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Fact Sheet

Polychlorinated Biphenyls (PCBs) Update: Impact on Fish Advisories

PCBs are a group of synthetic organic chemicals that contain 209 possible individual chlorinated biphenyl compounds. These chemically related compounds are called congeners and vary in their physical and chemical properties and toxicity. There are no known natural sources of PCBs. Although banned in the United States from further production in 1979, PCBs are distributed widely in the environment because of their persistence and widespread use. PCB mixtures found in the environment are different from the commercially produced PCB mixtures (known as Aroclors in the United States) because of differences in chemical properties, persistence, and bioaccumulation among the different congeners. The most common analytical method used to detect PCBs in the environment is based on Aroclor analysis; however, congener-specific methods have been developed and currently are being tested. PCB exposure is associated with a wide array of adverse health effects in experimental animals. Experimental animal studies have shown toxic effects to the liver, gastrointestinal system, blood, skin, endocrine system, immune system, nervous system, and reproductive system. In addition, developmental effects and liver cancer have been reported. Skin rashes and a severe form of acne have been documented in humans; however, other effects of PCB exposure in humans are not well understood. EPA has classified PCBs as probable human carcinogens (Group B2). As of 1998, 37 states have issued 679 fish advisories for PCBs. These advisories inform the public that high concentrations of PCBs have been found in local fish at levels of public health concern. State advisories recommend either limiting or avoiding consumption of certain fish from specific waterbodies or, in some cases, from specific waterbody types (e.g., all freshwater lakes or rivers).

The purpose of this fact sheet is to summarize current information on sources, fate and transport, occurrence in human tissues, range of concentrations in fish tissue, fish advisories, fish consumption limits, toxicity, and regulations for PCBs. The fact sheets also illustrate how this information may be used for developing fish consumption advisories. An electronic version of this fact sheet and fact sheets for dioxins/furans, mercury, and toxaphene are available at **http://www.epa.gov/OST/fish**. Future revisions will be posted on the web as they become available.

Sources of PCBs in the Environment

There are no known natural sources of PCBs; therefore, all sources of PCBs are related to commercial manufacture, use, storage, and disposal. Manufacture of PCBs was banned in the United States in 1979. However, PCB-containing materials still in service at the time of the ban were not required to be removed from use, and, therefore, some are still in use. For example, the life expectancy of electrical transformers that contain PCBs is 30 years or more.

Currently, the major source of PCBs is environmental reservoirs from past releases. PCBs have been detected in soil, surface water, air, sediment, plants, and animal tissue in all regions of the earth. PCBs are highly persistent in the environment with reported half-lives in soil and sediment ranging from months to years. Because PCBs have very low solubility in water and low volatility, most PCBs are contained in sediments that serve as environmental reservoirs from which PCBs may continue to be released over a long period of time. PCBs may be mobilized from sediments if disturbed (e.g., flooding, dredging). Volatilization from land and surface water is also an important source for the global distribution of PCBs and is discussed below.

Fate and Transport of PCBs

The global cycling of PCBs results from their evaporation from soils and surface waters to the atmosphere and their redeposition back to land and surface water. Adsorption to sediments and revolatilization are the primary loss mechanisms from surface water.

PCBs are highly lipophilic (fat soluble) and are rapidly accumulated by aquatic organisms and bioaccumulated through the aquatic food chain. Concentrations of PCBs in aquatic organisms may be 2,000 to more than a million times higher than the concentrations found in the surrounding waters, with species at the top of the food chain having the highest concentrations. Bioaccumulation factors vary among the congeners and generally increase with chlorine content from the trichlorobiphenyls up through the hexachlorobiphenyls and then generally

decrease with higher chlorine content (hepta- and octa-chlorobiphenyls).

PCBs have been included in several major fish contaminant monitoring programs. A summary of the National Contaminant Biomonitoring Program (NCBP) data conducted by the U.S. Fish and Wildlife Service, from 1976 through 1984, indicated a significant downward trend in the geometric mean concentration in whole fish samples of total PCBs (from 0.89 ppm in 1976 to 0.39 ppm in 1984); however, PCB residues in fish tissue remain widespread, being detected at 91% of the sites monitored in 1984. Maximum total PCB tissue residue concentrations (wet weight) during this same period also declined, from 70.6 ppm in 1976 to 6.7 ppm in 1984. Coinciding declines in tissue residue concentrations of three Aroclors (1248, 1254, and 1260) were also observed. An analysis of the 1984-1985 data from the NCBP study showed there was no significant difference in residues in bottom feeding and predatory fish for Aroclor 1248 and 1254; however, there were significantly higher concentrations of Aroclor 1260 in predator species as compared to bottom feeders. Mean tissue concentrations of Aroclor 1248, 1254, and 1260 were 0.06 ± 0.32 0.21 ± 0.39, and 0.14 ± 0.24 ppm, respectively, for bottom feeders (e.g., carp, white suckers, and channel catfish) and 0.08 ± 0.31, 0.35 ± 0.69, and 0.23 ± 0.38 ppm, respectively, for predator species (e.g., rainbow, brown, brook, and lake trout, largemouth bass, and walleye).

Total PCBs also were detected at 91% of 374 sites surveyed in EPA's National Study of Chemical Residues in Fish (NSCRF). Maximum, arithmetic mean, and median total PCB concentrations reported were 124, 1.89, and 0.209 ppm (wet weight), respectively. As is shown in Table 1, the tri-, tetra-, penta-, hexa-, and heptachlorobiphenyls were detected in fish tissue samples at >50% of the sites. Mean tissue concentrations were highest for the tetra- and pentachlorobiphenyls with concentrations of 0.696 and 0.565 ppm, respectively. The median fish tissue concentrations were highest for the hexa- followed by the pentachlorobiphenyls with concentrations of 0.077 and 0.072 ppm, respectively.

Table 1. Summary of PCBs Detected in Fish Tissue[a] as Part of the National Study of Chemical Residues in Fish[b] (1986-1989)

Congener Group	% of sites where detected	ppm		
		Maximum	Mean	Median
Monochlorobiphenyl	13.8	0.235	0.001	ND
Dichlorobiphenyl	30.7	5.072	0.021	ND
Trichlorobiphenyl	57.5	18.344	0.150	0.002
Tetrachlorobiphenyl	72.4	60.764	0.696	0.023
Pentachlorobiphenyl	86.7	29.578	0.565	0.072
Hexachlorobiphenyl	88.7	8.862	0.356	0.077
Heptachlorobiphenyl	69.1	1.850	0.097	0.017
Octachlorobiphenyl	34.8	0.593	0.017	ND
Nonachlorobiphenyl	9.7	0.413	0.003	ND
Decachlorobiphenyl	3.3	0.038	0.001	0.003
Total PCBs*	91.4	-----	1.898	0.209

* Total PCBs refers to the sum of the concentrations of compounds with 1 to 10 chlorines.
[a] Concentrations are reported on a wet weight basis.
[b] Species included freshwater, estuarine, and marine finfish; and a small number of marine shellfish.
Source: U.S. EPA, 1992.

Potential Sources of Exposure and Occurrence in Human Tissues

Exposure to PCBs is predominantly through the diet, and especially from fish and seafood products. Red meat, poultry, eggs, and dairy products also may be important dietary sources of PCBs.

Individuals in the general population who may be exposed to higher than average levels of PCBs include recreational and subsistence fishers who routinely consume large amounts of locally caught fish, subsistence hunters who routinely consume the meat and organ tissues of marine mammals, and persons who live near hazardous waste sites contaminated with PCBs.

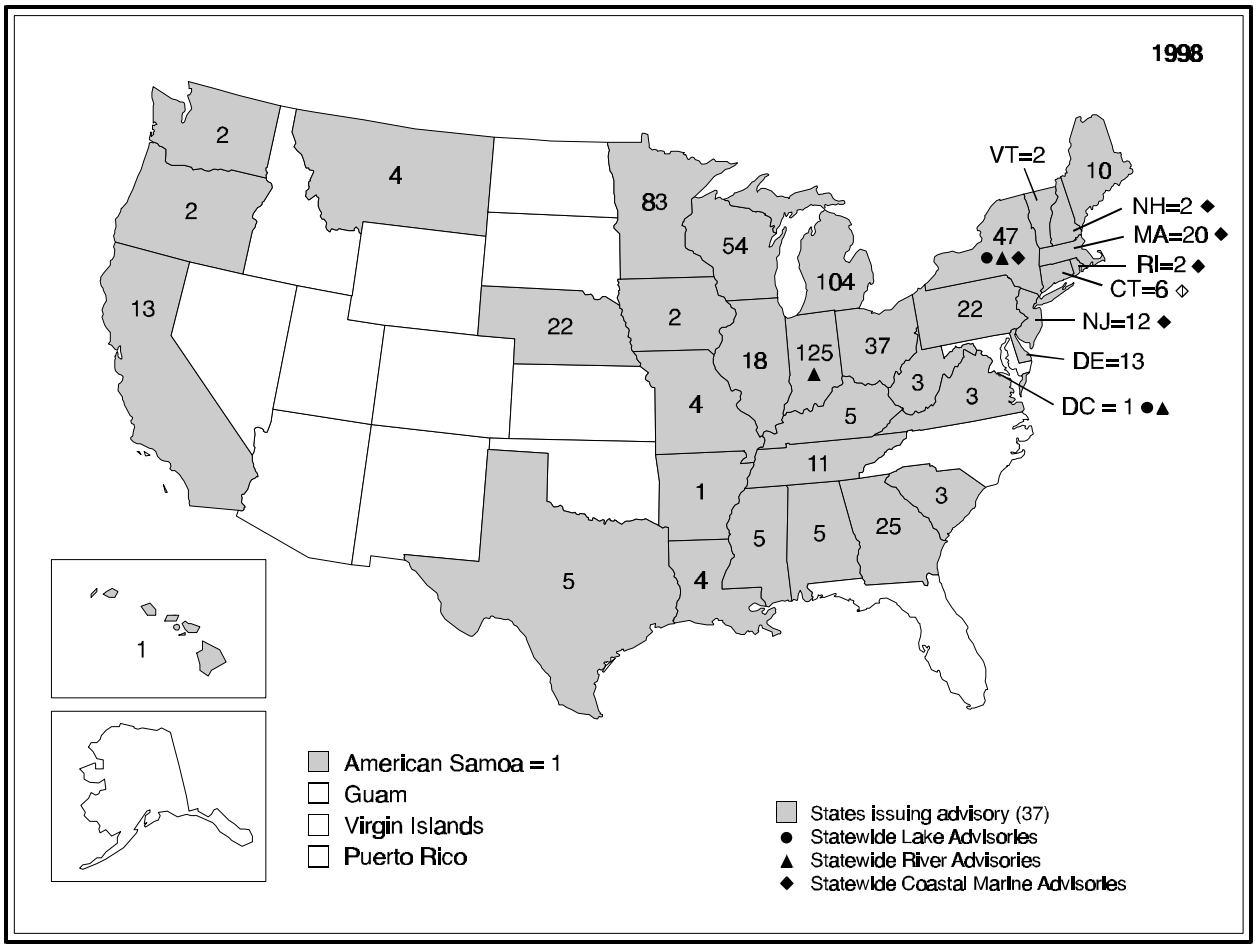
Analytical methods are available to measure PCBs in blood, tissue, breast milk, and environmental media. However, currently there are no standard methods for analyzing for PCB congeners. There is a standard method for Aroclor analysis, but PCBs in environmental samples and human tissues are not adequately characterized as Aroclors. In the absence of peer-reviewed analytical methods for congener specific chemical analysis of fish tissue, EPA's Office of Water recommends the continued use of total Aroclor chemical analysis of fish tissue when conducting human health risk assessment for PCBs. Recently, new risk assessment methods for the PCB congeners have been published and peer-reviewed. However, the analytical methods for congener analysis have not been verified or peer reviewed. When standard methods for congener analysis have been verified and peer reviewed, the Office of Water will reevaluate the use of these methods because of their increased accuracy and precision over Aroclor analysis.

Fish Advisories

The states have primary responsibility for protecting their residents from the health risks of consuming contaminated noncommercially caught fish. They do this by issuing consumption advisories for the general population, including recreational and subsistence fishers, as well as sensitive subpopulations (such as pregnant women/fetus, nursing mothers, and children). These advisories inform the public that high concentrations of chemical contaminants, such as PCBs, have been found in local fish. The advisories recommend either limiting or avoiding consumption of certain fish from specific waterbodies or, in some cases, from specific waterbody types (e.g., freshwater lakes or rivers).

As of December 1998, PCBs were the chemical contaminants responsible, at least in part, for the issuance of 679 fish consumption advisories by 37 states, including the District of Columbia and the U.S. Territory of American Samoa (see Figure 1). Almost 27% of all advisories issued in the United States are

Figure 1. 1998 Fish Advisories for PCBs.



a result of PCB contamination in fish and shellfish. The number of advisories for PCBs is second only to that for mercury advisories. Advisories for PCBs have increased 112% from 319 advisories in 1993 to 679 advisories in 1998. The number of states that have issued PCB advisories increased only slightly from 31 to 35 states from 1993 to 1994 and then declined to 34 states for 1995 and 1996. In 1997 and 1998, the number of states issuing advisories rose to 36 and 37, respectively. Advisories for PCBs increased nearly 15% from 1997 (588 advisories) to 1998 (679 advisories).

Three states (Indiana, New York, and the District of Columbia) have issued statewide advisories for PCBs in their freshwater lakes and/or rivers. Another 6 states—Connecticut, Massachusetts, New Jersey, New Hampshire, New York, and Rhode Island—have statewide PCB advisories in effect for their coastal marine waters. To date, 79% of the 679 PCB advisories in effect have been issued by the following 10 states; Indiana (125), Michigan (104) Minnesota (83), Wisconsin (54), New York (47), Ohio (37), Georgia (25), Pennsylvania (22), Nebraska (22), and Massachusetts (20).

General recommendations regarding food preparation, such as trimming the fat and skinning the fish prior to cooking, also may be included in the general advisory information. Lipophilic chemicals, such as PCBs, accumulate mainly in fatty tissues (belly flap, lateral line, subcutaneous and dorsal fat, dark muscle, gills, eye, brain, and internal organs). Therefore, removal of internal organs and skin and trimming the fat before cooking will decrease exposure. In addition, various cooking procedures can also reduce the amount of PCBs consumed (see Appendix section "Dose Modification Due to Food Preparation and Cooking" of *EPA's Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories*, Volume 2).

Fish Consumption Limits—Table 2 shows the recommended monthly fish consumption limits for PCBs for fish consumers based on EPA's default values for risk assessment parameters. Consumption limits have been calculated as the number of allowable fish meals per month, based on the ranges of PCBs in the consumed fish tissue. The following assumptions were used to calculate the consumption limits:

- Consumer adult body weight of 72 kg
- Average fish meal size of 8 oz (0.227 kg)
- Time-averaging period of 1 month (30.44 days)
- EPA's reference dose for PCBs (2×10^{-5} mg/kg-d) from EPA's Integrated Risk Information System (U.S. EPA, 1999)
- EPA's cancer slope factor for PCBs (2 per mg/kg-d) from EPA's Integrated Risk Information System (U.S. EPA, 1999c)
- Maximum acceptable cancer risk level (10^{-5} over a 70-year lifetime)

For example, when PCB levels in fish tissue are 0.05 ppm, then three 8-oz. meals per month (based on the noncancer health endpoint—EPA's reference dose) or a half of an 8-oz. meal per month (based on the cancer health endpoint—EPA's cancer slope factor) can safely be consumed. EPA recommends using the more conservative of the two limits, for PCBs, this is the consumption limit based on the cancer endpoint.

Table 2. Monthly Fish Consumption Limits for PCBs

Risk-Based Consumption Limit	Noncancer Health Endpoints	Cancer Health Endpoints
Fish Meals/Month	Fish Tissue Concentrations (ppm, wet weight)	Fish Tissue Concentrations (ppm, wet weight)
16	>0.006 - 0.012	>0.0015 - 0.003
12	>0.012 - 0.016	>0.003 - 0.004
8	>0.016 - 0.024	>0.004 - 0.006
4	>0.024 - 0.048	>0.006 - 0.012
3	>0.048 - 0.064	>0.012 - 0.016
2	>0.064 - 0.097	>0.016 - 0.024
1	>0.097 - 0.19	>0.024 - 0.048
0.5	>0.19 - 0.39	>0.048 - 0.097
None (<0.5)[a]	>0.39	>0.097

[a]None = No consumption recommended.
NOTE: In cases where >16 meals per month are consumed, refer to EPA's *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories,* Volume 2, Section 3 for methods to determine safe consumption limits.

For sensitive populations, such as pregnant women, nursing mothers, and young children, some states have issued either "no consumption" advisories or "restricted consumption" advisories for PCBs. Additional information on calculating specific limits for these sensitive populations is available in *EPA's Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories*, Volume 2, Section 3.

Toxicity of PCBs

Pharmacokinetics—PCBs are absorbed through the gastrointestinal tract and distributed throughout the body. Studies of individual chlorobiphenyl congeners indicate, in general, that PCBs are readily absorbed, with an oral absorption efficiency of 75% to greater than 90%. Because of their lipophilic nature, PCBs, especially the more highly chlorinated congeners (tetra- through hexachlorobiphenyl), tend to accumulate in lipid-rich tissues. Greater relative amounts of PCBs are usually found in the liver, adipose tissue, skin, and breast milk. It has been shown that absorption by nursing infants of tetra- and higher chlorinated congeners from breast milk ranges from 90% to 100% of the dose. Offspring can also be

exposed to PCBs through placental transfer. PCBs have also been measured in other body fluids including plasma, follicular fluid, and sperm fluid.

The retention of PCBs in fatty tissues is linked to the degree of chlorination and also to the position of the chlorine atoms in the biphenyl ring. In general, higher chlorinated PCBs persist for longer periods of time. Pharmacokinetic modeling of PCB disposition indicates that PCB movement in the body is a dynamic process, with exchanges between various tissues that depend on fluctuating exposure levels to specific congeners. The result is elimination of congeners that are more easily metabolized and retention of those that resist metabolism. In occupationally-exposed individuals, lower chlorinated congeners had half-lives between 1 and 6 years and higher chlorinated PCBs had half-lives ranging from 8 to 24 years.

PCBs induce mixed function oxidases, and different congeners induce specific forms (isozymes) of the cytochrome P-450 system. Although there has been much research into the mechanisms of PCB toxicity, there is no clear definition of the mechanisms for most congeners. The congeners appear to act by a variety of mechanisms. Some PCB congeners are similar to dioxins and bind to a cytosolic protein, the Ah receptor, which regulates the synthesis of a variety of proteins. The toxicity of these congeners is similar to dioxins. The toxicity of other PCB congeners seems to be unrelated to the Ah receptor. Ultimately, the toxicity of a PCB mixture may depend on the toxicity of the individual congeners and their interactions.

Acute Toxicity—Acute high-level exposures of laboratory animals to PCBs have resulted in liver and kidney damage, neurological effects, developmental effects, endocrine effects, hematological effects, and death. LD_{50} values for Aroclor mixtures range from about 1,000 mg/kg to more than 4,000 mg/kg. No human deaths have been associated with acute exposure to PCBs.

Chronic Toxicity—In animal studies, numerous effects have been documented, including hepatic, gastrointestinal, hematological, dermal, body weight changes, endocrine, immunological, neurological, and reproductive effects. Most of the studies have involved oral exposure. Despite the variety of adverse effects observed in animals exposed to PCBs, overt adverse effects in humans have been difficult to document. This has been attributed to the fact that, in most cases, the dosages tested in animals were considerably higher than those found in occupational exposures and the difficulties with interpreting epidemiological studies. These include multiple confounding factors, uncertain exposure estimates, and statistical limitations. Skin rashes and a persistent and severe form of acne (chloracne) have been reported following exposures to PCBs. Occupational and accidental exposures have indicated that PCBs may affect many organs including the gastrointestinal, respiratory, immune, central nervous, and cardiovascular systems.

Developmental Toxicity—PCB mixtures have been shown to cause adverse developmental effects in experimental animals. Some human studies have suggested that PCB exposure may cause adverse effects in children and in developing fetuses while other studies have not shown effects. Reported effects include lower IQ scores, low birth weight, and lower behavior assessment scores. However, study limitations, including lack of control for confounding variables, deficiencies in the general areas of exposure assessment, selection of exposed and control subjects, the comparability of exposed and control samples, and different findings from different studies provide inconclusive evidence that PCBs cause developmental effects in humans.

A study was conducted of pregnancy outcomes in women who had consumed PCB-contaminated fish from Lake Michigan over an average of 16 years (exposure both prior to and during pregnancy). Consumption of contaminated fish and levels of total PCBs in cord serum correlated with lower birth weight, smaller head circumference, and shorter gestational age. Fish consumption was correlated with delayed neuromuscular maturity, and, at 7 months, the children had subnormal visual recognition memory. Children from this cohort were examined at ages 4 and 11. At age 4, cord serum PCB levels were associated with impaired short-term memory. Activity level was inversely related to 4-year serum PCB level and also to maternal milk PCB level. At age 11, prenatal exposure to PCBs was associated with lower full-scale and verbal IQ scores after controlling for potential confounding variables such as socio-economic status. The results from this series of studies were confounded by possible maternal exposure to other chemicals and by the fact that the exposed group, on average, drank more alcohol and caffeine prior to and during pregnancy, weighed more, and took more cold medications during pregnancy than the nonexposed group.

Other relevant studies generally found no significant differences between control groups and exposed groups regarding stillbirths, multiple births, preterm births, congenital anomalies, and low birth weight.

Information on chronic developmental toxicity is available from studies in Rhesus monkeys. Exposure periods ranged from 12 to 72 months. Inflammation of tarsal glands, nail lesions, and gum recession were noted in offspring of monkeys exposed to Aroclor 1254. Adverse neurobehavioral effects were reported following exposure to Aroclor 1016 and Aroclor 1248. Other observed effects include reduction in birth weight and increased infant death for Aroclor 1248.

Exposure via lactation is a significant concern for neonates. Animal studies indicate that lactational exposure may be more significant than prenatal exposure. In monkeys, signs of PCB intoxication were observed in lactationally-exposed offspring, but not in offspring exposed only prenatally.

PCB Exposure and Development Effects— The data from some studies in humans suggest that exposure to PCBs may cause developmental effects. However, limitations of these studies diminish the validity of the results. Animal studies indicate that PCBs can cause developmental effects following prenatal or postnatal exposure.

Mutagenicity—The majority of mutagenicity assays of PCBs have been negative. However, an increase in the percentage of chromosomal aberrations in peripheral lymphocytes and an increase in the sister chromatid exchange rate were reported in a study of workers manufacturing PCBs for 10 to 25 years. Although workers and controls were matched for smoking and drinking, concurrent exposure to other known human genotoxic chemicals occurred. Another study found an increased incidence of chromatid exchanges in lymphocytes from workers exposed to PCBs in an electric power substation fire compared to unexposed controls. It is possible that toxic chlorinated dioxins and/or furans generated during the fire may have been responsible for the effects.

The weight of evidence from the in vitro and in vivo genotoxicity studies suggests that PCBs are not likely to be genotoxic to humans. However, exposure to PCBs may enhance the genotoxic activity of other chemicals.

Carcinogenicity—PCBs are classified by EPA as Group B2—probable human carcinogens. This is based on studies that have found liver tumors in rats exposed to Aroclors 1260, 1254, 1242, and 1016. Evaluation of the animal data indicates that PCBs with 54% chlorine content induces a higher yield of liver tumors in rats than other PCB mixtures.

Human epidemiological studies of PCBs have not yielded conclusive results. There is some suggestive evidence that xenoestrogens, including PCBs, may play a role in breast cancer induction. Some studies have indicated an excess risk of several cancers including: liver, biliary tract, gall bladder, gastrointestinal tract, pancreas, melanoma, and non-Hodgkins's lymphoma. As with all epidemiological studies, it is very difficult to obtain clear unequivocal results because of the long latency period required for cancer induction and the multiple confounders arising from concurrent exposures, lifestyle differences, and other factors. The currently available evidence is considered inadequate, but suggestive that PCBs may cause cancer in humans.

Summary of EPA Health Benchmarks

- Chronic Toxicity-Reference Dose: $2x10^{-5}$ mg/kg-d (Aroclor 1254) (U.S. EPA, 1999c)
- Carcinogenicity: 1 (central estimate) to 2 (upper bound) per mg/kg-d (U.S. EPA, 1999c)
- Developmental Toxicity: $7x10^{-5}$ mg/kg-d (Aroclor 1016) (U.S. EPA, 1999c)

Special Susceptibilities—There is evidence that embryos, fetuses, and neonates are more susceptible to PCBs due to their under-developed enzymatic systems, which may lead to increased accumulation in the body. Breast-fed infants may have an increased risk because of bioconcentration of PCBs in breast milk and high intake rates relative to body weights. In addition, there is evidence that a steroid present in human milk inhibits glucuronyl transferase activity which could in turn, inhibit glucuronidation and excretion of PCB metabolites. Other individuals with potentially greater risk include those with liver and blood diseases or those with syndromes associated with impairment to the metabolic systems that help eliminate PCBs from the body.

Interactive Effects—PCBs induce microsomal enzymes; therefore, the effects of exposure to PCBs or other compounds depend on the role of oxidative metabolism. For example, preexposure to PCBs may enhance the liver toxicity of some chemicals (trichloroethylene, mirex, kepone, carbon tetrachloride, tetrachloroethylene) but decrease the liver toxicity of 1,1-dichloroethylene. Other interactive effects include increased metabolism and excretion of pentobarbital, increased genotoxicity of numerous carcinogens, increased duodenal ulcerogenic activity of acrylonitrile, and increased kidney toxicity of trichloroethylene.

Critical Data Gaps—The following studies could help to fill in some of the key data gaps for PCBs: congener-specific PCB levels in human tissues, epidemiological studies of population living near PCB contaminated sites and occupational settings where exposure to PCBs still occurs; reproductive studies in humans and animals including fertility studies in males of a sensitive species; developmental and neurodevelopmental studies; immunotoxicity studies in humans and animals; neurotoxicity studies in humans with high PCB body burdens and in animals; chronic studies to determine the most sensitive

animal target organ and species; and comparative toxicity of Aroclors and bioaccumulated PCBs.

EPA Regulations and Advisories

- Maximum Contaminant Level in drinking water = 0.0005 mg/L
- Water Quality Criteria:
  - Continuous chronic criteria (freshwater) = 0.014 µg/L
  - Continuous chronic criteria (saltwater) = 0.03 µg/L
  - Human health = 0.00017 µg/L
- Listed as a hazardous air pollutant under Section 112 of the Clean Air Act
- Reportable quantity = 1 lb
- Listed as a hazardous substance

Sources of Information

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The 1998 update of the database *National Listing of Fish and Wildlife Advisories* is available for downloading from the following Internet site: http://www.epa.gov/OST