Polychlorinated Biphenyls (PCBs)

TEACH Chemical Summary

U.S. EPA, Toxicity and Exposure Assessment for Children's Health

This TEACH Chemical Summary is a compilation of information derived primarily from U.S. EPA and ATSDR resources, and the TEACH Database. The TEACH Database contains summaries of research studies pertaining to developmental exposure and/or health effects for each chemical or chemical group. TEACH does not perform any evaluation of the validity or quality of these research studies. Research studies that are specific for adults are not included in the TEACH Database, and typically are not described in the TEACH Chemical Summary.

I. INTRODUCTION

Polychlorinated biphenyls (PCBs) are a family of man-made organic chemicals with a common structure (a pair of benzene rings) that vary primarily in their degree of chlorination (1, 2). Each single form of PCB is called a congener, and is often identified by number (e.g., PCB 153). PCBs in pure form are odorless or mildly aromatic solids or oily liquids, often found in mixtures with other organic chemicals (1, 2).

PCBs have been present in a variety of types of industrial equipment (e.g., electrical, heat transfer, and hydraulic equipment) and consumer products (e.g., plasticizers in paints, plastics, and rubber products), although the manufacturing of PCBs was stopped in the U.S. in 1977. PCB levels in the U.S. are decreasing, although PCBs continue to persist in the environment, and to bioaccumulate in fish and other animals. PCBs also exist in machinery built before 1977 (e.g., fluorescent light fixtures and electrical appliances) and at some hazardous waste sites (1). PCBs often occur in mixtures of congeners, some of which were used in applications and are known by their industrial trade name (Aroclor, Clophen, and Kanechlor). Some combinations of congeners are identified by a number; for example, Aroclor 1254 is a mixture of mono- to heptachlorinated biphenyl congeners with an average chlorine content of 54% (indicated in the second half of the Aroclor number).

An important route of exposure for children is ingestion of foods containing PCBs, including fish and other foods (3-9), and human breast milk (10-30). Children living near old landfills, incinerators, and hazardous waste sites are also at risk for PCB exposure from contaminated drinking water and incidental ingestion of contaminated dirt after putting dirty hands or dirt-covered objects in their mouths (1).

The primary targets of PCBs are the endocrine (hormonal) and nervous systems (1). PCB exposure during prenatal and early childhood development has been associated with low birth weight (31-38), neurobehavioral developmental delays (reviewed in (39-47), cognitive deficits (reviewed in (39-44, 48, 49), changes in production of thyroid hormones (reviewed in (50-52), and altered reproductive system development in males and females (reviewed in (53-56). PCB exposure has also been associated with chloracne (a specific type of often severe and persistent skin lesion), with liver damage in humans, and with liver cancer in experimental animals (1).

II. EXPOSURE MEDIA AND POTENTIAL FOR CHILDREN'S EXPOSURE [1]

Exposure Media	Relative Potential for Children's Exposure[2,3]	Basis[4]
Diet	Higher	Diet is a major source of exposure. PCBs can be found in fish, meat, and dairy products, and tend to bioaccumulate in animal fats. PCBs have also been detected in human breast milk (see Considerations for Decision Makers).
Sediment	Medium	PCBs can remain in sediment for many years, and the PCBs that persist are often called the "weathered" PCBs. Persistent weathered PCBs often contain more highly chlorinated PCBs (with 6-9 chlorine atoms) than PCBs recently released into the environment. Highly chlorinated PCBs may be more toxic than PCBs with lower chlorination. PCBs in sediment can bioaccumulate in fish.
Soil	Medium	PCBs can remain in soil for many years with limited degradation.
Ambient Air	Lower	Generally, low levels of PCBs are found in ambient air; in cases of disruption or movement of PCB-containing materials, PCBs might be released to nearby air.
Indoor Air	Lower	Generally, low levels of PCBs are found in indoor air.
Surface Water	Lower	Most PCBs partition to sediment, solids, or oils in water, not the water column; generally, low levels of PCBs have been found in surface water.
Drinking Water	Lower	PCB contamination of drinking water can be a concern in areas close to sites contaminated with PCBs.

[1] For more information about child-specific exposure factors, please refer to the "Child-Specific Exposure Factors Handbook" (*http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55145*).

[2] The Relative Potential for Children's Exposure category reflects a judgment by the TEACH Workgroup, U.S. EPA, that incorporates potential exposure pathways, frequency of exposure, level of exposure, and current state of knowledge. Site-specific conditions may vary and influence the relative potential for exposure. For more information on how these determinations were made, go to *http://www.epa.gov/teach/teachprotocols_chemsumm.html*.

[3] Childhood represents a lifestage rather than a subpopulation, the distinction being that a subpopulation refers to a portion of the population, whereas a lifestage is inclusive of the entire population.

[4] Information described in this column was derived from several resources (e.g., 1) including studies listed in the TEACH Database (*http://www.epa.gov/teach*).

III. TOXICITY SUMMARY[5, 6]

Acute high dose exposure to PCBs resulted in chloracne (distinctive skin lesions) in children and adults, which can be severe and disfiguring lesions, and persist from 1 year to permanently (1, 53, 57, 58). Very high dose PCB exposure (including exposure to furans) has also been associated with an increased incidence of respiratory problems and ear infections in children (59-61). Acute prenatal exposure resulted in intrauterine growth retardation, reduced birth weight, delayed developmental milestones, and other abnormalities in infants and children (62-64). Acute exposure has also been associated with liver and kidney damage in adults (1).

Chronic PCB exposure has been associated with other health effects. In adult males, decreased sperm integrity or sperm counts were associated with increasing blood PCB concentrations (65-68). Increased cord blood PCB concentrations were significantly associated with lower birth weight in most (31-35, 37, 38, 69), but not all studies (70, 71).

In children, neurological and behavioral effects were associated with increased blood PCB concentrations, including reduced I.Q., reduced scores on neurobehavioral assessments, memory deficits, and learning deficits (25, 39-49, 72-84). PCB exposure of children and pregnant women has been associated with changes in thyroid hormone levels in infants and children (50-52, 85-92) and changes in the developing reproductive system (53-56). In children, prenatal PCB exposure was also associated with impaired response inhibition (i.e., ability to withhold responding to irrelevant stimuli), and with decreased volume (MRI-measured) of the splenium of the corpus callosum, a brain structure related to this behavior (93, 94).

Some studies reported significant associations between chronic PCB exposure and impaired ability to fight infection and other immune system effects in children (13, 59-61, 95-99).

Experimental animal studies have shown an association between chronic PCB exposure and increased incidence of liver tumors in adult animals (1). Increased numbers of cervical, vaginal, and mammary tumors per mouse was reported in adult experimental animals who were neonatally exposed to PCBs (100, 101). Numerous experimental animal studies have reported a range of health effects following developmental exposure to PCBs. Effects included reduced birth weight following maternal PCB exposure during pregnancy (102-104). Neurodevelopmental delays (40, 45, 46) and reproductive tract development changes (105-117) following developmental exposure have also been reported. Associations between developmental PCB exposure and thyroid hormone concentrations have also been reported (118-126).

Carcinogenicity Weight-of-Evidence Classification[7]: PCBs are classified by the U.S. EPA as B2, probable human carcinogens, based on liver tumors in adult rats (http://www.epa.gov/iris/subst/0294.htm, II.A.1) (127). The World Health Organization International Agency for Research on Cancer (IARC) in 1998 classified PCBs as Group 2A, probably carcinogenic in humans (http://monographs.iarc.fr/ENG/Monographs/vol18/volume18.pdf) (128).

[5] Please refer to research article summaries listed in the TEACH Database for details about study design considerations (e.g., dose, sample size, exposure measurements).

[6] This toxicity summary is likely to include information from workplace or other studies of mature (adult) humans or experimental animals if child-specific information is lacking for the chemical of interest. Summaries of articles focusing solely on adults are not listed in the TEACH Database because the TEACH Database contains summaries of articles pertaining to developing organisms.

[7] For recent information pertaining to carcinogen risk assessment during development, consult "Guidelines for Carcinogen Risk Assessment and Supplemental Guidance on Risks from Early Life Exposure" at *http://www.epa.gov/cancerguidelines*.

IV. EXPOSURE AND TOXICITY STUDIES FROM THE TEACH DATABASE

This section provides a brief description of human and animal studies listed in the TEACH Database. These descriptions generally include the overall conclusion in each study without evaluation or assessment of scientific merit by TEACH. For more details about doses and exposure levels, query the TEACH Database. Any consideration of adverse events should include an understanding of the relative exposure on a body weight basis. In many cases, exposure levels in animal studies are greater than exposure levels normally encountered by humans.

A. *HUMAN EXPOSURE AND EFFECTS*

Many human and experimental animal studies of developmental exposures to PCBs have been published, and almost 500 articles are listed in the TEACH Database. An overview and summary of large poisoning incidents and more recent developmental studies of PCB exposure are provided here. Summaries of additional articles can be obtained in the TEACH Database. Additional detailed and comprehensive discussions of available reproductive and developmental studies are provided elsewhere (1, 41).

- One key route of exposure to PCBs for children is via diet (1-9). PCBs bioaccumulate in the fatty tissue of fish and other animal products such as beef, chicken, and dairy (1, 3-8). PCBs have been detected in several species of fish and shellfish (3, 4). PCBs have been detected in some fish oil supplements manufactured outside the U.S. (129). See Considerations for Decision Making for more information about PCB concentrations in foods and fish oil supplements.
- PCBs have been detected in maternal blood or serum (36, 130-139), cord blood (11-15, 130, 137, 138, 140-146), fetal adipose tissue (147), placenta (12, 26), infant blood (146, 148-150), children's blood or serum (11, 12, 15, 136, 143, 151-157), and breast milk (10-26, 28-30).
- Prenatal exposure during pregnancy to high levels of PCBs has occurred in two separate large-scale poisoning incidents (reviewed in (53, 57, 58). The Yusho (oil disease) incident in 1968 affected about 1,800 people in Japan who ingested cooking oil contaminated with PCBs and other chemicals. The Yu-Cheng (oil disease) incident in 1979 affected about 2,000 people in Taiwan, who ingested rice oil contaminated with PCBs. Prenatal exposure following maternal ingestion of these contaminated oils was associated with intrauterine growth retardation, low birth weights, delayed developmental milestones, abnormal behaviors, chloracne (skin lesions), shortened finger bones, and other abnormalities (56-60, 62-64, 158).
- Possible effects of PCB exposure on sperm development have been investigated (65-68, 159-161). Serum PCB concentrations in adult men were significantly associated with decreased sperm integrity (a measure of sperm quality) in men from 3 out of 5 countries tested (68). Another study found significantly greater percentages of abnormal sperm in men exposed 20 years prior in the Yucheng poisoning incident, as compared to men who were not exposed (65). A third study found significantly decreased sperm motility (movement), but no significant differences in sperm counts, associated with serum PCB 153 concentrations in adult men (66).
- The birth weight of infants following maternal PCB exposure during pregnancy has been studied (reviewed in (31, 67). Maternal serum PCB concentrations were associated with significantly lower birth weight in some studies (32-38); in other studies there was no difference (70, 162) or increased birth weight (71). Differences in results may be due in part to the different congeners of PCBs measured, or different mixtures of PCBs to which individuals were exposed (31).

- Neurological and behavioral effects have been associated with prenatal and lactational exposure to PCBs (39-44, 46-49, 163-166). Significant effects included decreased scores on the Intelligence Quotient (I.Q.) assessment, memory deficits, learning deficits, and lower scores on tests of neurobehavioral parameters (25, 72-83). For example, highly-chlorinated PCB concentrations in newborn blood were significantly associated with lower scores on neurobehavioral tests in infants in one study (73) but not another (167). In children, prenatal PCB exposure was also associated with impaired response inhibition (i.e., ability to withhold responding to irrelevant stimuli), and with decreased volume (MRI-measured) of the splenium of corpus callosum, a brain structure related to this behavior (93, 94).
- Some PCBs are classified as endocrine disruptors, or hormonally-active compounds, because of the ability of some congeners to modify the endocrine system (1, 53-55, 85). Effects on the reproductive hormonal system have been observed. Increased maternal serum or cord blood PCB concentrations were associated with earlier age of onset of menstruation (168) and significantly shorter menstrual cycle length in their daughters (56).
- Effects of PCB exposure on maternal, fetal, infant, and children's thyroid hormone concentrations have been investigated (reviewed in (50-52). Changes in levels of some thyroid hormones in infants and children have been associated with PCB exposure (85-92). For example, increasing placental PCB concentrations were associated with decreasing fetal T4 levels (90). Increased maternal concentrations of microsomal-inducing PCBs were associated with increased infant thyroid stimulating hormone concentrations (91).
- In infants, some immune system effects have been reported to be significantly associated with PCB exposure during pregnancy (13, 60, 61, 95, 99). For example, increased cord blood PCB concentrations were significantly associated with increased susceptibility to ear infections in one study (13) but not another (169). Also, higher cord blood PCB concentrations were associated with a higher incidence of respiratory problems and ear infections in infants (60, 61).
- In children, some immune system effects were associated with prenatal or early life exposure to PCBs (13, 59, 61, 96-99, 170). For example, one study demonstrated subtle immune response changes (e.g., increased numbers of total lymphoctyes and cytotoxic T lymphocytes) in children at 42 months of age that correlated with PCB exposure (96). In a separate study, changes in several immune system parameters (e.g., percentage of eosinophils, natural killer cells, and T lymphocytes; antibodies to allergens; and frequency of re-occurrence of childhood diseases) were significantly associated with total PCB body burden (97).
- Children exposed to high levels of PCBs in the Yusho and Yu-Cheng poisoning incidents (see the Introduction) experienced chloracne (skin lesions), eye discharge, hyperpigmentation, fatigue, and nausea (57, 58, 63, 158). A 24-year follow-up of this group reported increased mortality (death) from liver problems and Lupus (171).

B. *EXPERIMENTAL ANIMAL EXPOSURE AND EFFECTS*

Many studies have been performed which have examined developmental effects of prenatal and early life exposures to PCBs in experimental animals. Studies have examined effects of single congeners of PCBs and PCB Aroclors (see Introduction). The studies included in this section are representative of available studies, which in total were too numerous to include here. Additional studies are summarized in the TEACH Database.

- A physiologically-based pharmacokinetic model of lactational transfer of PCB 153 with or without PCB 126 in mice was reported (172).
- Ingestion of PCBs was associated with adverse reproductive effects in adult experimental animals (including rats, mice, minks, and monkeys) as well as their offspring. Reproductive effects in females included prolonged estrus, decreased sexual receptivity, decreased implantation rate, decreased conception rate, prolonged menstruation, and decreased fertility (106-108, 173). Reproductive effects in males included decreased ability of sperm to fertilize eggs (109), decreased seminal vesicle weight and sperm counts (109-112, 117), and increased abnormalities in sperm morphology (113, 114). Also, prenatal exposure of rats to Aroclor 1254 resulted in prolonged estrous periods in adult females (115) and smaller testes in adult males (116).
- PCBs have been shown to be toxic to fetuses and teratogenic (cause birth defects) during gestation when administered at high dosages (174-178). Observed effects included fetal cleft palate (174-178), decreased thymus size (176), and decreased numbers of live pups per litter (175-178).
- Developmental toxicity was observed in offspring exposed to PCBs during gestation and/or lactation (reviewed in (179, 180). For example, Aroclor 1254 delivered by gavage to pregnant rats caused a reduction in birth weight and postnatal growth (104). In monkeys, maternal exposure to Aroclor 1016 and 1248 via diet resulted in significant reductions in birth weight (103). In contrast, there was no significant difference in birth weight in a separate study of low-dose exposure to Aroclor 1254 in monkeys (107).
- Neurological effects have been observed in offspring following prenatal or lactational exposure to PCBs (reviewed in (40, 45, 46). Observed effects in rats and monkeys included delayed development of air righting ability (an index of neuromuscular maturation), delayed onset of the startle response, and decreased performance in learning or memory tests (116, 181-188). One study in rats showed no effect in two behavioral tests in offspring following maternal PCB exposure via ingestion during pregnancy and lactation (189). Several specific neurochemical changes have been identified in specific regions of the brain following PCB exposure, e.g., decreased dopamine levels in the frontal cortex and increased serotonin and serotonin metabolites in several brain regions (190-197).
- Some effects on neurological development following early life PCB exposure were shown to persist well after exposure was stopped (reviewed in (40, 45, 46). For example, monkeys exposed to a PCB mixture (from birth to 20 weeks of age via diet) showed learning deficits when tested 3 years after exposure was stopped (183-185, 198); PCB concentrations in these studies were comparable to those found in human breast milk.
- Effects of early life PCB exposure on tumor incidence has been investigated. Significantly increased tumor incidence of a variety of tumor types during adulthood was observed following early life exposure (during the first 5 days of birth and then stopped) of rats to hydroxylated PCBs (PCB metabolites) (100). In rats treated during early life with PCBs prior to treatment with the mammary-tumor-inducing agent methylnitrosourea (MNU), significantly increased numbers of mammary tumors were observed in one study (101) but not another (199).

- Immunological effects have been studied in animals exposed to PCBs during gestation and/or lactation (reviewed in (99). For example, infant monkeys exposed to Aroclor 1254 *in utero* and postnatally via breast milk had reductions in serum concentrations of some specific antibodies (192, 200). Other immune system effects included reduced interleukin (IL)-2 production in mice (201), and reduced NK cell activity in rats (202).
- Thyroid hormone regulation has been shown to be affected in rats following prenatal and/or lactational exposure to either mixtures or individual congeners of PCBs. A decrease in circulating thyroxine (T4) levels has been consistently seen in mothers and their offspring, while effects on triiodothyronine (T3) levels have been more variable and may depend on the type of PCB to which animals were exposed (118-126). Injection of T4 reduced the neurological deficits which occurred following prenatal exposure to Aroclor 1254 (118).

V. CONSIDERATIONS FOR DECISION-MAKERS

This section contains information that may be useful to risk assessors, parents, caregivers, physicians, and other decision-makers who are interested in reducing the exposure and adverse health effects in children for this particular chemical. Information in this section focuses on ways to reduce exposure, assess possible exposure, and, for some chemicals, administer treatment.

A. *Information about Reducing or Preventing Exposure to PCBs*

- Particular consideration should be given to women of childbearing age and their infant children for preventing PCB exposures because PCBs cross the placenta and can be transferred via breast milk (1, 10).
- The CDC stated that, "in most cases, the benefits of breast feeding outweigh any risks from exposure to PCBs in mother's milk" (1). Therefore, even though PCBs are known to be present in breast milk, women are encouraged to breast feed their infants. This recommendation may be modified, however, in circumstances of acute, high dose PCB exposures (1).
- Reducing children's exposures to PCBs in the diet is best accomplished by complying with recommendations of fish advisories. Pregnant women, women of childbearing age, and children are usually considered sensitive populations, and are given recommendations for number of servings of specific types of fish to eat weekly or monthly, depending on PCB concentrations (203, 204). Fish that are bottom feeders, such as catfish, and large freshwater fish that are higher up on the food chain (i.e., large Chinook salmon) are likely to have higher PCB levels. Recently, PCB levels were found to be high in many farmed Atlantic salmon, and the PCBs were thought to have come from the fish meal fed to these salmon (205, 206). For recent updates on contaminant levels in fish, go to http://epa.gov/waterscience/fish/ (204).
- Sports fisherman and their families who consume the fish they eat should be aware of fish advisories for local waters (207). In the U.S., PCBs are included in several fish contaminant monitoring programs at tribal, state, and national levels (204, 207-209). Local municipal or state public health offices can be consulted for specific fish advisories.

- Exposure to PCBs is also of concern for children who live close to some landfills, incinerators, and hazardous waste sites (1). Children may be exposed though incidental ingestion of PCB-contaminated soil and through contaminated drinking water. Workers exposed to PCBs can also carry PCBs home on their clothing, which contribute to their children's exposures to PCBs in house dust (1, 210).

B. *Other Information about PCBs*

- PCB concentrations in serum, adjusted for lipid content, of people twelve years of age and older were measured as part of the National Health and Nutrition Examination Survey (NHANES), and results for numerous congeners have been reported since 1999 (157, 211).
- Detailed compilations and analyses of information pertaining to exposure and health effects of PCBs are available in the Toxicological Profile for Polychlorinated Biphenyls (1). Summaries of this detailed information are also available (212, 213).
- Several PCB congeners are identified as having dioxin-like activity via binding to the aryl hydrocarbon receptor (AhR) and are predominantly non-ortho substituted coplanar PCBs (e.g., PCB 78, 81, 126, 169). Dioxin-like PCBs constitute a minority of PCBs found in environmental and biological samples. Dioxin-like PCBs share a similar mechanism of action (MOA) as dioxin, and have been shown to lead to dioxin-like adverse health effects (e.g., immunotoxicity, developmental toxicity, and hormonal changes). Dioxin-like PCBs are considered in risk assessments for dioxins and dibenzofurans, with application of toxic equivalency factors (TEQs) as a measure of toxicity relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). Extensive information about exposure and risk characterization for dioxin-like PCBs is available (214, 215).
- The U.S. EPA used 2002 PCB air emissions data for all 50 states to report county-level emissions, modeled ambient air concentration estimates, modeled human exposure estimates, and estimated risk (216).
- PCBs as a group are ranked as number 5 on the 2005 Priority List of Hazardous Substances for the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) section 104(i), as amended by the Superfund Amendments and Reauthorization Act (SARA). This is a list, in the order of priority of concern, of substances most commonly found at sites listed on the National Priorities list (NPL); there are currently 275 substances on this list. In addition to listing PCBs as a group on this Priority List, 10 Aroclors are listed. The highest ones are Aroclor 1254 as number 13 and Aroclor 1260 as number 14 on the priority list (217).
- Consult the U.S. EPA "Child-Specific Exposure Factors Handbook" (EPA/600/R-06/096F) for factors to assess children's ingestion rates (218).

VI. TOXICITY REFERENCE VALUES

Polychlorinated Biphenyls as a group (CASRN 1336-36-3)

A. Oral/Ingestion

The U.S. EPA used a tiered approach to determine the cancer potency of PCB mixtures that depends on the information available to the U.S. EPA at the time that reference values were calculated. Details about risk, persistence, and criteria for use of different slope factors is available on the IRIS Web site (www.epa.gov/iris/subst/0294.htm, II.B.3) (127). The 3 tiers are 1) high risk and persistence; 2) low risk and persistence; and 3) lowest risk and persistence. The U.S. EPA provides an upper-bound slope factor and central-estimate slope factor for each tier, and provides criteria for use for each slope factor.

"Because of the potential magnitude of early-life exposure, the possibility of greater perinatal sensitivity, and the likelihood of interactions among thyroid and hormonal development, it is reasonable to conclude that early-life exposures may be associated with increased risks. Due to this potential for higher sensitivity early in life, the 'high risk' tier is used for all early-life exposure" (www.epa.gov/iris/subst/0294.htm, II.B.3) (127).

U.S. EPA Cancer Oral Slope Factor: High risk and persistence, upper-bound: 2.0 per (mg/kg)/day; low risk and persistence, upper-bound, 0.4 per (mg/kg)/day; lowest risk and persistence, upper-bound, 0.07 per (mg/kg)//day, based on liver tumors in adult rats (www.epa.gov/iris/subst/0294.htm, II.B.3) (127). Last Workgroup Verification Date 8/22/96.

U.S. EPA Cancer Drinking Water Upper Bound Unit Risk: 1E-5 (or 0.00005) per ppb (µg/L) (upper-bound, unit risk), based on liver tumors in adult rats; these estimates should not be used if drinking water concentrations exceed 1000 µg/L; for food chain exposure or ingestion that includes contaminated sediment or soil, the slope factor for "high risk and persistence" should be used instead (www.epa.gov/iris/subst/0294.htm, II.B.3) (127). Last Workgroup Verification Date 8/22/96.

U.S. EPA Drinking Water Concentrations at Specified Risk Levels: 1E-4 (or 1 in 10,000), 10 µg/L; 1E-5 (or 1 in 100,000), 1 µg/L; 1E-6 (or 1 in 1,000,000), 0.1 µg/L (www.epa.gov/iris/subst/0294.htm, II.B.3) (127). Last Workgroup Verification Date 8/22/96.

U.S. EPA Maximum Contaminant Level (MCL) for Drinking Water: 0.0005 mg/L, based on skin changes, thymus gland problems, immune deficiencies, reproductive or nervous system difficulties, and increased risk of cancer in adults (http://www.epa.gov/safewater/contaminants/index.html) (219). Last revised 5/09.

U.S. EPA Maximum Contaminant Level Goal (MCLG): 0 mg/L. Last revised 5/09.

Reference Toxicity Values continued on next page

B. Inhalation

U.S. EPA Inhalation Upper Bound Unit Risk: 1x10-4 per µg/cu.m. (www.epa.gov/iris/subst/0294.htm, II.C.3) (127). Last Workgroup Verification Date 8/22/96.

U.S. EPA Air Concentrations at Specified Risk Levels: 1E-4 (or 1 in 10,000), 1 µg/cu.m.; 1E-5 (or 1 in 100,000), 0.1 µg/cu.m.; 1E-6 (or 1 in 1,000,000), 0.01 µg/cu.m.; these estimates should not be used if ambient air concentrations exceed 100 µg/cu.m.; for inhalation of an aerosol or dust contaminated with PCBs, the slope factor for "high risk and persistence" should be used instead (www.epa.gov/iris/subst/0294.htm, II.C.3) (127). Last Workgroup Verification Date 8/22/96.

U.S. EPA Carcinogenic Risk from Inhalation Exposure Air Unit Risk: 0.4 per (mg/kg)/day (upper-bound, slope factor), based on liver tumors in adult rats (www.epa.gov/iris/subst/0294.htm, II.C.3) (127). Last Workgroup Verification Date 8/22/96.

Aroclor 1016 (CASRN 12674-11-2)

A. Oral/Ingestion

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: 7E-5 (or 0.00007) mg/kg-day based on reduced birth weight in monkeys (www.epa.gov/iris/subst/0462.htm, I.A.1) (220). Last Workgroup Verification Date 11/4/92.

Aroclor 1248 (CASRN 12672-29-6)

A. Oral/Ingestion

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: The health effects data for Aroclor 1248 were determined to be inadequate for the derivation of an oral RfD (www.epa.gov/iris/subst/0649.htm, I.A.1) (221). Last Workgroup Review Date 7/20/93.

Aroclor 1254 (CASRN 11079-69-1)

A. Oral/Ingestion

U.S. EPA Reference Dose (RfD) for Chronic Oral Exposure: 2E-5 (or 0.00002) mg/kg-day based on ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails, and decreased antibody (IgG and IgM) response to sheep erythrocytes in monkeys (http://www.epa.gov/iris/subst/0389.htm, I.A.1) (222). Last Workgroup Verification Date 2/16/94.

ATSDR Minimal Risk Level (MRL): 0.03 µg/kg-day (oral intermediate; neurological endpoint in monkeys); 0.02 µg/kg-day (oral chronic; immunological endpoint in monkeys) (http://www.atsdr.cdc.gov/mrls/index.html) (223). Last revised 12/08.

VII. U.S. FEDERAL REGULATORY INFORMATION

- The U.S. FDA has set tolerance levels of 2 parts per million PCBs in the edible portion of fish in the commercial food supply and sold in interstate commerce (224). The U.S. FDA periodically tests fish sold in markets for levels of PCBs and other contaminants, and confiscates products that contain PCB levels that exceed tolerance levels. It is not within the jurisdiction of the U.S. FDA to monitor PCB concentrations in supplements, including fish oil (224, 225).
- The U.S. FDA has set tolerance levels between 0.2-3 ppm in other foods, with the lowest tolerance level of 0.2 ppm for infant and junior foods (226). The U.S. FDA also has set tolerances for PCBs in bottled water at a level of 0.0005 mg/L (227).
- PCBs are listed as a group as one of 188 hazardous air pollutants (HAPs) listed under section 112(b) of the 1990 Clean Air Act Amendments and is regulated from more than 170 industrial source categories (228).
- The U.S. EPA requires reporting of quantities of certain chemicals that exceed a defined reportable quantity, and that quantity varies for different chemicals (229). PCBs are classified under the Emergency Planning and Community Right-to-Know Act (EPCRA) Section 313 "Toxic Chemicals" as persistent, bioaccumulative and toxic compounds (PBT) and as such, reporting quantities of PCBs greater than 10 pounds manufactured or processed, or otherwise used, is required; PCB congeners that are dioxin-like have a reporting threshold of 0.1 grams. Under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), reporting releases of PCBs of any quantity exceeding 1 pound is required. Reporting releases of any quantity exceeding 1 pound for each of Aroclor 1016, 1221, 1232, 1242, 1248, 1254, and 1260 is also required under CERCLA (229).

VIII. BACKGROUND ON CHEMICAL

CAS Number: Polychlorinated biphenyls, 1336-36-3; Aroclor 1016, 12674-11-2; Aroclor 1248, 12672-29-6; Aroclor 1254, 11079-69-1.

Physicochemical Properties: PCBs are a class of compounds in which 1 to 10 chlorine atoms are attached to the biphenyl structure (1). PCBs are subdivided according to degree of chlorination and there are 209 possible chlorobiphenyl congeners. Commercial PCB mixtures are known in the United States by their industrial trade name, Aroclor. PCBs can be found as odorless or mildly aromatic solids or oily liquids, often in mixtures with other organic compounds. For more information, go to the National Library of Medicine ChemID Web site (*http://chem.sis.nlm.nih.gov/chemidplus)* and search for PCBs or polychlorinated biphenyls.

Production: PCBs were produced commercially in the U.S. from 1929 until 1979 (1). Annual U.S. production peaked in 1970 with a volume of 85 million pounds of Aroclors (1).

Uses: Before 1974, PCBs were used both in nominally closed (e.g., capacitor and transformers; minimal volatilization) and in open-ended (e.g., plasticizers, surface coatings, inks, adhesives, pesticide extenders, and carbonless duplicating paper) applications (1). By 1974, use of PCBs in the U.S. was restricted to nominally closed applications such as the production of capacitors and transformers. After 1977, Aroclors were no longer used in the production of new capacitors (1). The U.S. EPA Toxic Release Inventory (TRI) reported PCB total releases and disposals of over 1.6 million pounds in 2007; total releases are likely to be greater than this estimate because not all sources of PCB releases are required to report (230).

Environmental Fate: PCBs can persist in the environment for long periods of time (1). Before 1979, PCBs entered the environment during their manufacture and use. Since then, PCBs have continued to be released into the environment from hazardous waste sites, illegal or improper dumping, and leaks from PCB-containing electrical transformers (1). PCBs may be carried long distances in the air before depositing onto soil, vegetation, and water bodies. PCBs accumulate in the sediment where they can persist and act as a source of contamination of the food chain over a period of years (1, 8). PCBs in water bioaccumulate, or build up, in fish and marine mammals, and can reach levels thousands of times higher than the levels in water (1, 209).

Synonyms and Trade Names: Aroclor, Arochlor 1221, Arochlor 1232, Arochlor 1242, Arochlor 1248, Arochlor 1254, Arochlor 1260, Arochlor 1262, Arochlor 1268, Arochlor 2565, Arochlor 4465, Arochlor 5442, Biphenyl, Polychloro-chlophen, Chlorextol, chlorinated biphenyl, and more; for a more complete list, go to the National Library of Medicine ChemID Web site (*http://chem.sis.nlm.nih.gov/chemidplus*) and search for PCBs or polychlorinated biphenyls.

Additional information on PCBs is available in the TEACH Database for PCBs, and at the following Web sites:

http://www.epa.gov/pcb/
http://www.epa.gov/ogwdw/dwh/c-soc/pcbs.html
http://www.epa.gov/waterscience/fish/pcb99.html
http://www.epa.gov/epaoswer/hazwaste/minimize/factshts/pcb-fs.pdf
www.atsdr.cdc.gov/tfacts17.html
www.epa.gov/glnpo/sediments.html

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