L in water) in interstitial water (water above bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 10 and 3 decimal places.

23. RA_IUNH3. Unionized ammonia concentration (mg/L in water) interstitial water (water within bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 10 and 3 decimal places.

24. RA_IH2S. Hydrogen sulfide concentration (mg/L in water) in interstitial water (water within bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 10 and 4 decimal places.

25. RABATCH. The batch number that the sample were run in, numeric character width 10.

26. RADATAQC. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 4. Data qualifier codes are as follows:

A. When the sample meets or exceeds the control criteria requirements, the value is reported as "-4".

B. When the sample has minor exceedances of control criteria but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, the QA evaluations should be consulted before using the data.

C. When the QA sample has major exceedances of control criteria requirements and the data is not usable for most assessments and reporting purposes, the value is reported as "-6".

D. When the sample has minor exceedances of control criteria and is unlikely to affect assessments, the value is reported as -3.

ABALONE LARVAL SHELL DEVELOPMENT TOXICITY TEST DATA. The following are descriptions of the field headings for the larval (Haliotis rufescens) shell development toxicity tests, presented in fields 27 through 30. Results are given for undiluted

subsurface water (100%).

27. HRS100_MN. Station mean percent normal development in 100% subsurface water. Numeric field, width 6 and 2 decimal places.
28. HRS100_SD. Station standard deviation of percent normal development in 100% subsurface water. Numeric field, width 6 and 2 decimal places.
29. HRS100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
30. HRS100_NH3. Unionized ammonia concentration (mg/L in water) in subsurface water for each station analyzed in abalone toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 6 and 3 decimal places.

The following are descriptions of the field headings for the sea urchin (Strongylocentrotus purpuratus) fertilization toxicity tests, presented in fields 31 through 41. Results are given for undiluted pore water (100% pore water), pore water that is diluted with Granite Canyon seawater to a 50% of original concentration (50% pore water), and pore water that is diluted with Granite Canyon seawater to a 25% of original concentration (25% pore water).

31. SPPF100_MN. Station mean percent fertilization in 100% pore water. Numeric field, width 6 and 2 decimal places.
32. SPPF100_SD.Station standard deviation of percent fertilization in 100% pore water. Numeric field, width 6 and 2 decimal places.
33. SPPF100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
34. SPPF100NH3. Unionized ammonia concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
35. SPPF100H2S. Hydrogen sulfide concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0"= not detected. Numeric field,

width 7 and 4 decimal places.

36. SPPF50_MN. Station mean percent fertilization in 50% pore water. Numeric field, width 6 and 2 decimal places.
37. SPPF50_SD. Station standard deviation of % fertilization in 50% pore water. Numeric field, width 6 and 2 decimal places.
38. SPPF50_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
39. SPPF25_MN. Station mean percent fertilization in 25% pore water. Numeric field, width 6 and 2 decimal places.
40. SPPF25_SD. Station standard deviation of percent fertilization in 25% pore water. Numeric field, width 6 and 2 decimal places.
41. SPPF25_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.

The following are descriptions of the field headings for the sea urchin embryo (Strongylocentrotus purpuratus) development tests, presented in fields 42 through 54. Results are given for undiluted pore water (100% pore water), pore water that is diluted with Granite Canyon seawater to a 50% of original concentration (50% pore water), and porewater that is diluted with Granite Canyon seawater to a 25% of original concentration (25% pore water).

42. SPPD100_MN. Station mean percent normal development in 100% pore water. Numeric field, width 6 and 2 decimal places.
43. SPPD100_SD. Station standard deviation of percent normal development in 100% pore water. Numeric field, width 6 and 2 decimal places.
44. SPPD100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
45. SPPD100NH3. Unionized ammonia concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
46. SPPD100H2S. Hydrogen sulfide concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-

9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0"= not detected. Numeric field, width 7 and 4 decimal places.

47. SPPD50_MN. Station mean percent normal development in 50% pore water. Numeric field, width 6 and 2 decimal places.
48. SPPD50_SD. Station standard deviation of percent normal development in 50% pore water. Numeric field, width 6 and 2 decimal places.
49. SPPD50_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
50. SPPD25_MN. Station mean percent normal development in 25% pore water. Numeric field, width 6 and 2 decimal places.
51. SPPD25_SD. Station standard deviation of percent normal development in 25% pore water. Numeric field, width 6 and 2 decimal places.
52. SPPD25_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
53. SPPDBATCH. The batch number that the samples were analyzed in, numeric character width 10.
54. SPPDQC. Data qualifier codes are notations used by data reviewers to briefly describe, or qualify data and the systems producing data, numeric character width 3. Data qualifier codes are as follows:
    A. When the sample meets or exceeds the control criteria requirements, the value is reported as "-4".
    B. When the sample has minor exceedances of control criteria but is generally usable for most assessments and reporting purposes, the value is reported as "-5". For samples coded "-5" it is recommended that if assessments are made that are especially sensitive or critical, the QA evaluations should be consulted before using the data.
    C. When the QA sample has major exceedances of control criteria requirements and the data is not usable for most assessments and reporting purposes, the value is reported as "-6".
    D. When the sample has minor exceedances of control criteria and is unlikely to affect assessments, the value is reported as -3.

The following are descriptions of the field headings for the sea urchin embryo (Strongylocentrotus purpuratus) cytogenetic tests, presented in fields 55 through 59. Results are given for undiluted pore water (100% pore water).

55. SPPC100_MN. Station mean percent normal mitosis in

100% pore water. Numeric field, width 6 and 2 decimal places.

56. SPPC100_SD. Station standard deviation of percent normal mitosis in 100% pore water. Numeric field, width 6 and 2 decimal places.
57. SPPC100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 6.
58. SPPC100NH3. Unionized ammonia concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 6 and 3 decimal places.
59. SPPC100H2S. Hydrogen sulfide concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0"= not detected. Numeric field, width 7 and 4 decimal places.

MUSSEL LARVAL SHELL DEVELOPMENT TOXICITY TEST DATA. The following are descriptions of the field headings for the larval (Mytilus edulis) shell development toxicity tests, presented in fields 60 through 63. Results are given for undiluted subsurface water (100%).

60. MES100_MN. Station mean percent normal development in 100% subsurface water. Numeric field, width 6 and 2 decimal places.
61. MES100_SD. Station standard deviation of percent normal development in 100% subsurface water. Numeric field, width 6 and 2 decimal places.
62. MES100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
63. MES100_NH3. Unionized ammonia concentration (mg/L in water) in subsurface water. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 6 and 3 decimal places.

The following are descriptions of the field headings for the larval (Mytilus edulis) shell development toxicity tests, presented in fields 64 through 68. Results are given for undiluted pore water (100% pore water).

64. MEP100_MN. Station mean percent normal development in 100% pore water. Numeric field, width 6 and 2 decimal places.
65. MEP100_SD. Station standard deviation of percent normal development in 100% pore water. Numeric field, width 6 and 2 decimal places.
66. MEP100_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.
67. MEP100_NH3. Unionized ammonia concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 6 and 3 decimal places.
68. MEP100_H2S. Hydrogen sulfide concentration (mg/L in water) in pore water samples (100%). When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0"= not detected. Numeric field, width 7 and 4 decimal places.

POLYCHAETE SURVIVAL TOXICITY TEST DATA. The following are descriptions of the field headings for the polychaete worm (Neanthes arenaceodentata) survival toxicity tests, presented in fields 69 through 71.

69. NASURV_MN. Station mean percent survival. Numeric field, width 6 and 2 decimal places.
70. NASURV_SD. Station standard deviation of % survival. Numeric field, width 6 and 2 decimal places.
71. NASURV_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not statistically significant. Character field, width 5.

POLYCHAETE WEIGHT TOXICITY TEST DATA. The following are descriptions of the field headings for the polychaete worm (Neanthes arenaceodentata) weight toxicity tests, presented in fields 72 through 80.

72. NAWT_MN. Station mean weight (gm). Numeric field, width 6 and 2 decimal places.
73. NAWT_SD. Station standard deviation of weight (gm). Numeric field, width 6 and 2 decimal places.
74. NAWT_SG. Station statistical significance, representing the significance of the statistical test between the home sediment and the sample. A single * represents significance at the .05 level, and double ** represents significance at the .01 level. ns = not

statistically significant. Character field, width 5.

75. NA_OTNH3. Total ammonia concentration (mg/L in water) in overlying water (water above bedded sediment used for polychaete tests) for each station analyzed using polychaete toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
76. NA_OUNH3. Unionized ammonia concentration (mg/L in water) in overlying water (water above bedded sediment used for polychaete tests) for each station analyzed using polychaete toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 7 and 3 decimal places.
77. NA_OH2S. Hydrogen sulfide concentration (mg/L in water) in overlying water (water above bedded sediment used for polychaete tests) for each station analyzed using polychaete toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 9 and 4 decimal places.
78. NA_ITNH3. Total ammonia concentration (mg/L in water) in interstitial water (water above bedded sediment used for polychaete tests) for each station analyzed using polychaete toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 9 and 3 decimal places.
79. NA_IUNH3. Unionized ammonia concentration (mg/L in water) in interstitial water (water within bedded sediment used for polychaete tests) for each station analyzed using polychaete toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 9 and 3 decimal places.
80. NA_IH2S. Hydrogen sulfide concentration (mg/L in water) in interstitial water (water within bedded sediment used for amphipod tests) for each station analyzed using amphipod toxicity tests. When the value is missing or not analyzed, the value is reported as "-9.0" = not analyzed. When the value is less than the detection limit of the analytical test, the value is reported as "-8.0" = not detected. Numeric field, width 9 and 4 decimal places.

CHEMICAL SUMMATIONS AND QUOTIENTS

In the following section, chemical summations (total chlordane, total DDT, total PCBs, LMW PAHs, HMW PAHs, total PAHs) and quotients (ERM and PEL) are presented. Beginning with samples collected during Leg 20 (June, 1993), additional analytes were added to the standard BPTCP synthetic organic analyte list. These additions were made to enable the data set to be more comparable with other monitoring programs. This included addition of analytes used for some of the chemical summations of the PAHs and total chlordane. Resulting summations may be conservative for the PAH and chlordane data for samples taken before Leg 20, because some of the constituents could not be included.

For purposes of these summations, samples which were found to have chemical concentrations less than the method detection limit (-8 in Appendix A) were adjusted to a value of one-half of the method detection limits given in the methods description. The summations were calculated as follows:

Total chlordane

Leg<15 (TTL_CHLR) = $\sum$ ([cis-Chlordane] [trans-Nonachlor])

Leg=15 (TTL_CHLR) = $\sum$ ([cis-Chlordane] [trans-Chlordane])

Leg>15 (TTL_CHLR) = $\sum$ ([cis-Chlordane] [trans-Chlordane] [cis-Nonachlor] [trans-Nonachlor] [Oxychlordane])

Total DDT

All Legs (TTL_DDT) = $\sum$ ([o',p' DDD] [p',p' DDD] [o',p' DDE] [p',p' DDE] [o',p' DDT] [p',p' DDT])

Total PCB

All Legs (TTL_PCB) = $\sum$ ([PCB8] [PCB18] [PCB28] [PCB44] [PCB52] [PCB66] [PCB101] [PCB105] [PCB118] [PCB128] [PCB138] [PCB153] [PCB170] [PCB180] [PCB187] [PCB195] [PCB206] [PCB209])

Low Molecular Weight PAHs

Leg<16 (LMW_PAH) = $\sum$ ([ACE] [ANT] [BPH] [DMN] [FLU] [MNP1] [MPH1] [PHN])

Leg$\geq$16 (LMW_PAH) = $\sum$ ([ACE] [ACY] [ANT] [BPH] [DMN] [FLU] [MNP1] [MNP2] [MPH1] [NPH] [PHN] [TMN])

High Molecular Weight PAHs

Leg<16 (HMW_PAH) = $\sum$ ([BAA] [BAP] [BEP] [CHR] [DBA] [FLA] [PER] [PYR])

Leg$\geq$16 (HMW_PAH) = $\sum$ ([BAA] [BAP] [BBF] [BKF] [BGP] [BEP] [CHR] [DBA] [FLA] [IND] [PER] [PYR])

Total PAHs

All legs (TTL_PAH) = $\sum$ ([LMW_PAH] [HMW_PAH])

ERM Quotients and PEL Quotients were calculated using summations of the individual chemicals for which ERMs and PELs have been

derived (Table 5). Chemical concentrations are divided by their respective ERM or PEL values to obtain a specific individual chemical quotient (example 1). A value greater than one indicates the chemical concentration in that sample exceeded its respective ERM or PEL. A value of five would indicate the chemical was five times higher than the ERM or PEL in that sample.

example - sample IDORG #199 Copper concentration= 170 mg/g
PEL for copper= 108.2

CopperQ= (170 mg/g) / (108.2 mg/g) = 1.57

Summations and averaging of the individual chemical quotients were calculated to give summary ERM Quotients (ERMQ) and PEL Quotients (PELQ). Each quotient summation is divided by the number of analytes used in the summation (Table 5) to yield an average summary quotient.

Summary ERM Quotient

ERMQ = ((ANTIMONYQ + ARSENICQ + CADMIUMQ + CHROMIUMQ + COPPERQ + LEADQ + MERCURYQ + SILVERQ + ZINCQ + TTL_DDTQ + TTL_CHLRQ + DIELDRINQ + ENDRINQ + TTL_PCBQ + LMW_PAHQ + HMW_PAHQ) / 16)

Summary PEL Quotient

PELQ = ((ARSENICQ + CADMIUMQ + CHROMIUMQ + COPPERQ + LEADQ + MERCURYQ + SILVERQ + ZINCQ + TTL_DDTQ + TTL_CHLRQ + DIELDRINQ + LINDANEQ + TTL_PCBQ + LMW_PAHQ + HMW_PAHQ) / 15)

Description of calculations for cumulative frequency distributions of percent area toxic.

The following identifies and describes each of the spreadsheet columns used to generate cumulative frequency functions for estimates of percent area toxic.

Idorg : lists all samples tested for each toxicity test protocol/pore water dilution.
Block#: lists assigned letter/number code for each area (block) based on EMAP block designations. See Figure 2.
 # samples/block: lists total number of samples collected in given block.
toxic: "1" indicates sample toxicity based on EMAP definition (both significant difference from laboratory control and toxicity value <80% of control value). Blank cell indicates no significant toxicity.
mn as % of control : lists sample toxicity means normalized to percentage of the control value.
Area/block : Area in km2 for block associated with each sample
Area/sample : Area in km2 represented by each sample, calculated as: Block area/number of samples collected in given block.
Area/sample as % of total : Area represented by each sample as a percent of the total area sampled.
Cum area/sample as % of total : Cumulative area per sample as a percent of the total area sampled.
% total area toxic/sample : Area represented by each toxic sample as a percent of the total area.
SUMS : Numbers in this row show column totals. Sum of Area/sample gives total area sampled for a given toxicity test protocol. Sum of % of total area toxic/sample gives the total area defined as toxic for given test protocol /pore water dilution.

Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments

Prepared for:

National Oceanic and Atmospheric Administration
United States Department of Commerce
501 West Ocean Boulevard, Suite 4470
Long Beach California 90802

Prepared by:

Donald D. MacDonald
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2376 Yellow Point Road, RR #3
Ladysmith, B.C.
V0R 2E0

August, 1994
(Revised April 1997)

United States, ET AL. v.
Montrose Chemical Corporation of California, ET AL.

Witness Statement of Donald D. MacDonald
MacDonald Environmental Sciences Ltd.
2376 Yellow Point Road, R.R. #3
Ladysmith, British Columbia
V0R 2E0

Phone: 250-722-3631 Fax: 250-722-3651

Background Information Relevant to the Preparation of this Witness Statement

Nature of the Evidence Given

The evidence provided in the following witness statement consists of both factual and opinion evidence.

Professional Qualifications

The professional experience and educational qualifications which qualify me to give the opinions that I give in this witness statement are set out in my curriculum vitae, which is attached as Appendix 6 of this witness statement. My experience in the field of sediment quality assessment includes:

- Development of an approach to the derivation of Canadian sediment quality guidelines;
- Development of numerical sediment quality assessment guidelines for 34 chemical substances in Florida coastal waters;
- Development of Canadian sediment quality guidelines for freshwater ecosystems;
- Development of Canadian sediment quality guidelines for marine and estuarine ecosystems;
- Participation in the development of numerical sediment quality guidelines for NOAA's National Status and Trends Program;
- Development of procedures for deriving site-specific sediment quality remediation objectives;
- Participation in the development of a sediment toxicity database for evaluating matching sediment chemistry and biological effects data; and,
- Development of Canadian sediment quality guidelines for toxaphene, DDTs, and PCBs.

Conflict of Interest

I have no personal interest in this case other than as a paid consultant to the Respondent. My prior involvement with U.S. government agencies has been as a paid consultant on specific projects related to hazard and environmental assessments. I have had no prior involvement with the issue involving Montrose Chemical Corporation of California and others. My company and I will be paid the same regardless of the outcome of this case.

Documents Used to Prepare Evidence

In preparing this evidence, I have reviewed numerous texts, articles, protocols, and publications relating to the fate and effects of sediment-associated contaminants on aquatic organisms, including those related to the effects of dichlorodiphenyl trichloroethane (DDT) and polychlorinated biphenyls (PCBs).

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Executive Summary

A review of the published and unpublished literature was conducted to evaluate the toxic effects of DDTs and PCBs in sediments. The primary objective of this review was to determine sediment effect concentrations (SECs) of DDTs and PCBs in Southern California Bight sediments. The secondary objective of this study was to determine if the concentrations of DDTs and PCBs in the sediments of the Southern California Bight would cause injury to one or more sediment-dwelling species that occur or would be expected to occur in this area. Injurious effects were considered to include any impairments to benthic habitats caused by DDTs or PCBs acting alone, or in any combination with other contaminants (for further information see glossary; Appendix 1).

In this review, DDTs were defined as *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD, and any metabolite or degradation product of these chemicals. The term "PCBs" is defined as all of the polychlorinated biphenyls found in Southern California Bight, plus the degradation products and metabolites of these chemicals (for further information see glossary; Appendix 1).

A tiered strategy was used to derive SECs for DDTs and PCBs in the Southern California Bight. This strategy relied preferentially on data from controlled laboratory studies to establish SECs for these substances (i.e., SECs were derived using the spiked-sediment bioassay approach; SSBA). Information from field studies and other sources were utilized when acceptable data from spiked-sediment bioassays were not available (i.e., SECs were derived using the weight-of evidence approach; WEA). These two approaches were considered to be complementary because cause-and-effect relationships between contaminant concentrations and toxic effects can be established from controlled laboratory experiments. While it is difficult to definitively identify the causes of toxic effects in field studies, SECs derived using the weight-of evidence approach may be more reliable than the SSBA because they reflect the toxicity of complex mixtures of contaminants. This is important in the Southern California Bight because DDTs, PCBs, and other substances occur in complex mixtures.

The SECs developed in this review were evaluated to determine their reliability and predictability. Reliability was evaluated to determine if the SECs accurately identified the concentrations of DDTs and PCBs that are likely to be associated with toxic effects in sediments from the Southern California Bight. The predictability of the SECs was evaluated to determine if information on the

concentrations of DDTs and PBs could be used to accurately predict toxic effects in sediments from elsewhere in the United States (i.e., outside the Southern California Bight). The reliability and predictability of several other concentrations of these substances were also evaluated to provide a basis of comparison with the SECs.

The potential for observing toxic effects due to DDTs and PCBs in Southern California Bight sediments was determined by comparing measured concentrations in toxic samples to the SECs derived in this study. If a group of substances was present in Southern California Bight sediments at concentrations in excess of the SEC, then that substance or group of substances was considered to have been associated with the observed sediment injury.

The available data on the toxic effects of SUM DDT (*p,p'*-DDT + *o,p'*-DDT), SUM DDE (*p,p'*-DDE + *o,p'*-DDE), SUM DDD (*p,p'*-DDD + *o,p'*-DDD), total DDT (sum of all six DDT compounds), Aroclor 1254, and total PCBs were summarized in this review. These groups of substances were the focus of this review because there was no evidence to suggest that there were significant differences in the toxicity of the o,p'- and p,p'-isomers of DDT, DDE, or DDD. In addition, insufficient toxicological information was available on the o,p'-isomers of DDTs or the individual PCB congeners to derive SECs. Acceptable dose-response data from controlled laboratory studies (i.e., spiked-sediment bioassays on sediment-dwelling arthropod species) were located for SUM DDT and Aroclor 1254. Therefore, the SECs for these groups of substances were derived using the SSBA. The SECs for the other groups of substances were derived using the WEA. The SECs for DDTs and PCBs were calculated using both summarized and unsummarized field data; both values have been reported in this document. The SECs derived for DDTs and PCBs are presented in the following table.

The results of the evaluations of reliability and predictability indicate that a high degree of confidence can be placed on the SECs that were derived in this study. Importantly, the dry weight-normalized SECs for all six groups of substances (SUM DDT, SUM DDE, SUM DDD, tDDT, Aroclor 1254, and tPCB) were considered to be reliable. That is, the SECs can be used to accurately classify, as toxic or non-toxic, sediments from the Southern California Bight. The SECs for SUM DDT, SUM DDD, tDDTs, and tPCBs were all highly predictive (i.e., predictability >80%); insufficient data were available to evaluate the predictability of the SECs for SUM DDE and Aroclor 1254. Therefore, most of the SECs can be used to accurately classify sediments from elsewhere in the United States. The agreement between the results of the reliability and predictability evaluations

increase the confidence that can be placed on the SECs. Higher reliability (i.e., 85%) and similar predictability (82%) were obtained for a somewhat higher SEC for SUM DDT (i.e., 0.06 mg/kg DW). A lower SEC for Aroclor 1254 (i.e., 0.4 mg/kg DW) would have been just as reliable (i.e., 95%) and could be used to classify more sediment samples from the Southern California Bight.

The results of these evaluations indicate that the organic carbon-normalized SECs are also reliable. The incidence of toxicity was ≥ 80% when any of these SECs were equalled or exceeded in sediments from the Southern California Bight, which indicates that the SECs provide effective tools for classifying sediments from this geographic area. Most of the SECs can also be used to accurately classify sediments from elsewhere in the United States. The predictability of the SECs for SUM DDT, tDDTs, and tPCBs ranged between 82% and 100%. Lower predictability (71%) was observed for the SEC for SUM DDD; a higher SEC (0.40 mg/kg $DW_{1\%OC}$) would have been more predictive of toxicity in the independent data set (i.e., 100% predictability). Insufficient data were available to evaluate the predictability of the SECs for SUM DDE and Aroclor 1254, indicating the concentrations of these substances tend to be much lower elsewhere in the United States than they are in the Southern California Bight. The general agreement between the results of the reliability and predictability evaluations increases the confidence that can be placed on the SECs.

Based on a review of the existing information, it was concluded that the concentrations of DDTs and PCBs in the sediments of the Southern California Bight were sufficient to cause injury to sediment-dwelling organisms. While much of the information reviewed was from studies conducted in the 1970's and 1980's, the most recent data examined indicate that the concentrations of SUM DDT, SUM DDE, SUM DDD, tDDT, and tPCB in surficial sediments (0 to 2 cm) exceeded the SECs derived in this study at many sites (Bay *et al.* 1994; Sapudar *et al.* 1994; Fairey *et al.* 1996; Fairey 1997). Higher concentrations of these groups of substances were observed in deeper sediments (e.g., 7.5 to 47.5 cm) collected from the Palos Verdes Shelf in 1985 (Swartz *et al.* 1991); the concentrations of Aroclor 1254 in deeper sediments exceeded the SEC derived in this study. The majority of the samples that exceeded the SECs were also toxic, considering amphipod survival, amphipod abundance, or the sea urchin fertilization. These data indicate that the concentrations of DDTs and PCBs in the Southern California Bight were sufficient to cause injury to sediment-dwelling organisms.

A summary of the sediment effect concentrations (SECs) for DDTs and PCBs in in the Southern California Bight.

Substance	Sediment Effect Concentration	Reliability	Predictability
SUM DDT	0.031 mg/kg DW	76%	82%
	0.06 mg/kg DW	**85%**	**82%**
	0.111 mg/kg DW (1%OC)	**91%**	**82%**
	0.333 mg/kg DW (3% OC)	91%	82%
	0.555 mg/kg DW (5% OC)	91%	82%
SUM DDE	***6.58 mg/kg DW***	**97%**	**NA**
	1.58 mg/kg DW (1% OC)	**87%**	**NA**
	4.74 mg/kg DW (3% OC)	87%	NA
	7.90 mg/kg DW (5% OC)	87%	NA
SUM DDD	***0.89 mg/kg DW***	**95%**	**83%**
	0.23 mg/kg DW (1% OC)	91%	71%
	0.40 mg/kg DW (1% OC)	**95%**	**100%**
	1.20 mg/kg DW (3% OC)	95%	100%
	2.00 mg/kg DW (5% OC)	95%	100%
tDDT	***7.15 mg/kg DW***	**95%**	**100%**
	2.00 mg/kg DW (1% OC)	**82%**	**100%**
	6.00 mg/kg DW (3% OC)	82%	100%
	10.0 mg/kg DW (5% OC)	82%	100%
Aroclor 1254	***0.4 mg/kg DW***	**95%**	**33%**
	2.1 mg/kg DW	93%	NA
	0.20 mg/kg DW (1% OC)	**95%**	**25%**
	1.08 mg/kg DW (1% OC)	100%	NA
	0.60 mg/kg DW (3% OC)	95%	25%
	1.00 mg/kg DW (5% OC)	95%	25%
tPCB	***0.835 mg/kg DW***	**87%**	**98%**
	0.577 mg/kg DW (1% OC)	**80%**	**100%**
	1.73 mg/kg DW (3% OC)	80%	100%
	5.40 mg/kg DW (5% OC)	80%	100%

The most highly recommended SECs are shown in bold italics..
NA - Insufficient data available to evaluate predictability.

Table of Contents

List of Tables

List of Appendices

Acknowledgements

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Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs in Sediments

1.0 Introduction

Bed sediments in the Southern California Bight are known to be contaminated by a variety of toxic and bioaccumulative substances (Word and Mearns 1979; Swartz *et al.* 1986; Mearns *et al.* 1991). Specifically, bed sediments on the Palos Verdes Shelf and elsewhere in the Bight have been contaminated by DDTs and PCBs, primarily as a result of discharges from the Whites Point sewage outfall (Mearns *et al.* 1991). These contaminants in the sediments of the Southern California Bight represent a significant environmental concern because:

(i) bed sediments provide essential and productive habitats for communities of benthic and epibenthic (for further information see glossary; Appendix 1) organisms;

(ii) sediment-dwelling biota are important elements of coastal ecosystems; and,

(iii) the presence of elevated concentrations of-sediment-associated contaminants, specifically DDTs and PCBs, could be harmful to sediment-dwelling organisms.

The purpose of this witness statement is to conduct a review of the toxic effects of DDTs and PCBs in sediments. In this review, the term "DDTs" is defined as *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD, and any metabolite or degradation product of these chemicals. The term "PCBs" is defined as all of the polychlorinated biphenyls found in Southern California Bight, plus the degradation products and metabolites of these chemicals. The primary objectives of this witness statement are:

(i) to review the published literature and other existing information on the effects of sediment-associated DDTs and PCBs;

(ii) to determine sediment effect concentrations (SECs) of DDTs and PCBs in Southern California Bight sediments; and,

(iii) to determine if DDTs and/or PCBs occur in the sediments of the Southern California Bight at concentrations that are sufficient to cause injury to one or more sediment-dwelling species that occur or would be expected to occur in this area.

In this study, SECs are defined as the concentrations of DDTs and PCBs in bed sediments that would be sufficient to cause injury to sediment-dwelling organisms (including infaunal and epibenthic organisms). Injurious effects were considered to include any impairments to benthic organisms and habitats caused by DDTs or PCBs acting alone or in any combination with other contaminants. Such impairments to sediment-dwelling organisms and benthic habitats were considered to be indicated by:

(i) acute or chronic mortality;

(ii) reduced growth;

(iii) impaired reproduction;

(iv) abnormal development;

(v) increased incidence of tumours or pre-cancerous lesions;

(vi) altered organ morphology or size;

(vii) avoidance behavior; or,

(viii) altered benthic invertebrate community structure.

The SECs derived in this study do not consider the potential effects of sediment-associated contaminants on fish or other species that reside in the water column. In addition, the SECs do not consider the potential for bioaccumulation in marine organisms nor the potential effects that could occur throughout the food web as a result of the bioaccumulation of these substances.

The SECs reported in this document may be used to determine the areal extent of sediment injury in the Southern California Bight. In addition, these SECs may be used as a scientific basis for establishing target clean-up levels for DDTs and PCBs in bed sediments, if the results of a damage assessment indicate the need for sediment remediation. **However, the sediment effect concentrations represent the concentrations of DDTs and PCBs at which adverse biological effects have been demonstrated in laboratory and/or field studies; therefore, target clean-up levels would have to be lower to ensure that bed sediments would support healthy and diverse populations of sediment-dwelling organisms.**

1.1 Glossaries of Terms and Acronyms

Many of the terms and acronyms used in this review may not be familiar to the reader. For this reason, glossaries of terms and acronyms have been included to facilitate interpretation of the information presented in this review (see Appendices 1 and 2). In addition, the following table provides a means of quickly converting data between the various units that have been utilized.

Table 1. Conversion factors for standard and scientific units.

Standard Units		Scientific Units
1 part per million (ppm)	=	1 mg/kg, 1 µg/g, 1000 µg/kg
1 part per billion (ppb)	=	1 µg/kg
1 mg/kg OC	=	0.01 mg/kg $DW_{1\%OC}$

2.0 Methods

2.1 Introduction

This review of the toxic effects of DDTs and PCBs in the Southern California Bight consisted of four main steps. First, procedures for deriving sediment effect concentrations (SECs) were selected from the existing approaches that have been established for developing sediment quality guidelines and criteria (for further information see glossary; Appendix 1). Next, the available data on the toxic effects of DDTs and PCBs were collected, evaluated, and compiled. Subsequently, SECs for DDTs and PCBs were derived using the available toxicity data. Finally, the SECs for DDTs and PCBs were evaluated to determine their reliability and predictability. Each of these steps is described in more detail below and in Appendix 3.

2.2 Selection of Procedures for Deriving SECs for DDTs and PCBs in the Southern California Bight

Sediment quality guidelines (SQG) and criteria (SQC) are numerical limits or narrative statements that are recommended to support and maintain the designated uses of an aquatic environment (CCME 1994). In contrast, the SECs derived in this study are intended to identify the concentrations of sediment-associated contaminants that are likely to cause, or to be associated with, adverse effects on sediment-dwelling organisms. While the level of protection afforded to sediment-dwelling organisms differs among the various assessment tools (e.g., SQGs, SQC, SECs, etc.), the general procedures used to derive these values may be similar.

A variety of approaches has been used to derive SQGs for sediment-associated contaminants (for reviews see Beak Consultants Ltd. 1987; 1988; Chapman 1989; Persaud *et al.* 1989; Sediment Criteria Subcommittee 1989; Adams *et al.* 1992; USEPA 1992; MacDonald *et al.* 1992). However, there is no general agreement as to which approach will provide the most reliable, flexible, and credible guidelines for evaluating sediment quality (Long *et al.* 1995a). For this reason, the procedures for deriving SECs were selected by considering both the need for reliable SECs and the availability of data on the toxic effects of DDTs and PCBs. Dose-response data, generated in controlled laboratory studies, provide information that can be used directly to evaluate the toxicity of sediment-associated contaminants; however, only a limited number of such studies (i.e., spiked-sediment bioassays) have been conducted to assess the effects of sediment-

associated DDTs and PCBs on estuarine and marine organisms. To optimize the use of the available information, a tiered strategy was established to develop SECs in DDTs and PCBs in the Southern California Bight. This strategy relies preferentially on the spiked-sediment bioassay approach (SSBA) for deriving SECs, with the weight-of-evidence approach (WEA) utilized when insufficient data are available to support the SSBA.

The information from spiked-sediment bioassays was used preferentially because it provided dose-response data for quantitatively determining the effects of contaminants in sediments. By establishing cause-and-effect relationships between contaminant concentrations and adverse biological effects, spiked-sediment bioassays generate high confidence in the resultant SECs. In addition, data from controlled laboratory studies can provide important information for identifying the sediment characteristics that affect the bioavailability and, hence, toxicity of sediment-associated contaminants (Swartz *et al.* 1990).

The weight-of-evidence approach was also a central component of the tiered strategy for deriving SECs. In contrast to the SSBA, the WEA integrates data on the effects of sediment-associated contaminants from many different sources, mainly from field studies. This is important because the biological effects associated with a chemical substance in the field may be affected by factors such as the occurrence of other contaminants, by the presence of substances or conditions that affect contaminant bioavailability (e.g., organic carbon), or by the nature of the resident biological community. By integrating data on many species, life stages, endpoints, exposure durations, and locations, the weight-of-evidence approach facilitates the development of SECs that are likely to be directly relevant for assessing the potential for observing toxic effects in field-collected sediments.

These two approaches are complementary because they address two major issues associated with the quality of the SECs, precision and reliability. Because the SSBA utilizes data from controlled laboratory studies, it tends to provide precise SECs (i.e., the results of toxicity tests conducted under similar experimental conditions in different laboratories are likely to be similar. Hence, effect concentrations for a specific species and life stage can be defined precisely; Lamberson and Swartz 1992). Because the WEA primarily utilizes data on the toxicity of field-collected sediments and on *in situ* benthic invertebrate community alterations, it tends to provide reliable SECs (i.e., the SECs are likely to correctly predict the toxic effects of chemicals in the field). Correspondence between the SECs derived using these two approaches generates additional confidence in the applicability of the SECs for assessing contaminated sediments.

It should be noted that safety factors (for further information see glossary; Appendix 1) were not applied to the SECs derived using either the spiked-sediment bioassay or weight-of-evidence approaches. Therefore, the SECs derived in this study are not considered to be protective of aquatic life and should not be used as target clean-up levels. Rather, the SECs represent the levels of DDTs and PCBs at which adverse biological effects on sediment-dwelling organisms in the Southern California Bight would be expected to occur. The development of sediment quality remediation objectives (i.e., target clean-up levels) for DDTs and PCBs in the Southern California Bight will require implementation of additional procedures (such as those described by MacDonald and Sobolewski 1993). Sediment quality remediation objectives would, necessarily, be lower than the SECs derived in this review, if they are intended to protect sediment-dwelling organisms.

2.3 Collection, Evaluation, and Compilation of Toxic Effects Data

Information on the toxic effects of DDTs and PCBs was collected in two stages. In the first stage, more than ten bibliographic databases were searched for information relevant to the derivation of SQGs. In addition, over 300 scientists were contacted by telephone or letter to obtain recent or unpublished information. This data collection effort resulted in the identification and retrieval of more that 780 references that, potentially, included information on the toxic effects of sediment-associated contaminants.

In the second stage, several additional bibliographic databases were searched to obtain more recent published information on DDTs and PCBs. These databases included Enviroline, CAS Online, Aquatic Sciences and Fisheries Abstracts, and the National Technical Information Service. Additionally, many researchers active in the sediment quality assessment field were contacted directly to acquire the most recent published and unpublished information on these substances. Several new references were obtained as a result of this ancillary data acquisition effort. The information that was collected during the course of this investigation was considered to provide a comprehensive basis for evaluating the toxic effects of DDTs and PCBs in sediments.

All of the references retrieved during the course of the study were critically reviewed to determine their applicability to the SEC derivation process. The screening criteria used in this data evaluation are described in Appendix 3. Data from the Southern California Bight which met all of the screening criteria were

incorporated into a project database and used to derive the SECs for DDTs and PCBs. Data from other areas that met all but the geographic screening criteria were incorporated into an independent database and used to evaluate the SECs.

2.4 Derivation of the SECs

A tiered strategy was used to develop SECs for DDTs and PCBs for the Southern California Bight. In the first tier of this strategy, SECs were derived using the spiked-sediment bioassay approach (SSBA). The weight-of-evidence approach (WEA) was utilized when a sufficient quantity of acceptable data were not available to support the SSBA. Specifically, SECs were derived as follows (the detailed procedures used to derive the SECs for DDTs and PCBs are described in Appendix 3):

(i) if acceptable data from spiked-sediment bioassays were available on at least one infaunal or epibenthic arthropod species, then the lowest observed effect level (LOEL) for the most sensitive life stage of the most sensitive species was identified and adopted as the SEC; and,

(ii) if acceptable data from spiked-sediment bioassays were not available, then the WEA was used to calculate an effects range-median (ER-M) from the information assembled in each toxicological data set. Both summarized and unsummarized data sets were used to calculate dry weight normalized and organic normalized ER-Ms (see Appendix 3). The lower of the two ER-Ms calculated was adopted as the SEC.

Dose-response data on the most sensitive life stage of the most sensitive species (from controlled laboratory studies) are considered to provide a precise basis for identifying the concentrations of DDTs and PCBs that would be sufficient to cause injury to sediment-dwelling species. However, SECs derived using this approach could underestimate the toxicity of contaminants. Toxicity could be underestimated because the test species and life stages used in bioassays may not fully represent the range of sensitivities of sediment-dwelling organisms in the Southern California Bight. In addition, short-term (acute) toxicity tests which measure mortality as the experimental endpoint may not be as sensitive as longer-term (chronic) tests that consider non-lethal endpoints (e.g., growth and reproduction). Furthermore, the presence of additional contaminants in sediments

Therefore, it is likely that effects on sensitive species in the environment would be observed when SECs, derived using the SSBA, are exceeded in sediments.

The ER-Ms derived using the WEA are considered to provide a reliable basis for identifying the concentrations of DDTs and PCBs that would be sufficient to cause injury to sediment-dwelling species. Effects range-median values often correspond with the results of spiked-sediment bioassays. For example, Long *et al.* (1995a) reported ER-Ms for cadmium, zinc, and fluoranthene of 9.6, 410, and 5.1 mg/kg DW for marine and estuarine ecosystems. By comparison, the 10 day median lethal concentrations (LC_{50}s; for further information see glossary; Appendix 2) of these substances to the amphipod, *Rhepoxynius abronius*, are 6.9 to 11.5 mg/kg DW (Kemp *et al.* 1986; Robinson *et al.* 1988), 276 mg/kg DW (Swartz *et al.* 1988), and 3.3 to 10.5 mg/kg DW (Swartz *et al.* 1987; Swartz *et al.* 1988) for cadmium, zinc, and fluoranthene, respectively. Therefore, effects on sensitive species in the environment are likely to be observed when ER-Ms are exceeded in sediments.

2.5 Evaluation of the SECs

The SECs derived in this study were evaluated in two ways. First, the reliability of the SECs was evaluated, using matching sediment chemistry and biological effects data from Southern California. In this study, reliability is a measure of the ability of the SECs to correctly predict toxic effects in sediments from the Southern California Bight. Second, the predictability of the SECs was evaluated using a series of independent data sets from marine and estuarine sites located elsewhere in the United States (i.e., matching sediment chemistry and biological effects data that were not used to derive the SECs). In this study, predictability is a measure of the ability of the SECs to correctly predict toxic effects in the sediments from outside the Southern California Bight. Together, the determinations of reliability and predictability provide a technical basis for determining the level of confidence that can be placed on the various SECs.

The reliability of the SECs was evaluated using matching sediment chemistry and biological effects data from sites located in the Southern California Bight (i.e., Point Conception to the United States-Mexico border). In the reliability evaluation, the concentrations of DDTs and PCBs in each sediment sample were compared to the SECs. These comparisons formed the basis for the predictions that were made regarding the toxicity of each sediment sample. Sediment samples with concentrations of DDTs and/or PCBs that equaled or exceeded the SECs

were predicted to be toxic. The reliability of the SECs was then evaluated by comparing the predictions with the results of solid phase and porewater toxicity tests and benthic invertebrate community assessments. In the reliability evaluation, the following endpoints were considered to be indicative of toxicity:

- Impaired amphipod survival;
- Impaired sea urchin survival, growth, and reproduction (as indicated by rates of fertilization and normal development);
- Impaired polychaete survival, growth, and reproduction (as indicated by the production of emergent juveniles);
- Impaired sand dollar growth and reproduction (as indicated by the rate of normal development);
- Impaired nematode reproduction (as indicated by population growth); and,
- Altered community composition (as indicated by reduced abundance of sensitive taxa; specifically, amphipods and arthropods).

The reliability of each SEC was calculated as the ratio of the number of samples that were correctly predicted to be toxic and the number of samples that were originally predicted to be toxic (expressed as a percentage; Ingersoll *et al.* 1996). Toxic samples were defined as those for which one or more of the measured endpoints were significantly different from control or reference samples (MacDonald *et al.* 1996). The SECs were considered to be reliable if greater than 75% of the samples that were predicted to be toxic actually were toxic (Long *et al.* In review).

The predictability of the SECs was evaluated using the same procedure that was used to evaluate the reliability of the SECs. In this case, however, an independent data set was used to evaluate the SECs. The independent data set was comprised of matching sediment chemistry and biological effects data from sites located throughout the United States that were not considered during the derivation of the SECs (i.e., outside the Southern California Bight). Data were included in the independent data set if they met all of the screening criteria outlined in Appendix 3 (except the geographic criterion). The independent data used in this evaluation are presented in Appendix 5.

In the predictability evaluation, the concentrations of the concentrations of DDTs and PCBs in each sediment sample were compared to the SECs. These comparisons formed the basis for the predictions that were made regarding the

toxicity of each sediment sample. Sediment samples with concentrations of DDTs and/or PCBs that equaled or exceeded the SECs were predicted to be toxic. The predictability of the SECs was then evaluated by comparing the predictions with the results of solid phase and porewater toxicity tests and benthic invertebrate community assessments. The following endpoints were considered to be indicative of toxicity in the evaluation of predictability:

- Impaired amphipod survival;
- Impaired sea urchin survival, growth, and reproduction (as indicated by rates of fertilization and normal development);
- Impaired polychaete survival, growth, and reproduction (as indicated by the production of emergent juveniles);
- Impaired sand dollar growth and reproduction (as indicated by the rate of normal development);
- Impaired nematode reproduction (as indicated by population growth); and,
- Altered community composition (as indicated by reduced abundance of sensitive taxa; specifically, amphipods and arthropods).

The predictability of each SEC was calculated as the ratio of the number of samples that were correctly predicted to be toxic and the number of samples that were originally predicted to be toxic (expressed as a percentage). In this evaluation, toxic samples were defined as those for which one or more of the measured endpoints were significantly different from control or reference samples (MacDonald *et al.* 1996). The SECs were considered to be predictive if greater than 75% of the samples that were predicted to be toxic actually were toxic (Long *et al.* In review).

The SECs are intended to identify the concentrations of DDTs and PCBs that are likely to be associated with adverse effects on sediment-dwelling organisms in the Southern California Bight. As such, the SECs should be established at levels that maximize the potential for correctly classifying of toxic samples as toxic and minimize the potential for incorrectly classifying non-toxic samples as toxic (i.e., false positives). The criteria for assessing the reliability and predictability of the SECs support this objective by establishing target levels for correct classification and false positives at $\geq$ 75% and $\leq$ 25%, respectively (Long *et al.* 1995a). By comparison, the overall incidence of toxicity in the dependent (i.e., with the Southern California Bight) and the independent (i.e., elsewhere in the United States) data sets ranged from 52 to 61% and from 10 to 64%, respectively.

3.0 Evaluation of the Toxicity of Sediment-Associated DDTs

3.1 Identity and Nomenclature of DDTs

Technical grade DDT is a broad spectrum chlorinated hydrocarbon insecticide that was used in a variety of applications for over 30 years in the United States (USEPA 1980a). As a persistent, non-systemic (i.e., not incorporated into the plant) contact and ingested insecticide, DDT provides effective control of a variety of insect pests over extended time periods (Worthing and Hance 1991). While this persistence decreases the need for repeated use of the pesticide at the application site, it also increases the potential for effects on non-target species. In addition, the physicochemical properties of this substance (i.e., low solubility in water and high solubility in lipids) are such that DDT and its metabolites tend to accumulate in the tissues of aquatic organisms and magnify in the food web (USEPA 1980a). While the use of this pesticide has been suspended in the United States, it is still manufactured and used in a number of countries worldwide.

Technical grade DDT (CAS RN: 50-29-3), or dichlorodiphenyltrichloroethane, is comprised primarily of six compounds, including *p,p'*-DDT (77.1%), *o,p'*-DDT (14.9%), *p,p'*-DDE (4.0%), *o,p'*- DDE (0.1%), *p,p'*-DDD (0.3%) and *o,p'*-DDD (0.1%). In addition, technical grade DDT also includes a number of compounds that have not been identified (3.5%; USEPA 1980a). DDT has several metabolites that are frequently found in the environment, including DDEs and DDDs (or Rhothane, which was also manufactured and used as an insecticide for several years; USEPA 1980a). Several other metabolites and degradation products have also been identified (see USEPA 1980a for more information).

In this review, the term "total DDT (tDDT)" is used to refer to the sum of the concentrations of six compounds, including *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, and *o,p'*-DDD. The term "SUM DDT" is used to refer to the sum of the concentrations of *p,p'*-DDT and *o,p'*-DDT isomers. The term "SUM DDE" is used to refer to the sum of the concentrations of *p,p'*-DDE and *o,p'*-DDE isomers. The term "SUM DDD" is used to refer to the sum of the concentrations of *p,p'*-DDD and *o,p'*-DDD isomers. The more general term "DDTs" is used to refer to any of the six substances identified above and their metabolites and degradation products.

While it would be desirable to identify sediment effect concentrations for each of the six DDT compounds, toxicological data were not always available on all of these substances. In some field studies (e.g., Word and Mearns 1979), only the concentrations of total DDT were reported. In other field studies (e.g., Swartz *et*

al. 1985; Swartz *et al.* 1986; Ferraro *et al.* 1991), the concentrations of the *p,p'*-isomers of DDE, DDT and/or DDD were reported; however, the concentrations of the *o,p'*-isomers were not measured. In the most recent field surveys, however, the concentrations of all six DDT compounds were reported (Anderson *et al.* 1988; Bay *et al.* 1994). Typically, the concentrations of total DDT have been reported in spiked-sediment bioassays, which is considered to be comprised of >90% *p,p'*-DDT and *o,p'*-DDT. The inconsistencies in the analytes represented in sediment chemistry data make it difficult to compare the results obtained for different substances and from different studies.

To facilitate the development of SECs, data were compiled for four groups of substances: SUM DDT; SUM DDE; SUM DDD; and, tDDT. This approach to data reduction was considered to be appropriate because few data were available on several of the individual DDT isomers. In addition, no information was located that indicated that the toxicity of, for example, DDE varied depending on which isomer was considered (i.e., *p,p'*-DDE or *o,p'*-DDE).

As indicated above, individual field studies have reported a variety of chemical concentration data. When information on the concentrations of both the *o,p'*- and *p,p'*-isomers was reported, the sum of the two values was calculated (e.g., *o,p'*-DDE + *p,p'*-DDE = SUM DDE). Alternatively, the sum of the concentrations of the two isomers was estimated from the concentration of the *p,p'*-isomer. The conversion factor used in these calculations was the mean ratio of the concentration of the *p,p'*-isomer to the concentration of the sum of the two isomers and was determined using the data reported by Bay *et al.* (1994). For example, the mean ratio of *p,p'*-DDE to SUM DDE in Palos Verdes sediments was 0.886 ± 0.021 (Bay *et al.* 1994). Therefore, SUM DDE concentrations were estimated, as necessary, by dividing the concentrations of *p,p'*-DDE by the conversion factor (0.886). The conversion factors used to estimate SUM DDT and SUM DDD concentrations were 0.894 and 0.863, respectively.

The concentrations of tDDT were determined using one of three methods. When the concentrations of all six analytes were reported, the sum of these values was calculated. When the concentrations of *p,p'*-DDT, *p,p'*-DDE, and *p,p*-DDD were reported, the sum of these three values was determined. Subsequently the concentration of tDDT was estimated by summing the estimated concentrations of the SUM DDT, SUM DDE, and SUM DDD. When only the concentration of *p,p'*-DDE was measured, this value was divided by an empirically derived conversion factor (0.787; which was determined using data from Bay *et al.* 1994) to estimate tDDT concentrations.

The sediment chemistry data used to estimate the concentrations of DDTs in field studies (i.e., Bay *et al.* 1994) were revised after these calculations had been completed. The data reported from Bay *et al.* (1994) reflect these revisions. However, the estimated concentrations of DDTs (i.e., derived using the above conversion factors) in other field studies were not revised. As such, the actual concentrations of DDTs could be higher or lower (by up to 1.1%) than the concentrations reported in this document. This uncertainty in the concentrations of DDTs was considered to be negligible; therefore, the data reported herein were not revised.

3.2 Toxic Effects of SUM DDT

Information on the toxic effects of sediment-associated SUM DDT are available from two types of studies, including controlled laboratory studies and field investigations. Once again, information from controlled laboratory spiked-sediment bioassays provides precise dose-response data for assessing the toxic effects of SUM DDT. By comparison, data from field surveys provide information that can be used to link contaminant concentrations to adverse biological effects; however, these data do not provide dose-response information for definitively identifying causality. A variety of factors, other than the concentration of DDTs, could also have contributed to, or been responsible for, the effects observed in these types of investigations. However, the co-occurrence analyses used to evaluate these field data identify the contaminants that are most strongly linked to the observed toxic effects. Both types of data are summarized in the following sections.

3.2.1 Spiked-Sediment Bioassay Data

Acute toxicity data are available on three species of benthic macroinvertebrates, including one polychaete, one decapod, and one amphipod. One of these species is known to occur in the Southern California Bight (an amphipod, *Rhepoxynius abronius*), while the other two species (a polychaete, *Nereis virens* and a decapod, *Crangon septemspinosa*) are closely related to species that occur in this area (i.e., members of the same genera were recorded in this area; SCAMIT 1994). Therefore, these toxicity data were considered to be relevant for identifying sediment effect concentrations of SUM DDT in the Southern California Bight. None of these studies provided information on the composition of the DDTs used in the bioassays (i.e., *p,p'*-DDT vs. *o,p'*-DDT); it was assumed that either technical

grade or reagent grade DDTs were utilized in these investigations. Both of these formulations consist of greater than 90% SUM DDT (USEPA 1980a).

The information available suggests that the sand shrimp, *Crangon septemspinosa*, is the most sensitive species to the effects of sediment-associated DDTs represented in the toxicological data set. McLeese and Metcalfe (1980) spiked sandy sediments (97% sand) by dissolving DDT in hexane and adding the mixture to the bottom of two liter beakers. After the solvent had evaporated, sediment and seawater were added to the beaker. The sand shrimp were added to the test chambers after the sediment had settled and were exposed to DDT-spiked sediment for 96 hours. Neither the sediment nor the overlying water were renewed at any time during this test (i.e., a static toxicity test design was employed). These investigators reported a median lethal concentration of 0.031 mg SUM DDT/kg DW for this species (at 0.28% TOC DW; McLeese and Metcalfe 1980). As the exposure system was not at equilibrium (for further information see glossary; Appendix 1) during the tests (i.e., concentrations of DDTs in sediments increased over the four day period), the LC_{50} was determined based on the average concentrations of DDT measured during the experiment. By dividing the original LC_{50} by the level of TOC in the sediment, it was possible to calculate an LC_{50} of 0.111 mg SUM DDT/kg $DW_{1\%OC}$ for the sand shrimp.

Data from a related study indicate that the sand worm, *Nereis virens*, is not as sensitive as the sand shrimp to the effects of SUM DDT (when mortality was considered as the experimental endpoint). In static renewal tests (i.e., the animals were transferred to newly prepared sediments every 4 days), McLeese *et al.* (1982) exposed sand worms to concentrations of up to 16.5 mg SUM DDT /kg DW (at 2% TOC DW) for a period of 288 hours (12 days). No mortalities were observed in any of the test chambers over this period. Therefore, an LC_{50} of >16.5 mg SUM DDT/kg DW, or >8.25 mg DDTs/kg $DW_{1\%OC}$, was calculated for this species. Based on the results of this test, these authors concluded that the sand worm was less sensitive to organochlorines (including DDTs) than was the sand shrimp. Once again, the methods used in this study did not produce equilibrium conditions in the test chambers; hence, calculated average concentrations of SUM DDT were used to determine the LC_{50}s.

In a more recent study, Plesha *et al.* (1988) examined the toxicity of a mixture of four chlorinated hydrocarbons to the amphipod, *Rhepoxynius abronius*. The chlorinated hydrocarbons used in this investigation included Aroclor 1254, hexachlorobutadiene, 2,2-bis(*p*-chlorophenyl)-1,1,1-trichloroethane (*p,p'*-DDT), and hexachlorobenzene, at nominal concentrations of 1.0, 0.1, 0.2, and 0.05 mg/kg DW. The sediment used in this study was a silty sand (54% sand) with 0.9%

organic carbon (0.9% OC DW). The contaminants were added to the sediments in an acetone solution, processed, and stored for 48 hours prior to adding the test organisms. The results of the acute (10 day) toxicity bioassays, in which mortality was the endpoint measured, indicated that the sediments with 0.2 mg/kg DW of SUM DDT were significantly toxic to amphipods (survival was reduced by more than 20% relative to control treatments). Much lower survival rates (i.e., <20% survival) were observed when the amphipods were exposed to sediments that had higher nominal concentrations of chlorinated hydrocarbons (1 mg/kg DW of SUM DDT). These results suggest that SUM DDT, when present in mixtures of chlorinated hydrocarbons, may be toxic to amphipods at concentrations as low as 0.200 mg/kg DW or 0.222 mg/kg $DW_{1\%OC}$.

3.2.2 Field Data

In the past 20 years, a number of studies have been conducted in the Southern California Bight to address environmental concerns associated with contaminated sediments. While the majority of these studies were conducted in the late 1970's and early 1980's (Word and Mearns 1979; Swartz *et al.* 1985; Swartz *et al.* 1986), several follow-up surveys have been implemented to assess trends in sediment contamination and/or toxicity. A total of seven studies from the Southern California Bight with matching chemistry and biological effects data were reviewed to support the development of SECs for SUM DDT (Anderson *et al.* 1988; Swartz *et al.* 1991; Ferraro *et al.* 1991; Bay *et al.* 1994; Sapudar *et al.* 1994; Fairey *et al.* 1996; Fairey 1997; Tables A4-1 to A4-4).

In 1980, Swartz *et al.* (1985) examined sediment toxicity, contamination, and benthic macroinvertebrate community structure at seven sites in the vicinity of the Los Angeles County Sanitation District's sewage outfalls on the Palos Verdes Shelf. Sediment toxicity was evaluated in laboratory bioassays that utilized the amphipod, *Rhepoxynius abronius*, as the test organism. The results of this study indicated that the sediments from three stations (1, 2, and 3) were acutely toxic to amphipods during 10-day exposures. These investigators reported that benthic macroinvertebrate community structure (as indicated by infaunal index) had also been altered at these sites. The average concentration of SUM DDT at the stations 1, 2, and 3 was 0.062 ± 0.058 mg/kg DW (0.016 ± 0.014 mg/kg $DW_{1\%OC}$). The average concentration of this substance at the non-toxic stations (4, 5, 6, and 7) was 0.021 ± 0.041 mg/kg DW (0.005 ± 0.011 mg/kg $DW_{1\%OC}$). Several other metrics (including species richness, crustacean abundance, echinoderm abundance, and total abundance) indicated that the benthic invertebrate community

structure was altered at sites that had mean SUM DDT concentrations of between 0.070 and 0.537 mg/kg DW (0.010 to 0.017 mg/kg $DW_{1\%OC}$). As the analytical detection limits were not reported in this study, the mean SUM DDT concentrations were calculated using an assumed value of 0.0 mg/kg DW for the sites without detectable levels of SUM DDT.

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes shelf and Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10 day) toxicity tests using amphipods (*Rhepoxynius abronius*) were conducted on each of the core sections. The results of this investigation indicates that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The concentrations of SUM DDT averaged 6.28 ± 3.07 mg/kg DW (0.799 ± 0.407 mg/kg $DW_{1\%OC}$) in these sediments. Shallower and deeper sediments (2.5 - 12.5 cm and 37.5 to 47.5 cm) from these sites were moderately toxic to amphipods (35.9 ± 12.1% mortality); these samples had, on average, 1.86 ± 1.77 mg/kg DW (0.475 ± 0.455 mg/kg $DW_{1\%OC}$) of SUM DDT. By comparison, the samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average SUM DDT concentrations of 0.191 ± 0.509 mg/kg DW (0.055 ± 0.100 mg/kg $DW_{1\%OC}$).

In 1987, Anderson *et al.* (1988) sampled sediments from seven sites in the vicinity of Palos Verdes and three sites in San Diego Harbor. Only the data from the sites located in the Palos Verdes area was used in this review. While this survey was explicitly designed to evaluate the toxicity of PAH-contaminated sediments, at least one of the sites sampled also had relatively elevated levels of DDTs. Matching sediment chemistry and biological effects data were available for all of these sites. The sediment chemistry data consisted of information on over 50 substances, including six DDT isomers. The biological effects information collected during this survey were generated using three laboratory bioassays (Microtox, amphipod, and sea urchin). However, the Microtox data (which are based on measurements of bacterial luminescence) were not considered because the ecological relevance of this test is uncertain. While benthic invertebrate community structure data were also collected, differences in the depth and other physical features (i.e., open ocean vs. sheltered) at the various sites that were sampled complicated the interpretation of this information. Therefore, these latter data were not used in this review.

The results of the 10-day acute toxicity tests conducted in this study indicated that the survival and reburial of the amphipod, *Grandidierella japonica*, were not correlated with concentrations of SUM DDT in Southern California Bight sediments (Anderson *et al.* 1988). For example, the mean concentration of SUM

DDT was 0.004 ± 0.002 mg/kg DW (0.002 ± 0.002 mg/kg $DW_{1\%OC}$) in sediments that were significantly toxic to this species. In contrast, the mean concentration of SUM DDT in sediments that did not affect amphipod survival was 0.070 ± 0.150 mg/kg DW (0.019 ± 0.035 mg/kg $DW_{1\%OC}$). As the concentrations of these substances were lower at the toxic sites than they were at the non-toxic sites, something other than the concentrations of SUM DDT must have been responsible for the observed toxicity.

The results of the 35-day chronic toxicity tests conducted by Anderson *et al.* (1988) indicate that the survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, were not correlated with the concentrations of sediment-associated DDTs (i.e., there was no concordance between sediment chemistry and the biological effects). However, sea urchin growth (as measured by increases in weight or test width; the test is the hard outer covering of the sea urchin) was linked with concentrations of SUM DDTs. Reduced growth rates were observed in sediments with a mean concentration of 0.191 ± 0.261 mg SUM DDT/kg DW (0.045 ± 0.063 mg/kg $DW_{1\%OC}$). No effects on growth were evident at the sites with an average concentration of 0.007 ± 0.009 mg SUM DDT/kg DW (0.005 ± 0.003 mg/kg $DW_{1\%OC}$). It should be noted that this analysis was significantly affected by one sample (Palos Verdes) that had concentrations of DDTs that were more than an order of magnitude higher than those that were measured at other sites.

In a study commissioned by the Santa Monica Bay Restoration Project, Bay *et al.* (1994) collected sediments from 12 sites located in the vicinity of the Palos Verdes Peninsula during June and July, 1992. A station located near Dana Point in Orange County was used as a reference site in this study. This study was designed to evaluate the toxicity of Palos Verdes sediments to infaunal and epibenthic organisms, and included both bulk sediment and porewater bioassays. The toxicity of bulk sediments was assessed in acute (10-day) tests on the amphipod, *Rhepoxynius abronius*, and chronic (28-day and 35-day) tests on the amphipod, *Grandidierella japonica*, and the white sea urchin, *Lytechinus pictus*. As poor survival was observed among all of the treatment groups (including controls) in the amphipod (*G. japonica*) growth bioassay, these data were not considered for identifying sediment effect concentrations of DDTs. In this survey, the toxicity of porewater was evaluated in short-term (< 2-hour) bioassays using purple sea urchin, *Strongylocentrotus purpuratus*, gametes.

Acute toxicity bioassays were conducted using sediments from five of the 13 sites sampled near the Palos Verdes Peninsula in the 1992 survey; none of these sediments were acutely toxic to the amphipod, *Rhepoxynius abronius* (Bay *et al.*

1994). While the survival, gonad size, and diameter of white sea urchins were not affected by exposure to Palos Verdes sediments, the growth rate [as indicated by changes in wet weight (WW) during the test] of this species was reduced in certain Palos Verdes sediments. Relatively low growth rates (i.e., ≤ 0.002 g WW/day) were observed at sites with a mean concentration of SUM DDT of 0.762 ± 1.31 mg/kg DW (0.24 ± 0.427 mg/kg $DW_{1\%OC}$). The average SUM DDT concentrations were lower (0.117 ± 0.156 mg/kg DW or 0.048 ± 0.050 mg/kg $DW_{1\%OC}$) in the sediments that were not associated with effects on sea urchin growth (i.e., growth rates of ≥ 0.005 g WW/day).

Impaired fertilization of sea urchin, *Strongylocentrotus purpuratus*, gametes was also associated with elevated SUM DDT concentrations in Palos Verdes sediments (Bay *et al.* 1994). High fertilization rates (80.0 ± 10.2%) were observed when sea urchin gametes were exposed to porewater from sediments with low concentrations of SUM DDT (0.048 ± 0.066 mg/kg DW or 0.021 ± 0.022 mg/kg $DW_{1\%OC}$). The fertilization of sea urchin eggs was lower (9.4 ± 16.2%) in porewater from sediments with higher SUM DDT concentrations (0.749 ± 1.12 mg/kg DW or 0.244 ± 0.359 mg/kg $DW_{1\%OC}$; Bay *et al.* 1994).

In 1992, sediments from a number of locations in the vicinity of Los Angeles and Long Beach Harbors were sampled to assess sediment contamination and toxicity in San Pedro Bay (as part of the Bay Protection and Toxics Cleanup Program; Sapudar *et al.* 1994). The toxicity of each sample was evaluated using two toxicity tests, including 48-hour red abalone (*Haliotus rufescens*) larval development test (using pore water) and 10-day amphipod (*Rhepoxynius abronius*) survival test (using bulk sediments). The data from the porewater test on abalone larval development were not used in this evaluation because they provided little ability to discriminate between sampling sites (i.e., nearly 90% of the samples were shown to be toxic based on the results of this test). Toxic samples were identified as those in which amphipod survival was more than 20% lower than the average survival in the control treatments (Long *et al.* In review).

The results of this investigation indicated that sediments from several locations in San Pedro Bay were acutely toxic to amphipods, including Los Angeles Inner Harbor, Inner Fish Harbor, East Basin, and Alamitos Bay. The average concentration of SUM DDT in the toxic samples was 0.008 ± 0.013 mg/kg DW or 0.004 ± 0.004 mg/kg $DW_{1\%OC}$. By comparison, SUM DDT concentrations averaged 0.003 ± 0.004 mg/kg DW or 0.003 ± 0.003 mg/kg $DW_{1\%OC}$ at the non-toxic sites in this survey. Analysis of these results suggests that dry weight concentrations of SUM DDT were associated with the observed toxicity to

amphipods; however, there was little concordance between amphipod mortality and organic carbon-normalized SUM DDT concentrations.

In a related study, sediments were collected at several locations on the Palos Verdes Shelf and in Santa Monica Bay during 1992 and 1993 (Fairey 1997). The results of 10-day toxicity tests with the amphipod, *Rhepoxynius abronius*, indicated that the sediments from 11 of the 19 sites tested were acutely toxic (as indicated by a > 20% difference from control survival; Long *et al.* In review). The average concentration of SUM DDT at these sites was 0.091 ± 0.228 mg/kg DW or 0.079 ± 0.230 mg/kg $DW_{1\%OC}$. The average concentrations of SUM DDT were 0.042 ± 0.034 mg/kg DW or 0.023 ± 0.024 mg/kg $DW_{1\%OC}$ at the non-toxic sites in this survey. These results indicate that amphipod survival was linked with both dry weight- and organic carbon-normalized concentrations of SUM DDT. Due to the small number of sediments tested (4), the results of the polychaete survival and growth tests were not used in this evaluation.

A total of 350 stations in the vicinity of San Diego were also sampled between October, 1992 and May, 1994 under the Bay Protection and Toxics Cleanup Program (Fairey *et al.* 1996). The data for the sediment samples from the middle portion of San Diego Bay were included in the data set assembled for this investigation because substantial concentration gradients were observed for in several of the contaminants of concern. Several solid phase and porewater toxicity tests were conducted on each of the samples collected in this portion of the bay (i.e., the vicinity of the naval shipyards), including amphipod (*Rhepoxynius abronius*) survival, sea urchin (*Strongylocentrotus purpuratus*) fertilization, and sea urchin (*S. purpuratus*) larval development. The results of the polychaete growth and survival test, the bay mussel larval development test, and the red abalone larval development tests were not used due to incomplete designation of toxic samples. The results of the power analyses conducted by Long *et al.* (In review) were used to designate toxic samples for the amphipod and sea urchin tests (i.e., samples were designated as toxic if the response measured differed by more than 20% from that of the control samples).

Based on the results of the solid phase tests, sediments from the eastern portion of middle San Diego Bay were the most toxic to amphipods. The average concentration of SUM DDT at the toxic sites was 0.005 ± 0.020 mg/kg DW (0.009 ± 0.053 mg/kg $DW_{1\%OC}$). By comparison, the average concentration of SUM DDT in the samples that were not acutely toxic to amphipods was 0.003 ± 0.003 mg/kg DW (0.002 ± 0.004 mg/kg $DW_{1\%OC}$). Because dry weight-normalized SUM DDT concentrations were similar in the toxic and non-toxic samples, it is unlikely that this substance contributed significantly to amphipod mortality in the

middle portion of San Diego Bay. However, the linkage between amphipod survival and SUM DDT was stronger when organic carbon-normalized concentrations were considered.

The results of the sea urchin fertilization and larval development tests also provide relevant information for evaluating the toxic effects of SUM DDT. In samples from San Diego Bay, decreased the fertilization success of sea urchin gametes was observed in porewater from sediments that contained, on average, 0.008 ± 0.028 mg/kg DW (0.004 ± 0.013 mg/kg $DW_{1\%OC}$) of SUM DDT. Higher rates of abnormal development of sea urchin embryos were also observed in pore water from sediments that contained average SUM DDT concentrations of 0.008 ± 0.026 mg/kg DW (0.014 ± 0.074 mg/kg $DW_{1\%OC}$). SUM DDT were lower in the sediment samples that did not influence sea urchin fertilization or larval development.

3.2.3 SECs for SUM DDT

Using the spiked-sediment bioassay approach, a sediment effect concentration of 0.031 mg/kg DW or 0.111 mg/kg $DW_{1\%OC}$ was derived for SUM DDT (i.e., the sum of the concentrations of *p,p'*-DDT and *o,p'*-DDT).

Rationale: Data from three spiked-sediment toxicity investigations (McLeese and Metcalfe 1980; McLeese *et al.* 1982; Plesha *et al.* 1988) indicate that median lethal concentrations of SUM DDT, expressed on a dry weight basis, ranged from 0.031 to >16.5 mg/kg DW (Tables A4-1 and A4-2). At 1% OC, these LC_{50}s ranged from 0.111 to >8.25 mg/kg $DW_{1\%OC}$ (Table A4-3 and A4-4). While the DDT formulations used in these studies probably included a variety of substances besides *p,p'*-DDT and *o,p'*-DDT, these data were considered for identifying sediment effect concentrations of SUM DDT because these formulations most likely contained at least 92% of the SUM DDT.

The available spiked-sediment bioassay data include toxicological information on three sediment-dwelling invertebrate species (including two arthropod species) and, therefore, satisfy the minimum data requirements established in this study (see Appendix 3). In accordance with the procedures identified in Section 2.4, the lowest observed effect level (LOEL) for the most

sensitive species, from an acceptable spiked-sediment bioassay, was identified as the sediment effect concentration of SUM DDT (0.031 mg/kg DW or 0.111 mg/kg $DW_{1\%OC}$ for the sand shrimp, *Crangon septemspinosa*). The LOEL is from an acute toxicity study in which lethality was the endpoint measured; no information was available to convert it to a chronic LOEL (i.e., an acceptable acute to chronic ratio was not available). Therefore, it should be recognized that adverse biological effects could occur in the Southern California Bight at SUM DDT concentrations below the recommended SEC.

The results of the toxicity test on the sand shrimp are supported by data on the toxicity of SUM DDT to amphipods. Plesha *et al.* (1988) reported significant mortality (>20%) when amphipods, *Rhepoxynius abronius*, were exposed to 0.200 mg SUM DDT/kg DW (0.222 mg/kg $DW_{1\%OC}$) for a period of ten days. These sediments contained a mixture of other chlorinated hydrocarbons, which probably enhanced the toxicity of SUM DDT. Sediments in the Southern California Bight are known to contain elevated concentrations of several chlorinated hydrocarbons (Mearns *et al.* 1991). Therefore, the results reported by Plesha *et al.* (1988) support the SEC derived for SUM DDT.

3.2.4 Evaluation of the SECs for SUM DDT

Reliability:

The degree of confidence that can be placed on the SECs for SUM DDT was evaluated using matching sediment chemistry and biological effects data from the Southern California Bight. A total of seven studies provided the requisite information on SUM DDT concentrations, in conjunction with the results of laboratory toxicity tests or benthic invertebrate community assessments. These studies included Swartz *et al.* (1985), Anderson *et al.* (1988), Swartz *et al.* (1991), Bay *et al.* (1994), Sapudar *et al.* (1994), Fairey *et al.* (1996), and Fairey (1997).

The results of this evaluation indicate that the dry weight-normalized SEC for SUM DDT provides a reliable basis for classifying sediment

samples in the Southern California Bight (Table 2). Of the 50 sediment samples with SUM DDT concentrations at or above the SEC, 38 (76%) were found to be toxic, as defined in Section 2.5. Therefore, there is a high probability of observing adverse effects on sediment-dwelling organisms when SUM DDT concentrations exceed the SEC.

The probability of observing adverse biological effects increased at higher concentrations of SUM DDT. For example, 85% of the sediment samples (33 of 39 samples) with SUM DDT concentrations at or above 0.06 mg/kg DW were found to be toxic. The incidence of adverse effects increased to 88% (21 of 24 samples) when SUM DDT concentrations equaled or exceeded 0.20 mg/kg DW. The increasing incidence of toxicity with increasing concentrations of SUM DDT indicates that a dose-response relationship exists for this substance in Southern California Bight sediments. The presence of such a relationship increases the confidence that can be placed in the SECs.

Using the data that were compiled in this study, it is apparent that the organic-carbon normalized SEC also provides a reliable basis for classifying sediments in the Southern California Bight (Table 3). The probability of observing adverse biological effects (considering a variety of species, life stages, and endpoints) when concentrations of SUM DDT equaled or exceeded 0.111 mg/kg $DW_{1\%OC}$ is approximately 91% (20 of 22 sediment samples).

There is a lower probability of observing adverse effects at lower concentrations of SUM DDT. For example, the incidence of toxic effects was roughly 73% (32 of 44 samples) when SUM DDT concentrations equaled or exceeded 0.02 mg/kg $DW_{1\%OC}$. The probability of observing toxic effects increased to 87% at SUM DDT at or above 0.06 mg/kg $DW_{1\%OC}$. Once again, the increasing incidence of effects with increasing concentrations of SUM DDT enhances the confidence that can be placed in the SECs.

Predictability

Matching sediment chemistry and biological effects data were compiled from a total of five studies to evaluate the predictability of the SECs

Table 2. An evaluation of the reliability of the dry weight-normalized SECs for SUM DDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.031 mg/kg DW	Swartz et al. 1985	7	3	3	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	24	17	70.8
	Bay et al. 1994	13	7	6	85.7
	Sapudar et al. 1994	103	5	5	100.0
	Fairey et al. 1996	85	2	2	100.0
	Fairey 1997	19	8	4	50.0
	Overall	267	50	38	76.0
0.060 mg/kg DW	Swartz et al. 1985	7	3	3	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	20	17	85.0
	Bay et al. 1994	13	7	6	85.7
	Sapudar et al. 1994	103	1	1	100.0
	Fairey et al. 1996	85	2	2	100.0
	Fairey 1997	19	5	3	60.0
	Overall	267	39	33	84.6
0.200 mg/kg DW	Swartz et al. 1985	7	0	0	NA
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	19	16	84.2
	Bay et al. 1994	13	3	3	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	1	1	100.0
	Overall	267	24	21	87.5

Table 3. An evaluation of the reliability of the organic carbon-normalized SECs for SUM DDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.02 mg/kg DW (1% OC)	Swartz et al. 1985	7	2	2	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	23	17	73.9
	Bay et al. 1994	13	9	6	66.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	4	4	100.0
	Fairey 1997	19	5	2	40.0
	Overall	267	44	32	72.7
0.060 mg/kg DW (1% OC)	Swartz et al. 1985	7	0	0	NA
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	19	17	89.5
	Bay et al. 1994	13	5	4	80.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	4	4	100.0
	Fairey 1997	19	2	1	50.0
	Overall	267	31	27	87.1
0.111 mg/kg DW (1% OC)	Swartz et al. 1985	7	0	0	NA
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	17	15	88.2
	Bay et al. 1994	13	2	2	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	2	2	100.0
	Fairey 1997	19	1	1	100.0
	Overall	267	22	20	90.9

for SUM DDT (Munns *et al.* 1991; Swartz et al. 1994; Long *et al.* 1994; Long *et al.* 1995b; Long 1997). All of these data applied to marine and estuarine sediments from outside the Southern California Bight (Table A5-1). Using these data, the dry weight-normalized SEC was found to accurately predict toxicity in sediments with elevated levels of SUM DDT (Table 4). Thirty-six of the 44 sediment samples (82%) with SUM DDT concentrations at or above 0.031 mg/kg DW were found to be toxic. The incidence of adverse biological effects was similar when SUM DDT concentrations equaled or exceeded 0.06 mg/kg DW (i.e., 82%; 23 of 28 samples) or 0.20 mg/kg DW (i.e., 85%; 11 of 13 samples).

Based on the results of this evaluation, the organic carbon-normalized SEC was as predictive of toxicity as the dry-weight normalized SEC (Table 5). The incidence of toxic effects was 82% (9 of 11 samples) when SUM DDT concentrations equaled or exceeded 0.111 mg/kg $DW_{1\%\ OC}$ in marine and estuarine sediments. The probability of observing toxic effects was lower when SUM DDT concentrations equaled or exceeded 0.02 mg/kg $DW_{1\%OC}$ (79%; 22 of 28 samples were toxic) or 0.06 mg/kg $DW_{1\%OC}$ (75%; 9 of 12 samples were toxic).

While no directly applicable to marine and estuarine sediments, the results of a study conducted in a freshwater system that was contaminated by DDTs provides additional insight into the predictability of the SECs for SUM DDT. In 1991, Hoke *et al.* (1994) evaluated the acute toxicity of field-collected sediments from the Huntsville Spring Branch-Indian Creek system in Alabama. In this study, the toxicity of sediments collected nearby the Redstone Army Arsenal was evaluated in 10-day toxicity tests using amphipods (*Hyalella azteca*). The concentrations of SUM DDT ranged from below detection limits (BDL) to 147 mg/kg DW in the 15 sediment samples that were collected. The SEC of 0.031 mg/kg DW for SUM DDT was exceeded in 13 of the 15 sediment samples. The results of the toxicity tests indicated that 12 of these samples were toxic to amphipods (statistical analyses of the mortality data were not performed by these investigators; samples with mortality of >20% were defined as toxic in this evaluation of the predictability of the SECs). Therefore, the predictability of the DW-normalized SEC for SUM DDT was calculated to be 92.3%. All of the samples that were

Table 4. An independent evaluation of the predictability of the dry weight-normalized SECs for SUM DDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.031 mg/kg DW	Munns et al. 1991	29	2	0	0.0
	Swartz et al. 1994	9	7	4	57.1
	Long et al. 1994	61	8	8	100.0
	Long et al. 1995	50	20	18	90.0
	Long 1997	105	7	6	85.7
	Overall	254	44	36	81.8
0.060 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	7	4	57.1
	Long et al. 1994	61	5	5	100.0
	Long et al. 1995	50	12	11	91.7
	Long 1997	105	4	3	75.0
	Overall	254	28	23	82.1
0.200 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	2	2	100.0
	Long et al. 1995	50	5	5	100.0
	Long 1997	105	1	0	0.0
	Overall	254	13	11	84.6

Table 5. An independent evaluation of the predictability of the organic carbon-normalized SECs for SUM DDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.02 mg/kg DW (1% OC)	Swartz et al. 1994	9	7	4	57.1
	Long et al. 1994	61	4	4	100.0
	Long et al. 1995	50	12	10	83.3
	Long 1997	105	5	4	80.0
	Overall	216	28	22	78.6
0.060 mg/kg DW (1% OC)	Swartz et al. 1994	9	6	4	66.7
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	4	4	100.0
	Long 1997	105	1	0	0.0
	Overall	216	12	9	75.0
0.111 mg/kg DW (1% OC)	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	4	4	100.0
	Long 1997	105	1	0	0.0
	Overall	216	11	9	81.8

predicted to be toxic using the OC-normalized SEC were toxic to amphipods (12 of 12 samples; predictability = 100%).

3.3 Toxic Effects of SUM DDE

3.3.1 Spiked-Sediment Bioassay Data

Only one controlled laboratory study (i.e., spiked-sediment bioassay) was located on the effects of sediment-associated SUM DDE on marine organisms. Bay *et al.* (1994) spiked sediments that were collected from a reference site (which was located near Dana Point in Orange County, California), with *p,p'*-DDE at concentrations ranging from 0 to 8.7 mg/kg DW. After an equilibration period of two days, sea urchins (*Lytechinus pictus*) were exposed to test sediments for a period of 35 days. While this study was designed explicitly as a bioaccumulation test, survival and growth were also monitored to provide data on the biological effects of this substance. No dose related effects in the survival, growth, or gonad weight of sea urchins exposed to DDE-spiked sediments were reported at any DDE concentration. Therefore, the LC_{50} and EC_{50}s (for further information see glossary; Appendix 2) of DDE for this species were all greater than 8.7 mg/kg DW. As the level of OC in these reference sediments was 1%, the OC-normalized concentrations of DDE at 1%OC were the same as the dry weight concentrations of DDE in this study.

3.3.2 Field Data

The results of nine field surveys provide data for evaluating the effects of SUM DDE in the Southern California Bight. In six of these studies, the concentrations of both DDE isomers were measured, which facilitated the calculation of SUM DDE concentrations. In the other three studies, only the concentrations of *p,p'*-DDE were measured; these data were converted to SUM DDE concentrations (as outlined in Section 3.1).

In the 1980 study conducted in the vicinity of the Los Angeles County Sanitation District's sewage outfalls, Swartz *et al.* (1985) examined sediment toxicity, contamination, and benthic macroinvertebrate community structure at seven sites on the Palos Verdes Shelf. The results of this study indicated that the sediments

from three sites were toxic to amphipods during 10-day exposures. Evaluation of a number of metrics (e.g., invertebrate biomass and infaunal index) indicated that the benthic macroinvertebrate community structure had also been altered at these sites. Sediments from the other four sites were not toxic. At the toxic sites, the concentration of SUM DDE averaged 5.82 ± 1.21 mg/kg DW or 1.47 ± 0.38 mg/kg $DW_{1\%OC}$. The mean concentration of this substance at the non-toxic sites was 2.91 ± 4.01 mg/kg DW or 0.852 ± 1.00 mg/kg $DW_{1\%OC}$.

In a follow-up survey conducted in 1983, Swartz *et al.* (1986) collected sediment and macrobenthos samples from a total of nine sites on the Palos Verdes Shelf and Santa Monica Bay. Matching sediment chemistry and biological effects (including acute toxicity bioassays and benthic invertebrate community structure) data were reported for six of these sites. These stations were considered to be located at the same sites as those sampled in the 1980 survey. The results of 10-day tests on the amphipod, *Rhepoxynius abronius*, indicated that the sediments collected from only one station (#6) were acutely toxic (Ferraro *et al.* 1991). The concentration of SUM DDE at this station was 3.15 mg/kg DW or 1.31 mg/kg $DW_{1\%OC}$. The mean concentration of SUM DDE at the non-toxic sites was 4.66 ± 3.83 mg/kg DW or 1.60 ± 1.14 mg/kg $DW_{1\%OC}$. The lack of concordance between the amphipod survival and SUM DDE concentrations indicates that this substance was not responsible for observed biological effects.

In the 1983 study, benthic invertebrate community structure was altered at several sites with elevated levels of SUM DDE. For example, low echinoderm densities were observed at sites with mean concentrations of SUM DDE of 5.26 ± 3.10 or 1.85 ± 0.83 mg/kg $DW_{1\%OC}$. In contrast, high densities of echinoderms were observed at a site with 0.124 mg SUM DDE/kg DW or 0.095 mg SUM DDE/kg $DW_{1\%OC}$. Similarly, low amphipod densities were observed at sites with mean concentrations of SUM DDE of 7.33 ± 1.51 mg/kg DW or 2.36 ± 0.59 mg/kg $DW_{1\%OC}$. Higher densities of amphipods were observed at sites with 1.49 ± 1.53 mg DDE/kg DW or 0.752 ± 0.614 mg/kg $DW_{1\%OC}$. Similar results were observed when total benthic invertebrate density, species richness, and infaunal index were considered.

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes shelf and Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10 day) toxicity tests using amphipods (*Rhepoxynius abronius*) were conducted on each of the core sections. The results of this investigation indicates that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The

mean concentration of SUM DDE in these sediment samples was 123 ± 45.5 mg/kg DW or 14.6 ± 2.88 mg/kg $DW_{1\%OC}$. Moderate toxicity to amphipods (35.9 ± 12.1% mortality) was observed in shallower and deeper sediments (2.5 to 12.5 cm and 37.5 to 47.5 cm); these samples had, on average, 19.1 ± 15.4 mg/kg DW (3.45 ± 2.34 mg/kg $DW_{1\%OC}$) of SUM DDE. The samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average SUM DDE concentrations of 1.44 ± 3.54 mg/kg DW (0.440 ± 0.596 mg/kg $DW_{1\%OC}$).

One year later (1986), another follow-up survey was conducted to evaluate trends in sediment contamination, toxicity, and benthic macroinvertebrate community structure on the Palos Verdes Shelf and Santa Monica Bay (Ferraro *et al.* 1991). A total of eight sites were sampled in this survey; none of the sediments tested were acutely toxic to the amphipod, *Rhepoxynius abronius*, during 10-day exposures. The mean concentration of DDE at the eight sites was 6.16 ± 4.16 mg/kg DW or 1.63 ± 0.82 mg/kg $DW_{1\%OC}$.

Data on benthic invertebrate community structure, which reflects continuous exposure to sediment-associated contaminants, indicates that adverse effects on sediment-dwelling organisms occur at elevated levels of SUM DDE (Ferraro *et al.* 1991). For example, low densities of amphipods (4.1 ± 2.5/0.1 m^2) were observed at three of the eight sites sampled on the Palos Verdes Shelf. At these sites, concentrations of SUM DDE averaged 9.47 ± 4.04 mg/kg DW or 2.36 ± 0.22 mg/kg $DW_{1\%OC}$. Higher amphipod (54.6 ± 5.1/0.1 m^2) densities were observed at the sites with lower concentrations of SUM DDE (0.976 ± 1.09 mg/kg DW or 0.439 ± 0.262 mg/kg $DW_{1\%OC}$). Similarly, low echinoderms densities (2.4 ± 5.7/0.1 m^2) were observed at seven of the eight sites sampled during this survey, with DDE concentrations averaging 7.02 ± 3.67 mg/kg DW or 1.82 ± 0.65 mg/kg $DW_{1\%OC}$. Echinoderm densities (208/0.1 m^2) were higher at the site with lower concentrations of SUM DDE (0.203 mg/kg DW or 0.254 mg/kg $DW_{1\%OC}$). Species richness, total benthic invertebrate density, and total benthic invertebrate biomass were not correlated with SUM DDE concentrations.

Matching sediment chemistry and biological effects data were collected at seven sites in the Southern California Bight during 1987 (Anderson *et al.* 1988). While this study was conducted primarily to evaluate the effects associated with PAH-contaminated sediments, it also provides some information for assessing the potential effects of mixtures of SUM DDE and other substances. In this study, the survival and reburial of the amphipod, *Grandidierella japonica*, (in 10-day acute toxicity tests) were not linked with concentrations of SUM DDE. The mean concentration of SUM DDE was 0.078 ± 0.081 mg/kg DW or 0.027 ± 0.027 mg/kg $DW_{1\%OC}$ in sediments that were significantly toxic to this species. By

comparison, the mean concentration of SUM DDE in sediments that did not affect survival was 0.861 ± 2.07 mg/kg DW or 0.212 ± 0.494 mg/kg $DW_{1\%OC}$. As the concentration of this substance was lower at the toxic sites than it was at the non-toxic sites, factors other than SUM DDE must have been responsible for the observed effects.

The survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, (in 35-day chronic exposure tests) were not correlated with the concentrations of sediment-associated SUM DDE measured in Palos Verdes sediments (i.e., there was no concordance between sediment chemistry and the biological effects; Anderson *et al.* 1988). However, sea urchin growth was correlated with concentrations of DDTs, with reduced growth rates observed in sediments with mean concentrations of 2.62 ± 3.47 mg/kg DW or 0.618 ± 0.852 mg/kg $DW_{1\%OC}$ of SUM DDE. It should be noted that this analysis was significantly affected by one sample (Palos Verdes) that had concentrations of DDEs that were more than an order of magnitude higher than those that were measured at other sites.

In a study commissioned by the Santa Monica Bay Restoration Project, Bay *et al.* (1994) collected sediments from 12 sites located in the vicinity of the Palos Verdes Peninsula and one reference site near Dana Point. Acute toxicity bioassays were conducted using sediments from five of the 13 sites sampled in the 1992 survey; none on these sediments were toxic to amphipods (*Rhepoxynius abronius*; Bay *et al.* 1994). The mean concentrations of SUM DDE at these sites were 5.53 ± 4.66 mg/kg DW or 2.18 ± 1.41 mg/kg $DW_{1\%OC}$. Similarly, the survival, avoidance behavior, gonad growth and growth (as indicated by diameter) of white sea urchins (*Lytechinus pictus*) were not affected at mean SUM DDE concentrations of 4.77 ± 4.66 mg/kg DW or 1.69 ± 1.18 mg/kg $DW_{1\%OC}$.

Unlike the other bioassay endpoints, the growth rate (as measured by wet weight change over a 35-day exposure period) of white sea urchins was influenced by exposure to Palos Verdes sediments (Bay *et al.* 1994). Reduced growth rates were observed at sites with mean concentrations of SUM DDE of 8.73 ± 5.49 mg/kg DW or 2.50 ± 1.34 mg/kg $DW_{1\%OC}$. The concentrations of this substance were lower in the sediments that were not associated with growth effects (3.02 ± 3.15 mg/kg DW or 1.33 ± 0.962 mg/kg $DW_{1\%OC}$. Similar results were obtained when the fertilization of sea urchin, *Strongylocentrotus purpuratus*, gametes was the experimental endpoint considered in the bioassay. The results of this short-term (< 2 hours) test indicated that low fertilization rates (9.4 ± 16.2%) were associated with exposure to porewater from sediments that had mean SUM DDE concentrations of 8.41 ± 4.19 mg/kg DW or 2.56 ± 0.935 mg/kg $DW_{1\%OC}$.

Exposure to porewater from sediments with 2.80 ± 3.60 mg SUM DDE/kg DW or 1.21 ± 1.05 mg SUM DDE/kg $DW_{1\%OC}$ was associated with higher fertilization rates.

As part of the Bay Protection and Toxics Cleanup Program, sediment samples were collected from a number of locations in the vicinity of Los Angeles and Long Beach Harbours in 1992 (i.e., San Pedro Bay; Sapudar *et al.* 1994). The toxicity of each sample was evaluated using two toxicity tests, including 48-hour red abalone (*Haliotus rufescens*) larval development test (using pore water) and 10-day amphipod (*Rhepoxynius abronius*) survival test (using bulk sediments). The data on the effects of pore water on abalone larval development were not used in this evaluation because they provided little ability to discriminate between sampling sites (i.e., nearly 90% of the samples were shown to be toxicity based on the results of this test). Toxicity to amphipods was designated based on comparisons to control survival, using the minimum significant difference reported by Long *et al.* (In review).

The results of the Sapudar *et al.* (1994) study indicate that the sediments from Los Angeles Inner Harbour, Inner Fish Harbour, East Basin, and Alamitos Bay were found to be the most toxic to amphipods. The average concentrations of SUM DDE in the toxic samples were 0.124 ± 0.103 mg/kg DW or 0.066 ± 0.059 mg/kg $DW_{1\%OC}$. By comparison, SUM DDE concentrations averaged 0.097 ± 0.077 mg/kg DW or 0.100 ± 0.109 mg/kg $DW_{1\%OC}$ in sediments from the non-toxic sites. The limited concordance between amphipod survival and SUM DDE concentrations indicates that SUM DDE probably contributed minimally to the observed toxicity.

During 1992 and 1993, sediments were also collected at several locations on the Palos Verdes Shelf and in Santa Monica Bay (Fairey 1997). Two toxicity tests were conducted during this investigation, including a 10-day acute lethality test with the amphipod, *Rhepoxynius abronius*; and, a 20-day lethality and growth test with the polychaete, *Neanthes arenaceodentata*. However, the results of the polychaete test were not used in this evaluation because only four sediment samples were tested. Of the 19 sites for which matching sediment chemistry and toxicity data were reported, sediments from 11 sites were found to be acutely toxic to amphipods. The average concentrations of SUM DDE at the toxic sites were 0.485 ± 0.991 mg/kg DW or 0.324 ± 0.648 mg/kg $DW_{1\%OC}$. By comparison, SUM DDE concentrations averaged 2.68 ± 0.516 mg/kg DW or 1.63 ± 1.02 mg/kg $DW_{1\%OC}$ in the sediments from the non-toxic sites. The lack of concordance between amphipod survival and SUM DDE concentrations indicates that this group of substances was not responsible for the observed biological effects.

Between October, 1992 and May, 1994, sediment samples were also collected at a total of 350 stations in the vicinity of San Diego under the Bay Protection and Toxic Cleanup Program (Fairey *et al.* 1996). The sediment samples from the middle portion of San Diego Bay (i.e., the vicinity of the naval shipyards) were included in the data set assembled for this investigation because substantial concentration gradients in several of the contaminants of concern were observed. Effects on several marine organisms and endpoints were examined in this study, including amphipod (*Rhepoxynius abronius*) survival, sea urchin (*Strongylocentrotus purpuratus*) fertilization, and sea urchin (*S. purpuratus*) larval development. The results of the polychaete growth and survival test, the bay mussel larval development test, and the red abalone larval development tests were not used due to incomplete designation of toxic samples. The results of the power analyses conducted by Long *et al.* (In review) were used to designate toxic samples for the amphipod and sea urchin tests (i.e., samples were designated as toxic if the response measured differed by more than 20% from that of the control samples).

Based on the measured toxicity to amphipods, sediments from the eastern portion of middle San Diego Bay were the most toxic. The average concentration of SUM DDE at the toxic sites was 0.010 ± 0.013 mg/kg DW (0.009 ± 0.0223 mg/kg $DW_{1\%OC}$). By comparison, the average concentration of SUM DDE in the samples that were not acutely toxic to amphipods was 0.014 ± 0.017 mg/kg DW (0.007 ± 0.007 mg/kg $DW_{1\%OC}$). Because SUM DDE concentrations in the toxic samples were similar to or lower than they were in the non-toxic samples, it is unlikely that SUM DDE contributed significantly to amphipod mortality in the middle portion of San Diego Bay. Similarly, little concordance was observed between sea urchin fertilization or embryo development and the concentrations of SUM DDE in sediment samples from middle San Diego Bay.

3.3.3 SECs for SUM DDE

Using the weight-of-evidence approach, SECs of 6.58 mg/kg DW and 1.82 mg/kg $DW_{1\%OC}$ were derived for SUM DDE (i.e., the sum of the concentrations of *p,p'*-DDE and *o,p'*-DDE).

Rationale: Data from only one spiked-sediment toxicity test (Bay *et al.* 1994) were available to evaluate the toxicity of sediment-associated SUM DDE. The results of this study indicated that SUM DDE was not toxic to the sea urchin, *Lytechinus pictus*, at

concentrations as high as 8.7 mg/kg DW (8.7 mg/kg $DW_{1\%OC}$). As no toxicological data were available on a sediment-dwelling arthropod species, a threshold concentration was not calculated using the spiked-sediment bioassay approach. Hence, the threshold concentration of SUM DDE was derived using the weight-of-evidence approach.

The summarized and unsummarized toxicological data sets for SUM DDE (mg/kg DW; Tables A4-5 and A4-6) contained a total of 23 and 108 effects data records (hits; designated by an *), respectively. Evaluation of these data sets, using the procedures described in Section 2.4, resulted in the calculation of ER-Ms (effects range median) of 6.58 and 7.07 mg/kg DW, respectively, for SUM DDE. The lower of these two values was identified as the SEC.

Similarly, the summarized and unsummarized toxicological data sets for SUM DDE (Tables A4-7 and A4-8), expressed on an OC-normalized basis (mg/kg $DW_{1\%OC}$) contained 17 and 88 effects data entries, respectively. The ER-Ms derived from these two data sets were 1.82 and 2.21 mg/kg $DW_{1\%OC}$, respectively. The lower of these two values was identified as the SEC.

3.3.4 *Evaluation of the SECs for SUM DDE*

Reliability:

Matching sediment chemistry and biological effects data were compiled from nine studies that were conducted in the Southern California Bight, including Swartz *et al.* (1985), Swartz *et al.* (1986), Anderson *et al.* (1988), Ferraro *et al.* (1991), Swartz *et al.* (1991), Bay *et al.* (1994), Sapudar *et al.* (1994), Fairey *et al.* (1996), and Fairey (1997). Both the dry weight-normalized and organic carbon-normalized SECs were evaluated using the procedures described in Section 2.5. The results of this evaluation indicate that the SECs for SUM DDE are generally reliable.

Based on the results of this assessment, the dry-weight normalized SEC can be used to reliably classify sediments from the Southern

California Bight (Table 6). Of the 29 sediment samples with concentrations of SUM DDE at or in excess of 6.58 mg/kg DW, 97% (28 samples) were found to be toxic. A lower value (2.00 mg/kg DW) was less reliable than the recommended SEC, as indicated by the incidence of toxic effects at or above this concentration (79.2%; 38 of 48 samples). In contrast, the reliability of a higher value of SUM DDE (10.0 mg/kg DW) was similar to that of the recommended SEC (94% correct classification; 17 of 18 samples).

The organic carbon-normalized SEC also provided a reliable basis for classifying sediment samples with elevated levels of SUM DDE in the Southern California Bight (Table 7). Based on the results of laboratory toxicity tests and benthic invertebrate community assessments, 87% (26 of 30) samples of the sediment samples with SUM DDE concentrations at or in excess of 1.82 mg/kg $DW_{1\% OC}$ were toxic to seidment-dwelling organisms. A lower value (0.50 mg/kg $DW_{1\% OC}$) was found to be less reliable, as indicated by the relatively lower incidence of toxic effects (68%; 41 of 60 samples) that was observed when SUM DDE concentrations equaled or exceeded this level. In contrast, a high incidence of toxic effects (100%; 14 of 14 samples) was observed when SUM DDE concentrations equaled or exceeded 3.00 mg/kg $DW_{1\% OC}$. Confidence in both SECs is enhanced by the concordance between the concentrations of SUM DDE and the incidence of toxic effects.

Predictability:

The predictability of the SECs for SUM DDE was evaluated using an independent data set, which was comprised of matching sediment chemistry and biological effects data for 254 sampling sites located outside the Southern California Bight. These studies were conducted in Narragansett Bay (Munns *et al.* 1991), San Francisco Bay (Swartz *et al.* 1994), Tampa Bay (Long *et al.* 1994), Hudson-Raritan Estuary (Long *et al.* 1995b), and Biscayne Bay (Long 1997; Table A5-2). None of the sediment samples from these marine and estuarine sites had concentrations of SUM DDE that equaled or exceeded the SECs (Tables 8 and 9). Therefore, it was not possible to evaluate the predictability of the SECs for SUM DDE. However, data from one freshwater site provide some relevant information for evaluating the predictability of the SECs.

Table 6. An evaluation of the reliability of the dry weight-normalized SECs for SUM DDE.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
2.00 mg/kg DW	Swartz et al. 1985	7	4	4	100.0
	Swartz et al. 1986	6	4	4	100.0
	Anderson et al. 1988	9	1	1	100.0
	Ferraro et al. 1991	8	6	6	100.0
	Swartz et al. 1991	31	17	16	94.1
	Bay et al. 1994	13	8	6	75.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	8	1	12.5
	Overall	281	48	38	79.2
6.58 mg/kg DW	Swartz et al. 1985	7	2	2	100.0
	Swartz et al. 1986	6	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	4	4	100.0
	Swartz et al. 1991	31	16	15	93.8
	Bay et al. 1994	13	5	5	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	281	29	28	96.6
10.0 mg/kg DW	Swartz et al. 1985	7	0	0	NA
	Swartz et al. 1986	6	0	0	NA
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	1	1	100.0
	Swartz et al. 1991	31	15	14	93.3
	Bay et al. 1994	13	2	2	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	281	18	17	94.4

Table 7. An evaluation of the reliability of the organic carbon-normalized SECs for SUM DDE.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.50 mg/kg DW (1% OC)	Swartz et al. 1985	7	5	4	80.0
	Swartz et al. 1986	6	5	4	80.0
	Anderson et al. 1988	9	1	1	100.0
	Ferraro et al. 1991	8	7	6	85.7
	Swartz et al. 1991	31	21	17	81.0
	Bay et al. 1994	13	11	7	63.6
	Sapudar et al. 1994	103	1	0	0.0
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	9	2	22.2
	Overall	281	60	41	68.3
1.82 mg/kg DW (1% OC)	Swartz et al. 1985	7	1	1	100.0
	Swartz et al. 1986	6	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	4	4	100.0
	Swartz et al. 1991	31	16	15	93.8
	Bay et al. 1994	13	6	4	66.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	1	0	0.0
	Overall	281	30	26	86.7
3.00 mg/kg DW (1% OC)	Swartz et al. 1985	7	0	0	NA
	Swartz et al. 1986	6	0	0	NA
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	0	0	NA
	Swartz et al. 1991	31	13	13	100.0
	Bay et al. 1994	13	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	281	14	14	100.0

Table 8. An independent evaluation of the predictability of the dry weight-normalized SECs for SUM DDE.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
2.00 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	0	0	NA
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	0	0	NA
6.58 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	0	0	NA
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	0	0	NA
10.0 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	0	0	NA
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	0	0	NA

Table 9. An independent evaluation of the predictability of the organic carbon-normalized SECs for SUM DDE.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.50 mg/kg DW (1% OC)	Swartz et al. 1994	9	4	4	100.0
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	216	0	0	100.0
1.82 mg/kg DW (1% OC)	Swartz et al. 1994	9	0	0	NA
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	216	0	0	NA
3.00 mg/kg DW (1% OC)	Swartz et al. 1994	9	0	0	NA
	Long et al. 1994	61	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	216	0	0	NA

Toxic samples were identified based on toxicity to amphipods, impaired fertilization of sea urchin gametes, or reduced amphipod abundance.

In 1991, Hoke *et al.* (1994) evaluated the acute toxicity of field-collected sediments from the Huntsville Spring Branch-Indian Creek system in Alabama. In this study, the toxicity of sediment samples collected nearby the Redstone Army Arsenal were evaluated using 10-day amphipod (*Hyalella azteca*) toxicity tests. The concentrations of SUM DDE ranged from below detection limits (BDL) to 12.2 mg/kg DW in the 15 samples collected in this investigation. The DW-normalized SEC of 6.58 mg/kg DW for SUM DDE was exceeded in three of the 15 sediment samples; hence, three samples were predicted to be toxic. The results of the toxicity tests that were performed indicated that all three samples were toxic to amphipods (statistical analyses of the mortality data were not performed by these investigators; samples with mortality of >20% were defined as toxic in this evaluation of the predictability of the SECs). All four of the samples that were predicted to be toxic using the OC-normalized SEC (1.68 mg/kg $DW_{1\%OC}$) were toxic to amphipods. Therefore, the predictability of both the DW-normalized and OC-normalized SECs for SUM DDE was calculated to be 100%.

3.4 Toxic Effects of SUM DDD

3.4.1 Spiked-Sediment Bioassay Data

No spiked-sediment bioassay data were located on the toxic effects of sediment-associated SUM DDD on marine organisms.

3.4.2 Field Data

In 1980, Swartz *et al.* (1985) examined sediment toxicity, contamination, and benthic macroinvertebrate community structure at seven sites in the vicinity of the Los Angeles County Sanitation District's sewage outfalls on the Palos Verdes Shelf. The results of this study indicated that the sediments from three stations (1, 2, and 3) were acutely toxic to amphipods, *Rhepoxynius abronius*, during 10-day tests. These investigators reported that benthic macroinvertebrate community structure (as indicated by infaunal index and invertebrate biomass) at these sites differed from that of the reference sites. The average concentration of SUM

DDD at the stations 1, 2, and 3 was 1.26 ± 0.769 mg/kg DW or 0.321 ± 0.173 mg/kg $DW_{1\%OC}$. The average concentration of this substance at the non-toxic stations (4, 5, 6, and 7) was 0.282 ± 0.411 mg/kg DW or 0.082 ± 0.105 mg/kg $DW_{1\%OC}$. Phoxocephalid amphipods were absent from stations that had average SUM DDD concentrations of 1.17 ± 0.574 mg/kg DW (0.299 ± 0.147 mg/kg $DW_{1\%OC}$), while this family was represented at stations that had 0.078 ± 0.075 mg SUM DDD/kg DW (0.032 ± 0.035 mg/kg $DW_{1\%OC}$).

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes Shelf and Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10 day) toxicity tests using amphipods (*Rhepoxynius abronius*) were conducted on each of the core sections. The results of this investigation indicated that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The average concentration of SUM DDD in these samples was 14.5 ± 6.34 mg/kg DW or 1.82 ± 0.829 mg/kg $DW_{1\%OC}$. Higher survival rates (35.9 ± 12.1% mortality) were observed in shallower and deeper sediments (2.5 - 12.5 cm and 37.5 to 47.5 cm); these samples had, on average, 3.00 ± 3.59 mg/kg DW (0.672 ± 0.587 mg/kg $DW_{1\%OC}$) of SUM DDD. The samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average SUM DDD concentrations of 0.303 ± 0.808 mg/kg DW (0.085 ± 0.155 mg/kg $DW_{1\%OC}$).

Matching sediment chemistry and biological effects data collected at seven sites in the vicinity of Palos Verdes in 1987 provide additional information for evaluating the potentially toxic effects of SUM DDD (Anderson *et al.* 1988). The results of the 10-day acute toxicity tests conducted in this study indicated that the survival and reburial of the amphipod, *Grandidierella japonica*, were not linked with concentrations of SUM DDD in Southern California Bight sediments (Anderson *et al.* 1988). The mean concentration of SUM DDD was 0.022 ± 0.017 mg/kg DW (0.013 ± 0.016 mg/kg $DW_{1\%OC}$) in sediments that were significantly toxic to this species. By comparison, the mean concentration of SUM DDD in sediments that did not affect amphipod survival was 0.106 ± 0.239 mg/kg DW (0.028 ± 0.056 mg/kg $DW_{1\%OC}$). As the concentrations of these substances were lower at the toxic sites than they were at the non-toxic sites, something other than the concentrations of SUM DDD must have been responsible for the observed effects.

The results of the 35-day chronic toxicity tests conducted by Anderson *et al.* (1988) indicate that the survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, were not linked to the

concentrations of sediment-associated DDDs (i.e., there was no concordance between the sediment chemistry and the biological effects). However, sea urchin growth (as measured by increases in weight or test width) was linked to concentrations of SUM DDDs, with reduced growth rates observed in sediments with a mean concentration of 0.310 ± 0.400 mg SUM DDD/kg DW (0.073 ± 0.099 mg/kg $DW_{1\%OC}$). No effects on growth were evident at the sites with an average concentration of 0.012 ± 0.014 mg SUM DDD/kg DW (0.009 ± 0.010 mg/kg $DW_{1\%OC}$). It should be noted that this analysis was significantly affected by one sample (Palos Verdes) that had concentrations of DDDs that were more than an order of magnitude higher than those that were measured at other sites.

In a study commissioned by the Santa Monica Bay Restoration Project, Bay *et al.* (1994) collected sediments from 12 sites located in the vicinity of the Palos Verdes Peninsula and a reference site near Dana Point. Acute toxicity bioassays were conducted using sediments from five of the 13 sites sampled in the 1992 survey; none of these sediments were acutely toxic to the amphipod, *Rhepoxynius abronius* (Bay *et al.* 1994). The mean concentration of SUM DDD at these stations was 0.893 ± 1.38 mg/kg DW (0.312 ± 0.431 mg/kg $DW_{1\%OC}$).

The survival, avoidance behavior, gonad size, and diameter of white sea urchins were not affected by exposure to Palos Verdes sediments (Bay *et al.* 1994). The mean concentration of SUM DDD at the 13 sites was 0.517 ± 0.883 mg/kg DW or 0.181 ± 0.276 mg/kg $DW_{1\%OC}$. However, the growth rate (as measured by WW change during the test) of this species was influenced by exposure to certain Palos Verdes sediments. Specifically, relatively low growth rates (i.e., ≤ 0.08 g WW/35 days) were observed at sites with a mean concentration of SUM DDD of 1.17 ± 1.47 mg/kg DW or 0.357 ± 0.481 mg/kg $DW_{1\%OC}$. The average concentrations of this substance were lower (0.227 ± 0.236 mg/kg DW or 0.103 ± 0.075 mg/kg $DW_{1\%OC}$) in the sediments that were not associated with growth effects (i.e. growth rates of ≥ 0.17 g WW/35 days). Similarly, the fertilization of sea urchin (*Strongylocentrotus purpuratus*) gametes was also linked to sediment SUM DDD concentrations, with low fertilization (9.4 ± 16.2%) observed in porewater from sediments with, on average, 1.10 ± 1.26 mg/kg DW (0.353 ± 0.402 mg/kg $DW_{1\%OC}$). Higher fertilization rates (80.0 ± 10.2%) were observed in porewater from sediments with mean concentrations of 0.170 ± 0.221 mg SUM DDD/kg DW (0.074 ± 0.068 mg SUM DDD/kg $DW_{1\%OC}$).

In 1992, sediment samples were collected from a number of locations in San Pedro Bay, including Los Angeles and Long Beach Harbours (Sapudar *et al.* 1994). The toxicity of each sample was evaluated using two toxicity tests, including 48-hour red abalone (*Haliotus rufescens*) larval development test (using

pore water) and 10-day amphipod (*Rhepoxynius abronius*) survival test (using bulk sediments). The data on the effects of pore water on abalone larval development were not used in this evaluation because they provided little ability to discriminate between sampling sites (i.e., nearly 90% of the samples were shown to be toxicity based on the results of this test).

The results of this investigation indicated that sediments from Los Angeles Inner Harbour, Inner Fish Harbour, East Basin, and Alamitos Bay were the most toxic to amphipods. In this study, toxic samples were identified as those in which amphipod survival was more than 20% lower than the average survival in the control treatments (Long *et al.* In review). The average concentrations of SUM DDD in the toxic samples were 0.029 ± 0.050 mg/kg DW or 0.012 ± 0.011 mg/kg $DW_{1\%OC}$. In the non-toxic samples, SUM DDD concentrations averaged 0.011 ± 0.016 mg/kg DW or 0.010 ± 0.007 mg/kg $DW_{1\%OC}$. While amphipod mortality was linked to dry weight-normalized concentrations of SUM DDD, the organic carbon-normalized SUM DDD concentrations appeared to contribute minimally to the biological effects that were observed.

In a related study, sediments were collected at several locations on the Palos Verdes Shelf and in Santa Monica Bay during 1992 and 1993 (Fairey 1997). Based on the results of 10-day toxicity tests, sediments from 11 of the 19 sites tested were acutely toxic to the amphipod, *Rhepoxynius abronius*. The average concentration of SUM DDD at the toxic sites was 0.107 ± 0.246 mg/kg DW or 0.092 ± 0.249 mg/kg $DW_{1\%OC}$. By comparison, SUM DDD concentrations averaged 0.116 ± 0.033 mg/kg DW or 0.072 ± 0.052 mg/kg $DW_{1\%OC}$ at the non-toxic sites in this survey. The limited concordance between amphipod survival and contaminant concentrations indicates that SUM DDD was unlikely to be responsible for the observed biological effects. While polychaete survival and growth tests were also conducted in this study, the data were not used in this evaluation because only four sediment samples were tested.

Under the Bay Protection and Toxics Cleanup Program, sediment samples were collected at a total of 350 stations in the vicinity of San Diego between October, 1992 and May, 1994 (Fairey *et al.* 1996). Sediment samples from the middle portion of San Diego Bay (i.e., the vicinity of the naval shipyards) were included in the data set assembled for this investigation because substantial concentration gradients in several of the contaminants of concern were observed. Several solid phase and porewater toxicity tests were conducted during this investigation, including an amphipod (*Rhepoxynius abronius*) survival test, a sea urchin (*Strongylocentrotus purpuratus*) fertilization tests, and a sea urchin (*S. purpuratus*) larval development test. The results of the polychaete growth and survival test,

the bay mussel larval development test, and the red abalone larval development tests were not used due to incomplete designation of toxic samples. The results of the power analyses conducted by Long *et al.* (In review) were used to designate toxic samples for the amphipod and sea urchin tests (i.e., samples were designated as toxic if the response measured differed by more than 20% from that of the control samples).

The results of this study indicated that sediments from the eastern portion of middle San Diego Bay were generally the most toxic to amphipods. The average concentration of SUM DDD at the toxic sites was 0.006 ± 0.018 mg/kg DW (0.010 ± 0.050 mg/kg $DW_{1\%OC}$). The average concentration of SUM DDD in the samples that were not acutely toxic to amphipods was 0.005 ± 0.005 mg/kg DW (0.003 ± 0.004 mg/kg $DW_{1\%OC}$). While there was little concordance between amphipod mortality and dry weight-normalized SUM DDD concentrations, the relationship was stronger for organic carbon-normalized SUM DDD concentrations.

The results of the tests with sea urchin gametes and embryos appear to agree with the results of the amphipod tests. Specifically, there was little concordance between sea urchin fertilization or embryo development and the dry weight-normalized concentrations of SUM DDD in sediment samples from middle San Diego Bay. However, sea urchin larval development was linked to organic carbon normalized concentrations of SUM DDD.

3.4.3 SECs for SUM DDD

Using the weight-of-evidence approach, SECs of 0.89 mg/kg DW or 0.23 mg/kg $DW_{1\%OC}$ were derived for SUM DDD (i.e., the sum of the concentrations of *p,p'*-DDD and *o,p'*-DDD).

Rationale: No dose-response data from controlled laboratory studies were located on the toxicity of SUM DDD. For this reason, SECs for SUM DDD could not be calculated using the spiked-sediment bioassay approach. Hence, the SECs for SUM DDD were derived using the weight-of-evidence approach.

A total of 15 effects records on the toxicity of-sediment-associated SUM DDD (hits; designated by an *) were included in the summarized toxicological data set (Table A4-9).

Evaluation of these data using the procedures described in Sections 2.4, resulted in the calculation of an ER-M (effects range median) of 1.17 mg/kg DW for SUM DDD. The unsummarized data set (Table A4-10) consisted of a total of 75 effects data records; the 50th percentile of this distribution was 0.89 mg/kg DW. The lower of these two values was identified as the SEC for SUM DDD.

The summarized toxicological data set for OC-normalized SUM DDD consisted of 16 effects data entries (Table A4-11). The median value in this data set was 0.299 mg/kg $DW_{1\%OC}$ for SUM DDD. Using the unsummarized toxicological data set (which had 72 effects data records; Table A4-12), an ER-M of 0.23 mg/kg $DW_{1\%OC}$ was determined for SUM DDD. The lower of these two values was identified as the SEC for this group of substances.

3.4.4 Evaluation of the SECs for SUM DDD

Reliability:

The reliability of the SECs for SUM DDD were evaluated using the procedures described in Section 2.5. To support this evaluation, matching sediment chemistry and biological effects data were compiled from a number of studies that were conducted in the Southern California Bight. These studies included Swartz *et al.* (1985), Swartz *et al.* (1986), Anderson *et al.* (1988), Ferraro *et al.* (1991), Swartz *et al.* (1991), Bay *et al.* (1994), Sapudar *et al.* (1994), Fairey *et al.* (1996), and Fairey (1997).

The results of this evaluation indicate that the dry weight-normalized SEC for SUM DDD provides a reliable basis for classifying sediment samples with elevated levels of this group of substances (Table 10). In total, 20 of the 21 (95%) sediment samples with SUM DDD concentrations at or in excess of the SEC were found to be toxic, based on the results of laboratory toxicity tests and benthic invertebrate community assessments. Therefore, there is a high probability of observing toxic effects at SUM DDD concentrations at or above the SEC. Most of the sediment samples with SUM DDD

Table 10. An evaluation of the reliability of the dry weight-normalized SECs for SUM DDD.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.5 mg/kg DW	Swartz et al. 1985	7	4	4	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	18	16	88.9
	Bay et al. 1994	13	5	5	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	1	1	100.0
	Overall	267	29	27	93.1
0.89 mg/kg DW	Swartz et al. 1985	7	3	3	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	17	16	94.1
	Bay et al. 1994	13	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	267	21	20	95.2
1.5 mg/kg DW	Swartz et al. 1985	7	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	15	14	93.3
	Bay et al. 1994	13	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	267	18	17	94.4

concentrations at or above 0.5 mg/kg DW (93%; 27 of 29 samples) and 1.5 mg/kg DW (94%; 17 of 18 samples) were also toxic to sediment-dwelling organisms. These results suggest that a lower value could be used to evaluate sediment quality in the Southern California Bight without substantially reducing the reliability of the SEC.

The organic carbon-normalized SEC also provides a reliable basis for classifying sediment samples in the Southern California Bight (Table 11). Twenty-one of the 23 sediment samples (91%) with SUM DDD concentrations at or above the SEC were found to be toxic to sediment-dwelling organisms. These results indicate that there is a high probability of observing adverse biological effects when SUM DDD concentrations exceed the organic carbon-normalized SEC. A lower value would have been slightly less reliable, as indicated by the incidence of toxic effects at or above 0.10 mg/kg $DW_{1\% OC}$ (86%; 30 of 35 samples were toxic). The increasing incidence of toxicity with increasing concentrations of SUM DDD generates additional confidence in the SECs.

Predictability:

The predictability of the SECs for SUM DDD was evaluated using data from five studies that were conducted outside Southern California Bight. These studies were conducted in Narragansett Bay (Munns *et al.* 1991). San Francisco Bay (Swartz *et al.* 1994), Tampa Bay (Long *et al.* 1994), Hudson-Raritan Estuary (Long *et al.* 1995b), and Biscayne Bay (Long 1997; Table A5-3). Of the 254 sediment samples that were collected in these studies, only six had SUM DDD concentrations that equaled or exceeded the dry weight-normalized SEC (Table 12). Five of these samples were toxic to sediment-dwelling organisms, as indicated by the results of laboratory toxicity tests (predictability = 83%). The incidence of toxic effects was the same for a lower concentration of SUM DDD, while predictability was somewhat lower (i.e., 80%; 4 of 5 samples were correctly predicted to be toxic) for a higher concentration of SUM DDD (1.50 mg/kg DW).

While the available data were limited, the results of this evaluation indicated that the organic carbon-normalized SEC was less predictive of toxicity than the dry weight-normalized SEC (Table 13). Only five

Table 11. An evaluation of the reliability of the organic carbon-normalized SECs for SUM DDD.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (SUM DDT > SEC)	Number of Samples Toxic to Amphipods	Predictability of SEC (% Correct)
0.10 mg/kg DW (1% OC)	Swartz et al. 1985	7	4	4	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	19	17	89.5
	Bay et al. 1994	13	7	6	85.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	1	1	100.0
	Fairey 1997	19	3	1	33.3
	Overall	267	35	30	85.7
0.23 mg/kg DW (1% OC)	Swartz et al. 1985	7	3	3	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	17	15	88.2
	Bay et al. 1994	13	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	1	1	100.0
	Fairey 1997	19	1	1	100.0
	Overall	267	23	21	91.3
0.40 mg/kg DW (1% OC)	Swartz et al. 1985	7	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	15	14	93.3
	Bay et al. 1994	13	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	1	1	100.0
	Overall	267	19	18	94.7

Table 12. An independent evaluation of the predictability of the dry weight-normalized SECs for SUM DDD.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.50 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	6	5	83.3
0.89 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	6	5	83.3
1.50 mg/kg DW	Munns et al. 1991	29	0	0	NA
	Swartz et al. 1994	9	4	3	75.0
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	254	5	4	80.0

Table 13. An independent evaluation of the predictability of the organic carbon-normalized SECs for SUM DDD.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.10 mg/kg DW (1% OC)	Swartz et al. 1994	9	7	4	57.1
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	1	1	100.0
	Long 1997	105	0	0	NA
	Overall	216	9	6	66.7
0.23 mg/kg DW (1% OC)	Swartz et al. 1994	9	6	4	66.7
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	216	7	5	71.4
0.40 mg/kg DW (1% OC)	Swartz et al. 1994	9	4	4	100.0
	Long et al. 1994	61	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	216	5	5	100.0

of the seven sediment samples with concentrations of SUM DDD equaled or exceeded the SEC were found to be toxic. Therefore, the predictability of the SEC was calculated to be 71%. By comparison, all of the sediment samples (5 of 5) were correctly classified when 0.40 mg/kg $DW_{1\%\ OC}$ was used as the sediment quality assessment value.

Matching sediment chemistry and biological effects data from one study conducted in a freshwater system provide additional information for evaluation the predictability of the SECs for SUM DDD. In 1991, Hoke *et al.* (1994) evaluated the acute toxicity of field-collected sediments from the Huntsville Spring Branch-Indian Creek system in Alabama. In this study, the effects of sediment-associated contaminants were evaluated in 10-day toxicity tests using amphipods (*Hyalella azteca*). The concentrations of SUM DDD ranged from below detection limits (BDL) to 42.3 mg/kg DW in the 15 samples collected nearby the Redstone Army Arsenal. The DW-normalized SEC of 1.17 mg/kg DW for SUM DDD was exceeded in 11 of the 15 sediment samples; hence, 11 samples were predicted to be toxic. The results of the toxicity tests indicated that all 11 samples were toxic to amphipods (statistical analyses of the mortality data were not performed by these investigators; samples with mortality of >20% were defined as toxic in this evaluation of the predictability of the SECs). In addition, all 11 of the samples that were predicted to be toxic using the OC-normalized SEC (0.299 mg/kg $DW_{1\%OC}$) were toxic to amphipods. Therefore, the predictability of both the DW-normalized and OC-normalized SECs for SUM DDD was calculated to be 100%.

3.5 Toxic Effects of Total DDT

3.5.1 Spiked-Sediment Bioassay Data

The data from four spiked-sediment toxicity investigations (McLeese and Metcalfe 1980; McLeese *et al.* 1982; Plesha *et al.* 1988; Bay *et al.* 1994) indicate that median lethal concentrations of tDDT ranged from 0.031 to >16.5 mg/kg DW (0.111 to >8.7 mg/kg $DW_{1\%OC}$). While these studies provide important information for assessing the toxic effects of specific DDT isomers, they may not

be directly relevant for evaluating the toxicity of tDDT. For example, it is likely that three of these studies utilized technical or reagent grade DDT in the toxicity tests. Both of these formulations are comprised primarily (i.e., >90%) of the *p,p'*-DDT and *o,p'*-DDT isomers (USEPA 1980a). However, Bay *et al.* (1994) reported that SUM DDE represented, on average, 88.4 ± 7.86% of the tDDT in the Palos Verdes sediments sampled in 1992; SUM DDT represented, on average, only 3.7 ± 3.89% of the tDDT in these samples. As there is some evidence (from water-only exposure studies) to suggest that *p,p'*-DDT and *o,p'*-DDT (i.e., SUM DDT) are more toxic to marine organisms than DDE or DDD (USEPA 1980a), consideration of data on SUM DDT alone could overestimate the toxicity of tDDT in the Southern California Bight. Likewise, data on the toxic effects of SUM DDE alone could underestimate the toxicity of tDDT, if DDE was significantly less toxic than the other DDT isomers. Therefore, the available spiked-sediment bioassay data were not used directly to determine SECs for tDDT in the Southern California Bight. However, these data were incorporated into the toxicological database and used to derive SECs using the WEA.

3.5.2 Field Data

In 1977, Word and Mearns (1979) collected sediment samples from a total of 70 stations between Point Conception and the United States-Mexico border. At each station, two samples were collected for assessing benthic invertebrate community characteristics and one sample was collected for chemical analysis. Total DDT concentrations were measured in the samples collected at each station. All of these samples were collected from a water depth of approximately 60 meters.

The results of the Word and Mearns (1979) study suggest that several benthic invertebrate community metrics are correlated with the concentration of tDDT in the Southern California Bight. For example, sites with an average of 70.2 ± 70.7 mg tDDT/kg DW had low densities of arthropods (4 ± 3.45 N/0.1 m^2). Higher arthropod densities (48.8 ± 19.5 N/0.1 m^2) were observed at sites with, on average, 3.2 ± 11.3 mg/kg DW of tDDT. The highest densities of arthropods (144 ± 42.7 N/0.1 m^2) were observed at the sites with the lowest concentrations of tDDT (0.101 ± 0.151 mg/kg DW). While echinoderm abundance demonstrated similar patterns relative to tDDT concentrations, species richness and total invertebrate abundance were not strongly linked to tDDT concentrations. As OC concentrations were not reported in this study, it was not possible to normalize the results to 1%OC.

In the 1980 study conducted in the vicinity of the Los Angeles County Sanitation District's sewage outfalls, Swartz *et al.* (1985) examined sediment toxicity, contamination, and benthic macroinvertebrate community structure at seven sites on the Palos Verdes Shelf. The results of this study indicated that the sediments from three sites were acutely toxic to amphipods during 10-day exposures. Benthic invertebrate community structure was also altered at these sites (as indicated by infaunal index and total biomass). Sediments from the other four sites were not toxic. At the toxic sites, the concentration of tDDT averaged 7.15 ± 1.89 mg/kg DW or 1.81 ± 0.55 mg/kg $DW_{1\%OC}$. The mean concentration of this group of substances at the non-toxic sites was 3.21 ± 4.46 mg/kg DW or 0.94 ± 1.12 mg/kg $DW_{1\%OC}$.

In a follow-up survey conducted in 1983, Swartz *et al.* (1986) collected sediment and macrobenthos samples from a total of nine sites on the Palos Verdes Shelf and in Santa Monica Bay. Matching sediment chemistry and biological effects (including acute toxicity bioassays and benthic invertebrate community structure) data were reported for six of these sites. These stations were considered to be the same locations as those sampled in the 1980 survey. The results of 10-day tests on the amphipod, *Rhepoxynius abronius*, indicated that the sediments collected from only one station (#6) were acutely toxic (Ferraro *et al.* 1991). The concentration of tDDT at this station was 3.55 mg/kg DW or 1.48 mg/kg $DW_{1\%OC}$. The mean concentration of tDDT at the non-toxic sites was 5.25 ± 4.31 mg/kg DW or 1.80 ± 1.29 mg/kg $DW_{1\%OC}$. The lack of concordance between the amphipod survival and tDDT concentrations indicates that this group of substances was not responsible for observed biological effects.

In the 1983 study (Swartz *et al.* 1986), benthic invertebrate community structure was altered at several sites with elevated levels of tDDT. For example, low echinoderm densities were observed at sites with mean concentrations of tDDT of 5.92 ± 3.49 mg/kg DW or 2.08 ± 0.934 mg/kg $DW_{1\%OC}$. In contrast, high densities of echinoderms were observed at a site with 0.140 mg tDDT/kg DW or 0.107 mg tDDT/kg $DW_{1\%OC}$. Similarly, low amphipod densities were observed at sites with mean concentrations of tDDT of 8.25 ± 1.70 mg/kg DW or 2.65 ± 0.659 mg/kg $DW_{1\%OC}$. Higher densities of amphipods were observed at sites with 1.68 ± 1.73 mg tDDT/kg DW or 0.846 ± 0.691 mg/kg $DW_{1\%OC}$. Similar results were observed when total benthic invertebrate density, species richness, and infaunal index were considered.

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes Shelf and in Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10

day) toxicity tests using amphipods, *Rhepoxynius abronius*, were conducted on each of the core sections. The results of this investigation indicated that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The mean concentration of tDDT in these sediment samples was 143 ± 52.3 mg/kg DW or 17.2 ± 3.13 mg/kg $DW_{1\%OC}$. Moderate toxicity to amphipods (35.9 ± 12.1% mortality) was observed in shallower and deeper sediments (2.5 - 12.5 cm and 37.5 to 47.5 cm); these samples had, on average, 23.9 ± 17.5 mg/kg DW (4.59 ± 3.10 mg/kg $DW_{1\%OC}$) of tDDT. The samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average tDDT concentrations of 1.94 ± 4.85 mg/kg DW (0.580 ± 0.835 mg/kg $DW_{1\%OC}$).

One year later (1986), another follow-up survey was conducted to evaluate trends in sediment contamination and benthic macroinvertebrate community structure on the Palos Verdes Shelf and in Santa Monica Bay (Ferraro *et al.* 1991). A total of eight sites were sampled in this survey; none of the sediments tested were acutely toxic to the amphipod, *Rhepoxynius abronius*, during 10-day exposures. The mean concentration of tDDT at the eight sites was approximately 6.94 ± 4.69 mg/kg DW or 1.83 ± 0.922 mg/kg $DW_{1\%OC}$.

Data on benthic invertebrate community structure, which reflects continuous exposure to sediment-associated contaminants, indicates that adverse effects on sediment-dwelling organisms occur at elevated levels of tDDT (Ferraro *et al.* 1991). For example, low densities of amphipods (4.1 ± 2.5 N/0.1 m^2) were observed at several sites sampled on the Palos Verdes Shelf. At these sites, concentrations of tDDT averaged 10.7 ± 4.55 mg/kg DW or 2.66 ± 0.253 mg/kg $DW_{1\%OC}$. Moderate amphipod densities (23.5 ± 5.9 N/0.1 m^2) were observed at the sites with lower concentrations of tDDT (7.12 ± 0.695 mg/kg DW or 1.90 ± 0.238 mg/kg $DW_{1\%OC}$). The highest amphipod densities (54.6 ± 5.1 N/0.1 m^2) were observed at the sites with the lowest tDDT concentrations (1.10 ± 1.23 mg/kg DW or 0.494 ± 0.295 mg/kg $DW_{1\%OC}$). Similarly, low echinoderms densities (2.4 ± 5.7 N/0.1 m^2) were observed at seven of the eight sites sampled during this survey, with tDDT concentrations averaging 7.90 ± 4.13 mg/kg DW or 2.05 ± 0.732 mg/kg $DW_{1\%OC}$ at these sites. The density of echinoderms (208 N/0.1 m^2) was higher at a site with lower concentrations of tDDT (0.229 mg/kg DW or 0.286 mg/kg $DW_{1\%OC}$). Species richness, total benthic invertebrate density, and total benthic invertebrate biomass were not correlated with tDDT concentrations, however.

Matching sediment chemistry and biological effects data were collected at seven sites in the Southern California Bight during 1987 (Anderson *et al.* 1988). While

this study was conducted primarily to evaluate the effects associated with PAH-contaminated sediments, it also provides some information for assessing the potential effects of mixtures of tDDT and other substances. In this study, the survival and reburial of the amphipod, *Grandidierella japonica*, (in 10-day acute toxicity tests) were not linked with concentrations of tDDT. The mean concentration of tDDT was 0.104 ± 0.094 mg/kg DW or 0.042 ± 0.044 mg/kg $DW_{1\%OC}$ in sediments that were significantly toxic to this species. The average concentration of tDDT in sediments that did not affect survival was 1.04 ± 2.45 mg/kg DW or 0.259 ± 0.585 mg/kg $DW_{1\%OC}$. As the concentration of this substance was lower at the toxic sites than it was at the non-toxic sites, something other than the concentrations of tDDT must have been responsible for the observed effects.

The survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, (in 35-day chronic exposure tests) were not correlated with the concentrations of sediment-associated tDDT measured in Palos Verdes sediments (i.e., there was no concordance between sediment chemistry and the biological effects; Anderson *et al.* 1988). However, effects on sea urchin growth were associated with elevated concentrations of DDTs. Reduced growth rates were observed in sediments with mean concentrations of 3.12 ± 4.13 mg/kg DW or 0.736 ± 1.01 mg/kg $DW_{1\%OC}$ of tDDT. It should be noted that this analysis was significantly affected by one sample (Palos Verdes) that had concentrations of DDTs that were more than an order of magnitude higher than those measured at other sites.

In a study commissioned by the Santa Monica Bay Restoration Project, Bay *et al.* (1994) collected sediments from 12 sites located in the vicinity of the Palos Verdes Peninsula and one reference site near Dana Point. Acute toxicity bioassays were conducted using sediments from five of the 13 sites sampled in the 1992 survey; none on these sediments were toxic to amphipods (*Rhepoxynius abronius*; Bay *et al.* 1994). The mean concentrations of tDDT at these sites were 7.09 ± 6.96 mg/kg DW or 2.71 ± 2.09 mg/kg $DW_{1\%OC}$. Similarly, the survival, avoidance behavior, gonad growth and growth (as indicated by diameter) of white sea urchins (*Lytechinus pictus*) were not affected at mean tDDT concentrations of 5.61 ± 5.83 mg/kg DW or 1.97 ± 1.57 mg/kg $DW_{1\%OC}$.

The growth rate (as measured by wet weight change over the 35-day exposure period) of white sea urchins was influenced by exposure to Palos Verdes sediments (Bay *et al.* 1994). Reduced growth rates were observed at sites with mean concentrations of tDDT of 10.7 ± 7.32 mg/kg DW or 3.09 ± 2.10 mg/kg $DW_{1\%OC}$. The concentrations of this group of substances were lower in the

sediments that were not associated with growth effects (3.36 ± 3.52 mg/kg DW or 1.48 ± 1.07 mg/kg $DW_{1\%OC}$). Similar results were obtained when the fertilization of sea urchin, *Strongylocentrotus purpuratus*, gametes was the experimental endpoint considered in the bioassay. The results of this short-term (< 2 hours) test indicated that low fertilization rates (9.4 ± 16.2%) were associated with exposure to porewater from sediments that had mean tDDT concentrations of 10.3 ± 5.83 mg/kg DW or 3.15 ± 1.61 mg/kg $DW_{1\%OC}$. Higher fertilization rates were associated with exposure to porewater from sediments with 3.02 ± 3.88 mg tDDT/kg DW or 1.31 ± 1.14 mg tDDT/kg $DW_{1\%OC}$.

Under the Bay Protection and Toxics Cleanup Program, sediment samples were collected from a number of locations in the vicinity of Los Angeles and Long Beach Harbours in 1992 (i.e., San Pedro Bay; Sapudar *et al.* 1994). The toxicity of each sample was evaluated using two toxicity tests, including 48-hour red abalone (*Haliotus rufescens*) larval development test (using pore water) and 10-day amphipod (*Rhepoxynius abronius*) survival test (using bulk sediments). The data on the effects of pore water on abalone larval development were not used in this evaluation because they provided little ability to discriminate between sampling sites (i.e., nearly 90% of the samples were shown to be toxicity based on the results of this test).

Based on the results of the amphipod tests, Sapudar *et al.* (1994) showed that sediments from Los Angeles Inner Harbour, Inner Fish Harbour, East Basin, and Alamitos Bay tended to be the most toxic. In this assessment, toxic samples were identified as those in which amphipod survival was more than 20% lower than the average survival in the control treatments (Long *et al.* In review). In the toxic samples from San Pedro Bay, the concentrations of tDDT averaged 0.161 ± 0.147 mg/kg DW or 0.081 ± 0.064 mg/kg $DW_{1\%OC}$. By comparison, tDDT concentrations averaged 0.111 ± 0.086 mg/kg DW or 0.113 ± 0.116 mg/kg $DW_{1\%OC}$ in the non-toxic samples. The limited concordance between amphipod survival and tDDT concentrations indicates that this group of substances likely contributed minimally to the amphipod mortality that was observed in Los Angeles and Long Beach Harbours.

During 1992 and 1993, sediment sampling was conducted at several locations on the Palos Verdes Shelf and in Santa Monica Bay (Fairey 1997). Matching sediment chemistry and toxicity data were reported for 19 sites in this area. The results of 10-day acute toxicity tests with the amphipod, *Rhepoxynius abronius*, indicated that the sediments from 11 of these sites were acutely toxic. The average concentrations of tDDT at these sites were 0.683 ± 1.28 mg/kg DW or 0.495 ± 1.04 mg/kg $DW_{1\%OC}$. At the non-toxic sites, tDDT concentrations

averaged 2.83 ± 0.521 mg/kg DW or 1.73 ± 1.07 mg/kg $DW_{1\%OC}$. The lack of concordance between amphipod survival and tDDT concentrations indicates that this group of substances was not responsible for the observed biological effects. While polychaete survival and growth tests were also conducted in this study, the data were not used in this evaluation because only four sediment samples were tested.

Between October, 1992 and May, 1994, a total of 350 samples were collected under the Bay Protection and Toxics Cleanup Program to evaluate sediment quality conditions in San Diego (Fairey *et al.* 1996). A portion of these data (i.e., the samples from the middle portion of San Diego Bay near the naval shipyards) were included in the data set assembled for this investigation because substantial concentration gradients existed for several of the contaminants of concern. The porewater and solid phase sediment toxicity tests conducted on each of the samples included: amphipod (*Rhepoxynius abronius*) survival; sea urchin (*Strongylocentrotus purpuratus*) fertilization; and, sea urchin (*S. purpuratus*) larval development. The results of the polychaete growth and survival test, the bay mussel larval development test, and the red abalone larval development tests were not used due to incomplete designation of toxic samples. The results of the power analyses conducted by Long *et al.* (In review) were used to designate toxic samples for the amphipod and sea urchin tests (i.e., samples were designated as toxic if the response measured differed by more than 20% from that of the control samples).

Based on the results of the 10-day acute lethality tests, sediments from the eastern portion of middle San Diego Bay were generally found to be the most toxic to amphipods. The concentration of tDDT at the toxic sites averaged 0.022 ± 0.049 mg/kg DW (0.028 ± 0.123 mg/kg $DW_{1\%OC}$). Similar concentrations of tDDT (0.022 ± 0.021 mg/kg DW or 0.013 ± 0.013 mg/kg $DW_{1\%OC}$) were observed in the samples that were not acutely toxic to amphipods, indicating that tDDT probably contributed little to amphipod mortality in the middle portion of San Diego Bay. Better concordance between the results of two of the toxicity tests (i.e., amphipod mortality and sea urchin larval development) and tDDT concentrations was observed when organic carbon-normalized values were considered, however.

In 1993, sediment samples were collected from 10 sites along the 60 meter contour on the Palos Verdes Shelf (Murdoch *et al.* In press). Acute (10 day) and short-term chronic (20 day) toxicity tests using the marine polychaete, *Neanthes arenaceodentata*, were conducted on sediments from all of these locations. In addition, a 120 day chronic toxicity test was conducted using the same species to

determine the effects on reproduction associated with long-term exposure to PCBs and DDTs. The results of this study indicated that acute and short-term chronic exposure to tDDT concentrations as high as 267 mg/kg DW (33.2 mg/kg $DW_{1\%OC}$) did not adversely affect polychaete survival or growth. The concentrations of tDDT averaged 49.4 ± 107 mg/kg DW (7.56 ± 12.6 mg/kg $DW_{1\%OC}$) in these non-toxic samples. However, polychaete reproductive success, as indicated by the number of emergent juveniles, was compromised in the sediments that had the highest concentrations of tDDTs (140 ± 180 mg/kg DW or 18.3 ± 21 mg/kg $DW_{1\%OC}$). In contrast, polychaete reproduction was not impaired in sediments that contained 4.14 ± 3.38 mg/kg DW (2.18 ± 1.23 mg/kg $DW_{1\%OC}$).

3.5.3 SECs for Total DDT

Using the weight-of-evidence approach, SECs of 7.15 mg/kg DW and 2.00 mg/kg $DW_{1\%OC}$ were derived for tDDT (i.e., the sum of the concentrations of *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD).

Rationale: As indicated previously, data on the toxicity of tDDT are available from four spiked-sediment bioassays. Two of the four studies included dose-response information on a benthic arthropod species, but both of these investigations were conducted using the technical or reagent grade DDT. As the tDDT in Southern California Bight sediment consists primarily of DDE, the data from these spiked-sediment bioassays could overestimate the toxicity of tDDT. For this reason, SECs were derived using the WEA rather than the SSBA.

The summarized and unsummarized toxicological data sets for tDDT (mg/kg DW) contained a total of 36 and 149 effects data records, respectively (hits; designated by an *; Tables A4-13 and A4-14). Evaluation of these data sets, using the procedures described in Section 2.4, resulted in the calculation of ER-Ms (effects range median) of 7.15 and 8.22 mg/kg DW using the summarized and unsummarized data sets, respectively, for tDDT. The lower of these two values was identified as the SEC.

Similarly, the summarized and unsummarized toxicological data sets for tDDT, expressed on an OC-normalized basis (mg/kg

$DW_{1\%OC}$) contained 28 and 100 effects data entries, respectively (Tables A4-15 and A4-16). The ER-Ms derived from these two data sets were 2.00 and 2.21 mg/kg $DW_{1\%OC}$, respectively. The lower of these two values was identified as the SEC.

3.5.4 Evaluation of the SECs for Total DDT

Reliability:

The reliability of the SECs for tDDTs was evaluated using the procedures described in Section 2.5. To support this evaluation, matching sediment chemistry and biological effects data were compiled from 11 studies that were conducted in the Southern California Bight. The results of an evaluation of the SECs derived in this study indicate that these assessment values provide a reliable basis for evaluating the toxic effects of sediment-associated tDDTs.

In the Southern California Bight, 40 sediment samples had concentrations of tDDTs that equaled or exceeded the SEC of 7.15 mg/kg DW (Table 14). Based on the results of various toxicity tests and benthic invertebrate community assessments, 38 of these samples (95%) were found to be toxic. This assessment indicates that a high degree of confidence should be placed in the SEC for tDDT.

A lower value was found to be less reliable than the SEC for tDDT. Of the 62 sediment samples with tDDT concentrations at or in excess of 2.0 mg/kg DW, 47 (76%) were found to be toxic. A higher incidence of toxicity (96%) was observed in sediment samples with tDDT concentrations at or in excess of 10 mg/kg DW. The increasing incidence of toxicity with increasing concentrations of tDDTs raises the level of confidence that can be placed in the SEC.

Based on the information contained in the toxicological data sets, the OC-normalized SEC is also provides a reliable basis for classifying sediment samples from the Southern California Bight (Table 15). Thirty-two of the 39 sediment samples (82%) with tDDT concentrations at or above the SEC were found to be toxic. A lower value (0.5 mg/kg $DW_{1\%\ OC}$) was found to be much less reliable, as only 63% of the samples with concentrations at or above this level

Table 14. An evaluation of the reliability of the dry weight-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
2.00 mg/kg DW	Word & Mearns 1979	41	6	6	100.0
	Swartz et al. 1985	7	4	4	100.0
	Swartz et al. 1986	6	4	4	100.0
	Anderson et al. 1988	9	1	1	100.0
	Ferraro et al. 1991	8	6	6	100.0
	Swartz et al. 1991	31	18	16	88.9
	Bay et al. 1994	13	8	6	75.0
	Murdoch et al. In Press	6	5	2	40.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	10	2	20.0
	Overall	328	62	47	75.8
7.15 mg/kg DW	Word & Mearns 1979	41	6	6	100.0
	Swartz et al. 1985	7	3	3	100.0
	Swartz et al. 1986	6	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	4	4	100.0
	Swartz et al. 1991	31	17	16	94.1
	Bay et al. 1994	13	5	5	100.0
	Murdoch et al. In Press	6	3	2	66.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	328	40	38	95.0

Table 14. An evaluation of the reliability of the dry weight-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
10.00 mg/kg DW	Word & Mearns 1979	41	6	6	100.0
	Swartz et al. 1985	7	0	0	NA
	Swartz et al. 1986	6	0	0	NA
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	1	1	100.0
	Swartz et al. 1991	31	15	14	93.3
	Bay et al. 1994	13	3	3	100.0
	Murdoch et al. In Press	6	2	2	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	328	27	26	96.3

Table 15. An evaluation of the reliability of the organic carbon-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.50 mg/kg DW (1% OC)	Swartz et al. 1985	7	6	4	66.7
	Swartz et al. 1986	6	5	4	80.0
	Anderson et al. 1988	9	1	1	100.0
	Ferraro et al. 1991	8	7	6	85.7
	Swartz et al. 1991	31	22	17	77.3
	Bay et al. 1994	13	11	7	63.6
	Murdoch et al. In Press	6	6	2	33.3
	Sapudar et al. 1994	103	1	0	0.0
	Fairey et al. 1996	85	1	1	100.0
	Fairey 1997	19	10	2	20.0
	Overall	287	70	44	62.9
2.00 mg/kg DW (1% OC)	Swartz et al. 1985	7	3	3	100.0
	Swartz et al. 1986	6	2	2	100.0
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	5	5	100.0
	Swartz et al. 1991	31	16	15	93.8
	Bay et al. 1994	13	5	4	80.0
	Murdoch et al. In Press	6	5	2	40.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	3	1	33.3
	Overall	287	39	32	82.1

Table 15. An evaluation of the reliability of the organic carbon-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
3.00 mg/kg DW (1% OC)	Swartz et al. 1985	7	0	0	NA
	Swartz et al. 1986	6	1	1	100.0
	Anderson et al. 1988	9	0	0	NA
	Ferraro et al. 1991	8	0	0	NA
	Swartz et al. 1991	31	15	14	93.3
	Bay et al. 1994	13	3	3	100.0
	Murdoch et al. In Press	6	3	2	66.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	3	1	33.3
	Overall	287	25	21	84.0

were toxic. A higher value, 3.0 mg/kg $DW_{1\% OC}$, was found to be slightly more reliable than the SEC [i.e., 21 of 25 (84%) of the sediment samples were correctly classified].

Predictability:

The predictability of the SECs for tDDT was evaluated using data from nine studies that were conducted elsewhere in the United States (i.e., outside the Southern California Bight; Chapman *et al.* 1987; Pastorok and Becker 1990; Munns *et al.* 1991; Casillas *et al.* 1992; Swartz *et al.* 1994; Long *et al.* 1994; Rice *et al.* 1995; Long *et al.* 1995b; Long 1997; Table A5-4). This data set included matching sediment chemistry and biological effects data on more than 300 sediment samples. Only three of these samples had concentrations of tDDTs that exceeded the dry weight-normalized SECs; all three of these sediment samples were toxic (Table 16). Notwithstanding the limitations on the data set, these results suggest that the SEC provides an accurate basis for classifying sediment samples with elevated levels of tDDTs. A somewhat lower level of predictability (83%; 5 of 6 samples were correctly classified) was achieved using a lower value of tDDTs (2.0 mg/kg DW).

Evaluation of the organic carbon-normalized SEC for tDDT was also limited by the available data (Table 17). That is, few sediment samples from sites outside the Southern California Bight had tDDT concentrations that exceeded the SEC. Nonetheless, all of the sediment samples with tDDT levels above 2.0 mg/kg $DW_{1\% OC}$ (i.e., 3 of 3 samples) were found to be toxic, suggesting that the SEC provided an accurate predictor of sediment toxicity. A lower value (0.050 mg/kg $DW_{1\%OC}$) also accurately classified sediments with elevated levels of tDDTs (predictability = 86%, 6 of 7 samples).

The results of independent study conducted in a freshwater ecosystem provide additional information for evaluating the predictability of the SEC that was developed for tDDT in the Southern California Bight. In 1991, Hoke *et al.* (1994) evaluated the acute toxicity of field-collected sediments from the Huntsville Spring Branch-Indian Creek system in Alabama. In this study, amphipods (*Hyalella azteca*) were exposed for a period of 10 days to sediments (15 samples) collected nearby the Redstone Army Arsenal. The concentrations of tDDT

Table 16. An independent evaluation of the predictability of the dry weight-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
2.00 mg/kg DW	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Munns et al. 1991	29	0	0	NA
	Casillas et al. 1992	15	0	0	NA
	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	1	1	100.0
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	313	6	5	83.3
7.15 mg/kg DW	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Munns et al. 1991	29	0	0	NA
	Casillas et al. 1992	15	0	0	NA
	Swartz et al. 1994	9	3	3	100.0
	Long et al. 1994	61	0	0	NA
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	313	3	3	100.0
10.0 mg/kg DW	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Munns et al. 1991	29	0	0	NA
	Casillas et al. 1992	15	0	0	NA
	Swartz et al. 1994	9	3	3	100.0
	Long et al. 1994	61	0	0	NA
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	313	3	3	100.0

Table 17. An independent evaluation of the predictability of the organic carbon-normalized SECs for tDDT.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tDDT > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.50 mg/kg DW (1% OC)	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Casillas et al. 1992	12	0	0	NA
	Swartz et al. 1994	9	5	4	80.0
	Long et al. 1994	61	1	1	100.0
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	1	1	100.0
	Long 1997	105	0	0	NA
	Overall	281	7	6	85.7
2.00 mg/kg DW (1% OC)	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Casillas et al. 1992	12	0	0	NA
	Swartz et al. 1994	9	3	3	100.0
	Long et al. 1994	61	0	0	NA
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	281	3	3	100.0
3.00 mg/kg DW (1% OC)	Chapman et al. 1987	9	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Casillas et al. 1992	12	0	0	NA
	Swartz et al. 1994	9	3	3	100.0
	Long et al. 1994	61	0	0	NA
	Rice et al. 1995	20	0	0	NA
	Long et al. 1995	50	0	0	NA
	Long 1997	105	0	0	NA
	Overall	281	3	3	100.0

ranged from below detection limits (BDL) to 199 mg/kg DW (BDL to 78.6 mg/kg $DW_{1\%OC}$) in these samples. The SEC of 7.15 mg/kg DW for tDDT was exceeded in 10 of the sediment samples; all 10 samples were predicted to be toxic. The results of the toxicity tests that were performed indicated that all 10 samples were toxic to amphipods (statistical analyses of the mortality data were not performed by these investigators; samples with mortality of >20% were defined as toxic in this evaluation of the predictability of the SECs). All 11 of the samples that were predicted to be toxic using the OC-normalized SEC (2.00 mg/kg $DW_{1\%OC}$) were toxic to amphipods. Therefore, the predictability of both the DW-normalized and OC-normalized SECs for tDDT was calculated to b 100%.

3.6 Discussion of the Toxic Effects of DDTs

There are a number of factors that could, potentially, affect the toxicity of sediment-associated DDTs. These factors include species and life stage tested, exposure route(s) considered, duration of exposure, endpoint measured, DDT isomers present, and the characteristics of the sediments under investigation. While it is difficult to evaluate the influence of these factors directly (due to the limitations of the available data), the following discussion is provided to identify the potential effects of these factors on the toxicity of DDTs.

Marine organisms exhibit a broad range of sensitivities to environmental contaminants. Therefore, the species and life stage used in a toxicity test can influence the apparent toxicity of a substance. No information was located that would directly support an evaluation of the relative sensitivities, to sediment-associated DDTs, of the biota that would normally occur in association with Southern California Bight sediments. However, data from water column exposure studies appear to corroborate the premise that aquatic organisms exhibit a broad range of sensitivities to DDTs. For example, USEPA (1980a) reported that acutely (96 hour) lethal (LC_{50}) or effective (EC_{50}) concentrations of DDT to marine biota ranged from 0.00014 mg/L for the brown shrimp (*Penaeus aztecus*) to 0.089 mg/L for the northern puffer (*Sphaeroides maculatus*). **Variability in species and life stage sensitivities to DDTs means that SECs established using limited data only may not accurately reflect the range of sensitivities that exist among the species that would normally occur in the Southern California**

Bight. Hence, adverse biological effects on important and sensitive species could occur below the SECs of DDTs developed in this study.

In coastal ecosystems, aquatic organisms may be exposed to DDTs via a number of routes. First, epibenthic species may be exposed to DDTs through direct contact with the sediments (i.e., dermal exposure) and in their diet (i.e., oral exposure). Contact with the overlying water is expected to be a minor exposure route for these species. Infaunal organisms may be exposed to DDTs by direct contact with sediment particles and porewater, as well as in their diet. However, dermal exposure may be reduced in tube-dwelling species. Lastly, benthic invertebrates that process sediments are likely to have the highest exposure to DDTs, as they may extract DDTs directly from sediment particles or from microorganisms that they ingest. In addition, they are likely to be exposed by direct contact with sediments and porewater. Therefore, it is important for the toxicological database to include data on species that are exposed to DDTs via various routes to increase the ecological applicability of the sediment effect concentrations subsequently developed. For example, SECs based on data for epibenthic species only could underestimate the toxicity of sediment-associated DDTs.

Duration of exposure is an important factor that affects the results of toxicity tests. Typically, high concentrations of a contaminant are required to elicit significant effects in short-term exposure tests (e.g., 96 hours), while lower effect concentrations are reported in longer tests on the same species and life stage. MacDonald (1993) evaluated toxicity data on over forty substances from water-only exposures and reported that short-term LC_{50}s were, on average, 7.7 times higher than long-term LC_{50}s. It is likely that biological responses to sediment-associated contaminants would also be affected by exposure duration. As most of the available toxicity data on DDTs are from relatively short-term studies in which lethality was the endpoint measured, threshold effect concentrations of DDTs (i.e., the levels that are associated with adverse effects) in the Southern California Bight could be lower than those indicated in this assessment.

The experimental endpoint measured in toxicity tests can also influence the apparent toxicity of DDTs to sediment-dwelling organisms. Exposure to DDTs has been associated with a diverse range of adverse effects in fish and other aquatic biota, including mortality, reproductive impairment, enzyme activity inhibition, hyperactivity, loss of equilibrium, and behavioral alterations (USEPA 1980a). No evidence of carcinogenicity in aquatic organisms was located in this review; however, inconclusive evidence of carcinogenicity in mammalian receptors has been reported (USEPA 1980a). MacDonald (1993) reported that effective

concentrations of water-borne contaminants can vary by as much as two orders of magnitude depending on the endpoint that was measured in the bioassay. Similar patterns of toxicity are likely to occur for sediment-associated contaminants, as well. As more sensitive effects are likely to occur at lower concentrations of DDTs, sediment effect concentrations derived from acute lethality data could underestimate the toxicity of DDTs in the Southern California Bight.

Evaluation of the toxicity of DDTs is further complicated by the availability of dose-response data (i.e., from spiked-sediment bioassays) for tDDT and the individual DDT isomers. For this reason, associative (or co-occurrence) information that links contaminant concentrations to adverse biological effects has also been used in this review. However, interpretation of associative data is complicated by the presence of multiple contaminants in each sediment sample, frequently including metals, PAHs, PCBs, and other substances. While the methodology used in this review provides a means of identifying the contaminants that are implicated in the toxic response, it is difficult to conclusively desegregate the effects of individual chemical contaminants. As the SECs reflect the effects of DDTs when they are present in mixtures, the toxicity of individual sediment-associated DDTs could be overestimated if they occurred in the Southern California Bight by themselves.

The toxicity of sediment-associated DDTs may also be affected by the mixture of DDTs present. Sediments in the Southern California Bight frequently contain the parent compounds and several degradation products. While few data are available to evaluate the relative toxicity of these substances in sediments, information on the effects of water-borne DDTs indicates that there could be significant differences in toxicity. For example, USEPA (1980a) reported data which indicated that the species mean acute values of water-borne DDT, DDE, and DDD for the eastern oyster (*Crassostrea virginica*) were 0.0079 mg/L, 0.014 mg/L, and 0.025 mg/L, respectively. In another species, the Korean shrimp (*Palaemon macrodactylus*), water-borne DDT was roughly ten times more toxic than DDD (when acute lethality was the endpoint measured). These data appear to indicate that DDT is more toxic than either of the two major metabolites, DDE and DDD. This is important when the toxicity of tDDT is being evaluated because reliance on toxicological information on one of the more toxic forms of DDT could bias the overall assessment. Likewise, reliance on toxicological data on the least toxic forms could result in SECs that underestimate toxicity.

The results of the evaluations presented in this review indicate that the SECs for DDT are generally reliable. That is, the dry weight- and organic carbon-normalized SECs for all four groups of DDTs can be used to accurately classify sediments

from the Southern California Bight. For SUM DDT, reliability could have been improved to 85% if an SEC of 0.06 mg/kg DW had been selected.

The evaluation of the predictability of the SECs that were developed for DDTs in this study was limited by the matching sediment chemistry and biological effects data that were available for sites located outside the Southern California Bight. That is, most of the sediment samples obtained from sites located elsewhere in the United States had concentrations of DDTs that were below the SECs. As such, it was not possible to evaluate the predictability of the SECs for SUM DDE in marine and estuarine sediments. Nonetheless, evaluation of the data that were compiled showed that most of the dry weight- and organic carbon-normalized SECs can be used to accurately classify sediments from outside the Southern California Bight. A higher SEC for SUM DDD (e.g., 0.4 mg/kg $DW_{1\%OC}$) would have been more predictive, however. The general agreement between the results of the reliability and predictability evaluations substantially increases the confidence that can be placed in the SECs.

The DW-normalized SECs for three of the four groups of DDTs accurately predicted toxicity in field-collected sediments. However, the SEC for SUM DDT would have predicted toxicity more accurately if it were higher (eg., 0.060 mg/kg $DW_{1\%OC}$). All of the OC-normalized SECs provided an accurate basis for predicting the toxicity of Southern California Bight sediments. While data from the Southern California Bight provide essential information for evaluating the SECs, data from other geographic areas provides an independent basis for assessing their predictability. Two independent data sets were used to evaluate the predicability of the SECs for DDTs (Swartz *et al.* 1994; Hoke *et al.* 1994). The results of this assessment indicate that the SECs provide an accurate basis for predicting sediment toxicity. However, a higher SEC for SUM DDT (e.g., 0.200 mg/kg DW) would have predicted sediment toxicity more accurately.

4.0 Evaluation of the Toxicity of Sediment-Associated PCBs

4.1 Identity and Nomenclature of PCBs

Polychlorinated biphenyls (PCBs) is the generic term that is applied to a group of 209 congeners that contain between one and 10 chlorine atoms on a biphenyl ring. The potential positions for chlorine substitution are numbered according to the American Chemical Society standard notation. PCB congeners with the same number of chlorine atoms on the biphenyl rings are grouped into a specific class (e.g., tetrachlorobiphenyl), with each congener in the class assigned a numeric prefix that describes the position of the chlorine atoms (e.g., 3,4,4',5-tetrachlorobiphenyl). The ten classes of PCBs, the empirical formulas for each class, and the number of possible congeners for each class are listed in Table 18.

Table 18. Empirical formulae and number of congeners for each class of PCBs.

PCB	Empirical Formula	Number of Possible Congeners
Monochlorobiphenyl	$C_{12}H_9Cl$	3
Dichlorobiphenyl	$C_{12}H_8Cl_2$	12
Trichlorobiphenyl	$C_{12}H_7Cl_3$	24
Tetrachlorobiphenyl	$C_{12}H_6Cl_4$	42
Pentachlorobiphenyl	$C_{12}H_5Cl_5$	46
Hexachlorobiphenyl	$C_{12}H_4Cl_6$	42
Heptachlorobiphenyl	$C_{12}H_3Cl_7$	24
Octachlorobiphenyl	$C_{12}H_2Cl_8$	12
Nonachlorobiphenyl	$C_{12}HCl_9$	3
Decachlorobiphenyl	$C_{12}Cl_{10}$	1

Commercially, PCBs have been produced as complex mixtures of many chlorobiphenyl congeners, whose usages depend primarily on the percentage of chlorine in the mixture. For example, mixtures of PCBs that contain between 21% and 54% chlorine (by weight) have been used extensively as dielectric fluids in closed electrical systems. In addition, PCB mixtures have been used as

plasticizers, heat transfer liquids, hydraulic fluids, fluids in vacuum pumps and compressors, lubricants, wax extenders, special adhesives, and surface coatings for carbonless copy paper (CCREM 1986). All of these latter uses were curtailed in 1971 in the United States (USEPA 1980b).

Polychlorinated biphenyls have been marketed under a number of trade names worldwide (e.g., Askerel, Eucarel, Sovol; McDonald and Tourangeau 1986); however, all of the PCBs manufactured in North America were produced under the trade name Aroclor (Moore and Walker 1991). The commercially available Aroclor mixtures are designated with a four digit number, which typically indicates the molecular structure and the percent chlorine by weight. For example, Aroclor 1254 is a chlorinated biphenyl which contains 54% chlorine by weight (which is designated by the '54'; Kalmaz and Kalmaz 1979). The lower chlorinated Aroclor formulations (e.g., 1221 and 1232) are primarily composed of mono-, di-, and trichlorobiphenyls, whereas the higher chlorinated Aroclor mixtures (e.g., 1254 and 1260) are dominated by tetra-, hexa-, and hepta-chlorobiphenyls (see Table 19; Rappe and Buser 1980; USEPA 1980b).

Table 19. Approximate molecular composition of Aroclor mixtures.

PCB	Percent Chlorine (by weight) of Aroclor Formulations					
	1221	1232	1242	1248	1254	1260
Biphenyl	11.0	6.0	-	-	-	-
Monochlorobiphenyl	51.0	26.0	1.0	-	-	-
Dichlorobiphenyl	32.0	29.0	17.0	1.0	-	-
Trichlorobiphenyl	4.0	24.0	40.0	23.0	-	-
Tetrachlorobiphenyl	2.0	15.0	32.0	50.0	16.0	-
Pentachlorobiphenyl	0.5	0.5	10.0	20.0	60.0	12.0
Hexachlorobiphenyl	-	-	0.5	1.0	23.0	46.0
Heptachlorobiphenyl	-	-	-	-	1.0	36.0
Octachlorobiphenyl	-	-	-	-	-	6.0
Nonachlorobiphenyl	-	-	-	-	-	-
Decachlorobiphenyl	-	-	-	-	-	-

Evaluation of the toxic effects of PCBs is complicated for a number of reasons. First, this group of compounds consists of 209 different congeners, each of which may have unique toxicological characteristics. However, dose-response data from spiked-sediment bioassays were not located on any of these substances. Therefore, it was not possible to assess the toxicity of individual PCB congeners to sediment-dwelling organisms.

Second, much of the available data on the toxicity of sediment-associated PCBs have been generated on several formulated PCB mixtures, including Aroclor 1242 and Aroclor 1254. These data are essential for evaluating the toxicity of these formulations in field-collected sediments. In the Southern California Bight, however, sediments are likely to contain many more PCB congeners than would be represented by measurements of Aroclor 1242 and/or Aroclor 1254 concentrations (i.e., mono-, di-, hepta-chlorobiphenyls may not be fully represented by these measurements). Hence, field-collected sediments could be more toxic than would be indicated by, for example, Aroclor 1254 concentrations alone.

Finally, the majority of field studies have reported the concentrations of total PCBs in sediments. However, a variety of procedures have been used to calculate these concentrations. For example, Bay *et al.* (1994) measured the concentrations of 80 PCB congeners and calculated total PCBs as the sum of these measurements. In other studies, total PCBs has been calculated as the sum of the concentrations of Aroclor 1242 and Aroclor 1254 (e.g., Anderson *et al.* 1988). In other cases, the concentration of total PCBs has been reported, but no information was provided on the procedures that were used to calculate the value. Therefore, measurements of total PCBs in sediment samples may vary depending on which procedure has been used to calculate the concentrations.

In this review, data were compiled separately for Aroclor 1254 and total PCBs in bed sediments. No attempt was made to determine the relationship between these two variables in the Southern California Bight. As such, total PCBs were not estimated from Aroclor 1254 measurements or vice versa. Instead, data from studies which reported Aroclor 1254 concentrations were compiled together. Likewise, data on the effects of total PCBs were compiled together, regardless of which procedure was used to calculate these values. The SECs for each group of substances were derived separately from the appropriate toxicological data sets.

4.2 Toxic Effects of Aroclor 1254

4.2.1 Spiked-Sediment Bioassay Data

A total of five studies were located which examined the effects of Aroclor 1254 in spiked-sediment bioassays. While these studies involved three different species which occur, or are closely related to species that occur, in the Southern California Bight, the experimental protocols used and ancillary information reported in several of these studies limit their application in this review. Nonetheless, the results of all of these studies are discussed below to provide the reader with the information needed to evaluate the results.

The results of a study on the effects of Aroclor 1254 indicate that the sand shrimp, *Crangon septemspinosa*, is relatively insensitive to sediment-associated PCB mixtures (McLeese and Metcalfe 1980). In this experiment, adult sand shrimp were exposed to two different PCB mixtures, Aroclor 1242 and 1254, for a period of 96 hours. As no mortalities were observed during the course of this test, it was not possible to calculate definitive median lethal concentrations for either of these formulated PCB products. Therefore, 96-hour LC_{50}s of >0.78 and >3.4 mg/kg DW were reported for Aroclor 1242 and 1254, respectively (at 0.28% TOC DW). Normalization of these values to 1%OC results in the calculation of 96-hour LC_{50}s of >2.79 and >12.1 mg/kg $DW_{1\%OC}$ for these formulated PCBs, respectively. It should be noted that no information was provided to assess whether or not equilibrium conditions were achieved in the test chambers and that only three organisms per treatment were tested in this study.

The results of two long-term experiments conducted on the sandworm, *Nereis diversicolor*, suggest that relatively high concentrations of a PCB mixture are required to affect this species. In the first test, Polikarpov *et al.* (1983) exposed sandworms to concentrations of a PCB, identified as DP5 (which is a formulated PCB product; B. Dexter. E.V.S. Consultants. Seattle, Washington. Personal communication), ranging from 18.7 to 89.6 mg/kg DW for 128 days. The concentration of DP5 in reference sediments was 0.19 mg/kg DW. High mortality was observed in all of the test groups, with 50% mortality occurring over 31.5 to 48.5 days at the various treatment concentrations. By comparison, 50% mortality occurred in reference sediments after 62.5 days. In the second test, significant adverse effects on survival were observed at 14.7 mg DP5/kg DW, but not at 1.7 mg/kg DP5 DW. The authors reported that sediments with 3.9 and 6.5 mg DP5/kg DW were only slightly toxic. Growth rate (as indicated by changes in body weight) was not a useful indicator of the toxic effects of the DP5 in either of these tests. It is important to note that no information was provided on

the composition of this PCB mixture, the experimental methods in the bioassays, or the characteristics of the sediments that were used in this study. Therefore, these data were not used to derive sediment effect concentrations of PCBs.

In a more recent study, Plesha *et al.* (1988) examined the toxicity of a mixture of four chlorinated hydrocarbons to the infaunal amphipod, *Rhepoxynius abronius*. The chlorinated hydrocarbons used in this investigation included Aroclor 1254, hexachlorobutadiene, 2,2-bis(*p*-chlorophenyl)-1,1,1-trichloroethane (*p,p'*-DDT), and hexachlorobenzene, at nominal concentrations of 1.0, 0.10, 0.20, and 0.05 mg/kg DW. The results of acute toxicity bioassays, in which mortality was the endpoint measured, indicated that the sediments with 1.0 mg/kg DW of Aroclor 1254 (at 0.9% OC) were significantly toxic to amphipods. Much lower survival rates were observed when the amphipods were exposed to sediments that had higher nominal concentrations of chlorinated hydrocarbons (5.0 mg/kg DW of Aroclor 1254). The results of this study suggest that Aroclor 1254 is toxic in the 1 mg/kg DW range, when it is present in mixtures of chlorinated hydrocarbons.

Swartz *et al.* (1988) conducted acute lethality bioassays on the infaunal amphipod, *Rhepoxynius abronius*, to assess the toxicity of Aroclor 1254, both alone and in mixtures with other substances. These investigators reported a 10 day LC_{50} of 8.8 mg/kg DW and a 10 day LC_{10} of 5.45 mg/kg DW for this formulated mixture of PCBs (at 0.41% OC; R. Swartz. USEPA. Newport, Oregon. Personal communication). Normalization of these results to 1%OC results in the calculation of a 10 day LC_{50} of 21.5 mg/kg $DW_{1\%OC}$ and a 10 day LC_{10} of 13.3 mg/kg $DW_{1\%OC}$ for Aroclor 1254.

A significant increase in mortality (to 55% in a 10 day test) was observed when amphipods were exposed to sediments (0.39% OC DW) containing fluoranthene (2.10 mg/kg DW) in combination with Aroclor 1254 (2.1 mg/kg DW; Swartz *et al.* 1988). However, significant mortality did not occur when Aroclor 1254 was present alone at this concentration (mortality <10%) or in combination with zinc (at 138 mg/kg DW; mortality <10%). These results indicate that the toxicity of Aroclor 1254 is enhanced by the presence of fluoranthene. Normalization of these results to 1%OC yields a calculated 10 day LC_{50} of <5.38 mg/kg $DW_{1\%OC}$ for Aroclor 1254 in the presence of 5.38 mg/kg $DW_{1\%OC}$ of fluoranthene.

More recently, the lethal and sublethal effects of Aroclor 1254 on the sediment-dwelling copepod, *Microarthridion littorale*, were investigated in a study that employed Aroclor 1254 spiked-sediments (DiPinto *et al.* 1993). In the initial acute toxicity bioassays, male and female copepods were exposed, separately, to concentrations ranging from 21 to 333 mg/kg of Aroclor 1254 for a period of 96

hours. The results of this study indicated male copepods were more sensitive to this PCB mixture than were female copepods, with 96 hour LC_{50}s of 117 and 251 mg/kg DW reported, respectively (at 3.8 to 4.0% OC). Normalization of these median lethal concentrations to 1%OC results in the calculation of 96 hour LC_{50} of 30.0 and 64.4 mg Aroclor 1254/kg $DW_{1\%OC}$ for male and female copepods, respectively. The lowest observed effect level (LOEL) for acute lethality was 83 mg/kg DW for Aroclor 1254 or 21.3 mg Aroclor 1254/kg $DW_{1\%OC}$.

In a related experiment, DiPinto *et al.* (1993) conducted short-term chronic bioassays to evaluate the effects of Aroclor 1254 on copepod reproduction. In these tests, copulating pairs of copepods were allowed to reproduce in PCB-contaminated sediments, in which Aroclor 1254 concentrations ranged between 4 and 83 mg/kg. The results of these tests, which were 12 days in duration, indicated that reproductive impairment can occur at relative low concentrations of sediment-associated Aroclor 1254. In the first of two experiments, the production of eggs and total reproductive capacity was significantly reduced when the adult copepods were exposed to Aroclor 1254 concentrations as low as 4.2 mg/kg DW. Reduced nauplii (i.e., larval copepods) production was observed in copepods exposed to 8.3 mg/kg DW. Similar trends of decreasing nauplii production and total reproductive capacity with increasing Aroclor 1254 concentrations were also observed in the second experiment, although these latter results were not statistically significant. Normalization of the lowest effective treatment concentration to 1%OC results in a calculated LOEL of 1.08 mg Aroclor 1254/kg $DW_{1\%OC}$. It should be noted that the shallow layer of sediment used in these bioassays tended to minimize contaminant exposure (DiPinto *et al.* 1993). Consequently, these data may underestimate the toxicity of Aroclor 1254 to this organism.

4.2.2 Field Studies

In the past 15 years, several studies have been conducted in the Southern California Bight to evaluate the environmental concerns associated with contaminated sediments. In three of these investigations, matching data on biological effects and the concentrations of Aroclor 1254 were collected (Word and Mearns 1979; Anderson *et al.* 1988; Swartz *et al.* 1991). These three studies on the Southern California Bight were reviewed to support the evaluation of sediment effect concentration for PCBs.

In 1977, Word and Mearns (1979) collected sediment samples from a total of 70 stations between Point Conception and the United States-Mexico border. At each station, two samples were collected for analysis of benthic invertebrate community characteristics and one sample was collected for sediment chemistry. Total PCB and Aroclor 1254 concentrations were measured in the samples collected at each station. All of these samples were collected from a water depth of approximately 60 metres.

The results of this study indicate that benthic invertebrate community structure may be affected by sediment-associated Aroclor 1254 (Word and Mearns 1979). For example, low arthropod abundance (4.0 ± 2.5 N/0.1 m^2) was observed in sediments that had, on average, 3.31 ± 2.27 mg/kg DW of Aroclor 1254. Higher densities of arthropods (144 ± 42.7 N/0.1 m^2) were observed at sites with 0.052 ± 0.070 mg Aroclor 1254/kg DW. Several other benthic invertebrate community metrics including species richness, infaunal index, and echinoderm abundance, were influenced by the concentrations of Aroclor 1254 in sediments. Neither polychaete abundance nor total benthic invertebrate abundance were reduced at the sites with elevated levels of Aroclor 1254. It was not possible to OC-normalize these results, as TOC was not reported in this study.

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes Shelf and in Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10 day) toxicity tests using amphipods, *Rhepoxynius abronius*, were conducted on each of the core sections. The results of this investigation indicated that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The mean concentration of Aroclor 1254 in these sediment samples was 10.6 ± 5.34 mg/kg DW or 1.24 ± 0.437 mg/kg $DW_{1\%OC}$. Moderate toxicity to amphipods (35.9 ± 12.1% mortality) was observed in shallower and deeper sediments (2.5 - 12.5 cm and 37.5 to 47.5 cm); these samples had, on average, 2.61 ± 6.10 mg/kg DW (0.456 ± 0.245 mg/kg $DW_{1\%OC}$) of Aroclor 1254. The samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average Aroclor 1254 concentrations of 0.322 ± 0.731 mg/kg DW (0.110 ± 0.112 mg/kg $DW_{1\%OC}$).

In 1987, Anderson *et al.* (1988) sampled sediments from seven sites in the vicinity of Palos Verdes and three sites in San Diego Harbor. Matching sediment chemistry and biological effects data were generated for each of these sites. The sediment chemistry data consisted of information on over 50 substances, including Aroclor 1254. The biological effects information collected during this survey were

generated using three laboratory bioassays (Microtox, amphipod, and sea urchin), as well as benthic invertebrate community analyses. The Microtox data (which are based on measurements of bacterial luminescence) were not considered because the ecological relevance of this test is uncertain. Only the data from the Palos Verdes area was used in this assessment.

The survival and reburial of the amphipod, *Grandidierella japonica*, in Southern California Bight sediments (in 10 day acute toxicity tests) was not correlated with concentrations of Aroclor 1254 in the Anderson *et al.* (1988) study. For example, the mean concentration of Aroclor 1254 in sediments that were toxic to amphipods was 0.238 ± 0.237 mg/kg DW (0.085 ± 0.097 mg/kg $DW_{1\%OC}$). By comparison, the mean concentration of Aroclor 1254 was 0.240 ± 0.455 mg/kg DW (0.075 ± 0.097 mg/kg $DW_{1\%OC}$) in sediments that did not affect the survival of this species. As the concentrations of Aroclor 1254 were similar at the toxic and the non-toxic sites, something other than the concentrations of this PCB formulation must have been responsible for the observed effects.

While amphipods were apparently unaffected by concentrations of Aroclor 1254 in Southern California Bight sediments, the survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, (in 35-day chronic exposure tests) were linked to the concentrations of sediment-associated Aroclor 1254 (i.e., there was concordance between sediment chemistry and the biological effects). The concentration of Aroclor 1254 was 0.484 mg/kg DW at the site that was associated with these biological effects. However, the linkages between these effects and Aroclor 1254 were weaker when the concentrations of this PCB mixture were expressed on an OC-normalized basis. Impairment of sea urchin growth was also observed at sites with elevated concentrations of Aroclor 1254 (0.817 ± 0.472 mg/kg DW or 0.161 ± 0.163 mg/kg $DW_{1\%OC}$).

4.2.3 SECs for Aroclor 1254

Using the spiked-sediment bioassay approach, SECs of 2.1 mg/kg DW and 1.08 mg/kg $DW_{1\%OC}$ were derived for Aroclor 1254.

Rationale: The available spiked-sediment bioassay data include toxicological information on three sediment-resident invertebrate species (including one amphipod species) and, therefore, satisfy the minimum data criteria established in this study. Data from four

spiked-sediment toxicity investigations (McLeese and Metcalfe 1980; Plesha *et al.* 1988; Swartz *et al.* 1988; DiPinto *et al.* 1993) were considered to be acceptable for deriving sediment effect concentrations of Aroclor 1254 in bed sediments. The results of these tests indicated that Aroclor 1254, alone, is acutely toxic to aquatic organisms at concentrations ranging from 8.8 to 251 mg/kg DW. Acutely toxic concentrations of Aroclor 1254 alone, expressed on a dry weight basis at 1%OC, ranged from 21.5 to 64.4 mg/kg $DW_{1\%OC}$.

Few data were available to evaluate the chronic toxicity of Aroclor 1254. However, the short-term chronic toxicity tests on copepod reproduction are relevant to the development of SECs. In the first of two experiments, reductions in the total reproductive capacity and egg production of copepods were observed at concentrations of Aroclor 1254 as low as 4.20 mg/kg DW (1.08 mg/kg $DW_{1\%OC}$; DiPinto *et al.* 1993). In the second experiment, exposure to Aroclor 1254 significantly reduced naupliar output and, perhaps, total reproductive capacity per live female. Although greater variability in the data reduced the statistical power of the second experiment, the trends of decreasing reproductive success with increasing Aroclor 1254 concentrations were similar in the two experiments.

Aroclor 1254 is more toxic to amphipods when it is present in complex mixtures than when it occurs alone. Swartz *et al.* (1988) observed significant mortality (55%) when amphipods were exposed to 2.10 mg Aroclor 1254/kg DW (5.38 mg/kg $DW_{1\%OC}$), in the presence of fluoranthene (at 2.10 mg/kg DW or 5.38 mg/kg $DW_{1\%OC}$). Fluoranthene was the least toxic of the PAHs evaluated by Long *et al.* (1995a; i.e., it had the highest ER-M). Therefore, Aroclor 1254 would be expected to be more toxic in mixtures with other PAHs at 2.1 mg tPAH/kg DW than in mixtures with fluoranthene alone. Bay *et al.* (1994) reported that tPAH concentrations ranged from 0.756 to 10.6 mg/kg DW in Southern California Bight sediments. Anderson *et al.* (1988) observed tPAH concentrations as high as 20.4 mg/kg DW. Therefore, PAHs occur in Southern California Bight sediments at concentrations that would be expected to enhance the toxicity of Aroclor 1254. Together, these data suggest that Aroclor 1254

may be acutely toxic to amphipods at concentrations as low as 2.10 mg/kg DW (5.38 mg/kg $DW_{1\%OC}$).

The lowest observed effect level (2.10 mg/kg DW) of Aroclor 1254 from the acute toxicity tests on amphipod survival was adopted as the DW-normalized SEC (Swartz *et al.* 1988). The OC-normalized SEC for Aroclor 1254 was the LOEL (1.08 mg/kg $DW_{1\%OC}$) obtained from the short-term chronic toxicity tests on copepod reproduction (DiPinto *et al.* 1993).

The SECs for Aroclor 1254 are supported by the results of additional spiked-sediment bioassays which indicate toxic effects at similar concentrations of this PCB mixture. For example, Swartz *et al.* (1988) reported a 10-day lethal threshold concentration (LC_{10}) of 5.45 mg/kg DW or 13.3 mg/kg $DW_{1\%OC}$ for the amphipod, *Rhepoxynius abronius*. In mixtures with other organic (fluoranthene) and inorganic (zinc and mercury) substances, significant toxicity to this amphipod species was observed at 2.10 to 4.60 mg/kg DW (5.38 to 9.02 mg/kg $DW_{1\%OC}$). Similarly, Plesha *et al.* (1988) reported significant toxicity to amphipods in 10 day toxicity tests at concentrations of Aroclor 1254 as low as 1.00 mg/kg DW or 1.11 mg/kg $DW_{1\%OC}$, when it was present in mixtures of chlorinated hydrocarbons. Greater than 80% mortality to amphipods was observed at 5.00 mg/kg DW (5.56 mg/kg $DW_{1\%OC}$) of Aroclor 1254 in the same mixtures of chlorinated hydrocarbons. As sediments on the Palos Verdes Shelf also contain complex mixtures of chlorinated hydrocarbons (e.g., DDTs; Swartz *et al.* 1991), PAHs (Anderson *et al.* 1988), and metals (Mearns *et al.* 1991), the data from spiked-sediment bioassays for mixtures of contaminants are likely to be relevant for establishing the SECs.

4.2.4 Evaluation of the SECs for Aroclor 1254

Reliability:

The reliability of the SECs for Aroclor 1254 were evaluated using the toxicological data sets that were assembled for the Southern California Bight. The results of this assessment indicate that the dry-weight

normalized SEC for Aroclor 1254 provides a reliable basis for classifying sediment samples as toxic or non-toxic (Table 20). Thirteen of the 14 samples with Aroclor 1254 concentrations at or above the SEC were found to be toxic, based on the results of laboratory toxicity tests and benthic invertebrate community assessments. Importantly, lower values of Aroclor 1254 would also have provided reliable SECs; the incidence of toxicity was 95% and 94% in sediment samples with Aroclor 1254 concentrations at or in excess of 0.4 and 1.15 mg/kg DW, respectively. These results indicate that adverse effects on sediment-dwelling organisms are likely to occur at Aroclor 1254 concentrations well below the SEC. Therefore, it would be appropriate to establish the SEC at a lower level for Aroclor 1254.

Although the data are limited, the results of this assessment show that the OC-normalized SEC also provides a reliable basis for classifying sediment samples in the Southern California Bight (Table 21). All of the sediment samples with Aroclor 1254 concentrations at or in excess of 1.08 mg/kg $DW_{1\% OC}$ (i.e. 5 of 5 samples) were found to be toxic to sediment-dwelling organisms. These results also indicate that a lower SEC could have been established for Aroclor 1254. For example, 18 of the 19 (95%) sediment samples with Aroclor 1254 concentrations at or above 0.20 mg/kg $DW_{1\% OC}$ were toxic. Therefore, the lower value would have provided a reliable SEC for Aroclor 1254.

Predictability:

No data were located for evaluating the predictability of the dry weight-normalized or organic carbon-normalized SECs for Aroclor 1254. All of the data sets compiled for marine and estuarine sites outside the Southern California Bight had Aroclor 1254 concentrations well below the recommended SECs (Table A5-5). However, a few of the samples collected in two of these studies (Word *et al.* 1988; Munns *et al.* 1991) had Aroclor 1254 levels that exceeded the lower values that were evaluated above. Only one toxicity test was conducted in each of these studies, an acute lethality test with amphipods. The results shown in Tables 22 and 23 indicate that neither of the values (i.e., 0.4 mg/kg DW and 0.2 mg/kg $DW_{1\% OC}$) provided an accurate basis for classifying sediment samples based on

Table 20. An evaluation of the reliability of dry weight-normalized SECs for Aroclor 1254.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (Aroclor 1254 > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.40 mg/kg DW	Word & Mearns 1979	42	6	6	100.0
	Anderson et al. 1988	9	2	2	100.0
	Swartz et al. 1991	31	18	17	94.4
	Overall	40	20	19	95.0
1.15 mg/kg DW	Word & Mearns 1979	42	5	5	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	15	14	93.3
	Overall	40	16	15	93.8
2.10 mg/kg DW	Word & Mearns 1979	42	3	3	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	14	13	92.9
	Overall	40	14	13	92.9

Table 21. An evaluation of the reliability of organic carbon-normalized SECs for Aroclor 1254.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (Aroclor 1254 > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.20 mg/kg DW (1% OC)	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	18	17	94.4
	Overall	40	19	18	94.7
1.08 mg/kg DW (1% OC)	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	5	5	100.0
	Overall	40	5	5	100.0
2.00 mg/kg DW (1% OC)	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	1	1	100.0
	Overall	40	1	1	100.0

Table 22. An independent evaluation of the predictability of dry weight-normalized SECs for Aroclor 1254.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (Aroclor 1254 > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.40 mg/kg DW	Word et al. 1988	20	1	1	100.0
	Munns et al. 1991	29	2	0	0.0
	Overall	49	3	1	33.3
1.15 mg/kg DW	Word et al. 1988	20	0	0	NA
	Munns et al. 1991	29	0	0	NA
	Overall	49	0	0	NA
2.10 mg/kg DW	Word et al. 1988	20	0	0	NA
	Munns et al. 1991	29	0	0	NA
	Overall	49	0	0	NA

Toxic samples were identified based on toxicity to amphipods, impaired fertilization of sea urchin gametes, or reduced amphipod abundance.

Table 23. An independent evaluation of the predictability of organic carbon-normalized SECs for Aroclor 1254.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (Aroclor 1254 > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.20 mg/kg DW (1% OC)	Word et al. 1988	20	4	1	25.0
	Overall	20	4	1	25.0
1.08 mg/kg DW (1% OC)	Word et al. 1988	20	0	0	NA
	Overall	20	0	0	NA
2.00 mg/kg DW (1% OC)	Word et al. 1988	20	0	0	NA
	Overall	20	0	0	NA

Aroclor 1254 concentrations. However, these results should not be considered to be definitive due to the limited number of samples that were considered.

4.3 Toxic Effects of tPCBs

4.3.1 Spiked-Sediment Bioassay Data

While data from five spiked-sediment bioassays provide information for evaluating the toxic effects of Aroclor 1254, no dose-response data from controlled laboratory studies were located on tPCBs.

4.3.2 Field Studies

In the past 15 years, several studies have been conducted in the Southern California Bight to evaluate the environmental concerns associated with contaminated sediments. One of these studies was conducted in the late 1970's (Word and Mearns 1979) and several follow-up surveys have been implemented to assess trends in contamination or toxicity (Anderson *et al.* 1988; Swartz *et al.* 1991; Bay *et al.* 1994; Murdoch *et al.* In press; Sapudar *et al.* 1994; Fairey *et al.* 1996; Fairey 1997). A total of eight studies with matching chemistry and biological effects data from the Southern California Bight were reviewed to support the evaluation of sediment effect concentration for PCBs. It should be noted that data from field surveys provide information that can be used to link contaminant concentrations to adverse biological effects; however, these data do not provide dose-response information for definitively identifying causality. A variety of factors, other than the concentration of PCBs, could also have contributed to, or been responsible for, the effects observed in these investigations.

In 1977, Word and Mearns (1979) collected sediment samples from a total of 70 stations between Point Conception and the United States-Mexico border. At each station, two samples were collected for analysis of benthic invertebrate community characteristics and one sample was collected for sediment chemistry. Total PCB concentrations were measured in the samples collected at each station. All of these samples were collected from a water depth of approximately 60 metres.

The results of this study suggest that several benthic invertebrate community metrics are correlated with the concentration of sediment-associated tPCB in the Southern California Bight. For example, high (229 ± 60.3/0.1 m^2) and moderate (73.8 ± 40.9/0.1 m^2) densities of echinoderms were present at sites with mean concentrations of 0.015 ± 0.017 and 0.032 ± 0.040 mg tPCB/kg DW, respectively. In contrast, much lower densities of echinoderms (3.1 ± 4.5/m^2) were observed at sites with tPCB concentrations of 1.48 ± 2.77 mg/kg DW. Similarly, low densities of arthropods and low species richness were observed at sites with 4.88 ± 4.09 and 1.05 ± 2.50 mg tPCB/kg DW, respectively. However the densities of polychaetes and total invertebrates were not reduced at elevated tPCB concentrations (2.28 ± 4.29 and 3.38 ± 5.08 mg/kg DW, respectively). As OC concentrations were not reported in this study, it was not possible to normalize the results to 1%OC.

In 1985, Swartz *et al.* (1991) collected sediment cores from four stations on the Palos Verdes Shelf and in Santa Monica Bay. Two cores (up to 50 cm in depth) were collected from each station and cut into 5 cm vertical sections. Acute (10 day) toxicity tests using amphipods, *Rhepoxynius abronius*, were conducted on each of the core sections. The results of this investigation indicated that the mid-depth (12.5 - 32.5 cm) sediments from the stations located nearby the Los Angeles County outfalls were the most toxic to amphipods (78.6 ± 8.7% mortality). The mean concentration of tPCB in these sediment samples was 20.7 ± 13.1 mg/kg DW or 2.37 ± 1.20 mg/kg $DW_{1\%OC}$. Moderate toxicity to amphipods (35.9 ± 12.1% mortality) was observed in shallower and deeper sediments (2.5 - 12.5 cm and 37.5 to 47.5 cm); these samples had, on average, 4.40 ± 9.21 mg/kg DW (0.693 ± 0.575 mg/kg $DW_{1\%OC}$) of tPCB. The samples that were the least toxic to amphipods (8.7 ± 3.3% mortality) had average tPCB concentrations of 0.429 ± 1.02 mg/kg DW (0.140 ± 0.157 mg/kg $DW_{1\%OC}$).

In 1987, Anderson *et al.* (1988) sampled sediments from seven sites in the vicinity of Palos Verdes and three sites in San Diego Harbor. Matching sediment chemistry and biological effects data were generated for each of these sites. The sediment chemistry data consisted of information on over 50 substances, including Aroclor 1242, Aroclor 1254, and tPCBs. Although it was not explicitly stated, it appears as though tPCB concentrations were calculated as the sum of the concentrations of Aroclor 1242 and Aroclor 1254. The biological effects information collected during this survey were generated using three laboratory bioassays (Microtox, amphipod, and sea urchin), as well as benthic invertebrate community analyses. The Microtox data (which are based on measurements of bacterial luminescence) were not considered because the ecological relevance of

this test is uncertain. Only the data from the Palos Verdes area were used in this assessment.

The survival and reburial of the amphipod, *Grandidierella japonica*, in Southern California Bight sediments (in 10 day acute toxicity tests) was not correlated with concentrations of tPCB in the Anderson *et al.* (1988) study. For example, the mean concentration of tPCB was 0.320 ± 0.340 mg/kg DW (0.105 ± 0.109 mg/kg $DW_{1\%OC}$) in sediments that were significantly toxic to this species. The mean concentrations of tPCB in sediments that did not affect amphipod survival was 0.314 ± 0.588 mg/kg DW (0.089 ± 0.135 mg/kg $DW_{1\%OC}$). As the concentrations of these substances were similar at the toxic and the non-toxic sites, something other than the concentrations of PCBs was likely responsible for the observed effects.

The survival, sediment preference (i.e., avoidance behavior), and gonad growth of the sea urchin, *Lytechinus pictus*, (in 35-day chronic exposure tests) were not correlated with the concentrations of sediment-associated tPCBs (i.e., there was no concordance between sediment chemistry and the biological effects). However, sea urchin growth was correlated with concentrations of tPCBs, with significant reductions in growth observed in sediments with mean concentrations of 1.09 ± 0.57 mg/kg DW or 0.211 ± 0.207 mg/kg $DW_{1\%OC}$ of tPCB. The growth of sea urchins was not affected by sediments with 0.096 ± 0.137 mg tPCB/kg DW or 0.061 ± 0.079 mg tPCB/kg $DW_{1\%OC}$.

In a study commissioned by the Santa Monica Bay Restoration Project, Bay *et al.* (1994) collected sediments from 12 sites located off the Palos Verdes Peninsula during June and July, 1992. A station located near Dana Point in Orange County was used as a reference site in this study. This study was designed to evaluate the toxicity of Palos Verdes sediments to infaunal and epibenthic organisms, and included both bulk sediment and porewater bioassays. The toxicity of bulk sediments was assessed in acute (10 day) tests on the amphipod, *Rhepoxynius abronius*, and chronic (28 day and 35 day) tests on the amphipod, *Grandidierella japonica*, and the white sea urchin, *Lytechinus pictus*. As poor survival was observed among all of the treatment groups (including controls) in the amphipod growth bioassay, these data were not considered for identifying sediment effect concentrations of tPCBs. In this survey, the toxicity of porewater was evaluated in short-term (< 2 hour) bioassays using purple sea urchin, *Strongylocentrotus purpuratus*, gametes.

Acute toxicity bioassays were conducted using sediments from five of the 13 sites sampled in the 1992 survey; none on these sediments were toxic to amphipods

(Bay *et al.* 1994). The mean concentration of tPCB at these sites was 1.31 ± 1.35 mg/kg DW or 0.497 ± 0.404 mg/kg $DW_{1\%OC}$. Likewise, avoidance behavior and growth of white sea urchins (as measured by test width; the test is the hard outer covering of the sea urchin) was not affected by any of the sediments tested in this study. However, the weight gain of white sea urchins was affected by exposure to Palos Verdes sediments. Low growth rates (0.002 ± 0.0002 g WW/d) were observed at sites with a mean concentration of 1.94 ± 1.39 mg tPCB/kg DW (0.565 ± 0.407 mg/kg $DW_{1\%OC}$). Higher growth rates (0.005 ± 0.001 g WW/d) were measured in sediments containing lower tPCB concentrations (0.645 ± 0.699 mg/kg DW or 0.281 ± 0.193 mg/kg $DW_{1\%OC}$).

As part of the Bay Protection and Toxics Cleanup Program, sediment samples were collected from a number of locations in San Pedro Bay in 1992, including Los Angeles and Long Beach Harbours (Sapudar *et al.* 1994). The toxicity of each sample was evaluated using two toxicity tests, including 48-hour red abalone (*Haliotus rufescens*) larval development test (using pore water) and 10-day amphipod (*Rhepoxynius abronius*) survival test (using bulk sediments). The data on the effects of pore water on abalone larval development were not used in this evaluation because they provided little ability to discriminate between sampling sites (i.e., nearly 90% of the samples were shown to be toxicity based on the results of this test).

The results of this investigation indicated that sediments from Los Angeles Inner Harbour, Inner Fish Harbour, East Basin, and Alamitos Bay were the most toxic to amphipods. In this study, toxic samples were identified as those in which amphipod survival was more than 20% lower than the average survival in the control treatments (Long *et al.* In review). The average concentration of tPCB in the toxic samples was 0.177 ± 0.152 mg/kg DW or 0.089 ± 0.059 mg/kg $DW_{1\%OC}$. By comparison, tPCB concentrations averaged 0.088 ± 0.092 mg/kg DW or 0.074 ± 0.046 mg/kg $DW_{1\%OC}$ at the non-toxic sites in this survey. These results indicate that amphipod survival was linked to the dry weight-normalized concentrations of tPCB. However, limited concordance was evident between amphipod survival and organic carbon-normalized concentrations of tPCB.

In a related study, sediments were collected from several locations on the Palos Verdes Shelf and in Santa Monica Bay during 1992 and 1993 (Fairey 1997). Matching sediment chemistry and toxicity data were reported for 19 sites in this area. The results of 10-day acute toxicity tests with the amphipod, *Rhepoxynius abronius*, indicated that the sediments from 11 of these sites were acutely toxic. The average concentration of tPCB at these sites was 0.241 ± 0.126 mg/kg DW or 0.104 ± 0.080 mg/kg $DW_{1\%OC}$. At the non-toxic sites, tPCB concentrations

averaged 0.265 ± 0.050 mg/kg DW or 0.158 ± 0.092 mg/kg $DW_{1\%OC}$. The lack of concordance between amphipod survival and tPCB concentrations indicates that this group of substances was not responsible for the observed biological effects. While polychaete survival and growth tests were also conducted in this study, the data were not used in this evaluation because only four sediment samples were tested.

Sediments were also collected at a total of 350 stations in the vicinity of San Diego as part of the Bay Protection and Toxics Cleanup Program between October, 1992 and May, 1994 (Fairey *et al.* 1996). The results of chemical analyses indicated that the sediments in San Diego Bay, Mission Bay, the San Diego River Estuary, and the Tijuana River Estuary contained a variety of contaminants. While most of the areas within San Diego Bay had relatively low levels of PCBs, some of the stations in the middle portion of the bay had relatively elevated PCBs concentrations. In particular, sediment samples collected in the vicinity of the naval shipyards had substantial PCB concentrations gradients and were, therefore, considered to be the most relevant for evaluating the toxicity effects of PCBs. In this study, total PCB concentrations were estimated from the sum of the 18 PCB congeners that are typically measured under the NOAA National Status and Trends Program (NOAA 1991).

Several porewater and solid phase sediment toxicity tests were conducted on each of the samples collected in the vicinity of the naval shipyards in San Diego Bay. Data on amphipod (*Rhepoxynius abronius*) survival, sea urchin (*Strongylocentrotus purpuratus*) fertilization, and sea urchin (*S. purpuratus*) larval development were obtained and used to evaluate the toxicity of sediment-associated PCBs. The results of the polychaete growth and survival test, the bay mussel larval development test, and the red abalone larval development tests were not used due to incomplete designation of toxic samples. The results of the power analyses conducted by Long *et al.* (In review) were used to designate toxic samples for the amphipod and sea urchin tests (i.e., samples were designated as toxic if the response measured differed by more than 20% from that of the control samples).

The results of acute toxicity test showed that sediments taken from the eastern portion of middle San Diego Bay (i.e., nearby the naval shipyards) were generally the most toxic to amphipods. The average concentration of tPCBs at the toxic sites was 0.281 ± 0.395 mg/kg DW (0.228 ± 0.459 mg/kg $DW_{1\%OC}$). By comparison, the average concentration of tPCB in the samples that were not acutely toxic to amphipods was 0.244 ± 0.360 mg/kg DW (0.218 ± 0.350 mg/kg $DW_{1\%OC}$). Because tPCB concentrations were similar in the toxic and non-toxic samples, it is unlikely that tPCBs contributed significantly to the biological effects

that were observed in the middle portion of San Diego Bay. This conclusion is supported by the analyses of the sea urchin fertilization and larval development tests, which showed no clear linkage between toxicity test results and tPCB concentrations.

As part of an investigation to evaluate the effects of organochlorines on the survival, growth, and reproduction of a marine polychaete, Murdoch *et al.* (In press) collected sediment samples from 10 sites along the 60 meter contour on the Palos Verdes Shelf in 1993. The toxicity tests conducted on these sediment samples included acute (10 day) and short-term chronic (20 day) survival and growth tests and a chronic (120 day) reproduction test using the marine polychaete, *Neanthes arenaceodentata*. The results of this study indicated that acute and short-term chronic exposure to tPCB concentrations as high as 31.8 mg/kg DW (3.95 mg/kg $DW_{1\%OC}$) did not adversely affect polychaete survival or growth. The concentration of tPCB averaged 5.85 ± 12.7 mg/kg DW (0.89 ± 1.51 mg/kg $DW_{1\%OC}$) in these non-toxic samples. However, polychaete reproductive success, as indicated by the number of emergent juveniles, was compromised in the sediments that had the highest concentrations of tPCB (16.6 ± 21.5 mg/kg DW; 2.16 ± 2.54 mg/kg $DW_{1\%OC}$). Polychaete reproduction was not impaired in sediments that contained 0.49 ± 0.42 mg/kg DW (0.26 ± 0.13 mg/kg $DW_{1\%OC}$), however.

4.3.3 SECs for tPCBs

Using the weight-of-evidence, SECs of 0.835 mg/kg DW and 0.577 mg/kg $DW_{1\%OC}$ were derived for tPCBs.

Rationale: As indicated previously, no spiked-sediment bioassay data were located that applied directly to tPCB. For this reason, the SECs for tPCB were derived using the WEA.

The summarized toxicological data set on tPCB had 18 effects data records (Table A4-21). The 50th percentile concentration of this data distribution was 1.086 mg/kg DW. There were a total of 110 effects data records in the unsummarized toxicological data set assembled for this group of substances (Table A4-22). Evaluation of these data using the procedures described in Appendix 3 results in an ER-M of 0.835 mg/kg DW.

The lower of these two values was selected as the SEC for tPCB.

Using the summarized and unsummarized data sets assembled on the biological effects of sediment-associated tPCB (OC-normalized), ER-Ms of 0.606 and 0.577 mg/kg $DW_{1\%OC}$ were derived (Table A4-23 and A4-24). The lower of these two values was identified as the SEC for tPCBs.

4.3.4 *Evaluation of the SECs for PCBs*

Reliability:

The reliability of the recommended SECs were evaluated using the matching sediment chemistry and biological effects data for the Southern California Bight that were assembled during this study. The results of this evaluation indicate that the SEC of 0.835 mg/kg DW provides a reliable basis for assessing PCB-contaminated sediments (Table 24). Eighty-seven percent of the sediment samples with tPCB concentrations at or above the SEC were found to be toxic to benthic invertebrates (34 of 39 samples).

Both lower and higher values of tPCBs would also provide a reliable basis for predicting sediment toxicity in the southern California Bight. For example, the incidence of toxicity was 87% (52 of 60 samples) in sediments with tPCB concentrations at or above 0.4 mg/kg DW, suggesting that a lower SEC would be just as reliable as the recommended SEC. Similarly, a higher level of tPCBs also correctly predicted toxicity in 20 of the 21 (95%) sediment samples with tPCB concentrations at or in excess of 2.0 mg/kg DW. The increasing incidence of toxicity with increasing concentrations of tPCB generates additional confidence in the SEC.

The results of this evaluation indicate that the OC-normalized SEC for tPCBs (0.577 mg/kg $DW_{1\%OC}$) is also reliable (Table 25). In the data set for the Southern California Bight, 20 of the 25 (80%) samples with tPCB concentrations at or in excess of the SEC were found to be toxic. However, a lower value was shown to be less reliable, as

Table 24. An evaluation of the reliability of the dry weight-normalized SECs for tPCBs.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tPCB > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.40 mg/kg DW	Word and Mearns 1979	42	6	6	100.0
	Anderson et al. 1988	9	2	2	100.0
	Swartz et al. 1991	31	18	17	94.4
	Bay et al. 1994	13	8	6	75.0
	Murdoch et al. In press	6	4	2	50.0
	Sapudar et al. 1994	103	7	6	85.7
	Fairey et al. 1996	85	14	12	85.7
	Fairey 1997	19	1	1	100.0
	Overall	308	60	52	86.7
0.835 mg/kg DW	Word and Mearns 1979	42	5	5	100.0
	Anderson et al. 1988	9	1	1	100.0
	Swartz et al. 1991	31	16	15	93.8
	Bay et al. 1994	13	6	5	83.3
	Murdoch et al. In press	6	3	2	66.7
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	8	6	75.0
	Fairey 1997	19	0	0	NA
	Overall	308	39	34	87.2
2.00 mg/kg DW	Word and Mearns 1979	42	4	4	100.0
	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	14	13	92.9
	Bay et al. 1994	13	2	2	100.0
	Murdoch et al. In press	6	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	0	0	NA
	Fairey 1997	19	0	0	NA
	Overall	308	21	20	95.2

Table 25. An evaluation of the reliability of the organic carbon-normalized SECs for tPCBs.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tPCB > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.20 mg/kg DW (1% OC)	Anderson et al. 1988	9	2	2	100.0
	Swartz et al. 1991	31	18	16	88.9
	Bay et al. 1994	13	9	6	66.7
	Murdoch et al. In press	6	5	2	40.0
	Sapudar et al. 1994	103	2	1	50.0
	Fairey et al. 1996	85	29	20	69.0
	Fairey 1997	19	1	0	0.0
	Overall	266	66	47	71.2
0.577 mg/kg DW (1% OC)	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	13	12	92.3
	Bay et al. 1994	13	3	3	100.0
	Murdoch et al. In press	6	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	8	4	50.0
	Fairey 1997	19	0	0	NA
	Overall	266	25	20	80.0
1.00 mg/kg DW (1% OC)	Anderson et al. 1988	9	0	0	NA
	Swartz et al. 1991	31	11	11	100.0
	Bay et al. 1994	13	1	1	100.0
	Murdoch et al. In press	6	1	1	100.0
	Sapudar et al. 1994	103	0	0	NA
	Fairey et al. 1996	85	3	2	66.7
	Fairey 1997	19	0	0	NA
	Overall	266	16	15	93.8

indicated by the lower incidence of toxicity (71%; 47 of 66 samples) at tPCB concentrations at or in excess of 0.20 mg/kg $DW_{1\%OC}$. In contrast, a somewhat higher value (1.0 mg/kg $DW_{1\%OC}$) provided a more reliable basis for classifying sediment samples with elevated concentrations of tPCBs (i.e., 94% correct classification; 15 of 16 samples). Once again, the increasing incidence of toxicity with increasing concentrations of tPCB indicates that the SEC is reliable.

Predictability:

The predictability of the SECs for tPCBs was evaluated using matching sediment chemistry and biological effects data from sites located outside of the Southern California Bight (i.e., elsewhere in the United States; Table A5-6). The results of this evaluation indicate that the SEC of 0.835 mg/kg DW provides an accurate basis for predicting the toxicity of tPCBs. Of the 43 sediment samples that had tPCB concentrations equal or greater than the SEC, 42 were found to be toxic. Therefore, 98% of the sediment samples were correctly classified using the recommended SEC (Table 26).

The predictability of higher and lower values were also evaluated to provide a basis for comparison with the SEC. The results of this assessment indicated that a lower value could also be used to accurately classify sediment samples collected outside the Southern California Bight. For example, the incidence of toxicity was 87% in sediment samples with tPCB concentrations at or above 0.4 mg/kg DW (67 of 77 samples were toxic). A higher value was also predictive of sediment toxicity, as indicated by the high incidence of effects (100%; 7 of 7 samples) in sediment samples with tPCB concentrations at or above 2.00 mg/kg DW.

The SEC of 0.577 mg/kg $DW_{1\%OC}$ accurately predicted the toxicity of tPCBs in sediments outside the Southern California Bight. Based on information from laboratory toxicity tests and benthic invertebrate community assessments, all ten of the sediment samples with tPCB concentrations at or above the SEC were found to be toxic (predictability = 100%; 10 of 10 samples; Table 27). An SEC of 0.2 mg/kg $DW_{1\%OC}$ would provide a slighty less accurate basis for predicting the toxicity of tPCB. Forty-nine of the 59 (83%) of the sediment samples that had tPCB concentrations equal to or greater

Table 26. An independent evaluation of the predictability of the dry weight-normalized SECs for tPCBs.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tPCB > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.40 mg/kg DW	Tietjen & Lee 1984	18	6	6	100.0
	Word et al. 1988	20	4	1	25.0
	Pastorok & Becker 1990	15	4	4	100.0
	Munns et al. 1991	19	2	0	0.0
	Casillas et al. 1992	15	4	3	75.0
	Long et al. 1994	61	8	8	100.0
	Rice et al. 1995	20	9	9	100.0
	Long et al. 1995	50	22	19	86.4
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	18	17	94.4
	Overall	332	77	67	87.0
0.835 mg/kg DW	Tietjen & Lee 1984	18	5	5	100.0
	Word et al. 1988	20	0	0	NA
	Pastorok & Becker 1990	15	2	2	100.0
	Munns et al. 1991	19	0	0	NA
	Casillas et al. 1992	15	2	2	100.0
	Long et al. 1994	61	4	4	100.0
	Rice et al. 1995	20	5	5	100.0
	Long et al. 1995	50	11	11	100.0
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	14	13	92.9
	Overall	332	43	42	97.7
2.00 mg/kg DW	Tietjen & Lee 1984	18	0	0	NA
	Word et al. 1988	20	0	0	NA
	Pastorok & Becker 1990	15	0	0	NA
	Munns et al. 1991	19	0	0	NA
	Casillas et al. 1992	15	0	0	NA
	Long et al. 1994	61	2	2	100.0
	Rice et al. 1995	20	1	1	100.0
	Long et al. 1995	50	1	1	100.0
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	3	3	100.0
	Overall	332	7	7	100.0

Table 27. An independent evaluation of the predictability of the organic carbon-normalized SECs for tPCBs.

SEC	Study	Total Number of Samples Collected	Number of Samples Predicted to be Toxic (tPCB > SEC)	Number of Toxic Samples	Predictability of SEC (% Correct)
0.20 mg/kg DW (1%OC)	Word et al. 1988	20	5	1	NA
	Pastorok & Becker 1990	15	4	4	100.0
	Casillas et al. 1992	15	4	3	75.0
	Long et al. 1994	61	8	7	87.5
	Rice et al. 1995	20	7	7	NA
	Long et al. 1995	50	18	15	83.3
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	13	12	92.3
	Overall	295	59	49	83.1
0.577 mg/kg DW (1%OC)	Word et al. 1988	20	0	0	NA
	Pastorok & Becker 1990	15	2	2	100.0
	Casillas et al. 1992	15	2	2	100.0
	Long et al. 1994	61	2	2	100.0
	Rice et al. 1995	20	1	1	NA
	Long et al. 1995	50	2	2	100.0
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	1	1	100.0
	Overall	295	10	10	100.0
1.00 mg/kg DW (1%OC)	Word et al. 1988	20	0	0	NA
	Pastorok & Becker 1990	15	1	1	100.0
	Casillas et al. 1992	15	1	1	100.0
	Long et al. 1994	61	1	1	100.0
	Rice et al. 1995	20	1	1	100.0
	Long et al. 1995	50	0	0	NA
	Chapman et al. 1987	9	0	0	NA
	Long 1997	105	1	1	100.0
	Overall	295	5	5	100.0

than 0.2 mg/kg $DW_{1\%OC}$ were toxic. These results indicate that a lower SEC could be used to accurately classify sediment samples with elevated levels of tPCBs. As was the case in the Southern California Bight, the incidence of toxicity increased with increasing concentrations of tPCB elsewhere in the United States.

4.4 Discussion of the Toxic Effects of PCBs

There are a number of factors that could, potentially, affect the toxicity of sediment-associated PCBs. These factors include species and life stage tested, exposure route(s), duration of exposure, endpoint measured, PCB congeners present, and the characteristics of the sediments under consideration. While it is difficult to evaluate the influence of these factors directly (due to limitations on the available data), the following discussion is provided to identify the potential effects of these factors on the toxicity of PCBs.

No information was located that would directly support an evaluation of the relative sensitivities, to sediment-associated PCBs, of the biota that would normally occur in association with Southern California Bight sediments. However, data from acute toxicity tests (24 to 96 hours in duration) in which aquatic organisms were exposed to water-borne Aroclor 1254 indicate that sensitivities can span several orders of magnitude. For example, the concentrations of Aroclor 1254 causing biological effects ranged from 0.015 mg/L for a natural phytoplankton assemblage (Moore and Harriss 1972) to 9.00 mg/L for the green alga, *Dunaliella tertiolecta* (Luard 1973), when photosynthetic rate was the experimental endpoint measured. In general, invertebrates appear to be somewhat more sensitive than phytoplankton, with median lethal concentrations (LC_{50}s) of Aroclor 1254 ranging from 0.0078 mg/L for the grass shrimp, *Palaemonetes pugio* (Roesijadi *et al.* 1976) to >0.100 mg/L for the oyster, *Crassostrea virginica* (Duke *et al.* 1970). By comparison, no mortality was observed when pinfish, *Lagodon rhomboides*, were exposed to 0.100 mg/L of Aroclor 1254 for a period of 48 hours. Together, these data suggest that aquatic organisms exhibit a wide range of sensitivities to PCBs.

Few data were located with which to evaluate the relative sensitivities of various life stages of marine organisms to PCBs. However, the available information from acute lethality tests indicates that the sensitivities of various life stages can span more than an order of magnitude. For example, the 96 hour LC_{50}s of Aroclor

1254 were 0.0078 mg/L for juvenile grass shrimp and 0.041 mg/L for adults of the same species (Roesijadi *et al.* 1976). Likewise, juvenile fiddler crabs, *Uca pugilator*, were much more sensitive to Aroclor 1254 than the adults of the same species, with LC_{50}s of 0.010 and >0.100 mg/L reported for these life stages, respectively. **This variability in species and life stage sensitivities means that SECs of PCBs, established using limited data only, may not accurately reflect the range of sensitivities that exist among the species that would normally occur in the Southern California Bight. Hence, adverse biological effects on important and sensitive species could occur below the SECs of PCBs developed in this study.**

In coastal ecosystems, aquatic organisms may be exposed to PCBs via a number of routes. First, epibenthic species may be exposed to PCBs through direct contact with the sediments (i.e., dermal exposure) and in their diet (i.e., oral exposure). Contact with the overlying water is expected to be a minor exposure route for these species. Sediment resident organisms may be exposed to PCBs by direct contact with sediment particles and porewater, as well as in their diet. However, dermal exposure may be reduced in tube-dwelling species. Lastly, benthic invertebrates that process sediments are likely to have the highest exposure to PCBs, as they may extract PCBs directly from sediment particles or from microorganisms that they ingest. In addition, they are likely to be exposed by direct contact with sediments and porewater. Therefore, it is important for the toxicological database to include data on species that are exposed to PCBs via various routes to increase the ecological applicability of the SECs subsequently developed.

Duration of exposure is an important factor that affects the results of toxicity tests. Typically, higher concentrations of a contaminant are required to elicit significant effects in short-term exposure tests (e.g., 96 hours) than would be required in longer term tests (e.g., ≥ 10 days). For example, MacDonald (1993) evaluated toxicity data on over forty substances from water-only exposures and reported that short-term LC_{50}s were, on average, 7.7 times higher than long-term LC_{50}s. Therefore, SECs of PCBs derived using data from short-term studies are likely to be higher than those that are based on chronic toxicity studies.

The experimental endpoint measured is an important factor in the evaluation of the toxic effects of PCBs. Exposure to PCBs has been associated with a diverse range of adverse effects in fish and other aquatic biota, including changes in community structure, mortality, reproductive impairment, inhibition of growth and photosynthesis, alteration of metabolic rates, and changes in gross morphology (Moore and Walker 1991). MacDonald (1993) reported that the concentrations of water-borne contaminants that cause adverse biological effects can vary over

several orders of magnitude depending on the endpoint that was measured in the bioassay. Similar patterns of toxicity are likely to occur for sediment-associated contaminants, as well. As more sensitive biological processes are likely to be affected by lower concentrations of PCBs, SECs derived from lethality data could underestimate the toxicity of PCBs in the Southern California Bight.

The analytical procedures used to generate data on PCB concentrations in the field could affect the SECs derived in this study. Derivation of an reliable SEC for PCBs from field data alone is contingent on the use of accurate and precise analytical procedures. Historically, the analytical methods for quantifying PCBs from field samples have been variable (i.e., different extraction and quantification procedures have been used). Therefore, it is likely that the precision and accuracy of these methods were also variable. This variability has the potential to influence the SECs that are derived from these data. However, it is unknown if this variability would result in under- or over-estimates of PCB concentrations. It should be noted, however, that measurements of formulated PCBs or individual congeners are likely to underestimate the concentrations of tPCBs.

Evaluation of the toxicity of PCBs is further complicated by limitations on the availability of dose-response data (i.e., from spiked-sediment bioassays) for most PCB mixtures. For this reason, associative (or co-occurrence) information that links contaminant concentrations to adverse biological effects has also been used in this review. However, interpretation of associative data is complicated by the presence of multiple contaminants in each sediment sample, frequently including metals, PAHs, PCBs, and other substances. While the methodology used in this review provides a means of identifying the contaminants that are implicated in the toxic response, it is difficult to conclusively desegregate the effects of individual chemical contaminants. Hence, the weight-of-evidence approach could overestimate or underestimate the toxicity of individual sediment-associated PCBs in the Southern California Bight. Nonetheless, this approach is considered to be applicable because the PCBs in the Southern California Bight do not occur in isolation; rather they are present in complex mixtures of organic and inorganic contaminants.

Lastly, differences in the toxicity of individual PCB congeners has the potential to affect the SECs for this group of substances. It is difficult to determine the influence of this factor because no data were located on the relative toxicity of the various PCB congeners or mixtures in sediments. Data from water-only exposure studies revealed no clear trends in the relative toxicity of water-borne PCB formulations (Aroclor 1220, 1224, 1248, 1254, and 1260; USEPA 1980b), which suggests that degree of chlorination is not a major determinant in PCB toxicity.

However, it is possible that coplanar PCBs may be the most toxic congeners. If their mode of toxicity is similar to that of dioxins (e.g., 2,3,7,8-tetrachlorodibenzo-*p*-dioxin), then toxic effects on sensitive endpoints, like reproduction and growth, would be expected to occur at concentrations that are much lower than acutely toxic concentrations. Moreover, extended exposure and observation periods would be required to detect these responses. Therefore, data from short-term lethality studies would underestimate the toxicity of these congeners and, hence, underestimate the toxicity of PCB mixtures in the field.

Consideration of all the factors discussed above suggests that the SECs for PCBs are more likely to underestimate than to overestimate the toxicity of these substances. Because the SECs for Aroclor 1254 are based on data from relatively short-term studies (i.e., ≤ 12 days), they probably underestimate the chronic toxicity of this formulation. This would be even more likely if this formulation contained a significant quantity of coplanar PCBs.

Evaluations of both the reliability and predictability indicate that the SECs for Aroclor 1254 are probably too high. For example, SECs of 0.400 mg/kg DW and 0.200 mg/kg $DW_{1\%OC}$ would have been approximately as reliable as the recommended values. These lower values predicted toxicity in Southern California Bight sediments with a high degree of accuracy (95% and 95%, respectively). The SECs for tPCBs provide a reliable basis for evaluating biological effects in field-collected sediments from the Southern California Bight. In addition, the SECs for tPCBs can be used to accurately classify sediment samples from elsewhere in the United States. This agreement between the evaluations of reliability and predictability increase the confidence that can be placed in the SECs.

5.0 Summary and Conclusions

A review of the published and unpublished literature was conducted to evaluate the toxic effects of DDTs and PCBs in sediments. The primary objective of this review was to determine sediment effect concentrations (SECs) of DDTs and PCBs in Southern California Bight sediments. The secondary objective of this study was to determine if the concentrations of DDTs and PCBs in the sediments of the Southern California Bight would cause injury to one or more sediment-dwelling species that occur or would be expected to occur in this area. Injurious effects were considered to include any impairments to benthic organisms or habitats caused by DDTs or PCBs acting alone, or in any combination with other contaminants.

In this review, DDTs were defined as *p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD, and any metabolite or degradation product of these chemicals. The term "PCBs" is defined as all of the polychlorinated biphenyls found in Southern California Bight, plus the degradation products and metabolites of these chemicals.

A tiered strategy was used to derive SECs for DDTs and PCBs in the Southern California Bight. This strategy relied preferentially on data from controlled laboratory studies to establish SECs for these substances (i.e., using the spiked-sediment bioassay approach; SSBA). Information from field studies and other sources were utilized when acceptable data from spiked-sediment bioassays were not available (i.e., SECs were derived using the weight-of evidence approach; WEA). These two approaches were considered to be complementary because cause-and-effect relationships between contaminant concentrations and toxic effects can be established from controlled laboratory experiments. While it is difficult to definitively identify the causes of toxic effects in field studies, SECs derived using the weight-of evidence approach may be more accurate because they reflect the toxicity of complex mixtures of contaminants. This is important in the Southern California Bight because DDTs, PCBs, and other substances occur in complex mixtures.

The SECs developed in this review were evaluated to determine their reliability and predictability. The reliability of the SECs was evaluated to determine if toxic effects could be accurately predicted in sediments from the Southern California Bight. The predictability of the SECs was evaluated to determine if toxic effects could be accurately predicted in sediments from elsewhere in the United States using data on concentrations of DDTs and PCBs alone.

The available data on the toxic effects of SUM DDT (*p,p'*-DDT + *o,p'*-DDT), SUM DDE (*p,p'*-DDE + *o,p'*-DDE), SUM DDD (*p,p'*-DDD + *o,p'*-DDD), total DDT (sum of all six DDT compounds), Aroclor 1254, and total PCBs were summarized in this review. These groups of substances were the focus of this review because there was no evidence to suggest that there were significant differences in the toxicity of the o,p'- and p,p'-isomers of DDT, DDE, or DDD. In addition, insufficient toxicological information was available on the o,p'-isomers of DDTs and the individual PCB congeners to derive SECs. Acceptable dose-response data from controlled laboratory studies (i.e., spiked-sediment bioassays on sediment-dwelling arthropod species) were located for SUM DDT and Aroclor 1254. Therefore, the SECs for these groups of substances were derived using the SSBA. The SECs for the other groups of substances were derived using the WEA. The SECs for DDTs and PCBs were derived using both summarized and unsummarized field data; both values have been reported. The SECs of DDTs and PCBs that were derived in this study are presented in Table 28. The SECs are reported on a dry weight basis and on an OC-normalized basis at three levels of organic carbon to simplify their use.

The results of the evaluations of reliability and predictability indicate that a high degree of confidence can be placed on the SECs that were derived in this study. Importantly, the dry weight-normalized SECs for all six groups of substances (SUM DDT, SUM DDE, SUM DDD, tDDT, Aroclor 1254, and tPCB) were considered to be reliable. That is, the recommended SECs can be used to accurately predict toxic effects in sediments from the Southern California Bight. The SECs for SUM DDT, SUM DDD, tDDTs, and tPCBs all had high predictability (i.e., >80%); insufficient data were available to evaluate the predictability of the SECs for SUM DDE and Aroclor 1254. Therefore, most of the SECs can be used to accurately predict toxic effects in sediments from elsewhere in the United States. The agreement between the results of the reliability and predictability evaluations increase the confidence that can be placed on the SECs. Higher reliability (i.e., 85%) and similar predictability (82%) were obtained for a somewhat higher SEC for SUM DDT (i.e., 0.06 mg/kg DW). A lower SEC for Aroclor 1254 (i.e., 0.4 mg/kg DW) would have been just as reliable (i.e., 95%) and could be used to classify more sediment samples from the Southern California Bight.

The results of these evaluations indicate that the organic carbon-normalized SECs are reliable. The incidence of toxicity was ≥ 80% when any of these SECs were equalled or exceeded in sediments from the Southern California Bight, which indicates that the SECs provide effective tools for assessing sediments from this geographic area. Most of the SECs can also be used to accurately predict toxic

Table 28. A summary of the sediment effect concentrations (SECs) for DDTs and PCBs in in the Southern California Bight.

Substance	Sediment Effect Concentration	Reliability	Predictability
SUM DDT	0.031 mg/kg DW	76%	82%
	0.06 mg/kg DW	**85%**	**82%**
	0.111 mg/kg DW (1%OC)	**91%**	**82%**
	0.333 mg/kg DW (3% OC)	91%	82%
	0.555 mg/kg DW (5% OC)	91%	82%
SUM DDE	***6.58 mg/kg DW***	**97%**	**NA**
	1.58 mg/kg DW (1% OC)	**87%**	**NA**
	4.74 mg/kg DW (3% OC)	87%	NA
	7.90 mg/kg DW (5% OC)	87%	NA
SUM DDD	***0.89 mg/kg DW***	**95%**	**83%**
	0.23 mg/kg DW (1% OC)	91%	71%
	0.40 mg/kg DW (1% OC)	**95%**	**100%**
	1.20 mg/kg DW (3% OC)	95%	100%
	2.00 mg/kg DW (5% OC)	95%	100%
tDDT	***7.15 mg/kg DW***	**95%**	**100%**
	2.00 mg/kg DW (1% OC)	**82%**	**100%**
	6.00 mg/kg DW (3% OC)	82%	100%
	10.0 mg/kg DW (5% OC)	82%	100%
Aroclor 1254	***0.4 mg/kg DW***	**95%**	**33%**
	2.1 mg/kg DW	93%	NA
	0.20 mg/kg DW (1% OC)	**95%**	**25%**
	1.08 mg/kg DW (1% OC)	100%	NA
	0.60 mg/kg DW (3% OC)	95%	25%
	1.00 mg/kg DW (5% OC)	95%	25%
tPCB	***0.835 mg/kg DW***	**87%**	**98%**
	0.577 mg/kg DW (1% OC)	**80%**	**100%**
	1.73 mg/kg DW (3% OC)	80%	100%
	5.40 mg/kg DW (5% OC)	80%	100%

The most highly recommended SECs are shown in bold italics..
NA - Insufficient data available to evaluate predictability.

effects in sediments from elsewhere in the United States. The predictability of the SECs for SUM DDT, tDDTs, and tPCBs ranged between 82% and 100%. Lower predictability (71%) was observed for the SEC for SUM DDD; a higher SEC (0.40 mg/kg $DW_{1\%OC}$) would have been more predictive of toxicity in the independent data set (i.e., 100% predictability). Insufficient data were available to evaluate the predictability of the SECs for SUM DDE and Aroclor 1254, indicating the the concentrations of these substances tend to be much lower elsewhere in the United States than they are in the Southern California Bight. The general agreement between the results of the reliability and predictability evaluations increases the confidence that can be placed on the SECs.

Based on a review of the existing information, it was concluded that the concentrations of DDTs and PCBs in the sediments of the Southern California Bight were sufficient to cause injury to sediment-dwelling organisms. While much of the information reviewed was from studies conducted in the 1970's and 1980's, the most recent data examined indicate that the concentrations of SUM DDT, SUM DDE, SUM DDD, tDDT, and tPCB in surficial sediments (0 to 2 cm) exceeded the SECs derived in this study at many sites (Bay *et al.* 1994; Sapudar *et al.* 1994; Fairey *et al.* 1996; Fairey 1997). Higher concentrations of these groups of substances were observed in deeper sediments (e.g., 7.5 to 47.5 cm) collected from the Palos Verdes Shelf in 1985 (Swartz *et al.* 1991). The concentrations of Aroclor 1254 in deeper sediments also exceeded the SECs derived in this study. The majority of the samples that exceeded the SECs were also toxic, considering amphipod survival, amphipod abundance, or the sea urchin fertilization. These data indicate that the concentrations of DDTs and PCBs in the Southern California Bight were sufficient to cause injury to sediment-dwelling organisms.

A number of limitations on the SECs have been identified throughout this review. It is important to consider these limitations when the SECs are utilized for assessing sediment injury in the Southern California Bight, including:

- The recommended SECs define contaminant concentrations which, if exceeded, have a high probability (i.e., >75%) of adversely affecting sediment-dwelling organisms in the Southern California Bight;

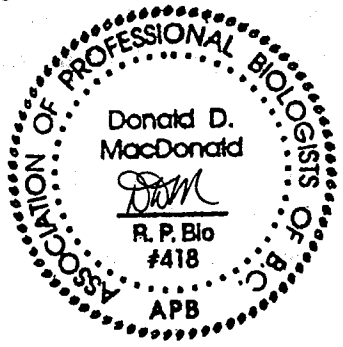
- The recommended SECs do not consider the potential effects of sediment-associated contaminants on fish and other species that reside in the water column;

- The recommended SECs do not consider the potential for adverse effects on higher trophic levels in the food web that could occur due to bioaccumulation;

- The recommended SEC for SUM DDT do not explicitly consider information on the toxicity of SUM DDT when it occurs in mixtures with other contaminants. It is likely that this group of substances would be more toxic when it is present in sediments with other contaminants;

- Because the SECs define contaminant concentrations that are likely to elicit adverse biological effects, remediation objectives would have to be lower to adequately protect sediment-dwelling organisms. Therefore, the recommended SECs should not be used directly as sediment quality remediation objectives or target clean-up levels;

- The recommended SECs were derived using data on a limited number of species and life stages of marine organisms, and, in some cases, using data from short-term tests in which lethality was the endpoint measured. The SECs would likely be lower if additional toxicological data on more sensitive species, more sensitive life stages, more sensitive endpoint, or longer exposure durations were available.



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Appendix 1

Glossary of Terms

Appendix 1

Glossary of Terms

Amphipod - any crustacean of the order Amphipoda (e.g., sand hoppers).

Arthropod - any member of the phyllum arthropoda, which includes crustaceans, insects, arachnids, and centipedes.

Approach - the philosophy and procedures used by a regulatory agency to establish criteria. The components of the approach can include the types of information considered, the management goal underlying the criteria (e.g., protection of aquatic life), relative priorities assigned to various types of information, and the ways that information is combined to set the criteria.

Background concentration - the concentration of a chemical substance that is present in water, sediment, or soil at a site or area that is considered to be relatively unaffected by industrial activity.

Benthic - is a term that refers to an organism that is generally associated with the sediments.

Community metrics - a group of parameters that are calculated to describe the structure of the benthic macroinvertebrate community.

Contact - a term that is used to describe pesticides that act on direct contact with the outside surface of the plant or animal (i.e., it does not have to be ingested).

Contaminant - any chemical substance whose concentration exceeds background concentrations or which is not naturally occurring in the environment.

Criteria - the numerical limits or narrative statements that are recommended to protect and maintain the specified uses of water, sediment, or soil.

Decapod - any crustacean of the order Decapoda (e.g., crabs, lobsters, shrimps, prawns).

Degradation product - a simpler substance that is formed as a result of the breakdown (or degradation) of a chemical.

Dry weight - is the weight of a sediment sample after it has been dried to remove moisture.

Echinoderm - any member of the phylum Echinodermata (e.g., sea urchin).

Epibenthic - a term that refers to an organism that generally lives on the surface of the sediments.

Equilibrium - a term that refers to sediments in which there is no net movement of a substance between the water and the sediments (i.e., the concentrations of the substance in water and sediments are constant over time).

Gonad - ovary or testes.

Guidelines - the numerical limits or narrative statements that are recommended to protect and maintain the specified uses of water, sediment, or soil.

Infaunal - is a term that refers to an organism that generally lives in the sediments.

Invertebrate - an animal without a backbone; with or without a hard external skeleton (e.g., shrimp, worm).

Lipid - fatty substances that are not soluble in water. Certain toxic chemicals are soluble in the lipids of organisms.

Macroinvertebrate - an invertebrate that is visible with the naked eye.

Metabolite - a simpler substance that is formed as a result of the breakdown (metabolism) of a chemical in an organism.

Microorganisms - animals and plants that are not visible to the naked eye.

Microtox™ - a toxicity test that is performed with a bacterium of the genus *Photobacterium*.

Northern puffer - a species of fish (vertebrate).

Objectives - the numerical limits or narrative statements that are established to protect and maintain the specified uses of water, sediment, or soil at a particular site. Objectives may be adopted directly from generic criteria or formulated to account for site-specific conditions.

Organic carbon - includes the nonvolatile organic compounds (sugars), volatile organic compounds (mercaptans, partially volatile compounds (oils), and particulate carbonaceous materials (cellulose) that are contained in sediments. Organic carbon is important because non-ionic compounds tend to partition into it and because it bears surface charges which can interact with other types of contaminants (e.g., ionic compounds). These interactions can affect the toxicity of sediment-associated contaminants.

Polychaete - marine worm.

Porewater - is a term that refers to water that surrounds the sediment particles and fills the voids between sediment particles (also known as interstitial water).

Process sediments - to ingest sediments to obtain the edible material that is present; the sediment is extruded after processing (e.g., worm casts).

Remediation - the management of a contaminated site so as to prevent, minimize, or mitigate damage to human health or the environment. Remediation options may include both direct physical actions (such as removal, destruction, and containment) and institutional controls (such as zoning designations or orders).

Safety factors - unitless digits which are applied to effective concentrations of a contaminant to account for a number of uncertainties associated with the derivation of sediment quality criteria from limited toxicological data sets. Safety factors are used to estimate "safe concentrations" of a substance in sediments to protect sensitive species that are not represented in the toxicological data set or for which the available data are not directly applicable.

Spiked-sediment bioassay - toxicity test in which a contaminant has been added to clean sediments at different concentrations to determine its toxicity to a test organism. Typically the sediments are thoroughly mixed and left to equilibrate before adding the test organism.

Standards - the numerical limits or narrative statements, which are usually adopted from criteria or objectives, that are recognized in the enforceable environmental control laws of one or more levels of government. Standards may be specified in a regulation, statute, contract, or any other legally binding document.

Steady State - a term that refers to sediments in which there is little net movement of a substance between the water and the sediments (i.e., the concentrations of the substance in water and sediments are relatively constant over time; equilibrium conditions have almost been achieved).

Wet weight - is the weight of a sediment sample before it has been dried to remove moisture.

Appendix 2

Glossary of Acronyms

Appendix 2

Glossary of Acronyms

+/-	=	Is the shorthand used when the mean plus or minus one standard deviation is reported
>, <, =	=	Greater than, Less than, Equal to
-	=	Indeterminate
*	=	Hit; see Methods-Appendix 3
µg/kg	=	Micrograms Per Kilogram; Parts Per Billion
µg/L	=	Micrograms Per Liter; Parts Per Billion
ACOE	=	Army Corps of Engineers
ADT	=	Adult
ADT/JUV	=	Adult and/or Juvenile
AET	=	Apparent Effects Threshold
AETA	=	Apparent Effects Threshold Approach
AVS	=	Acid Volatile Sulphides
BEDS	=	Biological Effects Database for Sediments
BDL	=	Below Detection Limit
CAS RN	=	Chemical Abstracts Service Registry Number
CCME	=	Canadian Council of Ministers of the Environment
C.L.	=	Confidence Limit
COA	=	Co-occurrence Analysis
CWA	=	Clear Water Act
d	=	Day
DDTs	=	*p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD and any metabolite or degradation product
DW	=	Dry Weight
EC_{50}	=	Effective Concentration to 50% of the tested organisms
EPA	=	Environmental Protection Agency
EqP	=	Equilibrium Partitioning
EqPA	=	Equilibrium Partitioning Approach
ER-L	=	Effects Range-Low
ER-M	=	Effects Range-Median
ej/prod.	=	Emergent Juveniles per Production
FCV	=	Final Chronic Value
FT	=	Flow-through Test
g	=	Gram
GAM	=	Gamete
h	=	Hour
HAT	=	Hatchlings
I	=	Instar

JUV	=	Juvenile
kg	=	Kilogram
L	=	Liter
LAR	=	Larval
LOEL	=	Lowest Observed Effect Level
LC_0	=	Lethal Concentration to 0% of the tested organisms
LC_{50}	=	Lethal Concentration to 50% of the tested organisms
LPL	=	Lower Prediction Limit
m	=	Month
20-m	=	20 minutes
MFO	=	Mixed-Function Oxidase
mg/kg	=	Milligrams Per Kilogram; Parts Per Million
mm/d	=	Millimeters Per Day; Growth Rate
N	=	Number of Organisms
NC	=	No Concordance
ND	=	Not Detected
NE	=	No Effect
NEO	=	Neonate
NG	=	No Gradient
NOAA	=	National Oceanic and Atmospheric Administration
NRDA	=	Natural Resources Damage Assessments
NSLC	=	National Screening Level Concentration
NSTP	=	National Status and Trends Program
NSTPA	=	National Status and Trends Program Approach
NYM	=	Nymph
OC	=	Organic Carbon
PAHs	=	Polycyclic Aromatic Hydrocarbons
PCBs	=	Polychlorinated Biphenyls plus degradation products and metabolites of these chemicals
ppb	=	Parts Per Billion
ppm	=	Parts Per Million
PSDDA	=	Puget Sound Dredge Disposal Analysis
S	=	Species
SAB	=	Science Advisory Board
SBA	=	Sediment Background Approach
SD	=	Standard Deviation
SDUs	=	Species Diversity Units
SDWA	=	Safe Drinking Water Act
SEC	=	Sediment Effect Concentration
SG	=	Small Gradient
SLCA	=	Screening Level Concentration Approach
sp.	=	Species
spp.	=	Species (plural)

SQ Criteria	=	Sediment Quality Criteria
sq.m.	=	Square meter
SQAGs	=	Sediment Quality Assessment Guidelines
SQO	=	Sediment Quality Objective
SRUs	=	Species Richness Units
SSBA	=	Spike Sediment Bioassay Approach
ST	=	Static Test
SUBADT	=	Sub Adult
SUM DDT	=	*p,p'*-DDT, *o,p'*-DDT
SUM DDD	=	*p,p'*-DDD, *o,p'*-DDD
SUM DDE	=	*p,p'*-DDE, *o,p'*-DDE
TC	=	Threshold Concentration
TLm	=	Median Tolerant Levels
TOC	=	Total Organic Carbon
Total DDT (or tDDT)	=	*p,p'*-DDT, *o,p'*-DDT, *p,p'*-DDE, *o,p'*-DDE, *p,p'*-DDD, *o,p'*-DDD
Total PCBs (or tPCBs)	=	Sum of the concentrations of 209 individual PCB congeners; tPCBs may be estimated from the sum of the concentrations of a subset of the 209 individual congeners
TU/g	=	Toxic Units / gram
USEPA	=	United States Environmental Protection Agency
WEA	=	Weight of Evidence Approach
wk	=	Week
WW	=	Wet Weight

Appendix 3

Methods for Deriving SECs

Appendix 3

Description of the Methods Used to Derive Sediment Effect Concentrations for DDTs and PCBs

A3.1 Spiked-Sediment Bioassay Approach

In the spiked-sediment bioassay approach (SSBA), sediment quality guidelines (SQGs) are derived using empirically generated information on the responses of test organisms to specific contaminant challenges under controlled laboratory conditions. The information that supports this approach is obtained from studies in which uncontaminated sediments are spiked with a range of known contaminant concentrations. By exposing test organisms to these spiked sediments, it is possible to establish quantitative cause-and-effect relationships between chemical concentrations and adverse biological responses. The preferred experimental endpoints in these studies include (Lamberson and Swartz 1992):

(i) mortality;
(ii) growth
(iii) reproduction;
(iv) physiological alterations; and,
(iv) other responses that have demonstrated ecological relevance (see Appendix 4).

Typically, the results of these tests are expressed as the concentrations that cause mortality (LC_{50}) or another effect (EC_{50}) to 50% of the organisms tested over a specific time period (e.g., 10 days). Chemicals may be tested alone or in combination to determine the effects of various concentrations of contaminants in sediment.

In this review, sediment effect concentrations (SECs) have been established preferentially from spiked-sediment bioassay data. Specifically, the lowest observed effect level (LOEL) for the most sensitive species represented in the toxicological database was identified as the SEC (CCME 1994). The LOEL is defined as the lowest concentration of a substance that has been demonstrated to cause an adverse biological effect on a sediment-dwelling organism. Where possible, the results of chronic (i.e., long-term) toxicity tests which measured non-lethal endpoints (such as growth or reproduction) were used in this review. If such data were not available, the results of

acute toxicity tests were used directly to define SECs for DDTs and PCBs.

To ensure that only the highest quality data were used to derive the SECs, the spiked-sediment bioassay data were evaluated prior to incorporation into the toxicological data sets. Data were included in the spiked-sediment bioassay database if:

(i) the contaminant concentrations in the various treatment groups were measured (i.e., estimated concentrations were not used);
(ii) the equilibrium adjustment period (i.e., the time between sediment spiking and toxicity testing) was reported or information was reported to determine how concentrations of the contaminant changed over time;
(iii) adequate environmental conditions were maintained in the test chambers;
(iv) the responses in the control treatments were within accepted limits (e.g., at least 90% for the amphipod, *Rhepoxynius abronius*; ASTM 1994a; 1994b);
(v) the endpoint(s) measured were ecologically-relevant (i.e., likely to influence the organism's viability in the field); and,
(vi) the organism tested was identified and representative of sediment-dwelling species that occur in the Southern California Bight. A test organism was considered to be representative of species that occur in the Southern California Bight if:
    - that species is known to occur in the Southern California Bight; or,
    - similar species are known to occur in the Southern California Bight (SCAMIT 1994).

Only data that met all of these criteria were incorporated into the toxicological data sets that were used to derive the SECs. SECs were derived using this approach only if acceptable data were available from at least one solid phase test on an epibenthic or infaunal arthropod species. This criterion was established to increase the likelihood that SECs would be applicable to sensitive sediment-dwelling species (i.e., species that are exposed to DDTs and PCBs from multiple routes, including direct contact with sediments, direct contact with porewater, and/or processing of sediments to obtain food). The LOEL for the most sensitive life stage of the most sensitive species represented in the toxicological data set was adopted as the SEC for the substance under consideration. This procedure was used to ensure that the SECs would

be as protective as possible. These SECs were expressed on a dry weight basis (mg/kg DW) and on a dry weight basis at various levels of organic carbon (e.g., mg/kg $DW_{1\%OC}$). If insufficient data were available to support the derivation of SECs using the SSBA (i.e., acceptable data on at least one sediment-dwelling arthropod species), then the weight-of-evidence (WEA) was used to derive the SECs.

A3.2 Weight-of-Evidence Approach

When acceptable spiked-sediment bioassay data were not available, SECs were derived using the weight-of-evidence approach (WEA; Long and Morgan 1990; Long 1992; Long *et al.* 1995a; MacDonald 1994). The derivation of SECs using the WEA involved four main steps. First, the available information on the effects of sediment-associated contaminants was identified and retrieved. Next, the available data were evaluated to determine their applicability to the SEC process. Data which met the screening criteria were either incorporated directly or further analyzed prior to incorporation into the appropriate toxicological data sets. Lastly, the information in the various toxicological data sets was evaluated to identify SECs for DDTs and PCBs. The procedures used to derive SECs using the weight-of-evidence approach (MacDonald 1994; Long *et al.* 1995a; MacDonald *et al.* 1996) are described below.

The first step in the SEC derivation process involved the collation of information on the effects of sediment-associated contaminants. To this end, more than ten bibliographic databases were searched for relevant published information. In addition, over 300 scientists were contacted by telephone or letter to obtain additional publications. In total, more that 800 potentially-relevant references were identified and retrieved. These references included data from spiked-sediment bioassays and field studies of sediment toxicity.

To ensure that only the highest quality data were used to derive the SECs, all of the data that were retrieved during this study were critically evaluated. Data from individual studies were considered to be acceptable for deriving SECs if:

(i) the study was conducted in a marine or estuarine area within the Southern California Bight (i.e., Point Conception to the US-Mexico Border);

(ii) matching (i.e., synoptically-collected) biological and chemical data were collected and reported;

(iii) appropriate procedures for collecting, handling, and storing sediments were utilized (ASTM 1994c);

(iv) sediment contaminant concentrations in each sample or treatment group were measured (estimated values were not used;

(v) the concentrations of the contaminants of concern differed by at least a factor of ten among sampling stations (Long and Morgan 1990; Long and MacDonald 1992; Long *et al.* 1995a). This criterion was established to ensure that the data utilized were from areas that had significant gradients in DDT or PCB concentrations. As such, DDTs and PCBs were more likely to be directly associated with the adverse effects reported in the study;

(vi) sediments were not frozen before toxicity tests were initiated (ASTM 1994a; 1994b);

(vii) the responses in the control treatments were within commonly accepted limits (e.g., at least 90% for the amphipod, *Rhepoxynius abronius* ASTM 1994a; 1994b);

(viii) adequate environmental conditions were maintained in test chambers during toxicity testing (ASTM 1994a; 1994b). This criterion was included to ensure that the biological responses were not caused by, for example, low dissolved oxygen levels or elevated water temperatures;

(ix) the endpoint(s) measured were ecologically-relevant (i.e., likely to influence the organism's viability in the field); and,

(x) the organism tested was identified and representative of sediment-dwelling species that occur in the Southern California Bight. A test organism was considered to be representative of species that occur in the Southern California Bight if:

- that species is known to occur in the Southern California Bight; or,
- similar species are known to occur in the Southern California Bight (SCAMIT 1994).

To ensure that the SECs considered a broad range of biological effects, the toxicological information considered in this review included:

(i) measurements of altered benthic communities (depressed species richness or total abundance) in field studies;

(ii) significantly or relatively elevated sediment toxicity in field studies; and,

(iii) effective (e.g. EC_{50}) or lethal (e.g. LC_{50}) concentrations determined in laboratory bioassays of sediments spiked with single chemicals or mixtures of chemicals.

The raw data that passed the initial screening steps were further evaluated and incorporated into the toxicological data sets that were developed for this project. A total of four toxicological data sets were created for each group of chemicals that was considered in this study. Two of the data sets included "summarized" toxicological data, one of which included data expressed on a dry weight basis and the other included data expressed on an dry weight basis at 1% OC. The other two data sets incorporated raw or "unsummarized" toxicological data, one of which included data expressed on a dry weight basis and the other included data expressed on an dry weight basis at 1% OC.

The summarized data sets listed the mean concentrations of DDTs or PCBs in the toxic and non-toxic samples for each endpoint measured in each study. By contrast, the "unsummarized" data sets listed all the concentrations of DDTs or PCBs in the toxic and non-toxic samples for each endpoint reported for each study. Both "summarized" and "unsummarized" data were utilized in this study to optimize the use of the available information. In addition, derivation of SECs using both types of data sets provided a means of assessing the influence of the data analysis procedures on the resultant SECs.

Chemical concentrations were normalized to 1% OC by dividing the original dry weight concentrations by the OC (%) content of the sediment. As such, the OC-normalized contaminant concentrations were reported as mg/kg $DW_{1\%\ OC}$. Organic carbon normalization of non-polar organic contaminant concentrations is considered to account for the influence of organic matter on contaminant bioavailability (Di Toro *et al.* 1991).

Raw data from individual field surveys that passed the initial screening steps were evaluated in "co-occurrence analyses" with either of two methods (Long 1992; Long and MacDonald 1992) prior to incorporation into the "summarized" toxicological data sets. If the statistical significance of the data was reported, then the mean chemical concentrations in the statistical groups (i.e., toxic and non-toxic) were calculated and compared. In some cases, the results of power analyses

conducted for various toxicity tests were used to identify toxic samples [i.e., when the minimum significant difference (MSD) from controls was exceeded; Thursby In prep.; Long *et al.* 1996]. If no such statistical evaluations were reported, the frequency distributions of the biological data were examined and mean concentrations in subjectively determined groups of samples were calculated and compared (e.g., most toxic versus least toxic). The available data from spiked-sediment bioassays were also included in the summarized toxicological data sets.

The raw data from individual field surveys were also evaluated in co-occurrence analyses prior to inclusion in the "unsummarized" toxicological data sets. If the statistical significance of the data was reported, then the concentration of the analyte in each "toxic" sample was compared to the mean concentration of the analytes in the "non-toxic" samples. The calculated MSDs for the various toxicity tests were also used to assign toxic and non-toxic designations to individual sediment samples. If no such statistical evaluations were reported, the frequency distributions of the biological data were examined. Subsequently, the concentration of the analyte in each of the samples that were considered to be toxic was compared to the mean concentration of the substance in the non-toxic samples. The available data from spiked-sediment bioassays were also included in the unsummarized toxicological data sets.

Each entry in all of the data sets was assigned an 'effects/no-effects' descriptor (see Tables A4-1 to A4-24; Long *et al.* 1995a; MacDonald *et al.* 1996). An entry was assigned an 'effects' descriptor (*) if:

(i) an adverse biological effect, such as acute toxicity, was reported; and,

(ii) concordance was apparent between the observed biological response and the measured chemical concentration.

Concordance was apparent when adverse biological effects were observed in association with at least a two-fold elevation in the chemical concentration above reference concentrations. This two-fold criterion provided a consistent basis for identifying chemical concentrations that were associated with the adverse effect that was measured (Long and Morgan 1990; Long and MacDonald 1992; Long *et al.* 1995a). A 'no gradient' (NG) descriptor was assigned when no differences in the concentrations of the chemical of concern was reported between the toxic and non-toxic stations. A 'small gradient' (SG) descriptor was assigned when the concentrations of a substance differed by less than a factor of two between the toxic and non-toxic

samples. A 'no concordance' (NC) descriptor was assigned when there was no concordance between the severity of the effect and the chemical concentration that was measured (i.e., the concentration of a chemical in the toxic samples was lower than the concentration of that substance in the non-toxic samples). For samples that were assigned the NG, SG, and NC descriptors, it was assumed that other factors (whether measured or not) were more important in the etiology of the observed effect than the concentration of the contaminant considered. Finally, a 'no effects' (NE) descriptor was applied to biological data from unaffected, background, or reference samples. Data entries from spiked-sediment bioassays were also assigned an 'effects' descriptor if significant biological effects were reported.

Collectively, the 'effects' data entries from laboratory and field studies were used to derive the SECs. All of the 'effects' data were given equal weight in the derivation of SECs using the WEA. Collectively, data assigned 'no gradient', 'small gradient', 'no concordance', and 'no effects' descriptors were regarded as the 'no-effects' data set. These latter data entries were included in the 'no effects' data set because the concentrations of the substance were not associated with the observed biological effects. Hence, the substance was not considered to be harmful at the concentration that was measured. While the 'no effects' data were not used to derive the SECs, they were used to evaluate the reliability of the resultant SECs.

The distributions of the effects data sets for each substance were determined using percentiles (Byrkit 1975). The median, or 50th percentile, of the effects data was determined and referred to as the Effects Range-Median (ER-M; Long and Morgan 1990; Long *et al.* 1995a). The lower of the ER-Ms derived using the summarized and unsummarized data sets for each substance was adopted as the SEC, when insufficient data were available to support the SSBA. Percentiles of aquatic toxicity data were used by Klapow and Lewis (1979) to calculate marine water quality standards; the authors noted that this approach tended to minimize the influence of single (potentially outlier) data points on the development of guidelines. The SECs derived using the WEA are considered to be appropriate for evaluating sediment quality in the Southern California Bight because there is a high probability of observing adverse effects when this concentration is exceeded (see Long and Morgan 1990; Long 1992; Long and MacDonald 1992; MacDonald 1994; CCME 1994; Long *et al.* 1995a; MacDonald *et al.* 1996 for more complete descriptions of the WEA). Therefore, the SECs are likely to define concentrations of sediment-associated contaminants that would injure sediment-dwelling organisms.

Sediment Injury in the Southern California Bight: Review of the Toxic Effects of DDTs and PCBs in Sediments

Volume II
Appendices 4, 5, and 6

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List of Appendices

Appendix 4

Toxicological Data Set for PCBs & DDTs

Table A4-1. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; summarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		2.1+/-1.15	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		2.1+/-1.15	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.71	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		2.1+/-1.15	Swartz et al. 1985[a]
0.0022 +/- 0.004		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4+/-0.6	Fairey et al. 1996
0.0026 +/- 0.003		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0028 +/- 0.004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.27+/-0.89	Sapudar et al. 1994
0.0028 +/- 0.003		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.87+/-0.88	Fairey et al. 1996
0.0043 +/- 0.002	0.1	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.0046 +/- 0.020	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.66+/-0.6	Fairey et al. 1996
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0070 +/- 0.009		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0074 +/- 0.009		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0077 +/- 0.026	3.0	*	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0078 +/- 0.013	2.8	*	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93+/-1.04	Sapudar et al. 1994
0.0084 +/- 0.028	3.8	*	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.91+/-0.79	Fairey et al. 1996
0.0207 +/- 0.041		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53+/-1.27	Swartz et al. 1985[a]
0.0207 +/- 0.041		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
0.0207 +/- 0.041		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
0.0310		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
0.0425 +/- 0.034		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.10+/-0.91	Fairey 1997
0.0447 +/- 0.051		*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		3.53+/-0.74	Swartz et al. 1985[a]
0.0480 +/- 0.123		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.0486 +/- 0.066		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.81+/-1.1	Bay et al. 1994
0.0530 +/- 0.130		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.0530 +/- 0.130		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.0530 +/- 0.130		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.0537 +/- 0.051		*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		3.34+/-1.38	Swartz et al. 1985[a]
0.0619 +/- 0.058	3.0	*	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	4+/-0.26	Swartz et al. 1985[a]
0.0619 +/- 0.058	3.0	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[a]

Table A4-1. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; summarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0619 +/- 0.058	3.0	*	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[a]
0.0671			*ER-M*							
0.0671 +/- 0.048		*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		3.95+/-0.24	Swartz et al. 1985[a]
0.0671 +/- 0.048		*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		3.95+/-0.24	Swartz et al. 1985[a]
0.0671 +/- 0.048		*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		3.95+/-0.24	Swartz et al. 1985[a]
0.0701 +/- 0.150		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
0.0914 +/- 0.228	2.2	*	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.83+/-1.77	Fairey 1997
0.1170 +/- 0.156		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al. 1994
0.1910 +/- 0.261	26	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
0.1910 +/- 0.261	27	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
0.1914 +/- 0.509		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991[a]
0.2000		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
0.3155 +/- 0.738		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.3155 +/- 0.738		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.3155 +/- 0.738		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.3155 +/- 0.738		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.6720 +/- 1.17		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	2+/-0.95	Bay et al. 1994
0.7490 +/- 1.12	15	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.22+/-1.26	Bay et al. 1994
0.7615 +/- 1.31	6.5	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al. 1994
0.7620 +/- 1.31	0.1	NC	Southern California	COA	35-d	Toxic (0.002+/-0.0003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.08+/-1.41	Bay et al. 1994
1.00		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.86 +/- 1.77	9.7	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991[a]
6.28 +/- 3.07	33	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991[a]
16.5		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	2	McLeese et al. 1982

[a]SUM DDT concentrations have been estimated from the concentrations of p,p'-DDT by dividing by 0.894 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported.
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		3.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		0.9	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		3.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		4.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.2	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.2	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Swartz et al. 1985[a]
ND		NC	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.2	Swartz et al. 1985[a]
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.6	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.48	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.27	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.02	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.9	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.09	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.77	Fairey et al. 1996

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.22	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.86	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.14	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.89	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.7	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.9	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.09	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.07	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.55	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.14	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.75	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.13	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.9	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.6	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.77	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.22	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.02	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.19	Fairey et al. 1996
0.0010 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.77	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.88	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.17	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.95	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.94	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.58	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.16	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.14	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.19	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.28	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.75	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.48	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.13	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.15	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.89	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	NR	2.77	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	NR	1.96	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	NR	2	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	NR	1	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	NR	0.97	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	NR	1.37	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	NR	0.81	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	NR	3.48	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	NR	2.61	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	NR	2.56	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	NR	2.06	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	NR	2.18	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	NR	2.16	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	NR	2.34	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	NR	0.99	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	NR	1	Sapudar et al. 1994
0.0010	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	NR	0.86	Sapudar et al. 1994
0.0010		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0010		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.67	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.35	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.75	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.78	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.19	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.59	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.88	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.11	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.14	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.48	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.27	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.18	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.28	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.03	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.09	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.67	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.78	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.09	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.81	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.37	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.24	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.72	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.78	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.07	Fairey et al. 1996
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	0.9	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	0.61	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	0.7	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	0.6	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	NR	1.5	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	NR	1.6	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	NR	0.38	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	NR	0.39	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	NR	2.9	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	NR	2.5	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	NR	1.5	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	NR	0.8	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	2.5	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	NR	0.4	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	NR	2.3	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	1.56	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	1.22	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	NR	2.13	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	NR	0.76	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	NR	0.72	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	1.03	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	NR	1.05	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	2.5	Sapudar et al. 1994
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	NR	2.34	Sapudar et al. 1994
0.0015	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	NR	2.3	Sapudar et al. 1994
0.0015		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	0.55	Sapudar et al. 1994

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0015		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	0.53	Sapudar et al. 1994
0.0015		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.16	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.16	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.26	Fairey et al. 1996
0.0016		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	NR	2.31	Sapudar et al. 1994
0.0016	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0016	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0016	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.65	Fairey et al. 1996
0.0016	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0017		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	NR	2.1	Sapudar et al. 1994
0.0017		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	NR	0.9	Sapudar et al. 1994
0.0017	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Sapudar et al. 1994
0.0017	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0018		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0018	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	NR	2.51	Fairey 1997
0.0018		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	NR	1.4	Sapudar et al. 1994
0.0020		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	0.31	Sapudar et al. 1994
0.0020	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0020	0.2	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0020		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	NR	0.29	Sapudar et al. 1994
0.0021	0.05	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	NR	2.46	Fairey 1997
0.0021		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	NR	0.8	Sapudar et al. 1994
0.0022		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	NR	0.34	Sapudar et al. 1994
0.0023		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0023		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0023		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0023	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.42	Fairey et al. 1996
0.0023		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.93	Fairey et al. 1996

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0023	1.0	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.42	Fairey et al. 1996
0.0023	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	NR	1.46	Sapudar et al. 1994
0.0023		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	NR	0.25	Sapudar et al. 1994
0.0024	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0024	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0025		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	NR	0.9	Sapudar et al. 1994
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	1.49	Sapudar et al. 1994
0.0026	1.0	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Sapudar et al. 1994
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	NR	0.28	Sapudar et al. 1994
0.0026	0.06	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	NR	2.42	Fairey 1997
0.0027	0.99	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	NR	0.6	Sapudar et al. 1994
0.0027		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	NR	0.7	Fairey 1997
0.0027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027	0.996	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	NR	1.39	Sapudar et al. 1994
0.0030	0.1	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0028	1.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	NR	1.36	Sapudar et al. 1994
0.0029	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.83	Fairey et al. 1996
0.0029		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	NR	1	Sapudar et al. 1994
0.0029		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Sapudar et al. 1994
0.0029	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.98	Fairey et al. 1996
0.0030		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0030		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Bay et al. 1994
0.0030		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0030		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0030		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0030		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0030		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	3.4	Sapudar et al. 1994
0.0030		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	NR	0.5	Sapudar et al. 1994
0.0030	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0031		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.53	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.53	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.9	Fairey et al. 1996
0.0033	1.5	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.88	Fairey et al. 1996
0.0033	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.29	Fairey et al. 1996
0.0033	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	NR	1.1	Fairey 1997
0.0034		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	NR	0.28	Sapudar et al. 1994
0.0035		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0035		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	NR	1.4	Sapudar et al. 1994
0.0036		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	0.29	Sapudar et al. 1994

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0036	0.2	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0036		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Fairey et al. 1996
0.0036	1.4	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.53	Fairey et al. 1996
0.0036	1.6	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.53	Fairey et al. 1996
0.0039	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.58	Fairey et al. 1996
0.0039	1.8	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.58	Fairey et al. 1996
0.0039	1.4	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0040	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	NR	2.86	Sapudar et al. 1994
0.0040	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	NR	1.98	Sapudar et al. 1994
0.0040		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0040		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.07	Fairey et al. 1996
0.0041	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0041	1.9	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0041	1.5	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.27	Fairey et al. 1996
0.0042		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	NR	0.9	Sapudar et al. 1994
0.0042		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	NR	0.49	Sapudar et al. 1994
0.0042		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	NR	1.11	Sapudar et al. 1994
0.0043		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.94	Fairey et al. 1996
0.0043	1.95	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.94	Fairey et al. 1996
0.0043	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Fairey et al. 1996
0.0047	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	NR	1.42	Sapudar et al. 1994
0.0048		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	NR	0.89	Sapudar et al. 1994
0.0051	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	NR	2.3	Sapudar et al. 1994
0.0055		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	NR	1.5	Sapudar et al. 1994
0.0055	2.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.37	Fairey et al. 1996
0.0055		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.37	Fairey et al. 1996
0.0055		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Fairey et al. 1996
0.0056	2.0	*	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	NR	1.28	Sapudar et al. 1994
0.0056		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991[a]
0.0056	2.0	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	NR	1.2	Sapudar et al. 1994
0.0056	2.0	*	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.33	Fairey et al. 1996
0.0058	2.1	*	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	NR	0.69	Sapudar et al. 1994
0.0059	2.3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.75	Fairey et al. 1996
0.0063	2.3	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	NR	1.95	Sapudar et al. 1994
0.0065		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0065	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0066	2.6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.26	Fairey et al. 1996
0.0066		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.26	Fairey et al. 1996

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0066		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Fairey et al. 1996
0.0069	2.5	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	NR	2.85	Sapudar et al. 1994
0.0070	3.1	*	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.28	Fairey et al. 1996
0.0070		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.28	Fairey et al. 1996
0.0070		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0070	0.1	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0070	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0070	0.03	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.0072		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.81	Fairey et al. 1996
0.0072		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0072		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	2.8	Sapudar et al. 1994
0.0074		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0085		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	NR	2	Sapudar et al. 1994
0.0088	3.4	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.93	Fairey et al. 1996
0.0088	3.9	*	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.93	Fairey et al. 1996
0.0088		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.67	Fairey et al. 1996
0.0088	3.2	*	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	NR	3	Sapudar et al. 1994
0.0090	3.5	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.04	Fairey et al. 1996
0.0090		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.0090		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0090		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0090		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0090		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0090		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0097	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	NR	5.3	Fairey 1997
0.0102	3.7	*	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	NR	0.7	Sapudar et al. 1994
0.0105	3.8	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	NR	1.6	Sapudar et al. 1994
0.0110		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0110		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0112		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991[a]
0.0127	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	NR	0.7	Fairey 1997
0.0133		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.38	Fairey et al. 1996
0.0134		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	NR	1.5	Sapudar et al. 1994
0.0134	4.8	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0140		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.8	Bay et al. 1994
0.0140		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0140		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0140		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0140		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0140		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0147	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	NR	5.67	Fairey 1997
0.0154	5.6	*	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	NR	1.4	Sapudar et al. 1994
0.0159		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	NR	1.95	Fairey 1997
0.0168		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991[a]
0.0168		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991[a]
0.0170		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0170		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0170		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0170		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0170		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0225		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	NR	2.8	Fairey 1997
0.0260		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.0260		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.0270		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0270		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0277		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	NR	4.54	Sapudar et al. 1994
0.0289		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	NR	2.92	Fairey 1997
0.0311		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
0.0336		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991[a]
0.0362	13	*	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	NR	2	Sapudar et al. 1994
0.0366	13	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	NR	4.28	Sapudar et al. 1994
0.0389	14	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	NR	4.27	Sapudar et al. 1994
0.0389		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	NR	1.98	Fairey 1997
0.0391		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991[a]
0.0435	16	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	NR	4.3	Sapudar et al. 1994
0.0447		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991[a]
0.0503		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991[a]
0.0553	1.3	SG	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	NR	3	Fairey 1997
0.0581		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	NR	2.79	Fairey 1997
0.0615	1.4	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	NR	1.86	Fairey 1997
0.0617	23	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	NR	4.6	Sapudar et al. 1994
0.0631		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	NR	0.85	Fairey 1997
0.0686	1.6	SG	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	NR	5.06	Fairey 1997
0.0716		*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		3.7	Swartz et al. 1985[a]
0.0716		*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[a]

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0716		*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		3.7	Swartz et al. 1985[a]
0.0716	3.5	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.7	Swartz et al. 1985[a]
0.0716		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.7	Swartz et al. 1985[a]
0.0716	3.5	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
0.0716	3.5	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
0.0716		*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
0.0783		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991[a]
0.0828		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.8	Swartz et al. 1985[a]
0.0828		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.0828		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.0828		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.8	Swartz et al. 1985[a]
0.0828		*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		3.8	Swartz et al. 1985[a]
0.0828		*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.0828		*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.0828		*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		3.8	Swartz et al. 1985[a]
0.1020	2.1	*	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4	Bay et al. 1994
0.1020		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.1020		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.1020		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.1020		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.1020		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.1098		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	NR	2.84	Fairey 1997
0.1121			*ER-M*							
0.1121	43	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.24	Fairey et al. 1996
0.1141	5.5	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.1	Swartz et al. 1985[a]
0.1141		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.1	Swartz et al. 1985[a]
0.1141		*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		4.1	Swartz et al. 1985[a]
0.1141	5.5	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
0.1141	5.5	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
0.1141		*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
0.1141		*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
0.1141		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.1	Swartz et al. 1985[a]
0.1250	48	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.1250	56	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.1250	45	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.37	Fairey et al. 1996
0.1380		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.8	Bay et al. 1994
0.1380		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.1380		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.1380		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.1380	1.2	SG	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.1380		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.1490		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.1490		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Bay et al. 1994
0.1490		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1490		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1490		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1490		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1490		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1750	3.6	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.3	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.1750	1.5	SG	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.2000		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
0.2840	5.9	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.5	Bay et al. 1994
0.2840		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.2840		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.2840		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.2840		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.2840		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.3755		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.3755		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.3755		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.3755		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
0.3755		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
0.3760	19	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.3760	19	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.4360	2.3	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991[a]
0.4520	9.3	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.4754		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991[a]
0.6260	3.3	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991[a]
0.7210	3.8	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991[a]
0.7730	18	*	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	NR	1	Fairey 1997
1.00		*	Laboratory	SSBA	10-d	Toxic (>90% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.14	6	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991[a]
1.28	6.7	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.6	Swartz et al. 1991[a]
1.48	7.8	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991[a]
1.50	7.8	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991[a]
1.91		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991[a]
2.69	14	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991[a]
2.71	14	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991[a]
2.73	23	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
2.73	56	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.1	Bay et al. 1994

Table A4-2. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.73		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Bay et al. 1994
2.73		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
2.73		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
2.73		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
2.73		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
4.57	24	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991[a]
5.37	28	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991[a]
6.26	33	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991[a]
6.64	35	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991[a]
7.46	39	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991[a]
8.70	46	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.7	Swartz et al. 1991[a]
9.84	51	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991[a]
9.98	52	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.2	Swartz et al. 1991[a]
16.5		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	2	McLeese et al. 1982

[a]SUM DDT concentrations have been estimated from the concentrations of p,p'-DDT by dividing by 0.894 (conversion factor was determined from the data reported by Bay et al. 1994).

ND = not detected; detection limit was not reported.

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.

Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-3. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; summarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0016 +/- 0.002		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0017 +/- 0.002		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0024 +/- 0.004		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0024 +/- 0.002	0.1	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0030 +/- 0.003		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0035 +/- 0.004	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0041 +/- 0.013	2.6	*	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0047 +/- 0.003		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0050 +/- 0.003		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054 +/- 0.011		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0054 +/- 0.011		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0054 +/- 0.011		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0093 +/- 0.053	3.9	*	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0115 +/- 0.013		*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0137 +/- 0.028		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0138 +/- 0.013		*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0141 +/- 0.074	8.4	*	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0150 +/- 0.030		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0150 +/- 0.030		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0150 +/- 0.030		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0157 +/- 0.014	2.9	*	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0157 +/- 0.014	2.9	*	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0157 +/- 0.014	2.9	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0172			***ER-M***							
0.0172 +/- 0.012		*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]

Table A4-3. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; summarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0172 +/- 0.012		*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0172 +/- 0.012		*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0194 +/- 0.035		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0210 +/ 0.022		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0229 +/- 0.024		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0454 +/- 0.063	9.1	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0454 +/- 0.063	9.7	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0480 +/- 0.050		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0546 +/- 0.100		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0788 +/- 0.230	3.4	*	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1070 +/- 0.237		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1070 +/- 0.237		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1070 +/- 0.237		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1070 +/ 0.237		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1110		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980
0.2220		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
0.2250 +/- 0.372		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.2400 +/- 0.427	2.2	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2439 +/ 0.359	12	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.4748 +/- 0.455	8.7	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.7992 +/- 0.407	15	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.11		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
8.25		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	1	McLeese et al. 1982

[a]SUM DDT concentrations have been estimated from the concentrations of p,p'-DDT by dividing by 0.894 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
ND		NC	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0003	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0003 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0003 <	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0003		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0004 <	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0004	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0004	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0004 <	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0004 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0004 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005 <	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0005 <	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0005 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0006 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006	0.03	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0006	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006	0.1	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006	0.04	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0006 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0006 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007 <	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0007	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0007	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0007 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0008 <	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008 <	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0008	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0008	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0009 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0009 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009 <	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009 <	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0009		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0009 <	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009 <	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0010 <	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010 <	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0010		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0010	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010 <	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0010 <	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0010	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0010		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0010	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0010	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0011 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0011 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0011 <	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0011	0.05	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0011 <	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0011		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0011	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0012	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0012	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0013		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0013		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0013		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0013		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0014	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0014		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0014	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0014		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0014		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0014	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0015 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0015 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0015	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0015	1.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0016	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0016	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0016	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0016		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0016		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0017		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0017		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0017	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0018	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0018 <	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0019	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0019	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0019		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0019		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0020	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0020	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0020	1.2	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0020	1.4	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0020	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0021	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0021	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0022		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0022	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0022	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0023		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0023		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0023		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0024	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0024	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0024	1.4	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0024	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0024	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0024	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0024	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0025		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0025		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0026		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0026	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0027	0.1	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0027		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0027		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0027		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0027		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0027		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0027		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0028		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0028		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0029		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0029	0.98	NC	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0029	2.0	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0030	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0031	0.2	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0031		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0031	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0032	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0032	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0033	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0036	0.2	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0036		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0036 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0037		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0038		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0038		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0038		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0038		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0038		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0038		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0038		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0039		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0042	2.9	*	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0042		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0043		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0044	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0045	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0046	2.8	*	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0046		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0047	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0047		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0052		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0054		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0060		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991*
0.0060		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0061		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0063		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0063		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0064		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0066	2.2	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0066	2.8	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0069		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0070		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0080		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0081		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0084	2.8	*	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0086	2.8	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0086		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0089		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0091	3.0	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0093		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0096		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0099		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0099		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988

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Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0099		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0101	3.4	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0104		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0109		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0110	3.7	*	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0116		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0121		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0123		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0131		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0134	4.5	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0136	0.6	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0146	4.8	*	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0175		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0175		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0175		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0175		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0175		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0175		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0181	6.0	*	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0181	0.8	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0184	0.8	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0193	3.6	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0193	3.6	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0193	3.6	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0193		*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0193		*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0193		*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0193		*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0193		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0196		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0197		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0208		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0217		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0217		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0218		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0218		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0218		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0218		*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0218		*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0218		*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0218		*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0218		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0230		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0274		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0274		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0278			*ER-M*							
0.0278	5.2	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0278	5.2	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0278	5.2	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0278		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0278		*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0278		*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0278		*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0278		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0283		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0283		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0283		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0283		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0283		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0331	1.4	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0363		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0363		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0363		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0363		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0363		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0363	0.7	NC	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0387		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0407	0.8	NC	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0407	1.97	SG	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0407		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0407		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0407		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0407		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0559		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0613	37	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0613	42	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0621		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0621		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0631	3.1	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0631		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0631		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0631		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0631		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0631		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0683	1.3	SG	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0729	3.5	*	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0729		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0729		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0729		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0729		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0729		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0742		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0797	1.5	SG	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0903	19	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0903	19	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0903		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0903		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0903		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0903		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0903		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1110		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980
0.1488	2.7	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.1614	7.8	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1614		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1614		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1614		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1614		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1614		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1614		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.2220		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
0.2612		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.2863	5.2	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3109		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3207	5.9	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3245	5.9	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3324	6.1	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3378	142	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.4671	322	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.5462	10	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.5749	11	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.6820	13	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.7730	34	*	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.8131	15	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.8800	18	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-4. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDT (mg/kg DW at 1% OC; unsummarized data set).

Sum DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.8813		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.8813		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.8813		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.8813		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.8813		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.8813	43	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.8822	16	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.9187	17	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.9784	18	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.03	19	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.11		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
1.47	27	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.52	28	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
8.25		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	1	McLeese et al. 1982

[a]SUM DDT concentrations have been estimated from the concentrations of p,p'-DDT by dividing by 0.894 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-5. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	23* Species	Life Stage	TOC (%)	Reference
0.0103 +/- 0.018	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.66+/-0.6	Fairey et al. 1996
0.0120 +/- 0.018		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4+/-0.6	Fairey et al. 1996
0.0121 +/- 0.013	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0133 +/- 0.014	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.91+/-0.79	Fairey et al. 1996
0.0134 +/- 0.027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0137 +/- 0.017		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.87+/-0.88	Fairey et al. 1996
0.0230 +/- 0.026		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0230 +/- 0.026		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0632		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[a]
0.0780 +/- 0.081	0.1	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.0969 +/- 0.077		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.27+/-0.89	Sapudar et al. 1994
0.1237 +/- 0.103	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93+/-1.04	Sapudar et al. 1994
0.1242		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1.3	Swartz et al. 1986[a]
0.1650	0.3	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1650	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1650	0.3	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.2032		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		0.8	Ferraro et al. 1991[a]
0.4847 +/- 0.991	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.83+/-1.77	Fairey 1997
0.6000 +/- 1.68		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.6540 +/- 1.79		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.6540 +/- 1.79		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.6540 +/- 1.79		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.6546 +/- 0.751		NE	Southern California	COA		Normal benthic community (74+/-7.99; infaunal index)	Benthic invertebrates		1.35+/-0.07	Swartz et al. 1986[a]
0.8610 +/- 2.07		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
0.9357 +/- 0.805		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		2.1+/-1.15	Swartz et al. 1985[a]
0.9357 +/- 0.805		NE	Southern California	COA		Not toxic (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		2.1+/-1.15	Swartz et al. 1985[a]
0.9357 +/- 0.805		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		2.1+/-1.15	Swartz et al. 1985[a]
0.9763 +/- 1.09		NE	Southern California	COA		High density (54.6+/-5.09 N/0.1 sq.m.)	Amphipods		1.8+/-1.41	Ferraro et al. 1991[a]
1.38 +/- 0.389		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.71	Swartz et al. 1985[a]
1.44 +/- 3.54		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991[b]
1.49 +/- 1.53		NE	Southern California	COA		High density (20.9+/-0.23 N/0.1 sq.m.)	Amphipods		1.7+/-0.61	Swartz et al. 1986[a]
1.49 +/- 1.53		NE	Southern California	COA		High species richness (65.6+/-5.37 S/0.1 sq.m.)	Benthic invertebrates		1.7+/-0.61	Swartz et al. 1986[a]
2.62 +/- 3.47	114	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988

Table A4-5. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	23* Species	Life Stage	TOC (%)	Reference
2.62 +/- 3.47	114	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
2.68 +/- 0.516		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.10+/-0.91	Fairey 1997
2.80 +/- 3.60		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.81+/-1.1	Bay et al. 1994
2.91 +/- 4.01		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
2.91 +/- 4.01		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
2.91 +/- 4.01		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53+/-1.27	Swartz et al. 1985[a]
3.02 +/- 3.15		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al. 1994
3.15	0.7	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Swartz et al. 1986[a]
3.15		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[a]
3.70 +/- 3.67	0.6	NC	Southern California	COA		Low biomass (13.4+/-3.32 g/0.1 sq.m.)	Benthic invertebrates		2.28+/-1.07	Swartz et al. 1986[a]
4.66 +/- 3.83	1.5	SG	Southern California	COA		Low density (358+/-76.8 N/0.1 sq.m.)	Benthic invertebrates		2.42+/-0.98	Swartz et al. 1986[a]
4.66 +/- 3.83		NE	Southern California	COA	10-d	Not toxic (7.8+/-4.44% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42+/-0.98	Swartz et al. 1986[a]
4.77 +/- 4.66		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
4.77 +/- 4.66		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
4.77 +/- 4.66		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
4.77 +/- 4.66		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
4.84 +/- 3.04	77	*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		3.53+/-0.74	Swartz et al. 1985[a]
5.02 +/- 2.84		NE	Southern California	COA		Normal benthic community (62.1+/-15.8; infaunal index)	Benthic invertebrates		3.04+/-1.08	Ferraro et al. 1991[a]
5.02 +/- 2.84		NE	Southern California	COA		High species richness (70.9+/-16.9 S/0.1 sq.m.)	Benthic invertebrates		3.04+/-1.08	Ferraro et al. 1991[a]
5.26 +/- 3.10	42	*	Southern California	COA		Low density (0.2+/-0.14 N/0.1 sq.m.)	Echinoderms		2.64+/-0.77	Swartz et al. 1986[a]
5.27 +/- 3.31	3.8	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		3.34+/-1.38	Swartz et al. 1985[a]
5.53 +/- 4.66		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	2+/-0.95	Bay et al. 1994
5.79 +/- 6.29	0.9	NC	Southern California	COA		Low biomass (11.3+/-4.37 g/0.1 sq.m.)	Benthic invertebrates		3.05+/-1.89	Ferraro et al. 1991[a]
5.81 +/- 3.77		NE	Southern California	COA		High biomas (33.3+/-8.63 g/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.42	Swartz et al. 1986[a]
5.82 +/- 1.21	2.0	*	Southern California	COA		Low biomass (9.37+/-2.78 g/0.1 sq.m.)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[a]
5.82 +/- 1.21	2.0	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[a]
5.82 +/- 1.21	2.0	*	Southern California	COA	10-d	Toxic (21+/-1.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	4+/-0.26	Swartz et al. 1985[a]
6.07 +/- 5.49	1.0	NC	Southern California	COA		Low density (415+/-94.4 N/0.1 sq.m.)	Benthic invertebrates		3.08+/-1.63	Ferraro et al. 1991[a]
6.16 +/- 4.16		NE	Southern California	COA	10-d	Not toxic (11.9+/-5.35% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.34+/-1.3	Ferraro et al. 1991[a]
6.29 +/- 2.43	9.6	*	Southern California	COA		Altered benthic community (53.7+/-3.43; infaunal index)	Benthic invertebrates		2.95+/-0.39	Swartz et al. 1986[a]
6.32 +/- 0.617	6.5	*	Southern California	COA		Moderate density (23.5+/-5.91 N/0.1 sq.m.)	Amphipods		3.77+/-0.42	Ferraro et al. 1991[a]
6.32 +/- 0.617		NE	Southern California	COA		High density (944+/-101 N/0.1 sq.m.)	Benthic invertebrates		3.77+/-0.42	Ferraro et al. 1991[a]
6.55 +/- 0.669		NE	Southern California	COA		High biomass (25.5+/-4.21 g/0.1 sq.m.)	Benthic invertebrates		3.63+/-0.44	Ferraro et al. 1991[a]

Table A4-5. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
6.58			*ER-M*							
6.58 +/- 1.81	7.0	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		3.95+/-0.24	Swartz et al. 1985[a]
6.58 +/- 1.81	7.0	*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		3.95+/-0.24	Swartz et al. 1985[a]
6.58 +/- 1.81	7.0	*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		3.95+/-0.24	Swartz et al. 1985[a]
7.02 +/- 3.67	35	*	Southern California	COA		Low density (2.4+/-5.65 N/0.1 sq.m.)	Echinoderms		3.7+/-0.87	Ferraro et al. 1991[a]
7.33 +/- 1.51	4.9	*	Southern California	COA		Low density (5.3+/-3.7 N/0.1 sq.m.)	Amphipods		3.13+/-0.15	Swartz et al. 1986[a]
7.33 +/- 1.51	4.9	*	Southern California	COA		Low species richness (38.4+/-3.29 S/0.1 sq.m.)	Benthic invertebrates		3.13+/-0.15	Swartz et al. 1986[a]
8.41 +/- 4.19	3.0	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.22+/-1.26	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.73 +/- 5.49	2.9	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.08+/-1.41	Bay et al. 1994
9.47 +/- 4.04	9.7	*	Southern California	COA		Low density (4.13+/-2.5;N/0.1 sq.m.)	Amphipods		3.93+/-1.27	Ferraro et al. 1991[a]
14.1	2.8	*	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
14.1	2.8	*	Southern California	COA		Altered benthic community (3.6; infaunal index)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
19.1 +/- 15.4	13	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991[b]
123 +/- 47.5	85	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991[b]

[a]SUM DDE concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.886 (conversion factor was determined from the data reported by Bay et al. 1994).
See Appendix 2 for glossary of acronyms.
[b]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; (conversion factors were determined from the data reported by Bay et al. 1994). SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0010 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.09	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.37	Fairey et al. 1996
0.0010 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.09	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.07	Fairey et al. 1996
0.0016	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.14	Fairey et al. 1996
0.0016	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.55	Fairey et al. 1996
0.0016	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.19	Fairey et al. 1996
0.0016		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.09	Fairey et al. 1996
0.0016		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.19	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Fairey et al. 1996
0.0018	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.88	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.35	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0019	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0020	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.07	Fairey et al. 1996
0.0020	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0020	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.48	Fairey et al. 1996
0.0020		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.48	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.24	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.67	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.67	Fairey et al. 1996
0.0025	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0026	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0028	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0028	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.6	Fairey et al. 1996
0.0028	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.6	Fairey et al. 1996
0.0029	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.17	Fairey et al. 1996
0.0029	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0029	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0029	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0030	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0030		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.78	Fairey et al. 1996
0.0030		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.78	Fairey et al. 1996
0.0030	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0031	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey et al. 1996
0.0032		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0032	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.89	Fairey et al. 1996
0.0032	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.58	Fairey et al. 1996
0.0032	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.9	Fairey et al. 1996
0.0032	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.9	Fairey et al. 1996
0.0033		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.11	Fairey et al. 1996
0.0034	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0034		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0035	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.95	Fairey et al. 1996
0.0035	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0035	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.77	Fairey et al. 1996
0.0036	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93	Fairey et al. 1996
0.0036	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.13	Fairey et al. 1996
0.0037	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0038	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.09	Fairey et al. 1996
0.0038	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0038	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.02	Fairey et al. 1996
0.0038	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.02	Fairey et al. 1996
0.0038		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.81	Fairey et al. 1996
0.0039	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0039	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0039	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Fairey et al. 1996
0.0039		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.59	Fairey et al. 1996
0.0039		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.26	Fairey et al. 1996
0.0039		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.16	Fairey et al. 1996
0.0039		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.16	Fairey et al. 1996
0.0041	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.13	Fairey et al. 1996
0.0046	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0046	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.75	Fairey et al. 1996
0.0046		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.75	Fairey et al. 1996
0.0047	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.48	Fairey et al. 1996
0.0049	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.7	Fairey et al. 1996
0.0050	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.89	Fairey et al. 1996
0.0050	0.01	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	High biomass (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	High biomass (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0060	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.9	Fairey et al. 1996
0.0060	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.86	Fairey et al. 1996
0.0063		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.18	Fairey et al. 1996
0.0070		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0070		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0070		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0070		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0070		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0070		NE	Southern California	COA	35-d	High biomass (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0070		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0072	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey et al. 1996
0.0074	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.15	Fairey et al. 1996
0.0074	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0076	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.65	Fairey et al. 1996
0.0076	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.75	Fairey et al. 1996
0.0076	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.77	Fairey et al. 1996
0.0076	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.77	Fairey et al. 1996
0.0076		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.72	Fairey et al. 1996
0.0077	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.98	Fairey et al. 1996
0.0078	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.19	Fairey et al. 1996
0.0078	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0078	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.14	Fairey et al. 1996
0.0078		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.14	Fairey et al. 1996
0.0081	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.27	Fairey et al. 1996
0.0081		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53	Fairey et al. 1996
0.0081		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.27	Fairey et al. 1996
0.0084		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.05	Sapudar et al. 1994
0.0085		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.88	Fairey et al. 1996
0.0086		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.28	Fairey et al. 1996
0.0087	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.28	Fairey et al. 1996
0.0087		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.9	Fairey et al. 1996
0.0087		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.53	Fairey et al. 1996
0.0087		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.53	Fairey et al. 1996
0.0087	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0092	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0092	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.94	Fairey et al. 1996
0.0092	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.22	Fairey et al. 1996
0.0092	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.22	Fairey et al. 1996
0.0092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0095		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	35-d	High biomass (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0095		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0099	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.33	Fairey et al. 1996
0.0100		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991*
0.0105	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0109	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.26	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.26	Fairey et al. 1996
0.0112	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0114	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Fairey et al. 1996
0.0114	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.94	Fairey et al. 1996
0.0114		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.94	Fairey et al. 1996
0.0115		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	35-d	High biomass (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0115		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0116	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.16	Fairey et al. 1996
0.0120		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.00	Bay et al. 1994
0.0120		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.00	Bay et al. 1994
0.0122		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0122		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.81	Fairey et al. 1996
0.0123	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0129	0.96	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0130	0.97	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.93	Fairey et al. 1996
0.0130	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.93	Fairey et al. 1996
0.0130		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.67	Fairey et al. 1996
0.0138	1.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0138		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.78	Fairey et al. 1996
0.0138		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.03	Fairey et al. 1996
0.0145	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.29	Fairey et al. 1996
0.0145	1.2	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.88	Fairey et al. 1996
0.0150		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991[a]
0.0154		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.07	Fairey et al. 1996
0.0155		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0157		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.34	Sapudar et al. 1994
0.0160	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Sapudar et al. 1994
0.0160	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.04	Fairey et al. 1996
0.0165	1.4	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.28	Fairey et al. 1996
0.0165		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0165		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.28	Fairey et al. 1996
0.0166	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0168		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.31	Sapudar et al. 1994
0.0169	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0169	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.58	Fairey et al. 1996
0.0169	1.4	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.58	Fairey et al. 1996
0.0178	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.27	Fairey et al. 1996

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0178	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0178	1.5	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0179	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.83	Fairey et al. 1996
0.0181	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.75	Fairey et al. 1996
0.0183	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.86	Sapudar et al. 1994
0.0191	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0191	1.4	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.14	Fairey et al. 1996
0.0194	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.99	Sapudar et al. 1994
0.0209	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.24	Fairey et al. 1996
0.0217	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0236		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.11	Sapudar et al. 1994
0.0256		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0280		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.49	Sapudar et al. 1994
0.0282		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Sapudar et al. 1994
0.0282		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.38	Sapudar et al. 1994
0.0284		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0309		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Sapudar et al. 1994
0.0314		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0357		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.72	Sapudar et al. 1994
0.0367		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0371		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.76	Sapudar et al. 1994
0.0375	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.97	Sapudar et al. 1994
0.0380		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0383	2.9	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.42	Fairey et al. 1996
0.0383	3.2	*	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.42	Fairey et al. 1996
0.0383		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.93	Fairey et al. 1996
0.0392		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0427	3.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.53	Fairey et al. 1996
0.0427	3.5	*	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.53	Fairey et al. 1996
0.0427		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Fairey et al. 1996
0.0442		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0451	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.0464		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0469	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0487	3.5	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.37	Fairey et al. 1996
0.0487	3.6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0487	4	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0489	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.48	Sapudar et al. 1994
0.0493	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.85	Sapudar et al. 1994
0.0509		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.61	Sapudar et al. 1994
0.0510		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.38	Fairey et al. 1996
0.0514		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Sapudar et al. 1994
0.0533	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Sapudar et al. 1994
0.0537		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.53	Sapudar et al. 1994
0.0542	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.81	Sapudar et al. 1994

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0545	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0545	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.67	Fairey 1997
0.0570		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	35-d	High biomass (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0570		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0570	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.06	Fairey 1997
0.0586	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.46	Sapudar et al. 1994
0.0586	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0586	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.46	Fairey 1997
0.0603	4.5	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.37	Fairey et al. 1996
0.0603		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Fairey et al. 1996
0.0603		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.37	Fairey et al. 1996
0.0614	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.3	Fairey 1997
0.0614		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0615		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0627	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey 1997
0.0629	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
0.0632	0.05	NC	Southern California	COA		Low density (616.6 N/0.1 sq.m.)	Benthic invertebrates		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.90	Swartz et al. 1985[a]
0.0632		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.90	Swartz et al. 1985[a]
0.0637	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0640	0.1	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	35-d	High biomass (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0640		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0650		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991[a]
0.0658	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0664		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0665	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0686		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0691		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0692	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.36	Sapudar et al. 1994
0.0700		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991[a]

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.0704	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0711	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Sapudar et al. 1994
0.0716	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey 1997
0.0723		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0752	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.51	Fairey 1997
0.0766		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0786		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0821		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.1	Sapudar et al. 1994
0.0838		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.4	Sapudar et al. 1994
0.0840		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0850		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0857		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.49	Sapudar et al. 1994
0.0890	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0900		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991[a]
0.0942		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.25	Sapudar et al. 1994
0.0979		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.0987	1.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.1009		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.1009		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.1023	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.1056	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.1080		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.55	Sapudar et al. 1994
0.1092	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Fairey 1997
0.1116	8.1	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.1146		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.1149	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.39	Sapudar et al. 1994
0.1197	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.42	Sapudar et al. 1994
0.1200		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991[a]
0.1230		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.1230		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.5	Sapudar et al. 1994
0.1242	0.02	NC	Southern California	COA		Low biomass (11.5 g/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[a]
0.1242	0.04	NC	Southern California	COA		Low density (490 N/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[a]
0.1242		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1.3	Swartz et al. 1986[a]
0.1242		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1.3	Swartz et al. 1986[a]
0.1242		NE	Southern California	COA		High species richness (66.6 S/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[a]
0.1242		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates		1.3	Swartz et al. 1986[a]
0.1242		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.3	Swartz et al. 1986[a]
0.1272	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.69	Sapudar et al. 1994
0.1480	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Sapudar et al. 1994
0.1547	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.06	Sapudar et al. 1994
0.1564	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.16	Sapudar et al. 1994
0.1600	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.56	Sapudar et al. 1994
0.1636		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.1648	0.3	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.1648	0.3	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1648	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1648		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.54	Anderson et al. 1988
0.1650	0.0	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.50	Anderson et al. 1988
0.1650	7.3	*	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.50	Anderson et al. 1988
0.1650	7.3	*	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.50	Anderson et al. 1988
0.1674	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.18	Sapudar et al. 1994
0.1684		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.56	Sapudar et al. 1994
0.1735	1.8	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.61	Sapudar et al. 1994
0.1798		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.1830		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.1833		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.31	Sapudar et al. 1994
0.1888		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.1931		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.1942	2.0	*	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.1950		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.1981	2.0	*	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.96	Sapudar et al. 1994
0.2032	0.03	NC	Southern California	COA		Low biomass (16.1 g/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[a]
0.2032	0.03	NC	Southern California	COA		Low density (398 N/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[a]
0.2032		NE	Southern California	COA		High density (58.2 N/0.1 sq.m.)	Amphipods		0.8	Ferraro et al. 1991[a]
0.2032		NE	Southern California	COA		High species richness (62 S/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[a]
0.2032		NE	Southern California	COA		Normal benthic community (92.4; infaunal index)	Benthic invertebrates		0.8	Ferraro et al. 1991[a]
0.2032		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		0.8	Ferraro et al. 1991[a]
0.2032		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Ferraro et al. 1991[a]
0.2033		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Sapudar et al. 1994
0.2210	2.3	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.2229		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.54	Sapudar et al. 1994
0.2242		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.13	Sapudar et al. 1994
0.2270		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.2374	2.4	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.27	Sapudar et al. 1994
0.2500		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991[a]
0.2520		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.9	Sapudar et al. 1994
0.2726		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.89	Sapudar et al. 1994
0.2800	2.9	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.6	Sapudar et al. 1994
0.2820	2.9	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.3	Sapudar et al. 1994
0.2830	2.9	*	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Sapudar et al. 1994
0.2887	3.0	*	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Sapudar et al. 1994
0.3180		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.4	Sapudar et al. 1994
0.3450		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991[a]
0.3673	3.8	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.28	Sapudar et al. 1994
0.3930		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.60	Bay et al. 1994
0.3930		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.60	Bay et al. 1994
0.3930		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.60	Bay et al. 1994
0.3930		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.60	Bay et al. 1994
0.3930		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.60	Bay et al. 1994

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
0.4730	4.9	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Sapudar et al. 1994
0.5470		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.80	Bay et al. 1994
0.5470		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.80	Bay et al. 1994
0.5470		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.80	Bay et al. 1994
0.5470		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.80	Bay et al. 1994
0.5470		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.80	Bay et al. 1994
0.5470		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.80	Bay et al. 1994
0.5950		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
0.5950		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
0.5950		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
0.5950		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
0.5950		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
0.5950		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.40	Bay et al. 1994
0.7000		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991[a]
0.7300		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991[a]
0.7390	0.2	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.10	Bay et al. 1994
0.7390		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.10	Bay et al. 1994
0.7390		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.10	Bay et al. 1994
0.7390		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.10	Bay et al. 1994
0.7390		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.10	Bay et al. 1994
0.7390		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.10	Bay et al. 1994
0.8010	0.6	NC	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991[a]
1.02		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991[a]
1.10	17	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		High biomass (20.9g/0.1 sq.m.)	Benthic invertebrates		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		2.20	Swartz et al. 1985[a]
1.10		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.20	Swartz et al. 1985[a]
1.19		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1.4	Swartz et al. 1986[a]
1.19		NE	Southern California	COA		High species richness (59.8 S/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[a]
1.19		NE	Southern California	COA		Normal benthic community (68.3; infaunal index)	Benthic invertebrates		1.4	Swartz et al. 1986[a]
1.19		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Swartz et al. 1986[a]
1.19	0.2	NC	Southern California	COA		Low biomass (10.2 g/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[a]
1.19	0.4	NC	Southern California	COA		Low density (355 N/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[a]
1.19	9.6	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1.4	Swartz et al. 1986[a]
1.33		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991[a]
1.57	0.6	NC	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.65	26	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		3.20	Swartz et al. 1985[a]

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
1.65		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA		High density (86 N/0.1sq.m.)	Crustaceans		3.20	Swartz et al. 1985[a]
1.65		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.20	Swartz et al. 1985[a]
1.75	0.3	NC	Southern California	COA		Low biomass (9.2 g/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[a]
1.75	0.3	NC	Southern California	COA		Low density (504 N/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[a]
1.75	8.6	*	Southern California	COA		Low density (15.2 N/0.1 sq.m.)	Echinoderms		2.8	Ferraro et al. 1991[a]
1.75		NE	Southern California	COA		High density (51 N/0.1 sq.m.)	Amphipods		2.8	Ferraro et al. 1991[a]
1.75		NE	Southern California	COA		High species richness (83.2 S/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[a]
1.75		NE	Southern California	COA		Normal benthic community (75; infaunal index)	Benthic invertebrates		2.8	Ferraro et al. 1991[a]
1.75		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Ferraro et al. 1991[a]
1.87		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991[a]
1.98		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Fairey 1997
5.08	223	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
5.08	223	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
2.24		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.20	Bay et al. 1994
2.24		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.20	Bay et al. 1994
2.25	0.8	NC	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.40	Bay et al. 1994
2.25		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1.40	Bay et al. 1994
2.25		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.40	Bay et al. 1994
2.25		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.40	Bay et al. 1994
2.25		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.40	Bay et al. 1994
2.25		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.40	Bay et al. 1994
2.32		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.84	Fairey 1997
2.37		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.92	Fairey 1997
2.43		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
2.52		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.85	Fairey 1997
3.15	1.2	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.86	Fairey 1997
3.15	0.7	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Swartz et al. 1986[a]
3.15	4.8	*	Southern California	COA		Altered benthic community (50.6; infaunal index)	Benthic invertebrates		2.4	Swartz et al. 1986[a]
3.15	25	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		2.4	Swartz et al. 1986[a]
3.15		NE	Southern California	COA		High density (21.2 N/0.1 sq.m.)	Amphipods		2.4	Swartz et al. 1986[a]
3.15		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[a]
3.15		NE	Southern California	COA		High species richness (70.4 S/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[a]
3.15		NE	Southern California	COA		High biomass (39.4 g/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[a]
3.18		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Fairey 1997
3.21		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Fairey 1997
3.41		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.79	Fairey 1997
4.45	1.5	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		4.20	Swartz et al. 1985[a]
4.45	1.5	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		4.20	Swartz et al. 1985[a]
4.45	1.7	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.20	Swartz et al. 1985[a]

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
4.45	3.2	*	Southern California	COA		Low density (661.4 N/0.1 sq.m.)	Benthic invertebrates		4.20	Swartz et al. 1985[a]
4.45	4.7	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		4.20	Swartz et al. 1985[a]
4.45	4.8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.20	Swartz et al. 1985[a]
4.45	4.8	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		4.20	Swartz et al. 1985[a]
4.45	71	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.20	Swartz et al. 1985[a]
5.08		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
5.08		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
5.08		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
5.08		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
5.08		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
5.38	3.7	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991[a]
5.61	1.0	NC	Southern California	COA		Low biomass (17.7 g/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[a]
5.61	1.8	SG	Southern California	COA		Low density (293 N/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[a]
5.61	3.8	*	Southern California	COA		Low density (4.3 N/0.1 sq.m.)	Amphipods		3.3	Swartz et al. 1986[a]
5.61	3.8	*	Southern California	COA		Low species richness (39.3 S/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[a]
5.61	8.6	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates		3.3	Swartz et al. 1986[a]
5.61	45	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.3	Swartz et al. 1986[a]
5.61		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.3	Swartz et al. 1986[a]
5.93	6.1	*	Southern California	COA		Moderate density (23.2 N/0.1 sq.m.)	Amphipods		4.1	Ferraro et al. 1991[a]
5.93	29	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		4.1	Ferraro et al. 1991[a]
5.93		NE	Southern California	COA		High species richness (87.4 S/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[a]
5.93		NE	Southern California	COA		High density (1054 N/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[a]
5.93		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[a]
5.93		NE	Southern California	COA		Normal benthic community (53.4; infaunal index)	Benthic invertebrates		4.1	Ferraro et al. 1991[a]
5.93		NE	Southern California	COA	10-d	Not toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.1	Ferraro et al. 1991[a]
5.95		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.40	Bay et al. 1994
5.95		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.40	Bay et al. 1994
6.00	6.2	*	Southern California	COA		Moderate density (17.8 N/0.1 sq.m.)	Amphipods		3.3	Ferraro et al. 1991[a]
6.00	30	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.3	Ferraro et al. 1991[a]
6.00		NE	Southern California	COA		High species richness (72 S/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[a]
6.00		NE	Southern California	COA		High density (856 N/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[a]
6.00		NE	Southern California	COA		High biomass (31.4 g/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[a]
6.00		NE	Southern California	COA		Normal benthic community (53.3; infaunal index)	Benthic invertebrates		3.3	Ferraro et al. 1991[a]
6.00		NE	Southern California	COA	10-d	Not toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.3	Ferraro et al. 1991[a]
6.33	2.2	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		4.10	Swartz et al. 1985[a]
6.33	2.2	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		4.10	Swartz et al. 1985[a]
6.33	2.5	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.10	Swartz et al. 1985[a]
6.33	4.6	*	Southern California	COA		Low density (371.8N/0.1 sq.m.)	Benthic invertebrates		4.10	Swartz et al. 1985[a]
6.33	6.7	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		4.10	Swartz et al. 1985[a]
6.33	6.8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.10	Swartz et al. 1985[a]

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
6.33	6.8	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		4.10	Swartz et al. 1985[a]
6.33	101	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.10	Swartz et al. 1985[a]
6.68			ER-M							
6.68	2.3	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		3.70	Swartz et al. 1985[a]
6.68	2.3	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		3.70	Swartz et al. 1985[a]
6.68	2.6	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.70	Swartz et al. 1985[a]
6.68	4.9	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		3.70	Swartz et al. 1985[a]
6.68	7.1	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		3.70	Swartz et al. 1985[a]
6.68	7.1	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		3.70	Swartz et al. 1985[a]
6.68	7.1	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		3.70	Swartz et al. 1985[a]
6.68	106	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.70	Swartz et al. 1985[a]
7.03	7.2	*	Southern California	COA		Moderate density (29.6 N/0.1 sq.m.)	Amphipods		3.9	Ferraro et al. 1991[a]
7.03	35	*	Southern California	COA		Low density (0.8 N/0.1 sq.m.)	Echinoderms		3.9	Ferraro et al. 1991[a]
7.03		NE	Southern California	COA		High species richness (89 S/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[a]
7.03		NE	Southern California	COA		High density (921 N/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[a]
7.03		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[a]
7.03		NE	Southern California	COA		Normal benthic community (49.5; infaunal index)	Benthic invertebrates		3.9	Ferraro et al. 1991[a]
7.03		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.9	Ferraro et al. 1991[a]
7.06	4.9	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991[a]
7.07	1.1	SG	Southern California	COA		Low biomass (13.5 g/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.07	1.1	SG	Southern California	COA		Low density (298 N/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.07	7.2	*	Southern California	COA		Low density (4.2 N/0.1 sq.m.)	Amphipods		3.2	Ferraro et al. 1991[a]
7.07	35	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.2	Ferraro et al. 1991[a]
7.07		NE	Southern California	COA		High species richness (43.8 S/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.07		NE	Southern California	COA		Normal benthic community (52.6; infaunal index)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.07		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Ferraro et al. 1991[a]
7.20	1.1	SG	Southern California	COA		Low density (517 N/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.20	7.4	*	Southern California	COA		Low density (6.6 N/0.1 sq.m.)	Amphipods		3.2	Ferraro et al. 1991[a]
7.20	35	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.2	Ferraro et al. 1991[a]
7.20		NE	Southern California	COA		High species richness (58.8 S/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.20		NE	Southern California	COA		High biomass (21.4 g/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.20		NE	Southern California	COA		Normal benthic community (58.3; infaunal index)	Benthic invertebrates		3.2	Ferraro et al. 1991[a]
7.20		NE	Southern California	COA	10-d	Not toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Ferraro et al. 1991[a]
7.48	2.7	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.80	Bay et al. 1994
7.48		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.80	Bay et al. 1994
7.66	2.7	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.50	Bay et al. 1994
7.66		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.50	Bay et al. 1994
7.66		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.50	Bay et al. 1994
7.66		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.50	Bay et al. 1994
7.66		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.50	Bay et al. 1994

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
7.66		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.50	Bay et al. 1994
7.89	1.4	SG	Southern California	COA		Low biomass (14.3 g/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[a]
7.89	2.5	*	Southern California	COA		Low density (320 N/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[a]
7.89	3.4	*	Southern California	COA		Low density (9.4 N/0.1 sq.m.)	Amphipods		3.1	Swartz et al. 1986[a]
7.89	5.3	*	Southern California	COA		Low species richness (34.8 S/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[a]
7.89	12	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		3.1	Swartz et al. 1986[a]
7.89	64	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.1	Swartz et al. 1986[a]
7.89		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Swartz et al. 1986[a]
8.48	2.7	*	Southern California	COA		Low density (334 N/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[a]
8.48	5.7	*	Southern California	COA		Low density (2.2 N/0.1 sq.m.)	Amphipods		3	Swartz et al. 1986[a]
8.48	5.7	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[a]
8.48	13	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		3	Swartz et al. 1986[a]
8.48	68	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3	Swartz et al. 1986[a]
8.48		NE	Southern California	COA		High biomass (27.2 g/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[a]
8.48		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Swartz et al. 1986[a]
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1.00	Bay et al. 1994
8.84	6.5	*	Southern California	COA		Low density (720.2 N/0.1 sq.m.)	Benthic invertebrates		3.80	Swartz et al. 1985[a]
8.84	9.4	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3.80	Swartz et al. 1985[a]
8.84	9.4	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		3.80	Swartz et al. 1985[a]
8.84	9.4	*	Southern California	COA		Low density (7 N/0.1 sq.m.)	Crustaceans		3.80	Swartz et al. 1985[a]
8.84	140	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.80	Swartz et al. 1985[a]
8.84		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		3.80	Swartz et al. 1985[a]
8.84		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		3.80	Swartz et al. 1985[a]
8.84		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.80	Swartz et al. 1985[a]
9.55	3.2	*	Southern California	COA	35-d	Toxic (0.00023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.80	Bay et al. 1994
9.55		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	3.80	Bay et al. 1994
9.55		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.80	Bay et al. 1994
9.55		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.80	Bay et al. 1994
9.55		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.80	Bay et al. 1994
9.55		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.80	Bay et al. 1994
11.0	7.6	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991[a]
12.0	4.3	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.10	Bay et al. 1994
12.0		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	3.10	Bay et al. 1994
12.0		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.10	Bay et al. 1994
12.0		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.10	Bay et al. 1994
12.0		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.10	Bay et al. 1994
12.0		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.10	Bay et al. 1994
12.0	4.0	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.10	Bay et al. 1994
12.7	4.5	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.30	Bay et al. 1994
12.7		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	4.30	Bay et al. 1994
12.7		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.30	Bay et al. 1994
12.7		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.30	Bay et al. 1994
12.7		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.30	Bay et al. 1994

Table A4-6. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	108* Species	Life Stage	TOC (%)	Reference
12.7	4.2	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.30	Bay et al. 1994
13.2	9.1	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991[a]
13.6		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991[a]
14.1	2.2	*	Southern California	COA		Low density (357 N/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
14.1	15	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		5.4	Ferraro et al. 1991[a]
14.1	70	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		5.4	Ferraro et al. 1991[a]
14.1	2.2	*	Southern California	COA		Low biomass (6.3 g/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
14.1	2.8	*	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
14.1	2.8	*	Southern California	COA		Altered benthic community (3.6; infaunal index)	Benthic invertebrates		5.4	Ferraro et al. 1991[a]
14.1		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.4	Ferraro et al. 1991[a]
32.1	22	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991[a]
39.2	27	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991[a]
43.7	30	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991[a]
54.3	38	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991[a]
78.7	55	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991[a]
91.2	63	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991[a]
92.6	64	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.60	Swartz et al. 1991[a]
129	89	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991[a]
134	93	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991[a]
147	102	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991[a]
180	125	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.20	Swartz et al. 1991[a]
196	136	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.70	Swartz et al. 1991[a]

*SUM DDE concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.886 (conversion factor was determined from the data reported by Bay et al. 1994).

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.

Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-7. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0071 +/- 0.007	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0074 +/- 0.007		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0076 +/- 0.015		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0086 +/- 0.022	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0087 +/- 0.014	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0093 +/- 0.011		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0156	0.1	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0156	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0156	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0165 +/- 0.018		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0170 +/- 0.018		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0265 +/- 0.027	0.1	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0660 +/- 0.059	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0702		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0955		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
0.0999 +/- 0.109		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1500 +/- 0.402		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1670 +/- 0.426		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1670 +/- 0.426		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1670 +/- 0.426		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2120 +/- 0.494		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2540		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
0.3243 +/- 0.648	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.3612 +/- 0.252		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.3612 +/- 0.252		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.3612 +/- 0.252		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.4391 +/- 0.262		NE	Southern California	COA		High density (54.6+/-5.09 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
0.4399 +/- 0.596		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[b]
0.4707 +/- 0.532		NE	Southern California	COA		Normal benthic community (74+/-7.99; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.5068 +/- 0.012		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.6180 +/- 0.852	36	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.6180 +/- 0.852	37	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.7517 +/- 0.614		NE	Southern California	COA		High density (20.9+/-0.23 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]

Table A4-7. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.7517 +/- 0.614		NE	Southern California	COA		High species richness (65.6+/-5.37 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.8521 +/- 1.00		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.8521 +/- 1.00		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.8521 +/- 1.00		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
1.21 +/- 1.05		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.29 +/- 0.733	18	*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
1.30 +/- 1.06	0.6	NC	Southern California	COA		Low biomass (13.4+/-3.32 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.31	0.8	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
1.31		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.33 +/- 0.962		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.36 +/- 0.854	2.7	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.43 +/- 1.16	0.8	NC	Southern California	COA		Low biomass (11.3+/-4.37 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.47 +/- 0.379	1.7	SG	Southern California	COA		Low biomass (9.37+/-2.78 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.47 +/- 0.379	1.7	SG	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.47 +/- 0.379	1.7	SG	Southern California	COA	10-d	Toxic (21+/-1.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
1.49 +/- 0.772		NE	Southern California	COA		Normal benthic community (62.1+/-15.8; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.49 +/- 0.772		NE	Southern California	COA		High species richness (70.9+/-16.9 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.59 +/- 1.07	0.9	NC	Southern California	COA		Low density (415+/-94.4 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.60 +/- 1.14	1.2	SG	Southern California	COA		Low density (358+/-76.8 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.60 +/- 1.14		NE	Southern California	COA	10-d	Not toxic (7.8+/-4.44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
1.63 +/- 0.819		NE	Southern California	COA	10-d	Not toxic (11.9+/-5.35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
1.63 +/- 1.02		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.68 +/- 0.528	4.7	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
1.68 +/- 0.528	4.7	*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.68 +/- 0.528	4.7	*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
1.69 +/- 1.18		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.69 +/- 1.18		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.69 +/- 1.18		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.69 +/- 1.18		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.69 +/- 0.211	3.8	*	Southern California	COA		Moderate density (23.5+/-5.91 N/0.1 sq.m)	Amphipods		1	Ferraro et al. 1991[a]
1.69 +/- 0.211		NE	Southern California	COA		High density (944+/-101 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.62			*ER-M*							
1.82 +/- 0.650	7.2	*	Southern California	COA		Low density (2.4+/-5.65 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]

Table A4-7. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; summarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.83 +/- 0.330		NE	Southern California	COA		High biomass (25.5+/-4.21 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.85 +/- 0.830	19	*	Southern California	COA		Low density (0.2+/-0.14 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
2.07 +/- 1.07		NE	Southern California	COA		High biomass (33.3+/-8.63 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.10 +/- 0.709	4.5	*	Southern California	COA		Altered benthic community (53.7+/-3.43; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.18 +/- 1.41		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
2.36 +/- 0.586	3.1	*	Southern California	COA		Low density (5.3+/-3.7 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
2.36 +/- 0.586	3.1	*	Southern California	COA		Low species richness (38.4+/-3.29 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.36 +/- 0.225	5.4	*	Southern California	COA		Low density (4.13+/-2.5;N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
2.50 +/- 1.34	1.9	SG	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.56 +/- 0.935	2.1	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.62	1.8	SG	Southern California	COA		Altered benthic community (3.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.62	1.8	SG	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
3.45 +/- 2.34	7.8	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[b]
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
14.6 +/- 2.88	24	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[b]

[a]SUM DDE concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.886 (conversion factor was determined from the data reported by Bay et al. 1994).

[b]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; (conversion factors were determined from the data reported by Bay et al. 1994). SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations.

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0004 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0013	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0014	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0014	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0014	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0014	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0015	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0016	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0017		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0017		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0017	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0017	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0017	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0018	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0019	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0019	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0020	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0020	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0022	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0025		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0025	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0025		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0026	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0026	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0026	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0026		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0026	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0027	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0028	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0028	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0028	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0029	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0029	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0029	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0030	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0030		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0031	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0032		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0032	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0032		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0032	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0032	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0033	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0033	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0034		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0034		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0034	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0034		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0034		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0035	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0035	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0036	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0036		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0036	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0036	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0037	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0037	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0037	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0037	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0038	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0042	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0043	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0044		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0044		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0045		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0045	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0046		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0047	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0050		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0050	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0050		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0053	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0053		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0055	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0055		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0055		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0061	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0063	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0065	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0065	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0065	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0066	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0066	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0066	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0068	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0068		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0068	0.03	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0068		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0069	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0075	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0075	1.0	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0077	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0078		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0078	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0080		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0081	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0082	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0083	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0087	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0087		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0087	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0087	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0096	0.01	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0097		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0098	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0099		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0100	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0104		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0104		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0106		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0110	1.5	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0113	0.01	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0115		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0116	0.01	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0120		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0120		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0120		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0120		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0120		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0121	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0121		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0125		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0125		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0134	1.8	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0134		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0138	1.9	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0141	0.1	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0142		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0154		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0156	0.1	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0156	0.9	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0156	0.9	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0156		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0157	0.1	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0157	0.1	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0157	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0158	1.7	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0158	2.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0162		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0163		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991*
0.0166	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0171		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0173	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0177	2.3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0184		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0186	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0194		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0196	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0196	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0211		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0213	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0213		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0217	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0221		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0238	0.01	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0239	2.6	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0239	3.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0254	3.4	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0254		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0259	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0274		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0279	3.0	*	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0279	3.7	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0299	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0308		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0318		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0318	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0356		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0364	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0370		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0387	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0391		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0393		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0401	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0410		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0426	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0443		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0445	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0455		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991*
0.0459		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0461		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0461		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0488		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0491		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0491		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0496		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0509	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0512	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0514	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0516		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0519	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0519	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0533	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0543		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0547	7.3	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0550	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0556	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0558		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0565		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0571	0.3	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0571		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0571		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0575		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0579	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0600		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0609	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0612		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0617	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0625	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0633		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0647		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0651	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0654		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0656	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0665	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0669	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0670	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0695		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0702	0.1	NC	Southern California	COA		Low density (616.6 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0702		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0702		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0724	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0742		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0747	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0751	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0754	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0762		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0766		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0768	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0780		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0788		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0792		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0794		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0796		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0825		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0827	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0830	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0834		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0837		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0843	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0858	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0869		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0871	11	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0873		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0876		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0879		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0899	0.06	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0908		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0935		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0943	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0955		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
0.0955	0.05	NC	Southern California	COA		Low biomass (11.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.0955	0.1	NC	Southern California	COA		Low density (490 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.0955		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
0.0955		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.0955		NE	Southern California	COA		High species richness (66.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.0955		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
0.0961	0.96	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1011	1.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1013		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1042	1.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1053		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1079		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1316	18	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1666		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1716		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1730		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.1843	1.8	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1964		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2095		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2460		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2464		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.2479		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2479		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2479		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2479		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2479		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2479		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.2540	0.1	NC	Southern California	COA		Low biomass (16.1 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.2540	0.2	NC	Southern California	COA		Low density (398 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.2540		NE	Southern California	COA		High density (58.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
0.2540		NE	Southern California	COA		High species richness (62 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.2540		NE	Southern California	COA		Normal benthic community (92.4; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.2540		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
0.2540		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
0.2931		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3604		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3768		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3942	3.9	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.4118		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.4465		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.4987		NE	Southern California	COA		High biomass (20.9g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4987		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.4987	7.1	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.4987		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.4987		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4987		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4987		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.4987		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.5150	7.4	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		High density (86 N/0.1sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.5150		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.5982		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.6068	1.4	SG	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.6248	0.3	NC	Southern California	COA		Low biomass (9.2 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.6248	0.4	NC	Southern California	COA		Low density (504 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.6248	2.5	*	Southern California	COA		Low density (15.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
0.6248		NE	Southern California	COA		High density (51 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
0.6248		NE	Southern California	COA		High species richness (83.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.6248		NE	Southern California	COA		Normal benthic community (75; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
0.6248		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
0.6421		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.6550		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6550		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6550		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6550		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6550		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718	0.5	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6718		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.6838		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6838		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6838		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6838		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6838		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6838		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.7248		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.8130		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.8165		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.8465	0.4	NC	Southern California	COA		Low biomass (10.2 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.8465	0.6	NC	Southern California	COA		Low density (355 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]

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Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.8465	8.9	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
0.8465		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
0.8465		NE	Southern California	COA		Normal benthic community (68.3; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.8465		NE	Southern California	COA		High species richness (59.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
0.8465		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
1.01		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.03		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.06	1.2	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.06	1.2	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.06	1.2	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
1.06	2.1	*	Southern California	COA		Low density (661.4 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.06	2.9	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
1.06	2.9	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.06	2.9	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
1.06	15	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
1.15		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.22	74	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.22	74	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.22		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
1.22		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
1.22		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.22		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.22		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.22		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.31	0.8	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
1.31	2.8	*	Southern California	COA		Altered benthic community (50.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.31	14	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
1.31		NE	Southern California	COA		High density (21.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
1.31		NE	Southern California	COA		High species richness (70.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.31		NE	Southern California	COA		High biomass (39.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.31		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.35	3.1	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.45	3.3	*	Southern California	COA		Moderate density (23.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
1.45	5.7	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
1.45		NE	Southern California	COA		High species richness (87.4 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.45		NE	Southern California	COA		High density (1054 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.45		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.45		NE	Southern California	COA		Normal benthic community (53.4; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.45		NE	Southern California	COA	10-d	Not toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
1.54	1.8	SG	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.54	1.8	SG	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.54	1.8	SG	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
1.54	3	*	Southern California	COA		Low density (371.8N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.54	4.3	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
1.54	4.3	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.54	4.3	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
1.54	22	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
1.57	0.965	NC	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.60		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.61	1.3	SG	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.61		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.61		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.61		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.61		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.61		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.69	1.04	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.70	0.8	NC	Southern California	COA		Low biomass (17.7 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.70	1.3	SG	Southern California	COA		Low density (293 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.70	2.3	*	Southern California	COA		Low density (4.3 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
1.70	2.3	*	Southern California	COA		Low species richness (39.3 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.70	3.6	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
1.70	18	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
1.70		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
1.70	1.4	SG	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.70		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.70		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.70		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.70		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.70		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.80		NE	Southern California	COA		High species richness (89 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.80		NE	Southern California	COA		High density (921 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.80		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.80		NE	Southern California	COA		Normal benthic community (49.5; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.80		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
1.80	4.1	*	Southern California	COA		Moderate density (29.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
1.80	7.1	*	Southern California	COA		Low density (0.8 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
1.81			*ER-M*							
1.81	2.1	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.81	2.1	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.81	2.1	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
1.81	3.6	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.81	5.0	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.81	5.0	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
1.81	5.0	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
1.81	26	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
1.82	4.1	*	Southern California	COA		Moderate density (17.8 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
1.82	7.2	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
1.82		NE	Southern California	COA		High species richness (72 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.82		NE	Southern California	COA		High density (856 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.82		NE	Southern California	COA		High biomass (31.4 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.82		NE	Southern California	COA		Normal benthic community (53.3; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
1.82		NE	Southern California	COA	10-d	Not toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
1.86		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.07	4.7	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
2.21	1.2	SG	Southern California	COA		Low biomass (13.5 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.21	1.3	SG	Southern California	COA		Low density (298 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.21	5.0	*	Southern California	COA		Low density (4.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
2.21	8.7	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
2.21		NE	Southern California	COA		High species richness (43.8 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.21		NE	Southern California	COA		Normal benthic community (52.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.21		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
2.22		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
2.25	1.3	SG	Southern California	COA		Low density (517 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.25	5.1	*	Southern California	COA		Low density (6.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
2.25	8.9	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
2.25		NE	Southern California	COA		High species richness (58.8 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.25		NE	Southern California	COA		High biomass (21.4 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.25		NE	Southern California	COA		Normal benthic community (58.3; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.25		NE	Southern California	COA	10-d	Not toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
2.33		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
2.33		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
2.33	4.6	*	Southern California	COA		Low density (720.2 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
2.33	6.4	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
2.33	6.4	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
2.33	6.4	*	Southern California	COA		Low density (7 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
2.33	33	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
2.33		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.48	5.7	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
2.48		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
2.48		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.51		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.51		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.51		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.51		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.51		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.51	1.9	SG	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.54	1.2	SG	Southern California	COA		Low biomass (14.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.54	1.9	SG	Southern California	COA		Low density (320 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.54	3.4	*	Southern California	COA		Low density (9.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
2.54	3.4	*	Southern California	COA		Low species richness (34.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.54	5.4	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.54	27	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
2.54		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
2.62	1.4	SG	Southern California	COA		Low biomass (6.3 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.62	1.5	SG	Southern California	COA		Low density (357 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.62	1.8	SG	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.62	1.8	SG	Southern California	COA		Altered benthic community (3.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[a]
2.62	6.0	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[a]
2.62	10	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[a]
2.62		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[a]
2.67		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.67		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.67		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.67		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.67		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.67		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
2.67	2.2	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.83	2.2	*	Southern California	COA		Low density (334 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.83	3.8	*	Southern California	COA		Low density (2.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[a]
2.83	3.8	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.83	6.0	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[a]
2.83	30	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[a]
2.83		NE	Southern California	COA		High biomass (27.2 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[a]

Table A4-8. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDE (mg/kg DW at 1% OC; unsummarized data set).

Sum DDE Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.83		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[a]
2.95	2.2	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.95	2.4	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.95		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.95		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.95		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.95		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.96		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
3.21	7.3	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
3.47		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
3.86		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.86		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.86		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.86		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.86		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
3.86	2.9	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.86	3.2	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
4.73	11	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
5.57	13	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
7.55	17	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
10.5	24	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
10.8	25	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
12.1	28	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
14.2	32	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
15.7	36	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
15.9	36	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
15.9	36	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
17.6	40	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
18.3	42	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]

[a]SUM DDE concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.886 (conversion factor was determined from the data reported by Bay et al. 1994).

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-9. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; summarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[a]
0.0048 +/- 0.005		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.87+/-0.88	Fairey et al. 1996
0.0050 +/- 0.005		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4+/-0.6	Fairey et al. 1996
0.0050 +/- 0.006		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0064 +/- 0.018	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.66+/-0.6	Fairey et al. 1996
0.0077 +/- 0.019	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0092 +/- 0.026	1.9	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.91+/-0.79	Fairey et al. 1996
0.0110 +/- 0.016		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.27+/-0.89	Sapudar et al. 1994
0.0117 +/- 0.014		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0120 +/- 0.014		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0220 +/- 0.017	0.2	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.0280	0.3	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0280	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0280	0.3	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0291 +/- 0.050	2.6	*	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93+/-1.04	Sapudar et al. 1994
0.0780 +/- 0.193		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.0780 +/- 0.075		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		2.1+/-1.5	Swartz et al. 1985[a]
0.0780 +/- 0.075		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		2.1+/-1.5	Swartz et al. 1985[a]
0.0780 +/- 0.075		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		2.1+/-1.5	Swartz et al. 1985[a]
0.0840 +/- 0.206		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.0840 +/- 0.206		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.0840 +/- 0.206		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.1060 +/- 0.239		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
0.1068 +/- 0.246	0.9	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.83+/-1.77	Fairey 1997
0.1157 +/- 0.033		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.10+/-0.91	Fairey 1997
0.1170 +/- 0.048		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.71	Swartz et al. 1985[a]
0.1700 +/- 0.221		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.81+/-1.1	Bay et al. 1994
0.2270 +/- 0.236		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al. 1994
0.2816 +/- 0.412		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53+/-1.27	Swartz et al. 1985[a]
0.2816 +/- 0.412		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
0.2816 +/- 0.412		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[a]
0.3033 +/- 0.808		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991[a]
0.3100 +/- 0.400	26	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988

Table A4-9. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; summarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.3100 +/- 0.400	26	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
0.5170 +/- 0.883		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.5170 +/- 0.883		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.5170 +/- 0.883		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.5170 +/- 0.883		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
0.8196 +/- 0.703		*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		3.53+/-0.74	Swartz et al. 1985[a]
0.8930 +/- 1.38		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	2+/-0.95	Bay et al. 1994
0.9361 +/- 0.722	8.0	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		3.34+/-1.38	Swartz et al. 1985[a]
1.10 +/- 1.26	6.5	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.22+/-1.26	Bay et al. 1994
1.17			*ER-M*							
1.17 +/- 0.573	15	*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		3.95+/-0.24	Swartz et al. 1985[a]
1.17 +/- 0.573	15	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		3.95+/-0.24	Swartz et al. 1985[a]
1.17 +/- 0.573	15	*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		3.95+/-0.24	Swartz et al. 1985[a]
1.17 +/- 1.47	5.2	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.08+/-1.41	Bay et al. 1994
1.26 +/- 0.664	4.5	*	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	4+/-.26	Swartz et al. 1985[a]
1.26 +/- 0.664	4.5	*	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		4+/-.26	Swartz et al. 1985[a]
1.26 +/- 0.664	4.5	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		4+/-.26	Swartz et al. 1985[a]
3.00 +/- 3.59	9.9	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991[a]
14.5 +/- 6.34	48	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991[a]

[a]SUM DDD concentrations have been estimated from the concentrations of p,p'-DDD by dividing by 0.863 (conversion factor was determined from the data reported by Bay et al. 1994).

ND = not detected; detection limit was not reported.

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Swartz et al. 1985[a]
0.0007 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.09	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.13	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.19	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.14	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.13	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey et al. 1996
0.0007 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.19	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.11	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.09	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.09	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.37	Fairey et al. 1996
0.0007 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.9	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.9	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.58	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.55	Fairey et al. 1996
0.0010	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0010		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.07	Fairey et al. 1996
0.0011	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0011	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.48	Fairey et al. 1996
0.0011	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0011		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.48	Fairey et al. 1996
0.0012	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0012	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.07	Fairey et al. 1996
0.0012	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.6	Fairey et al. 1996
0.0012	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.6	Fairey et al. 1996
0.0012	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0012	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.89	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.59	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.28	Fairey et al. 1996
0.0013	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.9	Fairey et al. 1996
0.0013	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0013	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0013	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.17	Fairey et al. 1996
0.0014	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0014	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Fairey et al. 1996
0.0014	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.16	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.16	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.26	Fairey et al. 1996
0.0015	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.78	Fairey et al. 1996
0.0015		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.78	Fairey et al. 1996
0.0015	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0016	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.7	Fairey et al. 1996
0.0016	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.02	Fairey et al. 1996
0.0016	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.02	Fairey et al. 1996
0.0016		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.81	Fairey et al. 1996
0.0016	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0016		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0017	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0017	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.65	Fairey et al. 1996
0.0017		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.67	Fairey et al. 1996
0.0017		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.67	Fairey et al. 1996
0.0017		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.24	Fairey et al. 1996
0.0018	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0018	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0018	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.27	Fairey et al. 1996
0.0018	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.89	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.27	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.27	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53	Fairey et al. 1996
0.0019	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.09	Fairey et al. 1996
0.0019	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.88	Fairey et al. 1996
0.0019		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.35	Fairey et al. 1996
0.0019		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.88	Fairey et al. 1996
0.0019		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0019	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.48	Fairey et al. 1996
0.0020		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0020		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0020		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0022	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.28	Fairey et al. 1996
0.0022		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.28	Fairey et al. 1996
0.0022		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0022	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0022		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.03	Fairey et al. 1996
0.0022		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.78	Fairey et al. 1996
0.0024	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0026	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0026	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.97	Sapudar et al. 1994
0.0027		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0027		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.49	Sapudar et al. 1994
0.0028	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.86	Fairey et al. 1996
0.0030		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.61	Sapudar et al. 1994
0.0030	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.22	Fairey et al. 1996
0.0030	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.22	Fairey et al. 1996
0.0030	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.94	Fairey et al. 1996
0.0031	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0031	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.77	Fairey et al. 1996
0.0031	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.95	Fairey et al. 1996
0.0031	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.28	Fairey et al. 1996
0.0031		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.34	Sapudar et al. 1994
0.0033	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey et al. 1996
0.0033	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.33	Fairey et al. 1996
0.0034		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0034		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0035		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.38	Sapudar et al. 1994
0.0035		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Sapudar et al. 1994
0.0035	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0035		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0035		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0035		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0035		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0035		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0035		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidicrella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0036	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.98	Fairey et al. 1996
0.0036	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.15	Fairey et al. 1996
0.0036	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0037		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.18	Fairey et al. 1996
0.0037	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0038		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0040	0.04	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0040		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0041		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.31	Sapudar et al. 1994
0.0042		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Sapudar et al. 1994
0.0043		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0043		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0043		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.76	Sapudar et al. 1994
0.0043		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.4	Sapudar et al. 1994
0.0044	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0044		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0046	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0047	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.75	Fairey et al. 1996
0.0047	0.98	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0047		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.75	Fairey et al. 1996
0.0047	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.88	Fairey et al. 1996
0.0047	0.98	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.29	Fairey et al. 1996
0.0048	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0049	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.86	Sapudar et al. 1994
0.0050		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0050		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0050		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0050	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.16	Fairey et al. 1996
0.0051		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.72	Sapudar et al. 1994
0.0051		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.11	Sapudar et al. 1994
0.0052	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.99	Sapudar et al. 1994
0.0052	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Sapudar et al. 1994
0.0052		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0053	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.58	Fairey et al. 1996

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0053	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.58	Fairey et al. 1996
0.0053	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0053	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0054	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.19	Fairey et al. 1996
0.0056	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.46	Sapudar et al. 1994
0.0057	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.77	Fairey et al. 1996
0.0057	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.77	Fairey et al. 1996
0.0057		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.72	Fairey et al. 1996
0.0057	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0058	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.04	Fairey et al. 1996
0.0058		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991[a]
0.0058		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.25	Sapudar et al. 1994
0.0061		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0062		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.53	Sapudar et al. 1994
0.0063	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.42	Fairey et al. 1996
0.0063	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.42	Fairey et al. 1996
0.0063		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.93	Fairey et al. 1996
0.0063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0063	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.94	Fairey et al. 1996
0.0063	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Fairey et al. 1996
0.0063		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.94	Fairey et al. 1996
0.0064	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.14	Fairey et al. 1996
0.0065	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.93	Fairey et al. 1996
0.0065	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.93	Fairey et al. 1996
0.0065		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.67	Fairey et al. 1996
0.0066		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0067		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.05	Sapudar et al. 1994
0.0069		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.53	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.53	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.9	Fairey et al. 1996
0.0071		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0073		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0074		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.5	Sapudar et al. 1994
0.0074		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0075		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0076		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.0076	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.36	Sapudar et al. 1994
0.0077	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.75	Fairey et al. 1996
0.0077	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0078		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.55	Sapudar et al. 1994
0.0080	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0084	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.81	Sapudar et al. 1994
0.0086	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.42	Sapudar et al. 1994
0.0086		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0088	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.69	Sapudar et al. 1994

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0090	1.8	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.26	Fairey et al. 1996
0.0090		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.26	Fairey et al. 1996
0.0090		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Fairey et al. 1996
0.0091		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.0092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0096	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0096		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0099	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.39	Sapudar et al. 1994
0.0099		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0099	1.98	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.53	Fairey et al. 1996
0.0099	1.98	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.53	Fairey et al. 1996
0.0099		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Fairey et al. 1996
0.0102	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0104		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.0108		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0110		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0110		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.1	Sapudar et al. 1994
0.0113	1.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Sapudar et al. 1994
0.0113		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.0115		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.0116		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991[a]
0.0116		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991[a]
0.0116		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991[a]
0.0116		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.0117		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.49	Sapudar et al. 1994
0.0119		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.07	Fairey et al. 1996
0.0120	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.18	Sapudar et al. 1994
0.0121	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.61	Sapudar et al. 1994
0.0126		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0130		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.56	Sapudar et al. 1994
0.0130	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.0131		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.0131	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.06	Sapudar et al. 1994
0.0131	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0134	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.56	Sapudar et al. 1994
0.0135	2.7	*	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.14	Fairey et al. 1996
0.0135	2.8	*	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0135		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.14	Fairey et al. 1996
0.0139		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0139	2.8	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.37	Fairey et al. 1996
0.0139		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.37	Fairey et al. 1996
0.0139		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Fairey et al. 1996
0.0142	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.16	Sapudar et al. 1994
0.0143		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.0145		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.9	Sapudar et al. 1994

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0146	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.46	Fairey 1997
0.0149		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Sapudar et al. 1994
0.0151	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Sapudar et al. 1994
0.0151	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0151	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Sapudar et al. 1994
0.0152		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.81	Fairey et al. 1996
0.0152		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0153		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0157	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey 1997
0.0157		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.13	Sapudar et al. 1994
0.0159	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.51	Fairey 1997
0.0159	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.96	Sapudar et al. 1994
0.0166	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.3	Fairey 1997
0.0168	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.0172		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Sapudar et al. 1994
0.0172		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.38	Fairey et al. 1996
0.0173	3.4	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.75	Fairey et al. 1996
0.0179	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.48	Sapudar et al. 1994
0.0186	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.67	Fairey 1997
0.0186	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.0195	3.9	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.24	Fairey et al. 1996
0.0197	3.9	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.83	Fairey et al. 1996
0.0198		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.31	Sapudar et al. 1994
0.0206	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0208	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0210		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0211	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.0211	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Sapudar et al. 1994
0.0221	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey 1997
0.0235	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
0.0237	4.9	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0246		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.89	Sapudar et al. 1994
0.0250		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.0250		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0250		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0250		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0250		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0250		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.0270	2.5	*	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0275	0.3	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0275	0.3	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0275	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0275		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.0275		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0275		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.0276	2.5	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Sapudar et al. 1994
0.0277		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.4	Sapudar et al. 1994
0.0278		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.0280	0.3	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.0280	2.4	*	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0280	2.4	*	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.0290		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991*
0.0298	2.7	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Sapudar et al. 1994
0.0310	0.1	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0310		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Bay et al. 1994
0.0310		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0310		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0310		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0310		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.0339	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Fairey 1997
0.0342	3.1	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.85	Sapudar et al. 1994
0.0350	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.06	Fairey 1997
0.0355		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0355		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0355		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0355		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0355		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.0355		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0360	0.3	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.0377	3.4	*	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0420		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0420		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0420		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0420		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0420		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.0470		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.8	Bay et al. 1994
0.0470		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0470		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0470		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0470		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0470		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.0695		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991*
0.0730		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Fairey 1997
0.0753		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991*
0.0753		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991*
0.0811		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991*

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0834		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.2	Swartz et al. 1985
0.0834		NE	Southern California	COA	10-d	Toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		3.2	Swartz et al. 1985[a]
0.0834		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		3.2	Swartz et al. 1985[a]
0.0880		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
0.1042		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.79	Fairey 1997
0.1050		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Fairey 1997
0.1129		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.92	Fairey 1997
0.1160		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.1160		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Bay et al. 1994
0.1166		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Fairey 1997
0.1172	23	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.1172	23	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.1172	24	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.37	Fairey et al. 1996
0.1217		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991[a]
0.1263		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.54	Sapudar et al. 1994
0.1377	1.2	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.86	Fairey 1997
0.1502		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.85	Fairey 1997
0.1506		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA	10-d	Toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		2.2	Swartz et al. 1985[a]
0.1506		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		2.2	Swartz et al. 1985[a]
0.1730	16	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.3	Sapudar et al. 1994
0.1750	16	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.6	Sapudar et al. 1994
0.1758		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.84	Fairey 1997
0.1934	18	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.27	Sapudar et al. 1994
0.1999	18	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.28	Sapudar et al. 1994
0.2450	1.4	SG	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4	Bay et al. 1994
0.2450		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.2450		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.2450		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.2450		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2450		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.3820	1.3	SG	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991[a]
0.4260		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.4260		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Bay et al. 1994
0.5122	1.8	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.2	Swartz et al. 1985
0.5122	1.8	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985
0.5122	1.8	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		4.2	Swartz et al. 1985
0.5122	4.4	*	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
0.5122	6.6	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[a]
0.5122	6.6	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.2	Swartz et al. 1985[a]
0.5122	6.6	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		4.2	Swartz et al. 1985[a]
0.5122		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.2	Swartz et al. 1985[a]
0.5340	3.1	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.5	Bay et al. 1994
0.5340		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.5340		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.5340		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.5340		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.5340		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
0.5450	2.4	*	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.5450		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.8	Bay et al. 1994
0.5450		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.5450		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.5450		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.5450		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
0.5928		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.5928		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.5928		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.5928		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
0.5928		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
0.5930	51	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.5930	51	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
0.6040	3.5	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
0.6040		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Bay et al. 1994
0.7184		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991[a]
0.7910	3.5	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.7910	4.6	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.3	Bay et al. 1994
0.7910		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.7910		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.7910		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.7910		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
0.8410	7.3	*	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.8922			ER-M							
0.8922	11	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.8922	11	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		3.8	Swartz et al. 1985[a]
0.8922	11	*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		3.8	Swartz et al. 1985[a]
0.8922		NE	Southern California	COA	10-d	Toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.8	Swartz et al. 1985[a]
0.8922		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.8922		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		3.8	Swartz et al. 1985[a]
0.8922	8	*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985
0.8922		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.8	Swartz et al. 1985[a]
1.00	3.3	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991[a]
1.35	4.5	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991[a]
1.51	5.3	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.7	Swartz et al. 1985[a]
1.51	5.3	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
1.51	5.3	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
1.51	13	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[a]
1.51	19.3	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985
1.51	19.3	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		3.7	Swartz et al. 1985
1.51	19.3	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		3.7	Swartz et al. 1985
1.51		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.7	Swartz et al. 1985[a]
1.77	6.3	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.1	Swartz et al. 1985[a]
1.77	6.3	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
1.77	6.3	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
1.77	15	*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
1.77	22.7	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[a]
1.77	22.7	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.1	Swartz et al. 1985[a]
1.77	22.7	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		4.1	Swartz et al. 1985[a]
1.77		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.1	Swartz et al. 1985[a]
2.00	6.6	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991[a]
2.64	8.7	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991[a]
3.04		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991[a]
3.32	19	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.1	Bay et al. 1994
3.32		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.32		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.32		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.32		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.32		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Bay et al. 1994
3.32	15	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.56	12	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991[a]
4.66	15	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991[a]

Table A4-10. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
6.47	21	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.6	Swartz et al. 1991[a]
8.38	28	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991[a]
9.55	32	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991[a]
9.55	32	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991[a]
10.5	35	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991[a]
13.1	43	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991[a]
14.5	48	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991[a]
21.1	70	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.2	Swartz et al. 1991[a]
21.9	72	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.7	Swartz et al. 1991[a]
24.1	80	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991[a]

[a]SUM DDD concentrations have been estimated from the concentrations of p,p'-DDD by dividing by 0.863 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported.
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.
Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-11. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; summarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0028 +/- 0.003		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0033 +/- 0.004		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0037 +/- 0.004		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0048 +/- 0.013	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0060 +/- 0.015	2.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0087 +/- 0.010		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0090 +/- 0.01		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0096 +/- 0.007		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0100 +/- 0.050	3.1	*	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0115 +/- 0.011	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0130 +/- 0.016	0.5	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0229 +/- 0.046		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0250 +/- 0.048		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250 +/- 0.048		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250 +/- 0.048		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0279 +/- 0.056		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0315 +/- 0.035		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0315 +/- 0.035		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0315 +/- 0.035		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0473 +/- 0.030		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0718 +/- 0.052		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0726 +/- 0.099	8.1	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0726 +/- 0.099	8.3	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0740 +/- 0.068		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0823 +/- 0.105		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0823 +/- 0.105		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0823 +/- 0.105		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0855 +/- 0.155		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0920 +/- 0.249	1.3	SG	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997

Table A4-11. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; summarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1030 +/- 0.075		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1810 +/- 0.276		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1810 +/- 0.276		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1810 +/- 0.276		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1810 +/- 0.276		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2151 +/- 0.173		*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.2368 +/- 0.182	5	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2991			*ER-M*							
0.2991 +/- 0.147	9	*	Southern California	COA		Low density (2+/-0.596.01 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.2991 +/- 0.147	9	*	Southern California	COA		Low species richness (8.7+/-6.01 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2991 +/- 0.147	9	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.3120 +/- 0.431		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.3205 +/- 0.172	3.9	*	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.3205 +/- 0.172	3.9	*	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.3205 +/- 0.172	3.9	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.3526 +/- 0.402	4.8	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.3570 +/- 0.481	3.5	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6720 +/- 0.587	7.9	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.82 +/- 0.829	21	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]

[a]SUM DDD concentrations have been estimated from the concentrations of p,p'-DDD by dividing by 0.863 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported.
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
ND		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
ND		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
ND		NE	Southern California	COA	10-d	Toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0003 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0004 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0005	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0005	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0006 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0006	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0007	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0007		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0007	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0007	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0008		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0008	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0008		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0009	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0009	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0010		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0011	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0011	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0011		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0012	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0012		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0013	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0013	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0014		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0014	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0014	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0015	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0015	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0015	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0016	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0016		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0016	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0016	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0016	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0017	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0017	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0017	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0017	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0019	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0020		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0020		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0020	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0020	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0020	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0021	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0021	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0021	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0021	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0022	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0022		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0022		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0022		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0023		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0023	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0025	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0025	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0025	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0026	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0026	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0026		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0026		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.1	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0026	0.3	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026	0.3	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0026		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0027	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0027	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0029	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0031	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0032	1.0	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0033	0.05	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0033		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0033	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0033		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0034		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0036		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0036		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0037	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0038		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0038	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0039	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0039		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0040	1.5	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0041		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0042		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0043	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0043	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0044	1.4	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0044		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0044		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0044		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0044		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0044		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0044		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0044		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0045		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0045		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0045	1.4	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0045		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0045	1.4	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0046	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0046		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0046		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0046		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0046	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0047	1.5	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0048	0.2	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0048		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0049		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0049		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0050		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0050		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0051	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0052	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0052		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0052	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0054		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0054		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0054	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0055		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0055		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0055	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0056		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0056	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0056		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0056	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0057	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0057	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0057		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0058	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0059	2.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0059		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0059	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0060	2.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0061	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0061		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0062		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0063	2.3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0063	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0064	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0064		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0064		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0064		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0065	1.7	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0065	2.4	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0065	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0066	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0066		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0066	2.0	*	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0067	1.8	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0067		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0067		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0069	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0070		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0070	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0071	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0071		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0071		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0072	2.6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0072		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0072		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0072	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0073	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0074		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0075		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0077		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0077		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0077	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0078		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0079		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0080	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0081	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0081		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0083		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0083		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0085		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0086		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0088		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0089		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0090		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0092	0.95	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0096		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0097	1.004	SG	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0097		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0102		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0102		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0103	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0104	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0104	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0104		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0104		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0104		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0104		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0104		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0104		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0108		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0108	3.9	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0108		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0113	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0116	3.6	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0116	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0117		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0118	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0118		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0119	4.3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0119		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0120	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0122		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0122		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0125		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0126		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0128	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0131		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0137	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0142		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0147	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0148		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0149		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0179		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0189	2.0	SG	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0200		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0201	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0227		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0232		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0245	2.6	*	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0248	2.6	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0261		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0261		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0261		NE	Southern California	COA	10-d	Toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0271		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0276		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0278		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0282	0.3	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0282		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0282		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0282		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0282		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0282		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0317	1.1	SG	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0317		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0325		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0336	0.5	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0373		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0374		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0375		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0380	4.0	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0387		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0402	4.2	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0443		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0453	4.7	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0461		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0467	4.9	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0476		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0497		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0575	15	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0575	21	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0588		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0588		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0588		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0588		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0588		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0588		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0589		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0619		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0663		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.0685		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.0685		NE	Southern California	COA	10-d	Toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.0700		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0700		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0700		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0700		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0700		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0740	1.03	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0813	29	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0967		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0967		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1187	1.6	SG	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1187		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1187		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1187		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1187		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1187		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1219	1.5	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.1219	1.5	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.1219	1.5	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.1219	3	*	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.1219	3.8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.1219	3.8	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.1219	3.8	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]

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Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1219		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.1257		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1425	16	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1425	16	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1425		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1425		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1425		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1425		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1425		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1434		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1434		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1434		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1434		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1434		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1434	1.4	SG	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1750	2.4	*	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1750		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1767		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1775		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.1775		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1840		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1840		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1840		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1840		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1840	1.8	SG	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1840	2.5	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al 1994
0.1908	2.2	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.2116	2.5	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.2157	2.9	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.2157		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2157		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2157		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2157		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2157		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2157		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.2348			*ER-M*							
0.2348		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2348		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2348		NE	Southern California	COA	10-d	Toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.2348	5.0	*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2348	7.3	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.2348	7.3	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.2348	7.3	*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.2348		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.2894	3.4	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.3168	97	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.3947		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.4071	5.0	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4071	5.0	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4071	5.0	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.4071	8.7	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4071	13	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.4071	13	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4071	13	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.4071		*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.4324	9.2	*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4324	5.3	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4324	5.3	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4324	5.3	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[a]
0.4324	14	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[a]
0.4324	14	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[a]
0.4324	14	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[a]
0.4324		*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[a]
0.4535	5.3	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.4949		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.5621	6.6	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.7523	8.8	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.7697	9	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
0.8410	11.7	*	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.9217	11	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.07	14	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.07		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.07		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-12. A summary of the available data on the biological effects associated with sediment-sorbed SUM DDD (mg/kg DW at 1% OC; unsummarized data set).

Sum DDD Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.07		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.07		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.07		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
1.07	10	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.24	15	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.40	16	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.49	17	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.79	21	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.84	22	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
1.97	23	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
2.05	24	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
2.07	24	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]
3.71	44	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[a]

[a]SUM DDD concentrations have been estimated from the concentrations of p,p'-DDD by dividing by 0.863 (conversion factor was determined from the data reported by Bay et al. 1994).
ND = not detected; detection limit was not reported.
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-13. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0147 +/- 0.017		NE	Southern California	COA		High density (229+/-60.3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0195 +/- 0.025		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4+/-0.6	Fairey et al. 1996
0.0213 +/- 0.034		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0216 +/- 0.021		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.87+/-0.88	Fairey et al. 1996
0.0216 +/- 0.049	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.66+/-0.6	Fairey et al. 1996
0.0277 +/- 0.050	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.0311		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
0.0312 +/- 0.063	1.6	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.91+/-0.79	Fairey et al. 1996
0.0319 +/- 0.040	2.2	*	Southern California	COA		Moderate density (73.8+/-40.9 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0420 +/- 0.045		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0420 +/- 0.045		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0627 +/- 0.093		NE	Southern California	COA		High species richness (133+/-32.5 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0632		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[c]
0.1011 +/- 0.150		NE	Southern California	COA		High density (144+/-42.7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1040 +/- 0.094	0.1	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.1107 +/- 0.086		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.27+/-0.89	Sapudar et al. 1994
0.1204 +/- 0.335	0.04	NC	Southern California	COA		Low density (68.2+/-47.8 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1233 +/- 0.345	0.04	NC	Southern California	COA		Low density (370+/-122 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1398		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1.3	Swartz et al. 1986[b]
0.1398 +/- 0.427	0.1	NC	Southern California	COA		Low density (79.8+/-32.6 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1606 +/- 0.147	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93+/-1.04	Sapudar et al. 1994
0.1651 +/- 0.470	2.6	*	Southern California	COA		Moderate species richness (81.1+/-8.90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1690 +/- 0.687		NE	Southern California	COA		Normal benthic community (83.8+/-12.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.2000		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
0.2287		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		0.8	Ferraro et al. 1991[b]
0.5229 +/- 1.17	0.2	NC	Southern California	COA		Moderate density (259+/-85.9 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.6828 +/- 1.28	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.83+/-1.77	Fairey 1997
0.7260 +/- 2.00		NE	Southern California	COA	10-d	Not toxic (76+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.7370 +/- 0.845		NE	Southern California	COA		Normal benthic community (74+/-7.99; infaunal index)	Benthic invertebrates		1.35+/-0.07	Swartz et al. 1986[b]
0.7920 +/- 2.12		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988

Table A4-13. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.7920 +/- 2.12		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.7920 +/- 2.12		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.8227 +/- 1.04	4.9	*	Southern California	COA		Altered benthic community (53.8+/-5.63; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.00		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.01 +/- 0.858		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		2.1+/-1.15	Swartz et al. 1985[c]
1.01 +/- 0.858		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		2.1+/-1.15	Swartz et al. 1985[c]
1.01 +/- 0.858		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		2.1+/-1.15	Swartz et al. 1985[c]
1.04 +/- 2.45		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
1.05 +/- 2.50	17	*	Southern California	COA		Low species richness (50.5+/-9.2 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.10 +/- 1.23		NE	Southern California	COA		High density (54.6+/-5.09 N/0.1 sq.m.)	Amphipods		1.8+/-1.41	Ferraro et al. 1991[b]
1.15 +/- 1.73	0.3	NC	Southern California	COA		Moderate density (786+/-107 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.48 +/- 2.77	101	*	Southern California	COA		Low density (3.13+/-4.49 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.49 +/- 0.342		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.71	Swartz et al. 1985[c]
1.68 +/- 1.73		NE	Southern California	COA		High density (20.9+/-0.23 N/0.1 sq.m.)	Amphipods		1.7+/-0.61	Swartz et al. 1986[b]
1.68 +/- 1.73		NE	Southern California	COA		High species richness (65.6+/-5.37 S/0.1 sq.m.)	Benthic invertebrates		1.7+/-0.61	Swartz et al. 1986[b]
1.94 +/- 4.85		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991[c]
2.28 +/- 4.29		NE	Southern California	COA		High density (1068+/-636 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.83 +/- 0.521		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.10+/-0.91	Fairey 1997
2.89 +/- 3.82		NE	Southern California	COA		High density (418+/-275 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
3.02 +/- 3.88		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.81+/-1.1	Bay et al. 1994
3.12 +/- 4.13	74	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
3.12 +/- 4.13	74	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
3.20 +/- 11.3	32	*	Southern California	COA		Moderate density (48.8+/-19.5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
3.21 +/- 4.46		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[c]
3.21 +/- 4.46		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		2.53+/-1.27	Swartz et al. 1985[c]
3.21 +/- 4.46		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53+/-1.27	Swartz et al. 1985[c]
3.36 +/- 3.52		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al 1994
3.38 +/- 5.08		NE	Southern California	COA		High density (1947+/-843 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
3.55	0.7	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Swartz et al. 1986[b]
3.55		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[b]
4.14 +/- 3.38		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (425+/-82 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1.71+/-0.995	Murdoch et al. In press
4.17 +/- 4.13	0.6	NC	Southern California	COA		Low biomass (13.4+/-3.32 g/0.1 sq.m.)	Benthic invertebrates		2.28+/-1.07	Swartz et al. 1986[b]
5.25 +/- 4.31	1.5	SG	Southern California	COA		Low density (358+/-76.8 N/0.1 sq.m.)	Benthic invertebrates		2.42+/-0.98	Swartz et al. 1986[b]

Table A4-13. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
5.25 +/- 4.31		NE	Southern California	COA	10-d	Not toxic (7.8+/-4.44% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42+/-0.98	Swartz et al. 1986[b]
5.61 +/- 5.83		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
5.61 +/- 5.83		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
5.61 +/- 5.83		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
5.61 +/- 5.83		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
5.65 +/- 3.20		NE	Southern California	COA		Normal benthic community (62.1+/-15.8; infaunal index)	Benthic invertebrates		3.04+/-1.08	Ferraro et al. 1991[b]
5.65 +/- 3.20		NE	Southern California	COA		High species richness (70.9+/-16.9 S/0.1 sq.m.)	Benthic invertebrates		3.04+/-1.08	Ferraro et al. 1991[b]
5.71 +/- 3.63	90	*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		3.53+/-0.74	Swartz et al. 1985[c]
5.92 +/- 3.49		*	Southern California	COA		Low density (0.2+/-0.14 N/0.1 sq.m.)	Echinoderms		2.64+/-0.77	Swartz et al. 1986[b]
6.26 +/- 3.89	4.2	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		3.34+/-1.38	Swartz et al. 1985[c]
6.52 +/- 7.08	0.9	NC	Southern California	COA		Low biomass (11.3+/-4.37 g/0.1 sq.m.)	Benthic invertebrates		3.05+/-1.89	Ferraro et al. 1991[b]
6.54 +/- 4.24		NE	Southern California	COA		High biomass (33.3+/-8.63 g/0.1 sq.m.)	Benthic invertebrates		2.7+/-0.42	Swartz et al. 1986[b]
6.84 +/- 6.18	1.0	NC	Southern California	COA		Low density (415+/-94.4 N/0.1 sq.m.)	Benthic invertebrates		3.08+/-1.63	Ferraro et al. 1991[b]
6.94 +/- 4.69		NE	Southern California	COA	10-d	Not toxic (11.9+/-5.35% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.34+/-1.3	Ferraro et al. 1991[b]
7.08 +/- 2.73	9.6	*	Southern California	COA		Altered benthic community (53.7+/-3.43; infaunal index)	Benthic invertebrates		2.95+/-0.39	Swartz et al. 1986[b]
7.09 +/- 6.96		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	2+/-0.95	Bay et al. 1994
7.12 +/- 0.695	6.5	*	Southern California	COA		Moderate density (23.5+/-5.91 N/0.1 sq.m.)	Amphipods		3.77+/-0.42	Ferraro et al. 1991[b]
7.12 +/- 0.695		NE	Southern California	COA		High density (944+/-101 N/0.1 sq.m.)	Benthic invertebrates		3.77+/-0.42	Ferraro et al. 1991[b]
7.15			*ER-M*							
7.15 +/- 1.89	2.2	*	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[c]
7.15 +/- 1.89	2.2	*	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		4+/-0.26	Swartz et al. 1985[c]
7.15 +/- 1.89	2.2	*	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	4+/-0.26	Swartz et al. 1985[c]
7.37 +/- 0.75		NE	Southern California	COA		High biomass (25.5+/-4.21 g/0.1 sq.m.)	Benthic invertebrates		3.63+/-0.44	Ferraro et al. 1991[b]
7.81 +/- 2.04	7.7	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		3.95+/-0.24	Swartz et al. 1985[c]
7.81 +/- 2.04	7.7	*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		3.95+/-0.24	Swartz et al. 1985[c]
7.81 +/- 2.04	7.7	*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		3.95+/-0.24	Swartz et al. 1985[c]
7.90 +/- 4.13	35	*	Southern California	COA		Low density (2.4+/-5.65 N/0.1 sq.m.)	Echinoderms		3.71+/-0.87	Ferraro et al. 1991[b]
8.25 +/- 1.7	4.9	*	Southern California	COA		Low density (5.3+/-3.7 N/0.1 sq.m.)	Amphipods		3.13+/-0.15	Swartz et al. 1986[b]
8.25 +/- 1.7	4.9	*	Southern California	COA		Low species richness (38.4+/-3.29 S/0.1 sq.m.)	Benthic invertebrates		3.13+/-0.15	Swartz et al. 1986[b]
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
10.3 +/- 5.83	3.4	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.22+/-1.26	Bay et al. 1994

Table A4-13. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
10.4		*	Palos Verdes Shelf, CA	COA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1994
10.7 +/- 4.55	9.7	*	Southern California	COA		Low density (4.13+/-2.5;N/0.1 sq.m.)	Amphipods		3.93+/-1.27	Ferraro et al. 1991[b]
10.7 +/- 7.32	3.2	*	Southern California	COA	35-d	Toxic (0.002+/-0.0003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.08+/-1.41	Bay et al. 1994
10.9	64	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
15.9	2.8	*	Southern California	COA		Altered benthic communtiy (3.6; infaunal index)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
15.9	2.8	*	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
16.5		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	2	McLeese et al. 1982
23.9 +/- 17.5	12	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991[c]
49.4 +/- 107		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.57+/-0.08 mg/d)	Neanthes arenaceodentata (polychaete)	JUV	3.1+/-2.7	Murdoch et al. In press
49.4 +/- 107		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (2.67+/-4.8% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.1+/-2.7	Murdoch et al. In press
70.2 +/- 70.7	694	*	Southern California	COA		Low density (4+/-2.45 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
140 +/- 180	34	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (205+/-63.6 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	5.85+/-3.1	Murdoch et al. In press
143 +/- 52.3	74	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991[c]

[a]Total DDT concentrations have been estimated from the concentrations of the p,p'-isomers (p,p'-DDT, p,p'-DDE, p,p'-DDD) by dividing by 0.889 (conversion factor was determined from the data reported by Bay et al. 1994).
[b]Total DDT concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.787 (conversion factor was determined from the data reported by Bay et al. 1994).
[c]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations (conversion factors were determined from the data reported by Bay et al. 1994).
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0010 <	0.00001	NC	Southern California	COA		Moderate density (833 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010 <	0.00001	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0010 <	0.0001	NC	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0010 <		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0010 <		NE	Southern California	COA		High species richness (124 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010 <		NE	Southern California	COA		High density (610 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0010	0.00002	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010	0.00002	NC	Southern California	COA		Low density (374 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010	0.00002	NC	Southern California	COA		Low density (47 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0010	0.00003	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0010	0.0001	NC	Southern California	COA		Moderate density (130 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0010	0.005	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0010	0.01	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0010	0.02	NC	Southern California	COA		Moderate species richness (72 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010	0.02	NC	Southern California	COA		Low species richness (62 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0010		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0020	0.00004	NC	Southern California	COA		Low density (442 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020	0.00005	NC	Southern California	COA		Low density (50 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0020	0.0001	NC	Southern California	COA		Low density (119 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0020	0.02	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0020	0.04	NC	Southern California	COA		Moderate species richness (67 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		Normal benthic community (94.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0027 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.09	Fairey et al. 1996
0.0027 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.37	Fairey et al. 1996
0.0027 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.09	Fairey et al. 1996
0.0030	0.0003	NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0030	0.001	NC	Southern California	COA		Altered benthic community (45; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.1	NC	Southern California	COA		Moderate species richness (83 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		High density (195 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0030		NE	Southern California	COA		High density (1231 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		High density (385 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0030		NE	Southern California	COA		High density (616 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0033	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.14	Fairey et al. 1996
0.0033	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.19	Fairey et al. 1996
0.0033		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.09	Fairey et al. 1996
0.0033		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.19	Fairey et al. 1996
0.0035		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Fairey et al. 1996
0.0035	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.55	Fairey et al. 1996
0.0036		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.07	Fairey et al. 1996
0.0040	0.0001	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.0001	NC	Southern California	COA		Low density (36 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0040	0.0001	NC	Southern California	COA		Low density (110 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0040	0.0005	NC	Southern California	COA		Moderate density (142 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0040	0.04	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0040	0.1	NC	Southern California	COA		Moderate species richness (73 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		Normal benthic community (95.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0041	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.07	Fairey et al. 1996
0.0042	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0042	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.48	Fairey et al. 1996
0.0042		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.48	Fairey et al. 1996
0.0046	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0047	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0047	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.88	Fairey et al. 1996
0.0047		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.35	Fairey et al. 1996
0.0047		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0048	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey et al. 1996
0.0048		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.24	Fairey et al. 1996
0.0048		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.67	Fairey et al. 1996
0.0048		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.67	Fairey et al. 1996
0.0050		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.11	Fairey et al. 1996
0.0050	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0050	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0050	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.6	Fairey et al. 1996
0.0050	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.6	Fairey et al. 1996
0.0052	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0052	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.58	Fairey et al. 1996
0.0052	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.9	Fairey et al. 1996
0.0052	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.9	Fairey et al. 1996
0.0052	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.17	Fairey et al. 1996
0.0052	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0052	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0053	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93	Fairey et al. 1996
0.0053	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.13	Fairey et al. 1996
0.0054	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.89	Fairey et al. 1996
0.0055	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0055		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.78	Fairey et al. 1996
0.0055		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.78	Fairey et al. 1996
0.0058	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0058	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.13	Fairey et al. 1996
0.0060	0.0001	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060	0.0001	NC	Southern California	COA		Low density (515 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060	0.0001	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0060	0.0001	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0060	0.0002	NC	Southern California	COA		Moderate density (212 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0060	0.0002	NC	Southern California	COA		Low density (112 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0060	0.1	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0060	0.1	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0060	0.1	NC	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0060	0.1	NC	Southern California	COA		Low species richness (41 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060		NE	Southern California	COA		Normal benthic community (91.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0060		NE	Southern California	COA		Normal benthic community (98.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0060		NE	Southern California	COA		High density (184 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0060		NE	Southern California	COA		High density (224 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0061	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0061		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0061		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.59	Fairey et al. 1996
0.0063	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0063	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0063	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Fairey et al. 1996
0.0064	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.02	Fairey et al. 1996
0.0064	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.02	Fairey et al. 1996
0.0064		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.81	Fairey et al. 1996
0.0065	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0066	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.09	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.26	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.16	Fairey et al. 1996
0.0069		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.16	Fairey et al. 1996
0.0075	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.7	Fairey et al. 1996
0.0076	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.48	Fairey et al. 1996
0.0076	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.95	Fairey et al. 1996
0.0076	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0076	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.77	Fairey et al. 1996
0.0077		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0077	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.89	Fairey et al. 1996
0.0082	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.9	Fairey et al. 1996
0.0090		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0099	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.86	Fairey et al. 1996
0.0100		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991[c]
0.0103	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0103	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.75	Fairey et al. 1996
0.0103		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.75	Fairey et al. 1996
0.0107	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0108		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0108		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0108		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0108		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0108		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0108		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0109	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.65	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.28	Fairey et al. 1996
0.0109	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.27	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.27	Fairey et al. 1996
0.0109		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.18	Fairey et al. 1996
0.0110	0.01	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0110	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0114		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.88	Fairey et al. 1996
0.0114	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey et al. 1996
0.0120	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.15	Fairey et al. 1996
0.0120	0.0002	NC	Southern California	COA		Low density (502 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120	0.0003	NC	Southern California	COA		Low density (51 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0120	0.0004	NC	Southern California	COA		Moderate density (173 sq.m.)	Polychaetes			Word and Mearns 1979
0.0120	0.1	NC	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0120	0.2	NC	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		Normal benthic community (95; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		High density (207 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0126	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0127	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0128	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.28	Fairey et al. 1996
0.0130	0.0003	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0130	0.0003	NC	Southern California	COA		Low density (84 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0130	0.0004	NC	Southern California	COA		Low density (134 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0130	0.001	NC	Southern California	COA		Moderate density (99 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0130	0.3	NC	Southern California	COA		Moderate species richness (89 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0130		NE	Southern California	COA		High density (120 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0130		NE	Southern California	COA		Normal benthic community (83.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0132	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.94	Fairey et al. 1996
0.0132	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.22	Fairey et al. 1996
0.0132	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.22	Fairey et al. 1996
0.0140		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0140		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0142	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.19	Fairey et al. 1996
0.0142	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.98	Fairey et al. 1996
0.0143	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.77	Fairey et al. 1996
0.0143	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.77	Fairey et al. 1996
0.0143		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.72	Fairey et al. 1996
0.0150		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0150		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991[c]
0.0150		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0160		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0160		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0161		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)		1.05	Sapudar et al. 1994
0.0163	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.75	Fairey et al. 1996
0.0166	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0170	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0170	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0170		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.78	Fairey et al. 1996
0.0170		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.03	Fairey et al. 1996
0.0174	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.0176	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.16	Fairey et al. 1996
0.0180	0.0004	NC	Southern California	COA		Low density (476 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0180	0.0004	NC	Southern California	COA		Low density (61 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0180	0.001	NC	Southern California	COA		Moderate density (213 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0180	0.002	NC	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0180	0.4	NC	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0180		NE	Southern California	COA		High density (130 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0180		NE	Southern California	COA		Normal benthic community (77.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0187		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.9	Fairey et al. 1996
0.0187		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.53	Fairey et al. 1996
0.0187		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.53	Fairey et al. 1996
0.0188	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.33	Fairey et al. 1996
0.0190	0.0004	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0190	0.0005	NC	Southern California	COA		Low density (85 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0190	0.001	NC	Southern California	COA		Moderate density (202 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0190	0.002	NC	Southern California	COA		Low density (11 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0190	0.4	NC	Southern California	COA		Moderate species richness (74 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0190		NE	Southern California	COA		High density (156 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0190		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0190	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0194		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		0.29	Sapudar et al. 1994
0.0203		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		0.7	Sapudar et al. 1994
0.0210		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)		0.34	Sapudar et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0221	1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Fairey et al. 1996
0.0221	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.94	Fairey et al. 1996
0.0221		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.94	Fairey et al. 1996
0.0222	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)		1	Sapudar et al. 1994
0.0223	1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0223	1.05	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.14	Fairey et al. 1996
0.0223		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.14	Fairey et al. 1996
0.0225	1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.29	Fairey et al. 1996
0.0225	1.2	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.88	Fairey et al. 1996
0.0229		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		0.31	Sapudar et al. 1994
0.0230	0.0005	NC	Southern California	COA		Low density (382 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0230	0.001	NC	Southern California	COA		Low density (144 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0230	0.001	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0230	0.003	NC	Southern California	COA		Moderate density (42 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0230	0.01	NC	Southern California	COA		Altered benthic community (59.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0230	0.2	NC	Southern California	COA		Moderate density (76 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0230	0.5	NC	Southern California	COA		Moderate species richness (90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0237	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.27	Fairey et al. 1996
0.0237	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0237	1.2	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0241	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)		0.86	Sapudar et al. 1994
0.0253	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)		0.6	Sapudar et al. 1994
0.0256	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)		0.99	Sapudar et al. 1994
0.0257	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.28	Fairey et al. 1996
0.0257		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0257		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.28	Fairey et al. 1996
0.0260	0.001	NC	Southern California	COA		Low density (39 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0260	0.001	NC	Southern California	COA		Moderate density (186 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0260	0.002	NC	Southern California	COA		Low density (464 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0260	0.003	NC	Southern California	COA		Moderate density (53 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0260	0.5	NC	Southern California	COA		Moderate species richness (94 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0260		NE	Southern California	COA		High density (101 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0260		NE	Southern California	COA		Normal benthic community (89.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0260	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.0260	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.58	Fairey et al. 1996
0.0260	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.58	Fairey et al. 1996
0.0264	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)		1	Sapudar et al. 1994
0.0265	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.14	Fairey et al. 1996
0.0265	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.26	Fairey et al. 1996
0.0265		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Fairey et al. 1996
0.0265		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.26	Fairey et al. 1996
0.0275		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0275		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0275		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0275		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0282	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.93	Fairey et al. 1996
0.0282	1.4	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.93	Fairey et al. 1996
0.0282		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.67	Fairey et al. 1996
0.0307	1.4	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.04	Fairey et al. 1996
0.0311		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
0.0313		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.07	Fairey et al. 1996
0.0320	0.001	NC	Southern California	COA		Moderate density (902 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0320	0.001	NC	Southern California	COA		Low density (136 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0320	0.001	NC	Southern California	COA		Moderate density (407 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0320	0.004	NC	Southern California	COA		Moderate density (24 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0320		NE	Southern California	COA		High density (233 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0320		NE	Southern California	COA		Normal benthic community (74.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0320		NE	Southern California	COA		High species richness (169 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0327		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)		0.38	Sapudar et al. 1994
0.0329		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)		1.11	Sapudar et al. 1994
0.0334		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		1.03	Sapudar et al. 1994
0.0346		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.0346		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.81	Fairey et al. 1996
0.0349		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)		0.49	Sapudar et al. 1994
0.0354		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)		0.39	Sapudar et al. 1994
0.0360	0.001	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0360	0.001	NC	Southern California	COA		Low density (247 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0360	0.001	NC	Southern California	COA		Low density (32 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0360	0.001	NC	Southern California	COA		Low density (66 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0360	0.001	NC	Southern California	COA		Low density (40 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0360	0.001	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0360	0.004	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0360	0.004	NC	Southern California	COA		Moderate density (37 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0360	0.4	NC	Southern California	COA		Moderate density (41 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0360	0.7	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0360	0.7	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0360		NE	Southern California	COA		High density (95 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0360		NE	Southern California	COA		Normal benthic community (83.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0360		NE	Southern California	COA		Normal benthic community (85.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0374	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)		1.95	Sapudar et al 1994
0.0379		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)		0.8	Sapudar et al. 1994
0.0390	0.001	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0390	0.001	NC	Southern California	COA		Low density (13 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0390	0.001	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0390	0.004	NC	Southern California	COA		Moderate density (33 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0390	0.4	NC	Southern California	COA		Moderate density (22 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0390	0.8	NC	Southern California	COA		Low species richness (32 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0390		NE	Southern California	COA		Normal benthic community (94.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0404		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)		0.8	Sapudar et al. 1994
0.0404	1.9	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.83	Fairey et al. 1996
0.0411	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)		0.97	Sapudar et al. 1994
0.0413	1.9	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.75	Fairey et al. 1996
0.0418		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)		0.72	Sapudar et al. 1994
0.0424		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)		0.76	Sapudar et al. 1994
0.0451		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		0.6	Sapudar et al. 1994
0.0460	0.001	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0460	0.001	NC	Southern California	COA		Low density (29 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0460	0.001	NC	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0460	0.01	NC	Southern California	COA		Moderate density (68 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0460	0.5	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0460	0.9	NC	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0460		NE	Southern California	COA		Normal benthic community (88.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0469	2.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.42	Fairey et al. 1996
0.0469	2.4	*	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.42	Fairey et al. 1996
0.0469		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.93	Fairey et al. 1996
0.0476		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)		1.1	Sapudar et al. 1994
0.0495		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		2	Sapudar et al. 1994
0.0542		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)		0.9	Sapudar et al. 1994
0.0543		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)		1.5	Sapudar et al. 1994
0.0549		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		0.61	Sapudar et al. 1994
0.0563	2.6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.53	Fairey et al. 1996
0.0563	2.9	*	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.53	Fairey et al. 1996
0.0563		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Fairey et al. 1996
0.0570	0.001	NC	Southern California	COA		Low density (440 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0570	0.001	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0570	0.002	NC	Southern California	COA		Low density (96 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0570	0.01	NC	Southern California	COA		Moderate density (93 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0570	0.6	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0570	1.2	SG	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0570		NE	Southern California	COA		Normal benthic community (90.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0614		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		0.53	Sapudar et al. 1994
0.0616		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)		0.9	Sapudar et al. 1994
0.0626	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.0632	0.04	NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		0.9	Swartz et al. 1985[c]
0.0632		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Swartz et al. 1985[c]
0.0636	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)		0.81	Sapudar et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0650	0.001	NC	Southern California	COA		Moderate density (641 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0650	0.002	NC	Southern California	COA		Moderate density (208 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0650	0.01	NC	Southern California	COA		Moderate density (107 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0650	1.3	SG	Southern California	COA		Moderate species richness (93 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0650		NE	Southern California	COA		High density (110 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0650		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0650		NE	Southern California	COA		High density (201 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0665	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)		1.46	Sapudar et al. 1994
0.0667	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)		0.7	Sapudar et al. 1994
0.0678	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)		3.48	Sapudar et al. 1994
0.0708		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)		1.5	Sapudar et al. 1994
0.0713	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)		2.3	Sapudar et al. 1994
0.0716		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)		1.1	Sapudar et al. 1994
0.0720		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)		1.5	Sapudar et al. 1994
0.0734	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)		1.1	Sapudar et al. 1994
0.0753	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)		2.46	Fairey 1997
0.0758		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		2.8	Sapudar et al. 1994
0.0764		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991[e]
0.0773	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)		1.37	Sapudar et al. 1994
0.0774		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)		0.9	Sapudar et al. 1994
0.0787		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)		1.5	Sapudar et al. 1994
0.0790	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.0796	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)		1.36	Sapudar et al. 1994
0.0797	3.7	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.37	Fairey et al. 1996
0.0797		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Fairey et al. 1996
0.0797		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.37	Fairey et al. 1996
0.0800	0.002	NC	Southern California	COA		Low density (230 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0800	0.002	NC	Southern California	COA		Low density (65 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0800	0.002	NC	Southern California	COA		Low density (71 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0800	0.01	NC	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0800	0.8	NC	Southern California	COA		Moderate density (20 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0800	1.6	SG	Southern California	COA		Low species richness (47 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0800		NE	Southern California	COA		Normal benthic community (82.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0810	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)		2.42	Fairey 1997
0.0815		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.38	Fairey et al. 1996
0.0830		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		0.9	Sapudar et al. 1994
0.0849	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)		2.86	Sapudar et al. 1994
0.0850		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.0850	0.002	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0850	0.002	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0850	0.003	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0850	0.8	NC	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0850	1.7	SG	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0850		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0850		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0859	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.0871	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.0877	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)		5.3	Fairey 1997
0.0878	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)		5.67	Fairey 1997
0.0882	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)		1.1	Sapudar et al. 1994
0.0891		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)		1	Sapudar et al. 1994
0.0891		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)		0.4	Sapudar et al. 1994
0.0904	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)		2.85	Sapudar et al. 1994
0.0922		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)		0.29	Sapudar et al. 1994
0.0928	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)		2.51	Fairey 1997
0.0948		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)		2.1	Sapudar et al. 1994
0.0970	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)		1.1	Fairey 1997
0.0983		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)		1.4	Sapudar et al. 1994
0.0984		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991[c]
0.0991	0.03	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)		0.7	Fairey 1997
0.0999		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		1.49	Sapudar et al. 1994
0.1023		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)		0.25	Sapudar et al. 1994
0.1035		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.1035		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.1035		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.1035		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.1035		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.1035		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.1040	0.1	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.1111		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)		0.28	Sapudar et al. 1994
0.1115		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.1115		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.1126		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)		1.28	Sapudar et al. 1994
0.1128		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991[c]
0.1132		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)		1.6	Sapudar et al. 1994
0.1150	0.002	NC	Southern California	COA		Moderate density (737 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1150	0.003	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1150	0.01	NC	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1150	1.1	SG	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1150		NE	Southern California	COA		Normal benthic community (83.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1150		NE	Southern California	COA		High species richness (106 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1150		NE	Southern California	COA		High density (614 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1173		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		0.55	Sapudar et al. 1994
0.1241	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)		2	Sapudar et al. 1994
0.1275	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)		1.39	Sapudar et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1278	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)		1.6	Sapudar et al. 1994
0.1311		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)		1.4	Sapudar et al. 1994
0.1316		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991[c]
0.1330	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)		1.42	Sapudar et al. 1994
0.1334		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)		0.5	Sapudar et al. 1994
0.1398		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1.3	Swartz et al. 1986[b]
0.1398		NE	Southern California	COA		High species richness (66.6 S/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[b]
0.1398		NE	Southern California	COA		Not toxic (79.6; infaunal index)	Benthic invertebrates		1.3	Swartz et al. 1986[b]
0.1398		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.3	Swartz et al. 1986[b]
0.1398	0.02	NC	Southern California	COA		Low biomass (11.5 g/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[b]
0.1398	0.04	NC	Southern California	COA		Low density (490 N/0.1 sq.m.)	Benthic invertebrates		1.3	Swartz et al. 1986[b]
0.1398		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1.3	Swartz et al. 1986[b]
0.1416	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)		1.4	Sapudar et al. 1994
0.1418	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)		0.69	Sapudar et al. 1994
0.1430		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.1487	6.9	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.1525	7.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.24	Fairey et al. 1996
0.1540	0.003	NC	Southern California	COA		Low density (359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1540	0.004	NC	Southern California	COA		Low density (23 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1540	0.005	NC	Southern California	COA		Low density (131 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1540	0.02	NC	Southern California	COA		Moderate density (118 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1540	1.5	SG	Southern California	COA		Moderate density (52 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1540	3.1	*	Southern California	COA		Low species richness (61 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1540		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1606	0.06	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)		5.06	Fairey 1997
0.1629	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)		2	Sapudar et al. 1994
0.1632	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)		1.98	Sapudar et al. 1994
0.1680	0.003	NC	Southern California	COA		Low density (271 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1680	0.004	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1680	0.01	NC	Southern California	COA		Low density (44 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1680	0.02	NC	Southern California	COA		Moderate density (154 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1680	1.7	SG	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1680	3.4	*	Southern California	COA		Low species richness (56 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1680		NE	Southern California	COA		Normal benthic community (98.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1688	1.5	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)		2.06	Sapudar et al. 1994
0.1710	0.003	NC	Southern California	COA		Low density (518 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1710	0.004	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1710	0.01	NC	Southern California	COA		Moderate density (335 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1710	0.02	NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1710	0.1	NC	Southern California	COA		Altered benthic community (57.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1710	1.7	SG	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1710	3.5	*	Southern California	COA		Low species richness (57 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1716	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)		2.16	Sapudar et al. 1994
0.1720	0.003	NC	Southern California	COA		Low density (198 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1720	0.004	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1720	0.01	NC	Southern California	COA		Low density (33 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1720	0.02	NC	Southern California	COA		Moderate density (78 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1720	1.7	SG	Southern California	COA		Moderate density (63 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1720	3.5	*	Southern California	COA		Low species richness (43 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1720		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1740	0.003	NC	Southern California	COA		Low density (405 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1740	0.004	NC	Southern California	COA		Low density (78 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1740	0.01	NC	Southern California	COA		Moderate density (193 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1740	0.02	NC	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1740	3.5	*	Southern California	COA		Low species richness (55 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1740		NE	Southern California	COA		High density (129 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1740		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1744	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)		2.56	Sapudar et al. 1994
0.1750		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		2.5	Sapudar et al. 1994
0.1804	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)		2.18	Sapudar et al. 1994
0.1824		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		1.56	Sapudar et al. 1994
0.1866	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)		2.61	Sapudar et al. 1994
0.1923		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)		0.28	Sapudar et al. 1994
0.1956		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)		2.3	Sapudar et al. 1994
0.1984	0.07	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)		3	Fairey 1997
0.1990	0.2	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	0.3	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	4.8	*	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990	4.8	*	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.1990		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.2000		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
0.2037		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		1.1	Sapudar et al. 1994
0.2046		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)		2.31	Sapudar et al. 1994
0.2070 <	0.004	NC	Southern California	COA		Low density (467 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2070 <	0.01	NC	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2070 <	0.01	NC	Southern California	COA		Low density (62 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2070 <	0.01	NC	Southern California	COA		Moderate density (301 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2070 <	1	SG	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2070 <	2.1	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2070 <		NE	Southern California	COA		Normal benthic community (72.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2072		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)		2.34	Sapudar et al. 1994
0.2073		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)		2.5	Sapudar et al. 1994
0.2120	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)		2.34	Sapudar et al. 1994
0.2150	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)		1.96	Sapudar et al. 1994
0.2180	0.004	NC	Southern California	COA		Low density (259 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2180	0.01	NC	Southern California	COA		Low density (152 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2180	0.01	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2180	0.02	NC	Southern California	COA		Low density (3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2180	0.1	NC	Southern California	COA		Altered benthic community (48.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2180	2.2	*	Southern California	COA		Moderate density (19 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2180	4.4	*	Southern California	COA		Low species richness (48 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2192		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		1.22	Sapudar et al. 1994
0.2270	0.03	NC	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2270	4.6	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2270		NE	Southern California	COA		High density (178 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2270		NE	Southern California	COA		Normal benthic community (63.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2270		NE	Southern California	COA		High density (1359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2270		NE	Southern California	COA		High density (305 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2270		NE	Southern California	COA		High density (806 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2287	0.03	NC	Southern California	COA		Low biomass (16.1 g/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[b]
0.2287	0.03	NC	Southern California	COA		Low density (398 N/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[b]
0.2287		NE	Southern California	COA		High density (58.2 N/0.1 sq.m.)	Amphipods		0.8	Ferraro et al. 1991[b]
0.2287		NE	Southern California	COA		High species richness (62 S/0.1 sq.m.)	Benthic invertebrates		0.8	Ferraro et al. 1991[b]
0.2287		NE	Southern California	COA		Normal benthic community (92.4; infaunal index)	Benthic invertebrates		0.8	Ferraro et al. 1991[b]
0.2287		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		0.8	Ferraro et al. 1991[b]
0.2287		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Ferraro et al. 1991[b]
0.2355	2.1	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)		2.3	Sapudar et al. 1994
0.2395		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)		2.5	Sapudar et al. 1994
0.2409		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)		2.13	Sapudar et al. 1994
0.2675		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)		2.9	Sapudar et al. 1994
0.2909	13	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.37	Fairey et al. 1996
0.2909	14	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.2909	15	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.2957		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991[c]
0.3020		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)		0.89	Sapudar et al. 1994
0.3048	2.8	*	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)		2.77	Sapudar et al. 1994
0.3129	2.8	*	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)		3	Sapudar et al. 1994
0.3487		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		3.4	Sapudar et al. 1994
0.3769		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)		4.54	Sapudar et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.4520		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.4697	4.2	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)		4.27	Sapudar et al. 1994
0.4740	0.01	NC	Southern California	COA		Low density (370 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4740	0.01	NC	Southern California	COA		Low density (99 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.4740	0.01	NC	Southern California	COA		Moderate density (188 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.4740	0.1	NC	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.4740	0.1	NC	Southern California	COA		Altered benthic community (58.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.4740	4.7	*	Southern California	COA		Moderate density (31 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.4740	9.6	*	Southern California	COA		Low species richness (58 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4928		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991[c]

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.4980		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.54 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.4980		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (443 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.4980		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (12% mortality)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.4985	4.5	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)		4.3	Sapudar et al. 1994
0.4990	0.01	NC	Southern California	COA		Low density (520 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4990	0.01	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.4990	0.01	NC	Southern California	COA		Moderate density (254 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.4990	0.1	NC	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.4990	10	*	Southern California	COA		Moderate species richness (91 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4990		NE	Southern California	COA		High density (142 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.4990		NE	Southern California	COA		Normal benthic community (60.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.5084	4.6	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)		1.2	Sapudar et al. 1994
0.5167	4.7	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)		4.6	Sapudar et al. 1994
0.6038	5.5	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)		4.28	Sapudar et al. 1994
0.6080		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.6080		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.6080		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.6080		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.6080		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.6080		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.8	Bay et al. 1994
0.6290		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.6290		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.6290		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.6290		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.6290		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.6290		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.7730	0.2	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.7730		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.7730		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.7730		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.7730		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.7730		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Bay et al. 1994
0.8145		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991[c]
0.8501		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991[c]
1.00		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.13		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991[c]
1.25	20	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		2.2	Swartz et al. 1985[c]
1.25		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.2	Swartz et al. 1985[c]
1.30	0.03	NC	Southern California	COA		Low density (281 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.30	0.03	NC	Southern California	COA		Low density (193 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
1.30	0.04	NC	Southern California	COA		Low density (56 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
1.30	0.1	NC	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.30	0.4	NC	Southern California	COA		Altered benthic community (46.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.30	13	*	Southern California	COA		Moderate density (23 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
1.30	26	*	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.33	0.2	NC	Southern California	COA		Low biomass (10.2 g/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[b]
1.33	0.4	NC	Southern California	COA		Low density (355 N/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[b]
1.33	9.6	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1.4	Swartz et al. 1986[b]
1.33		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1.4	Swartz et al. 1986[b]
1.33		NE	Southern California	COA		High species richness (59.8 S/0.1 sq.m.)	Benthic invertebrates		1.4	Swartz et al. 1986[b]
1.33		NE	Southern California	COA		Not toxic (68.3; infaunal index)	Benthic invertebrates		1.4	Swartz et al. 1986[b]
1.33		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Swartz et al. 1986[b]
1.50		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991[c]
1.73	27	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		3.2	Swartz et al. 1985[c]
1.73		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Swartz et al. 1985[c]
1.79	0.04	NC	Southern California	COA		Low density (478 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.79	0.04	NC	Southern California	COA		Low density (160 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
1.79	0.1	NC	Southern California	COA		Moderate density (217 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
1.79	0.2	NC	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.79	0.5	NC	Southern California	COA		Altered benthic community (58.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.79	18	*	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
1.79	36	*	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.90	1	NC	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991[c]
1.97		NE	Southern California	COA		High density (51 N/0.1 sq.m.)	Amphipods		2.8	Ferraro et al. 1991[b]
1.97		NE	Southern California	COA		High species richness (83.2 S/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[b]
1.97		NE	Southern California	COA		Normal benthic community (75; infaunal index)	Benthic invertebrates		2.8	Ferraro et al. 1991[b]
1.97		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Ferraro et al. 1991[b]
1.97	0.3	NC	Southern California	COA		Low biomass (9.2 g/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[b]
1.97	0.3	NC	Southern California	COA		Low density (504 N/0.1 sq.m.)	Benthic invertebrates		2.8	Ferraro et al. 1991[b]
1.97	8.6	*	Southern California	COA		Low density (15.2 N/0.1 sq.m.)	Echinoderms		2.8	Ferraro et al. 1991[b]
2.06		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)		1.95	Fairey 1997
2.38		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Bay et al. 1994
2.38		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.2	Bay et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.52		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)		2.92	Fairey 1997
2.52		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)		0.7	Fairey 1997
2.60	0.9	NC	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4	Bay et al. 1994
2.60		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
2.60		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
2.60		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
2.60		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
2.60		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
2.60		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)		2.84	Fairey 1997
2.73		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)		0.85	Fairey 1997
2.85		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.48 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
2.85		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (533 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
2.85		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
3.06		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991[c]
3.19	1.1	SG	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)		1	Fairey 1997
3.33		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)		1.98	Fairey 1997
3.34		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)		2.8	Fairey 1997
3.34	1.2	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)		1.86	Fairey 1997
3.55	0.7	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Swartz et al. 1986[b]
3.55	4.8	*	Southern California	COA		Toxic (50.6; infaunal index)	Benthic invertebrates		2.4	Swartz et al. 1986[b]
3.55	25	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		2.4	Swartz et al. 1986[b]
3.55		NE	Southern California	COA		High density (21.2 N/0.1 sq.m.)	Amphipods		2.4	Swartz et al. 1986[b]
3.55		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[b]
3.55		NE	Southern California	COA		High species richness (70.4 S/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[b]
3.55		NE	Southern California	COA		High biomass (39.4 g/0.1 sq.m.)	Benthic invertebrates		2.4	Swartz et al. 1986[b]
3.57		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)		2.79	Fairey 1997
4.72		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.55 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
4.72		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (369 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
4.72		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
4.96	1.5	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[c]
4.96	1.5	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		4.2	Swartz et al. 1985[c]
4.96	1.5	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.2	Swartz et al. 1985[c]
4.96	3.3	*	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[c]
4.96	4.9	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.2	Swartz et al. 1985[c]
4.96	4.9	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		4.2	Swartz et al. 1985[c]
4.96	4.9	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		4.2	Swartz et al. 1985[c]
4.96	78	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.2	Swartz et al. 1985[c]
6.04	145	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
6.04	145	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
6.04		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
6.04		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
6.04		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
6.04		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
6.04		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
6.32	0.97	NC	Southern California	COA		Low biomass (17.7 g/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[b]

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
6.32	1.8	SG	Southern California	COA		Low density (293 N/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[b]
6.32	3.8	*	Southern California	COA		Low density (4.3 N/0.1 sq.m.)	Amphipods		3.3	Swartz et al. 1986[b]
6.32	3.8	*	Southern California	COA		Low species richness (39.3 S/0.1 sq.m.)	Benthic invertebrates		3.3	Swartz et al. 1986[b]
6.32	8.6	*	Southern California	COA		Toxic (58.6; infaunal index)	Benthic invertebrates		3.3	Swartz et al. 1986[b]
6.32	45	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.3	Swartz et al. 1986[b]
6.32		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.3	Swartz et al. 1986[b]
6.53		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Bay et al. 1994
6.53		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
6.67		NE	Southern California	COA		High species richness (87.4 S/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[b]
6.67		NE	Southern California	COA		High density (1054 N/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[b]
6.67		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		4.1	Ferraro et al. 1991[b]
6.67		NE	Southern California	COA		Normal benthic community (53.4; infaunal index)	Benthic invertebrates		4.1	Ferraro et al. 1991[b]
6.67		NE	Southern California	COA	10-d	Not toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.1	Ferraro et al. 1991[b]
6.67	6.1	*	Southern California	COA		Moderate density (23.2 N/0.1 sq.m.)	Amphipods		4.1	Ferraro et al. 1991[b]
6.67	29	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		4.1	Ferraro et al. 1991[b]
6.76		NE	Southern California	COA		High species richness (72 S/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[b]
6.76		NE	Southern California	COA		High density (856 N/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[b]
6.76		NE	Southern California	COA		High biomass (31.4 g/0.1 sq.m.)	Benthic invertebrates		3.3	Ferraro et al. 1991[b]
6.76		NE	Southern California	COA		Normal benthic community (53.3; infaunal index)	Benthic invertebrates		3.3	Ferraro et al. 1991[b]
6.76		NE	Southern California	COA	10-d	Not toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.3	Ferraro et al. 1991[b]
6.76	6.2	*	Southern California	COA		Moderate density (17.8 N/0.1 sq.m.)	Amphipods		3.3	Ferraro et al. 1991[b]
6.76	30	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.3	Ferraro et al. 1991[b]
7.92		NE	Southern California	COA		High species richness (89 S/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[b]
7.92		NE	Southern California	COA		High density (921 N/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[b]
7.92		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		3.9	Ferraro et al. 1991[b]
7.92		NE	Southern California	COA		Normal benthic community (49.5; infaunal index)	Benthic invertebrates		3.9	Ferraro et al. 1991[b]
7.92		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.9	Ferraro et al. 1991[b]
7.92	7.2	*	Southern California	COA		Moderate density (29.6 N/0.1 sq.m.)	Amphipods		3.9	Ferraro et al. 1991[b]
7.92	35	*	Southern California	COA		Low density (0.8 N/0.1 sq.m.)	Echinoderms		3.9	Ferraro et al. 1991[b]
7.95	1.1	SG	Southern California	COA		Low biomass (13.5 g/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
7.95	1.1	SG	Southern California	COA		Low density (298 N/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
7.95	7.2	*	Southern California	COA		Low density (4.2 N/0.1 sq.m.)	Amphipods		3.2	Ferraro et al. 1991[b]
7.95	35	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.2	Ferraro et al. 1991[b]
7.95		NE	Southern California	COA		High species richness (43.8 S/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
7.95		NE	Southern California	COA		Normal benthic community (52.6; infaunal index)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
7.95		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Ferraro et al. 1991[b]
8.11	1.1	SG	Southern California	COA		Low density (517 N/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
8.11	7.4	*	Southern California	COA		Low density (6.6 N/0.1 sq.m.)	Amphipods		3.2	Ferraro et al. 1991[b]
8.11	35	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.2	Ferraro et al. 1991[b]
8.11		NE	Southern California	COA		High species richness (58.8 S/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
8.11		NE	Southern California	COA		High biomass (21.4 g/0.1 sq.m.)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
8.11		NE	Southern California	COA		Normal benthic community (58.3; infaunal index)	Benthic invertebrates		3.2	Ferraro et al. 1991[b]
8.11		NE	Southern California	COA	10-d	Not toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.2	Ferraro et al. 1991[b]
8.22	2.6	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[c]
8.22	2.6	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		4.1	Swartz et al. 1985[c]
8.22	2.6	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.1	Swartz et al. 1985[c]
8.22	5.5	*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[c]
8.22	8.1	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		4.1	Swartz et al. 1985[c]
8.22	8.1	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		4.1	Swartz et al. 1985[c]
8.22	8.1	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		4.1	Swartz et al. 1985[c]
8.22	130	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		4.1	Swartz et al. 1985[c]
8.26			*ER-M*							
8.26	2.6	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[c]
8.26	2.6	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		3.7	Swartz et al. 1985[c]
8.26	2.6	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.7	Swartz et al. 1985[c]
8.26	5.6	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[c]
8.26	8.1	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		3.7	Swartz et al. 1985[c]
8.26	8.1	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		3.7	Swartz et al. 1985[c]
8.26	8.1	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		3.7	Swartz et al. 1985[c]
8.26	131	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.7	Swartz et al. 1985[c]
8.48	2.8	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.5	Bay et al. 1994
8.48		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
8.48		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
8.48		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
8.48		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
8.48		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
8.51		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.59 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
8.51		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (354 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
8.51		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
8.54	2.8	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
8.54		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.86	5	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991[c]
8.88	1.4	SG	Southern California	COA		Low biomass (14.3 g/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[b]
8.88	2.5	*	Southern California	COA		Low density (320 N/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[b]
8.88	3.4	*	Southern California	COA		Low density (9.4 N/0.1 sq.m.)	Amphipods		3.1	Swartz et al. 1986[b]
8.88	5.3	*	Southern California	COA		Low species richness (34.8 S/0.1 sq.m.)	Benthic invertebrates		3.1	Swartz et al. 1986[b]
8.88	12	*	Southern California	COA		Toxic (52.8; infaunal index)	Benthic invertebrates		3.1	Swartz et al. 1986[b]

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
8.88	64	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		3.1	Swartz et al. 1986[b]
8.88		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Swartz et al. 1986[b]
9.54	2.7	*	Southern California	COA		Low density (334 N/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[b]
9.54	5.7	*	Southern California	COA		Low density (2.2 N/0.1 sq.m.)	Amphipods		3	Swartz et al. 1986[b]
9.54	5.7	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[b]
9.54	13	*	Southern California	COA		Toxic (52.8; infaunal index)	Benthic invertebrates		3	Swartz et al. 1986[b]
9.54	68	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3	Swartz et al. 1986[b]
9.54		NE	Southern California	COA		High biomass (27.2 g/0.1 sq.m.)	Benthic invertebrates		3	Swartz et al. 1986[b]
9.54		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Swartz et al. 1986[b]
9.56	5	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991[c]
9.82	6.6	*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[c]
9.82	9.7	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		3.8	Swartz et al. 1985[c]
9.82	9.7	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[c]
9.82	9.7	*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		3.8	Swartz et al. 1985[c]
9.82	155.	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		3.8	Swartz et al. 1985[c]
9.82		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		3.8	Swartz et al. 1985[c]
9.82		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		3.8	Swartz et al. 1985[c]
9.82		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.8	Swartz et al. 1985[c]
10.2	3	*	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
10.2		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
10.2		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
10.2		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
10.2		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
10.2		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.8	Bay et al. 1994
12.6	3	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (160 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
12.6		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.72 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
12.6		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
13.6	4.1	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
13.6	4.5	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.3	Bay et al. 1994
13.6		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
13.6		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
13.6		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
13.6		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
14.8	8	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991[c]
15.0	8	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991[c]
15.9	2.2	*	Southern California	COA		Low density (357 N/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
15.9	15	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		5.4	Ferraro et al. 1991[b]
15.9	70	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		5.4	Ferraro et al. 1991[b]
15.9	2.8	*	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
15.9	2.8	*	Southern California	COA		Changed benthic community (3.6; infaunal index)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
15.9	2.2	*	Southern California	COA		Low biomass (6.3 g/0.1 sq.m.)	Benthic invertebrates		5.4	Ferraro et al. 1991[b]
15.9		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.4	Ferraro et al. 1991[b]
16.5		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	2	McLeese et al. 1982
18.0	5.4	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
18.0	6	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.1	Bay et al. 1994

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
18.0		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
18.0		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
18.0		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
18.0		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
18.0		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Bay et al. 1994
18.5		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991[c]
23.9	0.5	NC	Southern California	COA		Low density (116 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
23.9	0.6	NC	Southern California	COA		Low density (38 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
23.9	0.7	NC	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
23.9	2.7	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
23.9	7.1	*	Southern California	COA		Altered benthic community (51; infaunal index)	Benthic invertebrates			Word and Mearns 1979
23.9	238	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
23.9	485	*	Southern California	COA		Low species richness (37 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
25.0	2.8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
25.0	7.5	*	Southern California	COA		Altered benthic community (54.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
25.0	250	*	Southern California	COA		Moderate density (48 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
25.0	508	*	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
25.0		NE	Southern California	COA		High density (3059 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
25.0		NE	Southern California	COA		High density (1004 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
25.0		NE	Southern California	COA		High density (1964 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
33.3	0.7	NC	Southern California	COA		Moderate density (710 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
33.3	0.996	NC	Southern California	COA		Moderate density (424 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
33.3	3.8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
33.3	9.9	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
33.3	332	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
33.3	676	*	Southern California	COA		Low species richness (45 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
33.3		NE	Southern California	COA		High density (235 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
46.6	24	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991[c]
46.8	24	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991[c]
47.8	25	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991[c]
48.5	0.97	NC	Southern California	COA		Moderate density (895 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
48.5	1.5	SG	Southern California	COA		Moderate density (371 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
48.5	5.5	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
48.5	485	*	Southern California	COA		Low density (7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
48.5	986	*	Southern California	COA		Low species richness (46 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
48.5		NE	Southern California	COA		Normal benthic community (64.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
48.5		NE	Southern California	COA		High density (486 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
53.0	1.1	SG	Southern California	COA		Low density (534 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
53.0	1.3	SG	Southern California	COA		Low density (26 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
53.0	1.6	NC	Southern California	COA		Low density (76 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
53.0	530	*	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
53.0	1077	*	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
53.0		NE	Southern California	COA		Normal benthic community (98.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
53.0		NE	Southern California	COA		High density (349 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
68.5	35	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991[c]

Table A4-14. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
100	52	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.6	Swartz et al. 1991[c]
107	55	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991[c]
113	58	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991[c]
147	76	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991[c]
151	78	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991[c]
166	85	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991[c]
175	20	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
175	52	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
175	1750	*	Southern California	COA		Low density (5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
175	3561	*	Southern California	COA		Low species richness (36 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
175		NE	Southern California	COA		High density (2140 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
175		NE	Southern California	COA		High density (310 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
175		NE	Southern California	COA		High density (1795 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
212	109	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.2	Swartz et al. 1991[c]
227	117	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.7	Swartz et al. 1991[c]
267	64	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (250 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press
267		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.53 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press
267		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (4% mortality)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press

[a]Total DDT concentrations have been estimated from the concentrations of the p,p'-isomers (p,p'-DDT, p,p'-DDE, p,p'-DDD) by dividing by 0.889 (conversion factor was determined from the data reported by Bay et al. 1994).
[b]Total DDT concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.787 (conversion factor was determined from the data reported by Bay et al. 1994).
[c]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations (conversion factors were determined from the data reported by Bay et al. 1994).
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.
Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-15. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0056 +/- 0.006		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0132 +/- 0.013		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0149 +/- 0.016		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0162 +/- 0.031	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0189	0.1	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0189	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0189	0.1	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0214 +/- 0.076	3.8	*	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0281 +/- 0.123	2.1	*	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0299 +/- 0.028		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0300 +/- 0.028		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0420 +/- 0.044	0.2	NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0702		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
0.0810 +/- 0.064	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1075		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
0.1110		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980
0.1125 +/- 0.116		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1870 +/- 0.475		NE	Southern California	COA	10-d	Not toxic (76+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2080 +/- 0.504		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2080 +/- 0.504		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2080 +/- 0.504		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2220		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
0.2590 +/- 0.585		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2859		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
0.3928 +/- 0.280		NE	Southern California	COA		High density (54.5+/-9.9 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
0.3928 +/- 0.280		NE	Southern California	COA		High species richness (80.8+/-13.7 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.3928 +/- 0.280		NE	Southern California	COA		High density (111+/-32 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
0.4943 +/- 0.295		NE	Southern California	COA		High density (54.6+/-5.09 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
0.4951 +/- 1.04	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.5299 +/- 0.598		NE	Southern California	COA		Not toxic (74+/-7.99; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.5541 +/- 0.018		NE	Southern California	COA		High density (2585+/-2124 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5800 +/- 0.835		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.7360 +/- 1.01	25	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.7360 +/- 1.01	25	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988

Table A4-15. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.8463 +/- 0.691		NE	Southern California	COA		High density (20.9+/-0.23 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
0.8463 +/- 0.691		NE	Southern California	COA		High species richness (65.6+/-5.37 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.9404 +/- 1.12		NE	Southern California	COA		High biomass (41.6+/-27.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.9404 +/- 1.12		NE	Southern California	COA		Normal benthic community (58.2+/-18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.9404 +/- 1.12		NE	Southern California	COA	10-d	Not toxic (8+/-5.7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
1.00 >		*	San Francisco Bay, CA	COA		Toxic (reduced abundance)	Amphipods		1	Swartz et al. 1994
1.00 <		NE	San Francisco Bay, CA	COA		Not toxic (normal abundance)	Amphipods		1	Swartz et al. 1994
1.11		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
1.31 +/- 1.14		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.46 +/- 1.19	0.6	NC	Southern California	COA		Low biomass (13.4+/-3.32 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.48 +/- 1.07		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.48	0.8	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
1.48		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.52 +/- 0.878	22	*	Southern California	COA		Low density (0.07+/-0.1 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
1.60 +/- 1.31	0.8	NC	Southern California	COA		Low biomass (11.3+/-4.37 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.61 +/- 1.01	2.9	*	Southern California	COA		Low density (535+/-184 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.67 +/- 0.869		NE	Southern California	COA		High species richness (70.9+/-16.9 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.67 +/- 0.869		NE	Southern California	COA		Not toxic (62.1+/-15.8; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.73 +/- 1.07		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.79 +/- 1.21	0.9	NC	Southern California	COA		Low density (415+/-94.4 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.80 +/- 1.29	1.2	SG	Southern California	COA		Low density (358+/-76.8 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.80 +/- 1.29		NE	Southern California	COA	10-d	Not toxic (7.8+/-4.44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
1.81 +/- 0.55	1.9	SG	Southern California	COA		Low biomass (9.37+/-5.07 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.81 +/- 0.55	1.9	SG	Southern California	COA		Altered benthic community (8.6+/-8.53; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.81 +/- 0.55	1.9	SG	Southern California	COA	10-d	Toxic (21+/-1.73% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
1.83 +/- 0.922		NE	Southern California	COA	10-d	Not toxic (11.9+/-5.35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
1.90 +/- 0.238	3.8	*	Southern California	COA		Moderate density (23.5+/-5.91 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
1.90 +/- 0.238		NE	Southern California	COA		High density (944+/-101 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.97 +/- 1.57		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.97 +/- 1.57		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.97 +/- 1.57		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.97 +/- 1.57		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.00			ER-M							
2.00 +/- 0.596	5.1	*	Southern California	COA		Low density (1+/-1.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]

Table A4-15. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; summarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.00 +/- 0.596	5.1	*	Southern California	COA		Low species richness (26+/-11.5 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.00 +/- 0.596	5.1	*	Southern California	COA		Low density (8.7+/-6.01 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
2.05 +/- 0.732	7.2	*	Southern California	COA		Low density (2.4+/-5.65 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.06 +/- 0.371		NE	Southern California	COA		High biomass (25.5+/-4.21 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.08 +/- 0.934	19	*	Southern California	COA		Low density (0.2+/-0.14 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
2.18 +/- 1.23		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (425+/-82 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.33 +/- 1.20		NE	Southern California	COA		High biomass (33.3+/-8.63 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.36 +/- 0.798	4.5	*	Southern California	COA		Toxic (53.7+/-3.43; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.65 +/- 0.659	3.1	*	Southern California	COA		Low density (5.3+/-3.7 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
2.65 +/- 0.659	3.1	*	Southern California	COA		Low species richness (38.4+/-3.29 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.66 +/- 0.253	5.4	*	Southern California	COA		Low density (4.13+/-2.5 n/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.71 +/- 2.09		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
2.95	1.8	SG	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.95	1.8	SG	Southern California	COA		Toxic (3.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
3.00		*	San Francisco Bay, CA	COA	10-d	Toxicity threshold	Amphipods	ADT	1	Swartz et al. 1994
3.00 <		NE	San Francisco Bay, CA	COA	10-d	Not toxic	Amphipods	ADT	1	Swartz et al. 1994
3.09 +/- 2.10	2.1	*	Southern California	COA	35-d	Toxic (0.002+/-0.0003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.15 +/- 1.61	2.4	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
4.59 +/- 3.10	7.9	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
7.56 +/- 12.6	1.0	NG	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.57+/-0.08 mg/d)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
7.56 +/- 12.6		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (2.67+/-4.8% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
8.25		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	1	McLeese et al. 1982
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
10.4		*	Palos Verdes Shelf, CA	COA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1994
17.2 +/- 3.13	30	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
18.3 +/- 21.0	8.4	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (205+/-63.6 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
25		*	San Francisco Bay, CA	COA	10-d	LC50	Eohaustorius estuarius (amphipod)	ADT	1	Swartz et al. 1994

[a]Total DDT concentrations have been estimated from the concentrations of the p,p'-isomers (p,p'-DDT, p,p'-DDE, p,p'-DDD) by dividing by 0.889 (conversion factor was determined from the data reported by Bay et al. 1994).
[b]Total DDT concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.787 (conversion factor was determined from the data reported by Bay et al. 1994).
[c]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; (conversion factors were determined from the data reported by Bay et al. 1994). SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations.
See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0003	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0005	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0006		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0008	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0009	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0010	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0011	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0011 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0013		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0014	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0016	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0018	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0020	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0021	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0021		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0023		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0023		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0024	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0025 <	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0025 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0025	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0025	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0025	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0027	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0027	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0027	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0028	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0028		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0028	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0028	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0028		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0029	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0029	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0030	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0030		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0031		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0031		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0031	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0031	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0033		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0035		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0035		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0036	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0039	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0041	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0042		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0043	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0043	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0043	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0043		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0043	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0044	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0045		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0047	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0047	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0048	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0048	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0048		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0050	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0050	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0051	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0051	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0052	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0052	0.9	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0053	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0055		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0056	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0057	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0057	1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0059	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0059		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0060		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0060		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0062	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0062	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0064	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0068	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0072		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0072		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0072	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0072	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0074		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0074		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0078	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0078		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0080	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0081	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0083		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0092		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0093	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0093	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0094	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0096		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0098	0.7	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0098		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0099		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0101	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0101	1.8	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0104	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0108	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0108	1.9	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0109	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0109	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0115		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.0116	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0116	2.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0119	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0119		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0120	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0123		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0147	0.1	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0147		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0150		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0150		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0150		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0152	1.1	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0153		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0155	0.009	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0158	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0163		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.0165	3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0165		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0165	0.01	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0167		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0167		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0167	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0189	0.1	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0189		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0190	0.1	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0190	0.1	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0190	0.1	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0190	0.6	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0190	0.6	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0192	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0194	1.3	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0194	3.5	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0195	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0196	3.5	*	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0196		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0203		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0210	3.8	*	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0210		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0213	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0222	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0235	1.6	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0235		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0248		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0248		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0248		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0248		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0248		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0248		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0248		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0248		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0250		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0252		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0257		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0258	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0261		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0261		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0264	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0271		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0279		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0281	0.2	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0290		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0297		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0297	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0306	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0310	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0317	0.3	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0317	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0324		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0335	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0336	6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0336		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0339		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0362		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0368	2.5	*	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0368	6.6	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0370	0.02	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0389		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0421		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0422	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0424	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0433		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0451		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0455	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0472		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0474		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0480		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0489	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0505		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0525		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0558		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0564	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0580		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0585	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0590		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0602		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0617		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0618	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0621	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0639		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.0651		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0661	0.04	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0664		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0667		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0667	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0671		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0671	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0681	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0681	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0684		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0700		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0702		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0702	0.1	NC	Southern California	COA		Low density (617 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		High density (65.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		High biomass (15.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		Normal benthic community (80.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		High species richness (73.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		High density (100 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA		High density (136 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
0.0702		NE	Southern California	COA	10-d	Not Toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
0.0708		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0712		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0715	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0729	5.5	*	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0738		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0752		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0785	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0795	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0799	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0802	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0815	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0817		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.0819	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0824	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0828	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0829		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0830		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0850		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0860		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0861		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0879		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0882	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0885		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0886		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0891		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0900		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0906	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0908		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0917	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0922		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0922		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0924	0.4	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0924		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0936		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0937	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0953	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0958		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0960		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.1011	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1024	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1026		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1043	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1049		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.1075		NE	Southern California	COA		High density (93.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
0.1075	0.05	NC	Southern California	COA		Low biomass (11.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.1075	0.1	NC	Southern California	COA		Low density (490 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.1075		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
0.1075		NE	Southern California	COA		High species richness (66.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.1075		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.1075		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1097	0.97	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1100	0.98	NC	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1100	0.98	NC	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1110		*	Laboratory	SSBA	4-d	LC50	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980
0.1123	0.998	NC	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1131		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1158		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1159	1	SG	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1169		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1411	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1416	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1426	9.6	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1426	25	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1797		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1852		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2047		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.2055	1.8	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2133		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2220		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
0.2228		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2621		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2621		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2621		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2621		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2621		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2621		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.2668		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2859	0.1	NC	Southern California	COA		Low biomass (16.1 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.2859	0.2	NC	Southern California	COA		Low density (398 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.2859		NE	Southern California	COA		High density (58.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
0.2859		NE	Southern California	COA		High species richness (62 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.2859		NE	Southern California	COA		Normal benthic community (92.4; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.2859		NE	Southern California	COA		High density (208 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
0.2859		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
0.3179		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3393		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3520		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.3968		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.4092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.4237	3.8	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.4671	83	*	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.4791		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.5199		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.5410	8	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA		High density (46 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.5410		NE	Southern California	COA		High biomass (68.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA		High density (4087 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA		Normal benthic community (51.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA		High species richness (96.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA		High density (86 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
0.5410		NE	Southern California	COA	10-d	Not toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
0.5533		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.54 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.5533		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (443 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.5533		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (12% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.5671	8	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		High density (52.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		High biomass (20.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		High density (1083 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		Normal benthic community (63; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		High species richness (72.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA		High density (147 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
0.5671		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
0.6655		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
0.6868		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.7027	0.5	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7027		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7027		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7027		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7027		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7027		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.7034		NE	Southern California	COA		High density (51 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
0.7034		NE	Southern California	COA		High species richness (83.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.7034		NE	Southern California	COA		Normal benthic community (75; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.7034		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
0.7034	0.3	NC	Southern California	COA		Low biomass (9.2 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.7034	0.4	NC	Southern California	COA		Low density (504 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
0.7034	2.5	*	Southern California	COA		Low density (15.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
0.7533		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7533		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7533		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7533		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7533		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.7600		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.7862	59	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.8185		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.8616		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.9171		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.9530	0.4	NC	Southern California	COA		Low biomass (10.2 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.9530	0.6	NC	Southern California	COA		Low density (355 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.9530	8.9	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
0.9530		NE	Southern California	COA		High density (20.8 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
0.9530		NE	Southern California	COA		High species richness (59.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.9530		NE	Southern California	COA		Normal benthic community (68.3; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
0.9530		NE	Southern California	COA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
1.06		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.11		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
1.18	1.3	SG	Southern California	COA		Low biomass (11.9 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.18	1.3	SG	Southern California	COA		Altered benthic community (2.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.18	1.3	SG	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
1.18	2.1	*	Southern California	COA		Low density (661 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.18	3.0	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
1.18	3.0	*	Southern California	COA		Low species richness (18.6 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
1.18	3.0	*	Southern California	COA		Low density (10.8 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
1.18	17	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
1.19		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.28		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.44	2.5	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
1.45	49	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.45	49	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.45		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
1.45		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
1.45		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.45		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.45		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
1.48	0.8	NC	Southern California	COA	10-d	Toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
1.48	2.8	*	Southern California	COA		Altered benthic community (50.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.48	14	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
1.48		NE	Southern California	COA		High density (21.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
1.48		NE	Southern California	COA		High species richness (70.4 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.48		NE	Southern California	COA		High biomass (39.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.48		NE	Southern California	COA		High density (1656 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.63		NE	Southern California	COA		High species richness (87.4 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.63		NE	Southern California	COA		High density (1054 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.63		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.63		NE	Southern California	COA		Normal benthic community (53.4; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
1.63		NE	Southern California	COA	10-d	Not toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
1.63	3.3	*	Southern California	COA		Moderate density (23.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
1.63	5.7	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
1.68		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.68		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.80	1	SG	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
1.82	3.2	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
1.86		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.86	1.4	SG	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.88	1.4	SG	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.88		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.88		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.88		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.88		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.88		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.91	0.8	NC	Southern California	COA		Low biomass (17.7 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.91	1.3	SG	Southern California	COA		Low density (293 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.91	2.3	*	Southern California	COA		Low density (4.3 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
1.91	2.3	*	Southern California	COA		Low species richness (39.3 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.91	3.6	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
1.91	18	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
1.91		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
1.98		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
1.98		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.99	3.6	*	Southern California	COA		Low density (372 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.00	2.1	*	Southern California	COA		Low biomass (6.4 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.00	2.1	*	Southern California	COA		Altered benthic community (4.6; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.00	2.1	*	Southern California	COA	10-d	Toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
2.00	5.1	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
2.00	5.1	*	Southern California	COA		Low species richness (15.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.00	5.1	*	Southern California	COA		Low density (1.4 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
2.00	29	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
2.03	4.1	*	Southern California	COA		Moderate density (29.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.03	7.1	*	Southern California	COA		Low density (0.8 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.03		NE	Southern California	COA		High species richness (89 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.03		NE	Southern California	COA		High density (921 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.03		NE	Southern California	COA		High biomass (24.6 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.03		NE	Southern California	COA		Normal benthic community (49.5; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.03		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
2.04		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.48 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.04		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (533 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.04		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.05			ER-M							
2.05	4.1	*	Southern California	COA		Moderate density (17.8 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.05	7.2	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.05		NE	Southern California	COA		High species richness (72 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.05		NE	Southern California	COA		High density (856 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.05		NE	Southern California	COA		High biomass (31.4 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.05		NE	Southern California	COA		Normal benthic community (53.3; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.05		NE	Southern California	COA	10-d	Not toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
2.23	2.4	*	Southern California	COA		Low biomass (9.8 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.23	2.4	*	Southern California	COA		Altered benthic community (18.4; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.23	2.4	*	Southern California	COA	10-d	Toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
2.23	4.0	*	Southern California	COA		Low density (307 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.23	5.7	*	Southern California	COA		Low density (2.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
2.23	5.7	*	Southern California	COA		Low species richness (28.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.23	5.7	*	Southern California	COA		Low density (15.6 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
2.23	32	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
2.35	4.1	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
2.49		NE	Southern California	COA		High species richness (43.8 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.49		NE	Southern California	COA		Normal benthic community (52.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.49		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
2.49	1.2	SG	Southern California	COA		Low biomass (13.5 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.49	1.3	SG	Southern California	COA		Low density (298 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.49	5	*	Southern California	COA		Low density (4.2 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.49	8.7	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.53		NE	Southern California	COA		High species richness (58.8 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.53		NE	Southern California	COA		High biomass (21.4 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.53		NE	Southern California	COA		Normal benthic community (58.3; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.53		NE	Southern California	COA	10-d	Not toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
2.53	1.3	SG	Southern California	COA		Low density (517 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.53	5.1	*	Southern California	COA		Low density (6.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.53	8.9	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.58	4.7	*	Southern California	COA		Low density (720 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.58	6.6	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1985[c]
2.58	6.6	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.58	6.6	*	Southern California	COA		Low density (7.0 N/0.1 sq.m.)	Crustaceans		1	Swartz et al. 1985[c]
2.58	37	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1985[c]
2.58		NE	Southern California	COA		High biomass (61.5 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.58		NE	Southern California	COA		Normal benthic community (37.1; infaunal index)	Benthic invertebrates		1	Swartz et al. 1985[c]
2.58		NE	Southern California	COA	10-d	Not toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1985[c]
2.69	1.8	SG	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.69		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.69		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.69		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.69		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.69		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.69		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.59 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.69		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (354 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.69		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.72		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
2.72		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
2.87	1.2	SG	Southern California	COA		Low biomass (14.3 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.87	1.9	SG	Southern California	COA		Low density (320 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.87	3.4	*	Southern California	COA		Low density (9.4 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
2.87	3.4	*	Southern California	COA		Low species richness (34.8 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.87	5.4	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
2.87	27	*	Southern California	COA		Low density (0.2 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
2.87		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]
2.94	1.8	SG	Southern California	COA		Low species richness (19.2 S/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.94	1.8	SG	Southern California	COA		Altered benthic community (3.6; infaunal index)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.95	1.4	SG	Southern California	COA		Low biomass (6.3 g/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.95	1.5	SG	Southern California	COA		Low density (357 N/0.1 sq.m.)	Benthic invertebrates		1	Ferraro et al. 1991[b]
2.95	6	*	Southern California	COA		Low density (1.6 N/0.1 sq.m.)	Amphipods		1	Ferraro et al. 1991[b]
2.95	10	*	Southern California	COA		Low density (0.4 N/0.1 sq.m.)	Echinoderms		1	Ferraro et al. 1991[b]
2.95		NE	Southern California	COA	10-d	Not toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Ferraro et al. 1991[b]
3.02		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
3.05		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.05		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.05		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.05		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.05		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.05		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
3.05	2.3	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
3.17	2.4	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
3.17	2.1	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.17		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.17		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.17		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.17		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
3.18	2.2	*	Southern California	COA		Low density (334 N/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
3.18	3.8	*	Southern California	COA		Low density (2.2 N/0.1 sq.m.)	Amphipods		1	Swartz et al. 1986[b]
3.18	3.8	*	Southern California	COA		Low species richness (41.2 S/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
3.18	6	*	Southern California	COA		Altered benthic community (52.8; infaunal index)	Benthic invertebrates		1	Swartz et al. 1986[b]
3.18	30	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms		1	Swartz et al. 1986[b]
3.18		NE	Southern California	COA		High biomass (27.2 g/0.1 sq.m.)	Benthic invertebrates		1	Swartz et al. 1986[b]
3.18		NE	Southern California	COA	10-d	Not toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1986[b]

Table A4-16. A summary of the available data on the biological effects associated with sediment-sorbed Total DDT (mg/kg DW at 1% OC; unsummarized data set).

Total DDT Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
3.19	1.8	SG	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
3.21		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
3.44	3	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (160 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.44		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.72 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.44		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.45		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.55 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.45		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (369 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.45		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.60		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
4.08	7.1	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
4.31	7.4	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
5.62	10	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
5.81		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
5.81		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
5.81		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
5.81		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
5.81		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
5.81	3.9	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
5.81	4.5	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
6.09	11	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
8.25		NE	Laboratory	SSBA	12-d	LC0	Nereis virens (sand worm)	ADT	1	McLeese et al. 1982
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on wet weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on diameter)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
8.70		NE	Laboratory	SSBA	35-d	Not toxic (no effect on gonad weight)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
11.0	19	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
11.7	20	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
13.2	23	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
16.7	29	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
17.4	30	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
17.4	30	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
17.8	31	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
18.6	32	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
20.8	36	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
21.2	37	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991[c]
33.2	64	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (250 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
33.2		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.53 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
33.2		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (4% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press

[a]Total DDT concentrations have been estimated from the concentrations of the p,p'-isomers (p,p'-DDT, p,p'-DDE, p,p'-DDD) by dividing by 0.889 (conversion factor was determined from the data reported by Bay et al. 1994).
[b]Total DDT concentrations have been estimated from the concentrations of p,p'-DDE by dividing by 0.787 (conversion factor was determined from the data reported by Bay et al. 1994).
[c]Total DDT equals the sum of SUM DDT, SUM DDD and SUM DDE. SUM DDT concentrations were calculated by dividing p,p'-DDT by 0.894; SUM DDD concentrations were calculated by dividing p,p'-DDD by 0.863; (conversion factors were determined from the data reported by Bay et al. 1994). SUM DDE was calculated as the sum of the reported p,p'-DDE and o,p'-DDE concentrations.
See Appendix 2 for glossary of acronyms.

Table A4-17. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; summarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0080 +/- 0.005		NE	Southern California	COA		High species richness (133+/-32.5 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0094 +/- 0.011		NE	Southern California	COA		High density (229+/-60.3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0261 +/- 0.038	2.8	*	Southern California	COA		Moderate density (73.8+/-40.9 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0515 +/- 0.070		NE	Southern California	COA		High density (144+/-42.7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0740 +/- 0.105		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0740 +/- 0.105		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0891 +/- 0.250	0.05	NC	Southern California	COA		Low density (68.2+/-47.8 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0948 +/- 0.257	0.04	NC	Southern California	COA		Low density (370+/-122 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1062 +/- 0.316	0.1	NC	Southern California	COA		Low density (79.8+/-32.6 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1177 +/- 0.332	2.3	*	Southern California	COA		Moderate density (48.8+/-19.5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1253 +/- 0.520		NE	Southern California	COA		Normal benthic community (83.8+/-12.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1258 +/- 0.382	16	*	Southern California	COA		Moderate species richness (81.1+/-8.90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2090 +/- 0.393		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2090 +/- 0.393		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2090 +/- 0.393		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2377 +/- 0.237		NC	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.2395 +/- 0.379		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.2404 +/- 0.455		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
0.3221 +/- 0.731		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991
0.4083 +/- 0.905	0.3	NC	Southern California	COA		Moderate density (259+/-85.9 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.4835	2.3	*	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	2	*	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	2	*	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6409 +/- 0.837	5.1	*	Southern California	COA		Altered benthic community (53.8+/-5.63; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.7230 +/- 1.60	90	*	Southern California	COA		Low species richness (50.5+/-9.2 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.8173 +/- 0.472	11	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
0.8173 +/- 0.472	11	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
0.8800 +/- 1.35	0.4	NC	Southern California	COA		Moderate density (786+/-107 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.00		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.02 +/- 1.74	109	*	Southern California	COA		Low density (3.13+/-4.49 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.43 +/- 2.60		NE	Southern California	COA		High density (1068+/-636 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
1.97 +/- 2.33		NE	Southern California	COA		High density (418+/-275 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
2.10			*ER-M*							
2.10		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988

Table A4-17. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; summarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.10		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (58% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (87% mortality; w/mercury, zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (48% mortality; w/mercury, zinc)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		NE	Laboratory	SSBA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		NE	Laboratory	SSBA	10-d	Not toxic (21% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.15 +/- 3.04		NE	Southern California	COA		High density (1947+/-843 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.61 +/- 6.10	8.1	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991
3.31 +/- 2.27	64	*	Southern California	COA		Low density (4+/-2.45 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
3.39		NE	Laboratory	SSBA	96-h	LC0	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
4.20		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
4.60		*	Laboratory	SSBA	10-d	Toxic (19% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		*	Laboratory	SSBA	10-d	Toxic (25% mortality; w/zinc, mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (4% mortality; w/zinc, mercury)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.80		NE	Laboratory	SSBA	10-d	Not toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
5.00		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
5.45		*	Laboratory	SSBA	10-d	LC10	Rhepoxynius abronius (amphipod)	ADT	0.41	Swartz et al. 1988
6.58	53	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
8.30		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
8.80		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.41	Swartz et al. 1988
9.40		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
10.6 +/- 5.34	33	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991
83.1		*	Laboratory	SSBA	96-h	Toxic (>10% mortality)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
117		*	Laboratory	SSBA	96-h	LC50 (males)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
182		*	Laboratory	SSBA	96-h	LC50 (both sexes)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
251		*	Laboratory	SSBA	96-h	LC50 (females)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0010 <	0.0002	NC	Southern California	COA		Low density (534 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010 <	0.0003	NC	Southern California	COA		Low density (26 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0010 <	0.0003	NC	Southern California	COA		Low density (76 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0010 <	0.01	NC	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0010 <	0.1	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0010 <		NE	Southern California	COA		Normal benthic community (98.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0010 <		NE	Southern California	COA		High density (349 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0020	0.001	NC	Southern California	COA		Moderate density (833 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020	0.001	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020	0.001	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0020	0.001	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0020	0.001	NC	Southern California	COA		Low density (112 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0020	0.04	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0020	0.04	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0020	0.2	NC	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0020	0.3	NC	Southern California	COA		Low species richness (41 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		Normal benthic community (98.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		High species richness (124 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020		NE	Southern California	COA		High density (224 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0020		NE	Southern California	COA		High density (610 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0030	0.001	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.001	NC	Southern California	COA		Low density (374 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.001	NC	Southern California	COA		Low density (442 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (36 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (47 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (50 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (110 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0030	0.002	NC	Southern California	COA		Low density (119 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0030	0.1	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0030	0.1	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0030	0.1	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0030	0.3	NC	Southern California	COA		Moderate density (130 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0030	0.3	NC	Southern California	COA		Moderate density (142 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0030	0.4	NC	Southern California	COA		Moderate species richness (67 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.4	NC	Southern California	COA		Moderate species richness (72 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030	0.4	NC	Southern California	COA		Moderate species richness (73 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		Normal benthic community (94.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		Normal benthic community (95.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0030		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040	0.002	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.002	NC	Southern California	COA		Low density (84 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0040	0.003	NC	Southern California	COA		Low density (134 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0040	0.4	NC	Southern California	COA		Moderate density (99 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040	0.5	NC	Southern California	COA		Moderate species richness (89 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		High density (120 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0040		NE	Southern California	COA		Normal benthic community (83.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0045		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0050	0.002	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (382 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (502 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Low density (32 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Low density (51 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Low density (144 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Moderate density (173 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Low density (40 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.003	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.04	NC	Southern California	COA		Altered benthic community (59.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050 <	0.1	NC	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate density (76 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050	0.5	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0050	0.5	NC	Southern California	COA		Moderate density (42 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0050	0.6	NC	Southern California	COA		Moderate species richness (90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.6	NC	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.6	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		High density (95 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050		NE	Southern California	COA		Normal benthic community (83.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		Normal benthic community (95; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		High density (207 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0060	0.003	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060	0.003	NC	Southern California	COA		Low density (464 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060	0.003	NC	Southern California	COA		Low density (29 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0060	0.003	NC	Southern California	COA		Low density (39 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0060	0.004	NC	Southern California	COA		Moderate density (186 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0060	0.004	NC	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0060	0.1	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0060	0.6	NC	Southern California	COA		Moderate density (53 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0060	0.6	NC	Southern California	COA		Moderate density (68 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0060	0.8	NC	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0060	0.8	NC	Southern California	COA		Moderate species richness (94 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0060		NE	Southern California	COA		High density (101 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0060		NE	Southern California	COA		Normal benthic community (88.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0060		NE	Southern California	COA		Normal benthic community (89.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0060		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0060		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0080	0.004	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0080	0.004	NC	Southern California	COA		Low density (13 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0080	0.01	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0080	0.2	NC	Southern California	COA		Moderate density (22 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0080	0.9	NC	Southern California	COA		Moderate density (33 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0080	1.0	NG	Southern California	COA		Low species richness (32 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0080		NE	Southern California	COA		Normal benthic community (94.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0085		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0085		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0085		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0085		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0085		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0085		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0090		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0100	0.005	NC	Southern California	COA		Moderate density (902 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0100	0.01	NC	Southern California	COA		Low density (136 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0100	0.01	NC	Southern California	COA		Moderate density (407 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0100	0.04	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0100	1.1	SG	Southern California	COA		Moderate density (24 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0100		NE	Southern California	COA		High density (233 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0100		NE	Southern California	COA		Normal benthic community (74.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0100		NE	Southern California	COA		High species richness (169 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0100		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0100		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0100		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0100		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0100		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0100		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0120	0.01	NC	Southern California	COA		Moderate density (737 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (440 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (476 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (61 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0120	0.01	NC	Southern California	COA		Moderate density (213 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0120	0.01	NC	Southern California	COA		Low density (96 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0120	0.2	NC	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0120	0.2	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0120	1.3	SG	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0120	1.3	SG	Southern California	COA		Moderate density (93 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0120	1.3	SG	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0120	1.5	SG	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120	1.5	SG	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		High density (130 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0120		NE	Southern California	COA		Normal benthic community (77.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		Normal benthic community (83.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		Normal benthic community (90.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		High species richness (106 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0120		NE	Southern California	COA		High density (614 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0150	0.01	NC	Southern California	COA		Low density (247 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0150	0.01	NC	Southern California	COA		Low density (66 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0150	0.01	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0150	0.3	NC	Southern California	COA		Moderate density (41 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0150	1.6	SG	Southern California	COA		Moderate density (37 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0150	1.9	SG	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0150		NE	Southern California	COA		Normal benthic community (85.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0200		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991
0.0210	0.01	NC	Southern California	COA		Moderate density (641 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0210	0.01	NC	Southern California	COA		Low density (515 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0210	0.01	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0210	0.01	NC	Southern California	COA		Moderate density (208 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0210	0.01	NC	Southern California	COA		Moderate density (212 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0210	0.4	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0210	2.2	*	Southern California	COA		Moderate density (107 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0210	2.6	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0210	2.6	*	Southern California	COA		Moderate species richness (93 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0210		NE	Southern California	COA		High density (110 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0210		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0210		NE	Southern California	COA		Normal benthic community (91.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0210		NE	Southern California	COA		High density (184 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0210		NE	Southern California	COA		High density (201 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0240	0.01	NC	Southern California	COA		Low density (467 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0240	0.01	NC	Southern California	COA		Low density (62 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0240	0.02	NC	Southern California	COA		Moderate density (301 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0240	0.5	NC	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0240	2.6	*	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0240	3.0	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0240		NE	Southern California	COA		Normal benthic community (72.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0250	0.01	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0250	0.01	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0250	0.02	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0250	0.5	NC	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0250	3.1	*	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0250		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0250		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0260	0.01	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0260	3.3	*	Southern California	COA		Low species richness (62 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0260		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0280	0.01	NC	Southern California	COA		Low density (271 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0280	0.01	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0280	0.02	NC	Southern California	COA		Low density (44 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0280	0.5	NC	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0280	3.0	*	Southern California	COA		Moderate density (154 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0280	3.5	*	Southern California	COA		Low species richness (56 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0280		NE	Southern California	COA		Normal benthic community (98.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0300		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991
0.0310	0.01	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310	0.02	NC	Southern California	COA		Low density (85 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0310	0.02	NC	Southern California	COA		Moderate density (202 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0310	3.3	*	Southern California	COA		Low density (11 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0310	3.9	*	Southern California	COA		Moderate species richness (74 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310		NE	Southern California	COA		High density (156 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0310		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0365		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japónica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0365		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0365		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0365		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0365		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0365		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0370		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0400		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991
0.0510	0.02	NC	Southern California	COA		Low density (359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0510	0.03	NC	Southern California	COA		Low density (23 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0510	0.04	NC	Southern California	COA		Low density (131 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0510	1.0	NC	Southern California	COA		Moderate density (52 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0510	5.4	*	Southern California	COA		Moderate density (118 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0510	6.4	*	Southern California	COA		Low species richness (61 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0510		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0530	0.02	NC	Southern California	COA		Low density (520 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0530	0.03	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0530	0.04	NC	Southern California	COA		Moderate density (254 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0530	5.6	*	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0530	6.6	*	Southern California	COA		Moderate species richness (91 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0530		NE	Southern California	COA		High density (142 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0530		NE	Southern California	COA		Normal benthic community (60.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0550		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991
0.0610	0.03	NC	Southern California	COA		Low density (370 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0610	0.03	NC	Southern California	COA		Low density (99 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0610	0.04	NC	Southern California	COA		Moderate density (188 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0610	0.5	NC	Southern California	COA		Altered benthic community (58.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0610	1.2	SG	Southern California	COA		Moderate density (31 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0610	6.5	*	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0610	7.6	*	Southern California	COA		Low species richness (58 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0750		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991
0.0860	0.7	NC	Southern California	COA		Altered benthic community (45; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0860	9.1	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0860	11	*	Southern California	COA		Moderate species richness (83 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0860		NE	Southern California	COA		High density (195 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0860		NE	Southern California	COA		High density (1231 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0860		NE	Southern California	COA		High density (385 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0860		NE	Southern California	COA		High density (616 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0900	0.04	NC	Southern California	COA		Low density (281 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0900	0.05	NC	Southern California	COA		Low density (193 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0900	0.1	NC	Southern California	COA		Low density (56 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0900	0.7	NC	Southern California	COA		Altered benthic community (46.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0900	1.7	SG	Southern California	COA		Moderate density (23 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0900	9.6	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0900	11	*	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0900		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991
0.0950		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991
0.1030	0.05	NC	Southern California	COA		Low density (405 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1030	0.1	NC	Southern California	COA		Low density (78 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1030	0.1	NC	Southern California	COA		Moderate density (193 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1030	11	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1030	13	*	Southern California	COA		Low species richness (55 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1030		NE	Southern California	COA		High density (129 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1030		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1080	0.1	NC	Southern California	COA		Low density (198 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1080	0.1	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1080	0.1	NC	Southern California	COA		Low density (33 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1080	2.1	*	Southern California	COA		Moderate density (63 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1080	11	*	Southern California	COA		Moderate density (78 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1080	14	*	Southern California	COA		Low species richness (43 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1080		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1250		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991
0.1360	0.1	NC	Southern California	COA		Low density (230 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1360	0.1	NC	Southern California	COA		Low density (65 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1360	0.1	NC	Southern California	COA		Low density (71 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1360	2.6	*	Southern California	COA		Moderate density (20 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1360	14	*	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1360	17	*	Southern California	COA		Low species richness (47 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1360		NE	Southern California	COA		Normal benthic community (82.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1400		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991
0.1550	0.1	NC	Southern California	COA		Low density (478 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1550	0.1	NC	Southern California	COA		Low density (160 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1550	0.1	NC	Southern California	COA		Moderate density (217 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1550	1.2	SG	Southern California	COA		Altered benthic community (58.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1550	3.0	*	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1550	16	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1550	19	*	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1700		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991
0.1880	0.1	NC	Southern California	COA		Low density (259 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1880	0.1	NC	Southern California	COA		Low density (152 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1880	0.1	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1880	1.5	SG	Southern California	COA		Altered benthic community (48.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1880	3.7	*	Southern California	COA		Moderate density (19 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1880	20	*	Southern California	COA		Low density (3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1880	24	*	Southern California	COA		Low species richness (48 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2000		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991
0.2195	0.9	NC	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.2195		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2195		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2195		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2195		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2195		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2200		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.2360	25	*	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2360	30	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2360		NE	Southern California	COA		High density (178 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2360		NE	Southern California	COA		Normal benthic community (63.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2360		NE	Southern California	COA		High density (1359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2360		NE	Southern California	COA		High density (305 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2360		NE	Southern California	COA		High density (806 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2360		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.2360		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2700		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991
0.3600		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991
0.4120	0.2	NC	Southern California	COA		Low density (518 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4120	0.2	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.4120	0.3	NC	Southern California	COA		Moderate density (335 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.4120	3.3	*	Southern California	COA		Altered benthic community (57.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.4120	8	*	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.4120	44	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.4120	52	*	Southern California	COA		Low species richness (57 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.4835	2	*	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	2.3	*	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	2.3	*	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	2.3	*	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4835	6.5	*	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.4835	6.5	*	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.4840		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.5150	1.6	SG	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991
0.5350	1.7	SG	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991
0.8750	2.7	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991
1.00		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
1.15			ER-M							
1.15		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
1.15	16	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.15	16	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.15		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
1.15		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.15		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.15		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.34	4.1	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991
1.43	0.7	NC	Southern California	COA		Low density (116 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.43	0.7	NC	Southern California	COA		Low density (38 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
1.43	1.0	SG	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
1.43	11	*	Southern California	COA		Altered benthic community (51; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.43	28	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
1.43	152	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.43	179	*	Southern California	COA		Low species richness (37 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.68	13	*	Southern California	COA		Altered benthic community (54.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.68	33	*	Southern California	COA		Moderate density (48 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
1.68	179	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.68	211	*	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.68		NE	Southern California	COA		High density (3059 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.68		NE	Southern California	COA		High density (1004 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
1.68		NE	Southern California	COA		High density (1964 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.10		*	Laboratory	SSBA	10-d	Toxic (48% mortality; w/mercury, zinc)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.10		NE	Laboratory	SSBA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		NE	Laboratory	SSBA	10-d	Not toxic (21% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (58% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.10		*	Laboratory	SSBA	10-d	Toxic (87% mortality; w/mercury, zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
2.15	6.7	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991
2.18	6.8	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991
2.30	1.1	SG	Southern California	COA		Moderate density (710 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.30	1.6	SG	Southern California	COA		Moderate density (424 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.30	18	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
2.30	45	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
2.30	244	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
2.30	287	*	Southern California	COA		Low species richness (45 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.30		NE	Southern California	COA		High density (235 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
2.84		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991
2.91	1.4	SG	Southern California	COA		Moderate density (895 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.91	2.0	*	Southern California	COA		Moderate density (371 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.91	57	*	Southern California	COA		Low density (7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
2.91	310	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
2.91	364	*	Southern California	COA		Low species richness (46 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.91		NE	Southern California	COA		Normal benthic community (64.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
2.91		NE	Southern California	COA		High density (486 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
3.39		NE	Laboratory	SSBA	96-h	LC0	Crangon septemspinosa (shrimp)	ADT	0.28	McLeese and Metcalfe 1980
3.68	11	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991
4.20		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (4% mortality; w/zinc, mercury)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		*	Laboratory	SSBA	10-d	Toxic (19% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.60		*	Laboratory	SSBA	10-d	Toxic (25% mortality; w/zinc, mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
4.80		NE	Laboratory	SSBA	10-d	Not toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Swartz et al. 1988
5.00		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	0.9	Plesha et al. 1988
5.78	18	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991
5.45		*	Laboratory	SSBA	10-d	LC10	Rhepoxynius abronius (amphipod)	ADT	0.41	Swartz et al. 1988
6.16	19	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991
6.52	20	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991
6.58	53	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
6.58	128	*	Southern California	COA		Low density (5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
6.58	700	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-18. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
6.58	823	*	Southern California	COA		Low species richness (36 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
6.58		NE	Southern California	COA		High density (2140 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
6.58		NE	Southern California	COA		High density (310 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
6.58		NE	Southern California	COA		High density (1795 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
6.82	21	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991
7.72	24	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991
8.30		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
8.80		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	0.41	Swartz et al. 1988
9.40		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.51	Swartz et al. 1988
9.81	30	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.6	Swartz et al. 1991
13.2	41	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991
13.3	41	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.2	Swartz et al. 1991
16.1	50	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.7	Swartz et al. 1991
19.8	61	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991
83.1		*	Laboratory	SSBA	96-h	Toxic (>10% mortality)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
117		*	Laboratory	SSBA	96-h	LC50 (males)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
182		*	Laboratory	SSBA	96-h	LC50 (both sexes)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993
251		*	Laboratory	SSBA	96-h	LC50 (females)	Microarthridion littorale (copepod)	ADT	3.9	DiPinto et al. 1993

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.

Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-19. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW at 1% OC; summarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0459	0.59	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0459	0.59	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0459	0.59	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0499 +/- 0.069		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0499 +/- 0.069		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0694 +/- 0.105		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0747 +/- 0.097		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0780 +/- 0.103		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0780 +/- 0.103		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0780 +/- 0.103		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0852 +/- 0.097	1.2	SG	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1104 +/- 0.112		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1613 +/- 0.163	3.2	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1613 +/- 0.163	3.2	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.4563 +/- 0.245	4.1	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.08		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
1.11		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
1.24 +/- 0.437	11	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.12		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
5.38			*ER-M*							
5.38		*	Laboratory	SSBA	10-d	Toxic (48% mortality; w/mercury, zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (21% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (58% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (87% mortality; w/mercury, zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.56		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (4% mortality; w/zinc, mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		*	Laboratory	SSBA	10-d	Toxic (19% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		*	Laboratory	SSBA	10-d	Toxic (25% mortality; w/zinc, mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
12.1		NE	Laboratory	SSBA	96-h	LC0	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980

Table A4-19. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW at 1% OC; summarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
12.3		NE	Laboratory	SSBA	10-d	Not toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
13.3		*	Laboratory	SSBA	10-d	LC10	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
18.4		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
21.3		*	Laboratory	SSBA	96-h	Toxic (>10% mortality)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
21.5		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
30.0		*	Laboratory	SSBA	96-h	LC50 (males)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
46.7		*	Laboratory	SSBA	96-h	LC50 (both sexes)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
64.4		*	Laboratory	SSBA	96-h	LC50 (females)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993

See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-20. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW at 1% OC; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0042		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0047		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0054		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0093		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0099		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0137		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0140	0.2	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0218		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0260		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0345		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0459		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0460	0.9	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0460	0.9	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0460	0.6	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0460	0.6	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0460	0.6	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0500	0.7	NC	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0551		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988

Table A4-20. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW at 1% OC; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0551		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0588		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0652		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0652		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0652		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0652		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0652		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0652		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0657		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0657		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0661		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0698		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0735		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1000		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1040		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1173		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1471		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1960		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1960		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1960		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1960		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1960		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1964		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1978		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2000	3.0	*	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2471	2.2	*	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2550	2.3	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2551	2.3	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2764		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2767		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2767		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2767		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2767		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2770	5.5	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2770	5.5	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3362	3.0	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.3916	3.5	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.4629		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.5129	4.6	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.7111	6.4	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.8221	7.4	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.8305	7.5	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.8913	8.1	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.9501	8.6	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991

Table A4-20. A summary of the available data on the biological effects associated with sediment-sorbed Aroclor 1254 (mg/kg DW at 1% OC; unsummarized data set).

Aroclor 1254 Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.9572	8.7	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.08		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
1.11		*	Laboratory	SSBA	10-d	Toxic (>30% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
1.14	10	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.31			*ER-M*							
1.31	12	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.51	14	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.56	14	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.12	19	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.12		*	Laboratory	SSBA	12-d	Toxic (reduced reproductive capacity)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
5.38		*	Laboratory	SSBA	10-d	Toxic (48% mortality; w/mercury, zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		NE	Laboratory	SSBA	10-d	Not toxic (21% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (55% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (58% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.38		*	Laboratory	SSBA	10-d	Toxic (87% mortality; w/mercury, zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
5.56		*	Laboratory	SSBA	10-d	Toxic (>80% mortality; w/chlorinated hydrocarbons)	Rhepoxynius abronius (amphipod)	ADT	1	Plesha et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/zinc)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (3% mortality; w/mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (4% mortality; w/zinc, mercury)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality; w/fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		*	Laboratory	SSBA	10-d	Toxic (19% mortality; w/zinc, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
9.02		*	Laboratory	SSBA	10-d	Toxic (25% mortality; w/zinc, mercury, fluoranthene)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
12.1		NE	Laboratory	SSBA	96-h	LC0	Crangon septemspinosa (shrimp)	ADT	1	McLeese and Metcalfe 1980
12.3		NE	Laboratory	SSBA	10-d	Not toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
13.3		*	Laboratory	SSBA	10-d	LC10	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
18.4		NE	Laboratory	SSBA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
21.3		*	Laboratory	SSBA	96-h	Toxic (>10% mortality)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
21.5		*	Laboratory	SSBA	10-d	LC50	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1988
30.0		*	Laboratory	SSBA	96-h	LC50 (males)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
46.7		*	Laboratory	SSBA	96-h	LC50 (both sexes)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993
64.4		*	Laboratory	SSBA	96-h	LC50 (females)	Microarthridion littorale (copepod)	ADT	1	DiPinto et al. 1993

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.

Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-21. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; summarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0147 +/- 0.017		NE	Southern California	COA		High density (229+/-60.3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0319 +/- 0.040	2.2	*	Southern California	COA		Moderate density (73.8+/-40.9 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0627 +/- 0.093		NE	Southern California	COA		High species richness (133+/-32.5 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0791 +/- 0.101		NE	Southern California	COA		High density (144+/-42.7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0876 +/- 0.092		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.27+/-0.89	Sapudar et al. 1994
0.0960 +/- 0.137		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.0960 +/- 0.137		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.38+/-1.29	Anderson et al. 1988
0.1204 +/- 0.335	0.04	NC	Southern California	COA		Low density (68.2+/-47.8 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1233 +/- 0.345	0.04	NC	Southern California	COA		Low density (370+/-122 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1398 +/- 0.427	0.1	NC	Southern California	COA		Low density (79.8+/-32.6 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1516 +/- 0.408	2	SG	Southern California	COA		Moderate density (48.8+/-19.5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1651 +/- 0.470	3	*	Southern California	COA		Moderate species richness (81.1+/-8.90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1690 +/- 0.687		NE	Southern California	COA		Normal benthic community (83.8+/-12.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1769 +/- 0.152	2.02	*	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93+/-1.04	Sapudar et al. 1994
0.1887 +/- 0.268	0.63	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.2008 +/- 0.386		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4+/-0.6	Fairey et al. 1996
0.2415 +/- 0.126	0.91	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.83+/-1.77	Fairey 1997
0.2441 +/- 0.360		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.87+/-0.88	Fairey et al. 1996
0.2652 +/- 0.050		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.10+/-0.91	Fairey 1997
0.2700 +/- 0.508		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2700 +/- 0.508		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2700 +/- 0.508		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.73+/-1.55	Anderson et al. 1988
0.2810 +/- 0.395	1.2	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.66+/-0.6	Fairey et al. 1996
0.3010 +/- 0.367		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.74+/-0.79	Fairey et al. 1996
0.3079 +/- 0.359	1.5	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.91+/-0.79	Fairey et al. 1996
0.3140 +/- 0.588		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	2+/-1.73	Anderson et al. 1988
0.3159 +/- 0.495		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	2.71+/-3.28	Anderson et al. 1988
0.3197 +/- 0.340	1.02	SG	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	4.13+/-5.55	Anderson et al. 1988
0.4286 +/- 1.02		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.74+/-1.31	Swartz et al. 1991
0.4940 +/- 0.421		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (425+/-82 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1.71+/-0.995	Murdoch et al. In press
0.5080 +/- 0.636		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.81+/-1.1	Bay et al. 1994
0.5229 +/- 1.17	0.2	NC	Southern California	COA		Moderate density (259+/-85.9 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979

Table A4-21. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; summarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.6450 +/- 0.699		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.9+/-1.25	Bay et al. 1994
0.6865	2.54	*	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865	3	*	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865	3	*	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.8227 +/- 1.04	4.9	*	Southern California	COA		Altered benthic community (53.8+/-5.63; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.05 +/- 1.09		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
1.05 +/- 1.09		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
1.05 +/- 1.09		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
1.05 +/- 1.09		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.26+/-1.36	Bay et al. 1994
1.05 +/- 2.5	17	*	Southern California	COA		Low species richness (50.5+/-9.2 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.09			*ER-M*							
1.09 +/- 0.565	11	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
1.09 +/- 0.565	11	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	7.35+/-4.51	Anderson et al. 1988
1.15 +/- 1.73	0.3	NC	Southern California	COA		Moderate density (786+/-107 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.31 +/- 1.35		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	2+/-0.95	Bay et al. 1994
1.48 +/- 2.77	101	*	Southern California	COA		Low density (3.13+/-4.49 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
1.94 +/- 1.39	3	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.08+/-1.41	Bay et al. 1994
1.98 +/- 1.09	4	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.22+/-1.26	Bay et al. 1994
2.28 +/- 4.29		NE	Southern California	COA		High density (1068+/-636 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.89 +/- 3.82		NE	Southern California	COA		High density (418+/-275 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
3.38 +/- 5.08		NE	Southern California	COA		High density (1947+/-843 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
4.40 +/- 9.21	10	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.86+/-2.29	Swartz et al. 1991
4.88 +/- 4.09	62	*	Southern California	COA		Low density (4+/-2.45 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
5.85 +/- 12.7		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.57+/-0.08 mg/d)	Neanthes arenaceodentata (polychaete)	JUV	3.1+/-2.7	Murdoch et al. In press
5.85 +/- 12.7		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (2.67+/-4.8% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.1+/-2.7	Murdoch et al. In press
10.9	64	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
16.6 +/- 21.5	34	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (205+/-63.6 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	5.85+/-3.1	Murdoch et al. In press
20.7 +/- 13.1	48	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.16+/-1.83	Swartz et al. 1991

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0020 <	0.0003	NC	Southern California	COA		Low density (534 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020 <	0.0003	NC	Southern California	COA		Low density (26 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0020 <	0.0004	NC	Southern California	COA		Low density (76 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0020 <	0.01	NC	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0020 <	0.02	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0020 <		NE	Southern California	COA		Normal benthic community (98.3; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0020 <		NE	Southern California	COA		High density (349 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Moderate density (833 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Low density (374 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Low density (47 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0040	0.001	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0040	0.002	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0040	0.002	NC	Southern California	COA		Low density (112 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0040	0.1	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0040	0.1	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0040	0.1	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0040	0.1	NC	Southern California	COA		Moderate species richness (72 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.06	NC	Southern California	COA		Low species richness (41 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040	0.3	NC	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040	0.3	NC	Southern California	COA		Moderate density (130 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		Normal benthic community (98.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		High species richness (124 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0040		NE	Southern California	COA		High density (224 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0040		NE	Southern California	COA		High density (610 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.001	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.001	NC	Southern California	COA		Low density (442 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.001	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (36 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (50 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (84 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (110 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (119 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.002	NC	Southern California	COA		Low density (134 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate density (39 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate species richness (67 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate species richness (73 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.1	NC	Southern California	COA		Moderate species richness (89 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0050	0.3	NC	Southern California	COA		Moderate density (99 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0050	0.3	NC	Southern California	COA		Moderate density (142 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0050		NE	Southern California	COA		High density (120 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0050		NE	Southern California	COA		Normal benthic community (83.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		Normal benthic community (94.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		Normal benthic community (95.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0050		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0070	0.002	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0070	0.002	NC	Southern California	COA		Low density (502 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0070	0.002	NC	Southern California	COA		Low density (32 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0070	0.002	NC	Southern California	COA		Low density (51 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0070	0.003	NC	Southern California	COA		Moderate density (173 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0070	0.003	NC	Southern California	COA		Low density (40 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0070	0.1	NC	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0070	0.1	NC	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0070	0.1	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0070	0.5	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0070		NE	Southern California	COA		High density (95 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0070		NE	Southern California	COA		Normal benthic community (83.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0070		NE	Southern California	COA		Normal benthic community (95; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0070		NE	Southern California	COA		High density (207 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0075		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0075		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.96	Anderson et al. 1988
0.0080		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0090	0.003	NC	Southern California	COA		Low density (232 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0090	0.003	NC	Southern California	COA		Low density (464 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0090	0.003	NC	Southern California	COA		Low density (29 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0090	0.003	NC	Southern California	COA		Low density (39 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0090	0.004	NC	Southern California	COA		Moderate density (186 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0090	0.004	NC	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0090	0.1	NC	Southern California	COA		Moderate density (46 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0090	0.1	NC	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0090	0.1	NC	Southern California	COA		Moderate species richness (94 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0090	0.6	NC	Southern California	COA		Moderate density (53 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0090	0.6	NC	Southern California	COA		Moderate density (68 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0090		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.11	Anderson et al. 1988
0.0090		NE	Southern California	COA		High density (101 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0090		NE	Southern California	COA		Normal benthic community (88.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0090		NE	Southern California	COA		Normal benthic community (89.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0100	0.003	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0100	0.003	NC	Southern California	COA		Low density (13 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0100	0.004	NC	Southern California	COA		Low density (18 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0100	0.1	NC	Southern California	COA		Moderate density (22 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0100	0.2	NC	Southern California	COA		Low species richness (32 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0100	0.7	NC	Southern California	COA		Moderate density (33 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0100		NE	Southern California	COA		Normal benthic community (94.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0110	0.003	NC	Southern California	COA		Low density (382 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0110	0.004	NC	Southern California	COA		Low density (144 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0110	0.005	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0110	0.1	NC	Southern California	COA		Altered benthic community (59.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0110	0.1	NC	Southern California	COA		Moderate density (76 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0110	0.2	NC	Southern California	COA		Moderate species richness (90 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0110	0.7	NC	Southern California	COA		Moderate density (42 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0120		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0120		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.91	Anderson et al. 1988
0.0133	0.05	NC	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0140	0.004	NC	Southern California	COA		Moderate density (902 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0140	0.005	NC	Southern California	COA		Low density (136 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0140	0.01	NC	Southern California	COA		Moderate density (407 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0140	0.95	NC	Southern California	COA		Moderate density (24 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0140		NE	Southern California	COA		High density (233 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0140		NE	Southern California	COA		Normal benthic community (74.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0140		NE	Southern California	COA		High species richness (169 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0160	0.005	NC	Southern California	COA		Low density (440 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0160	0.01	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0160	0.01	NC	Southern California	COA		Low density (96 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0160	0.2	NC	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0160	0.3	NC	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0160	1.1	SG	Southern California	COA		Moderate density (93 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0160		NE	Southern California	COA		Normal benthic community (90.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0165	0.1	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0165		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.73	Anderson et al. 1988
0.0168		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.25	Sapudar et al. 1994
0.0176		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.0177		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.4	Sapudar et al. 1994
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.48	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.42	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.53	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.09	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.37	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.42	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.53	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.19	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.93	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.09	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.37	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.78	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.19	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.48	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.28	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.03	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.09	Fairey et al. 1996
0.0180 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.37	Fairey et al. 1996
0.0180 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.0181	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.14	Fairey et al. 1996
0.0183	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.0186		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.38	Fairey et al. 1996
0.0193		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0193	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.0194		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Fairey et al. 1996
0.0200	0.01	NC	Southern California	COA		Low density (247 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0200	0.01	NC	Southern California	COA		Low density (476 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0200	0.01	NC	Southern California	COA		Low density (61 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0200	0.01	NC	Southern California	COA		Low density (66 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0200	0.01	NC	Southern California	COA		Moderate density (213 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0200	0.01	NC	Southern California	COA		Low density (97 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0200	0.3	NC	Southern California	COA		Moderate density (41 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0200	0.3	NC	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0200	0.3	NC	Southern California	COA		Low species richness (60 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0200	1.4	SG	Southern California	COA		Moderate density (37 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0200	1.4	SG	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0200		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.92	Swartz et al. 1991
0.0200		NE	Southern California	COA		High density (130 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0200		NE	Southern California	COA		Normal benthic community (77.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0200		NE	Southern California	COA		Normal benthic community (85.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0203		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.5	Sapudar et al. 1994
0.0218	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0225		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0242		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.34	Sapudar et al. 1994
0.0243		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.31	Sapudar et al. 1994
0.0247		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0254	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.65	Fairey et al. 1996
0.0272	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.27	Fairey et al. 1996
0.0272		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.53	Fairey et al. 1996
0.0272		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.27	Fairey et al. 1996
0.0281	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.9	Fairey et al. 1996
0.0284		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.0300	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.93	Fairey et al. 1996
0.0300	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.13	Fairey et al. 1996
0.0300		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.87	Swartz et al. 1991
0.0310	0.01	NC	Southern California	COA		Moderate density (641 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310	0.01	NC	Southern California	COA		Low density (408 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310	0.01	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0310	0.01	NC	Southern California	COA		Moderate density (208 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0310	0.01	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0310	0.4	NC	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0310	0.5	NC	Southern California	COA		Moderate species richness (69 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310	0.5	NC	Southern California	COA		Moderate species richness (93 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0310	2.1	*	Southern California	COA		Moderate density (107 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0310		NE	Southern California	COA		High density (110 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0310		NE	Southern California	COA		Normal benthic community (69.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0310		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0310		NE	Southern California	COA		High density (204 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0310		NE	Southern California	COA		High density (201 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0320	0.01	NC	Southern California	COA		Low density (467 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0320	0.01	NC	Southern California	COA		Low density (62 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0320	0.01	NC	Southern California	COA		Moderate density (301 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0320	0.4	NC	Southern California	COA		Moderate density (80 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0320	0.5	NC	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0320	2.2	*	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0320		NE	Southern California	COA		Normal benthic community (72.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0327	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.0327	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.27	Fairey et al. 1996
0.0327	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.0329	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0333	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.0343		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0352		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0364		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.07	Fairey et al. 1996
0.0366	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Sapudar et al. 1994
0.0367	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0368		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.49	Sapudar et al. 1994
0.0370	0.01	NC	Southern California	COA		Low density (331 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0370	0.6	NC	Southern California	COA		Low species richness (62 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0370		NE	Southern California	COA		Normal benthic community (79.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0372		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.05	Sapudar et al. 1994
0.0376		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.39	Sapudar et al. 1994
0.0392	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.97	Sapudar et al. 1994
0.0393		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.29	Sapudar et al. 1994
0.0395		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0395		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.56	Anderson et al. 1988
0.0397		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.11	Sapudar et al. 1994
0.0400	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.9	Fairey et al. 1996
0.0400	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.58	Fairey et al. 1996
0.0400	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.9	Fairey et al. 1996
0.0400	0.01	NC	Southern California	COA		Low density (515 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0400	0.01	NC	Southern California	COA		Low density (68 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0400	0.02	NC	Southern California	COA		Moderate density (212 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0400	0.5	NC	Southern California	COA		Moderate density (34 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0400	0.6	NC	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0400		NE	Southern California	COA		Normal benthic community (91.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0400		NE	Southern California	COA		High density (184 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0402	0.2	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
0.0413		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.38	Sapudar et al. 1994
0.0416	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0422	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.02	Fairey et al. 1996
0.0422	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.02	Fairey et al. 1996
0.0422		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.81	Fairey et al. 1996

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0429		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.28	Sapudar et al. 1994
0.0432	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.55	Fairey et al. 1996
0.0437	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.28	Fairey et al. 1996
0.0441		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0443	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.14	Fairey et al. 1996
0.0443	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.0443		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.14	Fairey et al. 1996
0.0451		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.26	Fairey et al. 1996
0.0451		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.16	Fairey et al. 1996
0.0451		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.16	Fairey et al. 1996
0.0455	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.07	Fairey et al. 1996
0.0460	0.01	NC	Southern California	COA		Low density (271 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0460	0.02	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0460	0.02	NC	Southern California	COA		Low density (44 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0460	0.6	NC	Southern California	COA		Moderate density (51 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0460	0.7	NC	Southern California	COA		Low species richness (56 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0460	3.1	*	Southern California	COA		Moderate density (154 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0460		NE	Southern California	COA		Normal benthic community (98.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0462		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.88	Fairey et al. 1996
0.0474	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0475		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.54	Swartz et al. 1991
0.0482	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Sapudar et al. 1994
0.0495	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.46	Sapudar et al. 1994
0.0496	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.42	Sapudar et al. 1994 *
0.0501		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.72	Sapudar et al. 1994
0.0504		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.61	Sapudar et al. 1994
0.0504		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0514	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.39	Sapudar et al. 1994
0.0515		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.76	Sapudar et al. 1994
0.0518		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0533		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Sapudar et al. 1994
0.0538	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.03	Fairey et al. 1996
0.0548		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.8	Sapudar et al. 1994
0.0573		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.53	Sapudar et al. 1994
0.0588	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.09	Fairey et al. 1996
0.0592	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.24	Fairey et al. 1996
0.0600	0.02	NC	Southern California	COA		Low density (520 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0600	0.02	NC	Southern California	COA		Low density (91 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0600	0.03	NC	Southern California	COA		Moderate density (254 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0600	0.96	NC	Southern California	COA		Moderate species richness (91 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0600	4.1	*	Southern California	COA		Low density (12 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0600		NE	Southern California	COA		High density (142 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0600		NE	Southern California	COA		Normal benthic community (60.9; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0610	0.02	NC	Southern California	COA		Low density (359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0610	0.02	NC	Southern California	COA		Low density (23 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0610	0.03	NC	Southern California	COA		Low density (131 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0610	0.8	NC	Southern California	COA		Moderate density (52 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0610	0.97	NC	Southern California	COA		Low species richness (61 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0610	4.1	*	Southern California	COA		Moderate density (118 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0610		NE	Southern California	COA		Normal benthic community (93.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0614		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0625		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Swartz et al. 1991
0.0630	0.02	NC	Southern California	COA		Low density (472 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0630	0.02	NC	Southern California	COA		Low density (85 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0630	0.03	NC	Southern California	COA		Moderate density (202 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0630	1.005	SG	Southern California	COA		Moderate species richness (74 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0630	4.3	*	Southern California	COA		Low density (11 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0630		NE	Southern California	COA		High density (156 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0630		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0638		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.6	Sapudar et al. 1994
0.0647		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0654		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.0670		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.54 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.0670		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (443 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.0670		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (12% mortality)	Neanthes arenaceodentata (polychaete)	JUV	0.9	Murdoch et al. In press
0.0672	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.88	Fairey et al. 1996
0.0672		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.35	Fairey et al. 1996
0.0672		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
0.0682	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0684	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.73	Fairey et al. 1996
0.0686	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.99	Sapudar et al. 1994
0.0690	0.02	NC	Southern California	COA		Low density (370 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0690	0.02	NC	Southern California	COA		Low density (99 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.0690	0.03	NC	Southern California	COA		Moderate density (188 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.0690	0.4	NC	Southern California	COA		Altered benthic community (58.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.0690	0.9	NC	Southern California	COA		Moderate density (31 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.0690	1.1	SG	Southern California	COA		Low species richness (58 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.0690	4.7	*	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.0702		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.0706	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.6	Fairey et al. 1996
0.0706	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.0706	0.4	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.6	Fairey et al. 1996
0.0707	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.69	Sapudar et al. 1994
0.0752	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Sapudar et al. 1994
0.0754		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.55	Sapudar et al. 1994
0.0758	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.86	Sapudar et al. 1994
0.0761		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.56	Sapudar et al. 1994
0.0775	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey et al. 1996
0.0784		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.0793		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0814		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.49	Sapudar et al. 1994
0.0825		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.0838		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0850		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.08	Swartz et al. 1991
0.0851	0.97	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.36	Sapudar et al. 1994
0.0867	0.99	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0880		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.24	Fairey et al. 1996
0.0880		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.67	Fairey et al. 1996
0.0880		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.67	Fairey et al. 1996
0.0886	1.01	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Sapudar et al. 1994
0.0891		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0910	1.04	SG	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0919		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.5	Sapudar et al. 1994
0.0936		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.0950		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.0953		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.11	Fairey et al. 1996
0.0961		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.0965	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.89	Fairey et al. 1996
0.0989	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.1000		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.37	Swartz et al. 1991
0.1021	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.16	Sapudar et al. 1994
0.1031	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.18	Sapudar et al. 1994
0.1034	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.53	Fairey et al. 1996
0.1034		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.9	Sapudar et al. 1994
0.1043	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.94	Fairey et al. 1996
0.1054	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.1	Fairey et al. 1996
0.1054	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.77	Fairey et al. 1996
0.1054	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Fairey et al. 1996
0.1064	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.13	Fairey et al. 1996
0.1075	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.61	Sapudar et al. 1994
0.1084		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1084	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.06	Sapudar et al. 1994
0.1088		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.59	Fairey et al. 1996
0.1090	0.03	NC	Southern California	COA		Low density (281 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1090	0.05	NC	Southern California	COA		Low density (56 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1090	0.1	NC	Southern California	COA		Low density (193 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1090	0.6	NC	Southern California	COA		Altered benthic community (46.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1090	1.4	SG	Southern California	COA		Moderate density (23 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1090	1.7	SG	Southern California	COA		Low species richness (53 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1090	7.4	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1099		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.31	Sapudar et al. 1994
0.1106		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.9	Sapudar et al. 1994
0.1110		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.1110		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.1110		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1110		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.1110		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.8	Bay et al. 1994
0.1110		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.8	Bay et al. 1994
0.1118	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.1119	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.56	Sapudar et al. 1994
0.1125		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.1132		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.34	Sapudar et al. 1994
0.1136	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.96	Sapudar et al. 1994
0.1152		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.1	Sapudar et al. 1994
0.1167	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.77	Fairey et al. 1996
0.1167	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.95	Fairey et al. 1996
0.1167	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.77	Fairey et al. 1996
0.1178	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey et al. 1996
0.1190	0.04	NC	Southern California	COA		Low density (405 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1190	0.04	NC	Southern California	COA		Low density (78 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1190	0.1	NC	Southern California	COA		Moderate density (193 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1190	1.9	SG	Southern California	COA		Low species richness (55 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1190	8.1	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1190		NE	Southern California	COA		High density (129 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1190		NE	Southern California	COA		Normal benthic community (65; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1205	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.77	Sapudar et al. 1994
0.1208		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Sapudar et al. 1994
0.1237	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Sapudar et al. 1994
0.1246		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.13	Sapudar et al. 1994
0.1250	0.04	NC	Southern California	COA		Low density (198 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1250	0.04	NC	Southern California	COA		Low density (17 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1250	0.1	NC	Southern California	COA		Low density (33 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1250	1.6	SG	Southern California	COA		Moderate density (63 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1250	1.99	SG	Southern California	COA		Low species richness (43 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1250	8.5	*	Southern California	COA		Moderate density (78 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1250		NE	Southern California	COA		Normal benthic community (97.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1262	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.89	Fairey et al. 1996
0.1280		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.1280		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.1280		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.1280		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.1280		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	0.6	Bay et al. 1994
0.1287	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.22	Fairey et al. 1996
0.1287	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.94	Fairey et al. 1996
0.1287	0.6	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.22	Fairey et al. 1996
0.1332	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.35	Fairey et al. 1996
0.1360	0.2	NC	Southern California	COA	35-d	Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.1360		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.1360		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.1360		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1360		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.1	Bay et al. 1994
0.1360		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.1	Bay et al. 1994
0.1371	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.86	Fairey et al. 1996
0.1382	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.81	Sapudar et al. 1994
0.1382	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.04	Fairey et al. 1996
0.1382		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.78	Fairey et al. 1996
0.1382		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.78	Fairey et al. 1996
0.1399	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.75	Fairey et al. 1996
0.1400	0.04	NC	Southern California	COA		Low density (230 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1400	0.05	NC	Southern California	COA		Low density (65 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1400	0.1	NC	Southern California	COA		Low density (71 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1400	1.8	SG	Southern California	COA		Moderate density (20 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1400	2.2	*	Southern California	COA		Low species richness (47 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1400	9.5	*	Southern California	COA		Moderate density (57 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1400		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.40	Swartz et al. 1991
0.1400		NE	Southern California	COA		Normal benthic community (82.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1404	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.1412	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.1412	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.17	Fairey et al. 1996
0.1412	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.1427		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.07	Fairey et al. 1996
0.1447	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.1447		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Sapudar et al. 1994
0.1493		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.4	Sapudar et al. 1994
0.1500		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.45	Swartz et al. 1991
0.1535		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.18	Fairey et al. 1996
0.1556	1.8	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.1620		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1620		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1620		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1620		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1620		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.1620		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.1700	0.1	NC	Southern California	COA		Moderate density (737 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1700	0.1	NC	Southern California	COA		Low density (15 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1700	2.1	*	Southern California	COA		Moderate density (54 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1700	12	*	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1700		NE	Southern California	COA		Normal benthic community (83.5; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.1700		NE	Southern California	COA		High species richness (106 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1700		NE	Southern California	COA		High density (614 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1746	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.7	Fairey et al. 1996
0.1790	0.1	NC	Southern California	COA		Low density (478 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1790	0.1	NC	Southern California	COA		Low density (160 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.1790	0.1	NC	Southern California	COA		Moderate density (217 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.1790	1.1	SG	Southern California	COA		Altered benthic community (58.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1790	2.3	*	Southern California	COA		Moderate density (75 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.1790	2.9	*	Southern California	COA		Moderate species richness (77 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.1790	12	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.1849	2.1	*	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Sapudar et al. 1994
0.1861		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.7	Fairey 1997
0.1886	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.48	Fairey et al. 1996
0.1921	2.2	*	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.1922	2.2	*	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.1950		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.70	Swartz et al. 1991
0.2032	2.3	*	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	2	Sapudar et al. 1994
0.2038		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.95	Fairey 1997
0.2108	0.8	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.06	Fairey 1997
0.2205	0.8	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.3	Fairey 1997
0.2247	0.8	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.67	Fairey 1997
0.2250		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.71	Swartz et al. 1991
0.2397	0.98	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.19	Fairey et al. 1996
0.2400		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.64	Swartz et al. 1991
0.2504	2.9	*	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.28	Sapudar et al. 1994
0.2533	0.95	NC	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.86	Fairey 1997
0.2533		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Fairey 1997
0.2540	1.5	SG	Southern California	COA		Altered benthic community (45; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2540	4.1	*	Southern California	COA		Moderate species richness (83 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2540	17	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2540		NE	Southern California	COA		High density (195 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2540		NE	Southern California	COA		High density (1231 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2540		NE	Southern California	COA		High density (385 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2540		NE	Southern California	COA		High density (616 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2560	0.1	NC	Southern California	COA		Low density (259 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2560	0.1	NC	Southern California	COA		Low density (152 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2560	0.1	NC	Southern California	COA		Low density (74 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2560	1.0	NG	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.2560	1.5	SG	Southern California	COA		Altered benthic community (48.2; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2560	3.2	*	Southern California	COA		Moderate density (19 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2560	4.1	*	Southern California	COA		Low species richness (48 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2560	17	*	Southern California	COA		Low density (3 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2560		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1.12	Anderson et al. 1988
0.2560		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2560		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2560		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2560		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2560		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.12	Anderson et al. 1988
0.2600		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.92	Fairey 1997
0.2612		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.89	Sapudar et al. 1994
0.2745	1.0	SG	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.46	Fairey 1997
0.2843		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.85	Fairey 1997

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2880	4.6	*	Southern California	COA		Moderate species richness (88 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2880	20	*	Southern California	COA		Low density (4 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.2880		NE	Southern California	COA		High density (178 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.2880		NE	Southern California	COA		Normal benthic community (63.1; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.2880		NE	Southern California	COA		High density (1359 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.2880		NE	Southern California	COA		High density (305 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.2880		NE	Southern California	COA		High density (806 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.2915	1.1	SG	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.51	Fairey 1997
0.2948		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.98	Fairey 1997
0.3008	0.999	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.26	Fairey et al. 1996
0.3008		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.03	Fairey et al. 1996
0.3008		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.26	Fairey et al. 1996
0.3116	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.3130	1.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.92	Fairey et al. 1996
0.3169		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Sapudar et al. 1994
0.3191		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.84	Fairey 1997
0.3204		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.79	Fairey 1997
0.3219	1.2	SG	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Fairey 1997
0.3276	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.16	Fairey et al. 1996
0.3305		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.3305		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.28	Anderson et al. 1988
0.3433	3.9	*	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.48	Sapudar et al. 1994
0.3495	1.2	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.77	Fairey et al. 1996
0.3495	1.7	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.77	Fairey et al. 1996
0.3495		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.72	Fairey et al. 1996
0.3520		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.5	Sapudar et al. 1994
0.3600		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.48 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
0.3600		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (533 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
0.3600		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1.4	Murdoch et al. In press
0.3600		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.82	Swartz et al. 1991
0.3620	4.1	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.3	Sapudar et al. 1994
0.3735	1.4	SG	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.42	Fairey 1997
0.3783	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.83	Fairey et al. 1996
0.3800		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.84	Swartz et al. 1991
0.3867	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.29	Fairey et al. 1996
0.3867	1.9	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.88	Fairey et al. 1996
0.3875	4.4	*	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.1	Sapudar et al. 1994
0.3929	4.5	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.85	Sapudar et al. 1994
0.3973	1.6	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.4	Fairey et al. 1996
0.3973	1.98	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	0.94	Fairey et al. 1996

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.3973		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	0.94	Fairey et al. 1996
0.4056	4.6	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.86	Sapudar et al. 1994
0.4090		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.2	Bay et al. 1994
0.4090		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.2	Bay et al. 1994
0.4319	1.63	SG	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Fairey 1997
0.4620	0.9	NC	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.4	Bay et al. 1994
0.4620		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.4620		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.4620		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.4620		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.4620		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1.4	Bay et al. 1994
0.4673	5.3	*	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	3	Sapudar et al. 1994
0.4738	5.4	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.6	Sapudar et al. 1994
0.4800		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.55 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
0.4800		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (369 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
0.4800		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1.37	Murdoch et al. In press
0.4842	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.14	Fairey et al. 1996
0.5045	5.8	*	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.27	Sapudar et al. 1994
0.5130	0.2	NC	Southern California	COA		Low density (518 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.5130	0.2	NC	Southern California	COA		Low density (117 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
0.5130	0.2	NC	Southern California	COA		Moderate density (335 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
0.5130	3.0	*	Southern California	COA		Altered benthic community (57.7; infaunal index)	Benthic invertebrates			Word and Mearns 1979
0.5130	6.5	*	Southern California	COA		Moderate density (29 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
0.5130	8.2	*	Southern California	COA		Low species richness (57 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
0.5130	35	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
0.5131	5.9	*	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.28	Sapudar et al. 1994
0.5150	1.2	SG	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.32	Swartz et al. 1991
0.5334	2.2	*	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.98	Fairey et al. 1996
0.5345	6.1	*	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.3	Sapudar et al. 1994
0.5450	1.3	SG	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.17	Swartz et al. 1991
0.5786		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.54	Sapudar et al. 1994
0.5804	1.9	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.93	Fairey et al. 1996
0.5804	2.9	*	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.93	Fairey et al. 1996
0.5804		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.67	Fairey et al. 1996
0.6372	2.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.04	Fairey et al. 1996
0.6865	2.0	*	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.6865	2.6	*	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865	2.6	*	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865	2.6	*	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865	7.2	*	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.6865	7.2	*	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	10.5	Anderson et al. 1988
0.6865		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	10.5	Anderson et al. 1988
0.7158	2.9	*	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.6	Fairey et al. 1996
0.7158	3.6	*	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.75	Fairey et al. 1996
0.7158		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1.75	Fairey et al. 1996
0.8347			*ER-M*							
0.8347	2.8	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.75	Fairey et al. 1996
0.8750	2.0	*	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.43	Swartz et al. 1991
0.9008		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.9	Fairey et al. 1996
0.9008		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.53	Fairey et al. 1996
0.9008		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.53	Fairey et al. 1996
0.9065	3.7	*	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.15	Fairey et al. 1996
0.9292		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
0.9292		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.81	Fairey et al. 1996
0.9780	3.2	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.04	Fairey et al. 1996
0.9780	4.0	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	0.37	Fairey et al. 1996
0.9780	4.9	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.04	Fairey et al. 1996
0.9930		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.4	Bay et al. 1994
0.9930		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.4	Bay et al. 1994
0.9954	3.3	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	2.58	Fairey et al. 1996
0.9954	4.1	*	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
0.9954	5.0	*	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.58	Fairey et al. 1996
1.02	5.1	*	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.28	Fairey et al. 1996
1.02		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.02	Fairey et al. 1996
1.02		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	3.28	Fairey et al. 1996
1.07		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.59 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
1.07		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (354 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
1.07		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.16	Murdoch et al. In press
1.34	3.0	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (160 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
1.34		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.72 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
1.34		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	3.66	Murdoch et al. In press
1.38	5.7	*	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1.22	Fairey et al. 1996
1.38		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1.35	Fairey et al. 1996
1.49	16	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.49	16	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.49		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
1.49		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	4.16	Anderson et al. 1988
1.49		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.49		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988
1.49		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.16	Anderson et al. 1988

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
1.55	3.6	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.24	Swartz et al. 1991
1.71	7.0	*	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.33	Fairey et al. 1996
1.74	2.7	*	Southern California	COA	35-d	Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
1.74	3.4	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.8	Bay et al. 1994
1.74		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	2.8	Bay et al. 1994
1.74		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.8	Bay et al. 1994
1.79	3.5	*	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.5	Bay et al. 1994
1.79		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
1.79		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
1.79		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
1.79		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
1.79		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.5	Bay et al. 1994
1.93	0.6	NC	Southern California	COA		Low density (116 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.93	0.7	NC	Southern California	COA		Low density (38 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
1.93	0.8	NC	Southern California	COA		Low density (64 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
1.93	11	*	Southern California	COA		Altered benthic community (51; infaunal index)	Benthic invertebrates			Word and Mearns 1979
1.93	24	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
1.93	31	*	Southern California	COA		Low species richness (37 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
1.93	131	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
2.08	12	*	Southern California	COA		Altered benthic community (54.8; infaunal index)	Benthic invertebrates			Word and Mearns 1979
2.08	26	*	Southern California	COA		Moderate density (48 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
2.08	33	*	Southern California	COA		Moderate species richness (79 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.08	141	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
2.08		NE	Southern California	COA		High density (3059 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.08		NE	Southern California	COA		High density (1004 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
2.08		NE	Southern California	COA		High density (1964 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.48	3.8	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
2.48	4.9	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	4.3	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
2.48		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	4.3	Bay et al. 1994
2.83	0.8	NC	Southern California	COA		Moderate density (710 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.83	1.2	SG	Southern California	COA		Moderate density (424 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
2.83	17	*	Southern California	COA		Altered benthic community (58.6; infaunal index)	Benthic invertebrates			Word and Mearns 1979
2.83	36	*	Southern California	COA		Low density (2 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979

Table A4-22. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
2.83	45	*	Southern California	COA		Low species richness (45 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
2.83	193	*	Soulhern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
2.83		NE	Southern California	COA		High density (235 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
2.94	6.9	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	4.25	Swartz et al. 1991
3.00	7.0	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.38	Swartz et al. 1991
3.42	5.3	*	Southern California	COA	35-d	Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.42	6.7	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	3.1	Bay et al. 1994
3.42		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.42		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.42		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.42		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	3.1	Bay et al. 1994
3.42		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	3.1	Bay et al. 1994
3.86	1.1	SG	Southern California	COA		Moderate density (895 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
3.86	1.7	SG	Southern California	COA		Moderate density (371 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
3.86	49	*	Southern California	COA		Low density (7 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
3.86	62	*	Southern California	COA		Low species richness (46 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
3.86	262	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
3.86		NE	Southern California	COA		Normal benthic community (64.4; infaunal index)	Benthic invertebrates			Word and Mearns 1979
3.86		NE	Southern California	COA		High density (486 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
3.97		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.14	Swartz et al. 1991
5.21	12	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	5.18	Swartz et al. 1991
8.47	20	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.49	Swartz et al. 1991
10.8	25	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	6.43	Swartz et al. 1991
10.9	64	*	Southern California	COA		Degraded benthic community (21; infaunal index)	Benthic invertebrates			Word and Mearns 1979
10.9	138	*	Southern California	COA		Low density (5 N/0.1 sq.m.)	Arthropods			Word and Mearns 1979
10.9	174	*	Southern California	COA		Low species richness (36 S/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
10.9	741	*	Southern California	COA		Low density (0 N/0.1 sq.m.)	Echinoderms			Word and Mearns 1979
10.9		NE	Southern California	COA		High density (2140 N/0.1 sq.m.)	Benthic invertebrates			Word and Mearns 1979
10.9		NE	Southern California	COA		High density (310 N/0.1 sq.m.)	Mollusca			Word and Mearns 1979
10.9		NE	Southern California	COA		High density (1795 N/0.1 sq.m.)	Polychaetes			Word and Mearns 1979
12.3	29	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	7.85	Swartz et al. 1991
12.5	29	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.12	Swartz et al. 1991
13.4	31	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.29	Swartz et al. 1991
20.2	47	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.6	Swartz et al. 1991
23.6	55	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.2	Swartz et al. 1991
26.4	62	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	8.45	Swartz et al. 1991
31.8	64	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (250 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press
31.8		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.53 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press
31.8		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (4% mortality)	Neanthes arenaceodentata (polychaete)	JUV	8.04	Murdoch et al. In press
34.0	79	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	10.7	Swartz et al. 1991
45.2	105	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	9.34	Swartz et al. 1991

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.

Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Where the concentration of the contaminant was less than detection limit (indicated by '<') in a toxic sample, 1/2 of the detection limit was used to compare to the mean concentration in the non-toxic samples.

Table A4-23. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; summarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0611 +/- 0.079		NE	Southern California	COA	35-d	Not toxic (0.01+/-0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0611 +/- 0.079		NE	Southern California	COA	35-d	Not toxic (0.02+/-0.005 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0651	0.7	NC	Southern California	COA	35-d	Toxic (0.001 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0651	0.7	NC	Southern California	COA	35-d	Toxic (69.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0651	0.7	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0743 +/- 0.046		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13.6+/-5.22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0890 +/- 0.135		NE	Southern California	COA	10-d	Not toxic (23.6+/-11.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0892 +/- 0.059	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (38.7+/-11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0945 +/- 0.120		NE	Southern California	COA	10-d	Not toxic (96+/-4.12% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0967 +/- 0.110	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3+/-21.9% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0980 +/- 0.128		NE	Southern California	COA	35-d	Not toxic (0.004+/-0.0005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0980 +/- 0.128		NE	Southern California	COA	35-d	Not toxic (23.7+/-8.35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0980 +/- 0.128		NE	Southern California	COA	35-d	Not toxic (0.27+/-0.78% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.1044 +/- 0.080	0.7	NC	Santa Monica Bay	COA	10-d	Significantly toxic (54.5+/-14.6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1050 +/- 0.109	1.1	SG	Southern California	COA	10-d	Toxic (51.6+/-14.8% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.1357 +/- 0.257		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (89.3+/-9.87% fertization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1396 +/- 0.157		NE	Southern California	COA	10-d	Least toxic (8.66+/-3.34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1495 +/- 0.160	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (24.5+/-26.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1537 +/- 0.153		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (87.6+/-8.2% normal development	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1576 +/- 0.092		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9.5+/-4.54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.2110 +/- 0.207	3.5	*	Southern California	COA	35-d	Toxic (0.003+/-0.0028 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2110 +/- 0.207	3.5	*	Southern California	COA	35-d	Toxic (0.004+/-0.006 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2178 +/- 0.350		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12.8+/-5.94% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2210 +/- 0.179		NE	Southern California	COA	1.3-h	Not toxic (80+/-10.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.2283 +/- 0.459	1.0	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (54.7+/-19.4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2551 +/- 0.127		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (425+/-82 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.2810 +/- 0.193		NE	Southern California	COA	35-d	Not toxic (0.005+/-0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3690 +/- 0.291		NE	Southern California	COA	35-d	Not toxic (0.025+/-0.004 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3690 +/- 0.291		NE	Southern California	COA	35-d	Not toxic (0.0008+/-0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3690 +/- 0.291		NE	Southern California	COA	35-d	Not toxic (23.8+/-4.91% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3690 +/- 0.291		NE	Southern California	COA	35-d	Not toxic (1.23+/-1.92% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-23. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; summarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.4970 +/- 0.404		NE	Southern California	COA	10-d	Not toxic (10.4+/-6.07% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.5650 +/- 0.407	2.01	*	Southern California	COA	35-d	Toxic (0.002+/-0.0002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6060			*ER-M*							
0.6060 +/- 0.303	2.7	*	Southern California	COA	1.3-h	Toxic (9.4+/-16.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.6931 +/- 0.575	5.0	*	Southern California	COA	10-d	Moderately toxic (35.9+/-12.1% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.8903 +/- 1.51		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.57+/-0.08 mg/d)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.8903 +/- 1.51		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (2.67+/-4.8% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.16 +/- 2.54	8.5	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (205+/-63.6 ej/prod.)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
2.37 +/- 1.20	17	*	Southern California	COA	10-d	Most toxic (78.6+/-8.65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991

See Appendix 2 for glossary of acronyms.
The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study.
Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0046 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0063 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0065 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0074 <	0.05	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0074 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (17.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0076 <	0.05	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (58.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0076 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0076 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (63.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0078		NE	Southern California	COA	10-d	Not toxic (15% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	10-d	Not toxic (97% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	35-d	Not toxic (24.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	35-d	Not toxic (2.2% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	35-d	Not toxic (0.011 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0078		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0080		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (21.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	35-d	Not toxic (-0.00009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.0080		NE	Southern California	COA	1.3-h	Not toxic (76% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0081		NE	Southern California	COA	10-d	Not toxic (16.5% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	10-d	Not toxic (96% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	35-d	Not toxic (35% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0081		NE	Southern California	COA	35-d	Not toxic (0.018 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0094 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0096	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0101 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0107	0.0	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (67% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0107		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0118 <	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0118 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0118 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (4.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0120	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0120		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (80% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0122 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0122 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (85.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0129		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0132		NE	Southern California	COA	10-d	Not toxic (42.2% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0132		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0132		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0132		NE	Southern California	COA	35-d	Not toxic (32.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0132		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0132		NE	Southern California	COA	35-d	Not toxic (0.008 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0132		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0133	0.1	NC	Santa Monica Bay	COA	10-d	Significantly toxic (84% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0133	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (43% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0135		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (83.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0143	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0144	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0150		NE	San Pedro Bay	COA	10-d	Not significantly toxic (23% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0150		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0151 <	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (23.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0151 <		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (96% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0155	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0155	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0159	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0160	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0160	0.1	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (35.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0161		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (75% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0165 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (2.8% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0165 <		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0165 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (93.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0175 <	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (15.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0175 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0194	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0210	0.1	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0210	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (2.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0217	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0218		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0226	0.3	NC	Southern California	COA	10-d	Toxic (36.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	10-d	Not toxic (89% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	35-d	Not toxic (19.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0226		NE	Southern California	COA	35-d	Not toxic (0.021 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0229		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0246		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (91% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0251		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0265	0.2	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (59.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0267	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0281	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0303	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0308		NE	Southern California	COA	10-d	Least toxic (11.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0312	0.2	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (17% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0320	0.1	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0325	0.4	NC	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0339	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (61% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0340		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0345		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0350	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0351		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0353		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0354		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0357		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0357	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (71% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0358		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0359		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0367	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0370	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0374		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0381		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0386	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0388	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (50.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0388		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0389		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0389		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (97.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0393		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0396	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0399	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0404	0.5	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0406		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0412	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0413	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0413	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0416	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0417	0.3	NC	Santa Monica Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0425	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (56% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0431		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0435	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0437	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (54% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0439		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0441	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0441	0.3	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0443		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0447	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0450		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0451	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (26% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0458		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0465		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (98% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0473	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0473	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0476		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0478	0.6	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0484		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0488		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0498		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0498		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (98.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0511	0.2	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (62% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0517		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0518		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0522	0.3	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (36% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0526	0.7	NC	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0547		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0549		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0551		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0558		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0559	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0560		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0574	0.4	NC	Santa Monica Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0576		NE	San Pedro Bay	COA	10-d	Not significantly toxic (16% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0580	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0585		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0595	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (77% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0613		NE	San Pedro Bay	COA	10-d	Not significantly toxic (18% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0626	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (44% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0629		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0629	0.8	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0643 <		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0651	0.7	NC	Southern California	COA	10-d	Toxic (66.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0651	1.1	SG	Southern California	COA	35-d	Toxic (0.001 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0651	1.1	SG	Southern California	COA	35-d	Toxic (0.001 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0651		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0654	0.7	NC	Southern California	COA	35-d	Toxic (0.0014 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0654	0.7	NC	Southern California	COA	35-d	Toxic (30.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0654	0.7	NC	Southern California	COA	35-d	Toxic (51.1% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0659	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0659	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (9.5% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0663	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0666		NE	San Pedro Bay	COA	10-d	Not significantly toxic (6% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0668	0.4	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0668		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0672		NE	San Pedro Bay	COA	10-d	Not significantly toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0675		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0675		NE	Southern California	COA	35-d	Not toxic (27.7% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0675		NE	Southern California	COA	35-d	Not toxic (0.029 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0675		NE	Southern California	COA	35-d	Not toxic (0.006 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0675		NE	Southern California	COA	35-d	Not toxic (0.0019 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.0675		NE	Southern California	COA	1.3-h	Not toxic (66% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.0676	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0677	0.3	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (98% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0678		NE	San Pedro Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0682	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0684		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (81% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0685		NE	San Pedro Bay	COA	10-d	Not significantly toxic (24% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0689	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (36% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0692	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0692	0.5	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0693	0.9	NC	San Pedro Bay	COA	10-d	Significantly toxic (34% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0696		NE	San Pedro Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0696		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0705		NE	Southern California	COA	10-d	Not toxic (23.3% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	10-d	Not toxic (98% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	35-d	Not toxic (12% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	35-d	Not toxic (0.012 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0705		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0711	0.96	NC	San Pedro Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0712		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0727		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0730		NE	Southern California	COA	10-d	Least toxic (7.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0737	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0744		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.54 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.0744		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (443 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.0744		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (12% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.0751		NE	San Pedro Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0751		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0771		NE	Southern California	COA	10-d	Not toxic (11.7% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	10-d	Not toxic (100% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	35-d	Not toxic (14.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	35-d	Not toxic (0.010 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.0772		NE	Southern California	COA	35-d	Not toxic (0.030 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.0776		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0776		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (91.9% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0783		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0785	0.5	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (67% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0789	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0790	1.1	SG	San Pedro Bay	COA	10-d	Significantly toxic (37% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0791		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.0800	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0826		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0838		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0859		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (92% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0864		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0881		NE	San Pedro Bay	COA	10-d	Not significantly toxic (8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0882	1.2	SG	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0891		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0905		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (7% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.0936	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0941	0.4	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0958	0.6	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.0958	0.7	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (60.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.0961	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0964		NE	San Pedro Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0973	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0986	1.3	SG	San Pedro Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.0987	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.0990		NE	San Pedro Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1000		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1016	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (26% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1025	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1027	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (6% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1030	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1031	1.4	SG	San Pedro Bay	COA	10-d	Significantly toxic (29% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1038		NE	Southern California	COA	10-d	Least toxic (1.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1045		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1055	0.7	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (52.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1055	0.8	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (40.4% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1063		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1071	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (52% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1081		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1084		NE	San Pedro Bay	COA	10-d	Not significantly toxic (15% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1087		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1110	0.5	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (56% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1116	0.7	NC	Santa Monica Bay	COA	10-d	Significantly toxic (65% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1124		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (11% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1147		NE	Southern California	COA	10-d	Least toxic (8.75% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1148		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1149		NE	San Pedro Bay	COA	10-d	Not significantly toxic (22% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1161	0.7	NC	Santa Monica Bay	COA	10-d	Significantly toxic (68% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1181	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (35% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1199	1.6	SG	San Pedro Bay	COA	10-d	Significantly toxic (38% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1207	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (80% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1229	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (55% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1236		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1236		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1236		NE	Southern California	COA	35-d	Not toxic (0.019 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1236		NE	Southern California	COA	35-d	Not toxic (0.0002 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1236		NE	Southern California	COA	1.3-h	Not toxic (90% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1236	0.4	NC	Southern California	COA		Toxic (0.0017 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1243	1.7	SG	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1262	0.8	NC	Middle San Diego Bay	COA	48-h	Significantly toxic (13.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1262	0.9	NC	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1274	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1274		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1301		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (75.2% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1313		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (80.7% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1313		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (88.1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1320		NE	Southern California	COA	10-d	Least toxic (6.25% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1321	1.8	SG	San Pedro Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1354		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1362	0.9	NC	Santa Monica Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1371		NE	San Pedro Bay	COA	10-d	Not significantly toxic (20% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1377	0.6	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (32% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1378	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1388		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1388		NE	Southern California	COA	35-d	Not toxic (13.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1388		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1388		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1388		NE	Southern California	COA	35-d	Not toxic (0.0017 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.1388		NE	Southern California	COA	1.3-h	Not toxic (92% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.1408		NE	San Pedro Bay	COA	10-d	Not significantly toxic (17% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1418	1.9	SG	San Pedro Bay	COA	10-d	Significantly toxic (41% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1440	0.9	NC	Santa Monica Bay	COA	10-d	Significantly toxic (51% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1468		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1477	1.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1477	1.1	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (77.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.1489		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (13% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1501	2.0	*	San Pedro Bay	COA	10-d	Significantly toxic (28% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1532		NE	San Pedro Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.1542	1.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1543	0.98	NC	Santa Monica Bay	COA	10-d	Significantly toxic (58% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.1558	2.1	*	San Pedro Bay	COA	10-d	Significantly toxic (46% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1574	2.1	*	San Pedro Bay	COA	10-d	Significantly toxic (27% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1630	1.1	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (25% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.1689	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (50% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1706	2.3	*	San Pedro Bay	COA	10-d	Significantly toxic (42% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1790	0.8	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (78% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1948	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (30% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.1956	2.6	*	San Pedro Bay	COA	10-d	Significantly toxic (31% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.1978		NE	Southern California	COA	10-d	Least toxic (10% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.1981		NE	San Pedro Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.2014	0.9	NC	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2032		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2057	1.5	SG	Middle San Diego Bay	COA	20-m	Significantly toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.2067	1.3	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.2071		NE	Southern California	COA	10-d	Least toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2096	1.4	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.2133		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2133		NE	Southern California	COA	35-d	Not toxic (18.6% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2133		NE	Southern California	COA	35-d	Not toxic (0.033 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2133		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2133		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.2286	2.6	*	Southern California	COA	10-d	Toxic (51.9% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	10-d	Not toxic (91% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	35-d	Not toxic (29.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	35-d	Not toxic (0.007 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2286		NE	Southern California	COA	35-d	Not toxic (0.013 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.2388	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (43% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.2388		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (90.8% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.2467	1.6	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (42% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.2517	1.8	SG	Southern California	COA	10-d	Moderately toxic (36.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2551	1.8	SG	Southern California	COA	10-d	Moderately toxic (31.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.2571		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.48 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.2571		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (533 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.2571		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.2659		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (4% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.2824	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (33% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2838	1.3	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (63% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2921		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (12% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.2926	1.9	SG	Santa Monica Bay	COA	10-d	Significantly toxic (47% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.2935		NE	San Pedro Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.2951	2.1	*	Southern California	COA	10-d	Moderately toxic (22.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.3035	2.0	SG	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.3111	2.3	*	Middle San Diego Bay	COA	20-m	Significantly toxic (54.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.3111		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (97.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.3300	1.5	SG	Southern California	COA	1.3-h	Toxic (38% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.3300		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3300		NE	Southern California	COA	35-d	Not toxic (20.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3300		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3300		NE	Southern California	COA	35-d	Not toxic (0.003 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3300		NE	Southern California	COA	35-d	Not toxic (0.0009 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3307		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (93.1% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.3345		NE	Santa Monica Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey 1997
0.3386		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.59 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3386		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (354 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3386		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3408		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	35-d	Not toxic (20.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	35-d	Not toxic (0.0010 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	10-d	Not toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.3408		NE	Southern California	COA	1.3-h	Not toxic (89% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.3504		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.55 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3504		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (369 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3504		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3523	4.7	*	San Pedro Bay	COA	10-d	Significantly toxic (72% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Sapudar et al. 1994
0.3560		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (95.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.3560		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (99.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.3572	5.8	*	Southern California	COA	35-d	Toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3572	5.8	*	Southern California	COA	35-d	Toxic (0.008 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3572		NE	Southern California	COA	10-d	Not toxic (32.6% mortality)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.3572		NE	Southern California	COA	10-d	Not toxic (93% reburial)	Grandidierella japonica (amphipod)	ADT	1	Anderson et al. 1988
0.3572		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d gonad growth rate)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3572		NE	Southern California	COA	35-d	Not toxic (22.3% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3572		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Anderson et al. 1988
0.3661	3.0	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (160 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3661		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.72 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3661		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
0.3858	2.5	*	Middle San Diego Bay	COA	48-h	Significantly toxic (0% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.3858	2.9	*	Middle San Diego Bay	COA	20-m	Significantly toxic (65.7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.3916	2.8	*	Southern California	COA	10-d	Moderately toxic (28.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.3978	1.8	SG	Southern California	COA	1.3-h	Toxic (7% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.3978		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3978		NE	Southern California	COA	35-d	Not toxic (27.4% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.3978		NE	Southern California	COA	35-d	Not toxic (0.028 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3978		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.3978		NE	Southern California	COA	35-d	Not toxic (0.0005 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4090	3.0	*	Middle San Diego Bay	COA	20-m	Significantly toxic (15% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.4090		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (82.3% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.4138		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	35-d	Not toxic (24.5% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	35-d	Not toxic (0.005 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	35-d	Not toxic (0.0013 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	10-d	Not toxic (9% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.4138		NE	Southern California	COA	1.3-h	Not toxic (75% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.4216	1.9	SG	Middle San Diego Bay	COA	10-d	Significantly toxic (49% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.4227	3.1	*	Middle San Diego Bay	COA	20-m	Significantly toxic (23% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.4227		NE	Middle San Diego Bay	COA	48-h	Not significantly toxic (73.5% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.4474	2.1	*	Middle San Diego Bay	COA	10-d	Significantly toxic (100% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.4579		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4579		NE	Southern California	COA	35-d	Not toxic (26.2% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4579		NE	Southern California	COA	35-d	Not toxic (0.025 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4579		NE	Southern California	COA	35-d	Not toxic (0.0004 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4579		NE	Southern California	COA	1.3-h	Not toxic (72% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.4579	1.6	SG	Southern California	COA		Toxic (0.0023 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.4694	3.4	*	Southern California	COA	10-d	Moderately toxic (40% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.4741		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (21% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.4794	3.1	*	Middle San Diego Bay	COA	48-h	Significantly toxic (54.4% normal development)	Strongylocentrotus purpuratus (sea urchin)	EMB	1	Fairey et al. 1996
0.4794	3.5	*	Middle San Diego Bay	COA	20-m	Significantly toxic (1.3% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
0.5767			*ER-M*							
0.5767	2.1	*	Southern California	COA		Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.5767	2.6	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.5767		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.5767		NE	Southern California	COA	35-d	Not toxic (21.9% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.5767		NE	Southern California	COA	35-d	Not toxic (0.027 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.5767		NE	Southern California	COA	35-d	Not toxic (0.0007 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214	2.8	*	Southern California	COA	1.3-h	Toxic (0% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
0.6214		NE	Southern California	COA	35-d	Not toxic (0% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214		NE	Southern California	COA	35-d	Not toxic (30.8% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214		NE	Southern California	COA	35-d	Not toxic (0.022 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214		NE	Southern California	COA	35-d	Not toxic (0.004 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214		NE	Southern California	COA	35-d	Not toxic (0.0006 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
0.6214		NE	Southern California	COA	10-d	Not toxic (5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
0.6463		NE	Southern California	COA	10-d	Least toxic (12.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.6918	5.0	*	Southern California	COA	10-d	Moderately toxic (47.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
0.7349	3.4	*	Middle San Diego Bay	COA	10-d	Significantly toxic (39% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.8159	3.7	*	Middle San Diego Bay	COA	10-d	Significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996

Table A4-24. A summary of the available data on the biological effects associated with sediment-sorbed Total Polychlorinated Biphenyls (mg/kg DW at 1% OC; unsummarized data set).

Total PCBs Conc.+/-SD	Ratio	Hit	Area	Analysis Type	Test Type	End-Point Measured	Species	Life Stage	TOC (%)	Reference
0.8663		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
0.9110		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
1.00		NE	Middle San Diego Bay	COA	10-d	Not significantly toxic (14% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
1.01	7.2	*	Southern California	COA	10-d	Most toxic (78.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.03		NE	Middle San Diego Bay	COA	20-m	Not significantly toxic (94% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Fairey et al. 1996
1.10	3.9	*	Southern California	COA		Toxic (0.002 g WW/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.10	5.0	*	Southern California	COA	1.3-h	Toxic (1% fertilization)	Strongylocentrotus purpuratus (sea urchin)	GAM	1	Bay et al. 1994
1.10		NE	Southern California	COA	35-d	Not toxic (4% mortality)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.10		NE	Southern California	COA	35-d	Not toxic (27.1% avoidance)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.10		NE	Southern California	COA	35-d	Not toxic (0.023 mm/d growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.10		NE	Southern California	COA	35-d	Not toxic (0.0003 g WW/d gonad growth)	Lytechinus pictus (sea urchin)	ADT	1	Bay et al. 1994
1.10		NE	Southern California	COA	10-d	Not toxic (19% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Bay et al. 1994
1.13	5.2	*	Middle San Diego Bay	COA	10-d	Significantly toxic (53% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
1.31	9.4	*	Southern California	COA	10-d	Most toxic (76.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.54	11	*	Southern California	COA	10-d	Most toxic (82.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.57	11	*	Southern California	COA	10-d	Moderately toxic (43.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.62	12	*	Southern California	COA	10-d	Moderately toxic (37.5% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
1.68	12	*	Southern California	COA	10-d	Most toxic (66.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.33	17	*	Southern California	COA	10-d	Most toxic (90% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.35	17	*	Southern California	COA	10-d	Most toxic (81.3% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
2.64	12	*	Middle San Diego Bay	COA	10-d	Significantly toxic (95% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Fairey et al. 1996
3.13	22	*	Southern California	COA	10-d	Most toxic (63.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
3.19	23	*	Southern California	COA	10-d	Most toxic (85% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991
3.96	64	*	Palos Verdes Shelf	COA	20-d	Significantly toxic (250 ej/replicate)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.96		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (0.53 mg WW/d growth)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
3.96		NE	Palos Verdes Shelf	COA	20-d	Not significantly toxic (4% mortality)	Neanthes arenaceodentata (polychaete)	JUV	1	Murdoch et al. In press
4.84	35	*	Southern California	COA	10-d	Most toxic (83.8% mortality)	Rhepoxynius abronius (amphipod)	ADT	1	Swartz et al. 1991

See Appendix 2 for glossary of acronyms.

The ratio was calculated by dividing the measured concentration of the substance in sediments at the toxic site by the mean concentration of that substance at the non-toxic sites for the same study. Information on the mean concentrations of the substance at the non-toxic sites can be found in the Summarized data tables.

Appendix 5

Independent Data Set for DDTs & PCBs

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.0002	0.0001	Tampa Bay, FL	5 % mort.	NT	72.4 % fert.	T			T	Long et al. 1994
0.0004	0.0030	Tampa Bay, FL	11 % mort.	NT	85.2 % fert.	NT			NT	Long et al. 1994
0.0004	0.0002	Tampa Bay, FL	4 % mort.	NT	22.6 % fert.	T			T	Long et al. 1994
0.0004	0.0003	Tampa Bay, FL	6 % mort.	NT	46 % fert.	T			T	Long et al. 1994
0.0004	0.0003	Tampa Bay, FL	7 % mort.	NT	91.6 % fert.	NT			NT	Long et al. 1994
0.0004	0.0002	Tampa Bay, FL	7 % mort.	NT	71.4 % fert.	T			T	Long et al. 1994
0.0004	0.0002	Tampa Bay, FL	7 % mort.	NT	0 % fert.	T			T	Long et al. 1994
0.0004	0.0014	Tampa Bay, FL	9 % mort.	NT	77.6 % fert.	NT			NT	Long et al. 1994
0.0004	0.0012	Tampa Bay, FL	13 % mort.	NT	46.2 % fert.	T			T	Long et al. 1994
0.0004	0.0003	Tampa Bay, FL	14 % mort.	NT	19.2 % fert.	T			T	Long et al. 1994
0.0004	0.0002	Tampa Bay, FL	15 % mort.	NT	78.4 % fert.	NT			NT	Long et al. 1994
0.0004	0.0048	Tampa Bay, FL	16 % mort.	NT	61.6 % fert.	T			T	Long et al. 1994
0.0004	0.0023	Tampa Bay, FL	19 % mort.	NT	91.6 % fert.	NT			NT	Long et al. 1994
0.0004	0.0022	Tampa Bay, FL	20 % mort.	NT	86.4 % fert.	NT			NT	Long et al. 1994
0.0004	0.0024	Tampa Bay, FL	23 % mort.	NT	9.6 % fert.	T			T	Long et al. 1994
0.0007	0.0003	Tampa Bay, FL	5 % mort.	NT	84.2 % fert.	NT			NT	Long et al. 1994
0.0007	0.0008	Tampa Bay, FL	15 % mort.	NT	82.8 % fert.	NT			NT	Long et al. 1994
0.0011	0.0030	Tampa Bay, FL	11 % mort.	NT	25 % fert.	T			T	Long et al. 1994
0.0011	0.0008	Tampa Bay, FL	16 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0014	0.0004	Tampa Bay, FL	19 % mort.	NT	43.8 % fert.	T			T	Long et al. 1994
0.0015	0.0008	Tampa Bay, FL	6 % mort.	NT	3.4 % fert.	T			T	Long et al. 1994
0.0019	0.0004	Tampa Bay, FL	5 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0019	0.0004	Tampa Bay, FL	12 % mort.	NT	0.6 % fert.	T			T	Long et al. 1994
0.0019	0.0074	Tampa Bay, FL	13 % mort.	NT	84.4 % fert.	NT			NT	Long et al. 1994
0.0019	0.0040	Tampa Bay, FL	15 % mort.	NT	88.4 % fert.	NT			NT	Long et al. 1994
0.0023	0.0022	Tampa Bay, FL	18 % mort.	NT	86.4 % fert.	NT			NT	Long et al. 1994
0.0023	0.0011	Tampa Bay, FL	12 % mort.	NT	16 % fert.	T			T	Long et al. 1994
0.0024	0.0022	Tampa Bay, FL	17 % mort.	NT	17.8 % fert.	T			T	Long et al. 1994
0.0025	0.0007	Tampa Bay, FL	19 % mort.	NT	43 % fert.	T			T	Long et al. 1994
0.0028	0.0009	Tampa Bay, FL	1 % mort.	NT	27.4 % fert.	T			T	Long et al. 1994
0.0030	0.0016	Tampa Bay, FL	10 % mort.	NT	3 % fert.	T			T	Long et al. 1994
0.0035	0.0010	Tampa Bay, FL	17.5 % mort.	T	0.6 % fert.	T			T	Long et al. 1994
0.0040	0.0009	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T			T	Long et al. 1994
0.0045	0.0038	Tampa Bay, FL	18 % mort.	NT	7.6 % fert.	T			T	Long et al. 1994
0.0045	0.0027	Tampa Bay, FL	22 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0046	0.0018	Tampa Bay, FL	10 % mort.	NT	5.8 % fert.	T			T	Long et al. 1994
0.0047	0.0016	Tampa Bay, FL	10 % mort.	NT	3.2 % fert.	T			T	Long et al. 1994
0.0049	0.0019	Tampa Bay, FL	6 % mort.	NT	0.4 % fert.	T			T	Long et al. 1994

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development	Overall Toxicity	Reference
0.0053	0.0055	Tampa Bay, FL	13 % mort.	NT	42.8 % fert.	T		T	Long et al. 1994
0.0055	0.0021	Tampa Bay, FL	10 % mort.	NT	5.6 % fert.	T		T	Long et al. 1994
0.0058	0.0036	Tampa Bay, FL	22 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0068	0.0019	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0094	0.0024	Tampa Bay, FL	16 % mort.	NT	1 % fert.	T		T	Long et al. 1994
0.0101	0.0026	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0102	0.0043	Tampa Bay, FL	10 % mort.	NT	27.2 % fert.	T		T	Long et al. 1994
0.0107	0.0046	Tampa Bay, FL	17 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0108	0.0033	Tampa Bay, FL	27 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0119	0.0022	Tampa Bay, FL	14 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0125	0.0027	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0141	0.0060	Tampa Bay, FL	18 % mort.	NT	74 % fert.	T		T	Long et al. 1994
0.0147	0.0100	Tampa Bay, FL	15 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0179	0.0061	Tampa Bay, FL	8 % mort.	NT	4 % fert.	T		T	Long et al. 1994
0.0184	0.0030	Tampa Bay, FL	15 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994
0.0306	0.0050	Tampa Bay, FL	17 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994
0.0378	0.0106	Tampa Bay, FL	13 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0589	0.0179	Tampa Bay, FL	23 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0600	0.0212	Tampa Bay, FL	55 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0615	0.0366	Tampa Bay, FL	24 % mort.	T	11 % fert.	T		T	Long et al. 1994
0.0898	0.0143	Tampa Bay, FL	22.5 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.2581	0.0589	Tampa Bay, FL	61 % mort.	T	0 % fert.	T		T	Long et al. 1994
2.67	0.8995	Tampa Bay, FL	52 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0004	0.0001	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0005	0.0001	Hudson-Raritan Estuary	100 % mort.	T				T	Long et al. 1995b
0.0007	0.0100	Hudson-Raritan Estuary	9 % mort.	NT				NT	Long et al. 1995b
0.0010	0.0040	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0010	0.0003	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0010	0.0006	Hudson-Raritan Estuary	4 % mort.	NT				NT	Long et al. 1995b
0.0020	0.0008	Hudson-Raritan Estuary	14 % mort.	NT				NT	Long et al. 1995b
0.0030	0.0011	Hudson-Raritan Estuary	81 % mort.	T				T	Long et al. 1995b
0.0040	0.0017	Hudson-Raritan Estuary	28 % mort.	T				T	Long et al. 1995b
0.0040	0.0085	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0050	0.0020	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0050	0.0019	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0090	0.0053	Hudson-Raritan Estuary	21 % mort.	T				T	Long et al. 1995b
0.0100	0.0050	Hudson-Raritan Estuary	24 % mort.	T				T	Long et al. 1995b

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development		Overall Toxicity	Reference
0.0110	0.0110	Hudson-Raritan Estuary	97 % mort.	T					T	Long et al. 1995b
0.0130	0.0087	Hudson-Raritan Estuary	82 % mort.	T					T	Long et al. 1995b
0.0170	0.0088	Hudson-Raritan Estuary	7 % mort.	T					T	Long et al. 1995b
0.0170	0.0077	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b
0.0190	0.0049	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0190	0.0043	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b
0.0220	0.0070	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0220	0.0072	Hudson-Raritan Estuary	49 % mort.	T					T	Long et al. 1995b
0.0250	0.0078	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0250	0.0100	Hudson-Raritan Estuary	17 % mort.	NT					NT	Long et al. 1995b
0.0260	0.0059	Hudson-Raritan Estuary	82 % mort.	T					T	Long et al. 1995b
0.0270	0.0074	Hudson-Raritan Estuary	24 % mort.	T					T	Long et al. 1995b
0.0280	0.0364	Hudson-Raritan Estuary	9 % mort.	NT					NT	Long et al. 1995b
0.0280	0.0093	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0280	0.0122	Hudson-Raritan Estuary	91 % mort.	T					T	Long et al. 1995b
0.0290	0.0104	Hudson-Raritan Estuary	12 % mort.	NT					NT	Long et al. 1995b
0.0320	0.0128	Hudson-Raritan Estuary	19 % mort.	T					T	Long et al. 1995b
0.0320	0.0133	Hudson-Raritan Estuary	85 % mort.	T					T	Long et al. 1995b
0.0320	0.0076	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b
0.0360	0.0113	Hudson-Raritan Estuary	6 % mort.	NT					NT	Long et al. 1995b
0.0460	0.0079	Hudson-Raritan Estuary	75 % mort.	T					T	Long et al. 1995b
0.0480	0.0079	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b
0.0520	0.0547	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b
0.0520	0.0104	Hudson-Raritan Estuary	69 % mort.	T					T	Long et al. 1995b
0.0620	0.0155	Hudson-Raritan Estuary	23 % mort.	T					T	Long et al. 1995b
0.0620	0.0295	Hudson-Raritan Estuary	47 % mort.	T					T	Long et al. 1995b
0.0690	0.0288	Hudson-Raritan Estuary	81 % mort.	T					T	Long et al. 1995b
0.0690	0.0135	Hudson-Raritan Estuary	48 % mort.	T					T	Long et al. 1995b
0.1010	0.0235	Hudson-Raritan Estuary	23 % mort.	NT					NT	Long et al. 1995b
0.1090	0.0454	Hudson-Raritan Estuary	32 % mort.	T					T	Long et al. 1995b
0.1390	0.0579	Hudson-Raritan Estuary	67 % mort.	T					T	Long et al. 1995b
0.2340	0.0490	Hudson-Raritan Estuary	98 % mort.	T					T	Long et al. 1995b
0.2340	0.1182	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b
0.4180	0.1310	Hudson-Raritan Estuary	86 % mort.	T					T	Long et al. 1995b
0.4990	0.1674	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b
0.7260	0.4939	Hudson-Raritan Estuary	63 % mort.	T					T	Long et al. 1995b
0.00003	0.0001	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.00005	0.0002	Biscayne Bay, FL	10 % mort.	NT	81 % fert.	NT	56 % norm. dev.	T	T	Long 1997

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.00005	0.0001	Biscayne Bay, FL	35 % mort.	T	110 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	89 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	19 % mort.	NT	110 % fert.	NT	3 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	9 % mort.	NT	96 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	11 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0004	Biscayne Bay, FL	9 % mort.	NT	72 % fert.	T	91 % norm. dev.	NT	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	69 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0005	Biscayne Bay, FL	0 % mort.	NT	96 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	117 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0000	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0004	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	2 % mort.	NT	68 % fert.	T	82 % norm. dev.	NT	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	2 % mort.	NT	111 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0007	Biscayne Bay, FL	3 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0001	0.0000	Biscayne Bay, FL	4 % mort.	NT	98 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	109 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	114 % fert.	NT	62 % norm. dev.	T	T	Long 1997
0.0001	0.0000	Biscayne Bay, FL	4 % mort.	NT	80.05 % fert.	NT	85 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	5 % mort.	NT	0 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	6 % mort.	NT	1 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0000	Biscayne Bay, FL	7 % mort.	NT	1 % fert.	T	2 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	7 % mort.	NT	110 % fert.	NT	53 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	8 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	9 % mort.	NT	95 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0001	0.0009	Biscayne Bay, FL	11 % mort.	NT	95 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	12 % mort.	NT	105 % fert.	NT	105 % norm. dev.	NT	NT	Long 1997
0.0001	0.0008	Biscayne Bay, FL	18 % mort.	NT	99 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	18 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0006	Biscayne Bay, FL	18 % mort.	NT	59 % fert.	T	64 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	23 % mort.	T	91 % fert.	NT	1 % norm. dev.	T	T	Long 1997

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development		Overall Toxicity	Reference
0.0001	0.0004	Biscayne Bay, FL	43 % mort.	T	34 % fert.	T	13 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	56 % mort.	T	0 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	4 % mort.	NT	78 % fert.	T	103 % norm. dev.	NT	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	19 % mort.	NT	115 % fert.	NT	45 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	5 % mort.	NT	117 % fert.	NT	2 % norm. dev.	T	T	Long 1997
0.0002	0.0001	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	98 % norm. dev.	NT	NT	Long 1997
0.0002	0.0001	Biscayne Bay, FL	2 % mort.	NT	62 % fert.	T	86 % norm. dev.	NT	T	Long 1997
0.0002	0.0002	Biscayne Bay, FL	0 % mort.	NT	83 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997
0.0002	0.0001	Biscayne Bay, FL	3 % mort.	NT	113 % fert.	NT	63 % norm. dev.	T	T	Long 1997
0.0002	0.0001	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	93.9 % norm. dev.	NT	NT	Long 1997
0.0002	0.0001	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	104 % norm. dev.	NT	NT	Long 1997
0.0002	0.0000	Biscayne Bay, FL	13 % mort.	NT	100 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0003	0.0006	Biscayne Bay, FL	6 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0003	0.0002	Biscayne Bay, FL	11 % mort.	NT	94 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0003	0.0004	Biscayne Bay, FL	17 % mort.	NT	115 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0003	0.0014	Biscayne Bay, FL	4 % mort.	NT	106 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0003	0.0007	Biscayne Bay, FL	1 % mort.	NT	96 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0004	0.0006	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0004	0.0002	Biscayne Bay, FL	2 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0004	0.0003	Biscayne Bay, FL	5 % mort.	NT	99 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997
0.0004	0.0003	Biscayne Bay, FL	8 % mort.	NT	66 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0004	0.0001	Biscayne Bay, FL	0 % mort.	NT	7 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0004	0.0003	Biscayne Bay, FL	0 % mort.	NT	47 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0005	0.0004	Biscayne Bay, FL	7 % mort.	NT	82 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997
0.0005	0.0004	Biscayne Bay, FL	1 % mort.	NT	95 % fert.	NT	101 % norm. dev.	NT	NT	Long 1997
0.0005	0.0003	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0006	0.0001	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0006	0.0001	Biscayne Bay, FL	7 % mort.	NT	92 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0007	0.0003	Biscayne Bay, FL	4 % mort.	NT	92 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0008	0.0008	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	18 % norm. dev.	T	T	Long 1997
0.0008	0.0004	Biscayne Bay, FL	4 % mort.	NT	112 % fert.	NT	10 % norm. dev.	T	T	Long 1997
0.0009	0.0004	Biscayne Bay, FL	1 % mort.	NT	80.4 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997
0.0009	0.0005	Biscayne Bay, FL	0 % mort.	NT	94 % fert.	NT	90 % norm. dev.	NT	NT	Long 1997
0.0009	0.0007	Biscayne Bay, FL	0 % mort.	NT	75 % fert.	T	1 % norm. dev.	T	T	Long 1997
0.0010	0.0002	Biscayne Bay, FL	3 % mort.	NT	62 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0011	0.0022	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	12 % norm. dev.	T	T	Long 1997
0.0014	0.0011	Biscayne Bay, FL	6 % mort.	NT	94 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997
0.0015	0.0002	Biscayne Bay, FL	5 % mort.	NT	88 % fert.	NT	0 % norm. dev.	T	T	Long 1997

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.0016	0.0005	Biscayne Bay, FL	0 % mort.	NT	19 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0017	0.0003	Biscayne Bay, FL	0 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0020	0.0003	Biscayne Bay, FL	9 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0021	0.0007	Biscayne Bay, FL	2 % mort.	NT	91 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997
0.0031	0.0010	Biscayne Bay, FL	31 % mort.	T	37 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0034	0.0024	Biscayne Bay, FL	59 % mort.	T	92 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0034	0.0010	Biscayne Bay, FL	5 % mort.	NT	98 % fert.	NT	75 % norm. dev.	T	T	Long 1997
0.0056	0.0015	Biscayne Bay, FL	6 % mort.	NT	99 % fert.	NT	103 % norm. dev.	NT	NT	Long 1997
0.0060	0.0016	Biscayne Bay, FL	59 % mort.	T	99 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0067	0.0018	Biscayne Bay, FL	9 % mort.	NT	83 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0077	0.0045	Biscayne Bay, FL	84 % mort.	T	80.4 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0096	0.0041	Biscayne Bay, FL	68 % mort.	T	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0108	0.0018	Biscayne Bay, FL	81 % mort.	T	104 % fert.	NT	13 % norm. dev.	T	T	Long 1997
0.0121	0.0051	Biscayne Bay, FL	98 % mort.	T	98 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0131	0.0049	Biscayne Bay, FL	65 % mort.	T	102 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0170	0.0370	Biscayne Bay, FL	69 % mort.	T	91 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0192	0.0069	Biscayne Bay, FL	91 % mort.	T	98 % fert.	NT	78 % norm. dev.	T	T	Long 1997
0.0208	0.0022	Biscayne Bay, FL	61 % mort.	T	100 % fert.	NT	79.8 % norm. dev.	T	T	Long 1997
0.0282	0.0072	Biscayne Bay, FL	49 % mort.	T	48 % fert.	T	3 % norm. dev.	T	T	Long 1997
0.0288	0.0020	Biscayne Bay, FL	90 % mort.	T	36 % fert.	T	74 % norm. dev.	T	T	Long 1997
0.0402	0.0096	Biscayne Bay, FL	59 % mort.	T	93 % fert.	NT	101 % norm. dev.	NT	T	Long 1997
0.0413	0.0066	Biscayne Bay, FL	61 % mort.	T	71 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0503	0.0193	Biscayne Bay, FL	33 % mort.	T	93 % fert.	NT	2 % norm. dev.	T	T	Long 1997
0.1012	0.0119	Biscayne Bay, FL	95 % mort.	T	96 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.1182	0.0134	Biscayne Bay, FL	91 % mort.	T	88 % fert.	NT	3 % norm. dev.	T	T	Long 1997
0.1495	0.0166	Biscayne Bay, FL	92 % mort.	T	14 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.2920	0.0960	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0001		Narragansett Bay, RI	3.3 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	10 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	2 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	2.7 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991

Appendix A5-1. A summary of the available information on the toxic effects of sediment-associated SUM DDT from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality	Sea Urchin Fertilization	Sea Urchin Development	Overall Toxicity	Reference
0.0003	<	Narragansett Bay, RI	6 % mort.	NT		NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	11.3 % mort.	NT		NT	Munns et al. 1991
0.0004		Narragansett Bay, RI	6.7 % mort.	NT		NT	Munns et al. 1991
0.0004		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0004		Narragansett Bay, RI	0 % mort.	NT		NT	Munns et al. 1991
0.0004		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0005		Narragansett Bay, RI	6.7 % mort.	NT		NT	Munns et al. 1991
0.0005		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0006		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0006		Narragansett Bay, RI	3.3 % mort.	NT		NT	Munns et al. 1991
0.0006		Narragansett Bay, RI	2.7 % mort.	NT		NT	Munns et al. 1991
0.0007		Narragansett Bay, RI	1.3 % mort.	NT		NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0011		Narragansett Bay, RI	8.7 % mort.	NT		NT	Munns et al. 1991
0.0059		Narragansett Bay, RI	10.7 % mort.	NT		NT	Munns et al. 1991
0.0093		Narragansett Bay, RI	6 % mort.	NT		NT	Munns et al. 1991
0.0432		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0530		Narragansett Bay, RI	8 % mort.	NT		NT	Munns et al. 1991
0.0030	0.0034	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.0180	0.0153	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.0850	0.0787	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
0.0890	0.0597	San Francisco Bay, CA	28 % mort.	NT		NT	Swartz et al. 1994
0.5380	0.1805	San Francisco Bay, CA	32 % mort.	T		T	Swartz et al. 1994
0.8810	0.6034	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
7.54	4.36	San Francisco Bay, CA	36 % mort.	T		T	Swartz et al. 1994
28.7	16.1	San Francisco Bay, CA	43 % mort.	T		T	Swartz et al. 1994
37.0	15.6	San Francisco Bay, CA	66.2 % mort.	T		T	Swartz et al. 1994

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.

Endpoint Measured: mort. = mortality; fert. = fertilization; norm. dev. = normal development.

NT = toxic; T = toxic.

Species tested included:

Long et al. 1994; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Long et al. 1995b; Ampelisca abdita (amphipod).

Long 1997; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Munns et al. 1991; Ampelisca abdita (amphipod).

Swartz et al. 1994; Eohaustorius estuarius (amphipod).

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.0003	0.0015	Tampa Bay, FL	20 % mort.	NT	86.4 % fert.	NT			NT	Long et al. 1994
0.0003	0.0023	Tampa Bay, FL	11 % mort.	NT	85.2 % fert.	NT			NT	Long et al. 1994
0.0003	0.0009	Tampa Bay, FL	11 % mort.	NT	25 % fert.	T			T	Long et al. 1994
0.0006	0.0032	Tampa Bay, FL	23 % mort.	NT	9.6 % fert.	T			T	Long et al. 1994
0.0007	0.0008	Tampa Bay, FL	15 % mort.	NT	82.8 % fert.	NT			NT	Long et al. 1994
0.0007	0.0082	Tampa Bay, FL	16 % mort.	NT	61.6 % fert.	T			T	Long et al. 1994
0.0008	0.0003	Tampa Bay, FL	15 % mort.	NT	78.4 % fert.	NT			NT	Long et al. 1994
0.0010	0.0005	Tampa Bay, FL	10 % mort.	NT	3 % fert.	T			T	Long et al. 1994
0.0012	0.0006	Tampa Bay, FL	5 % mort.	NT	72.4 % fert.	T			T	Long et al. 1994
0.0014	0.0030	Tampa Bay, FL	15 % mort.	NT	88.4 % fert.	NT			NT	Long et al. 1994
0.0016	0.0083	Tampa Bay, FL	19 % mort.	NT	91.6 % fert.	NT			NT	Long et al. 1994
0.0017	0.0007	Tampa Bay, FL	5 % mort.	NT	84.2 % fert.	NT			NT	Long et al. 1994
0.0017	0.0008	Tampa Bay, FL	6 % mort.	NT	3.4 % fert.	T			T	Long et al. 1994
0.0018	0.0010	Tampa Bay, FL	6 % mort.	NT	46 % fert.	T			T	Long et al. 1994
0.0024	0.0069	Tampa Bay, FL	13 % mort.	NT	46.2 % fert.	T			T	Long et al. 1994
0.0025	0.0009	Tampa Bay, FL	1 % mort.	NT	27.4 % fert.	T			T	Long et al. 1994
0.0026	0.0012	Tampa Bay, FL	12 % mort.	NT	16 % fert.	T			T	Long et al. 1994
0.0028	0.0016	Tampa Bay, FL	7 % mort.	NT	91.6 % fert.	NT			NT	Long et al. 1994
0.0028	0.0022	Tampa Bay, FL	16 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0032	0.0102	Tampa Bay, FL	9 % mort.	NT	77.6 % fert.	NT			NT	Long et al. 1994
0.0033	0.0032	Tampa Bay, FL	18 % mort.	NT	86.4 % fert.	NT			NT	Long et al. 1994
0.0036	0.0008	Tampa Bay, FL	5 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0038	0.0014	Tampa Bay, FL	4 % mort.	NT	22.6 % fert.	T			T	Long et al. 1994
0.0044	0.0012	Tampa Bay, FL	17.5 % mort.	T	0.6 % fert.	T			T	Long et al. 1994
0.0044	0.0010	Tampa Bay, FL	12 % mort.	NT	0.6 % fert.	T			T	Long et al. 1994
0.0045	0.0037	Tampa Bay, FL	18 % mort.	NT	7.6 % fert.	T			T	Long et al. 1994
0.0051	0.0019	Tampa Bay, FL	7 % mort.	NT	0 % fert.	T			T	Long et al. 1994
0.0053	0.0035	Tampa Bay, FL	14 % mort.	NT	19.2 % fert.	T			T	Long et al. 1994
0.0059	0.0018	Tampa Bay, FL	19 % mort.	NT	43.8 % fert.	T			T	Long et al. 1994
0.0060	0.0021	Tampa Bay, FL	7 % mort.	NT	71.4 % fert.	T			T	Long et al. 1994
0.0063	0.0038	Tampa Bay, FL	22 % mort.	NT	0.2 % fert.	T			T	Long et al. 1994
0.0063	0.0040	Tampa Bay, FL	22 % mort.	NT	0 % fert.	T			T	Long et al. 1994
0.0081	0.0022	Tampa Bay, FL	19 % mort.	NT	43 % fert.	T			T	Long et al. 1994
0.0088	0.0060	Tampa Bay, FL	15 % mort.	NT	0 % fert.	T			T	Long et al. 1994
0.0101	0.0043	Tampa Bay, FL	10 % mort.	NT	27.2 % fert.	T			T	Long et al. 1994
0.0125	0.0501	Tampa Bay, FL	13 % mort.	NT	84.4 % fert.	NT			NT	Long et al. 1994
0.0133	0.0079	Tampa Bay, FL	24 % mort.	T	11 % fert.	T			T	Long et al. 1994
0.0157	0.0048	Tampa Bay, FL	27 % mort.	T	0 % fert.	T			T	Long et al. 1994

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration			Endpoint Measured						
			Amphipod		Sea Urchin			Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development	Toxicity	Reference
0.0192	0.0036	Tampa Bay, FL	14 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0204	0.0052	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0233	0.0065	Tampa Bay, FL	13 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0240	0.0052	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0259	0.0099	Tampa Bay, FL	10 % mort.	NT	5.6 % fert.	T		T	Long et al. 1994
0.0286	0.0264	Tampa Bay, FL	17 % mort.	NT	17.8 % fert.	T		T	Long et al. 1994
0.0341	0.0359	Tampa Bay, FL	13 % mort.	NT	42.8 % fert.	T		T	Long et al. 1994
0.0377	0.0094	Tampa Bay, FL	16 % mort.	NT	1 % fert.	T		T	Long et al. 1994
0.0406	0.0140	Tampa Bay, FL	10 % mort.	NT	3.2 % fert.	T		T	Long et al. 1994
0.0464	0.0178	Tampa Bay, FL	10 % mort.	NT	5.8 % fert.	T		T	Long et al. 1994
0.0471	0.0143	Tampa Bay, FL	23 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0535	0.0212	Tampa Bay, FL	6 % mort.	NT	0.4 % fert.	T		T	Long et al. 1994
0.0582	0.0206	Tampa Bay, FL	55 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0609	0.0097	Tampa Bay, FL	22.5 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0645	0.0178	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0710	0.0162	Tampa Bay, FL	61 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0820	0.0280	Tampa Bay, FL	8 % mort.	NT	4 % fert.	T		T	Long et al. 1994
0.0870	0.0368	Tampa Bay, FL	18 % mort.	NT	74 % fert.	T		T	Long et al. 1994
0.0974	0.0160	Tampa Bay, FL	15 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994
0.0985	0.0160	Tampa Bay, FL	17 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994
0.1009	0.0431	Tampa Bay, FL	17 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.1396	0.0299	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.6035	0.2032	Tampa Bay, FL	52 % mort.	T	0 % fert.	T		T	Long et al. 1994
0.0006	0.0086	Hudson-Raritan Estuary	9 % mort.	NT				NT	Long et al. 1995b
0.0010	0.0004	Hudson-Raritan Estuary	14 % mort.	NT				NT	Long et al. 1995b
0.0010	0.0040	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0030	0.0011	Hudson-Raritan Estuary	81 % mort.	T				T	Long et al. 1995b
0.0040	0.0085	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0050	0.0029	Hudson-Raritan Estuary	4 % mort.	NT				NT	Long et al. 1995b
0.0080	0.0028	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0090	0.0018	Hudson-Raritan Estuary	100 % mort.	T				T	Long et al. 1995b
0.0110	0.0143	Hudson-Raritan Estuary	9 % mort.	NT				NT	Long et al. 1995b
0.0110	0.0041	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0110	0.0036	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b
0.0110	0.0046	Hudson-Raritan Estuary	28 % mort.	T				T	Long et al. 1995b
0.0120	0.0027	Hudson-Raritan Estuary	82 % mort.	T				T	Long et al. 1995b
0.0140	0.0140	Hudson-Raritan Estuary	97 % mort.	T				T	Long et al. 1995b

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod		Sea Urchin				Overall		
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference	
0.0150	0.0034	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b	
0.0160	0.0044	Hudson-Raritan Estuary	24 % mort.	T					T	Long et al. 1995b	
0.0160	0.0168	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b	
0.0160	0.0080	Hudson-Raritan Estuary	24 % mort.	T					T	Long et al. 1995b	
0.0170	0.0044	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b	
0.0170	0.0061	Hudson-Raritan Estuary	12 % mort.	NT					NT	Long et al. 1995b	
0.0180	0.0093	Hudson-Raritan Estuary	7 % mort.	T					T	Long et al. 1995b	
0.0220	0.0072	Hudson-Raritan Estuary	49 % mort.	T					T	Long et al. 1995b	
0.0230	0.0092	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b	
0.0230	0.0092	Hudson-Raritan Estuary	19 % mort.	T					T	Long et al. 1995b	
0.0260	0.0060	Hudson-Raritan Estuary	23 % mort.	NT					NT	Long et al. 1995b	
0.0260	0.0153	Hudson-Raritan Estuary	21 % mort.	T					T	Long et al. 1995b	
0.0300	0.0093	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b	
0.0300	0.0095	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b	
0.0330	0.0220	Hudson-Raritan Estuary	82 % mort.	T					T	Long et al. 1995b	
0.0340	0.0107	Hudson-Raritan Estuary	6 % mort.	NT					NT	Long et al. 1995b	
0.0390	0.0129	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b	
0.0390	0.0163	Hudson-Raritan Estuary	32 % mort.	T					T	Long et al. 1995b	
0.0450	0.0214	Hudson-Raritan Estuary	47 % mort.	T					T	Long et al. 1995b	
0.0460	0.0184	Hudson-Raritan Estuary	17 % mort.	NT					NT	Long et al. 1995b	
0.0490	0.0123	Hudson-Raritan Estuary	23 % mort.	T					T	Long et al. 1995b	
0.0550	0.0095	Hudson-Raritan Estuary	75 % mort.	T					T	Long et al. 1995b	
0.0560	0.0112	Hudson-Raritan Estuary	69 % mort.	T					T	Long et al. 1995b	
0.0580	0.0121	Hudson-Raritan Estuary	98 % mort.	T					T	Long et al. 1995b	
0.0590	0.0185	Hudson-Raritan Estuary	86 % mort.	T					T	Long et al. 1995b	
0.0590	0.0246	Hudson-Raritan Estuary	67 % mort.	T					T	Long et al. 1995b	
0.0590	0.0246	Hudson-Raritan Estuary	85 % mort.	T					T	Long et al. 1995b	
0.0610	0.0205	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b	
0.0620	0.0122	Hudson-Raritan Estuary	48 % mort.	T					T	Long et al. 1995b	
0.0630	0.0263	Hudson-Raritan Estuary	81 % mort.	T					T	Long et al. 1995b	
0.0740	0.0322	Hudson-Raritan Estuary	91 % mort.	T					T	Long et al. 1995b	
0.0770	0.0126	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b	
0.0820	0.0558	Hudson-Raritan Estuary	63 % mort.	T					T	Long et al. 1995b	
0.0830	0.0198	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b	
0.0860	0.0391	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b	
0.1030	0.0520	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b	
0.00003	0.0002	Biscayne Bay, FL	11 % mort.	NT	95 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997	
0.00004	0.0002	Biscayne Bay, FL	10 % mort.	NT	81 % fert.	NT	56 % norm. dev.	T	T	Long 1997	

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development		Overall Toxicity	Reference
0.0001	0.0003	Biscayne Bay, FL	18 % mort.	NT	99 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	89 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	69 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	0 % mort.	NT	96 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	117 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	111 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0004	Biscayne Bay, FL	3 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0001	0.0003	Biscayne Bay, FL	4 % mort.	NT	106 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	109 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0001	0.0001	Biscayne Bay, FL	5 % mort.	NT	117 % fert.	NT	2 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	6 % mort.	NT	1 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0000	Biscayne Bay, FL	7 % mort.	NT	1 % fert.	T	2 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	7 % mort.	NT	110 % fert.	NT	53 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	11 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	12 % mort.	NT	105 % fert.	NT	105 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	18 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0003	Biscayne Bay, FL	18 % mort.	NT	59 % fert.	T	64 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	19 % mort.	NT	115 % fert.	NT	45 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	19 % mort.	NT	110 % fert.	NT	3 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	35 % mort.	T	110 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	43 % mort.	T	34 % fert.	T	13 % norm. dev.	T	T	Long 1997
0.0001	0.0000	Biscayne Bay, FL	56 % mort.	T	0 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0002	Biscayne Bay, FL	6 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	5 % mort.	NT	0 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	17 % mort.	NT	115 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	9 % mort.	NT	96 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997
0.0001	0.0004	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	4 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	114 % fert.	NT	62 % norm. dev.	T	T	Long 1997
0.0001	0.0005	Biscayne Bay, FL	9 % mort.	NT	72 % fert.	T	91 % norm. dev.	NT	T	Long 1997
0.0002	0.0003	Biscayne Bay, FL	23 % mort.	T	91 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0002	0.0001	Biscayne Bay, FL	8 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0002	0.0003	Biscayne Bay, FL	4 % mort.	NT	78 % fert.	T	103 % norm. dev.	NT	T	Long 1997
0.0003	0.0005	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	1 % norm. dev.	T	T	Long 1997

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development		Overall Toxicity	Reference
0.0003	0.0002	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	98 % norm. dev.	NT	NT	Long 1997
0.0003	0.0008	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0003	0.0003	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	18 % norm. dev.	T	T	Long 1997
0.0003	0.0007	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0004	0.0003	Biscayne Bay, FL	7 % mort.	NT	82 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997
0.0004	0.0004	Biscayne Bay, FL	2 % mort.	NT	68 % fert.	T	82 % norm. dev.	NT	T	Long 1997
0.0004	0.0003	Biscayne Bay, FL	11 % mort.	NT	94 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0004	0.0007	Biscayne Bay, FL	9 % mort.	NT	95 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0004	0.0003	Biscayne Bay, FL	8 % mort.	NT	66 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0005	0.0002	Biscayne Bay, FL	2 % mort.	NT	62 % fert.	T	86 % norm. dev.	NT	T	Long 1997
0.0006	0.0005	Biscayne Bay, FL	1 % mort.	NT	95 % fert.	NT	101 % norm. dev.	NT	NT	Long 1997
0.0006	0.0004	Biscayne Bay, FL	0 % mort.	NT	75 % fert.	T	1 % norm. dev.	T	T	Long 1997
0.0006	0.0012	Biscayne Bay, FL	1 % mort.	NT	96 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0006	0.0004	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0006	0.0005	Biscayne Bay, FL	5 % mort.	NT	99 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997
0.0007	0.0004	Biscayne Bay, FL	2 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0007	0.0004	Biscayne Bay, FL	0 % mort.	NT	47 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0008	0.0009	Biscayne Bay, FL	0 % mort.	NT	83 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997
0.0008	0.0014	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0008	0.0016	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	12 % norm. dev.	T	T	Long 1997
0.0009	0.0003	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	104 % norm. dev.	NT	NT	Long 1997
0.0009	0.0003	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	93.9 % norm. dev.	NT	NT	Long 1997
0.0010	0.0005	Biscayne Bay, FL	0 % mort.	NT	94 % fert.	NT	90 % norm. dev.	NT	NT	Long 1997
0.0011	0.0003	Biscayne Bay, FL	4 % mort.	NT	98 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997
0.0011	0.0005	Biscayne Bay, FL	1 % mort.	NT	80.4 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997
0.0011	0.0002	Biscayne Bay, FL	13 % mort.	NT	100 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0013	0.0010	Biscayne Bay, FL	6 % mort.	NT	94 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997
0.0014	0.0006	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0014	0.0008	Biscayne Bay, FL	3 % mort.	NT	113 % fert.	NT	63 % norm. dev.	T	T	Long 1997
0.0017	0.0007	Biscayne Bay, FL	4 % mort.	NT	92 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.0020	0.0006	Biscayne Bay, FL	4 % mort.	NT	80.05 % fert.	NT	85 % norm. dev.	NT	NT	Long 1997
0.0021	0.0007	Biscayne Bay, FL	2 % mort.	NT	91 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997
0.0024	0.0005	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0025	0.0005	Biscayne Bay, FL	0 % mort.	NT	7 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0026	0.0004	Biscayne Bay, FL	9 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0031	0.0005	Biscayne Bay, FL	0 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0033	0.0005	Biscayne Bay, FL	5 % mort.	NT	88 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0035	0.0005	Biscayne Bay, FL	7 % mort.	NT	92 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0037	0.0011	Biscayne Bay, FL	0 % mort.	NT	19 % fert.	T	0 % norm. dev.	T	T	Long 1997

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development		Overall Toxicity	Reference
0.0046	0.0023	Biscayne Bay, FL	4 % mort.	NT	112 % fert.	NT	10 % norm. dev.	T	T	Long 1997
0.0070	0.0018	Biscayne Bay, FL	9 % mort.	NT	83 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0076	0.0044	Biscayne Bay, FL	84 % mort.	T	80.4 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0077	0.0021	Biscayne Bay, FL	6 % mort.	NT	99 % fert.	NT	103 % norm. dev.	NT	NT	Long 1997
0.0084	0.0061	Biscayne Bay, FL	59 % mort.	T	92 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0093	0.0028	Biscayne Bay, FL	5 % mort.	NT	98 % fert.	NT	75 % norm. dev.	T	T	Long 1997
0.0136	0.0058	Biscayne Bay, FL	68 % mort.	T	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0137	0.0036	Biscayne Bay, FL	3 % mort.	NT	62 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0138	0.0053	Biscayne Bay, FL	33 % mort.	T	93 % fert.	NT	2 % norm. dev.	T	T	Long 1997
0.0142	0.0047	Biscayne Bay, FL	31 % mort.	T	37 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0146	0.0038	Biscayne Bay, FL	59 % mort.	T	99 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0230	0.0059	Biscayne Bay, FL	49 % mort.	T	48 % fert.	T	3 % norm. dev.	T	T	Long 1997
0.0261	0.0094	Biscayne Bay, FL	91 % mort.	T	98 % fert.	NT	78 % norm. dev.	T	T	Long 1997
0.0264	0.0575	Biscayne Bay, FL	69 % mort.	T	91 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0285	0.0119	Biscayne Bay, FL	98 % mort.	T	98 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0293	0.0031	Biscayne Bay, FL	61 % mort.	T	100 % fert.	NT	79.8 % norm. dev.	T	T	Long 1997
0.0303	0.0073	Biscayne Bay, FL	59 % mort.	T	93 % fert.	NT	101 % norm. dev.	NT	T	Long 1997
0.0307	0.0101	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0316	0.0050	Biscayne Bay, FL	61 % mort.	T	71 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0346	0.0129	Biscayne Bay, FL	65 % mort.	T	102 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0402	0.0028	Biscayne Bay, FL	90 % mort.	T	36 % fert.	T	74 % norm. dev.	T	T	Long 1997
0.0486	0.0081	Biscayne Bay, FL	81 % mort.	T	104 % fert.	NT	13 % norm. dev.	T	T	Long 1997
0.0746	0.0088	Biscayne Bay, FL	95 % mort.	T	96 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0903	0.0102	Biscayne Bay, FL	91 % mort.	T	88 % fert.	NT	3 % norm. dev.	T	T	Long 1997
0.1037	0.0115	Biscayne Bay, FL	92 % mort.	T	14 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0001		Narragansett Bay, RI	2.7 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	6 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	2 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	10 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0004		Narragansett Bay, RI	3.3 % mort.	NT					NT	Munns et al. 1991
0.0006		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0006	<	Narragansett Bay, RI	11.3 % mort.	NT					NT	Munns et al. 1991
0.0006		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0007		Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0012		Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0013		Narragansett Bay, RI	6.7 % mort.	NT					NT	Munns et al. 1991

Appendix A5-2. A summary of the available information on the toxic effects of sediment-associated SUM DDE from locations outside the Southern California Bight.

Concentration			Endpoint Measured				
			Amphipod	Sea Urchin		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Toxicity	Reference
0.0015		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0018		Narragansett Bay, RI	8.7 % mort.	NT		NT	Munns et al. 1991
0.0023		Narragansett Bay, RI	1.3 % mort.	NT		NT	Munns et al. 1991
0.0031		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0037		Narragansett Bay, RI	3.3 % mort.	NT		NT	Munns et al. 1991
0.0037		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0048		Narragansett Bay, RI	6.7 % mort.	NT		NT	Munns et al. 1991
0.0050		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0059		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0060		Narragansett Bay, RI	0 % mort.	NT		NT	Munns et al. 1991
0.0069		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0072		Narragansett Bay, RI	2.7 % mort.	NT		NT	Munns et al. 1991
0.0131		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0164		Narragansett Bay, RI	10.7 % mort.	NT		NT	Munns et al. 1991
0.0358		Narragansett Bay, RI	8 % mort.	NT		NT	Munns et al. 1991
0.0425		Narragansett Bay, RI	6 % mort.	NT		NT	Munns et al. 1991
0.0020	0.0023	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.0110	0.0093	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.0290	0.0269	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
0.0490	0.0329	San Francisco Bay, CA	28 % mort.	NT		NT	Swartz et al. 1994
0.1230	0.0842	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
0.6150	0.2064	San Francisco Bay, CA	32 % mort.	T		T	Swartz et al. 1994
0.6720	0.3884	San Francisco Bay, CA	36 % mort.	T		T	Swartz et al. 1994
0.9040	0.5079	San Francisco Bay, CA	43 % mort.	T		T	Swartz et al. 1994
1.64	0.6895	San Francisco Bay, CA	66.2 % mort.	T		T	Swartz et al. 1994

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.

Endpoint Measured: mort. = mortality; fert. = fertilization; norm. dev. = normal development.

NT = toxic; T = toxic.

Species tested included:

Long et al. 1994; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Long et al. 1995b; Ampelisca abdita (amphipod).

Long 1997; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Munns et al. 1991; Ampelisca abdita (amphipod).

Swartz et al. 1994; Eohaustorius estuarius (amphipod).

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Sea Urchin Fertilization		Sea Urchin Development	Overall Toxicity	Reference
0.0011	0.0005	Tampa Bay, FL	15 % mort.	NT	78.4 % fert.	NT		NT	Long et al. 1994
0.0014	0.0007	Tampa Bay, FL	6 % mort.	NT	3.4 % fert.	T		T	Long et al. 1994
0.0015	0.0010	Tampa Bay, FL	14 % mort.	NT	19.2 % fert.	T		T	Long et al. 1994
0.0016	0.0116	Tampa Bay, FL	11 % mort.	NT	85.2 % fert.	NT		NT	Long et al. 1994
0.0016	0.0046	Tampa Bay, FL	11 % mort.	NT	25 % fert.	T		T	Long et al. 1994
0.0016	0.0181	Tampa Bay, FL	16 % mort.	NT	61.6 % fert.	T		T	Long et al. 1994
0.0016	0.0081	Tampa Bay, FL	20 % mort.	NT	86.4 % fert.	NT		NT	Long et al. 1994
0.0016	0.0090	Tampa Bay, FL	23 % mort.	NT	9.6 % fert.	T		T	Long et al. 1994
0.0017	0.0004	Tampa Bay, FL	12 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994
0.0017	0.0056	Tampa Bay, FL	9 % mort.	NT	77.6 % fert.	NT		NT	Long et al. 1994
0.0018	0.0007	Tampa Bay, FL	5 % mort.	NT	84.2 % fert.	NT		NT	Long et al. 1994
0.0018	0.0004	Tampa Bay, FL	5 % mort.	NT	0.2 % fert.	T		T	Long et al. 1994
0.0021	0.0008	Tampa Bay, FL	4 % mort.	NT	22.6 % fert.	T		T	Long et al. 1994
0.0021	0.0012	Tampa Bay, FL	6 % mort.	NT	46 % fert.	T		T	Long et al. 1994
0.0021	0.0012	Tampa Bay, FL	7 % mort.	NT	91.6 % fert.	NT		NT	Long et al. 1994
0.0021	0.0007	Tampa Bay, FL	7 % mort.	NT	71.4 % fert.	T		T	Long et al. 1994
0.0021	0.0008	Tampa Bay, FL	7 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0021	0.0060	Tampa Bay, FL	13 % mort.	NT	46.2 % fert.	T		T	Long et al. 1994
0.0021	0.0024	Tampa Bay, FL	15 % mort.	NT	82.8 % fert.	NT		NT	Long et al. 1994
0.0021	0.0045	Tampa Bay, FL	15 % mort.	NT	88.4 % fert.	NT		NT	Long et al. 1994
0.0021	0.0111	Tampa Bay, FL	19 % mort.	NT	91.6 % fert.	NT		NT	Long et al. 1994
0.0022	0.0089	Tampa Bay, FL	13 % mort.	NT	84.4 % fert.	NT		NT	Long et al. 1994
0.0024	0.0013	Tampa Bay, FL	5 % mort.	NT	72.4 % fert.	T		T	Long et al. 1994
0.0027	0.0008	Tampa Bay, FL	19 % mort.	NT	43.8 % fert.	T		T	Long et al. 1994
0.0028	0.0015	Tampa Bay, FL	10 % mort.	NT	3 % fert.	T		T	Long et al. 1994
0.0030	0.0008	Tampa Bay, FL	19 % mort.	NT	43 % fert.	T		T	Long et al. 1994
0.0030	0.0014	Tampa Bay, FL	12 % mort.	NT	16 % fert.	T		T	Long et al. 1994
0.0030	0.0028	Tampa Bay, FL	17 % mort.	NT	17.8 % fert.	T		T	Long et al. 1994
0.0031	0.0033	Tampa Bay, FL	13 % mort.	NT	42.8 % fert.	T		T	Long et al. 1994
0.0045	0.0035	Tampa Bay, FL	16 % mort.	NT	0.2 % fert.	T		T	Long et al. 1994
0.0045	0.0044	Tampa Bay, FL	18 % mort.	NT	86.4 % fert.	NT		NT	Long et al. 1994
0.0053	0.0023	Tampa Bay, FL	10 % mort.	NT	27.2 % fert.	T		T	Long et al. 1994
0.0059	0.0020	Tampa Bay, FL	1 % mort.	NT	27.4 % fert.	T		T	Long et al. 1994
0.0069	0.0058	Tampa Bay, FL	18 % mort.	NT	7.6 % fert.	T		T	Long et al. 1994
0.0078	0.0017	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T		T	Long et al. 1994
0.0081	0.0031	Tampa Bay, FL	10 % mort.	NT	5.6 % fert.	T		T	Long et al. 1994
0.0088	0.0034	Tampa Bay, FL	10 % mort.	NT	5.8 % fert.	T		T	Long et al. 1994
0.0091	0.0031	Tampa Bay, FL	10 % mort.	NT	3.2 % fert.	T		T	Long et al. 1994

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin			Overall		
DW	DW @1%OC	Area	Mortality		Fertilization		Development	Toxicity	Reference	
0.0092	0.0026	Tampa Bay, FL	17.5 % mort.	T	0.6 % fert.	T		T	Long et al. 1994	
0.0095	0.0033	Tampa Bay, FL	8 % mort.	NT	4 % fert.	T		T	Long et al. 1994	
0.0100	0.0019	Tampa Bay, FL	14 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0101	0.0025	Tampa Bay, FL	16 % mort.	NT	1 % fert.	T		T	Long et al. 1994	
0.0105	0.0042	Tampa Bay, FL	6 % mort.	NT	0.4 % fert.	T		T	Long et al. 1994	
0.0116	0.0070	Tampa Bay, FL	22 % mort.	NT	0.2 % fert.	T		T	Long et al. 1994	
0.0130	0.0082	Tampa Bay, FL	22 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0131	0.0036	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0148	0.0063	Tampa Bay, FL	18 % mort.	NT	74 % fert.	T		T	Long et al. 1994	
0.0168	0.0043	Tampa Bay, FL	9 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0175	0.0119	Tampa Bay, FL	15 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0196	0.0084	Tampa Bay, FL	17 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0228	0.0037	Tampa Bay, FL	17 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994	
0.0265	0.0057	Tampa Bay, FL	11 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0303	0.0085	Tampa Bay, FL	13 % mort.	NT	0 % fert.	T		T	Long et al. 1994	
0.0327	0.0195	Tampa Bay, FL	24 % mort.	T	11 % fert.	T		T	Long et al. 1994	
0.0424	0.0129	Tampa Bay, FL	27 % mort.	T	0 % fert.	T		T	Long et al. 1994	
0.0457	0.0073	Tampa Bay, FL	22.5 % mort.	T	0 % fert.	T		T	Long et al. 1994	
0.0481	0.0079	Tampa Bay, FL	15 % mort.	NT	0.6 % fert.	T		T	Long et al. 1994	
0.0991	0.0350	Tampa Bay, FL	55 % mort.	T	0 % fert.	T		T	Long et al. 1994	
0.1142	0.0347	Tampa Bay, FL	23 % mort.	T	0 % fert.	T		T	Long et al. 1994	
0.3356	0.0766	Tampa Bay, FL	61 % mort.	T	0 % fert.	T		T	Long et al. 1994	
1.86	0.6255	Tampa Bay, FL	52 % mort.	T	0 % fert.	T		T	Long et al. 1994	
0.0004	0.0057	Hudson-Raritan Estuary	9 % mort.	NT				NT	Long et al. 1995b	
0.0005	0.0001	Hudson-Raritan Estuary	100 % mort.	T				T	Long et al. 1995b	
0.0010	0.0040	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b	
0.0030	0.0011	Hudson-Raritan Estuary	81 % mort.	T				T	Long et al. 1995b	
0.0030	0.0018	Hudson-Raritan Estuary	4 % mort.	NT				NT	Long et al. 1995b	
0.0040	0.0085	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b	
0.0060	0.0021	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b	
0.0060	0.0060	Hudson-Raritan Estuary	97 % mort.	T				T	Long et al. 1995b	
0.0080	0.0030	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b	
0.0090	0.0032	Hudson-Raritan Estuary	12 % mort.	NT				NT	Long et al. 1995b	
0.0090	0.0046	Hudson-Raritan Estuary	7 % mort.	T				T	Long et al. 1995b	
0.0090	0.0020	Hudson-Raritan Estuary	100 % mort.	T				T	Long et al. 1995b	
0.0100	0.0042	Hudson-Raritan Estuary	28 % mort.	T				T	Long et al. 1995b	
0.0110	0.0036	Hudson-Raritan Estuary	0 % mort.	NT				NT	Long et al. 1995b	

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.0120	0.0039	Hudson-Raritan Estuary	49 % mort.	T					T	Long et al. 1995b
0.0130	0.0040	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0140	0.0044	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0160	0.0064	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0160	0.0041	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0160	0.0053	Hudson-Raritan Estuary	0 % mort.	NT					NT	Long et al. 1995b
0.0190	0.0076	Hudson-Raritan Estuary	19 % mort.	T					T	Long et al. 1995b
0.0200	0.0211	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b
0.0200	0.0133	Hudson-Raritan Estuary	82 % mort.	T					T	Long et al. 1995b
0.0210	0.0047	Hudson-Raritan Estuary	82 % mort.	T					T	Long et al. 1995b
0.0210	0.0066	Hudson-Raritan Estuary	6 % mort.	NT					NT	Long et al. 1995b
0.0220	0.0086	Hudson-Raritan Estuary	14 % mort.	NT					NT	Long et al. 1995b
0.0220	0.0129	Hudson-Raritan Estuary	21 % mort.	T					T	Long et al. 1995b
0.0260	0.0087	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b
0.0270	0.0123	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b
0.0310	0.0072	Hudson-Raritan Estuary	23 % mort.	NT					NT	Long et al. 1995b
0.0310	0.0148	Hudson-Raritan Estuary	47 % mort.	T					T	Long et al. 1995b
0.0310	0.0129	Hudson-Raritan Estuary	32 % mort.	T					T	Long et al. 1995b
0.0350	0.0175	Hudson-Raritan Estuary	24 % mort.	T					T	Long et al. 1995b
0.0360	0.0182	Hudson-Raritan Estuary	100 % mort.	T					T	Long et al. 1995b
0.0380	0.0152	Hudson-Raritan Estuary	17 % mort.	NT					NT	Long et al. 1995b
0.0400	0.0125	Hudson-Raritan Estuary	86 % mort.	T					T	Long et al. 1995b
0.0410	0.0532	Hudson-Raritan Estuary	9 % mort.	NT					NT	Long et al. 1995b
0.0420	0.0116	Hudson-Raritan Estuary	24 % mort.	T					T	Long et al. 1995b
0.0430	0.0108	Hudson-Raritan Estuary	23 % mort.	T					T	Long et al. 1995b
0.0780	0.0186	Hudson-Raritan Estuary	80 % mort.	T					T	Long et al. 1995b
0.0790	0.0155	Hudson-Raritan Estuary	48 % mort.	T					T	Long et al. 1995b
0.0890	0.0371	Hudson-Raritan Estuary	67 % mort.	T					T	Long et al. 1995b
0.0910	0.0379	Hudson-Raritan Estuary	85 % mort.	T					T	Long et al. 1995b
0.0940	0.0162	Hudson-Raritan Estuary	75 % mort.	T					T	Long et al. 1995b
0.0980	0.0408	Hudson-Raritan Estuary	81 % mort.	T					T	Long et al. 1995b
0.1080	0.0470	Hudson-Raritan Estuary	91 % mort.	T					T	Long et al. 1995b
0.1100	0.0220	Hudson-Raritan Estuary	69 % mort.	T					T	Long et al. 1995b
0.1150	0.0189	Hudson-Raritan Estuary	71 % mort.	T					T	Long et al. 1995b
0.1240	0.0259	Hudson-Raritan Estuary	98 % mort.	T					T	Long et al. 1995b
0.2760	0.1878	Hudson-Raritan Estuary	63 % mort.	T					T	Long et al. 1995b
0.00003	0.0001	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997
0.00005	0.0002	Biscayne Bay, FL	10 % mort.	NT	81 % fert.	NT	56 % norm. dev.	T	T	Long 1997

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin					Overall		
DW	DW @1%OC	Area	Mortality	Fertilization		Development			Toxicity	Reference	
0.00005	0.0001	Biscayne Bay, FL	35 % mort.	T	110 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	89 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	91 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	91 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	19 % mort.	NT	110 % fert.	NT	3 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	9 % mort.	NT	96 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	11 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0004	Biscayne Bay, FL	9 % mort.	NT	72 % fert.	T	91 % norm. dev.	NT	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	69 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0005	Biscayne Bay, FL	0 % mort.	NT	96 % fert.	NT	1 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	0 % mort.	NT	117 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	0 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0000	Biscayne Bay, FL	0 % mort.	NT	112 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0004	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	1 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	2 % mort.	NT	68 % fert.	T	82 % norm. dev.	NT	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0003	Biscayne Bay, FL	2 % mort.	NT	111 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0007	Biscayne Bay, FL	3 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0000	Biscayne Bay, FL	4 % mort.	NT	98 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	109 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	4 % mort.	NT	114 % fert.	NT	62 % norm. dev.	T	T	Long 1997	
0.0001	0.0000	Biscayne Bay, FL	4 % mort.	NT	80.05 % fert.	NT	85 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	5 % mort.	NT	0 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0003	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	6 % mort.	NT	1 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0000	Biscayne Bay, FL	7 % mort.	NT	1 % fert.	T	2 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	7 % mort.	NT	110 % fert.	NT	53 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	8 % mort.	NT	108 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	9 % mort.	NT	95 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0009	Biscayne Bay, FL	11 % mort.	NT	95 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	12 % mort.	NT	105 % fert.	NT	105 % norm. dev.	NT	NT	Long 1997	
0.0001	0.0008	Biscayne Bay, FL	18 % mort.	NT	99 % fert.	NT	1 % norm. dev.	T	T	Long 1997	
0.0001	0.0003	Biscayne Bay, FL	18 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0006	Biscayne Bay, FL	18 % mort.	NT	59 % fert.	T	64 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	23 % mort.	T	91 % fert.	NT	1 % norm. dev.	T	T	Long 1997	

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod		Sea Urchin				Overall		
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference	
0.0001	0.0004	Biscayne Bay, FL	43 % mort.	T	34 % fert.	T	13 % norm. dev.	T	T	Long 1997	
0.0001	0.0001	Biscayne Bay, FL	56 % mort.	T	0 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0001	0.0002	Biscayne Bay, FL	4 % mort.	NT	78 % fert.	T	103 % norm. dev.	NT	T	Long 1997	
0.0001	0.0003	Biscayne Bay, FL	19 % mort.	NT	115 % fert.	NT	45 % norm. dev.	T	T	Long 1997	
0.0001	0.0003	Biscayne Bay, FL	5 % mort.	NT	117 % fert.	NT	2 % norm. dev.	T	T	Long 1997	
0.0002	0.0001	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	98 % norm. dev.	NT	NT	Long 1997	
0.0002	0.0001	Biscayne Bay, FL	2 % mort.	NT	62 % fert.	T	86 % norm. dev.	NT	T	Long 1997	
0.0002	0.0002	Biscayne Bay, FL	0 % mort.	NT	83 % fert.	NT	96 % norm. dev.	NT	NT	Long 1997	
0.0002	0.0001	Biscayne Bay, FL	3 % mort.	NT	113 % fert.	NT	63 % norm. dev.	T	T	Long 1997	
0.0002	0.0001	Biscayne Bay, FL	2 % mort.	NT	101 % fert.	NT	93.9 % norm. dev.	NT	NT	Long 1997	
0.0002	0.0001	Biscayne Bay, FL	1 % mort.	NT	97 % fert.	NT	104 % norm. dev.	NT	NT	Long 1997	
0.0002	0.0000	Biscayne Bay, FL	13 % mort.	NT	100 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997	
0.0003	0.0006	Biscayne Bay, FL	6 % mort.	NT	114 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0003	0.0002	Biscayne Bay, FL	11 % mort.	NT	94 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997	
0.0003	0.0004	Biscayne Bay, FL	17 % mort.	NT	115 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0003	0.0014	Biscayne Bay, FL	4 % mort.	NT	106 % fert.	NT	1 % norm. dev.	T	T	Long 1997	
0.0003	0.0007	Biscayne Bay, FL	1 % mort.	NT	96 % fert.	NT	99 % norm. dev.	NT	NT	Long 1997	
0.0004	0.0006	Biscayne Bay, FL	2 % mort.	NT	97 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997	
0.0004	0.0002	Biscayne Bay, FL	2 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0004	0.0003	Biscayne Bay, FL	5 % mort.	NT	99 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997	
0.0004	0.0003	Biscayne Bay, FL	8 % mort.	NT	66 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0004	0.0001	Biscayne Bay, FL	0 % mort.	NT	7 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0004	0.0003	Biscayne Bay, FL	0 % mort.	NT	47 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0005	0.0004	Biscayne Bay, FL	7 % mort.	NT	82 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997	
0.0005	0.0004	Biscayne Bay, FL	1 % mort.	NT	95 % fert.	NT	101 % norm. dev.	NT	NT	Long 1997	
0.0005	0.0003	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0006	0.0001	Biscayne Bay, FL	0 % mort.	NT	67 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0006	0.0001	Biscayne Bay, FL	7 % mort.	NT	92 % fert.	NT	0 % norm. dev.	T	T	Long 1997	
0.0007	0.0003	Biscayne Bay, FL	4 % mort.	NT	92 % fert.	NT	100 % norm. dev.	NT	NT	Long 1997	
0.0008	0.0008	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	18 % norm. dev.	T	T	Long 1997	
0.0008	0.0004	Biscayne Bay, FL	4 % mort.	NT	112 % fert.	NT	10 % norm. dev.	T	T	Long 1997	
0.0009	0.0004	Biscayne Bay, FL	1 % mort.	NT	80.4 % fert.	NT	89 % norm. dev.	NT	NT	Long 1997	
0.0009	0.0005	Biscayne Bay, FL	0 % mort.	NT	94 % fert.	NT	90 % norm. dev.	NT	NT	Long 1997	
0.0009	0.0007	Biscayne Bay, FL	0 % mort.	NT	75 % fert.	T	1 % norm. dev.	T	T	Long 1997	
0.0010	0.0002	Biscayne Bay, FL	3 % mort.	NT	62 % fert.	T	0 % norm. dev.	T	T	Long 1997	
0.0011	0.0022	Biscayne Bay, FL	0 % mort.	NT	86 % fert.	NT	12 % norm. dev.	T	T	Long 1997	
0.0014	0.0011	Biscayne Bay, FL	6 % mort.	NT	94 % fert.	NT	95 % norm. dev.	NT	NT	Long 1997	
0.0015	0.0002	Biscayne Bay, FL	5 % mort.	NT	88 % fert.	NT	0 % norm. dev.	T	T	Long 1997	

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured							
			Amphipod		Sea Urchin				Overall	
DW	DW @1%OC	Area	Mortality		Fertilization		Development		Toxicity	Reference
0.0016	0.0005	Biscayne Bay, FL	0 % mort.	NT	19 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0017	0.0003	Biscayne Bay, FL	0 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0020	0.0003	Biscayne Bay, FL	9 % mort.	NT	2 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0021	0.0007	Biscayne Bay, FL	2 % mort.	NT	91 % fert.	NT	97 % norm. dev.	NT	NT	Long 1997
0.0031	0.0010	Biscayne Bay, FL	31 % mort.	T	37 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0034	0.0024	Biscayne Bay, FL	59 % mort.	T	92 % fert.	NT	1 % norm. dev.	T	T	Long 1997
0.0034	0.0010	Biscayne Bay, FL	5 % mort.	NT	98 % fert.	NT	75 % norm. dev.	T	T	Long 1997
0.0056	0.0015	Biscayne Bay, FL	6 % mort.	NT	99 % fert.	NT	103 % norm. dev.	NT	NT	Long 1997
0.0060	0.0016	Biscayne Bay, FL	59 % mort.	T	99 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0067	0.0018	Biscayne Bay, FL	9 % mort.	NT	83 % fert.	NT	102 % norm. dev.	NT	NT	Long 1997
0.0077	0.0045	Biscayne Bay, FL	84 % mort.	T	80.4 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0096	0.0041	Biscayne Bay, FL	68 % mort.	T	95 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.0108	0.0018	Biscayne Bay, FL	81 % mort.	T	104 % fert.	NT	13 % norm. dev.	T	T	Long 1997
0.0121	0.0051	Biscayne Bay, FL	98 % mort.	T	98 % fert.	NT	97 % norm. dev.	NT	T	Long 1997
0.0131	0.0049	Biscayne Bay, FL	65 % mort.	T	102 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0170	0.0370	Biscayne Bay, FL	69 % mort.	T	91 % fert.	NT	96 % norm. dev.	NT	T	Long 1997
0.0192	0.0069	Biscayne Bay, FL	91 % mort.	T	98 % fert.	NT	78 % norm. dev.	T	T	Long 1997
0.0208	0.0022	Biscayne Bay, FL	61 % mort.	T	100 % fert.	NT	79.8 % norm. dev.	T	T	Long 1997
0.0282	0.0072	Biscayne Bay, FL	49 % mort.	T	48 % fert.	T	3 % norm. dev.	T	T	Long 1997
0.0288	0.0020	Biscayne Bay, FL	90 % mort.	T	36 % fert.	T	74 % norm. dev.	T	T	Long 1997
0.0402	0.0096	Biscayne Bay, FL	59 % mort.	T	93 % fert.	NT	101 % norm. dev.	NT	T	Long 1997
0.0413	0.0066	Biscayne Bay, FL	61 % mort.	T	71 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.0503	0.0193	Biscayne Bay, FL	33 % mort.	T	93 % fert.	NT	2 % norm. dev.	T	T	Long 1997
0.1012	0.0119	Biscayne Bay, FL	95 % mort.	T	96 % fert.	NT	0 % norm. dev.	T	T	Long 1997
0.1182	0.0134	Biscayne Bay, FL	91 % mort.	T	88 % fert.	NT	3 % norm. dev.	T	T	Long 1997
0.1495	0.0166	Biscayne Bay, FL	92 % mort.	T	14 % fert.	T	0 % norm. dev.	T	T	Long 1997
0.2920	0.0960	Biscayne Bay, FL	6 % mort.	NT	95 % fert.	NT	94 % norm. dev.	NT	NT	Long 1997
0.0002		Narragansett Bay, RI	3.3 % mort.	NT					NT	Munns et al. 1991
0.0002		Narragansett Bay, RI	10 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	4 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	2.7 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	6 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	2 % mort.	NT					NT	Munns et al. 1991
0.0003	<	Narragansett Bay, RI	11.3 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0003		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991
0.0005		Narragansett Bay, RI	4.7 % mort.	NT					NT	Munns et al. 1991

Appendix A5-3. A summary of the available information on the toxic effects of sediment-associated SUM DDD from locations outside the Southern California Bight.

Concentration			Endpoint Measured					
			Amphipod	Sea Urchin		Overall		
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Toxicity	Reference
0.0006		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	6.7 % mort.	NT		NT	Munns et al. 1991
0.0012		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0015		Narragansett Bay, RI	3.3 % mort.	NT		NT	Munns et al. 1991
0.0016		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0019		Narragansett Bay, RI	6.7 % mort.	NT		NT	Munns et al. 1991
0.0020		Narragansett Bay, RI	1.3 % mort.	NT		NT	Munns et al. 1991
0.0021		Narragansett Bay, RI	8.7 % mort.	NT		NT	Munns et al. 1991
0.0028		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0032		Narragansett Bay, RI	0 % mort.	NT		NT	Munns et al. 1991
0.0038		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0047		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0049		Narragansett Bay, RI	2.7 % mort.	NT		NT	Munns et al. 1991
0.0058		Narragansett Bay, RI	2 % mort.	NT		NT	Munns et al. 1991
0.0074		Narragansett Bay, RI	6 % mort.	NT		NT	Munns et al. 1991
0.0241		Narragansett Bay, RI	10.7 % mort.	NT		NT	Munns et al. 1991
0.0263		Narragansett Bay, RI	4.7 % mort.	NT		NT	Munns et al. 1991
0.0270		Narragansett Bay, RI	8 % mort.	NT		NT	Munns et al. 1991
0.0306		Narragansett Bay, RI	4 % mort.	NT		NT	Munns et al. 1991
0.0060	0.0069	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.0540	0.0458	San Francisco Bay, CA	24 % mort.	NT		NT	Swartz et al. 1994
0.2550	0.2361	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
0.2810	0.1886	San Francisco Bay, CA	28 % mort.	NT		NT	Swartz et al. 1994
1.19	0.3997	San Francisco Bay, CA	32 % mort.	T		T	Swartz et al. 1994
1.74	1.19	San Francisco Bay, CA	23 % mort.	NT		NT	Swartz et al. 1994
17.8	10.3	San Francisco Bay, CA	36 % mort.	T		T	Swartz et al. 1994
18.3	10.3	San Francisco Bay, CA	43 % mort.	T		T	Swartz et al. 1994
39.1	16.4	San Francisco Bay, CA	66.2 % mort.	T		T	Swartz et al. 1994

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.
Endpoint Measured: mort. = mortality; fert. = fertilization; norm. dev. = normal development.
NT = toxic; T = toxic.
Species tested included:

Long et al. 1994; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).
Long et al. 1995b; Ampelisca abdita (amphipod).
Long 1997; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).
Munns et al. 1991; Ampelisca abdita (amphipod).
Swartz et al. 1994; Eohaustorius estuarius (amphipod).

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured										Overall	
			Amphipod	Sea Urchin		Sand Dollar			Polychaete					
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development		Mortality		Mortality	Growth		Toxicity	Reference
0.0020		Puget Sound, WA				0.9 mm inc. diameter	NT	0 % mort.	NT				NT	Casillas et al. 1992
0.0020		Puget Sound, WA				1.5 mm inc. diameter	NT	0 % mort.	NT				NT	Casillas et al. 1992
0.0020		Puget Sound, WA				2.4 mm inc. diameter	NT	0 % mort.	NT				NT	Casillas et al. 1992
0.0040	0.0013	Puget Sound, WA				0.5 mm inc. diameter	NT	62 % mort.	T				T	Casillas et al. 1992
0.0050	0.0024	Puget Sound, WA				0.5 mm inc. diameter	NT	52 % mort.	T				T	Casillas et al. 1992
0.0050	0.0041	Puget Sound, WA				1.9 mm inc. diameter	T	5 % mort.	NT				T	Casillas et al. 1992
0.0060	0.0031	Puget Sound, WA				0.2 mm inc. diameter	NT	88 % mort.	T				T	Casillas et al. 1992
0.0070	0.0013	Puget Sound, WA				2.3 mm inc. diameter	NT	0 % mort.	NT				NT	Casillas et al. 1992
0.0070	0.0044	Puget Sound, WA				2 mm inc. diameter	T	2 % mort.	NT				T	Casillas et al. 1992
0.0080	0.0033	Puget Sound, WA				0 mm inc. diameter	T	100 % mort.	T				T	Casillas et al. 1992
0.0080	0.0100	Puget Sound, WA				0.9 mm inc. diameter	T	1 % mort.	NT				T	Casillas et al. 1992
0.0110	0.0014	Puget Sound, WA				1.2 mm inc. diameter	NT	2 % mort.	NT				NT	Casillas et al. 1992
0.0270	0.0024	Puget Sound, WA				1.5 mm inc. diameter	T	0 % mort.	NT				T	Casillas et al. 1992
0.0450	0.0062	Puget Sound, WA				0.2 mm inc. diameter	T	3 % mort.	NT				T	Casillas et al. 1992
0.0540	0.0265	Puget Sound, WA				0.2 mm inc. diameter	T	15 % mort.	T				T	Casillas et al. 1992
0.0004	0.0007	San Francisco Bay, CA	9 % mort.	NT									NT	Chapman et al. 1987
0.0007	0.0005	San Francisco Bay, CA	24 % mort.	T									T	Chapman et al. 1987
0.0008	0.0006	San Francisco Bay, CA	13 % mort.	NT									NT	Chapman et al. 1987
0.0008	0.0006	San Francisco Bay, CA	4 % mort.	NT									NT	Chapman et al. 1987
0.0011	0.0010	San Francisco Bay, CA	13 % mort.	NT									NT	Chapman et al. 1987
0.0015	0.0012	San Francisco Bay, CA	9 % mort.	NT									NT	Chapman et al. 1987
0.0022	0.0016	San Francisco Bay, CA	37 % mort.	T									T	Chapman et al. 1987
0.0029	0.0007	San Francisco Bay, CA	95 % mort.	T									T	Chapman et al. 1987
0.0036	0.0011	San Francisco Bay, CA	24 % mort.	T									T	Chapman et al. 1987
0.0024	0.0118	Tampa Bay, FL	20 % mort.	NT	86.4 % fert.	NT							NT	Long et al. 1994
0.0024	0.0169	Tampa Bay, FL	11 % mort.	NT	85.2 % fert.	NT							NT	Long et al. 1994
0.0024	0.0010	Tampa Bay, FL	15 % mort.	NT	78.4 % fert.	NT							NT	Long et al. 1994
0.0026	0.0147	Tampa Bay, FL	23 % mort.	NT	9.6 % fert.	T							T	Long et al. 1994
0.0028	0.0311	Tampa Bay, FL	16 % mort.	NT	61.6 % fert.	T							T	Long et al. 1994
0.0030	0.0085	Tampa Bay, FL	11 % mort.	NT	25 % fert.	T							T	Long et al. 1994
0.0035	0.0039	Tampa Bay, FL	15 % mort.	NT	82.8 % fert.	NT							NT	Long et al. 1994
0.0037	0.0020	Tampa Bay, FL	5 % mort.	NT	72.4 % fert.	T							T	Long et al. 1994
0.0041	0.0217	Tampa Bay, FL	19 % mort.	NT	91.6 % fert.	NT							NT	Long et al. 1994
0.0041	0.0017	Tampa Bay, FL	5 % mort.	NT	84.2 % fert.	NT							NT	Long et al. 1994
0.0043	0.0025	Tampa Bay, FL	6 % mort.	NT	46 % fert.	T							T	Long et al. 1994
0.0046	0.0023	Tampa Bay, FL	6 % mort.	NT	3.4 % fert.	T							T	Long et al. 1994

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.0050	0.0142	Tampa Bay, FL	13 % mort. NT	46.2 % fert. T						T	Long et al. 1994
0.0053	0.0031	Tampa Bay, FL	7 % mort. NT	91.6 % fert. NT						NT	Long et al. 1994
0.0053	0.0172	Tampa Bay, FL	9 % mort. NT	77.6 % fert. NT						NT	Long et al. 1994
0.0054	0.0115	Tampa Bay, FL	15 % mort. NT	88.4 % fert. NT						NT	Long et al. 1994
0.0064	0.0023	Tampa Bay, FL	4 % mort. NT	22.6 % fert. T						T	Long et al. 1994
0.0068	0.0035	Tampa Bay, FL	10 % mort. NT	3 % fert. T						T	Long et al. 1994
0.0072	0.0047	Tampa Bay, FL	14 % mort. NT	19.2 % fert. T						T	Long et al. 1994
0.0073	0.0016	Tampa Bay, FL	5 % mort. NT	0.2 % fert. T						T	Long et al. 1994
0.0077	0.0028	Tampa Bay, FL	7 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0079	0.0037	Tampa Bay, FL	12 % mort. NT	16 % fert. T						T	Long et al. 1994
0.0080	0.0018	Tampa Bay, FL	12 % mort. NT	0.6 % fert. T						T	Long et al. 1994
0.0084	0.0065	Tampa Bay, FL	16 % mort. NT	0.2 % fert. T						T	Long et al. 1994
0.0085	0.0030	Tampa Bay, FL	7 % mort. NT	71.4 % fert. T						T	Long et al. 1994
0.0100	0.0031	Tampa Bay, FL	19 % mort. NT	43.8 % fert. T						T	Long et al. 1994
0.0101	0.0098	Tampa Bay, FL	18 % mort. NT	86.4 % fert. NT						NT	Long et al. 1994
0.0113	0.0038	Tampa Bay, FL	1 % mort. NT	27.4 % fert. T						T	Long et al. 1994
0.0136	0.0037	Tampa Bay, FL	19 % mort. NT	43 % fert. T						T	Long et al. 1994
0.0158	0.0133	Tampa Bay, FL	18 % mort. NT	7.6 % fert. T						T	Long et al. 1994
0.0166	0.0664	Tampa Bay, FL	13 % mort. NT	84.4 % fert. NT						NT	Long et al. 1994
0.0171	0.0049	Tampa Bay, FL	17.5 % mort. T	0.6 % fert. T						T	Long et al. 1994
0.0224	0.0136	Tampa Bay, FL	22 % mort. NT	0.2 % fert. T						T	Long et al. 1994
0.0251	0.0158	Tampa Bay, FL	22 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0255	0.0109	Tampa Bay, FL	10 % mort. NT	27.2 % fert. T						T	Long et al. 1994
0.0339	0.0314	Tampa Bay, FL	17 % mort. NT	17.8 % fert. T						T	Long et al. 1994
0.0358	0.0078	Tampa Bay, FL	11 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0395	0.0151	Tampa Bay, FL	10 % mort. NT	5.6 % fert. T						T	Long et al. 1994
0.0410	0.0077	Tampa Bay, FL	14 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0410	0.0279	Tampa Bay, FL	15 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0425	0.0447	Tampa Bay, FL	13 % mort. NT	42.8 % fert. T						T	Long et al. 1994
0.0473	0.0120	Tampa Bay, FL	9 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0545	0.0188	Tampa Bay, FL	10 % mort. NT	3.2 % fert. T						T	Long et al. 1994
0.0572	0.0143	Tampa Bay, FL	16 % mort. NT	1 % fert. T						T	Long et al. 1994
0.0598	0.0229	Tampa Bay, FL	10 % mort. NT	5.8 % fert. T						T	Long et al. 1994
0.0689	0.0272	Tampa Bay, FL	6 % mort. NT	0.4 % fert. T						T	Long et al. 1994
0.0689	0.0209	Tampa Bay, FL	27 % mort. T	0 % fert. T						T	Long et al. 1994
0.0844	0.0233	Tampa Bay, FL	9 % mort. NT	0 % fert. T						T	Long et al. 1994
0.0915	0.0256	Tampa Bay, FL	13 % mort. NT	0 % fert. T						T	Long et al. 1994
0.1075	0.0640	Tampa Bay, FL	24 % mort. T	11 % fert. T						T	Long et al. 1994

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.1094	0.0373	Tampa Bay, FL	8 % mort. NT	4 % fert. T						T	Long et al. 1994
0.1158	0.0491	Tampa Bay, FL	18 % mort. NT	74 % fert. T						T	Long et al. 1994
0.1312	0.0561	Tampa Bay, FL	17 % mort. NT	0 % fert. T						T	Long et al. 1994
0.1519	0.0247	Tampa Bay, FL	17 % mort. NT	0.6 % fert. T						T	Long et al. 1994
0.1638	0.0269	Tampa Bay, FL	15 % mort. NT	0.6 % fert. T						T	Long et al. 1994
0.1786	0.0382	Tampa Bay, FL	11 % mort. NT	0 % fert. T						T	Long et al. 1994
0.1965	0.0312	Tampa Bay, FL	22.5 % mort. T	0 % fert. T						T	Long et al. 1994
0.2173	0.0768	Tampa Bay, FL	55 % mort. T	0 % fert. T						T	Long et al. 1994
0.2202	0.0669	Tampa Bay, FL	23 % mort. T	0 % fert. T						T	Long et al. 1994
0.6647	0.1517	Tampa Bay, FL	61 % mort. T	0 % fert. T						T	Long et al. 1994
5.13	1.7282	Tampa Bay, FL	52 % mort. T	0 % fert. T						T	Long et al. 1994
0.0017	0.0243	Hudson-Raritan Estuary	9 % mort. NT							NT	Long et al. 1995b
0.0030	0.0120	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0090	0.0034	Hudson-Raritan Estuary	81 % mort. T							T	Long et al. 1995b
0.0090	0.0053	Hudson-Raritan Estuary	4 % mort. NT							NT	Long et al. 1995b
0.0100	0.0020	Hudson-Raritan Estuary	100 % mort. T							T	Long et al. 1995b
0.0120	0.0255	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0150	0.0052	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0224	0.0072	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0240	0.0090	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0250	0.0098	Hudson-Raritan Estuary	14 % mort. NT							NT	Long et al. 1995b
0.0250	0.0104	Hudson-Raritan Estuary	28 % mort. T							T	Long et al. 1995b
0.0310	0.0310	Hudson-Raritan Estuary	97 % mort. T							T	Long et al. 1995b
0.0430	0.0097	Hudson-Raritan Estuary	100 % mort. T							T	Long et al. 1995b
0.0440	0.0177	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0440	0.0227	Hudson-Raritan Estuary	7 % mort. T							T	Long et al. 1995b
0.0520	0.0135	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0550	0.0196	Hudson-Raritan Estuary	12 % mort. NT							NT	Long et al. 1995b
0.0560	0.0184	Hudson-Raritan Estuary	49 % mort. T							T	Long et al. 1995b
0.0570	0.0335	Hudson-Raritan Estuary	21 % mort. T							T	Long et al. 1995b
0.0590	0.0133	Hudson-Raritan Estuary	82 % mort. T							T	Long et al. 1995b
0.0610	0.0305	Hudson-Raritan Estuary	24 % mort. T							T	Long et al. 1995b
0.0660	0.0210	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0660	0.0440	Hudson-Raritan Estuary	82 % mort. T							T	Long et al. 1995b
0.0680	0.0212	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0740	0.0296	Hudson-Raritan Estuary	19 % mort. T							T	Long et al. 1995b
0.0800	0.1039	Hudson-Raritan Estuary	9 % mort. NT							NT	Long et al. 1995b

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.0830	0.0275	Hudson-Raritan Estuary	0 % mort. NT							NT	Long et al. 1995b
0.0850	0.0234	Hudson-Raritan Estuary	24 % mort. T							T	Long et al. 1995b
0.0880	0.0926	Hudson-Raritan Estuary	71 % mort. T							T	Long et al. 1995b
0.0910	0.0286	Hudson-Raritan Estuary	6 % mort. NT							NT	Long et al. 1995b
0.1090	0.0436	Hudson-Raritan Estuary	17 % mort. NT							NT	Long et al. 1995b
0.1300	0.0591	Hudson-Raritan Estuary	100 % mort. T							T	Long et al. 1995b
0.1380	0.0657	Hudson-Raritan Estuary	47 % mort. T							T	Long et al. 1995b
0.1540	0.0386	Hudson-Raritan Estuary	23 % mort. T							T	Long et al. 1995b
0.1580	0.0367	Hudson-Raritan Estuary	23 % mort. NT							NT	Long et al. 1995b
0.1790	0.0746	Hudson-Raritan Estuary	32 % mort. T							T	Long et al. 1995b
0.1820	0.0758	Hudson-Raritan Estuary	85 % mort. T							T	Long et al. 1995b
0.1930	0.0460	Hudson-Raritan Estuary	80 % mort. T							T	Long et al. 1995b
0.1950	0.0336	Hudson-Raritan Estuary	75 % mort. T							T	Long et al. 1995b
0.2100	0.0913	Hudson-Raritan Estuary	91 % mort. T							T	Long et al. 1995b
0.2100	0.0412	Hudson-Raritan Estuary	48 % mort. T							T	Long et al. 1995b
0.2180	0.0436	Hudson-Raritan Estuary	69 % mort. T							T	Long et al. 1995b
0.2300	0.0958	Hudson-Raritan Estuary	81 % mort. T							T	Long et al. 1995b
0.2400	0.0393	Hudson-Raritan Estuary	71 % mort. T							T	Long et al. 1995b
0.2870	0.1196	Hudson-Raritan Estuary	67 % mort. T							T	Long et al. 1995b
0.3730	0.1884	Hudson-Raritan Estuary	100 % mort. T							T	Long et al. 1995b
0.4160	0.0870	Hudson-Raritan Estuary	98 % mort. T							T	Long et al. 1995b
0.5170	0.1621	Hudson-Raritan Estuary	86 % mort. T							T	Long et al. 1995b
0.5860	0.1966	Hudson-Raritan Estuary	80 % mort. T							T	Long et al 1995b
1.0840	0.7374	Hudson-Raritan Estuary	63 % mort. T							T	Long et al. 1995b
0.0001	0.0002	Biscayne Bay, FL	0 % mort. NT	89 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0002	0.0006	Biscayne Bay, FL	10 % mort. NT	81 % fert. NT	56 % norm. dev. T					T	Long 1997
0.0002	0.0005	Biscayne Bay, FL	0 % mort. NT	91 % fert. NT	100 % norm. dev. NT					NT	Long 1997
0.0002	0.0004	Biscayne Bay, FL	35 % mort. T	110 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0002	0.0007	Biscayne Bay, FL	0 % mort. NT	91 % fert. NT	99 % norm. dev. NT					NT	Long 1997
0.0002	0.0016	Biscayne Bay, FL	11 % mort. NT	95 % fert. NT	97 % norm. dev. NT					NT	Long 1997
0.0002	0.0004	Biscayne Bay, FL	19 % mort. NT	110 % fert. NT	3 % norm. dev. T					T	Long 1997
0.0002	0.0014	Biscayne Bay, FL	3 % mort. NT	97 % fert. NT	102 % norm. dev. NT					NT	Long 1997
0.0002	0.0005	Biscayne Bay, FL	5 % mort. NT	117 % fert. NT	2 % norm. dev. T					T	Long 1997
0.0002	0.0015	Biscayne Bay, FL	18 % mort. NT	99 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0003	0.0004	Biscayne Bay, FL	0 % mort. NT	69 % fert. T	0 % norm. dev. T					T	Long 1997
0.0003	0.0004	Biscayne Bay, FL	0 % mort. NT	114 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0003	0.0004	Biscayne Bay, FL	0 % mort. NT	112 % fert. NT	0 % norm. dev. T					T	Long 1997

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.0003	0.0010	Biscayne Bay, FL	0 % mort. NT	96 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0003	0.0003	Biscayne Bay, FL	0 % mort. NT	117 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0003	0.0002	Biscayne Bay, FL	0 % mort. NT	108 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0003	0.0006	Biscayne Bay, FL	2 % mort. NT	111 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0003	0.0002	Biscayne Bay, FL	6 % mort. NT	1 % fert. T	0 % norm. dev. T					T	Long 1997
0.0003	0.0001	Biscayne Bay, FL	7 % mort. NT	1 % fert. T	2 % norm. dev. T					T	Long 1997
0.0003	0.0003	Biscayne Bay, FL	12 % mort. NT	105 % fert. NT	105 % norm. dev. NT					NT	Long 1997
0.0003	0.0013	Biscayne Bay, FL	18 % mort. NT	59 % fert. T	64 % norm. dev. T					T	Long 1997
0.0003	0.0008	Biscayne Bay, FL	43 % mort. T	34 % fert. T	13 % norm. dev. T					T	Long 1997
0.0003	0.0001	Biscayne Bay, FL	56 % mort. T	0 % fert. T	0 % norm. dev. T					T	Long 1997
0.0003	0.0006	Biscayne Bay, FL	19 % mort. NT	115 % fert. NT	45 % norm. dev. T					T	Long 1997
0.0003	0.0004	Biscayne Bay, FL	4 % mort. NT	91 % fert. NT	100 % norm. dev. NT					NT	Long 1997
0.0003	0.0010	Biscayne Bay, FL	9 % mort. NT	72 % fert. T	91 % norm. dev. NT					T	Long 1997
0.0003	0.0004	Biscayne Bay, FL	9 % mort. NT	96 % fert. NT	96 % norm. dev. NT					NT	Long 1997
0.0003	0.0002	Biscayne Bay, FL	5 % mort. NT	0 % fert. T	0 % norm. dev. T					T	Long 1997
0.0003	0.0006	Biscayne Bay, FL	7 % mort. NT	110 % fert. NT	53 % norm. dev. T					T	Long 1997
0.0003	0.0005	Biscayne Bay, FL	2 % mort. NT	97 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0003	0.0006	Biscayne Bay, FL	23 % mort. T	91 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0004	0.0002	Biscayne Bay, FL	8 % mort. NT	108 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0004	0.0004	Biscayne Bay, FL	4 % mort. NT	109 % fert. NT	100 % norm. dev. NT					NT	Long 1997
0.0004	0.0009	Biscayne Bay, FL	18 % mort. NT	114 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0004	0.0009	Biscayne Bay, FL	6 % mort. NT	114 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0004	0.0006	Biscayne Bay, FL	4 % mort. NT	78 % fert. T	103 % norm. dev. NT					T	Long 1997
0.0005	0.0006	Biscayne Bay, FL	11 % mort. NT	114 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0005	0.0020	Biscayne Bay, FL	4 % mort. NT	106 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0005	0.0007	Biscayne Bay, FL	17 % mort. NT	115 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0005	0.0003	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	98 % norm. dev. NT					NT	Long 1997
0.0006	0.0013	Biscayne Bay, FL	6 % mort. NT	95 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0007	0.0021	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	102 % norm. dev. NT					NT	Long 1997
0.0007	0.0005	Biscayne Bay, FL	4 % mort. NT	114 % fert. NT	62 % norm. dev. T					T	Long 1997
0.0008	0.0004	Biscayne Bay, FL	2 % mort. NT	62 % fert. T	86 % norm. dev. NT					T	Long 1997
0.0009	0.0016	Biscayne Bay, FL	2 % mort. NT	101 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0010	0.0009	Biscayne Bay, FL	2 % mort. NT	68 % fert. T	82 % norm. dev. NT					T	Long 1997
0.0010	0.0012	Biscayne Bay, FL	0 % mort. NT	83 % fert. NT	96 % norm. dev. NT					NT	Long 1997
0.0011	0.0007	Biscayne Bay, FL	11 % mort. NT	94 % fert. NT	94 % norm. dev. NT					NT	Long 1997
0.0011	0.0021	Biscayne Bay, FL	1 % mort. NT	96 % fert. NT	99 % norm. dev. NT					NT	Long 1997
0.0012	0.0004	Biscayne Bay, FL	2 % mort. NT	101 % fert. NT	93.9 % norm. dev. NT					NT	Long 1997
0.0013	0.0006	Biscayne Bay, FL	2 % mort. NT	67 % fert. T	0 % norm. dev. T					T	Long 1997

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.0013	0.0004	Biscayne Bay, FL	4 % mort. NT	98 % fert. NT	99 % norm. dev. NT					NT	Long 1997
0.0013	0.0021	Biscayne Bay, FL	9 % mort. NT	95 % fert. NT	102 % norm. dev. NT					NT	Long 1997
0.0013	0.0010	Biscayne Bay, FL	5 % mort. NT	99 % fert. NT	89 % norm. dev. NT					NT	Long 1997
0.0013	0.0010	Biscayne Bay, FL	8 % mort. NT	66 % fert. T	0 % norm. dev. T					T	Long 1997
0.0013	0.0012	Biscayne Bay, FL	7 % mort. NT	82 % fert. NT	95 % norm. dev. NT					NT	Long 1997
0.0014	0.0011	Biscayne Bay, FL	1 % mort. NT	95 % fert. NT	101 % norm. dev. NT					NT	Long 1997
0.0015	0.0003	Biscayne Bay, FL	13 % mort. NT	100 % fert. NT	94 % norm. dev. NT					NT	Long 1997
0.0015	0.0009	Biscayne Bay, FL	0 % mort. NT	67 % fert. T	0 % norm. dev. T					T	Long 1997
0.0015	0.0009	Biscayne Bay, FL	0 % mort. NT	47 % fert. T	0 % norm. dev. T					T	Long 1997
0.0016	0.0005	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	104 % norm. dev. NT					NT	Long 1997
0.0016	0.0006	Biscayne Bay, FL	0 % mort. NT	112 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0017	0.0017	Biscayne Bay, FL	0 % mort. NT	86 % fert. NT	18 % norm. dev. T					T	Long 1997
0.0021	0.0011	Biscayne Bay, FL	0 % mort. NT	94 % fert. NT	90 % norm. dev. NT					NT	Long 1997
0.0021	0.0036	Biscayne Bay, FL	2 % mort. NT	97 % fert. NT	102 % norm. dev. NT					NT	Long 1997
0.0022	0.0012	Biscayne Bay, FL	3 % mort. NT	113 % fert. NT	63 % norm. dev. T					T	Long 1997
0.0022	0.0016	Biscayne Bay, FL	0 % mort. NT	75 % fert. T	1 % norm. dev. T					T	Long 1997
0.0025	0.0011	Biscayne Bay, FL	1 % mort. NT	80.4 % fert. NT	89 % norm. dev. NT					NT	Long 1997
0.0028	0.0008	Biscayne Bay, FL	4 % mort. NT	80 % fert. NT	85 % norm. dev. NT					NT	Long 1997
0.0032	0.0064	Biscayne Bay, FL	0 % mort. NT	86 % fert. NT	12 % norm. dev. T					T	Long 1997
0.0034	0.0007	Biscayne Bay, FL	0 % mort. NT	7 % fert. T	0 % norm. dev. T					T	Long 1997
0.0039	0.0016	Biscayne Bay, FL	4 % mort. NT	92 % fert. NT	100 % norm. dev. NT					NT	Long 1997
0.0049	0.0007	Biscayne Bay, FL	5 % mort. NT	88 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0050	0.0039	Biscayne Bay, FL	6 % mort. NT	94 % fert. NT	95 % norm. dev. NT					NT	Long 1997
0.0056	0.0009	Biscayne Bay, FL	0 % mort. NT	2 % fert. T	0 % norm. dev. T					T	Long 1997
0.0056	0.0017	Biscayne Bay, FL	0 % mort. NT	19 % fert. T	0 % norm. dev. T					T	Long 1997
0.0059	0.0030	Biscayne Bay, FL	4 % mort. NT	112 % fert. NT	10 % norm. dev. T					T	Long 1997
0.0063	0.0022	Biscayne Bay, FL	2 % mort. NT	91 % fert. NT	97 % norm. dev. NT					NT	Long 1997
0.0067	0.0014	Biscayne Bay, FL	0 % mort. NT	67 % fert. T	0 % norm. dev. T					T	Long 1997
0.0076	0.0011	Biscayne Bay, FL	9 % mort. NT	2 % fert. T	0 % norm. dev. T					T	Long 1997
0.0124	0.0089	Biscayne Bay, FL	59 % mort. T	92 % fert. NT	1 % norm. dev. T					T	Long 1997
0.0130	0.0019	Biscayne Bay, FL	7 % mort. NT	92 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0151	0.0040	Biscayne Bay, FL	9 % mort. NT	83 % fert. NT	102 % norm. dev. NT					NT	Long 1997
0.0151	0.0041	Biscayne Bay, FL	6 % mort. NT	99 % fert. NT	103 % norm. dev. NT					NT	Long 1997
0.0152	0.0039	Biscayne Bay, FL	3 % mort. NT	62 % fert. T	0 % norm. dev. T					T	Long 1997
0.0164	0.0095	Biscayne Bay, FL	84 % mort. T	80.4 % fert. NT	97 % norm. dev. NT					T	Long 1997
0.0181	0.0060	Biscayne Bay, FL	31 % mort. T	37 % fert. T	0 % norm. dev. T					T	Long 1997
0.0182	0.0055	Biscayne Bay, FL	5 % mort. NT	98 % fert. NT	75 % norm. dev. T					T	Long 1997
0.0223	0.0059	Biscayne Bay, FL	59 % mort. T	99 % fert. NT	97 % norm. dev. NT					T	Long 1997

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured								
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development	Mortality	Mortality	Growth	Toxicity	Reference
0.0244	0.0105	Biscayne Bay, FL	68 % mort. T	95 % fert. NT	0 % norm. dev. T					T	Long 1997
0.0470	0.0170	Biscayne Bay, FL	91 % mort. T	98 % fert. NT	78 % norm. dev. T					T	Long 1997
0.0478	0.0199	Biscayne Bay, FL	98 % mort. T	98 % fert. NT	97 % norm. dev. NT					T	Long 1997
0.0489	0.1062	Biscayne Bay, FL	69 % mort. T	91 % fert. NT	96 % norm. dev. NT					T	Long 1997
0.0522	0.0195	Biscayne Bay, FL	65 % mort. T	102 % fert. NT	96 % norm. dev. NT					T	Long 1997
0.0554	0.0141	Biscayne Bay, FL	49 % mort. T	48 % fert. T	3 % norm. dev. T					T	Long 1997
0.0579	0.0060	Biscayne Bay, FL	61 % mort. T	100 % fert. NT	79.8 % norm. dev. T					T	Long 1997
0.0613	0.0103	Biscayne Bay, FL	81 % mort. T	104 % fert. NT	13 % norm. dev. T					T	Long 1997
0.0657	0.0253	Biscayne Bay, FL	33 % mort. T	93 % fert. NT	2 % norm. dev. T					T	Long 1997
0.0702	0.0048	Biscayne Bay, FL	90 % mort. T	36 % fert. T	74 % norm. dev. T					T	Long 1997
0.0785	0.0188	Biscayne Bay, FL	59 % mort. T	93 % fert. NT	101 % norm. dev. NT					T	Long 1997
0.0869	0.0139	Biscayne Bay, FL	61 % mort. T	71 % fert. T	0 % norm. dev. T					T	Long 1997
0.1871	0.0220	Biscayne Bay, FL	95 % mort. T	96 % fert. NT	0 % norm. dev. T					T	Long 1997
0.2186	0.0247	Biscayne Bay, FL	91 % mort. T	88 % fert. NT	3 % norm. dev. T					T	Long 1997
0.2722	0.0302	Biscayne Bay, FL	92 % mort. T	14 % fert. T	0 % norm. dev. T					T	Long 1997
0.3451	0.1135	Biscayne Bay, FL	6 % mort. NT	95 % fert. NT	94 % norm. dev. NT					NT	Long 1997
0.0007		Narragansett Bay, RI	10 % mort. NT							NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	2.7 % mort. NT							NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	3.3 % mort. NT							NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	6 % mort. NT							NT	Munns et al. 1991
0.0008		Narragansett Bay, RI	4 % mort. NT							NT	Munns et al. 1991
0.0009		Narragansett Bay, RI	11.3 % mort. NT							NT	Munns et al. 1991
0.0009		Narragansett Bay, RI	4.7 % mort. NT							NT	Munns et al. 1991
0.0013		Narragansett Bay, RI	4.7 % mort. NT							NT	Munns et al. 1991
0.0015		Narragansett Bay, RI	4.7 % mort. NT							NT	Munns et al. 1991
0.0017		Narragansett Bay, RI	4 % mort. NT							NT	Munns et al. 1991
0.0026		Narragansett Bay, RI	6.7 % mort. NT							NT	Munns et al. 1991
0.0045		Narragansett Bay, RI	4.7 % mort. NT							NT	Munns et al. 1991
0.0049		Narragansett Bay, RI	8.7 % mort. NT							NT	Munns et al. 1991
0.0050		Narragansett Bay, RI	1.3 % mort. NT							NT	Munns et al. 1991
0.0058		Narragansett Bay, RI	3.3 % mort. NT							NT	Munns et al. 1991
0.0063		Narragansett Bay, RI	2 % mort. NT							NT	Munns et al. 1991
0.0071		Narragansett Bay, RI	6.7 % mort. NT							NT	Munns et al. 1991
0.0072		Narragansett Bay, RI	2 % mort. NT							NT	Munns et al. 1991
0.0091		Narragansett Bay, RI	4 % mort. NT							NT	Munns et al. 1991
0.0096		Narragansett Bay, RI	0 % mort. NT							NT	Munns et al. 1991
0.0100		Narragansett Bay, RI	4.7 % mort. NT							NT	Munns et al. 1991

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured										Overall	
			Amphipod	Sea Urchin		Sand Dollar			Polychaete					
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development		Mortality		Mortality		Growth	Toxicity	Reference
0.0102		Narragansett Bay, RI	2 % mort. NT										NT	Munns et al. 1991
0.0127		Narragansett Bay, RI	2.7 % mort. NT										NT	Munns et al. 1991
0.0280		Narragansett Bay, RI	4.7 % mort. NT										NT	Munns et al. 1991
0.0320		Narragansett Bay, RI	4 % mort. NT										NT	Munns et al. 1991
0.0465		Narragansett Bay, RI	10.7 % mort. NT										NT	Munns et al. 1991
0.0566		Narragansett Bay, RI	2 % mort. NT										NT	Munns et al. 1991
0.0591		Narragansett Bay, RI	6 % mort. NT										NT	Munns et al. 1991
0.1158		Narragansett Bay, RI	8 % mort. NT										NT	Munns et al. 1991
0.0010	0.0011	Puget Sound, WA	9 % mort. NT			3 % abnorm. dev.	NT			8 % mort.	NT		NT	Pastorok & Becker 1990
0.0015	0.0009	Puget Sound, WA	11 % mort. NT			5 % abnorm. dev.	NT			0 % mort.	NT		NT	Pastorok & Becker 1990
0.0020	0.0013	Puget Sound, WA	13 % mort. NT			2 % abnorm. dev.	NT			0 % mort.	NT		NT	Pastorok & Becker 1990
0.0020	0.0013	Puget Sound, WA	15 % mort. NT			23 % abnorm. dev.	NT			4 % mort.	NT		NT	Pastorok & Becker 1990
0.0050	0.0029	Puget Sound, WA	13 % mort. NT			12 % abnorm. dev.	T			8 % mort.	NT		T	Pastorok & Becker 1990
0.0050	0.0029	Puget Sound, WA	100 % mort. T			100 % abnorm. dev.	T			4 % mort.	NT		T	Pastorok & Becker 1990
0.0060	0.0038	Puget Sound, WA	22 % mort. NT			3 % abnorm. dev.	NT			4 % mort.	NT		NT	Pastorok & Becker 1990
0.0070	0.0047	Puget Sound, WA	100 % mort. T			12 % abnorm. dev.	T			4 % mort.	NT		T	Pastorok & Becker 1990
0.0070	0.0044	Puget Sound, WA	100 % mort. T			98 % abnorm. dev.	T			12 % mort.	NT		T	Pastorok & Becker 1990
0.0080	0.0044	Puget Sound, WA	16 % mort. NT			4 % abnorm. dev.	NT			16 % mort.	NT		NT	Pastorok & Becker 1990
0.0080	0.0073	Puget Sound, WA	16 % mort. NT			6 % abnorm. dev.	T			0 % mort.	NT		T	Pastorok & Becker 1990
0.0110	0.0079	Puget Sound, WA	9 % mort. NT			16 % abnorm. dev.	T			8 % mort.	NT		T	Pastorok & Becker 1990
0.0270	0.0129	Puget Sound, WA	100 % mort. T			100 % abnorm. dev.	T			60 % mort.	T		T	Pastorok & Becker 1990
0.0450	0.0346	Puget Sound, WA	31 % mort. T			100 % abnorm. dev.	T			8 % mort.	NT		T	Pastorok & Becker 1990
0.0540	0.0318	Puget Sound, WA	54 % mort. T			100 % abnorm. dev.	T			36 % mort.	T		T	Pastorok & Becker 1990
0.0010	0.0010	Hudson-Raritan Estuary				2.3 mm inc. length	NT	0 % mort.	NT	12 % mort.	NT	12.5 mg inc. NT	NT	Rice et al. 1995
0.0010	0.0100	Hudson-Raritan Estuary	2 % mort. NT										NT	Rice et al. 1995
0.0010	0.0100	Hudson-Raritan Estuary				2.4 mm inc. length	NT	7 % mort.	NT				NT	Rice et al. 1995
0.0010	0.0100	Hudson-Raritan Estuary	2 % mort. NT			2.6 mm inc. length	NT	0 % mort.	NT				NT	Rice et al. 1995
0.0020	0.0040	Hudson-Raritan Estuary	6 % mort. NT			1.9 mm inc. length	NT	0 % mort.	NT	3 % mort.	NT	15.2 mg inc. NT	NT	Rice et al. 1995
0.0030	0.0100	Hudson-Raritan Estuary	5 % mort. NT			1.6 mm inc. length	T	2 % mort.	NT	27 % mort.	NT	16.1 mg inc. NT	T	Rice et al. 1995
0.0150	0.0071	Hudson-Raritan Estuary				2.4 mm inc. length	NT	2 % mort.	NT	27 % mort.	NT	1.4 mg inc. T	T	Rice et al. 1995
0.0170	0.0077	Hudson-Raritan Estuary	15 % mort. T			1.7 mm inc. length	T	0 % mort.	NT	23 % mort.	NT	11 mg inc. NT	T	Rice et al. 1995
0.0210	0.0210	Hudson-Raritan Estuary	10 % mort. NT			2.2 mm inc. length	NT	0 % mort.	NT	7 % mort.	NT	9.7 mg inc. T	T	Rice et al. 1995
0.0270	0.0093	Hudson-Raritan Estuary				1.7 mm inc. length	NT	0 % mort.	NT	3 % mort.	NT	12.4 mg inc. T	T	Rice et al. 1995
0.0340	0.0227	Hudson-Raritan Estuary				2.2 mm inc. length	NT	2 % mort.	NT	40 % mort.	NT	7.1 mg inc. T	T	Rice et al. 1995
0.0370	0.0116	Hudson-Raritan Estuary	5 % mort. NT			2.1 mm inc. length	NT	0 % mort.	NT	13 % mort.	NT	9.1 mg inc. T	T	Rice et al. 1995
0.0470	0.0121	Hudson-Raritan Estuary				2.2 mm inc. length	NT	0 % mort.	NT	0 % mort.	NT	17.1 mg inc. T	T	Rice et al. 1995

Appendix A5-4. A summary of the available information on the toxic effects of sediment-associated Total DDT from locations outside the Southern California Bight.

Concentration			Endpoint Measured													
			Amphipod	Sea Urchin		Sand Dollar			Polychaete				Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth and Development		Mortality		Mortality		Growth		Toxicity	Reference
0.0850	0.0213	Hudson-Raritan Estuary				1.8 mm inc. length	NT	2 % mort.	NT	100 % mort.	T	0 mg inc.	T	T	Rice et al. 1995
0.1200	0.0480	Hudson-Raritan Estuary	14 % mort.	T		0.4 mm inc. length	T	1 % mort.	NT	15 % mort.	NT	2.8 mg inc.	T	T	Rice et al. 1995
0.1650	0.0423	Hudson-Raritan Estuary				2.3 mm inc. length	NT	0 % mort.	NT	17 % mort.	NT	6.6 mg inc.	T	T	Rice et al. 1995
0.2300	0.1533	Hudson-Raritan Estuary	9 % mort.	NT		2.3 mm inc. length	NT	0 % mort.	NT	17 % mort.	NT	9.9 mg inc.	T	T	Rice et al. 1995
0.2900	0.0784	Hudson-Raritan Estuary				2.2 mm inc. length	NT	0 % mort.	NT	13 % mort.	NT	1.9 mg inc.	T	T	Rice et al. 1995
0.3100	0.0756	Hudson-Raritan Estuary	8 % mort.	NT		1.7 mm inc. length	T	2 % mort.	NT	33 % mort.	NT	7.2 mg inc.	T	T	Rice et al. 1995
0.7500	0.1563	Hudson-Raritan Estuary	16 % mort.	T		2.2 mm inc. length	NT	0 % mort.	NT					T	Rice et al. 1995
0.0110	0.0126	San Francisco Bay, CA	24 % mort.	NT										NT	Swartz et al. 1994
0.0830	0.0703	San Francisco Bay, CA	24 % mort.	NT										NT	Swartz et al. 1994
0.3690	0.3417	San Francisco Bay, CA	23 % mort.	NT										NT	Swartz et al. 1994
0.4190	0.2812	San Francisco Bay, CA	28 % mort.	NT										NT	Swartz et al. 1994
2.34	0.7866	San Francisco Bay, CA	32 % mort.	T										T	Swartz et al. 1994
2.75	1.88	San Francisco Bay, CA	23 % mort.	NT										NT	Swartz et al. 1994
26.0	15.0	San Francisco Bay, CA	36 % mort.	T										T	Swartz et al. 1994
47.8	26.9	San Francisco Bay, CA	43 % mort.	T										T	Swartz et al. 1994
77.7	32.7	San Francisco Bay, CA	66 % mort.	T										T	Swartz et al. 1994

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.

Endpoint Measured: mort. = mortality; fert. = fertilization; norm. dev. = normal development; abnorm. dev. = abnormal development; inc. = increase.

NT = toxic; T = toxic.

Species tested included:

Casillas et al. 1992; Dendraster excentricus (sand dollar).

Chapman et al. 1987; Rhepoxynius abronius (amphipod).

Long et al. 1994; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Long et al. 1995b; Ampelisca abdita (amphipod).

Long 1997; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Munns et al. 1991; Ampelisca abdita (amphipod).

Pastorok & Becker 1990; Rhepoxynius abronius (amphipod), Dendraster excentricus (sand dollar), Neanthes arenaceodentata (polychaete).

Rice et al. 1995; Rhepoxynius abronius (amphipod), Dendraster excentricus (sand dollar), Armandia brevis (amphipod).

Swartz et al. 1994; Eohaustorius estuarius (amphipod).

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Aroclor 1254 from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Overall Toxicity	Reference
0.0031		Narragansett Bay, RI	8.7 % mort.	NT	NT	Munns et al. 1991
0.0079		Narragansett Bay, RI	12 % mort.	NT	NT	Munns et al. 1991
0.0141		Narragansett Bay, RI	5.3 % mort.	NT	NT	Munns et al. 1991
0.0221		Narragansett Bay, RI	8.6 % mort.	NT	NT	Munns et al. 1991
0.0426		Narragansett Bay, RI	4 % mort.	NT	NT	Munns et al. 1991
0.0471		Narragansett Bay, RI	4.7 % mort.	NT	NT	Munns et al. 1991
0.0487		Narragansett Bay, RI	6 % mort.	NT	NT	Munns et al. 1991
0.0949		Narragansett Bay, RI	2 % mort.	NT	NT	Munns et al. 1991
0.1260		Narragansett Bay, RI	6.7 % mort.	NT	NT	Munns et al. 1991
0.1320		Narragansett Bay, RI	10.7 % mort.	NT	NT	Munns et al. 1991
0.1820		Narragansett Bay, RI	8 % mort.	NT	NT	Munns et al. 1991
0.2020		Narragansett Bay, RI	1.3 % mort.	NT	NT	Munns et al. 1991
0.2040		Narragansett Bay, RI	8.7 % mort.	NT	NT	Munns et al. 1991
0.2210		Narragansett Bay, RI	4.7 % mort.	NT	NT	Munns et al. 1991
0.2330		Narragansett Bay, RI	3.3 % mort.	NT	NT	Munns et al. 1991
0.2840		Narragansett Bay, RI	2 % mort.	NT	NT	Munns et al. 1991
0.3480		Narragansett Bay, RI	4.7 % mort.	NT	NT	Munns et al. 1991
0.4970		Narragansett Bay, RI	6 % mort.	NT	NT	Munns et al. 1991
0.5050		Narragansett Bay, RI	4 % mort.	NT	NT	Munns et al. 1991
0.0500	0.1250	Oakland Inner Harbor, CA	7 % mort.	NT	NT	Word et al. 1988
0.0500	0.0424	Oakland Inner Harbor, CA	16 % mort.	NT	NT	Word et al. 1988
0.0500	0.0130	Oakland Inner Harbor, CA	18 % mort.	NT	NT	Word et al. 1988
0.0500	0.0467	Oakland Inner Harbor, CA	22 % mort.	NT	NT	Word et al. 1988
0.0500	0.0329	Oakland Inner Harbor, CA	26 % mort.	NT	NT	Word et al. 1988
0.0500	0.0463	Oakland Inner Harbor, CA	26 % mort.	NT	NT	Word et al. 1988
0.0500	0.0467	Oakland Inner Harbor, CA	30 % mort.	NT	NT	Word et al. 1988
0.0500	0.0258	Oakland Inner Harbor, CA	31 % mort.	NT	NT	Word et al. 1988
0.0600	0.0339	Oakland Inner Harbor, CA	21 % mort.	NT	NT	Word et al. 1988
0.0800	0.0455	Oakland Inner Harbor, CA	23 % mort.	NT	NT	Word et al. 1988
0.0900	0.0570	Oakland Inner Harbor, CA	24 % mort.	NT	NT	Word et al. 1988
0.1400	0.0848	Oakland Inner Harbor, CA	30 % mort.	NT	NT	Word et al. 1988
0.1700	0.0842	Oakland Inner Harbor, CA	30 % mort.	NT	NT	Word et al. 1988
0.3300	0.2260	Oakland Inner Harbor, CA	23 % mort.	NT	NT	Word et al. 1988
0.3700	0.1737	Oakland Inner Harbor, CA	24 % mort.	NT	NT	Word et al. 1988
0.3800	0.2011	Oakland Inner Harbor, CA	22 % mort.	NT	NT	Word et al. 1988

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Aroclor 1254 from locations outside the Southern California Bight.

Concentration DW	Concentration DW @1%OC	Area	Endpoint Measured: Amphipod Mortality		Overall Toxicity	Reference
0.5000	311	Oakland Inner Harbor, CA	32 % mort.	T	T	Word et al. 1988
0.0500	30.9	Oakland Inner Harbor, CA	38 % mort.	T	T	Word et al. 1988
0.0850	57.4	Oakland Inner Harbor, CA	35 % mort.	T	T	Word et al. 1988
0.1100	72.8	Oakland Inner Harbor, CA	36 % mort.	T	T	Word et al. 1988

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.
Endpoint Measured: mort. = mortality.
NT = toxic; T = toxic.
Species tested included:

Munns et al. 1991; Ampelisca abdita (amphipod).
Word et al. 1988; Rhepoxynius abronius (amphipod).

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode	Overall	
DW	DW @1% OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.0070		Puget Sound, WA				0.9 mm inc. d. NT	0 % mort. NT				NT	Casillas et al. 1992
0.0070		Puget Sound, WA				1.5 mm inc. d. NT	0 % mort. NT				NT	Casillas et al. 1992
0.0070		Puget Sound, WA				2.4 mm inc. d. NT	0 % mort. NT				NT	Casillas et al. 1992
0.0560	0.0373	Puget Sound, WA				0.5 mm inc. d. NT	62 % mort. T				T	Casillas et al. 1992
0.1000	0.0625	Puget Sound, WA				2 mm inc. d. T	2 % mort. NT				T	Casillas et al. 1992
0.1100	0.0647	Puget Sound, WA				0.5 mm inc. d. NT	52 % mort. T				T	Casillas et al. 1992
0.1500	0.0938	Puget Sound, WA				0.2 mm inc. d. NT	88 % mort. T				T	Casillas et al. 1992
0.1500	0.1000	Puget Sound, WA				2.3 mm inc. d. NT	0 % mort. NT				NT	Casillas et al. 1992
0.1600	0.0889	Puget Sound, WA				0 mm inc. d. T	100 % mort. T				T	Casillas et al. 1992
0.1700	0.1000	Puget Sound, WA				1.9 mm inc. d. T	5 % mort. NT				T	Casillas et al. 1992
0.2100	0.1909	Puget Sound, WA				0.9 mm inc. d. T	1 % mort. NT				T	Casillas et al. 1992
0.4200	0.3000	Puget Sound, WA				1.2 mm inc. d. NT	2 % mort. NT				NT	Casillas et al. 1992
0.5900	0.2810	Puget Sound, WA				1.5 mm inc. d. T	0 % mort. NT				T	Casillas et al. 1992
0.8400	0.6462	Puget Sound, WA				0.2 mm inc. d. T	3 % mort. NT				T	Casillas et al. 1992
1.70	1.00	Puget Sound, WA				0.2 mm inc. d. T	15 % mort. T				T	Casillas et al. 1992
0.0057	0.0095	S. Francisco Bay, CA	9 % mort. NT								NT	Chapman et al. 1987
0.0111	0.0089	S. Francisco Bay, CA	4 % mort. NT								NT	Chapman et al. 1987
0.0175	0.0120	S. Francisco Bay, CA	24 % mort. T								T	Chapman et al. 1987
0.0266	0.0242	S. Francisco Bay, CA	13 % mort. NT								NT	Chapman et al. 1987
0.0270	0.0218	S. Francisco Bay, CA	13 % mort. NT								NT	Chapman et al. 1987
0.0368	0.0281	S. Francisco Bay, CA	9 % mort. NT								NT	Chapman et al. 1987
0.0573	0.0398	S. Francisco Bay, CA	37 % mort. T								T	Chapman et al. 1987
0.1798	0.0446	S. Francisco Bay, CA	95 % mort. T								T	Chapman et al. 1987
0.2553	0.0813	S. Francisco Bay, CA	24 % mort. T								T	Chapman et al. 1987
0.0017	0.0010	Tampa Bay, FL	6 % mort. NT	46 % fert. T							T	Long et al. 1994
0.0027	0.0191	Tampa Bay, FL	11 % mort. NT	85.2 % fert. NT							NT	Long et al. 1994
0.0032	0.0351	Tampa Bay, FL	16 % mort. NT	61.6 % fert. T							T	Long et al. 1994
0.0032	0.0159	Tampa Bay, FL	20 % mort. NT	86.4 % fert. NT							NT	Long et al. 1994
0.0038	0.0109	Tampa Bay, FL	13 % mort. NT	46.2 % fert. T							T	Long et al. 1994
0.0040	0.0086	Tampa Bay, FL	15 % mort. NT	88.4 % fert. NT							NT	Long et al. 1994
0.0047	0.0263	Tampa Bay, FL	23 % mort. NT	9.6 % fert. T							T	Long et al. 1994
0.0049	0.0020	Tampa Bay, FL	5 % mort. NT	84.2 % fert. NT							NT	Long et al. 1994
0.0055	0.0062	Tampa Bay, FL	15 % mort. NT	82.8 % fert. NT							NT	Long et al. 1994
0.0105	0.0062	Tampa Bay, FL	7 % mort. NT	91.6 % fert. NT							NT	Long et al. 1994
0.0106	0.0557	Tampa Bay, FL	19 % mort. NT	91.6 % fert. NT							NT	Long et al. 1994
0.0107	0.0346	Tampa Bay, FL	9 % mort. NT	77.6 % fert. NT							NT	Long et al. 1994
0.0116	0.0331	Tampa Bay, FL	11 % mort. NT	25 % fert. T							T	Long et al. 1994
0.0130	0.0054	Tampa Bay, FL	15 % mort. NT	78.4 % fert. NT							NT	Long et al. 1994

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode	Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.0171	0.0053	Tampa Bay, FL	19 % mort. NT	43.8 % fert. T							T	Long et al. 1994
0.0200	0.0070	Tampa Bay, FL	7 % mort. NT	71.4 % fert. T							T	Long et al. 1994
0.0235	0.0086	Tampa Bay, FL	4 % mort. NT	22.6 % fert. T							T	Long et al. 1994
0.0251	0.0165	Tampa Bay, FL	14 % mort. NT	19.2 % fert. T							T	Long et al. 1994
0.0270	0.0134	Tampa Bay, FL	6 % mort. NT	3.4 % fert. T							T	Long et al. 1994
0.0323	0.0299	Tampa Bay, FL	17 % mort. NT	17.8 % fert. T							T	Long et al. 1994
0.0324	0.0073	Tampa Bay, FL	12 % mort. NT	0.6 % fert. T							T	Long et al. 1994
0.0325	0.0121	Tampa Bay, FL	7 % mort. NT	0 % fert. T							T	Long et al. 1994
0.0330	0.0177	Tampa Bay, FL	5 % mort. NT	72.4 % fert. T							T	Long et al. 1994
0.0369	0.0080	Tampa Bay, FL	5 % mort. NT	0.2 % fert. T							T	Long et al. 1994
0.0383	0.0107	Tampa Bay, FL	13 % mort. NT	0 % fert. T							T	Long et al. 1994
0.0485	0.0132	Tampa Bay, FL	19 % mort. NT	43 % fert. T							T	Long et al. 1994
0.0611	0.0156	Tampa Bay, FL	9 % mort. NT	0 % fert. T							T	Long et al. 1994
0.0674	0.0709	Tampa Bay, FL	13 % mort. NT	42.8 % fert. T							T	Long et al. 1994
0.0713	0.2852	Tampa Bay, FL	13 % mort. NT	84.4 % fert. NT							NT	Long et al. 1994
0.0719	0.0157	Tampa Bay, FL	11 % mort. NT	0 % fert. T							T	Long et al. 1994
0.0784	0.0603	Tampa Bay, FL	16 % mort. NT	0.2 % fert. T							T	Long et al. 1994
0.0886	0.0252	Tampa Bay, FL	17.5 % mort. T	0.6 % fert. T							T	Long et al. 1994
0.0897	0.0421	Tampa Bay, FL	12 % mort. NT	16 % fert. T							T	Long et al. 1994
0.1091	0.0466	Tampa Bay, FL	10 % mort. NT	27.2 % fert. T							T	Long et al. 1994
0.1364	0.0257	Tampa Bay, FL	14 % mort. NT	0 % fert. T							T	Long et al. 1994
0.1431	0.0745	Tampa Bay, FL	10 % mort. NT	3 % fert. T							T	Long et al. 1994
0.1572	0.1526	Tampa Bay, FL	18 % mort. NT	86.4 % fert. NT							NT	Long et al. 1994
0.1625	0.0620	Tampa Bay, FL	10 % mort. NT	5.6 % fert. T							T	Long et al. 1994
0.1791	0.0449	Tampa Bay, FL	16 % mort. NT	1 % fert. T							T	Long et al. 1994
0.1964	0.1650	Tampa Bay, FL	18 % mort. NT	7.6 % fert. T							T	Long et al. 1994
0.2020	0.1374	Tampa Bay, FL	15 % mort. NT	0 % fert. T							T	Long et al. 1994
0.2174	0.0921	Tampa Bay, FL	18 % mort. NT	74 % fert. T							T	Long et al. 1994
0.2259	0.0893	Tampa Bay, FL	6 % mort. NT	0.4 % fert. T							T	Long et al. 1994
0.2802	0.1698	Tampa Bay, FL	22 % mort. NT	0.2 % fert. T							T	Long et al. 1994
0.2840	0.0463	Tampa Bay, FL	17 % mort. NT	0.6 % fert. T							T	Long et al. 1994
0.2976	0.1272	Tampa Bay, FL	17 % mort. NT	0 % fert. T							T	Long et al. 1994
0.3042	0.1166	Tampa Bay, FL	10 % mort. NT	5.8 % fert. T							T	Long et al. 1994
0.3069	0.1047	Tampa Bay, FL	8 % mort. NT	4 % fert. T							T	Long et al. 1994
0.3124	0.1059	Tampa Bay, FL	1 % mort. NT	27.4 % fert. T							T	Long et al. 1994
0.3403	0.0729	Tampa Bay, FL	11 % mort. NT	0 % fert. T							T	Long et al. 1994
0.3524	0.0974	Tampa Bay, FL	9 % mort. NT	0 % fert. T							T	Long et al. 1994
0.3813	0.1315	Tampa Bay, FL	10 % mort. NT	3.2 % fert. T							T	Long et al. 1994
0.3852	0.2423	Tampa Bay, FL	22 % mort. NT	0 % fert. T							T	Long et al. 1994
0.5146	0.1559	Tampa Bay, FL	27 % mort. T	0 % fert. T							T	Long et al. 1994

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured								Overall	
			Amphipod	Sea Urchin			Sand Dollar	Polychaete		Nematode		
DW	DW @1% OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.5215	0.0858	Tampa Bay, FL	15 % mort. NT	0.6 % fert. T							T	Long et al. 1994
0.6964	0.2461	Tampa Bay, FL	55 % mort. T	0 % fert. T							T	Long et al. 1994
0.8014	0.4770	Tampa Bay, FL	24 % mort. T	11 % fert. T							T	Long et al. 1994
1.58	0.4795	Tampa Bay, FL	23 % mort. T	0 % fert. T							T	Long et al. 1994
1.60	0.2546	Tampa Bay, FL	22.5 % mort. T	0 % fert. T							T	Long et al. 1994
3.58	0.8166	Tampa Bay, FL	61 % mort. T	0 % fert. T							T	Long et al. 1994
16.7	5.62	Tampa Bay, FL	52 % mort. T	0 % fert. T							T	Long et al. 1994
0.0000	0.0000	Hudson-Raritan Est.	9 % mort. NT								NT	Long et al. 1995b
0.0080	0.0320	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.0400	0.0153	Hudson-Raritan Est.	81 % mort. T								T	Long et al. 1995b
0.0840	0.0840	Hudson-Raritan Est.	97 % mort. T								T	Long et al. 1995b
0.1050	0.2234	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.1090	0.0641	Hudson-Raritan Est.	4 % mort. NT								NT	Long et al. 1995b
0.1130	0.0395	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.1130	0.0582	Hudson-Raritan Est.	7 % mort. T								T	Long et al. 1995b
0.1160	0.0414	Hudson-Raritan Est.	12 % mort. NT								NT	Long et al. 1995b
0.1200	0.0388	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.1270	0.1337	Hudson-Raritan Est.	71 % mort. T								T	Long et al. 1995b
0.1340	0.0439	Hudson-Raritan Est.	49 % mort. T								T	Long et al. 1995b
0.1360	0.0306	Hudson-Raritan Est.	82 % mort. T								T	Long et al. 1995b
0.1490	0.0336	Hudson-Raritan Est.	100 % mort. T								T	Long et al. 1995b
0.1800	0.0359	Hudson-Raritan Est.	100 % mort. T								T	Long et al. 1995b
0.1900	0.0592	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.2060	0.0858	Hudson-Raritan Est.	28 % mort. T								T	Long et al. 1995b
0.2150	0.0683	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.2270	0.0850	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.2310	0.0924	Hudson-Raritan Est.	19 % mort. T								T	Long et al. 1995b
0.2310	0.1050	Hudson-Raritan Est.	100 % mort. T								T	Long et al. 1995b
0.2370	0.1612	Hudson-Raritan Est.	63 % mort. T								T	Long et al. 1995b
0.2390	0.0752	Hudson-Raritan Est.	6 % mort. NT								NT	Long et al. 1995b
0.2470	0.0818	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.2520	0.0653	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.2540	0.3299	Hudson-Raritan Est.	9 % mort. NT								NT	Long et al. 1995b
0.2700	0.1059	Hudson-Raritan Est.	14 % mort. NT								NT	Long et al. 1995b
0.3200	0.1600	Hudson-Raritan Est.	24 % mort. T								T	Long et al. 1995b
0.4000	0.2353	Hudson-Raritan Est.	21 % mort. T								T	Long et al. 1995b
0.4560	0.1256	Hudson-Raritan Est.	24 % mort. T								T	Long et al. 1995b
0.4840	0.1936	Hudson-Raritan Est.	17 % mort. NT								NT	Long et al. 1995b
0.5070	0.1271	Hudson-Raritan Est.	23 % mort. T								T	Long et al. 1995b

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured								Overall	
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode		
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.5390	0.3593	Hudson-Raritan Est.	82 % mort. T								T	Long et al. 1995b
0.5640	0.2686	Hudson-Raritan Est.	47 % mort. T								T	Long et al. 1995b
0.5700	0.2289	Hudson-Raritan Est.	0 % mort. NT								NT	Long et al. 1995b
0.5760	0.2400	Hudson-Raritan Est.	32 % mort. T								T	Long et al. 1995b
0.5830	0.1956	Hudson-Raritan Est.	80 % mort. T								T	Long et al. 1995b
0.6160	0.1931	Hudson-Raritan Est.	86 % mort. T								T	Long et al. 1995b
0.6710	0.1560	Hudson-Raritan Est.	23 % mort. NT								NT	Long et al. 1995b
0.8370	0.4227	Hudson-Raritan Est.	100 % mort. T								T	Long et al. 1995b
1.09	0.2131	Hudson-Raritan Est.	48 % mort. T								T	Long et al. 1995b
1.21	0.2079	Hudson-Raritan Est.	75 % mort. T								T	Long et al. 1995b
1.32	0.3152	Hudson-Raritan Est.	80 % mort. T								T	Long et al. 1995b
1.34	0.5583	Hudson-Raritan Est.	85 % mort. T								T	Long et al. 1995b
1.36	0.5679	Hudson-Raritan Est.	67 % mort. T								T	Long et al. 1995b
1.45	0.6322	Hudson-Raritan Est.	91 % mort. T								T	Long et al. 1995b
1.61	0.6704	Hudson-Raritan Est.	81 % mort. T								T	Long et al. 1995b
1.80	0.2951	Hudson-Raritan Est.	71 % mort. T								T	Long et al. 1995b
1.97	0.4128	Hudson-Raritan Est.	98 % mort. T								T	Long et al. 1995b
2.85	0.5700	Hudson-Raritan Est.	69 % mort. T								T	Long et al. 1995b
0.0011	0.0017	Biscayne Bay, FL	3 % mort. NT	97 % fert. NT	102 % norm. dev. NT						NT	Long 1997
0.0011	0.0013	Biscayne Bay, FL	43 % mort. T	34 % fert. T	13 % norm. dev. T						T	Long 1997
0.0011	0.0016	Biscayne Bay, FL	18 % mort. NT	59 % fert. T	64 % norm. dev. T						T	Long 1997
0.0012	0.0012	Biscayne Bay, FL	12 % mort. NT	105 % fert. NT	105 % norm. dev. NT						NT	Long 1997
0.0013	0.0012	Biscayne Bay, FL	0 % mort. NT	108 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0013	0.0014	Biscayne Bay, FL	0 % mort. NT	117 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0013	0.0015	Biscayne Bay, FL	0 % mort. NT	112 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0014	0.0017	Biscayne Bay, FL	0 % mort. NT	69 % fert. T	0 % norm. dev. T						T	Long 1997
0.0014	0.0012	Biscayne Bay, FL	56 % mort. T	0 % fert. T	0 % norm. dev. T						T	Long 1997
0.0014	0.0014	Biscayne Bay, FL	4 % mort. NT	109 % fert. NT	100 % norm. dev. NT						NT	Long 1997
0.0015	0.0013	Biscayne Bay, FL	4 % mort. NT	114 % fert. NT	62 % norm. dev. T						T	Long 1997
0.0017	0.0038	Biscayne Bay, FL	0 % mort. NT	96 % fert. NT	1 % norm. dev. T						T	Long 1997
0.0017	0.0043	Biscayne Bay, FL	4 % mort. NT	106 % fert. NT	1 % norm. dev. T						T	Long 1997
0.0017	0.0019	Biscayne Bay, FL	11 % mort. NT	114 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0018	0.0026	Biscayne Bay, FL	5 % mort. NT	117 % fert. NT	2 % norm. dev. T						T	Long 1997
0.0018	0.0028	Biscayne Bay, FL	19 % mort. NT	115 % fert. NT	45 % norm. dev. T						T	Long 1997
0.0018	0.0023	Biscayne Bay, FL	0 % mort. NT	114 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0019	0.0033	Biscayne Bay, FL	18 % mort. NT	114 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0020	0.0018	Biscayne Bay, FL	6 % mort. NT	1 % fert. T	0 % norm. dev. T						T	Long 1997
0.0021	0.0034	Biscayne Bay, FL	35 % mort. T	110 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0022	0.0032	Biscayne Bay, FL	7 % mort. NT	110 % fert. NT	53 % norm. dev. T						T	Long 1997

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode	Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.0022	0.0034	Biscayne Bay, FL	19 % mort. NT	110 % fert. NT	3 % norm. dev. T						T	Long 1997
0.0023	0.0040	Biscayne Bay, FL	2 % mort. NT	111 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0023	0.0048	Biscayne Bay, FL	0 % mort. NT	91 % fert. NT	100 % norm. dev. NT						NT	Long 1997
0.0023	0.0095	Biscayne Bay, FL	18 % mort. NT	99 % fert. NT	1 % norm. dev. T						T	Long 1997
0.0024	0.0018	Biscayne Bay, FL	5 % mort. NT	0 % fert. T	0 % norm. dev. T						T	Long 1997
0.0026	0.0127	Biscayne Bay, FL	11 % mort. NT	95 % fert. NT	97 % norm. dev. NT						NT	Long 1997
0.0030	0.0021	Biscayne Bay, FL	8 % mort. NT	108 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0033	0.0057	Biscayne Bay, FL	6 % mort. NT	114 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0040	0.0021	Biscayne Bay, FL	7 % mort. NT	1 % fert. T	2 % norm. dev. T						T	Long 1997
0.0041	0.0053	Biscayne Bay, FL	4 % mort. NT	91 % fert. NT	100 % norm. dev. NT						NT	Long 1997
0.0044	0.0060	Biscayne Bay, FL	17 % mort. NT	115 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0045	0.0124	Biscayne Bay, FL	0 % mort. NT	91 % fert. NT	99 % norm. dev. NT						NT	Long 1997
0.0046	0.0054	Biscayne Bay, FL	9 % mort. NT	96 % fert. NT	96 % norm. dev. NT						NT	Long 1997
0.0048	0.0054	Biscayne Bay, FL	0 % mort. NT	89 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0050	0.0021	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	104 % norm. dev. NT						NT	Long 1997
0.0050	0.0066	Biscayne Bay, FL	2 % mort. NT	97 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0051	0.0057	Biscayne Bay, FL	0 % mort. NT	83 % fert. NT	96 % norm. dev. NT						NT	Long 1997
0.0057	0.0191	Biscayne Bay, FL	10 % mort. NT	81 % fert. NT	56 % norm. dev. T						T	Long 1997
0.0057	0.0076	Biscayne Bay, FL	4 % mort. NT	78 % fert. T	103 % norm. dev. NT						T	Long 1997
0.0062	0.0184	Biscayne Bay, FL	9 % mort. NT	72 % fert. T	91 % norm. dev. NT						T	Long 1997
0.0073	0.0050	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	98 % norm. dev. NT						NT	Long 1997
0.0077	0.0029	Biscayne Bay, FL	4 % mort. NT	80.05 % fert. NT	85 % norm. dev. NT						NT	Long 1997
0.0092	0.0149	Biscayne Bay, FL	23 % mort. T	91 % fert. NT	1 % norm. dev. T						T	Long 1997
0.0119	0.0053	Biscayne Bay, FL	0 % mort. NT	112 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0123	0.0095	Biscayne Bay, FL	8 % mort. NT	66 % fert. T	0 % norm. dev. T						T	Long 1997
0.0123	0.0062	Biscayne Bay, FL	2 % mort. NT	62 % fert. T	86 % norm. dev. NT						T	Long 1997
0.0123	0.0071	Biscayne Bay, FL	3 % mort. NT	113 % fert. NT	63 % norm. dev. T						T	Long 1997
0.0126	0.0221	Biscayne Bay, FL	2 % mort. NT	101 % fert. NT	1 % norm. dev. T						T	Long 1997
0.0136	0.0121	Biscayne Bay, FL	2 % mort. NT	68 % fert. T	82 % norm. dev. NT						T	Long 1997
0.0151	0.0137	Biscayne Bay, FL	7 % mort. NT	82 % fert. NT	95 % norm. dev. NT						NT	Long 1997
0.0155	0.0095	Biscayne Bay, FL	0 % mort. NT	67 % fert. T	0 % norm. dev. T						T	Long 1997
0.0175	0.0328	Biscayne Bay, FL	1 % mort. NT	96 % fert. NT	99 % norm. dev. NT						NT	Long 1997
0.0185	0.0149	Biscayne Bay, FL	1 % mort. NT	95 % fert. NT	101 % norm. dev. NT						NT	Long 1997
0.0196	0.0415	Biscayne Bay, FL	6 % mort. NT	95 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0204	0.0110	Biscayne Bay, FL	2 % mort. NT	67 % fert. T	0 % norm. dev. T						T	Long 1997
0.0217	0.0040	Biscayne Bay, FL	9 % mort. NT	2 % fert. T	0 % norm. dev. T						T	Long 1997
0.0280	0.0065	Biscayne Bay, FL	0 % mort. NT	7 % fert. T	0 % norm. dev. T						T	Long 1997
0.0283	0.0150	Biscayne Bay, FL	4 % mort. NT	112 % fert. NT	10 % norm. dev. T						T	Long 1997
0.0292	0.0160	Biscayne Bay, FL	0 % mort. NT	94 % fert. NT	90 % norm. dev. NT						NT	Long 1997
0.0326	0.0103	Biscayne Bay, FL	0 % mort. NT	19 % fert. T	0 % norm. dev. T						T	Long 1997

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured								Overall	
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode		
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.0328	0.0947	Biscayne Bay, FL	1 % mort. NT	97 % fert. NT	102 % norm. dev. NT						NT	Long 1997
0.0371	0.0082	Biscayne Bay, FL	13 % mort. NT	100 % fert. NT	94 % norm. dev. NT						NT	Long 1997
0.0376	0.0087	Biscayne Bay, FL	0 % mort. NT	67 % fert. T	0 % norm. dev. T						T	Long 1997
0.0391	0.0656	Biscayne Bay, FL	9 % mort. NT	95 % fert. NT	102 % norm. dev. NT						NT	Long 1997
0.0424	0.0137	Biscayne Bay, FL	4 % mort. NT	98 % fert. NT	99 % norm. dev. NT						NT	Long 1997
0.0426	0.0187	Biscayne Bay, FL	1 % mort. NT	80.4 % fert. NT	89 % norm. dev. NT						NT	Long 1997
0.0435	0.0070	Biscayne Bay, FL	5 % mort. NT	88 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0446	0.0137	Biscayne Bay, FL	2 % mort. NT	101 % fert. NT	93.9 % norm. dev. NT						NT	Long 1997
0.0466	0.0295	Biscayne Bay, FL	0 % mort. NT	47 % fert. T	0 % norm. dev. T						T	Long 1997
0.0496	0.0387	Biscayne Bay, FL	6 % mort. NT	94 % fert. NT	95 % norm. dev. NT						NT	Long 1997
0.0499	0.0083	Biscayne Bay, FL	7 % mort. NT	92 % fert. NT	0 % norm. dev. T						T	Long 1997
0.0516	0.0179	Biscayne Bay, FL	31 % mort. T	37 % fert. T	0 % norm. dev. T						T	Long 1997
0.0548	0.0922	Biscayne Bay, FL	2 % mort. NT	97 % fert. NT	102 % norm. dev. NT						NT	Long 1997
0.0620	0.0220	Biscayne Bay, FL	2 % mort. NT	91 % fert. NT	97 % norm. dev. NT						NT	Long 1997
0.0697	0.0119	Biscayne Bay, FL	0 % mort. NT	2 % fert. T	0 % norm. dev. T						T	Long 1997
0.0709	0.0450	Biscayne Bay, FL	11 % mort. NT	94 % fert. NT	94 % norm. dev. NT						NT	Long 1997
0.0963	0.0778	Biscayne Bay, FL	5 % mort. NT	99 % fert. NT	89 % norm. dev. NT						NT	Long 1997
0.1133	0.0302	Biscayne Bay, FL	3 % mort. NT	62 % fert. T	0 % norm. dev. T						T	Long 1997
0.1163	0.0828	Biscayne Bay, FL	0 % mort. NT	75 % fert. T	1 % norm. dev. T						T	Long 1997
0.1185	0.0478	Biscayne Bay, FL	4 % mort. NT	92 % fert. NT	100 % norm. dev. NT						NT	Long 1997
0.1238	0.1251	Biscayne Bay, FL	0 % mort. NT	86 % fert. NT	18 % norm. dev. T						T	Long 1997
0.1628	0.3246	Biscayne Bay, FL	0 % mort. NT	86 % fert. NT	12 % norm. dev. T						T	Long 1997
0.2023	0.0541	Biscayne Bay, FL	9 % mort. NT	83 % fert. NT	102 % norm. dev. NT						NT	Long 1997
0.2091	0.1213	Biscayne Bay, FL	84 % mort. T	80.4 % fert. NT	97 % norm. dev. NT						T	Long 1997
0.2130	0.1535	Biscayne Bay, FL	59 % mort. T	92 % fert. NT	1 % norm. dev. T						T	Long 1997
0.3138	0.0946	Biscayne Bay, FL	5 % mort. NT	98 % fert. NT	75 % norm. dev. T						T	Long 1997
0.4728	0.1248	Biscayne Bay, FL	59 % mort. T	99 % fert. NT	97 % norm. dev. NT						T	Long 1997
0.5647	0.1525	Biscayne Bay, FL	6 % mort. NT	99 % fert. NT	103 % norm. dev. NT						NT	Long 1997
0.6284	0.2703	Biscayne Bay, FL	68 % mort. T	95 % fert. NT	0 % norm. dev. T						T	Long 1997
0.6601	0.2545	Biscayne Bay, FL	33 % mort. T	93 % fert. NT	2 % norm. dev. T						T	Long 1997
0.8201	0.1382	Biscayne Bay, FL	81 % mort. T	104 % fert. NT	13 % norm. dev. T						T	Long 1997
0.8807	0.2248	Biscayne Bay, FL	49 % mort. T	48 % fert. T	3 % norm. dev. T						T	Long 1997
0.9145	1.9870	Biscayne Bay, FL	69 % mort. T	91 % fert. NT	96 % norm. dev. NT						T	Long 1997
0.9347	0.3381	Biscayne Bay, FL	91 % mort. T	98 % fert. NT	78 % norm. dev. T						T	Long 1997
1.07	0.2578	Biscayne Bay, FL	59 % mort. T	93 % fert. NT	101 % norm. dev. NT						T	Long 1997
1.08	0.1729	Biscayne Bay, FL	61 % mort. T	71 % fert. T	0 % norm. dev. T						T	Long 1997
1.08	0.1140	Biscayne Bay, FL	61 % mort. T	100 % fert. NT	79.8 % norm. dev. T						T	Long 1997
1.15	0.3805	Biscayne Bay, FL	6 % mort. NT	95 % fert. NT	94 % norm. dev. NT						NT	Long 1997
1.22	0.4556	Biscayne Bay, FL	65 % mort. T	102 % fert. NT	96 % norm. dev. NT						T	Long 1997
1.30	0.5423	Biscayne Bay, FL	98 % mort. T	98 % fert. NT	97 % norm. dev. NT						T	Long 1997

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode	Overall	
DW	DW @1% OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
1.95	0.2215	Biscayne Bay, FL	91 % mort. T	88 % fert. NT	3 % norm. dev. T						T	Long 1997
2.00	0.2362	Biscayne Bay, FL	95 % mort. T	96 % fert. NT	0 % norm. dev. T						T	Long 1997
2.50	0.1721	Biscayne Bay, FL	90 % mort. T	36 % fert. T	74 % norm. dev. T						T	Long 1997
2.89	0.3224	Biscayne Bay, FL	92 % mort. T	14 % fert. T	0 % norm. dev. T						T	Long 1997
0.0033		Narragansett Bay, RI	8.7 % mort. NT								NT	Munns et al. 1991
0.0081		Narragansett Bay, RI	12 % mort. NT								NT	Munns et al. 1991
0.0143		Narragansett Bay, RI	5.3 % mort. NT								NT	Munns et al. 1991
0.0223		Narragansett Bay, RI	8.6 % mort. NT								NT	Munns et al. 1991
0.0428		Narragansett Bay, RI	4 % mort. NT								NT	Munns et al. 1991
0.0473		Narragansett Bay, RI	4.7 % mort. NT								NT	Munns et al. 1991
0.0489		Narragansett Bay, RI	6 % mort. NT								NT	Munns et al. 1991
0.0951		Narragansett Bay, RI	2 % mort. NT								NT	Munns et al. 1991
0.1262		Narragansett Bay, RI	6.7 % mort. NT								NT	Munns et al. 1991
0.1322		Narragansett Bay, RI	10.7 % mort. NT								NT	Munns et al. 1991
0.1822		Narragansett Bay, RI	8 % mort. NT								NT	Munns et al. 1991
0.2022		Narragansett Bay, RI	1.3 % mort. NT								NT	Munns et al. 1991
0.2042		Narragansett Bay, RI	8.7 % mort. NT								NT	Munns et al. 1991
0.2212		Narragansett Bay, RI	4.7 % mort. NT								NT	Munns et al. 1991
0.2332		Narragansett Bay, RI	3.3 % mort. NT								NT	Munns et al. 1991
0.2842		Narragansett Bay, RI	2 % mort. NT								NT	Munns et al. 1991
0.3482		Narragansett Bay, RI	4.7 % mort. NT								NT	Munns et al. 1991
0.4982		Narragansett Bay, RI	6 % mort. NT								NT	Munns et al. 1991
0.5052		Narragansett Bay, RI	4 % mort. NT								NT	Munns et al. 1991
0.0110	0.0126	Puget Sound, WA	9 % mort. NT			3 % abn. dev. NT		8 % mort. NT			NT	Pastorok & Becker 1990
0.0440	0.0275	Puget Sound, WA	15 % mort. NT			23 % abn. dev. NT		4 % mort. NT			NT	Pastorok & Becker 1990
0.0560	0.0329	Puget Sound, WA	11 % mort. NT			5 % abn. dev. NT		0 % mort. NT			NT	Pastorok & Becker 1990
0.0560	0.0373	Puget Sound, WA	13 % mort. NT			2 % abn. dev. NT		0 % mort. NT			NT	Pastorok & Becker 1990
0.1000	0.0625	Puget Sound, WA	100 % mort. T			98 % abn. dev. T		12 % mort. NT			T	Pastorok & Becker 1990
0.1100	0.0647	Puget Sound, WA	13 % mort. NT			12 % abn. dev. T		8 % mort. NT			T	Pastorok & Becker 1990
0.1500	0.0938	Puget Sound, WA	22 % mort. NT			3 % abn. dev. NT		4 % mort. NT			NT	Pastorok & Becker 1990
0.1500	0.1000	Puget Sound, WA	100 % mort. T			12 % abn. dev. T		4 % mort. NT			T	Pastorok & Becker 1990
0.1600	0.0889	Puget Sound, WA	16 % mort. NT			4 % abn. dev. NT		16 % mort. NT			NT	Pastorok & Becker 1990
0.1700	0.1000	Puget Sound, WA	100 % mort. T			100 % abn. dev. T		4 % mort. NT			T	Pastorok & Becker 1990
0.2100	0.1909	Puget Sound, WA	16 % mort. NT			6 % abn. dev. T		0 % mort. NT			T	Pastorok & Becker 1990
0.4200	0.3000	Puget Sound, WA	9 % mort. NT			16 % abn. dev. T		8 % mort. NT			T	Pastorok & Becker 1990
0.5900	0.2810	Puget Sound, WA	100 % mort. T			100 % abn. dev. T		60 % mort. T			T	Pastorok & Becker 1990
0.8400	0.6462	Puget Sound, WA	31 % mort. T			100 % abn. dev. T		8 % mort. NT			T	Pastorok & Becker 1990
1.70	1.00	Puget Sound, WA	54 % mort. T			100 % abn. dev. T		36 % mort. T			T	Pastorok & Becker 1990

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured														Overall	
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode								
DW	DW @1% OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth							Toxicity	Source
0.0020	0.0200	Hudson-Raritan Est.	2 % mort. NT														NT	Rice et al. 1995
0.0020	0.0200	Hudson-Raritan Est.	2 % mort. NT			2.6 mm inc. len. NT	0 % mort. NT										NT	Rice et al. 1995
0.0030	0.0300	Hudson-Raritan Est.				2.4 mm inc. len. NT	7 % mort. NT										NT	Rice et al. 1995
0.0060	0.0060	Hudson-Raritan Est.				2.3 mm inc. len. NT	0 % mort. NT	12 % mort. NT	12.5 mg inc. NT								NT	Rice et al. 1995
0.0450	0.1500	Hudson-Raritan Est.	5 % mort. NT			1.6 mm inc. len. T	2 % mort. NT	27 % mort. NT	16.1 mg inc. NT								T	Rice et al. 1995
0.0900	0.1800	Hudson-Raritan Est.	6 % mort. NT			1.9 mm inc. len. NT	0 % mort. NT	3 % mort. NT	15.2 mg inc. NT								NT	Rice et al. 1995
0.2100	0.1000	Hudson-Raritan Est.				2.4 mm inc. len. NT	2 % mort. NT	27 % mort. NT	1.4 mg inc. T								T	Rice et al. 1995
0.2200	0.2200	Hudson-Raritan Est.	10 % mort. NT			2.2 mm inc. len. NT	0 % mort. NT	7 % mort. NT	9.7 mg inc. T								T	Rice et al. 1995
0.2400	0.1600	Hudson-Raritan Est.				2.2 mm inc. len. NT	2 % mort. NT	40 % mort. NT	7.1 mg inc. T								T	Rice et al. 1995
0.3900	0.1054	Hudson-Raritan Est.				2.2 mm inc. len. NT	0 % mort. NT	13 % mort. NT	1.9 mg inc. T								T	Rice et al. 1995
0.3900	0.1773	Hudson-Raritan Est.	15 % mort. T			1.7 mm inc. len. T	0 % mort. NT	23 % mort. NT	11 mg inc. NT								T	Rice et al. 1995
0.4300	0.1049	Hudson-Raritan Est.	8 % mort. NT			1.7 mm inc. len. T	2 % mort. NT	33 % mort. NT	7.2 mg inc. T								T	Rice et al. 1995
0.4700	0.3133	Hudson-Raritan Est.	9 % mort. NT			2.3 mm inc. len. NT	0 % mort. NT	17 % mort. NT	9.9 mg inc. T								T	Rice et al. 1995
0.7600	0.1949	Hudson-Raritan Est.				2.2 mm inc. len. NT	0 % mort. NT	0 % mort. NT	17.1 mg inc. T								T	Rice et al. 1995
0.7800	0.1950	Hudson-Raritan Est.				1.8 mm inc. len. NT	2 % mort. NT	100 % mort. T	0 mg inc. T								T	Rice et al. 1995
0.8500	0.2931	Hudson-Raritan Est.				1.7 mm inc. len. NT	0 % mort. NT	3 % mort. NT	12.4 mg inc. T								T	Rice et al. 1995
0.9700	0.3031	Hudson-Raritan Est.	5 % mort. NT			2.1 mm inc. len. NT	0 % mort. NT	13 % mort. NT	9.1 mg inc. T								T	Rice et al. 1995
1.27	0.3256	Hudson-Raritan Est.				2.3 mm inc. len. NT	0 % mort. NT	17 % mort. NT	6.6 mg inc. T								T	Rice et al. 1995
1.64	0.3417	Hudson-Raritan Est.	16 % mort. T			2.2 mm inc. len. NT	0 % mort. NT										T	Rice et al. 1995
2.88	1.15	Hudson-Raritan Est.	14 % mort. T			0.4 mm inc. len. T	1 % mort. NT	15 % mort. NT	2.8 mg inc. T								T	Rice et al. 1995
0.0004		Hudson Raritan Est.								0.085	NT	0.06	NT				NT	Tietjen & Lee 1984
0.0005		Hudson Raritan Est.								0.107	NT	0.069	NT				NT	Tietjen & Lee 1984
0.0008		Hudson Raritan Est.								0.093	NT	0.049	NT				NT	Tietjen & Lee 1984
0.0060		Hudson Raritan Est.								0.06	NT	-0.043	T				T	Tietjen & Lee 1984
0.0300		Hudson Raritan Est.								0.013	T	0.007	T				T	Tietjen & Lee 1984
0.0300		Hudson Raritan Est.								-0.012	T	0.02	T				T	Tietjen & Lee 1984
0.0680		Hudson Raritan Est.								0.053	NT	-0.009	T				T	Tietjen & Lee 1984
0.1840		Hudson Raritan Est.								0.092	NT	0.019	NT				NT	Tietjen & Lee 1984
0.1980		Hudson Raritan Est.								0.072	NT	0.021	NT				NT	Tietjen & Lee 1984
0.2700		Hudson Raritan Est.								0.09	NT	-0.064	T				T	Tietjen & Lee 1984
0.3200		Hudson Raritan Est.								-0.082	T	-0.104	T				T	Tietjen & Lee 1984
0.4000		Hudson Raritan Est.								0.055	NT	-0.008	T				T	Tietjen & Lee 1984
0.6950		Hudson Raritan Est.								-0.039	T	-0.004	T				T	Tietjen & Lee 1984
0.8400		Hudson Raritan Est.								0.012	T	-0.039	T				T	Tietjen & Lee 1984
1.11		Hudson Raritan Est.								0.013	T	-0.065	T				T	Tietjen & Lee 1984
1.11		Hudson Raritan Est.								-0.199	T	-0.127	T				T	Tietjen & Lee 1984
1.30		Hudson Raritan Est.								-0.143	T	0.014	T				T	Tietjen & Lee 1984
1.56		Hudson Raritan Est.								0.055	NT	-0.139	T				T	Tietjen & Lee 1984

Appendix A5-5. A summary of the available information on the toxic effects of sediment-associated Total Polychlorinated Biphenyls from locations outside the Southern California Bight.

Concentration			Endpoint Measured									
			Amphipod	Sea Urchin		Sand Dollar		Polychaete		Nematode	Overall	
DW	DW @1%OC	Area	Mortality	Fertilization	Development	Growth & Developmen	Mortality	Mortality	Growth	Population Growth	Toxicity	Source
0.1500	0.3750	Oakland Inner H., CA	7 % mort.	NT							NT	Word et al. 1988
0.1500	0.1271	Oakland Inner H., CA	16 % mort.	NT							NT	Word et al. 1988
0.1500	0.0391	Oakland Inner H., CA	18 % mort.	NT							NT	Word et al. 1988
0.1500	0.1402	Oakland Inner H., CA	22 % mort.	NT							NT	Word et al. 1988
0.1500	0.0987	Oakland Inner H., CA	26 % mort.	NT							NT	Word et al. 1988
0.1500	0.1389	Oakland Inner H., CA	26 % mort.	NT							NT	Word et al. 1988
0.1500	0.1402	Oakland Inner H., CA	30 % mort.	NT							NT	Word et al. 1988
0.1500	0.0773	Oakland Inner H., CA	31 % mort.	NT							NT	Word et al. 1988
0.1500	0.0926	Oakland Inner H., CA	38 % mort.	T							T	Word et al. 1988
0.1600	0.0904	Oakland Inner H., CA	21 % mort.	NT							NT	Word et al. 1988
0.1800	0.1023	Oakland Inner H., CA	23 % mort.	NT							NT	Word et al. 1988
0.1850	0.1250	Oakland Inner H., CA	35 % mort.	T							T	Word et al. 1988
0.1900	0.1203	Oakland Inner H., CA	24 % mort.	NT							NT	Word et al. 1988
0.2100	0.1391	Oakland Inner H., CA	36 % mort.	T							T	Word et al. 1988
0.2400	0.1455	Oakland Inner H., CA	30 % mort.	NT							NT	Word et al. 1988
0.3300	0.1634	Oakland Inner H., CA	30 % mort.	NT							NT	Word et al. 1988
0.5500	0.3767	Oakland Inner H., CA	23 % mort.	NT							NT	Word et al. 1988
0.5900	0.3122	Oakland Inner H., CA	22 % mort.	NT							NT	Word et al. 1988
0.7200	0.4472	Oakland Inner H., CA	32 % mort.	T							T	Word et al. 1988
0.7800	0.3662	Oakland Inner H., CA	24 % mort.	NT							NT	Word et al. 1988

Concentration: DW = dry weight; @1%OC = at 1% organic carbon.

Area: Est. = estuary; S. Francisco = San Francisco; Oakland Inner H. = Oakland Inner Harbor.

Endpoint Measured: mort. = mortality; fert. = fertilization; norm. dev. = normal development; abn. dev. = abnormal development; inc. len. = increase length; inc. d. = increase diameter.

NT = toxic; T = toxic.

Species tested included:

Casillas et al. 1992; Dendraster excentricus (sand dollar).

Chapman et al. 1987; Rhepoxynius abronius (amphipod).

Long et al. 1994; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Long et al. 1995b; Ampelisca abdita (amphipod).

Long 1997; Ampelisca abdita (amphipod); Arbacia punctulata (sea urchin).

Munns et al. 1991; Ampelisca abdita (amphipod).

Pastorok & Becker 1990; Rhepoxynius abronius (amphipod), Dendraster excentricus (sand dollar), Neanthes arenaceodentata (polychaete).

Rice et al. 1995; Rhepoxynius abronius (amphipod), Dendraster excentricus (sand dollar), Armandia brevis (amphipod).

Swartz et al. 1994; Eohaustorius estuarius (amphipod).

Tietjen & Lee 1984; Chromadorina germanica and Diplolaimella punicea (nematodes).

Word et al. 1988; Rhepoxynius abronius (amphipod).

Appendix 6

CV of Donald D. MacDonald

DONALD D. MACDONALD

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EDUCATION:

Bachelor of Science, Zoology
(Fisheries Biology; Environmental Physiology, Comparative Biochemistry)
University of British Columbia, 1982

SPECIALIZATION:

Principal of MacDonald Environmental Sciences Limited, which was established to provide scientific consulting services in the fields of fisheries and aquatic resource management, stream ecology, environmental quality guidelines and policy development, environmental risk and hazard assessment, and information and technology transfer.

Specialist environmental toxicology and chemistry, ecosystem-based resource management, water quality/water use interactions, and sediment quality assessment.

PROFESSIONAL MEMBERSHIPS:

American Fisheries Society
Past-President, Canadian Aquatic Resources Section; Nominations Committee; Chair, Wetlands Conservation Committee; Newsletter Committee; Membership Committee.

Aquaculture Association of Canada

Association of Professional Biologists of British Columbia

Canadian Association on Water Pollution Research and Control

International Association on Water Pollution Research and Control

Pacific Fisheries Biologists

Society of Environmental Toxicology and Chemistry

DONALD D. MACDONALD

A6-2

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EXPERIENCE:

AQUATIC BIOLOGIST - February, 1989 to Present

MacDonald Environmental Sciences Limited
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Independent consulting on fisheries and aquatic resource management, environmental quality, stream ecology, computer data management, and information and technology transfer. Recent projects have been focused on the development of water quality guidelines, sediment quality guidelines, tissue residue guidelines, environmental quality monitoring programs, fisheries co-management programs, and the assessment of environmental quality.

WATER QUALITY OBJECTIVES OFFICER - September, 1984 to February, 1989

Water Quality Branch, Inland Waters, Environment Canada
502 - 1001 West Pender Street, Vancouver, B.C. V6E 2M9

Compilation, management and statistical analysis of existing and new information generated to support the formulation of water quality objectives in waters of significant federal interest; generation of water quality criteria information through toxicological, water quality, and other studies; design and implementation of monitoring programs to assess compliance with water quality objectives; preparation of reports and other publications on information developed to formulate water quality objectives; organization of workshops and information exchange sessions on water quality guidelines and objectives; provision of information and advice to technical committees established to resolve the International Joint Commission reference on the Flathead River.

Supervisor: Dr. D. Valiela, Head Water Quality Objectives Division

TECHNICAL PLANNING COORDINATOR - November, 1983 to September, 1984

Water Quality Branch, Inland Waters, Environment Canada
502 - 1001 West Pender Street, Vancouver, B.C. V6E 2M9

Planning and development of regional water quality programs, including long- and short-term logistics and budgetary requirements and inter-project coordination; planning, organization, expedition, and supervision of special field studies and sampling projects for water quality analysis; pollution surveillance and sediment sampling; planning and implementation on national water quality monitoring programs to assess national trends and conditions.

Supervisor: Dr. W.E. Erlebach, Chief Water Quality Branch

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PUBLICATIONS AND TECHNICAL REPORTS:

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