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POLYCHLORINATED BIPHENYL HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

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SUMMARY

This account summarizes recent technical literature on the environmental chemistry of polychlorinated biphenyls (PCBs); lists PCB background concentrations in fish, wildlife, and invertebrates; documents their toxic and sublethal properties; and reviews and provides recommendations for the protection of sensitive species of aquatic organisms and wildlife.

PCBs, a group of 209 synthetic halogenated aromatic hydrocarbons, have been used extensively in the electricity generating industry as insulating or cooling agents in transformers and capacitors. Due to human activities and the chemical characteristics of the products, PCBs are now distributed worldwide, with measurable concentrations reported in aquatic organisms and wildlife from North America, Europe, the United Kingdom, and the Atlantic and Pacific oceans. PCBs elicit a variety of biologic and toxic effects including death, birth defects, reproductive failure, liver damage, tumors, and a wasting syndrome. They are known to bioaccumulate and to biomagnify within the food chain. As a result of legislation, virtually all uses of PCBs and their manufacture have been prohibited in the United States since 1979. In general, the ban has been accompanied by declines in PCB residues in fishery and wildlife resources. However, the current environmental burden of PCBs in water, sediments, disposal sites, deployed transformers, and other PCB containers is now estimated at more than 82 million kg, much of it localized, and this continues to represent a potential hazard to associated fish and wildlife.

The toxicological properties of individual PCBs are influenced primarily by two factors: the partition coefficient based on solubility in N-octanol/water (Kow); and steric factors, resulting from different patterns of chlorine substitution. In general, PCB isomers with high Kow values, and high numbers of substituted chlorines in adjacent positions, constitute the greatest environmental concern. Unfortunately, basic chemical information is lacking on many isomers. Also, biological responses to individual isomers or mixtures vary widely, even among closely related taxonomic species. The issue is further confounded by the presence of toxic impurities, such as polychlorinated dibenzofurans, which may have been formed during the PCB manufacturing process, or result from product usage. At this time, total PCB residues give a more reliable measure of environmental PCB contamination than do measurements of any Aroclor or other commercial mixtures. In view of the demonstrated differential toxicities within the array of PCB congeners, it may finally become necessary to modify existing standards and criteria based on the more toxic PCBS.

For aquatic life, water concentrations of less than 0.014 ug total PCBs/1 (ppb) appear to afford a satisfactory degree of protection, although concentrations as low as 0.006 ug/l resulted in measurable accumulation by various species of filter-feeding shellfish. Among sensitive species of teleosts, total PCB residues (in ug/kg fresh weight) in excess of 500 in diets, 400 in whole body, and 300 in eggs were demonstrably harmful, and should be considered as presumptive evidence of significant PCB contamination. Among small mammals, the mink (*Mustela vison*) is one of the most susceptible species tested; dietary levels as low as 100 ug PCBs/kg fresh weight caused death and reproductive toxicity. A tolerable daily limit for mink has been estimated at less than 1.5 ug total PCBs/kg body weight. Tolerable daily PCB levels for rhesus monkey (*Macaca mulatta*) dog (*Canis* sp.), and rat (*Rattus* spp.) were 1.0, 2. , and 5.0 ug/kg body weight, respectively. For birds, total PCB levels (in ug/kg fresh weight) in excess of 3,000 in diet, 16,000 in egg, or 54,000 in brain were frequently associated with PCB poisoning.

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TABLES

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INTRODUCTION

As a result of human activity, polychlorinated biphenyls (PCBS) are now distributed worldwide, with measurable concentrations reported in polar bears (*Ursus martinus*) from the Canadian Arctic, birds and fish from the Great Lakes, wildlife in Europe, Scandinavia, and the United Kingdom, marine organisms of the Atlantic and Pacific Oceans, and up to 91% of the adult human population in the United States. Their presence in the organisms has been shown to cause reproductive failure, birth defects, skin lesions, tumors, liver disorders, and, among sensitive species, death. PCB toxicity is further enhanced by their ability to bioaccumulate and to biomagnify within the food chain due to extremely high liposolubility. These, and other, biological effects of PCBs have been extensively reviewed by Ayer (1976), Roberts et al. (1978), NAS (1979), EPA (1980), Pal et al. (1980), D'Itri and Kamrin (1983), Fleming et al. (1983), Ernst (1984), Stickel et al. (1984), Safe (1984), Simmons (1984), and Lucier and Hook (1985a,b).

PCBs, a group of synthetic halogenated aromatic hydrocarbons, were first prepared in 1881, and since 1930 have been in general use in products that include heat transfer agents, lubricants, dielectric agents, flame retardants, plasticizers, and waterproofing materials (Roberts et al. 1978). After 1971, they were used almost exclusively as insulating or cooling agents in closed electrical systems, such as transformers and capacitors (NAS 1979). Environmental contamination resulted from industrial discharges, from leaks of supposedly closed systems, from disposal of PCB wastes to municipal sewage treatment plants, landfills, and equipment dumps, and especially through atmospheric transport of incompletely incinerated PCBS. Long-range atmospheric transport of PCBs by wind, rain, and snow is now well documented (NAS 1979). PCBs tend to bond tightly to particulate matter, notably soils and sediments of lakes, estuaries, and rivers, where they may remain available for resuspension for at least 8 to 15 years (Swain 1983). The North Atlantic Ocean seems to be the dominant sink for PCBs, accounting for 50 to 80% of the PCBs in the environment, while freshwater sediment is a major continental reservoir (NAS 1979). Other significant reservoirs of mobile PCBs still exist along with even larger, currently immobile, pools. The latter includes those materials containing PCBs that are still in service, and those deposited in landfills and dumps.

Between 1930 and 1975, more than 630 million kg of PCBs were manufactured domestically (Safe 1984). At present, PCBs are not produced in the United States. PCB legislation under the Toxic Substances Control Act, effective January 1977, required the U.S. Environmental Protection Agency (EPA) to establish labeling and disposal requirements for PCBs, and mandated an eventual ban on the manufacture and processing of PCBs (Bremer 1983). Effective July 1979, the final PCB ban rule was implemented, which prohibits the manufacture, processing, distribution in commerce, and use of PCBs except in a totally enclosed system, unless specifically exempted by EPA (Bremer 1983). Although the ban has been in effect for about 6 years, the current environmental burden of PCBs in water, sediments, disposal sites, and in deployed transformers and other containers is sufficiently large, estimated at 82 million kg (D'Itri and Kamrin 1983), to present potentially significant hazards to fish and wildlife resources.

In this account, I summarize the recent technical literature documenting environmental hazards associated with PCBs, with emphasis on aquatic organisms and wildlife, and review quality criteria recommendations for the protection of sensitive species. This account is part of a continuing series of synoptic reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

ENVIRONMENTAL CHEMISTRY

PCBs are organic compounds commercially produced by chlorination of a biphenyl (BP) with anhydrous chlorine in the presence of iron filings or ferric chloride as the catalyst. The purified product is a complex mixture of chlorobiphenyls containing 18 to 79% chlorine; the precise composition depends on the conditions under which chlorination occurred (EPA 1980). Ten possible degrees of chlorination of the biphenyl molecule give rise to ten PCB congener groups: mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decachlorobiphenyl (Figure 1). Within any congener group, a number of positional isomers (discrete chemical compounds) are possible, depending on the number of chlorines in the molecule. For example, the tetrachloro- and pentachlorobiphenyl congener groups are composed of 30 and 46 possible isomers, respectively. Not all 209 possible isomers are likely to be formed during the manufacturing process. In general, the most common ones are those that have either an equal number of chlorine atoms on both rings, or a difference of only one

chlorine atom between rings (NAS 1979). Although chlorine substitution is favored at the ortho and para positions (Figure 1), the commercial products are complex mixtures of isomers and congeners with no apparent positional preference for halogen substitution (Safe 1984).

Recent advances in the identification and quantification of PCB isomers through mathematical and computer-assisted techniques (Dunn et al. 1984; Schwartz et al. 1984) will prove useful in data interpretation of metabolic fate studies. Unfortunately, the results of PCB analyses vary widely among cooperating laboratories. An interlaboratory comparison of spiked and unspiked samples of herring oil by 23 participating European and North American laboratories showed that calculated spike recoveries ranged from 23 to 136% (Musial and Uthe 1983). This was attributed to serious deficiencies in most steps in the analytical procedures. It seems that until these deficiencies are corrected, PCB analyses will have credibility from only a few selected laboratories.

Toxic materials as impurities in PCBs include polychlorinated dibenzofurans (PCDF) in some domestic and foreign mixtures at levels of 0.8 to 33 mg/kg. The concentrations of PCDFs in Great Lakes fish were related to the concentrations of PCBs (Stalling et al. 1983). Sometimes, PCBs used in electrical capacitors and transformers are converted under the action of heat or electrical arcing to form PCDFs including 2,3,7,8-tetrachlorobenzofuran and 2,3,4,7,8-pentachlorobenzofuran. Kanechlor 400 (Table 1), for example, with an initial PCDF content of 20 mg/kg, yielded fluid with a PCDF content of 4,975 to 11,765 mg/kg after use as a heat transfer fluid in a heat exchanger. These PCDFs were identified as the agents that poisoned more than one thousand humans in Japan in 1968 (EPA 1980; Lucier and Hook 1985a,b). Additional research is needed on PCDFs and other toxic impurities in PCBS.

PCBs are extremely stable compounds, and slow to chemically degrade under environmental conditions. Microbial degradation of PCBs depends on the degree of chlorination and the position of the chlorine atom on the biphenyl molecule; lower chlorinated BPs are readily transformed by bacteria, but not the higher chlorinated compounds (NAS 1979). Higher chlorobiphenyls, i.e., those with five or more chlorine atoms, were more persistent in the environment than those with three or less chlorine atoms; tetrachloro BPs were intermediate in persistence (EPA 1980). Passage of PCBs through activated sludge in sewage treatment plants for 48 hours resulted in 81% degradation for Aroclor 1221 (21% chlorine by weight), 26% for Aroclor 1242 (42% chlorine), and only 15% for Aroclor 1254 (54% chlorine) (NAS 1979). Because of their wide range of physical properties, their chemical stability, and their miscibility with organic compounds, PCBs have been used extensively as hydraulic fluids, plasticizers, adhesives, heat transfer fluids, wax extenders, dedusting agents, lubricants, flame retardants, and especially as dielectric fluids in capacitors and transformers. The current uses of PCBs in the United States have been severely curtailed and production was stopped during the 1970's, although significant quantities of PCBs are still used as dielectric fluids in older transformers and capacitors (Safe 1984).

Commercial PCB formulations are sold under a variety of trade names (Roberts et al. 1978; NAS 1979; EPA 1980; D'Itri and Kamrin 1983; Safe 1984). In the United States, Aroclor is the most familiar requested trademark, but PCBs have also been marketed as Chloretol, Dyknol, Inerteem, Noflamol, and Pyranol. In other countries, PCB formulations have been sold as Pyralene (France), Phenoclor (France), Kanechlor (Japan), Santotherm (Japan), Fenclor (Italy), Apirolio (Italy), Soval (USSR), Delor (Czechoslovakia), and Clophen (West Germany). Some formulations are similar; for example, Kanechlor 600, Phenoclor DP6, Clophen A60, and Aroclor 1260 all contain an average of 60% chlorine, although the former three preparations are composed of a mixture of hexachloro BPs, while Aroclor 1260 contains a variety of forms (Table 1; NAS 1979; EPA 1980).

Chlorination levels of PCB formulations differ markedly (Table 1). Among Aroclor formulations commercially produced by the Monsanto Corporation, Aroclor 1221 contained an average of 21% chlorine by weight and was a clear mobile oil. Aroclor 1254 contained 54% chlorine and was a yellow viscous liquid; 1260 contained 60% chlorine by weight and resembled a yellow sticky resin; and Aroclor 1268 was a white solid (Safe 1984). Aroclor 1254 contained less than 1% of biphenyl and monochloro BP, 0.5% dichloro BP, 1% trichloro BP, 21% tetrachloro BP, 48% pentachloro BP, 24% hexachloro BP, and 6% heptachloro BP (NAS 1979). Aroclor 1016 was similar to Aroclor 1242, with both containing an average of about 42% chlorine by weight, although 1242 contained 9% PCBs with five or more chlorines and 1016 only 5% (Roberts et al. 1978). In general, PCBs are relatively insoluble in water but freely soluble in nonpolar organic solvents and in biological lipids (EPA 1980). Monochlorobiphenyls are comparatively soluble in water (1,190-5,900 ug/1), but this decreases rapidly with increasing chlorination: dichlorobiphenyls, 80 to 1,880 ug/l; trichloro BP, about 8 ug/l; and tetrachloro BP, 3 to 170 ug/l (NAS 1979). The solubilities of various PCB isomers in water also decreased with increasing chlorine

content: 2, 4'-dichloro BP was soluble to 637 ug/l; 2,2',5-trichloro BP to 248 ug/l; 2,2',5,5'-tetrachloro BP to 26.5; 2,2',4,5,5'-pentachloro BP to 10.3; and 2,2',4,4',5,5'-hexachloro BP to 0.95 ug/l (Menzie 1978). Water solubilities of various Aroclor formulations, in ug/l, were 240 for 1242, 50 for 1248, 10 for 1254, and 3 for 1260 (NAS 1979). PCB octanol/water partition coefficients ranged between 10,000 and 20,000 for representative tri-, tetra-, and pentachlorobiphenyls. High partition coefficients with this biphasic solvent system correlate well with PCB biomagnification in fatty tissues of aquatic organisms (EPA 1980), and with incorporation into sediments (Fox et al. 1983). In fact, PCBs are strongly adsorbed on soils, sediments, and particulates in the environment, with levels usually highest in aquatic sediments containing microparticulates (EPA 1980; Duinker et al. 1983) and high organic or clay content (NAS 1979). Uptake of PCBs from contaminated marine sediments by benthic invertebrates is governed by processes that include ingestion of contaminated sediment particles, and exchange of PCBs directly from sediment particles (Larsson 1984). PCB-laden sediments of Raritan Bay, New Jersey, and lower New York Harbor were effective in producing population perturbations of benthic marine invertebrates when sediments contained high silt and clay composition; low silt and clay sediments were ineffective (Stainken 1984).

The number and position of the chlorine atoms on the biphenyl rings affects the biological properties of the compound. For example, PCBs with hydrogen atoms on two adjacent carbon atoms in one or both rings are more readily metabolized than those with hydrogen atoms adjacent only to chlorines, due to metabolic reactions involving arene oxide intermediates (EPA 1980). Furthermore, PCBs with chlorines in the 2 and 6 (ortho) positions are easily metabolized by humans while those with chlorines in the 4 and 4' (para) positions or 3, 4, or 3, 4, 5 positions on one or both rings tend to be biologically active and well-retained in tissues (EPA 1980).

In mammals, PCBs are readily absorbed through the gut, respiratory system, and skin. Initally, PCBs concentrate in liver, blood, and muscle; eventually, accumulations are highest in adipose tissue and skin. Phenolic derivatives or dihydrodiols are the major metabolites, but susceptibility of individual PCB isomers to metabolism is a function of the number of chlorine atoms present on the biphenyl rings and their arrangement. In general, most readily metabolized PCBs are also rapidly excreted in urine and bile. The highly chlorinated isomers are difficult to metabolize and accumulate almost indefinitely. PCBs can be transferred to young mammals either transplacentally or in breast milk. Retention of PCBs is highly species specific: nonhuman primates, for example, retained PCBs more efficiently than rodents (EPA 1980). PCB patterns, especially in warm-blooded animals, only vaguely resemble the mixture from which they originated (Hansen et al. 1983; Ernst 1984). Subtle differences between chlorobiphenyls are further amplified by differences among various animals to absorb, distribute, biotransform, and excrete individual PCB isomers (Hansen et al. 1983).

Biological activities of PCB isomers differ substantially. Among three symmetrical hexachlorobiphenyl (HCBP) isomers, 3,4,5,3',4',5'-HCBP was the most toxic in dietary lethal studies to domestic chickens (*Gallus* spp.), 2,4,5,21,4',5'-HCBP was least toxic, and 2,4,6,2',4',6'-HCBP intermediate in toxicity (Ayer 1976). A tetrachlorobiphenyl (3,4,3',4') was more than 1000 times more potent in producing effects in chicks than 2,4,5,2',4',5'-HCBP both as an hepatotoxin and as an inducer of cytochrome P mediated mixed function oxidases (Rifkind et al. 1984). The biological half-life of intravenously administered PCB formulations in liver of rat (*Rattus* spp.) increased with increasing chlorination: half-lives of 86 hours were determined for 4-monochlorobiphenyl, 99 hours for 4,4'-dichlorobiphenyl, 193 hours for 2,2',4,5,5'-pentachlorobiphenyl, and 1,308 hours for 2,2',4,4',5,5'-hexachlorobiphenyl (Menzie 1978).

PCBs are usually taken up by animals and stored in lipids under circumstances of increasing lipid content in organs. However, recent studies with marine fishes indicate that PCB components remain mobilizable from organs whose lipid contents increased (Boon et al. 1984). The degree of mobilization in codfish (*Gadus morhua*) and sole (*Solea solea*) appeared to be related to polar lipid components such as phospholipids and glycerols. In other aquatic species, the role of diet and tissue specific sites are important. The patterns of pentachloro BP and higher chlorinated BP (not present in seawater) were equal in marine clams, worms, and sediments, and strongly indicate that uptake was via the diet or from sediments (Duinker et al. 1983). In freshwater fishes, direct partitioning across the gill membrane of the blood:water interface controls PCB accumulation; however, dietary PCBs may significantly affect accumulation and exchange rates at the gill membrane (Rohrer et al. 1982).

Biological availability and uptake of individual PCBs from aqueous solution are influenced primarily by two factors: the partition coefficient (Kow) based on the solubilities of compounds in N-octanol/water; and steric

factors resulting from different patterns of chlorine substitution. Log Kow values for various isomers of Aroclors 1242, 1254, and 1260 are high, varying from 4.0 to 9.35, indicating high biological uptake potential. Steric effect coefficients are based on the number of chlorine atoms in the biphenyl molecule and their arrangement (Shaw and Connell 1982). For example, three chlorines in the ortho positions were assigned a steric effect coefficient of 0.3; four chlorines in the ortho postions 0.2; three or four adjacent chlorines on one ring 0.6, and on both rings 0.3; chlorines in the meta position on one ring 0.8, and on both rings 0.6. The product of log Kow and the steric effect coefficient seem to be directly related to bioaccumulation (Shaw and Connell 1982). Thus, maximum uptake was found with penta- and hexachlorobiphenyls predominant in Aroclor 1254, which have high values for log Kow and for steric effect coefficients. Comparatively less uptake was found with di-, tri-, and tetrachlorobiphenyls, typical of Aroclor 1242, which have lower values for log Kow, and with hepta- and octachlorobiphenyls, predominant in Aroclor 1260, which have lower steric effect coefficients. Structure-activity relationships, particularly for PCBS, can be applied to predictive models of contaminant cycling and biomagnification--and promises to become a fruitful research area.

Pal et al. (1980), in a review of PCBs in plant-soil systems, indicated three important research needs. First, many isomers of several PCB formulations have not been studied with respect to their biotransformation, volatilization, photodecomposition, adsorption, and transport. Second, highly chlorinated biphenyls, especially tetra-, penta-, and hexachlorobiphenyls, have not been evaluated for uptake by crops, volatilization, or distribution in soil layers. The relative build up and assimilation in soil-plant systems of hepta-, octa-, nona-, and decachlorobiphenyls have not been evaluated or studied. Finally, data are lacking on partition coefficients of PCBs between soil and water, water and atmosphere, atmosphere and plant populations, and during flow in food chains. A similar case can be made for most fish and wildlife species of current concern.

BACKGROUND CONCENTRATIONS

GENERAL

PCB concentrations have been determined for a variety of flora and fauna, and in certain nonbiological materials (Table 2). These selected data seem to represent current state-of-the-art analytical procedures for accurate and precise measurement of PCBs at detection limits of less than 0.01 ppm for most isomers. Characterization of Aroclor PCB residues in living and nonliving resources is difficult due to the chemical, physical, and metabolic transformations between product manufacture and detection in environmental samples. Schmitt et al. (1985) suggest that total PCBs in samples is a more reliable measure of environmental contamination than measurements of any commercial PCB mixtures such as Aroclor PCBS. Also, Brown et al. (1985) indicated that only a few PCB congeners (i.e., those most toxic) are appropriate for meaningful interpretation of PCB residues in biota.

There is general agreement that more samples from recent collections have detectable PCB residues, that absolute concentrations seem to be declining in some areas of known high PCB contamination, and that lower chlorinated PCBs--resembling Aroclor 1242--are disappearing. Nevertheless, many fish and wildlife species, including salmon, trout, turtles, eagles, herons, fish-eating birds, mink (*Mustela vison*), river otters (*Lutra canadensis*), and bats, all contain measurable, and in some cases potentially harmful, PCB residues, especially in adipose (fatty) tissues.

ALGAE AND TERRESTRIAL MACROPHYTES

In the Great Lakes, atmospheric deposition of PCBs is the most significant source of contamination. More than 90% of atmospheric PCBs are transported in the vapor phase and deposited by turbulent impaction. Deposition of PCBs is also associated with particulate matter, as well as direct partitioning from the vapor phase into the surface organic microlayer at the air-water interface, and these account for the remaining input (Rohrer et al. 1982). Once in the water column, the hydrophobic PCBs partition into the more apolar compartments of the ecosystem or are physically adsorbed on particulate matter. Transfer of PCBs on microparticulate materials and into phytoplankton is well documented, as is partitioning from aqueous solution into algal lipids (Rohrer et al. 1982). PCBs incorporated into phytoplankton exert inhibitory effects on photosynthesis and cell motility. In addition to direct toxic effects on algae, accumulated PCBs are readily introduced into the aquatic food chain (Rohrer et al. 1982).

Selected species of terrestrial plants collected in upstate New York showed decreases in total PCB concentrations of 42% between 1978 and 1980; however, levels in hay in 1979 varied between 0.08 and 0.10 ppm dry weight, or nearly half the Food and Drug Administration (FDA) limit of 0.2 ppm for PCBs in feeds for livestock (Buckley 1983). Plants grown on soils amended with lake sediments contaminated with Aroclors 1248, 1254, and 1260, accumulated PCBs (Sawhney and Hankin 1984). Uptake of Aroclors by beets (*Beta vulgaris*), turnips (*Brassica rapa*), and beans (*Phaseolus vulgaris*) was in the order 1248>1254>1260, indicating that lower chlorinated isomers (which are more soluble in water and more volatile) were more abundant in crop plants than higher chlorinated isomers. Based on these studies, continued monitoring of plants used as forage for livestock and wildlife appears necessary, especially in locations where soils are amended with PCB-contaminated aquatic sediments or sewage sludges.

INVERTEBRATES

Aquatic invertebrates assume an important role in the cycling of PCBs within and between ecosystems. Mysid crustaceans from Lake Michigan appear to have a low assimilation efficiency for PCBs and a high efficiency for fecal excretion of ingested PCBS; fecal pellets sink rapidly (80-170 m/day) and many probably reach the sediments intact before the onset of microbial degradation (Evans et al. 1982). By vertically migrating to the surface at night, mysids may transport PCBs into the upper regions; the PCBs will continue to recycle through the Lake Michigan ecosystem if the PCBs are excreted, egested, lost with molts, or if the mysid is consumed by fish. PCB levels of freshwater oligochaete worms from the Niagara River in New York State were positively correlated with sediment PCB levels (Fox et al. 1983). Uptake of PCBs from the sediment by chironomid (*Chironomus plumosus* - type) larvae was also directly related to the concentration of PCBs in the sediment (Larsson 1984). When larvae metamorphosed to adults, PCB compounds were concentrated and transferred from the aquatic to the terrestrial environment. In a field study near a Swedish sewage plant outfall, transport of PCBs by chironomids from the aquatic to the terrestrial environment was estimated at 20 ug of PCBs per m2 annually. Terrestrial predators that feed on emerging aquatic insects whose larval stage inhabits PCB-contaminated sediments may be exposed to PCBs (Larsson 1984).

FISH

Trace concentrations of the more persistent, more highly chlorinated PCBs are detected in fish from almost every major river in the United States (Schmitt et al. 1983, 1985). The ubiquity of PCB residues probably results from the dispersal of contaminated sediments, from atmospheric transport, and from patterns of continued use and disposal. Maximum concentrations in whole fish did not change appreciably at most locations in recent years; concentrations approaching 100 ppm fresh weight and 500 ppm in lipids were measured as recently as 1978 (Schmitt et al. 1983). Residues of PCBs in fish were more widely distributed than reported previously, but appeared to be declining in some areas of high concentration, and the less chlorinated PCBs (resembling Aroclor 1242) were disappearing. PCB concentrations in whole fish, collected Nationwide between 1976 and 1979, tended to be highest in the industrialized regions of the Northeast and the Midwest. Fish from the Hudson River, near Poughkeepsie, New York, contained 4 to 6X more PCBs than any of the other 108 stations analyzed; mean residue levels were 33.9 ppm fresh weight in 1976-1977 and 44.1 in 1978-1979 (Schmitt et al. 1983). However, data from 1982 (Brown et al. 1985) indicated a general decline in PCB content of fish from the Hudson River (although levels in some species remained substantially higher than the 5.0 ppm temporary tolerance level established by the U.S. Food and Drug Administration); this was attributed primarily to declines in the less chlorinated PCB congeners, especially Aroclor 1016. Accumulations in excess of 1.0 ppm PCBs fresh weight were reported in fish from stations in the Northeast, the Ohio River system, the Missouri River system, the Great Lakes, portions of the Mississippi River in Wisconsin, and from the Manoa Stream near Honolulu, Hawaii (Schmitt et al. 1983). In general, frequency of detectable PCB residues and total PCB residue levels in fish did not change significantly from 1974 to 1979. One exception to this pattern was the decline in residues resembling Aroclor 1242 from 14% of all samples in 1973, to 5% in 1974, to zero in 1976-1977; apparently, components most prevalent in the 1242 mixture are rapidly degraded. Furthermore, the continuing presence of residues resembling Aroclor 1248 suggest that relatively unaltered PCBs continue to enter the environment (Schmitt et al. 1983). The most recent Nationwide monitoring survey (Schmitt et al. 1985) demonstrated a significant downward trend in whole fish body burdens of PCBs in 1981-1982, with no significant increases at any station, confirming that residues were highest at stations in the industrialized regions of the Northeast, the Great Lakes, the Upper Mississippi River System, the Ohio River System, and the Cape

Fear River in North Carolina. Schmitt et al. (1985) suggested that total PCBs measure environmental PCB contamination more reliably than do measurements of any commercial mixtures such as Aroclor PCBs.

About 10% of coho salmon (*Oncorhynchus kisutch*) and 58% of chinook salmon (*0. tshawytscha*) collected in 1980 from the Great Lakes exceeded the current FDA action lever of 5.0 ppm total PCBs on a fresh weight basis. All chinook and about half the coho salmon exceeded the proposed FDA action level of 2.0 ppm total PCBs in fillets (Rohrer et al. 1982). PCBs detected in salmon from the Great Lakes in 1980 most closely resembled Aroclor 1254, although higher and lower chlorohomologues were present. PCBs resembling Aroclor 1260 were detected in only 10% of coho salmon from Lake Huron and may be the result of widespread historical use of Aroclor 1254 and more limited use of Aroclor 1260 (Rohrer et al. 1982). Marked variations in PCB content of selected tissues were evident; skinless fillets of Great Lakes salmon contained significantly lower PCB residues than skin-on fillets, with Aroclor 1254 concentrations 3.5 to 4X lower in the skinless fillets (Table 2). Also, a strong correlation was observed between the concentration of PCBs and DDE in Great Lakes salmon collected in 1980 (Rohrer et al. 1982), probably due to the chemicals' similar polarity and molecular size. The chemical interactions of PCBs with other chlorinated hydrocarbon contaminants, and their biological properties, are areas requiring additional research effort.

Although diet is a major route of PCB uptake in many species of fish, there are notable exceptions--even among closely related species. In lake trout (*Salvelinus namaycush*), for example, most (more than 99%) accumulated PCBs are from the diet and less than 1% from the medium. However, water is the major PCB uptake route in coho salmon (Rohrer et al. 1982). In the windowpane (*Scopthalmus aquosus*), a marine flounder from Long Island Sound, New York, stomach contents contained up to 0.45 ppm of PCBs fresh weight, suggesting a potentially large amount of dietary PCBs available to this species (Greig et al. 1983).

Elevated levels of PCBs (mostly Aroclor 1254) found in gonads of striped bass (*Morone saxatilis*), up to 1.4 ppm fresh weight and 2.3 on a lipid basis, may be associated with poor reproductive success of this species in Nova Scotia (Ray et al. 1984). This is similar to levels of 2.8 ppm PCBs in lipids of eggs of rainbow trout (*Salmo gairdneri*) that experienced heavy fry mortality (Rohrer et al. 1982), and to levels of 0.12 ppm PCBs on a fresh weight basis in gonads of flounder (*Platichthys flesus*) with inhibited reproduction (Ray et al. 1984). However, eggs of Atlantic salmon (*Salmo salar*) with 6.0 ppm PCBs in lipids hatched normally (Ray et al. 1984). It is clear that considerable interspecies differences exist in the responses of teleosts to PCB loadings.

Equilibrium levels of stable lipophilic contaminants in fish are directly proportional to the ambient water concentration of the chemical. Atmospheric deposition and high sediment contamination have also been implicated as major sources of PCB contamination (Rohrer et al. 1982). Lowest PCB levels in Great Lakes salmon were at stations with high flushing and high sedimentation rates (Rohrer et al. 1982). PCBs were detectable at low concentrations in brook trout (*Salvelinus fontinalis*) from remote New England lakes, tending to confirm that PCBS, and other recorded contaminants, reached the lakes by atmospheric deposition (Haines 1983).

REPTILES

Water snakes (*Nerodia* spp.) collected in Louisiana reflected PCB levels similar to those of CB residues in their prey species, primarily fresh- and brackish-water teleosts. PCBs in water snakes were detected in 95% of all fat samples and 52% of liver and muscle tissues; Aroclor 1260 accounted for most of the PCBs (Sabourin et al. 1984). Snapping turtles (*Chelydra serpentia*) are capable of storing high concentrations of PCBs in their fat without any apparent detrimental effects, and may be useful as biological indicators for lipophilic substances, including PCBs (Olafsson et al. 1983). Sea turtles contain relatively lower levels of PCBs than snapping turtles (Table 2). PCB concentrations were higher in saltwater loggerhead turtles (*Caretta caretta*), an omnivore, than green turtles (*Chelonia mydas*), which are vegetarians, again demonstrating that diet is an important route of PCB transfer (McKim and Johnson 1983).

BIRDS

In general, most bird tissues and eggs collected had measurable concentrations of PCBS, and the frequency of occurrence appears to be increasing. In 1976, 21% of European starlings (*Sturnus vulgaris*) collected Nationwide had detectable PCBS; in 1979 it was 83% (Cain and Bunck 1983). For mallards (*Anas platyrhynchos*), PCBs were detected in 39% of wing tissues in 1976-1977, and 95% In 1979-1980 (Fleming et

al. 1983). PCBs were detected in all of the eggs of six species of South African seabirds and in the majority of eggs from a seventh species in 1981-1983 (de Kock and Randall 1984); in 50 to 55% of eggs of the black-crowned night-heron (*Nycticorax nycticorax*) from Colorado, Wyoming, Washington, and Nevada taken in 1978-1980 (McEwen et al. 1984; Henny et al. 1984); in all Norwegian seabird fledglings in 1976-1977 (Fimreite and Bjerk 1983); in 77% of eggs of the black skimmer (*Rynchops nigra*) from Texas in 1981 (White et al. 1984); in 85 to 100% of all game bird tissues examined in West Germany during 1982 (Brunn et al. 1985); and in 99% of the eggs of the endangered American bald eagle (*Haliaeetus leucocephalus*), including 89% occurrence in breeding areas (Wiemeyer et al. 1984).

Populations of double-crested cormorants (*Phalacrocorax auritus*) from Lake Huron are now recovering rapidly, presumably due to a decrease in CB and other contaminant residues in eggs (Weseloh et al. 1983). In the late 1960's and early 1970's, cormorants nesting on the Great Lakes, and in particular Lake Huron, had eggs that were more highly contaminated with PCBS, DDE, and mercury, than did cormorant eggs from anywhere else in Canada. Concomitantly, egg survival, reproductive success, and colony size were either dangerously low or decreasing. In .1972, colonies were small, they showed high egg breakage and egg loss (95%), and nearly total reproductive failure (less than 0.11 young/nest). Eggshells were about 24% thinner than normal. Levels of DDE (14.5 ppm fresh weight) and PCBs (23.8) in eggs collected in 1972 were higher than other Canadian cormorant populations (Weseloh et al. 1983).

In the past, PCB residues in birds tended to be higher in areas of local contamination and heavy industrial use or discharge. But with the current stringent restrictions on PCB use, geographical distinctions are not as clear (Custer et al. 1983a; McLane et al. 1984), although residues in birds from industrialized areas still remain comparatively high (Fleming et al. 1983; White et al. 1984). For example, birds collected near the Sheboygan River, Wisconsin, contained PCB residues that would be considered harmful to some species tested in the laboratory (Table 2). The main source of PCBs at that location was a diecasting plant. Granular oil absorbent material behind the plant contained up to 120,000 ppm of PCBS; runoff and seepage from rain and floodwaters carried PCBs into the river, subsequently contaminating fish, especially carp (*Cyprinus carpio*). Whole fresh carp from the Sheboygan River contained more than 155 ppm of PCBs (as quoted in Heinz et al. 1984), suggesting that fish-eating birds may be especially at risk.

In 1981 and 1982, duck hunters in New York and New Jersey were cautioned about the consumption of wild waterfowl. At the time, waterfowl from the Hudson and Niagara rivers contained PCBs in excess of tolerances established by FDA for poultry (more than 5 ppm fresh weight), although PCB concentrations found in these waterfowl were below the levels associated with reproductive impairment or decreased survival of birds (Fleming et al. 1983).

Residues of PCBs in birds are modified by numerous biotic factors including fat content, tissue specificity, sex, and developmental stage. PCB residues in birds may also reflect levels due to aerosol transport of these compounds (Weber 1983), as well as water transport (Norheim and Kjos-Hanssen 1984). Highest PCB residues were in birds with low fat content and in poor condition on capture (Falandysz and Szefer 1984). Sexual differences in PCB content are pronounced due to the female's ability to shed a significant portion of the PCB burden into eggs (Lemmetyinen and Rantamaki 1980). Developmental stage is an important consideration when collecting bird samples for PCB analysis (Lemetyinen and Rantamaki 1980); for example, PCB level is reduced from egg to fledgling (Fimreite and Bjerk 1983). Finally, residues in brain appear to be good indicators of PCB stress in birds (Stickel et al. 1984). Concentrations greater than 300 ppm of PCBs in brain (fresh weight) were consistently recorded in dead or dying ring-billed gulls (*Larus delawarensis*) and ring-necked pheasants (*Phasianus colchicus*) poisoned by PCBs, as quoted in Heinz et al. (1984).

MAMMALS

Among mammals, the mink is especially sensitive to PCBS; only 0.64 ppm PCBs in their diets caused reproductive failure, and 1.0 ppm caused death (as quoted in Fleming et al. 1983). In western Maryland and in northern Oregon during 1978-1979 (areas with no recognized large-scale PCB pollution), levels of PCBs in livers of some wild mink were comparable to those reported for female ranch mink that failed to reproduce after eating a diet contaminated with 0.64 ppm of PCBs for 160 days (O'Shea et al. 1981; Henny et al. 1981). Some fish from the Oregon collection sites contained PCB residues (0.24-2.8 ppm fresh weight) that were equivalent to, or higher, than dietary levels fed to mink under controlled conditions (Henny et al. 1981).

PCB concentrations in river otters from the Columbia River, Oregon, during 1978 and 1979, were substantially higher than those reported in the same species from Alabama. The significance of this is not clear, but declining harvests of Columbia River otter populations (as opposed to upward trends elsewhere in Oregon) suggest that PCBs may be a contributory agent (Henny et al. 1981).

In little brown bats (*Myotis lucifugus*) and big brown bats (*Eptesicus fuscus*) from Maryland, pups found dead at birth had significantly more Aroclor than live littermates; moreover, females with elevated Aroclor 1260 residues tended to produce litters with a greater frequency of stillbirths (Clark and Lamont 1976; Clark and Krynitsky 1978).

PCBs were recently detected in 13 of 26 Florida manatees (*Trichechus manatus*), an endangered species. All individuals with detectable PCB residues were recovered from locations in the relatively urbanized areas of northeastern Florida, primarily in the lower St. Johns River and Brevard County (O'Shea et al. 1984).

In harbor seals (*Phoca vitulina*), PCB concentrations decrease with increasing blubber thickness (Van Der Zande and De Ruiter 1983). As expected, lower chlorinated PCBs were eliminated more rapidly from blubber lipids than higher chlorinated PCBS. For harbor seals in particular, blubber PCB residues contained a small fraction of lower chlorinated components when compared to PCB residues in fish that they eat (Van Der Zande and De Ruiter 1983). PCBs in adult Weddell seals (*Leptonychotes weddelli*) were predominantly penta- and hexachlorobiphenyls; a slightly higher amount of lower chlorinated biphenyls were found in newborns (Hidaka et al. 1983), suggesting that lower chlorinated biphenyls are more easily transferred from mother to pup through transplacental action than higher chlorinated biphenyls. Weddell seals contain low concentrations of PCBs in blubber compared to 10 other pinniped species and small cetaceans. This probably reflects the low PCB burdens in their diets attributed, in part, to the heavy ice and snow cover during much of the year that prevents atmospheric deposition of PCBs from entering the water and contaminating their diet (Hidaka et al. 1983).

INTEGRATED STUDIES

Biomagnification of PCBs through marine food chains in an Australian estuary became increasingly important with upper level carnivores such as gulls and pelicans, but was relatively unimportant at lower trophic levels (Shaw and Connell 1982). A similar situation was observed in Central Puget Sound in Washington in 1979 (Malins et al. 1980). PCB body burdens in marine organisms, especially benthic organisms, were directly related to the log of PCB concentration in sediments (Shaw and Connell 1982). Furthermore, PCBs were found in every tissue analyzed from fish and invertebrates in Puget Sound in 1979 (Malins et al. 1980). High PCB levels, especially in sediments, have been recorded from highly industrialized areas worldwide (Baldi et al. 1983).

PCBs used in the manufacture of electrical equipment at two facilities located on the upper Hudson River in Washington County, New York, have resulted in severe PCB contamination of the sediments--reportedly more than 100X greater than any other major river system (Sloan et al. 1983). Edible portions of fish collected from this area often exceeded the FDA tolerance level of 5.0 ppm fresh weight (Sloan et al. 1983). Carcich and Tofflemire (1982) estimated that more than 272,000 kg (perhaps as much as 603,000 kg) of PCBs were discharged into the River, and that most still resides in sediments north of Troy, New York. In the Hudson River, levels of higher chlorinated PCB components typical of Aroclor 1254 have stabilized during 1977 to 1981, suggesting that a dynamic equilibrium has been reached, and that these compounds may continue to exist at concentrations close to present levels (Sloan et al. 1983). Removal of PCB contaminated sediments by dredging from the Hudson River is now being studied, with greatest effort confined to the most highly contaminated 8% of the river bed. Disposal, by total containment and isolation of contaminated materials within a land burial facility, is one of the more preferred options (Carcich and Tofflemire 1982).

TOXICITY

AQUATIC ORGANISMS

LC-50 values of sensitive species of freshwater and marine organisms subjected to various Aroclor PCBs varied from 0.1 to 10.0 ug/l during exposure of 7 to 38 days (Table 3). In general, toxicity increased with increasing exposure, crustaceans and younger developmental stages were the most sensitive groups tested, and lower chlorinated biphenyls were more toxic than higher chlorinated biphenyls. In most toxicity tests, mortality patterns in PCB-exposed fish did not stabilize within 30 days (Johnson and Finley 1980).

BIRDS

As a group, birds were more resistant to acutely toxic effects of PCBs than mammals (Table 4). LD-50s for various species of birds varied from 604 to more than 6,000 mg Aroclor/kg diet, and for mallards more than 2,000 mg/kg body weight administered orally (Table 4). Signs of PCB poisoning among birds included morbidity, tremors (which may become continuous),. beak pointed upwards, and muscular incoordination. At necropsy, the liver frequently contained hemorrhagic areas and the gastrointestinal tract was filled with blackish fluid (Stickel et al. 1984).

For all avian species, PCB residues of 310 mg/kg fresh weight or higher in brain were associated with an increased likelihood of death from PCB poisoning. Residues in brains of starlings, red-winged blackbirds (*Agelaius phoeniceus*), common grackles (*Quiscalus quiscula*), and brown-headed cowbirds (*Molothrus ater*), that died after eating diets containing 1,500 mg Aroclor 1254/kg, varied from 349 to 763 mg/kg; surviving birds, at the 50% mortality point, contained 54 to 301 mg/kg (Stickel et al. 1984). Similar results are reported for ringed turtle-doves (*Streptopelia risoria*) after 105 days fed a 10 mg Aroclor 1254/kg diet, chickens fed Phenoclor, Clophen, and Aroclor PCBS, and finches and cormorants fed Aroclor 1254 (as quoted in Stickel et al. 1984). In the field, PCBs were the probable cause of mortality of many ring-billed gulls that died in southern Ontario in late summer and early autumn of 1969 and 1973; PCB residues in brains were 310 to 1,110 mg/kg fresh weight in 67% of the samples analyzed, and 200 to 310 in the remainder (as quoted in Stickel et al. 1984).

MAMMALS

The mink is the most sensitive wildlife species tested for which data are available (Table 4). Diets containing 6.7 to 8.6 mg Aroclor PCBs/kg fresh weight killed 50% of the mink in 9 months; single dosages administered orally produced LD-50 values of 750 to 4,000 mg/kg body weight, those administered intraperitoneally produced LD-50s between 500 and 2,250 mg/kg body weight (Table 4). Recent data (Aulerich et al. 1985) indicate that certain hexachlorobiphenyls (HCBP), such as 3,4,5,3',4',5' HCBP, are extremely toxic to mink; concentrations as low as 0.1 mg/kg fresh weight diet produced an LD-50 in 3 months, and completely inhibited reproduction in survivors. However, other HCBPS, such as 2,4,5,2',4',5' HCBP and 2,3,6,2',3',6' HCBP, were not fatal to mink under similar conditions, and did not produce adverse reproductive effects (Aulerich et al. 1985). Additional research is needed on the toxicodynamics of PCB congeners. Signs of PCB poisoning in mink included anorexia, bloody stools, fatty liver, kidney degeneration, and hemorrhagic gastric ulcers (Aulerich and Ringer 1977). The reasons for mink sensitivity to PCBs are unknown, but large variations in sensitivity to PCBs among species are common, even among those closely-related taxonomically. The European ferret (*Mustela putorius furo*), for example, is at least three times more resistant than mink to Aroclor 1242 (Table 4).

Rats fed diets containing 1,000 mg of Aroclor 1254/kg diet all died in 53 days; mortality started at day 28 (Hudson et al. 1984). These, and other feeding studies, suggest that a total intake of about 500 to 2,000 mg of Aroclor 1254 per kg body weight is the lethal level in rats for dietary exposures of 1 to 7 weeks. Prior to death, rats showed lack of muscular coordination, eyelid drooping, blanched retinas, morbidity, nasal secretions, and (with Aroclor 1268) a reddish exudate from their eyes (Hudson et al. 1984). Some rats died at 100 mg Aroclor 1254/kg body weight administered orally, or about 1/5 of the dietary LD-50 (Hudson et al. 1984).

SUBLETHAL EFFECTS

GENERAL

PCBs elicit a variety of biologic and toxic effects including skin lesions, a wasting syndrome, immunotoxicity, reproductive toxicity, genotoxic and epigenetic effects, hepatomegaly and related liver damage, and the induction of hepatic and extrahepatic drug-metabolizing enzymes. PCB accumulations from the diet and from other sources are high, and retention is lengthy in fatty tissues. Interspecies differences in sensitivity to PCBs are large, even between species that are closely related taxonomically.

TERRESTRIAL MACROPHYTES

A 5X increase in somatic mutations was observed in ostrich ferns (*Matteuccia struthiopteris*) growing near the Housatonic River in Pittsfield, Massachusetts, on sediments containing mean PCB residues of 26 mg/kg (mostly as Aroclor 1254), when compared to ostrich ferns from control areas (Klekowski 1982). No attempt was

made to duplicate these observations under controlled conditions, and no evidence of genetic damage to other plants of the PCB-contaminated area was found (Klekowski 1982).

AQUATIC ORGANISMS

Bioconcentration of Aroclor 1254 from the medium by selected species of freshwater and marine organisms varied from 60 to 340,000X (Table 5). Various species of algae also concentrate PCBs over water levels by 10,000 to 100,000X (Ernst 1984). Oysters (*Crassostrea virginica*) held for 65 days in seawater solutions containing 0.0055 to 0.06 ug/l of di-, tri-, tetra-, penta-, or hexachlorobiphenyls had bioconcentration factors of 1,200 to 4,800X; uptake was lowest for dichlorobiphenyls and became progressively higher with increasing chlorination of PCB congeners (Ernst 1984). Similar results were recorded for *Daphnia magna* exposed to five different C-14 labeled PCBs for 24 hours. BCFs varied from 473 to 11,232; formulations lowest in water solubilty (highest chlorination) were accumulated most readily (Zhang et al. 1983). For all PCBS, BCFs were generally higher with increasing exposure period, with increasing PCB concentration, and with increasing chlorination of PCB congeners (NAS 1979; Johnson and Finley 1980; EPA 1980).

Since PCBs are highly lipophilic, greatest concentrations were expected, and occurred, in fatty tissues. For example, lipid content in muscle of brown trout (*Salmo trutta*) was the best correlate of PCB concentration in muscle (Spigarelli et al. 1983). Differences in PCB content of tissue lipids were negatively correlated with phospholipid fractions in total lipid extracts; the higher the phospholipid fraction, the lower the PCB content in organ lipids. This has been verified experimentally in codfish and other species of marine teleosts (Boon et al. 1984), and probably should be explored in greater detail for other trophic levels. Other factors known to modify PCB accumulations in aquatic biota include: temperature magnitude and variation for trout (Spigarelli et al. 1983); time of incubation and age of larvae for mosquitos (Gooch and Hamdy 1983); presence of mineral oils on PCB-contaminated substrates for chironomid larvae (Meier and Rediske 1984); and diet for teleosts (Pizza and O'Connor 1983; Spigarelli et al. 1983; O'Connor and Pizza 1984). Of these, diet contributes most of the total PCB body burdens of upper level carnivores. For example, diet accounted for 90% of the total PCB body burden in brown trout (Spigarelli et al. 1983), and 51 to 83% in striped bass (Pizza and O'Connor 1983). PCB body burdens in striped bass were lower in winter during nonfeeding periods, and lower when fish migrated to a new area where dietary PCB levels were lower (O'Connor and Pizza 1984). Prey species of carnivores accumulate PCBs through contaminated sediments. PCB transfer through aquatic ecosystems has been reported in the Great Lakes using a sediment-lake trout model (Jensen 1984), and in New York Harbor from contaminated sediments to clams, shrimp, and especially nereid worms (Rubenstein et al. 1983).

Depuration of accumulated PCBs is slow, and slower yet at reduced temperatures (Zhang et al. 1983). Larvae of codfish exposed to PCBs as eggs showed no elimination after 12 days. Uptake of PCBs by yolk sac larvae was higher than in eggs; 60% of the PCBs remained after 15 days (Solbakken et al. 1984b). Larvae of chironomids (*Glyptotendipes barbipes*), held for 24 days in substrates containing 1,000 mg Aroclor 1242/kg, contained 18.0 mg Aroclor 1242/kg fresh weight; 7 days later, larvae still retained 97.8% of the total (Meier and Rediske 1984). Tissue samples of a Bermuda brain coral (*Diploria strigosa*) taken 9 months after initial exposure for 24 hours to radiolabeled 2,4,5,2',4',5'-hexachlorobiphenyl contained 84% of the original radioactivity (Solbakken et al. 1984a). Rainbow trout fed 1,150 mg Aroclor 1254/kg body weight contained high residues in various tissues 38 days posttreatment: 70 mg/kg in muscle on a fresh weight basis, 33 in liver, and 6 in gill filament (Kiessling et al. 1983). Factors affecting elimination of Aroclor 1254 by marine crustacean copepods (*Acartia tonsa*) include diet and reproductive state (McManus et al. 1983). Copepods fed during depuration eliminated PCBs more rapidly than unfed copepods. PCBs in copepod eggs were up to 4X the concentration in females producing them. Females eliminated PCBs twice as rapidly as males, indicating that egg production is an important route for PCB elimination. Fecal pellets were the most significant elimination route, but levels in fecal pellets from both sexes decreased over time suggesting a multiphasic elimination pattern. In fish, egg maturation and spawning result in a significant reduction in the body burden of persistent PCBs such as 2,5,2',5'-tetrachloro BP (Vodicnik and Peterson 1985). The percent lipid, and the percent of total lipid deposited in their eggs, markedly influences PCB transfer from fish to eggs (Niimi 1983). Mean percent PCB residue levels translocated to eggs in five species of Great Lakes fish varied from a low of 5.4 in rainbow trout to a high of 29.3 in yellow perch, and these (and intermediate) percentages reflected lipid percent levels transferred (Niimi 1983). Structural features in PCB forms and congeners cause different elimination rates between individual components in fish, resulting in differences in PCB composition between tissues and the source of uptake (Boon et al. 1984).

Decreased growth of aquatic organisms during exposure to PCBs is well documented. Concentrations as low as 0.1 ug/l of Aroclor 1254 produced growth reductions in marine diatoms and a freshwater alga (*Scenedesmus quadricauda*), and altered the population structure of phytoplankton communities (EPA 1980). Among sensitive species of freshwater algae treated with Aroclor 1242, including *S. quadricauda*, disruption of internal chloroplast membranes and failure of cytokinesis were the major changes observed (Mahanty et al. 1983). Marine algae exhibited greater-than-expected reductions in photosynthesis when stressed with mixtures of PCBs and DDE (Ernst 1984), and demonstrates the importance of toxicant evaluation of complex mixtures containing PCBs. Decreased shell growth of oysters was reported in acute tests with Aroclor 1016 at 10.1 ug/l, with Aroclor 1248 at 17.0 ug/l, with Aroclor 1254 at 14.0 ug/l, and with Aroclor 1260 at 60.0 ug/l (EPA 1980); similar results were reported for shrimp (Ernst 1984). Fry of brook trout held for 48 days at 1.5 ug Aroclor 1254/1 also showed decreased growth (Johnson and Finley 1980).

Reproductive toxicity of PCBs is reported for Baltic flounder (*Platichthys flesus*) when ovaries exceeded 0.12 mg of PCBs/kg fresh weight, and for cyprinid minnows (*Phoxinus phoxinus*) when gonads contained more than 24 mg PCBs/kg fresh weight; these are the threshold values beyond which reduced survival of developing eggs can be expected in those species Ernst 1984). Gonadal levels of 24 mg/kg in minnows were obtained with diets of 20 mg Clophen A-50/kg for 40 days, followed by 260 days on untreated diets (Ernst 1984). Rainbow trout with whole body residues of 0.4 mg Aroclor 1242/kg fresh weight produced eggs with low survival, and numerous (70%) fry deformities (EPA 1980). Rainbow trout eggs with 0.33 mg Aroclor 1254/kg fresh weight incurred 10 to 28% mortality prehatch, and numerous posthatch deformities (Niimi 1983). Eggs and fry of Atlantic salmon with PCB contents of 0.6 to 1.9 mg/kg fresh weight, or 14.4-34.0 mg/kg lipid weight, experienced 46 to 100% mortality (Niimi 1983). Embryos of sheepshead minnow (*Cyprinodon variegatus*) containing 7 mg Aroclor 1254/kg fresh weight had low survival; these values were effected through exposure of parent fish to 0.14 ug Aroclor 1254/1 for 28 days (EPA 1980). Brook trout experienced complete reproductive failure during exposure to 200 ug Aroclor 1254/1 for 71 weeks; the no effect level was 0.94 ug/l (EPA 1980). Eggs of the sea urchin, *Arbacia punctulata*, exposed for one hour to 0.5 mg Aroclor 1254/1 prior to fertilization showed reduced fertilization success and lowered survival; eggs were markedly more resistant to PCBs at the time of insemination and afterwards (Aroclor 1254) on early development and mortality in Arbacia eggs. (Adams 1983).

Mutagenic properties of Aroclor 1221 and 4-monochlorobiphenyl to bacteria were indicated by positive Ames tests, and Aroclor 1260 and Kanechlor 500 were demonstrably carcinogenic to mice and rats (NAS 1979). Unexpectedly, Aroclor 1254 prevented carcinogenesis and mutagenesis in rainbow trout. Trout fed 100 ppm dietary Aroclor 1254 for 3 months were significantly more resistant to liver carcinomas (induced by dietary aflatoxins) when PCBs were prefed prior to carcinogenic insult (Shelton et al. 1983). At the time of aflatoxin administration, trout contained 594 mg/kg PCBs in fat which declined rapidly over the next 12 months to 3.9 mg/kg. Aflatoxin-induced mutagenesis in trout liver cells was also significantly inhibited (67%) by Aroclor 1254 under similar conditions (Shelton et al. 1983).

Histopathology was reported in sensitive marine teleosts following exposure for 2 weeks to PCB concentrations of 0.5 ug/l; similar damage effects were recorded in oysters held for 24 weeks in 5 ug/l of Aroclor 1254, but not for 30 weeks in 1 ug/l (Ernst 1984).

A wide variety of biochemical perturbations were recorded among teleosts stressed by PCBS. The primary biochemical effect of PCBs is to induce hepatic mixed function oxidase systems, thus increasing the organism's capacity to biotransform or to detoxify xenobiotic chemicals and endogenous steroids (Melancon and Lech 1983; Shelton et al. 1983). Coho salmon injected intraperitoneally with 50 to 100 ug Aroclor 1254/kg body weight just prior to smoltification contained elevated levels of PCBs in liver (500 to 1,200 ppb) 2 weeks after injection when compared to controls (25 to 45 ppb), showed depressed gill Na-K ATPase and plasma thyroxin levels, and experienced great difficulty in adapting to seawater (Folmar et al. 1982). Biochemical indicators suggested that tissue accumulations of PCBs (at concentrations that clearly could be derived through a contaminated diet or from water column exposure) delayed events preparatory to, and involved in, saltwater adaptation in coho salmon. Mixtures of Aroclor 1242 and 1254 fed to rainbow trout and coho salmon at dietary concentrations of 500 ppm PCBs for 7 to 10 weeks produced inhibited growth, enlarged livers, elevated muscle water content, and lowered muscle lipid content (Leatherland and Sonstegard 1981). Salmon showed disrupted calcium and magnesium metabolism in blood, muscle, and skeleton. After about a week on PCB diets, both trout and salmon showed signs of poor muscle coordination and tetany, accompanied by lateral or ventral caudal flexion (scoliosis or lordosis). Brown trout fed diets containing 10 ppm of Clophen A-50 for 43 days were

anemic, hyperglycemic, and showed altered cholesterol metabolism (EPA 1980). Brook trout held in Aroclor 1254 solutions of more than 0.43 ug/l for 48 days had decreased concentrations of hydroxyproline in collagen isolated from the backbone (Johnson and Finley 1980). However, Aroclor 1254 did not markedly affect adrenaline response in gills of rainbow trout, or glycogen storage in muscle (Kiessling et al. 1983).

Maximum Acceptable Toxicant Concentration (MATC) values bracket the "no effect," and "measurable effect" levels, and are based on chronic exposure, and variables such as growth, reproduction, and metabolic upset. MATC values for selected species of aquatic organisms and Aroclor PCBS, with one exception, varied from 0.1 to 5.4 ug/l for the no effect level, and from 0.4 to 15.0 ug/l for measurable effects (Table 6). The exception was larvae of the sheepshead minnow (which was especially sensitive) with an MATC of 0.06 to 0.16 ug/l for Aroclor 1254 (Table 6).

BIRDS

Among sensitive avian species, PCBs disrupt normal patterns of growth, reproduction, metabolism, and behavior. In general, PCB accumulation is rapid and depuration is lengthy. Diet is an important route of PCB accumulation. Concentrations in liver (mg/kg fresh weight) were highest (900) in birds that fed on fish, followed by species that feed on small birds and mammals (50), worms and insects (0.65), and lowest (0.2) in herbivorous species (NAS 1979).

Delayed reproductive impairment was documented in ringed turtle-doves given 10 ppm of dietary Aroclor 1254 for 3 months; residues in the fat of adults was 736 ppm, and in their eggs 16 ppm fresh weight (as quoted in Heinz et al. 1984). Hatchability of ringed turtle-dove eggs from the first clutch was not reduced by consumption of Aroclor by the adults. However, 6 months later, the hatchability of the second clutch (accompanied by abnormal incubation behavior) was reduced to 10% of controls; embryos also contained chromosomal aberrations (Peakall et al. 1972). Mourning doves (*Zanaida macroura carolinensis*) given dietary Aroclor 1254 for 6 weeks at 0, 16, or ppm were observed for courtship behavior and reproductive effort during days 14 to 44 posttreatment (Tori and Peterle 1983). Doves fed 10 ppm spent twice as much time as controls in the courtship phase (billing, cooing, nest site selection), but only 50% of these pairs completed the courtship phase and progressed into nest building and incubation; of those that nested, nest initiation was significantly delayed. Doves fed 40 ppm spent the 30 days posttreatment in courtship without nesting; most of this group, especially females, did not respond normally to the presence of a mate. Tori and Peterle (1983) suggested that the disrupted reproductive behavior observed in PCB-treated doves was due to reduced estrogen and androgen levels. PCBs have been shown to degrade estrogens and androgens by increasing the activity of hepatic microsomal enzymes (as quoted in Tori and Peterle 1983). Hatchability of chicken eggs was reduced when hens were fed diets containing 20 ppm of various Aroclor PCBs (1232, 1242, 1248, or 1254). PCB residues in samples of fat from treated hens varied from 45 to 125 ppm, and in their eggs from 3 to 14 ppm (as quoted in Heinz et al. 1984). Reproductive impairment in chickens was recorded at Aroclor dietary levels as low as 5 ppm; effects at the 2 ppm level were not significant (Heinz et al. 1984). American kestrels (*Falco sparverius*) given 33 ppm of dietary Aroclor 1254 for 62 to 69 days (equivalent to 9 to 10 mg/kg body weight/daily), showed a significant decline in sperm concentration, but no compensatory increase in semen volume (Bird et al. 1983). PCB residues in treated kestrels, in ppm lipid weight, were 107 in muscle and 128 in testes; for controls, these values were 0.4 in muscle and 1.0 in testes. These results suggest that migratory flesh-eating birds feeding on a PCB-contaminated food chain might consume enough toxicant to alter their semen quality in that breeding season; when coupled with altered courtship, this could reduce the fertility of the eggs and reproductive fitness of the individual (Bird et al. 1983).

Among comparatively resistant species of birds, no significant reproductive effects were observed during long-term exposures at high Aroclor 1254 feeding levels in Japanese quail *Coturnix coturnix* (50 ppm in diets), northern bobwhites *Colinus virginianus* (50 ppm) 979), and mallards (25 ppm) (Custer and Heinz 1980). Screech owls (*Otus asio*), given 3 ppm of Aroclor 1248 in their diets for two breeding seasons, laid eggs containing 3.9 to 17.8 mg PCBs/kg fresh weight compared to control values of 0.0 to 0.6; however, reproductive variables, including eggs per clutch, hatchability, chick malformations, survival, and eggshell thickness, were not affected (McLane and Hughes 1980).

For most avian species, a reduction in eggshell thickness of 15 to 20% is suggested as a critical value beyond which population numbers will decline (Nygard 1983). Pheasants fed 50 mg of Aroclor 1254 weekly produced fewer eggs, but an effect on eggshell thinning was not apparent before other effects became obvious

(Roberts et al. 1978). However, eggshell thickness of the peregrine falcon (*Falco peregrinus*) from Norway declined 85% between 1854 and 1976; addled eggs containing dead embryos collected in 1976 had 724 ppm of PCBs in lipids, and up to 110 ppm on a fresh weight basis (Nygard 1983) Peregrine populations have declined in Norway, but the high DDT levels (which cause eggshell thinning) in tissues and eggs--together with measurable residues of dieldrin and mercury--made it difficult to ascribe thinning or population declines exclusively to PCBs (Nygard 1983). Mean PCB residues were significantly lower in eggs from successful nests of the American bald eagle than unsuccessful nests (1.3 ppm fresh weight vs. 7.2), and may be associated with eggshell thinning (Wiemeyer et al. 1984). PCB concentrations in eggs were inversely correlated with shell thickness in the bald eagle (Wiemeyer et al. 1984) as well as the black-crowned night-heron (McEwen et al. 1984; Henny et al. 1984). However, PCB content is frequently correlated positively with DDE content- (Norheim and Kjos-Hanssen 1984), which is known to interfere with avian calcium metabolism and to induce thin eggshells. The observed thickening of eggshells in black-crowned night-heron eggs between 1973 and 1979 in colonies from Rhode Island locations was associated with marked reductions in both PCBs and DDE (Custer et al. 1983a,b). At present, the evidence implicating PCBs as a major source of eggshell thinning is inconclusive.

Loss rates were followed in common grackles fed 150 ppm dietary Aroclor 1254 for 8 days, then given untreated food and killed at 1 to 32 weeks posttreatment (Stickel et al. 1984). PCB levels in bodies of grackles declined from 1,300 ppm fresh weight on the day clean food was restored, to 169 ppm 32 weeks later. The overall loss rate was estimated at 0.77% daily with a calculated biological half-life of 89 days. Similar loss rates were observed in pheasants given a single capsule dosage of Aroclor 1254 (as quoted in Stickel et al. 1984). In general, PCB residues in brain are good indicators of PCB exposure. For example, Japanese quail fed 1,000 ppm of Aroclor 1260, and that subsequently died, contained 780 ppm in brain; treated survivors contained 250 ppm (as quoted in Heinz et al. 1984). Also, various species of small birds that were killed by dietary exposure to Aroclor 1254 had PCB brain residues of 349 to 763 ppm (Heinz et al. 1984).

PCBs are associated with a variety of biochemical, histopathological, and behavioral responses in birds. PCBs affect zinc and calcium metabolism in chickens, disrupt vitamin A use in quail, and potentiate vitamin A deficiency in chickens by interfering with selenium use (Roberts et al. 1978). Body temperature, serum chemistry, and thyroid function of ringed turtle-doves was significantly altered by 3,4,3',4'-tetrachlorobiphenyl (Spear and Moon 1985). Metabolism of various respiratory pigments was disrupted by PCBs in birds and mammals (Roberts et al. 1978). PCBs are good inducers of drug-metabolizing enzymes that are vital in detoxification processes. Aroclor 1254 injected once into liver parenchymal tissue of the barn owl (*Tyto alba*), at 30 mg/kg body weight, produced increases in the levels of liver cytochrome P-450 activities (Rinzky and Perry 1983). Ringed turtle-doves fed 10 or 100 ppm of dietary Aroclor 1254 showed depressed levels of dopamine and norepinephrine; PCB residues in the brains of these doves averaged 2.8 in the 10 ppm group, and 18.3 ppm in the 100 ppm group (Heinz et al. 1984). Pelicans (Pelecanus sp.) fed 100 ppm of dietary Aroclor 1254 for 10 weeks showed increase liver histopathology (NAS 1979). Certain PCB congeners produce acute histopathologic changes in chick embryo liver, and these same congeners selectively induce cytochrome P-448 mediated mixed function oxidases; adverse effects were noted within 24 hours at concentrations as low as 146 mg of 3,4,3',4'-tetrachlorobiphenyl and 3,4,5,3',4',5'-hexachlorobiphenyl on a whole egg fresh weight basis (Rifkind et al. 1984). PCBs also have been associated with abnormal behavior in European robins (*Erithacus rubecula*), pheasants, quail, and other avian species according to Heinz et al. (1984).

MAMMALS

The mink is one of the most sensitive mammalian species to PCBs (Aulerich et al. 1985). Signs of PCB poisoning in mink include anorexia, weight loss, lethargy, and unthrifty appearance; prior to death, dark fecal stools indicative of the presence of blood from the upper gastrointestinal tract (confirmed by necropsy) was observed. Enlarged livers of mink given PCB diets were typical; a similar pattern has been associated in practically all species studied. PCB residues (10 to 15 ppm whole body fresh weight) within Great Lakes fish incorporated into the diet of mink caused reproductive problems and death in commercially ranched mink as long ago as 1965. Diets supplemented with as little as 2 ppm of Aroclor 1254 for 8 months, or 5 ppm for 4 months, resulted in near reproductive failure--with normal breeding and whelping, but a high death rate of kits; reproduction was not affected at dietary levels of 1 ppm of Aroclor 1254. Certain hexachlorobiphenyl isomers can produce death and reproductive toxicity in mink at dietary concentrations as low as 0.1 mg/kg, while other hexachlorobiphenyls are relatively innocuous (Aulerich et al. 1985). Biologically modified PCBs are more toxic to mink than corresponding technical mixtures. For example, tissues from cattle that had been dosed with

Aroclor 1254 and fed to mink at levels as low as 0.64 ppm fresh weight of diet caused severe reproductive effects, possibly because cattle tissues retained more of the highly chlorinated congeners of the PCB mixture once they have been metabolized. But Aroclors 1016 and 1221, at dietary concentrations of 2 ppm, produced no adverse reproductive effects in mink over a 9-month period, nor did Aroclor 1242 at 5 ppm during a similar period; the reasons for this are unknown but may be due to the ability of mink to metabolize selected PCB congeners. Mink, for example, easily eliminate 2,2',4,4',5,5'-hexachlorobiphenyl, but in rats, domestic pigeons (*Columba livia*), and trout, this congener remains almost indefinitely (Hornshaw et al. 1983). Placental transfer of PCBs occurs in mink, as has been demonstrated for rats, rabbits, cattle, rhesus monkeys, ferrets, and humans, and gives rise to the embryotoxicity demonstrated by these species (Ringer 1983). A significantly greater quantity of PCBs enters the growing offspring of mink from mammary transfer than from placental transfer. This PCB transfer by the lactating mink probably resulted in the high offspring mortality observed when Great Lakes fish were fed to commercial ranch mink in the late 1960's. Aroclor 1254 residues in subcutaneous fat of adult mink was up to 38X dietary levels, and some individual congeners accumulated up to 200X. The time for 50% elimination of PCBs from adipose tissues was about 98 days, and about 199 days for 100% elimination. Comparable data for other mammals indicate that PCBs are eliminated more rapidly by these species than mink, with 50% half-life times of 33 hours to 69 days for tissues of rat and dairy cows (EPA 1980; Stickel et al. 1984). In general, PCB residues in fat of mink were highest in winter when fat deposits were mobilized during cold weather and PCB residues were concentrated in remaining fat stores; this is consistent with the results obtained from pigeons subjected to low temperatures and starvation (Hornshaw et al. 1983).

The European ferret was significantly more resistant to PCBs than mink, and demonstrates that interspecies sensitivity to PCBs varies widely, even among taxonomically close species (Ringer 1983; Bleavins et al. 1984). In ferrets, total reproductive failure was documented in 9-month feeding studies of Aroclor 1242 at dietary levels of 20 ppm, but Aroclor 1016 at 20 ppm did not affect reproduction during a similar period. PCBs in maternal body fat stores of ferrets represent a reservoir that can be transferred to the developing fetus and growing neonate (Aroclor 1254) in the European ferret (Bleavins et al. 1984). Placental transfer of Aroclor 1254 to kits of the European ferret was about 0.01% per kit of the female's absorbed dietary dose when exposure occurred during the first trimester of pregnancy, and 0.04% when PCBs were administered during the third trimester. Transfer of PCBs through the dam's milk was also documented, and this route was 6 to 15X more effective than placental transfer.

The rhesus monkey (*Macaca mulatta*) is extremely sensitive to PCBs (Roberts et al. 1978; NAS 1979 EPA 1980; Safe 1984). Females fed diets containing 2.5 to 5.0 ppm of Aroclor 1248 for 6 months showed altered menstrual cycles, an increased frequency of stillbirths and abortions, a lowered birth rate, hyperpigmentation, skin eruptions, eye problems, and negatively altered behavioral patterns. Males were less sensitive than females; however, when fed diets containing 300 ppm of Aroclor 1248 for one month, both sexes showed hair loss, purulent discharges from the eyes, acneform skin eruptions, and hypertrophy of the liver and gastric mucosa. Rhesus monkeys can efficiently absorb Aroclor 1248 from the gut; 90% of a single oral dose of 1,500 or 3,000 mg/kg body weight was reported absorbed from the gastrointestinal tract. This, and the fact that rhesus monkeys were unable to efficiently eliminate or metabolize certain PCB congeners (i.e., 2,5,2',5'-tetrachlorobiphenyl) when compared to other species, may partially account for the sensitivity of this species. Infant rhesus monkeys, born to mothers exposed to 2.5 ppm of Aroclor 1248 in the diet during pregnancy and lactation, survived over 4 months; PCB levels in their fat declined over a period of 8 to 23 months.

In Japan, humans were accidentally poisoned by rice oil containing 2 to 3 mg of Kanechlor 400/kg (EPA 1980; Lucier and Hook 1985a,b). Symptoms included increased eye discharges and swelling of upper eyelids, acneform skin eruptions, skin pigmentation, hearing and vision problems, gastrointestinal disturbances, and altered blood chemistry. Infants born of Japanese women married to afflicted males were small for their age, had unusual pigmentation, premature eruption of teeth, and exophthalmia (popeyes). Three years after exposure, 50% of the patients were improving, 40% were unchanged, and 10% were worse. Even among those said to be improving, many still complained of headaches, fatigue, weakness and numbness of the limbs, and weight loss. Impurities in the Kanechlor 400 mixture included polychlorinated dibenzofurans and dioxins at levels up to 5 ppm, and these may be responsible, in part, for the observed symptoms in victims.

Mutagenic, carcinogenic, and teratogenic properties of PCBs are documented. Certain PCB congeners, such as 4-chlorobiphenyl, were highly mutagenic to *Salmonella typhimurium* in Ames tests (EPA 1980). Aroclor 1221 was less mutagenic, while Aroclors 1254 and 1268 were essentially inactive. In general, mutagenic

activity tends to decrease with increasing chlorination (EPA 1980). The carcinogenic effects of PCBs have been established in mice and rats with various Aroclor and Kanechlor PCBs and these, in turn, may enhance the carcinogenicity of other chemicals (EPA 1980). Experimental data clearly shows that commercial PCBs cause liver damage which leads to putative preneoplastic changes and hepatocellular carcinomas; however, these lesions are observed only after lengthy (11 to 21 months) exposures to relatively high doses (100 to 1,200 ppm in diets) of these chemicals (NAS 1979; Safe 1984). PCBs were also shown to inhibit the growth of experimental tumors in rats (EPA 1980); administration of Aroclor 1254 (either dietary or injected) for 5 days before or after tumor inoculation was more effective than administration between days 5 and 10. Teratogenic effects of PCBs observed in monkeys and rabbits include abnormal skull formation of fetuses exposed to high levels of Aroclor 1254 in utero, and retarded growth (EPA 1980).

Aroclor 1254 at dietary levels of 25 to 100 ppm for up to 3 weeks can significantly reduce sleeping times in animals that normally aestivate or hibernate, such as white-footed mice, *Peromyscus leucopus* (Sanders and Kirkpatrick 1977), and raccoons, *Procyon lotor* (Montz et al. 1982). The implications of this are not clear, and additional research is needed. Other systemic effects of PCBs that occur in a wide variety of animal species include hepatic disorders distinguished by altered porphyrin metabolism, increased thyroxin metabolism and ultrastructural changes in the thyroid, inhibition of ATPases, interference with oxidative phosphorylation, alterations in steroid hormone activities, immunosuppressive effects, and altered vitamin A metabolism (EPA 1980; Safe 1984).

CURRENT RECOMMENDATIONS

Effective July 1979, under Section 6e of the Toxic Substances Control Act, and unless specifically exempted by the U.S. Environmental Protection Agency, the manufacturing, processing, commercial distribution, and use of PCBs (except in a totally enclosed system) were prohibited (Bremer 1983). Similar actions had been initiated by Michigan, Wisconsin, and Minnesota in 1977 (Bremer 1983). However, PCBs remain universally distributed in the environment, and releases still include those from manufacturing, leaks from supposedly closed systems, and disposal of PCBs manufactured prior to 1971 (Ayer 1976). PCB burdens in waters, sediments, soils, disposal sites, and in deployed transformers and other containers of PCB is estimated at 82 million kg (D'Itri and Kamrin 1983). At this time, total PCB residues in organisms appear to be a more reliable measure of environmental PCB levels than measurements of any commercial mixtures (Schmitt et al. 1985). In light of the demonstrated differential toxicities within the array of PCB congeners, existing standards and criteria may need to be modified in order to reflect the more toxic PCBs (Brown et al. 1985).

PCB tolerance levels have been recommended for protection of various environmental resources and human health (Table 7). The recommended freshwater aquatic life protection criterion of 0.014 ug/l (24-hour average) is lower than 0.1 ug/l, a concentration known to adversely affect the growth of freshwater algae and fish (EPA 1980). This criterion would probably afford a satisfactory degree of protection to freshwater life if it were changed from 0.014 ug/l (24-hour average) to 0.014 ug/l (maximum). Criteria based on average daily concentrations usually indicate that high doses of toxicants may occur within a short period. Unfortunately, data bases existing for PCBs are inadequate to predict long-term effects on growth, uptake, and other variables when repeated high doses occur in short intervals.

The criterion of 0.03 ug/l (24-hour average) recommended for saltwater aquatic life protection is unsatisfactory. Concentrations of 0.1 ug/l of Aroclor 1254 are fatal to sheepshead minnows in 21 days, and concentrations as low as 0.006 ug/l result in significant uptake by oysters over a period of 65 days (Ernst 1984). Until additional data become available, the saltwater aquatic life protection criterion should not differ from the freshwater criterion (0.014 ug/l, maximum).

Fish diets containing 1.0 mg of Aroclor 1254 per kg fresh weight produced pathological changes in the kidney of rainbow trout after 11 months, and diets containing 1.2 mg Aroclor 1248/kg fresh weight produced progressive degenerative changes in the liver of lake trout after 9 months (Roberts et al. 1978). The current recommended level for PCBs in fish diets of less than 0.5 mg/kg fresh weight is based on investigations with striped bass by O'Connor and Pizza (1984). They found that food items containing less than 0.5 mg/kg fresh weight will not, in the course of one growing season, cause body burdens in striped bass to exceed 2 mg/kg fresh weight, a proposed Food and Drug Administration guideline for PCB burdens in fish. Striped bass are mobile, pelagic, and highly migratory; accordingly, dietary levels may have to be revised downwards for benthic, nonmigratory species that frequent localized areas of high PCB contamination (O'Connor and Pizza 1984).

Whole body residues of 0.4 mg PCBs/kg fresh weight are associated with reproductive toxicity in rainbow trout (EPA 1980). The large discrepancy between this value and the current recommended level of 5.0 mg/kg fresh weight in fish and shellfish (Table 7) will be discussed later. Eggs of rainbow trout containing 0.33 mg PCBs/kg fresh weight showed reduced hatch, and a significant increase in larval deformities (Niimi 1983). PCB residues of 0.12 mg/kg fresh weight in gonads of field-collected Baltic flounders may be associated with population declines of that species (Ernst 1984), but this needs to be verified experimentally.

Birds seem relatively resistant to PCBS. Among sensitive species, female screech owls fed 3.0 mg of PCBs/kg fresh weight diet laid eggs containing up to 17.8 mg/kg fresh weight; however, no other adverse effects were observed in either parents or progeny (McLane and Hughes 1980). Higher dietary exposures of 5 mg/kg in chickens, and 10 mg/kg in mourning doves resulted in reproductive impairment (Tori and Peterle 1983; as quoted in Heinz et al. 1984). Fertilized eggs of ringed turtle-doves containing 16.0 mg PCBs/kg fresh weight showed delays in growth and development (Peakall et al. 1972), and residues of this magnitude should be considered as presumptive evidence of significant PCB contamination. Residues in brain appear to be good indicators of PCB exposure in birds. Concentrations in excess of 301 mg PCBs/kg brain fresh weight is strong evidence of PCB poisoning, while concentrations in excess of 54 mg/kg fresh weight were common in brain of various avian species that survived high PCB dosages (Stickel et al. 1984).

Rats and dogs (*Canis* sp.) fed various Aroclor PCBs for 2 years showed no measurable effects at dietary levels equivalent to 0.255 (dogs) and 0.5 (rats) mg/kg body weight daily. Using a safety factor of 100, tolerable exposure limits of 2.5 (dog) and 5.0 (rat) ug PCBs/kg body weight daily were derived (Table 7). For the rhesus monkey, a comparatively sensitive species, the new temporary tolerance exposure is 1.0 ug/kg body weight daily (Grant 1983). The mink is the most sensitive animal tested to PCBs, with death and reproductive toxicity documented at 100 to 640 ug PCBs/kg fresh weight of diet (Table 7). The feeding level at which no measurable effects occur is not known with certainty. However, the calculated maximum tolerance level for mink is less than 1.54 ug PCBs/kg body weight daily. This value was derived by known growth rates of female mink between ages 7 and 31 weeks (NAS 1968)--when their body weight increased from 560 g to 1,130 g--by the percent of body weight consumed as food on a daily basis (16.4 to 27.2), and by a safety factor of 100 applied to a dietary level of 0.64 mg PCBs/kg fresh weight of diet. Since safety factors are usually applied to the no observed effect levels, a tolerable level of PCBs for mink may be less than 1.0 ug/kg body weight daily. Other species of mammalian wildlife tested were more resistant to PCBs than mink, and tolerance levels for livestock (Table 7) may also afford a reasonable degree of protection for wildlife, except mink.

Sound management of fishery and wildlife resources--including those resources that are artificially propagated and released--requires noninterference with desired uses such as health and well being of humans and other organisms at various trophic levels. Prior to the legislative restrictions on PCB use, Substantial losses to the atmosphere resulted from evaporation of plasticizers and from improper incineration, directly impacting occupational workers (EPA 1980), as well as aquatic ecosystems (Ayer 1976). In recent years, PCB levels have significantly declined in all human food items, with the possible exception of fish; most samples of fish containing more than 5.0 mg PCBs/kg fresh weight originated from the Great Lakes area (Hoeting 1983). In Michigan, all of a sample of 1,057 mothers had measurable PCBs in their breast milk at an average level of 2.3 mg/kg. Nursing infants from Michigan mothers might consume 10 to 25X the maximum daily dose rate of 1.0 ug PCBs/kg body weight that is currently recommended by the U.S. Food and Drug Administration for human adult intake (Swain 1983). The Michigan Department of Public Health has since established a Public Health Advisory related to fish consumption. They recommend that children, pregnant women, nursing mothers, and women who expect to bear children should not consume fish from the Great Lakes area (Swain 1983). Canadian PCB tolerance levels in food items for human health protection are markedly lower than those of the United States (Table 7). In one case, the current USA health tolerance level of 5.0 mg/kg fresh weight in fish and shellfish presents a distinct hazard to piscivorous teleosts and to fish-eating birds and mammals. A lowering from 5.0 to 2.0 mg PCBs/kg fresh weight in fish and shellfish has been proposed by FDA, but the tolerance level has not yet been changed; the delay appears to be based on economic reasons (Hoeting 1983). In the Great Lakes, for example, 55% of the domestic fish samples collected in 1979-1980 exceeded 2.0 mg PCBs/kg fresh weight; in 1980-1981, this was 17%; and in 1981-1982, 10% of the samples exceeded 2 mg/kg, including chinook salmon and their eggs, and lake trout (Hoeting 1983). In every collection year, measurable PCB residues were recorded in at least 28% of the Great Lakes fish samples collected (Hoeting 1983). At present, three courses of action appear warranted: continuation of the Nationwide monitoring program of fish and wildlife for PCBs and other environmental pollutants (O'Shea and Ludke 1979), additional investigations on the fate of PCBs under

conditions prevailing in the natural environment, and controlled studies on the toxicological significance of chlorinated dibenzofurans and other trace impurities found in commercial PCB mixtures and used PCB containing fluids.

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Table 1. Average chlorine content, chlorine atoms per molecule, and molecular weight of selected Aroclor and Kanechlor PCB formulations (after Roberts et al. 1978).

Trade name and formulation		Percent chlorine	Mean no. chlorine atoms per molecule	Mean molecular weight
Aroclor	Kanechlor			
1221	---	20.5-21.5	1.15	192
1232	200	31.5-32.5	2.04	221
1242	300	42	3.10	261
1248	400	48	3.90	288
1254	500	54	4.96	327
1260	600	60	6.30	372
1262	---	61.5-62.5	6.80	389
1268	---	68	8.70	453

Table 2. PCB concentrations in field collections of selected species of flora and fauna. Values shown are in mg/kg (ppm) fresh weight (FW), dry weight (DW), or lipid weight (LW).

Taxonomic group, organism tissue, location and other variables	Concentration[a] (ppm)	Reference[b]
Plants		
Northeast and central New York		
18 spp., 1979		
Foliage	0.03-0.29 DW	Buckley 1983
Hay	0.08-0.10 DW	
Brome grass, *Bromus inermis*		
Hay, 1979	0.14 DW	
Perennial rye, *Lolium perenne*		
Hay, 1979	0.08 DW	
Timothy, *Phleum pratense*		
Hay, 1979	0.10 DW	
Trembling aspen, *Populus tremuloides*		
Leaves		
1978	0.12 DW	
1979	0.09 DW	
1980	0.07 DW	
Goldenrod, *Solidago graminifolia*		
Leaves		
1978	0.32 DW	
1979	0.25 DW	
1980	0.18 DW	

Invertebrates		
Chironomid, *Chironomus plumosus*,		
near Swedish sewage plant outfall		
From sediments with	0.04 FW; 0.3 DW	Larsson 1984
Larvae	0.1 FW; 10.0 LW	
Adults	0.2 FW; 8.9 LW	
From sediments with	0.6 FW; 4.2 DW	
Larvae	8.6 FW; 757.0 LW	
Adults	19.4 FW; 665.0 LW	
From sediments with	7.0 FW; 28.0 DW	
Larvae	35.5 FW; 3,583.0 LW	
Adults	124.0 FW; 4,234.0 LW	
Mysid, *Mysis relicta*		
Lake Michigan, 1980-1981		
Whole	0.1-0.5 DW	Evans et al. 1982
Fecal pellets		
Aroclor 1254	1.3 DW	
Aroclor 1242	1.2 DW	
Fish		
Goldfish, *Carassius auratus*		
Hudson River, New York		
Whole		
1979	6,761.0 LW	Brown et al. 1985
1982	310.0 LW	
Brown bullhead, *Ictalurus nebulosus*		
Hudson River, New York		
Whole		
1979	2,510.0 LW	
1982	428.0 LW	
Pumpkinseed, *Lepomis gibbosus*		
Hudson River, New York		
Whole		
1979	1,079.0 LW	
1982	36.0 LW	
Largemouth bass, *Micropterus salmoides*		
Hudson River, New York		
Muscle		
1977	29.5-145.3 FW	
1981	<1.0-10.2 FW	
Whole		
1979	6,010.0 LW	
1982	1,000.0 LW	
Brook trout, *Salvelinus fontinalis*		

Whole, 1979-1980		
New Hampshire	0.08 FW	Haines 1983
Maine and Vermont	<0.03 FW	
Codfish, *Gadus morhua*		
Liver		
Fish weight (g)		
86	9.3 LW	Ernst 1984
243	8.9 LW	
471	10.0 LW	
917	21.0 LW	
2,628	22.0 LW	
North Sea, 1974-1976		
Muscle	<0.001 FW	
Liver	0.03 FW	
Windowpane flounder, *Scopthalmus aquosus*		
Long Island Sound, New York, 1980-1982		
Liver	0.9-1.9 FW	Greig et al. 1983
Stomach contents	0.03-0.45 FW	
Striped bass, *Morone saxatilis*		
Nova Scotia		
Muscle	0.02 (Max. 0.09) FW; 0.22 (Max. 0.73) LW	Ray et al. 1984
Gonad	0.04-1.4 FW; 0.08-2.3 LW	
Hudson River, New York		
Muscle		
1978	9.9 FW	Brown et al. 1985
1982	2.6 FW	
USA, Nationwide, 109 stations		
Whole		
Aroclor 1248		
1976-1977	0.15 (Max. 51.9) FW; 0.64 (Max. 465.0) LW	Schmitt et al. 1983
1978-1979	0.14 (Max. 66.9) FW; 0.73 (Max. 348.0) LW	
1980-1981	0.1 (Max. 8.5) FW; 0.8 (Max. 143.8) LW	Schmitt et al. 1985
Aroclor 1254		
1976-1977	0.47 (Max. 16.4) FW; 4.0 (Max. 278.0) LW	Schmitt et al. 1983
1978-1979	0.46 (Max. 22.0) FW; 4.7 (Max. 115.0) LW	
1980-1981	0.2 (Max. 4.2) FW; 2.1 (Max. 87.5) LW	Schmitt et al. 1985

Aroclor 1260		
1976-1977	0.87 (Max. 70.6) FW; 8.0 (Max. 738.0) LW	Schmitt et al. 1983
1978-1979	0.84 (Max. 92.3) FW; 8.8 (Max. 483.0) LW	
1980-1981	0.3 (Max. 2.6) FW 2.6 (Max. 61.0) LW	Schmitt et al. 1985
Total PCBs		
1976-1977	1.5 FW; 12.6 LW	Schmitt et al. 1983
1978-1979	1.4 FW; 14.2 LW	
Great Lakes, 48 watersheds, 1979		
Whole		
16 watersheds	<2.0 FW	Kuehl et al. 1983
13 watersheds	2.0-5.0 FW	
17 watersheds	5.0-20.0 FW	
2 watersheds	>20.0 FW	
Antioch, Illinois		
Walleye, *Stizostedion vitreum vitreum*		
Aroclor 1242	18.0 FW	
Aroclor 1248	22.0 FW	
Aroclor 1254	7.3 FW	
Total PCBs	47.3 FW	
Monroe, Michigan		
Carp, *Cyprinus carpio*		
Aroclor 1242	77.0 FW	
Aroclor 1248	27.0 FW	
Aroclor 1254	7.7 FW	
Total PCBs	111.7 FW	
Great Lakes, tributaries, 1980		
Coho salmon, *Oncorhynchus kisutch*		
Fillets, skin-on	Max. 6.1 FW	Rohrer et al. 1982
Fillets, skinless	Max. 1.6 FW	
Great Lakes area, 1980-1981		
Whole		
Ohio		
Astabula River	1.7-10.7 FW	De Vault 1985
Black River	1.3 FW	
Wisconsin		
Sheboygan River		
Total PCBs	38.6-98.4 FW	
Aroclor 1248	21.3-51.4 FW	
Aroclor 1254	15.8-42.4 FW	
Aroclor 1260	1.5-4.7 FW	

Milwaukee River	6.6-15.5 FW	
Menominee River	0.8-3.2 FW	
Kinnickinnic River	17.7 FW	
Fox River	2.0-20.9 FW	
Wolf River	0.2-0.8 FW	
Chequamegon Bay	0.4-0.7 FW	
North Saskatchewan River, Alberta, Canada, 5 spp.		
Fat	Max. 81.3 FW	Chovelon et al. 1984
Muscle	ND-0.3 FW	
Reptiles		
Loggerhead sea turtle, *Caretta caretta* Florida, east coast		
Muscle	0.005-0.046 FW	McKim and Johnson
Liver	0.008-0.182 FW	1983
Green sea turtle, *Chelonia mydas*		
Florida, east coast		
Muscle	0.005-0.009 FW	
Liver	0.043-0.080 FW	
Snapping turtle, *Chelydra serpentia*		
Fat		
Upper Hudson River	3,608.0 LW	Olafsson et al.
Lake Ontario	633.0 LW	1983
Water snake, *Nerodia cyclopion*		
Louisiana, near Baton Rouge, 1977-1979		
Whole, less skin and head	0.3 FW	Sabourin et al. 1984
Embryo	1.3 FW	
Water snake, *Nerodia rhombifera*		
Louisiana, near Baton Rouge, 1977-1979		
Fat	5.2-8.2 LW	
Liver	0.2-0.7 FW	
Muscle	up to 0.2 FW	
Whole, less skin and head	0.3-0.6 FW	
Embryo	0.8-1.3 FW	
Birds		
Cooper's hawk, *Accipiter cooperii*		
Egg, 1980		
Wisconsin	Max. 2.9 FW	Pattee et al. 1985
Maryland	Max. 4.0 FW	
Michigan	Max. 4.0 FW	
Connecticut	Max. 6.4 FW	
Pennsylvania	Max. 25.0 FW	
Spotted sandpiper, *Actitis macularia*		
Sheboygan River, Wisconsin, August 1979		
Carcass	28.0-106.0 FW	Heinz et al. 1984

Northern pintail, *Anas acuta*		
California, 1980-1981		Ohlendorf and Miller
Wings	0.07-0.74 FW	1984
Northern shoveler, *Anas clypeata*		
California, 1980-1981		
Carcass		
Males	0.33 (0.18-0.63) FW	
Females	0.11 (0.04-0.31) FW	
Mallard, Anas platyrhynchos		
New York, 1981-1982		
Subcutaneous fat	0.3-14.0 LW	Kim et al. 1985
New York, 1979-1980		
Subcutaneous fat	Max. 26.0 LW	Kim et al. 1984
Breast muscle	Max. 0.8 FW	
Liver	Max. 1.6 FW	
Brain	Max. 3.0 FW	
American black duck, *Anas rubripes*		
New York, 1979-1980		
Subcutaneous fat	Max. 19.0 LW	
Breast muscle	Max. 0.3 FW	
Liver	Max. 1.0 FW	
Brain	Max. 0.9 FW	
Great blue heron, *Ardea herodias*		
Sheboygan River, Wisconsin		
Dead on collection		
Brain, 1976	220.0 FW	Heinz et al. 1984
Brain, 1980	50.0 FW	
Carcass, 1980	23.0 FW	
Alive when captured		
Carcass, 1979	36.0 FW	
Stomach contents, 1979	20.0 FW	
Long-eared owl, *Asio otus*, Netherlands		
Liver		
1968-1969	191.0 FW	Koeman 1973
1969-1970	84.8 FW	
1970-1971	6.9 FW	
Lesser scaup, *Aythya affinis*		
California, 1980-1981		Ohlendorf and Miller
Wings	0.17-1.25 FW	1984
Canvasback, *Aythya valisineria*		
California, 1980-1981		
Wings		
San Francisco Bay		

Males	1.6 FW	
Females	0.3 FW	
Salton Sea	0.3 FW	
Canada goose, *Branta canadensis*		
New York, 1979-1980		
Breast muscle	Max. 0.5 FW	Kim et al. 1984
Liver	Max. 0.4 FW	
Great horned owl, *Bubo virginianus*		
New York, 1981		Stone and Okoniewski
Brain	360.0 LW	1983
Green-backed heron, *Butorides striatus*		
Northeastern Louisiana, 1980		
Adults		
Whole	0.7 (0.2-4.8) FW	Niethammer et al.
Muscle	0.1 FW	1984
Liver	0.2 FW	
Fat	6.0 FW	
Immature		
Whole	0.4 (ND-2.0) FW	
Belted kingfisher, *Ceryle alcyon*		
Sheboygan River, Wisconsin, August 1979		
Carcass	65.0-218.0 FW	Heinz et al. 1984
Stomach contents	12.0-58.0 FW	
Northern bobwhite, *Colinus virginianus*		
Southeastern Texas 1975-1981		Flickinger and
Carcass	<0.5 LW	Swineford 1983
Kestrel, *Falco tinnunculus*, Netherlands		
Liver		
1968-1969	21.4 FW	Koeman 1973
1969-1970	18.7 FW	
1970-1971	44.1 FW	
American bald eagle, *Haliaeetus leucocephalus*		
Egg		
Alaska		
1975	Max. 2.5 FW	Wiemeyer et al. 1984
1976	Max. 4.8 FW	
Arizona, 1977	Max. 8.5 FW	
California, 1977	Max. 2.6 FW	
Delaware		
1977	Max. 52.0 FW	
1978	Max. 32.0 FW	
Florida		
1975	Max. 16.0 FW	

1976	Max. 28.0 FW
1977	Max. 8.1 FW
1979	Max. 5.7 FW
Louisiana, 1979	Max. 7.4 FW
Maine	
1974	Max. 75.0 FW
1975	Max. 45.0 FW
1976	Max. 39.0 FW
1977	Max. 20.0 FW
1978	Max. 11.0 FW
1979	Max. 36.0 FW
Maryland	
1973	Max. 8.9 FW
1977	Max. 64.0 FW
1978	Max. 35.0 FW
1979	Max. 5.7 FW
Michigan	
1969	Max. 30.0 FW
1974	Max. 7.0 FW
1978	Max. 37.0 FW
Minnesota	
1972	Max. 44.0 FW
1976	6.1 (Max. 18.0) FW
1977	4.2 (Max. 13.0) FW
1978	4.7 (Max. 19.0) FW
1979	Max. 12.0 FW
New York	
1971	Max. 9.6 FW
1977	Max. 6.2 FW
1978	Max. 3.2 FW
Ohio, 1976	Max. 67.0 FW
Oregon, 1979	Max. 5.6 FW
Virginia	
1976	Max. 56.0 FW
1977	23.0-218.0 FW
1979	Max. 81.0 FW
Wisconsin	
1976	12.0 (Max. 61.0) FW
1977	5.4 (Max. 33.0) FW
1978	14.0 (Max. 98.0) FW
1979	3.0 (Max. 68.0) FW
Herring gull, *Larus argentatus*	
Lake Michigan and Green Bay	

Egg		
1977	100.0 (77.0-136.0) FW	Heinz et al. 1985
1978	65.0 (40.0-160.0) FW	
1980	54.0 (28.0-76.0) FW	
Norway, 1976-1977		
Fledgling	0.6-1.2 FW	Fimreite and Bjerk
Egg	7.0-9.8 FW	1983
Great Lakes, 1981		
Egg		
Snake Island,		
Lake Ontario	86.0 (45.0-127.0) FW	Fleming et al. 1983
Other colonies	23.0 FW	
Hooded merganser, *Lophodytes cacullatus*		
New York, 1981-1982		
Subcutaneous fat	45.0 LW	Kim et al. 1985
Common merganser, *Mergus merganser*		
New York, 1981-1982		
Subcutaneous fat	124.0 LW	
New York, 1979-1980		
Subcutaneous fat	Max. 9.8 LW	Kim et al. 1984
Breast muscle	Max. 20.0 FW	
Liver	Max. 2.9 FW	
Brain	Max. 2.3 FW	
Red-breasted merganser, *Mergus serrator*		
Lake Michigan, 1977-1978		
Egg	4.9-229.0 FW	Fleming et al. 1983
Black-crowned night-heron, *Nycticorax nycticorax*		
Carcass		
Lake Michigan, 1978	23.0-127.0 FW	Heinz et al. 1985
Brain, Lake Michigan, 1978	4.0-160.0 FW	
Egg		
New England, 1979		
Laid during different quarters of breeding season		
1st	8.4 FW	Custer et al. 1983b
2nd	8.1 FW	
3rd	6.9 FW	
4th	5.5 FW	
North Carolina, 1979		
Laid during different quarters of breeding season		
1st	2.6 FW	
2nd	2.0 FW	
3rd	1.4 FW	
4th	0.6 FW	

Rhode Island		
Gould Island		
1973	10.3 FW	Custer et al. 1983a
1979	6.1 FW	
Hope Island, 1979	10.0 FW	
Massachusetts, Clark's Island		
1973	7.2 FW	
1979	6.2 FW	
North Carolina		
Middle Marsh, 1979	2.4 FW	
Annex, 1979	0.8 FW	
Washington, Oregon, Nevada		
1979-1980	2.0-19.0 FW	Henny et al. 1984
Colorado, Wyoming		
1979	1.0 (0.4-7.0) FW	McEwen et al. 1984
Lake Michigan and Green Bay		
1977	92.0 FW	Heinz et al. 1985
1978	15.0-23.0 FW	
1980	24.0 FW	
Gray partridge, *Perdix perdix*		
Breast		
Low chlorinated PCBs		
Fat	7.8 FW	Brunn et al. 1985
Nonfat	0.04 FW	
High chlorinated PCBs		
Fat	0.8 FW	
Nonfat	0.001 FW	
Double-crested cormorant, *Phalacrocorax auritus*		
Lake Michigan and Green Bay		
Egg		
1977	12.0-16.5 FW	Heinz et al. 1985
1978	4.4-11.0 FW	
1980	2.0 FW	
Lake Huron 1972		
Egg	23.8 (10.3-25.6) FW	Weseloh et al. 1983
Cormorant, *Phalacrocorax carbo*, Netherlands, 1970		
Adults		
Liver	320.0 (93.0-470.0) FW	Koeman 1973
Whole body	29.0-460.0 FW	
Brain	190.0 FW	
Nestlings		
Liver	0.4 FW	
Whole body	2.4 FW	

Brain	0.7 FW	
Ring-necked pheasant, *Phasianus colchicus*		
Breast		
Low chlorinated PCBs		
Fat	6.4 FW	Brunn et al. 1985
Nonfat	0.03 FW	
High chlorinated PCBs		
Fat	4.9 FW	
Nonfat	0.007 FW	
Woodcock, *Philohela minor*		
Wing lipids		
17 States		
1971	5.7 LW	McLane et al. 1984
1972	1.7 LW	
1975	2.8 LW	
Fat, East Central Illinois		
1978-1979	Max. 12.5 LW	Edwards et al. 1983
Kittiwake, *Rissa tridactyla*		
Norway, 1976-1977		
Fledgling	0.65 FW	Fimreite and Bjerk
Egg	2.1 FW	1983
Black skimmer, *Rynchops niger*		
Egg		
Corpus Christi, Texas		
1978	7.2 (4.3-12.0) FW	White et al. 1984
1979	4.1 (1.4-9.0) FW	
1980	3.2 (1.0-20.0) FW	
1981	2.6 (0.8-5.0) FW	
Carcass		
Texas, 1983	1.6 (<0.5-10.0) FW	White et al. 1985
Mexico, 1983	1.0 (<0.5-16.0) FW	
Seabirds		
Baltic Sea, 1981-1983		
Adipose fat		
Auks, Family Alcidae		
4 spp.	92.0 (trace-1,100.O) LW	Falandysz and Szefer
Mergansers, *Mergus* spp.	54.0 LW	1984
Black-throated diver,		
Colymbus arcticus	19.0 LW	
Grebes, *Podiceps* spp.	16.0 LW	
West Coast Spitzbergen, 5 spp., 1979-1980		
Liver	<0.1-6.1 FW	Norheim and Kjos-
Fat	3.1-82.0 LW	Hanssen 1984

Eastern Cape, South Africa, 7 spp.		de Kock and Randall
Egg, 1981-1983	0.05-0.9 FW	1984
Arctic tern, *Sterna paradisaea*		
Finland, 1969-1974		
Liver		
Males	80.1 LW; 4.4 FW	Lemmetyinen and
Females	38.7 LW; 1.7 FW	Rantamaki 1980
Muscle		
Males	74.6 LW; 4.0 FW	
Females	40.3 LW; 2.1 FW	
Egg		27.7 LW; 2.9 FW
Chicks		
Liver		
Newly-hatched	44.0 LW; 4.3 FW	
Age 2-3 weeks	10.8 LW; 0.3 FW	
Muscle		
Age 2-3 weeks	14.1 LW; 0.6 FW	
European starling, *Sturnus vulgaris*		
Whole, minus feet, beak, wing tips, and skin		
Nationwide, USA		
1976	0.01 (ND-0.9) FW	Cain and Bunck 1983
1979	0.05 (ND-2.2) FW	
Wisconsin	1.6 FW	
North Carolina	2.2 FW	
Alabama	1.0 FW	
Maryland	0.8 FW	
Brown booby, *Sula leucogaster*		
Equatorial Atlantic, 1979-1980		
Muscle	0.2 FW; 0.2 DW	Weber 1983
Egg	0.1 FW; 0.1 DW	
Solitary sandpiper, *Tringa solitaria*		
Sheboygan River, Wisconsin, August 1979		
Carcass	23.0 FW	Heinz et al. 1984
Robin, *Turdus migratorius*		
East central Illinois, 1978-1979		
Fat	Max. 6.7 LW	Edwards et al. 1983
Attwater's greater prairie chicken, *Tympanuchus cupido attwateri*		
Southeastern Texas, 1975-1981		Flickinger and
Carcass	<0.5 LW	Swineford 1983
Guillemot, *Uria aalge*		
Norway, 1976-1977		
Fledgling	0.5-0.6 FW	Fimreite and

Egg	2.0-3.6 FW	Bjerk 1983
Waterfowl		
New York, 1979-1980, 17 spp.		
Subcutaneous fat	7.5 LW; 8.1 DW	Kim et al. 1984
Muscle	1.3 FW; 4.2 DW	
Liver	0.8 FW; 2.7 DW	
Brain	0.6 FW; 2.4 DW	
New York, 1981-1982, 9 spp.		
Subcutaneous fat	6.1 LW; 10.1 DW	Kim et al. 1985
Breast muscle	0.3 FW; 0.8 DW	
Mourning dove, *Zenaidura macroura*		
East central Illinois, 1978-1979		
Fat	Max. 0.4 LW	Edwards et al. 1983
Mammals		
Big brown bat, *Eptesicus fuscus*		
Laurel, Maryland		
May 1974		
Adult females, whole	1.3 (1.1-1.6) FW	Clark and Lamont
Young, whole	0.4 (0.2-3.3) FW	1976
June 1974		
Adult females, whole	2.0 (1.6-2.4) FW	
Young, whole	1.2 (0.8-1.7) FW	
Hare, *Lepas europans*		
Muscle		
Low chlorinated PCBs		
Fat	8.5 FW	Brunn et al. 1985
Nonfat	0.02 FW	
High chlorinated PCBs		
Fat	2.0 FW	
Nonfat	0.003 FW	
River otter, *Lutra canadensis*		
Lower Columbia River, Oregon, 1978-1979		
Liver		
Males	9.3 (4.8-23.0) FW	Henny et al. 1981
Females	3.5 (1.7-7.0) FW	
Muscle		
Males	3.7 (1.1-8.3) FW	
Females	2.7 (1.6-4.5) FW	
Mink, *Mustela vison*		
Oregon, 1978-1979		
Liver		
Males	0.5-3.5 FW	
Females	0.7-1.7 FW	

Muscle	0.6-1.6 FW	
Western Maryland, 1978-1979		
Liver		
Males	1.5 (1.1-2.0) FW	O'Shea et al. 1981
Females	1.4 (0.6-2.4) FW	
Atlantic Coast, 1970		
Fat		
Massachusetts		
Dead on collection	6.0-60.0 FW	Friedman et al. 1977
Healthy	0.3-0.5 FW	
Virginia		
Healthy	0.4-0.5 FW	
Gray bat, *Myotis grisescens*		
Missouri		
Brain		
1976	ND	Clark et al. 1980
1977	ND-9.3 FW	
Carcass		
1976	163.0 (71.0-425.0) LW	
1977	295.0 (86.0-1,000.0) LW	
Little brown bat, *Myotis lucifugus*		
Laurel, Maryland, 1976, carcass		
Adult females	11.4 (3.6-24.0) FW	Clark and Krynitsky
Young	4.2 (ND-25.0) FW	1978
Maryland and West Virginia, 1973		
Adults		
Carcass	3.2-11.6 FW	Clark and Prouty
Stomach contents	1.4 FW	1976
Guano	0.5-1.0 FW	
Ringed seal, *Phoca hispida*		
Liver	0.2 FW	Bowes and Lewis
Fat	0.6 FW	1974
Harbor seal, *Phoca vitulina*		
Dutch coast, 1981		
Blubber		
Age 1 year	31.0 LW	Van Der Zande and
Age 3 years	65.0 LW	De Ruiter 1983
Age 4 years	35.0 LW	
Age 5 years	55.0 LW	
Sperm whale, *Physeter macrocephalus*, Spain		
Males		
Blubber	9.9 LW	Aguilar 1983
Muscle	24.0 LW	

Sperm oil	10.5 LW	
Liver	30.1 LW	
Kidneys	9.4 LW	
Brain	1.4 LW	
Females		
Blubber	15.5 LW	
Muscle	30.7 LW	
Sperm oil	5.0 LW	
Liver	18.6 LW	
Kidneys	9.2 LW	
Brain	1.1 LW	
Milk	4.7 LW	
Manatee, *Trichechus manatus*		
Florida, 1977-1981, dead on collection		
Blubber	1.4 (0.5-4.6) FW	O'Shea et al. 1984
Polar bear, *Ursus martinus*		
Liver	4.0-4.9 FW	Bowes and Lewis
Fat	17.4-19.4 FW	1974
Cuvier's goosebeaked whale, *Ziphius cavirostris*		
Blubber	9.4 (7.9-12.3) LW	
Flesh	0.16 FW	Knap and Jickells
Liver	0.25 FW	1983
Kidney	0.10 FW	
Heart	0.04 FW	
Integrated studies		
Upper Hudson River, New York		
Water		
1977	0.00054 FW	Sloan et al. 1983
1978	0.00042 FW	
1979	0.00039 FW	
1980	0.00026 FW	
1981	0.00013 FW	
1982	0.00011 FW	Brown et al. 1985
Water column, including organisms, silt, and detritus		
1977	671.0 LW	Sloan et al. 1983
1978	792.0 LW	
1979	267.0 LW	
1980	186.0 LW	
1981	626.0 LW	
Fish, whole		
1977	4,217.0 LW	
1978	3,951.0 LW	
1979	1,332.0 LW	

1980	1,431.0 LW	
Aroclor 1221	33.0 LW	
Aroclor 1016	664.0 LW	
Aroclor 1254	734.0 LW	
Sediments	20.0-150.0 DW	Carcich and
Water column	0.0001-0.001 FW	Tofflemire 1982
Fish	10.0-130.0 FW	
Macroinvertebrates	3.0-10.0 FW	
Dredge spoils	5.0-50.0 DW	
Industrial landfills		
Waste material	500.0-5,000.0 DW	
Leachate	0.05-0.5 FW	
Dust	17.0 DW	
Plants		
Near industrial landfill	10.0-500.0 FW	
Near dredge spoil area	0.2-1.3 FW	
Lower Hudson River, New York		
Fish		
1977	1,604.0 LW	Sloan et al. 1983
1978	969.0 LW	
1979	371.0 LW	
1980	327.0 LW	
Aroclor 1221	36.0 LW	
Aroclor 1016	106.0 LW	
Aroclor 1254	185.0 LW	
1981	319.0 LW	
Aroclor 1221	19.0 LW	
Aroclor 1016	87.0 LW	
Aroclor 1254	213.0 LW	
Sediments	1.0-15.0 DW	Carcich and
Water column	ND-0.0008 FW	Tofflemire 1982
Fish		
Resident	5.0-10.0 FW	
Migratory	0.5-15.0 FW	
Macroinvertebrates	1.0-13.0 FW	
Turtles		
Muscle	5.0 FW	
Eggs	25.0 FW	
Lake Ontario, 1981		
Surficial sediments	0.3-0.8 DW	Fox et al. 1983
Oligochaetes	1.5-5.3 DW	
Amphipods	2.6-11.0 DW	
Mysid shrimp	3.0 DW	

Lake trout, whole, Age 1+	6.3 DW	
Australian estuary		
Muscle		
Herbivores Shaw and Connell	0.3 FW; 11.5 LW	
Omnivores 1982	1.2 FW; 16.3 LW	
Lower carnivores	0.9 FW; 26.8 LW	
Middle carnivores	0.1 FW; 41.0 LW	
Top carnivores	8.2 FW; 170.0 LW	
Central Puget Sound, Washington, 1979		
Sediments	<0.001-1.2 DW	Malins et al. 1980
Clam, soft parts	0.02-1.3 DW	
Shrimp, whole	0.1-3.0 DW	
Worms, whole	0.2-1.8 DW	
Crab, hepatopancreas	0.4-33.0 DW	
Fish, liver	0.6-35.0 DW	
Escambia Bay and River, Florida		
Sediments		
1970	Max. 78.0 DW	NAS 1979
1971	Max. 8.1 DW	
1972	Max. 5.8 DW	

[a]Concentrations are listed as mean, minimum-maximum, or maximum (Max.) values recorded.

[b]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

Table 3. Acute toxicities of Aroclor PCBs to selected aquatic species.

Ecosystem, organism, compound tested	Exposure period (days)	LC-50 (ug/l)	Reference[a]
Freshwater			
Invertebrates			
Crayfish, *Orconectes nais*			
1242	7	30	NAS 1979
1254	7	80-100	
Scud, *Gammarus pseudolimnaeus*			
1242	4	10	
1242	10	5	
1248	4	52	
1254	4	2,400	
Glass shrimp, *Palaemonetes kadiakensis*			

1254	7	3	
Damselfly, *Ischnura verticalis*			
1242	4	400	Johnson and Finley 1980
1254	4	200	
Dragonfly, *Macromia* sp.			
1242	4	800	
1254	5	800	
Cladoceran, *Daphnia magna*			
1254	14	1.8-24.0	EPA 1980
1254	21	1.3	
Stonefly, *Pteronarcella badia*			
1016	4	424-878	Johnson and Finley 1980
Hydra, *Hydra oligactis*			
1016	3	5,000	Adams and Haileselassie 1984
1254	3	10,000	
Fish			
Rainbow trout, *Salmo gairdneri*			
1016	4	114-159	Johnson and Finley 1980
1242	5	67	
1248	5	54	
1254	5	142	
1254	10	8	NAS 1979
1260	20	21	
Bluegill, *Lepomis macrochirus*			
1016	4	390-540	Johnson and Finley 1980
1242	5	125	
1242	15	54	NAS 1979
1248	20	10	
1254	25	54	
1260	30	150	
Channel catfish, *Ictalurus punctatus*			
1016	4	340-560	Johnson and Finley 1980
1242	15	110	NAS 1979
1248	15	130	
1254	15	740	
1260	30	140	
Salmonids, 4 spp.			
1016	4	134-1,154	Johnson and Finley 1980
Catostomids, 2 spp.			
1016	4	222-582	
Cutthroat trout, *Salmo clarki*			
1221	4	1,170	

1232	4	2,500	
1242	4	5,420	
1248	4	5,750	
1254	4	42,500	
1260	4	60,900	
1262	4	>50,000	
1268	4	>50,000	
Yellow perch, *Perca flavescens*			
1016	4	240	
1242	4	>150	
1248	4	>100	
1254	4	>150	
1260	4	>200	
Marine			
Invertebrates			
Grass shrimp, *Palaemonetes pugio*			
1254	4	6.1-7.8	Ernst 1984
1016	4	12.5	EPA 1980
Brown shrimp, *Penaeus aztecus*			
1016	4	10.5	
Pink shrimp, *Penaeus duorarum*			
1254	12	1.0	
Fish			
Sheepshead minnow, *Cyprinodon variegatus*			
1254			
Fry	21	0.1-0.32	Ernst 1984
Adult	21	0.9	EPA 1980
Spot, *Leiostomus xanthurus*			
1254	38	0.5	Ernst 1984
Pinfish, *Lagodon rhomboides*			
1254	12	0.5	

[a]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

Table 4. Toxicities of Aroclor PCBs to selected species of birds and mammals administered via dietary, oral, dermal, or intraperitoneal routes.

Taxonomic group, route of administration, units, organism, and compound	Exposure period	.LD-50	Reference[a]
Birds			
Dietary (mg/kg diet)			

Northern bobwhite, *Colinus virginianus*			
1221	5 days on	>6,000	Heath et al. 1972
1232	treated diet	3,002	
1242	plus 3 days	2,098	
1248	untreated	1,175	
1254		604	
1260		747	
1262		871	
Mallard, *Anas platyrhynchos*			
1242	5 days on	3,182	
1248	treated diet	2,798	
1254	plus 3 days	2,699	
1260	untreated	1,975	
1262		3,004	
Ring-necked pheasant, *Phasianus colchicus*			
1221	5 days on	>4,000	
1232	treated diet	3,146	
1242	plus 3 days	2,078	
1248	untreated	1,312	
1254		1,091	
1260		1,260	
1262		1,234	
Japanese quail, *Coturnix coturnix japonica*			
1221	5 days on	>6,000	
1232	treated diet	>5,000	
1242	plus 3 days	>6,000	
1248	untreated	4,844	
1254		2,898	
1260		2,186	
1262		2,291	
European starling, *Sturnus vulgaris*			
1254	4 days	1,500	Stickel et al. 1984
Red-winged blackbird, *Agelaius phoeniceus*			
1254	6 days	1,500	
Brown-headed cowbird, *Molothrus ater*			
1254	7 days	1,500	
Oral (grams/kg body weight)			
Mallard			
1242	Single dose	>2	NAS 1979
1254	"	>2	
1260	"	>2	
1268	"	>2	
Mammals			

Dietary (mg/kg diet)			
Mink, *Mustela vison*			
1242	9 months	8.6	Ringer 1983
1254	9 months	6.7	
European ferret, *Mustela putorius furo*			
1242	9 months	>20	
White-footed mice, *Peromyscus leucopus*			
1254	3 weeks	>100	Sanders and Kirkpatrick 1977
Rat, *Rattus norvegicus*			
1254	6 days	>75	Hudson et al. 1984
Mice, Swiss-Webster PCB-resistant strain			
1254	18 weeks	>250	Talcott and Koller 1983
Raccoon, *Procyon lotor*			
1254	8 days	>50	Montz et al. 1982
Cottontail rabbit, *Sylvilagus floridanus*			
1254	12 weeks	>10	Zepp and Kirkpatrick 1976
Oral (grams/kg body weight)			
Rat			
1221	Single dose	1.0- 4.0	EPA 1980; NAS 1979
1232	"	1.3- 4.5	
1242	"	0.8- 8.7	
1248	"	0.8-11.0	
1260	"	1.3-10.0	
1262	"	1.3- 3.2	
1268	"	2.5-11.3	
Various	"	1.0-12.0	Safe 1984
1254	"	0.5-1.4	Hudson et al. 1984
Mink			
1221	Single dose	0.75-1.0	Aulerich and Ringer
1242	"	3.0	1977; Ringer 1983
1254	"	4.0	
Dermal (grams/kg body weight)			
Rabbit			
1221	Single dose	4.0	EPA 1980
1232	"	4.5	
1242	"	8.7	
1248	"	11.0	
1260	"	10.0	
1262	"	11.3	

1268	"	10.9	
Rat			
Various	Single dose	0.8-3.2	Safe 1984
Intraperitoneal (grams/kg body weight)			
Mink			
1221	Single dose	0.5-0.75	Aulerich and Ringer
1242	"	1.0	1977
1254	"	1.25-2.25	

[a]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

Table 5. Aroclor 1254 bioconcentration factors (BCF) for selected species of whole aquatic organisms.

Ecosystem, organism, exposure duration in days, (tissue)	Aroclor 1254 concentration in medium (ug/l)	BCF	Reference[a]
Freshwater			
Invertebrates			
Daphnid, *Daphnia magna*			
4 (whole)	1.1	47,000	NAS 1979
Phantom midge, *Chaoborus punctipennis*			
4 (whole)	1.3	23,000	
14 (whole)	1.3	25,000	
Scud, *Gammarus pseudolimnaeus*			
4 (whole)	1.6	24,000	
21 (whole)	1.6	27,000	
Mosquito larvae, *Culex tarsalis*			
4 (whole)	1.5	18,000	
Crayfish, *Orconectes nais*			
4 (whole)	1.2	1,700	
21 (whole)	1.2	5,100	
Glass shrimp, *Palaemonetes kodiakensis*			
4 (whole)	1.3	12,000	
21 (whole)	1.3	17,000	
Protozoan, *Tetrahymena pyriformis*			
4 (whole)	1.0	60	EPA 1980
Vertebrates			
Fish			
Cichlid, *Cichlasoma facetum*			
3 (spleen)	isotope	1,862	Gooch and
3 (fins)	"	268	Hamdy 1983

3 (liver)	"	173	
3 (muscle)	"	164	
Marine			
Invertebrates			
American oyster, *Crassostrea virginica*			
168 (soft parts)	5.0	85,000	Ernst 1984
Rotifer, *Brachionus plicatilis*			
45 (Lipid)	-	340,000	EPA 1980
45 (Dry tissue)	-	51,000	
Vertebrates			
Fish			
Pinfish, *Lagodon rhomboides*			
35 (whole)	5.0	21,800	Ernst 1984
Spot, *Leiostomus xanthurus*			
56 (whole)	1.0	27,800	

[a]Each reference applies to data in the same row and in other rows that immediately follow for which no reference is indicated.

Table 6. Maximum acceptable toxicant concentration (MATC) values for Aroclor PCBs and selected species of aquatic organisms, based on exposure for life cycle, partial life cycle, or early life stage (from EPA 1980).

Ecosystem, organism, Aroclor PCB	MATC[a] (ug/l)
Freshwater	
Cladoceran, *Daphnia magna*	
1248	1.2-3.5
1254	2.5-7.5
Amphipod, *Gammarus pseudolimnaeus*	
1242	2.8-8.7
1248	2.5-5.1
Insect (midge), *Tanytarsus dissimilis*	
1254	0.5-1.2
Brook trout, *Salvelinus fontinalis*	
1254	0.7-1.5
Fathead minnow, *Pimephales promelas*	
1242	5.4-15.0
1248	0.1-0.4
1254	1.8-4.6
1260	1.3-4.0

Marine	
Sheepshead minnow, *Cyprinodon variegatus*	
Early life stage	
1016	3.4-15.0
1254	0.06-0.16

[a]Lower value in each pair indicates highest concentration tested producing no measurable effect on growth, reproduction, survival, and metabolic upset during chronic exposure; higher value indicates lowest concentration tested producing a measurable effect.

Table 7. Proposed PCB criteria for protection of various resources and human health.

Resource and criterion	PCB concentration[a]	Reference[b]
Aquatic life		
Freshwater	<0.014 ug/l, 24-h average	EPA 1980
Saltwater	<0.030 ug/l, 24-h average	
Fish		
Diets	<0.5 mg/kg FW	O'Connor and
Residues		Pizza 1984
Whole body	<0.4 mg/kg FW	EPA 1980
Eggs	<0.33 mg/kg FW	Niimi 1983
Laboratory Animals		
Rat	<5.0 ug/kg BW daily	Grant 1983
Dog	<2.5 ug/kg BW daily	
Rhesus monkey	<1.0 ug/kg BW daily	
Livestock		
Finished animal feedsc[c]	<0.2 mg/kg FW	Hoeting 1983
Animal feed components[d]	<2.0 mg/kg FW	
Food packaging materials[e]	<10.0 mg/kg	
Wildlife		
Mink	<100 ug/kg FW diet	Aulerich et al. 1985
Mink	<640 ug/kg FW diet;	Ringer 1983;
<1.5 ug/kg BW daily	Hornshaw et al. 1983	
Birds		
Diet	<3.0 mg/kg FW	McLane and Hughes
Residues		1980
Egg	<16.0 mg/kg FW	Peakall et al. 1972
Brain	<54.0 mg/kg FW	Stickel et al. 1984

Human health		
Adult daily intake	<1.0 ug/kg BW	Swain 1983
Fish and shellfish[f]		
USA	<5.0 mg/kg FW	Hoeting 1983
Canada	<2.0 mg/kg FW	Grant 1983
Poultry		
USA	<3.0 mg/kg LW	Hoeting 1983; Kim et al. 1985
Canada	<0.5 mg/kg LW	Grant 1983
Eggs, whole less shell		
USA	<0.3 mg/kg FW	Hoeting 1983
Canada	<0.1 mg/kg FW	Grant 1983
Dairy products		
USA	<1.5 mg/kg LW	Hoeting 1983
Canada	<0.2 mg/kg LW	Grant 1983
Fish oil		
Canada	<2.0 mg/kg LW	
Beef		
Canada	<2.0 mg/kg LW	
Infant and junior foods	<0.2 mg/kg FW	Hoeting 1983
Drinking water [g]	zero	EPA 1980
Lifetime safety limit	200 mg	Rohrer et al. 1982
Overt human effects	500 mg	
Air		
Occupational, 40-h week	<1.0 ug/m^3	EPA 1980

[a] FW = fresh weight; BW = body weight; LW = lipid weight.

[b] Each reference applies to data in the same row and in other rows that immediately follow for which no reference is indicated.

[c] Except feed concentrates, feed supplements, and feed premixes.

[d] Including fish meal and other byproducts of marine origin, and finished feed concentrates, supplements, and premixes.

[e] Paper products intended for use in contact with human food and finished animal feed.

[f] Excluding heads, scales, viscera, and inedible bones.

[g] The zero drinking water criterion for human health protection is based on the nonthreshold assumption for PCBs. However, a zero level threshold may not be attainable at this time. A measurable reduction in potential carcinogenic effects due to exposure of PCBs through ingestion of contaminated water may be affected through ingestion of water containing less than 0.0008 ug PCBs/1.

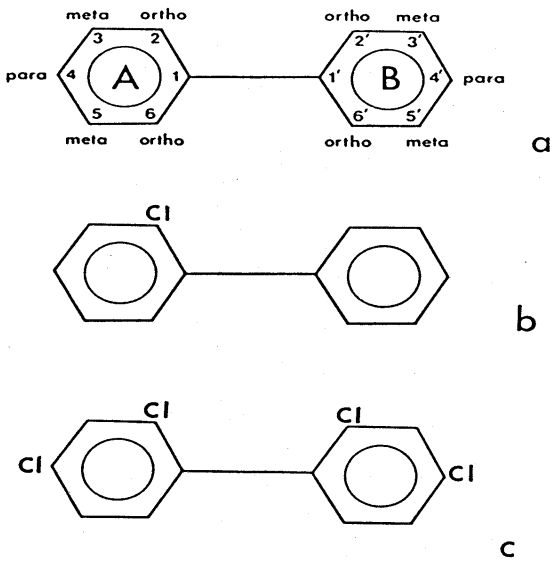

Figure 1. a = Structure of biphenyl (modified from Safe 1984); b= 2-monochlorobiphenyl; c= 2,2',4,4'=tetrachlorobiphenyl.