

## 6. POTENTIAL FOR HUMAN EXPOSURE

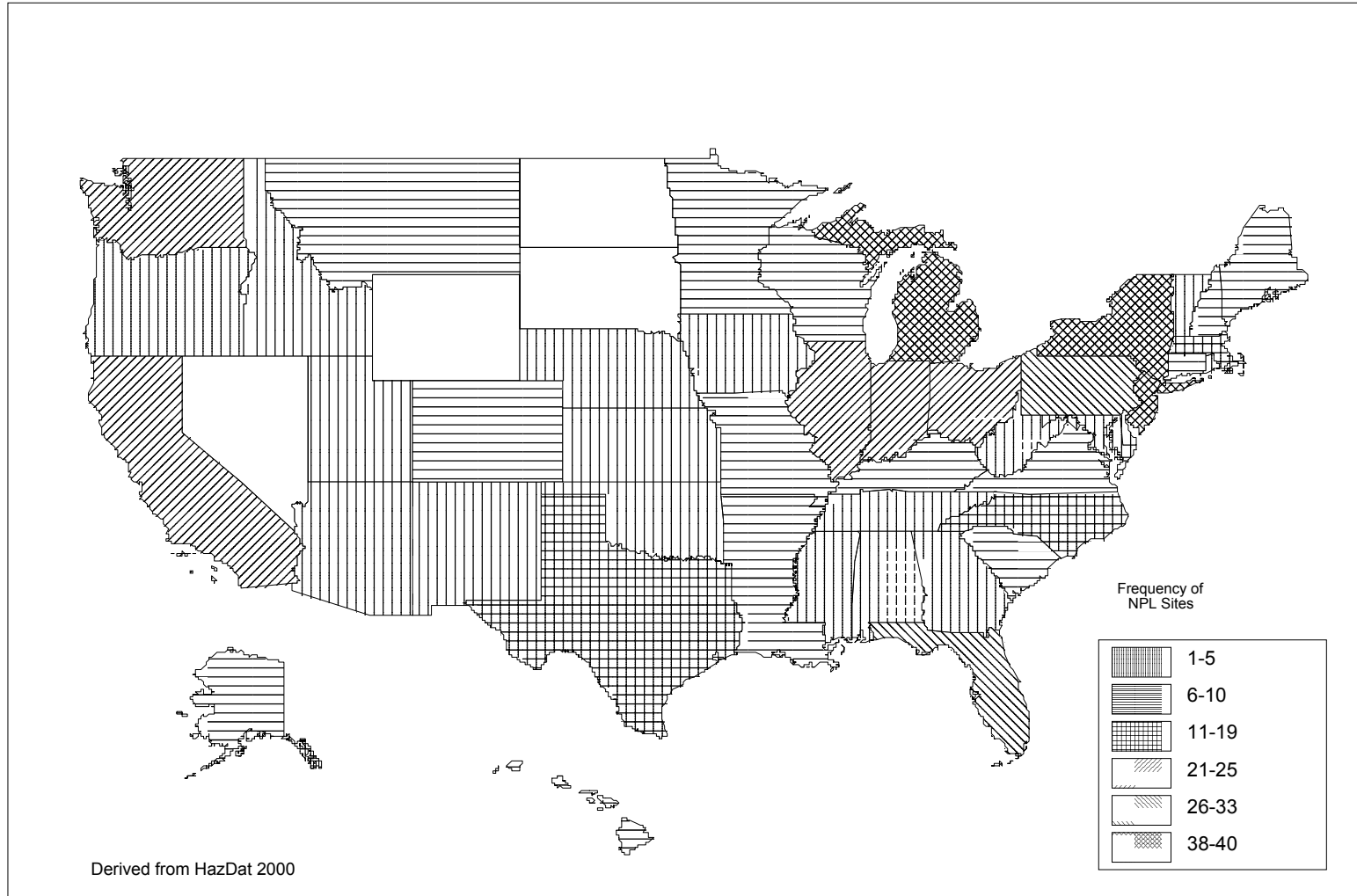
### 6.1 OVERVIEW

Polychlorinated biphenyls (PCBs) have been identified in at least 500 of the 1,598 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2000).

However, the number of sites evaluated for PCBs is not known. The frequency of these sites can be seen in Figure 6-1. Of these sites, 499 are located within the United States and 1 is located in the U.S. Territory of Guam (not shown).

PCBs have been released to the environment solely by human activity. Aroclors are no longer produced in the United States, except under exemption (see Section 5.3), and are no longer used in the manufacture of new products. Because PCBs are no longer manufactured or imported in large quantities, significant releases of newly manufactured or imported materials to the environment do not occur. Rather, PCBs predominantly are redistributed from one environmental compartment to another (e.g., soil to water, water to air, air to water, sediments to water) (Eisenreich et al. 1992; Larsson 1985; Larsson and Okla 1989; Lin and Que Hee 1987; Mackay 1989; Murphy et al. 1985, 1987; Swackhamer and Armstrong 1986). Thus, for example, the majority of PCBs in air result from volatilization of PCBs from soil and water. Some PCBs may be released to the atmosphere from uncontrolled landfills and hazardous waste sites; incineration of PCB-containing wastes; leakage from older electrical equipment in use; and improper disposal or spills (Blumbach and Nethe 1996; Boers et al. 1994; Bremle and Larsson 1998; Eisenreich et al. 1992; Hansen and O'Keefe 1996; Hansen et al. 1997; Hermanson and Hites 1989; Larsson 1985; Lewis et al. 1985; Morselli et al. 1985, 1989; Murphy et al. 1985; Oehme et al. 1987; Sakai et al. 1993; Sawhney and Hankin 1985; Swackhamer and Armstrong 1986; Tiernan et al. 1983; Wallace et al. 1996). PCBs may be released to water from accidental spillage of PCB-containing hydraulic fluids; improper disposal; combined sewer overflows (CSOs) or storm water runoff; and from runoff and leachate from PCB-contaminated sewage sludge applied to farmland (Crawford et al. 1995; Durell and Lizotte 1998; Gan and Berthouex 1994; Gunkel et al. 1995; Loganathan et al. 1997; Pham and Proulx 1997; Shear et al. 1996). PCBs may be released to soil from accidental leaks and spills; releases from contaminated soils in landfills and hazardous waste sites; deposition of vehicular emissions near roadway soil; and land application of sewage sludges containing PCBs (Alcock et al. 1995; Benfenati et al. 1992; Choi et al. 1974; Gan and Berthouex 1994; Gutenmann et al. 1994; Liberti et al. 1992; McLachlan et al. 1994; Morris and Lester 1994; O'Connor et al. 1990; Ohsaki and Matsueda 1994).

Figure 6-1. Frequency of NPL Sites with PCB Contamination



Derived from HazDat 2000

## 6. POTENTIAL FOR HUMAN EXPOSURE

PCBs are globally circulated and are present in all environmental media. Atmospheric transport is the most important mechanism for global dispersion of PCBs. Biphenyls with 0–1 chlorine atom remain in the atmosphere, those with 1–4 chlorines gradually migrate toward polar latitudes in a series of volatilization/deposition cycles, those with 4–8 chlorines remain in mid-latitudes, and those with 8–9 chlorines remain close to the source of contamination (Wania and Mackay 1996). PCBs enter the atmosphere from volatilization from both soil and water surfaces (Hansen 1999). Once in the atmosphere, PCBs are present both in the vapor phase and sorbed to particles. PCBs in the vapor phase appear to be more mobile and are transported further than particle-bound PCBs (Wania and Mackay 1996). Wet and dry deposition remove PCBs from the atmosphere (Dickhut and Gustafson 1995; Eisenreich et al. 1981; Golomb et al. 1997; Hoff et al. 1996; Leister and Baker 1994; Nelson et al. 1998). The dominant source of PCBs to surface waters is atmospheric deposition; however, redissolution of sediment-bound PCBs also accounts for water concentrations (Hansen 1999). PCBs in water are transported by diffusion and currents. PCBs are removed from the water column by sorption to suspended solids and sediments as well as by volatilization from water surfaces. Higher chlorinated congeners are more likely to sorb, while lower chlorinated congeners are more likely to volatilize (Eisenreich et al. 1983, 1992; Pearson 1996). PCBs also leave the water column by concentrating in biota. PCBs accumulate most in higher trophic levels through the consumption of contaminated food, a process referred to as biomagnification (EPA 1983c; Geyer et al. 1999; Koslowski et al. 1994; Looser and Ballschmiter 1998; Oliver and Niimi 1988; Porte and Albaiges 1993; Willman et al. 1999; Wilson et al. 1995). PCBs in soil are unlikely to migrate to groundwater because of strong binding to soil (EPA 1979h, 1988a; Sklarew and Girvin 1987). Volatilization from soil appears to be an important loss mechanism; it is more important for the lower chlorinated congeners than for the higher chlorinated congeners (Hansen 1999). Vapor-phase PCBs accumulate in the aerial parts of terrestrial vegetation and food crops by vapor-to-plant transfer (Bohm et al. 1999).

The ability of PCBs to be degraded or transformed in the environment depends on the degree of chlorination of the biphenyl molecule as well as on the isomeric substitution pattern. The vapor-phase reaction of PCBs with hydroxyl radicals is the dominant transformation process in the atmosphere (Brubaker and Hites 1998), while photolysis appears to be the only viable chemical degradation process in water (EPA 1979h). Biodegradation has been demonstrated under both aerobic (Dowling et al. 1993; EPA 1983c, 1988a; Fava et al. 1993; Gibson et al. 1993; Haluska et al. 1995; Sugiura 1992; Thomas et al. 1992) and anaerobic conditions (Abramowicz 1990, 1995; Anid et al. 1993; Brown et al. 1988; Chen et al. 1988; EPA 1983c, 1988a; Larsson and Lemkemeier 1989; Pardue et al. 1988; Rhee et al. 1989) and is the major degradation process for PCBs in soil and sediment.

## 6. POTENTIAL FOR HUMAN EXPOSURE

Typical atmospheric concentrations of PCBs have been found to be much lower in rural locations compared to urban locales. For example, the concentration of PCBs in urban Baltimore, Maryland ranged from 0.38 to 3.36 ng/m<sup>3</sup>, while in rural Baltimore, the concentration ranged from 0.02 to 0.34 ng/m<sup>3</sup> (Offenberg and Baker 1999). PCB levels in more remote areas are even lower with mean concentrations ranging from 0.025 ng/m<sup>3</sup> over the Norwegian Sea to 0.074 ng/m<sup>3</sup> over the Eastern Arctic (Harner et al. 1998). Monitoring studies conducted over the years have shown that atmospheric concentrations of PCBs have decreased since the late 1970s. Water monitoring studies indicate that PCB concentrations are generally higher near sites of anthropogenic input and in in-shore waters. The concentration of PCBs in the waters of the Great Lakes (Superior, Michigan, Huron, Erie, and Ontario) typically range from 0.070 to 1.6 ng/L (Anderson et al. 1999). Concentrations of PCBs in drinking water are generally <0.1 µg/L and thus, drinking water is not considered a significant pathway for exposure. Concentrations of PCBs in most soils are generally <100 µg/kg; however, PCB concentrations in contaminated soils can be several orders of magnitude higher. Subsurface soil and sludge collected on-site at a New York hazardous waste site near Akwesasne (a Native American community) had maximum concentrations of 750 mg/kg and 41,500 mg/kg, respectively (ATSDR 1995). PCB concentrations in fish have been of particular interest due to their influence on human exposure. Composite fish samples from the U.S. North Coast analyzed from 1988 to 1991 had a mean PCB concentration of 1.64 µg/g wet weight (Kennish and Ruppel 1995). Chinook salmon sampled from Lakes Ontario and Huron from 1991 to 1994 had mean concentrations of 0.835 and 0.338 µg/g wet weight, respectively (Feeley and Jordan 1998). Even in remote areas, PCBs have been detected in fish tissue. For example, lake trout caught in the Sierra Nevada mountains from 1993 to 1994 had PCB concentrations ranging from 0.018 to 0.430 µg/g wet weight (Datta et al. 1999).

The general population may be exposed to PCBs by ingesting contaminated food, especially fish from contaminated waters, and by inhaling contaminated air. Food consumption has and continues to be the major contributor to body burden of PCBs in the general population. The estimated dietary intake of PCBs for an average adult was 0.027 µg/kg/day in 1978 and had declined to <0.001 µg/kg/day by 1991 (Gunderson 1995). Several studies indicate that diets high in fish, from PCB-contaminated waters, can significantly increase a persons dietary intake of PCBs. For example, it was found that the mean concentration of PCBs in blood of 252 males who frequently consumed contaminated fish was 4.8 ng/mL, while in 57 males who were infrequent consumers, the mean concentration was 1.5 ng/mL (Hanrahan et al. 1999). In child-bearing women, this can be especially important since PCBs can concentrate in breast milk. Infants who are breast fed may therefore be at increased risk for PCB exposure if the mother has a diet high in contaminated fish (Dewailly et al. 1993; Fitzgerald et al. 1998). PCB exposure has also been

## 6. POTENTIAL FOR HUMAN EXPOSURE

attributed to inhalation of indoor air especially at locations which still use electrical equipment containing PCBs.

The detection of PCBs in blood, adipose tissue, breast milk, and other tissue samples from the general population indicates widespread exposure to PCBs from environmental sources. People who live near hazardous waste sites where PCBs have been detected may be exposed primarily by consuming contaminated fish from adjacent waterbodies and by breathing air that contains PCBs. Children playing near these sites or adults working near these sites may be exposed to additional PCBs by dermal contact with PCB-contaminated soil and by ingesting contaminated soil from their unwashed hands. Despite the prohibition on production and the restrictions regarding PCB use (Section 5.3), occupational exposure to PCBs can be orders of magnitude higher than general population exposure (Section 6.5).

### 6.2 RELEASES TO THE ENVIRONMENT

From 1929 until 1977, approximately 99% of all PCBs used by U.S. industries were manufactured by the Monsanto Chemical Company at a production facility in Sauget, Illinois (Durfee 1976; IARC 1978). During that period, over 571,000 metric tons ( $1,250 \times 10^6$  pounds) of PCBs were produced and/or used in the United States (Erickson 1997; Hansen 1999). In 1976, the U.S. Congress banned the manufacture, processing, distribution in commerce, and use of PCBs under the Toxic Substances Control Act (TSCA) and the Resource Conservation and Recovery Act (RCRA). Exemptions may be granted to individual petitioners for use with optical microscopy, and for research and development (see Section 5.3; EPA 1998u).

Because PCBs are no longer manufactured or imported in large quantities, significant releases of newly manufactured or imported materials to the environment do not occur. Rather, PCBs predominantly are redistributed from one environmental compartment to another (e.g., soil to water, water to air, sediments to water) (Eisenreich et al. 1992; Larsson 1985; Larsson and Okla 1989; Lin and Que Hee 1987; Mackay 1989; Murphy et al. 1985, 1987; Swackhamer and Armstrong 1986). Thus, for example, the majority of PCBs in air result from volatilization of PCBs from soil and water. Some PCBs may be released to the atmosphere from uncontrolled landfills and hazardous waste sites; incineration of PCB-containing wastes; leakage from older electrical equipment in use; and improper disposal or spills (Blumbach 1996; Boers et al. 1994; Bremle and Larsson 1998; Eisenreich et al. 1992; Hansen et al. 1997; Hermanson and Hites 1989; Larsson 1985; Lewis et al. 1985; Morselli et al. 1985, 1989; Murphy et al. 1985; Oehme et al. 1987; Sakai et al. 1993; Sawhney and Hankin 1985; Swackhamer and Armstrong 1986; Tiernan et al.

## 6. POTENTIAL FOR HUMAN EXPOSURE

1983; Wallace et al. 1996). PCBs may be released to water from accidental spillage of PCB-containing hydraulic fluids; improper disposal; CSOs or storm water runoff; and from runoff and leachate from PCB-contaminated sewage sludge applied to farmland (Crawford et al. 1995; Durell and Lizotte 1998; Gan and Berthouex 1994; Gunkel et al. 1995; Loganathan et al. 1997; Pham and Proulx 1997; Shear et al. 1996). PCBs may be released to soil from accidental leaks and spills; releases from contaminated soils in landfills and hazardous waste sites; deposition of vehicular emissions near roadway soil; and land application of sewage sludges containing PCBs (Alcock et al. 1995; Benfenati et al. 1992; Choi et al. 1974; Gan and Berthouex 1994; Gutenmann et al. 1994; Liberti et al. 1992; McLachlan et al. 1994; Morris and Lester 1994; O'Connor et al. 1990; Ohsaki and Matsueda 1994).

**6.2.1 Air**

From 1929 to 1977, unknown quantities of PCBs were released to the air during Aroclor production and processing and when PCB-contaminated equipment was incinerated (Durfee 1976). Similarly, transformer and capacitor producers discharged PCB-containing wastes to air during the various filling processes (Durfee 1976). Emissions are no longer discharged into the air through production activities; however, emissions may be discharged during the overhaul, repair, or reuse of materials containing PCBs. PCBs may have been released to the atmosphere from various past uses containing PCBs, for example, plasticizers, surface coatings, inks, adhesives, flame retardants, pesticide extenders, paints, and micro-encapsulation of dyes for carbonless duplicating paper; and, in addition, from the accidental losses of PCB fluids from capacitors and transformers (EPA 1976a; IARC 1978; Safe 1984; Welsh 1995).

The major source of PCB release to the atmosphere (2 million pounds/year) is the redistribution of the compounds that are already present in soil and water (Eisenreich et al. 1992; Murphy et al. 1985). Smaller amounts of PCBs may be released to the atmosphere from uncontrolled landfills and hazardous waste sites containing transformers, capacitors, and other PCB wastes (Bremle and Larsson 1998; Hansen and O'Keefe 1996; Hermanson and Hites 1989; Lewis et al. 1985; Murphy et al. 1985); incineration of PCB-containing wastes due to incomplete combustion of PCBs (Blumbach 1996; Boers et al. 1994; Kurokawa et al. 1996; Sakai et al. 1993); leakage from older electrical equipment still in use (Wallace et al. 1996); explosions or overheating of transformers containing PCBs (Schechter and Charles 1991); and improper (or illegal) disposal or spills of the compounds to open areas (Larsson 1985; Morselli et al. 1985, 1989; Murphy et al. 1985; Oehme et al. 1987; Sawhney and Hankin 1985; Swackhamer and Armstrong 1986; Tiernan et al. 1983). Historically, the amount of PCBs released from landfills and incinerators have been estimated to be 10–100 kg/year (22–220 pounds/year) and 0.25 kg/stack/year

## 6. POTENTIAL FOR HUMAN EXPOSURE

(0.55 pounds/stack/year), respectively, and are small compared to the quantity of PCBs released into the atmosphere through cycling from environmental processes (Murphy et al. 1985). PCBs have been identified in 31 air samples collected at 500 of the 1,598 NPL hazardous waste sites where they were detected in some environmental media (HazDat 2000), as well as in the air surrounding landfills during fires (Ruokojarvi et al. 1995). EPA regulations under TSCA regarding the incineration of PCBs requires that the combustion efficiency should be at least 99.9% (EPA 1998b); however, the small percentage of the PCBs not destroyed by incineration will be released into the atmosphere. According to the Toxics Release Inventory (TRI), a total of 446 pounds of PCBs were directly released into the air by 10 of the 14 RCRA hazardous waste and solvent recovery industries that processed them in 1998 (TRI98 2000). The TRI data for 1998 (TRI98 2000) are shown in Table 6-1. The TRI data should be used with caution since only certain types of facilities are required to report.

### 6.2.2 Water

From 1929 to 1977, the Monsanto Chemical Company released some PCB-containing waste water to municipal sewers during Aroclor production and processing. Waterborne discharges of PCBs from the Monsanto plant, estimated to be <1 pound/day (0.45 kg/day) in 1974, were greatly reduced over the years leading up to production cessation (Durfee 1976). Similarly, transformer and capacitor producers also discharged PCB-containing wastes to municipal sewers (Durfee 1976). High levels of PCBs were also detected in waste water from the manufacture of carbonless copy papers; from leaking hydraulic fluids used in, for example, die cast machines in iron, steel, and aluminum foundries; from pulp and paper mill effluents due to recycling of waste papers containing carbonless copy papers; and from electrical industry waste water contaminated by, for example, accidental loss of capacitor and transformer fluids. These waste waters may also have been discharged directly into surface waters. Treated waste waters may also have entered surface waters indirectly via effluents discharged from municipal publicly owned treatment works (POTWs) and industrial treatment plants.

Currently, the major source of PCB release to surface water is the environmental cycling process (Larsson 1985; Lin and Que Hee 1987; Mackay 1989; Murphy et al. 1985, 1987; Swackhamer and Armstrong 1986). Small amounts of PCBs may enter surface water by runoff of water from accidental spillage of PCB-containing hydraulic fluids, disposal of waste oils into street drains, or from farmland to which sewage sludge containing small quantities of PCBs has been applied (Gan and Berthouex 1994; Gunkel et al. 1995). PCBs may also reach surface waters via CSOs or storm water runoff (Crawford et al. 1995; Loganathan et al. 1997; Shear et al. 1996). The annual contribution of 26 water pollution control plants

**Table 6-1. Releases to the Environment from Facilities that Manufacture or Process Polychlorinated Biphenyls**

State <sup>b</sup>	Number of facilities	Total reported amounts released in pounds per year <sup>a</sup>					Total on-site release <sup>d</sup>	Total off-site release <sup>e</sup>	Total on and off-site release
		Air <sup>c</sup>	Water	Underground injection	Land				
AL	1	5	0	0	0	579180	579185	0	579185
AZ	1	0	0	0	0	134160	134160	1	134161
CA	2	5	0	0	0	1691574	1691579	0	1691579
IL	1	0	0	0	0	0	0	130	130
KS	1	25	0	0	0	0	25	4525	4550
MI	2	10	0	0	0	72000	72010	95	72105
NV	1	0	0	0	0	5200	5200	0	5200
NY	2	1	1	0	0	870000	870002	1067	871069
OR	1	0	0	0	0	151435	151435	0	151435
SC	1	0	0	0	0	0	0	1	1
TN	1	0	0	0	0	0	0	0	0
TX	4	178	250	5	0	46561	46994	47	47041
UT	2	222	0	0	0	192026	192248	12106	204354
WI	1	0	0	0	0	0	0	0	0
<b>Total</b>	<b>21</b>	<b>446</b>	<b>251</b>	<b>5</b>	<b>0</b>	<b>3742136</b>	<b>3742838</b>	<b>17972</b>	<b>3760810</b>

Source: TRI98 2000

<sup>a</sup>Data in TRI are maximum amounts released by each facility.

<sup>b</sup>Post office state abbreviations are used.

<sup>c</sup>The sum of fugitive and stack releases are included in releases to air by a given facility.

<sup>d</sup>The sum of all releases of the chemical to air, land, water, and underground injection wells.

<sup>e</sup>Total amount of chemical transferred off-site, including to publicly owned treatment works (POTWs).



## 6. POTENTIAL FOR HUMAN EXPOSURE

in New York City and New Jersey to the New York/New Jersey Harbor Estuary is estimated to be 88 kg (Durell and Lizotte 1998), while the Montreal, Quebec, waste water treatment plant contributed approximately 1 kg PCBs per year in 1993 to the St. Lawrence River (Pham and Proulx 1997). PCBs, particularly the lower chlorinated congeners, may enter groundwater through leaching of land-applied sewage sludge to soils containing low organic matter or through leaching from soils at hazardous waste sites (Griffin and Chou 1981). PCBs have been identified in 93 surface water and 192 groundwater samples collected at 500 of the 1,598 NPL hazardous waste sites where they were detected in some environmental media (HazDat 2000). According to the TRI, 251 and 5 pounds of PCBs were respectively discharged into surface water and injected into groundwater directly by RCRA hazardous waste and solvent recovery industries in 1998 (TRI98 2000). The TRI data for 1998 (TRI98 2000) are shown in Table 6-1. The TRI data should be used with caution, however, since only certain types of facilities are required to report. This is not an exhaustive list.

**6.2.3 Soil**

From 1929 to 1977, the Monsanto Chemical Company released some PCB-containing wastes to landfills as a result of Aroclor production. Similarly, transformer and capacitor producers disposed of PCB-containing wastes (e.g., capacitors, solid wastes, Fuller's earth media) directly into landfills (Durfee 1976). The amount of PCBs released to soil has decreased over the years due to the prohibition on production in the United States and the severe restrictions on processing and reuse of existing PCB-containing materials. PCBs may have been released to soils from various past uses containing PCBs (e.g., plasticizers, surface coatings, inks, adhesives, flame retardants, pesticide extenders, paints, and micro-encapsulation of dyes for carbonless duplicating paper) and, in addition, from the accidental losses of PCB fluids from capacitors and transformers (EPA 1976a; IARC 1978; Safe 1984; Welsh 1995).

Currently, the environmental cycling process involving deposition of atmospheric PCBs is expected to be the major source of surface soil contamination (Larsson and Okla 1989). Since PCBs are no longer produced in the United States, accidental leaks and spills from old transformers and capacitors containing PCBs and releases from containers in landfills and hazardous waste sites may be sources of PCBs in soil. Accidental spills of PCBs during transportation of electrical transformers and other PCB-containing equipment (Liberti et al. 1992); vehicular emissions (Benfenati et al. 1992; Ohsaki and Matsueda 1994) may also be sources of PCBs in soils. PCBs accumulation in POTW sewage sludge originates from domestic sources (e.g., human excretion from the recycling of PCB residues in foodstuffs) and from industrial facilities (Choi et al. 1974; McIntyre and Lester 1982; Morris and Lester 1994). Land

## 6. POTENTIAL FOR HUMAN EXPOSURE

application of municipal sludges results in elevated PCB concentrations in these soils (Alcock et al. 1995; Gan and Berthouex 1994; Gutenmann et al. 1994; McLachlan et al. 1994; O'Connor et al. 1990).

PCB concentrations in sludge reported in the 1970s and 1980s varied from <0.01 to 1960 mg/kg (ppm) (dry weight) (Jacobs et al. 1987), but median PCB concentration in municipal sludges was in the lower end of this range, 0.99 (Clevenger et al. 1983) and 4 mg/kg (Furr et al. 1976). However, total PCBs were detected in only 1 out of 16 sewage sludge samples (4.6 ppm dry weight; limit of detection=0.25 ppm dry weight) taken from large cities in the United States (Gutenmann et al. 1994). PCBs have been identified in 465 soil samples and 219 sediment samples collected at 500 of the 1,598 NPL hazardous waste sites where they were detected in some environmental media (HazDat 2000). The amount of PCBs released to land by industry has increased from 752 pounds (341 kg) in 1988 (TRI98 2000) to 134,160 pounds (60,854 kg) in 1998 (TRI98 2000). An additional 3,607,976 pounds were released to land in 1998 by RCRA hazardous waste and solvent recovery industries not represented in the 1988 to 1997 TRI data. During 1998, PCBs were not discharged by industry into POTWs (TRI98 2000). The TRI data for 1998 (TRI98 2000) are shown in Table 6-1. The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list.

### 6.3 ENVIRONMENTAL FATE

#### 6.3.1 Transport and Partitioning

PCBs are globally circulated and are present in all environmental media. Atmospheric transport is the most important mechanism for global dispersion of PCBs. Biphenyls with 0–1 chlorine atoms remain in the atmosphere, those with 1–4 chlorines gradually migrate toward polar latitudes in a series of volatilization/deposition cycles, those with 4–8 chlorines remain in mid-latitudes, and those with 8–9 chlorines remain close to the source of contamination. PCBs enter the atmosphere from volatilization from both soil and water surfaces. Once in the atmosphere, PCBs are present in both the vapor phase and sorbed to particles. PCBs in the vapor phase appear to be more mobile and transported further than particle-bound PCBs. Wet and dry deposition remove PCBs from the atmosphere. The dominant source of PCBs to surface waters is atmospheric deposition; however, redissolution of sediment-bound PCBs also accounts for water concentrations. PCBs in water are transported by diffusion and currents. PCBs are removed from the water column by sorption to suspended solids and sediments as well as from volatilization from water surfaces. Higher chlorinated congeners are more likely to sorb, while lower chlorinated congeners are more likely to volatilize. PCBs also leave the water column by concentrating in

## 6. POTENTIAL FOR HUMAN EXPOSURE

biota. PCBs accumulate more in higher trophic levels through the consumption of contaminated food, a process referred to as biomagnification. PCBs in soil are unlikely to migrate to groundwater because of strong binding to soil. Volatilization from soil appears to be an important loss mechanism; it is more important for the lower chlorinated congeners than for the higher chlorinated congeners. Vapor-phase PCBs accumulate in the aerial parts of terrestrial vegetation and food crops by vapor-to-plant transfer.

Wania and Mackay (1996) report that most PCBs are volatile enough to cycle between the air, water, and soil at environmental temperatures, and that atmospheric transport is the most important mechanism for the global movement of PCBs. These authors further categorized the transport and partitioning behavior of PCB congeners according to the number of chlorines present on the biphenyl molecule. Volatile mono-Bs remain primarily in the atmosphere. PCBs that have 1–4 chlorines and are *ortho*-rich (i.e., number of *ortho* chlorines >1) congeners tend to migrate toward polar latitudes by a series of volatilization/deposition cycles between the air and the water and/or soil. PCBs with 4–8 chlorines remain in mid-latitudes, and those with 8–9 chlorines remain close to the source of contamination. The more heavily chlorinated and *ortho*-poor (i.e., number of *ortho* chlorines #1)/*para*-rich (i.e., number of *para* chlorines >1) PCBs are less volatile and more readily condensed from the atmosphere. Thus, these PCBs are considered less mobile (Macdonald et al. 2000; Wania and Mackay 1993, 1996).

The atmosphere is a net recipient of PCBs from soil, water, and (indirect) sediment fluxes (Hansen 1999). These fluxes are the highest in summer as a result of warmer temperatures (Hoff et al. 1992). The importance of volatilization to atmospheric concentrations of PCBs is well established. This conclusion is also supported by the estimated Henry's law constants for Aroclors and PCB congeners, which range from  $2.9 \times 10^{-4}$  to  $4.6 \times 10^{-3}$  atm-m<sup>3</sup>/mol and  $1.5 \times 10^{-5}$  to  $2.8 \times 10^{-4}$  atm-m<sup>3</sup>/mol, respectively (see Tables 4-3 and 4-7) (Thomas 1982). The Great Lakes in particular appear to be a source of PCBs to the atmosphere (Arimoto 1989; Hornbuckle et al. 1993; Swackhamer and Armstrong 1986). The estimated PCB gas fluxes out of the Great Lakes to the atmosphere in 1994 were 1,700, 2,700, 420, and 440 kg/year for Lakes Superior, Michigan, Erie, and Ontario, respectively (Hoff et al. 1996). A pseudo first-order rate constant for the volatilization of total PCBs from Lake Superior is estimated to be 0.4/year ( $t_{1/2}$ =2 years) (Jeremiason et al. 1994). This latter estimated rate indicates that approximately one-half of the total water-borne mass of PCBs in Lake Superior enters the atmosphere over a 7-month period.

PCBs are transported from soil and sediment to the atmosphere. In the absence of water, the rate of movement of PCBs from the soil surface to the atmosphere is controlled by diffusive transfer (Cousins and Jones 1998). For example, Agrell et al. (1999) demonstrated that diffusive exchange from soils is the

## 6. POTENTIAL FOR HUMAN EXPOSURE

dominant transport mechanism of PCBs cycling between the atmosphere and terrestrial surfaces along the Baltic Sea region. As atmospheric sources of PCBs diminish, the flux between the atmosphere and soil will eventually achieve equilibrium (i.e., fluxes in and out of soil will be equal). For instance, Harner et al. (1995) estimated the net volatilization/accumulation of PCBs from soil for four congeners (PCBs 28, 52, 138, and 153) during the period 1942 through 1992. Presently, the more mobile PCBs 28 and 52 should have achieved equilibrium between the soil and air. However, PCBs 138 and 153 are much slower to volatilize from soil and will continue to slowly out-gas until equilibrium is attained.

Contaminated sediments exposed directly to the atmosphere during water level changes (e.g., tidal fluctuations) or during removal to landfills may rapidly transfer the volatile congeners directly to the air through covaporization with water (Chiarenzelli et al. 1996, 1997). This is illustrated by Bremle and Larsson (1998), who studied the concentration of PCBs in air during the landfilling of wet contaminated sediment. They found that the overlying air was enriched in the more volatile, lower molecular weight congeners compared to the deposited sediment, which suggests that volatilization was the major transport process out of the sediment for these congeners. However, other studies have demonstrated that once sediments become dehydrated, the binding of PCBs is tighter and the net volatilization is reduced (Chiarenzelli et al. 1996, 1997).

PCBs in air are present in both the vapor phase and adsorbed to aerosol particles (Eisenreich et al. 1981; Hermanson and Hites 1989; Wania and Mackay 1996). PCBs in the vapor phase appear to be more mobile and are transported further than particle-bound PCBs, while the heavier and coplanar PCBs tend to be particle-bound and/or more readily degraded in the atmosphere (Hansen 1999). PCBs with vapor pressures  $>10^{-4}$  mm Hg (mono- and di-CBs) appear to exist in the atmosphere almost entirely in the vapor phase, while PCBs with vapor pressures  $<10^{-8}$  mm Hg appear to exist almost entirely in the adsorbed phase, and PCBs with vapor pressures  $\#10^{-4}$  and  $\$10^{-8}$  mm Hg (tri- to hepta-CBs) exist in both the adsorbed and vapor phase (Eisenreich et al. 1981; Erickson 1992). The vapor pressures of the Aroclors and several PCB congeners are found in Tables 4-2 and 4-7. PCBs in the vapor phase are enriched (relative to commercial Aroclor mixtures) in di- and tri-*ortho* congeners within each homolog group due to their higher vapor pressures and limited tendency to bind to aerosol particulates. Also, being less volatile, coplanar non-*ortho* and higher chlorinated PCBs are present at very low proportions in the vapor phase, and tend to be associated with aerosols, thereby increasing their chances of removal from the atmosphere by wet and dry deposition (Falconer and Bidleman 1994; Hippelein and McLachlan 1998; Jones et al. 1992; Monosmith and Hermanson 1996; Muir et al. 1996a, 1996b; Panshin and Hites 1994; Simcik et al. 1998; Wania and Mackay 1993). For example, Falconer and Bidleman (1995) reported

## 6. POTENTIAL FOR HUMAN EXPOSURE

preferential sorption of non- and mono-*ortho* PCBs to urban aerosols to a greater extent than multi-*ortho* congeners of the same homolog.

PCBs are physically removed from the atmosphere by wet deposition (i.e., rain and snow scavenging of vapors and aerosols); by dry deposition of aerosols; and by vapor adsorption at the air-water, air-soil, and air-plant interfaces (Cousins et al. 1999; Currado and Harrad 1999; Dickhut and Gustafson 1995; Eisenreich et al. 1981; Franz and Eisenreich 1998; Golomb et al. 1997; Gregor et al. 1996; Hart et al. 1993; Hoff et al. 1996; Leister and Baker 1994; Nelson et al. 1998; Wania et al. 1999). Wet deposition occurs episodically by in-cloud scavenging or rain-out of vapor phase PCBs, and by below-cloud scavenging or wash-out of aerosol PCBs. At low temperatures, wet deposition may fall as snow. Franz and Eisenreich (1998) found that snow can be a very efficient medium for aerosol scavenging to an even greater extent than for rain; and they found that aerosol washout accounted for between 79 and 88% of the total PCB content in snow. However, Wania et al. (1999) recently reevaluated this study and pointed out that the contribution of gaseous scavenging may have been underestimated by these authors. Also, Wania et al. (1998) pointed out that adsorption of PCBs to ice surfaces is a major scavenging mechanism for gaseous PCBs in the atmosphere. In contrast to wet deposition, dry removal of PCBs from the atmosphere results from the gravitational settling of particulate PCBs (i.e., dry particulate deposition) and by the impaction of vapor phase PCBs on terrestrial or aquatic surfaces (i.e., dry gaseous deposition). Dry gaseous deposition is a complex process which depends on the physical-chemical properties of the PCBs, characteristics of the adsorbing surface, and environmental conditions (e.g., windspeed). In the ambient atmosphere, dry particulate deposition is predominantly in the form of fine aerosols (<1  $\mu\text{m}$ ), which deposit on surfaces by rapid, vibratory (Brownian) diffusion (Holsen and Noll 1992). However, in urban areas, PCBs are associated with coarse aerosols (>1  $\mu\text{m}$ ), and these particulates represent the majority of the dry deposition flux even though PCBs are largely in the vapor phase (Holsen et al. 1991).

PCB inputs into aquatic and marine reservoirs are predominantly from wet and dry deposition and from the recycling of sediment-sorbed PCBs into the water column. Eisenreich et al. (1983) demonstrated for the Great Lakes water column that the concentration of PCBs is elevated at both the air/water and water/sediment interfaces as a result of inputs from the atmosphere and sediments, respectively. In addition, Eisenreich et al. (1992) estimated that the upper Great Lakes receive the majority of the total inputs from deposition from the atmosphere (Superior 90%, Michigan 58%, Huron 78%), while the lower Great Lakes receive a lower but significant percentage from these sources (Erie 13%, Ontario 7%). The lower lakes receive a large loading of PCBs from the connecting channels (Detroit River and Niagara River) by neighboring industrial discharges and leakage from waste dump sites. In another study, Franz et al.

## 6. POTENTIAL FOR HUMAN EXPOSURE

(1998) concluded that dry deposition dominates atmospheric PCB loadings to Lake Michigan, suggesting that these loadings are more than 3 times greater than loadings from wet deposition. Similarities between the pattern of PCB congeners in the dry deposition and surficial sediments, and also the magnitude of their fluxes to the water column support their assertion. However, Pearson et al. (1996) concluded that input/output budgets for Lake Michigan also showed large imbalances, indicating failure to understand all of the processes of PCB transport to the water column to this body of water. For Lake Superior, Jeremiason et al. (1994) estimated PCBs inputs for 1984 from riverine, wet deposition, dry deposition, and other sources as 110 (36%), 125 (41%), 32 (10%), and 41 (13%) kg PCBs/year, respectively. Thus, wet deposition contributed the largest load of PCBs to this lake. However, for all of the Great Lakes, Hoff et al. (1996) noted that wet and dry deposition fluxes into the lakes appear to be getting smaller, and the net PCB flux is out of the lakes, i.e., volatilization.

Recycling of PCBs, due to volatilization of PCBs from the water column and subsequent release of PCBs from the sediments, occurs when inputs from the atmosphere decrease (Achman et al. 1996; Sanders et al. 1996). The process of recycling tends to increase with higher PCB solubility (Sanders et al. 1996). There are several mechanisms by which PCBs can exchange between the sediment bed and the overlying water. For example, PCBs dissolved or associated with colloidal particles can exchange across the sediment-water interface by diffusive and/or advective processes (Berner 1980; Formica et al. 1988). The rate of redissolution of PCBs from sediment to water will always be greater in summer than in winter because of more rapid volatilization of PCBs from water with higher summer temperatures (Larsson and Sodergren 1987). In summer, recycling of PCBs directly to the water column by dissolution appears to be the most important process (Sanders et al. 1996), while in winter, sediment resuspension is the predominant mechanism for recycling of PCBs (Sanders et al. 1996). Environmental redistribution of PCBs from aquatic sediment is most significant for the top sediment layers, while PCBs in the lower layers may be effectively sequestered from redistribution (Baker et al. 1985; EPA 1979h, 1988a; Kleinert 1976; Swackhamer and Armstrong 1986). In the lower Hudson River estuary, a high surface sediment concentration of PCBs resulted in the exchange of PCBs from sediment to water (Achman et al. 1996). The average fluxes from sediments were between 2 and 100 times more than the flux coming down the river, and clearly dominated other fluxes from direct atmospheric deposition and waste water treatment plant discharges.

PCBs in water are transported by diffusion and currents. PCBs in surface water essentially exist in three phases: dissolved, particulate, and colloid associated (Baker and Eisenreich 1990). The heavier and less soluble congeners in the water column are more likely to be associated with particulates and colloids, and

## 6. POTENTIAL FOR HUMAN EXPOSURE

do not freely exchange into the vapor phase. However, the more water soluble, lower chlorinated (and *ortho*-rich) congeners are predominantly in the dissolved state in the water column and can readily partition into the vapor phase. In New Bedford Harbor, Massachusetts, Burgess et al. (1996) reported that the ratio of colloid associated PCBs to freely dissolved PCBs increased from 1.2 to 8.0 (di-CBs to octa-CBs, respectively) as the degree of chlorination increased. However, at this site, the majority of the PCBs were associated with the particulate phase regardless of solubility or chlorination.

Experimental and monitoring data have shown that PCB concentrations in sediment and suspended matter are higher than in the associated water column (Eisenreich et al. 1983). In a study of the Saginaw River in Michigan, Verbrugge et al. (1995) reported that the ratio of the total PCBs bound to suspended particulates relative to dissolved PCBs, was 2 to 1. However, in a study examining the water column in Lake Superior, 75% of PCBs were in the dissolved phase, while 25% exist in the suspended particulate phase (Eisenreich et al. 1983). These studies suggest that the partitioning behavior of PCBs in the water column is location specific.

PCBs leave the water column by partitioning onto sediments and suspended particulates, and by volatilization at the air/water interface. PCBs can be immobilized for relatively long periods of time in aquatic sediments. The adsorption of dissolved PCBs onto solids (suspended particulates and sediments) is greatest for solids composed primarily of organic matter and clay (EPA 1980b). The more highly chlorinated components (and *ortho*-poor) PCBs, which have lower water solubilities and higher octanol-water partition coefficients ( $K_{ow}$ ), have a greater tendency to bind to solids as a result of strong hydrophobic interactions (see Table 4-2). In contrast, the low molecular weight PCBs, which have higher water solubilities and lower  $K_{ow}$ s, sorb to a lesser extent on solids and remain largely in the water column (see Table 4-2). Volatilization of highly chlorinated PCBs in the water column is reduced significantly by the sequestration on solids compared to the lightly chlorinated PCBs, in which volatilization may be only slightly effected (EPA 1985b; Lee et al. 1979). The estimated residence times (in years) of PCBs in the water columns of the Great Lakes are: Superior (3.3), Michigan (1.3), Huron (1.0), Erie (0.2), and Ontario (1.1); and the percent loss of PCBs from these lakes due to sedimentation, volatilization, and outflow to other water bodies are summarized in Table 6-2 (Arimoto 1989; Eisenreich et al. 1992). For PCBs in the Great Lakes, sedimentation and volatilization were the primarily loss mechanisms, while the contribution of outflow was comparatively low. For Lake Michigan between 1980 and 1991, the calculated half-lives for the PCB homologs (assuming a first-order processes; in years) due to both sedimentation and volatilization were: di- (11), tri- (15), tetra- (10), penta- (12), hexa- (5.3), hepta- (7), and octa- (5) (Pearson et al. 1996).

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-2. Percentage of Loss of Polychlorinated Biphenyls  
from the Great Lakes Waters**

Waterbody	Volatilization	Sedimentation	Outflow to other bodies of water
Lake Superior	86.6	11.4	2.0
Lake Michigan	68.1	30.6	1.3
Lake Huron	75.3	19.4	5.3
Lake Erie	46.0	45.2	8.8
Lake Ontario	53.4	29.3	17.3

Source: Eisenreich et al. 1992



## 6. POTENTIAL FOR HUMAN EXPOSURE

In addition to volatilization and sorption onto sediments, PCBs can leave the water column by concentrating in biota directly from water (EPA 1983c; Porte and Albaiges 1993). Bioconcentration is defined as uptake of a chemical from water alone; and bioaccumulation is the result of combined uptake via food, sediment, and water. The bioconcentration factors (BCFs; ratio of the concentration of PCBs in the organism over the concentration of PCBs in water) of PCBs in aquatic organisms are directly proportional to partition coefficients and lipid contents of the organism, and are congener specific (Geyer et al. 1999). BCFs in various fresh water and marine species are generally in the range of  $5 \times 10^2$ – $4 \times 10^4$  for lower chlorinated PCB congeners and about  $1 \times 10^3$ – $3 \times 10^5$  for tetra- to hexa-PCBs (70, 101, 110, and 136) (Geyer et al. 1999; see Table 6-3). Median BCFs for accumulation from water by phytoplankton range from  $1 \times 10^4$  to  $1 \times 10^6$ , and are generally the greatest for the tetra- to hepta-PCBs and for the coplanar tri-, tetra-, and penta-PCBs (Willman et al. 1999). Coplanar PCBs and the more highly chlorinated congeners can have aquatic organism BCFs as high as  $2 \times 10^6$  (Hansen 1999). However, the BCFs for the higher chlorinated homologs drop off after a certain point because these larger molecules do not readily pass through biological membranes. BCFs for freshwater and marine species are illustrated in Tables 6-4 and 6-5, respectively, for Aroclors mixtures (ASTER 1996).

Bioaccumulation factors (BAFs; the ratio of the concentration of PCBs in the organism over the combined concentration of PCBs in sediment, food, and water) of PCBs increase with higher chlorination and lower water solubility (Coristine et al. 1996; Zhang et al. 1983). In contrast to BCFs, a direct relationship between bioaccumulation, partition coefficients, and organism lipid content does not always exist, and other factors (e.g., reproductive cycles) may affect the uptake and accumulation of PCBs (Hansen 1999; Stow et al. 1997). Less chlorinated PCBs (1–4 chlorines) are readily taken up by organisms, but are readily eliminated and metabolized. Thus, these homologs are not bioaccumulated to a great extent (see Section 6.3.2; McFarland and Clarke 1989). The most highly chlorinated congeners (7–10 chlorines) occur in low concentrations in the environment, and are tightly bound with soil, sediment, and organic matter. Thus, these PCBs are also not significantly bioaccumulated (Bergen et al. 1993; Lacorte and Eggens 1993; McFarland and Clarke 1989). These PCBs, which have  $\log K_{ow}$  values  $>5$ , appear to enter biota through food-web transfer from sediment, which is less efficient (Koslowski et al. 1994). On the other hand, the penta-, hexa-, and hepta-PCBs are both bioavailable and resistant to degradation in organisms; and these PCB homologs bioaccumulate in organisms to the greatest extent (see Section 6.3.2; Bremle et al. 1995; Koslowski et al. 1994; McFarland and Clarke 1989; Porte and Albaiges 1993; Willman et al. 1997). For example, the PCBs that dominate congener profiles in the tissues of mussels, crabs, and seals are hexa-PCB isomers 138 and 153 (Hansen 1999; Porte and Albaiges 1993). The differences in congener retention in organisms apparently accounts for the differences in

**Table 6-3. Bioconcentration Factors (BCFs) and Bioaccumulation Factors (BAFs) for Select Congeners and Total Polychlorinated Biphenyls in Various Aquatic Organisms**

PCB	Organism	Laboratory BCF	Field BAF	Location	Reference
PCB 18	Rainbow trout ( <i>Oncorhynchus mykiss</i> )	81,000	590,000	Lake Ontario	Oliver and Niimi 1985
PCB 40	Rainbow trout ( <i>O. mykiss</i> )	49000	240,000	Lake Ontario	Oliver and Niimi 1985
PCB 52	Rainbow trout ( <i>O. mykiss</i> )	200,000	1,900,000	Lake Ontario	Oliver and Niimi 1985
PCB 101	Rainbow trout ( <i>O. mykiss</i> )	200,000	8,400,000	Lake Ontario	Oliver and Niimi 1985
PCB 153	Rainbow trout ( <i>O. mykiss</i> )	740,000	10,000,000	Lake Ontario	Oliver and Niimi 1985
Total	Perch ( <i>Perca fluviatilis</i> )		2,050–7,580	Lake Jarnsjon, Sweden	Bremle et al. 1995
Total	Tilapia ( <i>Oreochromis mossambicus</i> )		10,000	Shing Mun River, Hong Kong	Chui et al. 1991
Total	Pumpkinseed ( <i>Lepomis gibbonsus</i> )		187,000– 2,079,000	Hudson River, New York	Sloan et al. 1985
Total	Amphipods ( <i>Pontoporeia affinis</i> )		718,000	Lake Ontario	Oliver and Niimi 1988
Total	Oligochaetes ( <i>Tubifex tubifex</i> and <i>Limnodrilus hoffmeisteri</i> )		164,000	Lake Ontario	Oliver and Niimi 1988
Total	Slimy sculpin ( <i>Cottus cognatus</i> )		1,450,000	Lake Ontario	Oliver and Niimi 1988
Total	Alewife ( <i>Alosa pseudoharengus</i> )		1,180,000	Lake Ontario	Oliver and Niimi 1988
Total	Rainbow smelt (small) ( <i>Osmerus mordax</i> )		564,000	Lake Ontario	Oliver and Niimi 1988
Total	Rainbow smelt (large) ( <i>O. mordax</i> )		1,272,000	Lake Ontario	Oliver and Niimi 1988
Total	Salmonids		3,910,000	Lake Ontario	Oliver and Niimi 1988

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-4. Bioconcentration Factors (BCFs) for Various Aroclors in Fresh Water Species**

Species	BCF	Duration (days)
Aroclor 1232		
White sucker ( <i>Catostomus commersoni</i> )	5,500	30
Aroclor 1242		
Scud ( <i>Gammarus pseudolimnaeus</i> )	36,000	60
Fathead minnow (male) ( <i>Pimephales promelas</i> )	274,000	255
Atlantic salmon ( <i>Salmo salar</i> )	600	4
Aroclor 1248		
Scud ( <i>G. pseudolimnaeus</i> )	108,000	60
Fathead minnow (female) ( <i>P. promelas</i> )	120,000	240
Channel catfish ( <i>Ictalurus punctatus</i> )	56,400	77
Bluegill ( <i>Lepomis macrochirus</i> )	52,000	77
Aroclor 1254		
Cladoceran ( <i>Daphnia magna</i> )	3,800	4
Scud ( <i>G. pseudolimnaeus</i> )	6,200	21
Crayfish ( <i>Orconectes nais</i> )	750	21
Dobsonfly ( <i>Corydalus cornutus</i> )	1,500	7
Stonefly ( <i>Pteronarcys californica</i> )	740	21
Mosquito ( <i>Culex tarsalis</i> )	3,500	7
Phantom midge ( <i>Chaoborus punctipennis</i> )	2,700	14
Fathead minnow (female) ( <i>P. promelas</i> )	238,000	240
Rainbow trout ( <i>Oncorhynchus mykiss</i> )	46,000	30
Brook trout ( <i>Salvelinus fontinalis</i> )	47,000	118
Brook trout ( <i>S. fontinalis</i> )	42,000	500
Brook trout ( <i>S. fontinalis</i> )	3,000 <sup>a</sup>	500
Steelhead trout ( <i>Salmo gairdneri</i> )	38,000	24
Channel catfish ( <i>I. punctatus</i> )	61,200	77
Aroclor 1260		
Fathead minnow (female) ( <i>P. promelas</i> )	270,000	240

Source: ASTER 1998

<sup>a</sup>Fillet sample rather than whole body sample

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-5. Bioconcentration Factors (BCFs) for Various Aroclors in Salt Water Species**

Species	BCF	Duration (days)
Aroclor 1016		
Eastern oyster ( <i>Crassostrea virginica</i> )	13,000	84
Horseshoe crab ( <i>Limulus polyphemus</i> )	1,298	96
Sheepshead minnow (adult) ( <i>Cyprinodon variegatus</i> )	25,300	28
Sheepshead minnow (juvenile) ( <i>C. variegatus</i> )	43,100	28
Sheepshead minnow (fry) ( <i>C. variegatus</i> )	14,400	28
Pinfish ( <i>Lagodon rhomboides</i> )	17,000	21–28
Aroclor 1242		
Diatom ( <i>Cylindrotheca closterium</i> )	1,000	14
Aroclor 1254		
Ciliate protozoans ( <i>Tetrahymena pyriformis</i> )	60	7
Eastern oyster ( <i>C. virginica</i> )	8,100	2
Eastern oyster ( <i>C. virginica</i> )	101,000	245
Polychaete ( <i>Arenicola marina</i> )	236	5
Polychaete ( <i>Nereis diversicolor</i> )	373	5
Grass shrimp ( <i>Palaemonetes pugio</i> )	27,000	16
Pink shrimp ( <i>Penaeus duorarum</i> )	140	2
Spot ( <i>Leiostomus xanthurus</i> )	37,000	28
Sheepshead minnow (adult) ( <i>C. variegatus</i> )	30,000	28
Pinfish ( <i>L. rhomboides</i> )	980	2

Source: ASTER 1996

## 6. POTENTIAL FOR HUMAN EXPOSURE

congener concentration profiles seen in the higher trophic levels. PCB bioaccumulation is also effected by the stereochemistry of the congener; optimal bioaccumulation occurs for planar molecules substituted with 5 or 7 chlorines (Koslowski et al. 1994). For instance, median BAFs for zooplankton and zebra mussels ranged from  $1 \times 10^4$  to  $1 \times 10^6$ , and were the greatest for the planar tetra- to hepta-PCBs, which had 54% larger BAFs compared to values for the non-coplanar homologs (Willman et al. 1999). Typical field-measured BAFs range from  $2.1 \times 10^3$  to  $3.9 \times 10^6$  for total PCBs (Table 6-3). BAFs for PCB isomer groups in a Lake Ontario food web ranged from  $4.2 \times 10^4$  to  $1.3 \times 10^7$  (Table 6-6). The highest BAFs occur at the lower end of the food chain. For example, Oliver and Niimi (1988) determined that the water-plankton-mysid food chain had the highest bioaccumulation of PCBs in the Lake Ontario trophic system.

Bioaccumulation of PCBs in aquatic animals depends on the water zone in which the animals predominantly reside and feed. Certain benthic organisms, such as crabs, clams, sandworms, and grass shrimp, accumulate PCBs from water at the water/sediment interface (PCB concentration is higher at this interface than in the surrounding water column, see above) and via intake of phytoplankton and zooplankton, which contain higher levels of PCBs than the water (Porte and Albaiges 1993; Pruell et al. 1993; Secor et al. 1993). When airborne PCBs are deposited onto the surface of water, lower chlorinated and *ortho*-rich congeners, especially, become enriched in the surface microlayer which results in concentrations that are 500 times higher than the average concentration in water. As a result, bioaccumulation by fish is several orders of magnitude higher in this zone (Sodergren et al. 1990). Greater bioaccumulation will occur in the fatty tissues (lipids) than in the muscle or whole body of aquatic organisms (EPA 1980b). Thus, organisms with higher lipid concentrations will accumulate a greater burden of PCBs via trophic transfer. Fish species, such as lake trout (*Salvelinius namaycush*) and coho salmon (*Oncorhynchus kisutch*), with high lipid contents, have a net trophic transfer efficiency from food ranges of 75–89 and 38% (average for tetra-CBs; however, higher chlorinated congeners ranged from 43 to 56%), respectively (Madenjian et al. 1999). In addition, insects that have lipid-rich cuticular (skin) layers can capture significant amounts of vapor-phase PCBs in their tissues and enter these PCBs into the food chain (Saghir and Hansen 1999).

Biomagnification of PCBs within the aquatic food chain results from higher trophic transfer and has been observed in aquatic organisms (Koslowski et al. 1994; Looser and Ballschmiter 1998; Oliver and Niimi 1988; Wilson et al. 1995). Biomagnification is apparent in shellfish that accumulate PCBs from the consumption of phytoplankton and zooplankton, and in marine mammals (seals, dolphins, and whales) that accumulate PCBs from plankton and fish (Andersson et al. 1988; Kuehl and Haebler 1995; Lake et al. 1995a; Salata et al. 1995; Schantz et al. 1993c; Secor et al. 1993). Food chain biomagnification also

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-6. Field Measured Bioaccumulation Factors for Isomeric Groups of Polychlorinated Biphenyls**

Organism	PCB Group					
	Tri-	Tetra-	Penta-	Hexa-	Hepta-	Octa-
Amphipods	387,000	667,000	615,000	938,000	2,400,000	1,400,000
Oligochaetes	127,000	180,000	154,000	150,000	259,000	310,000
Slimy sculpin	87,000	633,000	1,490,000	3,125,000	5,185,000	7,500,000
Alewife	173,000	833,000	1,380,000	2,125,000	2,960,000	3,100,000
Rainbow smelt						
Small	42,000	367,000	590,000	1,063,000	1,590,000	1,600,000
Large	93,000	933,000	1,380,000	2,375,000	3,148,000	3,300,000
Salmonids	293,000	2,170,000	4,100,000	8,125,000	11,300,000	13,000,000

Source: Oliver and Niimi 1988

## 6. POTENTIAL FOR HUMAN EXPOSURE

occurs in several species of fish-consuming birds (Ankley et al. 1993; Hebert et al. 1994; Mackay 1989; Metcalfe and Metcalfe 1997; Shaw and Connell 1982; Winter and Streit 1992). Biomagnification of PCBs in the aquatic food chain is congener specific and is more predominant for congeners with  $K_{ow}$  values between 5 and 7 (Koslowski et al. 1994; Metcalfe and Metcalfe 1997). For example, in the food web of the western basin of Lake Erie, concentrations of PCB 138 increased from plankton (14  $\mu\text{g}/\text{kg}$ ) to piscivores (1.4x10<sup>3</sup>  $\mu\text{g}/\text{kg}$  in silver bass muscle tissue) to herring gulls (3.0x10<sup>4</sup>  $\mu\text{g}/\text{kg}$ ) (Koslowski et al. 1994). However, no biomagnification was observed for PCBs 77, 126, and 169 (Koslowski et al. 1994). As previously observed for bioaccumulation, differences in retention also account for differences in congener biomagnification in higher trophic levels.

PCBs are strongly sorbed to soils as a result of low water solubility and high  $K_{ow}$  (see Table 4-2), and will not leach extensively (EPA 1979h, 1988a; Sklarew and Girvin 1987). The tendency to leach will be greatest among the least chlorinated congeners and is expected to be greatest in soil with low organic carbon (Sklarew and Girvin 1987; Streck and Weber 1982a). Leaching of PCBs in most soils should not be extensive, particularly for the more highly chlorinated congeners. However, PCBs will leach significantly in the presence of organic solvents that may be present at municipal landfills or hazardous waste sites (Griffin and Chou 1981). Partition coefficients ( $K_d$ ) for PCBs 8, 52, and 153 for sorption onto soil with variable organic carbon content (0.2–2.3 by weight percent) are 74–825, 533–5,508, and 14,258–68,485 L/kg, respectively (Girvin and Scott 1997). Soil and sediment sorption coefficients ( $K_{oc}$ ) for biphenyl and PCB congeners are listed in Table 6-7.

Soils received net PCB inputs from water and air during the peak emissions of the 1960s and early 1970s. However, at present, soils appear to be reservoirs for releasing PCBs into the atmosphere (Hansen 1999). The mechanisms involved in the soil-to-air transfer of PCBs will involve a combination of direct soil organic matter-to-air transfer and soil pore water-to-air transfer (Cousins et al. 1997). Wicking (i.e., the movement of a compound in solution to replace evaporative surface water loss) has been demonstrated as a process that can increase the volatilization of PCBs from soil; thus, volatilization rates will be greatest in moist soils from the co-vaporization of PCBs and water (Bushart et al. 1998; Chiarenzelli et al. 1996, 1997, 1998). For example, Chiarenzelli et al. (1996, 1997) demonstrated that for small amounts of St. Lawrence River solids originally contaminated with Aroclor 1248, several *ortho*-chlorinated congeners were preferentially lost by volatilization, which could be positively correlated with water loss by vaporization. Soils with low organic carbon will have the greatest rate of volatilization of PCBs (Shen and Tofflemire 1980). For example, researchers at General Electric demonstrated that the rate of volatilization of Aroclor 1242 from soil is much less from the organic topsoil than from the course sand

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-7. Observed Soil and Sediment Sorption Coefficients ( $K_{oc}$ ) for Polychlorinated Biphenyls Congeners**

Compound	Log $K_{oc}$	Reference(s)
Biphenyl	3.27	Meylan et al. 1992
2-CB	3.47	Chiou et al. 1983
2,2'-CB	3.92	Chiou et al. 1983
2,4'-CB	4.13	Chiou et al. 1983
	4.57, 4.56	Girvin and Scott 1997
2,2',4-CB	4.84	Chiou et al. 1987
2,2',5-CB	4.57	Chin and Weber 1989
2,4,4'-CB	4.62	Chiou et al. 1983
2,2',5,5'-CB	3.43	Haque and Schmedding 1976
	4.23–5.15	Hassett et al. 1984
	4.97	Chin and Weber 1989
	5.42, 5.38	Girvin and Scott 1997
2,2',6,6'-CB	5.11	Steen et al. 1978
2,3',4',5-CB	5.02	Steen et al. 1978
2,2',4,5,5'-CB	5.79, 5.93	Gschwend and Wu 1985
2,2',3,4,4',5'-CB	6.16	Gschwend and Wu 1985
2,2',4,4',5,5'-CB	5.62	Karickhoff 1981
	4.78–6.87	Horzempa and Di Toro 1983
	6.85, 6.47	Girvin and Scott 1997
2,2',4,4',6,6'-CB	6.08	Karichoff 1981

Sources: Saçan and Balcioğlu (1996); Sklarew and Girvin (1987); McGroddy et al. (1996)



## 6. POTENTIAL FOR HUMAN EXPOSURE

fraction (Shen and Tofflemire 1980). In another study, Grundy et al. (1996) examined nine soil plots treated with Aroclors 1254 and 1260 representing dry barren, dry moss, and wet grass cover in the Canadian Arctic (Northwest Territory). Rate constants (first-order processes) for loss of total PCBs of approximately 0.5/year ( $t_{1/2} = 1.1$  year), with a range of 0.3–1.0/year for individual congeners, was estimated. For the dry barren area, loss was correlated with vapor pressure. For the two vegetated areas, the volatilization rate appeared to be reduced by organic matter from both living and dead vegetation.

PCBs accumulate in terrestrial vegetation by the following possible mechanisms: (1) uptake from soil through the roots; (2) dry deposition on aerial parts (particle-bound or gaseous); and (3) wet deposition on aerial parts (particle-bound or solute). The primary mode of uptake for total PCBs in terrestrial vegetation is by vapor-to-plant transfer (Bohm et al. 1999; Lober et al. 1994; O'Connor et al. 1990; Schönherr and Riederer 1989). However, Bohm et al. (1999) reported that vapor-to-plant partitioning is most important for tri-CBs, while aerial dry deposition is most important for hepta- and octa-CBs. For example, Ye et al. (1992b) found that the more highly chlorinated congeners (7-chlorines) are adsorbed by aerial plant tissues (e.g., tomato plant leaves) primarily by vapor-to-plant transfer, while the lower chlorinated congeners (3–6-chlorines) are both adsorbed on and absorbed in aerial plant tissues. The lower chlorinated (and *ortho*-enriched) congeners, which have the highest concentrations in the atmosphere, are the most efficiently scavenged by terrestrial vegetation by vapor-to-plant transfer (Jones and Duarte-Davidson 1997; Thomas et al. 1998); and leafy vegetation (e.g., lettuce, grass) appears to accumulate the highest levels of total PCBs by this mechanism (Cullen et al. 1996). The air-to-grass transfer is the first link in the grass-to-cattle-to-human food chain, and this food chain provides an appreciable fraction of human exposure to PCBs (Currado and Harrad 1999; see Section 6.4). Strong sorption of PCBs to soil organic matter and clay inhibits the uptake of PCBs in plants through the roots (Bacci and Gaggi 1985; Chu et al. 1999; Gan and Berthouex 1994; Paterson et al. 1990; Streck et al. 1982b; Webber et al. 1994; Ye et al. 1992a). As a result, below-ground vegetation, such as potatoes, will accumulate the lowest levels of total PCBs, lower proportions of the more lightly chlorinated congeners, and will predominately accumulate the moderately chlorinated congeners (e.g., penta-CBs 99, 101, and 110) directly from soil (Cullen et al. 1996). However, higher uptake from soil can occur in certain root crops (e.g., carrots) by the partitioning of PCBs into the lipid-rich epidermal layer (skin) or by soil particles adhering to the root (Cullen et al. 1996; O'Connor et al. 1990; Pal et al. 1980). Plants grown on PCB-contaminated sludge or sludge-amended soils will be free of vapor-phase PCB contamination as a result of strong sorption of PCBs to sludge organic matter (Gan and Berthouex 1994; O'Connor et al. 1990). For example, Gan and Berthouex demonstrated that for corn grown on PCB contaminated sludge-amended farmland, bioconcentration of PCBs did not occur in either the grain or stover. Plant BCFs of

## 6. POTENTIAL FOR HUMAN EXPOSURE

PCBs from soil are summarized in Table 6-8, and are estimated to be  $<0.02$  for most terrestrial plant species (Cullen et al. 1996; O'Connor et al. 1990; Pal et al. 1980).

### 6.3.2 Transformation and Degradation

The ability of PCBs to be degraded or transformed in the environment depends on the degree of chlorination of the biphenyl molecule as well as on the isomeric substitution pattern. The vapor-phase reaction of PCBs with hydroxyl radicals is the dominant transformation process in the atmosphere, while photolysis appears to be the only viable abiotic degradation process in water. Biodegradation in the environment, although slow, occurs under both aerobic and anaerobic conditions. In sediments, aside from the aerobic surface layer, anaerobic microbial degradation will be primarily responsible for transformation, particularly of the more highly chlorinated congeners. Aerobic biodegradation in soil, surface water, and sediments is limited to the less chlorinated congeners.

#### 6.3.2.1 Air

In the atmosphere, the vapor-phase reaction of PCBs with hydroxyl radicals (photochemically formed by sunlight) is the dominant transformation process (Brubaker and Hites 1998). The calculated tropospheric lifetime values for this reaction increases as the number of chlorine substitutions increases. The tropospheric lifetime values (determined using the calculated OH radical reaction rate constant and assuming an annual diurnally averaged OH radical concentration of  $5 \times 10^5$  molecule/cm<sup>3</sup>) are: 5–11 days for monochlorobiphenyls, 8–17 days for dichlorobiphenyls, 14–30 days for trichlorobiphenyls, 25–60 days for tetrachlorobiphenyls, and 60–120 days for pentachlorobiphenyls (Atkinson 1987). In another study, the estimated tropospheric lifetimes of PCBs (calculated using the estimated OH radical reaction rate constant and assuming a 24-hour average OH radical concentration of  $9.7 \times 10^5$  molecule/cm<sup>3</sup>) range from 2 days for biphenyl to 75 days for hexachlorobiphenyl, and a total global PCB loss rate of 8,300 tons/year was estimated (Anderson and Hites 1996; Atkinson 1996). Rate constants for PCBs more chlorinated than hexa are not easily measured due to their low vapor pressures. It is difficult to introduce a significant amount of these PCBs into the gas phase and to collect their gas phase reaction products which could have even lower volatilities. For the PCBs that do react with OH radicals, a possible reaction scheme is the formation of a 2-hydroxybiphenyl intermediate, which quickly degrades by a series of dark reactions to chlorinated benzoic acid (see Figure 6-2; Brubaker and Hites 1998). The little information available suggests that photolysis of gas-phase PCBs in the troposphere will be negligible for those PCBs with #4 chlorine atoms, and this may be the case for the more chlorinated PCBs as well (Atkinson 1996).

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-8. Plant Uptake (Bioaccumulation) of PCBs<sup>a</sup>**

Crop (growth media)	Application rate	BAF <sup>b</sup>	Reference <sup>a</sup>
Carrot (soil)	Aroclor 1254 at 100 ppm mixed in top 6 inches of soil	<1 (Aroclor 1254) #0.16 (roots)	Iwata et al. 1974
Carrot (acid soil and brown sand)	Aroclor 1254 at 0.05, 0.5, and 5 ppm (acid soil); 0.5 ppm (brown sand)	0, << 1, <1, 0.16 (roots) <1, 0.16 (roots peels)	Wallnöfer et al. 1975
Carrot (soil)	PCB 4 at 1 ppm in dry soil, mixed in top 10 cm	<1 (di-PCB) 0.25 (roots) 0.25 (leaves)	Moza et al. 1976
Carrot (soil)	None	1.5 (PCB 52) 0.35 (PCB 101) 0.38 (PCB 138) 0.28 (PCB 153)	Cullen et al. 1996
Corn (field)	Aroclor 1254 and 1260 contaminated sludge (92–144 µg PCBs/L sludge)	<1	Lawrence et al. 1977
Lettuce (soil)	None	6.0 (PCB 52) 1.5 (PCB 101) 1.1 (PCB 138) 0.74 (PCB 153)	Cullen et al. 1996
Potato (soil)	None	0.29 (PCB 52) 0.01 (PCB 101) 0.17 (PCB 138) 0.28 (PCB 153)	Cullen et al. 1996
Radish (acid soil and brown sand)	Aroclor 1254 at 0.05 ppm (acid soil or brown sand), 0.5 ppm (acid soil); Aroclor 1224 at 0.2 ppm (brown sand);  Aroclor 1254 at 5 ppm (acid soil) with moisture 40% of maximum water holding capacity	0, 0, 0.02  0.005	Wallnöfer et al. 1975
Soybean sprouts (sandy soil)	Aroclor 1242 at 100 ppm	0.002	Suzuki et al. 1977
Sugarbeet (brown soil)	Aroclor 1254 at 0.3 ppm in soil	0.01 (leaves) to 0.5 (whole plant) 0.17 (root peels) 0.03 (peeled root)	Wallnöfer et al. 1975
Sugarbeet (field soil)	PCB 4 at 0.24 ppm in 0–10 cm soil layer and 0.17 ppm in 10–20 cm soil layer	0.07 (roots) 0.03 (leaves)	Moza et al. 1976
Tomato (soil and vermiculite)	PCB 4; PCB 7; PCB 18; PCB 52; PCB 101 (concentration not specified)	0 for all PCBs (mature plants)	Pal et al. 1980

## 6. POTENTIAL FOR HUMAN EXPOSURE

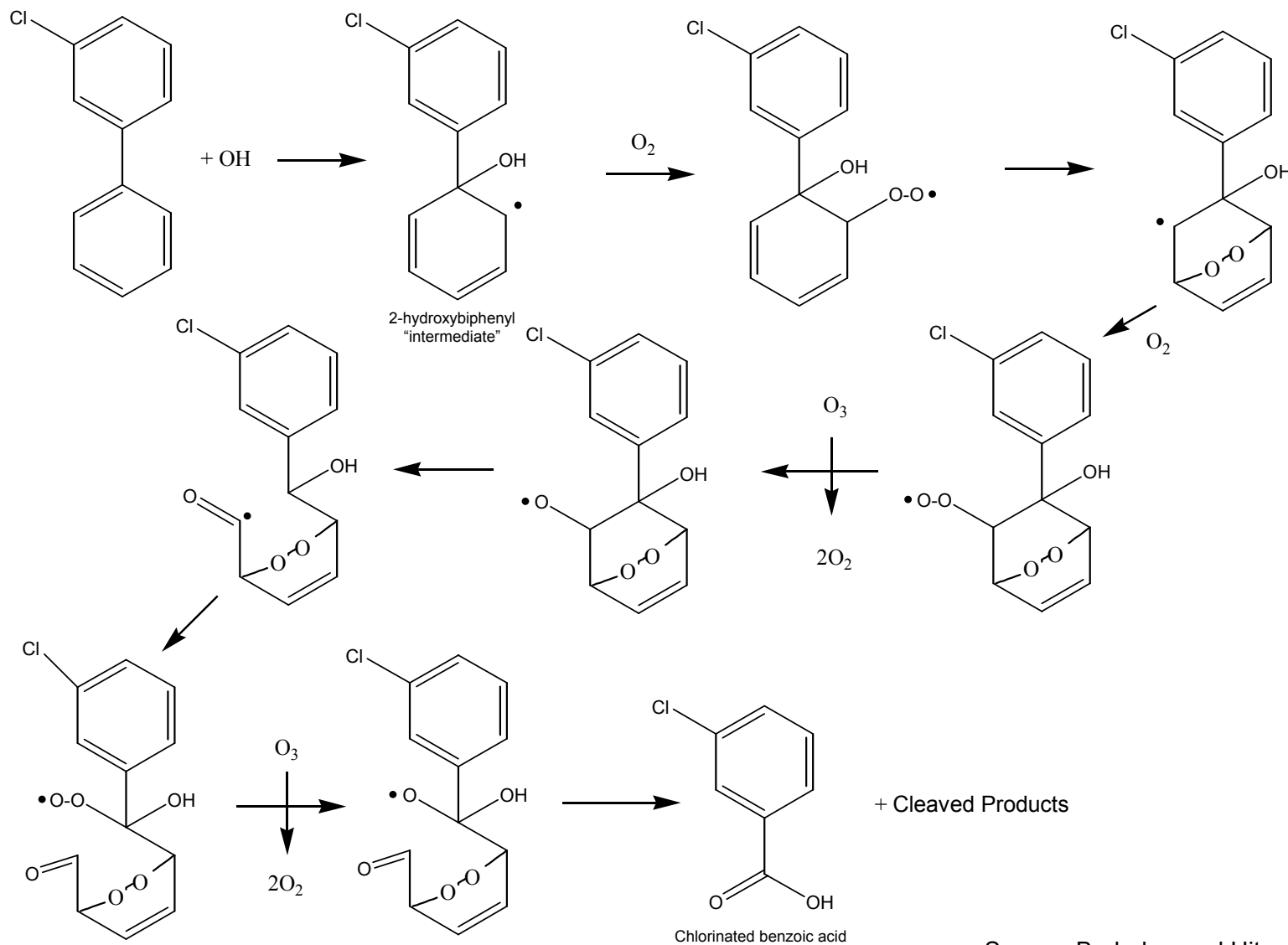
**Table 6-8. Plant Uptake (Bioaccumulation) of PCBs<sup>a</sup> (continued)**

Crop (growth media)	Application Rate	BAF <sup>b</sup>	Reference <sup>a</sup>
Tomato	None	0.64 (PCB 52) 0.23 (PCB 101) 0.15 (PCB 138) 0.01 (PCB 153)	Cullen et al. 1996

<sup>a</sup>Sources: Cullen et al. 1996; Pal et al. 1980

<sup>b</sup>BAF = bioaccumulation factor; concentration of PCBs in plant tissue divided by the concentration in growth medium

Figure 6-2. Pathways for OH Radical-initiated Reaction of 3-Chlorobiphenyl



Source: Brubaker and Hites 1998

## 6. POTENTIAL FOR HUMAN EXPOSURE

At the present time, insufficient data are available to assess the importance of photolysis and/or chemical reactions of particle phase congeners, although studies of Tysklind and Rappe (1991) and Koester and Hites (1992) on the photodegradation of polychlorinated dioxins and furans suggest that photolysis for particulate phase PCBs is not important (Atkinson 1996).

**6.3.2.2 Water**

In water, abiotic transformation processes such as hydrolysis and oxidation do not significantly degrade PCBs (EPA 1979h). Photolysis appears to be the only significant abiotic degradation process in water (EPA 1979h). Photolysis of PCBs occurs by photolytic cleavage of a carbon-chlorine bond followed by a stepwise replacement of chlorine with hydrogen which degrades PCBs (Barr et al. 1997; EPA 1979h). In all cases, the ring with the greatest degree of chlorination is the primary ring where dechlorination occurs. These dechlorination reactions have been reported to proceed by the loss of chlorine in the order of *ortho*>*para*>*meta* (Barr et al. 1997). Lepine et al. (1991) reported that for dechlorination of Aroclor 1254 (in cyclohexane) under natural sunlight, preferential removal of *ortho* chlorines led not only to decreases in many of the highly chlorinated PCB congeners but also to an increase in the concentrations of the toxic non-*ortho* coplaner congeners, PCBs 77 and 126. The estimated photolysis half-lives of mono- through tetrachlorobiphenyls with summer sunlight at a shallow water depth (<0.5 m) range from 17 to 210 days (EPA 1979h). Photolysis rates with sunlight are slower during winter (EPA 1979h). Nonetheless, as the number of chlorine substitutions increases, the light absorption band shifts toward longer wavelengths, and the photolysis rate for hepta- through deca-chlorinated biphenyls increases (EPA 1979h). The estimated photolysis half-lives (first-order) of 4-monoCB, 2,4-diCB, 2,4,6-triCB, 2,2',5,5'-tetraCB, and decachloro-CB (in 75% acetonitrile) were 210, 17, 53, 180, and <0.06 days, respectively (EPA 1983c). Bunce et al. (1978) predicted that for PCBs in shallow waters, on average up to 5% of the lightly chlorinated PCBs might lose a chlorine atom each year, but that at least one chlorine should be lost from every highly chlorinated PCB molecule annually. However, because the conditions of their experiments do not represent actual conditions in the environment, the PCB photolysis rate may be significantly lower.

The rate of PCB biodegradation in water is dependent on both individual congener structure and environmental conditions, as is explained in more detail in Section 6.3.2.3. Biodegradation in surface waters is primarily an aerobic process; in some, particularly oligotrophic waters, a substantial percentage of the total PCB concentration can be found in the dissolved phase (see Section 6.3.1; Eisenreich et al. 1983). The less chlorinated mono- and dichlorobiphenyl congeners are more likely to dissolve in water

## 6. POTENTIAL FOR HUMAN EXPOSURE

than the more chlorinated congeners. These congeners are also more likely to biodegrade under aerobic conditions (Bailey et al. 1983; Wong and Kaiser 1975). In a river die-away study using filtered river water, the individual monochlorobiphenyl congeners were degraded by 50% within 2–5 days (Bailey et al. 1983). However, 2,2',4,4'-tetrachlorobiphenyl was not degraded over the 98-day study. The biodegradation of Aroclor 1221 proceeded rapidly in unfiltered lake water after a 4-day lag phase; within 1 month, the original mixture was completely degraded into metabolites of lower molecular weight (Wong and Kaiser 1975). However, Aroclor 1260 was not degraded over a 12-week period in three different unfiltered natural water samples (Oloffs et al. 1972). An Aroclor 1254 mixture was not mineralized over 96 days in a fresh water study; a further study using individual congeners, 2-chlorobiphenyl and 2,4'-dichlorobiphenyl, showed that the former was degraded to chlorobenzoic acid, a common intermediate in the aerobic biodegradation of PCBs, while the latter was not biodegraded (Shiarls and Sayler 1982). In general, these results are similar to those reported in aerobic soil and sediment studies where mono-, di-, and trichlorobiphenyl structures are fairly readily biodegraded, biphenyl rings containing five or more chlorine substituents are considered to be persistent, and tetrachlorobiphenyl congeners exhibit an intermediate persistence (Abramowitz 1990; Alcock et al. 1996; EPA 1979i; Gan and Berthouex 1994). Biodegradation rates in marine water may be slower than those reported in freshwater. The monochlorobiphenyl congeners had half-lives of approximately 8 months in sea water incubated at 10 EC (Carey and Harvey 1978). Aroclors 1221 and 1254 were individually added to sea water and exposed for 4–8 weeks in enclosures in the North Sea; no degradation of either PCB mixture was reported over this time (Kuiper and Hanstveit 1988). Biodegradation is potentially a more important process in soil and sediment than in water, particularly for the more highly-chlorinated congeners, for at least three reasons: the higher numbers of microorganisms present, the opportunity for anaerobic biodegradation, and the preferential partitioning of PCBs to soil and sediment (see Section 6.3.2.3).

**6.3.2.3 Sediment and Soil**

PCBs, particularly the highly chlorinated congeners, adsorb strongly to sediment and soil where they tend to persist with half-lives on the order of months to years (see Section 6.3.1; Gan and Berthouex 1994; Kohl and Rice 1998). There is no abiotic process known that significantly degrades PCBs in soil and sediment. However, photolysis of PCBs from surface soil may occur, and PCBs may also undergo base-catalyzed dechlorination (Chiarenzelli et al. 1995; Taniguchi et al. 1997); albeit, both of these processes are likely to be insignificant removal mechanisms.

## 6. POTENTIAL FOR HUMAN EXPOSURE

Biodegradation has been shown to occur under both aerobic and anaerobic conditions and is a major degradation process for PCBs in soil and sediment, as reviewed by Higson (1992), Robinson and Lenn (1994), Bedard and Quensen (1995), and most recently by Wiegel and Wu (2000). While photolysis of PCBs from soil surfaces may also occur, and PCBs may also undergo base-catalyzed dechlorination (Chiarenzelli et al. 1995; Taniguchi et al. 1997), neither of these processes is likely to be a significant removal mechanism in soil and sediment.

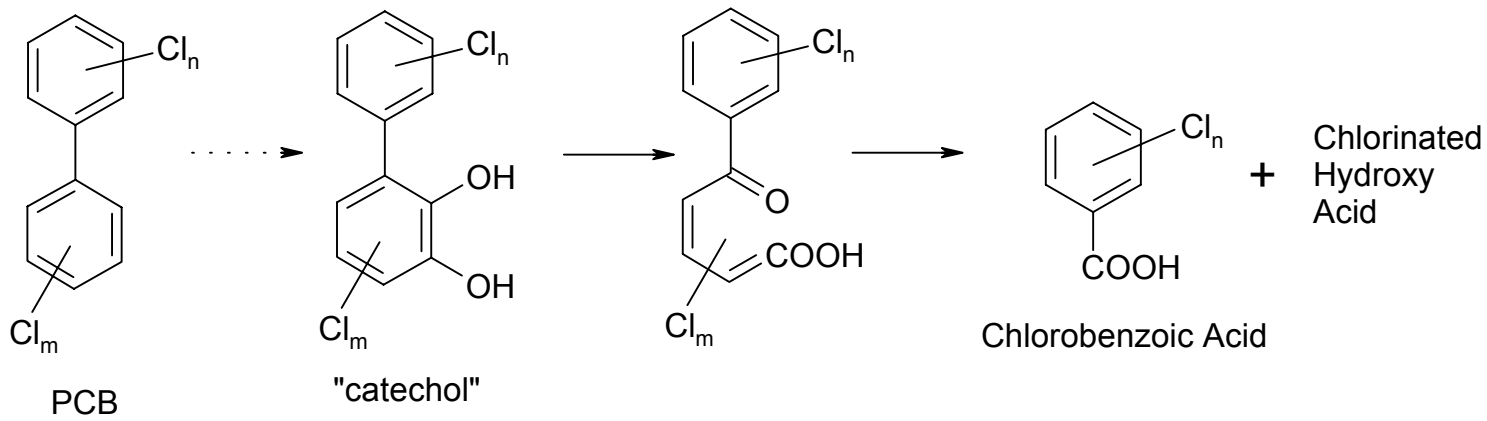
Numerous bacterial and some fungal isolates have been reported to aerobically biodegrade PCBs in the literature (Abramowicz 1990). Experiments with both pure and mixed microbial cultures show that some congeners of PCBs, usually containing from one to four chlorine substituents, are readily biodegraded aerobically (Abramowitz 1990), although biodegradation of congeners containing up to six or seven chlorine atoms has been shown under enrichment conditions (Abramowitz 1990; EPA 1983c; Gibson et al. 1993). The most common process for the aerobic degradation of PCBs by bacterial cultures proceeds in two distinct steps: first bioconversion to the corresponding chlorinated benzoic acid and secondly, mineralization of the chlorobenzoate to carbon dioxide and inorganic chlorides (Robinson and Lenn 1994; Thomas et al. 1992). Each step requires a separate group of genes (Afghan and Chau 1989; Robinson and Lenn 1994; Sondossi et al. 1992; Unterman et al. 1989). This pathway is further detailed in Figure 6-3 (Abramowicz 1990; Robinson and Lenn 1994). The initial attack of the biphenyl structure involves addition of O<sub>2</sub> by a biphenyl 2,3-oxygenase forming the corresponding unstable dihydro-dihydroxybiphenyl, subsequent dehydrogenation to the dihydroxybiphenyl, followed by *meta* ring cleavage to the corresponding chlorinated benzoic acid and a 5-carbon hydroxy-acid (Abramowicz 1990; Flanagan and May 1993; Robinson and Lenn 1994; Sylvestre and Sondossi 1994; Thomas et al. 1992). Steric hindrance of 2,3-dioxygenation, where the chlorine substituents prevent access of the 2,3 carbon atoms to the enzyme's active site is believed to be responsible for the inability of many higher chlorinated congeners to be oxidized (Abramowitz 1990; Sylvestre and Sondossi 1994). Aerobic oxidation of PCBs has been identified in the environment. A study by Flanagan and May (1993) reported the presence of chlorobenzoic acids, as well as other metabolites where the biphenyl ring is retained and in contaminated sediment cores taken from the Hudson River, but not in uncontaminated cores obtained upstream from the site of contamination. 2,3-Dioxygenase attack can also result in the formation of ring-chlorinated acetophenone from 3-chlorophenyl, 2,5- or 2,4,5-chlorophenyl PCB rings (Bedard 1990; Bedard et al. 1987).

Aerobic biodegradation of PCBs in the environment occurs mainly in soils and surficial sediments. PCB congeners with three or less chlorine substituents (major components in Aroclors 1221 and 1232) are

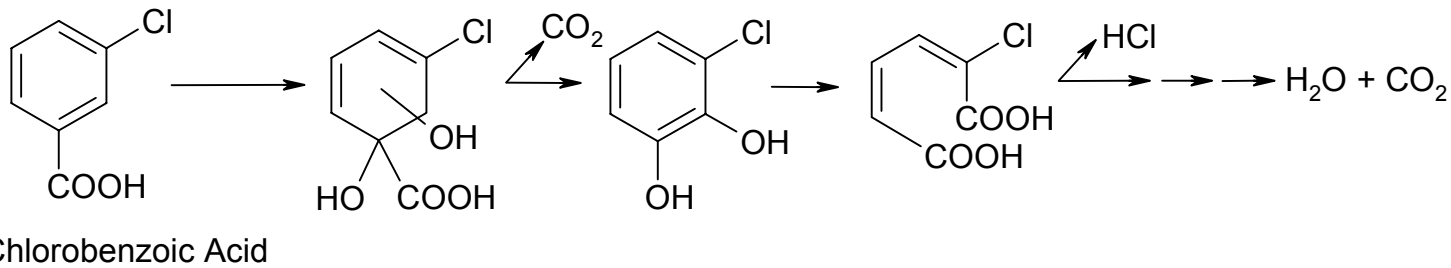


Figure 6-3. Pathways for Aerobic Degradation of PCBs

Cometabolism



Example of mineralization



Source: adapted from Adrigens et al. 1991; Robinson and Lenn 1994

## 6. POTENTIAL FOR HUMAN EXPOSURE

considered to be non-persistent, while those with five or more chlorines (major components in Aroclors 1248, 1254, and 1260) are not readily degraded and considered to be persistent (Abramowitz 1990; Alcock et al. 1996; EPA 1979i; Gan and Berthouex 1994; Iwata et al. 1973). Tetrachlorobiphenyls (major components in Aroclors 1016 and 1242) are intermediate in persistence. Thus, the addition of a PCB mixture to an aerobic environment results in a fractionating effect where the less chlorinated species biodegrade first and leave behind, for long-term build-up, the more highly chlorinated species. For example, Williams and May (1997) report that the addition of Aroclor 1242 to aerobic Hudson River sediment samples and incubated at 4 EC for several months resulted in biodegradation following an initial lag phase of 1.4 months. More than 50% loss was reported in 5 months, particularly among specific di- and trichlorobiphenyls. A single added hexachlorobiphenyl congener (2,2',4,4',6,6'-hexachlorobiphenyl) was not degraded over 300 days. In sludge-amended soil, the dissipation half-lives (first-order kinetics) of PCB 18 and 28, and total PCBs were reported to be on the order of <1–8.5 years (Alcock et al. 1996). Gan and Berthouex (1994) reported that the disappearance of PCBs in farmland soil amended with PCB-contaminated sludge was slow with half-lives (first-order kinetics) of di-CBs ranging from 7 to 11 months, while the half-lives of the tri-CBs and tetra-CBs ranged from 5 to 17 months and from 11 to 58 months, respectively. The mineralization of 2-, 3-, 4-, 2,2',5,5'-, 2,2',4,4'-, 2,2',3,3',5,5'-, and 2,2',4,4',5,5'-chlorobiphenyl was measured in a Flanagan silt loam containing 3% organic material; after 98 days, 19.7, 16.5, 16.1, 0.7, 0.4, 0.8, and 0.1% of the initially added radiolabel was found as CO<sub>2</sub> (Fries and Marrow 1984). In addition to the degree of chlorination, the chlorine substitution pattern affects the biodegradation rate of PCBs. For example, Afghan and Chau (1989) and Furukawa and Matsumura (1976) demonstrated that PCBs containing chlorine substituents on only one ring are degraded more quickly than PCBs containing an equivalent number of chlorine substituents distributed between both rings. Additionally, PCBs with chlorines found in *ortho* positions, such as 2,2'- and 2,6-dichlorobiphenyl, are more resistant to aerobic biodegradation than those with chlorines found in either the *para* or *meta* positions (Bedard and Haberl 1990; Furukawa et al. 1978; Robinson and Lenn 1994). The incubation of Aroclor 1242 in aerobic Hudson River sediment resulted in an enrichment of di- and trichlorobiphenyl congeners with di-*ortho* chlorines on one ring or di-*para* chlorines; other di- and trichlorobiphenyls in the mixture were readily degraded (Williams and May 1997).

Aerobic degradation rates of PCBs can be highly variable, depending not only on structural characteristics as outlined above, but also on a number of other factors including previous exposure to PCBs or PCB-like compounds, bioavailability, initial concentration, moisture, temperature, available nutrients such as carbon sources, and the presence of inhibitory compounds. Biodegradation of PCBs in aerobic soil is slow, especially in soils that have a high organic carbon content. PCBs that remain firmly bound in soil

## 6. POTENTIAL FOR HUMAN EXPOSURE

and sediment may not be bioavailable to the degrading organisms at sufficient concentrations. For example, in two soils containing >10% organic matter, only 5% biodegradation of Aroclor 1254 was observed after 1 year (Iwata et al. 1973). However, >25% biodegradation was observed after 1 year in a loamy, sandy soil containing only 0.1% organic carbon. In this study, the authors also observed that the less chlorinated PCB congeners were biodegraded more rapidly than the more highly chlorinated and tightly bound congeners (Iwata et al. 1973). Temperature also influences the rate of aerobic degradation. Williams and May (1997) evaluated the low temperature (4 EC) aerobic degradation of PCBs in sediment using Aroclor 1242-spiked samples of PCB-contaminated sediment from the Hudson River for several months. A 3- to 4-fold decrease in the rate of degradation was noted for sediment samples incubated at 4 EC versus those incubated at 25 EC. The aerobic biotransformation and biodegradation of PCBs can be enhanced by the use of adapted (pre-exposed) microbial populations and the addition of amenable substrates for co-metabolic and co-oxidative biotransformation. In a controlled laboratory aerobic microcosm sediment/water system, the half-lives (first-order kinetics) of Aroclors 1232, 1248, and 1254 were 61, 78, and 82 days, respectively, with no addition of substrates; 33, 39, and 36 days, respectively, with the addition of an amenable substrate; and 27, 32, and 36 days, respectively, with the addition of an amenable substrate and adapted microbes (Portier and Fujisaki 1988). Biphenyl or monochlorobiphenyls are commonly added as both growth substrates; they act to increase degradation rates through a cometabolic effect (Hurme and Puhakka 1997), as well as to induce the catabolic pathway required to sustain the growth of the PCB-degrading microbial population (Abramowicz 1990). Other studies report enhanced degradation rates in the presence of an added carbon source, such as sodium acetate, due to cometabolism (Pal et al. 1980). The efficiency of PCB degradation may also be controlled by the metabolite production pattern. Mono- and dichlorobenzoates, and possibly other higher chlorobenzoates formed from aerobic degradation of PCBs, have been shown to act as inhibitors towards further degradation of higher chlorinated PCBs (Guilbeault et al. 1994; Hickey et al. 1993; Robinson and Lenn 1994).

PCBs are slowly biodegraded in anaerobic environments by reductive dechlorination resulting in the formation of less toxic mono- and dichlorobiphenyl congeners, which are aerobically biodegradable (Abramowicz 1990, 1995; Anid et al. 1993; Brown et al. 1988; Chen et al. 1988; EPA 1983c; Larsson and Lemkemeier 1989; Pardue et al. 1988; Rhee et al. 1989). Until the 1980s, PCBs were not believed to be susceptible to anaerobic biodegradation based on studies measuring total PCB concentrations over time. Previous studies measured PCB loss as the change in total number of moles of PCBs over time. This generally remained the same as the biphenyl ring is not metabolized and only chlorine is released during reductive dechlorination. However, the overall congener distribution profile is markedly different

## 6. POTENTIAL FOR HUMAN EXPOSURE

following anaerobic biodegradation. The profile shows a decrease in concentration of the more highly chlorinated congeners and a corresponding increase in overall proportion of the less chlorinated congeners (Bedard and Quensen 1995). For example, Aroclor 1242 added to anaerobic Hudson River sediment was incubated for 73 weeks; at the end of this period, di-, tri-, tetra-, penta-, and hexachlorinated congeners were reduced by 11, 73, 66, 73, and 94%, respectively, while the concentration of monochlorobiphenyl congeners increased by 76% (Anid et al. 1993). The original homolog distribution of Aroclor 1254 versus that after 13 months incubation in anaerobic sediment (100 mg/L treatment) is as follows (in mole percent): tri-, 2 versus 18%; tetra-, 22 versus 51%; penta-, 48 versus 22%; hexa-, 21 versus 8.5%; hepta-, 6 versus 0.5% (Hurme and Puhakka 1997). Microbial PCB dechlorination is widespread in many anaerobic environments, including freshwater (pond, lake, and river) (Bedard and Quensen 1995; Wiegel and Wu 2000), estuarine (Brown and Wagner 1990; Tiedje et al. 1993), and marine sediments (Ofjord et al. 1994) for congeners with up to 10 chlorine substituents (Hartkamp-Commandeur et al. 1996), although other authors report dechlorination occurring for up to 7 (Quensen et al. 1990), 8 (Abramowitz 1990; Kuipers et al. 1999), or 9 (Kuipers et al. 1999) chlorines only. During reductive dechlorination, anaerobic bacteria use chlorine as the terminal electron acceptor in a two-electron transfer reaction involving the addition of the electron to the carbon-chlorine bond, followed by chlorine ( $\text{Cl}^-$ ) loss and subsequent hydrogen abstraction. The process of reductive dechlorination is illustrated in Figure 6-4 (Abramowicz 1990). Hydrogen ( $\text{H}_2$ ) is assumed to be directly or indirectly the electron donor and water the source of protons (Nies and Vogel 1991), although other sources are possible. For reductive dechlorination to occur, a low redox potential similar to methanogenesis ( $E_h < -400$  mV) and the absence of oxygen are thought to be required (May et al. 1992; Oremland 1988; Ye et al. 1992a), although some studies have shown that sulfidogenic redox conditions may also allow reductive dechlorination to proceed, but at a comparatively slower rate (Bedard and Quensen 1995; Hartkamp-Commandeur et al. 1996).

The most important structural factors determining whether a chlorine atom will be removed from a particular congener during anaerobic biodegradation include the position of the chlorine in relation to the opposite phenyl ring, the configuration of the surrounding chlorine atoms, the chlorine configuration of the opposite ring and, as summarized above, the total number of chlorine atoms. There are at least eight distinct, documented, reductive dechlorination pathways or processes, each resulting in a different congener distribution profile. These processes, M, Q, H, H', P, and N (and LP and T as in Wiegel and Wu 2000) are summarized in Table 6-9 (Bedard and Quensen 1995; Wiegel and Wu 2000; Wu et al. 1997). In any particular anaerobic environment, one or several of these processes may be occurring depending on the specificity that is developed by the adapted microbial population for dechlorination at a

**Table 6-9. Positions of Chlorines Removed by Each Dechlorination Process<sup>a</sup>**

Dechlorination process	Characteristic dechlorination products <sup>a</sup>	Susceptible chlorines	Susceptible Aroclors	Source of microorganisms
M	2 2, 2N/ 2, 6 <sup>b</sup> 2, 4N 2, 2N4 2, 4, 4N 2, 2N 6	Flanked and unflanked <i>meta</i>	1242 1248 <sup>a</sup> 1254 <sup>a</sup>	Upper Hudson Silver Lake
Q	2 2, 2N/ 2, 6 <sup>b</sup> 2, 3N 2, 2N 5 2, 2N 6 2, 3N 6	Flanked and unflanked <i>para</i>  <i>Meta</i> of 2, 3 group	1242 1248 1254	Upper Hudson
H !	2, 3N 2, 4N 2, 2N 4 2, 2N 5 2, 3N 4 2, 3N 5 2, 3N 6 2, 4, 4N/ 2, 4N 5 <sup>b</sup> 2, 2N 4, 4N <sup>c</sup> 2, 2N 4, 5N 2, 2N 5, 5N 2, 2N 3, 4N 5 <sup>c</sup> 2, 2N 3, 5, 5N 2, 2N 3, 4N 6 <sup>c</sup> 2, 2N 3, 5N 6 <sup>c</sup>	Flanked <i>para</i>  <i>Meta</i> of 2, 3 and 2, 3, 4 groups	1242 1248 1254 1260	Upper Hudson Lower Hudson New Bedford

Table 6-9. Positions of Chlorines Removed by Each Dechlorination Process<sup>a</sup> (continued)

Dechlorination process	Characteristic dechlorination products <sup>a</sup>	Susceptible chlorines	Susceptible Aroclors	Source of microorganisms
H	2, 3N	Flanked <i>para</i>	1242	Upper Hudson
	2, 3N 4		1248	Lower Hudson
	2, 3N 5	Doubly flanked <i>meta</i>	1254	New Bedford
	2, 3N 6		1260	Silver Lake
	2, 4, 4N 2, 4N 5 <sup>c</sup>			
	2, 2N 4, 4N			
	2, 2N 4, 5N			
	2, 2N 5, 5N			
	2, 2N 2, 4N 5			
	2, 2N 3, 5, 5N			
	2, 2N 3, 4N 6			
P	2, 2N 3, 5N	Flanked <i>para</i>	1254 <sup>a</sup>	Woods Pond
	2, 2N 4, 5N		1260	Silver Lake
	2, 2N 5, 5N			
	2, 2N 3, 3N 5			
	2, 2N 3, 5, 5N			
N	2, 4, 4N	Flanked <i>meta</i>	1254	Upper Hudson
	2, 2N 4, 4N		1260	Silver Lake
	2, 2N 4, 5N			Woods Pond
	2, 2, 4, 6N			
	2, 2N 4, 4N 6			
	2, 2N 3, 4N 5, 6			
LP	2, 2N 4	Flanked and unflanked <i>para</i>	1260	Woods Pond
	2, 2N 5			
	2, 2N 6			
T	2, 2N 4, 4N 5, 5N	Flanked <i>meta</i> of 2, 3, 4, 5 group	1260	Woods Pond
	2, 2N 3N 4, 4N 5			
	2, 2N 3, 4, 4N 5N 6	in hepta- and octa-CBs		

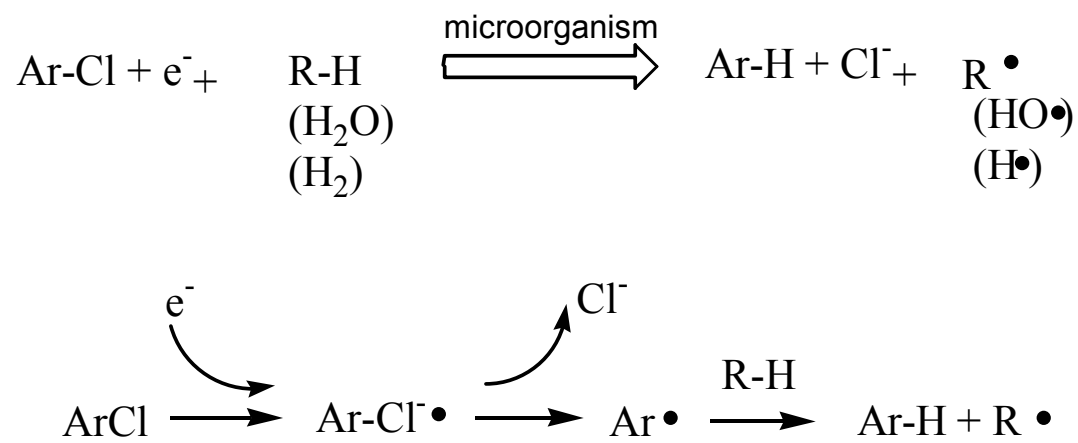
Source: Wiegel and Wu 2000; Wu et al. 1997; Bedard and Quensen 1995

<sup>a</sup>Products will vary depending on the congener composition of the PCB mixture being dechlorinated.

<sup>b</sup>Overlapping gas chromatograph peaks

<sup>c</sup>Proposed products from Aroclors 1254 and 1260

Figure 6-4. Possible Mechanism for Reductive Dechlorination by Anaerobic Microorganisms



Source: Abramowicz 1990

## 6. POTENTIAL FOR HUMAN EXPOSURE

particular position or dechlorination of a particular type of PCB (Alder et al. 1993; Bedard and Quensen 1995; Kuipers et al. 1999; May et al. 1989; Morris et al. 1992; Quensen et al. 1998; Robinson and Lenn 1994; Sokol et al. 1994; Tiedje et al. 1993; Wiegel and Wu 2000; Ye et al. 1992a; Zwiernik et al. 1998).

These processes can be combined at a single site producing different distribution profiles; combinations of M and H or M and H' have been observed (Bedard and Quensen 1995) as well as a combination of processes M and Q commonly called process C (Bedard and Quensen 1995). For example, it was observed that chlorine substituents were removed from only *meta* and *para* positions in Hudson River sediment contaminated with Aroclor 1242 resulting in a high proportion of *ortho* substituted mono- and di-chlorinated PCBs. The distribution profile of this sediment was believed to be due to dechlorination processes M, Q, and H or H' (Abramowicz et al. 1995; Bedard and Quensen 1995; Brown et al. 1984, 1987a, 1987b). In Silver Lake sediment contaminated with both Aroclor 1254 and 1260, *meta* and *para* chlorines were also preferentially removed (Williams 1994). However, *ortho* dechlorination of 2,4,6-CB was observed in cultures of Silver Lake sediment, suggesting a combination of dechlorination processes H, P, N, M, and Q (Bedard and Quensen 1995; Brown et al., 1984, 1987a, 1987b). While *ortho* dechlorination has been shown in the environment (Berkaw et al. 1996; Van Dort and Bedard 1991; Wu et al. 1998), most of the commonly reported processes outlined in Table 6-9 do not dechlorinate *ortho* chlorines resulting in the accumulation of less chlorinated, primarily *ortho*-substituted, PCB congeners due to anaerobic biodegradation (Abramowicz 1990, 1995; Bedard and May 1996; Berkaw et al. 1996; Brown et al. 1987; David et al. 1994; Morris et al. 1992; Nies and Vogel 1990; Rhee et al. 1993a, 1993b; Robinson and Lenn 1994; Tiedje et al. 1993; Van Dort and Bedard 1991; Wiegel and Wu 2000; Ye et al. 1992a).

The rate, extent, and specificity of anaerobic dechlorination can vary greatly even in the same sediment based on a number of environmental factors (Wiegel and Wu 2000). These include previous exposure to PCB or PCB-like compounds, electron acceptor availability, bioavailability, presence of co-contaminants, oxygen tension, redox level, temperature, pH, salinity, inhibitory compounds, available carbon and nutrients, and trace metals. Optimum rates of PCB dechlorination usually occur in the concentration range of 100–1,000 ppm (wet weight). Below a certain threshold concentration (<50 ppm), the rate of dechlorination is often very slow or non-quantifiable (Quensen et al. 1988; Rhee et al. 1993a; Robinson and Lenn 1994; Sokol et al. 1995, 1998). For example, Abramowicz et al. (1995) found that 93% of sediment samples containing >100 µg/g PCBs were extensively dechlorinated compared with only 63% of samples containing 5–10 µg/g. However, it should be noted that the reductive dechlorination of many PCB congeners in Aroclor mixtures has been observed even when their individual concentrations were



## 6. POTENTIAL FOR HUMAN EXPOSURE

<1 µg/g (Quensen et al. 1990; Schultz et al. 1989). PCBs generally remain tightly bound in soil and sediment, and may not be bioavailable to the biodegrading organisms even at optimum concentrations. The requirement of an optimum concentration (>50 ppm) may make this bioavailability factor critical for dechlorination of PCBs (Robinson and Lenn 1994; Tiedje et al. 1993). Bimodal desorption kinetics with both PCB-spiked and environmental sediments has been observed; approximately 50% of the initially present PCB mixture was found to be resistant to desorption with 50% of the resistant fraction desorbed over the following 6 months (Carroll et al. 1994; Harkness et al. 1993). Other authors report that desorption may not be as important given the slow rate of dechlorination of the more chlorinated congeners (Alder et al. 1993; Bedard et al. 1993). Addition of compounds, such as sodium lignisulfonate, that can increase the solubility of PCBs in soil and sediment has been shown to increase the rate of biodegradation (Sugiura 1992). Temperature is also an important factor controlling the rate of microbial dechlorination (Tiedje et al. 1993; Wiegel and Wu 2000; Wu et al. 1996, 1997). Temperatures in the range of 12–25 EC supported dechlorination, while dechlorination was not observed at temperatures >37 EC (Tiedje et al. 1993). Wu et al. (1997) reported optimal temperatures for overall chlorine removal of 20–27 EC in Woods Pond sediment contaminated with Aroclor 1260. Acid/base conditions may also affect the reductive dechlorination process. For example, the optimal pH for removal of chlorines in Woods Pond sediment contaminated with Aroclor 1260 and spiked with 2,3,4,6-tetraCB was approximately 7.0–7.5 (Wiegel and Wu 2000). The stereospecificity of dechlorination also varied as a function of pH with flanked *meta* dechlorination (Process N) occurring at pH 5.0–8.0, unflanked *para* dechlorination (Process LP) at pH 6.0–8.0, and *ortho* dechlorination at pH 6.0–7.5 (Wiegel and Wu 2000). As PCB-dechlorinating microorganisms are not able to cleave and utilize the biphenyl ring as a carbon and electron source, other compounds (e.g., mineral nutrients, electron donors, and carbon compounds) are required to help co-metabolize PCBs (Alder et al. 1993; Klasson et al. 1996; Sugiura 1992; Tiedje et al. 1993; Wiegel and Wu 2000). Alder et al. (1993) demonstrated that repeated addition of fatty acids (e.g., acetate, propionate, butyrate, and hexanoic acid) stimulated dechlorination of PCBs in carbon-limited sediment slurries, but not in sediment slurries with higher organic carbon contents. The omission of trace metals resulted in a slight reduction in the rate and extent of Aroclor 1242 dechlorination by Hudson River microorganisms (Abramowicz et al. 1993). Inhibition of PCB dechlorination can occur in the environment. Sokol et al. (1994) reported that high concentrations of co-contaminants at a site in the St. Lawrence River prevented dechlorination from occurring. Electron acceptors present in the environment may influence PCB reductive dechlorination. While most studies show reductive dechlorination of PCBs only under methanogenic and sometimes sulfidogenic conditions (Kuo et al. 1999; Ye et al. 1999), Morris et al. (1992) reports that dechlorination was shown under

## 6. POTENTIAL FOR HUMAN EXPOSURE

denitrifying and iron(III) reducing conditions as well (Bedard and Quensen 1995). Rates of dechlorination have been shown to be fastest in methanogenic (the most reducing) environments.

In the environment, aerobic and anaerobic biodegradation processes are often not readily separated and a combination of the two may be fairly common in aquatic environments. Hudson River sediment microcosms, spiked with Aroclor 1242, were designed with an aerobic surface sediment layer overlying a deeper anaerobic layer (Fish and Principe 1994). The distribution profile of congeners following degradation, was characterized by the authors as corresponding to a combination of process M dechlorination and aerobic biodegradation. Total PCB concentration decreased from 64.8 to 18.0  $\mu\text{mol/kg}$  sediment in 140 days. More recent studies have examined the potential of sequential anaerobic-aerobic treatment to degrade PCBs. As shown above, reductive dechlorination of PCBs in the environment often results in the accumulation of mono- and dichlorobiphenyls, the most commonly reported being the *ortho*-substituted congeners: 2-chlorobiphenyl, and 2,2'-, 2,4'-, 2,6-, 2,4-, and 2,3-dichlorobiphenyls (Adriaens and Grbic-Galic 1994). Hudson River sediment, containing 700  $\mu\text{g/g}$  Aroclor 1242, showed 55% removal of total chlorine after 16 weeks, but only from *meta* and *para* positions; the percentage of mono- and dichlorobiphenyls increased from 9 to 88% (Quensen et al. 1988). In a 73-day field study, aerobic biodegradation of a anaerobic sediment previously contaminated with Aroclor 1242 (upon release containing 9%, but at the time of the study containing 62–73%, mono- and dichlorobiphenyls) resulted in 35–55% further degradation of the less chlorinated PCBs, particularly when oxygen, biphenyl, and inorganic nutrients were provided (Harkness et al. 1993). However, the extent and type of dechlorination is not predictable from site to site, and congeners remaining from anaerobic biodegradation may also be resistant to aerobic biodegradation. For example, dechlorination of Aroclor 1254 in sediment resulted in the accumulation of tri- and tetrachlorobiphenyl congeners; they were not dechlorinated further to mono- and dichlorobiphenyls in this sediment and would be expected to be comparatively more resistant to aerobic biodegradation (Hurme and Puhakka 1997). The reductive dechlorination of Aroclor 1254 in a marine sediment system resulted in the accumulation of *ortho* tetra- and pentachlorobiphenyls. When this culture was then subjected to aerobic biodegradation, no biodegradation was shown over 2 months. Many of the remaining congeners had either two *ortho* or two *para* groups, making them resistant to aerobic biodegradation as well (Mannisto et al. 1997).

Biodegradation of PCBs in aerobic or anaerobic groundwater has not been studied, although PCBs have been reported in groundwater environments (Section 6.4.2). In aerobic groundwater, the less-chlorinated PCB congeners, which would be more likely to leach, would presumably degrade based on studies in aerobic surface waters and soil. However, groundwater is also commonly anaerobic and, as is covered in

## 6. POTENTIAL FOR HUMAN EXPOSURE

this section, microbial degradation under this oxygen condition proceeds for even the more highly chlorinated congeners. In a contaminant plume, as might be seen at a landfill site, sequential dechlorination of the more highly chlorinated PCB congeners may occur in the anaerobic plume while aerobic biodegradation at the anaerobic/aerobic interface of the plume edge may degrade some of the less chlorinated PCBs.

**6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT**

Reliable evaluation of the potential for human exposure to PCBs depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. With respect to PCB analysis, comparisons among various studies are complicated by the fact that authors may report PCB concentrations as Aroclors, as homologs, or as congeners. Historically, Aroclor analysis has been performed by most laboratories. This procedure can, however, result in significant error in determining total PCB concentrations (Schwartz et al. 1987) and in assessing the toxicological significance of PCBs because it is based on the assumption that distribution of PCB congeners in environmental samples and parent Aroclors is similar. The distribution of PCB congeners in Aroclors is, in fact, altered considerably by physical, chemical, and biological processes after their release into the environment, particularly when the process of biomagnification is involved (Oliver and Niimi 1988; Smith et al. 1990). Only recently has it become more common to determine the concentration of individual PCB congeners. However, major problems have been associated with the identification of the individual congeners, as only a limited number of standards have been available (Larsen 1995). In addition, in those studies that report results as total PCBs, the definition of what constitutes total PCBs (i.e., how many and which congeners are summed) is often not the same in the various studies. Problems related to chemical analysis procedures and reporting of total PCBs are discussed in greater detail in Chapter 7. In reviewing data on levels monitored or estimated in the environment, it should be noted that the amount of the chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. Monitoring studies indicate that atmospheric concentrations of PCBs tend to be dominated by lower chlorinated, more volatile congeners, especially at northern latitudes (Halsall et al. 1999; Harner et al. 1998; Ockenden et al. 1998). Concentrations in soils and sediments, which are dominated by highly chlorinated congeners, have followed a downward trend over time and appear to have reached a steady state concentration in several locations (Bopp et al. 1998; Lead et al. 1997; Van Metre et al. 1998). Water monitoring studies indicate that PCB concentrations have also decreased since the late 1970s due to the cessation of production and manufacturing (Anderson et al. 1999; Jeremiason et al. 1998). Aquatic species, including fish and sea mammals, have shown a similar downward trend in PCB contamination with highly chlorinated

























































## 6. POTENTIAL FOR HUMAN EXPOSURE

early 1970s to the late 1980s and that they appeared to have been in a steady state condition. For example, the concentration of hexachlorobiphenyls in cod-liver oil were 3.2 (1971), 2.5 (1975), 7.3 (1980), 3.3 (1985), 4.0 (1986), and 3.8  $\mu\text{g/g}$  (1989). In general, the hexa- and pentachlorobiphenyls were most predominant in the cod liver oil samples (Falandysz 1994).

Proximity to industrialized regions has been shown to influence PCB concentrations in fish. Forage fish collected in 1991 from coastal wetlands and beaches in Green Bay, Lake Michigan were analyzed for PCB concentrations (Brazner and DeVita 1998). The concentration of PCBs in fish species appeared to vary spatially within Green Bay, according to proximity to the city. For example, PCB concentrations in fish (lipid normalized) ranged from 0.048 to 0.458  $\mu\text{g/g}$  in the lower bay (closest to the city), 0.040–0.078  $\mu\text{g/g}$  in middle bay, and 0.003–0.011  $\mu\text{g/g}$  in the upper bay (furthest from the city). Mean levels of PCBs were also measured in fish above and below dams on rivers that feed into Lakes Michigan and Huron (Giesy et al. 1995). Researchers found that concentrations were higher downstream from the dams than upstream. For example, mean PCB concentrations in fish below dams found along the Manistee, Muskegon and Au Sable Rivers were 1.90, 3.40, and 1.10  $\mu\text{g/g}$  wet weight, respectively, while above the dam, mean concentrations were 0.020, 0.195, and 0.061  $\mu\text{g/g}$  wet weight, respectively. In a National Study of Chemical Residues in Fish conducted by EPA between 1986 and 1989, PCBs were detected at a mean concentration of 1.90  $\mu\text{g/g}$  in bottom-feeding and game fish collected from 91% of 362 sites surveyed (EPA 1992c; Kuehl et al. 1994). Fish collected from 26% of the sites contained PCB residues  $>1$   $\mu\text{g/g}$ , and fish from 2.5% of the sites contained PCB residues at  $>10.0$   $\mu\text{g/g}$ . Fish collected at sites near wood-preserving facilities, industrial/urban areas, pulp and paper mills, refineries/other industrial sites, and Superfund sites were more highly contaminated with PCBs than fish collected near agricultural areas, near POTW sites, or at U.S. Geologic Survey (USGS) National Stream Quality Accounting Network sites (EPA 1992c; Kuehl et al. 1994). Concentration patterns of various homologs in fish tissues were as follows: mean concentrations of total mono-, di-, octa-, nona-, and decachlorobiphenyl were  $<0.025$  ppm; mean concentrations of total tri- and heptachlorobiphenyl were  $<0.150$  ppm; and mean concentrations of total tetra-, penta-, and hexachlorobiphenyl were 0.699, 0.565, and 0.356 ppm, respectively (EPA 1992c). Table 6-16 compares PCB concentrations in tissues of six species of fish collected near the Mohawk Reservation at Akwesasne adjacent to the PCB contaminated General Motors Foundry site and downstream in the St. Lawrence River and its tributaries (ATSDR 1995). The highest mean concentration of PCBs in standard fillet tissues (20.55  $\mu\text{g/g}$ ) was detected in brown bullheads collected at the General Motors Corporation site. These values are far higher than the background concentrations of  $<0.100$   $\mu\text{g/g}$  for the total PCBs in fish fillets in New York State (Sloan and Jock 1990).

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-16. Mean Total PCB Levels in Standard Fillets of Fish Collected from the Vicinity of a Superfund Site**

Species	CM-CFD <sup>a</sup> site mean (range) <sup>b</sup>	St. Lawrence River and tributaries <sup>c</sup> mean (range) <sup>b</sup>
Brown bullhead	20.55 (<0.15–81.49)	1.82 (<0.15–10.73)
Northern pike	2.73 (0.48–5.12)	0.42 (<0.15–3.52)
Rock bass	1.04 (<0.15–4.02)	0.18 (0.15–0.86)
White sucker	6.39 (0.29–11.0)	0.17 (<0.15–0.63)
Yellow perch	3.41 (0.20–12.26)	0.61 (0.15–0.86)

Source: ATSDR 1995

<sup>a</sup>General Motors Corporation/Central Foundry Division, Massena, New York, plant is a Superfund site adjoining the Mohawk Indian Nation at Akwesasne. Fish and wildlife studies and human health studies have been conducted at this site.

<sup>b</sup>(ppm, wet/weight)

<sup>c</sup>PCB contamination has been detected within the Akwesasne reservation boundaries in the St. Lawrence River and several tributaries downstream from the General Motors Corporation site.

## 6. POTENTIAL FOR HUMAN EXPOSURE

Fish that inhabit remote areas of the world have also been shown to bioaccumulate PCBs. From 1993 to 1994, PCB residues were evaluated in Kokanee fish and lake trout from the Sierra Nevada ecosystem to determine the extent of organochlorine pollution in high altitude, alpine regions (Datta et al. 1999). Analysis of fish muscle revealed that the concentration of total PCBs ranged from 0.018 to 0.430  $\mu\text{g/g}$  wet weight for lake trout. Compared to trout, Kokanee fish generally had lower PCB concentrations ranging from 0.013 to 0.044  $\mu\text{g/g}$  wet weight. Residue analysis indicated that the congeners most commonly found ranged from penta- to heptachlorobiphenyls (Datta et al. 1999). Although Lake Tahoe does not have any known point sources of pollution from industry or agriculture, the levels of PCBs in lake trout samples were approximately equal to or slightly lower than those found in lake trout from Lake Superior. This suggests that PCBs have been introduced to Lake Tahoe through atmospheric deposition. For further information, refer to Table 6-17.

The remediation of contaminated sediments has been shown to decrease PCB concentrations in fish inhabiting contaminated rivers. For example, one study monitored PCB concentrations in fish before and after remediation of PCB contaminated sediment (Bremle and Larsson 1998). It was found that PCB concentrations in lake water decreased from 0.0086 to 0.0027  $\mu\text{g/L}$ , while concentrations in fish were halved after remediation was completed. Although concentrations of PCBs in fish decreased after remediation, the relative composition of PCB congeners remained relatively the same as before remediation. Fish still concentrated higher chlorinated congeners relative to levels detected in lake water (Bremle and Larsson 1998).

PCB concentrations have also been monitored in zebra mussels collected from the lower Saginaw River and Saginaw Bay, Michigan in the winter of 1991 (Endicott et al. 1998). The results indicated that the concentration of PCBs decreased in zebra mussels collected further away from the mouth of the Saginaw River. For example, the PCB concentration in zebra mussels from the mouth of the river was approximately 1.1  $\mu\text{g/g}$ , while concentration in zebra mussels collected 20 km away was approximately 0.45  $\mu\text{g/g}$ . The same concentration gradient was also observed in water samples taken from the zebra mussel collection sites. This suggests that PCB levels in zebra mussels are directly related to PCB concentrations in water (Endicott et al. 1998). Data from the Mussel Watch, plus additional data on shellfish (oysters) from North and South American coastal locations, indicate PCB congeners in shellfish were highest from South American locations and lowest in Central America (Sericano et al. 1995). Among 51 sites along the north Gulf of Mexico coast, samples with concentrations  $>0.100 \mu\text{g/g}$  were reported from 15 sites. Nevertheless, average concentrations of PCBs in shellfish from these 51 Gulf of

**Table 6-17. Mean PCB Concentrations in Fish from Remote Areas**

Species	Location	Year	PCB concentration µg/g (wet weight) <sup>a</sup>	Source
Char	Arctic Quebec, Canada	1989–1990	0.152 ± 0.042 <sup>b</sup>	Dewailly et al. 1993
Trout	Schrader Lake in the Alaskan Arctic	1992	0.0066	Wilson et al. 1995
Grayling	Schrader Lake in the Alaskan Arctic	1992	0.0013	Wilson et al. 1995
Lake trout	Sierra Nevadas	1993–1994	0.018–0.430	Datta et al. 1999
Kokanee fish	Sierra Nevadas	1993–1994	0.013–0.044	Datta et al. 1999
Lake trout	Siskiwit Lake	1996–1997	(0.040–0.460)	Kannan et al. 2000

<sup>a</sup>Ranges in parenthesis

<sup>b</sup>Lipid weight



## 6. POTENTIAL FOR HUMAN EXPOSURE

Mexico sites decreased between 1986 and 1993. For more information on PCB concentrations in crustaceans, refer to Table 6-18.

PCB concentrations in the tissues of edible turtles, and in some cases frogs, are also of concern with respect to human exposure, particularly for populations engaged in recreational and subsistence hunting. Hebert et al. (1993) evaluated the concentrations of PCBs in muscle tissue of 78 adult snapping turtles collected from 16 sites in southern Ontario, Canada. Mean concentrations (wet weight) of PCBs from all 16 sites ranged from <0.200 to 0.655  $\mu\text{g/g}$ . Skinner (1992) also reported concentrations of PCBs in fat, liver, and muscle tissue from snapping turtles collected near Akwesasne, where turtles are a source of food for a Native American community of nearly 10,000 people. This author reported concentrations of total PCBs (wet weight) ranging from 36.10 to 1,347 ppm in fat, 2.85–94.77 ppm in liver tissue, and not detected to 2.98 ppm in muscle tissue of snapping turtles. Northern leopard frogs from six wetlands located along the Fox River and around Green Bay were collected from 1994 to 1995 and analyzed for total PCB concentrations (Huang et al. 1999). PCB levels in frog tissues ranged from 0.002 to 0.200  $\mu\text{g/g}$  wet weight with the highest concentrations found in frogs from the upper Fox River, furthest away from Green Bay, Wisconsin. Mean residues of Aroclors 1254 and 1260 in tissues of frogs, collected along the Canadian shores of Lakes Erie and Ontario, and the St. Lawrence River, ranged from 0.310 to 1.699  $\mu\text{g/g}$  lipid weight in green frogs and 0.276–1.566  $\mu\text{g/g}$  lipid for leopard frogs (Gillan et al. 1998). Based on frog tissue content and sediment PCB content, biota-sediment accumulation factors of 33.28–1.06 and 23.02–0.42 were calculated for leopard frogs and green frogs, respectively.

PCB concentrations were analyzed in fat, liver, and muscle tissue of commonly hunted red and grey squirrels, beaver, muskrat, snowshoe hares, cottontail rabbits, and white-tailed deer (Skinner 1992). PCBs were typically found only in fatty tissues and occasionally in liver tissues, but were not detected in muscle tissue. Only two liver-tissue samples from muskrats contained detectable concentrations of PCBs. The highest concentration 0.7  $\mu\text{g/g}$  wet weight was detected in a male muskrat. Total PCBs were above detection limits more frequently in mammalian fatty tissues, but only in muskrat and cottontail rabbits. Maximum concentrations of 0.8  $\mu\text{g/g}$  wet weight and 4.0  $\mu\text{g/g}$  lipid weight occurred in male muskrat from the St. Lawrence River near Raquette Point and the St. Regis River, respectively (both sites are within the New York State portion of the Mohawk Nation Reservation at Akwesasne). Wild game provides an important food source for both recreational and subsistence hunters; eating wild game is also a significant cultural activity for many Native Americans (Skinner 1992).

**Table 6-18. Mean Concentration of PCBs in Crustaceans**

Species	Location	Year	PCB concentration µg/g (wet weight)	PCB concentration µg/g (lipid weight)	Source
Blue mussel	Belgian Fisheries	1983		2.4±0.3	Roose et al. 1998
		1993		1.6±0.1	
Brown shrimp	Belgian Fisheries	1983		0.49±0.08	Roose et al. 1998
		1993		0.28±0.05	
Oysters	Galveston Bay, Texas	1986	0.098 <sup>a</sup>		Jackson et al. 1998
		1988	0.100 <sup>a</sup>		
		1990	0.099 <sup>a</sup>		
		1992	0.058 <sup>a</sup>		
		1993	0.036 <sup>a</sup>		
Mussels/Oysters	U.S. Nationwide	1986	(0.009–6.808) <sup>a</sup>		Sericano et al. 1995
Blue mussels	Nordic Seas	1989–1990		(0.038–3.3)	Gustavson and Jonsson 1999
Zebra mussels	Mouth of Saginaw River	1991	1.1		Endicott et al. 1998
Zebra mussels	Saginaw Bay (59 km from mouth of Saginaw River)	1991	0.076		Endicott et al. 1998
Flat tree oysters	Morrocoy National Park, Venezuela	1991	(0.0006–0.012)		Jaffe et al. 1998
Zebra mussels	Saginaw Bay	1991	0.45		Endicott et al. 1998
Grass shrimp	Coastal Georgia	1996	0.33		Maruya and Lee 1998
Gei wai shrimp	Mai Po, Hong Kong	1997	0.0064		Liang et al. 1999
Caridean shrimp	Mai Po, Hong Kong	1997	0.0046		Liang et al. 1999

<sup>a</sup>Dry weight; Ranges in parenthesis

## 6. POTENTIAL FOR HUMAN EXPOSURE

Waterfowl may also be an important source of human exposure, especially for avid hunters. Table 6-19 presents temporal PCB concentrations found in several species. Tissues of fish- and shellfish-eating waterfowl (i.e., goldeneye and mergansers) contained significantly higher PCB concentrations than tissues of dabbling ducks (i.e., black ducks and mallards), which, in turn, contained higher concentrations of PCBs than tissues of grazers (i.e., Canada geese) whose food preferences include aquatic vegetation, upland grass, and grain (Rathke and McRae 1989). The concentration of total PCBs was determined in breast meat of Canadian geese collected from northeastern Illinois in 1994 (Levengood et al. 1999). Breast meat samples were baked, allowing fat to drip free, and assayed for PCB residues. Overall, PCBs were detected in only 5 of 87 tissue samples in baked breast meat (detection limit 0.01 µg/g) with residues occurring more frequently in muscle than in skin. PCB concentrations ranged from 0.114 to 0.480 µg/g (Levengood et al. 1999).

Herring gull eggs have routinely been collected by the Canadian Wildlife Service since 1974 to monitor contaminant trends. The PCB concentrations in eggs taken from several nesting colonies on the Great Lakes decreased from 1974 to 1986 (Rathke and McRae 1989). The trend analysis determined that the PCB concentration in herring gull eggs taken from two colonies on Lake Ontario was . 140 mg/kg wet weight in 1974 and fell to approximately 40 mg/kg in 1986. Similarly, the concentration of PCBs in eggs from a colony on Lake Erie was 60 mg/kg in 1974, but fell to <40 mg/kg in 1986. An analysis of PCB concentration in gull eggs in the Great Lakes from 1978 to 1992 indicates that egg concentrations have stabilized (Stow 1995b). Similar conclusions have been determined by other research efforts. Levels of PCBs in herring gull eggs from Great Lakes breeding colonies declined rapidly following the ban of PCBs in 1972. Since the mid 1980s, however, the concentration of PCBs in gull eggs has essentially stabilized in Lakes Superior, Michigan, Huron, and Ontario (Donaldson et al. 1999). In Lake Ontario, herring gull egg PCB concentrations show annual variation due to increased feeding on alewives during colder weather when alewives are particularly abundant (Hebert et al. 1997). Unhatched eggs and plasma samples from preledged bald eagles were analyzed for PCB concentrations in the Canadian Great lakes Basin (Donaldson et al. 1999). The study found the mean PCB concentration in unhatched bald eagle eggs collected along Lake Erie from 1974 to 1980 was 84 mg/kg wet weight and decreased to 26.4 mg/kg wet weight from 1989 to 1994. From 1990 to 1996, however, no significant decrease in plasma residue levels was observed from either Lake Erie or Lake Superior (Donaldson et al. 1999).

Bottlenose dolphins (*Tursiops truncatus*) collected during a 1990 mortality event along the Gulf Coast of the United States contained mean PCB concentrations of 93, 7.2, 49, 21, and 4 µg/g lipid basis in adult males, adult females, immature dolphins, suckling dolphins, and fetuses, respectively (Kuehl and Haebler

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-19. Mean PCB Concentrations in Animals**

Species	Location	Year	PCB concentration µg/g (wet weight) <sup>a</sup>	Source
Bufflehead duck	New York State	1983–1984	0.15±0.19	Foley 1992
Scaup duck	New York State	1983–1984	0.13±0.12	Foley 1992
Mallard duck	New York State	1983–1984	0.08±0.06	Foley 1992
Black duck	New York State	1983–1984	0.07±0.07	Foley 1992
Wood duck	New York State	1983–1984	0.05±0.01	Foley 1992
Canada geese	New York State	1983–1984	0.05±0.01	Foley 1992
Waterfowl	Eastern Lake Ontario/ St. Lawrence River	1983–1985	(<0.01–0.27)	Rathke and McRae 1989
Mallard duck	Wisconsin	1984–1989	(ND–0.021)	Botero et al. 1996
Grebe duck	British Columbia	1989	0.542 (liver tissue)	Elliott and Martin 1998
Seaduck	British Columbia	1989	1.770 (liver tissue)	Elliott and Martin 1998
Polar bear	Arctic Quebec	1989–1990	7.002±1.276 (lipid weight)	Dewailly et al. 1993
Mink	Georgia	1989–1991	0.154 (liver tissue)	Osowski et al. 1995
Mink	South Carolina	1989–1991	0.219 (liver tissue)	Osowski et al. 1995
Mink	North Carolina	1989–1991	0.216 (liver tissue)	Osowski et al. 1995
Mallard duck	Hamilton Harbor, Canada	1990	0.161	Gebauer and Weseloh 1993
Mink	Northwest Territories, Canada	1991–1995	0.007–0.0731 (liver tissue)	Poole et al. 1998
Sea otters	Aleutian Islands	1992–1998	0.310±0.480	Bacon et al. 1999
Sea otters	California Coast	1992–1998	0.190±0.350	Bacon et al. 1999
Sea otters	Southeast Alaska	1992–1998	0.008±0.014	Bacon et al. 1999

<sup>a</sup>Ranges in parenthesis

ND = not detected

## 6. POTENTIAL FOR HUMAN EXPOSURE

1995). This trend generally reflects increased accumulation with age, with the exception of the adult females. The lower PCB residues in adult females are possibly due to the loss of PCBs via placental transfer and via lactation by the adult females who are suckling their young (Kuehl and Haebler 1995). In a similar study with stranded bottlenose dolphins, Salata et al. (1995) reported a mean total PCB concentration of 36.1  $\mu\text{g/g}$ . The mean concentrations of homolog groups di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decachlorobiphenyls were 0.046, 1.1, 2.2, 6.16, 15.1, 8.58, 2.23, 0.42, and 0.264  $\mu\text{g/g}$ , respectively. Similar to what is observed in other aquatic organisms, highly chlorinated PCB congeners are preferentially bioconcentrated. Blubber and liver samples from six striped dolphins found dead in the Mediterranean sea from 1989 to 1990 were analyzed for PCBs (Reich et al. 1999). Researchers found that the mean concentration of total PCBs in dolphin blubber was  $35.6 \pm 47.7$   $\mu\text{g/g}$  wet weight and in liver samples was  $8.74 \pm 11.7$   $\mu\text{g/g}$  wet weight. Of the 37 congeners monitored in dolphin tissue, PCBs 138, 153, 170, and 180 comprised approximately 60% of total PCBs (Reich et al. 1999). Non-*ortho* and mono-*ortho* PCB congeners, however, contributed <1% of total PCBs in blubber and liver tissues. Of these, PCB 77 was detected most frequently and in the highest concentrations.

By comparing concentrations of PCBs in a mother dolphin and her unborn calf, the authors determined that the mother transferred 3.7% of her total PCB body burden to the calf during her pregnancy. It was also found that the lower molecular weight PCB congeners were more easily transferred than the higher molecular weight congeners and that congeners with 9 or 10 chlorine atoms may not have been transferred at all transplacentally. PCB concentrations were measured in milk from five captive bottlenose dolphins originally collected in the Gulf of Mexico (Ridgway and Reddy 1995). Maximum concentrations of PCBs in the milk were 4.45 ppm wet weight (14.1 ppm lipid weight) as Aroclor 1254, found in the milk produced by a 34-year-old female, the oldest of the population sampled. The lowest concentration, 0.281 ppm wet weight (1.38 ppm lipid weight), was in milk from one of the youngest, a 16-year-old female. For more information concerning PCB concentrations detected in blubber of several sea mammals, see Table 6-20. Based on a review of the literature on PCB residues in mammalian species, Kamrin and Ringer (1994) concluded that the lowest residues were found in the Antarctic, while the highest were in northern latitudes, particularly the Baltic Sea, with overall trends showing decreasing residues of PCBs over the past 10–15 years. Among male beluga whales from the St. Lawrence River estuary, tissue PCB concentrations decreased by a factor of 1.9 from 1982 to 1994 (Muir et al. 1996a). In the 1993–1994 samples, male and female beluga whales had geometric mean total PCB concentrations of 29.6 and 78.9  $\mu\text{g/g}$  lipid basis, respectively (Muir et al. 1996b). PCB 126 was detected most often and in the highest concentrations of the four coplanar PCBs (77, 81, 126, and 169).

**Table 6-20. Mean PCB Concentrations in Blubber of Sea Mammals**

Species	Location	Year	PCB concentration µg/g wet weight <sup>a</sup>	PCB concentration µg/g lipid weight	Source
Harbour porpoise	Kattegat-Skagerrak Seas, Norway	1978–1981 1989–1990		40±22 13±5.2	Berggrena et al. 1999
Porpoise	Irish Sea	1987–1989		6.19	Troisi et al. 1998
Dolphin	Irish Sea	1987–1989		(2.80–15.48)	Troisi et al. 1998
Striped dolphin	Mediterranean Sea	1989–1990	35.6±47.7		Reich et al. 1999
Beluga whale	Arctic Quebec	1989–1990		1.002±0.469	Dewailly et al. 1993
Seal	Arctic Quebec	1989–1990		0.527±0.692	Dewailly et al. 1993
Bottlenose dolphin (adult male)	U.S. Gulf Coast	1990		93	Kuehl and Haebler 1995
Bottlenose dolphin (adult female)	U.S. Gulf Coast	1990		7.2	Kuehl and Haebler 1995
Bottlenose dolphin (juvenile)	U.S. Gulf Coast	1990		49	Kuehl and Haebler 1995
Bottlenose dolphin (suckling)	U.S. Gulf Coast	1990		21	Kuehl and Haebler 1995
Bottlenose dolphin (fetus)	U.S. Gulf Coast	1990		4	Kuehl and Haebler 1995
Harbor seal pup	Mouth of Puget Sound	1990	(1.3–2.1)		Hong et al. 1996
Harbor seal pup	Head of Puget Sound	1990	(9.2–16)		Hong et al. 1996
Adult harbor seal	Mouth of Puget Sound	1990	(0.17–0.32)		Hong et al. 1996
Adult harbor seal	Head of Puget Sound	1990	(1.1–2.3)		Hong et al. 1996

**Table 6-20. Mean PCB Concentrations in Blubber of Sea Mammals (continued)**

Species	Location	Year	PCB concentration µg/g wet weight <sup>a</sup>	PCB concentration µg/g lipid weight	Source
Striped dolphin	Aegean Sea	1991		21.52±2.76	Troisi et al. 1998
Harbor seal	San Francisco Bay	1991–1992	0.050 <sup>a</sup>		Young et al. 1998
Beluga whale (fetus)	Alaskan North Coast	1992	1.35		Wade et al. 1997
Beluga whale (adult female)	Alaskan North Coast	1992	(0.70–2.16)		Wade et al. 1997
Beluga whale (adult male)	Alaskan North Coast	1992	(5.24–9.42)		Wade et al. 1997
Ringed seal (female)	Canadian Arctic	1993		0.387±0.073	Letcher et al. 1998
Ringed seal (male)	Canadian Arctic	1993		0.447±0.092	Letcher et al. 1998
Beluga whale (adult male)	St. Lawrence River	1993–1994		29.6	Muir et al. 1996b
Beluga whale (adult female)	St. Lawrence River	1993–1994		78.9	Muir et al. 1996b
Sperm whale (male)	North Sea	1994–1995		4.5	Holsbeek et al. 1999
Seal	Caspian Sea	1996–1997		(1.12–19.08)	Hall et al. 1999

<sup>a</sup>Concentration reported in blood; ranges in parenthesis

## 6. POTENTIAL FOR HUMAN EXPOSURE

PCBs have also been detected in several consumer products manufactured outside the United States. PCB concentrations in recycled paper products (envelopes, toilet paper, tissue paper, and cardboard boxes) of central European origin were in the range of 5–6,000  $\mu\text{g}/\text{kg}$  (ppb) (Welling et al. 1992). PCBs (Aroclor 1254) also were detected in both anhydrous lanolin and lanocerin and in finished cosmetic products produced in Italy (Mariani et al. 1994). PCB concentrations detected in the various products included 1.2 ppm in anhydrous lanolin, 4.8 ppm in lanocerin, 0.64 ppm in anhydrous cream for children, 0.52 ppm in oil/water emulsion-emollient cream, and 3.8 ppm in water/oil emulsion-emollient cream.

### 6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

PCBs are no longer manufactured or used (except under exemption) in the United States. Nonetheless, PCBs are present in the environment due to the recycling of the compounds released into the environment by historic anthropogenic production activities. The general population may be exposed to PCBs primarily via consumption of contaminated foods, particularly fish, meat, and poultry, and inhalation (Gunderson 1988). No known consumer product currently manufactured in the United States contains PCBs. Exposure of the general population to PCBs is evidenced by the body burden of the compounds among the nonoccupationally exposed population. PCBs have been detected in the blood, adipose tissue, and breast milk of nonoccupationally exposed members of the general population (EPA 1986b; Greizerstein et al. 1999; Gunderson 1995; Ouw et al. 1976). Overall, PCBs 138, 153, and 180 are the most consistently detected and quantitatively dominant congeners found in human tissues (Hansen 1998). These three congeners have been used to monitor both geographical and temporal trends in human exposure studies due to their high prevalence and persistence (Koopman-Esseboom et al. 1994a, 1994b; Schechter et al. 1989). Other congeners that are commonly detected include PCBs 28, 118, and 170. As the summaries in Section 6.4 indicate, there is a general overall trend for decreasing concentrations of PCBs in most environmental media over the past 2 decades; air concentrations have decreased slightly, and levels in water, sediments, and fish have decreased, in some cases significantly. As noted in this section, PCB body burdens in humans also have decreased, as evidenced by lower levels reported in human adipose tissue, blood serum, and breast milk.

The National Human Adipose Tissue Survey (NHATS), conducted in 1982 using packed column gas chromatography (GC), found that the concentrations of total PCBs in composite human adipose tissue ranged from 14 to 1,700  $\text{ng}/\text{g}$  (0.014–1.7 ppm) (lipid basis) (EPA 1986b). The maximum concentration was found in a sample composite collected from the South Atlantic region (Virginia, North Carolina, South Carolina, Georgia, and Florida). PCBs were detected in 83% of 46 composite samples analyzed in



## 6. POTENTIAL FOR HUMAN EXPOSURE

the NHATS study. In a subsequent study, Kutz et al. (1991) reported that an examination of the NHATS data from 1970 to 1983 revealed that 93.5% of the U.S. population had detectable concentrations of PCBs, 66.4% had concentrations <1 ppm, 28.9% had concentrations >1 ppm, and 5.1% had concentrations >3 ppm. The 0–14-year-old age group had the smallest percentage of individuals with detectable concentrations of PCBs and the smallest percentage of individuals exceeding the 1 and 3 ppm PCB concentrations; males had a higher percentage of individuals exceeding 1 ppm PCBs than did females; and the Northeast Census Region had the greatest percentage of individuals exceeding 3 ppm. A time-trend analysis of the data also revealed that the percentage of the population having PCB concentrations from 1 to 3 ppm steadily declined from over 62% in 1972 to <2% in 1984. The percentage of the population that had tissue levels >3 ppm also declined during this same period from a high near 10% in 1978 to zero in 1984 (Fensterheim 1993). PCB concentrations in human adipose tissues in the United States appear to have decreased significantly over the years.

Results of congener-specific analysis of PCBs in human adipose tissues obtained from Atlanta, Georgia, in 1986 have been reported (Lordo et al. 1996; Patterson et al. 1994). The sums of three coplanar PCB congeners (77, 126, and 169) were 0.24 ng/g in 14 males with a mean age of 43 and 0.18 ng/g in 14 females with a mean age of 54. Age was significantly correlated with the concentrations of the three congeners such that older people had higher concentrations of PCBs than did younger individuals. From 1990 to 1994, 105 autopsied bodies from Greenland were analyzed for PCBs (Dewailly et al. 1999). Researchers also found that the mean concentration of total PCBs in omental fat increased with age. The study found that in people ages 41–54, 55–69, and >70, mean PCB concentrations were 4,909, 5,337, and 7,357 ng/g lipid weight, respectively. The concentrations of congener-specific PCBs in intra-abdominal, subcutaneous, adrenal, liver, kidney, muscle, and spleen tissues obtained from five North American patients with no known occupational exposure are also available (Schechter et al. 1989, 1994). Differences were observed in the PCB congener pattern of distribution within a given tissue and between the various tissues of the donors. On a lipid weight basis, the highest concentrations of PCBs typically were detected in the adipose and liver tissue. Preferential accumulation in adipose tissue was also noticed in the Dewailly et al. (1999) study.

PCB serum levels measured from 1973 to 1996 in the general population are summarized in Table 6-21. Since the 1970s, researchers have noticed a decrease in PCB concentrations in human blood serum. In a study of 1,631 individuals from 1978 to 1979 living in the United States, the mean PCB concentration in human blood serum was 6.4 ng/g (Kreiss et al. 1982). Currently, mean serum PCB levels range from

**Table 6-21. Serum Polychlorinated Biphenyl (PCB) Levels in Non-occupationally Exposed U.S. Populations That Do Not Consume Fish from PCB-Contaminated Waters (1973–1996)**

Area and sampling method	Number of subjects	Year	PCB level ng/mL (ppb)				Range	Reference
			Arithmetic mean	Geometric mean	Arithmetic standard deviation	95% Confidence interval		
Nonconsumers of Great Lakes sport fish	41	1996		1.2			0.46–2.9	Anderson et al. 1998
Infrequent male consumers of Great Lakes sport caught fish	57	1994–1995		1.5			0.5–9.7	Hanrahan et al. 1999
Infrequent female consumers of Great Lakes sport caught fish	42	1994–1995		0.9			0.5–3.3	Hanrahan et al. 1999
Females from Cornwall and Mississauga Ontario, Canada	35	1992		3.2 <sup>b</sup>			1.3–12.0	Kearney et al. 1999
Males from Cornwall and Mississauga Ontario, Canada	45	1992		3.9 <sup>b</sup>			1.1–12.0	Kearney et al. 1999
Los Angeles–Long Beach, California work force <sup>a</sup>	738	1982–1984	5	4 <sup>b</sup>	4.37	–	<1–37	Sahl et al. 1985a, 1985b
Jefferson, Ohio, volunteers	59	1983	5.8	4.4	6.5	4–8	1–45	Welty 1983
Fairmont, West Virginia, volunteers	40	1983	6.7	5	5.3	5–8	1–23	Welty 1983
Norwood, Massachusetts, volunteers	990	1983	4.9	4.2	3.5	4–6	2–30	Condon 1983
Old Forge, Pennsylvania, volunteers	138	1981	3.6	–	–	–	<3–43	Reid and Fox 1982

**Table 6-21. Serum Polychlorinated Biphenyl (PCB) Levels in Non-occupationally Exposed U.S. Populations That Do Not Consume Fish from PCB-Contaminated Waters (1973–1996) (continued)**

Area and sampling method	Number of subjects	Year	PCB level ng/mL (ppb)				Reference	
			Arithmetic mean	Geometric mean	Arithmetic standard deviation	95% Confidence interval		Range
Maternity patients from western Michigan control group of nonfish eaters	71	1982	4	–	–	–	–	Schwartz et al. 1983
Lake Michigan random nonfish eaters	418	1980	–	6.6 <sup>b</sup>	–	–	<3–60	Humphrey 1983
Canton, Massachusetts, volunteers	10	1980	7.1	5.2	5.2	3–11	1–18	Condon 1983
Billings, Montana, random packinghouse workers	17	1979	7.5	5.8	6.8	4–11	2–30	Drotman 1981
Franklin, Idaho, volunteers	105	1979	–	–	–	–	<5	Drotman 1981
Random unexposed railroad workers at unspecified location	19	1979	12	–	–	–	10–27	Chase et al. 1982
Newton, Kansas, volunteers	7	1979	4.9	4.2	3.1	2–8	2–11	Vernon 1981
Michigan PBB cohort	1,631	1978–1979	7.7	6.4	–	–	<1–57	Kreiss et al. 1982
Bloomington, Indiana, volunteers and controls	110	1977	18.8	–	10.8	17–21	6–79	Baker et al. 1980
Lake Michigan random nonfish eaters	29	1973	17.3	15 <sup>b</sup>	–	–	<5–41	Humphrey 1983

Source: Adapted from Kreiss 1985; Massachusetts Department of Public Health 1987; Sahl et al. 1985a, 1985b

<sup>a</sup>Pre-employment survey of utility company workers

<sup>b</sup>Median

## 6. POTENTIAL FOR HUMAN EXPOSURE

0.9 to 1.5 ng/mL in individuals who do not have a diet high in fish, especially fish from the Great Lakes (Anderson et al. 1998; Hanrahan et al. 1999).

Congener-specific analysis of PCBs in serum show that the mean concentration of three coplanar congeners (77, 126, and 169) in the general population in the United States (sampled in 1988) is 176 pg/g (ppt) (lipid basis) (Patterson et al. 1994). Analysis of pooled serum samples show a decrease in the level of three coplanar (77, 126, and 169) PCBs from 1982 to 1989 (Patterson et al. 1994). The mean concentration of the total non-*ortho*-, mono-*ortho*-, and di-*ortho*-substituted PCBs in the whole blood of 50 Vietnam veterans in Michigan measured in 1991–1992 was 167 ng/g (ppb) with a concentration range of 50–628 ng/g (ppb) (Schechter et al. 1993). A breakdown of total PCBs revealed that 0.227 ng/g were contributed by the coplanar PCBs (77, 126, and 169), 50 ng/g by the mono-*ortho* PCBs (28, 74, 105, 118, and 156), and 117 ng/g by the di-*ortho* PCBs (99, 128, 138, 153, 170, 180, 183, 185, and 187) (Schechter et al. 1993). The three most predominant congeners in the whole blood samples were congener 153 (40 ppb), 138 (26 ppb), and 180 (19 ppb). The PCB levels in the veterans did not reflect exposure in Vietnam (Schechter et al. 1993).

PCB concentrations in human breast milk have also been closely monitored since the early 1970s (Mes and Davies 1979; Mes et al. 1986; Newsome and Ryan 1999). Temporal trend studies indicate that the PCB levels detected in human breast milk have decreased over time (Lunden and Noren 1998; Schade and Heinzon 1998). Recent studies indicate that the mean concentration of PCBs in human breast milk appears to range from 238 to 271 ng/g lipid weight (Kostyniak et al. 1999; Newsome et al. 1995). For more information concerning concentrations in human breast milk, please refer to Section 6.6 Exposures of Children.

Since the early 1960s, the FDA has conducted Total Diet Studies, also known as the Market Basket Surveys. These annual studies analyze ready-to-eat foods collected in markets from cities nationwide to determine the intake of selected contaminants in the American diet. Tables 6-22 through 6-25 present the results of the Total Diet Studies from 1976 to 1997 with respect to PCBs. Since the mid-1970s, individual diets for adult males, toddlers, and infants have been analyzed, and the total PCB levels have shown a downward trend in concentration from the mid-1970s to the mid-1980s. For example, the estimated daily dietary intake of PCBs in an adult diet in 1977 was 0.016 µg/kg/day while in the study from 1982 to 1984, the estimated daily intake was 0.0005 µg/kg/day (Gartrell et al. 1985a, 1985b, 1986a). Temporal monitoring studies of PCBs in Total Diet Studies from 1982 to 1997 have revealed that PCB intake has remained relatively steady (Bolger 1999; Gunderson 1995). For example, total diet studies

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-22. Estimated Daily Dietary Intake ( $\mu\text{g}/\text{kg}/\text{day}$ ) of Polychlorinated Biphenyls for Adults, Toddlers, and Infants<sup>a</sup>**

Fiscal year	Adult	Toddler	Infant
1982–1984	0.0005	0.0008	0.0012
1981–1982	0.003	Not detected	Not detected
1980	0.008	Not detected	Not detected
1979	0.014	Not detected	Not detected
1978	0.027	0.099	0.011
1977	0.016	0.030	0.025
1976	Trace	Not detected	Trace

Source: Derived from Gartrell et al. 1985a, 1985b, 1986a, 1986b; Gunderson 1988

<sup>a</sup>From food components (not individual food items) analysis

**Table 6-23. Mean Daily Intakes of PCBs Per Unit of Body Weight ( $\mu\text{g}/\text{kg}$  body weight/day)<sup>a,b</sup>**

Children						Adult males					
6 months			2 years			14–16 years			25–30 years		
82/84	84/86	86/91	82/84	84/86	86/91	82/84	84/86	86/91	82/84	84/86	86/91
0.001	0.001	<0.001	0.001	0.002	0.002	<0.001	0.002	<0.001	<0.001	0.001	<0.001

<sup>a</sup>Gunderson 1995<sup>b</sup>Study years: 1982–1984, 1984–1986, 1986–1991

**Table 6-24. Children Total Diet Studies — PCB Intakes from 265 Foods for the Years 1991–1997<sup>a</sup>**

	Dietary intake of PCBs µg/kg/day					
	6–11 months	2 years	6 years	10 year	14–16 years	14–16 years
Total Diet Study	Infant	Child	Child	Child	Female	Male
1991 (3 <sup>rd</sup> quarter)	0.002	0.015	0.023	0.021	0.013	0.013
1993 (1 <sup>st</sup> quarter)	<0.001	0.003	<0.001	<0.001	<0.001	0.002
1993 (2 <sup>nd</sup> quarter)	0.001	0.011	<0.001	<0.001	0.002	<0.001
1994 (1 <sup>st</sup> quarter)	0.001	0.034	0.018	0.018	0.010	0.011
1994 (2 <sup>nd</sup> quarter)	0.003	0.002	0.002	0.002	<0.001	<0.001
1995 (1 <sup>st</sup> quarter)	0.008	0.006	0.007	0.004	0.001	0.003
1997 (3 <sup>rd</sup> quarter)	<0.001	0.008	0.003	0.003	0.001	0.003
Average intake	0.002	0.012	0.008	0.007	0.004	0.005

<sup>a</sup> Bolger 1999

**Table 6-25. Adult Total Diet Studies — PCB Intakes from 265 Foods for the Years 1991–1997<sup>a</sup>**

Total Diet Study	Dietary intake of PCBs µg/kg/day							
	25–30 years		40–45 years		60–65 years		70 years	
	Female	Male	Female	Male	Female	Male	Female	Male
1991 (3 <sup>rd</sup> quarter)	0.007	0.008	0.006	0.009	0.008	0.012	0.008	0.012
1993 (1 <sup>st</sup> quarter)	<0.001	<0.001	<0.001	<0.001	0.001	0.001	0.001	0.001
1993 (2 <sup>nd</sup> quarter)	<0.001	0.001	0.001	0.001	<0.001	0.001	0.001	0.001
1994 (1 <sup>st</sup> quarter)	0.012	0.013	0.011	0.015	0.012	0.014	0.011	0.010
1994 (2 <sup>nd</sup> quarter)	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
1995 (1 <sup>st</sup> quarter)	0.003	0.002	0.004	0.003	0.004	0.005	0.002	0.004
1997 (3 <sup>rd</sup> quarter)	0.002	0.002	0.001	0.002	0.002	0.003	0.001	0.003
Average intake	0.004	0.004	0.003	0.004	0.004	0.005	0.003	0.004

<sup>a</sup> Bolger 1999c



## 6. POTENTIAL FOR HUMAN EXPOSURE

conducted from 1982 to 1984 for adults between the ages of 25 and 30 indicated that the mean daily intake of PCBs was  $<0.001 \mu\text{g}/\text{kg}$  body weight/day while in the 1997 study, the mean was  $0.002 \mu\text{g}/\text{kg}$  body weight/day. The FDA reported that the source of PCBs in the past was the meat-fish-poultry composite (63–100% of total dietary intake) with fish being the major contributing source (Jelinek and Corneliussen 1976). This observation appears to have continued in the recent Total Diet Studies conducted from 1991 to 1997 where meat, fish, and poultry remain the primary sources of PCBs in the human diet with fish being the major contributing factor (Bolger 1999). A recent market-basket study analyzed PCB congener levels in pooled food samples from supermarkets in five U.S. cities representing the northeast, mid-south, south, mid-west, and west (Schechter et al. 1997). Coplanar PCB concentrations ranged from  $0.2 \text{ pg}/\text{g}$  wet weight in a simulated vegetarian diet to  $531.4 \text{ pg}/\text{g}$  wet weight in fresh fish. Mono-*ortho* PCBs ranged from  $15 \text{ pg}/\text{g}$  wet weight in a vegetarian diet to  $2,350 \text{ pg}/\text{g}$  wet weight in fresh fish. Di-*ortho* PCBs ranged from  $144 \text{ pg}/\text{g}$  in a vegetarian diet to  $4,600 \text{ pg}/\text{g}$  in fresh fish. Schechter and Lingjun (1995) measured levels of mono-*ortho* and di-*ortho* PCBs in three types of fast foods sampled at the same five representative U.S. cities. Average total concentrations for mono-*ortho* PCBs were 380, 440, and  $500 \text{ pg}/\text{g}$  for hamburger, pizza, and chicken, respectively, and for di-*ortho* PCBs were 577, 740, and  $670 \text{ pg}/\text{g}$  for hamburger, pizza, and chicken, respectively.

As is the case with U.S. dietary exposure, PCB exposure via ingestion of drinking water and inhalation has also decreased over time. The average adult inhales  $.20 \text{ m}^3$  of air per day while the average numbers of hours a person spends outdoors, within vehicles, and indoors are approximately 1.77, 1.77, and 20.4 hours, respectively (EPA 1997d). Assuming that outdoor air at a typical urban location contains an average PCB concentration of  $5 \text{ ng}/\text{m}^3$  (range,  $1\text{--}10 \text{ ng}/\text{m}^3$ ) (Eisenreich et al. 1992), the average daily exposure via inhalation would be  $100 \text{ ng}$  (range,  $20\text{--}200 \text{ ng}$ ). However, the concentrations of PCBs in indoor air can be at least an order of magnitude higher than outdoor air concentrations (see Section 6.4.1) (Balfanz et al. 1993; MacLeod 1981; Wallace et al. 1996). It has been suggested that the emissions from certain appliances and devices (e.g., fluorescent lighting ballasts) that have PCB-containing components contribute to these higher indoor air concentrations. Individuals who spend more time indoors in these types of surroundings may be exposed to higher PCB concentrations than people who spend more time outdoors. The exact inhalation exposure for the general population depends on the amount of time an individual spends outdoors and indoors.

The general population is exposed to  $<200 \text{ ng}/\text{day}$  PCBs from drinking water (assuming drinking water concentrations of  $<0.1 \mu\text{g}/\text{L}$  [ppb] PCBs and a consumption rate of  $2 \text{ L}/\text{day}$ ). However, the daily exposure to PCBs via most drinking water in the United States is likely to be much lower than  $200 \text{ ng}$ ,

## 6. POTENTIAL FOR HUMAN EXPOSURE

since PCBs were detected in only one finished groundwater supply and two finished surface water supplies surveyed in NOMS I and II out of 113 cities surveyed nationwide. This value ( $<0.1 \mu\text{g/L}$ ) is based on the NOMS study conducted from 1975 to 1977. A study of municipal drinking water sources in Canada from 1985 to 1988 detected PCBs in only 1 out of 280 water samples at a concentration of  $0.006 \mu\text{g/L}$  (O'Neill et al. 1992). Consequently, for persons in the general population, food consumption and inhalation appear to represent greater sources of human exposure to PCBs than ingestion of drinking water.

From 1970 to 1976, occupational exposure to PCBs in the United States may have affected . 12,000 individuals per year (NIOSH 1977). PCB levels in blood, plasma, serum, and body tissues were 10–1,000 times higher in individuals exposed to PCBs in the workplace than in nonoccupationally exposed individuals (Wolff 1985; Yakushiji et al. 1978). Serum PCB levels in some occupationally exposed populations are reported in Table 6-26. Within 46 months following the cessation of PCB use, serum PCB levels of the lower chlorinated (mostly tri- and tetra-chlorinated) PCBs in capacitor manufacturing workers in the United States decreased by an average of 25–90%, although the higher chlorinated congeners did not decrease significantly (Wolff et al. 1992). A study conducted in Finland found that the median serum levels of three co-planar PCB congeners (77, 126, and 169) in capacitor manufacturing workers were 3–20 times higher than levels in the control population (Luotamo et al. 1993). The same congeners in the blood of exposed Finnish laboratory personnel, however, were not elevated above those of a control group (Hesso et al. 1992). PCB blood levels were compared for employees in the scrap metal industry where soils were contaminated with PCBs (Malkin 1995). Serum PCB levels of  $<1\text{--}65.3 \mu\text{g/L}$  were observed. No difference in PCB serum levels were found between outdoor or indoor workers. This lack of difference was associated with the workers' practice of eating lunch outdoors and consequent hand-to-mouth transmission.

Occupational exposure to PCBs via inhalation was estimated to be more than an order of magnitude higher than exposure via dermal contact in workers at a facility that recovers PCBs from transformers (Perkins and Knight 1989). Although occupational exposure to PCBs in the United States is no longer due to the production of PCBs or PCB-containing products (e.g., capacitors, transformers, and electrical equipment), it may still occur as a result of repairing electrical equipment that contains PCBs or accidents involving such equipment (Schechter and Charles 1991; Wolff 1985).

**Table 6-26. Serum Polychlorinated Biphenyl (PCB) Levels in Populations with Occupational Exposure**

Facility	Number of subjects	PCB levels ng/mL (ppb)				Reference
		Arithmetic mean	Geometric mean	95% Confidence interval	Range	
Railway car maintenance	86	33.4	–	–	10–312	Chase et al. 1982
Capacitor plant	34	394.0 <sup>a</sup>	–	234–554	trace–1,700	Ouw et al. 1976, 1979
Capacitor plant	290	48.0 <sup>a</sup>	21.0 <sup>b</sup>	38–546 <sup>b</sup>	1–546 <sup>c</sup>	Wolff et al. 1982a
Capacitor plant	80	342.0 <sup>a</sup>	–	–	41–1,319	Maroni et al. 1981a
Capacitor plant	221	–	119.0 <sup>c</sup> 25.3 <sup>b</sup>	–	1–2,220 <sup>c</sup> 1–250 <sup>b</sup>	Smith et al. 1982
Public utility	14	–	24.0 <sup>c</sup> 24.0 <sup>c</sup>	15–39 <sup>c</sup> 16–35 <sup>b</sup>	5–52 <sup>c</sup> 7–24 <sup>b</sup>	Smith et al. 1982
Transformer repair workers (recent exposure)	35	–	43.7 <sup>d</sup>	–	4.3–253	Fait et al. 1989
Transformer repair workers (past exposure)	17	–	30.0 <sup>c</sup>	–	1.5–143	Fait et al. 1989
Scavenging copper from PCB-contaminated capacitors at waste sites	11	–	12.0	–	–	Stehr-Green et al. 1986b
Private utility	25	–	22.0 <sup>c</sup> 29.0 <sup>b</sup>	17–25 <sup>c</sup> 20–43 <sup>b</sup>	9–48 <sup>c</sup> 7–250 <sup>b</sup>	Smith et al. 1982
Utility	1,058	4.0	3.0 <sup>d</sup>	3.65 <sup>f</sup>	<1–26	Sahl et al. 1985b

Source: Adapted from Kreiss 1985

<sup>a</sup>Blood level

<sup>b</sup>Higher-chlorinated PCBs

<sup>c</sup>Lower-chlorinated PCBs

<sup>d</sup>Median

<sup>e</sup>Standard deviation

## 6. POTENTIAL FOR HUMAN EXPOSURE

In addition, occupational exposure to PCBs may occur as a result of waste site cleanup or disposal activities. The median serum levels of two PCB congeners (77 and 81) in Finnish workers at a hazardous waste incinerator site where PCB-containing capacitors are destroyed were 3–4 times higher than control population levels (Luotamo et al. 1993). The serum levels of two other coplanar congeners (126 and 169) in incinerator workers were not significantly different from control population levels. Elevated PCB levels in blood (compared with background levels) were found in some exposed workers, including firefighters, electricians, and others who entered the building following the Binghamton State Office Building transformer fire in Binghamton, New York (Schechter 1987). In seven firefighters, the serum PCB levels measured approximately 10 months after the fire decreased by 20–95% from levels measured immediately after the exposure (Schechter et al. 1994). Compared to persons not occupationally exposed to PCBs, electrical workers appeared to retain more of PCB congener 126 after exposure than any other congener; however, the unusually high concentration of PCB 126 in serum of electrical workers was later attributed to an unidentified peak by the analyst (see Hansen 1999, Appendix Table 5) (Fait et al. 1989). Maintenance workers and welders who work with metals coated with PCB-containing paints may also be at higher risk of exposure because scraps from different railroad car paints were found to contain 4–625 mg/kg of Aroclor 1254 (Welsh 1995).

According to the National Occupational Exposure Study (NOES) conducted by NIOSH from 1981 to 1983, the following estimated number of workers were potentially exposed to Aroclors in the workplace: 2,214 to Aroclor 1242; 3,702 to Aroclor 1254; 991 to Aroclor 1260; and 1,558 to Aroclor 1016 (NIOSH 1989). Occupational exposure to Aroclors occurs in miscellaneous workers in the transformer industry, noncellulose fiber industry, semiconductor and related industries, and in sawmills and planing mills. It also occurs in clinical laboratory technicians and technologists of general medical and surgical hospitals. The NOES database does not contain information on the frequency, concentration, or duration of occupational exposure to any of the chemicals listed. The survey provides estimations of the numbers of workers for whom potential exposure in the workplace is an issue. Since this study was conducted from 1981 to 1983, it does not accurately represent current workplace exposure to PCBs.

### 6.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans. Differences from adults in susceptibility to hazardous substances are discussed in Section 3.7 Children's Susceptibility.

## 6. POTENTIAL FOR HUMAN EXPOSURE

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, put things in their mouths, sometimes eat inappropriate things (such as dirt or paint chips), and spend more time outdoors. Children also are closer to the ground, and they do not use the judgment of adults to avoid hazards (NRC 1993).

Children are exposed to PCBs in the same manner as the general population—primarily via consumption of contaminated foods, particularly meat, fish, and poultry (Bolger 1999; Gunderson 1985). Infants and young children consume a greater amount of food per kilogram of body weight and, therefore, may have a proportionately greater exposure to PCBs than adults (Cordle et al. 1982). Infants and children also have different diets than adults due to their age. This has been reflected in the FDA total diet studies (Gunderson et al. 1995). Breast-fed infants may be exposed to higher than average concentrations of PCBs because PCBs tend to accumulate in breast milk fat. Factors that can affect the levels of PCBs in human breast milk include mother's age, number of deliveries and lactations, place of residence, and changes in the mother's weight during lactation (Czaja et al. 1999a, 1999b). Women with the highest number of deliveries have higher levels of PCBs in their breast milk (Czaja et al. 1997a). However, while lactation may be one of the means of excreting PCBs from the body, it is age rather than the number of deliveries that seems to affect the concentration of PCBs with older women having higher concentrations. Also, women in industrial areas can have elevated levels of PCBs in their breast milk compared to women living in rural areas (Czaja et al. 1997b). It is estimated that an infant that is breast fed for 6 months will receive 6.8–12% of its lifetime PCB body burden (Kimbrough 1995; Patandin et al. 1999). Blood samples were taken from 80 full-term German neonates within the first 12 hours of life, before the first oral feeding (Lackmann et al. 1999). The median serum concentration of total PCBs was 0.96 µg/L (<0.30–3.14, range), with PCBs 138, 153, and 180 detected at median levels of 0.34 (<0.10–1.01), 0.42 (<0.10–1.42), and 0.17 (<0.10–0.78) µg/L, respectively. Lanting et al. (1998a) measured the levels of PCB congeners 118, 138, 153, and 180 in plasma from 42-month-old children (n=126) living in the Groningen area, The Netherlands. In 42-month-old children who were fully breast-fed for at least 6 weeks, the median total plasma PCB level 0.81 µg/L (range, 0.23–2.2), compared to the formula-fed children that had levels of 0.18 µg/L (range, 0.07–1.49) (see Section 3.7).

## 6. POTENTIAL FOR HUMAN EXPOSURE

A recent study conducted from 1991 to 1993 of human breast milk from 213 women living along Lake Ontario in New York State determined that the mean concentration of PCBs was  $271 \pm 116$  ng/g lipid weight (Kostyniak et al. 1999). In 1992, breast milk from 497 women living in Canada had a mean PCB concentration of 238 ng/g lipid weight (Newsome et al. 1995). Several studies indicate that PCB concentrations in human breast milk have decreased since the early 1970s (Hansen 1999; Lunden and Noren 1998). For example, in a study of human breast milk of Swedish women studied from 1972 to 1992, researchers determined that the concentration of PCBs decreased over time. PCB concentrations in 1972, 1980, 1984–1985, 1990, and 1992 were 1,090, 780, 600, 510, and 380 ng/g lipid weight, respectively (Lunden and Noren 1998). In Germany, researchers studied PCB levels from 1986 to 1996 in human breast milk of women between the ages of 27 and 31 who had only given birth once. They found that the concentrations of PCBs in 1986, 1988, 1990, 1992, 1994, and 1996 were 1,300, 1,050, 1,000, 750, 650, and 450 ng/g lipid weight, respectively (Schade and Heinzow 1998). Congener-specific analysis of human milk indicates that the congeners 138, 153, 118, 180, and 105 are most prevalent and that the three coplanar congeners (77, 126, and 169) were either not detected or detected at concentrations  $<1$  ng/g (ppb) on a milk fat basis (Bohm et al. 1993; Mes et al. 1993). For a representative trend analysis of PCB concentrations in human breast milk over time, please refer to Table 6-27. PCB exposure of infants whose mothers have a diet high in fish is discussed in Section 3.7 and 6.7. In general, mothers who consume fish contaminated with PCBs have higher levels in their breast milk compared to nonconsumers.

Unborn children may also be at risk of higher PCB exposure, especially in areas that have been heavily contaminated with PCBs. To illustrate this, researchers analyzed placental cord serum of 755 infants born to mothers residing in towns adjacent to a PCB-contaminated harbor in southeastern Massachusetts (Altshul et al. 1999). Infants whose cord serum was analyzed were born between 1993 and 1998 in the towns of New Bedford, Acushnet, Fairhaven, and Dartmouth, Massachusetts. Of the 51 PCB congeners analyzed, only 13 were above the detection limit (0.01 ng/g serum). The median concentration of PCBs was 0.56 ng/g serum (Altshul et al. 1999). Researchers found that the relative predominance of less chlorinated congeners in the cord blood was generally consistent with the characteristics of the contaminated site. PCB concentrations were also measured in cord blood from both 134 women who consumed Great Lakes fish and 145 women who had never consumed Great Lakes fish (Stewart et al. 1999). Although researchers did not find any difference between fish consumption levels and total PCBs in umbilical cord serum, it was established that fish eaters had marked elevations of the most heavily chlorinated PCB homologues. In particular, levels of hepta- to nonachlorobiphenyls were greater in fish eaters than non-fish eaters (Stewart et al. 1999). Another study examined PCB concentrations in nine

**Table 6-27. Mean Concentration of PCBs in Human Breast Milk**

Location	Sample Size	Year	PCB concentration (ng/g lipid) <sup>a</sup>	PCB concentration (ng/g milk)	Source
National Canadian Study	No data	1970		6	Mes and Davies 1979
	100	1975		12	Mes and Davies 1979
	210	1982		26	Mes et al. 1986
	412	1986		6.35	Mes et al. 1993
	497	1992	238	7.21	Newsome et al. 1995
National Sweden Study	135	1972	1,090		Lunden and Noren 1998
	153	1976	910		
	431	1980	780		
	102	1984–1985	600		
	120	1988–1989	650		
	60	1990	510		
	60	1991	410		
Akwesasne Indian Reservation	19	1986–1989	602		Fitzgerald et al. 1998
	38	1990	352		
	40	1991–1992	254		
Warren and Schoharie County, New York (rural)	52	1986–1989	375		Fitzgerald et al. 1998
	57	1990	404		
	45	1991–1992	318		
Northern Germany (age 27–31, primiparae)	15	1986	1,300		Schade and Heinzow 1998
	68	1988	1,050		
	84	1990	1,000		
	43	1992	750		
	29	1994	650		
	14	1996	450		

**Table 6-27. Mean concentration of PCBs in Human Breast Milk (continued)**

Location	Sample Size	Year	PCB concentration (ng/g lipid) <sup>a</sup>	PCB concentration (ng/g milk)	Source
Zagreb, Croatia	40	1987–1990	243 <sup>b</sup>		Krauthacker et al. 1998
	54	1991–1993	213 <sup>b</sup>		
	45	1994–1995	212 <sup>b</sup>		
16 Counties in New York state adjacent to Lake Ontario	213	1991–1993	271±116	8.28±4.66	Kostyniak et al. 1999
New York State	7	1991–1993	(239–428)	3.5–14.1	Greizerstein et al. 1999
Helsinki, Finland (urban)	20	1992–1994	296		Kiviranta et al. 1999
Kuopio, Finland (rural)	64	1992–1993	198		Kiviranta et al. 1999
New Bedford Harbor, Massachusetts (near superfund site)	4	1993	(1,107–2,379)		Korrick and Altshul 1998
Murmansk, Russia (industrialized area)	15	1993	429.4		Polder et al. 1998
Monchegorsk, Russia (industrialized area)	15	1993	490.5		Polder et al. 1998
Keewatin, Northern Canada	12	1996–1997	247		Newsome and Ryan 1999

<sup>a</sup>Ranges in parenthesis<sup>a</sup>Median



## 6. POTENTIAL FOR HUMAN EXPOSURE

stillborn fetuses from the Netherlands in 1993 and found that median (range) concentration of PCBs in adipose tissue was 235 (97–768) ng/g lipid weight (Lanting et al. 1998b).

As indicated above, PCB measurements of breast milk and placental cord blood have been used as surrogate measures of exposure in studies of children. Cord blood is the most direct marker of fetal exposure, but because of its relatively low fat content, it requires sensitive analytical methods for accurate PCB analysis; analysis of breast milk does not present this difficulty. Analytical techniques have improved enormously in recent years, such that cord blood analysis of PCBs is now more accurate and reliable, but still of concern due to the low concentration of fat in cord blood.

For most young children, it appears that the dietary intake of PCBs has reached a steady state in the United States. During the 1980s, dietary intake of PCBs for infants (6–11 months) declined from 0.011 to 0.0012  $\mu\text{g}/\text{kg}/\text{day}$ , and dietary intake of PCBs for toddlers (2 years) declined from 0.099 to 0.0008  $\mu\text{g}/\text{kg}/\text{day}$  (Gartrell et al. 1985a, 1985b, 1986a, 1986b; Gunderson 1988). In the most recent study conducted in 1997, the estimated dietary intakes for infants (6 months) and toddlers (2 years) are  $<0.001$  and 0.008  $\mu\text{g}/\text{kg}/\text{day}$ , respectively (Bolger 1999). Assuming that the average infant weighs 9 kg, the average daily dietary exposure would be  $<0.009$   $\mu\text{g}$ . Assuming that the average toddler weighs 13 kg, the average daily dietary exposure would be 0.104  $\mu\text{g}$ . See Tables 6-22 through 6-25 in Section 6.5 for more details. A potential source of dietary intake of PCBs for infants may also come from the consumption of contaminated baby formulas. In a study of eight soybean infant formulas obtained in Spain, researchers discovered detectable quantities of PCBs (Ramos et al. 1998). The mean total concentration of PCBs in soybean infant formula was 10.25 ng/g lipid weight. Of the 15 congeners analyzed for, PCB 101 contributed the most to the total amount of PCBs.

Additional exposure to PCBs could occur for children who live near hazardous waste sites. Since children spend a lot of time playing on the ground, both indoors and out, they come into more contact with contaminants found on dust and dirt particles. They may be exposed to PCBs by dermal contact with PCB-contaminated soil and by ingesting contaminated soil from their unwashed hands and other hand-to-mouth behavior. The determination of PCBs in dust and dirt can therefore be important for predicting children's exposure. However, quantitative information regarding the bioavailability and amount of PCBs that children are exposed to through contact with contaminated soils are unavailable.

Between 1994 and 1995, house dust and yard soil were analyzed for PCB concentrations from 34 homes surrounding New Bedford Harbor, Massachusetts during the dredging of PCB-contaminated sediments

## 6. POTENTIAL FOR HUMAN EXPOSURE

(Vorhees et al. 1999). House dust samples were collected from the carpet, while yard soil was collected from the main entryway. The results indicated that house dust samples were 10 times higher (260–23,000 ng/g) than yard soil concentrations (15–1,800 ng/g). Although yard soil concentrations from neighborhoods closest to the harbor were significantly higher than comparison neighborhoods distant from the harbor, house dust concentrations did not differ significantly between the two locales (Vorhees et al. 1999). In general, the house dust samples contained higher concentrations of the more volatile, less chlorinated PCBs than the soil samples. The results of the house dust data were compared to results obtained from homes that were not located near known PCB sources. PCBs measured in house dust in nine Seattle, Washington homes had concentrations ranging from 240 to 760 ng/g and eight Columbus, Ohio, homes had concentrations ranging from 210 to 1,900 ng/g (Vorhees et al. 1999). Clearly, PCB concentrations were generally lower in these locations compared to the New Bedford Harbor neighborhood homes. Street dust and dirt samples, analyzed in August 1993, from the streets of Buffalo, New York, also contained detectable amounts of PCBs (Irvine and Loganathan 1998). Total PCB concentrations for the dust and dirt samples ranged from 90 to 1,700 ng/g, dry weight. In every case, the higher-chlorinated congeners were detected more frequently and in greater concentrations. In particular, PCBs 153, 138, 101, 118, and 180 contributed >50% of the total concentration of PCBs in each sample (Irvine and Loganathan 1998). Residing in proximity to incinerators may also increase exposure levels for children. Blood samples from 298 children living near a toxic waste incinerator in Germany had a mean concentration of PCBs of 0.49 µg/L (Osius et al. 1999). Given that PCB congeners have  $\log K_{oc}$  values ranging from 3.27 to 6.87 (Horzempa and DiToro 1983; Meylan et al. 1992), they will generally adsorb strongly to soil and dust particles. This should decrease bioavailability of PCBs. More scientific data, however, are necessary to determine the degree of PCB exposure through hand-to-mouth activities. This is suggested as a data need for future study.

Consumption of contaminated groundwater may be an additional source of PCB exposure for children. PCBs have been detected in groundwater samples at 500 of the 1,598 NPL sites where they were detected in some environmental media (HazDat 2000).

Indoor air at schools could be a potential source of PCB exposure for children. Indoor air in seven public buildings (schools and offices) in Minnesota was monitored during 1984 for Aroclors 1242, 1254, and 1260 (Oatman and Roy 1986). The mean total Aroclor concentration ( $\pm 1$  standard deviation) in the indoor air of the three buildings using PCB transformers ( $457 \pm 223$  ng/m<sup>3</sup>) was found to be nearly twice as high as that in the air of the four buildings not using PCB transformers ( $229 \pm 106$  ng/m<sup>3</sup>). The Aroclor

## 6. POTENTIAL FOR HUMAN EXPOSURE

levels detected in the indoor air of all seven buildings were significantly higher than those detected in ambient outdoor air (Balfanz et al. 1993; Eisenreich et al. 1992; MacLeod 1981; Oatman and Roy 1986).

Children may also be exposed to PCBs through play activities involving PCB containing materials. Children who had played with parts of a capacitor that had once contained PCBs had elevated serum PCB (similar to Aroclor 1242) levels compared with a background population consisting of other household members and a reference group of persons in the same geographical area (Wolff and Schechter 1991). The serum PCB levels in the exposed children declined to about half of the initial values over a period of 11 months. Exposure could also occur if children were to play near an area where there was a transformer fire. Although there are no data on children, elevated PCB levels in blood were found in some workers who entered a building following a transformer fire (Schechter 1987).

PCBs have been detected in several consumer products manufactured outside the United States, and it has not been determined whether any of these products would be likely to be imported into the United States. Many of these products could be used by children. PCB concentrations detected in various products include 1.2 ppm in anhydrous lanolin, 4.8 ppm in lanocerin, 0.64 ppm in anhydrous cream for children, 0.52 ppm in oil/water emulsion-emollient cream, and 3.8 ppm in water/oil emulsion-emollient cream (Welling et al. 1992). Various fish oils used as dietary supplements were collected from around the world between 1994 and 1995 and analyzed for PCB levels (Jacobs et al. 1998). Researchers found that PCB congeners 138, 153, and especially 118 were detected most frequently and in the highest concentrations. None of the samples, however, exceeded the FDA regulatory limit of 2.0 ppm for total PCBs. In fact, total PCB concentrations ranged from <5 to 1,132 µg/L with a mean of 332.0 µg/L (Jacobs et al. 1998). Due to the lack of data concerning the amount of these products that are used by children, it is difficult to determine the degree of importance these items have on a child's exposure to PCBs.

Studies also have indicated that PCBs may be transported from the workplace to the home. Children living with parents who work with PCBs (i.e., occupations associated with hazardous waste) may have higher exposure levels. There have been several cases reported of the transport of PCBs from the workplace to the home and in some cases, the secondary exposure of family members. PCBs with a pattern resembling Aroclor 1254 were found in the blood of two railway maintenance workers who repaired transformers (77 and 101 ng/mL). The PCB levels for the wives who laundered their husbands' clothes were not elevated, but their PCB pattern resembled the Aroclor 1254 pattern of their husbands, suggesting that the PCBs found in the women's blood were derived from contact with their husbands (Fischbein and Wolff 1987). In Indiana, PCBs were released into the municipal sewage treatment plant

## 6. POTENTIAL FOR HUMAN EXPOSURE

by an electrical manufacturing firm. PCBs were found in the blood serum of sewage treatment workers (75.1 ppb), their family members (33.6 ppb), community residents (24.4 ppb), and people who applied sludge from the plant in their yards (17.4 ppb) (Baker et al. 1980). Thus, the worker's family members had higher concentrations of PCBs in their blood serum than the other nonoccupational groups. Based on these observations, children living in homes of parents who are exposed to PCBs may in turn be exposed through contaminated clothing and shoes.

### 6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to individuals who are occupationally exposed to PCBs (Section 6.5), there are several groups within the general population with potentially high exposures (higher than background levels) to PCBs. These groups include recreational and subsistence fishers who typically consume larger amounts of locally caught fish than the general population; Native American populations such as the North American Inuits or other subsistence hunters/fishers; breast-fed infants of mothers who consume large amounts of contaminated fish or wild game; farmers and their families who were exposed to PCB-contaminated foods via food stored in PCB contaminated silos; and individuals living in proximity to incinerators, other PCB-disposal facilities, or the 500 current or former NPL hazardous waste sites where PCBs have been detected (HazDat 2000).

Consumption of sportfish, particularly from waters contaminated with PCBs, will increase the level of human exposure to PCBs. PCB exposures for adults and children associated with the consumption of contaminated fish are available for residents of the Great Lakes region (Anderson et al. 1999; Hanrahan et al. 1999), Massachusetts (Massachusetts Department of Public Health 1987), Michigan (Anderson 1989; Courval et al. 1996; Hovinga et al. 1992; Humphrey 1983; Humphrey and Budd 1996; Schwartz et al. 1983), New York (Fitzgerald et al. 1996); Wisconsin (Anderson 1989; Fiore et al. 1989), northern Illinois (Pellettieri et al. 1996), and Alabama (Anderson 1989; Kreiss et al. 1981) as well as for populations in Canada (Ryan et al. 1997). Direct relationships are found between serum PCB levels and the quantities of fish consumed or numbers of fish meals consumed (Fiore et al. 1989; Hovinga et al. 1993; Humphrey and Budd 1996; Schwartz et al. 1983). One recent study monitored the PCB concentrations in blood serum from both frequent and infrequent consumers of Great Lakes sport fish (Hanrahan et al. 1999). It was found that the mean concentration of PCBs in blood of 252 males who were frequent consumers was 4.8 ng/mL while in 57 males who were infrequent consumers, the concentration was 1.5 ng/mL. A similar study conducted in 1992 compared PCB blood serum levels between males from Cornwall and Mississauga, Canada who consumed waterfowl and fish from Lake Ontario and non-consumers (Kearney

## 6. POTENTIAL FOR HUMAN EXPOSURE

et al. 1999). The PCB concentrations in blood serum of 101 male consumers and 45 non-consumers were 5.5 and 3.9 ng/mL wet weight, respectively. A multivariate regression analysis was used to show that the significantly elevated serum PCB levels observed in sportfish eaters in Michigan compared with controls was due to historic fish consumption rather than recent consumption (Hovinga et al. 1993). In a study of maternity patients from western Michigan, Schwartz et al. (1983) reported an increase in serum PCB levels in women who consumed larger numbers of fish meals per year than those who consumed no fish meals per year. For example, in the control group of non-fish eaters, the mean serum PCB level was 4 ppb. However, for women consuming 6–11, 12–23, 24–51, and 52–183 fish meals per year, the mean serum PCB levels were 5.5, 5.5, 5.9, and 9.0 ppb, respectively. A recent multimedia study characterized environmental exposures to PCBs among residents in nine homes in the Lower Rio Grand Valley of Texas (Berry et al. 1997; Butler et al. 1997). As part of this study, PCB blood serum concentrations were obtained and compared to the National Health and Nutrition Examination Survey II (NHANES II) 95th percentile. Blood plasma PCB concentrations for two individuals with maximum values, as Aroclor 1260, exceeded the NHANES II 95th percentile for PCBs. Upon further investigation, it was found that the residents caught and ate carp from a nearby irrigation ditch. Analysis of the fish showed high PCB concentrations of 399 mg/kg (ppm), indicating a likely source for the high blood serum concentration in these individuals. For all of the other participants in the study, blood serum PCB concentrations were less than 4.2 µg/L, the NHANES II median value. PCBs were not detected in drinking water or dietary samples other than the fish samples. Serum PCB levels were 2.5 times higher in people who regularly eat fish (consumption rate of >24 pounds/year [ $>11$  kg/year]) compared to those who occasionally or never eat fish (consumption rate of 0–6 pounds/year [ $0$ – $2.7$  kg/year]) (Humphrey 1988). For more information concerning PCB serum levels in people who consume fish, please refer to Table 6-28.

Recreational and subsistence fishers within the general population consume larger quantities of fish and shellfish than the general population. Because of this, these populations are at greater risk of exposure to PCBs and other chemical contaminants if the waters they fish frequently are contaminated. The EPA advises states to use a screening value of 0.01 ppm of total PCBs (sum of Aroclors) as a criteria to evaluate their fishable waterbodies (EPA 1993h). Currently, 678 advisories have been issued by 35 states, the District of Columbia, and American Samoa restricting the consumption of PCB-contaminated fish and shellfish (EPA 1999i). In one study, however, of 8,306 Great Lakes sportfishers surveyed, only about 8.4% of them consumed fish from the Great Lakes (Tilden et al. 1997). Of those,

**Table 6-28. Serum Polychlorinated Biphenyl (PCB) Levels in Non-occupationally Exposed U.S. Populations that Consume Fish from PCB-contaminated Waters (1973–1995)**

Population	Number of subjects	Year	PCB level ng/mL (ppb)				Reference
			Arithmetic mean	Geometric mean	Arithmetic standard deviation	Range	
Frequent male consumers of Great Lakes sport caught fish	252	1994–1995		4.8		0.7–58.2	Hanrahan et al. 1999
Frequent female consumers of Great Lakes sport caught fish	187	1994–1995		2.1		0.5–12.1	Hanrahan et al. 1999
Native American Indian males from Akwesasne near the St. Lawrence River in New York, Ontario, and Quebec	139	1992–1995	4.9	2.8	5.6	<0.10–31.7	Fitzgerald et al. 1999
Sport fisherman who ate fish from Lake Michigan	10	1993		8.6		3.6–15.2	Anderson et al. 1998
Sport fisherman who ate fish from Lake Huron	11	1993		5.7		1.3–12.9	Anderson et al. 1998
Sport fishermen who ate fish from Lake Erie	11	1993		2.2		1.2–3.2	Anderson et al. 1998
Females from Cornwall and Mississauga Ontario, Canada	51	1992		3.4 <sup>a</sup>		0.7–23.0	Kearney et al. 1999
Males from Cornwall and Mississauga Ontario, Canada	101	1992		5.5 <sup>a</sup>		0.9–21.0	Kearney et al. 1999
Lake Michigan volunteers eating <6 lbs sportfish annually	95	1989	–	6.8	–	2–42.1	Hovinga et al. 1993

**Table 6-28. Serum Polychlorinated Biphenyl (PCB) Levels in Non-occupationally Exposed U.S. Populations that Consume Fish from PCB-contaminated Waters (1973–1995) (continued)**

Population	Number of subjects	Year	PCB level ng/mL (ppb)				Reference
			Arithmetic mean	Geometric mean	Arithmetic standard deviation	Range	
Lake Michigan volunteer sportfishers eating >24 lbs sportfish annually	112	1989	–	19.0	–	4.9–173.8	Hovinga et al. 1993
Wisconsin anglers that consumed both sport-caught fish meals and meals of species listed on PCB consumption advisory	191	1985–1988	–	–	–	–	Fiore et al. 1989
19.6 fish meals;	6.x10 <sup>7</sup>					<0.6	
7.1 advisory fish meals						0.6–2.0	
25.3 fish meals,							
10.8 advisory fish meals						2–5.0	
32.0 fish meals,							
13.8 advisory fish meals						>5.0	
33.3 fish meals,							
16.9 advisory fish meals							
Maternity patients from western Michigan	193	1982	5.5	4.6 <sup>a</sup>	3.7	–	Schwartz et al. 1983
6–11 fish meals/year			5.5	–	–	–	
12–23 fish meals/year			5.5	–	–	–	
24–51 fish meals/year			5.9	–	–	–	
52–183 fish meals/year			9.0	–	–	–	
New Bedford, Massachusetts, known exposure to contaminated seafood	110	1981–1982	13.34	9.48 <sup>a</sup>	14.02	1.40–87.97	Massachusetts Department of Public Health 1987

**Table 6-28. Serum Polychlorinated Biphenyl (PCB) Levels in Non-occupationally Exposed U.S. Populations that Consume Fish from PCB-contaminated Waters (1973–1995) (continued)**

Population	Number of subjects	Year	PCB level ng/mL (ppb)				Reference
			Arithmetic mean	Geometric mean	Arithmetic standard deviation	Range	
New Bedford, Massachusetts, random sample	840	1981–1982	5.84	3.88 <sup>a</sup>	7.78	0.38–154.2	Massachusetts Department of Public Health 1987
Lake Michigan volunteer sportfishers	572	1980	–	21.0 <sup>a</sup>	–	<3–203	Humphrey 1983
Lake Michigan volunteer sportfishers	90	1973	72.7	56.0 <sup>a</sup>	–	25–366	Humphrey 1983
Triana, Alabama, volunteer sportfishers	458	1973	22.2	17.2	22.3	3–158	Kreiss et al. 1981

Source: Adapted from Kreiss 1985; Massachusetts Department of Public Health 1987; Sahl et al. 1985a, 1985b

<sup>a</sup>Median



## 6. POTENTIAL FOR HUMAN EXPOSURE

58% of males were aware of a health advisory, while only 39% of women were aware of a health advisory. It appears that although health advisories do exist, there is a need to better communicate these warnings to the public. The number of waterbodies under advisory for PCBs in each state is shown in Figure 6-5.

Elevated PCB concentrations have also been detected in human adipose tissue from populations with diets high in fish and seafood. Levels of PCBs were determined in liver, brain, omental fat, and subcutaneous abdominal fat samples collected from 105 deceased Inuit Greenlanders between 1992 and 1994 (Dewailly et al. 1999). The population studied represented individuals who had a history of a diet high in sea mammal fat and fish. Of the 14 congeners analyzed for, PCBs 138, 153, and 180 were found in the highest concentrations in all tissue samples and represented 63–68% of total PCB concentration. The mean concentration of total PCBs in omental fat samples from 41 Greenlanders was 5,719  $\mu\text{g}/\text{kg}$  lipid basis (range=1,019–12,716  $\mu\text{g}/\text{kg}$  lipid basis). Using the same analytical methods, the total concentrations of PCBs 138, 153, and 180 were compared to adipose tissue from 17 women living in Quebec City, Canada from 1991 to 1992. Researchers found that PCB concentrations in the Greenlanders were 18 times higher than those from Canada. The study also found that older individuals had higher levels of PCBs in their adipose tissue. For example, the mean PCB concentrations for people ages 41–54, 55–69, and  $\geq 70$  years were 4,909, 5,337, and 7,357  $\mu\text{g}/\text{kg}$  lipid basis, respectively (Dewailly et al. 1999). In general, PCBs accumulated preferentially in omental/subcutaneous fat followed by liver, and accumulated the least in brain tissue.

Fat extracted from the Ooligan fish is a widely consumed traditional condiment and medicine among the indigenous people of coastal British Columbia. The average total PCB concentrations of 24–57 ng/g (ppb) lipid were reported in Ooligan fish fat and in fish from various coastal locations in the region (Chan et al. 1996). High consumption patterns of these products could increase PCB exposure of the native peoples of this region. In the summer of 1996, research was conducted to estimate the daily average intake of PCBs from consumption of local fish by the Mohawk community at the Kahnawake reservation, located south of Montreal, Canada (Chan et al. 1999). A total of 131 fish, representing 6 species, were caught and analyzed for total PCB concentrations. The mean concentration of PCBs in the fish ranged from 29.23 to 153.89 ng/g wet weight. Based on an average diet of 23 g of fish/day (compared to 1.2 g/day in the average Canadian diet), the estimated daily intake of PCBs were 0.026  $\mu\text{g}/\text{kg}$  body weight/day for men and 0.033  $\mu\text{g}/\text{kg}$  body weight/day for women. Based on an average body weight of 81 kg for men and 65 kg for women, the daily intake of PCBs for the Mohawk Indians would be 2.106 and 2.145  $\mu\text{g}$ , respectively (Chan et al. 1999). Similar results were found in a study conducted in

**Figure 6-5. 1998 Fish Advisories for Polychlorinated Biphenyls**



## 6. POTENTIAL FOR HUMAN EXPOSURE

1994 of the Dene and Metis Inuits of Western Northwest Territories, Canada whose diet consists mainly of herbivorous animals and fish (Berti et al. 1998). Researchers analyzed their dietary intake and found that the mean dietary intake of PCBs was 0.023  $\mu\text{g}/\text{kg}/\text{day}$ . Researchers noted that individuals with diets high in whale blubber were at increased risk of high PCB exposure. Some examples of other populations that have diets high in fish and sea mammals include Alaskan Inuits who consume 304 g/day (Wolfe and Walker 1987), recreational anglers from Michigan who consume 27 g/day (Hovinga et al. 1992), and Indian tribes from Oregon and Washington who consume 58.7 g/day (CRITFC 1994). Since the average daily intake of fish in the U.S. diet is 6.2 g/day (EPA 1993h), these high fish consuming populations represent communities at higher risk for PCB exposure.

Infants that are breast fed by women living in these communities may also be exposed to higher PCB concentrations. It is estimated that an infant that is breast fed for 6 months will receive 6.8–12% of its lifetime PCB body burden in that period (Kimbrough 1995; Patandin et al. 1999). For example, the diet of the Mohawk Indians from the Akwesasne reservation in New York has been impacted by the General Motors Foundry Mill, which released PCBs into the St. Lawrence River (Fitzgerald et al. 1998). Fish consumed by the Mohawk Indians come from the St. Lawrence River and have consequently been contaminated. Comparison studies were conducted to determine whether PCB levels in breast milk differed from that of the general population that was not affected by the mill. The study found that in milk samples collected in 1986–1989, the mean concentration of PCBs in American Indian milk was 602 ng/g lipid weight, while in the control group, the mean concentration was 375 ng/g lipid weight (Fitzgerald et al. 1998). From 1991 to 1992, however, PCB levels decreased to a mean of 254 ng/g lipid in Mohawk Indian breast milk, while in the control group, it only decreased to 318 ng/g lipid weight. The reduction in breast milk PCB concentrations paralleled a corresponding decrease in local fish consumption (Fitzgerald et al. 1998). The authors found that from 1986 to 1992, the number of fish meals consumed by pregnant Mohawk women decreased from 10.7 meals per year in 1986–1989, to 3.6 meals per year in 1990, and to 0.9 meals per year in 1991–1992. No such decreasing trend in consumption was noted among the Caucasian control group whose PCB levels did not change significantly over the course of the study. The mean concentration of total PCBs in human milk from native Inuit women who consumed large quantities of marine mammal tissue was 1,052 ng/g lipid weight in a 1989–1990 study (Dewailly et al. 1993). This was 7 times greater than levels measured in Caucasian women from southern Quebec (157 ng/g, lipid basis). The concentrations of PCB congeners in breast milk from a remote maritime population from Arctic Quebec (Inuit women) were compared with those of control Caucasian women in Quebec, Canada (Dewailly et al. 1994). Di-*ortho* congeners (138, 153, 170, and 180), mono-*ortho* congener (118), and non-*ortho* coplanar congeners (126 and 169) were detected at

## 6. POTENTIAL FOR HUMAN EXPOSURE

concentrations of 862.5, 58.7, and 0.455 ng/g lipid weight in Inuit women compared to 106, 17.4, and 0.121 ng/g lipid weight in Caucasian women. Because of their high consumption of marine mammal tissues and seafood, the Inuit women have high concentrations of PCBs in their breast milk, in spite of the remoteness of Arctic Quebec (Dewailly et al. 1993, 1994). Daily PCB intake for native northern Quebec women was calculated to be 0.3 µg/kg (ppb) body weight while daily intake among infants was calculated to be 10 µg/kg due to breast feeding. This level is 143 times greater than the reference dose (RfD) of 0.07 µg/kg body weight established by the EPA (Ayotte et al. 1995). In general, these studies indicate that lactating women whose diets are high in contaminated fish can potentially increase the PCB exposure for their breast-fed infants. For more information concerning PCB concentrations in human breast milk, please refer to Table 6-27 in Section 6.6.

Unborn children may also be at risk of higher PCB exposure, especially in areas that have been heavily contaminated with PCBs. To illustrate this, researchers analyzed placental cord serum of 755 infants born to mothers residing in towns adjacent to a PCB-contaminated harbor in southeastern Massachusetts (Altshul et al. 1999). Infants whose cord serum was analyzed were born between 1993 and 1998 in the towns of New Bedford, Acushnet, Fairhaven, and Dartmouth, Massachusetts. Of the 51 PCB congeners analyzed, only 13 were above the detection limit (0.01 ng/g serum). The median concentration of PCBs was 0.56 ng/g serum (Altshul et al. 1999). Researchers found that the relative predominance of less chlorinated congeners in the cord blood was generally consistent with the characteristics of the contaminated site. PCB concentrations were also measured in cord blood from both 134 women who consumed Great Lakes fish and 145 women who had never consumed Great Lakes fish (Stewart et al. 1999). Although researchers did not find any difference between fish consumption levels and total PCBs in umbilical cord serum, it was established that fish eaters had marked elevations of the most heavily chlorinated PCB homologues. In particular, levels of hepta- to nonachlorobiphenyls were greater in fish eaters than non-fish eaters (Stewart et al. 1999). Another study examined PCB concentrations in nine stillborn fetuses from the Netherlands in 1993 and found that median (range) concentration of PCBs in adipose tissue was 235 (97–768) ng/g lipid weight (Lanting et al. 1998b).

Similarly, Native American populations such as the Inuit of Alaska or other subsistence hunters (particularly those living in high-latitude areas of the United States) may be exposed to higher levels of PCBs in wild game (e.g., beluga whales, seals, polar bears, and other game species) (Dewailly et al. 1993; Kuhnlein et al. 1995; Schantz et al. 1993c). Because these populations typically are hunters of the highest trophic levels, they are particularly exposed to PCBs (Ayotte et al. 1995). Kuhnlein et al. (1995) compared PCB concentrations in the diet of Arctic indigenous women from both eastern and western

## 6. POTENTIAL FOR HUMAN EXPOSURE

Canada. These authors reported that PCB intakes in the western Arctic population were lower than the eastern Arctic population because of the food preferences for caribou, moose, and fish as compared to ringed seals, caribou, narwhal, and walrus. Mean total blood plasma PCB concentrations among 499 Inuit adults from Nunavik in the Quebec Arctic was 4.1 mg/kg (ppm) lipid weight, compared to 0.13 mg/kg lipid weight for control samples from individuals in southern Quebec (Ayotte et al. 1997). Hing (1998) summarized data from northern and Arctic Canadian studies associated with exposure to PCBs from consumption of native foods. Mean total PCB concentrations in traditional foods, expressed as  $\mu\text{g/g}$  (ppm) wet weight, were 0.080, 1.90, 0.006, 0.010, 0.052, 0.290, and 0.006, respectively, for marine mammal meat, marine mammal blubber, terrestrial mammal meat, terrestrial mammal organs, fish, birds, and plants. Based on dietary habits among two individual communities, total dietary PCB intakes were estimated to be 16  $\mu\text{g/day}$  from marine mammal meat, 57  $\mu\text{g/day}$  from marine mammal blubber, 1.2  $\mu\text{g/day}$  from terrestrial mammal meat, 0.3  $\mu\text{g/day}$  from terrestrial mammal organs, 0.4  $\mu\text{g/day}$  for fish, 0.4  $\mu\text{g/day}$  for birds, and 0  $\mu\text{g/day}$  for plants. Total estimated PCB intakes for the two communities were 6.0 and 7  $\mu\text{g/day}$ , respectively. Samples from herds of Yukon Territory and Northwest Territory caribou in Canada showed relatively low non-*ortho* PCB concentrations, considered equivalent to background levels, although PCB congener 126 and 169 concentrations were higher among caribou sampled from eastern herds (Bathurst, Northwest Territory), probably due to differences in atmospheric transport patterns (Hebert et al. 1996). Wilson et al. (1995) also reported high concentrations of PCBs in two fish species from remote Arctic lakes in Alaska. The most abundant group of organochlorine compounds detected in the fish were PCBs. Concentrations of 6.6 and 1.3 ng/g (ppb) wet weight were detected in lake trout and grayling muscle tissue, respectively. While the problem of PCB contamination in the Arctic is clearly greater in the eastern Arctic, it is increasingly being detected in Alaska as well. Clearly, increased exposure of native hunting and fishing peoples to PCBs can occur, and their infants are also at risk of greater exposure via consumption of PCB-contaminated breast milk.

During the 1940s and 1950s, concrete silos on many Midwest farms were coated on the inside with sealants containing the PCB mixture Aroclor 1254. Over time, the sealant peeled off and became mixed with silage used to feed beef and dairy cattle. Farmers and their families who lived on farms where PCB-containing sealants were used in silo construction, and who regularly ate beef and dairy products produced on their own farms, were exposed to PCB-contaminated foods (Hansen 1987a; Humphrey 1983). Most of these silos, however, have been dismantled and removed. The high serum PCB levels (100–200  $\mu\text{g/L}$  [ppb]) detected in the most exposed individuals, however, suggests that monitoring should be continued (Humphrey 1983). Schantz et al. (1994) monitored serum PCB levels in Michigan mothers and children from farms with PCB-contaminated silos. These authors reported blood serum PCB levels

## 6. POTENTIAL FOR HUMAN EXPOSURE

of 9.6 ng/mL (ppb) for mothers and 6.8 ng/mL (ppb) for children. Maternal serum PCB levels and the number of weeks of breast-feeding accounted for 47% of the variance in the children's serum PCB levels, confirming that breast milk was a primary source of the PCB exposure for the children.

Another population that may receive higher PCB exposures than the general population includes people who live in the vicinity of incinerators, PCB disposal facilities, or hazardous waste sites. PCB concentrations measured downwind from a landfill were at least an order of magnitude higher than those measured upwind from the landfill, although effective dilution reduced concentrations of PCBs in air samples monitored 15 km from the landfill to a level comparable or even slightly lower than urban air levels (Hermanson and Hites 1989). This dilution effect was also evident from the observed decrease in the atmospheric concentrations of PCBs within a short distance from another landfill during remediation (Hermanson and Hites 1989). This study suggests that people who reside in the immediate vicinity of PCB-containing landfills may be exposed to PCBs in the air at levels higher than the general population. Despite the potential for individuals living near processing facilities or hazardous waste sites to be exposed to higher levels of PCBs in air, water, and soil, higher PCB levels in human tissues have not been conclusively demonstrated (i.e., elevated levels of PCBs in the serum/blood of the susceptible population). One possible explanation for the low prevalence of elevated PCB levels in the serum/blood of people who live near these contaminated sites is that most of these people are not exposed to PCBs at elevated levels or that the PCB concentrations to which they are exposed are not completely bioavailable. Another explanation is that, unlike occupational exposure scenarios where concentrations can be orders of magnitude higher than background levels, slightly elevated environmental exposure may not result in appreciable elevation of PCB levels in serum/blood. Beginning in 1982, pilot studies involving a total of 766 subjects were conducted at 12 hazardous waste sites in the United States to determine human exposure to PCBs (Stehr-Green et al. 1988). Although environmental PCB contamination levels as high as 2.5 ppb in well water and 330,000 ppb in soil samples were measured, serum PCB levels in people from 10 of the 12 sites were not any higher than serum PCB levels in unexposed individuals in the general population. The higher serum PCB levels found in people at two sites may be attributable to the historic prevalence of occupationally related exposures. Another study that evaluated the PCB exposure of 89 individuals living near a toxic waste site in Paoli, Pennsylvania, also reported similar serum PCB levels in exposed individuals and unexposed populations (ATSDR 1987).

Individuals living near processing sites or NPL sites where PCBs have been detected may also be exposed to higher levels of PCBs in their drinking water if they obtain tap water from wells located near these sites. PCBs have been detected in groundwater samples at 192 of the 500 NPL sites where they were

## 6. POTENTIAL FOR HUMAN EXPOSURE

detected in some environmental media (HazDat 2000). Consumption of contaminated groundwater, therefore, may be an additional source of PCB exposure for both adults and children. Children and adults may also receive higher PCB exposures from dermal contact if they play or work with PCB-contaminated soils. In an *in vivo* study with Rhesus monkeys, percutaneous absorption of Aroclor 1242 and 1254 from a clay loam soil containing 0.9% organic matter was determined to be in the range of 13.8–14.1% (Wester et al. 1993). In addition, children and adults may receive potentially higher oral exposures from ingestion of PCB-contaminated soils from their unwashed hands, while playing or working in PCB-contaminated areas.

### 6.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of PCBs is available. Where adequate information is not available, ATSDR, in conjunction with the National Toxicology Program (NTP), is required to assure the initiation of a program of research designed to determine the health effects (and techniques for developing methods to determine such health effects) of PCBs.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

#### 6.8.1 Identification of Data Needs

**Physical and Chemical Properties.** Some of the physical and chemical properties (i.e., octanol/water partition coefficient [ $K_{ow}$ ], Henry's law constant, reaction rate constants) often useful in estimating environmental fate and transport processes for PCBs are available primarily for the Aroclors as mixtures and not for the individual congeners (see Table 4-2) (Burkhard et al. 1985; EPA 1979h, 1985b; Hollifield 1979; Paris et al. 1978). The experimental determination of the physical and chemical properties of many more of the individual congeners is needed for accurately predicting the environmental fate of the individual congeners.

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Production, Import/Export, Use, and Release and Disposal.** PCBs are no longer produced, imported/exported, or used on an industrial scale in the United States. The available literature contains adequate documentation of their past production (Durfee 1976; EPA 1976a; IARC 1978; Kimbrough 1987), import/export volume (Durfee 1976; EPA 1988c; IARC 1978; USITC 1978, 1979, 1980, 1982), use (EPA 1976a; IARC 1978; Safe 1984; Welsh 1995), release (Durfee 1976; EPA 1979e; Larsson 1985; Liberti et al. 1992; Mackay 1989; Morselli et al. 1985; Murphy et al. 1985; Swackhamer and Armstrong 1986; Tiernan et al. 1983; TRI93 1995), and disposal (Arbon et al. 1994; Baukal et al. 1994; Chuang et al. 1995; EPA 1979e, 1995a; IRPTC 1985; Timberlake and Garbaciak 1995; TRI93 1995; Zhang and Rusling 1995; Zhang et al. 1993). There are current EPA regulations regarding the disposal of PCBs; however, information on current disposal practices in the case of accidental transformer leakage would be useful.

**Environmental Fate.** A number of studies indicate that PCBs are very persistent in the environment (Brown et al. 1988; EPA 1979h, 1983c; Gan and Berthouex 1994; Portier and Fujisaki 1988). The volatilization of PCBs from soil and water, followed by dry/wet deposition of airborne PCBs into soil and water, results in the continuous recycling of undegraded PCBs in the environment (Macdonald et al. 2000; Wania and Mackay 1993, 1996). Sediment is a repository for PCBs that can later be released to air and water. However, some critical data regarding the potential for long-term release of PCBs from sediments and the role of deep ocean sediments as an ultimate sink for PCBs are lacking. There is a lack of quantitative data on the photodegradation potential of PCBs in air, water, and soil in the presence of natural sunlight. Information on the concentrations of chlorinated benzoic acids in the vicinity of PCB sources is needed to assess the degree of importance of OH-radical reactions in the atmosphere for degrading PCBs (Brubaker and Hites 1998). No extensive and systematic studies have been done on the influence of different types of soil/sediment (including with different PCB adsorption properties) on the reductive dehalogenation of PCBs (Wiegel and Wu 2000). Since the toxicity and the environmental fate of PCBs depend on specific PCB congeners, development of more data regarding congener-specific fate and transport of PCBs in the environment are needed.

**Bioavailability from Environmental Media.** The absorption and distribution of PCBs as a result of inhalation, ingestion, and dermal exposure are discussed in Sections 3.3.1, 3.3.2, and 3.3.3. Few studies that describe the bioavailability of PCBs from ambient air, surface water and groundwater, or soil exist. Additional studies determining the effect of particle size and organic matter content on the bioavailability of PCBs from soil and the role of microparticle-sorbed PCBs on the bioavailability of PCBs from



## 6. POTENTIAL FOR HUMAN EXPOSURE

drinking water are needed. Such studies would be useful in assessing the health effects of PCBs on people living near hazardous waste sites.

**Food Chain Bioaccumulation.** Based on available information, estimates can be made about the bioaccumulation of PCBs in fish, shellfish, and marine mammals (Andersson et al. 1988; ASTER 1996; EPA 1983c; Kuehl and Haebler 1995; Kuehl et al. 1994; Lake et al. 1995a, 1995b; Porte and Albaiges 1993; Salata et al. 1995; Schantz et al. 1993a; Zhang et al. 1983), the bioconcentration potential from soil to plants (Bohm et al. 1999; Lober et al. 1994; O'Connor et al. 1990; Schönherr and Riederer 1989) and from atmospheric vapors and particulates to plants (Jones and Duarte-Davidson 1997; O'Connor et al. 1990; Thomas et al. 1998; Ye et al. 1992a). The existing data indicate that PCBs bioaccumulate significantly in aquatic and terrestrial food chains and biomagnify in predators, due to consumption of contaminated prey. More information on bioaccumulation and biomagnification of PCB congeners in edible fish and shellfish species is needed in assessing human health risks.

**Exposure Levels in Environmental Media.** The relative importance of different routes of exposure to PCBs in the past (late 1970s through early 1980s) is detailed in the current literature. FDA studies indicate that the dietary intake of PCBs has steadily decreased since 1978 (Gartrell et al. 1985a, 1985b, 1986a). According to these studies, the major contributing factor in dietary PCB intake has changed from fish to meat in recent years (Gunderson 1988). However, the FDA dietary intake values are estimated for marketed foods and do not provide an indication of PCB intake from consumption of fish obtained by sport and subsistence fishing. Despite recent studies by Berry et al. (1997) and Buckley et al. (1997), more recent data on the concentrations of PCBs in foods, collected using a market-basket approach, are needed to determine whether concentrations of PCBs in foods consumed by the general population have declined further since the mid-1980s. Data on the PCB concentrations in foods grown in contaminated areas, particularly in the vicinity of hazardous waste sites, are also needed. Also, more data on congener-specific PCB analysis of food, especially plant products, would be useful. Recent investigations also show that the concentration of PCBs in indoor air can be at least an order of magnitude higher than outdoor air (Balfanz et al. 1993; Vorhees et al. 1997; Wallace et al. 1996). Therefore, due to the decreased intake of PCBs from food in recent years, it is possible that the intake from inhalation exposure may currently exceed PCB intake from food. However, a direct comparison of the importance of exposure from inhalation and diet is difficult because the reported data do not always include the same PCBs (e.g., Aroclor 1016, 1254, etc.) for the purpose of quantifying the total PCB concentrations and in evaluating the subsequent intake. It would be useful to conduct further research to resolve this important issue. More recent monitoring data on the concentrations of total PCBs as well as congeners in air in

## 6. POTENTIAL FOR HUMAN EXPOSURE

urban areas near hazardous waste sites and incinerators are needed. More recent survey data on PCB concentrations in finished drinking water nationwide would be helpful in assessing the importance of this exposure route. Congener-specific analysis of drinking water would also be useful in determining exposure risks, particularly for the dioxin-like congeners.

**Exposure Levels in Humans.** PCB levels are reported in the current literature for blood (Schechter et al. 1993, 1994), serum (Anderson et al. 1998; Fitzgerald et al. 1999; Hanrahan et al. 1999; Hovinga et al. 1992, 1993; Kearney et al. 1999; Massachusetts Department of Public Health 1987; Patterson et al. 1994), breast milk (Dekoning and Karmaus 2000; Fitzgerald et al. 1998; Kostyniak et al. 1999; Newsome and Ryan 1999; Newsome et al. 1995; Schade and Heinzow 1998), and adipose tissue of the general population (Dewailly et al. 1999; EPA 1986b; Fensterheim 1993; Jensen 1989; Kutz et al. 1991; Ouw et al. 1976; Patterson et al. 1994; Schechter et al. 1989, 1991, 1994) and occupationally exposed individuals (Fait et al. 1989; Perkins and Knight 1989; Schechter and Charles 1991; Schechter et al. 1994; Welsh 1995; Wolff 1985; Yakushiji et al. 1978). However, few systematic surveys have ever been conducted in the United States to evaluate the trend of PCB concentrations in human tissues over the years, and the reasons for the apparent slower decrease in PCB concentrations in tissues (compared to the more rapid decrease in environmental levels) are not completely known. It would be helpful to develop a database of information on congener-specific PCB levels in tissues of exposed and control cases for studying clinical and epidemiological outcomes. In particular, a comprehensive study that monitors congener specific concentrations in fish species and relates them directly to congener levels in human tissue would be extremely useful. Additional data regarding the concentrations of PCBs in body fluids or tissues of people who reside near hazardous waste sites are needed. This information is necessary for assessing the need to conduct health studies on these populations.

**Exposures of Children.** Children may be exposed to PCBs by a variety of exposure pathways. The most important pathway appears to be consumption of contaminated foods, particularly meat, fish, and poultry (Gunderson 1988). Children can also be exposed to PCBs from mother's milk (Fitzgerald et al. 1998; Kimbrough 1995; Patandin et al. 1999; Rogan et al. 1987; Wickizer et al. 1981). More data are needed on the levels of PCB exposure in nursing women from occupational situations or consumption of fish or wild game and of from those of the general population. Exposure and body burden studies related to consumption of fish in the U.S. population are needed to determine exposure levels, particularly in children of recreational and subsistence fishers. Exposure and body burden studies are also needed in Native American communities that consume high levels of game and marine mammals. Information related to the exposure of children living near hazardous waste sites is also needed. In particular,

## 6. POTENTIAL FOR HUMAN EXPOSURE

information related to the potential for children to be exposed to PCBs bound to soil and dust particles through pica or unintentional hand-to-mouth activity within homes located in these areas. Quantitative information regarding the bioavailability and amount of PCBs that children are exposed to through contact with contaminated soils are unavailable. Therefore, any information concerning this subject would be useful in evaluating children's exposure.

Additional information on weight-adjusted intakes would be helpful for determining the health risks for young children, particularly those in Native American populations. Infants and young children consume a greater amount of food per kilogram of body weight and, therefore, may have a proportionately greater exposure to PCBs than adults (Cordle et al. 1982).

Child health data needs relating to susceptibility are discussed in Section 3.12.2 Identification of Data Needs: Children's Susceptibility.

**Exposure Registries.** No exposure registries for PCBs were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to this substance.

Information is particularly needed on the size of the populations potentially exposed to PCBs through contact with contaminated media in the vicinity of hazardous waste sites. The development of an exposure registry would provide a useful reference tool in assessing exposure levels and frequencies. It would also facilitate the conduct of epidemiological or health studies to assess any adverse health effects resulting from exposure to PCBs. In addition, a registry developed on the basis of exposure sources would allow an assessment of the variations in exposure levels from one source to another and the effect of geographical, seasonal, and regulatory action on the level of exposure within a certain source. These assessments, in turn, would provide a better understanding of the needs for research or data acquisition on the current exposure levels.

## 6. POTENTIAL FOR HUMAN EXPOSURE

**6.8.2 Ongoing Studies**

A search in Federal Research in Progress (FEDRIP 2000) identified ongoing research studies that may fill some of the data needs discussed in Section 6.8.1. These studies are listed in Table 6-29.

## 6. POTENTIAL FOR HUMAN EXPOSURE

**Table 6-29. Ongoing Studies on Environmental Fate and Treatment of Polychlorinated Biphenyls**

Investigator	Affiliation	Title	Sponsor
Bopp, Richard	Mount Sinai School of Medicine New York, New York	Sources and pathways of persistent chlorinated hydrocarbon exposure in New York City	NIEHS
Bush, Brian	Wadsworth Center	Adsorption/desorption of PCBs on Hudson River clay	National Center for Research Resources
Custer, Christine M	Upper Midwest Environmental Sciences Center	Bioaccumulation and effects of PCBs on tree swallows nesting along the Housatonic River, Massachusetts	U.S. EPA, Boston, Massachusetts and U.S. Fish and Wildlife Service
Estes, James A	Western Ecological Research Center	Monitoring program for environmental contaminants in the nearshore marine ecosystem at Adak Island, Alaska	U.S. Department of Agriculture, Cooperative State Research Service
Fischer, Lawrence	Michigan State University	Health hazards from groundwater contamination	NIEHS
Hansen, LG	Veterinary Bioscience, University of Illinois	Identification of PCB congeners associated with fish consumption	U.S. Department of Agriculture, Cooperative State Research Service
Hickey, William J	University of Wisconsin Madison, Wisconsin	Research on molecular and biochemical diversity of chlorobenzoate degrading bacteria	NSF, Division of International Programs
Hong, Chia-swee	State University of New York Albany, New York	Photocatalytic remediation of PCB-contaminated water and sediment	NIEHS
Huwe, JK	Agricultural Researcher Service	Dioxins and other environmental contaminants in food	U.S. Department of Agriculture
Landrigan, Philip J	Mount Sinai School of Medicine New York, New York	Study the current urban sources, environmental distribution and toxic effects on human health of PCBs in New York City	National Institute of Environmental Health Sciences
Manny, Bruce A	Great Lakes Science Center	Contamination of surface soils and wildcelery tubers at Grassy Island in the Wyandotte National Wildlife Refuge in the Detroit River	U.S. Department of Agriculture, Cooperative State Research Service
Matthews, HB	NIEHS, NIH RTP, North Carolina	Bioavailability of PCBs from soil	NIEHS
Mora, Miguel A	Columbia Environmental Researcher Center	Effects of environmental contaminants on major wildlife species of the lower Rio Grande Valley, Texas	Department of the Interior
Rhee, G-Yull	State University of New York Albany, New York	Bioremediation of PCB- contaminated sediments in the St. Lawrence River	NIEHS
Richmond, Milo E	New York Cooperative Fish and Wildlife Research Unit	Organochlorine and <i>metal</i> contaminants in Hudson River mammals	New York State
Santerre, CR	Food and Nutrition Purdue University	Xenobiotics in farm-raised and wild fish	Indiana State
Sarofim, Adel F	Massachusetts Institute of Technology	The formation of PCBs during the pyrolysis and oxidation of wastes at Superfund sites	NIEHS

**Table 6-29. Ongoing Studies on Environmental Fate and Treatment of Polychlorinated Biphenyls (continued)**

## 6. POTENTIAL FOR HUMAN EXPOSURE

Investigator	Affiliation	Title	Sponsor
Tiedje, J	Michigan State University, Crop and Soil Sciences East Lansing, Michigan	Microbial ecology of soil and biodegradation	U.S. Department of Agriculture, Cooperative State Research Service

Source: FEDRIP 2000

EPA = Environmental Protection Agency; NIEHS = National Institute of Environmental Health Sciences; NIH = National Institute of Health; NSF = National Science Foundation; RTP = Research Triangle Park