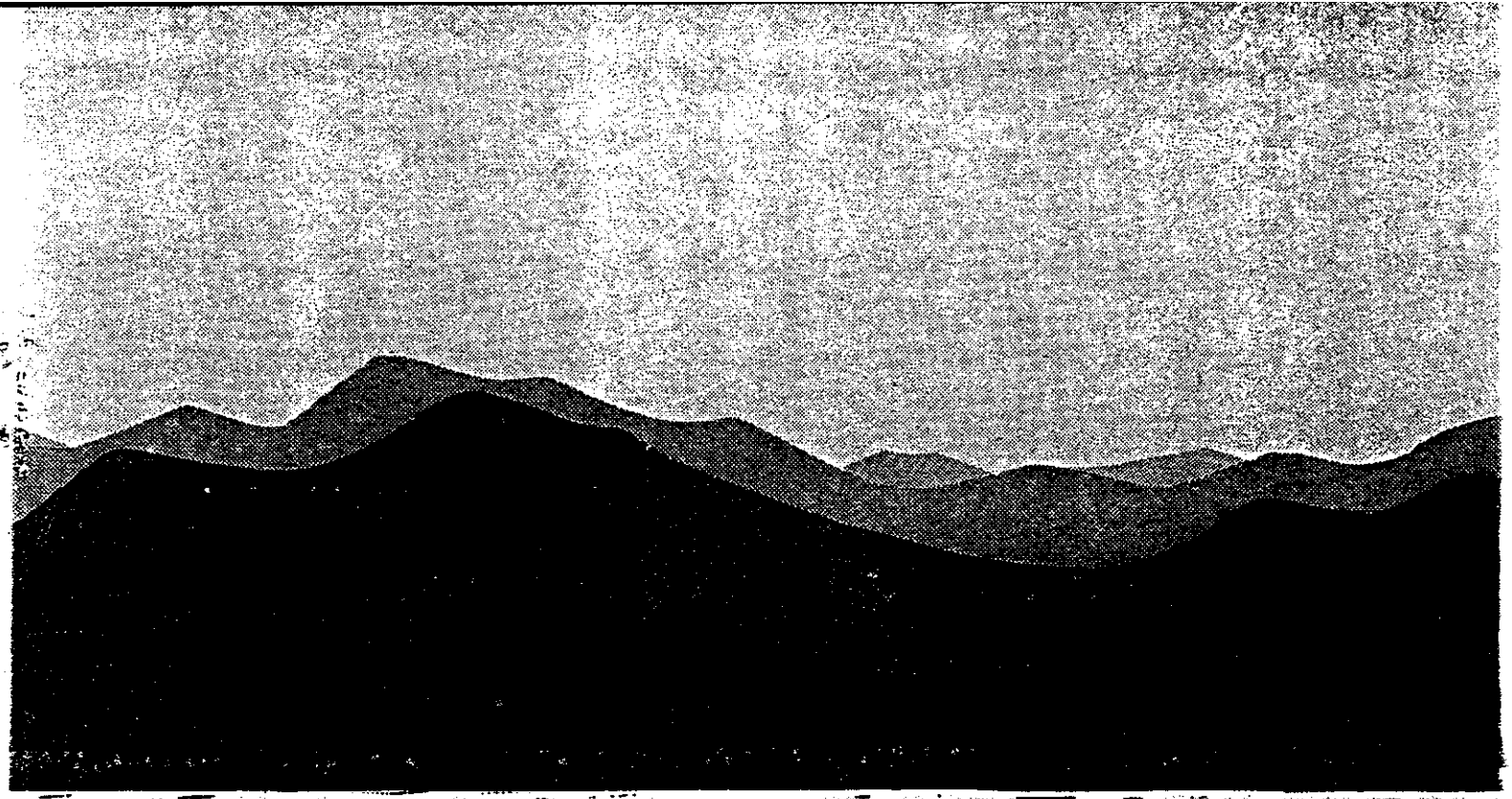

POLYCHLORINATED BIPHENYLS (PCBs)

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PREFACE

This is the second in a series of reports issued by the State Water Resources Control Board on industrial and agricultural chemicals. These reports deal with priority chemicals of concern to water quality and the protection of beneficial

[redacted]

Industrial Chemicals program based on the premise that the production and use of chemicals should not occur at the expense of water quality protection.

Chemicals are of inestimable value to society, and most are considered relatively safe under normal conditions of use. There are some chemicals whose environmental and health effects have been proven harmful. The possibility that toxic chemicals in the environment can cause cancer in humans and severely impair the health of wildlife has led to increased action by government to foster the safe use and disposal of these chemicals.

The chronic effects of persistent chemicals (e.g. impaired growth and reproduction) may be more devastating in the long run than immediately apparent effects, such as fish kills. Preventative measures are invariably less costly to society than corrective actions required after toxic chemical pollution has occurred.

Some current chemical use and disposal practices may have an adverse impact on water quality. These activities can usually be modified to minimize adverse environmental effects. Where existing or potential water quality problems have been identified, the State Board will recommend appropriate measures to correct or prevent such adverse impacts.

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SUMMARY

PCBs are found in air, water, sediment and biota throughout the world. Because they are ubiquitous, PCBs have been described as constituents rather than contaminants of the environment. PCBs are toxic substances. The information and analyses presented in this report conclude that PCBs are hazardous to humans and aquatic life. Prudence dictates that human contact with these chemicals should be minimized. Further dissemination into the environment of these long-lived compounds should be prevented.

Properties and Uses

PCBs are chlorinated hydrocarbons that are chemically stable and have low flammability, high boiling point, and low electrical conductivity. PCBs have been used as fluids in electrical transformers, capacitors and electromagnets, and heat transfer and hydraulic systems. They have also been used as plasticizers in paints, adhesives, and caulking compounds; fillers for investment casting waxes; and dye carriers in carbonless copy paper.

Of the 1.4 billion pounds of PCBs produced in the United States since 1929, over half (750 million pounds) are still in service. Monsanto, the major PCB producer, voluntarily reduced production in 1970. Before 1970, 60 percent of PCB sales were for "closed" uses (electrical and heat transfer systems), while the remaining 40 percent were for "open " uses (plasticizers, hydraulic fluids and lubricants). By 1972, all PCB production was for closed systems. The Toxic Substances Control Act (TSCA) of 1976 specifically banned the manufacture of new PCBs and prohibited the use of PCBs except in a "totally enclosed" manner or where specifically exempted. Approximately one-quarter (162 million pounds) of the PCBs still in service are used by electrical utilities in transformers and capacitors.

All commercial PCB formulas contain contaminants, such as polychlorinated dibenzofurans (PCDFs). These contaminants are more toxic to humans and aquatic life than PCBs.

Environmental Fate

Airborne transport and deposition is the major pathway for the global distribution of PCBs and other persistent chlorinated hydrocarbons. Over 90 percent of the PCB input to Lake Superior is attributed to atmospheric fallout. This extreme persistence, coupled with the resuspension of PCB-contaminated sediments in the Great Lakes, could cause their recycling through the aquatic food chain for up to 150 years. Breakdown of PCBs by biological degradation or photodecomposition is extremely slow.

With extremely low water solubility and high affinity for organic material, PCBs

Mammalian Toxicology and Human Health

In the past two years, controversy has risen over whether PCBs pose a significant risk to human health. The most recent industry-sponsored reviews by Drill et al. (1982) and Ecology and Environment, Inc. (1981) as well as the 1982 USEPA response to these reviews are in substantial agreement on the subject of how PCBs are absorbed, distributed throughout the body and excreted. Disagreement centers around the degree of toxicity of the commercial mixture. This report stresses that PCB mixtures are a combination of many different chlorinated biphenyl compounds (isomers), each having different chemical and physical properties. These compounds have a wide range of potential toxicities. The structure (location of chlorine atoms and degree of chlorination of the biphenyl molecule) of individual PCB isomers affects all metabolic processes and toxicity. The following table summarizes the scientific disagreement.

Although PCBs are not acutely toxic to mammals except at high doses, their chronic effects are pronounced at relatively low doses, particularly in the liver and in reproductive systems. Mink and rhesus monkeys are the more sensitive mammalian species compared to mice, rats and rabbits. PCBs have been shown to have adverse effects in the sensitive species at levels lower than the U.S. FDA action level of 5 ppm in some foods.

Reproduction

Drill et al. (1982) and the USEPA Response (1982) differ in their assessments of the effects of PCBs on reproduction: while the former believes that adverse reproductive effects occur only at high doses, the USEPA Response states that reproductive effects can be observed at low levels, sometimes at lower doses than where other manifestations of toxicity are measured. Adverse impacts on reproduction by PCBs were observed in the following feeding studies:

1. In rats, a dietary level of 20 ppm Aroclor 1254 reduced the number of offspring (Linder et al., 1974).

2. Mink showed total reproductive failure at 5 ppm (Kimbrough et al., 1978), and Aroclor 1254 affects reproduction at 2 ppm (Auerlich and Ringer, 1977).

3. The rhesus monkey showed adverse effects at levels of 2.5 ppm Aroclor 1248 (Allen and Barsotti, 1976).

Animal Carcinogenicity

The USEPA Response (1982) disagrees with the reviews by Drill et al. (1982) and Ecology and Environment, Inc. (1981) on the issue of whether PCBs are animal carcinogens. Drill et al. (1982) have concluded that evidence of PCBs acting as carcinogens is weak, at best. Ecology and Environment, Inc. (1981) concluded that PCBs are perhaps weak promoters of cancer, but are not initiators. In contrast, the USEPA Response (1982) supports the International Agency for Research on Cancer (IARC) position that there is experimental evidence that PCBs are carcinogens in some species of rats and mice.

Ten PCB animal bioassays have been reported for cancer effects (two in mice, seven in rats, and one in the dog). These include abstracts and unpublished studies. These bioassays show that commercial PCB mixtures are carcinogenic in both male mice and female rats. A recent abstract (Weltman and Norback, 1982) reports that Aroclor 1260 is carcinogenic in male as well as female rats. Other experimental studies have been reported as negative.

TOXICOLOGY OF PCBs: AREAS OF SCIENTIFIC DISAGREEMENT

EFFECT	DRILL, FREISS et al., (1982)	ECOLOGY AND ENVIRONMENT INC.(1981)-CMA	EPA RESPONSE (1982)	SWRCB POSITION
Reproduction	At "relatively high doses" (examples include those fed to monkeys).	Little effect at "moderate-to-high doses".	At doses that may be lower than for other toxic effects.	Rats - 20 ppm Minks - 1-5 ppm Monkeys - 2.5 ppm (FDA action level is 5 ppm for some foods.)
Carcinogenicity	No convincing evidence.	Weak promoters but not direct-acting carcinogens.	Positive	• Positive based on different interpretation of same studies reviewed by Drill et al. (1982) and EEI (1981). • Recent positive rat bioassays. • Positive based on re-evaluation of major studies.
Liver Enzyme Systems Stimulation	Cytochrome P-450 system only	Cytochrome P-450 and P-448 systems.	Cytochrome P-450 and P-448 systems.	• Cytochrome P-450 and P-448 systems. • Cytochrome P-448 stimulation exerts toxicity at same receptor site as 2,3,7,8-TCDD and 2,3,7,8-PCDF.
Occupational/ Epidemiological Studies	Reversible effects only (chloracne, liver enzyme stimulation).	Reversible effects.	Studies not sensitive enough.	Insufficient data; studies inadequate to identify cancer risk and other low incidence or chronic effects.
Yusho (1968) and Taiwan (1979) Rice Oil Poisonings	Unique incident due to contaminants.	Due to contaminants.	Probably due to combination of contaminants and PCBs.	Major effects probably due to contaminants.
Contaminants (chlorinated dibenzofurans, etc.)	Not assessed, but highly toxic.	Not assessed but potential hazard.	Present in all commercial PCB mixtures, not feasible to remove contaminants.	Highly toxic, must be considered in any assessment of commercial PCBs.

There is disagreement among reviewers over interpretation of data from the cancer bioassays. Issues raised include: (1) adequacy of study design; (2) interpretation of tissue lesions; (3) benign versus malignant tumors; and (4) relative significance of positive and negative findings. However, the available data suggest that at least some commercial PCB mixtures are animal carcinogens.

Mutagenicity

Although mutagenicity is highly correlated with carcinogenicity, Letz (1981) has observed that many of the highly chlorinated animal carcinogens are negative in short-term tests for mutagenicity. Both Drill et al. (1982) and Ecology and Environment, Inc. (1981) state that there is no significant evidence that PCBs are mutagenic in test systems. The USEPA Response (1982) notes that since a few tests have suggested that PCBs have mutagenic potential, the issue of mutagencity is still open and more data are needed. Thus, a lack of mutagenic potency by PCBs in short-term tests may not be appropriate for assessment of carcinogenic potential.

Stimulation of Liver Enzyme Systems

A new and important area of toxicological research involves biochemical stimulation by PCBs of certain enzyme systems in the liver. This work has shown some PCB

Aquatic Toxicology

Fish can accumulate PCBs up to one million times the concentrations found in water. The impact of PCBs on aquatic life is pronounced at very low concentrations in water. Fish subjected to low-level chronic exposure (1-2 ppb PCB in water) show decreased reproductive ability and decreased viability of eggs and fry. Early life stages of fish and invertebrates are more sensitive to PCBs than adult stages. The reproductive success and propagation of existing fisheries is highly vulnerable to parts-per-million levels.

The biological activity of PCBs depends upon the number of chlorine atoms and their position on the biphenyl rings. As in the case of human toxicity, assessing PCB aquatic toxicity is complicated by the presence in commercial mixtures of contaminants (e.g., dibenzofurans), which are also highly toxic to aquatic organisms.

Criteria and Standards

The 1980 EPA ambient water criterion to protect human health from consumption of PCB-contaminated fish, shellfish, and water is 0.079 parts per trillion (ppt) in water (using a one-in-one-million cancer risk level). EPA also developed two corresponding criteria to protect aquatic life. The 24-hour average criteria for freshwater and saltwater, respectively, are 14 ppt and 30 ppt. The National Academy of Science (1980) has recommended "Suggested No-Adverse Response Levels" (SNARL) for PCBs in drinking water of 350 ppb (24-hour exposure) and 50 ppb (7-day exposure).

The National Academy of Sciences (1973) proposed a 0.5 ppm criterion in fish tissue to protect consumers of contaminated fish. The Food and Drug Administration (FDA) has established action levels to control PCB levels in food. The action levels represent limits at or above which FDA will take legal action to remove adulterated products from the market. The FDA action level in fish is 5 ppm.

At the state level, the State Water Resources Control Board (SWRCB) established effluent limitations for total chlorinated pesticides and PCBs in the California Ocean Plan. The limiting concentrations for the 6-month median, daily maximum, and instantaneous maximum are, respectively: 2.0 ppb, 4.0 ppb, 6.0 ppb.

Presence of PCBs in California

Since 1978, PCB findings in fish by the State Board's Toxic Substances Monitoring (TSM) Program have been infrequent (PCBs were detected at 12 out of 44 stations) and at low levels (only one station exceeded the NAS guideline). Of the 28 streams sampled in the TSM Program since 1978, PCBs have been detected in each of three rivers (Sacramento, Feather, Santa Ana) at least twice. In 1977 and 1978, the TSM Program detected PCB concentrations above 500 ppb in fish on the lower Feather River at Nicolaus. An intensive follow-up survey of the Ponderosa Reservoir in 1980-81 found PCB levels as high as 7,700 ppb in suckers (1980). The State Board's Cooperative Striped Bass Study (COSBS) found PCB concentrations (ppm-wet weight) in ovaries (0.30-5.43), testes (0.06-0.60), liver (1.03-4.01) and muscle (0.36-1.36) from adult pre-spawning striped bass. PCB concentrations in Delta bass fillets collected in 1980 and 1981 were below the FDA action level of 5 ppm but did exceed the IJC 0.1 ppm limit. PCB levels in most prespawning Delta bass ovaries equalled levels representing approximately 25 percent mortality in Atlantic Salmon eggs.

Since 1977, the State Board's Marine Monitoring Program (Mussel Watch) has analyzed mussels from coastal and bay areas for PCBs. Data from most of the monitoring stations indicate that PCB levels have stabilized at relatively high concentrations. Stations with elevated levels include San Diego Harbor (7,300 ppb), Los Angeles-Long

Beach Harbor (2,000 ppb), and Newport Harbor Bay (5,000 ppb). The major exception

RECOMMENDATIONS

Despite the ban on manufacture and the restrictions on use, the known and potential impacts of PCBs on California's water quality are numerous and serious. There is no feasible method for removing all PCBs from the environment. The proposed State Board strategy for controlling PCBs is to (1) minimize their impairment of beneficial uses, and (2) ensure their safe handling, storage and disposal. To implement this strategy, the State Board has developed specific recommendations for consideration by affected state and federal agencies. The rationale is presented after the recommendations, which are listed below:

State Water Resources Control Board

1. Adopt a statewide policy for PCBs to:
   "Prohibit the discharge of PCBs to water from controllable sources."

2. Adopt a statewide PCB control program that includes:

   (a) Minimum Requirement for fish and shellfish consumption:

   - Fish Tissue: 5 ppm

   (b) Response Levels that trigger source identification and feasible control measures:

   - Fish Tissue: 0.5 ppm
   - Sediment: 1 ppm
   - Effluent: 1 ppb
   - Ambient Water: 15 ppt

   (c) Guideline for spill and abandoned waste clean-up:

   - 50 ppm

   (d) Reportable Quantity for spills:

   - 1 lb.

3. Focus PCB water quality monitoring on tissue and sediment analysis.

4. Conduct intensive water quality monitoring of identified "hot spots":

   - Survey location of PCB-containing equipment before selecting sampling sites (fish and mussels).

   - Monitor for PCB and major contaminants (e.g., chlorinated dibenzofurans).

5. Develop a procedures manual for RWQCB and other field staff on the state's PCB waste management program.

Department of Health Services

1. Improve coordination between DHS and the State and Regional Boards to ensure that a high priority is given to the protection of beneficial uses of water from PCB contamination. Shared functions should include: review of annual PCB facility inventories, PCB disposal facility permits, and spill prevention countermeasure plans; facility inspections; and spill investigations.

2. Implement the regulation which bans land disposal of PCB liquids over 50 ppm after January 1, 1984.

3. Approve privately owned and operated PCB destruction processes and facilities in California.

4. Request authority from EPA to prohibit the use of PCB-containing small capacitors that threaten water quality, food, or feed in state facilities after October 1, 1985.

Department of Health Services and Office of the State Architect

1. Seek state legislation to provide funding to:

   o Implement the PCB management program proposed by the Office of the State Architect for state facilities.

   o Replace PCB-containing small capacitors that pose a threat to water quality, food, or feed in state facilities.

U.S. Environmental Protection Agency

1. Authorize the State Department of Health Services to prohibit the use of PCB-containing small capacitors that pose a threat to water quality, food, or feed in state facilities after October 1, 1985.

2. Amend the PCB regulation relating to disposal facilities to incorporate performance standards that permit on-site encapsulation of contaminated soil.

3. Support research on:
   o Human and animal toxicology of individual PCB isomers to further assess the health effects.

   o Alternative PCB treatment technologies, including microbial degradation.

Department of Industrial Relations

1. Adopt the proposed occupational health standard on PCBs.

Major California Utilities

1. Establish a policy of requiring the installation of failure warning devices on switched large PCB capacitor banks that lack secondary containment or restricted access.

Rationale for Recommendations

The preceding recommendations are based on the scientific evidence that PCBs are hazardous to humans and aquatic life. Federal and state regulatory programs for PCBs are in place. Some of the recommendations support the continuation of existing control measures. Others propose changes in the Federal PCB regulations. The recommendations also address California's expanding hazardous waste management program.

Water Quality Strategy

The State and Regional Board's traditional approach to setting water quality objectives assumes that the discharge of pollutants can be regulated. This traditional approach is in most cases impractical for PCBs. First, receiving water objectives for PCBs are generally inappropriate. PCBs have extremely low water solubility. When present in the aquatic environment, they concentrate in sediment and fish tissue. Reliance on receiving water objectives could underestimate the threat that PCBs pose to beneficial uses. Second, protective receiving water levels often cannot be established for PCBs. Many PCB concentrations found in California fish tissue and sediment currently exceed levels necessary to protect human health and aquatic life. Third, most PCB discharges from point sources have ceased as a result of the EPA ban on manufacture and restriction of use. Fourth, site-specific environmental and economic factors determine the degree to which PCB sources can be controlled. While some PCB sources are relatively controllable, others may not be, because of the environmental damage that the clean-up operation would cause or the cost of pollutant removal and disposal.

PCB sources consist of PCB-containing electrical equipment and PCB spills and abandoned wastes. EPA and DHS regulate PCB-containing electrical equipment, and the major California utilities have developed PCB management programs. Therefore, this strategy focuses on PCB spills and abandoned wastes.

Controllable sources are spills and abandoned wastes for which clean-up is environmentally and economically feasible. Environmental and economic feasibility will be determined on a case-by-case basis. The evaluation will indicate whether the PCB source can be 1) eliminated, 2) encapsulated to prevent environmental contamination, or 3) mitigated to reduce the level of environmental contamination. This flexible and discretionary approach is the only practical way to determine appropriate remedial action.

This strategy (1) adopts as a minimum requirement the current U.S. Food and Drug Administration action level of 5 ppm PCBs in fish flesh; (2) identifies PCB response levels in sediment (1 ppm), fish tissue (0.5 ppm), effluent (1 ppb), and ambient water (15 ppt) to trigger source identification and control; and (3) sets a 50 ppm soil or sediment spill clean-up guideline which can be modified depending on water quality, technical and economic considerations.

The proposed ambient and effluent response levels, 15 ppt and 1 ppb respectively, are based on detection limits achieved in routine sampling programs. The sediment level of 1 ppm is based on bioconcentration factors observed in California fish and the water solubility of PCBs.

The fish tissue response level of 0.5 ppm represents a compromise between recommended criteria and California monitoring data. In 1972, the National Academy of Sciences proposed the 0.5 ppm fish tissue level to protect wildlife predators. Data on PCB levels in fish tissue from the State Board's Toxic Substances Monitoring Program range between 0.1 and 7.7 ppm. A very low (i.e., stringent) response level could trigger source identification and control for every sample taken. The proposed response level strikes a regulatory balance that provides a basis for establishing priorities in the handling of PCB problems.

The response levels are indicators of water quality problems. They are not meant to protect beneficial uses, but rather to serve as an early warning signals that trigger further investigation. It may not be possible, in some cases, to reduce concentrations below the response levels. However, responsible parties should attempt to cleanup as much of the contamination as is technologically, environment-

ally, and economically feasible. Where feasible, cleanup or source control should achieve levels that adequately protect beneficial uses. These levels would always be more strigent than the response levels.

The proposed revision to the California Ocean Plan exemplifies an approach to regulating PCB discharges for the protection of beneficial uses. This approach is compatible with the proposed PCB policy. The draft Ocean Plan recommends a marine water quality objective of 3 ppt based on the chronic toxicity of PCBs to aquatic life. For its purpose, which is to derive effluent limitations for the regulation of point source discharges, this is an appropriate use of a receiving water objective for PCBs.

The spill cleanup guideline is 50 ppm in soil or sediment, the minimum level at which DHS requires disposal in EPA approved chemical waste landfills. The 50 ppm level is a guideline as opposed to a minimum requirement, to provide regulatory flexibility and discretion. In some cases, clean-up more stringent than 50 ppm may be warranted. In other cases, clean-up down to 50 ppm may be infeasible.

The federal Superfund law (CERCLA) adopts by reference the 10-pound reportable quantity established under Section 311 of the Clean Water Act. SWRCB set a reportable quantity of one pound because major utilities are already reporting all spills to DHS. This smaller reportable quantity seemed more consistent with the informal DHS reporting policy than the larger quantity.

Federal Regulation of PCBs

The key elements of the federal program developed to implement the 1979 PCB regulation included:

(1) Ban PCB manufacture;
(2) Permit totally enclosed (and other exempted) uses;
(3) Establish labeling, storage, inspection, spill response and disposal requirements; and
(4) Require the destruction of PCB wastes containing 500 ppm PCB or more.

In response to the U. S. Court of Appeals' order, EPA has issued revised regulations on August 25, 1982, and October 21, 1982, which:

(1) Permit minor production of PCBs (incidental to manufacturing processes) where insignificant release to the environment occurs;
(2) Permit nontotally enclosed use of PCBs in electrical equipment, previously classified as totally enclosed, with some stricter inspection requirements;
(3) Require phase-out of certain PCB transformers and large PCB capacitors in 3 and 6 years, respectively.

Additional controls on the use of PCB-containing capacitors are necessary based on the threat that extremely low levels of PCBs pose to humans and aquatic life. For large switched bank capacitors that lack restricted access or secondary containment, failure detection devices should be installed. This recommendation can be implemented on a voluntary basis (See the discussion below related to Major California Utilities).

For small capacitors, SWRCB believes that accelerated phase-out is warranted for equipment that poses a threat to water quality, food, or feed. According to the pre-emption provisions of the Toxic Substances Control Act, California must request an exemption in order to regulate the use of PCB-containing equipment differently from EPA's program. SWRCB proposes that DHS, the lead state agency for PCB regulation, seek this additional authority.

State Management of PCBs and Other Hazardous Waste

State and federal superfund monies have been appropriated for PCB spills and abandoned hazardous wastes that meet the eligibility requirements. The DHS program to implement the PCB regulations is administered by its Hazardous Materials Management Branch.

The expansion of the state hazardous waste management program must address other state agencies with related responsibilities. In particular, the State Water Resources Control Board and Regional Water Quality Control Boards must be integrated into this program. These agencies share responsibility for protecting ground and surface water quality, including land disposal affecting water quality. Coordination between DHS and the State and Regional Boards is especially crucial because most controllable PCB releases to the environment come from 1) sediment deposits in water and 2) spills or abandoned hazardous wastes that contaminate soil or water.

Destruction of PCBs is the safest disposal option. The Air Resources Board has adopted a policy which established incineration standards that limit release of PCBs and their highly toxic contaminants (e.g., dibenzofurans and dibenzodioxins) to the atmosphere.

The Department of Industrial Relations (DIR) has proposed an occupational health standard for PCBs. This proposed standard addresses protective equipment and clothing, respiratory protection, work practices and employee decontamination. It also includes occupational safeguards that are intended to protect workers from harmful exposure to PCBs.

Inspection of Utility-Owned Large PCB Capacitors

The August 25, 1982, PCB regulation eliminates inspection requirements for all large PCB capacitors based on the fact that visual inspections cannot provide evidence of potential leaks. However, California utilities have developed failure detection devices which can be installed on switched bank capacitors. These devices, such as Southern California Edison's capacitor unbalance sensing relay, monitor temperatures within the capacitors to predict imminent failure. The utility industry experience with these failure detection devices indicates that they are 90 percent effective.

Because failure of PCB capacitors could release PCBs to the environment and the

The safest PCB disposal alternative is chemical destruction. There is limited landfill capacity for PCBs in California. The State should support the establishment of privately owned and operated PCB destruction facilities in California. Such facilities would reduce environmental hazards and the costs of transporting PCB wastes for out-of-state disposal.

The requirement for disposal of other PCB wastes in EPA-approved chemical waste landfills should be modified to allow on-site encapsulation under certain circumstances. Destruction of PCB-contaminated soil may be infeasible if very large volumes of soil are involved.

At the present time, federal regulations for PCB waste disposal in landfills preclude on-site encapsulation. Although DHS plans to encapsulate contaminated soil at several illegal PCB dump sites, these sites do not presently meet federal or state requirements for PCB disposal. The federal design criteria should be modified to permit encapsulation of PCB wastes to minimize migration. Where water contamination occurs, treatment and other control measures must be provided to protect beneficial uses.

State Legislation

The risk that PCBs pose to aquatic life and humans is serious enough to warrant preventing additional discharges to the environment wherever controllable.

SWRCB proposes that the State Legistature allocate funds to (1) implement the PCB management program proposed by the Office of the State Architect (OSA) and to (2) accelerate phase-out of PCB-containing small capacitors that threaten water quality, food, or feed in state facilities.

The OSA report on PCB equipment in state facilities proposed four alternatives for PCB management. The State Legislature adopted the recommended minimum program, which entails: (1) replacing leaking PCB equipment, (2) retaining PCB equipment in good condition, and (3) building temporary storage facilities for equipment removed from service. Additional funds are required to implement this program in all state facilities. The OSA has prepared a budget change proposal to carry out this recommendation.

Most small capacitors are located in non-utility facilities, including federal, state and local government installations and private industries and businesses. The rupture of small capacitors at the Sonoma State Hospital in 1981 highlighted the need for additional controls over this type of PCB-containing equipment. Because there are numerous small PCB-containing capacitors in locations which pose a health risk, SWRCB recommends gradual accelerated replacement starting with state facilities. This replacement program in state facilities will serve as a model for other facilities regulated by the state and local health departments.

Research

Additional research is needed in both the scientific and technological areas. Further toxicity studies of individual PCB isomers must be performed to resolve the persent controversy surrounding the human health effects of PCBs.

Regarding PCB treatment technology, additional research on microbial degradation is needed to develop this technique for use under field conditions. Further testing of alternatives to incineration should also take place to provide as many disposal options for PCBs as feasible.

1.0 INTRODUCTION

Polychlorinated biphenyls are chlorinated hydrocarbons which were commercially synthesized beginning in the 1920s. Their chemical stability, low flammability, high boiling point, and low electrical conductivity made them useful as fluids in electrical transformers, capacitors and heat transfer and hydraulic systems. Between 1929 and 1978 over 700 million tons of PCBs were manufactured in the United States. Worldwide production in 1970, the year when U.S. production peaked, was estimated at 110,000 tons (Risebrough and DeLappe, 1972). In addition to the U.S., other major producers of PCBs were France, Germany, Italy, Japan and the Soviet Union. Of the total volume of U.S.-manufactured PCBs, one half is still in service. The remainder is disseminated throughout the environment in air, water, soil and living organisms.

Until the mid-1960s, PCBs were believed to be inert and harmless. In 1966 Swedish scientists found PCBs in the tissues of fish from the Baltic Sea. The PCB concentrations in fish far exceeded levels identified in the water. Later evidence has established PCBs as ubiquitous components of the global environment. They have been found in seals from the North Atlantic, polar bears in the Arctic, fish in Peruvian waters, and birds from such diverse locations as Great Britain, New Zealand, Alaska, Chile, and Antarctica (Risebrough and DeLappe, 1972). The source of much of this dissemination is atomspheric fallout. For example, the presence of PCBs in freshwater fish of tundra lakes can only be explained by aerial contamination.

The risk that PCBs pose to fish and mammals consists mainly of long-term adverse effects. Chronic toxicity of PCBs to fish is well established. Fish subjected to low-level long term exposure (1-2 ppb in water) are less fertile and their eggs and fry suffer higher mortality rates. In 1969, with the discovery and description of the "Yusho" disease epidemic in Japan, the presence of PCBs in man was documented.

Even today, there is considerable controversy as to the effects of PCBs in humans. Government scientists agree that they pose a significant health risk. Industry scientists argue that the observed adverse effects result from contaminants in the PCB formulation. All commercial PCB formulations contain contaminants, such as polychlorinated dibenzofurans, which are more toxic to humans and aquatic life than PCBs.

The International Agency for Research on Cancer (IARC) has classified PCBs as probable human carcinogens. Recent studies of PCB chronic toxicity have also identified stimulation of liver enzymes and impairment of reproductive function as adverse effects in mammals.

Despite the 1977 USEPA ban on PCB manufacture and the 1979 regulation restricting their processing, distribution, and use, PCBs continue to be detected in California waters. Monitoring by the State Water Resources Control Board has detected PCBs in fresh and salt waters, sediments, and aquatic organisms. Some of the concentrations have been traced to specific sources, and subsequent control actions have reduced levels. However, many of the PCB levels found in water and biota result from redistribution of PCBs in the environment.

The widespread dispersion of PCBs stems largely from past production patterns and disposal practices. In 1976 Congress singled out PCBs for special regulation in enacting the Toxic Substances Control Act. The U.S. Environmental Protection Agency promulgated regulations in 1977 covering PCB manufacture, use, storage, inspection, and disposal. The Environmental Defense Fund challenged the 1977 regulation, and EPA issued several revisions to the regulation to comply with the court decision.

The federal regulations focus on PCBs in electrical equipment and implementation has emphasized compliance by utility companies. Management programs for non-utility PCB equipment are not as highly developed. Major problems have been identified with PCB equipment in California state facilities. As a result, the state legislature has funded development of a management program to control PCBs in state-owned facilities.

Apart from electrical equipment, PCBs are found in abandoned hazardous waste sites and in spills. These problems are addressed under the federal and state programs dealing with spill prevention, emergency response, and abandoned hazardous waste clean-up. PCB disposal in California is hampered by the lack of in-state disposal facilities.

This report assesses the properties and uses of PCBs, their environmental fate and toxicology, and the impact of PCBs on human health and California's aquatic resources. The report proposes a water quality strategy for controlling PCBs and a series of actions for consideration by affected state, local, and federal agencies.

2.0 PROPERTIES AND USES

2.1 Nomenclature

The biphenyl molecule ($C_{12}H_{10}$) can be chlorinated by replacing any or all of its hydrogen atoms with chlorine. Specific chlorinated biphenyl molecules are described by use of the ring numbering system indicated in Figure 2.1. The phenyl moiety containing the fewest chlorines is assigned prime numbers. Examples of the nomenclature used for chlorinated biphenyls are given in Figure 2.1.

The biphenyl molecule has ten possible sites for chlorine substitution; classes of chlorinated biphenyls range from monochlorinated to decachlorinated biphenyls. A total of 209 chlorinated biphenyl isomers are possible depending upon the number and pattern of chlorine substitution (Table 2.1).

2.2 Commercial Polychlorinated Biphenyl (PCB) Manufacture

In the commercial synthesis of chlorobiphenyls, the biphenyl is catalytically chlorinated with anhydrous chlorine using iron filings or ferric chloride as the catalyst. Commercial preparations of polychlorinated biphenyls (PCBs) are mixtures of isomers rather than a specific compound. The degree of chlorination depends chiefly on the time of contact (12-36 hours) of biphenyl with the anhydrous chlorine. The former United States manufacturer of PCBs, the Monsanto Chemical Company, used the trade name Aroclor for these mixtures (Nisbet & Sarofins, 1972). Several grades of Aroclor have been designated by numbers such as 1221, 1242, 1254, and 1260. The last two digits indicate the percent by weight of chlorine, and the "12" represents the biphenyl moiety. Aroclor 1016 consists primarily of tri- and tetrachlorobiphenyl compounds with pentachlorobiphenyls and above removed. Aroclor 1016 contains 41 percent chlorine by weight and was introduced by Monsanto in 1971 to replace Aroclor 1242. Table 2.2 gives the approximate molecular composition of seven Aroclor mixtures. PCB products manufactured in Japan were marketed as "Santotherm", and "Kanechlor". Kanechlors 300, 400, 500, and 600 contain approximately 42 percent, 48 percent, 54 percent, and 60 percent chlorine respectively. German PCB products have been marketed as "Clophens", A50 (54 percent chlorine) and A60 (60 percent chlorine). Other countries reported to have produced PCBs are France (Phenochlor and Pyralenes), Italy, (Fenclor), the Soviet Union (Sovol), Spain, Czechoslovakia, Poland, Argentina, Brazil, and India (NIOSH, 1977).

About half of the 209 possible chlorinated biphenyl isomers are known to occur in commercial mixtures. In addition to chlorinated biphenyls, commercial PCBs also contain other compounds, including biphenyl, chlorinated dibenzofurans, and chlorinated naphthalenes (Figure 2.2). A number of commercial mixtures have been analyzed for the presence of chlorinated dibenzofurans. The results are shown in Table 2.3. Chlorinated dibenzofurans have been shown to be far more toxic than PCBs, so presence of chlorinated dibenzofurans in low amounts in PCB mixtures may contribute to observed toxic effects of commercial PCBs. Further, there is indirect evidence that chlorinated dibenzofuran concentrations increase during use for such purposes as heat transfer fluids (Pomerantz et al., 1978; ARB, 1980).

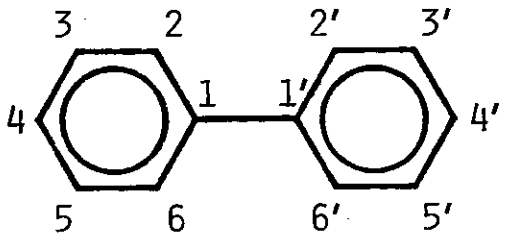
Some commercial Aroclors contained chlorinated terphenyls in addition to chlorinated biphenyls: Aroclors 2565 and 4465 contained 75 percent and 60 percent chlorinated biphenyls and 25 percent and 40 percent chlorinated terphenyl compounds, respectively (both mixtures contained 65 percent chlorine).

2.3 Physical and Chemical Properties

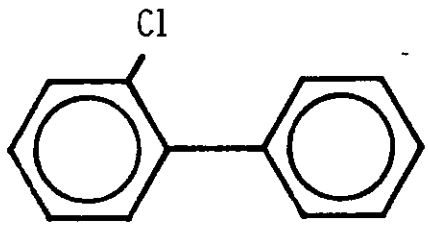
Physical properties of the ten chlorinated biphenyl Aroclors are summarized in Table 2.4. It should be emphasized that these physical properties are for mixtures

FIGURE 2.1

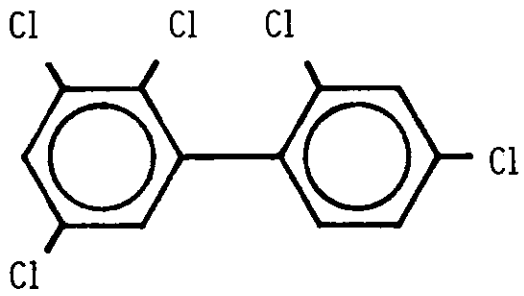
NUMBERING SYSTEM AND NOMENCLATURE FOR POLYCHLORINATED BIPHENYLS



THE BIPHENYL MOLECULE



A MONOCHLOROBIPHENYL
(2-Chlorobiphenyl)



A PENTACHLOROBIPHENYL
(2,2',3,4',5-Pentachlorobiphenyl)

TABLE 2.1

NUMBER OF POSSIBLE ISOMERS AND PERCENT CHLORINE
FOR THE 10 CHLOROBIPHENYL (PCB) CLASSES
(Adapted from NIOSH, 1977)

CHLOROBIPHENYL	EMPIRICAL FORMULA	NO. OF POSSIBLE ISOMERS	PERCENT CHLORINE BY WEIGHT
biphenyl	$C_{12}H_{10}$	1	0
monochlorobiphenyl	$C_{12}H_9Cl$	3	18.79
dichlorobiphenyl	$C_{12}H_8Cl_2$	12	31.77
trichlorobiphenyl	$C_{12}H_7Cl_3$	24	41.30
tetrachlorobiphenyl	$C_{12}H_6Cl_4$	42	48.56
pentachlorobiphenyl	$C_{12}H_5Cl_5$	46	54.30
hexachlorobiphenyl	$C_{12}H_4Cl_6$	42	58.93
heptachlorobiphenyl	$C_{12}H_3Cl_7$	24	62.77
octachlorobiphenyl	$C_{12}H_2Cl_8$	12	65.98
nonachlorobiphenyl	$C_{12}HCl_9$	3	68.73
decachlorobiphenyl	$C_{12}Cl_{10}$	1	71.18

TABLE 2.2

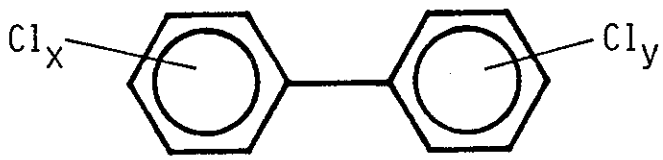
APPROXIMATE MOLECULAR COMPOSITION OF AROCLOR MIXTURES (PERCENT)
(Adapted from USEPA, 1980)

CHLOROBIPHENYL	1221	1232	1016	1242	1248	1254	1260
$C_{12}H_{10}$	11	6	Tr	-	-	-	-
$C_{12}H_9Cl$	51	26	1	1	-	-	-
$C_{12}H_8Cl_2$	32	29	20	17	1	-	-
$C_{12}H_7Cl_3$	4	24	57	40	23	-	-
$C_{12}H_6Cl_4$	2	15	21	32	50	16	-
$C_{12}H_5Cl_5$	0.5	0.5	1	10	20	60	12
$C_{12}H_4Cl_6$	-	-	Tr	0.5	1	23	46
$C_{12}H_3Cl_7$	-	-	-	-	-	1	36
$C_{12}H_2Cl_8$	-	-	-	-	-	-	6
$C_{12}HCl_9$	-	-	-	-	-	-	-
$C_{12}Cl_{10}$	-	-	-	-	-	-	-

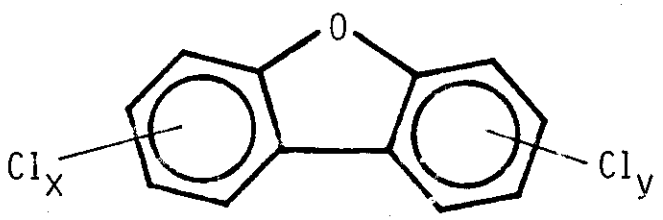
Tr - Trace (<0.1 percent)

FIGURE 2.2

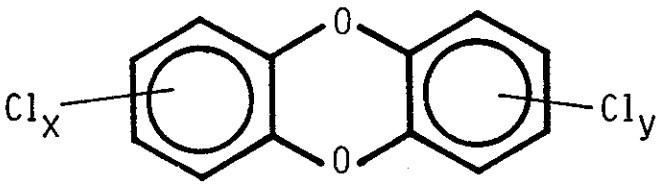
SKELETAL STRUCTURES OF CHLORINATED
BIPHENYLS, DIBENZOFURANS AND DIBENZODIOXINS
(Bowes et al., 1978)



POLYCHLORINATED BIPHENYL (PCB)



POLYCHLORINATED DIBENZOFURAN



POLYCHLORINATED DIBENZODIOXIN

TABLE 2.3

CHLORODIBENZOFURAN TYPES AND CONCENTRATIONS (ug/g)
IN COMMERCIAL PCB PREPARATIONS
(Adapted from NIOSH, 1977)

	Mixture	Chlorodibenzofurans di	tri	tetra	penta	hexa	hepta	Total
Aroclor	1016	0.5						0.5
Aroclor	1016			<0.001	<0.001	<0.001		
Aroclor	1248			0.5	1.2	0.3		2.0
Aroclor	1254			0.1	0.2	1.4		1.7
Aroclor	1254			0.2	0.4	0.9		1.5
Aroclor	1260			0.1	0.4	0.5		1.0
Aroclor	1260			0.2	0.3-	0.3		0.8
Clophen	A-60			1.4	5.0	2.2		8.4
Phenoclor	DP-6			0.7	10.0	2.9		13.6
Kanechlor	K300							1-1.5
Kanechlor	K400							17-18
Kanechlor	K500							2.5-4
Kanechlor	K600							3-5

TABLE 2.4

PHYSICAL PROPERTIES OF COMMERCIAL PCBs (Aroclors)
(USEPA, 1980)

PROPERTY	1221	1232	1016	1242
Chlorine, percent	20.5-21.5	31.4-32.5	41	42
Specific Gravity	1.182-1.192 (25°/15.5°C)	1.270-1.280 (25°/15.5°C)	1.362-1.372 (25°/15.5°C)	1.391-1.392
Distillation Range °C Corrected	275-320	290-325	323-356	325-366
Vapor Pressure (mm/HS)				4.06×10^{-4}
Evaporation loss (%) 100°C, 6 hr.	1.0-1.5	1.0-1.5		0-0.4
USTA D-6 Mod. 160°C, 5 hr.				3.0-3.6
Pour Point°C	1 (Crystal)	-35.5		-19
(WTM E97) F	34 (Crystal)	-32		2
Water Solubility at 25°C (ug/l)	200		225-250	240

TABLE 2.4 (cont.)

PHYSICAL PROPERTIES OF COMMERCIAL PCBs (Aroclors)
(USEPA, 1980)

PROPERTY	1248	1254	1260	1262
Chlorine, percent	48	54	60	61.5-62.5
Specific Gravity	1.405-1.415 (65°/15.5°C)	1.495-1.555 (65°/15.5°C)	1.555-1.566 (90°/15.5°C)	1.572-1.583 (90°/15.5°C)
Distillation Range °C Corrected	365-390	365-390	385-420	390-425
Vapor Pressure (mm/HS)	4.94×10^{-14}	7.71×10^{-5}	4.05×10^{-5}	
Evaporation loss (%) 100°C, 6 hr.	0-0.3	0-0.2	0-0.1	0-0.1
USTA D-6 Mod. 160°C, 5 hr.	3.0-4.0	1.1-1.3	0.5-0.8	0.5-0.2
Pour Point °C (WTM E97) F	-7 19.4	10 50	31 88	35-38 99
Water Solubility at 25°C (ug/l)	54	12	2.7	

TABLE 2.4 (cont.)

PHYSICAL PROPERTIES OF COMMERCIAL PCBs (Aroclors)
(USEPA, 1980)

PROPERTY	1268	1270
Chlorine, percent	68	71
Specific Gravity	1.604-1.611 (25°/25°C)	1.944-1.960 (25°/25°C)
Distillation Range °C Corrected	435-450	450-460
Vapor Pressure (mm/HS)		
Evaporation loss (%) 100°C, 6 hr.	0-0.6	
USTA D-6 Mod. 160°C, 5 hr.	0.1-0.2	
Pour Point °C (WTM E97) F		
Water Solubility at 25°C (ug/l)		

of biphenyl and various chlorinated biphenyl isomers: an estimated 40 to 60 different chlorinated biphenyl compounds are present in each of the higher commercial Aroclors (Pomerantz et al., 1978). The lower chlorinated Aroclors (1221, 1232, 1016, 1242, and 1248) are colorless mobile oils. Aroclor 1254 is a viscous liquid, and Aroclors 1260 and 1262 are sticky resins. The highest Aroclors, 1268 and 1270, are white powders. With the exception of Aroclors 1221 and 1268, the Aroclors do not crystallize upon heating or cooling. Instead, at a specific temperature, the "pour point", the mixtures change into a resinous state.

Chlorinated biphenyls appear to volatilize readily from water in laboratory aquaria. However, in the presence of sediments or particulates (which characterize natural waters), volatilization is markedly reduced due to adsorption onto particles. Chlorinated biphenyls are strongly adsorbed on solid surfaces, including glass and metal in laboratory apparatus, and on media such as soils, sediments, and particulates. In aquatic environments, chlorinated biphenyls are associated with sediments, and are usually present at much higher concentration in sediment than

TABLE 2.5

DISTRIBUTION OF PCB USAGE BASED ON
DOMESTIC SALES, 1957 to 1975
(million pounds)
(EPRI, 1979)

YEAR	DOMESTIC SALES OF PCBs	TOTAL PCB USE IN CAPACITORS	TOTAL PCB USE IN TRANSFORMERS	TOTAL PCB USE IN ELECTRICAL EQUIPMENT
1957	32.299	17.028	12.955	29.983
1958	26.061	14.099	5.719	19.818
1959	31.310	16.499	5.984	22.483
1960	35.214	16.967	7.921	24.888
1961	37.538	15.935	6.281	22.216
1962	38.043	15.382	7.984	23.366
1963	38.132	15.606	7.290	22.896
1964	44.869	19.540	7.997	27.537
1965	51.796	23.749	8.657	32.406
1966	59.078	28.884	8.910	37.794
1967	62.466	29.703	11.071	40.774
1968	65.116	29.550	11.585	41.135
1969	67.194	25.022	12.105	37.127
1970	73.061	26.708	13.828	40.356
1971	34.301	14.141	11.134	25.275
1972	26.408	20.321	5.335	25.656
1973	37.743	23.566	14.176	37.742
1974	34.000	22.000	12.000	34.000
1975	31.944	20.644	11.300	31.944
Total (1957-1975)	826.572	395.344	182.232	577.576
Total (1930-1957)	426.428	234.656	152.768	387.424
Total (1930-1975)	1,253.000	630.000	335.000	965.000

TABLE 2.6

END USES OF AROCLOR MIXTURES
(IARC, 1978)

End Use	1016	1221	1232	1242	1248	1254	1260	1262	1268
Current									
Capacitors	x	x		x		x			
Transformers				x		x	x		
Former									
Heat transfer				x					
Hydraulics/lubricants									
Hydraulic fluids			x	x	x	x	x		
Vacuum pumps					x	x			
Gas-transmission turbines									
turbines		x		x					
Plasticizers									
Rubbers		x	x	x	x	x			x
Synthetic resins					x	x	x	x	x
Carbonless paper				x					
Miscellaneous									
Adhesives		x	x	x	x	x			
Wax extenders				x		x			x
Dedusting agents						x	x		
Inks						x			
Cutting Oils						x			
Pesticide extenders						x			
Sealants and caulking									
compounds						x			

were for closed systems. After 1972, only those Aroclors designated 1016, 1221, 1242, and 1254 were produced in the United States, with total annual production of about 40 million pounds. Imported PCBs amounted to about one-half million pounds. A decachlorobiphenyl produced in Italy was imported for use in investment casting waxes and accounted for 80-90 percent of total imports. The remaining imports came from France and were used for semi-enclosed heat transfer applications. A review of the various types of PCB-containing equipment and materials is presented in Appendix A.

2.5 Inventory of PCBs in the United States and California

2.5.1 National and Regional Inventory

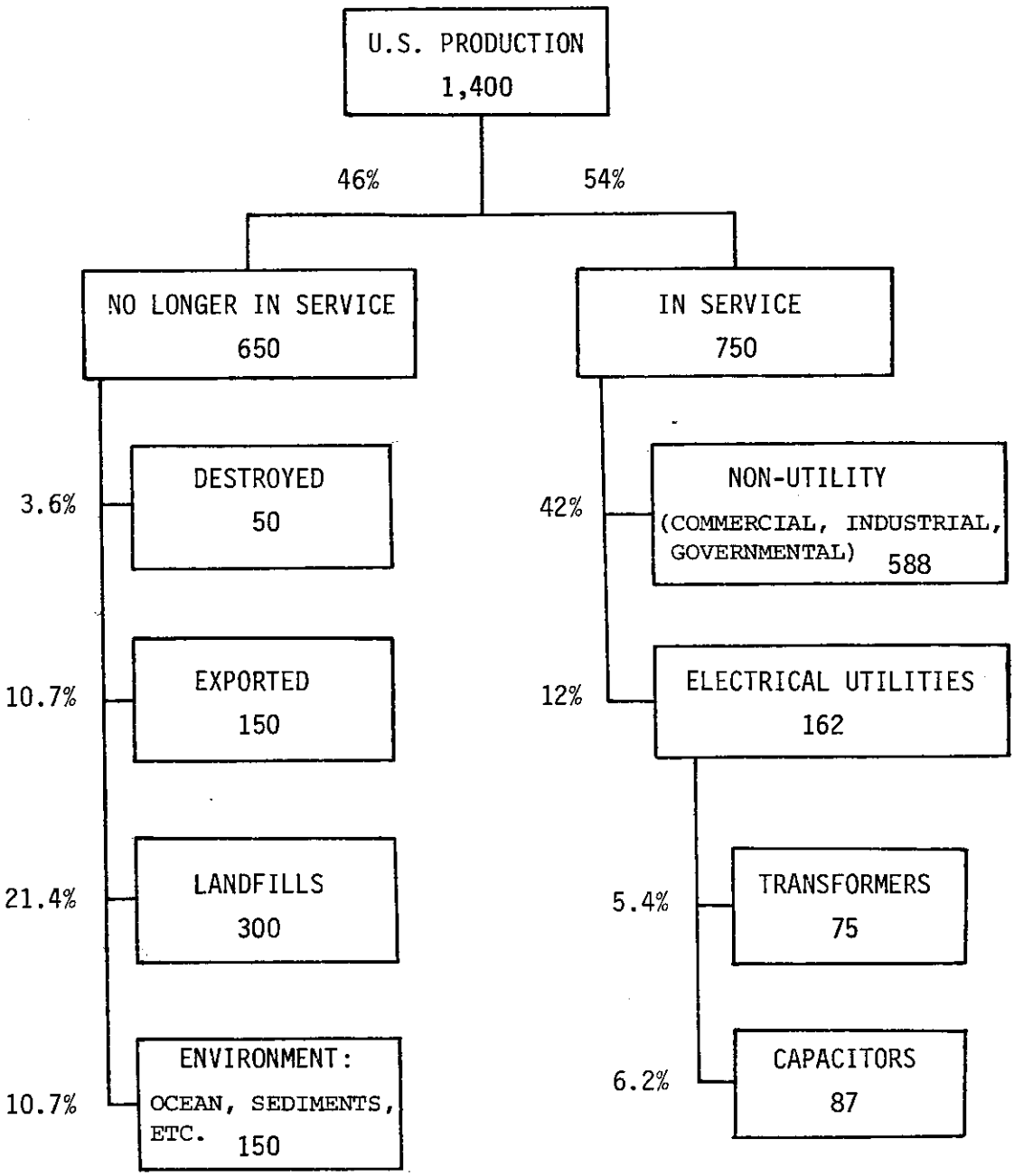
About 20 percent of the 750 million pounds of PCBs still in service in electrical equipment are in the electrical utility industry (Figure 2.3). Of the PCBs no longer in use, 50 million pounds have been destroyed, 150 million pounds have been exported, 300 million pounds have been placed in landfills, and 150 million pounds are present in the environment (Figure 2.3).

Closed system uses of PCBs, specifically in transformers and capacitors, are of interest because these types of electrical equipment have long service lives and can be used until replacement is necessary (capacitors) or retrofilled with non-PCB fluids (transformers). In the electrical utility industry, capacitors and transformers represent large amounts of PCBs that must ultimately be stored and destroyed. The Electric Power Research Institute (EPRI) has calculated that the average transformer contains 2,150 pounds and the average large, high voltage capacitor contains 25 pounds of PCBs.

In 1979, EPRI produced a four-volume report, "Disposal of PCBs and PCB-Contaminated Material" (EPRI, 1979). The objective was to develop guidelines for evaluating alternative methods of PCB disposal. In order to determine the amount of disposal facilities needed, EPRI estimated total PCB transformers and capacitors still in use nationwide and further estimated PCBs in use on a regional basis (using EPA's ten regions). Based upon available data, EPRI distributed utility-owned

FIGURE 2.3

FATE OF POLYCHLORINATED BIPHENYLS PRODUCED IN THE U.S.A.
($\times 10^6$ lbs.)
(Adapted from Miller, 1982)



The State Board obtained the preliminary survey results submitted to ARB and compiled the data by classification of PCB users (Table 2.7). The estimate probably represents a large proportion of PCBs in use in utility-owned equipment and understates other PCB uses in California as of 1980. Of the 2.3 million gallons (29.7 million pounds) of liquids containing PCBs at 50 ppm or greater, 1.5 million gallons (19.4 million pounds) were in use by the State's five major utilities. The five-utility ARB estimate is 40 percent of the EPRI Region IX estimate. California state agencies accounted for an additional 0.45 million gallons (5.8

TABLE 2.7

CALIFORNIA LIQUID PCB SURVEY
(ARB, 1980)

Classification of PCB User	Number Surveyed	Total PCBs (Gals) [1]	Status of Equipment	Total Gallons	Use: Transformers	Use: Capacitors	Use: Other Liquids
1. Electrical Utilities [2]	5	1,501,875	in service	1,431,443	796,265	635,178	--
			in storage	70,432	69,040	1,392	
2. California State Agencies (estimated)	?	450,000		450,000	450,000		
3. U.S. Military Facilities	20	151,262	in service	115,694	108,439	655	6,600
			in storage	35,568	5,103	19	30,446
4. Oil and Pipeline Companies	19	69,368	in service	68,215	52,224	15,991	--
			in storage	1,153	10	127	1,016
5. Manufacturers	12	65,842	in service	57,397	54,939	687	1,771
			in storage	8,445	8,371	24	50
6. U.S. Department of Energy Facilities	2	20,450	in service	16,910	13,859	3,051	--
			in storage	3,540	2,000	--	1,540
7. California Cities	11	18,156	in service	15,931	12,826	3,102	3
			in storage	2,225	414	1,811	--
8. Miscellaneous	6	10,582	in service	10,420	10,200	220	--
			in storage	162	162	--	--
9. Special District Irrigation Dis.	1	8,299	in service	8,299	1,983	6,316	--
10. Transportation	3	4,173	in service	4,171	2,900	1,271	--
			in storage	2	--	2	--
	80	2,300,007	in service	2,178,480	1,503,635	666,471	8,374
			in storage	121,527	85,100	3,375	33,052
				2,300,007	1,588,735	669,846	41,426

1/ Gallons of liquids containing greater than 50 ppm PCBs

2/ Los Angeles Department of Water and Power, Pacific Gas & Electric, Southern California Edison, San Diego Gas and Electric, Sacramento Municipal Utility District

3.0 ENVIRONMENTAL FATE

3.1 Introduction

The higher chlorinated isomers of PCBs are virtually indestructable by natural processes and will recycle in an aquatic ecosystem for many decades (Figure 3.1). Biological transformation of PCBs is extremely slow and limited to the lesser chlorinated isomers. Bioaccumulation is rapid and extremely high (10^4 - 10^6 times). Because of their water insolubility and highly lipophilic (e.g., attraction to organic matter) nature, PCBs accumulate in tissues and sediments. The migration of PCBs through soil is dependent upon soil structure, soil organic content, and the presence of solvents mixed or in contact with the PCBs.

3.2 Distribution Pathways

3.2.1 Air

Atmospheric transport and deposition is the primary mode of PCB global distribution. Airborne transport and deposition may account for 90 percent of the total PCBs in Lake Superior (Eisenrich et al., 1980). Atmospheric deposition has also been implicated as the major source of PCB input to Lake Michigan and many other waterways throughout the world. PCB distribution in the air is affected by:

1. The volatility of the PCB isomer;
2. Meteorological conditions, (e.g., wind, temperature, humidity, rainfall); and
3. Possible photodecomposition of the higher chlorinated isomers. (Photodecomposition has only been shown under laboratory conditions, and may not occur in the atmosphere.)

3.2.2 Surface Waters

With the exception of the lesser chlorinated PCB isomers, most are extremely hydrophobic. The distribution of PCBs in water is affected by:

1. The aqueous solubility of the PCB isomers which will fractionate into water or be adsorbed by organic matter (Veith and Comstock, 1975);

2. Water flow and circulation which affect the distribution and desorption of PCBs from matter and the transport of contaminated material (PCB desorption from sediments has been shown to be negligible in several studies);

3. The amount of organic material suspended or surrounding the contaminated water;

4. The co-distillation or evaporative rate at the water surface, as affected by temperature and other meteorological conditions.

Most PCB isomers will not be found in the aqueous phase, but adsorbed onto matter within the water column.

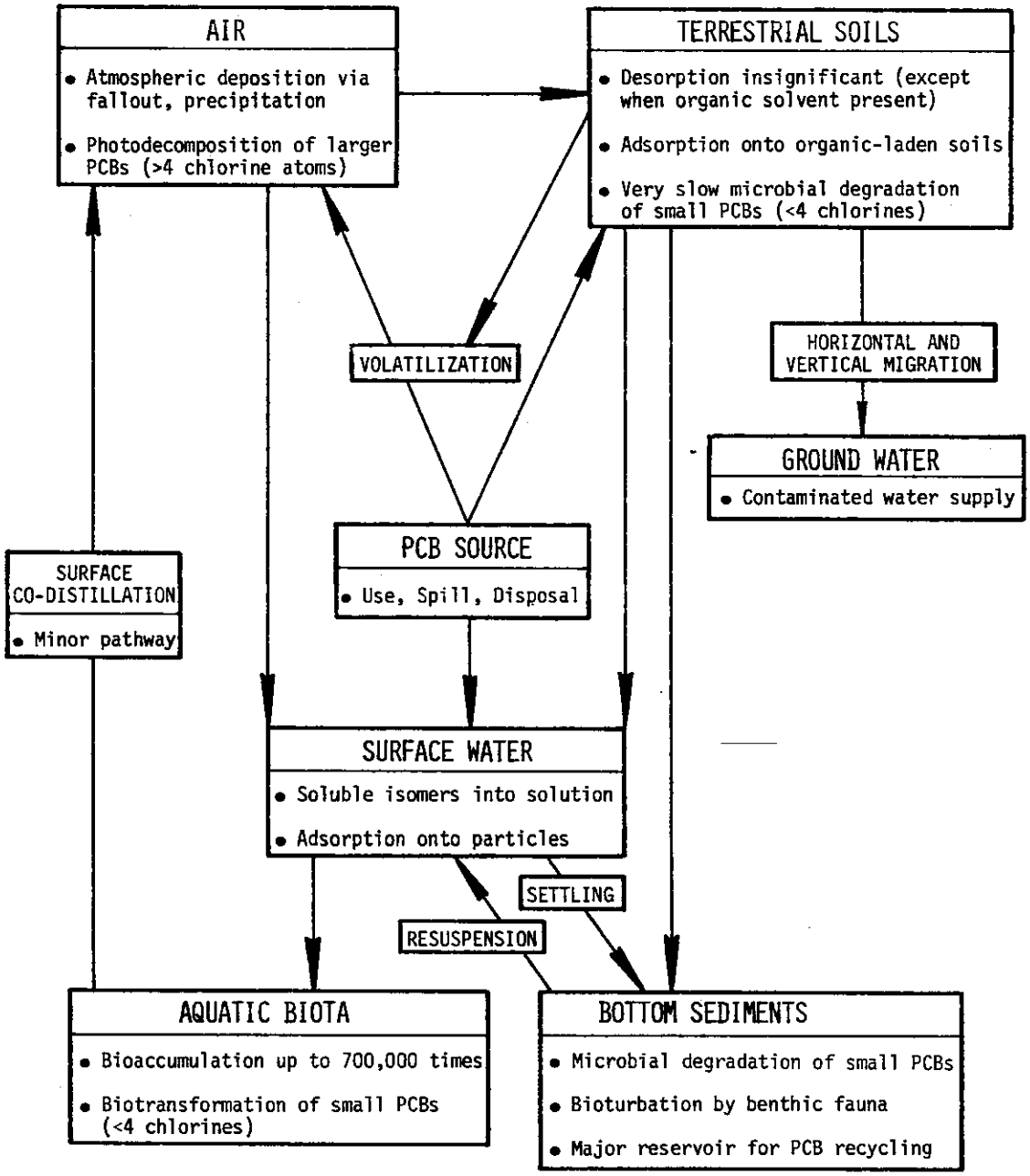
3.2.3 Aquatic Biota

Bioaccumulation

Organisms readily take up PCBs because of their lipophilic and hydrophobic qualities. Bioaccumulation varies significantly within the organs and tissues of an organism as well as among different species. Bioaccumulation is affected by:

FIGURE 3.1

PCB ENVIRONMENTAL PATHWAYS AND COMPARTMENTS



1. The partition coefficient of the PCB isomer;
2. The trophic level and feeding mechanisms of the organism (e.g. benthic, planktonic, nekton);
3. Surface area and size of the organism; and
4. Lipid content of the organisms and target organs.

PCB accumulation by fish appears to be a two-step process: from hydrosoil to water, then water to fish (Halter and Johnson, 1977).

Biotransformation and Biodegradation

Biodegradation is the only process known to transform PCBs under environmental conditions (Callahan et al., 1979). Biodegradation appears dependent upon the number of chlorine atoms in the isomer. Those isomers with 5 or more chlorine atoms are resistant to biodegradation.

3.2.4 Bottom Sediment (Hydrosoils)

Adsorption to sediments is the major process affecting PCB distribution in the aquatic environment (Callahan et al., 1979). While sediment adsorption is effective at immobilizing PCBs, resuspension of sediment can cause long-term contamination. For example, an estimate of 30-150 years has been suggested for biological recycling of PCBs in surficial Lake Superior and Lake Michigan sediments (Eisenrich et al., 1980). PCB distribution in bottom sediments is affected by:

1. Adsorption and desorption of PCBs by sediments. PCB desorption to equilibrium is a slow process and contributes negligible amounts of PCBs to flowing waters (Wildish et al., 1980). For example, in 120-day desorption tests, the estimated loss of PCB from hydrosoil containing 500 ppm was only 0.35 percent (Halter and Johnson, 1977).

2. Organic carbon content in sediments. The threshold concentration required for significant PCB concentrations to be bound to sediments was found to be about 2 percent organic carbon in Lake Superior (Eisenreich et al., 1980).

3. Overlying water flow conditions, which affect resuspension and deposition rates of sediments. Under static and flowing water conditions, hydrosoil PCB levels of 10 and 500 ppm produced equilibrium levels of 0.13 to 7.60 ppb and 0.11 to 0.54 ppb PCB in water, respectively (Halter and Johnson, 1977). Although PCB desorption is negligible, overlying water movement does affect the transport and burial of contaminated hydrosoils in a river.

4. Downward mixing of sediment from bioturbation by benthic fauna or other biological activity (e.g., spawning activity). Bioturbation depths appear to depend on the total sedimentation rate and benthic organism activity. The net effect of PCB fluxes to the sediment and mixing due to bioturbation is to increase the exposure time of sedimentary PCBs to the overlying water and biotic activity after PCB input has stopped.

5. Microbial degradation rates (limited to degradation of lighter PCBs). PCB sediment profiles reflect recent influxes of PCBs, followed by sediment reworking by bioturbation and water currents. The National Enforcement Investigations Center (NEIC) of EPA in Denver, Colorado successfully identified a major PCB source in Lake Michigan by sampling sediments and recommends the use of sediment analyses in forensic investigations (Meigs, 1980).

3.2.5 Terrestrial Soils

Adsorption and Desorption

As with aquatic sediments, PCBs are rapidly adsorbed by soils containing high organic content. Soil type and conditions (e.g. pH, moisture) affect the adsorption rate, distribution, and microbial degradation rate. Desorption occurs (1) when the adsorptive retention capacity of the soils is exceeded by supersaturation, (2) when soil conditions are altered (Farquhar et al., 1979), or (3) if an organic solvent was spilled at the same site.

PCBs appear to remain immobile in soils leached with water or landfill leachate (Griffin and Chou, 1981). Farquhar et al. (1979) measured water desorption of a highly PCB-contaminated soil to be 0.11 to 0.55 percent.

Microbial Degradation

The rate at which PCB isomers degrade in soil appears to be related to the molecular characteristics of the isomer. The more highly chlorinated isomers (4 or more chlorines) showed no breakdown in a 15-month study (Figure 3.2) (Tinsley, 1979). There are exceptions to this generalization that pronounced degradation is inversely proportional to chlorine number. In any case, degradation is very slow for PCBs with more than 4 chlorine atoms (Halter and Johnson, 1977).

3.2.6 Ground Water

Ground water contamination was observed in a New Jersey study (Page, 1981). Since limited circulation, dilution, and microbial activity occur in ground water, PCB contamination is a long-term problem. Factors which affect the soil mobility of PCBs would determine the likelihood of contamination. These factors include soil type and conditions, nature of leachate, and the presence of organic solvents in the PCB mixture.

3.2.7 Terrestrial Plants

Plant foliage has been found to accumulate PCBs from the atmosphere. The amounts accumulated vary among plant species. One study showed differential accumulation factors between plant species to be constant for over two orders of magnitude of PCB concentrations (Buckley, 1982). The study also showed that both PCBs and polybrominated biphenyls in soil contaminate plant roots but cause little contamination of leaves and stems. PCBs that were found in leaves and stems were carried by vapor transport from the soil (volatilization) and not translocated through the plant.

3.3 Water Contamination Potential

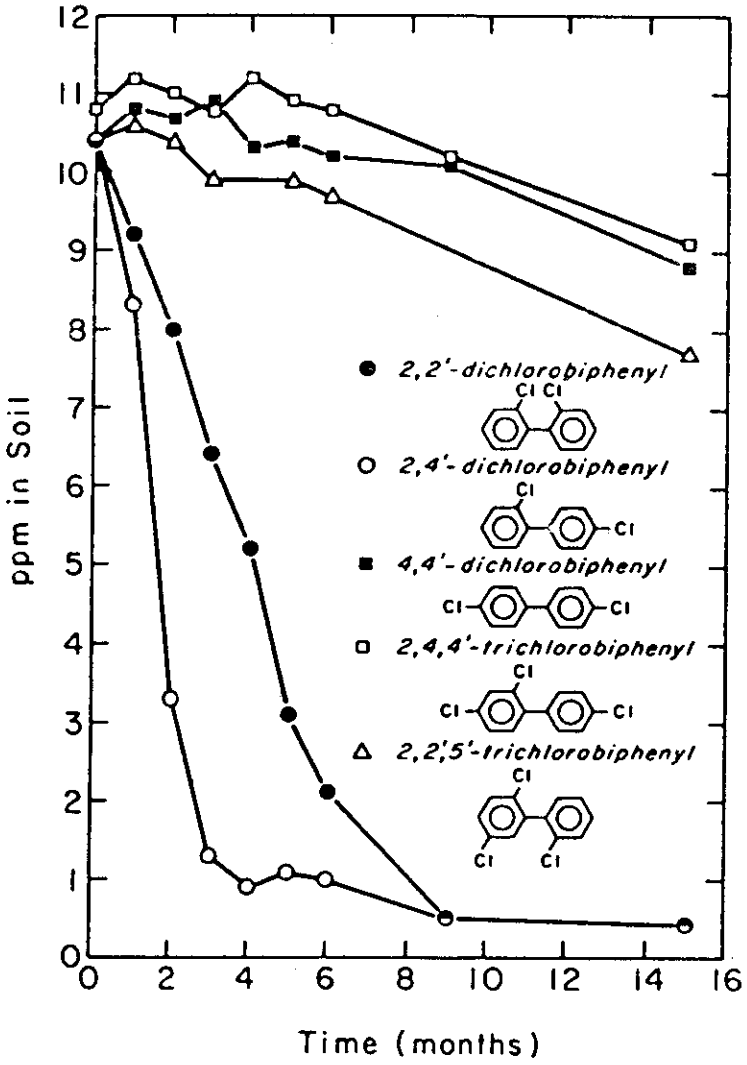
3.3.1 Surface Water

PCB contamination of lakes, rivers, and coastal ocean waters has been well documented (Eisenrich et al., 1980; Turk, 1980; Hom et al., 1974). PCB contamination of surface waters is inevitable because of atmospheric transport and deposition.

PCB mixtures, whether from airborne deposition or direct discharge, will fractionate into water- and organic-soluble mixtures. The more water-soluble mixtures, such as Aroclors 1221 and 1232, may be detected in water analyses. The lesser water soluble fractions will accumulate in organic matter in sediments, suspended particles in the water column or aquatic organisms. This strong affinity for organic matter

FIGURE 3.2

RATE OF DEGRADATION OF PCB ISOMERS IN SOIL
(Tinsley, 1979)



is due to the lipid solubility of PCBs. As a result, surface water contamination by PCBs is less likely to be an identified problem than sediment or biotic accumulation.

Destruction of PCBs in an aquatic system is very slow and limited to biological transformation and degredation of the lesser chlorinated (4 or less) isomers. The stability of the more chlorinated mixtures creates a serious long-term contamination problem.

3.3.2 Ground Water

PCB contamination of ground water was detected during a 1977-79 study of surface and ground water contamination problems in New Jersey (Page, 1981). The results from this survey of over 600 wells showed the highest levels to be 3.4 ppb (Aroclor 1242), 5.4 ppb (Aroclor 1248), and 0.4 ppb (Aroclor 1254). Aroclors 1242 and 1248 had greater average detectable concentrations in surface water than ground water. For Aroclor 1254 there was no significant difference (at the 0.05 significance level) between the average detectable concentration in surface water and ground water.

The factors which dictate the extent of PCB movement through soil into ground water are related to the mobility of the PCB isomers in soil and the proximity of the PCB source to the water table or well. Porous, loosely compacted aggregate and soils with low organic content such as sand and fractured clay material would not inhibit the downward mobility of PCBs (NRCC, 1980). However, if the soil is rich in organic matter, PCBs would be readily adsorbed.

3.4 Monitoring Strategy

Based on present knowledge of the environmental distribution and fate of PCBs, the best monitoring strategy for detecting PCBs would be to focus sampling and analytical efforts on the most probable compartments (water, sediments, biota, etc.) where PCBs could be found. Analysis of the water column alone would be inadequate to detect most PCBs. Only the more water soluble fractions could possibly be detected in a routine water monitoring program. The hydrophobic and lipophilic nature of the PCBs makes soil or sediment analyses, supplemented with collections of organisms, a more appropriate and cost effective approach in monitoring contamination (Chapman et al., 1982). A recommended monitoring strategy for detecting most PCB mixtures is shown in Table 3.1.

Some of the key recommendations made by the National Research Council of Canada (Roberts et al., 1978) for monitoring of PCBs in birds and fish are:

1. The lower chlorinated PCBs, which are more readily metabolized, might be developed as indicators of recent contamination.

2. The higher chlorinated PCBs commonly used as standards may best be interpreted as indicators of long term exposure.

3. It is important to report the size and lipid content of the organism along with residue data for meaningful interpretation of contamination trends and patterns.

4. Residue tolerances should be developed on the basis of levels needed to protect the most sensitive species.

5. Bioconcentration factors should be reported for a defined weight and a defined lipid content.

TABLE 3.1

RECOMMENDED MONITORING STRATEGY FOR PCBS IN AN AQUATIC ECOSYSTEM

Aroclor	Specific Gravity	Aqueous Solubility (ppm)	Estimated Log P Values	Compartment to Monitor		
				Surface Water	Sediment & Soil [1/]	Aquatic Biota
1016	1.33	0.42	5.58		X	X
1221	1.15	15.0	4.09	X	X	X
1232	1.24	1.45	4.54	X	X	X
1242	1.35	0.1-0.3	5.58		X	X
1248	1.41	0.54	6.11		X	X
1254	1.50	0.01-0.06	6.03		X	X
1260	1.58	.0027	7.14		X	X

[1/] Correlations have been found with many organic compounds which show that those with a water solubility of less than 5.0 ppm and an octanol/water partition coefficient greater than 10^5 tend to accumulate in the sediments (Hites and Lopez-Avila, 1980).

Most analytical laboratories currently report PCB concentrations in reference to standards of Aroclor 1254 and 1260 which are primarily composed of the higher chlorinated isomers (Table 2.2). Reporting of values for all identified isomers or gas chromatogram peaks would be of further value.

4.0 AQUATIC TOXICOLOGY

4.1 Introduction

Mortality of aquatic life from short term exposure to PCBs is likely to result from episodic events (e.g. spills). The predominant situation in nature involves exposure to low levels of PCBs over long time periods. Laboratory derived LC50 values are generally several times greater than the actual water solubility values for PCBs. The expected toxic effects from PCBs in the field result from bioaccumulation in body tissues such as the reproductive organs and liver. Some of the sublethal effects observed in the laboratory include poor survival and abnormal development of fish eggs and larvae. PCB residues in fatty tissue of parental female fish will mobilize to lipids in ovaries and eggs during the reproductive stages. This transfer is of major concern since developing larvae depend on stored lipids as an endogenous food supply for several days after hatching. Although toxic effects have been observed in PCB contaminated eggs and larvae, there has not been any field confirmation. Making such observations is complicated by the difficulty in observing fish egg and larval development in nature.

The concern for sublethal effects from PCB bioaccumulation led to the development of guidelines that limit PCB residues in aquatic organisms, animal feed, and human food (see Appendix G, Criteria and Standards for PCBs). The National Academy of Sciences (NAS) and EPA jointly recommend a maximum concentration of 0.5 ppm-ww (wet weight) in whole fish samples. This NAS/EPA guideline was established for the protection of aquatic life and predators. The International Joint Commission (IJC) also set a 0.1 ppm-ww limit on fish taken from the Great Lakes. The U.S. Food and Drug Administration (FDA) has action level limits of 5.0 ppm-ww (edible portion) for fish and a 2.0 ppm-ww limit for animal feed components.

PCB concentrations have been detected in the blood of fishermen who consume fish from the Great Lakes (see Chapter 5, Mammalian Toxicology and Human Health). The bioconcentration and transfer of PCBs in the ecological food web represents a threat to the well-being of all organisms including man.

4.2 PCB Dynamics in Aquatic Organisms

4.2.1 Uptake and Bioconcentration Factors

PCBs can be accumulated by aquatic organisms through contaminated food sources and exposure to contaminated water and sediment. Studies have shown:

1. About 70 to 80% of the PCBs in a contaminated food source are taken up by fish. There is higher body retention of chlorinated biphenyls with 5 or more chlorines. Gruger et al. (1975) observed 70% retention of a hexachlorinated biphenyl and 47% retention of a tetrachlorinated biphenyl in coho salmon (Figure 4.1).

2. The intestinal absorption of PCB components is equal (Lieb et al., 1974) and differences in the relative PCB composition in the tissues from the original

FIGURE 4.1

PERCENT CHLOROBIPHENYLS IN WHOLE JUVENILE
COHO SALMON FED FOR 24, 53 AND 108 DAYS
(Gruger et al., 1975)

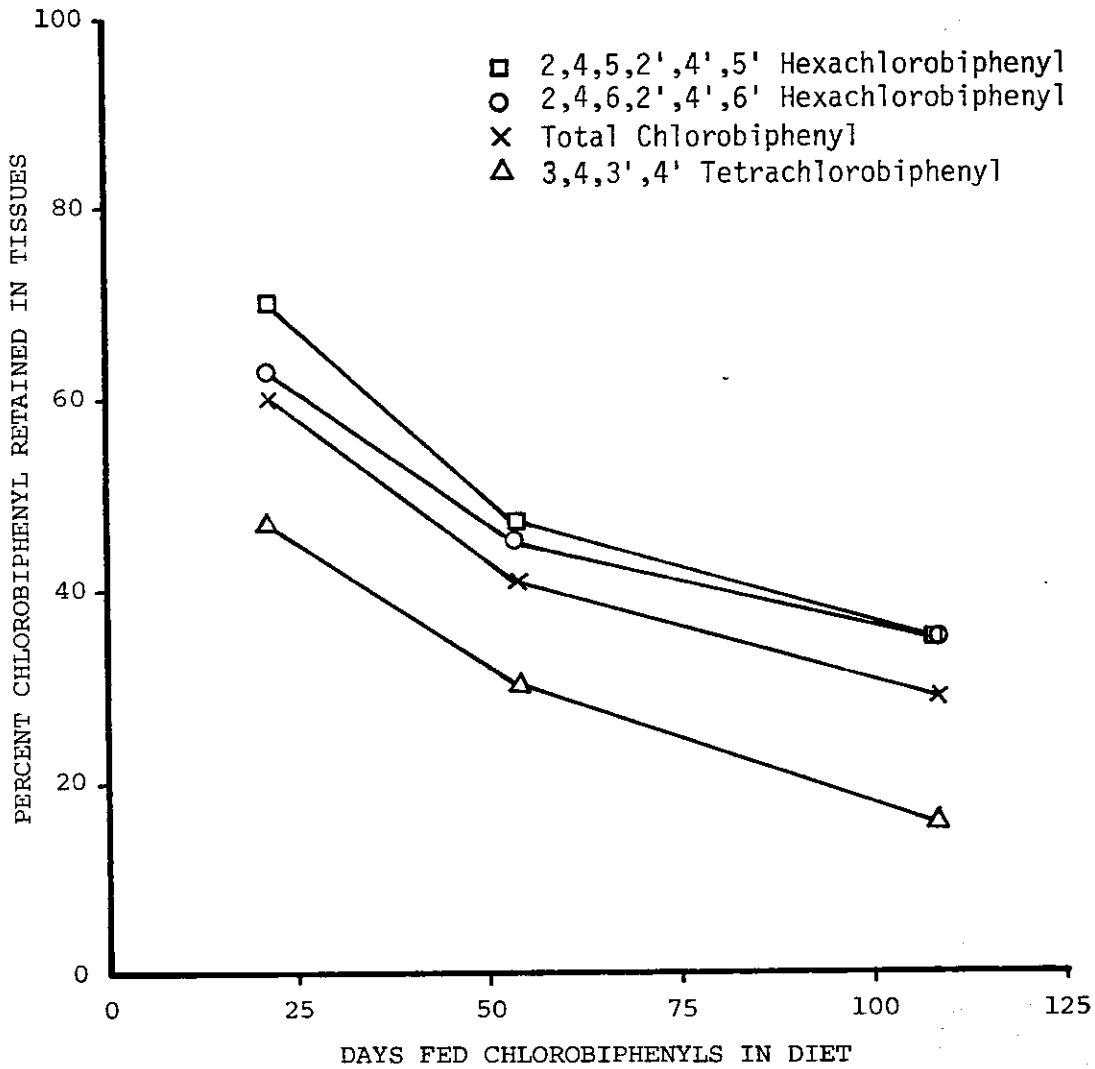
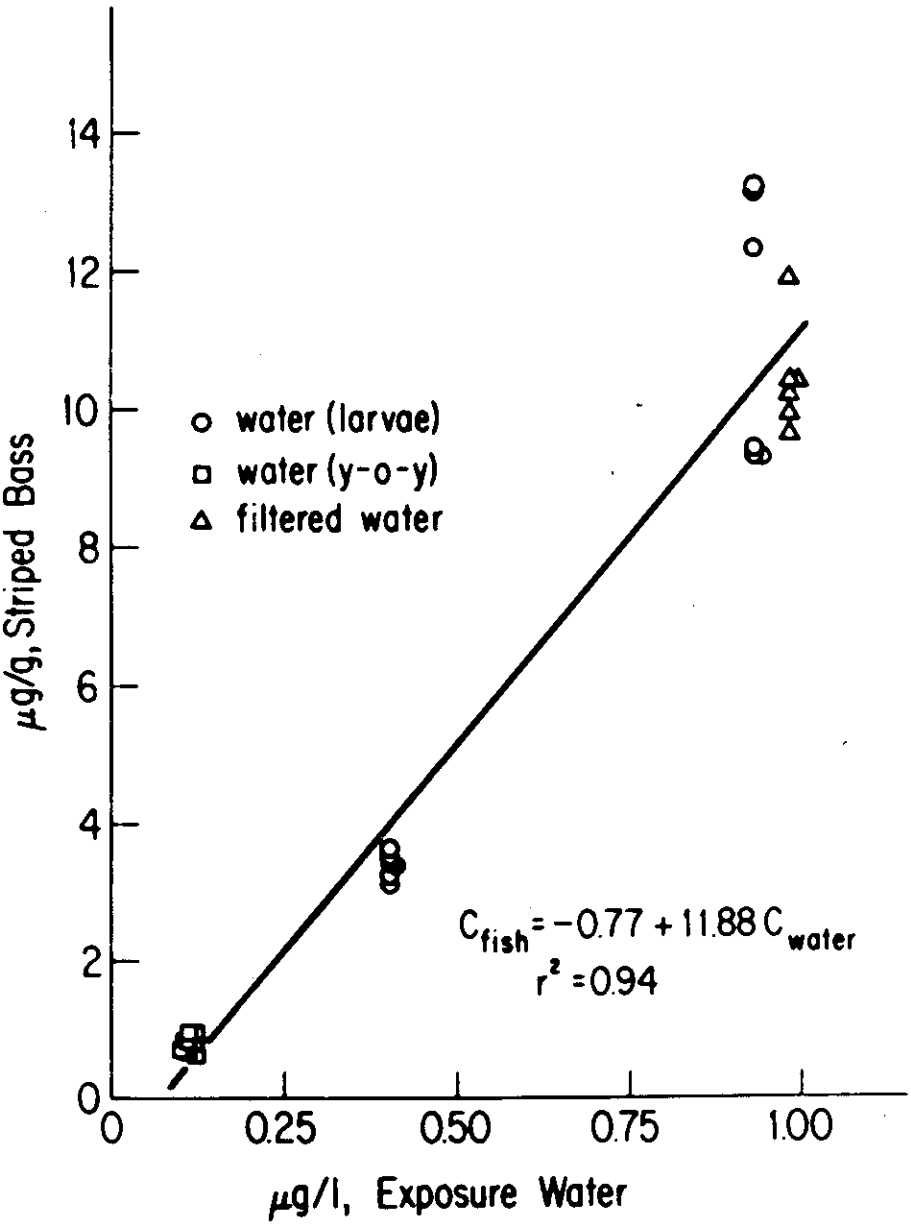


FIGURE 4.2

PCB ACCUMULATION BY YOUNG-OF-YEAR STRIPED BASS
(Califano et al., 1982)



Frederick (1975) also made a similar observation in a study of white suckers. Branson et al. (1975) observed a constant uptake rate from 1.6 to 9.0 mg/l of a tetrachlorinated biphenyl in water by rainbow trout fingerlings. Studies on preferential absorption by this route suggest that the different water solubilities of the individual PCB isomers may explain differences in PCB tissue residues from those in the water (Sanders and Chandler, 1972; Camp et al., 1974; Frederick, 1975). Uptake from water is probably the dominant route of PCB accumulation by aquatic life in the field.

Bioconcentration has been reported to be up to 10^4 times ambient water levels by plankton and appears to be related to surface area (Ware and Addison, 1974; Morris, 1975). Organisms may absorb PCBs from direct contact and ingestion of contaminated sediment or water near the sediment. PCBs have been detected in benthic organisms from contaminated areas (Nimmo et al., 1971; Nimmo et al., 1974).

The bioconcentration factor (BCF) for PCBs appears to be independent of the ambient water concentration because of their highly lipophilic nature. Whole-body residues in brook trout fry exposed for 118 days were 40,000 to 47,000 times the ambient water concentration (Mauck et al., 1978). Channel catfish (Ictalurus punctatus) accumulated PCBs from 56,370 to 61,190 times the water level in 77 days from exposure to Aroclors 1248 and 1254, respectively (Mayer et al., 1977).

For invertebrates, the bioaccumulation of PCBs ranges from 160 times the water concentration after one-day exposure for crayfish exposed for two weeks (in the laboratory) to 125,000 times for mysids (in the field). However, it is difficult using current analytical procedures to distinguish between PCBs adsorbed on the surface from those absorbed by the organism. Rinsing microorganisms and small invertebrates with organic solvents to extract PCBs dissolves cellular material as well. As a result, the bioconcentration factors for small organisms with large surface areas are high.

BCFs for freshwater invertebrates range from 2,700 to 108,000 and from 3,000 to 2,333,000 in freshwater fish. BCFs for marine invertebrates range from 800 to over 230,000 and 14,400 to over 670,000 in marine fish. USEPA (1980) estimates for laboratory-derived bioconcentration factors are less than those for field measurements (Figure 4.3). The lower laboratory-derived values appear to be due to substantial partitioning of the PCB isomers to particles in the water and aquaria.

4.2.2 Distribution in Body Tissues

The distribution of PCBs in fish tissue is closely related to lipid content and mass in relation to other parts of the body (Figure 4.4). Because of the lipophilic nature of PCBs, the liver, brain, and ovaries are typical target organs. However, redistribution of PCBs will occur from physiological changes or stress (e.g. spawning or migration). During such periods lipid mobilization may occur. Food deprivation may decrease fat deposits significantly enough to result in higher concentrations of PCB residues in tissues. Whipple (1982) observed equal partitioning of PCBs in liver and ovaries of prespawning, maturing female striped bass. She also noted that sexually mature female bass had more PCBs in the gonads than sexually immature fish.

The distribution of PCBs within the tissues also varies among species. Guiney and Peterson (1980), using a radio-labeled ^{14}C PCB isomer, compared the distribution and elimination of PCBs between a nonfatty fish (yellow perch) and a fatty fish (rainbow trout). In perch, the major sites of storage were viscera and carcass, whereas in trout, skeletal muscle and carcass were the major distribution sites. The difference may have been due to lipid content of the tissues.

FIGURE 4.3

FIELD AND LABORATORY BIOCONCENTRATION FACTORS (BCF)
FOR AQUATIC ORGANISMS EXPOSED TO PCBs
(USEPA, 1980)

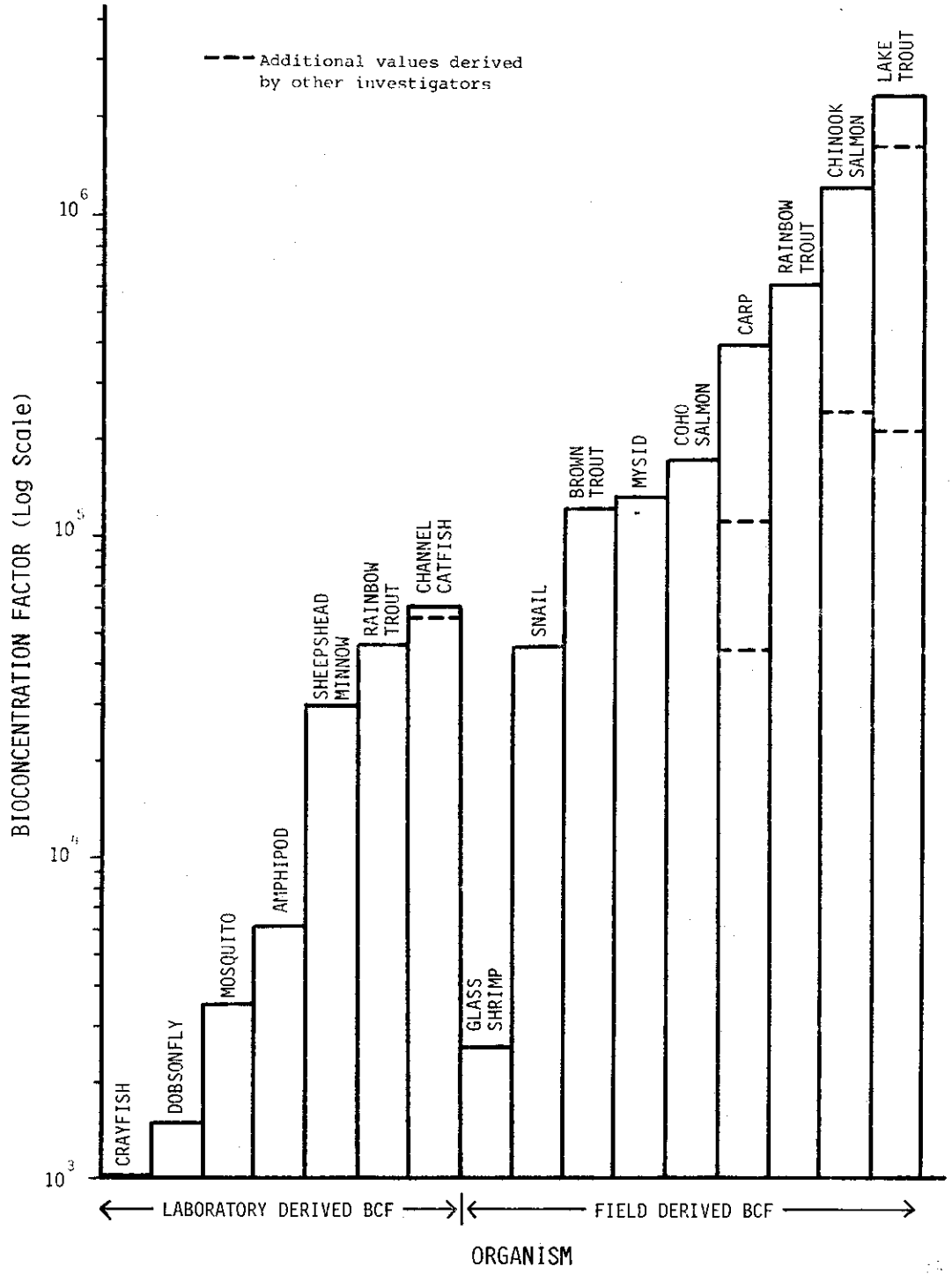
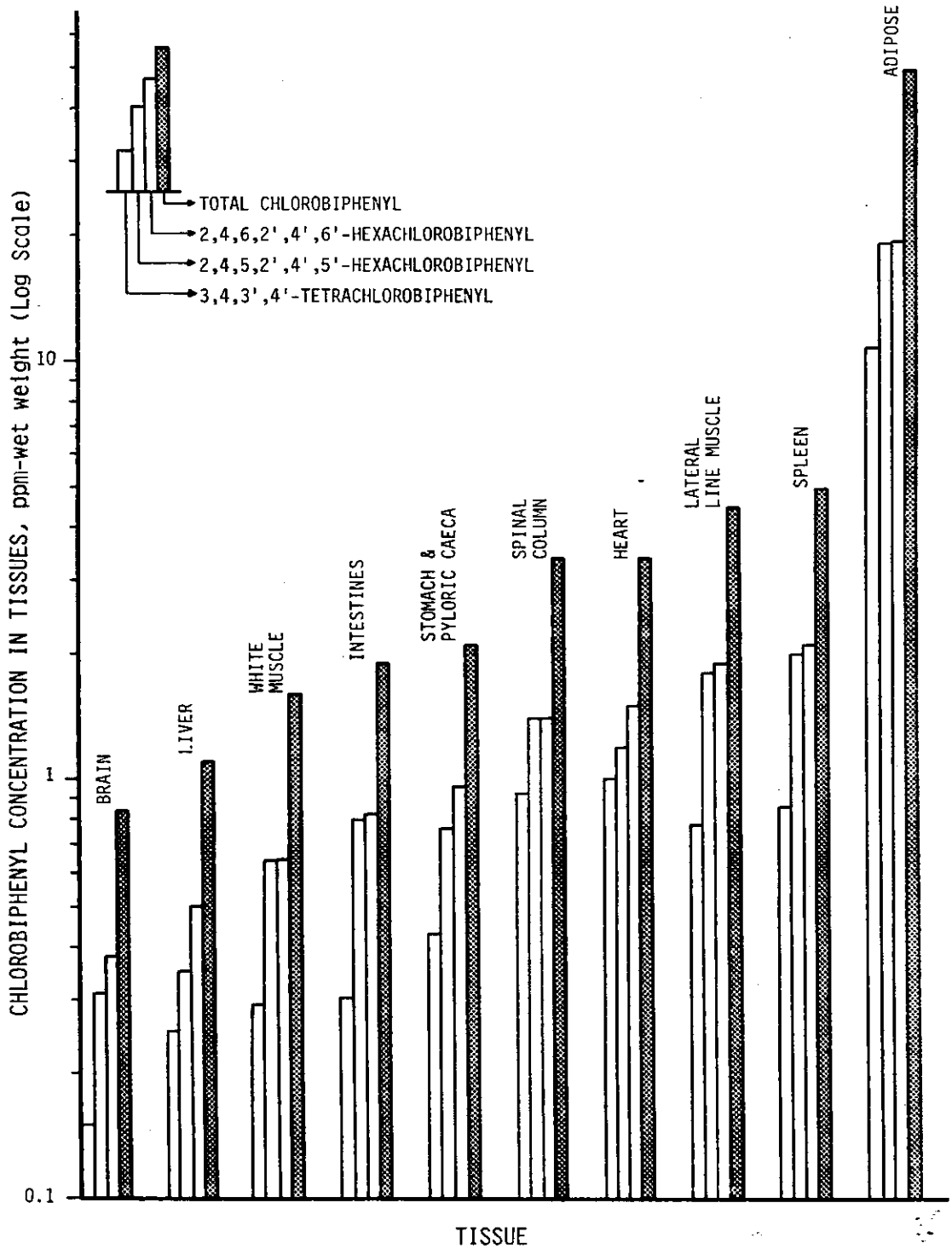


FIGURE 4.4

CONCENTRATIONS OF PCBs IN VARIOUS TISSUES OF
JUVENILE COHO SALMON FED PCB DIETS
(Adapted from Gruger et al., 1975)



4.2.3 Metabolism and Elimination

A variety of biota are capable of metabolizing the chlorinated biphenyls with less than five chlorine atoms. Baxter et al. (1975) reported degradation of a tri-, tetra-, and pentachlorinated biphenyl by the salt-marsh caterpillar. Sanborn et al. (1975) reported a similar observation in bluegill sunfish. Lower chlorinated biphenyls are metaboiized into polar compounds in the liver and excreted.

The rates of metabolism and elimination of PCBs are closely related to: (1) the number of chlorine atoms, (2) position of the chlorine and hydrogen atoms on the biphenyl ring, and (3) the amount of PCB residues bound to fat tissues. Chlorobiphenyls with five or more chlorine atoms and without two adjacent unsubstituted carbon atoms are generally more resistant to biodegradation and therefore more persistent. The elimination of PCBs from tissue is a lengthy process (Roberts et al., 1978).

While the lower chlorinated biphenyls can be metabolized and eliminated from the body, the higher chlorinated isomers are similar to DDT and DDE in their environmental persistence. Guiney et al. (1979) measured the half-life (t-1/2) for whole body elimination of a tetrachlorinated biphenyl at 1.76 years in female rainbow trout and 1.43 years in males.

However, during the spawning season the half-lives were 0.52 year in females and 0.54 year in male trout. The increased elimination rate is primarily due to the voiding of contaminated eggs and sperm. The study showed a redistribution of PCBs during the reproductive cycle and illustrated the enhancing effect of egg and sperm maturation and spawning on whole body elimination of PCBs in fish. PCB can also alter sex hormone metabolism in adult salmonid fish and affect the mortality and viability of eggs and fry (Freeman and Idler, 1975).

4.3 Aquatic Toxicity

4.3.1 Factors Affecting Toxicity

A comparison of acute and sublethal toxicity data for PCBs is difficult. The reported water concentrations of PCBs during these tests may actually underestimate the sensitivity of aquatic organisms to PCBs. Zitko (1970) reported coagulation of PCB test solutions containing particulate matter because of the low aqueous solubility of PCB components. Results from such tests are difficult to reproduce and may not reflect the true lethal tolerances of test organisms to PCBs. This may partially explain the wide range in reported LC50 values (parts per thousand to parts per million). It is more accurate to refer to the LC50 values as "nominal" or "starting" doses.

The task of comparing toxicity data becomes even more complex due to the lack of standardized test protocols for laboratory test conditions (e.g. flow, temperature), developmental stage of the test organism (e.g. larval, adult), and composition of the "PCB mixture" (e.g. isomers, contaminants, carrier solvent).

Researchers often introduce lipophilic compounds into water at levels much higher than the water solubility of the compounds. This is achieved most often by mixing the compounds with organic solvents (e.g. acetone). This practice can be defended if the researcher wants to determine if the compounds are toxic to the organism being studied or wants to know the mechanism of action of the compound in the organism once it has proven to be toxic. However, the practice of adding compounds at levels higher than their natural water solubility should not be followed if one wants to determine accurate acute or chronic toxicity values. Compounds introduced

above their natural water solubilities will have the undissolved portion adsorbed onto surfaces of particles in the water or container or test organism. Consequently, a significant underestimation of the toxicity may result because the toxic effects will occur at a concentration below that of the natural water solubility level.

4.3.2 Lethal Concentrations

Acute toxicity values for selected aquatic organisms are shown in Table 4.1. Some LC50 values listed in Table 4.2 greatly exceed the known water solubilities of the PCB mixtures. This suggests that PCBs will rarely cause immediate mortalities of aquatic life in the field.

Fish

A time-dependent dose relationship of mortality from PCB exposure has been observed. The LC50 values for PCBs under short term exposure ($\leq$ 96 hours) are 10-100 times greater than those reported from longer duration studies (Table 4.2). The early stages of fish appear to be more sensitive to PCBs than adult fish. Hansen et al. (1974) observed mortality of sheepshead minnow eggs containing 7.0 mg/g-ww or more of Aroclor 1254 within 24 to 48 hours after hatching. In a similar study Hansen et al. (1975) noted no apparent reduction in survival of sheepshead minnow embryos and fry containing 77 ug/g-ww of Aroclor 1016. This comparison illustrated a significant effect on developing fish. Halter and Johnson (1974) observed decreases in egg hatchability, mean hatching time, and alevin survival and growth for coho salmon eggs and alevin exposed to Aroclor 1254.

Freeman and Idler (1975) observed a 78% hatching success for eggs from PCB-treated brook trout fertilized with sperm from PCB-treated trout. Hatching success in the control group was 100 percent. When fertilized eggs from either treated or control fish were incubated in water containing 0.2 ppm Aroclor 1254 and 3.8 ppm Corexit 7664, less than one percent of the control eggs and none from the PCB treated fish hatched. Jensen et al. (1970) reported a reduction in Atlantic salmon egg hatchability from 16 to 100% for eggs containing 7.7 to 34 ug/g-lw PCB.

Shimmel et al. (1974) noted significantly fewer sheepshead minnow embryos developing in a 10.0 ug/l Aroclor 1254 solution (7 day exposure) and fewer fry surviving at concentrations greater than 0.1 ug/l. He concluded fry were more susceptible to Aroclor 1254 than embryos, juveniles, or adults.

Temperature may also affect test results. The 10-day LC50 value for Aroclor 1248 at 20°C in channel catfish and bluegill sunfish was more than half that at 27°C (Stalling and Mayer, 1972). The higher test temperature resulted in higher acute toxicity possibly due to the additional stress produced by warmer conditions.

Aquatic Invertebrates

Static and flow-through toxicity tests with aquatic invertebrates show that these species are more sensitive than fish to the toxic effects of PCBs (Mayer et al., 1977). The acute toxicity LC50 values for several freshwater invertebrates are listed in Table 4.1. Crustaceans were found to be more susceptible to PCBs than aquatic insects.

Aroclor 1242 acute toxicities range from 10 ppb for scud (amphipod) to 400 ppb in the damselfly. However, Aroclor 1254 acute toxicities range from 200 ppb for damselfly to 2,400 ppb for the scud. The toxicity of the PCB isomer appears to depend on the percentage of chlorination.

TABLE 4.1

ACUTE TOXICITY OF PCBs to AQUATIC ORGANISMS
(Adapted from Mayer et al., 1977 and USEPA, 1980)

ORGANISM AND COMPOUND	EXPOSURE (days) and TEST TYPE[1]	LC50 (ug/L)[2]
Amphipod		
Aroclor 1242	4F	10
Aroclor 1248	4S	52
Aroclor 1254	4S	2,400
2,3,4' trichlorobiphenyl	4S	70
4,4' dichlorobiphenyl	4S	100
2,4' dichlorobiphenyl	4S	120
2,4,6,2',4',6' hexachlorobiphenyl	4S	150
2,4,5,2',5' pentachlorobiphenyl	4S	210
Crayfish		
Aroclor 1242	7S	30
Aroclor 1254	7S	100
Glass Shrimp		
Aroclor 1254	7F	3
Damselfly		
Aroclor 1242	4F	400
Aroclor 1254	4F	200
Dragonfly		
Aroclor 1242	7S	800
Aroclor 1254	7S	1,000
Fathead Minnow		
Aroclor 1242	4F	15
Aroclor 1254	4F	8
Fathead Minnow (juvenile)		
Aroclor 1242	4F	300
Cutthroat Trout		
Aroclor 1242	4S	5,400
Aroclor 1248	4S	5,700
Aroclor 1254	4S	42,000
Aroclor 1260	4S	61,000

[1] F = flow-through toxicity test; S = static toxicity test

[2] Some LC50 values exceed the water solubility of the PCB compound tested.

TABLE 4.2

COMPARISON OF TOXICITY VALUES DERIVED FROM SHORT- AND LONG-TERM PCB EXPOSURES (Mayer et al., 1977; USEPA, 1980)

Organism	Aroclor [1/] 1242	1248	1254	1260	Other	Time of Exposure	Comments
CHRONIC							
Rainbow trout, *Salmo gairdneri*	12	3.4	27	49		25 days	LC50, 17°C
Bluegills, *Lepomis macrochirus*	120	100	239			25 days	LC50, 17°C
Channel catfish, *Ictalurus punctatus*	132	104	181	465		25 days	LC50, 17°C
ACUTE							
Fathead minnow, *Pimephales promelas*	15		7.7		-	96 hour	LC50, flow thru
Fathead minnow (juvenile)	300					96 hour	LC50, flow thru
Redear sunfish, *Lepomis microlophus*					Capacitor 21 19	96 hour	LC50, flow thru
Largemouth bass, *Micropterus salmoides*					Capacitor 21 2.3	96 hour	LC50, flow thru
Rainbow trout, *Salmo gairdneri*					Capacitor 21 2.0	96 hour	LC50, flow thru

1/ Concentration expressed as µg/1 (ppb)

Nebeker et al. (1974) found the relative toxicity (three week LC50) of eight Aroclor mixtures on water fleas (Daphnia magna) to differ by as much as a factor of ten under static tests. With the exception of Aroclor 1268, the acute toxicity decreased with increasing chlorination. When flow through tests were conducted, the 2 week LC50s for Aroclor 1254 and 1258 were about 0.1-0.05 of the 3 week LC50s derived from static tests. In the same study, midge pupae (Tanytarsus dissimilis) were more sensitive to Aroclor 1254 than the larvae. Results from static tests are generally considered underestimations of toxicity because of aging and adsorption of the test material to the bioassay tanks. Flow through bioassays provide better conditions with which to test toxicity.

Nimmo et al. (1971) observed juvenile pink shrimp (Penaeus duovarum) to be more sensitive to Aroclor 1254 than adult shrimp. A 1 ppb Aroclor 1254 concentration resulted in mortalities within 15 days while adult shrimp mortaltites occurred at 2.4 to 4.3 ppb within 17 to 53 days. They suggested that it was related to molting stage. A similar observation by Wildish (1970) was reported for newly molted scuds (Gammarus sp.).

4.3.3 Sublethal Effects

Reproductive Effects

Fish

Organochlorine residues have been suggested as causal agents in the decline of the

FIGURE 4.5

PERCENT MORTALITY OF ATLANTIC SALMON EGGS
DURING INCUBATION IN RELATION TO PCB RESIDUES
(Jensen et al., 1970)

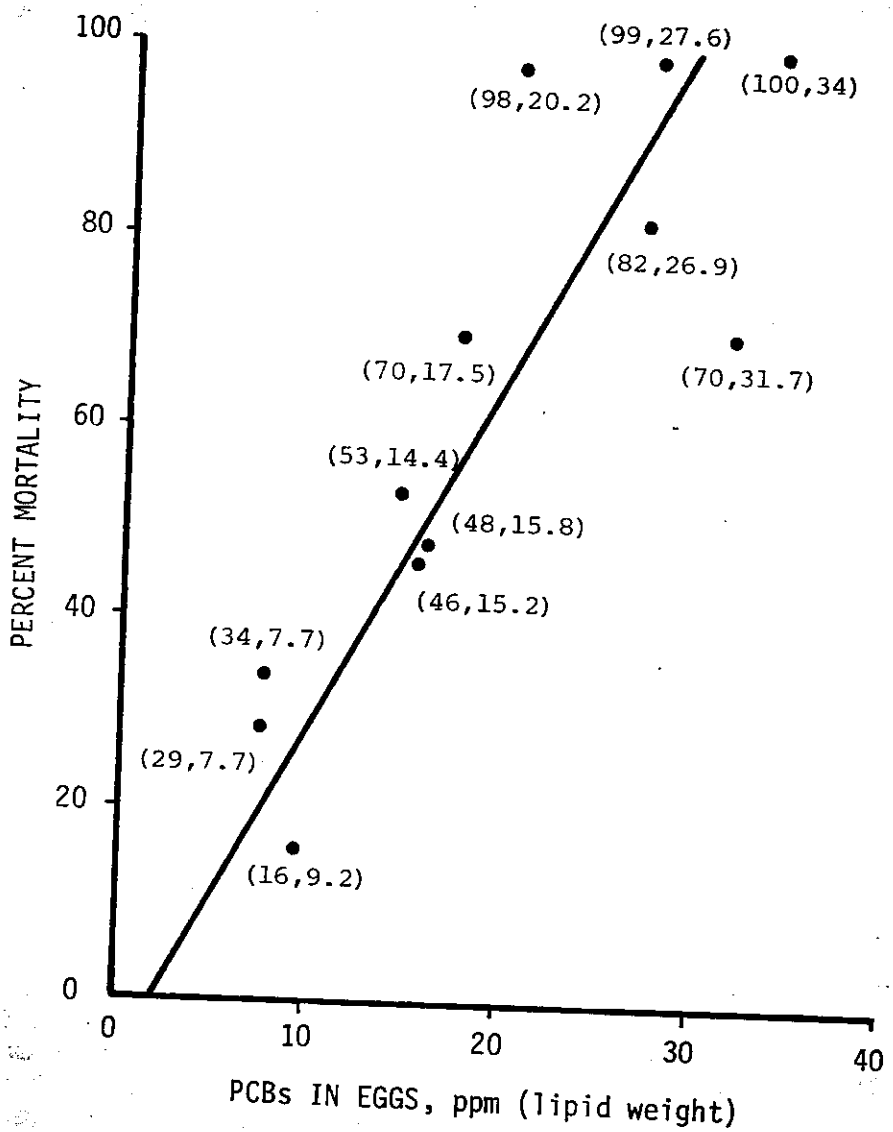


FIGURE 4.6

PERCENT VIABLE HATCH OF BALTIC FLOUNDER EGGS
IN RELATION TO PCB RESIDUES IN OVARIES
(Von Westernhagen et al., 1981)

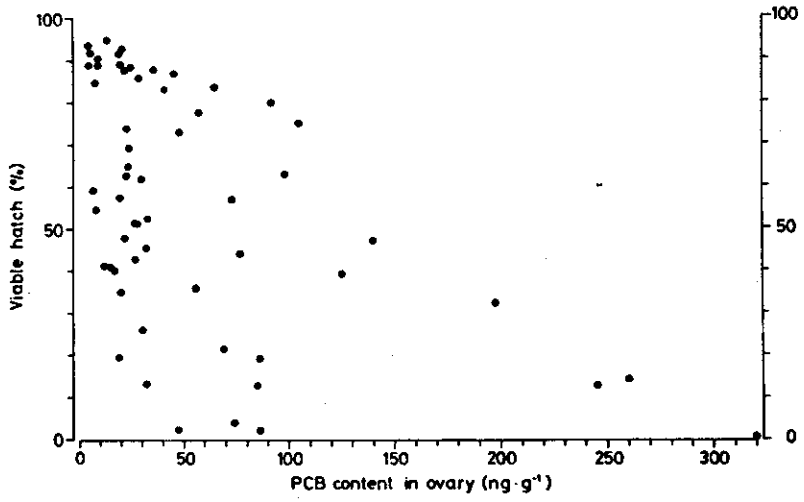


TABLE 4.3

TOXIC EFFECTS OF PCBs IN FISH
(USEPA, 1980)

ORGANISM	AROCLOR 1248	AROCLOR 1254	AROCLOR 1260	TIME OF EXPOSURE	BIOLOGICAL RESPONSE
Brook trout (eggs & sperm) Salvelinus fontinalis		0.2 ppm (mg/l)		21 days	78% hatching success compared to control
Brook trout hatchlings		1.4 ppb (ug/l)		48 days	Reduced growth rate
Brook trout hatchlings		1.4 ppb		118 days	Abnormal bone development
Brook trout hatchlings		0.43 ppb		118 days	No effect level for normal bone development
Fathead minnow Pimephales promelas		0.4 ppb		240 days	20% reduction in standing crop in 2nd generation fish due to death of larvae soon after hatching
Fathead minnow	4.7 ppb		3.3 ppb	30 days (acute)	LC50, flow through bioassay

Invertebrates

Hansen (1974) observed changes in species composition of communities developing from planktonic larvae at 1 ug/l Aroclor 1254. A significant reduction in growth rate and population densities of the ciliate protozoan Tetrahymena pyriformis occurred at the same concentration (Cooley et al. 1972). A reduction in production of young waterfleas and both freshwater (Gammarus pseudolimnoeus) and marine scuds (Gammarus oceanicus) was observed at PCB levels below LC50 values (Wildish, 1970). Chronic effects have been seen for Daphnia magna at 4.3 ppb of Aroclor 1248 and at 2.1 to 15 ppb for Aroclor 1254 (USEPA, 1980).

Phytoplankton

Unicellular algae are affected by PCBs at concentrations similar to those which cause chronic effects in animals. Reduced growth and cell division were observed in unicellular algae (Table 4.4) exposed to Aroclor 1254 at 0.1 ug/l.

The mechanism of PCB toxicity to phytoplankton is not defined. Reductions in growth may be due to inhibition of enzymes involved with nutrient uptake and fixation (Fisher, 1975). In laboratory hatch culture studies, disruption of chloroplast and vacuolar morphology has been reported in the range of 10-50 ug/l (Glooschenko and Glooschenko, 1975).

Plants

PCBs disrupt photosynthesis by damaging chloroplasts and reducing chlorophyll levels and total RNA cellular material (Mahanty and McWha, 1976; Mahanty and Fineran, 1976). Mahanty (1975) observed a population reduction of the floating aquatic plant Spirodela oligorrhiza at 500 ug/l Aroclor 1242 (lowest level tested). This amount is well above the reported water solubility of Aroclor 1242.

Behavioral Effects

In short-term tests (90 minutes), grass shrimp, mosquitofish, and pinfish avoided Aroclor 1254 at concentrations as low as 0.1 mg/l while sheepshead minnow and pink shrimp did not avoid Aroclor 1254 below 10 mg/l (Hansen, 1974). Observations on fish sluggishness and lethargy have also been reported (Johansson et al., 1972).

Tissue Damage

Numerous pathological responses have been observed in fish exposed to PCBs. Tissue damage has been reported in the gills, liver, kidneys, pancreas, and spleen. Pronounced effects on the liver such as degeneration (e.g. lesions) and lipid deposition have been reported (Schoettger, 1971; Hansen et al., 1974; Roberts et al., 1978). In addition to tissue damage, a reduction in growth rate associated with changes in thyroid activity resulting from PCB exposure has been reported (Nebeker, 1976).

Parrish et al. (1972) and Duke et al. (1970) reported growth inhibition of oysters exposed to Aroclor 1254. Mauck et al. (1978) found that 48-day exposure of brook trout hatchlings to 1.4 ug/l Aroclor 1254 resulted in significantly reduced growth rates. No difference in growth of surviving fry was observed after 118 days, presumably due to the mortality of weakened fish. There is evidence that exposure to PCBs results in abnormal bone development. Backbone development in fry exposed for 118 days was significantly altered. The no-effect concentration of Aroclor 1254 for brook trout fry backbone composition, growth and mortality were respectively 0.43, 0.7 and 1.5 ug/l.

TABLE 4.4

EFFECTS OF AROCLOR 1254 ON PLANKTON ORGANISMS
(USEPA, 1980)

SPECIES	EFFECTS	CONCENTRATION (ug/l)[1/]
Diatoms:	Saltwater Species	
Rhizosolenia setiger	No growth in 48 hr. Reduced growth thereafter	0.1
Thalassiosira pseudonana	Reduced growth	25 to 100
Thalassiosira pseudonana	Reduced cell division	1.0
Skeletonema costatum	Reduced growth and reduced cell division	10
Cyllindrotheca closterium	Reduced growth	100
Chaetoceros socilis	Reduced cell division	10
Nitzschia longissima	No effect on cell division	100
Phytoplankton:		
Monochrysis lutheri	Reduced cell division	10
Isochrysis galbana	Reduced cell division	1
Dunallella tertiolecta	Increased cell division	100
Mixed Communities:		
Phytoplankton populations	Toxicity in 24 hrs.	15
Phytoplankton populations	Toxicity in 24 hrs.	6.5
Natural phytoplankton community	Decreased diversity, species ratio altered	100
Phytoplankton communities	Reduced biomass and size	1
Diatoms, Thalassiosira pseudonana and Skeletonema costatum	Reduced growth and carbon fixation in 48 hr.	10
Diatom, Thalassiosira pseudonana and green alga, Dunallella tertiolecta	Species ratio change	1
Diatom, Thalassiosira pseudonana and green alga, Dunallella tertiolecta	Species ratio change	0.1

1/ Some values exceed the solubility of Aroclor 1254 (12 ug/l).

Effects on Immune System

PCBs can indirectly reduce the ability of an organism to combat infection and disease by affecting the thymus and spleen. These organs are an integral part of the immune system of fish. Incidences of fin rot in Dover sole off some major Southern California ocean outfalls correlated with areas with extremely high PCB levels in the sediment (McDermott et al., 1976). Studies have also shown increased viral infection rates in pink shrimp exposed to PCBs (Couch, 1976). Fungus-like lesions on the body and mouth and hemorrhaging around the mouth was observed in pinfish as a result of PCB poisoning (Hansen et al., 1971).

4.4 Conclusion

PCBs are complex mixtures of chlorinated biphenyls and other contaminants. Their effects on aquatic life are primarily a function of the physical and chemical properties of specific isomers. All PCB isomers bioaccumulate rapidly up to 10^4 times in the laboratory and up to 10^6 times ambient water concentrations in the field. This phenomenon to bioconcentrate is a major threat to vital organs of aquatic organisms. The lesser chlorinated biphenyls (less than five chlorine atoms) are more readily biodegradable. The differences in aqueous solubilities, lipophilic nature, and stability account for the pattern of PCB distribution in tissue. The ability of an organism to detoxify and eliminate from the body the lower chlorinated biphenyls makes them of lesser environmental concern than the higher chlorinated mixtures. However, this elimination of PCBs from tissue remains a very slow process.

Early life stages of fish and invertebrates are more sensitive to PCBs than adult stages. The reproductive success and propagation of existing fisheries is highly vulnerable to parts-per-million levels. The detection of PCBs in aquatic life throughout the world is significant. Tissue burdens in Baltic flounder, Atlantic salmon, and striped bass have often exceeded levels where poor reproductive success has been reported in other fish species. In view of their high bioaccumulation

5.0 MAMMALIAN TOXICOLOGY AND HUMAN HEALTH

5.1 Introduction

Controversy has recently risen over whether PCBs pose a significant risk to human health. The central theme of this chapter is to compare differing interpretations by three groups of existing scientific studies: (1) a detailed review conducted by the consulting toxicology firm of Drill, Freiss, Hayes, Loomis, and Shaffer, Inc. in February, 1982 for the Edison Electric Institute (EEI), (2) a more cursory review prepared by Ecology and Environment, Inc. in November 1981 for the Chemical Manufacturers Association (CMA), and (3) the USEPA's Response of August 1982 to these reviews. We have also provided our own evaluation of existing studies.

In each subchapter, points of difference and/or agreement between the USEPA and the EEI and CMA consultants are compared. In addition, we have based this review on (1) an assessment of the existing literature, (2) communication with several researchers active in the field, and (3) comments by peer reviewers.

This review highlights recent findings in the areas of animal cancer bioassays, metabolism, and biochemical bases for toxicity. A discussion of the significance of existing epidemiological studies is also included.

Although commercial mixtures of polychlorinated biphenyls (PCBs) show very low acute toxicity in mammalian systems, these compounds are of concern because they are bioaccumulated and highly resistant to metabolism and biodegradation. Concern over possible effects on human health has concentrated on long-term PCB exposure. In populations with no history of exposure to PCBs, these substances have been detected in human blood, adipose tissue, and mothers' milk. Animal studies have shown that chronic PCB exposures have adverse reproductive effects and increase the activity of certain liver enzymes. Although a number of animal studies have been negative, some studies in mice and rats have shown PCBs to be animal carcinogens. The Working Group of the International Agency for Research on Cancer (IARC) in 1978 placed polychlorinated biphenyls on a list of 18 chemicals that "are probably carcinogenic in humans" (IARC, 1978). The National Toxicology Program's Second Annual Report on Carcinogens (December 1981) listed PCBs among a total of 88 substances that are either known or anticipated to be carcinogens.

Much of the existing information on the toxicity and health effects of PCBs is equivocal. Thus, it is possible for reviewers to differ in any assessment of risks posed by these chemicals. The effect of highly toxic contaminants in commercial mixtures complicates understanding of the toxic effects of "pure" PCBs. The many different polychlorinated biphenyls themselves have diverse physical, chemical, and biological properties. And it is important to distinguish between studies that investigate specific isomers of chlorinated biphenyls, those that utilize commercial mixtures, and those that examine environmental residues.

In "Summary of the Health Effects of PCBs," prepared by Ecology and Environment, Inc. for the Chemical Manufacturers Association, the authors concluded that "any exposure to PCBs does not pose a significant health risk to humans" (Ecology and Environment, Inc. 1981, p. 1). They also stated that animal data indicate a low acute hazard potential; minimal mutagenic, reproductive, and teratogenic risks; and that "the carcinogenic potential of this compound has not been convincingly demonstrated in an animal model relevant to man." The authors assert that, at most, PCBs act as weak promoters, not initiators of cancer, and that there are thresholds that suggest safe levels to PCBs. Epidemiological studies indicate that the usual consequences of PCB exposure are reversible skin disorders. "Therefore, it appears

that PCBs are not a remarkable toxicant, but a chemical which requires high doses to produce harmful effects" (Ecology and Environment, Inc., 1981, p. 3).

The Drill et al. report (1982), "Potential Health Effects in the Human from Exposure to Polychlorinated Biphenyls and Related Impurities," prepared for the Edison Electric Institute, reached conclusions similar to those of Ecology and Environment, Inc. Of effects in animals, only dermatological effects have been noted in humans occupationally exposed to PCBs. "Since the risk to human health from even high level occupational exposures has been shown by the studies available to be low, it may be concluded that much lower human exposure levels do not present significant risks" (Drill et al., 1982, p. 13).

On August 4, 1982, Dr. Seymour Freiss, a principal partner in DFHL&S, the firm authoring the Drill et al. report, summarized his interpretation of existing health effects data to staff members of several California regulatory agencies. A summary of Dr. Freiss' interpretation is contained in Appendix B of this report.

Dr. Freiss concluded that the two health effects observed in workers exposed to PCBs and in laboratory tests animals were reversible and, therefore, not serious. These effects are the skin disorder known as chloracne and induction of certain enzymes known as mixed function oxidases.

In support of this interpretation, Dr. Freiss noted that in laboratory experiments, "the bulk of the studies on animals yielded negative results on cancer in all tissues in all species, with an exception of some unverified results on liver cancer in mice and rats" (see Appendix B, p.2). The adverse reproductive effects observed in animals generally occurred "at very high doses", and the probability of

chemicals exert their effects on mammalian systems. The pharmacokinetics of individual polychlorinated biphenyls is affected not only by the degree to which the biphenyl ring is chlorinated, but also by the location of chlorine atoms on the ring.

The absorption, storage, and metabolism of polychlorinated biphenyls have been reviewed by Safe (1980), in a chapter that carefully distinguishes between the pharmacokinetics of PCB isomers and PCB commercial mixtures. Because there is variation between the chemical and physical properties of individual PCB isomers, there are differences with respect to rates of mammalian uptake, retention, and metabolism. Thus, there are differences between the chromatographic profile of a parent commercial mixture (containing 50 or more isomers) and the tissue sample obtained from an experimental animal that consumed the commercial mixture (Safe 1980). There are also species differences: rats and dogs will metabolize and excrete certain isomers (for example, 4,4'- dichlorobiphenyl and 2,2',3,3',6,6'- hexachlorobiphenyl) several times more rapidly than monkeys (Matthews and Kato, 1979).

Little pharmacokinetic data has been reported on humans (Letz, 1981), but it is known that gas chromatographic profiles of PCBs in human tissues differ from commercial products (Wolff et al., 1982).

5.2.1 Absorption

Polychlorinated biphenyls are readily absorbed by dermal, inhalative and ingestive routes of entry. Data obtained from laboratory rats show similar patterns of systemic toxicity after dermal, oral, or inhalational administration. Although specific information on the rate or degree of PCB absorption by any route for any [redacted] (Letz, 1981), some general observations can be made

distribution and excretion. Four isomers were tested: a one chlorine biphenyl (4-chloro-), a two chlorine biphenyl (4,4'-dichloro-), a five chlorine biphenyl (2,2',4,5,5'-pentachloro), and a six chlorine biphenyl (2,2',4, 4',5,5'-hexachlorobiphenyl). The PCBs moved rapidly from the blood and initially were stored largely in liver and muscle. Subsequent redistribution to adipose tissue was related to degree of chlorination (Figure 5.1). Maximum storage of the monochlorobipenyl in adipose tissue occurred one hour after injection and accounted for 11.6 percent of the administered dose. Storage of the dichlorobiphenyl was maximum at two hours with 52.7 percent of the administered dose contained in adipose. Maximum pentachlorobiphenyl storage in adipose occurred at one day (23.5 percent). In contrast, the hexachlorobiphenyl had not reached maximum storage 42 days after injection, at which time, 85.2 percent of the administered dose was contained in adipose tissue. Excretion accounted for more than 90 percent of the mono-, di-, and pentachlorobiphenyl during the 42 day period, but only 15 percent of the hexachlorobiphenyl was excreted (Matthews and Anderson, 1975). Over time, PCBs - particularly those most resistant to metabolic conversion-accumulate at highest levels in the fat and skin depots, with distribution following the order of adipose tissue >skin >liver >muscle (Safe, 1980).

Gas chromatogram PCB profiles in human tissue are similar for adipose, blood, and breast milk, a finding suggesting that, while certain tissues accumulate higher concentration of PCBs, there is not tissue specific, preferential partitioning of individual isomers (Safe, 1982). In charcoal fractionation studies of human adipose tissue, it has been shown that there is preferential concentration of PCB isomers with a lower degree of ortho substitution than is observed in commercial mixtures (Safe, 1982).

(Note: the carbon atom numbering system for the biphenyl molecule is given in Figure 2.1. The ortho positions are carbons 2 and 6, the meta positions are carbons 3 and 5, and the para position is carbon 4.)

[redacted]

FIGURE 5.1

STORAGE OF FOUR PCB ISOMERS IN RAT ADIPOSE TISSUE
(Data from Matthews and Anderson, 1975)

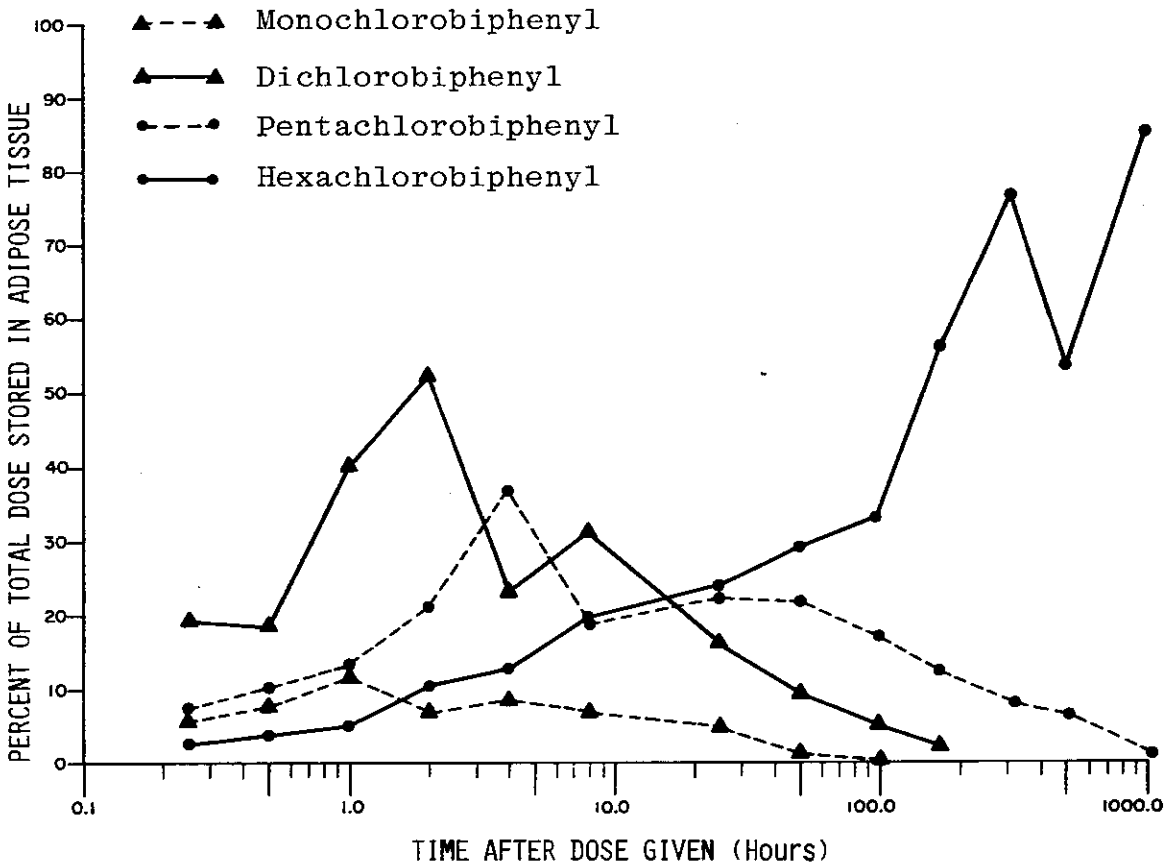


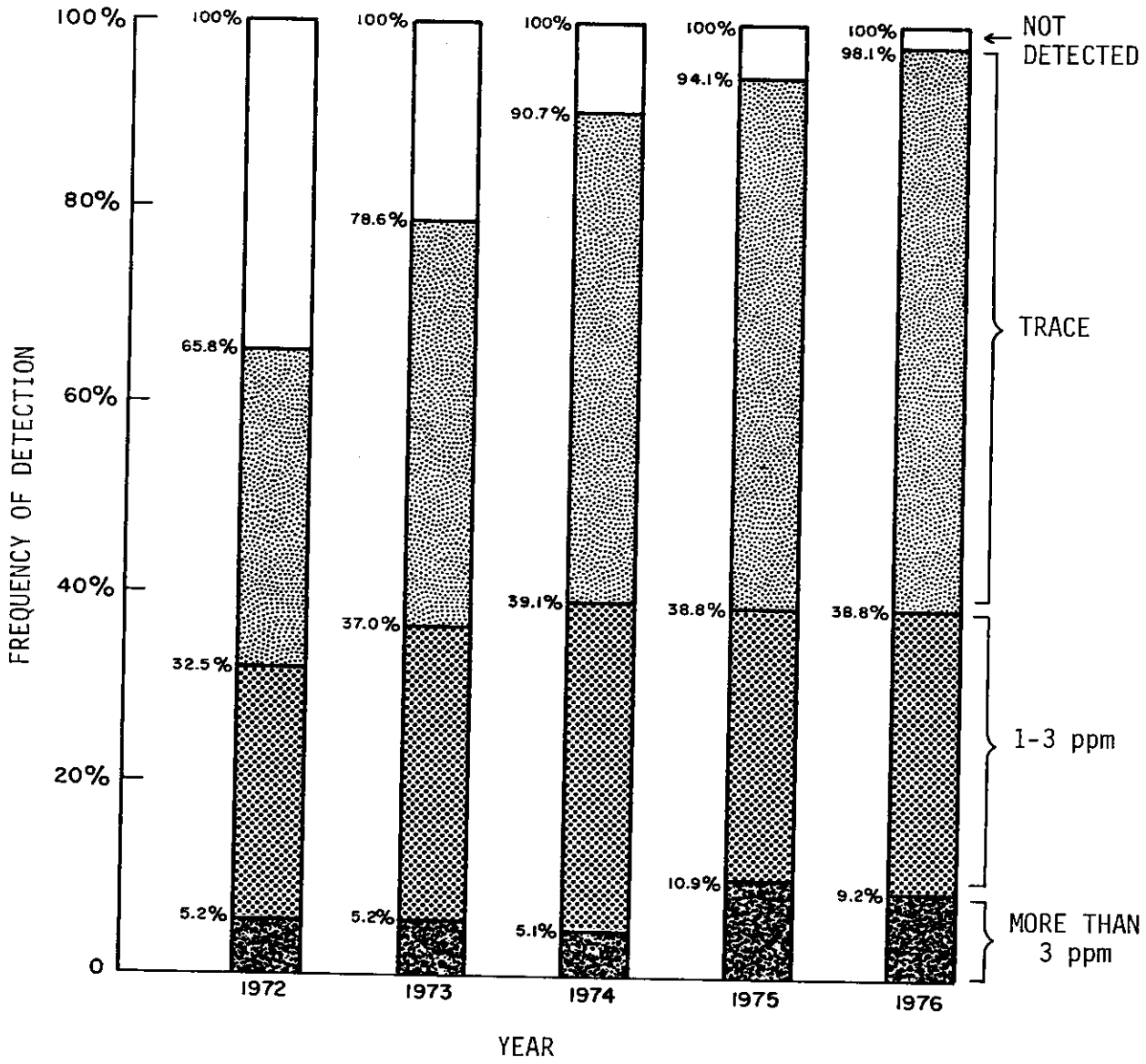
TABLE 5.1

PCB RESIDUES IN HUMAN TISSUES

MEDIAN LEVEL	RANGE	LOCATION	POPULATION	REFERENCE
1. FAT TISSUE				
1 ppm	0.3-10.0 ppm	US, Europe, Japan	general population	Wasserman, et al., 1979
0.8 ppm	0-2.8 ppm	Norway	general population	Landrigan, 1980
0.9 ppm	0.1-6.6 ppm	Canada	general population	Landrigan, 1980
75 ppm		Japan	Yusho patients	Wasserman et al., 1979
24 ppm	2-271 ppm	USA	occupational (capacitor manufacturing facility)	Wolff et al., 1982
2. SERUM				
17.2 ppb	3.2-158 ppb	Triana, Alabama	community	Kreiss et al., 1981
18.8 ppb	6-79 ppb	Bloomington, Indiana	community	Kreiss et al., 1981
77 ppb	25-366 ppb	Michigan	fisherman	Kreiss et al., 1981
3. PLASMA				
33 ppb	11-720 ppb	Taiwan	PCB poisoning patients	Chen et al., 1980
83 ppb	2-1,412 ppb	USA	capacitor workers	Wolff et al., 1982
	100-650 ppb	Japan	capacitor workers	Wasserman et al., 1979
	75-1,900 ppb	Finland	capacitor workers	Wasserman et al., 1979
4. MILK (Fat Weight)				
1.1 ppm	0.1-2.5 ppm	Canada		Landrigan, 1980
1.2 ppm	0.2-4.9 ppm	Japan		Landrigan, 1980
1.35 ppm	Tr - 5.1 ppm	Michigan		Wickizer and Brilliant, 1981

FIGURE 5.2

PCB LEVELS IN HUMAN ADIPOSE TISSUE
(USEPA, 1977)



NOTE: Because analytical methods and limit of detection were not specified in profile report, histograms may not be indicative of trends.

mixtures of PCBs to laboratory animals has shown that the lower chlorinated isomers are preferentially eliminated (Safe, 1980). Because PCBs are very complicated mixtures, they are not suitable for metabolic studies. Rather, studies have focused on pure isomers (Sundstrom et al., 1976). The chief metabolic products are biphenyls, biphenyldiols, and dihydrohydroxy-biphenyls, although other metabolites such as biphenyltriols and methoxy derivatives have been noted (USEPA, 1980). In a recent review, Safe (1980) summarized what has been learned about the oxidation of chlorinated biphenyl isomers:

1. Hydroxylation is favored at the para position in the least chlorinated ring (unless there is steric hindrance such as 3,5 dichloro substitution).

2. In lower chlorinated biphenyls, several sites are readily hydroxylated: the para positions of both rings and the carbon atoms located opposite to the chlorine substitution.

3. While not a requirement, the presence of two adjacent, unsubstituted carbon atoms facilitates oxidative metabolism of a PCB.

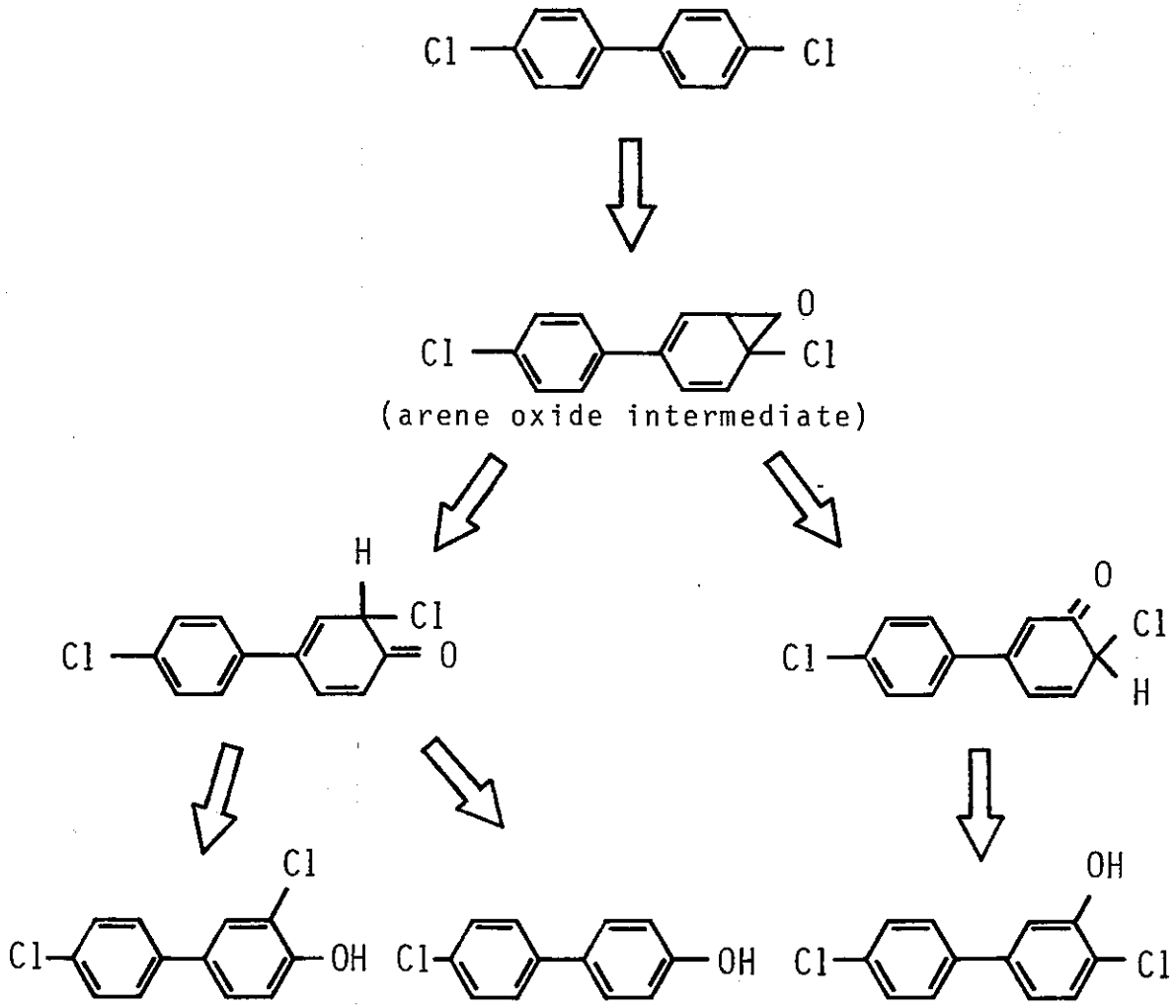
4. With increasing chlorination of both phenyl rings, the rate of excretion of metabolites is decreased.

5. The distribution of various metabolic products from a given PCB isomer is species dependent.

Animal feeding studies have demonstrated an inverse relationship between degree of chlorination and rate of metabolism. Not only are the less chlorinated more

FIGURE 5.3

POSSIBLE METABOLIC PATHWAYS FOR 4,4'-DICHLOROBIPHENYL IN THE RABBIT
(Adapted from Sundstrom et al., 1976)



PERCENT EXCRETED AS:

	3,4'-dichloro-biphenylol	4'-chloro-4-biphenylol	4,4'-dichloro-3-biphenylol
Feces	0.7	0.2	2.0
Urine	2.0	2.2	7.5

is about one day in the rat and dog, but the half-life is two to three weeks in the monkey. Thus, 4,4'-dichlorobiphenyl can potentially bioaccumulate in the monkey but not in dogs or rats (Matthews and Kato, 1979). Similarly, the half-life of 2,2',3,3',6,6'-hexachlorobiphenyl is about one day in the rat and dog, but five days in the monkey (Matthews and Kato, 1979). The dog seems particularly able to metabolize certain isomers. Although 2,2',4,4', 5,5'-hexachlorobiphenyl has a very

There is substantial scientific agreement on the following aspects of PCB pharmacokinetics:

1. The structure (location of chlorine atoms and degree of chlorination of the biphenyl molecule) of individual PCB isomers determines the absorption, metabolism, and excretion of the compound. It is important to distinguish between the activity of (1) commercial PCB mixtures, (2) environmental and tissue patterns of PCBs, and (3) pure isomers.

2. PCBs are readily absorbed through the gut, the skin and the lungs.

3. PCBs may initially concentrate in blood, liver, and muscle; however, long term mammalian storage is primarily in adipose (fat) tissue and skin.

4. PCBs that are readily metabolized are most rapidly excreted, with the lower-chlorinated PCBs predominate in urine.

5. Many of the higher-chlorinated biphenyl isomers are accumulated almost indefinitely.

6. PCBs can be transferred either across the placenta or by breast milk. Quantitatively, transfer in breast milk is probably more significant (Letz, 1981).

7. Non-human primates (such as monkeys) may retain PCBs more efficiently than rodents, or other mammals. Dogs, in particular, rapidly metabolize many chlorinated biphenyl isomers. Obvious species differences exist in the ability of mammals to oxidatively metabolize chlorinated biphenyls.

8. Major metabolic products are produced either through formation of arene oxide intermediates or by direct hydroxylation. Those chlorinated biphenyls with one or more pairs of adjacent, unsubstituted carbon atoms are most readily metabolized.

9. Because arene oxide intermediates have been shown to covalently bind to cellular macromolecules, there is the potential for PCBs and their metabolic products to induce damage in the cellular genetic material.

5.3 Animal Toxicology

Several detailed reviews have been written in recent years that assess the toxicology of PCBs (Kimbrough et al., 1978; USEPA, 1980; Letz, 1981; Drill et al., 1982; USEPA, 1982); this section highlights a few areas of interest: acute toxicity and aspects of chronic toxicity such as reproductive, gastric, immunosuppressive, and endocrine effects. Other aspects of toxicity, the subjects of carcinogenicity, mutagenicity, enzyme induction, and human toxicology are discussed in subsequent sections. A systematic evaluation of the PCB toxicology literature is complicated by a number of factors that are listed in the review of Drill et al. (1982).

5.3.1 Acute Toxicity

Commercial mixtures of PCBs exhibit low acute toxicity in laboratory animals, a finding consistent with the lack of acute effects observed from human occupational exposure. Oral LD_{50} values range from 1 to 10 g/kg in mice, rats, and rabbits (Letz, 1981), a range classified as "slightly toxic" to "practically non-toxic" by the American Industrial Hygiene Association. Two general observations about the acute toxicity of PCBs have been made: the young are more sensitive than adults and females are more susceptible than males (Kimbrough et al., 1978). The pathological findings in rats subjected to acutely toxic doses of Aroclor 1242 include central nervous system (CNS) depression, anorexia, liver and kidney damage, coma, and death (Kimbrough et al., 1978; Letz, 1981). Consistent findings after deaths in rats, rabbits, and guinea pigs have involved the liver and include fatty infiltration, atrophy, and occasionally necrosis (Letz, 1981). In rats, the acute oral toxicity of PCB mixtures decreases with increasing chlorine content, a finding that may be explained by observations that highly chlorinated biphenyls are absorbed less and undergo less metabolic transformation (Letz, 1981). The lower chlorinated biphenyls may derive much of their acute toxicity from the activity of hydroxylated metabolic products (J.D. McKinney, Written Communication, August 12, 1982).

5.3.2 Subacute and Chronic Toxicity

Continuous low level exposure to both commercial PCB mixtures and individual polychlorinated biphenyls show cumulative toxic responses that are of greater concern than the relatively low acute toxicity of PCBs (Kimbrough et al., 1978). There is considerable variation between animal species with the most consistent finding being various adverse effects on the liver (Letz, 1981).

Some specific aspects of chronic toxicity are reported in subsequent sections. Section 5.3.3 discusses reproductive effects, and 5.5.4 mentions immunosuppression, stomach lesions, and endocrine effects.

One mammalian species, mink, seems especially sensitive to PCBs. There was 100 percent mortality when mink were fed 3.6 ppm Aroclor 1254 in the diet for 105 days (Platonow and Karstad, 1973).

In rats fed 100 ppm PCBs (Aroclor 1248, 1254, and 1262) in their diet for six weeks, symptoms included liver hypertrophy, marked fatty infiltration, and parenchymal cell degeneration (Allen and Abrahamson, 1973). PCB mixtures with lower chlorine content showed higher toxicity (Allen and Abrahamson, 1973). In an eight month study, Kimbrough, Linder and Gaines (1972) noted that morphological changes in rat livers were generally more pronounced with Aroclor 1254 than with Aroclor 1260.

Non-human primates were more sensitive than rodents to PCBs (Letz, 1981; IARC, 1978). At chronic doses of 2.5 ppm Aroclor 1248, given for one year, facial edema and acne were observed in rhesus monkeys (Kimbrough et al., 1978). At 100 and 300 ppm (approximately 10 mg/kg day) of Aroclor 1248 for 2-3 months, monkeys experienced high morbidity within one month and almost 100 percent mortality within three months (Allen, 1975). In contrast, rats were able to survive for one year on the 100 ppm dose (Allen, 1975).

Based on their studies with the rhesus monkey, Allen and his colleagues (Allen, 1975; Allen et al., 1979) have proposed that this animal provides a suitable model for evaluating PCB intoxication in humans. Table 5.2 compares some responses of rats, non-human primates, and humans to PCBs. For a number of health effects, the human response is closer to that of non-human primates than to the effect on rats.

TABLE 5.2

RESPONSES OF PRIMATES AND RATS TO PCBs
(Adapted from Letz, 1981; IARC, 1978)

RESPONSE	MAN	MONKEY	RAT
Susceptibility to toxicity	High	High	Moderate
Acne	Yes	Yes	No
Hyperpigmentation of skin	Yes	Only infants	No
Conjunctivitis	Yes	Yes	No
Swelling of eyelids	Yes	Yes	No
Subcutaneous swelling	Yes	Yes	No
Keratin cysts in hair follicles	Yes	Yes	No
Hyperplasia of hair follicle	Yes	Yes	No
Gastric hyperplasia	not analyzed	Yes	No
Liver hypertrophy	Yes	Yes	Yes
Liver enzyme change	not analyzed	Yes	Yes
Decreased no. of red-blood cells	Yes	Yes	No
Decreased hemoglobin	Yes	Yes	No
Increased leucocytes (white blood cells)	Yes	Yes	No
Reproductive effects (inhibition)	?	Yes	Yes
Carcinogenesis	?	?	Yes
Rate of Metabolism and Excretion (half-life of PCB isomers)	?	Slow	Rapid
4,4'-dichlorobiphenyl	?	2-3 weeks	1 day
2,2',3,3',6,6'- hexachloro-biphenyl	?	5 days	1 day

Recent studies with pure isomers of PCBs have shown that some compounds exhibit toxicity at low levels of exposure. McNulty et al. (1980) compared the effects of two tetrachlorobiphenyls and Aroclor 1242 in young rhesus males. A diet containing 0.3 to 3.0 ppm of the ortho-unsubstituted 3,3',4,4'-tetrachlorobiphenyl caused chloracne, weight loss and death in one to six months. The lesions at autopsy included squamous metaplasia, atrophy of the sebaceous glands, involution of the thymus, and hyperplasia of the gastric mucosa. A diet at the same doses of the ortho-substituted 2,2',5,5'-tetrachlorobiphenyl produced no adverse clinical or pathological effects. These clinical and pathological responses are similar for higher doses of Aroclor 1242, but were not observed in this series of experiments at levels of up to 5 ppm Aroclor 1242 (McNulty et al., 1980).

In a companion study (Becker et al., 1979), using dietary doses of 3, 10, 30, and 100 mg/kg of Aroclor 1242, gastric lesions were observed at 30 and 100 mg/kg in two weeks but not until 10 weeks with the lower doses. Since the 3,3',4,4'-tetrachlorobiphenyl isomer constitutes only 0.2 percent of Aroclor 1242, it was concluded that this isomer probably did not account for the toxicity of the Aroclor 1242 mixture (McNulty et al., 1980).

Comparison of the effects of ortho-substituted and ortho-unsubstituted tetrachlorobiphenyls is consistent with the hypothesis that PCB toxicity is inversely related to the degree of chlorination in the ortho positions of the biphenyl ring (McNulty et al., 1980).

It is significant that the series of experiments by McNulty et al. with rhesus monkeys showed mortality at lower dose levels with pure PCB isomers than with commercial Aroclor mixtures. This finding casts doubt on the interpretation that severe biological effects of commercial PCB mixtures are attributable solely to contaminants such as the chlorinated dibenzofurans.

5.3.3 Reproductive Effects

Drill et al. (1982) and USEPA (1982) disagree on the reproductive effects of PCBs. Drill et al. (1982) suggest that contaminants in commercial PCB mixtures may account for adverse effects, and "in most species, PCBs produce deleterious effects at high dosages." USEPA (1982), in contrast, states that, "some PCBs have the ability to alter reproductive processes in mammalian species, sometimes even at doses that do not cause other signs of toxicity." The USEPA document notes the female reproductive system appears to be more sensitive than the male, perhaps as a result of PCB-induced alterations in the metabolism of sex hormones. On the issues of fetotoxicity and developmental toxicity, EPA believes that these are areas for concern, even though some aspects have not been well-studied.

PCBs have significant adverse reproductive effects in mammalian species. For commercial Aroclor mixtures, the magnitude of the effect increases with higher doses and generally decreases with increased degree of chlorination (Letz, 1981). Reproductive effects include alterations in estrus cycles, failure of implantation, spontaneous abortions, still births, low birth weight offspring, and decreased post-natal survival (Letz, 1981). Reproductive processes may be affected at levels that do not cause other signs of toxicity (USEPA, 1982).

In mice fed Clophen A-60 (a German-manufactured commercial PCB mixture with a chlorine content comparable to Aroclor 1260) at a dose of 1.0 mg/kg/day for 10 days, lengthened estus cycles and a decreased number of successfully implanted ova occurred (Orberg and Kihlstrom, 1973). Since both the estus cycle and implantations are regulated by sex-hormones, USEPA (1982) has interpreted this finding as representing an effect upon the metabolism of sex hormones.

Rats exposed to Aroclor 1254 at levels of 20 ppm or more in the diet had fewer offspring per litter than did controls; in contrast, a 500 ppm dietary level of Aroclor 1260 reduced litter size (Linder et al., 1974). Dietary doses of 5 ppm Aroclor 1254 and 100 ppm Aroclor 1260 did not affect rat reproduction through two generations (Linder et al., 1974).

Mink show very sensitive reproductive effects from administration of PCBs (Auerlich and Ringer, 1977). Reproductive failure at commercial mink farms has been linked to PCB-containing Great Lakes Coho salmon processed as fish meal (USEPA, 1980). In a study of female mink, all eleven animals died from a dose of 30 ppm (10 ppm each of Aroclors 1242, 1248, and 1254) (USEPA, 1980). Reproductive failure was observed in mink at dose levels of one to five ppm (Letz, 1981), with complete reproductive failure at 5 ppm (Kimbrough et al., 1978). Auerlich and Ringer (1977) noted that Aroclor 1254 exerted a detrimental effect on reproduction at levels of 2 ppm, an effect not noted with Aroclors 1016, 1221, or 1242.

In a series of studies conducted at Madison, Wisconsin (Allen, 1975; Allen and Barsotti, 1976; Allen et al., 1979; and Barsotti et al., 1976), the reproductive effects of Aroclor 1248 on female rhesus monkeys have been reported. Allen and Barsotti (1976) fed Arcolor 1248 to female rhesus monkeys at dietary doses of 2.5 and 5.0 ppm for 18 months and observed changes in menstrual cycles, frequent resorption, and spontaneous abortions (Table 5.3). Not only was breeding performance affected, but infant body weights were diminished (Table 5.4). At lower dietary doses of 0.5 and 1.0 ppm, these reproductive effects were not observed.

Spontaneous outbreaks of disease, featuring weight loss and reproductive dysfunction, have been reported in a United States primate colony laboratory. The disease has been linked to PCBs present in the cement floor sealer (McConnell et al., 1979; McConnell, 1980).

Another possible reproductive effect of PCBs and other toxic substances is teratogenicity. Teratogens are substances that cause defects of fetal development (malformations).

Several reviewers have commented that PCBs have no known (Drill et al., 1982) or clearly defined teratogenic effect in mammals (Kimbrough et al., 1978; Letz, 1981). The recent EPA response (USEPA, 1982) to the Drill et al. (1982) report notes that teratogenicity has several definitions and addresses, instead, the subject of "developmental toxicity," defined as "developmental deviation, either structural or functional in nature, which occur as a result of adverse effects of the environment on developing systems." (USEPA, 1982, p. 29). Types of developmental deviations include: (1) death of the developing organism, (2) structural abnormality (malformation), (3) growth retardation, and (4) functional deficiency. USEPA (1982) concluded that PCBs do present a risk to developing individuals.

On the issues of fetotoxicity and teratogenicity, Drill et al. (1982) cite a number of negative studies and conclude that "PCBs present no appreciable risk of teratogenicity in offspring of human females under occupational conditions." Drill et al. (1982) do note that at relatively high doses (greater than 10 mg/kg), there have been positive fetotoxic results in rats, dogs, and rabbits. They also cite a study by Marks et al. (1981) reporting teratogenicity from a dose of 2 mg/kg/day (2 ppm) hexachlorobiphenyl isomers in mice. However, Drill et al. conclude that "commercial PCB mixtures showed no teratogenic activity in mice, rats, rabbits and monkeys," (Drill et al., 1982, p. 111) and "the scientific literature presently supports the conclusion that PCBs present no appreciable risk of teratogenicity for humans, in our opinion, in view of the essentially negative test results in four test species and the questionable positive findings in the dog and in swine." (Drill et al., 1982, p. 112).

TABLE 5.3

REPRODUCTIVE EFFECTS OF AROCLOR 1248 ON RHESUS MONKEYS
(Allen et al., 1979)

	Breeding Performance			
	During PCB Exposure		One Year After PCB Exposure	
	2.5 ppm	5.0 ppm	2.5ppm	5.0 ppm
Total impregnated	8/8	6/8	8/8	7/7
Absorptions/resorptions	3/8	4/8	1/8	1/7
Stillborn	0/8	1/8	0/8	1/7
Normal births	5/8	1/8	7/8	5/7

TABLE 5.4

BODY WEIGHTS OF INFANT MONKEYS BORN TO MOTHERS RECEIVING 2.5 or 5.0 PPM PCB (AROCLOR 1248) IN THEIR DIETS PRE- AND POSTPARTUM
(Allen et al., 1979)

Age (weeks)	Body Weight (grams)* Control	Experimental
0	507 ± 59	399 ± 22
2	601 ± 47	463 ± 40
4	711 ± 58	517 ± 12
6	796 ± 81	601 ± 38
12	1003 ± 71	765 ± 57

* Mean ± one standard deviation

Work with pure hexachlorobiphenyl isomers (Marks and Staples, 1980; Marks et al., 1981) has indicated that some isomers are teratogens in mice. Administration of 3,3',4,4',5,5'-hexachlorobiphenyl at doses of 0.1, 1, 2, 4, 8, and 16 mg/kg/day by gavage during 6 to 15 days of gestation showed a significant increase in the percentage of malformed fetuses at the higher doses: 2 mg/kg/ day (11.7%); 4 mg/kg/day (36.9%); 8 mg/kg/day (65.6%) and 16 mg/kg/day (60.6%) versus the control group (0.9%). None of the dosages was lethal to the gestating mothers (Marks et al., 1981). A similar study by Marks and Staples (1980) showed that 2,2',3,3',4,4' -hexachlorobiphenyl was teratogenic. Four other hexachlorobiphenyl isomers (2,2',3,3'5,5'-, 2,2',3,3'6,6'-, 2,2'4,4'5,5'- and 2,2'4,4'6,6'-) were not teratogenic.

5.3.4 Other Chronic Toxicological Effects

Other reported mammalian responses to PCBs include suppression of the immune system, stomach lesions, and effects on the endocrine system.

Immunosuppressive effects have been reviewed by several authors (Kimbrough et al., 1978; Vos et al., 1980; Letz, 1981; Drill et al., 1982; and USEPA, 1982). The Kimbrough review cites the following observations: (1) lymphoid atrophy in rabbits and guinea pigs, (2) suppression of humoral immune reactions in rabbits and guinea pigs, and (3) suppression of cell-mediated immune responses in guinea pigs. Because the PCBs tested in the above studies were all commerical mixtures, the role of contaminants, such as dibenzofurans, may be implicated in the response (Kimbrough et al., 1978).

Allen and Barsotti (1976) measured the effects of PCB intoxication on infant monkeys. They fed female rhesus monkeys doses of 2.5 and 5.0 ppm Aroclor 1248 in the diet before and during gestation and while the infants were nursing. Three of the six infants (one live birth of six conceived in the 5.0 ppm group and five live births of eight conceived in the 2.5 ppm group) died within 8 months of birth. At necropsy, lymph nodules of the spleen were small and without apparent germinal centers; only a remnant of the thymus was present. Microscopic examination of the thymus revealed a loss of cortical thymocytes and few medullary thymocytes (Allen and Barsotti, 1976).

McNulty et al. (1980) reported that 3.0 ppm in the diet of 3,3',4,4'-tetrachlorobiphenyl caused involution of the thymus in young male rhesus monkeys. Upon autopsy, the thymus gland was "not grossly recognizable". No effect occurred from administration of a second tetrachlorobiphenyl, the 2,2',5,5'- isomer.

Drill et al.(1982) agree that immunosuppression may be demonstrable at higher doses. However, at low doses levels, they state that there is no likelihood of significant immunosuppression. Drill et al. (1982) suggest that a dietary dose of 2.5 to 5.0 ppm for 6 months may be an immune response threshold in the rhesus monkey. This review does not reference the 1980 work of McNulty et al. with pure isomers of tetrachlorobiphenyl.

In its response to Drill et al., USEPA (1982) has concluded that PCBs are significant immunosuppressive agents. Because the immune system provides an organism with defense against disease, and because PCBs are bioaccumulated within the organism and the food chain, low levels of PCBs are of concern. "Immunosuppressive effects of PCBs may be important because they are seen at low dosages, and in the absence of general toxicity" (USEPA, 1982, p. 96).

Several studies have reported various gastric changes in rhesus monkeys. Allen and Barsotti (1976) reported invasion of "glandular elements" into muscularis mucosa

and penetration of acute inflammatory cells into submucosa in rhesus infants born to and nursed by mothers receiving PCBs in the diet. At high doses (300 ppm), Allen (1975) observed thickening of the gastric mucosa in rhesus monkeys. McConnell et al. (1979) reported severe hypertrophy of the glandular stomach of monkeys in primate colonies that had been unintentionally exposed to PCBs.

Becker et al. (1979) fed Aroclor 1242 at dietary doses of 3, 10, 30, and 100 ppm to young male rhesus monkeys. Serial biopsies were taken to observe morphological changes in the gastric mucosa. Severity of gastric lesions correlated directly with both duration and level of exposure. At 30 and 100 ppm, changes were observed at two weeks; at the lower doses of 3 and 10 ppm, changes were not seen until ten weeks. Becker et al. noted that rhesus monkeys developed pathological changes, such as gastric lesions and liver alterations, at much lower doses (less than 5 ppm) than do rats, mice, and rabbits. The latter animals require dietary doses of 100 ppm or more for many months (Becker et al., 1979).

Severe disruption of the gastric mucosa of the stomach has been reported in the rhesus from administration of 3,3',4,4'-tetrachlorobiphenyl at dietary levels of 3.0 ppm (McNulty et al., 1980).

Drill et al. (1982) note that the finding of PCB-induced gastric lesions in monkeys are variable and may be species specific, since these lesions have not been reported in other animals. The USEPA Response (1982) disagrees, stating that these lesions are probably not species specific.

Endocrine effects noted by Allen (1975) in the rhesus monkey were the increased metabolism of steroid and steroid-like compounds, alterations that may affect the ability to conceive. Allen et al. (1979) also noted a prolongation and flattening of the progesterone peak during the rhesus menstrual cycle.

The Drill et al. (1982) review does not comment specifically on PCB effects on the endocrine system. The USEPA Response (1982) suggests that PCB-altered sex steroid metabolism may play a role in adverse reproductive effects.

5.3.5 Comment on Animal Toxicology

Most laboratory studies have tested commercial PCB mixtures, although some studies of individual isomers have been undertaken. Recently, work with PCB isomers has shown that some chlorinated biphenyl compounds are highly toxic while other compounds are not very toxic. Such findings indicate that understanding and interpreting the effects of the complex mixtures that comprise commercial PCBs will be a difficult task. However, it is now clear that the toxic properties of PCB mixtures can not be attributed solely to the presence of highly toxic contaminants. As discussed later in this chapter, it may be only of academic importance to distinguish the effects of contaminants from "pure" PCBs. Commercial PCB mixtures contain these contaminants, and it is not feasible to remove them from commercial mixtures.

There are obvious species differences in the degree of response to PCBs. Overall, mink are perhaps the most sensitive mammalian species. Rhesus monkeys are more sensitive than mice, rats, or rabbits for a wide range of responses. While Drill et al. (1982) believe that there is no basis for choosing a particular species as an animal model to predict PCB effects on humans, other workers (Allen et al., 1979) have suggested using the rhesus monkey as a model. As illustrated in Table 5.3, man and monkey share many similar responses to PCB intoxication.

In view of recent reports by Marks et al. (1981) that some PCB isomers are teratogenic in mice, it is premature to dismiss the subject of teratogenicity. More research should be conducted on potential fetal malformations from PCB exposure.

Commercial PCB mixtures clearly have adverse reproductive effects. In the rat, a dietary level of 20 ppm of Aroclor 1254 affected the number of offspring (Linder et al., 1974). Mink show total reproductive failure at 5 ppm (Kimbrough et al., 1978), and Aroclor 1254 affects reproduction at 2 ppm (Auerlich and Ringer, 1977). The rhesus monkey shows adverse reproductive effects at levels of 2.5 ppm Aroclor 1248 (Allen and Barsotti, 1976).

The U.S. FDA action level for PCBs has been set at 5 ppm in fresh fish, a level greater than dietary doses observed to have adverse reproductive effects in laboratory mink and monkeys. Reproductive failure in commercially raised mink has been linked to PCBs present in fishmeal processed from spawning Great Lakes Coho salmon. The reproductive effects of PCBs have been observed from both low dose laboratory feeding studies and diets consisting of environmentally occurring concentrations. These observations stand in contrast to Dr. Seymour Freiss's statement to represen-

TABLE 5.5

SUMMARY OF ANIMAL BIOASSAYS FOR CARCINOGENICITY

SEX & STRAIN OF ANIMAL (INVESTIGATORS)	PCB SOURCE	DOSE	COMMENT
A. MICE			
1. Male dd strain (Ito et al., 1973)	Kanechlors 300,400,500[1/]	100, 250, 500 ppm	Positive for liver cancer; short study of 32 weeks, small sample size; liver cancer reported only at highest dose of Kanechlor 500
2. Male BALB/cj strain (Kimbrough and Linder, 1974)	Aroclor 1254	300 ppm	Negative for liver cancer; short study of 6 to 11 months; preneoplastic growths
B. RATS			
1. Male and female Donryu strain (Kimura and Baba, 1973)	Kanechlor 400	40 to 600 ppm	Negative for liver cancer; small size and short duration; neoplastic nodules
2. Male Wister strain (Ito et al., 1974)	Kanechlors 300,400,500	100, 500 1000 ppm	Negative for liver cancer; small sample size and short duration; neoplastic nodules in experiment animals, but not in controls
3. Male and female Charles River strain (Calandra, 1976)	Aroclors 1242, 1254 1260	1, 10, 100 ppm	Negative for liver cancer; dose dependent hepatomas and nodular hyperplasia
4. Male and female	Aroclors 1254, 1260	20, 100, 500, 1000	Negative for liver cancer; short duration with adenofibrosis observed

mice. Neither lesion occurred in any of the other groups nor in the 6 control animals.

This study is significant because of the occurrence of hepatocellular carcinoma in the mouse group receiving the highest dose of Kanechlor 500. Levinskas (1981a) has criticized this bioassay, noting that in an earlier publication (a brief communication) referencing this work (Nagosaki et al., 1972), the "well-differentiated hepatocellular carcinomas" were described as "hepatomas". Further, Levinskas (1981a) and Drill et al. (1982) note that no hyperplastic nodules or hepatocellular carcinomas were observed at lower doses of Kanechlor 500 or with Kanechlor 300 and 400.

The USEPA Response (1982) places a different emphasis on the Ito et al. study: the negative findings with Kanechlor 300 and 400 are unacceptable evidence for lack of carcinogenicity because of the short treatment time (32 weeks) and small number of animals per group (12 mice). Further, the induction of hepatocellular carcinoma by Kanechlor 500 in only 32 weeks is "an unusually clear indication of such potential".

2. Kimbrough and Linder, 1974: Aroclor 1254
Male BALB/cJ mice were fed Aroclor 1254 at 300 ppm for either 6 or 11 months. Of 22 surviving mice in the eleven month feeding group, nine animals had hepatic hepatomas and all 22 had adenofibrosis. Of 24 surviving animals fed Aroclor 1254 for 6 months, one had a hepatoma, and there was no adenofibrosis. Adenofibrosis and hepatoma were not observed in any of the 53 surviving control animals. No hepatocellular carcinomas were observed.

Drill et al.(1982) cite this study in their review, noting the occurrence of adenofibrosis and hepatomas in the livers of experimental animals and the lack of hepatocellular carcinomas. The USEPA Response (1982) only briefly cites this study. The IARC (1978) monograph states that "Kanechlor 500 and Aroclor 1254 are carcinogenic in mice..." Since the Kimbrough and Linder (1974) study is the only mouse study testing Aroclor 1254 cited by IARC, the appearance of hepatomas is apparently considered a carcinogenic response.

Rats

1. Kimura and Baba (1973): Kanechlor 400
Kanechlor 400 was fed to Donryu rats (10 males and 10 females) at dietary doses varying from 40 to 600 ppm in a 400 day experiment. Multiple adenomateous nodules were found in 6 of 10 female rats but neither in the experimental males nor the control animals (5 males and 5 females). Drill et al.(1982) noted that no hepatocellular carcinomas occurred in the Kimura and Baba study. The USEPA Response (1982) states that the experiment was not of sufficient duration because it was less than the rat's lifespan. The number of animals per experimental group (10) and control group (5) was small.

2. Ito et al. (1974): Kanechlors 300, 400, 500
Ito et al. (1974) studied the effects of Kanechlors 300, 400 and 500 on male Wistar rats at dietary levels of 100, 500, and 1,000 ppm for periods of up to 52 weeks. Cholangiofibrosis (adenofibrosis) was observed with all three Kanechlors but only at the highest dose. Nodular hyperplasia, which is synonymous with neoplastic nodules and has been classified as a primary neoplasm (Institute of Laboratory Animal Resources, [ILAR], 1980), occurred after the feeding of all three Kanechlors. No hepatocellular carcinomas were observed.

Drill et al. (1982) considers both adenofibrosis and neoplastic nodules to be benign lesions. The USEPA Response (1982) comments that, "The induction of hyperplastic nodules [neoplastic nodules], however, does suggest that such carcinogenic

potential exists." Both the Drill et al. and USEPA (1982) reviews note that this experiment was of insufficient duration to be conclusive.

3. Calandra (1976): Aroclors 1242, 1254, 1260

In 1969, the Monsanto Company sponsored a series of studies at Industrial Bio-Test Laboratories, including two-year chronic feeding studies of Aroclors 1242, 1254, and 1260 to dogs and rats (Levinskas, 1981b). These studies were summarized by Calandra at the National Conference on Polychlorinated Biphenyls in November 1975 (USEPA, 1976). Although the studies have not been reported in published journals, the rat bioassays have been referenced in reviews by Kimbrough et al. (1978), USEPA (1980), and Drill et al. (1982) as Monsanto Company literature. The study is discussed in some detail below because it has not been adequately described in the published literature.

Recently, in a Monsanto Company paper, Levinskas (1981b) presented the Calandra results in greater detail by analyzing the worksheets from the Industrial Biotest studies and by summarizing unpublished evaluations of liver sections by pathologists. This paper by Levinskas was included in the reference material used by Ecology and Environment, Inc. in their 1981 Summary of the "Health Effects of PCBs" for the Chemical Manufacturers Association.

One thousand Charles River rats were divided into ten treatment groups, including

4. Kimbrough, Linder and Gaines (1972): Aroclor 1254 and 1260

Kimbrough et al. (1972) observed adenofibrosis in both male and female rats fed either Aroclor 1254 or Aroclor 1260 for eight months. A higher incidence at a lower dietary level (100 and 500 ppm) occurred with Aroclor 1254 than with Aroclor 1260 (100, 500 and 1,000 ppm). In general, Aroclor 1254 had a more pronounced effect on the rat liver than did Aroclor 1260. In a later review, Kimbrough (1978) noted that adenofibrosis is frequently observed in concert with hepatocellular carcinomas in rats fed hepatocarcinogens. Kimbrough et al. (1973) has also reported that adenofibrosis occurred after six months of a 500 ppm dietary level of Aroclor 1254 in male Sherman rats and that the lesion was still present ten months later.

The duration of these studies was insufficient to test for carcinogenicity. The USEPA Response (1982) noted that the issue of whether an adenofibrosis can later develop into a cholangiocarcinoma is still being debated. The Drill et al. (1982) review considers adenofibrosis to be a benign lesion.

5. National Cancer Institute (1978): Aroclor 1254

Aroclor 1254 was fed to eight groups (four male and four female) of 24 rats each at dietary levels of 100, 50, 25 and 0 ppm for 105 weeks. The incidence of liver carcinoma and adenoma was dose dependent but not statistically significant. The incidence of hepatic nodular hyperplasia was dose dependent: in males, 0 percent at 0 ppm; 21 percent at 25 ppm; 33 percent at 50 ppm; and 50 percent at 100 ppm. In females, the dose-dependent response was 0 percent at 0 ppm; 25 percent at 25 ppm; 41 percent at 50 ppm; and 71 percent at 100 ppm. In addition to hepatic lesions, adenomas of the digestive tract were observed in two treated males and two treated females. One carcinoma was reported in a treated male. Dose-related increases of focal lesions in the glandular stomach of these animals was also noted (Morgan et al., 1981).

In discussing this bioassay, the NCI team concluded that, under the test protocol used, Aroclor 1254 was not carcinogenic in Fisher 344 rats; however, the high incidence of hepatocellular proliferative lesions was related to treatment. The carcinomas of the gastrointestinal tract "may be associated" with the Aroclor 1254 treatment.

Subsequently, Morgan et al. (1982) restained and reexamined stomach tissue from all

"gastric intestinal metaplasia and adenocarcinoma remains to be established, they commonly co-exist and may share initiating mechanisms." The results of the 1978 Aroclor 1254 NCI study are discussed in several sections of the review by Drill et al. (1982).
They note that there was no statistically significant increase in either hepatocellular carcinomas or gastrointestinal carcinomas and that there were increases in proliferative hepatic lesions.

The 1982 USEPA Response states that"...the NCI (1978) study, together with the recent work of Morgan et al. (1981), provides evidence for the carcinogenicity of Aroclor 1254 in Fisher 344 rats" (USEPA, 1982, p. 71). While not statistically significant, there was a dose-related increase in hepatocellular carcinoma and adenoma. The dose-related increase in hyperplastic nodules of the liver was

The recent study by Weltman and Norback was not referenced by Drill et al. (1982). The USEPA Response (1982) referenced this study and reported as follows: "While these results strongly suggest further evidence for the hepatocarcinogenicity of Aroclor 1260 in rats, few details are presented in the abstracts; further details are needed prior to EPA's acceptance of this work as an adequate evaluation of oncogenic potential." (USEPA, 1982, p. 75).

Dogs

Calandra (1976): Aroclors 1242, 1254 and 1260
This study, like the rat bioassay reported by Calandra, has not been published; however, a very brief summary was presented in the proceedings from the National Conference on Chlorinated Biphenyls (1976). This work has subsequently been referenced in several reviews (Kimbrough et al., 1978; Drill et al., 1982; and USEPA, 1982). Groups of 4 male and 4 female dogs were fed at levels of 0, 1, 10 and 100 ppm either of Aroclors 1242, 1254 or 1260 for 2 years and no "remarkable" histopathological changes were noted (Calandra, 1976). The Drill et al. review stated that neither hepatocellular carcinomas nor hepatic nodular hyperplasia occurred. The USEPA Response (1982) notes that, based on the dog's life span, seven years or more of exposure should be given for dog carcinogenicity bioassays; therefore, the duration of the Calandra dog study was far too short.

Interpretation of Animal Bioassays

There is disagreement as to how the above carcinogenicity bioassays should be interpreted. Issues raised by different reviewers (IARC, 1978; USEPA, 1980; Levinskas, 1981a; Drill et al., 1982; and USEPA, 1982) include (1) duration of a study in relation to the life span of the tested species; (2) study design; (3) the interpretation of tissue lesions by pathologists; (4) benign versus malignant tumors; and (5) the relative significance of positive and negative findings.

Because of the long latency period associated with the onset of most cancers in humans, long-term animal tests are required to maximize our ability to examine the carcinogenicity of a given substance. The significance of non-malignant tumors observed in animal cancer bioassays is controversial. For example, "preneoplastic" lesions may or may not become malignant. Lifetime animal studies might clarify the significance of preneoplastic growths, but lifetime studies have not been reported. Rather, animals are treated for a fixed period, sacrificed, and autopsied. Kimbrough (1979) considers neoplastic nodules to be part of the response spectrum elicited by hepatocarcinogens in rodents: (1) areas of liver alteration occur; followed by (2) development of neoplastic nodules, some of which (3) are transformed into hepatocellular carcinomas. In contrast, the review by Drill et al. (1982) considers areas of liver alteration and neoplastic nodules to be reversible and benign lesions, and these lesions are not discussed in their chapter on carcinogenesis.

The California Department of Health Services (DHS, 1982a), in a recent report, Carcinogen Identification Policy: A Statement of Science as a Basis of Policy, summarizes the recommended design of a cancer bioassay as suggested by the National Cancer Institute, USEPA, International Agency for Research on Cancer (IARC), U.S. Inter-Agency Regulatory Liaison Group (IRLG), and the Organization for Economic Cooperation and Development (OECD) as follows:

1. two species of test animals (usually rats and mice of both sexes) tested at two, preferably three, dose levels, with the high dose at approximately the estimated maximum tolerated dose;

2. dosing and observation for most of the animals' natural lifetime, usually 2 years for rodents;

3. adequate number of animals (at least 50 per sex) in each test group;

4. adequate number of concurrent controls;

5. detailed pathological examination of tissues; and

6. appropriate statistical evaluation of results. (DHS, 1982a, p. 25).

The DHS Document (1982a) then states several types of evidence that can lead to a conclusion of carcinogenicity from animal experiments, including:

1. statistically significant increases in tumors relative to controls at one or more of the dose levels tested;

2. an increase in the occurrence of rare tumors (those having a zero or low spontaneous incidence rate among historical controls); and/or

3. early appearance of cancer in the treated animals. (DHS, 1982a, p. 26).

The DHS document (1982a) distinguishes between benign tumors in human clinical medicine and in experimental animals. In human medicine, the term "cancer" is generally reserved for malignant tumors. In contrast, in animal studies, "experience has shown that at many sites benign tumors are early stages in a progression that leads to malignancy if the animals live long enough. Benign tumors that never progress to malignancy appear to be infrequent in test animals" (DHS, 1982a, p. 33).

Because of disagreement among pathologists over the classification of rat liver tumors, a workshop was convened under the auspices of the National Cancer Institute in 1974. The workshop concluded that neoplastic nodules (nodular hyperplasia) were new growths (tumors) rather than merely proliferation of tissue. Most workshop participants believed that areas or foci of cellular proliferation may be part of the progression to neoplastic nodules (Squire and Levitt, 1975). Neoplastic nodules "are proliferative lesions and are known to be induced by carcinogens and, at the least, they indicate an increased probability for the development of hepatocellular carcinoma. Although they may occur in control animals, the incidence is low, as is the incidence of naturally occurring hepatocellular carcinoma" (Squire and Levitt, 1975, p. 3215). However, there is still disagreement over whether neoplastic nodules will, in time, develop into hepatocellular carcinoma or if they just occur together with carcinomas.

After the NCI sponsored workshop, the National Academy of Sciences assembled a group of pathologists who produced the document "Histological Typing of Liver Tumors of the Rat" in the Journal of National Cancer Institute (Institute of Laboratory Animal Resources (ILAR), 1980). This publication classified neoplastic nodules under the category of primary neoplasms, a group also including trabecular carcinoma pattern, adenocarcinoma pattern, poorly differentiated carcinoma pattern, hepatoblastoma, sarcoma, and hemangiosarcoma. "The neoplastic nodule is a manifestation of the process of hepatocarcinogenesis, earlier states of which are represented by the foci and areas of cellular alterations described above. It is induced by a variety of hepatocarcinogens but not by noncarcinogenic agents" (ILAR, 1980, p. 185).

In discussing the sensitivity of animal cancer bioassays, DHS (1982a) notes that a test with 50 animals per sex per group is relatively insensitive - it cannot reliably predict an increase in cancer incidence of less than 15 percent. Yet such a test in two species currently requires three years and $500,000 to carryout. Further, negative results from tests of limited duration in animals are not good evidence for noncarcinogenicity (CDHS, 1982a).

The review by Levinskas (1981a) differs from the DHS position. "Nevertheless negative studies even if they are of relatively short duration are part of the overall evidence which has to be considered." (Levinskas, 1981a, p. 6). Levinskas considers benign lesions to be reversible.

The two Monsanto publications by Levinskas (1981a,b) served as the technical basis for the analysis of the rodent cancer bioassays performed by Ecology and Environment, Inc. (1981) for the Chemical Manufacturer's Association. Ecology and Environment, Inc. (1981) states in their Executive Summary (p. 1-2) that "... numerous studies involving both mice and rats have been reported, but only one study exists to date that suggests PCBs may cause an increase in hepatocellular carcinoma". Also, their statement that "PCBs produce only liver tumors" is contradicted by the Morgan et al. (1981) review of the 1978 NCI bioassay that reports significant levels of adenocarcinomas of the rat stomach.

The Drill et al. review (1982) is somewhat equivocal on the significance of benign liver lesions. "...(I)t may be considered that the hyperplastic foci and nodular hyperplasia (neoplastic nodules) occurring in the liver are benign lesions rather than carcinomas. However, such nodules may be part of a sequence of neoplastic changes that eventually progress on to hepatocellular carcinoma." (Drill et al., 1982, p. 69). Drill et al. (1982) do not include the occurrence of hyperplastic foci and neoplastic nodules in animal cancer bioassays in their discussion of experimental carcinogenicity studies. The conclusions drawn in the Drill et al. review (1982) on carcinogenicity are as follows: "(a) the evidence for carcinogenicity is negative for gastrointestinal carcinoma in rats and hepatocellular carcinoma in the dog; (b) some studies have reported an increase in hepatocellular carcinoma in mice and rats exposed to commercial PCBs chronically, whereas other studies have afforded negative results" (Drill et al., 1982, p. 6).

The USEPA Response (1982) states that two of the animal bioassays have been of aedequate design and duration to assess the carcinogenic potential of PCBs: the Kimbrough et al. (1975) study of Aroclor 1260 in female rats and the 1978 NCI study of Aroclor 1254 in rats in combination with the stomach tissue analyses by Morgan et al. (1981). According to the USEPA Response (1982), the Kimbrough et al. (1975) study "implicates Aroclor 1260 as a liver carcinogen" in Sherman female rats and the NCI (1978) study "provides evidence for the carcinogenicity of Aroclor 1254 in Fisher 344 rats". The 1973 Ito et al. study in mice for only 32 weeks of treatment also provides evidence for the carcinogenicity of Kanechlor 500. Further, several rodent studies conducted for shorter periods suggest carcinogenicity of several PCB mixtures due to induction of neoplastic nodules, hepatomas, and adenofibrosis. The USEPA Response (1982) notes a 1974 NCI workshop that resolved such nodules are "proliferative lesions and are known to be induced by carcinogens and, at the least, they indicate an increased probability for the development of hepatocellular carcinoma" (Squire and Levitt, 1975, p. 3215). The lack of hepatocellular carcinomas in these shorter term studies cannot be considered negative evidence due to inadequate duration of the bioassays (USEPA, 1982). Finally, the work of Weltman and Norback (1982), although only available in abstract form,

"strongly suggests further evidence for the hepatocarcinogenicity of Aroclor 1260 in rats,..." (USEPA, 1982).

5.4.2 Comment on Animal Carcinogenicity

Neither of the two studies on the mouse (Ito et al., 1973; Kimbrough and Linder, 1974) was of adequate duration to thoroughly test for carcinogenic potential. However, the occurrence of 5 hepatocellular carcinomas in 12 animals with the high dose of Kanechlor 500 represent evidence for carcinogenicity under the California Department of Health Services criteria (CDHS, 1982).

Of the seven bioassays performed in rats, four were of adequate duration: Kimbrough et al., 1975; Calandra (1976), as reported by Levinskas (1981b); NCI (1978) supple-

The Calandra (1976) two-year dog study of Aroclor 1242, 1254 and 1260 was clearly of inadequate duration to test for carcinogenicity. The minimal time for a study of dogs would be seven years (USEPA, 1982).

In summary, the available data suggest that polychlorinated biphenyl mixtures are carcinogens in male mice and in female (and possibly male) rats. "Unless there is a solid body of evidence for refuting positive results obtained from adequately designed and conducted studies, prudent public health policy requires that positive results be given greater weight than negative results" (DHS, 1982a, p. 35).

5.4.3. Mutagenicity

A number of reviewers (Kimbrough et al., 1978; USEPA, 1980; McConnell, 1980; Levinskas, 1981a; Letz, 1981; Drill et al., 1982; and USEPA, 1982) have examined the literature on the mutagenicity of polychlorinated biphenyls. There is general agreement among reviewers that research to date has not implicated PCBs as mutagens. However, PCBs belong to the group of highly chlorinated animal carcinogens, including chlordane, kepone, mirex, TCDD, chloroform and carbon tetrachloride, which are not positive in short-term tests for mutagenicity (Letz, 1981). In addition, because many PCBs are metabolized through arene oxide intermediates, there is the possibility that some PCB isomers possess mutagenic potential (USEPA, 1980)

Drill et al. (1982) note that in four classes of mutagenicity testing (bacterial test systems, cytogenic analysis in vivo, cytogenic analysis in vitro, and dominant lethality in the rodent), the results have been negative. One study reported by Wyndham et al. (1976) found Aroclor 1221 and 4-chlorobiphenyl to be mutagenic in the Ames bacterial assay, but the results have not been repeated in subsequent attempts to confirm the Wyndham et al. study (USEPA, 1980; Levinskas, 1981a; Letz, 1981; and Drill et al., 1982).

In studies with commercial PCBs (Aroclors 1242 and 1254), dominant lethal mutations have not been observed in rats (Green et al., 1975b); in vivo chromosomal aberrations have not been noted in rat bone marrow or spermatogonia (Green et al., 1975a); and chromosomal aberrations in human lymphocyte cultures subjected to Aroclor 1254 did not occur (Hoopingarner et al., 1972). Levinskas (1981a) concluded that PCBs are not mutagenic, although Levinskas cited a study that showed 2,2'5,5'-tetrachloro-biphenyl and its arene oxide and phenolic metabolites caused single strand DNA breaks (Stadnicki et al., 1979). The 3,4 epoxide metabolite of the tetrachlorobiphenyl was the most potent in induction of single strand breakage. Based on the Levinskas (1981a) review, Ecology and Environment, Inc. (1981) stated that, "Chlorinated biphenyls have not demonstrated any mutagenic potential when tested with the majority of commonly used and well-substantiated mutagenicity procedures" (Ecology and Environment, Inc., 1981, p. 1-4). Drill et al. (1982) concluded that, "there is no significant evidence that PCBs are mutagenic in test systems, and no reports of such activity in human populations" (Drill et al., 1982, p. 19).

Letz (1981) noted that "PCB mixtures have not been observed to have mutagenic activity nor to measurably affect chromosomes in repeated studies using a variety of in vitro or in vivo test systems" (Letz, 1981, p. 23).

In the USEPA Response (1982) to reviews by Drill et al. (1982) and Ecology and Environment, Inc. (1981), the Agency agreed that those PCBs which have been tested do not have chromosome breaking activity, but noted that not all isomers have been tested. The Agency believes the information on induction of point mutations is less certain and observes that the Ames bacterial mutagenesis test is frequently negative for chlorinated hydrocarbons. In summary, the USEPA Response (1982)

states, "The bulk of the data for PCBs indicate little or no mutagenic activity. However, the Agency is of the opinion that some of the positive results indicate a need to maintain an open mind and continue to develop and evaluate new data." (USEPA, 1982, p. 102).

5.5 Biochemical Effects

5.5.1 Enzyme Induction

The growth and maintenance of cells in a living organism require a system to utilize chemicals (food) as energy. The fundamental unit of this metabolic system is the enzyme-catalyzed reaction, which through biotransformation may either synthesize or breakdown chemicals. Enzymes are catalysts made of protein which greatly accelerate the rate of chemical reactions. There are a number of ways to regulate these reactions within cells: one important means is to control an enzyme's concentration by regulating its rate of synthesis. The blueprint for synthesis of a particular enzyme is contained at a site on the DNA (genetic material) of the cell. The rate at which a particular enzyme is synthesized can be controlled by repression or induction of that site. Enzyme induction is the process by which the rate of an enzyme's synthesis is stimulated.

Through enzyme induction, organisms react to many foreign compounds by increasing concentrations of enzymes capable of biotransforming these compounds. This makes the organisms more able to biotransform not only the substance initiating the induction, but other compounds as well. For example, the activity of one biotransformation group, the cytochrome P-450-containing enzyme system, is increased by exposure to over 300 different drugs, pesticides, and industrial chemicals (Neal, 1980).

The principal biochemical effect of polychlorinated biphenyls is the stimulation (induction) of certain enzyme systems. Although induction occurs in a number of tissues, the effects are greatest in the liver.

The PCBs are potent inducers of major oxidative biotransformation systems (Drill et al., 1982). For PCBs, the enzyme biotransformation system of greatest interest is the mixed function oxidase system (MFO), located in the microsomal fraction of the cell's cytoplasm. The MFO oxidizes target molecules using a family of iron-containing enzymes (hemeproteins), collectively referred to as cytochrome P-450. At least seven cytochrome P-450's have been identified in the rat liver; these isozymes differ in substrate specificity and inducibility (USEPA, 1982).

The cytochrome P-450 enzyme can be separated into two functional groups, P-448 and P-450 (Parkinson and Safe, 1981). For P-450, the model compound leading to enzyme induction is phenobarbitol; for P-448, the prototype is 3-methylcholanthrene. Induction of the P-448 cytochrome system leads to increased aryl hydrocarbon hydroxylase activity and induction of P-450 increases microsomal monooxygenese activity (Letz, 1981).

There is considerable evidence that the mutagenic and carcinogenic properties of benzpyrene and other polycyclic hydrocarbons results from transformation by cytochrome P-448 enzymes to metabolites that bind to DNA (Goldstein, 1980). Thus, P-448 inducing PCB isomers could magnify the carcinogenic potential of a compound such as benzpyrene by increasing its rate of metabolism (Goldstein, 1980).

The induction of mixed function oxidase can enhance the toxicity of compounds which are transformed to reactive metabolites. In particular, the phenobarbital

type cytochrome P-450 inducers have been shown to increase the hepatotoxicity of trichloroethylene and tetrachloroethylene (Goldstein, 1980). Thus, enzyme induction by either cytochrome P-448 or cytochrome P-450 inducers can potentially initiate secondary disease resulting from increased metabolism of polycyclic aromatics, halogenated hydrocarbons, and various administered drugs. And, unlike phenobarbitol, where the effect declines after administration, enzyme induction by PCBs is persistent long after exposure (Letz, 1981).

Reviewers disagree on the significance of cytochrome P-450 system induction and the type of P-450 induced. The Ecology and Environment, Inc. (1981) report to the Chemical Manufacturers Association observes that "PCB mixtures are also uniquely capable of inducing both cytochrome P-448 and P-450." Drill et al. (1982) state that (1) the main effect of PCBs is induction of cytochrome P-450, not P-448, (2) any P-448 induction may be due to the contaminant dibenzofurans which are present at about 2 ppm in commercial Aroclor mixtures, and (3) enzyme induction is a reversible process.

The USEPA Response (1982) "agrees with the view that cytochrome P-448 induction is relevant to carcinogenesis." However, the induction of enzymes by cytochrome P-450 inducers is also toxicologically important.

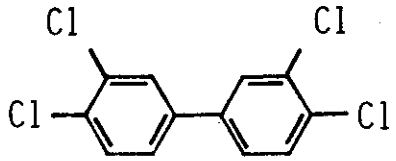
The commercial mixture Aroclor 1254 has a spectral effect on the cytochrome P-450 system intermediate between the effects of phenobarbitol and 3-methylcholanthrene. Aroclor 1254 induces two forms of cytochrome P-450b (phenobarbitol type) and one form of cytochrome P-450c (3-methylcholanthrene type) (Goldstein, 1980). Immunological studies have demonstrated that the collection of P-450 cytochromes are distinct hemeproteins. It has also been shown by spectral, electrophoretic, and immunological criteria that the mixture of hemeproteins induced by Aroclor 1254 is indistinguishable from the hemeproteins induced by a combination of phenobarbitol and 3-methylcholanthrene (Ryan et al., 1979). Studies of purified PCB isomers have shown that both the position and degree of chlorination are important (Goldstein, 1980). Some isomers induce P-448, some P-450, some both P-448 and P-450, and others are inactive. PCB isomers with two or more ortho chlorines are typically P-450 inducers, while those heavily chlorinated in the meta and para positions (such as 3,3',4,4',5,5'-hexachlorobiphenyl) are P-448 inducers. Some isomers, containing one ortho chlorine and the proper location of meta and para chlorines may induce both P-448 and P-450 (Albro and McKinney, 1981). Potency of induction is related to the pattern of chlorine atom substitution for a given polychlorinated biphenyl.

Recently, a correlation has been noted between the toxicity of individual halogenated aryl hydrocarbons (polychlorinated biphenyls, dibenzofurans, dibenzo-p-dioxins and polybrominated biphenyls) and their ability to induce aryl hydrocarbon hydroxylase, a cytochrome P-448-dependent monooxygenase (Yoshimura et al., 1979; Goldstein, 1979; Parkinson & Safe, 1981; Poland, Greenlee, and Kende, 1979). Aryl hydrocarbon hydroxylase is the enzyme that metabolizes the precarcinogen, benzpyrene (Goldstein, 1980).

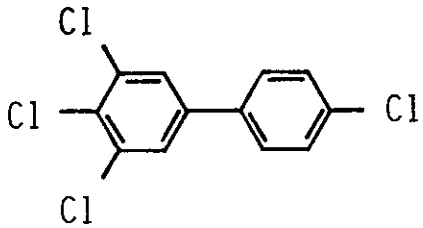
Only four of the possible 209 PCB isomers have been classified as strictly 3-methylcholanthrene inducers (P-448): 3,3',4,4'-tetra; 3,4,4',5-tetra; 3,3,'4,4',5-penta; and 3,3',4,4',5,5'-hexachlorobiphenyl. These isomers are chlorinated in lateral positions of the biphenyl nucleus [meta (3,3' and 5,5') and para (4,4')] and share structural similarities with the highly toxic tetrachloro dibenzo-p-dioxins and dibenzofurans (Figure 5.4). Two of these 4 PCB isomers (3,3',4,4'-tetra and 3,3',4,4',5,5'-hexa) have been tested and shown to bind to the aryl hydrocarbon

FIGURE 5.4

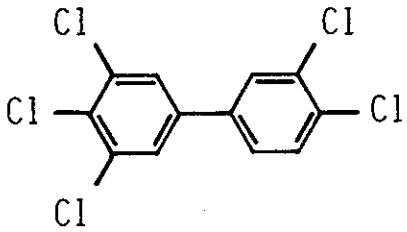
STRUCTURES OF FOUR METHYLCHOLANTHRENE-TYPE PCB ISOMERS AND TCDD AND TCDF
(Adapted from Parkinson and Safe, 1981)



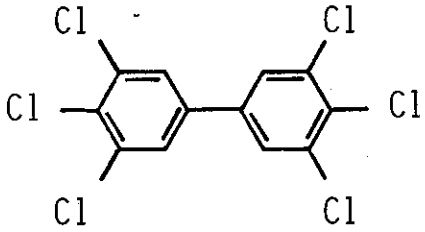
3,3',4,4'-TETRA-
CHLOROBIPHENYL



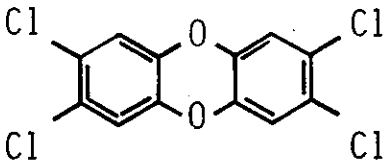
3,4,4',5-TETRA-
CHLOROBIPHENYL



3,3',4,4',5-PENTA-
CHLOROBIPHENYL

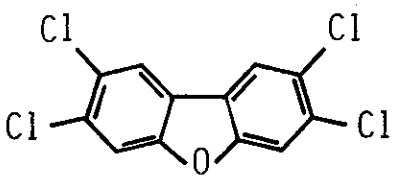


3,3',4,4',5,5'-HEXA-
CHLOROBIPHENYL



TCDD

2,3,7,8-TETRACHLORO-
DIBENZO-P-DIOXIN



TCDF

2,3,7,8-TETRACHLORO-
DIBENZOFURAN

hydroxylase receptor to which the most toxic chlorinated dibenzofurans and dibenzodioxins bind. Although it is generally accepted that the first step in the mechanism of halogenated aryl hydrocarbon toxicity is binding to the receptor site, there is disagreement over the causal relationship between increased AHH activity and various mammalian toxic responses to AHH-inducing PCBs.

Poland, Greenlee, and Kende (1979) have proposed a model to explain the mechanism of toxic action of these halogenated cytochrome P-448 inducers. In the model, toxicity and aryl hydrocarbon hydroxylase induction are initiated by reversible binding of the compound to a stereospecific recognition site, a protein in the cytoplasm of the hepatic cell. The chemical enters the cells and binds to the cytosolic protein at a stereospecific site. The halogenated compound and protein complex then migrates to the cell nucleus where, in an unknown manner, the complex initiates transcription and translation of gene(s) that code for cytochrome P-448. Evidence suggests that the gene locus is the structural gene for the cytosolic receptor protein. Poland et al. (1979) have identified two chlorobiphenyls (3,3',4,4'- tetra and 3,3',4,4',5,5'-hexa) that induce hepatic aryl hydrocarbon and compete for the cytosolic receptor site. The halogenated aromatic compounds that act as cytochrome P-448 inducers, have the following generalized structure-activity relationship:

1. the molecule fits roughly into a rectangle of dimensions 3$\overset{o}{A}$ by 10 $\overset{o}{A}$ (angstroms) with halogens in the four corners;
2. the molecule is planar or nearly planar; and
3. the molecule binds to the receptor protein.

Poland et al. (1979) have noted a correlation between the potency of a compound to induce cytochrome P-448 and its toxic potency that is linked to binding to the cytosolic site. Those P-448 inducers that have the highest binding affinity for the cytosolic site have the highest toxic potency. In summary, toxicity is mediated through the cytosolic protein and the initial event in toxic activity is the stereospecific recognition and binding of the halogenated compound by the cytosolic site. The model fails to explain why those polynuclear aromatic hydrocarbons that induce cytochrome P-448, such as benzpyrene and 3-methylcholanthrene, do not exert the same effects. Poland et al. (1979) postulate that because the halogenated aryl hydrocarbon hydroxylase inducers are persistent in the cell and resistant to metabolism, they remain bound to the cytosolic receptor much longer than the polyaromatic hydrocarbons which are more readily metabolized.

McKinney and Singh (1981) have proposed a similar mechanism that does not require planarity of the molecule. They note that the cytochrome P-448 inducers among polychlorinated and polybrominated biphenyls are the most toxic of these compounds. Even though they have the same carbon skeleton, inducers of P-448 and non-inducers behave like two separate classes of compounds. Based on various x-ray techniques, McKinney and Singh (1981) concluded that neither molecular planarity nor symmetry was required for binding to the cytosolic receptor. The basic requirements for structural specificity and binding were:

1. four lateral halogens arranged in a 3$\overset{o}{A}$ x 10 $\overset{o}{A}$ rectangle; and
2. a net (stereoelectronic) polarizability of the molecule. Coplanarity effects steric fit to the binding site and polarizability effects binding affinity.

Parkinson and Safe (1981) have suggested that some toxic responses result from receptor binding whereas other toxic responses to the class of P-448-inducing aryl hydrocarbons are not mediated through receptor binding. In particular, it has been

postulated that toxic reproductive effects may be a direct outcome of receptor binding because of disruption in the metabolism of certain lipophilic chemicals, such as steriod hormones (Parkinson and Safe, 1981).

5.5.2 Comment on Enzyme Induction

Recent research has elucidated the biochemical mechanisms accounting for some of the toxic effects exerted by cytochrome P-448-inducing chemicals. The signifi-

The effect of these shortcomings is that the epidemiological studies of PCB exposure have very low sensitivity. The California Department of Health Services (1982a) recently discussed the issue of low sensitivity of epidemiological studies in terms of assessing cancer risk:

> "When a cohort study population is relatively small (<1,000), it may be impossible to identify an agent that increases the risk of a specific cancer by a factor of less than 5 to 10. Even large-scale studies may fail to detect increase of less than 50% in cancer incidence. For this reason, negative results in cohort studies of this size seldom provide strong evidence that an agent is not carcinogenic. To obtain a positive result, the population studied must be very large; or a smaller-sized population must be exposed to a very potent carcinogen in large doses for several years." (DHS, 1982a, p. 23-24).

There are two principle types of epidemiological data on PCBs available: (1) results of worker exposure to PCBs and (2) followup of the Yusho and Taiwan incidents, where the toxic effects of the polychlorinated dibenzofuran (PCDF) contaminants may have exceeded the effects of the PCBs.

5.6.1. Occupational Exposure

Information on human occupational exposure has been compiled by Letz (1981) and is summarized in Appendix C (Table C.8). Dermal responses of workers exposed to PCBs have included chloracne, contact or allergic dermatitis, and brown skin discoloration. The occurrence of chloracne has been linked to exposure to a number of chlorinated hydrocarbons. Systemic symptoms of occupational exposure include nausea, digestive disturbances, headaches, upper respiratory problems, and persistent body odor (Letz, 1981).

Early investigations of PCB-induced liver damage have been reported, but these studies were flawed due to lack of control for exposure to other chemicals, inadequate medical histories, and the presence of compounding variables such as alcohol consumption. A 1979 study reported no significant difference in liver function tests (LFT) between capacitor workers exposed to low levels of PCBs and non-exposed workers (Fischbein et al., 1979). A 1981 study of electrical workers showed an apparent correlation between abnormal LFT's and amount of PCB exposure. These workers showed no other symptoms or laboratory abnormalities except for some cases of chloracne (Letz, 1981).

There is little occupational evidence to judge whether or not PCBs are a human carcinogen. A study of 2,567 capacitor workers recently completed by NIOSH showed no significant difference for any cause of death among exposed workers (Brown and Jones, 1981). However, there were some limitations in the sample design: (1) a relatively small sample, (2) an average of only 15 years follow-up for each worker (the latency period between exposure and death from cancer is probably longer), and (3) over 50 percent of the sample had 2 years or less exposure to PCBs (Letz, 1981). In a recent review, Kimbrough (1980) noted that, "No conclusive evidence has thus far been reported which demonstrates that occupational exposure to PCBs has caused an increased incidence of cancer." (Kimbrough, 1980, p. 385).

A recent epidemiological study of New York State capacitor workers showed a significant statistical difference between the weight of babies born to mothers with high PCB exposure compared to babies of mothers with low PCB work exposure. The high exposure group showed a reduction in gestational age of approximately one week and a reduction in birth weight of about 153 grams. This study by Taylor was reported at a conference sponsored by EPA, "Recent Advances in Exposure Health and Environmental Effects Studies of PCBs," held at Bethesda, Maryland on May 12 and

13, 1982. The Taylor study has not been published, and the biological implication of reduced gestational age are open to interpretation (R.D. Kimbrough, written summary of the conference: No. 4-Human Studies).

In assessing the occupational exposure data, the Ecology and Environment, Inc. (1981) report to the Chemical Manufacturers Association states that, "In summary, epidemiological studies have demonstrated that PCBs are not remarkably toxic chemicals after acute exposure, and that when excess exposure does occur, the usual consequences are dermatologic and not of a serious or permanent nature..... Therefore, it appears that PCBs are not a remarkable toxicant, but a chemical which requires high doses to produce harmful effects." (Ecology and Environment, Inc., 1981, p. 3). The Drill et al., (1982) report to the Edison Electric Institute observes that of the effects noted in animals, only dermatological effects have been seen in humans at the dose levels similar to occupational exposures. "Since the risk to human health from even high level occupational exposures has been shown by the studies available to be low, it may be concluded that much lower human exposure levels do not represent significant risks." (Drill et al., 1982, p. 13).

The 1982 USEPA Response discusses epidemiology in an appendix and does not reach a specific conclusion on data gathered from occupational exposure to PCBs. Much of the discussion is of a general nature and stresses the limits of epidemiological studies. "Thus epidemiological studies alone are rarely definitive and their significance is often open to much subjective interpretation." (USEPA, 1982, p. 177). The Agency notes that, while chloracne is a reversible chronic effect on humans exposed to PCBs, chloracne is also painful, disfiguring, and may require a long recovery before the symptoms disappear.

In contrast to Ecology and Environment, Inc. (1981) and Drill et al. (1982), Letz (1981) provided the following comment:

> "Although many problems have been identified in the studies evaluating the health effects of PCBs, it is clear that occupational exposure, at a minimum, can produce dermatologic effects. The long half-life of PCBs and their bioaccumulation in various human tissues leaves open the possibility of substantial chronic and delayed effects analogous to those seen in animal bioassays. These effects have only recently begun to be studies in a rigorous manner, and although the epidemiological evidence is neither complete nor entirely consistent there can be no question of the necessity to protect the worker from exposure." (Letz, 1981, p. 35).

Kimbrough (written communication, August 16, 1982) has observed that studies on the effects of PCBs in humans have been limited and have not adequately addressed possible consequences for reproduction and infant development. "Thus far, no studies have been conducted to determine whether exposure to PCBs affects human reproduction or survival of the neonate. No good information exists on possible toxic effects of PCB concentrations in human milk."

5.6.2 Yusho Incident

The 1968 Yusho incident in Japan was a mass outbreak of food poisoning, involving more than 1,600 people, caused by ingestion of rice oil contaminated with PCBs and other compounds (Kuratsune, 1980). Yusho disease differs from occupational exposure in that the contaminating fluid, Kanechlor 400, contained much higher levels of polychlorinated dibenzofurans (PCDFs) and polychlorinated quaterphenyls (PCQs) than normal commercial Kanechlor 400. These high levels of contaminants may have been formed while the PCB mixture was subjected to high temperatures in the heat exchanger. The contaminated rice oil was analyzed to contain PCBs at 1,000 ppm, PCQs at 1,000 ppm, and PCDFs at 5 ppm (Kuratsune, 1980). Clinical signs of Yusho disease have

TABLE 5.6

PERCENT DISTRIBUTION OF SYMPTOMS OF YUSHO REPORTED BY 189 PATIENTS EXAMINED BEFORE OCTOBER 31, 1968. (Letz, 1981)

SYMPTOMS	MALES (N=89)	FEMALES (N=100)
Dark brown pigmentation of nails	83.1	75.0
Distinctive hair follicles	64.0	56.0
Increased sweating at palms	50.6	55.0
Acnelike skin eruptions	87.6	82.0
Red plaques on limbs	20.2	16.0
Itching	42.7	52.0
Pigmentation of skin	75.3	72.0
Swelling of limbs	20.2	41.0
Stiffened soles in feet and palms of hands	24.7	29.0
Pigmented mucous membrane	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperemia of conjunctiva	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of upper eyelids	71.9	74.0
Feeling of weakness	58.4	52.0
Numbness in limbs	32.6	39.0
Fever	16.9	19.0
Hearing difficulties	18.0	19.0
Spasm of limbs	7.9	8.0
Headache	36.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

been formed while the PCB mixture was subjected to high temperatures in the heat exchanger. The contaminated rice oil was analyzed to contain PCBs at 1,000 ppm, PCQs at 1,000 ppm, and PCDFs at 5 ppm (Kuratsune, 1980). Clinical signs of Yusho disease have been reviewed by Kuratsune (1980) and Letz (1981). Table 5.6 lists occurrence of symptons in a sample of Yusho patients.

The problems of attempting to extrapolate Yusho exposure data to occupational exposure are as follows (Letz, 1981):

- Yusho exposure levels were higher than most occupational exposures. Yusho patients consumed an average dose of 2 grams and a minimum of 0.5 grams. Further, the route of entry was ingestion, whereas most occupational exposures have been through the skin or by inhalation.

- The exact chemical composition of the Kanechlor 400 present in the heat exchanger in 1968 is not known.

- Frying foods with the contaminated rice oil may have produced new compounds and may have altered the toxicity of the PCBs and other contaminants.

- Yusho oil contained high levels of PCDFs, whose toxicity is much greater than PCBs.

- The accuracy of PCB levels measured in foods in 1968 is in question since the methods for analyzing PCBs in foods were not well worked-out.

Regardless of the relative contribution of various halogenated hydrocarbons (PCBs, PCDFs, and PCQs), the Yusho incident allows documentation of the potential for human reproductive and transplacental effects resulting from exposure to these compounds. Several effects were noted in infants born to victims of Yusho disease. Eleven mothers with Yusho had 13 births, of which two were stillbirths. Ten infants experienced a transient grey or darkbrown pigmentation, five had pigmentation of the gums and/or nails, and nine experienced an increased ocular discharge. Twelve of the thirteen children were small compared to the Japanese national average. Effects were observed in infants born to severely exposed Yusho patients up to three years after the incident, although the responses were less severe.

Congenital abnormalities were also observed in Yusho infants: 2/13 had premature teeth eruption and 3/13 had abnormally wide sagittal sutures. It was also documented that Yusho could occur through ingestion of mother's milk: in infants born before the food poisoning incident, toxic responses were observed after 3 to 4 months of breast feeding (Letz, 1981).

In a 1977 cancer follow-up study of 1,665 Yusho victims (9 years after exposure), rate of death from cancer was 35 percent (11 of 31 cause-confirmed deaths), compared to 21 percent for the population of the Prefecture. However, there has probably not been sufficient time elapsed for onset of possible carcinogenesis from the Yusho exposure.

In the reference documents for the Ecology and Environment, Inc. report (1981) to the Chemical Manufacturers Associated, the Yusho illness is linked to high levels of polychlorinated dibenzofurans. Further, this report states that the PCB body burden of occupationally exposed capacitor workers have exceeded PCB levels ingested by Yusho victims, and these levels have not induced Yusho illness in capacitor workers.

In distinguishing Yusho disease from other PCB exposure, the Drill et al. (1982) report to the Edison Electric Institute noted several differences between the Yusho rice oil poisoning and occupational exposure to commercial grade PCBs:

1. Other halogenated compounds, including polychlorinated dibenzofurans (PCDFs)

2. The dosage patterns of Yusho patients and occupationally exposed workers are different: Yusho patients ingested large quantities of contaminated rice oil in a relatively short span of time whereas workers experienced low-level, long-term exposures primarily by inhalation and dermal absorption.

3. The Yusho disease pattern is very similar to the Taiwan incident; in each case, rice oil was contaminated by PCB fluids that had been subjected to high

effects in humans. And, as Kimbrough has observed, there is a lack of information about possible effects of maternal PCB exposure upon the developing fetus and infant. Thus, based on existing studies, it is premature to dismiss the potential health effects of PCBs on humans as not significant.

Occupational exposure to PCBs through absorption by skin and inhalation is not comparable to PCB exposure in the natural environment where these highly chlorinated, persistent isomers are those most resistant to metabolism and least subject to volatilization. That is, a person ingesting biomagnified PCB contaminants in fish possibly receives a more toxic PCB mixture than a capacitor worker inhaling the lower chlorinated PCBs on the production line. In cases where biomagnified biphenyls residues were fed to test animals, the potencies were always higher compared to the animals exposed to the commercial mixtures of PCBs (J.D. McKinney, 1982 - personal communication).

The unfortunate 1978 reoccurrence in Taiwan of Yusho poisoning through ingestion of PCB-contaminated rice oil demonstrates the need to remove these chemicals and associated PCB equipment from areas where food and feed are processed. The Yusho incidents suggest that if PCBs can be heated sufficiently to create higher concentrations of toxic contaminants, then human health effects can be severe. Because toxic contaminants such as polychlorinated dibenzofurans are present in commercial PCB mixtures and it is not feasible to remove them, they should be included in health effects assessments of these mixtures.

5.7 Contaminants in PCB Commercial Mixtures

As discussed in Chapter 2 and previously in this chapter, commercial PCB mixtures contain highly toxic contaminants—in particular the polychlorinated dibenzofurans

hexachlorobiphenyl (McConnell, 1980). Very little, if any, information is available on the reproductive, teratogenic, or mutagenic effects of PCDFs (McConnell, 1980). In general, information on PCDF toxicology is far sparser than material related to PCBs.

The Ecology and Environment, Inc. (1981) report to the Chemical Manufacturers Association limited their discussion of health effects to PCBs alone. However, since the review assessed primarily the effects of commercial grade PCBs, the effect of contaminants, in particular the polychlorinated dibenzofurans (PCDFs), has been factored into the assessments and "(t)he concern for low level exposures to PCDFs is still expected to be minimal." However, Ecology and Environment, Inc. note that "the PCDF content of PCBs may be increased under conditions of high temperatures, and the possibility of increased toxicity by the presence of this contaminant or others should always be considered when dealing with PCB waste mixtures of uncertain origin." (Ecology and Environment, Inc., 1981, p. 4-2).

The Drill et al. (1982) health effects review suggests that the clinical symptoms manifested in the Japanese Yusho incident are chiefly attributable to PCDFs. Because of the fact that at least the PCDFs in the Yusho oils are potentially more toxic than the PCBs on the basis of animal data, "it is not possible to extrapolate the Yusho experience to prediction of acute and sub-chronic effects of commercial PCB mixtures in the human...." (Drill et al., 1982, p. 2).

The 1982 USEPA Response does not consider debate on the presence of contaminants to be relevant. Contaminants are present in commercial mixtures; thus debate is only important if the contaminants can be removed, "which is not feasible." Further, "the premise is dubious, however, that PCBs are harmless and that their impurities should get all the blame for adverse effects." (USEPA, 1982, p. 176).

6.0 MONITORING FOR PCBS IN CALIFORNIA

6.1 Introduction

Several inland and coastal surveillance programs in California have confirmed the widespread contamination of PCBs in aquatic life and their persistence in [redacted] field residues are Aroclors 1254 and 1260; these

[redacted]

PCB findings in California's aquatic environment should be interpreted with

TABLE 6.1

HISTORICAL SUMMARY OF PCBs DETECTED IN FISH TISSUE (ppm Fresh Weight) BY TSM PROGRAM, 1978-1981 (Schafer, 1982)

Station	1978[1/]	1979[1/]	1980[2/]	1981[3/]
Klamath River			NA	
Trinity R.			NA	NA
Eel R.			NA	
Lake Pillsbury	NS	NS	NS	NA
Russian R.			NA	
Mad R.	NS	NS	NS	
Coyote Creek	NS	NS	NS	0.050 (1260)
Guadalupe R.	NS	NS	NS	
San Lorenzo			NA	
L. Nacamiento	NS	NS	NS	
Pajaro R.	NS	NS		
Salinas R.				NA
Shasta L.	NS	NS	NS	
Santa Clara R.	NS	NS	NS	
Sacramento R. (Keswick)	NS	NS		
Sacramento R. (Hamilton City)	NS	NS	NS	
Yuba R.			NA	
Feather R.	0.696		0.080[4/]	
Sutter Bypass	NS	NS	NS	
Reclamation Slough	NS	NS	0.35[4/]	
Colusa Drain (Abel Road)	NS	NS	0.35[4/]	
Colusa Drain (Knights Landing)	NS	NS	NS	
Clear L.	NS	NS	NA	
Cache Cr.				
No. F. American R.	NS	NS	NS	
So. F. American R.	NS	NS	NS	
American R.		0.136		
Sacramento R. (Hood)		0.100 (1260)	0.090 (1260)	0.058 (1260)
Mokelumne R.				
San Joaquin R.			0.050 (1260)	
Stanislaus R.		0.130		
Folsom L.	NS	NS	NS	
Tuolumne R.		0.160		
Merced R.				
O'Neill Forebay	NS	NS		0.130 (1248)
Don Pedro Reservoir	NS	NS	NS	
Susan R.	NS	NS	NS	
Truckee R.				
Colorado R.				
Salton Sea-North	NS	NS	NS	
Salton Sea-South	NS	NS		
Alamo R.		NS		
New R.			0.070	
Santa Ana R.	0.074		0.100	0.420[4/] (1248)

1/ Values reported as total of PCB 1240, 1254, 1268 unless otherwise noted.

2/ Values reported as total of PCB 1242, 1248, 1254, 1268 unless otherwise noted.

3/ Values reported as total of PCB 1242, 1245, 1254, 1260 unless otherwise noted.

4/ Highest value reported.

Legend: NA = Not Analyzed.
NS = Not Sampled.
All other stations Below Detection Limits; i.e., below 0.050 ppm.

In summary, between 1978 and 1981, PCB findings have been infrequent (PCBs were detected at 12 out of 44 stations) and at low levels (only one station exceeded the NAS guideline). Chronic low level contamination at the Sacramento and Santa Ana River stations has been identified during this period. The findings in Reclamation Slough and the Colusa Drain are notable because both of these waterways drain agricultural areas which, like urban areas, can also contribute PCB loadings to surface waters.

6.3 Cooperative Striped Bass Study (COSBS)

In 1979 the State Board, National Marine Fisheries Service (NMFS), and California Department of Fish and Game (DFG) formed the Cooperative Striped Bass Study (COSBS) to examine the effects of water pollution on the San Francisco Bay-Delta striped bass (Morone saxatilis) fishery. Field and laboratory studies of adult spawning bass have shown correlations between certain toxic pollutants and reduced reproductive capacity, fecundity, and egg viability (Whipple, 1983).

Analyses of striped bass tissues for PCBs and other compounds were conducted by the COSBS program during 1980 and 1981 (Table 6.2). Striped bass from the San Joaquin and Sacramento Rivers had consistently higher PCB levels in gonads, liver, and muscle tissue than other male and female striped bass taken from Coos River Oregon. This is especially significant considering the Oregon female bass averaged 2-3 times larger than the Delta bass.

The reported PCB wet weight concentrations in Delta bass fillets collected in 1980 were below the current FDA action level of 5 ppm. The mean muscle values for Sacramento river bass did exceed the NAS 0.5 ppm limit for the protection of predators. The lowest PCB muscle residues also exceeded the IJC limit of 0.1 ppm. The PCB levels in most prespawning Delta bass ovaries equalled levels representing approximately 25 percent mortality in Atlantic salmon eggs (Jensen, 1971).

6.4 National Pesticide Monitoring Program (NPMP)

The U.S. Fish and Wildlife Service (USFWS) Columbia (Missouri) National Fisheries Research Laboratory (CNFRL) monitors pollutant levels in freshwater fish and water fowl as part of the National Pesticide Monitoring Program (NPMP). CNFRL has

[redacted]

Joaquin River at Los Banos; Klamath River at Hornbrook; Colorado River at Imperial Reservoir; Colorado River at Lake Havasu; and Colorado River at Yuma, Arizona. Table 6.3 summarizes PCB residues data from fish collected at the Sacramento, San Joaquin, and Klamath River Stations from 1969 to 1981.

PCB levels have been consistently low, exceeding the FDA action level only once at

TABLE 6.2

SUMMARY OF PCB CONCENTRATIONS IN STRIPED BASS
ANALYZED BY COSBS (ppm Wet Weight)
(SWRCB, 1983)

River	Sex	GONADS				LIVER			MUSCLE		
		1980		1981		1980	1981		1980	1981	
		NMFS 1/	CDFG 2/	Aroclor 1260 3/	Aroclor 1254 3/	NMFS 1/	Aroclor 1260 3/	Aroclor 1254 3/	NMFS 1/	Aroclor 1260 3/	Aroclor 1254 3/
San Joaquin	♀	5.43	0.30	- -	- -	1.00	- -	- -	0.36	- -	- -
	♂	0.24	- -	- -	- -	4.01	- -	- -	0.63	- -	- -
Sacramento	♀	3.31	1.87	1.70	1.84	2.77	1.03	1.67	1.02	1.36	1.18
	♂	0.60	- -	- -	- -	2.45	- -	- -	.52	- -	- -
Coos (Oregon)	♀	1.46	0.15	0.35	0.83	0.41	0.08	0.25	0.22	0.05	0.42
	♂	0.06	- -	- -	- -	0.80	- -	- -	0.32	- -	- -

1. Mean concentrations of PCBs reported by NMFS in Whipple (1983), Tables 20 (liver), 21 (gonads), and 22 (muscle), pages 87-89.

2. Average concentrations of PCB 1260 in 1980 (gonads only) reported by CDFG in Whipple (1983), Table 19, page 86.

3. Mean residue levels for Aroclor 1260 and 1254 reported by Crosby et al (1983), Tables 1 (page 196) and 2 (page 197).

TABLE 6.3

POLYCHLORINATED BIPHENYLS (AROCLOR 1254) 1/
DETECTED IN FISH TISSUE BY NPMP, 1969-81 1/
(USFWS, 1983)

	YEAR												
STATION	1969	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981
SACRAMENTO RIVER (Sacramento)	0.1	4.0	3.0	8.2	1.4	2.5	NS 2/	NS	0.2	NS	0.1	NS	0.1
KLAMATH RIVER (Hornbrook)	0.3	0.3	0.2	NS	0.1	0.8	NS	NS	ND 3/	NS	0.1	NS	0.1
SAN JOAQUIN RIVER (Los Banos)	0.1	3.7	0.5	1.0	ND	NS	NS	NS	ND	NS	0.1	NS	ND

1/ Values reported as ppm - wet weight, whole fish. Highest annual value reported for each station is listed.

2/ NS = Not Sampled

3/ ND = Not Detected

Most of the PCB concentrations found at coastline stations have stabilized, fluctuating slightly from 1977 to the present (Table 6.4). PCB levels in mussels from the Royal Palms site (Palos Verdes Peninsula) decreased by one order of magnitude between 1971 (2500 ppb) and 1981 (150 ppb). The highest concentrations of PCBs in mussel tissues are reported for enclosed bays and estuaries. PCBs in mussel tissues from bay transplant stations in 1981 show a pattern of higher PCB inputs in Los Angeles - Long Beach and Newport harbors and San Diego Bay and lower inputs in San Francisco Bay.

The stations showing the highest PCB levels in resident Mytilus edulis during 1981 were Port Hueneme (1400 ppb), Marina del Rey (1000 ppb), and Anaheim Bay (840 ppb). The stations showing the highest PCB levels in transplanted Mytilus californianus during 1981 were San Diego Bay (800-7300 ppb), Los Angeles - Long Beach Harbor (2000 ppb) and Newport Harbor (5000 ppb)(Table 6.5). Intensive surveys are underway in these areas to identify specific sources of PCBs.

In summary, most open coastal areas exhibit lower PCB levels than enclosed bays and harbors. The Mussel Watch data indicate long-term persistence of residual levels of PCBs in the coastal environment. Intensive monitoring in southern California bays and harbors has identified these areas as PCB "hot spots." This suggests that a principal source of PCBs is surface runoff since the majority of the point source waste discharges in southern California are directly to the open ocean (SWRCB 1982b).

6.6 Southern California Coastal Water Research Project (SCCWRP)

The Southern California Coastal Water Research Project (SCCWRP), was founded in 1969, when five local government agencies (Ventura County, the cities of San Diego and Los Angeles, and the County Sanitation Districts of Los Angeles and Orange Counties) entered into a joint powers agreement to conduct environmental studies of the coastal waters from Point Conception to the Mexican border.

Table 6.6 summarizes the combined annual PCB emission rates from the five largest ocean dischargers (Oxnard, Ventura County; Hyperion, City of Los Angeles; Orange County Sanitation District; Joint Water Pollution Control Plant (JWPCP), Los Angeles County; Point Loma, City of San Diego) between 1971 and 1981. PCB discharges have stabilized since 1979 and remain at about 1200 kilograms per year. The discharge of PCBs is distributed fairly evenly among the dischargers with Orange County (35%), Hyperion (30%), JWPCP (26%) and Point Loma (6%) accounting for 97% of the mass discharge (Schafer 1982). Figure 6.1 shows the downward trend for PCB dischargers over the last 11 years.

Although PCBs do accumulate in pelagic seafood species off southern California, typical concentrations were 10 times less than FDA guidelines for human consumption (Schafer et al., 1982). Edible muscle tissue of sportfish residing in areas near sewage outfalls or in the Los Angeles harbor contained elevated levels (i.e. above background) of PCBs (0.01-0.16 mg/kg wet weight) but the levels did not exceed the World Health Organization 1971 recommended maximum safe daily intake of 5 ug/kg/day (Gosset et al., 1982).

TABLE 6.4

TABLE 6.5

CONCENTRATIONS OF PCB 1254 IN TISSUES OF TRANSPLANTED MYTILUS CALIFORNIANUS AT CALIFORNIA ISLAND, BAY, AND HARBOR STATIONS, 1979 - 1981 2/
(SWRCB, 1982b)

Location	1979	1980	1981 2/
Humboldt Bay - North Samoa Bridge	180	26	29
Humboldt Bay - South Samoa Bridge	81	34	--
Humboldt Bay - Eureka Slough	130	65	<10
Bodega Harbor	38	10	--
Tomales Bay	NA	27	--
Bolinas Lagoon	30	98	--
San Francisco Bay - Angel Island	770	830	--
San Francisco Bay - Treasure Island	630	1500	220
San Francisco Bay - Redwood City	NA	1200	200
San Francisco Bay - Dumbarton	NA	1300	250
Santa Cruz Harbor	110	150	--
Elkhorn Slough Bridge	65	100	--
Morro Bay - Virg's	NA	120	--
Channel Island Marina	NA	770	--
Port Hueneme	1300	2000	--
Anacapa Island	NA	36	--
Marina del Rey	NA	1800	--
Los Angeles - Long Beach Harbor	780	1800	2000
Newport Harbor	250	540	5000
Mission Bay	NA	190	--
San Diego Bay - Shelter Island	1800	1000	--
San Diego Bay - G Street	NA	1200	1000
San Diego Bay - Coronado Bridge	2800	2700	800
San Diego Bay - General Dynamics	--	--	7300

NA - Not analyzed
-- = No Data Available
1/ All values expressed as ng/g on a dry weight basis.
2/ Source: State Water Resources Control Board (SWRCB). California State Mussel Watch, 1981-82, Water Quality Monitoring Report, Sacramento, California (In Preparation).

TABLE 6.6

COMBINED ANNUAL PCB MASS EMISSION RATES OF SOUTHERN CALIFORNIA'S FIVE LARGEST MUNICIPAL WASTEWATER DISCHARGES, 1971-1981 (SCCWRP, 1982a)

Year	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981
FLOW, MGD	931	922	955	967	985	1027	966	1015	1054	1097	1097
TOTAL PCB, Kg/Yr 1/											
Discharger Values	8730	9830	4620	9390	6011	4310	2183	2510	1190	1129	1250
SCCWRP Values 2/	ND	19490	3390	5420	3070	2820	1560	590	1466	--	--

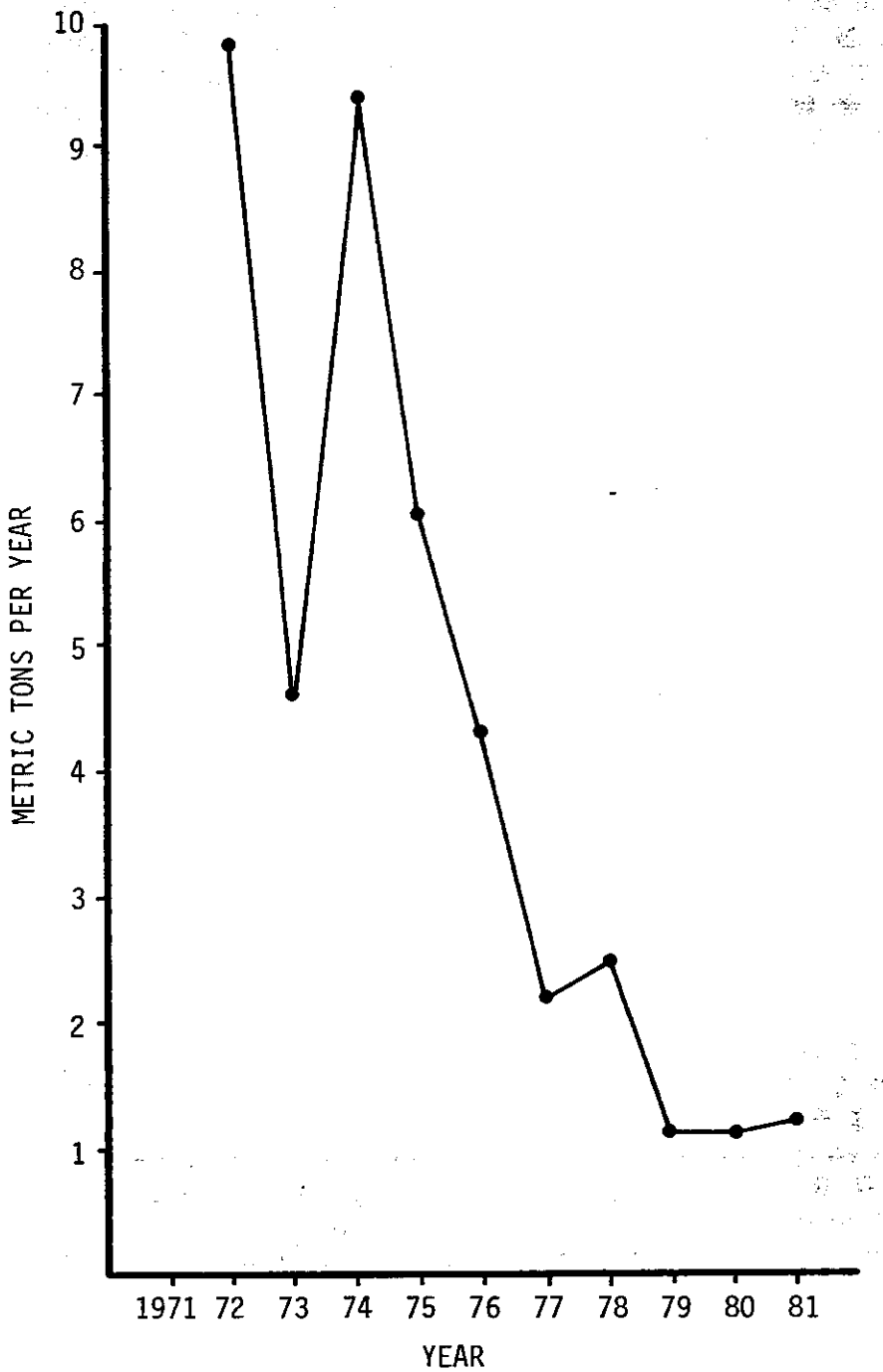
-- No data available

1/ Data based on analysis of monthly 24-hour composites.

2/ 1976-1979 data based on analysis of two 7-day composites.

FIGURE 6.1

COMBINED MASS EMISSIONS OF PCBs (1971-1981)
FROM SOUTHERN CALIFORNIA OCEAN DISCHARGERS
(Schafer, 1982)



7.0 PCB REGULATORY PROGRAM

7.1 Introduction

In California the PCB regulatory program primarily involves the U.S. Environmental Protection Agency, Department of Health Services, State Water Resources Control Board and Regional Water Quality Control Boards, and owners of electrical equipment

of 1976. In 1979 EPA issued a regulation which addressed PCB manufacture, use,

TABLE 7.1

MAJOR CHANGES IN PCB REGULATION

REGULATORY PROVISION	MAY 31, 1979 [1/] REGULATION	AUGUST 25, 1982 [2/] REGULATION	SWRCB RECOMMENDATION
• Definitions - disposal	did not specifically include spills	specifically includes spills	Agree
• PCB-contaminated - electrical equipment	regulation applied to transformers, capacitors and electromagnets	regulation applies to capacitors, transformers, electromagnets, voltage regulators, switches, circuit breakers, reclosers and cables	Agree
• Use of PCB-contaminated and PCB equipment			
- PCB transformers & PCB-filled electromagnets posing risk to food or feed	permitted as totally enclosed use	use prohibited after October 1, 1985; weekly inspection required until that date	Agree
- Other PCB transformers	"	use permitted for remainder of useful life; quarterly inspection required	Agree
- Large PCB Capacitors	"	use permitted for remainder of useful life if located in restricted access sub-stations or contained and restricted access indoor installations; no inspection required	Install failure warning devices on switched bank capacitors
		uses of other large PCB capacitors prohibited after October 1, 1988. No inspection required until that date	Install failure warning devices on switched bank capacitors until October 1, 1988
- Small PCB Capacitors	"	No restriction on use	Prohibit use in state facilities where risk to food, feed or water quality exists after October 1, 1985
- PCB-containing mineral oil filled electrical equipment	permitted as totally enclose use	Use permitted for remainder of useful life	Agree
- Electrical equipment posing exposure risk to food or feed	not covered	meaning clarified	Agree
- Oil-filled cable	not covered	Assumed to contain less than 50 ppm PCB if actual concentration unknown	Agree

(Table continued on next page)

TABLE 7.1 (continued)

MAJOR CHANGES IN PCB REGULATION

REGULATORY PROVISION	MAY 31, 1979 REGULATION [1/]	AUGUST 25, 1982 REGULATIONS [2/]	SWRCB RECOMMENDATION
• Storage and Record Keeping			
- Storage or disposal of non-leaking large high voltage PCB capacitors and PCB-contaminated electrical equipment	storage outside of qualified storage facilities after January 1, 1983 prohibited	storage outside of qualified storage facilities permitted	Agree
- Records of inspection and maintenance histories	required to be kept for 5 years	required to be kept for 3 years	Agree
• Spill Clean-up	did not specify level of cleanup required	does not specify level of cleanup required	50 ppm soil/sediment guideline that can be modified depending on site-specific circumstances

1/ Source: Federal Register, 1982a

2/ Source: Federal Register, 1982b

eliminated inspection requirements for capacitors. Failure detection devices should be installed on switched bank large capacitors. The technology for failure detection devices that are 90 percent effective exists, as demonstrated by Southern California Edison's experience with its capacitor unbalance sensing relay (CUSR). Installation of this type of equipment would reduce the risk of accidental releases

for implementing the PCB program. In 1981 EPA established five pilot cooperative enforcement programs in California, Ohio, Michigan, Connecticut, and Maryland to facilitate state PCB program implementation. The programs, which ran for 12-15 months, were designed to implement state priorities for PCB control and to familiarize states with the federal requirements. The health agency's priorities under the pilot program included spill reporting by major PCB users, inspection of on-line PCB equipment, and investigation of PCB spills. DHS plans to apply for authority to administer the current PCB program in California.

DHS has developed and implemented spill reporting and disposal monitoring procedures with all utilities and other major PCB users. These procedures require participating facilities to report any spill of PCBs, regardless of quantity, to DHS.

DHS has also investigated past and present disposal practices for PCBs. Although TSCA requires all facilities with PCB equipment to file and annual report with EPA, many non-utility facilities have not complied.

Finally, DHS has investigated all major spills and 10-15 percent of minor spills reported by users, EPA and other parties. DHS officials inspected sites, required cleanup, and made referrals for legal action, where necessary. The criteria for differentiating between major spills requiring DHS involvement and minor spills include:

1. Threat or potential threat to humans, animals or food crops;
2. Distance to usable ground or surface water;
3. Accessibility of site;
4. Volume and concentration of PCB material; and
5. Public concern.

While the pilot program ended in 1982, DHS has not received formal EPA authorization to carry out the federal program. Nonetheless, the major aspects of the pilot program have continued. Major PCB users report all spills to DHS. DHS also

In addition to DHS, many state agencies participate in emergency response to toxic spills, depending on the site of the spill and its effect on humans and the environment. The California Highway Patrol is the lead agency for coordinating public

prohibits the discharge of toxic substances in concentrations that impair beneficial uses. Appendix G contains criteria and standards developed by various federal and state agencies for PCBs.

The State and Regional Boards' traditional approach to setting water quality objectives assumes that the discharge of pollutants can be regulated. This traditional approach is impractical for PCBs. First, receiving water objectives for PCBs are inappropriate. PCBs have extremely low water solubility. When present in in the aquatic environment, they concentrate in sediment and fish tissue. Reliance on receiving water objectives could underestimate the threat that PCBs pose to beneficial uses. Second, protective receiving water levels often cannot be established for PCBs. Many PCB concentrations found in California fish tissue and sediment currently exceed levels necessary to protect human health and aquatic life. Third, most PCB discharges from point sources have ceased as a result of the EPA ban on manufacture and restrictions of use. Fourth, site-specific environ mental and economic factors determine the degree to which PCB sources can be controlled. While some PCB sources are relatively controllable, others may not be, because of the environmental damage that the clean-up operation would cause or the cost of pollutant removal and disposal.

Reportable Quantities for Spills

The Porter Cologne Act (Section 13271) requires the State Water Resources Control Board to set reportable quantities for hazardous substances that are spilled. Spills at or above the reportable quantity must be reported to the Office of Emergency Services for response action. The State Board has not yet set reportable quantity for PCBs (see Section 7.3.1 for DHS policy on reportable quantity).

Services regulations. California utilities have taken action to reduce the risk of PCB releases from electrical equipment, both on-line and in storage. For example, they have removed PCB-containing equipment posing a threat to water quality before mandated deadlines. They have developed operation and maintenance manuals covering the handling and disposal of PCB liquids and materials. They have also developed analytical methods for detecting PCBs in mineral oil fluids and early warning devices to detect capacitor failure. Furthermore, they have sponsored research into alternative technologies for PCB destruction to supplement the presently available disposal methods of landfilling (for specified PCB items) and incineration. The following discussion highlights aspects of the PCB programs carried out by three California utilities: Southern California Edison Company, Pacific Gas and Electric Company, and San Diego Gas and Electric.

Southern California Edison Company (SCE)

To prevent PCB spills from failed equipment, SCE developed the capacitor unbalance sensing relay (CUSR), a failure detection device. The utility installed this device on all switched bank capacitors in its distribution system by the end of 1981. The device has proven to be 90 percent effective in detecting imminent capacitor failures. SCE also developed an analytical procedure for measuring PCB concentrations in mineral oil and soils. Based on the concentrations detected, the company determines which equipment requires retrofilling, replacement, or other special handling (SCE, 1981). Furthermore, the company is evaluating alternative PCB treatment technologies including: thermal reactor destruction and microbial destruction of PCBs in contaminated soil, aerobic metabolism of less chlorinated biphenyls and anaerobic dehalogenation of higher chlorinated PCBs.

Pacific Gas and Electric (PG&E)

Pacific Gas and Electric has developed a PCB management plan for its Central Valley watershed facilities (PG&E 1980a, 1980b, 1981). The major plan elements include: (1) removal of PCB equipment; (2) cleaning of powerhouse drain sumps to remove any accumulated pollutants; (3) safe use of solvents to prevent contamination of waterways; and (4) implementation of spill prevention and cleanup plans. By mid-1981 the company had removed all PCB-filled transformers from Central Valley hydroelectric facilities. PG&E is replacing PCB-filled generator surge capacitors and expects to complete this changeout in 1983.

San Diego Gas and Electric (SDG&E)

San Diego Gas ane Electric initiated a program to voluntarily remove or relocate PCB capacitors from locations near waterways and to retire these units from service before the end of their useful lives (SDG&E, 1980). SD&E also sponsored the demonstration of the Sun Ohio PCBX detoxification process as part of the application to the Department of Health Services for a permit to use the process in California.

7.4.2 Management of PCB Equipment in California State Facilities

In 1980, the State Legislature directed the Office of the State Architect to assess and make recommendations regarding the management of PCBs in state facilities. This management program focuses on equipment containing PCB concentrations of 500 ppm or more. The first phase of the management program consisted of a survey of 94 state facilities. The survey assessed the adequacy of containment, replacement, storage and disposal of PCB equipment in these facilities. The survey also identified sites where PCBs posed an imminent contamination hazard because the equipment was leaking, or was located near mechanical equipment.

The Office of the State Architect issued a report on the survey which provided detailed information on state-owned facilities (OSA, 1981). It supplemented the information that EPA has collected through its inspections and reports submitted by owners of PCB equipment, and the inspections of PCB facilities that the Department of Health Services has conducted.

The report assessed four PCB management alternatives:

1. Replacement of all PCB equipment;
2. Replacement of PCB equipment in poor condition and retrofilling the equipment in good condition to achieve a PCB concentration of less than 50 ppm;
3. Replacement of leaking PCB equipment and retention of PCB equipment in good condition; and
4. Replacement of leaking PCB equipment that cannot be repaired and repair and retrofilling of equipment to achieve a PCB concentration of less than 500 ppm.

The report also recommended the establishment of one large and two small central PCB storage facilities to comply with federal requirements. The report evaluated nine temporary storage sites and recommended four of them for development. Finally, the report proposed a PCB management plan for state agencies. The management plan addresses on-site management, transportation, storage and disposal.

The State Legislature adopted the minimum program recommended (Alternative 3 above), and rejected the recommendation to construct central storage facilities. The legislature appropriated funds to: (1) remove 100 pieces of leaking equipment from use and store them on-site, (2) take spill prevention measures (e.g., build dikes and seal drains) for 900 units in place, and (3) build 41 facilities for temporary on-site storage of PCB equipment removed from use. Seven of the temporary storage facilities had been built by the end of 1982.

7.5 PCB Disposal Alternatives

PCB disposal involves the disposition of equipment removed from service, and the clean-up of spills and abandoned wastes. The California Office of Appropriate Technology (OAT) estimated that approximately 400 tons of liquid PCBs drained from on-line equipment presently await disposal (OAT, 1981). The Air Resources Board (ARB) estimated that up to 500 tons of PCB liquids will require disposal over each of the next 10 years (ARB, 1980). The major source of PCB solids from on-line equipment are capacitors and transformers, which contain various concentrations of PCBs. OAT estimated that about 450 tons per year of PCB solids and sludges are disposed of in California.

It is difficult to estimate the extent of contamination caused by PCB spills and abandoned wastes. The case studies presented in Appendix D include spills and improper disposal that contaminated large volumes of soil and other items. As

Several of the case studies included in Appendix D used on-site encapsulation for large volumes of PCB-contaminated soil. The soil would have been very costly to remove, transport and dispose. On-site encapsulation does not conform with federal requirements, which specify design criteria for landfills. Because on-site encapsulation may be the only feasible solution in some cases, this disposal alternative should be formally recognized.

PCB disposal alternatives to landfilling have been evaluated by numerous groups (Environment Canada, 1983; EPRI, 1979; OAT, 1981; ARB, 1980). One new technology is dechlorination. Several companies, including Goodyear Tire and Rubber Company, Sunohio, Inc. (Canton, Ohio), and Accurex Corporation (Mountain View, California), have developed chemical dechlorination processes which strip chlorine atoms from highly chlorinated compounds, such as PCBs, and produce a non-toxic residue. EPA Region 9 (San Francisco) has approved the Sunohio PCBX and Accurex processes for use in California. DHS has approved selected uses of these processes on a case by case basis. DHS is currently reviewing the Sunohio PCBX chemical dechlorination process for general use in the state. In addition, the ARB has identified seven remotely located cement kilns as possible test burn sites for PCB solids.

The lack of in-state disposal options for most PCBs is a serious problem. Disposal of PCB wastes outside of California involves greater risks of environmental contamination from accidents and higher costs for transportation. A comprehensive PCB management program requires a variety of disposal options available within the state.

7.6 Conclusion

The PCB regulatory program is complex, with overlapping responsibilities in the areas of waste disposal and spill and abandoned waste cleanup. In addition, the revised federal regulations have changed management of on-line PCB equipment.

SWRCB has developed recommendations that respond to the need for:

1) a water quality strategy that accounts for the unique properties and uses of PCBs,
2) improved management of PCBs in state-owned facilities, and
3) in-state PCB disposal options.

Specific recommendations to meet these needs are presented in the Recommendations section of this report.

APPENDIX A

TYPES OF PCB-CONTAINING
EQUIPMENT AND MATERIALS

A.1 Introduction

A broad category of nonflammable synthetic chlorinated hydrocarbon insulating liquids used in electrical capacitors, transformers, nuclear reactors and accessory equipment is designated by the generic term "Askarel". PCBs have been the major components of most askarels since 1932. Two general groups of askarels were used, capacitor grade and transformer grade. Aroclor 1242 was the major capacitor fluid in the United States prior to 1971; afterwards, Aroclor 1016 was used primarily. Transformer grade askarels have usually been mixtures of trichlorobenzenes and the more highly-chlorinated biphenyls (42 percent to 60 percent chlorine). Examples include the following: 100 percent Aroclor 1242; 70 percent Aroclor 1254 and 30 percent trichlorobenzene; 60 percent hexachlorobiphenyl and 40 percent trichlorobenzene; 45 percent hexachlorobiphenyl and 55 percent of a mixture of tri- and tetrachlorinated benzenes; and 70 percent pentachlorobiphenyl and 30 percent trichlorobenzene.

The Toxic Substances Control Act of 1976 specifically restricted the use of polychlorinated biphenyls except in a "totally enclosed" manner. However, the Act authorized the Administrator of EPA to exempt certain uses if they did not present an unreasonable risk of injury to health or the environment. Major existing uses of PCBs are in transformers and capacitors. An estimated 750 million pounds of PCBs are still in service, of which 162 million pounds are used by electrical utilities in transformers and capacitors.

A.2 Transformers

Transformers are frequently filled with a dielectric fluid which serves to increase the unit's resistance to arcing and acts as a heat transfer fluid to cool the transformer coils and windings. Although most transformers contain mineral oil, an estimated 2-3 percent of liquid-filled transformers use askarels, which contain 45-100 percent PCBs. Askarel transformers contain from 3 to 3,000 gallons of PCB liquids and are generally used in hazardous locations where fire is of concern. Of the estimated (1979) 140,000 PCB transformers currently in service, 90 percent are used in four major economic sectors: industrial, electrical utilities, commercial buildings, and railroads and subways (USEPA, 1981). A list of transformer manufacturers and PCB fluid trade names is given in Table A.1.

Some 51,000 PCB transformers are used in industries such as metals, chemicals, paper and lumber, mining, automotive, food, textile, and stone, clay and glass. These transformers are located inside buildings in hazardous areas, in on-site powerhouses, and near high voltage equipment that create high temperatures (kilns, metal processing equipment, etc.).

The electrical utilities use an estimated 38,000 to 40,000 PCB transformers at distribution substations and generating facilities. PCB transformers located at generating facilities generally are placed only at hazardous locations, for example, at nuclear facilities, near coal conveyers, and with electrostatic precipitators. The utilities estimate that their askarel transformers contain some 75 million pounds of PCBs (Miller, 1982). An additional 262,000 pounds of PCBs are present in mineral oil transformers that are PCB-contaminated in the 50-500 part-per-million range, including 1.9 million distribution transformers and 25,000 power transformers.

TABLE A.1

TRANSFORMERS MANUFACTURERS AND PCB FLUID TRADE NAMES
(USEPA, 1981)

TRANSFORMER MANUFACTURER	PCB FLUID TRADE NAME

About 34,000 PCB transformers are present in commercial buildings and are in service to meet fire code restrictions. Railroads and subways use 1,000 PCB transformers. Railroads use them on-board to reduce high voltage current from overhead lines in electric locomotives and in self-propelled cars serving the Washington to Boston Corridor. Subway transformers are usually located underground and distribute power to subway cars (USEPA, 1981).

A.3 Capacitors

A typical capacitor consists of conducting surfaces separated by a dielectric. Capacitors are used to accumulate and hold a charge of electricity. The breadbox-sized capacitors mounted on utility poles help maintain constant voltage in residences. The PCB regulations (40 CFR 761) refer to three groups of capacitors: small; large, high voltage; and large, low voltage. Small capacitors are defined as those containing less than three pounds of dielectric fluids. Large capacitors contain three or more pounds of dielectric fluid. High voltage capacitors operate at 2,000 volts A.C. Small capacitors that are incorporated in electrical equipment such as television sets, home air conditioners and light fixtures contain 2-340 ml of PCBs and have a service life of at least 10 years. In 1975 about 95 percent of the 100 million capacitors produced annually in the United States contained PCBs. Non-PCB capacitors manufactures since July 1, 1978, must be labeled "No PCBs" (USEPA, 1981). Generally, industrially important capacitors have used liquid-impregnated cellulose paper. Unlike transformers, which can be drained and refilled, capacitors are generally replaced rather than repaired. Table A.2 summarizes information on five types of capacitors.

In 1979, it was estimated that United State electrical utilities had about 1.3 million large capacitors in service. The large capacitors are usually located in distribution substations. Large capacitors are also used in mines, usually at the power substation that supplies electricity to the total facility, and are often installed in control boxes for large electrical motors. In steel and die casting plants, capacitors are used for power factor correction of motor control circuits and as part of the circuitry of electric induction furnaces. Table A.3 lists capacitor manufacturers and PCB dielectric fluid by trade name.

A.4 Hydraulic Systems

Hydraulic fluids are liquids used as force transmitters. PCBs were widely used by steel manufacturing and die casting plants in hydraulic systems handling hot metals in order to reduce fire hazards. Because hydraulic systems that contained PCBs can be retrofilled with non-PCB fluids, residual PCB contamination may remain. Monsanto manufactured at least 19 PCB hydraulic fluids under the general name of Pydrauls. Hydraulic systems normally leak several times their capacity each year since the fluid is pressurized to several thousand pounds per square inch and may leak at connections.

A.5 Other PCB Equipment

A.5.1 Heat Transfer Fluid

Heat transfer systems that contained PCBs were drained in the early 1970s. However, when analyzed 6 to 8 years later, many contained 2-3 percent PCB residuals. The federal regulation requires that these systems be tested and decontaminated until PCB levels are maintained at less than 50 ppm. Monsanto sold PCB heat transfer fluids under the trade name of Therminols.

TABLE A.2

AVERAGE TOTAL WEIGHT, PCB WEIGHT, AND
OPERATING LIFE OF SEVERAL CAPACITOR TYPES
(EPRI, 1979)

Capacitor Type	Total Weight (lb)	Weight of PCBs (lb)	Operating Life (yr)
Large High Voltage	120	25	20
Large Low Voltage	20	3.5	15
High Intensity Discharge Lighting	8	2	20
Fluorescent Lighting Ballast	3.5	0.1	15
Small Appliances	NA	1	NA

TABLE A.3

CAPACITOR MANUFACTURERS AND PCB FLUID TRADE NAMES
(USEPA, 1981)

CAPACITOR MANUFACTURER	PCB FLUID TRADE NAME
Aerovox	Hyvol
Axel Electronics	
Capacitor Specialists	
Cornell Dubilier	Dykanol
Electrical Utilities Corp.	Eucarel
Electromagnetic Filter Co.	
General Electric	Pyranol
Jard Corp.	Clorphen
McGraw Edison	Elemex
Monsanto (fluid only)	Aroclor, Capacitor 21, MCS 1489
P.R. Mallory & Co.	Aroclor B
R.F. Interonics	
Sangamo Electric Co.	Diaclor
Sprague Electric Co.	Clorinol
Tobe Deutschmann Labs	
Universal Manufacturing Corp.	Askarel*
Westinghouse	Inerteen
York Electronics	

* Generic name for non-flammable insulating liquids in transformers and capacitors.

A.5.2 Vacuum Pump Fluid

Monsanto sold vacuum pump PCB fluid under the trade name of Santovac until 1979. These vacuum pumps were used under conditions of high temperatures and with reactive gases such as chlorine. Fluids in these pumps are required to be decontaminated until PCB levels are less than 50 ppm.

A.5.3 Gas Turbine Fluid

Natural gas pipeline companies tested PCBs as a working fluid in compressors in the early 1970s. PCBs were found to be unsatisfactory because they tend to foam. However, even after draining, PCB residual levels in compressor fluids have been measured as high as 1,000 ppm.

A.5.4 Electric Motor Fluid

In the late 1960s and early 1970s, PCBs were used in some liquid-cooled electric motors manufactured by Reliance Electric for Joy Manufacturing Company. Joy Manufacturing has also supplied mining machine electric motors that contain PCBs as a coolant fluid for cutting head motors and traction motors. As of January 1, 1982, PCBs could no longer be added to these motors. The federal regulation also requires that, whenever these motors are serviced or repaired, they be rebuilt either as air-cooled or non-PCB liquid containing motors.

A.5.5 Electromagnets

Separator magnets containing PCBs have been used on coal conveyer belts. The regulation prohibits servicing or rebuilding of PCB electromagnets which require removal of the coil from the casing.

A.5.6 Investment Casting Wax

Prior to 1978, a number of investment casting waxes used PCBs as a filler component.

A.5.7 Carbonless Copy Paper (before 1973)

Between 1943 and 1972, the NCR Corporation used PCBs as a dye carrier in carbonless copy paper, and the copy paper contained about 3 percent PCBs by weight. When Monsanto discontinued the sale of PCBs for open uses, NCR switched to a substitute for PCBs. Without chemical analysis, the presence of PCBs in copy paper cannot be determined.

A.5.8 Resins

Monsanto marketed several resins prior to 1972 that contain polychlorinated terphenyls with PCBs as impurities.

A.5.9 Other Products

Other open system PCB products manufactured before 1973 included sealants (for windshields, silos, etc.), lubricants (bridge bearings, transmission fluid additives), paints, adhesives, textile coatings (ironing boards, etc.), and use as components in fire retardants.

APPENDIX B

HIGHLIGHTS OF THE PCB HEALTH EFFECTS STUDY

Source: Drill, Freiss, Hays, Loomis & Shaffer, Inc. 1982.

DFHL&S
DRILL, FRIESS, HAYS, LOOMIS & SHAFFER, INC. ~ Consultants in Toxicology ~ 1901 N. Fort Myer Drive
Suite 204
Arlington, Virginia 22209
Phone (703) 527-6450

Highlights of the PCB Health Effects Study
(Executed for NEMA/EEI by DFHL&S, Inc.)

Introduction

Mixtures of chemicals known as polychlorinated biphenyls (PCBs) were manufactured commercially in the United States until 1977 and they are used throughout the world in a variety of applications. Over the years, people have been exposed to these chemicals both in the workplace and in the natural environment. Exposures in the workplace were generally far more intensive, especially in terms of inhalation and skin contact, than the typical low-level or occasional exposure in the natural environment. Much information on possible health effects resulting from PCB exposures has accumulated in the scientific literature, starting back in the 1930s. This report reviews this information and uses it as the basis for an objective assessment of the potential health effects of PCBs on people.

Methodology of the Study

Two teams of specialists, representing the disciplines of medicine, toxicology, pharmacology, biochemistry and epidemiology, participated in the study. The first team reviewed the PCB health effects literature in detail through 1981, and prepared draft reports on the relationships between PCB exposure and specific health effects in test animals and in exposed people. The second team of scientists from universities independently reviewed the draft material. Conclusions and opinions in the final report reflect contributions from both teams.

The health effects information reviewed was of two major types: first, results from large numbers of studies in which laboratory test animals were exposed to graded dosages of PCBs for variable time periods; second, data on effects detected in occupationally exposed workers as deduced from epidemiological and clinical studies. In the major recent studies, which are continuing, several thousands of occupationally-exposed workers are being followed.

From the entire collection of data on people and test animals, conclusions and opinions as to the possibility of occurrence of specific potential health effects in people from exposures to PCBs have been drawn. The results fall into two categories of effects, as follows:

Health Effects Observed in People and Animals

Two health effects, neither of them serious, have been observed in workers occupationally exposed to PCBs and in experimental animals. They are:

1. . . . A skin disorder known as chloracne. The intensity of this effect is related to degree of exposure and to PCB chemical structure. Chloracne diminishes on termination of exposure, and is not a serious health problem.

2. . . . In the process of metabolizing PCBs, excess amounts of proteins (enzymes) known as mixed function oxidases (MFOs) are produced, chiefly in the liver. This condition, even following extended industrial exposures, is not of significance to health over the lifetime of a PCB-exposed person. At most, elevated MFO levels can cause changes in the required dosages of medicines or other therapeutic agents that are subject to ready oxidation in the body. Similar MFO elevations are caused by certain drugs, hydrocarbons, and other substances.

Health Effects Observed Only in Test Animals

Several health effects have been observed in test animals, but have not been observed in people, even after many years of occupational exposure. These are:

1. The possibility that exposure to PCBs can lead to cancer has been studied in test animals and in exposed people. The bulk of the studies on animals yielded negative results on cancer in all tissues in all species, with an exception of some unverified results on liver cancer in mice and rats. In studies of occupationally-exposed workers, the test results were also negative with an exception of a single report of excess skin cancer in one group of workers. This report was not confirmed in other studies. It can be concluded that there are no demonstrated connections between PCB exposure and cancer in people, even when occupationally exposed.

2. Adverse effects on reproduction have been observed in experimental animals, generally at very high dosages. These are:

    a. Effects of PCBs on the developing embryo or fetus leading to birth defects (teratological effects) have been observed in a limited number of experimental animals.

    b. Toxic effects on the developing fetus in test animals can be produced by high dosages of PCB.

No evidence for such effects in people has been recorded, even for pregnant women occupationally exposed to PCBs. It is concluded that the probability of such effects occurring in people is very low.

3. PCBs are able to produce adverse effects on liver tissues in test animals, reversible at low doses and increasingly irreversible at higher exposure levels. No adverse effects on liver functions have been observed in occupationally-exposed workers, leading to the conclusion that these chemicals do not cause human liver disease under these conditions.

4. PCBs exhibit a low to moderate degree of acute toxicity in test animals. However, there is little evidence in people of any acute toxicity in the course of PCB intake, storage in tissues, metabolism and excretion. Further, there is no evidence to indicate the PCBs produce any adverse effects on the human immune system or blood supply.

Other Observations and Conclusions

Mutagenesis: The possibilities of harmful, heritable changes in genetic material of cells and tissues under the action of PCBs have been studied in biological test systems, experimental animals, and occupationally-exposed workers. All test results are negative for such changes. The risk of genetic damage in people from PCB exposure is negligible. This conclusion is consistent with the lack of a demonstrated connection between PCB exposure and cancer.

Yusho Disease: Because of the uniqueness of the Yusho episodes in terms of the level of contamination of the PCBs, the dosage patterns and the symptoms observed, Yusho cannot be considered a model to reflect the possible effects of PCB exposure in man. It is impossible to stipulate whether any of the health effects observed are causally related to PCB exposure.

Summary

At human exposure levels as high as those previously encountered in occupational settings, PCBs can cause chloracne and increases in the levels of one or more liver enzymes. Neither effect represents a serious or life-threatening health problem. From present evidence, no firm associations can be made between PCB exposure and any other human health effects, even after long-term occupational exposures.

Since the risk to human health from even high level occupational [illegible] has been shown by the studies available to be low, it may be

APPENDIX C

SUPPLEMENTARY TABLES TO CHAPTER 5

(MAMMALIAN TOXICOLOGY AND HUMAN HEALTH)

TABLE C.1

EVIDENCE FOR CARCINOGENICITY IN MICE: KIMBROUGH AND LINDER (1974) 11 MONTH STUDY OF MALE BALB/cJ MICE TREATED WITH 300 PPM AROCLOR 1254 (Kimbrough and Linder, 1974)

NUMBER TREATED	NUMBER SURVIVING	EXPOSURE TIME	ADENOFIBROSIS	HEPATOMA
50	22	11 months	22	9
50	24	6 months[1/]	0	1
100	58	-	0	0

[1/] Mice were fed 300 ppm Aroclor 1254 for 6 months, followed by 5 months of control diet (0 ppm).

TABLE C.2

EVIDENCE FOR CARCINOGENICITY IN MICE: Ito et al. (1973) 32 WEEK STUDY OF MALE dd MICE TREATED WITH KANECHLORS 300, 400, and 500. (Ito et al., 1973)

KANECHLOR	SURVIVING ANIMALS[1/]	PPM IN DIET	NODULAR HYPERPLASIA	HEPATOCELLULAR CARCINOMA
500	12	500	7	5
	12	250	0	0
	12	100	0	0
400	12	500	0	0
	12	250	0	0
	12	100	0	0
300	12	500	0	0
	12	250	0	0
	12	100	0	0
Controls	6	-	0	0

[1/] Animals dying before completion of 32 week experimant not included in results

TABLE C.3

EVIDENCE FOR CARCINOGENIC EFFECTS OF PCBs IN RATS: KIMURA AND BABA (1973) 400 DAY STUDY AND ITO ET AL. (1974) 378 DAY STUDY
(Adapted from USEPA, 1980)

STUDY	SEX/NUMBER TREATED OR SURVIVING	PCB	DIETARY LEVEL	Liver Nodules		
				ADENOFIBROSIS	NEOPLASTIC NODULES	HEPATOCELLULAR CARCINOMA
Kimura and Baba, 1973; 400 days	M/10	Kanechlor 400	40-600 ppm	-	0	-
	F/10	400	40-600 ppm	-	6	-
	M/5	None	-	-	-	-
	F/5	None	-	-	-	-
Ito et al., 1974; 378 days	M/13	Kanechlor 500	1,000	4	5	-
	M/16	"	500	0	5	-
	M/25	"	100	0	3	-
	M/10	Kanechlor 400	1,000	2	3	-
	M/8	"	500	0	0	-
	M/16	"	100	0	2	-

TABLE C.3 (cont'd)

EVIDENCE FOR CARCINOGENIC EFFECTS OF PCBs IN RATS: KIMURA AND BABA (1973) 400 DAY STUDY AND ITO ET AL. (1974) 378 DAY STUDY
(Adapted from USEPA, 1980)

STUDY	SEX/NUMBER TREATED OR SURVIVING	PCB	DIETARY LEVEL	Liver Nodules		
				ADENOFIBROSIS	NEOPLASTIC NODULES	HEPATOCELLULAR CARCINOMA
	M/15	Kanechlor 300	1,000	2	0	-
	M/19	"	500	0	0	-
	M/22	"	100	0	1	-
	M/18	None	-	0	0	-

TABLE C.4
EVIDENCE FOR CARCINOGENIC EFFECTS IN RATS: THE CALANDRA 24 MONTH STUDY OF CHARLES RIVER RATS[1/]
(Adapted from Levinskas, 1981)

PCB SOURCE	DIETARY LEVEL (ppm)	NUMBER SURVIVING	Liver Nodules		
			NODULAR HYPERPLASIA	HEPATOMAS	HEPATO-CELLULAR CARCINOMA
Aroclor	100	19	8	3	0
1242	10	29	1	0	0
	1	23	1	0	0
Aroclor	100	26	14	4	0
1254	10	26	3	0	0
	1	30	0	0	0
Aroclor	100	25	6	7	0
1260	10	25	7	1	0
	1	26	0	0	0
Control	0	23	1	0	0

[1/] Twenty-four Month Data: Each initial treatment group consisted of 100 rats, 50 males and 50 females. At 3,6, and 12 months after onset of experiment, 10 rats (5 male and 5 female) per group were sacrificed. Thus, if all rats not deliberately sacrificed had survived, each treatment group would have had 70 rats at termination of the study.

TABLE C.5

EVIDENCE FOR CARCINOGENIC EFFECTS OF PCBs IN RATS: 1978
NATIONAL CANCER INSTITUTE STUDY OF AROCLOR 1254
(Adapted from USEPA, 1980)

STRAIN AND SEX	SURVIVING RATS	DIETARY LEVEL (ppm)	EXPOSURE TIME	Observed Changes		
				NODULAR HYPERPLASIA	HEPATOCELLULAR CARCINOMA AND ADENOMA	COMBINED HEMATOPOIETIC AND LIVER
Fisher	24	0	-	0	0	5
344, Male	24	25	105 weeks	5	0	2
	24	50	"	8	1	9
	24	100	"	12	3	12
Fisher 344,	23	0	-	0	0	4
Female	24	25	105 weeks	6	1	13
	22	50	"	9	1	8
	24	100	"	17	2	9

TABLE C.6

EVIDENCE FOR CARCINOGENIC EFFECTS OF PCBs IN RATS: 1975 KIMBROUGH ET AL. 21 MONTH FEEDING STUDY OF AROCLOR 1260 IN FEMALE SHERMAN RATS[1/]
(Adapted from USEPA, 1980)

PCB SOURCE	DIETARY LEVEL (ppm)	NUMBER TREATED	NUMBER SURVIVING	CYTOPLASMIC ALTERATION[2/]	NEOPLASTIC NODULES	HEPATOCELLULAR CARCINOMA
Aroclor 1260	100	200	184	182	144	26
None	-	200	174	28	0	1

1/ 630 Day Study of Female Sherman Rats

2/ Foci or Areas of Cytoplasmic Alteration (Kimbrough et al., 1975)

TABLE C.7

EVIDENCE FOR CARCINOGENIC EFFECTS IN RATS: WELTMAN AND NORBACK (1982)
24 MONTH FEEDING STUDY OF AROCLOR 1260 IN SPRAGUE - DAWLEY RATS[1/]

PCB SOURCE	NUMBER AND SEX OF RAT	DIETARY LEVEL	Incidence of Liver Tumors (Percent)		
			NEOPLASTIC NODULES	TRABECULAR HEPATOCELLULAR CARCINOMA	HEPATO-CHOLANGIOCELLULAR CARCINOMA
Aroclor 1260	47 F	100 ppm (16 months) + 50 ppm (8 months)	4	51	41
Aroclor 1260	46 M	"	11	4	-
Control	49 F, 32 M	-	-	1[2/]	-

1/ Data from abstract in Federation Proceedings (Weltman and Norback, 1982): Aroclor 1260 fed at 100 ppm for 16 months, followed by 50 ppm for 8 months, plus 5 additional months of control diet. Rats were examined from months 18 to 29.

2/ Identified only as one spontaneous hepatocellular lesion.

TABLE C.8

OCCUPATIONAL EXPOSURE TO PCBS
(Letz, 1981)

Study	Exposure Level & Time	Study Populations Exposed	Controls	Findings: Dermal Effects	Liver Function	Blood Concentrations	Cancer/ Mortality	Comments
Meigs, 1954: Outbreak of dermatitis in a chemical plant	0.1 mg/m^3 Aroclor - 5 to 19 mos. intermittent exposure through vapor leakage	14 potentially exposed	0	7 mild to moderate chlor-, acne, contact dermatitis	6 normal, 1 borderline abnormal			There was not a good correlation between apparent degree of exposure and development of signs of disease
Hesegawa et al. 1972: Study of 6 industrial plants including PCB manufacturing, capacitor manufacture, and biphenyl recovery	Vapors: 13-965 ug/m^3 Particulates: 4-650 (6,270 in a spill) <1 to 20 yrs.	99	32	Various	Slightly abnormal	Exposed: 370 ppm Nonexposed: 20 ppm		Dermal ailments were unrelated to blood concentrations; based on 3 plants, there was no relationship of exposure to blood concentrations, fat metabolism was apparently affected.

TABLE C.8 (cont'd)

OCCUPATIONAL EXPOSURE TO PCBS
(Letz, 1981)

Study	Exposure Level & Time	Study Populations Exposed	Controls	Findings: Dermal Effects	Liver Function	Blood Concentrations	Cancer/ Mortality	Comments
Hara et al., 1973-1974: Study concentrat on 17 immersion process workers	Level of exposure not reported in NIOSH Criteria Document	118		45% blackheads, 37% acne, 13% irritation	not reported	Exposed: 7-300 ppb		Effects correlated with duration of exposure; one year later blood concentrations were decreased by varying amounts. The longer the duration of exposure, the longer the PCB half-life in blood
Ouw et al., 1976: Study of capacitor manufacturer workers.	Aroclor 1242: 1.1 - 1.4 mg/m^3 (19 workers); 0.32 mg/m^3 (15 workers) Workers wore no pro-	34	30	Mild burning, irritation of face, eyes and skin; 5 had rashes	BSP elevated in 4 of 7 with blood levels> 500 ppb	Exposed: 100 - 602 ppb (mean 400 ppb). Not detected in non-exposed.		Systemic effects reported such as nausea and persistent body odor. There was no adverse response at blood concentrations below 200 ppb.

TABLE C.8 (cont'd)

OCCUPATIONAL EXPOSURE TO PCBS
(Letz, 1981)

Study	Exposure Level & Time	Study Populations Exposed	Study Populations Controls	Findings: Dermal Effects	Findings: Liver Function	Findings: Blood Concentrations	Findings: Cancer/ Mortality	Comments
	tective clothing Time 1 mo. to 23 yrs.			1 chloracne, several dermatitis				
Bahn, et al., 1976: Study of workers in a refinery	Aroclor 1254 over a 9 yr. period	31 males and 41 females					2 malignant melanomas observed, 0.04 expected (based on TNCS data)	These were preliminary results reported in a letter to the editor. Workers were also exposed to other chemicals. Study is in progress.

Findings		
Blood Concentrations	Cancer/ Mortality	Comments
All cause mortality was lower than expected (163 obs. vs. 174 exp.) All cancer mortality was lower than expected (39 obs. vs. 40.6 exp.) Rectal and liver cancer were slightly elevated but not significantly.		Lower observed mortality attributable to the "health worker" effect NIOSH will continue to followup mortality experience.
Average: 820 ppb (range: 320 - 2100 ppb)		No relationship was found between concentration in blood and duration of exposure

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TABLE C.8 (cont'd)

OCCUPATIONAL EXPOSURE TO PCBS
(Letz, 1981)

Study	Exposure Level & Time	Study Populations Exposed	Study Populations Controls	Findings: Dermal Effects	Findings: Liver Function	Findings: Blood Concentrations	Findings: Cancer/ Mortality	Comments
Karppanen and Kolho 1972: Study of 3 groups 1) no exposure 2) analytical lab workers 3) capacitor impregnation workers (Aroclor 1242)	Low, medium and high exposure <1 mg/m^3 (with skin protection)	High exposure: 8 males 4 females Medium exposure: 6 females	Low Exposure: 4 males 5 females			Unexposed: 5.6 - 12 ppb Medium: 36-63 ppb High exposure: 74-1,900 ppb		No biologic effect observed.
Bumgarner et al., 1973; Study of refuse workers exposed to PCB in incineration of waste	Incinerated waste	37	36 lumber yard workers			Measurable concentrations in 32 of the 37 refuse workers (4-14 ppb)		Concentrations not well correlated with duration of exposure

TABLE C.8 (cont'd)

OCCUPATIONAL EXPOSURE TO PCBS
(Letz, 1981)

Study	Exposure Level & Time	Study Populations Exposed	Study Populations Controls	Findings: Dermal Effects	Findings: Liver Function	Findings: Blood Concentrations	Findings: Cancer/ Mortality	Comments
Baker, Landrigan et. al., 1980 Study of exposure to PCBs in sewage sludge.	Liquid sewage entering plant 30-470 ppb. upstream sewage 1250-5500 ppb (Aroclor 1016). Concentrations in sludges (Aroclor 1242) were as high as 1,700 ppm (mean 479.1 ppb) and 107.3 ppm in treated soil (mean 17.1 ppm).	89 sludge users, 18 workers exposed to PCBs 19 members of workers' families	22 community members	Acne, increased pigmentation in 4 workers		Sludge users 17.4 ppb; workers 75.1 ppb; families 33.6 ppb; community 24.2 ppb		Plasma triglyceride levels increased significantly with serum PCB concentrations.

.8 (cont'd)

XPOSURE TO PCBS
1981)

Findings				
mal ects	Liver Function	Blood Concen- trations	Cancer/ Mortality	Comments
arent rease pre- ence vari- com- ints lud- 35 h tory acne ce on- of loy- t and with acne- m rash on mination.	Statisti- cal sig- nificance associ- ated with SGOT with serum levels of PCB but low incidence of "abnormal" values; no association with other liver enzymes.			Results se- verely limited by fact that there was no attempt to correlate exposure or blood levels with endpoints being measured -so that no possibility of observing "dose-response" effect or even exposed vs. control differ- ences.

APPENDIX C

PCB CASE STUDIES

APPENDIX D
PCB CASE STUDIES

D.1 Introduction

Six case studies have been selected to illustrate: (1) the variety of PCB spill and contamination problems, (2) the diversity of approaches used to address them and (3) gaps in current regulatory programs that require correction.

Types of PCB problems include improper disposal, dumping, and discharge from uncontrolled sources. Approaches to dealing with these problems range from source control to treatment of contaminated soil and liquids. Gaps in regulatory programs include interagency coordination and the priority assigned to spill prevention. One New York case study, the Hudson River contamination, was chosen to provide a perspective on the California experience.

D.2 Hudson River, New York

D.2.1 Problem Identification

One of the most severe cases of environmental contamination by PCBs occurred in the upper Hudson River Valley of New York. It is discussed in detail below to indicate the potential environmental impacts of uncontrolled discharge. The problem was first recognized in 1975, with the discovery that fish in the Hudson River contained PCBs at levels substantially greater than the FDA limit of 5 ppm. In the same

TABLE D.1

TYPICAL PCB CONCENTRATIONS IN THE HUDSON RIVER

	North of Troy	Troy to NYC
Sediments	20-150 µg/g	1-15 µg/g
Water	0.1-1 µg/l	Less than 0.8 µg.l
Fish	10-130 µg/g	5-15 µg/g
Macroinvertebrates	3-10 µg/g	1-3 µg/g
Turtles	6000 µg/g	600 µg/g

PCBs IN DRINKING WATER

Location	Raw Water	Finished Water
Above Hudson Falls	No	No
Waterford	Yes	Occasionally - less than DOH guidelines
Poughkeepsie	Yes	Occasionally - less than DOH guidelines

PCB LEVELS IN FISH AND WILDLIFE

Species	Ft. Edward to Troy	Troy to NYC
Smallmouth Bass	14 times FDA limit for interstate shipping	3 times FDA limit for interstate shipping
Yellow Perch	26 times FDA limit	Approx. equal to FDA limit
White Sucker	14 times FDA limit	3 times FDA limit
Carp (gold fish)	100 times FDA limit	--
Striped Bass	---	3 times FDA limit

The FDA limit on fish shipped interstate = 5 µg/g

FDA = U.S. Food and Drug Administration

DOH = N.Y. Department of Health

Source: Malcolm Pirnie, Inc. PCB Hot Spot Dredging Program - Upper Hudson River, New York. Draft Environmental Impact Statement, prepared for State of New York, Department of Environmental Conservation, September 1980.

an estimated 170,000 pounds of PCBs by placing PCB-contaminated sediments in encapsulation sites. General Electric has agreed to design the landfill sites, pay for all engineering studies and remedial actions, and monitor and maintain the sites for 30 years after completion of the remedial work. Included in the General Electric remedial program is the removal of wastes containing 330,000 pounds of PCBs from unsecure dump sites to places of more controlled containment.

D.2.3 Extent of Contamination

The amount of environmental contamination was significant. During a 30-year period ending in 1977, two General Electric capacitor manufacturing plants discharged over 500,000 pounds of PCBs to the Upper Hudson River. Much of the contaminated material accumulated behind Fort Edward Dam, which was removed in 1973. Removal caused release of contaminated sediments, a problem exacerbated by subsequent floods, including a 100-year frequency flood in 1976. It is estimated that sediments of the Upper Hudson contain 350,000 pounds of PCBs, and lower Hudson sediments contain 150,000 pounds.

New York State monitoring determined that nearly all fish in the Upper Hudson contained PCBs at levels greater than the FDA limit of 5 ug/g. Several hundred to several thousand pounds of PCBs are tied up within Hudson River biota, a mass less than one percent of total PCBs in the river system. Water column concentrations of PCBs in the Upper Hudson River are relatively low (0.1 to 1.0 ppb). Levels in other media are much higher: sediment, 20 to 150 ppm; fish 10 to 130 ppm, and turtles, up to 6,000 ppm. Some typical levels are summarized in Table D.1.

Significantly, drinking water PCB levels (sand -filter treated) were below 0.16 ug/l. Levels in river water ranged from below the 0.1 ug/l level of detection up to 5 ug/l and were related to the river flow rate.

D.3 Llano (Antelope Valley) PCB Dump Site

D.3.1 Introduction

This case study documents the actions that the Lahontan Regional Water Quality Control Board (Region 6) took to deal with an illegal hazardous waste site near Llano in Los Angeles County. The case study illustrates: (1) the difficulty of assigning responsibility for hazardous waste disposal and (2) the need for accurate laboratory analysis and on-site problem identification.

D.3.2 Problem Identification

In April 1980, a resident informed the Los Angeles County Department of Health Services (LADHS) of an illegal chemical dump of nineteen 55-gallon drums near Llano. Many of the drums had been punctured by bullet holes and their contents had leaked into the soil. An LADHS official took a sample from one of the drums and had it analyzed by the State Department of Health Services (DHS) laboratory. DHS reported the existence of the dump site to the Regional Water Quality Control Board (RWQCB) several days later. RWQCB staff investigated the site. The initial lab report indicated the presence of polychlorinated benzenes, but no PCBs.

Given the lab report and features of the site, the RWQCB determined that water quality was not immediately threatened and assumed that DHS would take responsibility for waste cleanup. Following a call from EPA Region IX, Regional Board staff contacted LADHS and DHS regarding cleanup of the site in August 1980. Neither EPA nor HDS nor LADHS took the initiative to obtain funds for cleanup.

In view of this bureaucratic inertia, the Regional Board decided to issue a Cleanup and Abatement Order. Eight additional drums had been discovered at two other sites, and RWQCB staff decided that ground water could become contaminated unless remedial action were taken. Additional site inspections were conducted and it was estimated, based on L.A. County Department of Health information, that two feet of contaminated soil would have to be disposed of.

RWQCB staff determined that neither the property owners where the drums were located, nor the state or county health departments had funds for waste cleanup. The Regional Board arranged to use the State Water Pollution Cleanup and Abatement Account to pay for disposing of the Llano wastes in November 1980. An additional laboratory analysis was required to determine if the wastes contained PCBs, which would limit disposal options. This analysis revealed that the liquid contained 50 percent PCB and 50 percent polychlorinated benzenes.

D.3.3 Problem Mitigation

The Regional Board contracted with Environmental Emergency Services to: (1) repackage the liquid wastes and transport them to Alabama for storage pending incineration in Arkansas; and (2) excavate contaminated soil and transport it and the empty drums to Beatty, Nevada, for landfilling. When disposal work began in December, it became evident that the PCB waste had penetrated deeper in the soil than originally estimated. This more extensive contamination meant that the initial allotment of $50,000 would not cover the cleanup cost. Cleanup activities, consisting of disposing of the PCB liquid and drums and 480 cubic yards of soil and installing a fence, gate, warning signs and headlight deflectors at the site, nearly depleted the cleanup and abatement account. A total of $131,000 was expended, and exploratory drilling at the site indicated that at least 500 additional cubic yards of contaminated soil required disposal.

Regional Board staff concluded that, following cleanup actions, the Llano site posed no short-term threat to water quality. As of April 1981, sampling indicated 1,000 ppm PCB in soil at depths greater than 10 feet. No PCBs above the detection level of 0.5 ppb were found in any of four wells sampled. Affected state and local agency officials recommended two remedial measures to prevent ground water pollution: placing a clay cap over the spill area and diverting surface drainage away from the site.

The State Department of Health Services has assumed responsibility for site cleanup. Soil samples taken in June 1981 indicated that the contamination had migrated to 25 feed of depth. Remedial action, funded by the federal and state superfund programs, is anticipated to fill in the excavated area and cap the site with a clay cover.

D.3.4 Conclusions and Recommendations

Sampling and Laboratory Analysis

Errors were made in analyzing liquid waste samples and soil contamination. The initial DHS laboratory analysis only covered the volatile portion of the sample and failed to indicate the presence of PCBs. With regard to the soil samples, RWQCB might have selected a different approach to eliminating the threat from contaminated soil had it recognized the extent of the problem. PCB migration in the soil has continued, based on sampling conducted during 1981. A higher priority might have been placed on capping the site if more accurate information on the extent of soil contamination had been available. More careful analysis, both in the laboratory and on-site, are essential to allow public officials to respond appropriately to hazardous waste threats to public health and the environment.

Responsibility for the Problem

Initially, the RWQCB was the only public agency willing to assume responsibility for cleaning up the Llano site. EPA, LADHS and DHS lacked funds for cleanup. The State Board's Cleanup and Abatement Account solved part of the problem: disposal of the liquids, drums, and part of the contaminated soil. Previous problem assessment and cleanup efforts had defined the scope of the public health threat. The establishment of the state superfund and DHS assignment of a high priority rating to the Llano site should mitigate the remainder of the problem.

D.4 Klamath River (Siskiyou County) Abandoned Barrel

D.4.1 Introduction

This case study documents the actions that the North Coast Regional Water Quality Control Board (Region 1) took to eliminate a PCB threat to water quality. The Regional Board assumed responsibility for arranging disposal of a PCB-contaminated, 55-gallon barrel deposited by the Klamath River. The case study illustrates: (1) the difficulty of assigning responsibility for disposal of a suspected hazardous waste and (2) the hazardous waste transport, storage, and disposal process as applied to a Regional Board remedial action.

D.4.2 Problem Identification

On October 27, 1981, the Regional Board was notified that a 55-gallon barrel labeled as containing a PCB mixture (Interteen) was on a mining claim near the Klamath River in Siskiyou County. The Regional Board informed the local health department of the barrel's presence on October 28. The health department staff determined that the barrel was secure and took a sample between October 28 and November 16 to verify its contents. Analysis of a suspected hazardous material is a prerequisite to further actions. The sample was transported to the DHS laboratory by car for analysis. Initial laboratory analysis did not reveal the presence of PCBs. The follow-up laboratory analysis, completed on December 3, showed 47.7 percent PCB content.

Following the intital contact of the local health department, Regional Board staff

Santa Rosa to Arkansas, (2) an Arkansas manifest, (3) service order, and (4) bill of lading. IT provided Regional Board staff with the required manifests and assisted in completing them. On January 4, 1982, an IT truck picked up the 55-gallon barrel for shipment to Arkansas. It took 42 days from initial notice to obtain analysis and make the cleanup and abatement arrangements. It took an additional 28 days to execute those arrangements.

D.4.4 Cost of Cleanup

The Siskiyou County health department sampled the barrel, and DHS analyzed the sample. SWRCB covered the major disposal costs, which are estimated as follows: $2,500 for IT Corporation to travel to Yreka, pick up the barrel, and transport it to Santa Rosa; $500 for an IT Corporation truck to transport the barrel (along with other hazardous wastes) to the Ensco facility in Arkansas; and $700 for incineration of the PCB waste. Including a 10 percent contingency, a total of $4,070 was budgeted from the SWRCB Cleanup and Abatement Fund for disposal of the barrel.

D.4.5 Conclusions and Recommendations

Laboratory Analysis

Both the quality and timeliness of DHS laboratory services were deficient. As in the case of the Llano incident, the initial laboratory analysis failed to detect the presence of PCBs. Laboratory analysis of abandoned wastes should more carefully check for the presence of hazardous or extremely hazardous substances.

With respect to turn-around time, it took 16 days for the DOHS laboratory to analyze the barrel sample. This time period represented a high priority rating. For an unknown, but potentially hazardous waste posing an imminent and unknown threat to humans or the environment, faster turn-around is essential. Operations of the DHS laboratory should be modified to provide prompt laboratory analysis of abandoned hazardous wastes.

Hazardous Waste Management

This dumping incident occurred just as the state health department was reorganizing to implement the state and federal superfund programs. DHS staff was changing positions and the Regional Board staff could not identify the responsible officials.

In addition, DHS has the authority to waive procedural requirements in emergency situations. Thus, the month-long delay that occurred because the Regional Board was complying with the letter of the regulations is avoidable. The Regional Board was not familiar with the waiver provision.

Now that this transition period has ended, communication between Regional Board and DHS staff should be more straightforward. While the DHS reorganization should address the logistics of coordination, the issue of integrating State and Regional Board water quality priorities in the DHS-headed hazardous waste management program remains. SWRCB/RWQCB and DHS have overlapping authority for both hazardous waste management and protection of human health. As the lead agency for federal and state superfund, DHS controls the major sources of spill and abandoned waste cleanup funds. To effectively carry out their responsibilities, the State and Regional Boards must have access to these funds.

D.5 PCBs in Feather River Fish (Butte County)

D.5.1 Introduction

In 1977 and 1978, the Toxic Substances Monitoring Program (TSM) detected PCBs in fish on the lower Feather River at Nicolaus. PCB concentrations in fish tissue exceeded 500 ppb in both years. In 1980, TSM staff began an action plan to intensively survey Feather River fish, water, and sediment for PCBs, an effort that involved staff from the SWRCB, Central Valley Regional Water Quality Control Board, and the Departments of Fish and Game and Water Resources. In addition, staff from the Regional Board conducted a survey of power transmission facilities located within the Feather River drainage to determine where PCB leaks and spills had occurred and to estimate the total volume of PCBs present in power transmission equipment in the region.

D.5.2 Results of Action Plan Sampling

In addition to the routine TSM Feather River monitoring station at Nicolaus, seven additional sites were monitored as part of the action plan (Figure D.1). Levels of PCBs detected in fish, water, and powerhouse sumps are presented in Table D.2. Concentrations of PCBs in fish from the North Fork of the Feather River were below the detectable tissue concentration of 50 ppb. In contrast, PCBs were detected in fish from two reservoirs on the South Fork. At Woodleaf Reservoir, brown trout contained 140 ppb PCBs, and sediment from the sump of an adjacent powerhouse contained 1200 ppb PCBs. Fish from Ponderosa Reservoir below the Forbestown Powerhouse bore high levels of PCB. A sample of six suckers contained 7700 ppb, a concentration exceeding the current FDA action level. Two other species examined, squawfish and suckers, had PCB levels greater than 2000 ppb (Figure D.2). PCBs were also detected in the water (at 0.022 ppb) and in soil samples taken near the Forbestown Powerhouse (up to 20,000 ppb). Identification of the Ponderosa Reservoir - Forbestown Powerhouse site as an area of high PCB concentration resulted in a full-scale investigation by Regional Board staff.

FIGURE D.1

ACTION PLAN SAMPLING LOCATIONS FOR PCBs
IN THE FEATHER RIVER SYSTEM, 1980

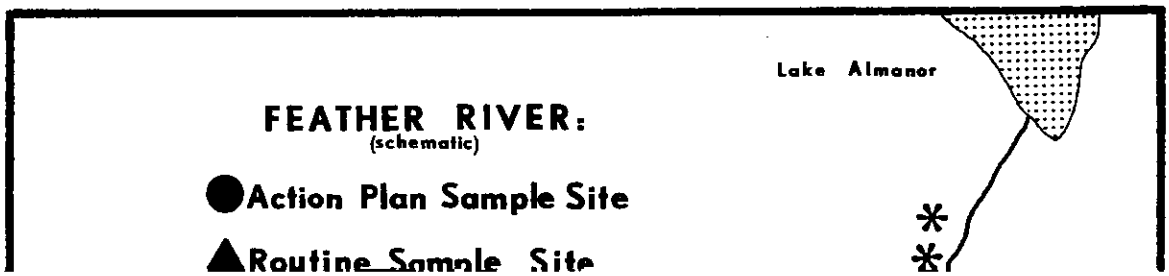

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FIGURE D.1

ACTION PLAN SAMPLING LOCATIONS FOR PCBs
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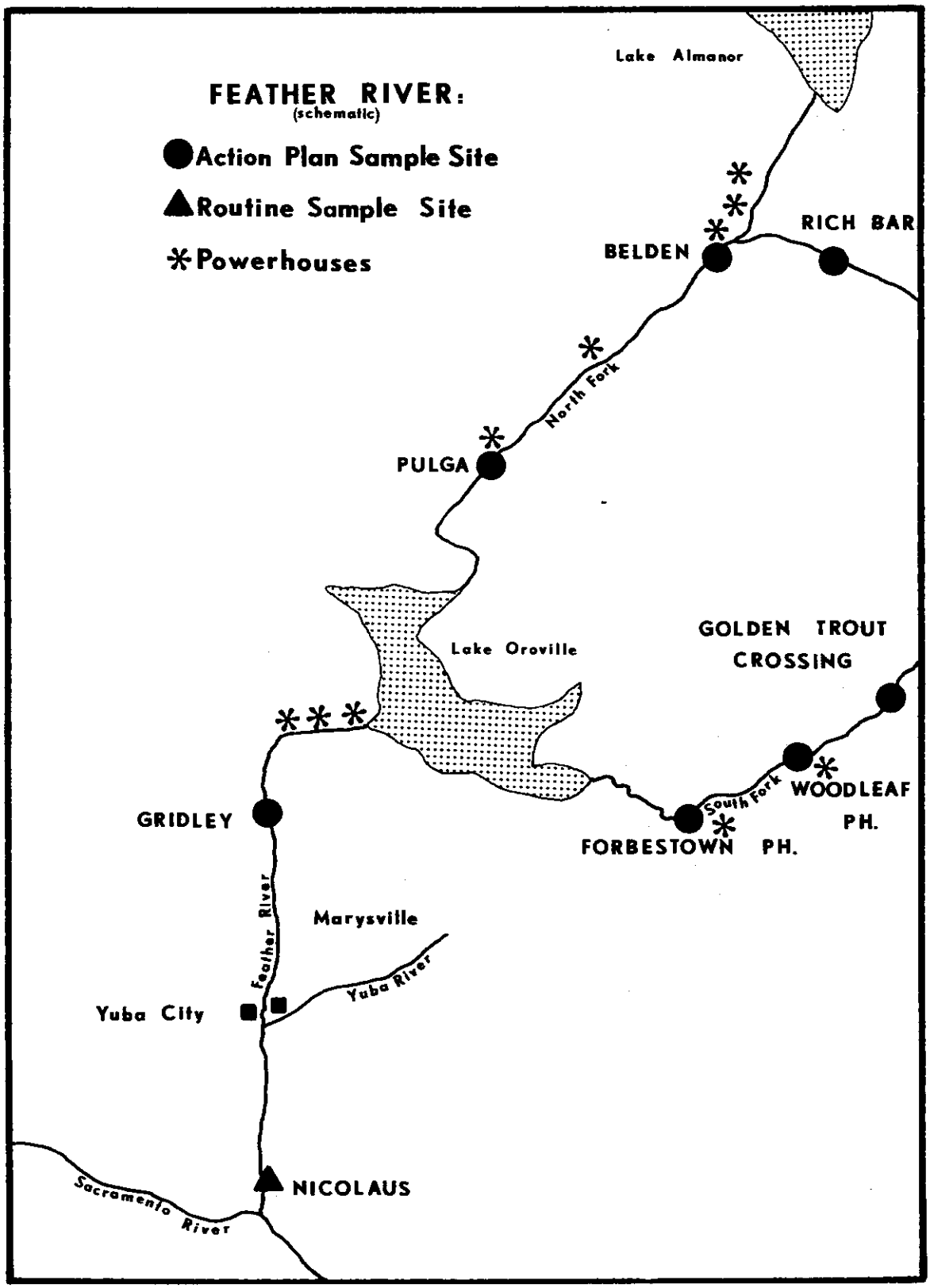


TABLE D.2

SUMMARY OF PCB ACTION PLAN DATA FROM FEATHER RIVER, 1980

		Biological Data						Environmental Data	
						Tissue Data		Powerhouse Sumps	Water
	Species	Sample Size	Mean Fork Length (mm)	Mean Weight (g)	Mean Age	% Lipid	PCB (ug/g fresh wt.)	PCB (ug/g dry wt.)	(ug/l)
Feather River									
Nicolaus (Routine Sta.)	smallmouth	5	257	305	3.0	0.09	<50	-	NA
" "	channel catfish	1	321	393	4.0	2.40	80	-	
" "	bluegill	4	167	117	2.5	0.04	<50	-	
" "	squawfish	5	211	85	1.5	0.12	60	-	
Gridley	white catfish	1	262	292	3.0	0.88	80	-	0.029 [1/]
S. Fk.; Forbestown PH.	squawfish	3	358	516	3.0	0.18	4800	<0.5 [3/]	0.022 [1/]
" "	channel catfish	1	335	560	4.0	0.29	140	-	
" "	suckers	6	396	768	4.0	3.20	7700	-	
" "	hardhead	1	342	503	5.0	3.20	2100	-	
" "	brown trout	4	268	221	2.5	0.39	350	-	
" "	rainbow trout	4	424	205	2+	0.76	320	-	
S. Fk.; Woodleaf Res.	brown trout	5	255	191	2.3	0.80	140	1.2 ppm	-
S. Fk.; Golden Trout Crossing	brown trout	6	232	163	2.5	0.79	<50	-	-
N. Fk.; Pulga	squawfish	1	413	144	5.0	0.10	<50	-	NA
N. Fk.; Belden	squawfish	1	198	85	2.0	0.09	<50	0.51ppm	.011 [2/]
" "	rainbow trout	2	291	306	3.0	0.57	<50	-	
N. Fk.; Rich Bar	brown trout	5	254	197	2.0	0.55	<50	-	0.037 [1/]

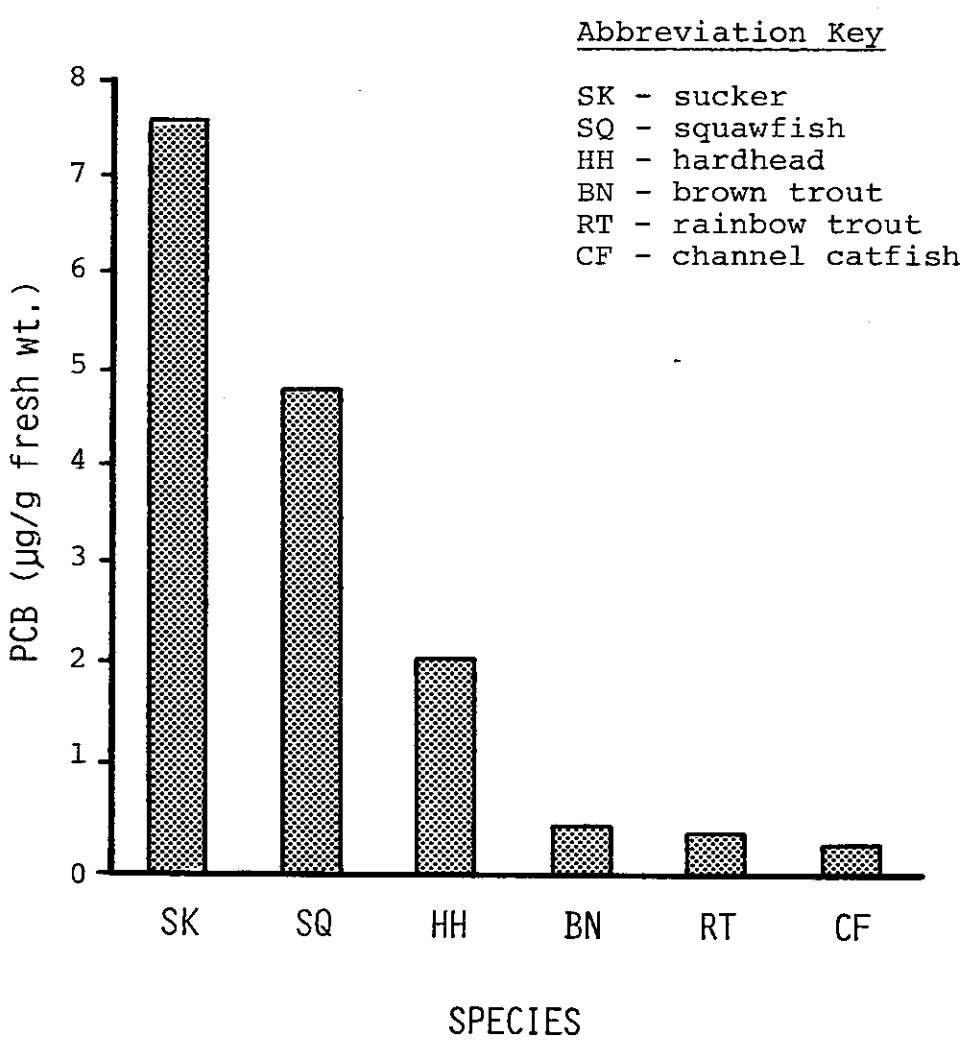
[1/] Results from the California Department of Water Resources (Bryte Lab.), 1980.

[2/] Results from Bodega Marine Laboratory, 1980.

[3/] Soil samples taken around Forbestown Ph. has as high as 20ppm PCB.

FIGURE D.2

RELATIVE CONCENTRATION OF PCB IN FISH COLLECTED IN PONDEROSA RESERVOIR BELOW FORBESTOWN POWERHOUSE



road into Ponderosa Reservoir. Five trenches were dug by backhoe, and soil logs and samples were taken. PCB levels ranged from below detection levels to 8300 ppb. The geologists concluded that little, if any, subsurface migration had occurred. The main route of migration from the road to the reservoir had been erosion of surface and near surface soil-contaminated with PCBs. Regional Board staff recommended paving the affected 1/4 mile of road to prevent further PCB discharges into the reservoir.

D.5.4 Conclusions and Recommendations

Although there are probably other sources of PCBs that contaminate the Feather River, this case study illustrates a success story. Follow-up action to initial findings of PCBs in Feather River fish led to mitigation of a major source as well as preventative work to clean up power transmission facilities that could cause PCB contamination problems in the future. In addition, PG&E is eliminating much of its PCB-containing equipment in the area. All PCB-filled transformers on the Feather River were replaced by the middle of 1981. A total of 5,510 distribution line capacitor banks had been removed by late 1982.

During the course of its investigation, Regional Board staff made several recommendations that would be useful in other cases where PCBs are found in the biota:

(1) Fish sampling points for follow-up work should concentrate on reservoirs and similar sites. The density of PCBs and the affinity of PCBs for organic material make reservoirs "prime sinks" for accumulating these compounds discharged into waterways.

(2) Physical surveys of potential PCB-containing equipment, such as examination of power transmission facilities, to identify potential sources of leaks should be conducted before selecting fish sampling points.

(3) PCB concentrations in fish tissue will vary considerably, influenced by factors such as species of fish, season of sampling, and lipid content.

(4) The detection of PCBs at significant levels in fish during routine monitoring is cause for follow-up work.

D.6 PCB Contamination of Soil and Groundwater (Oakland, California)

D.6.1 Introduction

This study documents a problem that has frequently occurred at sites where PCB transformers and capacitors have been fabricated. In the past, lack of awareness of the toxicity and persistence of PCBs led to indiscriminate on-site disposal.

believed to have hydraulic continuity with San Francisco Bay, contained PCB concentrations up to 1 part per billion. The San Francisco Bay Regional Water Quality Control Board (Region 2) issued a Cleanup and Abatement Order in December 1980.

D.6.3 Problem Mitigation

General Electric had previously encountered PCB soil and ground water problems at its Pittsfield, Massachusetts electrical equipment manufacturing plant. Using the Massachusetts experience, GE, with its consultants, developed a two-phase "Immediate Correction Plan" in January 1980. This preliminary design included: (1) oil plume control and (2) surface runoff control. The oil plume control consists of two parts: (1) collection of PCB-containing oils with a "French drain" and (2) a ground water treatment system to remove organics. The French drain consists of a trench--30 feet long, 20 feet deep and three feet wide--containing a perforated pipe filled with gravel. By pumping groundwater to establish a gradient of flow toward the drain, contaminated water is removed, and groundwater contamination is slowed. The ground water is then treated and discharged to the East Bay Municipal Utility District's waste treatment plant. The surface runoff control system consists of a bentonite claysoil cap placed on unpaved areas of the site.

D.6.4 Conclusions and Recommendations

Inspection and Enforcement Priorities

The General Electric transformer manufacturing site provides an example of extensive PCB contamination caused by past disposal practices. Similar Bay Area problems are known to exist at Westinghouse plants in Sunnyvale and Emeryville.

Past disposal practices at PCB equipment manufacturing sites are likely to have caused potential or actual water quality problems. PCBs were not considered harmful, and stringent disposal requirements were not in effect. Regional Water Quality Control Boards and other field staff conducting compliance inspections should assign a high priority to these sites. Early identification and mitigation of PCB water quality problems will minimize adverse effects on beneficial uses.

Problem Mitigation

The Immediate Correction Plan consists of both a preventative and corrective component. The clay cap will prevent water quality contamination from surface runoff. The collection and treatment of PCB-containing liquids will remove PCBs from ground water. This program can serve as a model for mitigating PCB water quality problems.

D.7 PCB Discharges at MGM Brakes Facility (Cloverdale, Sonoma County)

D.7.1 Introduction

Prior to 1971, PCBs were used extensively as hydraulic fluids and lubricants. Recycling of PCB-containing fluid led to contamination of many liquids used for these purposes. Subsequent illegal dumping or backlot disposal of PCB-containing waste oils has created the potential for widespread soil contamination. as illustrated in this case study.

D.7.2 Problem Identification

In August 1981, staff of the North Coast Regional Water Quality Control Board (Region 1) received a complaint indicating that a Cloverdale metal casting shop was illegally discharging wastewater and oil to the soil behind the plant. A subsequent inspection confirmed the illegal discharge. Eighteen days after receiving the complaint the Regional Board issued a Cleanup and Abatement Order.

One month after the initial complaint Region 1 staff again inspected the site to determine if the Cleanup and Abatement Order was being followed. Soil that had been identified as contaminated had been collected and stockpiled. Soil samples were taken and sent to the IT Corporation for chemical analysis prior to disposal. The IT Corporation notified the casting plant that PCBs were present in the waste oil-contaminated soil. Additional samples were collected from (1) the stockpiled soil and (2) the area where the contaminated soil had been removed. PCB levels were 357 ppm in stockpiled soil and ranged from 21 to 276 ppm in the area from which soil had been removed.

The PCB source appeared to be hydraulic fluids present in the hydraulic machinery used to cast metal parts for trucks. Operating under very high pressure, these machines typically leak up to ten times their fluid capacity annually. The company historically had collected leaking hydraulic oils into a storage tank to separate water and oil. The oil and water mixture separated into three layers in the tank: the top layer consisting of oils lighter than water, a polar phase, and the bottom layer of heavier-than-water oils. The plant routinely drew off the middle aqueous phase and discharged it to the surface of the back lot.

Although the oil layers were saved for ultimate recycling, crude separation of oil and water allowed for PCB contamination of the aqueous layer. Previously the metal casting plant had used a Monsanto hydraulic fluid, Hydrol 312, that contained 47 percent PCBs (Hydrol 312 has now been reformulated into a non-PCB product, Hydrol 312-C). According to information provided Regional Board staff there has been no recall of PCB-containing products. Therefore, some firms may have continued use of stored PCB hydraulic fluid until supplies were used up. Further,in hydraulic fluid systems, the replacement of PCB liquids is basically a dilution process: several replacements are necessary before sufficient dilution has occurred.

In November 1981, the Regional Board issued a second Cleanup and Abatement Order, requiring the casting plant to determine the extent of PCB contamination and then to clean up and abate the the effects of the discharge. In response with the

Decontamination of the plant (floors, walls, machinery, etc.) to remove PCBs cost $250,000. More extensive soil sampling indicated that an estimated 3,000 cubic yards of soil are PCB-contaminated. Consultants estimate the cost of soil removal to be $900,000. Because removal and disposal of all contaminated soil would be prohibitively expensive. the migration plan recommended by the consultants is to encapsulate and cover the site with a clay liner and provide surface runoff diversion drains to prevent further dissemination of PCBs. To meet the Cleanup and Abatement order requirement of no detectable PCB levels in ground or surface water, some soil removal may be necessary. The consultants claim that PCBs are immobilized in the soil and no migration will occur. The regulatory agencies are not convinced that encapsulation will prevent water pollution. If samples from monitoring wells reveal PCB contamination of off-site water, then the migration plan may have to be revised to include additional soil removal or treatment of contaminated water. The final form of the migration plan had not been decided as of December 1982.

D.7.4 Conclusion and Recommendations

Problem Identification

This case study illustrates the importance of comprehensive sampling to identify the full extent of and potential for soil and water contamination. Eighteen months after the problem was reported to the Regional Board, its magnitude had grown considerably. If the initial mitigation program of total removal and disposal of contaminated soil had been implemented in early 1982, a year later the consultants and regulatory agencies would have learned that they had just scratched the surface.

Solving such a hazardous waste disposal problem must proceed in tandem with defining the problem. First, any immediate threat to public health or the environment must be eliminated. Second, the potential long-term problem must be assessed to ensure that appropriate mitigation measures are carried out. It is important for the regulatory agencies to separate these two distinct phases of the cleanup program--emergency and remdial response--so that they base the implementation plan on the most complete information available at the time.

This case study is a good example of this two-part approach. The Regional Board took prompt action to ensure that no water quality threat existed. Then the regulatory agencies revised their long-term solution from total cleanup to on-site encapsulation based on additional soil sampling results. The requirement of extensive monitoring will ensure appropriate mitigation measures.

Feasibility of Control Measures

The case study is undoubtedly one of many examples of extensive soil contamination problems in California. Removal of all contaminated soil can be extremely costly. Soil encapsulation may be an acceptable option where off-site migration of toxics such as PCBs can be prevented. In cases where it is uncertain that encapsulation can prevent migration, a variety of mitigation measures, such as soil removal and leachate control, must be evaluated. The feasible solution to each specific PCB problem must consider environmental and economic factors in controlling the effects of the discharge.

APPENDIX E

OVERVIEW OF FEDERAL AND STATE
PCB REGULATIONS

E.1 PCB Manufacture, Use, Storage, and Transportation

Federal regulations applicable to PCB storage or transportation relate to: spill prevention (EPA), Interstate Commerce (Department of Transportation) and flammable liquids (Occupational Safety and Health Administration). State regulation of PCB storage and transportation is based on the classification of PCBs as hazardous or extremely hazardous wastes, depending on the PCB concentration. Table E.1 summarizes federal (TSCA) and state regulation of PCB use, storage and disposal.

E.1.1 Manufacture, Processing, Distribution and Use

EPA has exclusive authority under Section 6 of TSCA to regulate the manufacturing, processing, distribution in commerce and use of PCBs. Furthermore, section 18 of TSCA preempts state requirements that are not identical to or more stringent than the federal requirements, unless special approval is obtained from EPA. EPA issued a regulation on May 31, 1979, (40 CFR 761) covering these activities. In summary, the regulation:

1. Prohibited all manufacturing of PCBs after July 2, 1979, unless specifically exempted by EPA;

2. Prohibited the processing, distribution in commerce, and use of PCBs except in a totally enclosed manner, or where specifically exempted after July 2, 1979; and

3. Authorized certain processing, distribution in commerce, and use of PCBs in a non-totally enclosed manner.

The Environmental Defense Fund challenged this regulation, and EPA has issued revised regulations to comply with the court order. EPA eliminated totally enclosed uses because evidence was presented that all electrical equipment leaks. The regulation also established phase-out deadlines for certain PCB transformers and all large PCB capacitors. See Chapter 7, Section 7.5, for a description of the major features of the revised regulations.

E.1.2 Recordkeeping, Labeling and Storage

The PCB regulations require major PCB users and owners of PCB storage or disposal facilities to compile an annual inventory of PCBs in use or handled. They also establish marking requirements for PCB containers, transformers, capacitors, and transport vehicles.

The PCB regulations establish standards for storage facilities and deadlines for storage of PCB items. PCBs stored for disposal before January 1, 1983, must be disposed of by January 1, 1984. PCBs stored after January 1, 1983, must be disposed of within one year. The regulations permit temporary storage of certain PCB items in facilities that do not meet all the design requirements.

Storage containers for PCB liquids must comply with the Department of Transportation Shipping Container Specification (49 CFR 178) and with the Occupational Safety and Health Act Standards for flammable and combustible liquids (29 CFR 1910). Temporary

TABLE E.1

REGULATION OF PCBs IN USE OR STORAGE

ACTIVITY	FEDERAL EPA (TSCA) 1/	STATE			OTHERS
		DHS 2/	SWRCB/RWQCB 3/	ARB 4/	
USE, PROCESSING, DISTRIBUTION IN COMMERCE	REGULATES				
LABELING	SETS REQUIREMENTS				
RECORD-KEEPING	REQUIRES OF OWNERS				
STORAGE	SETS STANDARDS, TIME LIMITS	ISSUES PERMITS			
INSPECTION	CONDUCTS	CONDUCTS	RWQCBs CONDUCT		OFFICE OF THE STATE ARCHITECT SURVEY
ENFORCEMENT	PERFORMS	PERFORMS	SWRCB, RWQCBs PERFORM	PERFORMS	CAL OSHA PERFORMS
DISPOSAL FACILITIES	SETS STANDARDS	SETS MORE STRINGENT STANDARDS, REGULATES		SETS POLICIES FOR INCINERATION	

1/ UNITED STATES ENVIRONMENTAL PROTECTION AGENCY (TOXIC SUBSTANCES CONTROL ACT)

2/ DEPARTMENT OF HEALTH SERVICES

3/ STATE WATER RESOURCES CONTROL BOARD/REGIONAL WATER QUALITY CONTROL BOARDS

4/ AIR RESOURCES BOARD

storage of PCB containers with 50 to 500 ppm in PCB liquid is permitted, provided a Spill Prevention Control and Countermeasure Plan (EPA Clean Water Act, Section 311, 40 CFR 112) has been prepared.

The State Department of Health Services (DHS) issues permits for PCB storage under Section 25200 of the Health and Safety Code. DHS classifies PCBs containing more than 500 ppm as extremely hazardous wastes and PCB solids containing 50 to 500 ppm as hazardous wastes. PCB liquids containing 7 to 500 ppm are hazardous wastes according to state requirements. State regulations require facilities that store hazardous or extremely hazardous waste for more than 60 days to obtain a hazardous waste facility permit. These regulations also require hazardous waste facilities to have operating plans, which include the volume and type of waste handled, operational procedures, and an emergency contingency plan.

E.1.3 Transportation of PCBs for Disposal

Transportation of PCBs for disposal is covered by U.S. Department of Transportation (DOT) regulations and by state regulations related to hazardous materials management. The DOT regulations (49 CFR 397, 171, 172, 173) establish standards and requirements for carriers and shipments. These provisions relate to transport operations, such as placarding and hours that drivers may work, rather than tracking the PCB waste from generator to disposal site.

The state agencies responsible for regulating PCB transport are the Department of Health Services and California Highway Patrol. DHS registers hazardous waste haulers and monitors their operators under Section 24100 of the Health and Safety Code. DHS administers a hazardous waste tracking system to comply with state law

TABLE E.2

FEDERAL AND STATE DISPOSAL REQUIREMENTS FOR PCBs

	Federal Requirements			
PCB Item	Incinceration	Chemical Waste Landfill	Alternative	Superceding State Requirements
Liquid PCBs (concentrations above 500 ppm)	Required	Not Acceptable	None	incinceration required for concentration over 50 ppm
Liquid PCBs (concentrations 50-500 ppm) (Includes mineral oil dielectric fluid from PCB-contaminated transformers, and other PCB-contaminated liquids)	Acceptable	Acceptable if liquid is not an ignitable waste	High-efficiency boiler that meets required criteria or alternative disposal method approved by the EPA Regional Administration.	Definition of PCB liquid expanded to include 7-49 ppm.
Non-Liquid PCBs (contaminated soil, rags, and other debris)	Acceptable	Acceptable	None	
Municipal sewage treatment sludge and dredged materials	Acceptable	Acceptable	Alternative method approved by the Regional Administration	No alternative method allowed.
PCB Transformers	Acceptable	Acceptable if drained, filled with solvent, allowed to stand for 18 hours, and then drained.	None	
PCB-contaminated transformers (concentrations 50-500 ppm), drained		Disposal not regulated.	Reuse	Incinceration or chemical waste landfill required for disposal.
PCB Large High or Low Voltage Capacitors	Acceptable	Acceptable	None	
PCB Small Capacitors, owned by manufacturers of PCB capacitors or PCB equipment	Acceptable	Acceptable	None	

TABLE E.2 (continued)

FEDERAL AND STATE DISPOSAL REQUIREMENTS FOR PCBs

PCB Item	Federal Requirements: Incinceration	Federal Requirements: Chemical Waste Landfill	Federal Requirements: Alternative	Superceding State Requirements
PCB Small Capacitors, other than the above			Municipal Solid Waste	Case-by-case determination required.
PCB Hydraulic Machines if drained of free flowing fluid			Municipal Solid Waste	Municipal solid waste not acceptable. Chemical waste landfill required.
Other PCB Articles	Acceptable	Acceptable if free flowing liquid is drained prior to disposal.	None	
PCB Containers, not decontaminated	Acceptable	Acceptable if liquid PCBs are drained.	None	
PCB Containers, containing only PCBs at concentrations below 500 ppm			Municipal Solid Waste if liquid PCBs are drained	Municipal solid waste not acceptable. Chemical waste landfill required.
PCB Containers, decontaminated in accordance with #761.43			Reuse or Municipal Solid Waste	Municipal solid waste not acceptable. Chemical waste landfill required.

APPENDIX F

SOME PCB SPILL/CONTAMINATION SITES REPORTED
BY REGIONAL WATER QUALITY CONTROL BOARDS (RWQCBs)

SITE	DESCRIPTION	APPROXIMATE AMOUNT
NORTH COAST REGION		
MGM Brakes, Inc., Cloverdale	Metal casting plant disposed PCB-containing hydraulic fluid from oil-water sump to back lot. Cleanup and Abatement Orders (C&A) issued by RWQCB in August and November, 1981. Mitigation plan being developed.	8,000 cubic yards of contaminated soil.
Klamath River	Barrel beached on sand bar in 1981, contained askarel mixture (47% PCB). Regional Board arranged for disposal.	55 gallon barrel.
Fort Bragg, "Glass Beach"	Capacitors disposed of on beach; ~3 cubic yards of contaminated soil removed by PG & E in 1981.	4 Capacitors
Requa, Del Norte County	Six barrels spilled from truck when it went off road in 1979. Several barrels rolled down hill into a small creek serving as water supply.	8-10 gallons
Gardner's Oil	Company recycled oil from MGM Brakes, Inc. Suspected road contamination as a result of oil used for dirt road dust control. Some tanks contained oils exceeding 50 ppm. DOHS required disposal at Casmalia landfill.	?
SAN FRANCISCO BAY REGION		
General Electric Co., Oakland	Improper waste management by transformer manufacturing facility during 1950's. C&A issued by RWQCB in December 1980. Remedial action includes isolation of contaminated soil, treatment of contaminated ground water, and surface runoff control.	PCBs have been detected as deep as 35 feet (210 ppm) and 40 feet (1 ppm in soil cores).
Westinghouse, Emeryville	PCB soil contamination (EPA is lead agency).	?
CENTRAL COAST REGION		
Berman Steel Co., Watsonville	PCB Contamination (Soil ?)	?

APPENDIX F (continued)

SOME PCB SPILL/CONTAMINATION SITES REPORTED
BY REGIONAL WATER QUALITY CONTROL BOARDS (RWQCBs)

SITE	DESCRIPTION	APPROXIMATE AMOUNT
CENTRAL VALLEY REGION		
Interstate 5, near Lake Shasta, Shasta Co.	Truck carrying leaking transformer (November 1980). IT Corporation removed 1,200 gallons of PCB-containing fluid.	Transformer contained 1,500 gallons at 220 ppm PCB.
U.S. Coast Guard, Loran C Station, near Middletown, Lake County	PCB levels up to 43,400 ppm in soil samples at spill site. Drainage to Putah Creek and Lake Berryessa, (1977).	
Ponderosa Reservoir, S. Fork Feather River above Lake Oroville	PCBs used for dust control on Powerhouse Road by Oroville-Wyandotte Irrigation District, (1980).	246 gallons PCB sprayed on road.
McClellan AFB, Sacramento	Scrap metal business operating on land bought by McClellan AFB. PCB levels up to 224,000 ppm in soil. PCBs also found in drainage ditches near site and young minnows in Rio Linda Creek. McClellan AFB	McClellan AFB removed 800 cubic yards; Sacramento County removed over 1,300 cubic yards.

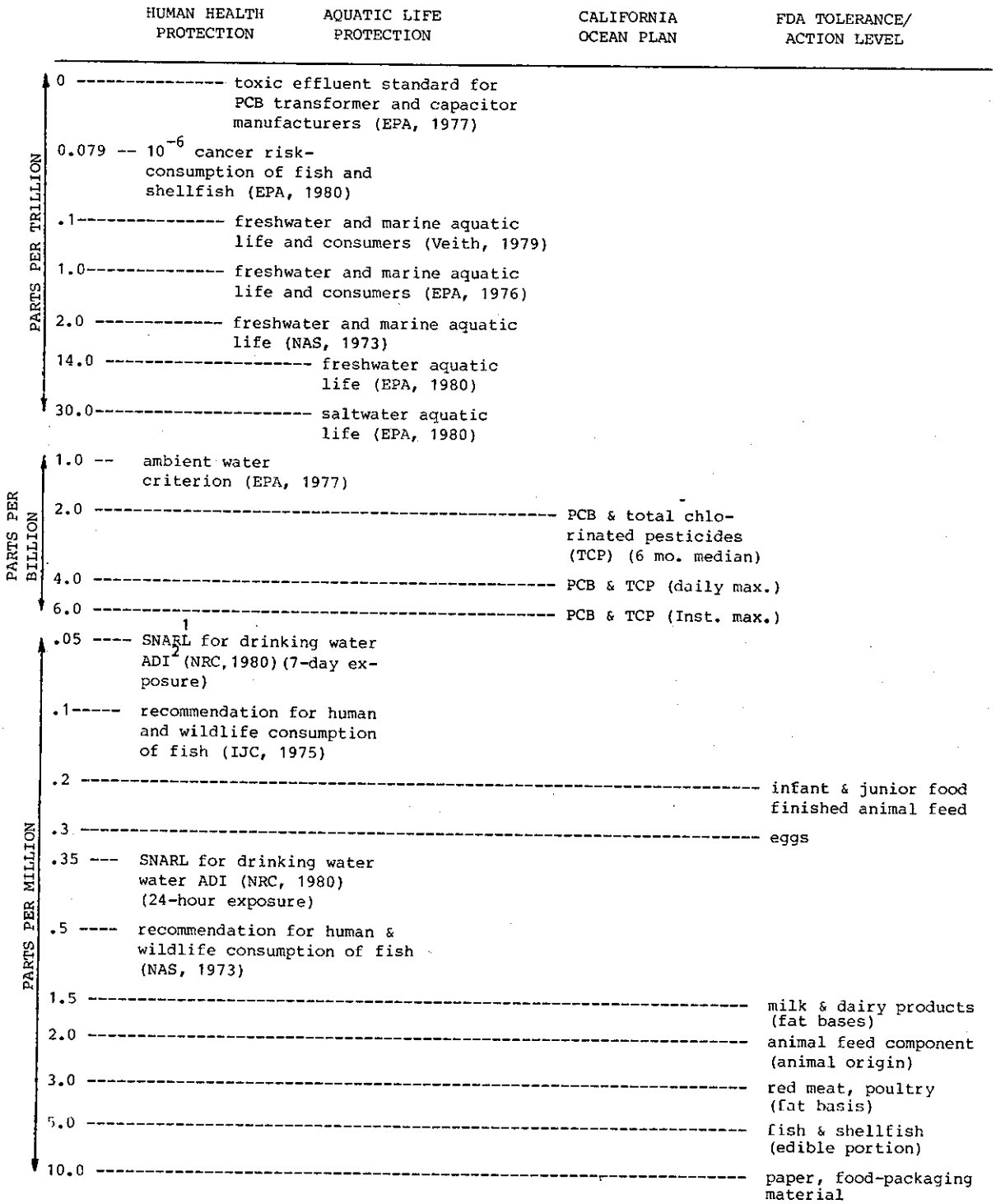
APPENDIX G
CRITERIA AND STANDARDS FOR PCBs

G.1 Water Quality, Fish Tissue and Food

Various federal and state agencies have adopted standards, criteria and policies to protect human health and the environment from adverse effects of PCBs. Figure G.1 lists PCB criteria and standards developed for (1) ambient water to protect humans, aquatic organisms and wildlife (2) drinking water to protect humans, (3) fish

FIGURE G.1

FEDERAL AND STATE CRITERIA AND STANDARDS FOR PCBs



1/ SNARL = suggested no adverse response level

2/ ADI = average daily intake

and wildlife consumption of fish. The International Joint Commission recommended a 0.1 ppm fish tissue concentration to protect human and wildlife consumers of fish (IJC, 1975).

G.1.4 Food

The United States Food and Drug Administration (FDA) established tolerances and action levels for PCBs in food and food-packaging materials (FDA, 1981). These tolerances and action levels represent limits at or above which FDA will remove products from the market. The FDA tolerances and action levels for PCBs range from 2 ppm for infant and junior food and finished animal feed to 10 ppm for paper and food packaging materials.

G.2 Air Quality

Various air quality standards and policies have been adopted for PCBs. The U.S. Occupational Safety and Health Administration (OSHA) adopted a 1 mg/m^3 standard (8-hour time-weighted average) for PCBs containing 42 percent chlorine and a 0.5 mg/m^3 standard for PCBs containing 54 percent chlorine (NIOSH, 1977). This OSHA standard applies for toxic effects other than cancer.

Cal OSHA proposed a standard for PCBs in June 1981 (General Industry Safety Order 5218). Present requirements pertaining to PCBs are contained in various standards and enforcement decisions are based on an assessment of each case. The proposed standard covers: permissible exposure limits, engineering controls, work practices, protective equipment and clothing, respiratory protection, employee decontamination, training and information, signs and labels, and medical surveillance. The proposed permissible exposure limit for the more dangerous 54 percent chlorine PCBs is 0.5 mg/m^3 (time weighted average) and 1 mg/m^3 for all other PCB mixtures. The proposed standard applies to PCB concentrations over 50 ppm.

In December 1981, the Air Resources Board adopted a policy of endorsing destruction of PCBs in cement kilns that have an efficiency rate of 99.998 percent, which is the limit of detection. The policy also requires test burns to evaluate levels of chlorinated dibenzofurans and dioxins for potential health hazards. The policy specifies cement kiln operating conditions covering contamination parameters and effluent pollutants. The policy also requires the preparation of a management plan identifying training programs and PCB storage and transportation methods.

APPENDIX H
REVIEW COMMENTS AND RESPONSE

Comments on the draft PCB Report were submitted by the following parties:

- Department of Fish and Game (DFG)
- Department of Health Services (DHS)
- Department of Industrial Relations (DIR)
- Air Resources Board (ARB)
- Regional Water Quality Control Boards (RWQCBs)
  - San Francisco Bay (Region 2)
  - Colorado River Basin (Region 7)
- Southern California Edison Company (SCE)
- Pacific Gas and Electric Company (PG&E)
- San Diego Gas and Electric Company (SDG&E)
- Sacramento Municipal Utility District (SMUD)

The major concerns expressed by these reviewers and the SWRCB response are presented below:

1. PCB Water Quality Strategy

COMMENT:
The proposed PCB water quality strategy incorporates minimum requirements for waste disposal adopted by DHS and for fish and shellfish consumption adopted by the United States Food and Drug Administration (FDA). These minimum requirements appear to duplicate existing standards and programs implemented by other agencies. Duplication of responsibilities should be avoided. (SCE, SDG&E).

RESPONSE:
The description of the proposed PCB water quality strategy has been clarified to indicate that the SWRCB program will support and rely upon existing PCB regulatory programs. We share the concern to avoid duplication of responsibilities.

COMMENT:
"Controllable sources of PCBs" is vague language, requires further definition and should not include regulated electrical equipment. (SCE, SDG&E, PG&E).

RESPONSE:
We agree and have defined controllable sources as spills and abandoned wastes for which cleanup is environmentally and economically feasible.

COMMENT:
The reportable quantity for PCBs should be 10 pounds, which is the Clean Water Act Section 311 limit adopted by reference in CERCLA. (SCE, SDG&E).

RESPONSE:
We proposed a 1 pound reportable quantity as compatible with the informal procedure that major California utilities have adopted of reporting all spills to DHS.

COMMENT:
The concept of response levels and minimum requirements needs clarification. The strategy should be flexible to allow changes as new information becomes available (SCE, PG&E, Region 2).

RESPONSE:
The rationale for the PCB water quality strategy has been expanded to provide additional information on the basis for and use of the limits. The strategy incorporates flexibility by tailoring actions to each individual case.

2. Utility Industry PCB Management Practices

COMMENT:
The report ignores PCB management practices by the utility industry for both on-line electrical equipment and spill response and cleanup. (SCE, SDG&E, SMUD).

RESPONSE:
Chapter 7 "PCB Regulatory Program" has been expanded to include a description of PCB California utility company management practices. The section includes a discussion of failure detection technology developed by the industry to prevent PCB spills.

3. Accelerated Phase-out of PCB-Containing Electrical Equipment

COMMENT:
EPA's August 25, 1982, PCB regulation established deadlines for phase-out of PCB-containing electrical equipment. SWRCB should endorse the federal program and not accelerate the removal of utility industry PCB capacitors. Lack of capacitor manufacturing and disposal capability makes accelerated phase-out infeasible. (SCE, SDG&E, PG&E).

RESPONSE:
We have revised the recommendation for accelerated phase-out to account for the identified constraints. First, the proposed acceleration of phase-out differentiates between large and small PCB capacitors and recommends early replacement of certain small capacitors, which EPA chose not to regulate. Second, early warning devices, rather than accelerated phase-out, are recommended for large PCB capacitors until they are replaced under the EPA-mandated deadline. Third, the recommendation for financial incentives to encourage industries to accelerate phase-out has been deleted. Instead, we propose that the state legislature finance replacement of certain small PCB capacitors in state facilities.

4. In-State Capability for PCB Destruction

COMMENT:
It is unclear how the recommended in-state capability to destroy PCB will be implemented. Public support for the establishment of a privately operated PCB destruction facility is recommended. (SCE)

RESPONSE:
We agree. The report recommends that privately operated PCB destruction facilities be established in California.

5. Occupational Exposure to PCBs

COMMENT:
The regulatory portions of the report omitted the Department of Industrial Relations regulatory program for worker protection from PCB exposure. (DIR)

RESPONSE:
The DIR regulatory program for PCBs has been incorporated in the report.

6. Economic Impact of the Proposed SWRCB, DHS and EPA Actions

COMMENT:
The report does not analyze the costs of proposed SWRCB, DHS, and EPA actions. (PG&E)

RESPONSE:
The proposed PCB water quality strategy requires a case-by-case assessment of feasible preventive and remedial measures. The precise cost of recommended SWRCB, DHS, and EPA regulatory actions cannot be estimated in advance.

7. August 25, 1982, EPA Regulation on PCBs

COMMENT:
The report should address the August 25, 1982 regulation adopted by EPA (PG&E)

RESPONSE:
We agree. The report has been revised to include summary and analysis of the final EPA rule.

8. Strict Liability on Users for Damages Caused by PCB Releases to the Environment.

COMMENT:
No additional legislation imposing strict liability on PCB users is needed because CERCLA already establishes this standard of liability and California is expected to follow the federal precedent. (SCE, PG&E)

RESPONSE:
The recommendation related to strict liability for damages to humans and the environment from PCBs has been deferred. The State Hazardous Waste Management Council is addressing the issue of strict liability in the broader context of all hazardous releases.

9. Isolation of PCB-Contaminated Material From Organics and Solvents in Landfills

COMMENT:
The recommendation which requires isolation of PCB-contaminated materials from organics and solvents in landfills is redundant. Federal regulations already require this precaution. (SCE)

RESPONSE:
We agree. The recommendation has been deleted.

10. Mammalian Toxicology and Human Health

COMMENT:
Only one study from "literally hundreds" of animal bioassays conducted on PCBs have shown PCBs to be animal carcinogens. (SCE)

RESPONSE:
Of the ten documented (published and unpublished) animal cancer bioassays, only three have been of sufficient duration to be considered major studies: Kimbrough (1975), Calandra (1976), and NCI (1978) (Drill et al., 1982). These ten studies are discussed in detail in Chapter 5, Section 5.4. Our interpretation is that the Kimbrough study (1975) of Aroclor 1260 was positive for liver cancer, and that the

NCI (1978) study of Aroclor 1254 was positive for stomach cancer. The validity of the Calandra study (1976), performed by Industricl Bio-test Laboratories (IBT) for the Monsanto Company, is in question. (See "The Murky World of Toxicity Testing," Science 220:1130-1133, June 10, 1983). Data from the IBT study, presented in Appendix C, Table C-4, show extremely high mortality for both control and experimental animals.

COMMENT:
The only proven adverse effects of PCBs on human health are the reversible conditions of chloracne and liver enzyme induction. (SCE)

RESPONSE:
The issue of the adequacy of human epidemiological studies is discussed in Chapter 5, Section 5.6. There are insufficient human data to conclude that PCBs will have no long term adverse effects on human health.

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