Environ. Sci. Technol. **2004**, *38,* 4519−4526

Food Web Pathway Determines How Selenium Affects Aquatic Ecosystems: A San Francisco Bay Case Study

A. ROBIN STEWART,*,† SAMUEL N. LUOMA,† CHRISTIAN E. SCHLEKAT,‡ MARTINA A. DOBLIN,§ AND KATHRYN A. HIEB||

U.S. Geological Survey, 345 Middlefield Road, Menlo Park, California 94025, Nickel Producers Environmental Research Association, 2605 Meridian Parkway, Suite 200, Durham, North Carolina 27713, Department of Ocean, Earth and Atmospheric Sciences, Old Dominion University, Norfolk, Virginia 23529, and California Department of Fish and Game, Central Valley Bay-Delta Branch, 4001 North Wilson Way, Stockton, California 95205

Chemical contaminants disrupt ecosystems, but specific effects may be under-appreciated when poorly known processes such as uptake mechanisms, uptake via diet, food preferences, and food web dynamics are influential. Here we show that a combination of food web structure and the physiology of trace element accumulation explain why some species in San Francisco Bay are threatened by a relatively low level of selenium contamination and some are not. Bivalves and crustacean zooplankton form the base of two dominant food webs in estuaries. The dominant bivalve *Potamocorbula amurensis* has a 10-fold slower rate constant of loss for selenium than do common crustaceans such as copepods and the mysid *Neomysis mercedis* (rate constant of loss, k_e = 0.025, 0.155, and 0.25 d^{-1}, respectively). The result is much higher selenium concentrations in the bivalve than in the crustaceans. Stable isotope analyses show that this difference is propagated up the respective food webs in San Francisco Bay. Several predators of bivalves have tissue concentrations of selenium that exceed thresholds thought to be associated with teratogenesis or reproductive failure (liver Se >15 $\mu g\ g^{-1}$ dry weight). Deformities typical of selenium-induced teratogenesis were observed in one of these species. Concentrations of selenium in tissues of predators of zooplankton are less than the thresholds. Basic physiological and ecological processes can drive wide differences in exposure and effects among species, but such processes are rarely considered in traditional evaluations of contaminant impacts.

Introduction

Large investments have been made in controlling chemical contamination of aquatic environments; however, identification of the ecological significance of contaminants in complex environmental settings remains problematic (*1*). One example of a significant effect on wildlife was the discovery of selenium (Se) poisoning at Kesterson Reservoir, CA, in 1983 (*2, 3*). Selenium, concentrated in irrigation drainage from the Western San Joaquin Valley (in the Central Valley of California; 300 $\mu g\ L^{-1}$; 3.8 mM), was transported into the Kesterson National Wildlife Refuge where it was accumulated by nesting birds, resulting in a significant deformity rate in bird hatchlings (64% nests affected for eared grebe, *Podiceps nigricollis*, and American coot, *Fulica americana*; *4*).

Concentrations of Se in solution and sediments were much reduced downstream from the reservoir, in San Francisco Bay (<1 $\mu g\ L^{-1}$ in water) (*5, 6*). Nevertheless, concentrations in some predatory fish (e.g., white sturgeon, *Acipenser transmontanus*) and some predatory birds (e.g., scoter, *Melanitta perspicillata*) were high (>10 $\mu g\ g^{-1}$ dry weight) (*7, 8*), while concentrations in other important predatory and prey species (striped bass, *Morone saxatilis*) were much lower. In this study we ask the question: Why did concentrations of Se differ so widely among predators in the Bay, and do those differences still occur? Does food web biomagnification of Se occur, and if so, why is it reflected differently in different predator species? Can stable isotopes be used help characterize different Bay food webs and help understand Se distributions? On the basis of bioaccumulation and stable isotope results, can we suggest what animals might potentially be most threatened by Se, and is there any evidence that effects are occurring in those specific species? Traditional evaluations of the implications of contamination, which include toxicological testing, geochemical speciation, or changes in community structure (*5*), do not address such questions. They may explain acute toxicity, cycling, sources, and bioavailability but not why species differ in their responses. So the issue of which species are most vulnerable to contamination remains poorly known.

Recent work shows that diet can be critical in determining contaminant exposures of animals. Where there is a strong dietary link in contaminant exposures, exposures of top predators can be explained by food web relationships (*9*). Diet dominates Se uptake (*10, 11*), but recent attempts to relate Se distributions to food webs have met with limited success (*12*). We also ask whether that lack of success stems from different processes affecting contaminant uptake by invertebrates at the lower trophic levels.

Experimental Section

Field Sampling. To limit the confounding influences of temporal and spatial variability in comparisons among species, sampling was constrained to a specific geographical area and season. Sampling for Se concentrations in invertebrates and fish was constrained to Suisun Bay and closely contiguous habitat in the northern reach of San Francisco Bay (Figure 1). We assume a homogeneous distribution of Se throughout the study region. Suisun Bay is near the head of the estuary, seaward from the confluence of the Sacramento−San Joaquin River system. It is a major part of the migration corridor and feeding ground for anadromous fish (e.g., Chinook salmon, *Oncorhynchus tshawytscha*; white sturgeon; and striped bass) and seasonally is a nursery area for fish that spawn either in freshwater (e.g., Sacramento splittail, *Pogonichthys macrolepidotus*; striped bass) or the ocean (e.g., Dungeness crab, *Cancer magister*; starry flounder, *Platichthys stellatus*). For this specific study, only samples collected in fall and early winter of 1999/2000 were used.

* Corresponding author phone: (650)329-4550; fax: (650)329-5590; e-mail: arstewar@usgs.gov.
† U.S. Geological Survey.
‡ Nickel Producers Environmental Research Association.
§ Old Dominion University.
|| California Department of Fish and Game.

10.1021/es0499647 CCC: $27.50
Published on Web 08/03/2004

TABLE 1. Bioaccumulation Model for the Crustacean *Neomysis mercedis* and the Bivalve *Potamocorbula amurensis*[a]

Food chain	Species	k_u (L g^{-1} d^{-1})	IR (g g^{-1} d^{-1})	AE (%)	k_e (d^{-1})	C_{ss} (μg g^{-1})
Bivalve	*P. amurensis*	0.003[b]	0.25[b]	45−80[c,d]	0.025[e]	2.1−12
Mysid	Copepods	0.024[f]	0.42[f]	50−53[e]	0.155[e]	0.7−2.2
	N. mercedis	0.027[e]	0.45[g]	73[e]	0.25[e]	0.9−2.7

[a] Mysid model was from diatoms to copepods to mysids, and bivalve model was from diatoms to bivalves. A single dissolved concentration (0.3 μg L^{-1}) and a range of particulate concentrations (0.5−1.5 μg g^{-1}) were used to predict steady-state tissue concentrations ($C_{ss} = ((k_u C_W)/k_e) + ((AE \times IR \times C_F)/k_e)$, where k_u is the dissolved metal uptake rate constant (L g^{-1} d^{-1}), C_W is the dissolved metal concentration (μg L^{-1}), AE is the assimilation efficiency (%), IR is the ingestion rate (g g^{-1} d^{-1}), C_F is the metal concentration in food (μg g^{-1}), and k_e is the efflux rate (d^{-1}). [b] USGS, unpublished data. [c] Ref *27*. [d] Ref *28*. [e] Ref *29*. [f] Ref *25*. [g] Ref *26*.

ships between Se concentrations and trophic level (δ^{15}N) we used linear regression.

Toxicity Thresholds. "Toxicity thresholds" are used in this paper to provide some perspective on the Se concentrations in tissues of both invertebrates and fish. The primary route of Se exposure to fish and invertebrates in nature is diet (*10*, *30*). In field studies, predators (fish) are the most sensitive species in the food web (e.g., ref *31*). The thresholds considered here are only from studies with predators and those that address concentrations that cause or coincide with teratogenesis or reproductive failure (the most sensitive end points). Thresholds for determining toxicity to predators are of two types. Threshold concentrations in food that cause adverse effects in predators and threshold concentrations in tissues of the predators themselves that coincide with the onset of effects. The predators for which the thresholds were derived were not the specific species we studied in San Francisco Bay.

In reviewing existing literature, Lemly (*30*) showed that concentrations of Se greater than 3 μg g^{-1} in the diet of fish result in deposition of elevated Se concentrations in developing eggs, particularly the yolk. Dietary Se concentrations within the range of 5−20 μg g^{-1} load eggs with Se beyond their teratogenic threshold. In the field, extinctions of numerous species of fish were observed in Belews Lake, in association with Se concentrations in invertebrates in the concentration range of 20−80 μg g^{-1} dry weight (*31*). We display a threshold value of 10 μg g^{-1} dry weight as representative of the field/laboratory range.

Lemly (*30*) also listed the proportion of deformities that were observed at different concentrations in fish tissues. In a variety of studies, the appearance of teratogenesis began at 5−10 μg g^{-1} dry weight whole tissue. High proportions of young were deformed above 20 μg g^{-1} dry weight whole tissue. Teratogenesis and reproductive failure consistently began to appear at tissue concentrations in excess of 15 μg g^{-1} dry weight. We chose to display 15 μg g^{-1} dry weight as representative of the threshold concentration in liver of fish. For both food and tissue thresholds, we recognize that the database is limited, the threshold may differ among species, and experts differ somewhat about the exact value representing a threshold (*32*).

Results and Discussion

Selenium concentrations ranged from low to potentially toxic in both invertebrates and fish (Figure 2A,B). Concentrations in lower trophic level crustaceans such as amphipods (*Ampelisca abdita*) ranged from 1 to 3 μg g^{-1} (dry weight) and were as high as 6 μg g^{-1} in zooplankton (although some of these were predaceous; *13*). In contrast, concentrations of Se in the filter-feeding bivalve, *P. amurensis*, were significantly higher than all the crustaceans at 5−20 μg g^{-1} (ANOVA, $P < 0.0001$) (Figure 2A). Suspended particulate Se concentrations in northern San Francisco Bay are relatively low, typically between 0.5 and 1.5 μg g^{-1} (Doblin, unpublished data). Thus, compared to suspended particulate material, Se is significantly biomagnified in *P. amurensis*, slightly biomagnified in zooplankton, and simply accumulated in other crustaceans.

Selenium uptake and elimination kinetics were examined to determine if these rates could explain the marked differences in concentrations seen in the field between clams and crustaceans. Both the bivalve *P. amurensis* and the mysid *N. mercedis* efficiently assimilated Se from their food (AEs > 50%) and accumulated dissolved Se slowly (Table 1). Neither AE nor uptake from solution differed greatly between the two species; so another explanation is needed for the differences in bioaccumulated Se seen in nature. The parameter that differed the most between bivalves and crustaceans was the elimination rate (k_e), which was 10 times lower for *P. amurensis* (0.025 d^{-1}) than for *N. mercedis* (0.25 d^{-1}) (Table 1). Results of the DYMBAM model, using Se concentrations in water and particulate material from Suisun Bay, showed that slower rates of elimination in bivalves resulted in higher steady-state concentrations for bivalves (maximum $C_{SS} = 12$ μg g^{-1}) than mysids (maximum $C_{SS} = 2.1$ μg g^{-1}) (Table 1). The DYMBAM forecasts also agreed reasonably closely with concentrations observed in these species in the Bay.

One physiological mechanism that might explain differences in Se loss between bivalves and crustaceans is the greater tendency of marine bivalves to re-absorb amino acids, or perhaps small proteins, that they lose as a result of catabolism. Selenium primarily occurs associated with proteins in the tissues of organisms and presumably is lost in that form. Wright and Manahan (*33*) and Manahan (*34*) showed direct absorption of dissolved organic material (DOM) or amino acids can occur across body surfaces of many soft-bodied marine invertebrate phyla, including bivalves. But the exception is marine arthropods, for which re-absorption is not efficient. A perhaps related phylogenetic distinction was observed by Schlekat et al. (*28*), who reported strong relationships between AEs and the proportion of Se in algal cell cytoplasm (in the form of dissolved organic selenides) for bivalves (including *P. amurensis*) but not for the amphipod *Leptocheirus plumulosus*.

Selenium concentrations were also highly variable among upper trophic level consumers including crab and fish. Mean liver Se concentrations ranged from 3.6 μg g^{-1} in yellowfin goby to 24 μg g^{-1} in white sturgeon (Figure 2B). Patterns in accumulation among the different species were not related to size or age. For example, larger, older Sacramento splittail (length 18 cm; age 1−2 yr; *35*) like white sturgeon (length 135−171 cm; age 14−20 yr; *36*) accumulated Se beyond the toxicity threshold to levels that have been correlated with adverse reproductive effects; but adult striped bass (length 49−94 cm; 3−10 yr; *37*) had much lower concentrations. Selenium is typically not detoxified in animal tissues by conjugation with metal-specific proteins or association with nontoxic inclusions. So mechanisms that semi-permanently sequester other metals and lead to progressive accumulation with size or age are not known for Se.

To determine whether differences in Se concentrations among fish could be explained by food-related variables, we examined feeding relationships among biota from San Francisco Bay. Stable isotope results were consistent with known dietary habits and gut-contents studies of the species collected and together were used to identify two crustacean-based and one clam-based food web along the salinity gradient (Figure 3). In Figure 3, ellipses enclose animals thought to be in similar food webs from knowledge of their

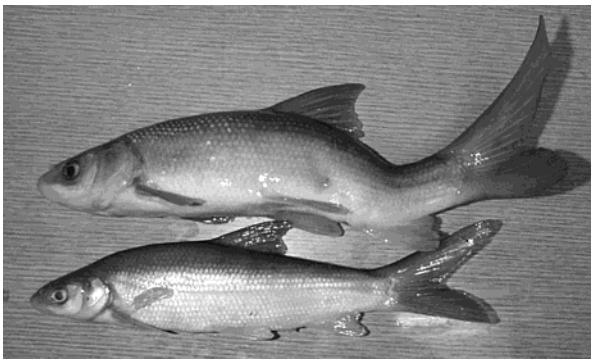

FIGURE 5. Sacramento splittail collected from North San Francisco Bay, CA, in 1999 displaying lordosis, a selenium-induced teratogenic deformity. Photo taken by Fred Feyrer, California Department of Water Resources.

no other studies that demonstrate, in the field, specific differences in Se uptake among primary consumers propagating to differences in contaminant accumulation at the top of the food web. In San Francisco Bay, processes that control Se uptake at the base of the food web appear to be the dominant factor controlling which species among top predators are exposed to the highest concentrations of this potential toxin.

The clam food web was also of the same length or shorter than either of the crustacean food webs (clam food web $\Delta\delta^{15}N = 4.9$; crustacean food web $\Delta\delta^{15}N = 7.6$). Therefore, food web length was not the most important factor determining Se concentrations in top predators. These results are contrary to those of other biomagnifying contaminants such as polychlorinated biphenyls, DDT, or mercury, which identify food chain length or carbon source as being a critical factor controlling concentrations in top predators (*50*, *51*). Additional anecdotal evidence highlights the relative importance of food web length and food web base concentrations in determining Se exposures. One exceptional white sturgeon (of 37 collected over 2 yr) appeared to be piscivorous, based upon $\delta^{15}N$ (18.75‰ vs a group mean = 15.65). That individual had the lowest liver Se level (5.5 $\mu g\ g^{-1}$, group mean = 22 $\mu g\ g^{-1}$). Conversely, this same sturgeon had the highest muscle mercury level (3.68 $\mu g\ g^{-1}$, dry weight; group mean = 1.12 $\mu g\ g^{-1}$) relative to other sturgeon. High Se concentrations were also observed in Dungeness crab (22 $\mu g\ g^{-1}$). Loss rates of Se are not known for this species, but it is a crustacean. It also preys upon clams; and, like other species in that food web, the clams are likely the source of the elevated Se. So food chain length can play a role in magnifying concentrations from one trophic level to the next (Se concentrations increase from clams to their predators), but that process only enhances the most significant increase resulting from enhanced uptake at the base of the food web.

A principal effect of Se is teratogenicity. Deformities occur in developing embryos when Se replaces sulfur in sulfur-rich hard tissues (*52*). Recent field surveys identified Sacramento splittail from Suisun Bay (where Se concentrations are highest) that have deformities typical of Se exposure (Figure 5). This suggests a toxicologic threat in at least some individuals of an important native species that has been listed under the Endangered Species Act (50 CFR Part 17).

Variable exposures among species complicate interpreting the influences of contaminants in nature. For this reason traditional approaches to understanding the environmental threat of contaminants may not be appropriate for Se. Biomagnification in San Francisco Bay makes upper trophic levels most vulnerable to Se effects. But unlike other contaminants that biomagnify, differences in the kinetics of uptake and loss at the first trophic step (clams and crustaceans) and propagation of those differences up trophic pathways cause some predators to be more exposed than others to Se. Presumably, this can influence what species might be most likely to disappear from a moderately contaminated environment. Similar principles may apply to other contaminants where diet is an important route of exposure (*49*). Combining ecological and environmental toxicological approaches, at the ecosystem level, with mechanistic laboratory experimentation, may help understand if effects of chemicals such as Se are going undetected in natural populations from such environments.

Acknowledgments

We thank D. W. Schindler, N. S. Fisher, G. A. Cutter, M.-N. Croteau, and Theresa Presser for their helpful comments on the manuscript and the USGS Menlo Park Metals Bioavailability Laboratory and crew of the R/V *Polaris* for assistance with sample collection. Research was supported by grant to S.N.L. and others from the CALFED Bay Delta Program (CALFED 98-2015000-00096), as well as from the NSF EGB Program (NSF Grant OCE-9707946) to G. A. Cutter.

Literature Cited

(1) Larison, J. R.; Likens, G. E.; Fitzpatrick, J. W.; Crock, J. G. *Nature* **2000**, *406*, 181−183.
(2) Presser, T. S. *Environ. Manage.* **1994**, *18*, 437−454.
(3) Presser, T. S.; Ohlendorf, H. M. *Environ. Manage.* **1987**, *11*, 805−821.
(4) Ohlendorf, H. M.; Hoffman, D. J.; Saiki, M. K.; Aldrich, T. W. *Sci. Total Environ.* **1986**, *52*, 49−63.
(5) Cutter, G. A. *Estuarine Coastal Shelf Sci.* **1989**, *28*, 13−34.
(6) Johns, C.; Luoma, S. N.; Elrod, V. *Estuarine Coastal Shelf Sci.* **1988**, *27*, 381−396.
(7) Lemly, A. D. In *Environmental Contaminants in Wildlife*; Beyer, W. N., Heinz, G. H., Redmon-Norwood, A. W., Eds.; CRC Press: Boca Raton, FL, 1996; Chapter 19, pp 427−446.
(8) Skorupa, J. P. In *Environmental Chemistry of Selenium*; Frankenberger, W. T., Jr., Engberg, R. A., Eds.; Marcel Dekker: New York, 1998; Chapter 18, pp 315−354.
(9) Kidd, K. A.; Schindler, D. W.; Muir, D. C. G.; Lockhart, W. L.; Hesslein, R. H. *Science* **1995**, *269*, 240−242.
(10) Luoma, S. N.; Johns, C.; Fisher, N. S.; Steinberg, N. A.; Oremland, R. S.; Reinfelder, J. R. *Environ. Sci. Technol.* **1992**, *26*, 485−491.
(11) Lemly, A. D. In *Environmental Chemistry of Selenium*; Frankenberger, W. T., Jr., Engberg, R. A. Eds.; Marcel Dekker: New York, 1998; Chapter 16, pp 281−296.
(12) Jarman, W. M.; Hobson, K. A.; Sydeman, W. J.; Bacon, C. E.; McLaren, E. B. *Environ. Sci. Technol.* **1996**, *30*, 654−660.
(13) Purkerson, D. G.; Doblin, M. A.; Bollens, S. M.; Luoma, S. N.; Cutter, G. A. *Estuaries* **2003**, *26*, 956−969.
(14) Linville, R. G.; Luoma, S. N.; Cutter, L.; Cutter, G. A. *Aquat. Toxicol.* **2002**, *57* (1−2), 51−64.
(15) Brown, C. L.; Luoma, S. N. *Mar. Ecol. Prog. Ser.* **1995**, *124*, 129−142.
(16) Peterson, B. J.; Fry, B. *Annu. Rev. Ecol. Syst.* **1987**, *18*, 293−320.
(17) France, R. L. *Limnol. Oceanogr.* **1995**, *40* (7), 1310−1313.
(18) Spiker, E. C.; Schemel, L. E. In *Distribution and Stable-Isotope Composition of Carbon in San Francisco Bay*; Conomos, T. J., Ed.; American Association of Advanced Science: 1979; pp 195−212.
(19) Cabana, G.; Rasmussen, J. B. *Proc. Natl. Acad. Sci. U.S.A.* **1996**, *93*, 10844−10847.
(20) Vander Zanden, M. J.; Rasmussen, J. B. *Ecology* **1999**, *80* (4), 1395−1404.
(21) Cutter, G. A. *Anal. Chem.* **1985**, *57*, 2951−2955.
(22) Luoma, S. N.; Fisher, N. In *Ecological Risk Assessments of Contaminated Sediments*; Ingersoll, C. G., Dillon, T., Biddinger, G., Eds.; SETAC Press: Pensacola, FL, 1997; pp 211−239.
(23) Schlekat, C. E., Lee, B.-G.; Luoma, S. N. In *Coastal and Estuarine Risk Assessment*; Newman, M. C.; Roberts, M. H.; Hale, R. C. Eds.; CRC Press: Boca Raton, FL, 2001; pp 151−188.
(24) Wang, W. X.; Fisher, N. S. *Sci. Total Environ.* **1999**, *237−238*, 459−472.
(25) Wang, W.-X.; Fisher, N. S. *Limnol. Oceanogr.* **1998**, *43*, 273−283.
(26) Johnston, N. T.; Lazenby, D. C. *Can. J. Zool.* **1982**, *60*, 813−824.
(27) Schlekat, C. E.; Dowdle, P. R.; Lee, B.-G.; Luoma, S. N.; Oremland, R. S. *Environ. Sci. Technol.* **2000**, *34*, 4504−4510.