6. POTENTIAL FOR HUMAN EXPOSURE

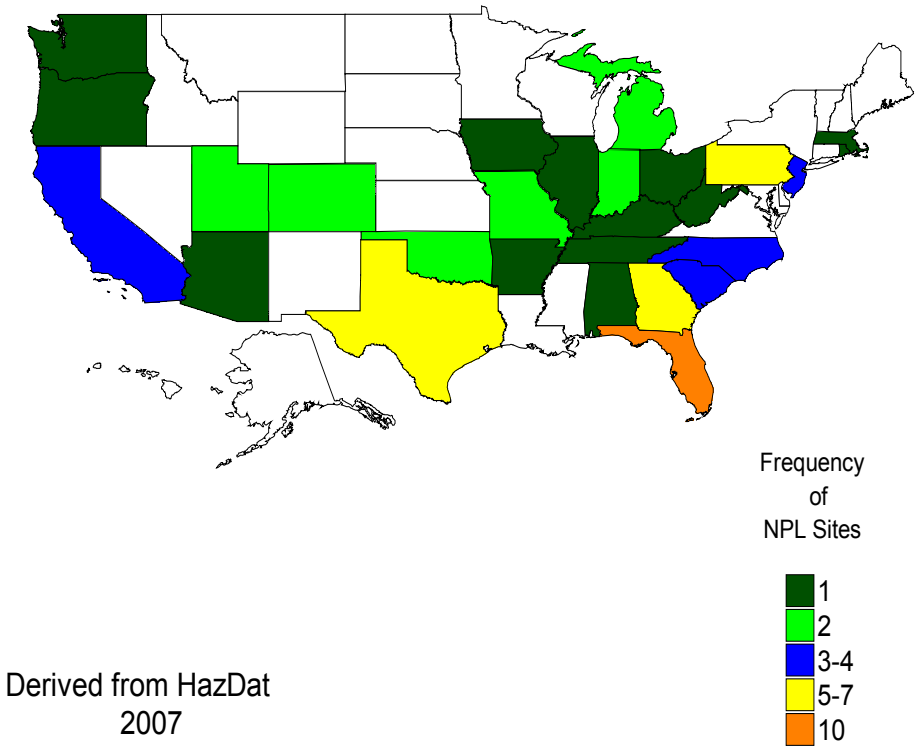
6.1 OVERVIEW

Toxaphene has been identified in at least 68 of the 1,699 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (HazDat 2007). However, the number of sites evaluated for toxaphene is not known. The frequency of these sites can be seen in Figure 6-1.

Toxaphene is a complex mixture of several hundred polychlorinated bicyclic terpene congeners (de Geus et al. 1999; Jansson and Wideqvist 1983; Lamb et al. 2008; Lau et al. 1996; Paris and Lewis 1973; Simon and Manning 2006). The transport and transformation of each of these components is influenced by its individual physical/chemical properties, in addition to those of the mixture as a whole. Although some data in the available literature indicate selective volatilization and metabolism of individual fractions of the mixture, the environmental fate of the mixture rather than of individual components has been studied by most investigators.

Toxaphene has been widely dispersed to the environment mainly as a result of its past use as an insecticide. The mixture partitions to the atmosphere, surface water and groundwater, soil and sediment particulates, and adipose tissue. As a result of its volatility and environmental persistence, toxaphene continues to be transported over long distances in the atmosphere (Andersson et al. 1988; Bidleman and Olney 1975; MacLeod et al. 2002; Paasivirta et al. 2009; Swackhamer and Hites 1988; Zell and Ballschmiter 1980). The half-life (first-order kinetics) for reaction of atmospheric toxaphene with photochemically produced hydroxyl radicals has been estimated to be at least 4–5 days for vapor-phase components of toxaphene (Howard 1991; Kelly et al. 1994); however, many congeners exist predominantly in the particulate phase and subsequently have longer atmospheric residence times and greater potential for long-range transport. Toxaphene strongly adsorbs to particles and is relatively immobile in soils (EPA 1981; Soubaneh et al. 2008; Swann et al. 1983; Wauchope et al. 1992). In water, toxaphene is strongly adsorbed to suspended particulates and sediments and is bioconcentrated by aquatic organisms to fairly high levels, with bioconcentration factors (BCFs) on the order of 4,200–60,000 (Sanborn et al. 1976; Schimmel et al. 1977). Toxaphene also appears to be biomagnified in aquatic food chains. Toxaphene is biotransformed relatively rapidly in soils and sediments under anaerobic conditions, with a half-life or half-disappearance time in the range of weeks to months (EPA 1979a). However, the mixture appears to be relatively resistant to biotransformation in these media under

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



aerobic conditions (half-life = years) (EPA 1979a; de Geus et al. 1999; Nash and Woolson 1967; Parr and Smith 1976; Smith and Willis 1978).

Recently, efforts have been made to differentiate between the form of toxaphene as it was formerly used as a pesticide, known as technical toxaphene, and the "weathered" form of this substance after years of environmental transport and degradation processes have had their effect (EPA 2010a). Weathered toxaphene is considered to be the most relevant form when assessing the current potential for human exposure to toxaphene. In order to achieve the best understanding of what individuals may be exposed to in the environment, recent studies have measured the levels of individual toxaphene congeners present in environmental samples. Congeners p-26, p-50, and p-62 are reported to be persistent in fish, marine mammals, human serum, and breast milk (Simon and Manning 2006). The toxicological implications of environmentally-persistent congeners of weathered toxaphene have not been adequately assessed.

Human exposure to toxaphene currently appears to be limited to ingestion of low concentrations of the mixture in food, particularly fish, and possibly to inhalation of ambient air. The most probable populations potentially exposed to relatively high concentrations of the mixture are individuals residing in the vicinity of hazardous waste disposal sites contaminated with toxaphene. Other subpopulations with potentially higher exposure rates may be northern Native American groups that eat aquatic mammals, which may contain residues of toxaphene (Muir et al. 1992), recreational or subsistence hunters in the southern United States that consume significant amounts of game animals (especially species like raccoons) (Ford and Hill 1990), and people who consume certain types of sportfish caught in the Great Lakes (ATSDR 2009).

6.2 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report releases into the environment (EPA 2005). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ 10 or more full-time employees; if their facility is included in Standard Industrial Classification (SIC) Codes 10 (except 1011, 1081, and 1094), 12 (except 1241), 20–39, 4911 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4931 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4939 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4953 (limited to facilities regulated under RCRA Subtitle C,

42 U.S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited to facilities primarily engaged in solvents recovery services on a contract or fee basis); and if their facility produces, imports, or processes ≥25,000 pounds of any TRI chemical or otherwise uses >10,000 pounds of a TRI chemical in a calendar year (EPA 2005).

Toxaphene has been detected in the atmosphere, soils, surface waters and sediments, rainwater, aquatic organisms, and foodstuffs. Historically, toxaphene has been released to the environment mainly as a result of its past use as an agricultural insecticide (EPA 1979b). Toxaphene-like mixtures of PCC congeners may also be released to the environment as unintentional byproducts from manufacturing processes involving chlorination, such as those used for paper and pulp (Rantio et al. 1993). There are no known natural sources of the mixture.

Because toxaphene is a Priority Pollutant under the Clean Water Act, it is required to be included in the TRI (EPA 2005). However, since most registered uses of toxaphene as a pesticide were canceled in 1982 (EPA 1982a) and all registered uses were canceled in the United States and its territories after 1990 (EPA 1990b), production of toxaphene for domestic pesticide use in the United States has ceased. Consequently, most releases of toxaphene reported to TRI for 2012 were disposals to landfills (TRI12 2013).

Current sources of toxaphene in the environment that may result in exposure for the U.S. population is long-range atmospheric transport from countries currently producing or using toxaphene (e.g., Mexico and countries in Central America, eastern Europe, the former Soviet Union, and parts of Asia) (Swackhamer et al. 1993; Voldner and Li 1993) and continued releases from previously contaminated U.S. soils and waters.

6.2.1 Air

Estimated releases of 10 pounds (0.005 metric tons) of toxaphene to the atmosphere from 11 domestic manufacturing and processing facilities in 2012, accounted for <0.41% of the estimated total environmental releases from facilities required to report to the TRI (TRI12 2013). These releases are summarized in Table 6-1.

As a result of its past use as an insecticide on crops in the southern United States, toxaphene was dispersed directly to the atmosphere by aerial and ground application (EPA 1979b). Volatilization of the

Table 6-1. Releases to the Environment from Facilities that Produce, Process, or Use Toxaphene[a]

		Reported amounts released in pounds per year[b]							
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
IL	1	0	0	0	1	0	0	1	1
MI	1	5	0	0	17	0	21	0	21
NE	1	2	0	0	0	0	2	0	2
NV	1	0	0	0	10	0	10	0	10
OH	3	2	0	0	133	0	133	1	134
OR	1	0	0	0	2,278	0	2,278	0	2,278
SC	1	0	7	0	0	0	7	0	7
TX	1	0	0	0	2	0	0	2	2
UT	1	1	0	0	0	0	1	0	1
Total	11	10	7	0	2,441	0	2,454	4	2,458

[a]The TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.
[b]Data in TRI are maximum amounts released by each facility.
[c]Post office state abbreviations are used.
[d]Number of reporting facilities.
[e]The sum of fugitive and point source releases are included in releases to air by a given facility.
[f]Surface water discharges, waste water treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).
[g]Class I wells, Class II-V wells, and underground injection.
[h]Resource Conservation and Recovery Act (RCRA) subtitle C landfills; other onsite landfills, land treatment, surface impoundments, other land disposal, other landfills.
[i]Storage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown
[j]The sum of all releases of the chemical to air, land, water, and underground injection wells.
[k]Total amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI12 2013 (Data are from 2012)

mixture from treated crop and soil surfaces following application also introduced substantial amounts of toxaphene to the atmosphere. For example, Willis et al. (1980, 1983) reported volatilization losses from treated cotton canopies of up to 80% of applied toxaphene within 11 days after treatment. Seiber et al. (1979) also reported that volatilization from leaf and soil surfaces was the major removal mechanism for toxaphene applied to cotton crops under field conditions. These investigators reported differential vaporization of the mixture (i.e., selectively greater loss of the more volatile components from soil and leaf surfaces), which was matched by a corresponding enrichment of these components in ambient air samples.

Toxaphene shows a strong tendency to sorb to particulates, and there has been a tendency to believe that toxaphene residuals in older hazardous waste sites would be relatively inert. Studies based primarily on theoretical considerations and computer screening models suggest that the PCCs could volatilize to the atmosphere unless a waste site has a clay cap thicker than approximately 0.3 m. The potential for volatilization increases if the soil matrix in which the toxaphene is buried has a significant sand fraction (Jury et al. 1990). These theoretical findings seem compatible with field measurements on several pesticides that showed the volatilization rates for toxaphene applied to soils were significantly higher than rates for triazine herbicides or alachlor (Glotfelty et al. 1989a). Toxaphene has been identified in air samples collected at 3 of the 68 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2007).

6.2.2 Water

Estimated releases of 7 pounds (0.003 metric tons) of toxaphene to surface water from 11 domestic manufacturing and processing facilities in 2012, accounted for 0.28% of the estimated total environmental releases from facilities required to report to the TRI (TRI12 2013). Estimated releases of 4 pounds (0.002 metric tons) of toxaphene off-site, which include transfers to publicly owned treatment works (POTWs), accounted for 0.16% of the estimated total environmental releases from facilities required to report to the TRI. The 2012 TRI release information is summarized in Table 6-1.

Toxaphene has been released to surface waters as a result of its direct application to lakes as a piscicide (EPA 1979b), in waste water releases from manufacturing and formulation plants (Durant and Reimold 1972), and in activities associated with the disposition of residual pesticides. For example, Mirsatari et al. (1987) described the release of aircraft rinse water to drainage ditches following aerial application of toxaphene, and the compound has been detected in surface water samples taken from disposal ponds at a

Superfund site (EPA 1986). NOAA (1974) reported that toxaphene concentrations in the effluent of a manufacturing plant decreased over a 4-year period from an average maximum monthly concentration of 2,332 ppb in August 1970 to 6 ppb in July 1974.

Because neat technical toxaphene sorbs to particulates and is markedly hydrophobic, it has been argued that toxaphene would not be able to migrate more than about 10 cm down a soil profile and, therefore, would not be of concern as a groundwater contaminant. Such arguments tend to overlook the fact that technical toxaphene used as a pesticide was usually mixed with a hydrocarbon solvent (e.g., xylene) as a carrier, which increased the mobility of toxaphene in soils. Data compiled by the EPA on pesticides in groundwater indicates that toxaphene was found in groundwater in one state as a result of normal agricultural use (Ritter 1990). Also, when such pesticide preparations have been introduced at old waste disposal sites, the toxaphene may be able to move into groundwater with the carrier-solvent. This scenario has been documented at a waste disposal site in California (Jaquess et al. 1989). The authors see this as a possibility at many waste disposal sites containing solvent materials, with toxaphene detections in groundwater at NPL sites, in the Mississippi Delta, and near Houston, Texas, supporting similar pollution pathways. Toxaphene has been identified in surface water and groundwater samples collected at 14 and 27 of the 68 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2007). For most groundwater supplies, however, any significant residence time in poorly oxygenated or anaerobic subsoil vadose zones would be expected to allow for anaerobic biochemical degradation of toxaphene.

6.2.3 Soil

Estimated releases of 2,441 pounds (1.11 metric tons) of toxaphene to soils from 11 domestic manufacturing and processing facilities in 2012, accounted for about 99.3% of the estimated total environmental releases from facilities required to report to the TRI (TRI12 2013). No underground injection releases were reported (TRI12 2013). The TRI release data are summarized in Table 6-1.

Toxaphene has been released directly to soils primarily as a result of its past use as an insecticide on agricultural crops (EPA 1979b). Disposal of spent livestock-dipping solutions (McLean et al. 1988) and wastes from manufacturing and formulation processes (EPA 1979b) were other significant sources of soil contamination. Mirsatari et al. (1987) reported that toxaphene has been found as a contaminant at pesticide disposal sites at concentrations in soils or sediment approaching or exceeding 100 ppm. Toxaphene was listed as a chemical of concern at the Crystal City Airport Superfund site in Crystal City,

Texas. The mixture was detected in surface soil samples taken at the airport following abandonment of agricultural chemicals at the site by defunct aerial application operators (EPA 1987b). Toxaphene was also found in pesticide contaminated soils at four other Superfund sites in Litchfield, Arizona; Albany, Georgia; Marrianna, Florida; and Malone, Florida; concentrations in these soils ranged from 18 to 1,505 mg/kg (ppm) (Troxler et al. 1993). Toxaphene has been identified in soil and sediment samples collected at 40 and 22 of the 68 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2007).

6.3 ENVIRONMENTAL FATE

6.3.1 Transport and Partitioning

A combination of monitoring and modeling efforts during the 1980s has firmly established the importance of atmospheric pathways as a major source of PCC inputs to regions in the upper latitudes far removed from regions where it was heavily used as an agricultural pesticide. Adaptations to regional transport models initially developed to study acid rain phenomena showed the physical possibility for atmospheric transport of toxaphene from locations in the southern United States to the Great Lakes Region of the northern United States and Canada (Hoh and Hites 2004; James and Hites 2002; MacLeod et al. 2002; Voldner and Schroeder 1989, 1990).

A series of studies by Canadian researchers has gathered detailed information on levels of toxaphene in various environmental compartments in regions ranging from Lake Baikal in Russia, to the Sargasso Sea, to the southeastern United States, to various areas in Canada and the Canadian Arctic (Barrie et al. 1993; Bidleman et al. 1989, 1992, 1993, 1995; Cotham and Bidleman 1991; Lockhart et al. 1992; McConnell et al. 1993; Muir et al. 1990, 1992). These studies help provide at least partial validation for the predictions from regional transport models and document the continued supply of PCC materials to areas in the northern hemisphere far removed from areas of former significant toxaphene use.

Researchers working with the atmospheric transport of toxaphene have assembled useful time series observations for sites along the southern Atlantic coast in the United States, in the Canadian Maritime provinces, and at stations in the Canadian Arctic (Bidleman et al. 1989, 1992, 1995). Comparisons of levels in environmental media during the 1990s with baseline concentrations in the 1970s and early 1980s did not suggest declines in toxaphene contaminants, with ambient air concentrations in particular remaining about the same or even increasing. Especially in high latitude areas, impacts from toxaphene

were still a matter of concern nearly a decade after the United States began phasing out the use of toxaphene as a pesticide agent.

Toxaphene is a mixture of many congeners, each of which has its own unique Henry's law constant. A Henry's law constant of $6x10^{-6}$ atm-m^3/mol at 20°C was measured for the mixture, which suggests that many components of toxaphene will volatilize to the atmosphere from water and soil surfaces. A half-life (first-order kinetics) of 6 hours to 12 days has been estimated for the volatilization of toxaphene from a model river, one meter deep, with a flow rate of 1 m/second and a wind velocity of 3 m/second (Howard 1991). The results of numerous field dissipation and atmospheric monitoring studies indicate that the atmosphere is indeed the most important environmental medium for transport of the mixture. In addition to the field dissipation studies cited in Section 6.2.1 (Seiber et al. 1979; Willis et al. 1980, 1983), significant partitioning of toxaphene to the atmosphere has been reported in a model agroecosystem study (Nash et al. 1977) and from fallow field soils (Glotfelty et al. 1989a).

The persistence of toxaphene in the atmosphere allows the mixture to be transported long distances from the application sites. The presence of toxaphene in surface waters of the Great Lakes originated from the aerial transport and deposition of the mixture from application sites in the southern United States (EPA 1984b; Hoh and Hites 2004; James and Hites 2002; Ma et al. 2005a, 2005b; MacLeod et al. 2002). Detection of toxaphene in the tissues of fish taken from a remote lake on Isle Royale in Lake Superior was also cited as evidence of long-range atmospheric transport (Swackhamer and Hites 1988).

Numerous other investigations have reported long-range atmospheric transport of toxaphene to remote locations. Toxaphene was detected in ambient air samples taken over the western North Atlantic Ocean and Bermuda. The source of the contamination was attributed to cotton-growing areas in the southern United States 1,200 km away (Bidleman and Olney 1975). The presence of toxaphene in biota of the Barents Sea in Northern Europe has been attributed to transport via air currents from areas of historical use in southeastern Europe and around the rivers that flow into the Aral Sea (Paasivirta et al. 2009). Maximum concentrations of toxaphene found in North American peat bogs corresponded to the period of maximum production and use of the compound in the United States in the mid-1970s (Rapaport and Eisenreich 1986). The composition of the toxaphene residues in the peat cores indicated that they were delivered to the peat surface by atmospheric transport and deposition with the dominant wind circulation patterns from primary source regions in the southern and southeastern United States. The presence of toxaphene in the following sources has also been attributed to its long-range atmospheric transport: fish taken from remote lakes in northern Canada (Muir et al. 1990); fish from pristine areas in the North

Atlantic Ocean, North Pacific Ocean, and Antarctic Ocean (Zell and Ballschmiter 1980); and fish, birds, and seals from the western North Atlantic Ocean, Arctic Ocean, Greenland, Canada, and Sweden (Andersson et al. 1988). Evidence of regional-scale transport of the mixture in the drainage basin of the Chesapeake Bay has also been reported (Glotfelty et al. 1989b).

Atmospheric toxaphene is transported back to soil and water surfaces by wet and dry deposition processes (Glotfelty et al. 1989b; Hoff et al. 1993a; Villeneuve and Cattini 1986). Several investigators have reported that washout in rain appears to be more important than the dry deposition of toxaphene (Bidleman et al. 1981; EPA 1984b). Hoff et al. (1993a) cited an unpublished 1992 report from the Great Lakes Protection Fund/Environment Canada in which the wet and dry deposition fluxes of PCCs to the Great Lakes were estimated to be 3.5–12.5 and 1.5–6.3 kg/year, respectively. Dry deposition accounted for only 15% of the input of atmospheric toxaphene into a rural estuary in South Carolina (Harder et al. 1980). Based on a range of assumptions about the concentration of PCCs in the Great Lakes, Hoff et al. (1993a) estimated that the annual loading of toxaphene by gas exchange may be more than an order of magnitude higher than the input by wet or dry deposition. The authors noted that even though potential errors in the assumptions for the gas transfer of PCCs were very large, they were not large enough to make wet and dry deposition fluxes comparable to the estimates of the gas phase mass transfer of toxaphene across the air/water interface. Burniston et al. (2005) measured toxaphene concentrations in precipitation into Lake Ontario from 1994 to 1998. These authors reported that estimates of wet deposition flux were 50% of the estimated gas deposition flux based on loadings of toxaphene for Lake Ontario via precipitation during 1998.

For higher latitude regions, there is more uncertainty about the importance of specific deposition mechanisms. Especially in Arctic areas, model estimates and available monitoring data suggest that dry particle deposition may be more important than scavenging through snowfall (Cotham and Bidleman 1991). The mechanisms for toxaphene show many similarities with fate and transport processes for hexachlorobenzene (HCB) and perhaps several other organochlorine toxicants. The hydrophobic properties of these organochlorines encourage partitioning in either a volatile or semi-volatile phase or in forms sorbed to particulates. These properties then facilitate the incorporation of the contaminants into food chains starting with algae, zooplankton, and macroinvertebrates. This in turn encourages biomagnification at higher trophic levels (Cotham and Bidleman 1991; Hargrave et al. 1992).

Toxaphene released to soils will persist for long periods of time. The high K_{oc} (soil organic carbon partition coefficient) values for toxaphene (log K_{oc}=3–5) (EPA 1981; Soubaneh et al. 2008; Wauchope et

al. 1992) suggest that the mixture should be strongly sorbed to soil particulates and, therefore, should be relatively immobile to leaching and inhibited from volatilizing from subsurface soils (Swann et al. 1983). Field studies have verified this behavior. Half-lives (first-order kinetics) ranging from approximately 1 year (Adams 1967) to 14 years (Nash and Woolson 1967) have been reported for toxaphene in soils. In surface soils, where volatilization will be a significant transport process, half-lives of 2 and 4 months have been reported for samples taken at the top 2.5 and 7.5 cm, respectively (Seiber et al. 1979). Between 85 and 90% of the total toxaphene residues were found in the upper 23 cm (cultivated layer) of a sandy loam test soil 13 years after the last foliar application of the mixture (Nash and Woolson 1968). Following multiple annual applications of toxaphene to cotton crops grown in a clay soil, Swoboda et al. (1971) detected 90–95% of toxaphene residues in the top foot of the 5-foot profile sampled; toxaphene was not detected in any of the drainage water samples taken from the site. About 93% of the toxaphene found in runoff from a treated cotton field on a silty clay soil was bound to the sediment fraction; only 7% was found in the aqueous fraction of the runoff (McDowell et al. 1981). Toxaphene concentrations in runoff varied seasonally, and losses in two of the years studied totaled only 0.5–1% of the amount applied. Runoff losses from a cotton crop grown in the Mississippi Delta were found to be 0.4% of applied toxaphene (Lorber and Mulkey 1982). Raff and Hites (2004) measured toxaphene levels in suspended sediment samples along the Mississippi River. Based on these data and water discharge rates, the authors estimated a release of 200–1,000 kg of toxaphene into the Gulf of Mexico from the main stem of the river during 2002. The source of toxaphene was attributed to nonpoint source runoff from agricultural lands.

According to the simulation models Foliar Washoff of Pesticides (FWOP), Chemical Runoff and Erosion from Agricultural Management Systems (CREAMS), and Pesticide Runoff Simulator (PRS), up to 3% of applied toxaphene may be lost in runoff and erosion from treated agricultural fields; all of the toxaphene would be associated with the sediment fractions (Smith and Carsel 1984). To evaluate the effects of toxaphene on groundwater and surface water quality under different land management practices, Donigian and Carsel (1987) used three models: the Pesticide Root Zone Model (PRZM); the Analytical Transient One-, Two-, and Three-Dimensional Simulation of Waste Transport in the Aquifer System (AT123D); and the Stream Transport and Agricultural Runoff of Pesticides for Exposure Assessment (STREAM). The dissolved mean toxaphene concentration in surface water predicted by the STREAM model for a 1.0 kg/ha application rate was 11.6 ppb for conventional-till, 4.9 ppb for reduced-till, and 3.4 ppb for no-till practices. Surface water runoff loadings and concentrations of toxaphene and several other pesticides typically decreased under the conservation tillage scenarios, but groundwater loadings and concentrations generally increased as a result of decreased runoff and increased groundwater recharge. The authors did not provide estimates of groundwater concentrations for toxaphene because

this pesticide did not demonstrate mean annual loadings high enough to require estimation of groundwater concentrations.

The mobility of toxaphene in soils also is influenced by soil moisture status and the presence of other organic solvating materials (Jaquess et al. 1989). Toxaphene did leach from laboratory columns of sand and sandy loam soils treated with organic solvents and emulsifiers when the columns were allowed to dry completely between wetting cycles. The mixture did not leach from the amended columns when a similar amount of water was applied on a continuous basis. Drying of the soil allowed crevices to form in the columns which expedited movement of the mixture. Toxaphene dissolved in the organic solvent or contained in the emulsifier amendment could leach through the macropores.

There is also evidence that voltatilization is the primary route of loss from toxaphene-treated foliage. In a study by Seiber et al. (1979), residues of toxaphene were analyzed in cotton leaves and associated air samples up to 58 days after a 9 kg/ha application of toxaphene to a cotton field in the San Joaquin Valley, California. Analyses of the cotton leaf samples indicated a 59% loss of toxaphene at 28 days post-application. Leaf residues declined from 661 ppm on the day of application to 135 ppm on day 50 post-application, with an observed trend toward greater loss of the more highly volatile components. A corresponding enrichment of volatile toxaphene components was observed in air samples. There was no indication of chemical degradation in these samples in spite of the presence of abundant sunlight, oxygen, and atmospheric oxidant throughout the study.

Toxaphene is highly insoluble in water (0.55 mg/L) (Murphy et al. 1987). Toxaphene in surface waters that is not volatilized to the atmosphere is sorbed to sediments or suspended particulates, which are ultimately deposited in sediments (EPA 1979a). The lower-solubility, more-chlorinated components of the mixture are preferentially sorbed to particulates and sediments. Paris et al. (1977) reported that the less soluble, more highly chlorinated fractions of toxaphene also appear to be selectively sorbed to aquatic microorganisms that are consumed by other organisms and, consequently, would be expected to bioaccumulate up the food chain.

Uptake factors (mg toxaphene sorbed per microorganism/concentration of toxaphene in the medium) ranged from $3.4x10^3$ to $1.7x10^4$ for a variety of bacteria, fungi, and algae *(Bacillus subtilis, Flavobacterium harrisonii, Aspergillus sp., Chlorella pyrenoidosa)* (Paris et al. 1977). Direct sorption of toxaphene onto sediment, plankton, and other suspended solids deposited in the sediment has also been

reported in three lakes in Wisconsin where the mixture was applied for the control of nongame fish. Toxaphene sorbed to sediments was not found to be readily desorbed (Veith and Lee 1971).

Toxaphene is bioconcentrated in the tissues of aquatic organisms. The major toxaphene congeners found in fish from pristine environments in the Canadian Rocky Mountains have been found to be the Cl_7–Cl_9 camphenes (i.e., hepta-, octa-, and nonachlorobornenes) (Bruns and Birkholz 1993). Experimentally determined bioconcentration factors (BCFs) for several aquatic organisms have been found to range from 4,200 to 60,000. In a flow-through bioassay conducted with the longnose killfish (*F. similis*), BCFs of up to 33,300 in fry and 60,000 in juvenile fish after 28 days of exposure were reported; BCFs in adults ranged from 4,200 to 6,800 after 14 days of exposure (Schimmel et al. 1977). Oysters (*C. virginica*) exposed to 1 ppb toxaphene have been found to accumulate up to 23 ppm in tissue after 24 weeks exposure; tissue concentrations decreased to nondetectable levels at the end of a 12-week depuration period (Lowe et al. 1971). In a model ecosystem study using radiolabeled toxaphene, BCFs of 6,902 for algae, 9,600 for snails, 890 for mosquitoes, and 4,247 for fish (*Gambusia affinis*) were reported (Sanborn et al. 1976).

Toxaphene has also been detected in the tissues of aquatic organisms in numerous field studies (see Section 6.4.4). For example, mean toxaphene concentrations of 11 ppm in lipid tissue for lake trout (*Salvelinus namaycush*) and 7 ppm in lipid tissue for whitefish (*Coregonus clupeaformis*) taken from a remote lake on Isle Royale in Lake Superior have been reported (Swackhamer and Hites 1988). Studies conducted in a natural ecosystem in northwestern Ontario on the fate of toxaphene in lake trout (*S. namaycush*) and white suckers (*Catastomus commersoni*) indicated depuration half-lives for total toxaphene ranging from 232 days (lake trout, initial intraperitoneal dose 7.0 μg/g) to 524 days (white suckers, initial intraperitoneal dose 3.5 μg/g), with first-order kinetics assumed (Delorme et al. 1993). Depuration half-lives for two of the more persistent toxaphene congeners, octachlorobornane T2 and nonachlorobornane T12, ranged from 294 days (lake trout; T2, initial intraperitoneal dose 7.0 μg/g) to 716 days (white suckers; T2, initial intraperitoneal dose 3.5 μg/g) with first-order kinetics assumed. The overall results of this study indicated significant interspecies differences in the ability to eliminate toxaphene, as well as possible intraspecies differences in the ability to eliminate different toxaphene congeners.

Toxaphene also appears to be biomagnified in aquatic food chains, although not to the extent of PCBs or other chlorinated insecticides, such as DDT. Stapleton et al. (2001) found that PCB burdens were greater than toxaphene burdens for each Great Lakes fish species collected during 1997–1998 with the exception

of deepwater sculpin. Evans et al. (1991) reported trophic biomagnification of toxaphene, with toxaphene concentration increasing by an average factor of 4.7 from plankton (mean concentration, 0.55 ppm) to fish (deepwater sculpin: mean concentration, 2.57 ppm). DDE and PCBs were found to be more strongly biomagnified, increasing 28.7 and 12.9 times, respectively, in average concentration from plankton to sculpin. Whittle et al (2000) measured food web toxaphene concentrations in four of the Great Lakes (Table 6-2). Based on these data, toxaphene biomagnification factors were determined to be 32.03 in Lake Superior, 24.33 in Lake Huron, 10.08 in Lake Erie, and 30.43 in Lake Ontario. In a study that included analyses of tissue residue levels in 16 species of fish, birds, amphibians, and reptiles, biomagnification of toxaphene was reported in three oxbow lakes in northeastern Louisiana (Niethammer et al. 1984). Tissue residue concentrations were highest in tertiary consumers (carnivores) and lowest in primary consumers (herbivores); toxaphene was not detected in the limited number of surface water or sediment samples taken from the lakes. The source of the toxaphene was apparently the surrounding cotton and soybean cropland, which had historically received heavy pesticide applications. Biomagnification was also reported in a study that included analyses of tissue residue levels in eight species of fish and water snakes in the area of the Yazoo National Wildlife Refuge, Mississippi (Ford and Hill 1991). Biomagnification of several organochlorine pesticides, including toxaphene, was apparent from soil sediments (geometric mean concentration, approximately 0.1 ppm) to mosquito fish, a larger secondary consumer and forage fish (geometric mean concentration, 0.25 ppm), to the spotted gar, a tertiary consumer (geometric mean concentration, 2.71 ppm). There was, however, no clear pattern of biomagnification in larger secondary consumers such as smallmouth buffalo and carp, or in tertiary consumers such as water snakes.

Biomagnification of toxaphene in marine ecosystems appears to be species dependent (de Boer and Wester 1993). The two main toxaphene congeners found in marine mammals such as seals and beluga whales are an octa- and a nonachlorobornane, which are present only as minor constituents in technical toxaphene (Vetter et al. 1993, 1994). No biomagnification of toxaphene in a Canadian arctic marine food chain was reported in a study conducted by Muir et al. (1988a). Toxaphene was detected in the muscle tissue of the arctic cod (*Boreogadus saida*) at a mean concentration of 0.018 ppm, but not in the blubber and liver of the ringed seal (*Phoca hispida*), which preys on the cod, or the fat of the polar bear (*Ursus maritimus*), which preys on the seal. Similar results were found by Andersson et al. (1988), who performed limited sampling of biota from various trophic levels in marine food chains in the western North Atlantic Ocean, Greenland, Sweden, and Canada. They reported that toxaphene concentrations in fish, bird, and seal tissues ranged from 0.33 to 17 ppm in fat tissue for all trophic levels versus 0.14–990 ppm for DDT and PCB residues. These results were interpreted as being indicative of less

Table 6-2. Food Web Total Toxaphene Concentrations (µg/g Wet Weight) Measured in Lake Superior, Lake Huron, Lake Erie, and Lake Ontario

Species	Lake Superior	Lake Huron	Lake Erie	Lake Ontario
Lake trout	1.926	0.365	0.081	0.639
Herring	1.024	–[a]	–	–
Sculpin	0.546	0.312	–	0.245
Smelt	0.291	0.119	0.016	0.066
Alewife	–	0.139	–	0.049
Diporeia	0.197	0.131	0.029	0.090
Mysis	0.091	0.020	–	0.034
Plankton	0.062	0.015	<0.015	0.021

[a]Not analyzed.

Source: Whittle et al. 2000

biomagnification and/or more effective metabolism of toxaphene at higher trophic levels, as compared with DDT and PCB.

In another study, however, toxaphene was found in the tissues of white-beaked dolphins (*Lagenorhynchus albirostris*) and pilot whales (*Globicephala melaena*) taken off the coast of Newfoundland in 1980 and 1982 (Muir et al. 1988b). The toxaphene peaks from the gas liquid chromatography (GLC) analyses of the dolphin blubber indicated considerable metabolism of the mixture, as compared with toxaphene residues detected in the local fish populations preyed upon by the dolphins. Other studies in the area of Baffin Bay, Canada, have found cetacean blubber with an average toxaphene congener concentration of 9.2 ppm for male narwhals. Tissue concentrations in individual males ranged up to 13.2 ppm (Muir et al. 1992). De Boer and Wester (1993) also found evidence of biomagnification of toxaphene in the marine food chain from fish to fish predators, and reported biomagnification factors (BMFs) of approximately 40 for harbor porpoise/fish and 100 for whitebeaked dolphin/fish. Comparison of the chromatograms from whitebeaked dolphin (blubber) and fish (hake liver) indicated similar metabolism of toxaphene for both species.

Tissue residue data from marine ecosystems have been used by Hargrave et al. (1993) to calculate the following ranges of BMFs (ng PCC/g lipid predator per ng PCC/g lipid prey) for various predator-prey links among arctic marine organisms. In a hypothetical food web, the following ranges in BMF values were reported: arctic cod and char/zooplankton (19.7–36.7); ringed seal/arctic cod and char (0.1–0.2); beluga/arctic cod and char (2.0–2.3); narwhal/arctic cod and char (3.3–3.4); small lysianassid amphipods/ arctic cod and char (0.7–2.7); small lysianassid amphipods/ringed seal (4.7–15.5); small lysianassid amphipods/beluga (0.4–1.1); *Eurythenes gyrillus*/arctic cod and char (9.1–11.1); *E. gyrillus*/narwhal (2.8–3.2); *E. gyrillus*/beluga (4.6–4.8); *E. gyrillus*/ringed seal (55.3–65.3); *E. gyrillus*/eelpout (4.4–19.2); and eelpout/small lysianassid amphipods (0.2–2.7).

6.3.2 Transformation and Degradation

Toxaphene is not a single molecular substance, but rather a mixture of hundreds of congeners including chlorinated bornanes, bornenes, bornadienes, camphenes, and dihydrocamphenes (see Section 4.2). The form of toxaphene as it was originally applied in the past as a pesticide is referred to as technical toxaphene. The composition of technical toxaphene released to the environment changes over time as the congeners degrade at different rates. Degradation proceeds mainly through dechlorination and dehydrochlorination, resulting in a shift in composition toward lower chlorinated homologs (Buser et al.

2000; Lamb et al. 2008). The changed form of toxaphene is commonly referred to as weathered toxaphene (Lamb et al. 2008; Simon and Manning 2006). In order to achieve the best understanding of what individuals may be exposed to in the environment, recent studies have measured the levels of individual toxaphene congeners present in environmental samples. Some of the toxaphene congeners that have been reported in the literature are listed in Table 6-3. Congeners p-26, p-50, and p-62 are reported to be persistent in fish, marine mammals, human serum, and breast milk (Simon and Manning 2006). The congeners Hx-Sed and Hp-Sed are known degradation products of toxaphene (Buser et al. 2000; EPA 2010a). Kapp and Vetter (2011) synthesized hydroxylated polychlorobornanes to better understand the transformation processes and the potential for the production of hydroxylated metabolites from the degradation of toxaphene. The authors concluded that hydroxylated compounds of technical toxaphene may be present from the degradation of toxaphene in the environment, but have not been described more frequently in literature due to their elusiveness in analytical detection.

6.3.2.1 Air

The worldwide, long-range atmospheric transport of the mixture suggests that toxaphene is relatively resistant to transformation in the atmosphere. Since the production of toxaphene involves exposing chlorinated camphenes to ultraviolet radiation, the congeners in the final mixture are resistant to degradation from direct photolysis (EPA 1976a; Korte et al. 1979). Consequently, toxaphene in the atmosphere is not expected to degrade readily by direct photolysis when attached to particulates. However, a half-life of approximately 4–5 days (first-order kinetics) has been estimated for the reaction of vapor-phase toxaphene with photochemically produced hydroxyl radicals (Howard 1991; Kelly et al. 1994). The higher chlorinated congeners have longer half-lives since they tend to exist in the particulate phase rather than the vapor phase. Rapaport and Eisenreich (1986) cited an atmospheric residence time of 46–70 days for the mixture. They noted that the toxaphene found in peat cores taken from remote regions in the northern United States and Canada was deposited from the atmosphere in a relatively untransformed state.

6.3.2.2 Water

Little information was found in the available literature on the biodegradation of toxaphene in aquatic systems. Toxaphene is resistant to chemical and biological transformation in aerobic surface waters (de Geus et al. 1999). It is not expected to undergo direct photolysis or photooxidation (EPA 1979a). Hydrolysis is also not an important fate process; a hydrolytic half-life (first-order kinetics) of >10 years

Table 6-3. Names and Parlar Identification Numbers of Some Toxaphene Congeners Reported in the Literature

Name	CAS number	Parlar number
2,2,3-exo,8,9,10(E)-Hexachlorocamphene	–	p-11
2-exo,3-endo,8,8,9,10(E)-Hexachlorocamphene	–	p-12
2,2,5,5,9,10,10-Heptachlorobornane	–	p-21
2-endo,3-exo,5-endo,6-exo,8,8,10,10-Octachlorobornane	142534-71-2	p-26
2,2,5-endo,6-exo,8,9,10-Heptachlorobornane (Toxicant B)	–	p-32
2,2,5,5,9,9,10,10-Octachlorobornane	–	p-38
2,2,3-exo,5-endo,6-exo,8,9,10-Octachlorobornane	–	p-39
2-endo,3-exo,5-endo,6-exo,8,9,10,10-Octachlorobornane	166021-27-8	p-40
2-exo,3-endo,5-exo,8,9,9,10,10-Octachlorobornane	165820-16-6	p-41
2,2,5-endo,6-exo,8,8,9,10-Octachlorobornane (Toxicant A1)	–	p-42a
2,2,5-endo,6-exo,8,9,9,10-Octachlorobornane (Toxicant A2)	–	p-42b
2-exo,5,5,8,9,9,10,10-Octachlorobornane	165820-17-7	p-44
2-endo,3-exo,5-endo,6-exo,8,8,9,10,10-Nonachlorobornane	6680-80-8	p-50
2,2,5,5,8,9,10,10-Octachlorobornane	–	p-51
2,2,5-endo,6-exo,8,8,9,10,10-Nonachlorohornane	–	p-56
2,2,5-endo,6-exo,8,9,9,10,10-Nonachlorobornane	–	p-59
2,2,5,5,8,9,9,10,10-Nonachlorobornane	154159-06-5	p-62
2-exo,3-endo,5-exo,6-exo,8,8,9,10,10-Nonachlorobornane	–	p-63
2,2,5,5,6-exo,8,9,9,10,10-Decachlorobornane	–	p-69
2-exo,3-endo,6-exo,8,9,10-Hexachlorobornane (Hx-Sed)	–	–
2-endo,3-exo,5-endo,6-exo,8,9,10-Heptachlorobornane (Hp-Sed)	–	–
2-exo,3-endo,5-exo,8,9,10,10-Heptachlorobornane (TMX-1)	–	–
2-exo,3-endo,-5-exo,9,9,10,10-Octachlorobornane (B7-1453)	–	–
2-endo,3-exo,5-endo,6-exo,8,8,9,10-Octachlorobornane (B8-1412)	–	–

Sources: de Geus et al. 1999; EPA 2010a; Gooch and Matsumura 1985; Lau et al. 1996; Vetter et al. 2001; Xia et al. 2009

for pH 5–8 at 25°C has been estimated (EPA 1976d, 1979a). Detoxification of toxaphene in eight Wisconsin lakes was reported to be due to adsorption rather than biodegradation (EPA 1977).

Buser et al. (2000) measured half-lives ranging from <1 day to several days for technical toxaphene congeners in anaerobic sewage sludge from a municipal waste water treatment plant. The non-gem-chloro-substituted congeners P26 and P50 degraded less rapidly than the gem-chloro-substituted congeners, which is consistent with the relatively high percentage of the P26 and P50 congeners detected in environmental samples (Buser et al. 2000; Lamb et al. 2008). Degradation was said to proceed through reductive dechlorination resulting formation of Hp-Sed and Hx-Sed and other metabolites. Lacayo et al. (2004) studied the degradation of toxaphene in water in aerobic and anaerobic bioreactors operating in sequence using a mixed culture inoculum. Reported degradation was 87% after 6 weeks and 98% after 39 weeks, with the majority of the degradation occurring under anaerobic conditions. Levels of toxaphene congeners with greater chlorine substitution decreased more rapidly than those with lesser chlorine substitution.

6.3.2.3 Sediment and Soil

Toxaphene has been reported to be quite persistent in aerobic surface soils. Nash and Woolson (1967) reported a half-life of 11 years (first-order kinetics) in an aerobic sand loam soil that had received high application rates (112 and 224 kg/ha, corresponding to approximately 50 and 100 ppm) of toxaphene. Seiber et al. (1979) reported half-lives of approximately 2 months (top 2.5 cm) and 4 months (top 7.5 cm) in aerated topsoil that had been treated with toxaphene at an application rate of 9 kg/ha. While the observed declines in toxaphene concentrations were primarily due to vaporization, at least one toxaphene component was reported to be significantly degraded. The mechanism of degradation was postulated to be dehydrochlorination or reductive chlorination, but this was not investigated further. Studies by Parr and Smith (1976) and Smith and Willis (1978) in a silty loam soil indicated no transformation of toxaphene in moist amended (i.e., alfalfa meal added) or unamended samples incubated under aerobic conditions, but rapid transformation (65–96% over 4 weeks) in amended and unamended samples incubated under anaerobic conditions. The transformation was reported to be a dechlorination reaction. No transformation was observed in autoclaved samples. A 50% loss of toxaphene in 6 weeks due to biodegradation in anaerobic, flooded soils was reported; however, no biodegradation was found in aerobic sediments (EPA 1979a).

There is conflicting information in the literature regarding the transformation of toxaphene in sediments. Seiber et al. (1979) found that in sediment samples taken from the bottom of a drainage ditch a year or more after application of toxaphene to an adjacent field (13.5 kg/ha), several major components of toxaphene, including toxicant B (congener p-32), were significantly degraded. Reductive dechlorination appeared to be a major mechanism of degradation. This mechanism results in lower weight products than occur in technical toxaphene, at least some of which are relatively stable in the environment. As a consequence, the authors emphasized that the environmental and toxicological significance of these products needs to be determined. Using a microcosm system, Williams and Bidleman (1978) reported that toxaphene transformation in an anaerobic salt marsh sediment was mediated chemically, rather than biologically. The transformation, believed to be a reductive dechlorination, was rapid, occurring within 2–6 days even in sterilized samples. In contrast, Mirsatari et al. (1987) found no transformation of toxaphene in autoclaved (i.e., sterile) sediment and soil samples over a 60-day test period. In addition, no transformation was observed in unsterile sediment samples incubated under aerobic conditions for 6 weeks. Rapid transformation (half-life 1 week) was observed only in unsterile sediment samples amended with organic matter and incubated under anaerobic conditions. The microbially mediated transformation was apparently a reductive dechlorination. Clark and Matsumura (1979) added radiolabeled toxaphene to sediments and incubated them for 30 days under aerobic and anaerobic conditions. As in the Mirsatari et al. (1987) study, no transformation was observed in autoclaved samples. However, toxaphene was transformed in the aerobically incubated samples by the bacterium, *Pseudomonas putida*. Clark and Matsumura (1979) stated that toxaphene biotransformation is likely to proceed initially as a dechlorination reaction under anaerobic conditions followed by oxidative transformation of the less chlorinated products under aerobic conditions. Thus, toxaphene apparently undergoes some biotransformation in the sediment layers of rivers and lakes under both anaerobic and aerobic conditions.

Lacayo-Romero et al. (2006) studied the degradation of toxaphene congeners in contaminated soils using anaerobic bioreactors. These authors reported that the congeners p-11 and p-12 were degraded while the concentration of p-15 increased, suggesting that the less chlorine substituent toxaphene congeners are formed during the degradation of the greater chlorine substituted congeners. Ruppe et al. (2004) identified 20 metabolites resulting from anerobic bacterial transformation of technical toxaphene in sediments and soils. The most recalcitrant of the toxaphene metabolites were 2-exo,3-endo, 6-exo,8,9,10-hexachlorobornane (B6-923) and 2-endo,3-exo,5-endo,6-exo,8,9,10-heptachlorobornane (B7-1001).

6.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to toxaphene depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of toxaphene in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on toxaphene levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. The analytical methods available for monitoring toxaphene in a variety of environmental media are detailed in Chapter 7.

As a result of its past widespread use as an insecticide and its persistence, toxaphene has been detected in ambient air, surface water and groundwater, soils and sediments, rainwater, and food. Data reported in this section have been obtained largely from national surveys in an attempt to present a representative national perspective of toxaphene contamination of various environmental media. However, toxaphene contamination of certain media may be a more serious problem on a regional basis than indicated by these national averages. For example, higher soil concentration levels can be expected in cotton growing areas of the South, and higher tissue residue levels have been found in fish taken from the Great Lakes.

A factor complicating the analysis of toxaphene in various environmental media is the difficulty in making trend comparisons for monitoring information collected before the early 1980s. Reliable detection of low levels of PCCs became possible only with the adoption of capillary column GC technology in the early 1980s. The prevailing earlier packed-column methods were usually unable to provide reliable total toxaphene readings for the large numbers of congeners (each present in minute amounts) encountered in most samples (Schmitt et al. 1990). For instance, U.S. Fish and Wildlife Service programs like the National Pesticide Monitoring Program (now the National Contaminant Biomonitoring Program or NCBP) started in the 1970s; however, due to problems in quantification with the older analytical technologies, results of these programs cannot be compared with toxaphene sampling results obtained since 1990 (Schmitt et al. 1990). These problems seriously interfere with drawing conclusions for such media as sediments or tissue samples, and make it almost impossible to make trend determinations for ambient water.

Another complicating factor is the mounting evidence that wastes from paper and pulp operations may be a source of toxaphene-like materials. Much of this research comes from countries where toxaphene was never used as a pesticide agent, but where anomalous findings of PCC materials were encountered. There

is a tendency in such cases to conclude that all of the PCC congeners are the result of hemispheric or global atmospheric transport pathways, but in some cases, PCC from paper and pulp wastes may help explain localized hotspots (Jarnuzi et al. 1992a, 1992b; Paasivirta and Rantio 1991; Rantio et al. 1993). Shanks et al. (1999) concluded that pulp and paper mills were not sources of toxaphene to Lake Superior or northern Lake Michigan at the time of the study based on similar concentrations measured in samples upstream from the mills compared with those measured in downstream samples.

Reliable evaluation of the potential for human exposure to toxaphene depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. In reviewing data on toxaphene levels monitored in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable. Also, analytical methods used in the past have been based on analysis of technical toxaphene and may not have detected some congeners that are expected to be present in the weathered form of toxaphene (EPA 2010a).

6.4.1 Air

Toxaphene has been detected in ambient air and rainwater samples collected at a number of sites in the United States; however, the available data are not current. No information was found in the available literature regarding ambient indoor exposure levels of toxaphene.

Toxaphene has also been detected in ambient air samples taken at remote locations. Toxaphene concentrations of <0.04–1.6 ng/m^3 in ambient air samples taken over the western North Atlantic Ocean from 1973 to 1974 have been reported (Bidleman and Olney 1975). Mean concentrations in ambient air samples from Bermuda were 0.81 ng/m^3 (±0.45 ng/m^3 standard deviation [SD]) and 0.72 ng/m^3 (±0.09 ng/m^3 SD).

In an ambient air monitoring study conducted at four urban sites (Baltimore, Maryland; Fresno, California; Riverside, California; and Salt Lake City, Utah) and at five rural sites (Buffalo, New York; Dothan, Alabama; Iowa City, Iowa; Orlando, Florida; and Stoneville, Mississippi) in the United States in 1967–1968, toxaphene was detected only in samples taken from the three agricultural areas in southern states. Maximum concentrations detected were 68 ng/m^3 (detected in 11 of 90 samples), 2,520 ng/m^3 (9 of 99 samples), and 1,340 ng/m^3 (55 of 98 samples) in Dothan, Alabama; Orlando, Florida; and Stoneville, Mississippi, respectively (Stanley et al. 1971). Toxaphene was included in the ambient air sampling of agricultural and urban areas conducted in 14–16 states as part of the National Air Pesticide

Monitoring Program. For the years 1970–1972, toxaphene was detected in 3.5% of the 2,479 samples collected at mean and maximum concentrations of 17 and 8,700 ng/m^3, respectively; the mean of the positive samples was 1,890 ng/m^3 (Kutz et al. 1976). In 1981, toxaphene was detected at maximum concentrations of 9.05, 1.73, 0.44, and 0.14 ng/m^3 in Greenville, Mississippi; Saint Louis, Missouri; Bridgeman, Michigan; and Beaver Island, Michigan, respectively (EPA 1984b; Rice et al. 1986).

Concentrations of chlorobornanes measured in air samples from Columbia, South Caroline during 1994–1995 ranged from 39 to 183 pg/m^3 (Bidleman et al. 1998). Air samples collected at a height of 40 cm above the soil at farms in Alabama, Louisiana, and Texas during June 1999 and June 2000 contained total toxaphene at concentrations ranging from 0.47 to 42.1 ng/m^3 (Bidleman and Leone 2004).

The average gas-phase concentrations of toxaphene were 1,600, 280, 34, and 10 pg/m^3 in air samples collected during 2000–2001 in Rohwer, Arkansas; Lubbock, Texas; Bloomington, Indiana; and Sleeping Bear Dunes, Michigan (Lake Michigan), respectively (James and Hites 2002). The average gas-phase concentrations of toxaphene were 61, 1,400, 60, and 23 pg/m^3 in air samples collected during 2002–2003 in Cocodrie, Louisiana; Rohwer, Arkansas; Bloomington, Indiana; and Sleeping Bear Dunes, Michigan (Lake Michigan), respectively (Hoh and Hites 2004). Based on these concentrations and analysis of air trajectories, the authors of these studies concluded that toxaphene detected in air from Indiana and the Great Lakes region originates in the southern United States (Hoh and Hites 2004; James and Hites 2002). Mean concentrations of total toxaphene and the congeners p-26 and p-50 measured in the air at locations over Lake Superior, Lake Huron, and Lake Erie were 28, 2.2, and 1.9 pg/L, respectively, in August 1996 and 12, 0.32, and 0.26 pg/L, respectively, in May 1997 (Jantunen and Bidleman 2003).

A seasonal variation in toxaphene concentrations in ambient air samples collected in Stoneville, Mississippi, from 1972 to 1974 was noted in a study by Arthur et al. (1976). The highest concentrations were observed in summer months, corresponding to the growing season, and the lowest in winter months. The sampling site was located in the middle of the most intensive cotton-growing area in Mississippi. The maximum concentration detected in weekly air samples was 1,747 ng/m^3. Average monthly levels were 258, 82, and 160 ng/m^3 for 1972, 1973, and 1974, respectively. A similar seasonal variation was found in atmospheric toxaphene concentrations in southern Ontario, which was attributed to increased volatilization of PCCs during the warmer summer months (Hoff et al. 1993b). During this 1988–1989 study, average monthly concentrations ranged from 0.08 pg/m^3 in February to 110 pg/m^3 in July; the overall maximum and mean concentrations (n=114) were 580 and 26 pg/m^3, respectively. Shoeib et al. (1999) measured total toxaphene concentrations ranging from 0.9 to 10.1 pg/m^3 in the air at Point Petre,

Ontario sampled during 1992 and from 1995 to 1997. The summer-to-winter concentration ratio was reported to be about 6. Glassmeyer et al. (1998) reported vapor-phase toxaphene concentrations ranging from 1.0 to 42 pg/m^3 measured in the air at Eagle Harbor, Michigan (Lake Superior) during 1996 and 1997.

Toxaphene has been identified in air samples collected at 3 of the 68 NPL hazardous waste sites where it was detected in some environmental media (HazDat 2007).

6.4.2 Water

Toxaphene has been detected very rarely in drinking water supplies. Toxaphene concentrations ranged from 5 to 410 ppt (0.005–0.410 ppb) in drinking water samples collected in Flint Creek, Alabama, between 1959 and 1963 (Faust and Suffet 1966). In an extensive water quality monitoring program conducted by the California Department of Health Services, toxaphene was detected (detection limit not specified) in only 2 of 5,279 public drinking water sources sampled from 1984 to 1992, at mean and maximum concentrations of 0.30 and 0.50 ppb, respectively (Storm 1994). Concentrations did not exceed the Maximum Contaminant Level (MCL) of 5.0 ppb.

The median toxaphene concentration detected in ambient surface waters in the United States in 1980–1982, according to analyses of EPA's STORET water quality database, was 0.05 ppb (Staples et al. 1985). The mixture was detected in 32% of the 7,325 samples collected over that period. Toxaphene was detected in only 3.4% of the 708 effluent samples taken during 1980–1983 at a median concentration of <0.2 ppb.

In a study of toxaphene concentrations in surface water and runoff from the Bear Creek, Mississippi, watershed conducted in 1976–1979, toxaphene concentrations in surface water were found to be measurable only after major runoff events (Cooper et al. 1987). At other times, only trace amounts of the compound (<0.01–1.07 ppb) were detected. However, runoff from two fields historically cultivated in cotton and soybeans contained toxaphene residues of 0.04–4.18 ppb and 289–2,964 ppm in the aqueous and particulate fractions, respectively. Petty et al. (1995) conducted studies using semipermeable membrane devices to determine bioavailable organochlorine pesticide residues in streams receiving irrigation drainwater from agricultural activity in the Lugert Altus Watershed in southwestern Oklahoma. Among the pesticides monitored, toxaphene was predominant, with calculated bioavailable (dissolved) water concentrations at six sampling sites ranging from 0.3 to 7 μg/L (ppb). In general, concentrations

were higher in summer than in spring. The authors noted that the Kow used in these calculations was an average for the toxaphene mixture and that, because Kow values for individual congeners may vary by an order of magnitude, water concentrations of toxaphene congeners could range from 0.9 to 9 ppb. There is an additional uncertainty in these estimates because they were derived from the dialysate data using models and preliminary data on uptake kinetics. The results do indicate, however, that significant concentrations of bioavailable toxaphene may still be present in this aquatic ecosystem several years after discontinuation of its use.

In contrast to agricultural areas, municipal areas do not show evidence of toxaphene in water samples. Toxaphene was not detected in 86 samples of municipal runoff collected from 15 cities in the United States in 1982 as part of the Nationwide Urban Runoff Program (Cole et al. 1984). Toxaphene was not detected (detection limits 0.06–0.2 ppb) in surface water samples collected in 1990–1993 from 13 sites in the Potomac River and Upper Chesapeake Bay areas (Hall et al. 1993, 1995). Sampling sites included both clean reference areas and suspected polluted areas.

Swackhamer et al. (1999) reported mean dissolved toxaphene concentrations of 1.12 ng/m^3 in Lake Superior surface water collected in 1996 and 0.38 ng/m^3 in Lake Michigan surface water collected in 1994–1995. Surface water concentrations were estimated to be <0.5 ng/m^3 in Lakes Huron, Erie, and Ontario. The higher levels in Lake Superior were attributed to colder temperatures (lower volatilization rate) and lower sedimentation rates (James et al. 2001; Swackhamer et al. 1999). In addition, Xia et al. (2011) used different fate models to suggest that higher toxaphene concentrations in Lake Superior are the result of differences in physical properties of the lake, such as large volume, large residence time, and cold temperatures, compared to the lower lakes. Measurements of mean toxaphene concentrations in surface water of the Great Lakes were reported as 718 pg/L in Lake Superior in 2002, 470 pg/L in Lake Huron in 1997, between 380 and 410 pg/L in Lake Michigan between 1994 and 1998, 230 and 96 pg/L in Lake Erie in 1993 and 1996, respectively, and 170 and 81 pg/L in Lake Ontario in 1993 and 2000, respectively (Xia et al. 2011). The mean concentration of total toxaphene and the congeners p-26 and p-50 measured in surface water collected at locations across Lake Superior in 1996 and 1997 were 918, 3.5, and 13 pg/L, respectively (Jantunen and Bidleman 2003).

Toxaphene has also been detected at hazardous waste sites in surface water, groundwater, and leachates. Toxaphene was detected at a maximum concentration of 17 ppb in surface water samples taken from two of nine disposal ponds at a Superfund site (EPA 1986). In a study of the chemical composition of leachates within existing landfills, toxaphene was not detected in any of the municipal landfill leachates

examined (Brown and Donnelly 1988). However, the mixture was detected in industrial landfill leachates at a concentration of ≤10 ppb. In a review of groundwater monitoring data collected in 1981–1984 from more than 500 wells at 334 hazardous waste disposal sites (RCRA and CERCLA sites) located in all 10 EPA regions and 42 states, Plumb (1987) reported that toxaphene was detected at 0.2% frequency at the 178 CERCLA sites examined and at 1.1% frequency at the 156 RCRA sites examined. Concentration data were not provided. Toxaphene has been identified in surface water and groundwater samples collected at 14 and 27 of the 68 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2007).

Toxaphene has been detected in rainwater samples taken in southern France near the Mediterranean Sea at mean concentrations of 7.2 ppt (range: not detected to 53 ppt) and 25.2 ppt (range: not detected to 81 ppt) in solution and sorbed to particulates, respectively (Villeneuve and Cattini 1986). Burniston et al. (2005) reported annual average toxaphene concentrations of 0.68–0.85 ng/L (38–47 ppt) measured in Lake Ontario precipitation samples collected continuously from November 1994 through December 1998. No additional information was found in the literature for concentrations of toxaphene in rainwater samples collected in the United States.

6.4.3 Sediment and Soil

Toxaphene has been detected in some samples of urban and agricultural soils from throughout the United States. Wiersma et al. (1972a) detected the mixture in concentrations that ranged from 0.11 to 52.7 ppm in samples of surface soils from three of eight U.S. cities in 1969. In another study of 14 cities conducted in 1970, toxaphene was detected at 3 of 28 sites (10.7%) at mean and geometric mean concentrations of 1.94 and 0.012 ppm, respectively; concentrations in the positive samples ranged from 7.73 to 33.4 ppm. In Sikeston, Missouri, toxaphene was detected at 1 of 27 sites at a concentration of 0.6 ppm. Carey et al. (1979a) monitored soils in five U.S. cities in 1971 and found toxaphene only in 11 of 43 samples (25.6%) taken from Macon, Georgia, at a mean concentration of 0.24 ppm (range, 0.23–4.95 ppm; geometric mean, 0.02 ppm). Toxaphene residues in domestic cropland soils were surveyed in the National Soils Monitoring Program (Carey et al. 1978, 1979b; Wiersma et al. 1972b). Toxaphene was found in 73 of 1,729 soil samples collected in 43 states during 1969 with a mean concentration of 0.07 ppm and a range of 0.10–11.72 ppm (Wiersma et al. 1972b). Toxaphene was found in 76 of 1,483 soil samples collected in 37 states during 1972 with a mean concentration of 0.24 ppm and a range of 0.22–46.58 ppm (Carey et al. 1979b).

Toxaphene was detected in 38 of 39 agricultural soil samples collected at locations across the state of Alabama (Harner et al. 1999). The geometric mean concentration of toxaphene in these samples was 84 ng/g dry weight and the maximum concentration was 2,423 ng/g dry weight. The concentrations of toxaphene measured in soil samples collected from 32 cotton fields in southern South Carolina and eastern Georgia ranged from 3.3 to 2,500 ng/g dry weight (Kannan et al. 2003). The median of the reported concentrations was 85.3 ng/g dry weight for the South Carolina soils and 67.25 for the Georgia soils. Soil samples collected at farms in Alabama, Louisiana, and Texas during June 1999 and June 2000 contained total toxaphene at concentrations ranging from 3.2 to 6,520 ng/g dry weight (Bidleman and Leone 2004).

Toxaphene levels were measured in soil samples collected during 2000–2001 from three schools and one field ballpark in Brunswick, Georgia (Agency for Toxic Substances and Disease Registry 2005). These sites are all located within 0.5 miles of the Hercules, Incorporated industrial facility, which manufactured toxaphene from the mid 1940s until 1982. Maximum toxaphene levels measured in the soil from the sampling locations were <0.010, 0.180, 0.030, and 0.380 ppm, respectively.

Rapaport and Eisenreich (1986) found toxaphene in samples of peat from bogs located in remote regions of the northern United States and Canada at concentrations ranging from <1 ppb (detection limit) to 30 ppb. Toxaphene was not detected (detection limit 0.5 ppm wet weight) in surface core samples (0–15 cm depth) of soils derived from dredged materials from nine confined disposal facilities in the Great Lakes region (Beyer and Stafford 1993).

Toxaphene has also been detected in sediment samples throughout the United States. Toxaphene was detected in 2.2% of 548 sediment samples collected in the lower Mississippi River and its tributaries in 1964 and from 1966 to 1967. Concentrations in the positive samples ranged from 0.1 to 13.18 ppm, the mean concentration was 6.5 ppm (Barthel et al. 1969). In southern Florida, toxaphene was detected, but not quantified, in 3.2% of 126 sediment samples collected from 1969 to 1972 (Mattraw 1975). Toxaphene was not detected in 27 sediment samples collected in Delaware and in the Raritan Canal, New Jersey, from 1979 to 1980 (Granstrom et al. 1984), or in sediment samples collected in Casco Bay, Maine, in 1991 (Kennicutt et al. 1994). At a site 1.4 miles from the outfall of a toxaphene plant on Terry Creek in Brunswick, Georgia, toxaphene was found at a concentration of 5.27 ppm in a 70–80-cm deep sediment sample collected in 1971 (IARC 1979). According to analyses of EPA's STORET water quality database, the median toxaphene concentration in sediment was 2.0 ppb; the compound was detected in 25% of the 1,603 samples taken during 1980–1983 (Staples et al. 1985).

During an investigation of organochlorine pesticides in soil sediments in the upper Steele Bayou watershed of Mississippi, toxaphene was found in 41% of 56 samples collected at two depths (2.54–7.62 and 25.4–30.48 cm) along eight different drainages (Ford and Hill 1991). The geometric mean and maximum wet weight toxaphene concentrations were 0.12 and 2.80 ppm for the shallow samples, and 0.07 and 4.60 ppm for the deeper samples, respectively. There was no significant difference in toxaphene concentrations between corresponding shallow and deep samples. Raff and Hites (2004) measured toxaphene levels ranging from 0.4 to 39 ng/g in suspended sediment samples collected from 32 locations along the Mississippi River during 2002–2003. The concentrations of toxaphene in the sediments were found to increase rapidly as the river passes through the cotton-growing regions of the southern United States. Studies in agricultural areas of the Mississippi Delta have provided indications of the persistence of toxaphene in soils and sediments under what might be construed as a worst case scenario. Results of investigations at Moon Lake and sites within its watershed just to the east of the main levees on the Mississippi River in Coahoma County, Mississippi, have been reported (Cooper 1991). In soils, which provide a generally aerobic redox environment, the average total toxaphene level based on 69 samples collected in the period 1983–1984 was 734 ppb. The toxaphene concentration in lake sediments averaged 12.4 ppb. In core samples from wetland flats displaying marked signs of anaerobic conditions, there was no detectable toxaphene. These findings underscore the fact that it is only in media providing appreciable residence times in biologically active anoxic conditions that one can expect significant biodegradation of toxaphene. In even moderately aerobic environments, and especially in soil or sediments rich in clay colloids, the pesticide agent is persistent for many years.

Shanks et al. (1999) reported toxaphene concentrations of 1.4–9.0 ng/g dry weight measured in sediment from rivers near pulp and paper mills near Lakes Michigan and Superior. These authors also measured toxaphene concentrations of 6.0–43 ng/g dry weight in sediments from rivers near sites where this pesticide was previously used. Maximum toxaphene concentrations measured in sediment cores collected from Lake Michigan, Lake Superior, and Lake Ontario during the early 1990s were 48, 42, and 29 ng/g, respectively (Pearson et al. 1997). Surficial accumulation rates of 0.097–1.01 ng/cm^2-year were determined (Pearson et al. 1997). Analysis of sediment cores showed that in most cases, toxaphene accumulations peaked in the early 1970s to early 1980s and then declined in following years (Pearson et al. 1997; Schneider et al. 2001). Howdeshell and Hites (1996) observed similar trends in eight Lake Ontario sediment cores collected in 1993 and cited contaminated flow from the Niagara River in addition to atmospheric deposition as sources of toxaphene in the lake. Analysis of sediment cores from two lakes in Canada that were treated with toxaphene during 1961–1962 revealed maximum toxaphene

concentrations of 500 and 1,602 ng/g dry weight at depths corresponding to the time of treatment (Miskimmin et al. 1995). Surface concentrations in these lakes were 53 and 112 ng/g dry weight. Toxaphene was not detected in untreated lake sediments. Toxaphene sediment concentrations from five Canadian lakes previously treated with this pesticide ranged from 2.6 to 110 µg/kg dry weight (Donald et al. 1998). Toxaphene was also detected at 0.2 µg/kg dry weight in an oligotrophic glacial fed lake that had no record of treatment.

Toxaphene has also been found in soils and sediments at hazardous waste disposal sites. Mirsatari et al. (1987) reported that toxaphene has been found as a contaminant at pesticide disposal sites at concentrations in soils or sediment approaching or exceeding 100 ppm. Toxaphene was also detected at a maximum concentration of 2,900 ppb (2.9 ppm) in sediment samples taken from two of nine disposal ponds at a Superfund site (EPA 1986). Toxaphene was found at concentrations ranging from 18 to 1,505 mg/kg (ppm) in pesticide contaminated soils at four other Superfund sites in Litchfield, Arizona; Albany, Georgia; Marrianna, Florida; and Malone, Florida (Troxler et al. 1993). More recently, toxaphene has been identified in soil and sediment samples collected at 40 and 22 of the 68 NPL hazardous waste sites, respectively, where it was detected in some environmental media (HazDat 2007).

6.4.4 Other Environmental Media

Several studies conducted to determine the levels of toxaphene in food indicate that this substance is found only infrequently in the U.S. food supply, generally at very low residue concentrations, which have decreased significantly since the restriction of its use in 1982 (EPA 1982a) and its total ban in 1990 (EPA 1990b). Except for fish and wild game animals from some areas of the United States (Agency for Toxic Substances and Disease Registry 2009; Ford and Hill 1990; Xia et al. 2009), the current U.S. food supply does not appear to contain levels of toxaphene that are of concern for human health.

Levels of toxaphene in food have been determined as part of the Food and Drug Administration's (FDA) Total Diet Studies. In a 1980–1982 survey of pesticides, toxaphene was detected in samples of food groups that comprised typical infant and toddler diets. Concentrations of 0.1–0.2 ppm (number positive samples, 3) and 0.7–0.12 ppm (number positive samples, 6) were found in the oils and fats food groups of infants' and toddlers' diets, respectively. The samples were collected in 13 U.S. cities. Toxaphene was not detected in drinking water or the other foods examined in the diet of either group. Other food groups examined included: whole milk; other dairy and dairy substitutes; meat, fish, and poultry; grain and cereal products; potatoes; vegetables; fruit and fruit juices; sugar and adjuncts; and beverages (Gartrell et

al. 1986a, 1986b). In a summary of data from 1985 to 1991 FDA Total Diet Studies on pesticide residues in infant foods and adult foods eaten by infants and children, toxaphene was found only in peanut butter at a maximum concentration of 0.16 ppm (number of positive samples, 27 of 27) (Yess et al. 1993).

Toxaphene was detected each year in regulatory monitoring of domestic and imported foods conducted by the FDA from 1988 to 1994 as part of its Pesticide Residue Monitoring Program (FDA 1989, 1990, 1991, 1992, 1993, 1994c, 1995). Concentrations were not reported; however, <1% of the surveillance samples had any pesticide residue levels that were above established tolerances. Toxaphene was also detected in the FDA Total Diet Studies in 1987, 1988, 1989, 1990, and 1991 (FDA 1988, 1990, 1991, 1992). From 1987 to 1990, it was listed among the most commonly found pesticides, with frequencies of detection of 1–2% (FDA 1988, 1989, 1990, 1991). Reports of 1992–1994 FDA Total Diet Studies indicated that the types of pesticide residues found and their frequencies of occurrence were consistent with those in previous years; however, there was no explicit statement that toxaphene was detected in the years 1992–1994 (FDA 1993, 1994c, 1995). Concentrations of toxaphene found in the FDA Total Diet Studies were not reported. However, in an overall summary for the 5-year period 1986–1991, average dietary intakes of toxaphene, in μg/kg body weight/day, for eight age/sex groups were reported to range from 0.0057 (25–30-year-old females) to 0.0224 (2-year-old children) (FDA 1993).

Overall, in 234 ready-to-eat foods tested 37 times each from 1982 to 1991 as part of the FDA Total Diet Studies, toxaphene was found 138 times at an average concentration of 0.04 μg/g (ppm) in 18 different foods: cantaloupe, raw carrots, boiled collards, corn chips, cucumbers, cooked frankfurters, dry-roasted peanuts, creamy peanut butter, dill pickles, cured ham, potato chips, radishes, boiled spinach, boiled summer squash, boiled winter squash, strawberries, tomato sauce, and cooked veal cutlet (KAN-DO Office and Pesticides Team 1995). Concentrations ranged from 0.0050 μg/g (ppm) (strawberries) to 0.12 μg/g (ppm) (dry-roasted peanuts). During the period 1989–1991, estimated toxaphene intakes were <0.01 μg/kg body weight/day for 6–11-month-old infants, 14–16-year-old males, and 60–65-year-old females, with a noticeable downward trend in all age categories (FDA 1990, 1991, 1992). (See Section 6.5 for more detailed information on estimated daily toxaphene intakes.) While progressive improvements in analytical technologies complicate comparisons of older values with more recent collections, the FDA Total Diet Studies clearly suggest that toxaphene residue levels in food and general population intake levels have fallen dramatically over the last decade.

Other studies further indicate that the occurrence of toxaphene in the U.S. food supply is very low. Toxaphene was not detected as a violative residue in a 1992–1993 statistically based FDA study of

pesticide residues in more than 3,000 samples of domestic and imported pears and tomatoes (Roy et al. 1995). A regional food basket study conducted in San Antonio, Texas, in the period from 1989 to 1991 screened 6,970 produce items for a suite of 111 pesticide analytes. Toxaphene was not detected in any produce items at levels above FDA violation thresholds (Schattenberg and Hsu 1992). A summary of results from the FOODCONTAM database (Minyard and Roberts 1991) for the period 1988–1989 showed no detectable toxaphene residues in food samples. This database involves 10 states that follow quality assurance/quality control (QA/QC) protocols consistent with those of such federal counterpart agencies as the USDA, EPA, and the FDA.

Toxaphene has been found in fish and shellfish in some areas of the United States at levels of concern for human health and, at present, there are fourteen fish consumption advisories in effect for this compound (see Section 6.7) (EPA 2010d).

Toxaphene is of particular concern as a major contaminant of Great Lakes fish. Xia et al. (2009) detected the toxaphene congeners, p-26, TMX-1, p-38, p-40, p-41, p-44, p-50, and p-62, in fish composites from Lake Michigan, Lake Superior, Lake Huron, Lake Ontario, and Lake Erie collected during 2004. Reported total toxaphene concentrations were 39 ng/g wet weight in Lake Erie walleye, 155 ng/g wet weight in Lake Huron lake trout, 243 ng/g wet weight in Lake Michigan lake trout, 113 ng/g wet weight in Lake Ontario lake trout, 398 ng/g in Lake Superior lake trout, and 846 ng/g wet weight in a Lake Superior lake trout Standard Reference Material labeled SRM 1946. Congeners p-26, p-50, and p-62 were reported to be the dominant peaks, together accounting for 2–44% of the amount of total toxaphene in the fish samples.

Swackhamer et al. (1998) measured toxaphene in plankton from Lake Michigan and fish from Lake Superior. Reported mean toxaphene concentrations were 51.3 ng/g dry weight in phytoplankton, 243 ng/g dry weight in zooplankton, 92.4 ng/g dry weight in mysis, 162 ng/g dry weight in bythotrephes, 411 ng/g dry weight in diporeia, 225 ng/g dry weight in sculpin, and 2,373 ng/g dry weight in lake trout. Mean total toxaphene concentrations of 92 and 198 ng/g wet weight were measured in bloater chub and alewife samples, respectively, collected from Grand Traverse Bay, Lake Michigan during 1997 and 1998 (Stapleton et al. 2002). Kucklick and Baker (1998) reported toxaphene concentrations of 99–210 ng/g wet weight in smelt, 560–720 ng/g wet weight in herring, 840–1,360 ng/g wet weight in bloater, 260–460 ng/g wet weight in sculpins, 21–40 ng/g in mysis, 110–180 ng/g wet weight in limnocatanus, 100 ng/g wet weight in amphipod, and 250–540 ng/g wet weight in lake trout collected from Lake Superior during the summer of 1994.

Whittle et al. (2000) reported toxaphene concentrations of 0.081–1.926 µg/g wet weight in lake trout, 1.024 µg/g wet weight in herring, 0.245–0.546 µg/g wet weight in sculpin, 0.016–0.291 µg/g wet weight in smelt, 0.049–0.139 µg/g wet weight in alewife, 0.029–0.197 µg/g wet weight in diporeia, 0.020–0.091 µg/g wet weight in mysis, and <0.015–0.062 µg/g wet weight in plankton collected from Lake Superior, Lake Huron, Lake Erie, and Lake Ontario. Results of this study are summarized in Table 6-2. Levels in Lake Superior samples were consistently higher than levels in samples from the other lakes. Henry et al. (1998) measured toxaphene in smallmouth bass collected from Fumee Lake in the Upper Peninsula of Michigan. Mean toxaphene concentrations were 137 ng/g wet weight in 0–20 cm length fish, 255 ng/g wet weight in 20–30 cm length fish, and 312 ng/g wet weight in >30 cm length fish.

Glassmeyer et al. (1997) measured toxaphene in lake trout, walleye, and smelt archival samples collected in 1982 and 1992/1994 from the Great Lakes. Reported 1982 toxaphene levels were 4.5–5.2 µg/g wet weight in lake trout, 0.25 µg/g wet weight in walleye, and 0.16–0.83 µg/g wet weight in smelt. Reported 1992/1994 levels were 0.54–6.7 µg/g wet weight in lake trout, 0.13 µg/g wet weight in walleye, and 0.059–0.16 µg/g wet weight in smelt. While concentrations in the Lake Superior samples were not significantly different between the 2 years, the results showed a decline in toxaphene concentrations in the fish from the other Great Lakes from 1982 to 1992.

Residues of toxaphene and other pesticides in fish were examined as part of the NCBP, formerly a part of the National Pesticide Monitoring Program conducted in 1984. Composite samples (n=321) of bottom-feeding and predatory fish were taken from 112 stations located along the major domestic rivers and in the Great Lakes. Toxaphene residues were detected in fish tissue samples collected at 69% of the stations. In earlier sampling periods, the percentages of stations where detectable residues were present were approximately 60% (1976–1977 and 1978–1979) and 88% (1980–1981). The maximum and geometric mean wet weight concentrations of the mixture in the 1984 samples were 8.2 and 0.14 ppm, respectively, the lowest values found in any NCBP sampling period. Maximum and geometric mean wet weight concentration data for earlier sampling periods were 12.7 and 0.34 ppm (1976–1977), 18.7 and 0.28 ppm (1978–1979), and 21.0 and 0.28 ppm (1980–1981), respectively (Schmitt et al. 1985, 1990).

Fillets of Great Lakes coho salmon collected from the five lakes in 1980 had mean concentrations of 0.19–1.53 ppm of "apparent toxaphene" (Clark et al. 1984). Lake trout collected from Lake Michigan have been found to contain residues of toxicant congeners A (p-42a and p-42b) and B (p-32) that were approximately one-tenth or less of the estimated total toxaphene residues (Gooch and Matsumura 1985,

1987). The percentages of toxicant A and toxicant B in the fish residues were, however, similar to those in the technical toxaphene, indicating that in the environment, the rates of degradation of these congeners are roughly the same as those of other toxaphene components.

Toxaphene concentrations in nearshore fish collected from the mouths of rivers and embayments around Lake Michigan in 1983 were determined in a study conducted by Camanzo et al. (1987). In 28 composite whole-fish samples collected from 14 sites, toxaphene was detected at a mean concentration of 0.04–3.46 ppm in samples of rock bass, northern pike, common carp, smallmouth bass, lake trout, bowfin, pumpkinseed, channel catfish, and largemouth bass. The investigators noted that bottom-feeding species (e.g., common carp, channel catfish) had higher residue levels than top predatory fish (e.g., northern pike), possibly as a result of the bottom-feeders being older, having more fat tissue, and living in proximity to contaminated sediments. Most of the residues differed from the GLC peaks for the toxaphene standard, indicating that some metabolism/transformation of the compound had taken place. In 1982, toxaphene (reported as a toxaphene-like compound) was detected (detection limit 1 mg/kg [ppm] wet weight) in all of 10 samples of lake trout collected in Lake Michigan (mean concentration 4.70.5 ppm), and in 9 of 10 samples of lake trout collected in Lake Superior (mean concentration 1.6±0.2 ppm) (Miller 1993). In this same study, toxaphene was detected in all of 10 samples of chinook salmon collected in Lake Michigan in 1982 (mean concentration 2.0±0.2 ppm), and in 4 of 8 samples of chinook salmon collected in Lake Michigan in 1983 (mean concentration 1.0±0.0 ppm). Fish fillet samples from 11 species of Great Lakes fish were found to have toxaphene levels ranging from not detected (detection limit 10 ppb [0.01 ppm] wet weight) in bass and bullhead to 936 ppb (0.936 ppm) wet weight in trout (Andrews et al. 1993; Newsome and Andrews 1993). The levels appeared to be species specific, with higher levels found in fish having higher fat content (trout, herring) than in fish having lower fat content (bass, bullhead, perch, pickerel, smelt, menominee).

Levels of toxaphene in fish to which consumers are actually exposed are dependent on the type of sample and the method of preparation, with higher concentrations generally found in the higher fat content skin-on fillets. Zabik et al. (1995a, 1995b) investigated the levels of pesticides in Great Lakes fish and the effects of processing and selected cooking methods on residue levels. Toxaphene was not detected (detection limit 0.050 ppm wet weight) in skin-on or skin-off fillets of carp from Lake Huron and Lake Michigan (Zabik et al. 1995a); however, in skin-on fillets of walleye and white bass from these lakes, concentrations ranged from not detected to 0.09 ppm (Zabik et al. 1995b). In chinook salmon, toxaphene was found in skin-on fillets at average concentrations of 0.41 and 0.34 ppm in Lake Huron and Lake Michigan, respectively; corresponding concentrations in skin-off fillets were 0.23 and 0.22 ppm (Zabik et

al. 1995a). Baking and charbroiling significantly reduced toxaphene concentrations in both skin-on and skin-off fillets of salmon (38–56% reduction), while canning skin-off fillets resulted in a 77% reduction of toxaphene concentration. Toxaphene was not found in any samples from Lake Erie (Zabik et al. 1995a, 1995b).

The mean concentrations of toxaphene measured in largemouth bass at five different locations in the Mobile River basin in Alabama ranged from 13 to 104 ng/g in 2004 (Hinck et al. 2009). Maruya and Lee (1998) reported toxaphene concentrations of 0.5–1 µg/g lipid in fish collected from the Turtle/Brunswick River Estuary near Brunswick, Georgia. In a national monitoring program measuring organochlorine chemical residues in piscivorous and bethivorous fish at 111 sites from 1995 to 2004 from large U.S. river basins, toxaphene was detected in 83 of 409 whole-body fish samples at a mean concentration of 0.03 µg/g wet weight (0.83 µg/g wet weight max) (Hinck et al. 2009). Toxaphene was found at maximum concentrations of 11 ppm in shellfish samples from California (4 positives in 85 samples) and 54 ppm in shellfish samples from Georgia (128 positives in 211 samples) in a National Pesticide Monitoring Program survey of estuarine molluscs conducted from 1965 to 1972, a period when toxaphene was heavily used (Butler 1973). Toxaphene was detected at concentrations <0.10 ppm wet weight in eggs, ovary, liver, and muscle tissue of three pallid sturgeon (*Scaphirnyncus albus*) samples from the Missouri River in North Dakota and Nebraska (Ruelle and Keenlyne 1993).

The concentrations of total toxaphene measured in 19 fish samples collected from different locations in the Yukon, Canada ranged from 42 to 242 ng/g with a mean of 107 ng/g (Chan and Yeboah 2000). The sum of the concentrations of the three congeners, p-26, p-50, and p-62, ranged from 10 to 55 ng/g. Donald et al. (1998) reported higher chlorobornane concentrations in fish (75.7–303 µg/kg wet weight) from untreated oligotrophic lakes at higher elevations than in fish (3.3–82 µg/kg wet weight) from treated trophic lakes at lower elevations in western Canada. Toxaphene concentrations of 1.1 ppm on a wet weight basis (24 ppm fat weight basis) in cod liver samples and 0.4–1.0 ppm wet weight basis (4.4–12 ppm fat weight basis) in herring fillets collected from the east coast of Canada were reported by Musial and Uthe (1983). Toxaphene was not detected in samples of deep sea scallops.

Egg yolk samples of loggerhead sea turtles collected in 2002 from 44 nests in North Carolina, eastern Florida, and western Florida contained total toxaphene concentration ranges of 0.238–8.95 (mean 3.22), 0.062–8.63 (mean 1.99), and <0.055–0.813 (mean 0.378) ng/g lipid, respectively (Alava et al. 2011).

Tuerk et al. (2005) reported total toxaphene concentrations of 13.0–10.7 µg/g wet mass measured in the blubber of Atlantic white-sided dolphins and 1.49–3.33 µg/g wet mass measured in the blubber of rough-toothed dolphins. The relative proportions of the toxaphene congeners, p-50, p-26, and p-62, in the blubber samples were approximately 50, 35, and 15%, respectively. Mean concentrations of total toxaphene were 11.7 and 1.03 µg/g lipid in the blubber of bottlenose dolphins from the Turtle/Brunswick River Estuary and the Savannah Area Estuary, respectively, along the coast of Georgia (Pulster et al. 2009). Fourteen toxaphene congeners were identified in the blubber samples. Congener p-42a, which is one of the most abundant congeners in technical toxaphene, was present in the highest concentrations (maximum of 3,950 µg/g lipid). Toxaphene congeners, p-25, p-40, p-50, Hx-Sed, and Hp-Sed, were frequently detected at concentrations ranging from 100 to 1,000 ng/g lipid.

Gouteux et al. (2003) measured toxaphene congeners in blubber samples of 26 male and 26 female beluga whales from the St. Lawrence Estuary. The mean concentrations of the toxaphene congeners p-26 and p-50 were 710 and 1,510 ng/g wet weight, respectively, in the males and 280 and 520 ng/g wet weight, respectively, in the females. Maximum concentrations of these congeners were 1,240 and 3,060 ng wet weight, respectively, in the males and 1,110 and 1,690 ng/g wet weight, respectively, in the females. The authors stated that on average, toxaphene concentrations decreased by a factor of two between 1988 and 1999. Gouteux et al. (2005) measured chlorobornanes in blubber samples from six seal species in the St. Lawrence marine ecosystem. Toxaphene congeners, p-26, p-40/41, p-44, p-50, and p-62, were all detected, with p-26 and p-50 comprising 50–80% of the total chlorobornanes in each sample. The mean concentrations of total chlorobornanes were 49 ng/g lipid weight in gray seals, 80 ng/g lipid weight in harbor seals, 18 ng/g lipid weight in ringed seals, 370 ng/g lipid weight in harp seals, and 680 ng/g lipid weight in hooded seals. Toxaphene was detected in all great blue heron egg samples collected from seven colonies along the St. Lawrence River in 2001 and 2002 at mean concentrations ranging from 20.2 to 159.1 ng/g wet weight. Major toxaphene congeners detected were octachlorobornane p-44 and the nonachlorobornane p-50 (Champoux et al. 2010).

Vetter et al. (2001) detected eight toxaphene congeners in the blubber of seals from the Baltic Sea, the North Sea, and the Antarctic. Congeners p-26, p-50, B8-1412, p-44, and p-62 were detected in the greatest concentrations, followed by B7-1453, p-40, and p-41. Total toxaphene concentrations ranged from 5 µg/kg wet weight in an Antarctic elephant seal to 1,457 µg/kg wet weight in a harp seal from the North Sea. Concentrations in three Weddell seals in Antarctica ranged from 161 to 489 µg/kg wet weight. Total toxaphene concentrations were 68–303 µg/kg in cod liver samples, 1,194 µg/kg in cod liver oil, and 4–98 µg/g in two penguins.

Alder et al. (1997) measured the levels of the three toxaphene congeners, p-26, p-50, and p-62, in >100 samples of fish species that are consumed in Germany. Reported mean concentrations for the sum of these congeners were 12.1 µg/kg wet weight in herring, 0.2 µg/kg wet weight in Alaska Pollock, 0.8 µg/kg wet weight in saithe, 15.1 µg/kg wet weight in redfish, 0.1 µg/kg wet weight in hake, 7.9 µg/kg wet weight in mackerel, 1.1 µg/kg wet weight in cod, 2.2 µg/kg wet weight in sardine, and 36.7 µg/kg wet weight in halibut. Mean concentrations of congeners p-26, p-50, and p-62 were 5.87, 8.70, and 1.59 µg/kg fresh weight, respectively, in salmon collected along the Swedish east coast of the Baltic Sea (Atuma et al. 2000).

Archived specimens of *Eurythenes gryllus*, a scavenging amphipod, collected from 2,075 to 4,250 m below the surface of the western and central Arctic Ocean during five expeditions between 1983 and 1998 contained toxaphene concentrations ranging from 1,530 to 154,000 ng/g lipid weight, showing the penetration of contaminants to the abyssal Arctic Ocean (Bidleman et al. 2013).

The chief regions where bioaccumulation or biomagnification in fish or wildlife might pose a serious public health concern are in high latitude areas outside the contiguous United States. Studies on marine mammals in eastern Canada (Muir et al. 1992) suggest risks to native Inuit groups that eat blubber or visceral tissues such as liver. While no comparable work has been done in Alaska, this is an area of the United States where there could be genuine concern for Native American Inuit groups that hunt and consume marine mammals.

Within the contiguous United States, there is concern for populations that regularly consume meat from omnivores or carnivores, such as raccoons. Studies reported in Ford and Hill (1990) on the Upper Steele Bayou near the Yazoo National Wildlife Refuge in Mississippi show wildlife still displaying toxaphene residues in adipose tissues in collections made in 1988. The residues were most pronounced for raccoons, where adipose concentrations of total toxaphene up to 31 ppm (weight mass basis) were observed. The Upper Steel Bayou region in Washington County was close to another area on the Big Sunflower River previously studied in 1980. Due to radical changes in the GC methods for analyzing toxaphene, researchers are hesitant to make quantitative comparisons (Ford and Hill 1990). Nevertheless, in the late 1970s, the U.S. Fish and Wildlife Service was concerned enough to issue advisories on human consumption of wildlife in the Mississippi Delta region. Many members of this region's rural subsistence-level population eat significant amounts of game meat, including raccoons.

Toxaphene was also reported to be a contaminant of tobacco crops and products. Gibson et al. (1974) reported that toxaphene was a sporadic contaminant of Kentucky Burley tobacco crops during the period 1963–1972. Toxaphene was detected in about 4% of the samples at maximum concentrations exceeding 100 ppm. Toxaphene was also detected in six brands of cigar tobacco sampled in 1972 at an average concentration of 0.92 ppm; four of the six samples had toxaphene concentrations of <0.5 ppm. McDonald and Hites (2003) measured the concentrations of toxaphene in 46 tree bark samples collected in the United States and Canada. Higher concentrations (>20 ng/g bark) were found in samples collected from the South and Southeastern United States, between 40 and 32 degrees latitude, where toxaphene was used heavily in the past. Two samples had toxaphene concentrations as high as 250 and 300 ng/g bark. Toxaphene concentrations generally ranged from 1 to 11 ng/g bark in samples collected at locations further north or south.

Toxaphene has also been found as a contaminant in anhydrous lanolin, which is used as a moisturizer in cosmetics and as a vehicle compound in pharmaceutical preparations (Heikes and Craun 1992). Toxaphene was detected (detection limit not reported) in 2 of 10 samples of anhydrous lanolin analyzed in 1989 at concentrations of 2.8 and 5.8 mg/kg (ppm), but not in any of 10 samples analyzed in 1991.

6.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Current human exposure to toxaphene in the United States appears to be very limited. Members of the general population may be exposed to low levels of the mixture through ingestion of contaminated foodstuffs and possibly through inhalation of ambient air (Kutz et al. 1991). Populations consuming large quantities of fish and shellfish potentially contaminated with toxaphene may be exposed to higher levels than the general public. Exposure to higher concentrations of toxaphene may also result from contact with contaminated media in the vicinity of waste disposal sites containing toxaphene-contaminated wastes. No information was found in the available literature regarding the size of the human population potentially exposed to toxaphene in the vicinity of hazardous waste sites.

Based on the toxaphene levels in their 1980–1982 food survey, the FDA estimated average dietary intakes, in μg/kg body weight/day of 0.080, 0.036, and 0.023 for infants, toddlers, and adults, respectively (Gartrell et al. 1986a, 1986b). However, actual intakes must be lower than the estimates because other reported average dietary intakes were based on the mean concentration of the positive samples. Toxaphene intakes, in μg/kg body weight/day, estimated for the total diet analyses were 0.0059, 0.0087, and 0.0046 in 1989 (FDA 1990); 0.0071, 0.0085, and 0.0093 in 1990 (FDA 1991); and 0.0033, 0.0059,

and 0.0024 in 1991 (FDA 1992) for 6–11-month-old infants, 14–16-year-old males, and 60–65-year-old females, respectively. An overall summary for the 5-year period 1986–1991 of average dietary intakes of toxaphene, in µg/kg body weight/day, by eight age/sex groups was reported: 6–11-month-old infants, 0.0071; 2-year-old children, 0.0224; 14–16-year-old females, 0.0062; 14–16-year-old males, 0.0089; 25–30-year-old females, 0.0057; 25–30-year-old males, 0.0067; 60–65-year-old females, 0.0078; and 60–65-year-old males, 0.0077 (FDA 1993; Gunderson 1995). These dietary intake estimates suggest a decreasing trend following the cancellation of most registered uses of toxaphene as an agricultural pesticide in the United States in 1982 (EPA 1982a) and a cancellation of all registered uses in 1990 (EPA 1990b).

Toxaphene has been detected at a concentration of 0.1 mg/kg on a milk fat basis in pooled human breast milk samples collected in Uppsala, Sweden (Vaz and Blomkvist 1985), and at an average concentration (n=16) of 2 mg/kg lipid weight in human breast milk samples from Nicaragua, where toxaphene is still being produced and used (de Boer and Wester 1993). Mean concentrations of total toxaphene and the toxaphene congeners p-26 and p-50 were 0.8, 0.4, and 0.6 ng/g fat, respectively, in 10 pools of human milk collected during 2002–2003 from 238 primiparous women living in Hong Kong and south China (Hedley et al. 2010). The toxaphene congener p-62 was not detected in any of the samples. Newsome and Ryan (1999) measured toxaphene levels in human milk samples collected from women living in northern and southern Canada. These authors found that toxaphene concentrations in the northern samples were approximately 10-fold higher than those measured in the southern samples and stated that this disparity may be due to differences in types of food consumed. Mean concentrations of total toxaphene, congener 26, and congener 50 were 6.03, 1.32, and 2.35 ng/g lipid, respectively, in samples collected across southern Canada in 1992 (n=58); 7.28, 1.32, and 1.15 ng/g lipid, respectively, in samples collected in the Great Lakes basin in 1992 (n=24); 12.1, 2.83, and 4.37 ng/g lipid, respectively, in samples collected across southern Canada in 1986 (n=30); and 67.7, 24.9, and 33.1 ng/g lipid, respectively, in samples collected in Keewatin, Northwest Territories in 1997 (n=12). Toxaphene was measured in pooled human milk samples collected from individuals living in sub-arctic and arctic locations in northwestern Russia (Polder et al. 2003). Concentrations of the toxaphene congeners p-26, p-50, and p-62 measured in these samples were 2.34–4.33, 3.70–5.75, and 1.32–1.67 µg/kg milk fat, respectively. Polder et al. (2008) reported a mean concentration of 11 ng/g lipid weight measured in 10 human milk samples collected during 2000–2001 from primipara mothers living in the town of Tromso in northern Norway. Skopp et al. (2002b) found the sum of congeners p-26, p-41, p-44, and p-50 to range from 7 to 24 µg/kg milk fat in breast milk samples from women in an area of northern Germany. Levels of toxaphene in human milk from U.S. populations are not available.

Barr et al. (2004) measured the levels of two toxaphene congeners, p-26 and p-50, in old serum pools originally collected in Atlanta, Georgia in 1987, Chicago, Illinois in 1992, and Cincinnati, Ohio in 1994. Reported concentrations in these samples were 14.3, 3.5, and 28.9 pg/mL, respectively, for p-26 and 10.5, 10.0, and 25.2 pg/mL, respectively, for p-50. Patel et al. (2004) measured toxaphene levels in pools of 108 serum samples collected from pregnant women in Barrow and Bethel, Alaska. p-26 and p-50 were detected in >50% of the samples with geometric mean concentrations of 1.10 and 1.61 ng/g lipid-weight, respectively.

When toxaphene was being manufactured and used as an insecticide, occupational exposure to toxaphene, particularly via the dermal and inhalation routes, may have been significant. Dermal exposures of 22.72 and 16.56 µg/hour were reported by Munn et al. (1985) for adults and youths, respectively, harvesting a toxaphene-treated onion crop in the Platte River Valley of Colorado in 1982. Any farmers, farm workers, or pesticide applicators who formerly used the mixture to control insects on livestock and crops may have been exposed to relatively high concentrations via these exposure routes.

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.

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 may be exposed to toxaphene by breathing contaminated air, drinking contaminated water, eating contaminated soil, or eating contaminated fish or animals. Children living near areas where toxaphene was used heavily or near hazardous waste sites contaminated with toxaphene may have higher exposure to this substance. Based on the maximum concentration (0.380 ppm) of toxaphene measured in

soil from school grounds and a park located near a former production facility in Brunswick, Georgia, an exposure dose of 0.000015 mg/kg/day was estimated for a child if exposure through pica is excluded (Agency for Toxic Substances and Disease Registry 2005). The estimated exposure dose for a child rose to 0.0006 mg/kg/day if exposure through pica was included. Both of these values were below the intermediate-duration oral MRL of 0.002 mg/kg/day derived for toxaphene.

Witt and Niessen (2000) measured levels of toxaphenes in the adipose tissue of 48 children living in Germany, Russia, and Kazakhstan. Median and maximum concentrations at the different sampling locations were 0.37–1.97 and 0.69–6.02 µg/kg, respectively, for congener p-26 and 0.65–2.36 and 1.22–6.12 µg/kg, respectively, for congener p-50. Levels of toxaphene measured in neonatal blood, cord blood, meconium fluid, or the blood or urine of children were not located.

Nursing infants may be at risk for potentially high exposure to toxaphene; however, no data on levels of toxaphene congeners in breast milk from U.S. women could be located in the available literature. There are several documented cases of toxaphene congeners in fats from human breast milk (Hedley et al. 2010; Mussalo-Rauhamaa et al. 1988; Newsome and Ryan 1999; Polder et al. 1998, 2003, 2008; Skopp et al. 2002b; Vaz and Blomkvist 1985). Toxaphene congeners were also found in the fats in human breast milk in Nicaragua, while toxaphene was still being produced and used (de Boer and Wester 1993). The high concentrations found, and the lack of correlation between the number of children a woman had and the toxaphene concentration in her breast milk, were cited as evidence that elimination of toxaphene via transfer to the infant was fully compensated for by a regular intake of toxaphene. Consequently, nursing infants of mothers who incur regular and potentially high exposures to toxaphene (e.g., from the consumption of contaminated fish or game) may be at a potentially high risk for exposure to toxaphene.

An additional subpopulation that could experience slightly higher levels of exposure are infants and young children who receive vitamin supplements from cod liver oil. This is of some concern in Europe where fish oil products may involve catches taken in polluted areas (Walter and Ballschmiter 1991). Oetjen and Karl (1998) measured levels of three toxaphene indicator congeners in fish oils from Europe ranging from 13 µg/kg fat in sand eel oil to 206 µg/kg fat in cod oil. While no recent literature was identified on fish oil products entering U.S. markets, studies conducted in the early 1980s did detect toxaphene residues in food products that would be part of typical toddler and infant diets (Gartrell et al. 1986a, 1986b). Cod liver samples taken from the east coast of Canada have also shown measurable concentrations of toxaphene (Musial and Uthe 1983).

6.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Members of the general population currently having potentially higher intakes of toxaphene include residents living near NPL sites and other hazardous waste sites contaminated with toxaphene; populations that consume large quantities of fish and shellfish from waterbodies where fish consumption advisories for toxaphene contamination are in effect; and Native American and subsistence hunter groups that consume large quantities of wild game animals in their diet. No information was found in the available literature regarding the size of these populations. The concentrations of toxaphene in all of the contaminated media to which these populations might be exposed have not been adequately characterized.

In September 2010, toxaphene was cited as the causative pollutant in three fish consumption advisories in Arizona (Gila River, Hassayampa River, and Salt River), two in Delaware (Army Creek and Army Pond), five in Georgia (Back River, Back River from Causeway to St. Simons Sound, Coastal Georgia, Middle and South Georgia, Terry And Dupree Creeks), one in Louisiana (Tensas River), two in Mississippi (Delta Region and Roebuck Lake), and one in Oklahoma (Bitter Creek) (EPA 2010d).

EPA has identified toxaphene as a target analyte and recommended that this chemical be monitored in fish and shellfish tissue samples collected as part of state toxics monitoring programs. Residue data obtained from these monitoring programs should be used by states to conduct risk assessments to determine the need for issuing fish and shellfish consumption advisories (EPA 2010d).

In much of the contiguous United States where toxaphene was once used as a pesticide agent, the incidence of toxaphene residues in freshwater fish appears to be declining. While changes in GC analysis technologies make it very hard to compare post-1980 records with analyses conducted in the 1970s, results from two sampling periods in the 1980s from the U.S. Fish and Wildlife Service NCBP show that the number of sites with detectable levels of total toxaphene in fish tissue samples dropped from 88% in 1980–1981 to 69% in samples collected in 1984 (Schmitt et al. 1990). There may still be the potential for localized contamination of fish in the vicinity of hazardous waste sites and in the Great Lakes.

As noted in Section 6.4.4, there could also be risks of high exposures for three U.S. subpopulations that consume large amounts of marine mammals or game animals. The first includes Native American groups in Alaska, although any quantification of the risks would have to be based on data collected from such groups as the Inuit in areas of Canada (Muir et al. 1992; Laird et al. 2013). Results of the International Polar Year Inuit Health Survey conducted in 2007-2008 measuring the body burden of persistent organic

pollutants in 2162 Inuit participants from 36 communities in Nunavut, Nunatsiavut, and the Inuvialuit Settlement Region in Canada showed that the mean blood plasma concentration of toxaphene was measured as 0.17 μg/L (range of 0.01-8.3 μg/L), and was higher than those in the Canadian general population. The second includes people such as recreational or subsistence hunters in rural areas of the Southeast where historically heavy use of toxaphene as a pesticide agent occurred. People in this area who eat large amounts of wild game animals, particularly such species as raccoons, could be at risk of higher exposures (Ford and Hill 1990). The third includes individuals who regularly consume sport fish caught from the Great Lakes (ATSDR 2009).

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 toxaphene is available. Where adequate information is not available, ATSDR, in conjunction with 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 toxaphene.

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. In general, physical and chemical properties of toxaphene have been sufficiently well characterized to permit estimation of its potential environmental fate (Bidleman et al. 1981; Budavari et al. 1989; EPA 1981; NIOSH/OSHA 1978; Worthing 1979). Since toxaphene is a complex mixture, the environmental fates of specific congeners in original product formulations will vary. Information on the physical and chemical properties of specific congeners is needed for more reliable prediction of environmental fate and transport processes for toxaphene mixtures. This information, in combination with additional information on the toxicities of toxaphene congeners and their degradation products, is necessary to permit more quantitative estimation of exposure risks and analysis of environmental exposures to toxaphene.

Production, Import/Export, Use, Release, and Disposal. According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit substance release and off-site transfer information to the EPA. The TRI, which contains this information for 2012, became available in November of 2013. This database is updated yearly and should provide a list of industrial production facilities and emissions.

Recent U.S. production data for toxaphene are not available; however, it is assumed that this substance is no longer being produced for use as a pesticide in the United State since all registered uses were canceled in 1990 (EPA 1990b; USDA 1995). The most recent estimate of U.S. production levels was in 1982, the year that EPA first restricted the use of toxaphene (EPA 1982a). Production levels that year were less than 2 million kg (EPA 1987a), substantially lower than in 1972 (21 million kg) when toxaphene was the most widely manufactured pesticide in the United States (Grayson 1981). The TRI lists facilities in Arizona, Idaho, South Carolina, and Texas that were involved in toxaphene production during 2012 (see Table 5.1) (TRI12 2013). No other information regarding recent production of toxaphene in the United States was found.

In other parts of the world, toxaphene use continues at very high levels (et al. 1989; Stern et al. 1993). Although reliable information on use levels outside western European countries is almost impossible to obtain, many researchers feel that global use levels are quite substantial (Lahaniatis et al. 1992; Stern et al. 1993). It has been estimated that total global usage of toxaphene from 1950 to 1993 exceeded 1.3 million tons (Voldner and Li 1993); however, this may be a significant underestimation (Swackhamer et al. 1993). Since toxaphene, once volatilized, can be transported atmospherically over very long distances, all terrestrial and aquatic ecosystems, including those in the United States, are still subject to low levels of exposure. Especially in terms of atmospheric inputs, the best available monitoring information shows no demonstrable downward trends (Bidleman et al. 1992). More reliable information on global usage and atmospheric emissions of toxaphene would be useful in estimating potential human exposures in the United States. Additional information on the amounts of PCCs released to the environment as by-products of the chlorinated pulp processes involving pine oils (pinene) (Rantio et al. 1993; Swackhamer et al. 1993) would also be useful in developing estimates of global production and emissions for toxaphene.

The TRI lists four states containing facilities that were involved with the import of toxaphene into the United States during 2012 (TRI12 2013) for industrial applications. Export of toxaphene to foreign

nations for use as a pesticide is not expected since nations around the globe have adopted similar bans under the Stockholm Convention. No other information was found regarding the import of toxaphene into or the export of toxaphene from the United States.

In 1982, the use of toxaphene was restricted by EPA to its use as a pesticide on livestock; to control grasshopper and army worm infestation on cotton, corn, and small grains (in emergency situations only); and on banana and pineapple crops in Puerto Rico and the Virgin Islands (EPA 1982a). After July 1990, the pesticide registrations for all toxaphene formulations were canceled in the United States and in all U.S. territories (EPA 1990b). Because of its historic use as a pesticide, toxaphene has been widely distributed in the air, soil, surface water and sediments, aquatic organisms, and foodstuffs. Information on the current distributional patterns, which may involve localized hotspots, would be helpful in estimating human exposure.

Incineration in a pesticide incinerator is the preferred method of disposal for toxaphene (EPA 1989). Additional information on the amount of toxaphene disposed of by this method, as well as the amount of toxaphene disposed of or abandoned at hazardous waste sites, would be helpful for estimating the potential for human exposure.

Environmental Fate. Information on the environmental fate of toxaphene congeners (as a chemical group) is only sufficient to permit a general understanding of the partitioning and widespread transport, of toxaphene mixtures in the environment. The composition of toxaphene mixtures varies among producers (Walter and Ballschmiter 1991; Worthing and Walker 1987), and only limited data are available on the transport and transformation of individual toxaphene congeners in these mixtures. Additional information on the identity, physical/chemical properties, and environmental fate of toxic fractions of toxaphene mixtures would be useful. However, the sampling and analytical methodology limitations that have contributed to the lack of availability of this type of data in the past have not been completely overcome (Andrews et al. 1993; Bidleman et al. 1993; Bruns and Birkholz 1993; de Boer and Wester 1993; EPA 2010a; Lamb et al. 2008; Muir and de Boer 1995; Vetter et al. 1993; Zhu et al. 1994). Therefore, the development of this information may be difficult. More information on the rates of biotransformation and abiotic reduction of toxaphene in soils and sediments under anaerobic conditions would improve the current understanding of toxaphene's environmental fate. The role of biotic transformations in aerobic environments following initial reductive dechlorination needs to be clarified. Toxaphene metabolites such as Hp-Sed and Hx-Sed have been identified (Buser et al. 2000; EPA 2010a). Further information

regarding the identity, toxicity, and environmental fate of the major toxaphene transformation products will be useful in making a more critical assessment of potential human exposure.

Bioavailability from Environmental Media. Animal studies and case reports of human exposure indicate that toxaphene is absorbed following inhalation, oral, and dermal exposure (Kutz et al. 1991; Munn et al. 1985). Pharmacokinetics data indicate that toxaphene present in water or food is extensively absorbed; however, the degree to which toxaphene is absorbed as a result of inhalation of contaminated air or dermal contact with contaminated environmental media has not been well studied. The high K_{oc} for toxaphene indicates that it is adsorbed relatively strongly to soil, but it is not possible to estimate the extent to which toxaphene present on ingested soil would be absorbed from the gastrointestinal tract. Toxaphene is not expected to be available to humans via ingestion of plants unless they have been recently treated with the mixture. Since all registered uses of toxaphene as a pesticide were canceled in the United States and U.S. Territories in July 1990, ingestion of domestically grown agricultural commodities should no longer be a source for toxaphene. More information on the extent of absorption of components of the mixture following contact with contaminated air, water, or soil would be helpful in determining the potential health effects resulting from human exposure.

Food Chain Bioaccumulation. Laboratory bioassay and field monitoring data clearly indicate that toxaphene components are bioconcentrated by aquatic organisms. Available model ecosystem and field monitoring studies of aquatic food chains are sufficient to indicate that toxaphene bioaccumulates in aquatic organisms (Lowe et al. 1971; Sanborn et al. 1976; Schimmel et al. 1977; Swackhamer and Hites 1988; Whittle et al. 2000). However, as the result of metabolism, toxaphene is not biomagnified to the same degree as other chlorinated compounds, such as DDT and PCBs (Evans et al. 1991; Ford and Hill 1991; Niethammer et al. 1984; Stapleton et al. 2001). While several studies show that toxaphene is biomagnified in some ecosystems, several other studies show that little or no biomagnification of toxaphene occurs in other ecosystems because of effective metabolism of toxaphene by higher trophic level mammalian species (Andersson et al. 1988; Muir et al. 1988a, 1988b, 1992). Further congener-specific information on the bioaccumulation and biomagnification potential of toxaphene in both terrestrial and aquatic food chains might help to resolve differences observed in different ecosystems. These data will be helpful in assessing the potential for human exposure as a result of ingestion of contaminated food.

Exposure Levels in Environmental Media. Reliable monitoring data for the levels of toxaphene in contaminated media at hazardous waste sites are needed so that the information obtained on levels of

toxaphene in the environment can be used in combination with the known body burden of toxaphene to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Although a large amount of monitoring data is available for toxaphene, most of the data were collected 20–30 years ago when the mixture was widely used as a pesticide (Cole et al. 1984; Cooper et al. 1987; EPA 1984b; Faust and Suffet 1966; Kutz et al. 1976; Plumb 1987; Stanley et al. 1971; Staples et al. 1985). Some recent monitoring data are available for air (Bidleman and Leone 2004; Hoh and Hites 2004; James and Hites 2002; Jantunen and Bidleman 2003), surface water (Jantunen and Bidleman 2003), soil (Agency for Toxic Substances and Disease Registry 2005; Bidleman and Leone 2004; Harner et al. 1999; Kannan et al. 2003), and sediment (Raff and Hites 2004; Schneider et al. 2001). Additional information on current levels in environmental media would be helpful in characterizing current concentrations to which humans could be exposed. This is particularly important for concentrations of toxaphene in air, soils, and surface waters in the vicinity of hazardous waste sites. The data currently available are too limited to be useful in estimating the exposure of populations coming into contact with the mixture through inhalation of contaminated air, consumption of contaminated surface water, groundwater, or foodstuffs, and/or contact with contaminated soil. Reliable information is needed on current exposure levels in all environmental matrices and food sources (fish, shellfish, and terrestrial wildlife) in the vicinity of hazardous waste sites. Additional biomonitoring studies of both aquatic and terrestrial wildlife populations near hazardous waste sites, near water bodies where fish consumption advisories are currently in place (EPA 2010d), and in areas where toxaphene was historically used in agriculture applications (Ford and Hill 1991) are needed. This information on levels of toxaphene in the environment would be useful in assessing the potential risk of adverse health effects in populations living in these areas.

Exposure Levels in Humans. Exposure levels for the populations with either short- or long-term contact with hazardous waste sites are unknown. These levels currently cannot be estimated because of the lack of toxaphene concentration data for contaminated media in the vicinity of hazardous waste sites. Exposure of the general population has been estimated from levels in foodstuffs (FDA 1990, 1991, 1992, 1993). Estimates of average dietary intakes for several age/sex categories are based on data obtained subsequent to the restriction of most uses of toxaphene in 1982 (EPA 1982a) and appear to be adequate. Inhalation is not expected to be a major exposure route for the general public; consequently, additional data are not necessary. Pharmacokinetic data indicate that toxaphene rapidly redistributes to body fat and toxaphene has been identified in human breast milk fat from non-U.S. nursing mothers (de Boer and

Wester 1993; Hedley et al. 2010; Mussalo-Rauhamaa et al. 1988; Newsome and Ryan 1999; Polder et al. 2008; Vaz and Blomkvist 1985). Levels of toxaphene have been measured in serum (Barr et al. 2004; Patel et al. 2004). Tissue levels have not been obtained from persons exposed to toxaphene as a result of contact with a hazardous waste site. This information would be useful in assessing the risk to human health for populations living in the vicinity of hazardous waste sites.

This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. Limited data are available regarding the exposures of children to toxaphene. Agency for Toxic Substances and Disease Registry (2005) assessed the potential for toxaphene exposure of children attending school near a former production facility. The estimated exposure dose for these children was calculated as 0.000015–0.0006 mg/kg/day. A few foreign studies are available that report toxaphene levels measured in human milk and adipose tissue of children (Hedley et al. 2010; Mussalo-Rauhamaa et al. 1988; Newsome and Ryan 1999; Polder et al. 2003, 2008; Vaz and Blomkvist 1985; Witt and Niessen 2000). Levels of toxaphene in human milk, amniotic fluid, meconium, umbilical cord blood, neonatal blood, childhood serum, or childhood adipose tissue of individuals living in the United States were not located.

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 toxaphene were located. This substance is not currently one of the compounds for which a sub-registry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for sub-registries 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 exposure to this substance.

6.8.2 Ongoing Studies

No ongoing studies were located regarding the potential for human exposure to toxaphene.