5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

Chlorinated dioxins (CDDs) are a family of compounds that includes some extremely toxic and potent congeners. The two most toxic of the CDDs in mammals are 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD (Buser 1987; Poland and Knutson 1982; Safe 1986; WHO 1997). In general, the more toxic congeners to mammals appear to be the 2,3,7,8-substituted tetra-, penta-, and hexachloro- compounds, (e.g., 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,4,7,8-HxCDD, 1,2,3,6,7,8-HxCDD, and 1,2,3,7,8,9-HxCDD) (Poland and Knutson 1982; Safe 1986; WHO 1997). A more detailed discussion of the relative toxicities of the different CDD congeners is given in Section 2.5, Relevance to Public Health.

CDDs usually occur in the environment concurrently with other chemicals such as chlorinated dibenzo-furans (CDFs). CDDs and CDFs are highly persistent compounds and have been detected in air, water, soil, sediments, animals, and foods. CDFs include 135 congeners, which are structurally similar to CDDs and which elicit a number of similar toxicological and biochemical responses in animals (for more information on CDFs see ATSDR 1994). CDDs and CDFs are released to the environment during combustion processes (e.g., municipal solid waste, medical waste, and industrial hazardous waste incineration, and fossil fuel and wood combustion); during the production, use, and disposal of certain chemicals (e.g., PCBs, chlorinated benzenes, chlorinated pesticides); during the production of bleached pulp by pulp and paper mills; and during the production and recycling of several metals (Buser et al. 1985; Czuczwa and Hites 1986a, 1986b; Oehme et al. 1987, 1989; Zook and Rappe 1994). The EPA has developed procedures for estimating risks associated with exposures to mixtures of CDDs and CDFs in environmental matrices (EPA 1989e). This approach is based on the assignment of 2,3,7,8-TCDD toxic equivalence factors (TEFs) to CDD/CDF congeners or homologues in complex mixtures. The rationale behind the use of TEFs is explained in Section 2.5, Relevance to Public Health. **Although the focus of this profile is CDDs, it should be recognized that most exposure scenarios involve exposure to CDDs, CDFs, and the non-ortho polychlorinated biphenyls (PCBs) that have CDD-like toxicity; many of these exposure scenarios are discussed in this chapter.** These exposures are usually reported in TEQs (for more information see Section 2.5, Relevance to Public Health, Toxic Equivalency Factors [TEFs] and Toxic Equivalents [TEQs]). Over the past several years sets of TEFs have been

developed, varying slightly from one to another. The reader is encouraged to consult the original literature for specific details on TEQs computation.

CDDs (TCDD, PeCDD, HxCDD, HpCDD, OCDD) are ubiquitous in the environment (Podoll et al. 1986). Although all of the sources or processes that contribute to CDDs in the environment have not been identified, CDDs are known to be formed in the manufacture of chlorinated intermediates and pesticides, during smelting of metals (EPA 1998j), in the incineration of municipal, medical, and industrial wastes (Podoll et al. 1986), and from the production of bleached wood pulp and paper (Fletcher and McKay 1993). CDDs are also found in emissions from the combustion of various other sources, including coal-fired or oil-fired power plants, wood burning, and home heating systems (Chiu et al. 1983; Czuczwa and Hites 1984; EPA 1998j; Gizzi et al. 1982; Thoma 1988). Generally, the more highly chlorinated CDDs are the most abundant congeners present in the emissions from these combustion sources. CDDs also occur in other combustion products (e.g., cigarette smoke) (Bumb et al. 1980; Lofroth and Zebuhr 1992; Muto and Takizawa 1989), automobile exhaust from cars running on leaded gasoline with chlorine scavengers and to a lesser extent from cars running on unleaded gasoline (Bingham et al. 1989; Marklund et al. 1987, 1990), and diesel exhaust (Jones 1995; Cirnies-Ross et al. 1996). CDDs/CDFs can form during the synthesis and combustion of chlorine-containing materials, such as polyvinylchloride (PVC), in the presence of naturally occurring phenols, vegetation treated with phenoxy acetic acid herbicides, paper and wood treated with chlorophenols, and pesticide-treated wastes (Arthur and Frea 1989).

CDDs occur as contaminants in the manufacture of various pesticides and, as a result, have been released to the environment during use of these pesticides. 2,3,7,8-TCDD is a by-product formed in the manufacture of 2,4,5-trichlorophenol (2,4,5-TCP) (Arthur and Frea 1989). 2,4,5-TCP was used to produce the bactericide, hexachlorophene, and the chlorophenoxyherbicide, 2,4,5-trichlorophenoxy acid (2,4,5-T). Trichlorophenol-based herbicides have been used extensively for weed control on crops, rangelands, roadways, right-of-ways, etc. Various formulations of 2,4-dichlorophenoxy acetic acid (2,4-D) contaminated mainly with higher chlorinated CDDs/CDFs and 2,4,5-T contaminated mainly with 2,3,7,8-TCDD were used extensively for defoliation and crop destruction by the American military during the Vietnam War. Although six herbicides were used (Orange, Purple, Pink, Green, White, and Blue), herbicide Orange (Agent Orange) was the primary defoliant (Wolf et al. 1985). Hexachlorophene use has been restricted by the FDA and its disposal is regulated by EPA under the Resource Conservation and Recovery Act (RCRA). In 1983, EPA canceled registration for all chlorophenoxy herbicides used on foods, rice paddies, pastures, and rangelands (IARC 1986b). 2,4,5-T can no longer be used legally in the

United States for any purpose (IARC 1986b). Other countries, including Canada, Sweden, the Netherlands, Australia, Italy, and the Federal Republic of Germany, have also canceled registrations for 2,4,5-T (IARC 1986b), but many other countries have not. Currently, 2,4,5-T can be produced with lower 2,3,7,8-TCDD concentrations than were previously possible. 2,4,5-TCP production has been discontinued in many countries, including the United States, Canada, the United Kingdom, the Federal Republic of Germany, and Austria (IARC 1986a). HxCDD, HpCDD, and OCDD are known contaminants of pentachlorophenol (PCP), primarily a wood preservative and pesticide, which was used extensively in the 1970s and is still used today (to a lesser extent) in the lumber industry. PCP is currently registered as a restricted-use pesticide in the United States (Sine 1990).

Although little definitive data exist to prove or disprove that CDDs form during natural processes, results from dated sediment cores have shown that there were significant increases in CDDs and CDFs after about 1940 (Czuczwa and Hites 1984, 1986b, 1986b) and lower levels of CDDs are currently found in persons from less industrialized countries (Schecter et al.1991a). The congener/homologue profile of the sediments was similar to that of atmospheric samples, strongly suggesting that combustion processes were the source of CDDs in the sediments. The historical increase in CDDs/CDFs also was similar to the trends for the production, use, and disposal of chlorinated organics, suggesting that accumulation of these compounds in the environment is a recent phenomenon related to the production, use, and subsequent incineration of chlorinated organic chemicals (Schecter et al. 1988).

CDDs are ubiquitous in the environment and are found at low background levels (parts per trillion [ppt] or parts per quadrillion [ppq]) in the air, water, and soil. Lower levels are found in biological and environmental samples from less industrialized rural regions than in those from more industrialized urban regions (Czuczwa and Hites 1986a; Des Rosiers 1987; Edgerton et al. 1989; Schecter et al. 1989e, 1989g, 1991a, 1994d; Tiernan et al. 1989b). HpCDD and OCDD are the most common CDDs found in environmental samples (Christmann et al. 1989b; Clement et al. 1985, 1989; Pereira et al. 1985; Reed et al. 1990; Tashiro et al. 1989a; Tiernan et al. 1989b).

The environmental fate and transport of CDDs involve volatilization, long-range transport, wet and dry deposition, photolysis, bioaccumulation, and biodegradation (Kieatiwong et al. 1990). CDDs strongly partition to soils and sediments. Due to their low vapor pressure and low aqueous solubility and their strong sorption to particulates, CDDs are generally immobile in soils and sediments. Although most biological and nonbiological transformation processes are slow, photolysis has been shown to be relatively

rapid. Photolysis is probably the most important transformation process in environmental systems into which sunlight can penetrate (Kieatiwong et al. 1990). Estimates of the half-life of 2,3,7,8-TCDD on the soil surface range from 9 to 15 years, whereas the half-life in subsurface soil may range from 25 to 100 years (Paustenbach et al. 1992). CDDs have been shown to bioaccumulate in both aquatic and terrestrial biota. CDDs have a high affinity for lipids and, thus, will bioaccumulate to a greater extent in organisms with a high fat content.

Over the past decade, typical concentrations of CDDs in urban air in the United States have averaged 2.3 pg/m^3, with OCDD and HpCDD homologues predominating and 2,3,7,8-TCDD being the least common congener (Smith et al. 1992). CDD concentrations range as follows: OCDD, 0.44–3.16 pg/m^3; HpCDD, 0.21–4.4 pg/m^3; HxCDD, 0.6–0.63 pg/m^3; PeCDD, not detected to 0.1 pg/m^3; and 2,3,7,8-TCDD, <0.04–0.18 pg/m^3 (Edgerton et al. 1989; Eitzer and Hites 1989a, 1989b; Hunt and Maisel 1992; Smith et al. 1992). Although 2,3,7,8-TCDD has been detected in some urban air, it is rarely detected in rural areas (Reed et al. 1990). Ambient air concentrations of 2,3,7,8-TCDD detected in the vicinity of a Superfund clean-up site were on the order of 1 pg/m^3 (Fairless et al. 1987). CDDs have been detected almost exclusively in raw surface waters, rather than in finished drinking water (Jobb et al. 1990). This is not unexpected because CDDs are hydrophobic, and the compounds tend to be adsorbed onto particulate matter in the water column. Conventional water treatment processes appear to be effective in removing the CDDs along with the particulates (Jobb et al. 1990; Meyer et al. 1989). OCDD is the congener most often detected in water supplies at concentrations ranging from 9 to 175 ppq (raw water) and from 19 to 46 ppq (finished water). 2,3,7,8-TCDD concentrations have not been detected in finished drinking water, but were detected in one raw sample at a concentration of 1.7 ppq (Meyer et al. 1989). Concentrations of 2,3,7,8-TCDD in most soils are <12 ppt (Des Rosiers 1987; Nestrick et al. 1986); however, levels in contaminated soils can be several orders of magnitude higher (1,750 ppb) (Tiernan et al. 1985). 2,3,7,8-TCDD and other CDDs, have also been detected in measurable amounts in the sediments of industrialized waterbodies throughout the United States (Bopp et al. 1991; Wenning and Erickson 1994; Wenning et al. 1992, 1993a, 1993b).

In the National Study of Chemical Residues in Fish conducted by the EPA between 1986 and 1989, four CDDs (2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD, and 1,2,3,4,6,7,8-HpCDD) were detected at over 50% (54 to 89%) of 388 sites surveyed nationwide (EPA 1992). The most frequently detected CDD compound (1,2,3,4,6,7,8,-HpCDD) was found in fish tissues at 89% of the sites. This compound was also detected at the highest concentrations of 249 ppt (mean 10.52 ppt) wet weight. 2,3,7,8-TCDD

and 1,2,3,7,8-PeCDD, the CDDs currently believed to be most toxic to vertebrates (WHO 1997), were found in fish tissue at 70% and 54% of the sites, respectively. 2,3,7,8-TCDD was found at a mean concentration of 6.9 ppt and a maximum concentration of 204 ppt, and 1,2,3,7,8-PeCDD was found at a mean concentration of 2.38 ppt and a maximum concentration of 54 ppt. With respect to source categories, fish collected near pulp and paper mills using chlorine had the highest median 2,3,7,8-TCDD concentration (5.66 ppt), compared to the second highest median 2,3,7,8-TCDD concentrations of 1.82 ppt at refinery/other industrial sites, and the third highest median 2,3,7,8- TCDD concentration of 1.27 ppt at Superfund sites. Similarly, with respect to source categories, fish collected near pulp and paper mills using chlorine had the highest median 1,2,3,7,8-PeCDD concentration (1.52 ppt), compared to the second highest median concentrations of 1.35 ppt at refinery/other industrial sites, and the third highest median concentration of 1.09 ppt at industrial/urban sites.

The detection of CDDs in blood, adipose tissue, breast milk, and other tissue samples from the general population indicates universal exposure to CDDs from environmental sources (Fürst et al. 1994; Orban et al. 1994; Patterson et al. 1986a; Ryan et al. 1986, 1993a; Schecter and Gasiewicz 1987a, 1987b; Schecter et al. 1986b, 1989e; Stanley 1986; Stanley et al. 1986). The general population is exposed to CDDs released from industrial and municipal incineration processes; exhausts from automobiles using leaded gasoline; cigarette smoke; and foods, including human milk (Pohl and Hibbs 1996; Schecter et al. 1994e). The major source (>90%) of exposure for the general population, however, is primarily associated with meat, dairy products, and fish (Beck et al. 1989a; Schaum et al. 1994; Schecter et al. 1994d, 1994e, 1996a). CDDs are transferred through the placenta to the fetus, by breast milk to infants and young children, and by lifelong dietary ingestion. Workers involved with incineration operations or those who have been or may be involved in the production, use, or disposal of trichlorophenol, phenoxyherbicides, hexachlorophene, pentachlorophenol and other compounds that contain impurities of CDDs are at a greater risk from exposure to CDDs and TEQs (Päpke et al. 1992; Schecter and Ryan 1988; Schecter et al. 1991). Individuals in the general population who may be exposed to potentially higher levels of CDDs include recreational and subsistence fishers (including many native Americans) and their families living in CDD-contaminated areas who consume large quantities of fish from contaminated waters (CRITFC 1994; Ebert et al. 1996), subsistence hunters such as the Inuit of Alaska who consume large quantities of wild game (particularly marine mammals) (Dewailly et al. 1993; Hebert et al. 1996; Norstrom et al. 1990), subsistence farmers and their families living in areas contaminated with CDDs who consume their own farm-raised beef and dairy products (EPA 1996b; McLachlan et al. 1994), individuals who live in the vicinity of an industrial or municipal incinerator, or individuals who live in the vicinity of the

126 hazardous waste sites where CDDs (and more especially where 2,3,7,8-substituted CDDs) have been detected (Gough 1991; Liem et al. 1991; Pohl et al. 1995; Riss et al. 1990; Wuthe et al. 1993).

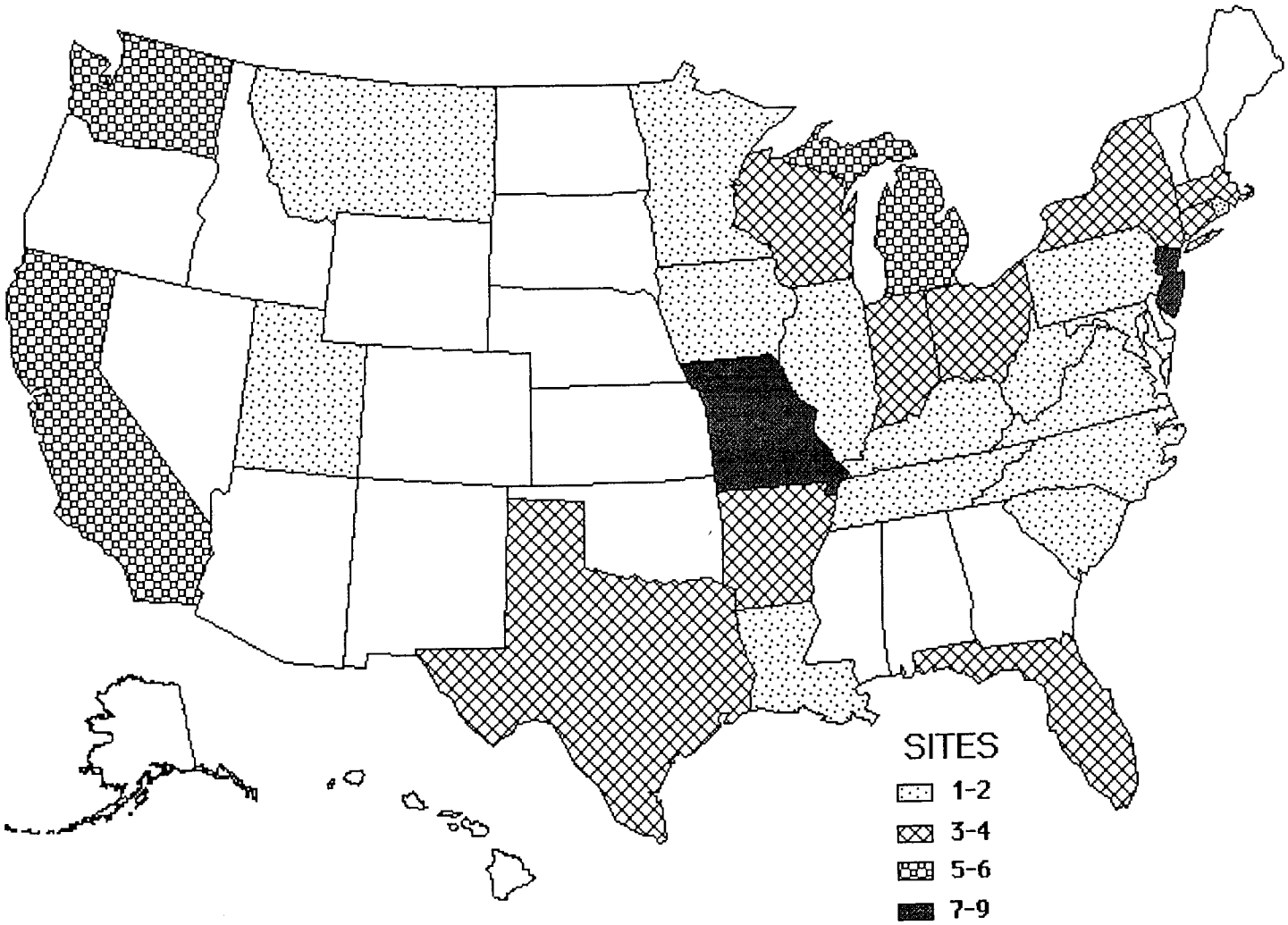
2,3,7,8-TCDD has been identified in at least 91 of 1,467 current or former EPA National Priorities List (NPL) hazardous waste sites (HazDat 1998). However, the number of sites evaluated for 2,3,7,8,-TCDD is not known. The frequency of these sites within the United States can be seen in Figure 5-1. Of these sites, 90 are located in the United States and 1 is located in the Commonwealth of Puerto Rico (not shown). Total CDDs (including TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD) have been identified in 126, 105, 34, 43, 49, and 53 sites, respectively, of the 1,467 hazardous waste sites on the NPL. The frequency of these sites within the United States for total CDDS, TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD, respectively, can be seen in Figures 5-2 through 5-7. Of the 126 sites with total CDD detections, 125 are located in the United States and 1 site is located in the Commonwealth of Puerto Rico (not shown). Of the 105 sites with total TCDD detections, 104 are located in the United States and 1 site is located in the Commonwealth of Puerto Rico (not shown). Of the sites with PeCDD, HxCDD, HpCDD, and OCDD detections, all 34, 43, 49, and 53 sites, respectively, are located in the United States.

5.2 RELEASES TO THE ENVIRONMENT

CDDs have been measured in all environmental media including ambient air, surface water, groundwater, soil, and sediment. While the manufacture and use of chlorinated compounds, such as chlorophenols and chlorinated phenoxy herbicides, were important sources of CDDs to the environment in the past, the restricted manufacture of many of these compounds has substantially reduced their current contribution to environmental releases. It is now believed that incineration/combustion processes are the most important sources of CDDs to the environment (Zook and Rappe 1994). Important incineration/combustion sources include: medical waste, municipal solid waste, hazardous waste, and sewage sludge incineration; industrial coal, oil, and wood burning; secondary metal smelting, cement kilns, diesel fuel combustion, and residential oil and wood burning (Clement et al. 1985; Thoma 1988; Zook and Rappe 1994).

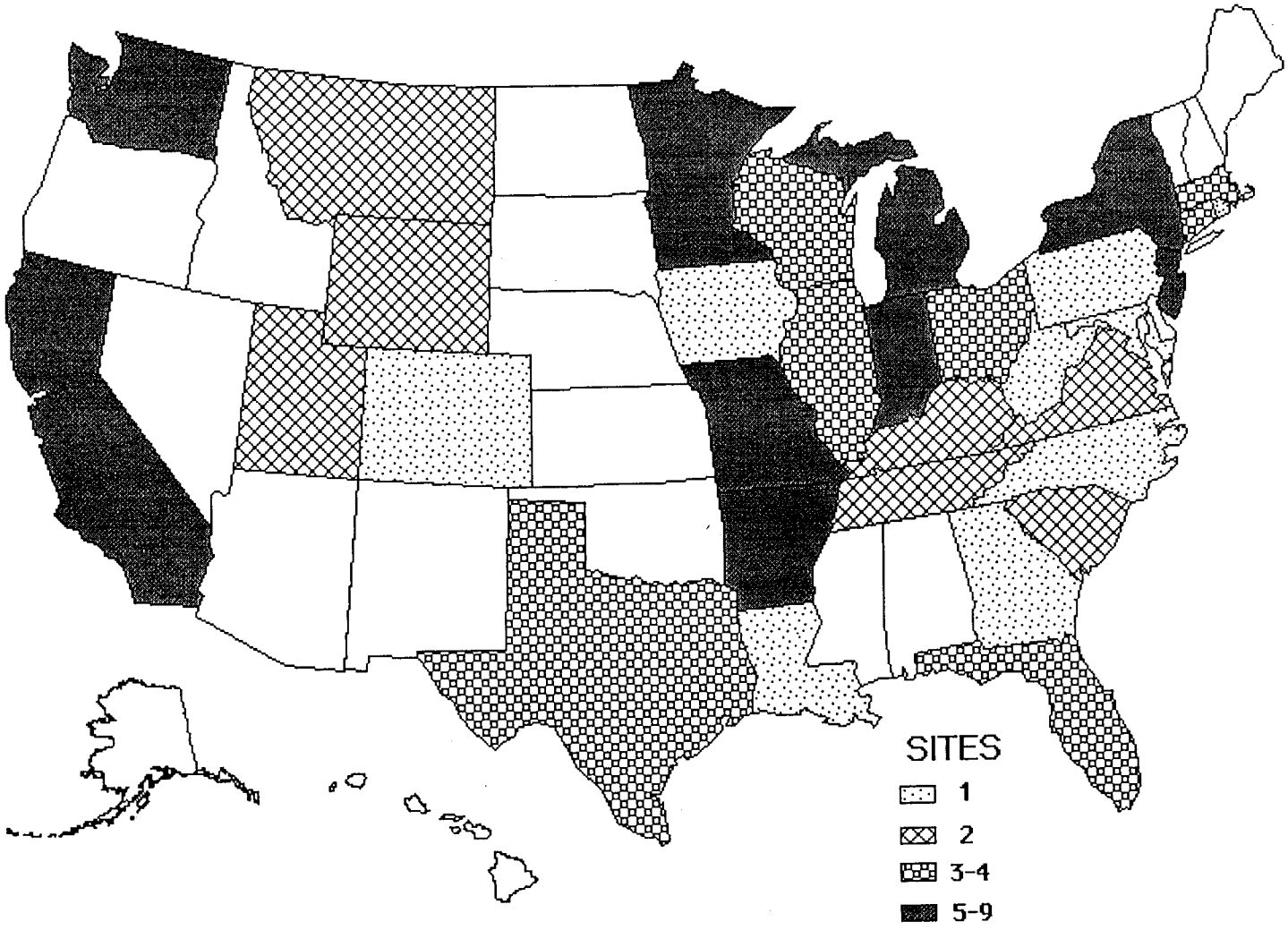
Emissions to the atmosphere from incineration and combustion sources result in the wide-spread distribution of CDDs. Consequently, CDDs are found at low levels in rural soils as well as in sediments of otherwise pristine waterbodies. Much of the CDD deposits from wet and dry deposition ultimately become components of urban runoff which enter rivers, streams, and estuaries directly or through stormwater outfalls and combined sewer overflows (CSOs). In a recent study, Huntley et al. (1997) used statistical

Figure 5-1. Frequency of NPL Sites with 2,3,7,8-TCDD Contamination



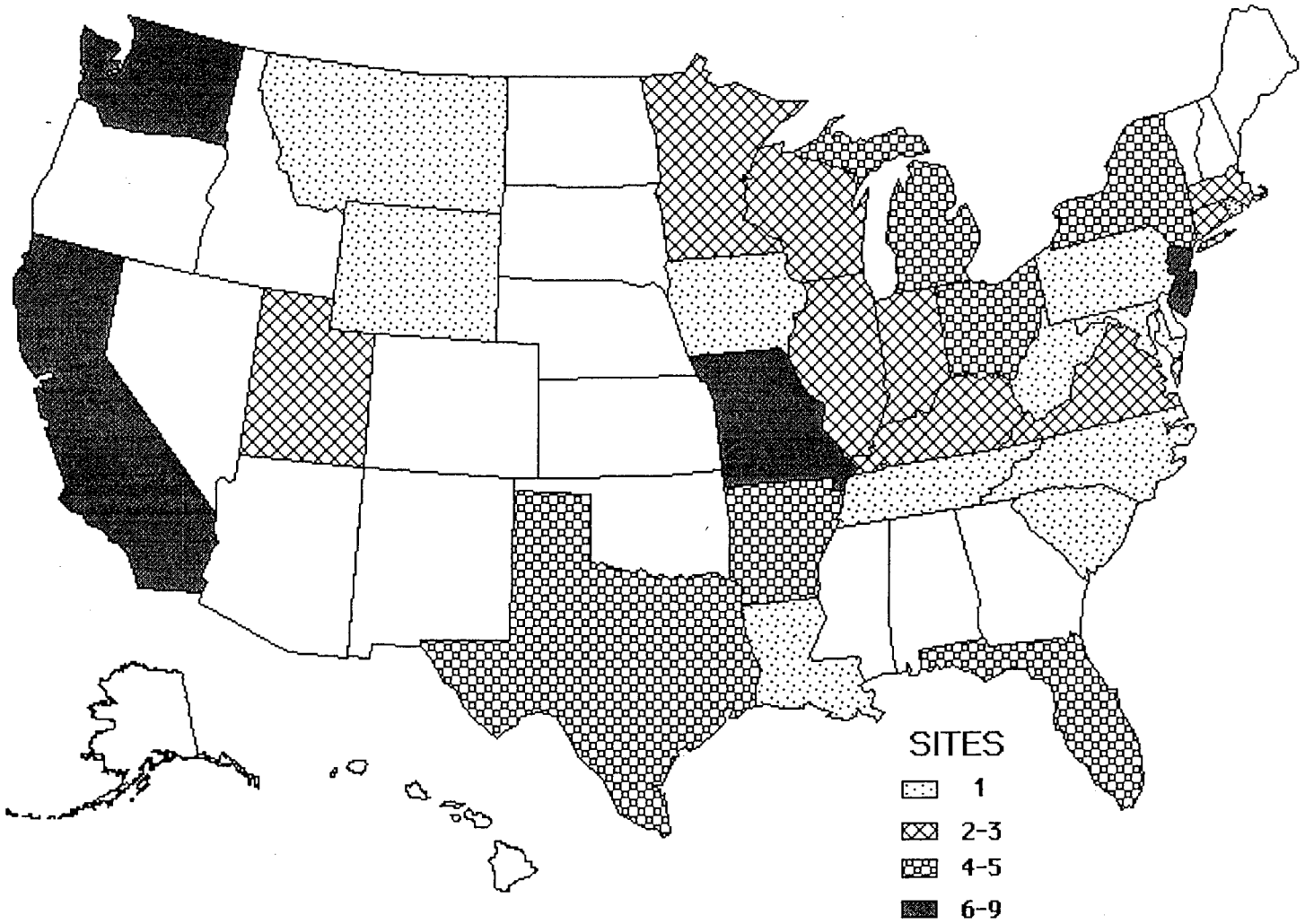
Derived from HazDat 1998

Figure 5-2. Frequency of NPL Sites with Total Dioxin Contamination



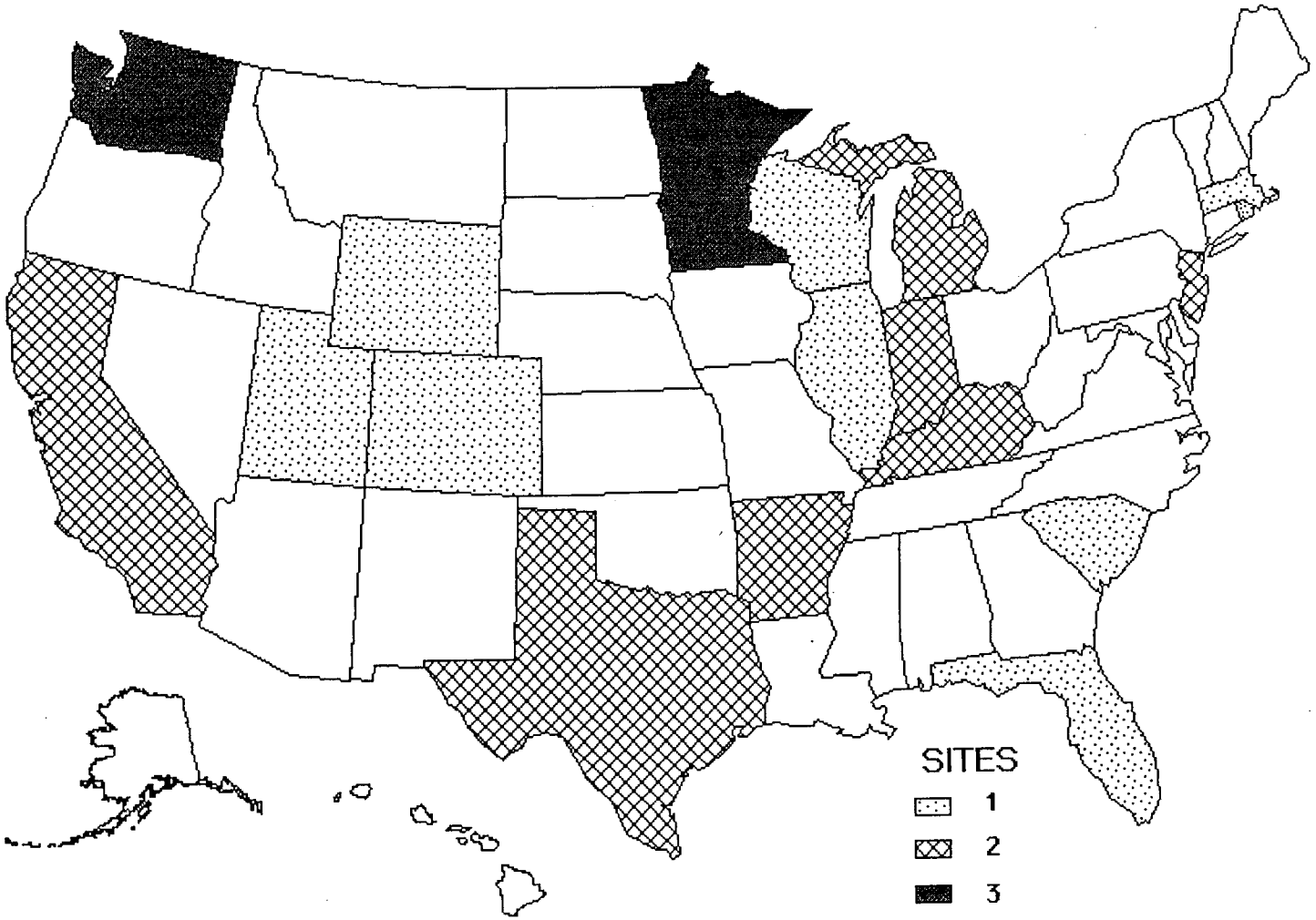
Derived from HazDat 1998

Figure 5-3. Frequency of NPL Sites with Tetra Dioxins Contamination



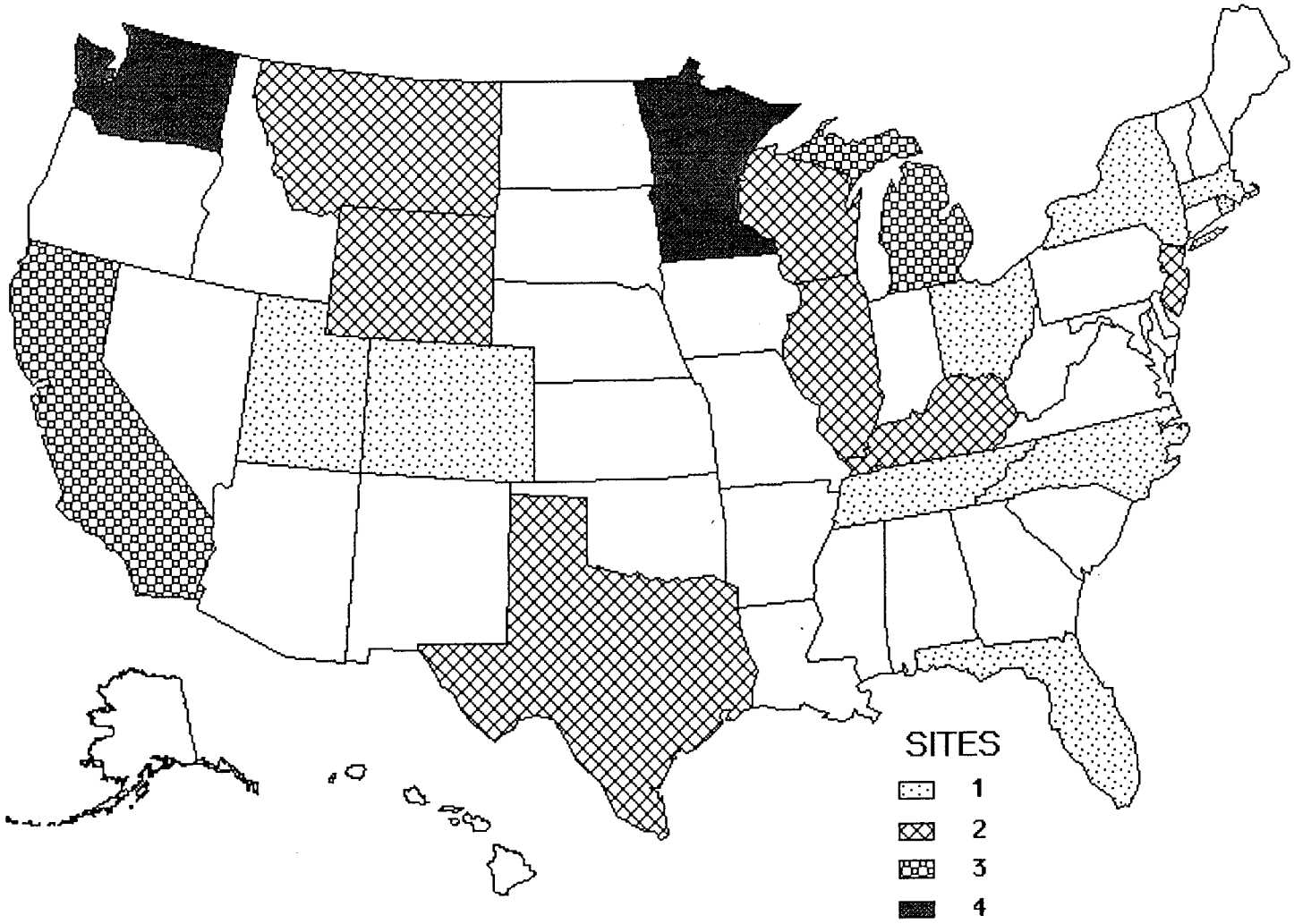
Derived from HazDat 1998

Figure 5-4. Frequency of NPL Sites with Penta Dioxins Contamination



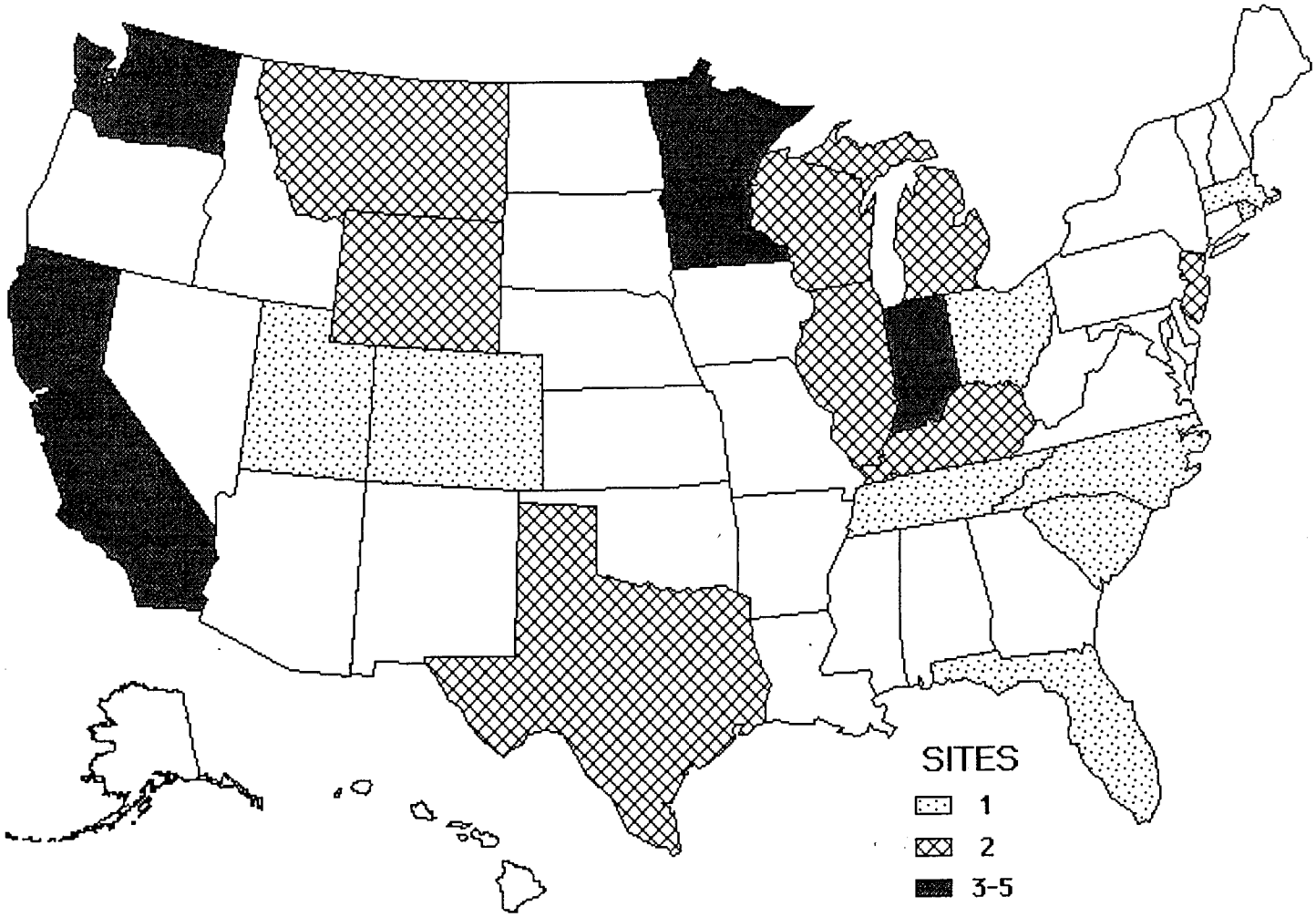
Derived from HazDat 1998

Figure 5-5. Frequency of NPL Sites with Hexa Dioxins Contamination



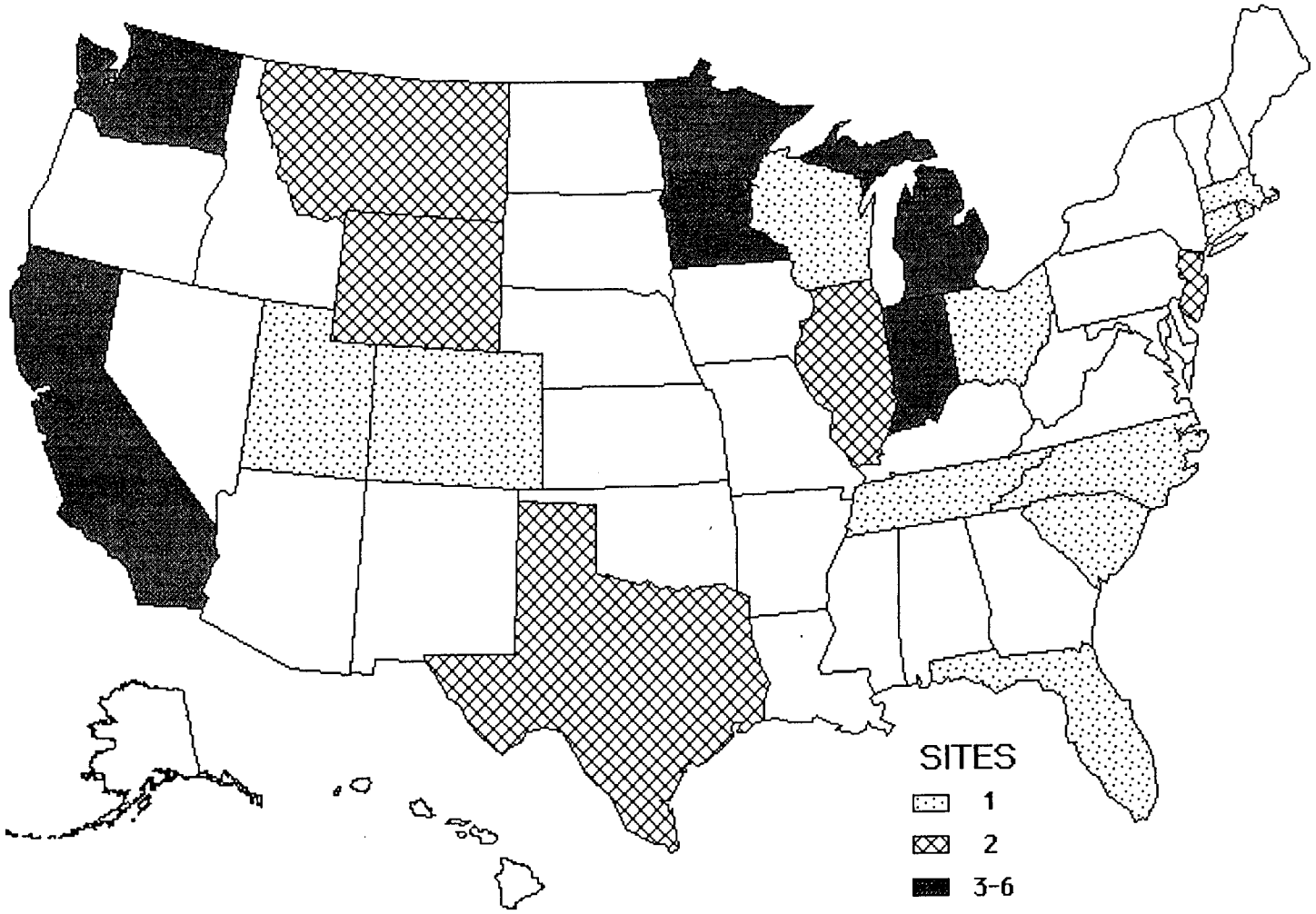
Derived from HazDat 1998

Figure 5-6. Frequency of NPL Sites with Hepta Dioxins Contamination



Derived from HazDat 1998

Figure 5-7. Frequency of NPL Sites with Octa Dioxins Contamination



Derived from HazDat 1998

pattern matching techniques (principal components analysis) to evaluate CDD congener patterns in sediment samples collected adjacent to several CSOs. According to these authors, the presence of these unique CDD/CDF congener patterns in sediment adjacent to CSOs suggested that these CSOs were a likely source given the industrial, residential, and stormwater inputs to the combined sewer overflow system. Such statistical techniques have been applied elsewhere to CDD congener pattern matching in an effort to identify specific sources of CDDs. Wenning et al. (1993a, 1993b) also applied principal components analysis to Newark Bay Estuary sediments and found that most of the congener fingerprint patterns were related to combustion/incineration sources. More recently, Ehrlich et al. (1994) applied polytopic vector analysis, a fingerprinting technique that "unmixes" the CDD/CDF patterns, and concluded that the primary sources of CDD/CDFs in Newark Bay Estuary sediments were combustion/incineration, sewage-related sources, and PCB-related sources. Statistical techniques that have proven useful for identifying sources of CDDs have recently been reviewed (Wenning and Erickson 1994). Future efforts to reduce the release of CDDs to the environment will require additional analysis of the distributional patterns of CDDs in environmental media, which may also provide information on sources still to be identified.

5.2.1 Air

The key sources of CDD releases to air are from anthropogenic combustion processes and the production and use of chemicals contaminated with CDDs. Some evidence suggests that natural combustion processes (e.g., forest fires or volcanic activity) may also be sources of CDDs, but to a much smaller extent. Toxics Release Inventory (TRI) data are not available for CDDs since CDD releases are not required to be reported (EPA 1995g).

Combustion Processes. Combustion processes generate CDDs, CDFs, and other halogenated aromatic compounds (Czuczwa and Hites 1984, 1986a, 1986b). Most of the direct releases of CDDs and CDFs from combustion processes are to the air (Czuczwa and Hites 1984, 1986a, 1986bc). CDDs and CDFs may be found in particulates released from the combustion of most types of organic material and limited evidence suggests that they may also result from trace chemical reactions in fire (Bumb et al. 1980; Crummett 1982; Safe 1990). The processes involved in the formation of CDDs and CDFs consist of numerous chemical reactions that occur during combustion of organic compounds in the presence of chlorinated material. The EPA has recently identified stationary source categories that release 2,3,7,8-TCDD TEQ to the atmosphere (EPA 1998j). The percentage contribution of the five highest source categories are: 68% from municipal waste incineration, 12.3% from medical waste incineration,

8.9% from Portland cement manufacture hazardous waste kilns, 3.5% from secondary aluminum smelting, and 3.0% from other biological incineration. These five source categories account for 95.9% of all stationary emissions of 2,3,7,8-TCDD TEQ to the air.

The "Trace Chemistries of Fire Hypothesis" suggests that CDDs and CDFs can also form during a variety of combustion processes including natural ones, such as forest fires and volcanic eruptions (Crummett 1982). However, there is very limited evidence suggesting that such natural processes could be minor sources of these compounds in the environment. Only data from one study were found that directly measured CDD/CDFs in actual emissions from forest fires. Tashiro et al. (1990) detected the concentration of total CDD/CDFs in air ranging from 15 to 400 pg/m^3. The samples were collected from fixed collectors 10 m above the ground and from aircraft flying through the smoke. Soil samples collected before the burn detected 43 ppt of OCDD in 1 of 4 samples tested. After the burn, OCDD was detected in 3 of 4 soil samples at concentrations of 46, 100, and 270 ppt. Because the small sample size precluded statistical analysis, no further conclusions were drawn by the authors. Thomas and Spiro (1995), however, estimated that forest and agricultural burning accounted for the third largest emission of CDD/CDF in the United States (30 kg/year), behind municipal waste incineration (200 kg/year) and hospital incinerators (40 kg/year) although the inclusion of agricultural burning, which may include acreage treated with long-lived organochlorine pesticides, may skew the values higher than would be expected from forest fires alone. Failure to find CDDs in ancient mummies or ancient frozen Eskimo tissues is another indication that the “Trace Chemistries of Fire Hypothesis” may have little bearing on human exposure (Ligon et al. 1989; Schecter et al. 1988; Tong et al. 1990). The EPA recently found elevated levels of 2,3,7,8-TCDD in two chickens that were traced to clay (used as an anti-caking additive in soybean animal meal) derived from clay deposits mined at the Kentucky-Tennesse Ball Clay Company in Crenshaw, Mississippi. (Chemical Regulation Reporter 1997a, 1997b). However, no information on the origin of the 2,3,7,8-TCDD, either natural or anthropogenic, was presented.

The issue of natural sources of CDD/CDF is interesting, but historical deposition records strongly implicate anthropogenic activity as the major source of CDD/CDFs (Thomas and Spiro 1996). These authors further suggest that the historic record on CDD/CDF deposition provided by sediment cores strongly implies that anthropogenic sources have been overwhelmingly dominant. Sediment cores from Siskwit Lake on a remote island in northern Lake Superior, provide a historic record of atmospheric CDD fluxes (Czuczwa and Hites 1986a). An 8-fold increase in the CDD/CDF deposition rate (from approximately 4–30 pg/cm^2/year) occurred between 1940 and 1970, corresponding to a great expansion in the

industrial use of chlorine (Thomas and Spiro 1996). The decrease in deposition rate of about 30% (from 30 to 24 pg/cm^2/year) from 1970 to the mid 1980s parallels decreased production and use of chlorophenols (pesticide registrations for 2,4,5-T and Silvex were discontinued in 1983 and 1984, respectively) (IARC 1977; Sine 1990) and reductions in municipal incinerator emission resulting from improvements in design, pollution controls, and operation of these facilities (Thomas and Spiro 1996). It is difficult to reconcile these trends with predominantly natural sources, especially since the total area of U.S. forests consumed by forest fires diminished by more than a factor of 4 between 1940 and 1970 through more effective fire control (Thomas and Spiro 1996).

Although the production of CDDs during combustion processes are highlighted here, most samples from combustion sources show a complex mixture of isomers and congeners of CDDs and CDFs which vary in their relative concentrations (Kolenda et al. 1994; Nestrick and Lamparski 1983; Vikelsoe et al. 1994). CDDs have been detected in emissions (flue gas and fly ash) from municipal, hazardous waste, and industrial incinerators (Buser 1987; Oppelt 1991; Sedman and Esparza 1991; Schecter 1983). Combustion of materials, such as vegetation treated with phenoxy acetic acid herbicides, paper and wood treated with chlorophenols, pesticide-treated wastes, and polyvinylchloride (PVC) in the presence of naturally occurring phenols, may lead to CDDs and CDD precursors (Arthur and Frea 1989). PVC is known to yield a small amount of chlorobenzene upon pyrolysis, which in turn thermally decomposes to CDDs and CDFs (Lustenhouwer et al. 1980). CDDs have also been detected in fly ash from an oil-fired power plant, in city dust, in commercial sludge fertilizer, in particulate deposits in car and truck mufflers, in exhaust from vehicles powered with leaded and unleaded gasoline and diesel fuel, in cigarette smoke, and in soot from home fireplaces and from PCB and chlorinated benzene contaminated transformer fires (Bumb et al. 1980; Hutzinger et al. 1985; Lofroth and Zebuhr 1992; Marklund et al. 1987, 1990; Muto and Takizawa 1989; Schecter 1983; Thoma 1988). Dichloroethane, the chlorinated additive in leaded gasoline, is also a source of CDDs (Marklund et al. 1987). The dichloroethane acts as a scavenger to prevent the deposition of lead compounds in engines (Safe 1990). Although the data indicate that CDDs result from diverse processes, the relative contributions of these sources and other unidentified sources to the presence of CDDs in the atmosphere are not known.

A mixture of CDDs (TCDD, PeCDD, HxCDD, HpCDD, OCDD) has been found in emissions (both particles and flue gases) from various combustion sources, including municipal incinerators, power plants, wood burning, home heating systems, and petroleum refining (Chiu et al. 1983; Czuczwa and Hites 1984; Gizzi et al. 1982; Nessel et al. 1991; Thoma 1988; Thompson et al. 1990). In individual samples of

emissions from an urban incinerator, HxCDDs and OCDD were often the most abundant CDDs found, although the homologue pattern can be quite variable (Gizzi et al. 1982). Emission of TCDD from municipal waste combustion ranged from 0.018 ng/m^3 to 62.5 ng/m^3 depending on the type of combustion facility (Roffman and Roffman 1991). A municipal solid waste incinerator sampled in 1988 contained an average TCDD concentration of 0.0012 ng/m^3, where OCDD was present at 1.2 ng/m^3, and HxCDD was present at >1 ng/m^3 (Nessel et al. 1991). In another study, no TCDDs were found in emissions from hazardous waste or municipal waste incinerators; the levels of PeCDD found in the emissions from municipal waste incinerators were three orders of magnitude higher than from hazardous waste incinerators (Oppelt 1991). Fly ash from a municipal incinerator and from coal-fired power plants was analyzed to study the CDD congener distributions typical of combustion samples (Czuczwa and Hites 1984). OCDD was the most abundant CDD in all fly ash samples. Coal fly ash samples differed significantly from municipal incinerator fly ash samples. Although some CDDs were detected in coal fly ash, no TCDDs or PeCDDs were detected. CDDs were present in much lower concentrations in fly ash from coal-fired power plants than in fly ash from a municipal incinerator. The levels of OCDD in the coal fly ash samples (2.2 ppb and 3.8 ppb) were at least 100 times lower than those found in the municipal incinerator fly ash (400 ppb). No isomers of TCDD were detected in municipal incinerator fly ash samples with a detection limit of 100 ppt (Czuczwa and Hites 1984).

CDDs have been detected in chimney soot samples from various home heating systems using unleaded heating oil, coal, and wood in Germany (Thoma 1988). A Canadian study of wood-burning stoves detected only OCDD in particulates from the stack emissions (Wang et al. 1983). Open-air burning of PCP-treated wood produced levels of CDDs ranging from 2 ppb (TCDD) to 187 ppb (OCDD) (Chiu et al. 1983). Combustion of untreated wood also produces CDDs (TCDD, PeCDD, HxCDD, HpCDD, OCDD) (Clement et al. 1985). Samples of bottom ash and chimney ash from 2 wood-burning stoves, 1 open fireplace, and outdoor open-air burning had detectable levels of CDDs ranging from 0.3 to 33 ppb. For each homologous class, the total concentrations ranged from not detectable to 11 ppb. Detection limits were equal to 10 ppt for TCDD and PeCDD and 50 ppt for HxCDD, HpCDD, and OCDD. The open-air burning ash produced the highest total CDD concentration of 33 ppb, with HpCDD (11 ppb) and OCDD (10 ppb) being the most abundant (Clement et al. 1985).

Fires involving capacitors or transformers containing chlorobenzene and PCBs are also sources of CDDs and CDFs. For example, in the transformer fire in the New York State Office Building in Binghamton, NY, TCDD, PeCDD, HxCDD, HpCDD, and OCDD were found in soot samples at levels ranging from

<2 ppm (PeCDD) to 7 ppm (HpCDD) (Tiernan et al. 1985). CDFs were more abundant and were detected at higher concentrations ranging from 28 to 1,920 ppm in soot.

Recently, Wikstrom et al. (1996) studied formation of CDDs, CDFs, and chlorobenzenes in the combustion process. These authors monitored combustion of an artificial fuel where the chlorine level and source were varied in the artificial waste. Different levels of organic chlorine (PVC) and inorganic chlorine ($CaCl_2$⁄6$H_2$0) were added to the fuel. When the level of chlorine in the fuel was <1%, there was no correlation between the quantities of CDDs, CDFs, and chlorobenzenes present. However, when the chlorine level was >1%, increased formation rates were noted for CDDs, CDFs, and chlorobenzenes.

Production and Use of Contaminated Chemicals and Certain Herbicides. CDDs are known trace contaminants of certain chlorinated industrial chemicals like chlorophenols (Buser 1987). CDDs can inadvertently form as by-products during the manufacture of chlorophenols. Since the 1930s, PCP and the tri- and tetrachlorophenols have gained recognition as fungicides, herbicides, insecticides, and precursors in the synthesis of other pesticides.

PCP was developed primarily for use as a wood preservative but has also been used as an herbicide on pineapple and sugarcane plantations. It has also been employed as a molluscicide against schistosomiasis, a severe human parasitic disease prevalent in much of tropical Asia, Africa, and South America (Hutzinger et al. 1985). The major contaminant of commercial PCP is OCDD, which may be present at concentrations between 500 and 1,500 mg/kg (ppm) (Dobbs and Grant 1979; Miller et al. 1989a). PCP may also contain mixed isomers of HxCDD and HpCDD (Pereira et al. 1985). It is currently registered as a restricted-use pesticide for use as a wood preservative (Sine 1990).

2,3,7,8-TCDD forms during the manufacture of 2,4,5-TCP. 2,4,5-TCP has been used in cooling towers and in paper, pulp, and leather processing (Hutzinger et al. 1985). 2,4,5-TCP was used to produce the bactericide hexachlorophene and phenoxy-herbicides like 2,4,5-trichlorophenoxy acids (2,4,5-T). 2,4,5-T, in turn, was used in the production of a wide variety of herbicides including Silvex (2-[2,4,5-trichloro-phenoxy]propionic acid) and Agent Orange (Hutzinger et al. 1985). Hexachlorophene, which is currently under EPA suspension, is reported to contain <15 µg/kg (ppb) 2,3,7,8-TCDD (IARC 1977; Sine 1990). 2,3,7,8-TCDD is an unwanted by-product formed during the production of hexachlorophene (Freeman et al. 1986). The 2,3,7,8-TCDD produced is primarily contained in still-bottom waste (waste oils) remaining after hexachlorophene is purified (Freeman et al. 1986). Still-bottom waste and other oils were used in the

early 1970s for dust control on roads, parking lots, horse arenas, and other sites around Missouri (Freeman et al. 1986). The herbicide 2,4,5-T produced commercially prior to 1965 contained up to 30 mg/kg (ppm) or more 2,3,7,8-TCDD (IARC 1977). The level of 2,3,7,8-TCDD in commercial 2,4,5-T was reduced to <0.05 mg/kg (ppm), and most of the commercial 2,4,5-T available before its registration was discontinued in the United States in 1983 contained <0.02 mg/kg (ppm) 2,3,7,8-TCDD (IARC 1977; Sine 1990). Chlorophenoxy herbicides, such as 2,4-D, are typically formulated as esters or amine salt derivatives (IARC 1986b). Of 16 samples of 2,4-D formulations from Canada analyzed for CDDs in the early 1980s, 8 of 9 ester formulations and 4 of 7 amine salt formulations were contaminated (IARC 1986b). The 2,4-D ester formulations contained 0.2–1.8 mg/kg (ppm) 1,3,6,8-TCDD (the only TCDD isomer detected), while the 2,4-D amine salt formulations contained 0.02–0.3 mg/kg (ppm) 1,3,6,8-TCDD (IARC 1986b). It should be noted that 1,3,6,8-TCDD is not one of the toxic CDDs with respect to mammals; however, 2,3,7,8-substituted CDDs/CDFs have been reported in 2,4-D from Russia (Schecter et al. 1993).

Agricultural and wartime uses of trichlorophenol-based herbicides such as 2,4,5-T and Silvex also have resulted in release of 2,3,7,8-TCDD at low concentrations in many countries (EPA 1987k). 2,4,5-T was used in aerial spraying operations for weed control on crops, along fence rows, ditch banks, farm roadways, pastures, and rangeland (Bovey 1980). Non-farm uses of 2,4,5-T included tree and bush control on rights-of-way, roadways, fire lanes, and railroads (Bovey 1980). Agent Orange, used as a defoliant in the Vietnam War from 1962 to 1970, was contaminated with an average of 2 ppm of 2,3,7,8-TCDD (Czuczwa and Hites 1986a, 1986b; Wolfe et al. 1985). An estimated 10–11 million gallons were applied in South Vietnam (EPA 1987k; Wolfe et al. 1985). This volume of Agent Orange contained an estimated 368 pounds of 2,3,7,8-TCDD (Wolfe et al. 1985). Agent Orange is an equal parts mixture of the butyl esters of 2,4,5,-T and 2,4-dichlorophenoxyacetic acid (2,4-D) (Josephson 1983). These herbicides were used extensively in silviculture for control of deciduous trees in conifer forests before their use was discontinued (EPA 1987k). The use of Silvex, a herbicide closely related to 2,4,5-T, was discontinued in the United States in 1984 (Sine 1990).

Industrial accidents have also released high levels of CDDs into the air. In 1976, at least 1.3 kg (2.87 pounds) of 2,3,7,8-TCDD was released into the air as a result of an industrial accident at the ICMESA chemical plant near Seveso, Italy, that was involved in 2,4,5-TCP synthesis (Cerlisi et al. 1989; Mocarelli et al. 1991). The 2,3,7,8-TCDD release contaminated a populated area of about 2.8 km^2 (1.08 mi^2) (Mocarelli et al. 1991).

2,3,7,8-TCDD has been detected in air samples collected at 9 of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). Total CDDs have been detected in air samples collected at 10 of the 126 NPL sites where they have been detected in some environmental media. Total TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in air samples at 10, 3, 3, 3, and 1 sites of the 105, 34, 43, 49, and 53 sites, respectively, where they have been detected in some environmental media (see Table 5-1).

5.2.2 Water

CDDs can enter water by a number of different mechanisms including urban runoff, combined sewer overflows (CSOs), and direct discharge by industrial facilities and publicly-owned treatment works (POTWs); deposition of particulates from combustion sources, runoff and drift from the use of chlorophenol-based pesticides; and leaching from chlorophenol-containing waste sites (Huntley et al. 1997; Muir et al. 1986a; Periera et al. 1985; Shear et al. 1996). Direct application or drift of 2,4,5-T or Silvex into water has also resulted in release of TCDD to surface water (Norris 1981); however, the contribution of CDDs from pesticide drift is now negligible since most CDD-containing pesticides have been banned. The migration of chemical wastes containing CDDs from disposal sites has resulted in contamination of surface water and groundwater (HazDat 1998).

CDDs/CDFs, specifically 2,3,7,8-TCDD and 2,3,7,8-TCDF, are also present in effluent and sludges from pulp and paper mills that employ the bleached kraft process (Clement et al. 1989; EPA 1991b; Swanson et al. 1988). 2,3,7,8-TCDD was detected in 7 of 9 bleached pulps at concentrations ranging from not detected (<1 ppt) to 51 ppt (median 4.9 ppt; mean 13 ppt) (Amendola et al. 1989). It was also detected in waste waters from 4 of 5 paper mills at levels ranging from not detected (<0.006 ppt) to 3.6 ppt (Amendola et al. 1989).

During 1988, the EPA and the U.S. pulp and paper industry jointly conducted a survey of 104 pulp and paper mills in the United States to measure concentrations of CDDs in effluent, sludge, and paper (EPA 1990d). This study is commonly called the 104-Mill Study and includes all U.S. mills where wood pulps are bleached with chlorine or chlorine derivatives. Higher chlorinated CDDs/CDFs are typically found in effluent when chlorine dioxide is used, but not when elemental chlorine is used. In 1992, the pulp and paper industry conducted its own survey (NCASI 1993). As part of an effort to develop revised effluent guidelines and standards for the pulp and paper industry, the EPA recently published the development

Table 5-1. Number of NPL Sites Where CDDs Have Been Detected in One or More Environmental Media[a]

Medium	2,3,7,8-TCDD[b]	**Total CDDs[c]**	Total TCDDs[d]	Total PeCDDs[e]	Total HxCDDs[f]	Total HpCDDs[g]	Total OCDDs[g]
Air	9	**10**	10	3	3	3	1
Surface water	9	**14**	10	1	4	4	6
Groundwater	15	**32**	21	3	10	14	16
Soil[i]	61	**94**	71	21	29	34	38
Sediment	17	**31**	22	7	10	9	13
Fish	11	**13**	12	1	0	0	1
Game animals	1	**1**	1	0	0	0	0
Total sites where chemical was detected	91	**126**	105	34	43	49	53

[a] A total of 1,467 hazardous waste sites have been identified on the NPL nationwide
[b] Includes only 2,3,7,8-TCDD (CAS# 001746-01-6)
[c] Includes all dioxins identified in footnotes d, e, f, g, and h.
[d] Includes 2,3,7,8-TCDD and other tetra Cdds (CAS# 41903-57-5)
[e] Includes PeCDDs (CAS# 039227-61-7 and 040321-76-4)
[f] Includes HxCDDs (CAS# 019408-74-3, 034465-46-8, 057653-85-7)
[f] Includes HpCDDs (CAS# 035822-46-9 and 037871-00-4)
[h] Includes OCDD (CAS# 003268-87)
[i] Soil contamination sites include those defined as:
1. surface/top soil
2. subsurface soil, or
3. soil depth not specified.

CDD = chlorinated dibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; NPL = National Priorities List; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: HazDat 1998

document for the guidelines and standards being proposed for this industry (EPA 1993a). This development document presents estimates of annual discharges of two congeners, 2,3,7,8-TCDD and 2,3,7,8-TCDF in effluents (from wastewater treatment systems) from this industry as of January 1993.

The joint EPA/paper industry study of 104 pulp and paper mills provides an estimate of the release of 2,3,7,8-TCDD and 2,3,7,8-TCDF in bleached pulp, waste water sludge, and waste water effluent from the U.S. pulp and paper industry as of mid-to-late 1988 (EPA 1990d). This was a time in the industry's history when only limited use of pulping and bleaching technologies and operating practices that demonstrated potential to reduce the formation of TCDDs and TCDFs had been implemented. In this study 2,3,7,8-TCDD was detected at 90 and 56% of the kraft and sulfite mills, respectively, that were surveyed, and no mill was found to be free of 2,3,7,8-TCDD/TCDF. For bleached pulp, the mean 2,3,7,8-TCDD concentration was 7.5 ppt (maximum 56 ppt) for kraft hardwoods, 12 ppt (maximum 116 ppt) for kraft softwoods, 7.1 ppt (maximum 15 ppt) for sulfite hardwoods, and 3.5 ppt (maximum 3.5 ppt) for sulfite softwoods. Mean waste water effluent concentrations of 2,3,7,8-TCDD were 0.076 ppt for kraft mills (maximum 0.64 ppt) and 0.013 ppt (maximum 0.023 ppt) for sulfite mills. Waste water sludges contained mean 2,3,7,8-TCDD concentrations of 101 ppt for kraft mills (maximum 1,390 ppt) and 13 ppt (maximum 58 ppt) for sulfite mills. Furthermore, for all kraft mills, about 38% of the 2,3,7,8-TCDD was partitioned to pulps, 33% to waste water sludges, and 29% to waste water effluents.

The NCASI (1993) report found that <10% of pulp and paper mills had 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in effluent above the detection limits of 10 ppq and 100 ppq, respectively; however, none of the more highly congener groups were measured. Similar results were obtained in the short- and long-term sampling reported for 18 mills (EPA 1993a). 2,3,7,8-TCDD and 2,3,7,8-TCDF were detected at four and nine mills, respectively. Waste water sludges at 75–90% of all mills contained <10 ppt of 2,3,7,8-TCDD and <100 ppt of 2,3,7,8-TCDF (NCASI 1993). Similar results were reported in the EPA (1993a) report except that 2,3,7,8-TCDD and 2,3,7,8-TCDF were found in sludges in 64 and 85%, respectively, of the mills sampled. NCASI (1993) reported that almost 90% of bleached pulps contained <2 ppt of 2,3,7,8-TCDD and <20 ppt of 2,3,7,8-TCDF. For bleached pulps, the mean 2,3,7,8-TCDD concentration was 0.9 ppt (maximum 10 ppt) and the mean 2,3,7,8-TCDF concentration was 6 ppt (maximum 323 ppt). The mean waste water effluent concentration of 2,3,7,8-TCDD was 0.006 ppt (maximum 0.08 ppt) and 0.031 ppt (maximum 0.510 ppt) for 2,3,7,8-TCDF. Waste water sludges contained a mean 2,3,7,8-TCDD concentration of 11 ppt (maximum 133 ppt) and 11 ppt (maximum 735 ppt) for 2,3,7,8-TCDF. In this study, mean pulp, waste water effluents, and waste water sludge concentrations of 2,3,7,8-TCDD all

declined by a factor of about 10 from those cited in the 104 Mill Study (EPA 1990d). Overall, NCASI (1993) reports a 90% reduction in TEQs generated by pulp and paper mills from 1988 to 1992 for all 2,3,7,8-TCDDs and 2,3,7,8-TCDFs.

2,3,7,8-TCDD has been detected in surface water and groundwater samples collected at 9 and 15 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). Total CDDs have been detected in surface and groundwater samples collected at 14 and 32 of the 126 NPL sites where they have been detected in some environmental media. Total TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in surface water samples collected at 10, 1, 4, 4, and 6 sites and in groundwater samples collected at 21, 3, 10, 14, and 16 of the 105, 34, 43, 49, and 53 NPL sites, respectively, where these homologues have been detected in some environmental media (see Table 5-1).

5.2.3 Soil

Historically, CDDs have been deposited onto soil through pesticide applications and disposal of CDD-contaminated industrial wastes, and via land application of paper mill sludges (EPA 1991b). Currently, however, atmospheric fall-out of CDD-laden particulates and gases appears to be the predominant source of CDDs to soil (Hutzinger et al. 1985).

The commercial production of trichlorophenol, as well as various derivative products such as 2,4,5-T and other biocides, has yielded large quantities of waste products containing substantial concentrations of CDDs. Extensive contamination of the environment with 2,3,7,8-TCDD occurred in Missouri in the early 1970s as a result of the spraying of horse arenas, roads, and parking lots with mixtures of used oil and chemical waste (Tiernan et al. 1985). The chemical waste, formed during the manufacture of 2,4,5-TCP and then used to make hexachlorophene, contained several hundred ppm of 2,3,7,8-TCDD (Tiernan et al. 1985). Several thousand gallons of this waste were dispersed over a sizable area of southwestern and eastern Missouri during the 1970s. Concentrations of 2,3,7,8-TCDD in soil samples from selected contaminated sites throughout Missouri ranged from 30 to 1,750 ppb (Tiernan et al. 1985). Concentrations of 2,3,7,8-TCDD in soil samples from Times Beach, Missouri, which had been heavily contaminated, ranged from 4.4 to 317 ppb (Tiernan et al. 1985).

In Seveso, Italy, an explosion occurred during the production of 2,4,5-T and a cloud of toxic material including 2,3,7,8-TCDD was released (Cerlisi et al. 1989; MMWR 1988; Mocarelli et al. 1991). Debris

from the cloud covered an area of approximately 700 acres (2.8 km^2). The total amount of 2,3,7,8-TCDD released during the accident was estimated to be 1.3 kg. Soil samples from this industrial accident were measured in three areas: Zone A, the most contaminated zone where residents were evacuated; Zone B, the moderately contaminated area where residents were advised not to eat locally raised produce; and Zone R, where 2,3,7,8-TCDD contamination in soil was lowest of the three areas. Mean soil concentrations in these 3 areas were: 230 $\mu g/m^2$ (maximum 5,477 $\mu g/m^2$) in Zone A, 3 $\mu g/m^2$ (maximum 43.9 $\mu g/m^2$) in Zone B, and 0.9 $\mu g/m^2$ (maximum 9.7 $\mu g/m^2$) in Zone R (MMWR 1988).

The migration of chemical waste containing CDDs from disposal sites has also resulted in environmental contamination of sediment. For example, at Love Canal in Niagara Falls, New York, where an estimated 200 tons of 2,4,5-TCP production waste were disposed of during the 1940s and early 1950s, 2,3,7,8-TCDD was detected at high concentrations (up to several hundred ppb) in storm sewer sediments (Smith et al. 1983; Tiernan et al. 1985).

2,3,7,8-TCDD has been detected in soil and sediment samples collected at 61 and 17 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). Total CDDs have been detected in soil and sediment samples collected at 94 and 31 of the 126 NPL sites where they have been detected in some environmental media. Total TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in soil samples at 71, 21, 29, 34, and 38 sites and in sediment samples at 22, 7, 10, 9, and 13 sites of the 105, 34, 43, 49, and 53 NPL sites, respectively, where these homologues have been detected in some environmental media (see Table 5-1).

5.3 ENVIRONMENTAL FATE

Combustion generated CDDs may be transported long distances (as vapors or associated with particulates) in the atmosphere (Czuczwa and Hites 1986a, 1986b; Tysklind et al. 1993). They may eventually be deposited on soils, surface waters, or plant vegetation as a result of dry or wet deposition. CDDs (primarily MCDD, DCDD, TrCDD) will slowly volatilize from the water column, while the more highly chlorinated CDDs will adsorb to suspended particulate material in the water column and be transported to the sediment (Fletcher and McKay 1993; Muir et al. 1992). CDDs deposited on soils will strongly adsorb to organic matter. CDDs are unlikely to leach to underlying groundwater but may enter the atmosphere on soil dust particles or enter surface waters on soil particles in surface runoff. Low water solubilities and high lipophilicity indicate that CDDs will bioconcentrate in aquatic organisms, although as a result of their

binding to suspended organic matter the actual uptake by such organisms may be less than predicted. This is also true of uptake and bioconcentration by plants, although foliar deposition and adherence may be significant.

5.3.1 Transport and Partitioning

Combustion processes appear to have contributed to the ubiquity of CDDs in the environment (Hites and Harless 1991; Tysklind et al. 1993). CDDs have relatively long residence times in the atmosphere, and combustion-generated CDDs associated with particulates can become distributed over large areas (Tysklind et al. 1993). During transport in the atmosphere, CDDs are partitioned between the vapor phase and particle-bound phase (Hites and Harless 1991). However, because of the very low vapor pressure of CDDs, the amount present in the vapor phase generally is negligible as compared to the amount adsorbed to particulates (Paustenbach et al. 1991). The two environmental factors controlling the phase in which the congener is found are the vapor pressure and the atmospheric temperature (Hites and Harless 1991). Congeners with vapor pressure $<10^{-8}$ mm Hg will be primarily associated with particulate matter while congeners with a vapor pressure $>10^{-4}$ mm Hg will exist primarily in the vapor phase. Those chemicals with vapor pressures between these values can be found in both the vapor phase and associated with particulates (Eisenreich et al. 1981). With a reported vapor pressure ranging from $7.4x10^{-10}$ to $3.4x10^{-5}$ mm Hg, 2,3,7,8-TCDD falls into the intermediate category.

The detection of CDDs in sediments from Siskiwit Lake, Isle Royale, suggests that CDDs can be transported great distances in air (Czuczwa and Hites 1986a, 1986b). Because this lake is landlocked on a wilderness island in Lake Superior, the only way that CDDs could reach these sediments is by atmospheric fall-out (i.e., by wet and dry deposition). Similar amounts of CDDs were also found in Lake Huron and Lake Michigan sediments, which indicates that atmospheric transport is a source of CDDs found on these Great Lake sites (Czuczwa and Hites 1986a, 1986b; Hutzinger et al. 1985). Atmospheric deposition of TCDD to Lake Erie may contribute up to 2% of the annual input of TCDD to the lake (Kelly et al. 1991). Through pattern analysis of herring gull monitoring data, Hebert et al. (1994) provided evidence that the sources of CDDs in Great Lakes food chains were mainly atmospheric, with the exception of 2,3,7,8-TCDD in Lake Ontario, and several CDDs in Saginaw Bay in Lake Huron where point sources were implicated.

CDDs are physically removed from the atmosphere via wet deposition (scavenging by precipitation), particle dry deposition (gravitational settling of particles), and gas-phase dry deposition (sorption of CDDs in the vapor phase onto plant surfaces) (Rippen and Wesp 1993; Welschpausch et al. 1995). Precipitation (rain, sleet, snow) is very effective in removing particle-bound CDDs from the atmosphere (Hites and Harless 1991; Koester and Hites 1992). Table 5-2 summarizes the average ppt scavenging ratios and percentage of washout due to particulates for congener groups of both CDDs and CDFs collected at two sites in Indiana. The scavenging ratio is the ratio of the concentration of the congener group in rain to the atmospheric concentration of the congener group and is a measure of the effectiveness of rain in removing the congener groups from the atmosphere. Table 5-2 also summarizes the percentages of the congener groups scavenged as particles in rain rather than as dissolved solutes in rain. Total rain scavenging ratios ranged from 10,000 to 150,000; HpCDDs and OCDD (the congeners most strongly associated with particulates) were the congeners scavenged most efficiently (Hites and Harless 1991; Koester and Hites 1992).

Environmental fate modeling of CDDs requires knowledge of a number of fundamental physical and chemical parameters, such as water solubility, vapor pressure, Henry's law constant, octanol-water partition coefficient (K_{ow}), and organic carbon partition coefficient (K_{oc}). CDDs are a class of high molecular weight, highly hydrophobic compounds. Although the class contains 8 homologues (congener groups) and 75 congeners, solubility values are available for only a handful of these congeners (Doucette and Andren 1988). CDDs have very low water solubilities, with solubility decreasing with increasing chlorine substitutions (Doucette and Andren 1988). The water solubility of 2,3,7,8-TCDD ranges from $7.9x10^{-6}$ to $33.2x10^{-4}$ mg/L (Shiu et al. 1988). See Table 3-2 for the water solubilities for specific congeners. Water solubilities at 25 EC for the congener groups have been estimated as follows: MCDD, 0.278–0.417 mg/L; DCDD, $3.75x10^{-3}$–$1.67x10^{-2}$ mg/L; TrCDD, $4.75x10^{-3}$–$8.41x10^{-3}$; TCDD, $7.9x10^{-6}$ to $6.3x10^{-4}$ mg/L; PeCDD, $1.18x10^{-4}$ mg/L; HxCDD, $4.42x10^{-6}$ mg/L; HpCDD, $2.4x10^{-6}$–$1.9x10^{-3}$ mg/L; and OCDD, $0.1x10^{-9}$–$7.4x10^{-8}$ mg/L (ASTER 1995; Doucette and Andren 1988; HSDB 1997; McCrady and Maggard 1993; Shiu et al. 1988).

CDDs generally exhibit very low vapor pressures, with the tendency of decreasing vapor pressure with increasing chlorine substitution (Friesen et al. 1985; Rordorf 1986, 1989). At 25 EC, the vapor pressure of 2,3,7,8-TCDD ranges from $7.4x10^{-10}$ to $3.4x10^{-5}$ mm Hg (HSDB 1997; Rordorf 1989). See Table 3-2 for the vapor pressures of specific congener groups. Vapor pressures at 25 EC for the other congener groups have been estimated as follows: MCDD, $9.0x10^{-5}$–$1.3x10^{-4}$ mm Hg; DCDD, $9.0x10^{-7}$–$2.9x10^{-6}$ mm Hg;

Table 5-2. Rain Scavenging Ratios (RS) and Percent Washout Due to Particulates (%W) for CDDs and CDFs in Ambient Air in Two Midwest Cities

Congener Group	Bloomington, IN		Indianapolis, IN	
	RS	%W	RS	%W
TCDD[a]	–	–	–	–
PeCDD	10,000	50	30,000	67
HxCDD	10,000	88	26,000	69
HpCDD	62,000	93	91,000	78
OCDD	90,000	80	150,000	60
TCDF	22,000	21	33,000	24
PeCDF	14,000	54	18,000	35
HxCDF	11,000	77	15,000	74
HpCDF	34,000	88	32,000	79
OCDF	21,000	52	41,000	87
Total CDD/CDF	—	68	—	64

[a]Rarely detected; no calculations performed

Sources: Hites and Harless 1991; Koester and Hites 1992

HpCDD = heptachlorodibenzo-*p*-dioxin; HpCDF = heptachlorodibenzofuran; HxCDD = hexachlorodibenzo-*p*-dioxin; HxCDF = hexachlorodibenzofuran; OCDD = octachlorodibenzo-*p*-dioxin; OCDF = octachlorodibenzofuran; PeCDD = pentachlorodibenzo-*p*-dioxin; PeCDF = pentachlorodibenzofuran; TCDD = tetrachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran

$6.46x10^{-8}$–$7.5x10^{-7}$; TCDD, $7.4x10^{-10}$–$4.0x10^{-3}$ mm Hg; PeCDD, $6.6x10^{-10}$ mm Hg; HxCDD, $3.8x10^{-11}$ mm Hg; HpCDD, $5.6x10^{-12}$–$7.4x10^{-8}$ mm Hg; and OCDD, $8.25x10^{-13}$–$1.68x10^{-12}$ mm Hg (HSDB 1997; McCrady and Maggard 1993; Rordorf 1989; Shiu et al. 1988). CDDs can be found in both the vapor and particle-bound phases (Eitzer and Hites 1989a; Hites and Harless 1991), with the low vapor pressure of OCDD resulting in its enrichment in the particulate phase in the atmosphere. When this particulate matter is deposited on water, OCDD-enriched sediments will result (Eitzer 1993). The less chlorinated CDD congeners (TCDD and PeCDD) occur in greater proportion in the vapor and dissolved phases of air and rain, whereas the more chlorinated congeners (HpCDD and OCDD) are associated with the particulate-bound phases (EPA 1991d). Data from one study of CDDs in the ambient atmosphere of Bloomington, IN, found that vapor-to-particle ratios for individual CDDs ranged from 0.01 to 30 and were dependent on the ambient temperature and the compound's vapor pressure (Eitzer and Hites 1989b). Since the less-chlorinated CDDs have higher vapor pressures, they are found to a greater extent in the vapor phase (Eitzer and Hites 1989a). As air moves, photodegradation of the vapor-phase CDDs occurs and they are lost more readily than the particulate-bound CDDs. Vapor-phase CDDs are not likely to be removed from the atmosphere by wet or dry deposition (Atkinson 1991), although this is a primary removal process for particulate-bound CDDs. Wet or dry deposition could result in greater concentrations of the more chlorinated CDDs reaching soil or water surfaces and eventually sediment (EPA 1991d). All CDDs are found to some extent in both the vapor phase and bound to particulates. At warmer temperatures (28 EC), CDDs, particularly the MCDDs, DCDDs, TrCDDs, and TCDDs will have a greater tendency to exist in the vapor phase. At cooler temperatures (16–20 EC and <3 EC), all CDDs will have less propensity to exist in the vapor phase and greater propensity to adsorb to particulates (Shroy et al. 1985). At a constant temperature, there is a positive relationship between increasing numbers of chlorine atoms on the molecule and decreased propensity to exist in the vapor phase relative to particulate adsorption (Eitzer and Hites 1989b; Paustenbach et al. 1991; Shroy et al. 1985).

CDDs are removed from the water column to a minor extent by volatilization to the atmosphere, with binding to particulates and sediment, or bioaccumulation by aquatic biota being more significant processes (Fletcher and McKay 1993; Muir et al. 1992; Paustenbach et al. 1992). CDDs have Henry's law constants ranging from 1.31×10^{-6} to 146×10^{-6} atm-m^3/mol (Shiu et al. 1988). These values indicate that volatilization from water is likely to be a slow, with the transfer rate controlled by the gas-phase resistance (i.e., the rate is controlled by slow diffusion through the air) (Lyman et al. 1982; Shiu et al. 1988). The more chlorinated homologous classes (TCDD, PeCDD, HxCDD, HpCDD, OCDD) have lower Henry's law constant values than the less chlorinated homologous classes (MCDD, DCDD, TrCDD). Thus,

volatilization from the water column is not expected to be a very significant loss process for the TCDD through OCDD congeners as compared to adsorption to particulates. In general, the Henry's law constants decrease with increasing chlorine number as a result of the decrease in vapor pressure and water solubility (Shiu et al. 1988). Volatilization half-lives for 2,3,7,8-TCDD were calculated for ponds and lakes (32 days) and for rivers (16 days) (Podoll et al. 1986). The primary removal mechanism for CDDs from the water column is sedimentation, with 70–80% of the CDDs being associated with the particulate phase (Muir et al. 1992). The remainder was associated with dissolved organic substances. CDDs bound to sediment particles may be resuspended in the water column if the sediments are disturbed. This could increase both the transport and availability of the CDDs for uptake by aquatic biota (Fletcher and McKay 1993).

Generally, CDDs are characterized by low vapor pressure, low aqueous solubility, and high hydrophobicity, suggesting that these compounds strongly adsorb to soil and that their vertical mobility in the terrestrial environment is low (Eduljee 1987b). In general, higher chlorinated CDDs also volatilize more slowly from soil and water surfaces than do lower chlorinated ones (Hutzinger et al. 1985). Nash and Beall (1980) reported that only 12% of 2,3,7,8-TCDD applied to bluegrass turf as a component of emulsifiable Silvex volatilized over a 9-month period. Because CDDs (particularly the more highly chlorinated PCDD, HxCDD, HpCDD, and OCDD) strongly adhere to soil and exhibit low solubility in water, leaching of CDDs would be unlikely if water were the only transporting medium. Instead, wind and erosion can cause the mixing and transport of CDD-contaminated soil. As a result of erosion, surface soil contaminated with CDDs is either blown away by wind or washed via surface water runoff into rivers, lakes, and streams, with burial in the sediments being the predominant fate of CDDs sorbed to soil (Hutzinger et al. 1985).

Adsorption is an important process affecting transport of hydrophobic compounds such as CDDs. The organic carbon fraction of the soil is believed to be the most important factor governing the degree of adsorption of hydrophobic organic contaminants. CDDs adsorb more strongly to soils with a higher organic carbon content than to soils with low organic carbon content (Yousefi and Walters 1987). Because of their very low water solubilities and vapor pressures, CDDs found below the surface soil (top few mm) are strongly adsorbed and show little vertical migration, particularly in soil with high organic carbon content (Yanders et al. 1989). Vertical movement of CDDs in soil may result from the saturation of sorption sites of the soil matrix, migration of organic solvents, or human or animal activity (Hutzinger et al. 1985). Adsorption/desorption of 2,3,7,8-TCDD in contaminated soils was studied by Des Rosiers (1986). Soil samples were taken from an abandoned 2,4,5-T manufacturing facility and a scrap metal yard in New

Jersey and from horse arenas, roadways, and residential property in Missouri. Historically, these samples were contaminated with either chemical residues or waste oils containing 2,3,7,8-TCDD. Mean log organic carbon partition coefficient (K_{oc}) values ranged from 7.39 to 7.58 (Des Rosiers 1986). This K_{oc} range indicates that 2,3,7,8-TCDD is immobile in soil (Swann et al. 1983). However, the mobility of 2,3,7,8-TCDD in soil will increase if organic co-solvents that can solubilize 2,3,7,8-TCDD are present in the soil (Podoll et al. 1986). This situation might occur at a hazardous waste site. In one study, only 1.5% of the CDDs applied to soil surfaces had leached to a depth of 2.5 cm below the soil surface after 15 months. Leaching of the CDDs through the soil was primarily associated with carriers such as petroleum oil (Orazio et al. 1992).

Most CDDs entering surface waters are associated with particulate matter (dry deposition of atmospheric particles) and eroded soil particulates contaminated with CDDs (Hallett and Brooksbank 1986). In the aquatic environment, significant partitioning of CDDs from the water column to sediment and suspended particulate organic matter may occur. Dissolved CDDs will partition to suspended solids and dissolved organic matter (detritus, humic substances) and are likely to remain sorbed once in the aquatic environment. From suspended sediment and water data collected from the Niagara River on the New York-Canada border, it was found that CDDs were strongly associated with suspended sediment (Hallett and Brooksbank 1986). Concentrations of total TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD in raw water ranged from below detection limits to 3.6 pg/L (3.6 ppq), while the concentration of these same homologue groups in suspended sediments ranged from below detected limits to 228 pg/g (ppt) (Hallett and Brooksbank 1986). The more highly chlorinated congeners (HxCDD, HpCDD, and OCDD) predominated in both water and suspended sediment samples.

A model has been developed to describe the vertical transport of low-volatility organic chemicals in soil (Freeman and Schroy 1986). The model was used to make predictions on the transport of 2,3,7,8-TCDD at the Eglin Air Force Base Agent Orange biodegradation test plots (Freeman and Schroy 1986). Trenches 10 cm deep were dug in the soil, and Agent Orange containing 40 ppb of 2,3,7,8-TCDD was applied to the trench bottom. The model predicted a vertical movement of 2,3,7,8-TCDD, buried in 1972, through the soil column. Soil-column-profile data confirm the vertical movement of 2,3,7,8-TCDD from core samples taken in 1984 (Freeman and Schroy 1986). The 2,3,7,8-TCDD in the Eglin Air Force Base biodegradation plots moved through the entire 10 cm of the soil column in 12 years (Freeman and Schroy 1986). The rates of migration and loss of 2,3,7,8-TCDD in contaminated soil were studied under natural conditions in experimental plots at the Dioxin Research Facility, Times Beach, Missouri (Yanders et al. 1989). The

TCDD concentration profiles of sample cores taken at Times Beach in 1988 (mean range 78–160 ppb) were virtually the same as those in cores taken in 1984 (mean range 76–162 ppb). The results show that little movement and essentially no loss due to volatilization of 2,3,7,8-TCDD had occurred in the experimental plots in the four years since the Dioxin Research Facility was established (Yanders et al. 1989).

CDDs are characterized by low water solubilities and high lipophilicities. K_{ow} values range from 10^4 to 10^{12} for MCDD through OCDD, with K_{ow} values increasing relative to increasing chlorination (Table 3-2). Because of these physicochemical properties, CDDs are expected to adsorb to bedded and suspended sediments and to bioaccumulate in aquatic organisms.

The bioconcentration factor (BCF) is the ratio of the concentration of CDDs in an organism over the concentration of CDDs in water. The BCF values for CDDs can be estimated from their K_{ow} values, and a number of regression equations are available for this purpose (Bysshe 1990). Experimentally measured BCFs for selected CDD congeners in various aquatic species are summarized in Table 5-3. Measurements of the bioconcentration of CDDs tend to increase with the degree of chlorination up to TCDDs, and then decrease as chlorination continues to increase up to the OCDD congener (Loonen et al. 1993). The more highly chlorinated congeners, such as OCDD, appear to have the lowest bioconcentration potential either because they are less bioavailable because of their rapid adsorption to sediment particles (Servos et al. 1989a, 1989b) or because their large molecule size may interfere with transport across biological membranes (Bruggeman et al. 1984; Muir et al. 1986a, 1986b).

The hydrophobic nature of CDDs, combined with their great affinity for organic carbon, suggests that a major proportion of CDDs in the aquatic environment is sorbed to organic matter and sediment. Because only a minute fraction of CDDs are dissolved in the natural environment, bioconcentration is not the primary route of exposure for most aquatic organisms. Whereas the term bioconcentration is defined as the uptake of a chemical from water only, the term bioaccumulation refers to the combined uptake of a chemical from both dietary sources (e.g., food) and water. A bioaccumulation factor (BAF) that includes the ingestion route of uptake can be calculated based on fish uptake from water, food, and sediment (Sherman et al. 1992).

The primary route of exposure to CDD congeners for lower trophic organisms (e.g., phytoplankton and various aquatic invertebrates) is uptake from the water column or from interstitial water (between sediment

Table 5-3. Bioconcentration Factors (BCFs) for Aquatic Organisms

Organism	Congener	Exposure period (days)	Media	BCF	References
Aquatic plants					
Oedogonium cardiacum *Elodea nuttali* *Ceratophylum demeusum*	2,3,7,8-TCDD	1–50	Water/sediment	208–2,083	Isensee 1978; Tsushimoto et al. 1982; Yockim et al. 1978
Invertebrates					
Physa sp. *Helosoma* sp. *Daphnia magna*	2,3,7,8-TCDD	1–32	Water/sediment	702–7,125	Isensee 1978; Yockim et al. 1978
Chironomus sp. *Hexagenia* sp. *Paragnetina* sp. *Pteronarcys* sp. *Acroneuria* sp.	1,3,6,8-TCDD	4	Water/sediment	1,375–18,439 (sand) 304–111,345 (silt)	Muir et al. 1983
Chironomus sp. *Hexagenia* sp. *Paragnetina* sp. *Pteronarcys* sp.	OCDD	4	Water/sediment	173–2,854 (sand) 331–2,296 (silt)	Muir et al. 1983
Fish					
Carp (*Cyprinus carpio*)	2,3,7,8-TCDD	71	Water	66,000	Cook et al. 1991
Rainbow trout fry (*Oncorhynchus mykiss*)	1,2,3,7-TCDD 1,3,6,8-TCDD 1,2,3,4,7-PeCDD 1,2,3,4,7,8-HxCDD 1,2,3,4,6,7-HpCDD OCDD	5	Water	874–1,577 1,400–2,938 810 1,715–2,840 1,059–1,790 34–136	Muir et al. 1986a, 1986b

Table 5-3. Bioconcentration Factors (BCFs) for Aquatic Organisms (continued)

Organism	Congener	Exposure period (days)	Media	BCF	References
Fathead minnow (*Pimephales promelas*)	1,2,3,7-TCDD 1,3,6,8-TCDD 1,2,3,4,7-PeCDD 1,2,3,4,7,8-HxCDD 1,2,3,4,6,7-HpCDD OCDD	5	Water	2,018–2,458 5,565–5,840 1,200–1,647 2,630–5,834 513–515 2,226	Muir et al. 1986a, 1986b
Fathead minnow (*Pimephales promelas*)	2,3,7,8-TCDD	71	Water	128,000	Cook et al. 1991
Fathead minnow (*Pimephales promelas*)	2,3,7,8-TCDD 2,3,7,8-TCDD		Water/sediment Water/sediment	2,500 5,800	Tsushimoto et al. 1982 Adams et al. 1986
Mosquitofish (*Gambusia affinis*)	OCDD	104	Experimental lake	>9,000	Servos et al. 1989b
White sucker (*Catostomus commersoni*)	2,3,7,8-TCDD		Water/sediment	4,875	Yockim et al. 1978

BCF = bioconcentration factor; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

particles). Certain benthic organisms accumulate highly lipophilic compounds (e.g., PCBs and CDDs/CDFs) from water at the water/sediment interface (the concentration of a lipophilic compound is generally higher at this interface than in the water column) and via intake of phytoplankton, zooplankton, and suspended particulate materials that contain higher concentrations of these chemicals than the surrounding water (Porte and Albaiges 1993; Pruell et al. 1993; Secor et al. 1993). For the higher trophic level organisms, such as foraging fish, predaceous fish, and piscivorous wildlife, the predominant route of exposure is via food chain transfer, with negligible contributions from CDDs in water and sediment (Muir and Yarechewski 1988). Exposure through direct consumption of CDD-contaminated sediment and detritus may occur in some bottom-feeding species such as carp and white suckers (Kuehl et al. 1987a, 1987b; Servos et al. 1989a, 1989b). Under natural conditions, in which a high proportion of these hydrophobic CDD compounds are sorbed to suspended and dissolved organic matter, direct uptake of these CDDs from water is not expected to be substantial (Muir et al. 1986a, 1986b). The estimated BCFs in such cases may not be a good indicator of the experimental bioaccumulation measured in the field. Another reason for the difference between estimated BCFs and experimentally measured bioaccumulation values is the ability of some aquatic organisms to metabolize and eliminate specific CDD congeners from their bodies and thereby change the congener profile pattern in their tissues.

Preferential bioconcentration and bioaccumulation of 2,3,7,8-TCDD and other 2,3,7,8-substituted CDDs by aquatic organisms have been reported (Branson et al. 1985; Kuehl et al. 1985, 1987a, 1987b, 1987c; Opperhuizen 1986; Paustenbach et al. 1992). In water-only exposure studies, BCF values for fish exposed to 2,3,7,8-TCDD ranged from 37,900 to 128,000 (Cook et al. 1991; Mehrle et al. 1988). Much lower BCF values ranging from 1,400 to 5,840 and 34 to 2,226 have been reported for fish exposed to 1,3,6,8,-TCDD and OCDD, respectively (Muir et al. 1986a, 1986b). These BCF values are approximately two orders of magnitude less than would be predicted using the K_{ow} values. Similarly, the lower BCFs for HpCDD in fathead minnows and OCDD in rainbow trout fry relative to the other CDDs tested resulted from lower uptake efficiencies from water. Elimination half-lives for TCDDs and PeCDDs were similar and rapid, averaging about 2.6 days in trout fry and 3 days in minnows. Elimination half-lives for HxCDD and HpCDD were longer, averaging about 16 days in rainbow trout and 20 days in fathead minnows (Muir et al. 1986b). The results of these studies also indicate that BCFs of the higher chlorinated CDDs (HxCDD, HpCDD, OCDD) from water are much lower than would be predicted based on their K_{ow} values. Servos et al. (1989a, 1989b) also noted that the BCF values were less than predicted based on the K_{ow} values, and these authors suggest that BCFs reported in the literature may underestimate the true BCF, unless the BCFs were calculated using truly dissolved CDD concentrations in the water column rather than

total dissolved concentrations, which would include complexes with large molecules of dissolved organic carbon.

BCF values measured in fish exposed to both water and sediment were much lower than equivalent exposures to water only and ranged from 2,500 to 5,800 (Adams et al. 1986; Cook et al. 1991; Tsushimoto et al. 1982) (Table 5-3). Loonen et al. (1993) also reported that bioaccumulation of CDDs was reduced in the presence of sediment and that the effects of sediment increased with increasing hydrophobicity (degree of chlorination) of the congeners. BCFs were reduced by 15–82% for various CDD/CDF congeners, with the greatest reduction associated with OCDD.

The bioavailability of CDDs/CDFs from municipal incinerator fly ash and sediment to freshwater fish has been studied in experimental situations. Like the BCF and BAF values, the biota-sediment-accumulation factor (BASF) (ratio of contaminant concentration in the organism normalized to lipid content to the concentration in fly ash or sediment, normalized to organic carbon content) generally decreased with an increasing degree of chlorination (Kuehl et al. 1985, 1987b, 1987c). The BASF values for benthic (bottom-dwelling) fish (e.g., carp, catfish) are generally higher than for those pelagic (water column) species (e.g., bass, trout, sunfish) because of the higher lipid content and increased exposure to contaminated sediments for the benthic species (Paustenbach et al. 1992).

Several authors have studied the disposition and metabolism of CDDs in fish. Studies on the disposition of 2,3,7,8-TCDD in rainbow trout and yellow perch indicate that fatty tissues (visceral fat, carcass, skin, and pyloric caeca) typically contain the bulk of 2,3,7,8-TCDD (78–90%) with only a small percentage (2–5%) associated with the skeletal muscle (Kleeman et al. 1986a, 1986b). For other congeners, such as 1,3,6,8-TCDD and OCDD, the greatest proportion of the total body burden is concentrated in the bile, with lesser concentrations in liver > caeca > kidney > spleen > skin > muscle (Muir et al. 1986a, 1986b). Differences in the distribution among various species may be a function of the exposure pathway (i.e., dietary versus water uptake) and differences in metabolic breakdown rates. For example, both the parent compound and metabolites of 2,3,7,8-TCDD and 1,3,6,8-TCDD were present in the bile of fish exposed under laboratory conditions (Branson et al. 1985; Muir et al. 1986a, 1986b). Kleeman et al. (1986b) reported the presence of several polar metabolites in the gall bladder of yellow perch exposed to a single dose of ^{14}C- 2,3,7,8-TCDD. One week later, the gall bladder, skin, skeletal muscle, and kidneys were removed. In contrast to liver, muscle, and kidney where the parent compound accounted for 96–99% of the extractable ^{14}C, the gall bladder contained almost entirely 2,3,7,8-TCDD metabolites, at least one of which

was a glucuronide conjugate. Although the metabolic breakdown was slow, it is clear that CDDs can be transformed by fish to polar metabolites that are subsequently excreted in the bile.

Freshwater aquatic invertebrates have been shown to bioaccumulate CDDs/CDFs through water, sediment, and food pathways (Isensee 1978; Muir et al. 1983; Yockim et al. 1978). The range in experimentally determined BCF values for freshwater invertebrates is presented in Table 5-3. As discussed previously, exposure to CDDs from sediment and water containing dissolved organic material markedly decreases the BCF values, especially for the more highly chlorinated CDDs. Sediment-dwelling organisms (e.g., *Chironomous sp.* larvae and *Hexagenia sp.* nymphs), stoneflies, and other predaceous nymphs showed poor accumulation of OCDD in comparison to 1,3,6,8-TCDD (Muir et al. 1983). The lower bioaccumulation of OCDD was attributed to greater adsorption of the OCDD onto sediment particles and organic matter, and the reduced uptake across biological membranes due to large molecular size. The potential ingestion of sediments during burrowing activities by sediment-dwelling insects was believed to result in greater tissue concentrations of CDDs than those observed for predaceous insects. It is also possible that predaceous insects may metabolize 1,3,6,8-TCDD more effectively, leading to a greater rate of elimination. Sediment-dwelling organisms are important food sources for fish and other predaceous insects; consequently, if rapid elimination of 1,3,6,8-TCDD and low accumulation of OCDD occur in the natural environment, bioaccumulation of these congeners in trophically higher-level organisms may not be significant (Muir et al. 1983).

Marine invertebrates have also shown an ability to bioaccumulate CDDs/CDFs to varying degrees in their tissues (Brown et al. 1994; Cai et al. 1994; Conacher et al. 1993; Hauge et al. 1994; Rappe et al. 1991), although no information on BCF values was found in the literature. Interestingly, several investigators have reported that shellfish species (crustaceans and molluscs) are better indicators of CDD/CDF contaminant levels than fish because their tissues contain larger numbers and higher residues of CDD/CDF congeners in addition to the 2,3,7,8-TCDD congeners and other 2,3,7,8-substituted congeners that are selectively accumulated in fish species (Brown et al. 1994; Conacher et al. 1993; Rappe et al. 1991). This is in contrast to what is observed in fish and fish-eating birds, in which there is selective retention of congeners with the 2,3,7,8-substitution positions occupied, which may be due to an increased ability to metabolize and eliminate non-2,3,7,8-substituted CDD/CDF congeners (Brown et al. 1994; Rappe et al. 1991). The use of shellfish species as target organisms in CDD/CDF-monitoring studies is recommended as these species provide a better overall representation of both the magnitude and congener-specific nature of the environmental contamination (Petreas et al. 1992). Conacher et al. (1993) present an example where

use of a shellfish species provides a much higher estimate of exposure to CDDs/CDFs as well as to total CDD equivalent toxicity (TEQs) than use of a fish species. This difference in congener bioaccumulation profiles between fish and shellfish species is a result of the ability of fish to metabolize CDDs/CDFs. Both the parent congeners and metabolites of 2,3,7,8-TCDD and 1,3,6,8-TCDD were present in the bile of fish exposed under laboratory conditions (Branson et al. 1985; Muir et al. 1986a). Kleeman et al. (1986a, 1986b) reported the presence of several polar metabolites, including glucuronide conjugates, in various fish exposed to 2,3,7,8-TCDD. Despite the slowness of the metabolic breakdown processes, it is clear that CDDs can be transformed within fish to polar metabolites that are subsequently excreted with the bile. It does not appear from the results obtained in studies conducted to date that shellfish species have the same ability to metabolize and eliminate non-2,3,7,8-substituted CDDs/CDFs (Brown et al. 1994; Cai et al. 1994).

It is apparent from the available data regarding the substantial bioaccumulation potential of CDDs/CDFs in aquatic organisms (particularly the 2,3,7,8-substituted congeners) as well as data on the extent of contamination of fish and shellfish in various freshwater and marine waterways, that ingestion of contaminated fish and shellfish is an important exposure pathway for CDDs/CDFs in humans.

CDDs have been found to accumulate in both surface and rooted aquatic vegetation, with BCF values ranging from 208 to 2,083 (Table 5-3) (Isensee 1978; Tsushimoto et al. 1982; Yockim et al. 1978). Corbet et al. (1983) reported that a rooted plant species (*Potemageton pectimatus*) and a surface-dwelling duckweed (*Lemna* sp.) accumulated concentrations of 1,3,6,8-TCDD of 280 and 105 ng/g (dry weight), respectively, following exposure to water containing 1,000 ng/L (ppt). The maximum concentrations were observed 8 days post-application and represented 6% of the total TCDD applied. These results are similar to those reported by Tsushimoto et al. (1982) in an outdoor pond study, in which a maximum bioaccumulation of 2,3,7,8-TCDD in the pond weeds *Elodea nuttali* and *Ceratophyllon demersum* equivalent to a BCF of 130 occurred after 5 days of exposure. In both studies, the tissue concentrations reached equilibrium in approximately 20 days and remained constant until the end of the experiment (approximately 58 and 170 days, respectively). These experimental data indicate that CDDs can accumulation in aquatic plant species through waterborne exposure.

Like many fish, several species of fish-eating birds have shown the ability for preferential bioaccumulation of 2,3,7,8-TCDD and other 2,3,7,8-substituted CDDs and TCDFs. Jones et al. (1994) monitored TEQ values for 2,3,7,8-TCDD in double-crested cormorants from three of the Great Lakes: Superior, Michigan,

and Huron. Biomagnification factors (BMF, the ratio of the concentration of TCDD-equivalents in bird eggs to concentrations in forage fish) were found to range from 11.7 to 56.8 (mean, 31.3). In another study, all of the CDDs and CDFs detected in double-crested cormorant and Caspian tern eggs were 2,3,7,8-substituted (Yamashita et al. 1992). Concentrations of 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,4,7,8-HXCDD, 1,2,3,6,7,8-HXCDD, 1,2,3,7,8,9-HXCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD ranged from 5.3 to 20, 3.2 to 9.4, 10 to 20, 3.6 to 11, and 7.8 to 16 pg TEQ/g, respectively, for double-crested cormorant eggs, and 8.2 to 22, 3.3 to 6.4, 8.7 to 17, 2.4 to 6.0, and 9.7 to 21 pg TEQ/g, respectively, for Caspian tern eggs. This same pattern was also reported to occur in California peregrine falcons and their eggs (Jarman et al. 1993). For this species, mean concentrations of 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 1,2,3,4,7,8-HxCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, 1,2,3,4,6,7,8-HpCDD and OCDD in eggs were 5.7, 11, 2, 11, 1.3, 3.8, and 5.3, respectively. Fish-eating birds are exposed to CDDs primarily through their diet. A rapid decline in contaminant levels in eggs of fish-eating birds, therefore, reflects a rapid decrease in contaminant levels of their prey. This has been shown to occur in Great blue heron chicks in British Columbia (Sanderson et al. 1994) in areas where CDD/CDF levels in pulp and paper mill effluents decreased substantially within a few years. The Great blue heron chicks also showed an increased hepatic microsomal ethoxyresorufin O-deethylase (EROD) activity in the areas of highest contamination. This indicates that the induction of cytochrome P-450 1A1 has occurred, and that the Ah-receptor-mediated process, by which 2,3,7,8-TCDD and related chemicals exert their toxicities, has been activated.

Ankley et al. (1993) studied the uptake of persistent polychlorinated hydrocarbons by four avian species at upper trophic levels of two aquatic food chains. Concentration of 2,3,7,8-TCDD toxic equivalents (TEQs) were evaluated in Forster's tern and common tern chicks and in tree-swallow and red-winged-blackbird nestlings from several areas in the watershed. Young birds accumulated small concentrations of 2,3,7,8-TCDD and several other 2,3,7,8-substituted CDDs and CDFs, including 1,2,3,6,7,8-HxCDD, 2,3,7,8-TCDF, 1,2,3,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDF, 1,2,3,7,8-PeCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD. The general trend in concentrations of CDDs from the greatest to least was Forster's tern - common tern > tree swallow > red-winged blackbird. The similarity in concentrations between the two tern species is expected given that they are both piscivores and their similar life histories and the close proximity of the two colonies. The greater concentrations in the tree swallows than in the red-winged blackbirds were somewhat unexpected given the presumed similarity of the diets (both species are insectivores). The authors suspect that the red-winged blackbirds foraged more on relatively uncontaminated upland food sources than the tree swallows, which fed primarily on chironomids emerging from the bay.

2,3,7,8-TCDD is generally considered to be bioavailable to terrestrial birds primarily through ingestion of TCDD-laden food items and soil particles (Nosek et al. 1992). These authors, using H^3TCDD-administered suspensions in various environmental matrices, found that 30% of the dose absorbed from suspensions of earthworms, 33% absorbed from soil suspensions, 41% absorbed from suspensions of paper mill sludge solids, and 58% absorbed from a suspension of crickets. These authors also reported that the percentage of the cumulative TCDD dose translocated to an individual egg was 1.1% for the first 15 eggs laid and that the percentage was not affected by the order in which the eggs were laid. Assuming an adult female could lay 30 eggs, 35% of the hen TCDD body burden could be translocated to all eggs laid. Results of these studies suggest that TCDD can be orally bioavailable from earthworms and crickets, important dietary sources for this species and other terrestrial species, as well as from nonfood items such as orally ingested soil and paper mill sludge solids.

For terrestrial mammals, the BCF value is the quotient of the concentration of CDD in the tissues divided by the concentration in food (Geyer et al. 1986a, 1986b). BCF values for 2,3,7,8-TCDD were calculated in the liver and/or fat of rats, cows, and monkeys (Geyer et al. 1986a; Kociba et al. 1978a). BCF values ranged from 10.9 to 24.5 in liver tissue and from 3.7 to 24.5 in fat tissue of rats fed 2,200, 210, or 22 ng/kg of 2,3,7,8-TCDD in their diet for 2 years (Geyer et al. 1986a; Kociba et al. 1978a). The BCF value calculated for this rat study, increased as the concentration in the animals' food decreased. In a cattle-feeding study, 24 ng 2,3,7,8-TCDD in the diet was fed to cows for 28 days after which time the BCF of 2,3,7,8-TCDD in the liver was 0.7 and in the fat was 3.5. Using a linear one compartment model, Geyer et al. (1986a) calculated that a steady state would be reached in 499 days and that the cattle fatty tissue would contain 594 ng/kg. The calculated BCF value for 2,3,7,8-TCDD would then be 24.8 (Geyer et al. 1986a; Jensen et al. 1981). This value is in good agreement with the BCF of 24.5 calculated for rats that received 22 ng TCDD/kg in their diet for years. This is a much higher BCF than has been reported by Fries and Paustenbach (1990). After 4 years of chronic exposure to 25 ng/kg 2,3,7,8-TCDD in their diet, the calculated BCF in fatty tissue of monkeys ranged from 24 to 40 (Geyer et al. 1986a). Using the 2,3,7,8-TCDD concentration in human adipose tissue (10.7 ppt whole weight) and in food (0.052–0.103 ng/kg), the calculated BCF is between 104 and 206 on a whole-weight basis, or between 115 and 229 on a lipid basis (90% lipid) (Geyer et al. 1986a). Using a pharmacokinetics model, the calculated BCF value is 153 (Geyer et al. 1986a). The authors further point out that the calculated BCFs for 2,3,7,8-TCDD in human adipose tissue are of the same order of magnitude as those calculated for PCBs, DDT, and hexachlorobenzene which are also persistent compounds with comparable lipophilicity (n-octanol/water partition coefficients). Based on this BCF range, 2,3,7,8-TCDD was ranked as having a

high bioconcentration potential in human adipose tissue (Geyer et al. 1986b). The half-life in humans was estimated to be approximately 7 years (Pirkle et al. 1989).

The primary mechanisms by which CDDs enter terrestrial food chains are by atmospheric wet and dry deposition of vapor-phase and particulate-bound chemicals (McCrady and Maggard 1993). Uptake of CDDs from soils by vegetables and other plants may occur (Schroll and Scheunert 1993). Accumulation of CDDs on vegetation may involve both of these mechanisms. Since 2,3,7,8-TCDD is lipophilic, adsorbs strongly to soil, and is not very soluble in water, root uptake and translocation to upper plant parts is only a minor source of vegetative contamination (Travis and Hattemer-Frey 1987) except perhaps for plant species belonging to the *Cucurbitaceas* (e.g., zucchini and pumpkin). For zucchini and pumpkin plants, root uptake of CDD/CDFs and subsequent translocation to the shoots and into the fruits is a main contamination pathway (Hulster et al. 1994). Hulster and Marschner (1993) reported that CDD levels in foliage were not related to CDD levels in soil. The contamination of plant foliage via atmospheric deposition is a more important contamination mechanism than root uptake and translocation to plant foliage (McCrady et al. 1990). Welschpausch et al. (1995) determined that dry deposition was the main pathway of uptake in grass of CDDs/CDFs from the atmosphere. Particles <2.9 μm in diameter were not important in atmospheric deposition, but large particles may contribute to HpCDD and OCDD accumulation. McCrady et al. (1990) conducted experiments with plants growing in nutrient solutions containing TCDD in a closed-laboratory system. These authors demonstrated that translocation from roots to shoots did not occur, but shoot contamination was associated with foliar uptake from the air. In general, there is little bioaccumulation of CDDs in plants (Hutzinger et al. 1985). BCFs for TCDD in plants have been estimated to be 0.0002, although most absorption occurs in the plant root with little or no translocation through the plant to the foliage (Wild and Jones 1992). A concentration of 0.06 ppm 2,3,7,8-TCDD was applied to the soil and root uptake from soil was then measured in oats and soybeans (Kearney et al. 1971). Oat and soybean plants (at all growth stages) accumulated very small quantities of 2,3,7,8-TCDD. A maximum of 0.15% (0.12 ppm) of 2,3,7,8-TCDD present in soils was translocated to the aerial portion of the oat and soybean plants. No detectable amounts of the compound were found in the oat or soybean plants harvested at maturity. The amount of 2,3,7,8-TCDD applied to these soils was many thousands of times greater than that which would occur in soils from herbicide applications containing a few ppm of 2,3,7,8-TCDD as an impurity. Even upon exposure to these high concentrations in the soil, significant amounts of 2,3,7,8-TCDD could not be measured in the plants (detection limit not reported) (Kearney et al. 1971).

Maize (corn) and bean cultivations grown in soils spiked with 22–1,066 ppt 2,3,7,8-TCDD showed 2,3,7,8-TCDD concentrations in roots ranging from 16 to 1,278 ppt for maize and from 37 to 1,807 for beans (Fachetti et al. 1986). The soil-grown crops did not show a significant increase of 2,3,7,8-TCDD in above-ground parts, either as a function of time or with increasing concentration of the pollutant in the soil (Fachetti et al. 1986).

Uptake of ^{14}C-labeled OCDD was studied in a closed, aerated-soil plant system for 7 days after application of the OCDD to soil (Schroll et al. 1994). The BCF (concentration of ^{14}C equivalent to the OCDD in plant dry matter divided by ^{14}C-labeled OCDD in dry soil) was 0.742 in carrot root and 0.085 in carrot shoots grown on OCDD-contaminated soil as compared to a BCF of not determinable and 0.084 in the control carrot root and shoots, respectively. There was no transport of ^{14}C-labeled OCDD between the roots and shoots or vice versa. The residues in roots were due only to root uptake from the soil; those in shoots were due only to foliar uptake from the air.

Muller et al. (1993) studied transfer pathways of CDD/CDFs to fruit. These authors found that homologue patterns of CDDs/CDFs in soil were different from those in both apples and pears grown in the contaminated soil. Concentrations of CDDs/CDFs ranged from 1 to 4 ng/kg (fresh weight) and were 4–8 times higher in the peel than in the pulp. These authors suggest that airborne CDDs/CDFs are a major source of contamination of fruits grown in contaminated soil. Muller et al. (1994) conducted field studies of CDD transfer pathways from soil to several edible plant varieties (carrots, lettuce, and peas). Plants were grown in soil with 5 ng TEQ/kg or total CDD/CDF concentrations of 363 ng/kg dry weight (control plots) and 56 ng TEQ/kg or total CDD/CDF concentrations of 3,223 ng/kg dry weight on the contaminated plots. CDD/CDF concentrations in carrot peels were three times higher on the contaminated plots than on the control plots. This was the result of a 10-fold increase in the CDD/CDF levels in the carrot peel. CDD/CDF concentrations in lettuce (17.7 and 21.1 ng/kg dry weight) and in peas (7.1 ng/kg dry weight) were not any higher when grown on the contaminated plot as compared to the control plots and were much lower than concentrations in the carrots (47.3 and 47.5 ng/kg, dry weight). This indicates that the CDD/CDFs in the lettuce and peas from both plots were of atmospheric origin. The CDD/CDF homologue pattern in the contaminated soil showed OCDFs and HpCDFs were the two most prevalent congeners, while the CDD/CDF homologue pattern from the peel of carrots grown on the contaminated plots contained TCDF, PeCDF, and HxCDF. Levels of TCDD were the lowest of all CDD/CDF homologues in both contaminated soils and carrot peels. The homologue profile in lettuce samples was largely dominated by lower chlorinated CDFs (TCDF and PeCDF) and higher chlorinated CDDs (HpCDD and OCDD), a

profile often found in samples of atmospheric deposition (Eitzer and Hites 1989a, 1989b). The lowest CDD/CDF levels of this study were found in peas with pea pods showing higher levels than seeds. The homologue profiles was dominated by lower chlorinated CDFs and higher chlorinated CDDs similar to the profile found in lettuce.

Since most of the CDDs released into the atmosphere settle onto water and soil surfaces, foliar deposition is the major route of vegetative contamination (Travis and Hattemer-Frey 1987). The translocation of foliar-applied 2,3,7,8-TCDD has been studied (Kearney et al. 1971). Labeled 2,3,7,8-TCDD was applied to the center leaflet of the first trifoliate leaf of 3-week-old soybean plants and the first leaf blade of 12-day-old oat plants. The compound was applied in an aqueous surfactant solution to enhance leaf adsorption and to keep the water-insoluble TCDD in solution. Plants were harvested 2, 7, 14, and 21 days after treatment, dissected into treated and untreated parts, and analyzed. 2,3,7,8-TCDD was not translocated from the treated leaf to other plant parts. Very little 2,3,7,8-TCDD was lost from soybean leaves, while a gradual loss (38% in 21 days) did occur from oat leaves (Kearney et al. 1971). The authors considered volatilization to be a possible mechanism for removal of 2,3,7,8-TCDD, but photolysis may also have contributed to the loss.

McCrady and Maggard (1993) measured the uptake and elimination mechanisms for 2,3,7,8-TCDD applied to grass foliage in a closed-laboratory system using [^{3}H]TCDD. The [^{3}H]2,3,7,8-TCDD was injected into the chamber as a vapor originating from a [^{3}H]2,3,7,8-TCDD generator. The total recovered radioactivity was 74%. Plant foliage accounted for 59% and the air and other chamber components accounted for 6 and 9%, respectively. This indicated that plant foliage was a major sink for [^{3}H]2,3,7,8-TCDD vapor. Less than 0.2% was recovered from the soil and associated with root tissues, further verifying an airborne mechanism of [^{3}H]2,3,7,8-TCDD uptake and negligible translocation. The authors also demonstrated that both photodegradation and volatilization were primary loss mechanisms for [^{3}H]2,3,7,8-TCDD. The photodegradation half-life (first-order kinetics) of 2,3,7,8-TCDD sorbed to grass and exposed to natural sunlight was 44 hours, while the half-life for volatilization of 2,3,7,8-TCDD from grass foliage was 128 hours.

In conclusion, CDDs may be transported long distances in the atmosphere. They eventually may be deposited on soils or surface water as a result of wet or dry deposition. CDDs will slowly volatilize from the water column or, more likely, will adsorb to suspended particulate materials in the water column and be transported to the sediment. CDDs deposited on soils will strongly adsorb to organic matter. They are

unlikely to leach to underlying groundwater, but may enter the atmosphere on soil or dust particles or enter surface water in runoff. Low water solubilities and high lipophilicity indicate that CDDs will bioconcentrate in aquatic organisms, although as a result of their binding to suspended organic matter, actual uptake by these organisms may be less than predicted. This is also true of uptake and bioconcentration by plants, although foliar deposition and adherence may be significant.

5.3.2 Transformation and Degradation

CDDs belong to a class of highly lipophilic compounds with low water solubility and low chemical reactivity that are resistant to microbial degradation. The dominant transformation processes affecting their fate have been shown to be surface photolysis and gas-phase diffusion/volatilization with subsequent photolysis (Yanders et al. 1989).

5.3.2.1 Air

The primary transformation reaction for CDDs in the atmosphere depends on whether the CDD is in the vapor or particulate phase. Vapor-phase CDDs are not likely to undergo reactions with atmospheric ozone, nitrate, or hydroperoxy radicals; however, reactions with hydroxyl radicals may be significant, particularly for the less-chlorinated congeners (MCDD through TCDD) (Atkinson 1991). Based on the photolysis lifetimes of CDDs in solution, it is expected that vapor-phase CDDs will also undergo photolysis in the atmosphere, although reactions with hydroxyl radicals will predominate. For TCDD, the photolytic lifetime ranges from 1.3 to 7.1 days, depending on the season (faster in summer), whereas the hydroxyl radical reaction lifetime is estimated to be 2 days (Atkinson 1991). A half-life of 8.3 days was estimated for the gas-phase reaction of 2,3,7,8-TCDD with photochemically produced hydroxyl radicals in the atmosphere (Podoll et al. 1986). Using the gas-phase hydroxyl radical reaction rate constant of 1×10^{-11} cm^3-$molecule^{-1}$ sec^{-1} and an average 12-hour daytime hydroxyl radical concentration of 1.5×10^6 molecules cm^{-3}, the atmospheric lifetimes of CDDs are estimated to range from 0.5 days for MCDD to 9.6 days for OCDD, with TCDD having a lifetime of 0.8–2 days (Atkinson 1991).

Particulate-bound CDDs are removed by wet or dry deposition with an atmospheric lifetime $10 days (Atkinson 1991) and, to a lesser extent, by photolysis. Miller et al. (1987) measured photolysis of 2,3,7,8-TCDD sorbed onto small-diameter fly ash particulates suspended in air. The results indicated that fly ash confers photostability to the adsorbed 2,3,7,8-TCDD. The authors reported little (8%) to no loss of

2,3,7,8-TCDD on the fly ash samples after 40 hours of illumination in simulated sunlight. Koester and Hites (1992) studied the photodegradation of CDDs naturally adsorbed to five fly ash samples (two from coal-fired plants, two from municipal incinerators, and one from a hospital incinerator). Although the authors reported that CDDs underwent photolysis in solution and on silica gel, no significant degradation was observed in 11 photodegradation experiments conducted for periods ranging from 2 to 6 days.

The selected transformation of the more and less chlorinated CDDs has been demonstrated by the analysis of CDDs found in soil samples compared with atmospheric concentrations of CDDs at the emission source (Marklund et al. 1991; Yamamoto and Fukushima 1993). Soil samples contained progressively greater concentrations of HpCDD and OCDD with increasing distance from the emission source, indicating that photolysis of the less chlorinated congeners was occurring (Eitzer 1993). In the air, the low vapor pressure of OCDD results in its partitioning primarily to the particulate phase rather than the vapor phase; therefore, atmospheric photodegradation is less likely to occur for this tightly bound congener (Eitzer 1993).

5.3.2.2 Water

Photolysis is the major route of CDD disappearance in aqueous solutions (Hutzinger et al. 1985). While photolysis is a relatively slow process in water, CDDs are rapidly photolyzed under certain conditions, (i.e., when exposed to ultraviolet light of the appropriate wavelength and in the presence of an organic hydrogen donor). These hydrogen donors can be expected to be present in chlorophenol pesticides either as formulation solvents (e.g., xylene or petroleum hydrocarbons), as active constituents of the formulation (e.g., the alkyl esters of 2,4-D and 2,4,5-T), or as natural organic films on soils (Crosby et al. 1973). The photolytic behavior of CDDs in an organic solvent or in a water-organic solvent, however, may not accurately reflect the photolytic behavior of these compounds in natural waters (Hutzinger et al. 1985). For example, Choudry and Webster (1989) reported that photolysis of 1,3,6,8-TCDD was slower in natural pond-water solutions than was predicted from studies with laboratory solutions. Conversely, Friesen et al. (1990) reported that photolysis of PeCDD and HpCDD proceeds faster in a pond or lake-water solutions than was predicted or measured in a laboratory solution. In general, however, lower chlorinated CDDs are degraded faster than higher chlorinated congeners. Chlorine atoms in the lateral positions (e.g., 2, 3, 7, 8) are also more susceptible to photolysis than are chlorine atoms in the para positions (e.g., 1, 4, 6, 9) (Choudhry and Hutzinger 1982; Crosby et al. 1973; Hutzinger et al. 1985).

Podoll et al. (1986) used the quantum yield data of Dulin et al. (1986) for a water:acetonitrile solution to calculate seasonal half-life values for dissolved 2,3,7,8-TCDD at 40 degrees north latitude in clear near-surface waters. Photolysis half-lives for dissolved 2,3,7,8-TCDD in sunlight range from 118 hours in winter, to 51 hours in fall, to 27 hours in spring, to 21 hours in summer (Podoll et al. 1986). Choudhry and Webster (1989) studied photolysis of a series of CDDs in a water:acetonitrile solution (2:1 v/v). These authors estimated the midday midsummer sunlight photolysis half-lives values at 40 degrees north latitude in clear near-surface waters as follows: 1,3,6,8-TCDD (0.3 days), 1,2,3,7-TCDD (1.8 days), 1,2,3,4,7-PeCDD (15 days), 1,2,3,4,7,8-HxCDD (6.3 days), 1,2,3,4,6,7,8-HpCDD (47 days), and OCDD (18 days) near the surface of water bodies (Choudhry and Webster 1989). Sunlight photolysis half-lives were also reported for the spring, fall, and winter for 1,2,3,4,6,7,8-HpCDD (57, 88, and 156 days, respectively) and for OCDD (21, 31, and 50 days, respectively) (Choudhry and Webster 1989). Photolysis half-lives for 1,2,3,4,6,7,8-HpCDD and OCDD in water-acetonitrile solutions irradiated at 313 nm were reported to be 8 and 7.7 days, respectively (Choudhry and Webster 1987, 1989). The half-lives of 1,3,6,8-TCDD and OCDD in lake water are 2.6 and 4 days, respectively, with removal by partitioning to the lake sediments (Servos et al. 1992).

The photodegradation profiles of 2,3,7,8-TCDD, 1,3,6,8-TCDD, and 1,2,3,4-TCDD in 1,4-dioxane solutions at various wavelengths under xenon lamp irradiation were studied (Koshioka et al. 1989a, 1989b, 1989c). Reductive dechlorination reactions were observed in the photolysis of TCDD isomers. After 200 minutes of irradiation with a xenon lamp, 2,3,7,8-TCDD formed 2,3,7-TrCDD, 2,7-DCDD, 2,8-DCDD, 2-MCDD, and DD. Photodegradation half-lives of 2,3,7,8-TCDD at the maximal photodegradation wavelengths of 252.6 nm and 318.6 nm were 72.6 minutes and 29.7 minutes, respectively (Koshioka et al. 1989b, 1989c). After 267 minutes of irradiation with a xenon lamp, 1,3,6,8-TCDD formed 1,3,6-TrCDD, 1,3-DCDD, 1,6-DCDD, 1-MCDD, 2-MCDD, and DD, while 1,2,3,4-TCDD formed 1,2,3-TrCDD, 1,2,4-TrCDD, 1,2-DCDD, 1,3-DCDD, 1,4-DCDD, 2,3-DCDD, 1-MCDD, 2-MCDD, and DD (Koshioka et al. 1989a).

The photolytic half-life of 2,3,7,8-TCDD in isooctane was estimated to be 40 minutes with a light source at 0.5 meters and 3 hours with a light source at 1 meter (Stehl et al. 1973). Very little change was observed in OCDD on exposure to artificial sunlight. Approximately 20% photolysis of OCDD was observed in isooctane at the end of 18 hours and about 6% photolysis of OCDD after 20 hours of exposure in 1-octanol (Stehl et al. 1973). Irradiation of pentachlorophenol (PCP) dissolved in sodium hydroxide at a wavelength

of 300 nm (equivalent to sunlight) for 16 hours produced OCDD (Crosby and Wong 1976). OCDD then underwent photoreduction to HpCDD as a PCP photolysis product (Crosby and Wong 1976).

Under equivalent light exposure conditions, photolytic half-lives were determined for each of the individual TCDD isomers in dilute hydrocarbon solution and as a diffuse molecular dispersion on a clean soft-glass surface (Nestrick et al. 1980). The photolytic behavior of 2,3,7,8-TCDD was atypical compared to other TCDD isomers. In a hydrocarbon solution, 2,3,7,8-TCDD had the fastest decomposition rate (half-life 56.8 minutes) and 1,4,6,9-TCDD had the slowest decomposition rate (half-life 8,400 minutes [5.8 days]). The half-lives of the remaining TCDD isomers ranged from 153 to 1,388 minutes (2.55–23.1 hours). However, as a diffuse molecular dispersion on a glass surface, the 2,3,7,8-TCDD had the slowest decomposition rate (half-life 8,400 minutes [5.8 days]), and 1,4,6,9-TCDD had the second slowest decomposition rate (half-life 830 minutes [13.8 hours]). The half-lives of the remaining TCDDs ranged from 121 to 560 minutes (2–9.3 hours). The majority of TCDD isomers photolytically decomposed faster on a glass surface than in a hydrocarbon solution under conditions of equivalent light intensity. 2,3,7,8-TCDD and 1,4,6,9-TCDD possess the highest degree of symmetry within the group, and these isomers demonstrated the largest change in the photodecomposition rate for surface and solution reactions, with the changes being in opposite directions. Additional photolysis tests were conducted using more highly chlorinated CDD congeners. In a hydrocarbon solution, the half-lives of 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,6,7,9-HpCDD, and OCDD were 1,800 minutes (1.3 days), 3,300 minutes (2.3 days), and 1,460 minutes (1.01 days), respectively, and 3,140 minutes (2.18 days), 2,400 minutes (1.67 days), and 48,900 minutes (33.96 days), respectively, on a glass surface (Nestrick et al. 1980).

2,3,7,8-TCDD decomposed rapidly when dissolved in methanol and exposed to ultraviolet (UV) light (Plimmer et al. 1973). Rate measurements showed that 2,3,7,8-TCDD is more rapidly photolyzed in methanol than OCDD (Plimmer et al. 1973). The photolysis half-lives for 2,3,7,8-TCDD, 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,6,7,9-HpCDD, and OCDD in *n*-hexadecane solution were 56.8 minutes, 1,800 minutes (1.25 days), 3,300 minutes (2.29 days), and 1,460 minutes (1.01 days), respectively (Mamantov 1984).

Solution-phase photolysis of HpCDD and OCDD has been reported (Dobbs and Grant 1979). Solutions of these CDDs in hexane (approximately 1 µg/mL) were exposed to natural sunlight as well as to fluorescent blacklight. The photolytic half-life for OCDD exposed to both types of radiation was 16 hours. HpCDD was generated by photolysis of OCDD (Dobbs and Grant 1979). The photolytic half-lives of

1,2,3,4,6,7,9-HpCDD and 1,2,3,4,6,7,8-HpCDD were 28 hours and 11 hours, respectively (Dobbs and Grant 1979).

It has been suggested that the potential for biological degradation of 2,3,7,8-TCDD in a wide variety of environmental samples is low (Arthur and Frea 1989). The fate of 2,3,7,8-TCDD in sediment and water from two lakes in Wisconsin was examined (Ward and Matsumura 1978). After incubation periods of up to 589 days, little metabolism of 2,3,7,8-TCDD was detected. The slight metabolism that was detected was stimulated by the presence of sediment and the addition of nutrients (Ward and Matsumura 1978). Also, 2,3,7,8-TCDD does not hydrolyze in water (Mabey et al. 1982; Miller et al. 1987).

5.3.2.3 Sediment and Soil

Photolysis of 2,3,7,8-TCDD on soils is a relatively slow process compared to photolysis in an aqueous media (Kieatiwong et al. 1990). 2,3,7,8-TCDD applied to soil or a solid surface seems to be extremely resistant to the action of sunlight and decomposes very slowly (Plimmer et al. 1973). A methanol solution of 2,3,7,8-TCDD (2.4 ppm) applied to glass plates coated with soil and illuminated 96 hours with a fluorescent UV lamp remained unchanged at the end of the period (Plimmer et al. 1973). Organic solvents added to the soil, however, can enhance the extent of photolysis. Use of a solvent mixture of tetradecane and 1-butanol to TCDD-treated soil, combined with exposure to sunlight, resulted in 61–85% photodegradation of TCDD after 60 days. The solvent was effective in transporting TCDD from deeper in the soil column (60 cm) to the soil surface via evaporation. At the soil surface, photodegradation could occur. TCDD concentrations at 60 cm decreased from 23.8 ng/g (ppb) to 7.1 ng/g (ppb) after 60 days (McPeters and Overcash 1993).

Photolysis of OCDD (10 mg/kg) on soils resulted in production of the lower chlorinated CDDs, notably 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, three HxCDD isomers substituted at the 2,3,7,8-positions, and 1,2,3,4,6,7,8-HpCDD. Photolysis of OCDD occurred in mean soil depths between 0.06 and 0.13 mm (Miller et al. 1989). Approximately 30–45% of OCDD was lost by day 5 of irradiation; no further significant loss of OCDD was observed following 10 additional days of irradiation. Although photolysis only occurred at shallow soil depths and the conversion of OCDD to the more toxic TCDD, PeCDD, and HxCDD homologues was small (0.5–1%) compared with the photodechlorination to HpCDD (67%), photolysis of OCDD may represent a significant source of these toxic isomers (Miller et al. 1989).

The loss of 2,3,7,8-TCDD in contaminated soil has been studied under natural conditions in experimental plots at the Dioxin Research Facility, Times Beach, Missouri (Yanders et al. 1989). The 2,3,7,8-TCDD concentration profiles of sample cores taken at Times Beach in 1988 were virtually the same as those in cores taken in 1984. The authors concluded that the loss of 2,3,7,8-TCDD due to photolysis at Times Beach was minimal in the 4 years covered by the study (Yanders et al. 1989). Estimates of the half-life of TCDD on the soil surface range from 9 to 15 years, whereas the half-life in subsurface soil may range from 25 to 100 years (Paustenbach et al. 1992).

A white rot fungus *(Phanerochaete chrysosporium)* has demonstrated the ability to degrade 2,3,7,8-TCDD in laboratory experiments (Bumpus et al. 1985; Des Rosiers 1986). In cultures containing 1.25 nmol of the 2,3,7,8-TCDD substrate, 27.9 pmol were mineralized to CO_2 in 30 days (2.23% metabolism) increasing to 49.5 pmol in 60 days (3.96% metabolism) (Des Rosiers 1986). It was suggested that the ability of this fungus to metabolize 2,3,7,8-TCDD is dependent on its extracellular lignin-degrading enzyme system (Bumpus et al. 1985; Des Rosiers 1986). More recently, Valli et al. (1992) reported that 2,7-DCDD also was degraded by *P. chrysosporium* via the removal of both aromatic chlorines before aromatic ring cleavage took place.

Cultures of *Pseudomonas testosteroni*, of an unidentified bacterium isolated from soil from Seveso, Italy, and of a mixture of 6 unidentified bacterial strains isolated from Seveso soil were incubated aerobically with ^{14}C-2,3,7,8-TCDD for 35, 54, and 12 weeks, respectively (Philippi et al. 1982). Results showed the occurrence of a metabolite of ^{14}C-2,3,7,8-TCDD in all three cultures. The polar metabolite amounted to approximately 1% of the input material and was found to be a hydroxylated derivative of ^{14}C-2,3,7,8-TCDD (Philippi et al. 1982).

Approximately 100 strains of pesticide-degrading microorganisms were tested for their ability to degrade 2,3,7,8-TCDD (Matsumura and Benezet 1973). The organisms were maintained in liquid axenic culture, and the production of metabolites from ring-labeled ^{14}C-2,3,7,8-TCDD was measured. Five strains were identified that showed some ability to degrade ^{14}C-2,3,7,8-TCDD. The degradative organisms included a fungus *(Trichoderma viride)*, a bacterium *(Pseudomonas putida)*, and three organisms referred to by coded numbers (Matsumura and Benezet 1973).

To determine the persistence of 2,3,7,8-TCDD, concentrations of 1, 10, and 100 ppm of unlabeled 2,3,7,8-TCDD were added to 300-g samples of silty loam and sandy soils and then assayed periodically for

residues (Kearney et al. 1971). Measurements of 2,3,7,8-TCDD residues after 20, 40, 80, 160, and 350 days of incubation at 28 EC in foil-sealed beakers indicated a relatively slow degradation process in both soils. After 350 days, 56% of the initially applied 2,3,7,8-TCDD was recovered from the sandy soil, while 63% was recovered from the silty clay loam for all concentrations (Kearney et al. 1971).

Parsons (1992) studied the influence of suspended sediment on the biodegradation of several CDDs. In this study, aqueous solutions of a mixture of 2-chloro-, 1,3-dichloro, 2,8-dichloro-, and 1,2,4-trichloro CDDs were incubated for 24 days with 100 mg/L suspended sediment. Subsequently, the degradation of the CDDs in the sediment suspensions by *Alcaligenes sp.* strain JB1 was compared to that in solutions without sediment. The amounts of all four CDD compounds degraded in the sediment suspensions after 7 days were greater than those initially present in the dissolved phase, based on their calculated sediment-water partition coefficients. The sorbed fractions were, therefore, sufficiently desorbed to be partly degraded. However, the biodegradation rates were slower in the sediment suspensions than in the solutions. The results indicate that sorbed fractions of CDDs formed after relatively short incubation periods are sufficiently labile to be available for biodegradation after desorption. Evidence that the presence of sediment lowers biodegradation rates in sediment suspension, however, implies that longer residence times, such as those observed under field conditions, may also lead to a significant lowering of the biodegradation rates in soil. This will apply even more to the more highly chlorinated CDD congeners. In another study, the degradation of highly chlorinated CDD congeners (5–7 chlorine/molecule) was studied for a period of 6 months in anaerobic microcosm incubations using PCB-contaminated Hudson River sediments and creosote-contaminated aquifer samples from Pensacola, Florida (Adriaens and Grbic-Galic 1994). The authors reported (pseudo-first-order) half-life values for 1,2,3,4,6,7,8-HpCDD of 4.1 and 2.9 years for the Hudson River and Pensacola aquifer-incubated microcosm samples, respectively. The half-life values for 1,2,3,4,7,8-HxCDD were 2 and 2.9 years for the Hudson River and Pensacola aquifer-incubated microcosm samples, respectively. The 1,2,4,6,8,9/ 1,2,4,6,7,9-HxCDD congeners were found not to be degraded, which was presumably due to the low concentration spiked. The authors reported that tentative identification of the degradation products indicate that para-dechlorination was the preferential route of reduction, as has been observed with 1,2,3,4,5,6,7,8-HpCDD in aquifer microcosms. This observation is contrary to photolytic dechlorination patterns of soil-sorbed CDDs.

Beurskens et al. (1995) reported that an anaerobic microbial consortium enriched from Rhine River sediments was able to remove chlorine substituents from CDDs. A model CDD, 1,2,3,4-TCDD, was reductively dechlorinated to both 1,2,3- and 1,2,4-TrCDD. These TrCDD compounds were further

dechlorinated to 1,3- and 2,3- DCDD and trace amounts of 2-MCDD. The TrCDD compounds were detected at low concentrations, but the 1,3- and 2,3- DCDD were detected at higher concentrations. The anaerobic culture dechlorinates 1,2,3,4-TCDD at a relatively rapid rate with a half-life value estimated at 15.5 days (first-order kinetics). The formation of metabolites with a conserved 2,3-substitution pattern from 1,2,3,4-TCDD indicates that dechlorination of highly chlorinated CDDs may result in metabolites that are potentially more toxic than the parent compounds.

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to CDDs depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. Historically, CDD analysis has been both complicated and expensive, and the analytical capabilities to conduct such analysis have been available through only a relatively few analytical laboratories. Limits of detection have improved greatly over the past decade with the use of high-resolution mass spectrometry, improvements in materials used in sample clean-up procedures, and with the use of known labeled and unlabeled chemical standards. Problems associated with chemical analysis procedures of CDDs in various media are discussed in greater detail in Chapter 6. In reviewing data on CDD levels monitored or estimated in the environment, it should be noted that the amount of the chemical identified analytically is not necessarily equivalent to the amount that is bioavailable (see Section 2.3) and that every measurement is accompanied with a certain analytical error.

5.4.1 Air

Indoor household dust samples gathered by a vacuum cleaner from rooms with furniture treated with a wood-preserving formulation were analyzed for CDDs (Christmann et al. 1989b). The wood-preserving formulation contained PCP, which is known to be contaminated with CDDs, particularly HxCDD, HpCDD, and OCDD. OCDD was the most abundant congener found in the dust samples at an average concentration of 191 µg/kg (ppb), followed by HpCDD (20 µg/kg), HxCDD (2.5 µg/kg), PeCDD (0.9 µg/kg), and TCDD (0.2 µg/kg) (Christmann et al. 1989b).

Indoor air concentrations of CDD/CDFs were measured in kindergarten classrooms in West Germany to evaluate releases from wood preservatives (e.g., PCP) that may have been used in building materials (Päpke et al. 1989a). Measured indoor air concentrations of total CDD/CDF ranged from 1.46 to

4.27 pg/m^3, while measured outdoor air concentrations ranged from 0.61 to 78.97 pg/m^3. The 2,3,7,8-substituted congeners predominated with mean concentrations as follows: OCDD (131.5 pg/m^3), 1,2,3,4,6,7,8-HpCDD (77 pg/m^3), 1,2,3,4,6,7,8-HpCDF (51 pg/m^3), and OCDF (25.3 pg/m^3).

Measured indoor air samples collected in an office building in Binghamton, New York, 2 years after a fire in an electrical transformer that contained PCBs and tri- and tetra-chlorobenzenes had concentrations of 2,3,7,8-TCDD ranging from 0.23 to 0.47 pg/m^3 (0.017–0.036 ppq) (Smith et al. 1986a). The 2,3,7,8-TCDD isomer constituted 23–30% of the 1.0–1.3 pg/m^3 (0.076–0.099 ppq) total TCDDs. The limit of detection for these samples was approximately 0.003 pg/m^3 (Smith et al. 1986a).

Background levels of CDD in air were measured in a semi-rural location in Elk River, Minnesota, located about 25 miles northwest of Minneapolis-St. Paul (Reed et al. 1990). No major industrial or commercial activity occurred in the area at the time of the study. Ambient air samples were collected in the winter and summer of 1988. 2,3,7,8-TCDD was not detected in any of the ambient air samples taken in the summer (detection limits for 2,3,7,8-TCDD ranged from 0.005 to 0.065 pg/m^3 [0.0004–0.0046 ppq]). 2,3,7,8-TCDD was noted in a wintertime sample at concentrations of 0.015 pg/m^3 (0.0011 ppq) and 0.019 pg/m^3 (0.0014 ppq). Detection limits in the remaining wintertime samples for 2,3,7,8-TCDD ranged from 0.005 to 0.01 pg/m^3 (0.0004–0.0007 ppq). Wintertime CDD concentrations were greater than those observed for summertime. The authors noted that this may be a result of increased numbers of combustion sources operating during the winter months. The wintertime CDD congener profile showed increasing concentrations with increasing chlorine substitutions. Average wintertime ambient air concentrations of HpCDD and OCDD ranged from approximately 0.5 to 4.1 pg/m^3 (0.029–0.236 ppq) and 0.74 to 8.2 pg/m^3 (0.039–0.436 ppq), respectively (Reed et al. 1990). Average summertime ambient air concentrations of HpCDD and OCDD ranged from approximately 0.204 to 0.246 pg/m^3 (0.011–0.014 ppq) and 0.018 to 0.024 pg/m^3 (0.001–0.0013 ppq), respectively (Reed et al. 1990). The authors found that, in general, the more highly chlorinated congeners were present at higher concentrations that the less chlorinated congeners.

A long-term study (1985–1988) of CDDs in the ambient atmosphere of Bloomington, IN (a suburban area), was carried out in order to provide base-line data against which the impact of a future incinerator on local CDD concentrations could be judged (Eitzer and Hites 1989b). Ambient air samples were analyzed for the presence of CDDs in both the particulate-bound phase and the vapor-phase forms. At the four sites sampled, the concentrations of CDDs (TCDD, PeCDD, HxCDD, HpCDD, and OCDD) increased with an increasing level of chlorination. All sites showed that the less chlorinated CDDs have a higher vapor-phase

fraction than the more chlorinated CDDs. In addition, all sites show OCDD to be the most abundant CDD, averaging from 0.44 to 0.69 pg/m^3 (0.023–0.032 ppq) (detection limit 0.001 pg/m^3 [5.3×10^{-5} ppq]) (Eitzer and Hites 1989b). A seasonal effect was seen on the proportion of the total atmospheric burden present in the vapor phase. During the warm summer months, the total vapor-to-particle bound ratio (V/P) was as great as 2, whereas in the winter it was <0.5. At warm temperatures, most of the less chlorinated CDDs are found in the vapor phase, whereas at cooler temperatures more of the CDDs were associated with the particle phase (Eitzer and Hites 1989b).

CDDs have been found in urban air particulates from Washington, D.C., and St. Louis, Missouri; OCDD was the predominant congener at concentrations of 200 ppb and 170 ppb for Washington and St. Louis, respectively (Czuczwa and Hites 1986a). Combustion of municipal and chemical wastes was the most likely source of these compounds. CDDs were detected in air samples from Albany, Binghamton, Utica, and Niagara Falls, NY (Smith et al. 1990b). Concentrations of CDD congener groups for all 4 cities were as follows: total TCDD, not detected (<0.21 pg/m^3 [0.016 ppq]); total PeCDD, <0.04–0.62 pg/m^3 (<0.003–0.043 ppq); total HxCDD, 0.10–2.4 pg/m^3 (0.007–0.15 ppq); total HpCDD, <0.21–4.4 pg/m^3 (0.012–0.25 ppq); and OCDD <0.54–4.6 pg/m^3 (0.029–0.244 ppq) (Smith et al. 1990b). In 1988–89, total CDDs measured downwind from an industrial source in Niagara Falls, NY, ranged from 0.3 pg/m^3 to 133 pg/m^3 and were approximately 2.5 times higher than upwind concentrations (Smith et al. 1990b). Between 1986 and 1990, total CDD concentrations averaged 2.3 pg/m^3, of which 65% was OCDD (Smith et al. 1992).

An extensive multi-year monitoring program for CDDs/CDFs was conducted at eight sampling locations in the Los Angeles South Coast Air Basin from 1987 to 1989 (Hunt and Maisel 1992). The monitoring network, which monitored for both vapor and particulates, included several sites situated in residential areas as well as sites in the vicinity of suspected CDD/CDF sources. Monitoring results indicated that 2,3,7,8-TCDD was virtually undetected. The most commonly detected 2,3,7,8-substituted congener was OCDD followed by 1,2,3,4,6,7,8-HpCDD. The predominance of 1,2,3,4,6,7,8-HpCDD as the most persistent congener is associated with stationary or mobile combustion source emissions. 1,2,3,4,6,7,8-HpCDD was found at all 7 sampling sessions at a mean concentration of 1.140 pg/m^3. OCDD also was found at all 7 sampling sessions at a mean concentration of 2.883 pg/m^3. The mean total TCDD concentration was 0.114 pg/m^3 and was measured during only 3 sampling sessions (Hunt and Maisel 1992).

The concentrations of CDDs in the ambient air at several sites in metropolitan Dayton, Ohio, have been determined (Tiernan et al. 1989b). No CDDs (TCDD, PeCDD, HxCDD, HpCDD, and OCDD) were found in rural regions, with average detection limits ranging from 0.03 pg/m^3 (TCDD) to 1.44 pg/m^3 (OCDD). The rural area was outside the impact zone of air pollutants from any regional industrial sources. CDDs in the industrialized regions appear to originate from a combination of sources, including municipal waste incinerators, motorized vehicles, and a PVC-coated metal incinerator, the latter being a major source of these pollutants. Suburban/roadside area samples were taken at ground level at a distance of about 3 meters from a street intersection through which approximately 60,000 cars passed each day. Other sampling sources were on the roofs of buildings in the downtown Dayton area, which lay in the emissions path from municipal solid-waste incinerators. TCDDs and PeCDDs (detection limits 0.01 and 0.03 pg/m^3, respectively) were not detected in the suburban/roadside area but were detected in the municipal waste-incinerator areas at 0.24 and 0.38 pg/m^3, respectively. HpCDD was detected in both the suburban/roadside areas and the municipal waste-incinerator areas at concentrations of 0.41 pg/m^3 (0.024 ppq) and 3.34 pg/m^3 (0.19 ppq), respectively. OCDD was also detected in the suburban/roadside areas (1.09 pg/m^3 [0.058 ppq]) and the municipal waste incinerator areas (4.69 pg/m^3 [0.25 ppq]). Concentrations of HxCDD were lower than HpCDD and OCDD, 0.05 pg/m^3 (0.003 ppq) in the suburban/ roadside areas and 2.56 pg/m^3 (0.160 ppq) in the vicinity of the municipal waste incinerators (Tiernan et al. 1989b).

Air samples were collected in Ohio in 1987 at an industrial area, an urban area downwind of a municipal incinerator, a high-traffic density area, and a rural area (Edgerton et al. 1989). No 2,3,7,8-TCDD was detected in any of the air samples with detection limits of <0.24 pg/m^3 (0.02 ppq) in any of the areas. The ambient concentrations of CDDs collected in the urban area were as follows: total HpCDD, 1.0–1.1 pg/m^3 (0.058–0.063 ppq); OCDD, 1.0–1.2 pg/m^3 (0.053–0.064 ppq); PeCDD, 0.1 pg/m^3 (0.03 pg/m^3); and total HxCDD, 0.6–0.63 pg/m^3 (0.038–0.039 ppq) (detection limit not specified). Concentrations of CDDs in the industrial area were: total HpCDD, 0.41–1.0 pg/m^3 (0.024–0.058 ppq), OCDD, 0.51–1.1 pg/m^3 (0.027–0.058 ppq), and total HxCDD, 0.43–0.78 pg/m^3 (0.027–0.049 ppq). Concentrations of total HpCDD, OCDD, total HxCDD in the high-traffic density area were 0.56 pg/m^3 (0.032 ppq), 0.96 pg/m^3 (0.051 ppq), and 0.15 pg/m^3 (0.008 ppq), respectively. Ambient air concentrations of total HpCDD, OCDD, and total HxCDD in the rural area were 0.48 pg/m^3 (0.028 ppq), 0.5 pg/m^3 (0.027 ppq), and 0.33 pg/m^3 (0.021 ppq), respectively. PeCDD was not detected in the industrial, high-traffic, or rural areas (Edgerton et al. 1989).

Air monitoring at Windsor, Ontario, downwind of a proposed municipal solid-waste incinerator in Detroit, Michigan, between 1987 and 1988 found a mean total CDD concentration of 2.12 pg/m^3. A sampling station located in a rural area 30 miles away provided background total CDD concentrations of 0.51 mg/m^3. At both stations, the primary congeners were HpCDD and OCDD in the particulate phase, whereas TCDD and PeCDD were not detected in the vapor or particulate phases above the detection limit (Bobet et al. 1990).

A mixture of CDDs (TCDD, PeCDD, HxCDD, HpCDD, OCDD) has been found in emissions from the combustion of various sources, including municipal incinerators, power plants, wood burning, house-heating systems, and petroleum-refining operations (Chiu et al. 1983; Clement et al. 1985; Thoma 1988; Thompson et al. 1990). CDDs were found in stack and fly ash samples from the following combustion sources (ranges given): municipal incinerator, 8 ppb (OCDD) to 390 ppb (HxCDD) (TCDD was found at 10 ppb); open-air burning of PCP-treated wood, 2 ppb (TCDD) to 187 ppb (OCDD); coal-fired power plant, 1 ppb (TCDD) to 6 ppb (PeCDD and HxCDD); hydroelectric power plant, 0.5 ppb (OCDD) to 5.2 ppb (TCDD) (Chiu et al. 1983); and petroleum refining, 0.8 (OCDD) to 3.4 ng/m^3 (PeCDD) (Thompson et al. 1990). Samples of ash from wood-burning stoves, a fireplace, and open-air wood burning contained detectable levels of CDDs ranging from 0.3 to 33 ppb (Clement et al. 1985). The open-air burning ash contained the highest total CDD concentration (33 ppb), with HpCDD being the most abundant homologue (11 ppb). The total CDD concentrations in 4 samples from wood-burning stoves ranged from 0.3 to 15 ppb, with the relative amounts of each homologue varying for each sample. Ash samples from the fireplace contained total CDD concentrations ranging from 3.1 to 5.4 ppb, with HxCDD (0.3–1.7 ppb) and OCDD (0.4–3.1 ppb) being the most abundant homologues present (Clement et al. 1985). TCDD was present in ash samples from open-air burning (0.8 ppb) and was detected in ash from the fireplace.

Ambient air monitoring in the vicinity of a Superfund clean-up site detected 2,3,7,8-TCDD levels on the order of 1 pg/m^3 (0.08 ppq) (Fairless et al. 1987). The surface and subsurface soils at the site were tested and found to contain 2,3,7,8-TCDD at concentrations above 1 ppb at most locations within the site.

2,3,7,8-TCDD has been detected in air samples (concentrations unspecified) collected at 9 of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). CDDs have been detected in air samples (concentrations unspecified) collected at 10 of the 126 NPL sites where they have been detected in some environmental media. TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD

have been detected in air samples (concentrations unspecified) at 10, 3, 3, 3, and 1 sites of the 105, 34, 43, 49, and 53 sites, respectively, where they have been detected in some environmental media (see Table 5-1).

In conclusion, most of the measurements of CDDs in air tend to be very close to current detection limits. CDDs are found at the greatest concentrations in urban air with OCDD being the most prevalent congener (up to 0.100 ppq), HpCDDs being the next most common congener, and 2,3,7,8-TCDD being the least common congener (0.014 ppq). Concentrations of all CDDs are highest in the air near industrial areas. Rural areas usually have very low or unquantifiable levels of all CDDs. In urban and suburban areas, concentrations of CDDs may be greater during colder months of the year when furnaces and wood stoves are used for home heating.

5.4.2 Water

Precipitation samples collected in a rural location (Dorset, Ontario) over an 8-month period between 1986 and 1987 were analyzed for CDDs (Tashiro et al. 1989a, 1989b). No TCDDs were found in any samples at detection limits of 4–30 ppq. OCDD concentrations were found in 3 samples in the 60–1,200 ppq range. Lower concentrations of HpCDD (70 ppq) were also found (Tashiro et al. 1989a). Precipitation samples were also collected in 1987–88 in urban and rural locations in Canada (Tashiro et al. 1989b). Varying levels of OCDD were detected throughout the sampling period, mainly at the rural location. OCDD was the only CDD detected at the rural site. OCDD concentrations ranged from 35 to 230 ppq, with the median value being slightly below 100 ppq. No seasonal pattern of OCDD concentrations was observed. OCDD was detected in only 2 of the urban precipitation samples at concentrations of 33 and 15 ppq (Tashiro et al. 1989b). Rain collected at Bloomington, IN, between June 1987 and July 1988 showed low concentrations of total CDDs, although OCDD was the most prominent congener in all samples at concentrations ranging from below the detection limit of 0.1 pg/L to 220 pg/L. Total TCDD was detected in only 3 of 28 samples at concentrations <9 pg/L (EPA 1991d).

An analysis of EPA's STORET (STOrage and RETrieval) database for 1980–82 showed that based on the statistical criteria used, 2,3,7,8-TCDD was detected but at concentrations too low to be quantified in surface-water samples collected at sampling sites (Staples et al. 1985). The sampling sites in the STORET database included both ambient and pipe sites. Ambient sites included streams, lakes, ponds, wells, reservoirs, canals, estuaries, and oceans and were intended to be indicative of general U.S. waterway conditions. Pipe sites referred to municipal or industrial influents or effluents (Staples et al. 1985).

Treated effluents from various Ontario pulp and paper plants using either the bleached kraft (8 mills) or sulfite bleaching process (2 mills) were analyzed for CDDs (Clement et al. 1989). 2,3,7,8-TCDD was not detected in any of the effluent samples with detection limits ranging from 0.07 to 0.7 ppt. A few samples contained a TCDD isomer (not 2,3,7,8-TCDD) at concentrations ranging from 0.06 to 0.12 ppt. PeCDD (0.07 ppt) was detected in one effluent sample, and OCDD (0.05–0.79 ppt) was detected in 4 effluent samples. Suspended particulates were collected from the final effluent from two plants. 2,3,7,8-TCDD and OCDD were detected in the particulates at a concentration range of 200–660 ppt and 180–210 ppt, respectively. The concentration of 2,3,7,8-TCDD determined in the particulates represents levels in the final effluent of 5–10 ppq, suggesting that 2,3,7,8-TCDD is associated with suspended particulate materials in the effluents (Clement et al. 1989).

In 1983, Jobb et al. (1990) conducted a survey of 49 drinking water supplies in Ontario, Canada, including supplies in the vicinity of chemical plants and pulp and paper mills. OCDD was detected in 36 of 37 positive samples ranging from concentrations of 9 to 175 ppq in raw samples (33 positive samples) and from 19 to 46 ppq in treated (filtered) water samples (4 positive samples). These low concentrations were found primarily in water obtained downstream of industrial areas in the St. Clair/Detroit River system. Concentrations of 2,3,7,8-TCDD were not detected in any sample. Because CDDs are hydrophobic, concentrations of these compounds in water tend to be adsorbed onto particulate matter in water. Conventional water treatment processes are expected to be effective in removing the CDDs along with the particulates. This is substantiated by the fact that only 4 of the 37 positive detections were found in treated drinking water, while 33 detections were found in raw water samples.

During 1986, a survey of 20 community water systems throughout the state of New York was conducted to evaluate CDD/CDF concentrations (Meyer et al. 1989). The sampling sites selected were representative of major surface water sources in the state used to obtain drinking water. The sites included surface water sources receiving industrial discharges and those known to contain CDD-contaminated fish, as well as water sources from more remote areas. Raw water sampled at the Lockport, NY, facility contained concentrations of TCDDs (1.7 ppq) as well as concentrations of TCDFs to OCDFs (18, 27, 85, 210, and 230 ppq, respectively). These data show that the CDF congener group concentrations increased with increasing chlorine numbers. TCDFs were also detected in finished water sampled at the Lockport facility (duplicate samples contained 2.1 and 2.6 ppq). Except for a trace of OCDF detected at one other location, no other CDDs/CDFs were detected in finished water at any of the other 19 community water systems surveyed.

Utility telecommunication and railway right-of-ways may be contaminated by leaching of CDDs associated with chlorophenol-treated railway ties and utility poles. A study in British Columbia showed that CDDs and CDFs were not detected in parkland ditch water (control area), but were detected in farmland, utility, and railway right-of-way ditch water (Wan and van Oostdam 1995). Total mean CDD concentrations (mainly OCDD and HpCDD) measured in farm ditch water, and railway ditch water, without and with utility poles were 2.22 µg/L, 45 µg/L, and 9,627 µg/L respectively. Mean total concentrations of CDDs were much higher in ditch water adjacent to utility poles (13,142 ng/L) than in ditch water 4 meters downstream (4,880 ng/L) or 4 meters upstream of the utility poles (2.72 ng/L). The authors concluded that utility poles and railway ties are a potential constant source of CDD/CDF contamination to both water and sediment in aquatic environment through ditch runoff.

2,3,7,8-TCDD has been detected in surface water samples collected at 9 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). CDDs have been detected in surface water samples collected at 14 of the 126 NPL sites where they have been detected in some environmental media. TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in surface water samples at 10, 1, 4, 4, and 6 sites of the 105, 34, 43, 49, and 53 NPL sites, respectively, where they have been detected in some environmental media (see Table 5-1).

Groundwater in the vicinity of an abandoned wood treatment facility was sampled from monitoring wells constructed at depths ranging from 6.1 to 30.5 meters and was analyzed for CDDs in January 1984 (Pereira et al. 1985). Concentrations of HxCDD, HpCDD, and OCDD in groundwater samples taken from wells at a depth of 6.1 meters were 61 ppt, 1,500 ppt, and 3,900 ppt, respectively. The authors noted that the high concentrations of CDDs in the sample from a depth of 6.1 meters probably resulted from the presence of microemulsions of oil that were difficult to separate from the sample. Groundwater samples collected from deeper wells (12.2–30.5 meters) contained HxCDD, HpCDD, and OCDD at concentration ranges of not detected to 21 ppt, not detected to 34 ppt, and not detected to 539 ppt, respectively (Pereira et al. 1985).

2,3,7,8-TCDD has been detected in groundwater samples collected at 15 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). CDDs have been detected in groundwater samples collected at 32 of the 126 NPL sites where they have been detected in some environmental media. TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in

groundwater samples at 21, 3, 10, 14, and 16 sites of the 105, 34, 43, 49, and 53 NPL sites, respectively, where they have been detected in some environmental media (see Table 5-1).

In conclusion, CDDs are rarely detected in drinking water at ppq levels or higher. Raw water samples generally have higher concentrations of CDDs (9–175 ppq) than finished drinking water samples (19–46 ppq) because conventional water treatment processes remove the CDDs along with the particulates from raw water. CDDs have been detected in treated effluent samples collected at pulp and paper mills using the bleach kraft or sulfite bleaching process. In groundwater samples collected near industrial sites, CDDs have been detected at concentrations up to 3,900 ppt.

5.4.3 Sediment and Soil

Soil. As part of this National Dioxin Study, EPA conducted a 2-year nationwide study to assess the extent of 2,3,7,8-TCDD contamination (EPA 1987n). Environmental samples (including soil, sediment, water, and fish) were analyzed for 2,3,7,8-TCDD concentrations at seven different tiers of sites (including NPL, various industrial, urban, and pristine rural sites). Soil concentrations at most of the Tier 1 and 2 sites (i.e., sites classified as or expected to be classified as NPL sites) were in the ppb range, although at a few of the sites where 2,4,5-TCP production waste storage or disposal occurred, concentrations were as high as 2,000 ppm. Offsite soil contamination of concern (in the ppb range) was confirmed at 7 of these 100 Tier 1 and 2 sites. At 11 of 64 Tier 3 sites (facilities and associated disposal sites where 2,4,5-TCP and its derivatives were formulated into pesticide products), soil concentrations exceeded 1 ppb, but in 7 of the 11 sites where contamination was found, only 1 or 2 samples exceeded 1 ppb. At 15 of 26 Tier 5 sites (areas where 2,4,5-TCP and other pesticide derivatives had been or were currently being used), soil concentrations were generally above 1 ppt with one detection at 6 ppb. Two-thirds of all detections at the Tier 5 sites were below 5 ppt. At 3 of 18 Tier 6 sites (organic chemical and pesticide manufacturing facilities where production processes could have resulted in 2,3,7,8-TCDD being introduced into the waste streams), soil concentrations exceeded the 1 ppt detection limit, although these concentrations were limited to one or two samples per site. In general, 2,3,7,8-TCDD was detected infrequently and at very low concentrations in background soil samples taken at sites (urban and rural areas) that did not have previously known sources of 2,3,7,8-TCDD contamination (1 ppt detection limit). Only 17 of 221 urban sites and 1 of 138 rural sites in Tier 7 (background sites not expected to have contamination) had detectable levels of 2,3,7,8-TCDD, with 11.2 ppt being the highest concentration reported (Des Rosiers 1987; EPA 1987n).

Background levels of CDDs in soil were measured at Elk River, Minnesota, a semi-rural area located about 25 miles northwest of Minneapolis-St. Paul (Reed et al. 1990). No major industrial or commercial activity occurred in the area at the time of the study. The soil data reflected generally low background concentrations of CDDs. 2,3,7,8-TCDD, total TCDD, and PeCDD were not detected (detection limit range 0.75–2.9 ppt). OCDD represented the highest baseline levels, ranging from 340 to 3,300 ppt. Levels of total HpCDD ranged from 62 to 640 ppt, while levels of total HxCDD ranged from 12 to 99 ppt (Reed et al. 1990).

Birmingham (1990) analyzed soil samples from industrial, urban, and rural sites in Ontario, Canada, and some Midwestern U.S. states for CDDs and CDFs. The concentrations of CDD/CDF in rural soils were generally not detectable, although HpCDDs and OCDD were found in a few samples. In urban soils, the tetra- through octa-congener groups were measured for both CDDs and CDFs. The HpCDDs and OCDD dominated the homologue profile and were two orders of magnitude greater than concentrations in rural soils. These urban soils also contained measurable quantities of TCDDs, PeCDDs, and HxCDDs. Industrial soils did not contain any TCDDs or PeCDDs, but they did contain the highest concentrations of the HpCDDs, OCDD, TCDFs, HpCDFs, and OCDFs. In an earlier study, soil concentrations of 2,3,7,8-TCDD were measured in industrialized areas of a group of mid-western and mid-Atlantic states (Illinois, Michigan, New York, Ohio, Pennsylvania, Tennessee, Virginia, and West Virginia) (see Table 5-4) (Nestrick et al. 1986). Many of the samples were taken within one mile of major steel, automotive, or chemical manufacturing facilities or of municipal solid-waste incinerators. The data show that in these typical industrialized areas, 2,3,7,8-TCDD soil concentrations are below 0.01 ppb (range, ND–9.4 ppt). The widespread occurrence of 2,3,7,8-TCDD in U.S. urban soils at levels of 0.001–0.01 ppb suggests that local combustion sources, including industrial and municipal waste incinerators, are the probable sources of the trace 2,3,7,8-TCDD soil concentrations found in those locations (Nestrick et al. 1986). Soil samples collected in the vicinity of a sewage sludge incinerator were compared with soil samples from rural and urban sites in Ontario, Canada (Pearson et al. 1990). Soil in the vicinity of the incinerator showed a general increase in CDD concentration with increasing degrees of chlorination. Of the CDFs measured, only OCDFs was detected (mean concentration, 43 ppt). Rural woodlot soil samples contained only OCDD (mean concentration, 30 ppt). Soil samples from undisturbed urban parkland revealed only concentrations of HpCDDs and OCDD, but all CDF congener groups from TCDF to OCDF were present. The parkland samples showed an increase in concentrations from the HpCDDs to OCDD and PeCDFs to OCDF. The TCDFs were found at the highest concentration (mean, 29 ppt) of all the CDF congener groups.

Table 5-4. 2,3,7,8-TCDD Levels Measured in Soil Samples Collected in 1984 from Industrialized Areas of U.S. Cities

Sample location[a]	2,3,7,8-TCDD (ppq)
Lansing, MI	3,000 (700)[b] ND (800)
Gaylord, MI	ND (200)
Detroit, MI	3,600 (700) 2,100 (400)
Chicago, IL	9400 4200
Middletown, OH	ND (300) ND (300)
Barberton, OH	5600
Akron, OH	6300
Nashville, TN	800 (300)
Pittsburgh, PA	2,600 (500)
Marcus Hook, PA	400 (300)
Philadelphia, PA	900 (300)
Clifton Heights, PA	ND (400)
Brooklyn, NY	2,600 (400)
South Carolina, WV	ND (400)
Arlington, VA	ND (400)
Newport News, VA	400 (300)

[a] Post office state abbreviations used
[b] Values in parentheses show the detection limit, 2.5 times noise, when the experimental result is less than 10 times the measured detection limit.

ND = not detected; ppq = parts per quadrillion; 2,3,7,8-TCDD = 2,3,7,8-tetrachlorodibenzo-*p*-dioxin

Source: Nestrick et al. 1986

A large-scale environmental survey was conducted by the Dow Chemical Company to determine soil levels of 2,3,7,8-TCDD on the Dow Midland Plant site and in the city of Midland, Michigan (Nestrick et al. 1986). The Dow Midland Plant site manufactures a variety of chlorophenolic compounds. Soil samples were taken from three different types of areas: locations known to be directly associated with current or historic chlorophenolic production and handling, locations known to be associated with incineration of chemical and conventional wastes and with ash storage, and locations away from established 2,3,7,8-TCDD sources that provide a measure of general background levels of 2,3,7,8-TCDD surface soil within the Dow property. Soil samples taken from chlorophenolic production areas showed a range of 2,3,7,8-TCDD concentrations from 0.041 to 52 ppb. Two localized areas of elevated concentrations (above 5 ppb) were identified with peaks at 34 ppb and 52 ppb. All other samples taken around this area had 2,3,7,8-TCDD soil concentrations below 1 ppb. Two of 10 surface soil samples with 2,3,7,8-TCDD concentrations above 1 ppb (2.0 and 4.3 ppb) were found near the waste incinerator. The concentrations observed there (0.018–4.3 ppb) closely matched the 2,3,7,8-TCDD content of the ash produced by the incinerator, which ranged up to 10 ppb. The background levels of 2,3,7,8-TCDD (0.0065–0.59 ppb) within the Dow Midland Plant site were well below 1 ppb. Soil samples taken within the city of Midland showed 2,3,7,8-TCDD soil concentrations below the 1 ppb concern level established by the U.S. Public Health Service, Centers for Disease Control and Prevention (CDC), for residential areas (Kimbrough et al. 1984). 2,3,7,8-TCDD soil concentrations in the city of Midland (0.6–450 ppt) were higher in areas nearer the Dow Chemical Company Midland Plant site (22–450 ppt) (Nestrick et al. 1986). This gradient suggests that operations on the Midland Plant site are associated with the appearance of the trace levels of 2,3,7,8-TCDD in the nearby environment.

Several studies have analyzed soil samples in the State of Missouri for 2,3,7,8-TCDD contamination and all reported values are comparable. Concentrations of 2,3,7,8-TCDD in soil samples from contaminated sites throughout Missouri ranged from 30 to 1,750 ppb and concentrations in Times Beach, MO, a heavily contaminated site ranged from 4.4 to 317 ppb (Tiernan et al. 1985). In another study, soil core samples taken from a roadside in Times Beach, MO, contained levels of 2,3,7,8-TCDD ranging from 0.8 to 274 ppb. Many roadways in Times Beach had been sprayed with waste oil containing CDDs for dust control (Freeman et al. 1986). In a third study conducted by Hoffman et al. (1986), 2,3,7,8-TCDD was measured in soil samples from the Quail Run Mobile Home Park in Gray Summit, MO. A maximum soil concentration of 2,200 ppb (single non-composited sample) was detected at one site; however, concentrations typically ranged from 39 to 1,100 ppb in composite soil samples.

2,3,7,8-TCDD has been detected in soil samples collected at 61 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). CDDs have been detected in soil samples collected at 94 of the 126 NPL sites where they have been detected in some environmental media. TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in soil samples at 71, 21, 29, 34, and 38 sites of the 105, 34, 43, 49, and 53 NPL sites, respectively, where they have been detected in some environmental media (see Table 5-1).

In conclusion, soil concentrations of CDDs are typically higher in urban areas than in rural areas. Soil concentrations associated with industrial sites are clearly the highest, with CDD levels ranging from the hundreds to thousands of ppt. In general, as the degree of chlorination increases, the concentrations increase. HpCDD and OCDD congeners are generally found at higher concentrations in soil and sediments than the TCDD, PeCDD, and HxCDD congeners.

Sediment. Highly stratified sediments from Green Lake in upstate New York had CDD concentrations that could be correlated with atmospheric deposition. CDDs could be detected as far back as 1860–1865 at a total CDD concentration of 7 ppt; 98% of all CDDs detected were OCDD. The CDD sediment profile showed a strong increase after 1923 and continued to increase until 1984 (the last year analyzed), with a maximum concentration of >900 ppt, of which 75% was OCDD (Smith et al. 1992).

In another study, surficial (surface) sediment samples taken from the Saginaw River and Bay and from southern Lake Huron showed that CDDs are ubiquitous in the samples studied, including the most remote locations (Czuczwa and Hites 1984). The concentrations were highest in those sediments collected closest to urban areas and lowest in open-lake cores. This indicates that the most of the CDDs found in these samples are anthropogenic in origin (Czuczwa and Hites 1984). The CDDs found closest to urban areas may be related to point source industrial inputs as well as atmospheric deposition, while CDDs found at the remote sites are likely to be only atmospheric in origin. In dated sediment cores, CDDs were absent before 1940. Thus, the authors suggest that accumulation of CDDs in the environment is a recent phenomenon and is related to industrial activities (Czuczwa and Hites 1986a, 1986b). Surface sediments taken from the Great Lakes showed that CDDs were ubiquitous in the sediments. OCDD was predominant at concentrations ranging from 560 to 4,800 ppt (dry weight) (Czuczwa and Hites 1986a, 1986b). The sediments also contained relatively high concentrations of HpCDD. The less chlorinated CDDs were not found in the sediments (Czuczwa and Hites 1986a). Sediment samples were collected from five sampling stations in the western basin of Lake Ontario near the mouth of the Niagara River and were analyzed for 2,3,7,8-TCDD

(Onuska et al. 1983). Measurable quantities of 2,3,7,8-TCDD were present in sediment at two of the stations. The highest concentration of 2,3,7,8-TCDD (13 ppt) was found at a depth of 3–5 cm, followed by a concentration of 4 ppt at a depth of 3 cm, and 3 ppt at a depth of 13–14 cm. Concentrations of 2,3,7,8-TCDD in the rest of the sediment samples were below the detection limit (0.1 ppt) (Onuska et al. 1983).

Surficial sediments collected from Jackfish Bay on the north shore of Lake Superior, near a pulp and paper manufacturer, contained moderate concentrations of the TCDFs (range of geometric mean, 2.4–6,223 pg/g) and OCDD congeners (range of geometric mean, 12–250 pg/g), with trace (< 60 ppt) concentrations of other congeners (Sherman et al. 1990). The OCDF and OCDD profile for a sediment core collected from Moberly Bay was similar to the surficial sediment pattern. These congener groups predominated at all sediment depths where detectable concentrations occurred. Low concentrations of the HpCDD, PeCDF, and HpCDF congeners also were detected. The concentration profile of the HpCDF congener group showed a relatively high value that dropped abruptly to nondetectable (<60 ppt) below a sediment depth of 10 cm. This abrupt change corresponded to a date of 1973 that reflected an operational change at the pulp mill.

Surficial harbor sediments collected near a PCP wood preserving plant in Thunder Bay, Ontario, Canada on the north shore of Lake Superior were found to contain CDDs and CDFs (McKee et al. 1990). The highest concentrations were detected at stations closest to the plant docking area and lower concentrations occurred at stations further from the source (McKee et al. 1990). No CDDs or CDFs were detected below the surficial layer. Concentrations of TCDD and PeCDD congeners were below detection limits (<1 ppt) in all samples. The concentrations of the HxCDD and OCDD congeners increased with the degree of chlorination. The maximum concentrations of the HxCDDs to OCDD ranged from 5,600 ppt (HxCDDs) to 980,000 ppt (OCDD). As with the CDD distribution profile, the concentrations of HxCDFs and OCDFs increased with the degree of chlorination.

Sediment samples taken from Love Canal storm sewers in Niagara Falls, NY, contained from 0.9 to 312 ng/g of 2,3,7,8-TCDD (0.9–312 ppb) (Smith et al. 1983). The highest concentration of 2,3,7,8-TCDD (312 ppb) was found immediately adjacent to the canal at its southern end; the next highest concentration (120 ppb) was found just upstream. A sample taken one street away from the canal, near the high altitude division of the storm sewer system where only a small amount of canal runoff occurs, contained only 0.9 ppb 2,3,7,8-TCDD (Smith et al. 1983).

Surface sediment samples were collected from various estuaries in the United States: Black Rock Harbor (an urban industrialized estuary in Connecticut), Long Island Sound (a relatively clean reference site in New York), Narragansett Bay (an estuary affected by input from chemical industries in Rhode Island), New Bedford Harbor (an estuary within a Superfund site boundary in Massachusetts), and Eagle Harbor (a creosote wood-treatment facility in Washington) (Norwood et al. 1989). 2,3,7,8-TCDD was detected in sediment from Black Rock Harbor (56–57 ppt), Narragansett Bay (15–19 ppt), and in some of the sediment samples found in New Bedford Harbor (4.2–4.6 ppt). 1,2,3,7,8-PeCDD was detected in estuarine sediments from Black Rock Harbor (79–95 ppt), New Bedford Harbor (21–29 ppt), and Eagle Harbor (5 ppt). HxCDD, HpCDD, and OCDD were also detected in sediments from all estuaries at concentrations ranging from approximately 10–100 ppt, 500–3,000 ppt and 2,000–37,000 ppt, respectively. The highest concentrations of HpCDD (>1,000 ppt) were detected in Narragansett Bay sediments, while the highest concentration of OCDD (37,000 ppt) was detected in Eagle Harbor sediments. The levels of CDDs reported for all samples were for dry weight (air dried) concentrations (Norwood et al. 1989).

Sediment samples collected in 1985–86 from estuarine areas (Passaic River and Newark Bay), near a Newark, NJ, facility that manufactured 2,4,5-T between 1948 and 1969, contained high concentrations of 2,3,7,8-TCDD and OCDD (Bopp et al. 1991). Concentrations of OCDD in the sediment were many times higher than concentrations of 2,3,7,8-TCDD. The study indicated that there probably was a significant regional source (i.e., combustion and/or use of the wood preservative PCP) for OCDD, a source that is lacking in significant concentrations of 2,3,7,8-TCDD relative to the local industrial source. A high correlation was found between 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations, suggesting that the industrial site was a major source of 2,3,7,8-TCDF to the natural waters of the area. Sediment core samples from a depth of 108–111 cm contained 2,3,7,8-TCDD at a concentration of 21,000 ppt, the highest concentration measured in the study. This residue value corresponds to deposition of sediments that occurred during the late 1950s to early 1960s during active 2,4,5-T production at the industrial site. Maximum concentrations of TCDD in the sediment cores corresponded to the period of maximum 2,4,5-T production, with more recently deposited sediments containing lower concentrations of TCDD. This study established the persistence of 2,3,7,8-TCDD and 2,3,7,8-TCDF in anaerobic sediments on a time scale of several decades (Bopp et al. 1991).

There has been considerable discussion about historic releases of CDDs/CDFs in Newark Bay, New Jersey. Bopp et al. (1991) suggested that a single source (pesticide production facility) is responsible for the presence of 2,3,7,8-TCDD/TCDF in the watershed. Recently, Wenning et al. (1992, 1993a, 1993b),

using chemometric comparisons of CDD/CDF residues in surficial sediments, found that congener patterns in Newark Bay were closely related to those found in sediments from other industrialized waterways with several different pollutant sources (e.g., New Bedford Harbor, Massachusetts; Black Rock Harbor, Connecticut; Providence River, Rhode Island; Eagle Harbor, Washington, as well as several European waterways). The similarities and differences among the various waterways examined in the analysis suggests that the presence of 2,3,7,8-substituted CDDs/CDFs in surficial sediments from Newark Bay are more likely due to multiple sources of contamination. Most recently, Ehrlich et al. (1994) identified the relative contributions of various sources of CDDs/CDFs to recently deposited sediments of Newark Bay using polytopic vector analysis, a multivariate statistical technique. These authors also concluded that the 2,3,7,8-substituted CDD/CDF patterns in the sediments of Newark Bay are consistent with discharges from multiple sources. In a recent study, Huntley et al. (1997) reported that combined sewer overflows may contribute substantially to surface sediment contamination of the nearby Passaic River. Several such sources that have existed over the past century in the vicinity include scrap metal refineries, pulp and paper mills, copper smelters, chemical manufacturing plants, municipal sewage treatment plants, and industrial/ municipal incinerators (EPA 1987n). 2,3,7,8-TCDD sediment concentrations ranged from below the detection limit (22 ppt) to 21,000 ppt (21 ppb), whereas OCDD concentrations ranged from 3.1 ppb to 42,000 ppt (42 ppb), although other sources of OCDD were thought to contribute to the elevated levels of OCDD (Bopp et al. 1991; Wenning et al. 1992).

Sludges from various Ontario pulp and paper plants using either the bleached kraft (8 mills) or sulfite bleaching process (2 mills) were analyzed for CDDs (Clement et al. 1989). 2,3,7,8-TCDD was detected in sludge samples at a concentration range of 170–370 ppt. Only one other TCDD isomer (180 ppt) was detected in a sludge sample, but it was not identified. PeCDDs and HxCDDs were not detected in any sludge samples, whereas HpCDD (400 ppt) was found in 1 sludge sample and OCDD (120–1,800 ppt) was found in 6 sludge samples (Clement et al. 1989).

Utility telecommunication and railway right-of-ways may be contaminated by leaching of CDDs associated with chlorophenol-treated railway ties and utility poles. A study in British Columbia showed that CDDs and CDFs were not detected in parkland ditch sediments (control area), but were detected in farmland, utility, and railway right-of-way ditch sediments (Wan and van Oostdam 1995). Total mean CDD concentrations (mainly OCDD and HpCDD) ranged from 18.8 to 277 ng/kg (ppt) (dry weight) in ditch sediments and ballasts respectively. Concentrations of CDDs were much higher in ditch sediment adjacent to utility poles (mean 2,576 ng/kg (ppt) [dry weight]) than in sediment 4 meters downstream (14 ng/kg

[dry weight]) or 4 meters upstream of the utility poles (not detected). CDD concentrations in ditch water were also higher close to the poles (mean 13,142 ng/L [ppt] than 4 meters downstream of the poles (mean 4,880 ng/L [ppt]). The authors concluded that utility poles and railway ties are a potential constant source of CDD/CDF contamination to both water and sediment in aquatic environment through ditch runoff.

2,3,7,8-TCDD has been detected in sediment samples collected at 17 sites of the 91 NPL hazardous waste sites where it has been detected in some environmental media (HazDat 1998). CDDs have been detected in sediment samples collected at 31 of the 126 NPL sites where they have been detected in some environmental media. TCDDs, PeCDDs, HxCDDs, HpCDDs, and OCDD have been detected in sediment samples at 22, 7, 10, 9, and 13 sites of the 105, 34, 43, 49, and 53 NPL sites, respectively, where they have been detected in some environmental media (see Table 5-1).

In conclusion, CDD congener profiles in sediment generally reflect those exhibited by the contamination source or sources. High concentrations of HxCDDs, HpCDDs, and OCDDs in sediment are usually the result of anthropogenic inputs via industrial processes and releases or urban runoff, and concentrations generally increase with the degree of chlorination, but decrease with distance from the source (McKee et al. 1990).

5.4.4 Other Environmental Media

Foods. The FDA has conducted limited analyses for the higher chlorinated CDDs (HxCDD, HpCDD, and OCDD) in market-basket samples collected from 1979 to 1984 under the FDA's Total Diet Program (Firestone et al. 1986). Food samples found to contain PCP residues >0.05 µg/g (ppm) were analyzed for 1,2,3,4,6,7,8-HpCDD and OCDD. In addition, selected samples of ground beef, chicken, pork, and eggs from the market-basket survey were analyzed for these CDD congeners (wet weight basis), regardless of the results of the PCP analysis. HxCDD was not found in any of the foods sampled; however, the detection limit (10–40 pg/g [ppt]) was very high. Generally low concentrations (<300 pg/g [ppt]) of HpCDD and OCDD were found in bacon, chicken, pork chops, and beef liver. Several beef livers had higher concentrations of OCDD residues (614–3,830 pg/g), and one beef liver contained 428 pg/g (ppt) of HpCDD. HxCDD, HpCDD, and OCDD were not detected in milk, ground beef, or seafood samples, but the detection limits (10–40 ppt) were very high. No CDDs were found in 17 egg samples collected in various parts of the United States. OCDD was detected in 2 of 18 pork samples (27 ppt and 53 ppt) and in 2 of the 16 chicken samples (29 ppt and 76 ppt). One chicken sample with PCP residues (>0.05 µg/g) contained

concentrations of 1,2,3,4,6,7,8-HpCDD (28 ppt) and OCDD (252 ppt). The CDD residues (21–1,610 pg/g) in eggs from Houston, Texas, and Mena, Arkansas, with PCP residues >0.05 µg/g collected in 1982 and 1983–84, respectively, contained 1,2,3,4,6,7,8-HpCDD concentrations ranging from 21 to 588 ppt, and OCDD concentrations ranging from 80 to 1,610 ppt. These residues were attributed to local PCP contamination problems in these areas (Firestone et al. 1986). Milk samples contaminated with PCP at levels ranging from 0.01 µg/g to 0.05 µg/g PCP contained no detectable CDDs. It should be noted that the reported limits of detection (10–40 ppt) for the FDA analyses from these older samples, are higher than concentrations of CDDs observed in foods from more recent studies. Samples of beef liver, pork chops, chicken, ground beef, and eggs collected in the United States and analyzed for HpCDD and OCDD contained average concentrations of HpCDD (2.2–9.6 ppt) and OCDD (6.3–47.6 ppt) (Jasinski 1989). Eggs contained the lowest levels of HpCDD and OCDD, and beef liver contained the highest levels.

LaFleur et al. (1990) analyzed the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF (wet weight basis) in a variety of food products collected randomly from grocery stores located in the southern, Midwestern, and northwestern regions of the United States. Concentrations of 2,3,7,8-TCDD ranged from 17 to 62 pg/kg for ground beef, were not detectable in ground pork, ranged from 12 to 37 pg/kg for beef hot dogs, and ranged from 7.2 to 9.4 pg/kg for canned corned beef hash on a whole-weight basis. Concentrations of 2,3,7,8-TCDF were generally much less than concentrations of 2,3,7,8-TCDD, with the exception of ground pork and corned beef hash. For ground pork, TCDD concentrations were not detectable and 2,3,7,8-TCDF concentrations ranged from 13 to 20 pg/kg and for corned beef hash, concentrations of TCDD ranged from 7.2 to 9.4 pg/kg, while concentrations of TCDF ranged from 9.8 to 10 pg/kg.

A study conducted by the province of Ontario, Canada analyzed concentrations of CDDs/CDFs in a variety of foods locally grown in Canada or imported from the United States and New Zealand (Birmingham et al. 1989). Concentrations of OCDD in various foods ranged from 3 to 210 ppt (wet weight basis). OCDD concentrations were detected in U.S. eggs (8 ppt), U.S. beef (24 ppt), Canadian hamburger (3 ppt), and Canadian chicken samples (210 ppt). Chicken also contained 15 ppt HpCDD, but no other CDDs were detected in these samples. Fruits and vegetables were generally free of CDD and CDF residues (detection limit = 1 ppt). OCDD concentrations ranging from 0.6 to 8 ppt (wet weight) were also detected in samples of U.S. potatoes (3 ppt), apples (8 ppt), peaches (0.6 ppt), and wheat (0.7 ppt). For these food items, OCDD was the only homologue detected. No 2,3,7,8-TCDD was detected in any of the food samples tested (detection limits of 1–4 ppt).

Beck et al. (1989a) analyzed the concentrations of CDDs/CDFs in 22 samples of foodstuffs collected in the Federal Republic of Germany. Twelve randomly collected food samples (chicken, eggs, butter, pork, redfish (ocean perch), cod, herring, vegetable oil, cauliflower, lettuce, cherries, and apples) were purchased in various stores in West Berlin. The highest 2,3,7,8-TCDD levels were observed in fish samples with concentrations of 4.7, 23, and 2.8 ppt (lipid basis) for herring, cod, and redfish, respectively. Concentrations of 2,3,7,8-TCDD (on a lipid basis) in meat, poultry, and dairy products were lower; with concentrations ranging from 0.01 ppt in sheep, 0.03 in pork, 0.2 in eggs, 0.3 in chicken, 0.6 in cattle, and 0.02 in cow's milk and 0.08 in butter (see Table 5-5). The EPA TEQ values for CDDs/CDFs calculated for these products ranged from 20.0–39.7 ppt in fish, 0.14–1.31 ppt in meat and poultry, and from 0.43 to 0.86 ppt in dairy products. In all samples tested, the 2,3,7,8-substituted congeners predominated in the samples and non-2,3,7,8-substituted congeners were not detected in fish, chicken and eggs. For meat, poultry, and dairy samples, the congener profile showed high concentrations of 1,2,3,4,6,7,8-HpCDD and OCDD with concentrations of most other congeners at or below 1 ppt (lipid basis). In fish samples, high concentrations of the 2,3,7,8-substituted congeners, TCDDs (range 2.8–23 ppt), PeCDD (range 1.3–12 ppt), HxCDD (range 0.01–17 ppt), and OCDD (range 11–83 ppt) resulted in TEQ values for CDDs/CDFs ranging from 20–40 ppt (lipid basis). In the five food samples of plant origin, no CDDs/CDFs were detected on a whole weight basis (detection limit . 0.01 ppt).

Congener-specific analyses for CDDs and CDFs were performed on 18 dairy, meat, and fish products obtained from a supermarket in upstate New York (Schecter et al. 1994d). Total CDD concentrations (on a wet weight basis) ranged from 0.35 to 2.91 ppt in fish, 0.6–59.3 ppt for meats, and 0.6–14 ppt in dairy products. A summary of the CDD/CDF concentrations and TEQ concentrations calculated for the 18 foods is presented in Table 5-6. The TEQ for both the CDDs and CDFs on a wet weight basis for these food samples ranged from 0.02 to 1.5 ppt, 0.02–0.13 ppt for fish products, 0.03–1.5 ppt for meat products, and 0.04–0.7 ppt for dairy products, with the highest TEQ found in ground beef.

Recently, the EPA and U.S. Department of Agriculture (USDA) completed the first statistically designed surveys of the occurrence and concentrations of CDDs/CDFs in beef fat (Ferrario et al. 1996; Winters et al. 1996), pork fat (Lorber et al. 1997), poultry fat (Ferrario et al. 1997), and the U.S. milk supply (Lorber et al. 1998). The congener specific results for various foods are shown in Table 5-7. It is clear from the results, that two congeners (1,2,3,4,6,7,8-HpCDD and 1,2,3,4,6,7,8-OCDD) were typically found at the highest concentrations in all food samples. Concentrations of 2,3,7,8-TCDD were highest in heavy fowl (0.43 ppt) and young turkeys (0.24 ppt); much lower concentrations were found in beef (0.05 ppt), pork

Table 5-5. CDD/CDF Concentrations in Food Samples of Animal Origin (ppt, lipid basis)

	Dairy products		Meat					Fish		
Congener	Cow's milk	Butter	Pork	Cattle	Sheep	Chicken	Eggs	Herring	Cod	Redfish
2,3,7,8-TCDF	0.7	0.15	0.11	0.3	0.6	2.1	1.1	57.0	98.0	78.0
2,3,7,8-TCDD	0.2	0.08	0.03	0.6	0.01	0.3	0.2	4.7	23.0	2.8
1,2,3,7,8-PeCDF	0.2	0.09	0.01	0.01	0.01	0.01	0.6	16.0	48.0	31.0
2,3,4,7,8-PeCDF	1.4	0.45	0.08	1.5	0.9	1.5	0.8	29.0	3.1	25.0
1,2,3,7,8-PeCDD	0.7	0.41	0.12	0.8	0.5	0.7	0.4	12.0	1.3	6.5
1,2,3,4,7,8-HxCDF	0.9	0.43	0.15	0.8	0.9	0.6	0.4	3.0	6.9	3.5
1,2,3,6,7,8-HxCDF	0.8	0.44	0.07	0.6	1.2	0.4	0.3	4.2	13.0	6.0
2,3,4,6,7,8-HxCDF	0.7	0.31	0.05	1.3	1.5	0.3	1.7	3.6	8.2	7.2
1,2,3,4,7,8-HxCDD	0.3	0.15	0.21	0.6	0.3	0.5	1.3	1.2	0.01	0.5
1,2,3,6,7,8-HxCDD	1.1	0.95	0.29	1.9	1.5	2.8	1.4	5.8	17.0	8.4
1,2,3,7,8,9-HxCDD	0.4	0.26	0.06	0.6	0.4	0.6	5.0	1.0	5.2	1.3
1,2,3,4,6,7,8-HpCDF	0.5	0.34	1.1	2.2	8.1	0.8	0.6	1.6	10.0	1.5
1,2,3,4,6,7,8-HpCDD	2.0	1.5	2.1	18.0	15.0	6.0	0.4	3.6	10.0	3.0
OCDF	1.0	0.25	0.41	0.2	0.3	0.6	0.2	1.4	2.1	0.3
OCDD	10.0	3.4	19.0	25.0	68.0	52.0	12.0	19.0	83.0	11.0
TEQ	0.86	0.43	0.14	1.31	0.52	1.16	0.80	21.3	39.7	20.0

Source: Beck et al. 1989a

TEQ = toxicity equivalency

Table 5-6. Dioxins, Dibenzofurans, and Dioxin Toxicity Equivalencies (TEQs) in U.S. Foods (ppt, wet weight)

	Total CDDs/CDFs		
Food type	CDD	CDF	TEQ
Fish			
Haddock	0.75	0.14	0.03
Haddock fillet	0.35	0.07	0.02
Crunchy haddock	2.91	0.51	0.13
Perch	1.55	1.14	0.02
Cod	0.82	0.09	0.02
Meats			
Ground beef	4.1	7.0	1.5
Beef rib sirloin tip	0.6	0.2	0.04
Beef rib steak	30.7	4.6	0.3
Pork chop	59.3	2.5	0.3
Cook ham	59.3	2.5	0.3
Lamb sirloin	8.95	0.85	0.4
Bologna	3.7	0.4	0.12
Chicken drumstick	0.95	0.14	0.03
Dairy			
Cottage cheese	0.6	0.3	0.04
Soft blue cheese	14.0	5.0	0.7
Heavy cream	5.0	2.0	0.4
Soft cream cheese	4.0	2.0	0.3
American cheese slices	4.0	2.0	0.3

Source: Schecter et al. 1994d

CDDs = chlorinated dibenzo-*p*-dioxins; CDFs = chlorinated dibenzofurans; TEQ = toxicity equivalency

Table 5-7. Overall National Averages of the Concentrations (ppt, or pg/g) of Dioxin and Furan Congeners in Fat of Meat and Milk on a Lipid Basis[a]

CDD/CDF Congener	Beef (n=63)	Pork fat (n=78)	Young chickens (n=39)	Light fowl (n=12)	Heavy fowl (n=12)	Young turkeys (n=15)	Milk (composites) (n=8)
2,3,7,8-TCCD	0.05 (0.03)	0.10 (0.01)	0.16 (0.15)	0.05 (0.03)	0.43 (0.42)	0.24 (0.24)	0.07 (0.07)
1,2,3,7,8-PeCDD	0.35 (0.04)	0.45 (0.01)	0.24 (0.12)	0.15 (0.00)	0.32 (0.22)	0.32 (0.23)	0.32 (0.32)
1,2,3,4,7,8-HxCDD	0.64 (0.18)	0.52 (0.10)	0.18 (0.05)	0.15 (0.00)	0.24 (0.13)	0.16 (0.03)	0.39 (0.39)
1,2,3,6,7,8-HxCDD	1.42 (1.21)	1.10 (0.80)	0.39 (0.33)	0.34 (0.29)	0.71 (0.70)	0.79 (0.77)	1.87 (1.87)
1,2,3,7,8,9-HxCDD	0.53 (0.26)	0.47 (0.04)	0.39 (0.29)	0.15 (0.01)	0.60 (0.51)	0.17 (0.06)	0.55 (0.55)
1,2,3,4,6,7,8-HpCDD	4.48 (4.39)	10.15 (9.93)	1.53 (1.53)	0.93 (0.93)	2.04 (2.02)	0.54 (0.52)	5.03 (5.03)
1,2,3,4,6,7,8,9-OCDD	4.78 (3.26)	52.77 (52.40)	5.31 (5.31)	2.07 (2.07)	7.67 (7.67)	0.75 (0.68)	4.89 (4.89)
2,3,7,8-TCDF	0.03 (0.00)	0.09 (0.004)	0.28 (0.28)	0.25 (0.25)	0.48 (0.47)	0.57 (0.57)	0.08 (0.08)
1,2,3,7,8-PeCDF	0.31 (0.00)	0.45 (0.00)	0.21 (0.08)	0.18 (0.05)	0.14 (0.02)	0.36 (0.25)	0.05 (0.00)
2,3,4,7,8-PeCDF	0.36 (0.06)	0.56 (0.14)	0.25 (0.12)	0.22 (0.11)	0.18 (0.09)	0.53 (0.47)	0.28 (0.28)
1,2,3,4,7,8-HxCDF	0.55 (0.27)	0.98 (0.60)	0.23 (0.10)	0.16 (0.04)	0.17 (0.06)	0.20 (0.13)	0.39 (0.39)
1,2,3,6,7,8-HxCDF	0.40 (0.12)	0.58 (0.58)	0.20 (0.07)	0.15 (0.03)	0.15 (0.01)	0.17 (0.03)	0.25 (0.25)
1,2,3,7,8,9-HxCDF	0.31 (0.00)	0.45 (0.00)	0.15 (0.00)	0.15 (0.00)	0.15 (0.00)	0.15 (0.00)	0.05 (0.00)
2,3,4,6,7,8-HxCDF	0.39 (0.10)	0.57 (0.16)	0.21 (0.08)	0.14 (0.02)	0.15 (0.02)	0.15 (0.03)	0.28 (0.28)
1,2,3,4,6,7,8-HpCDF	1.00 (0.75)	3.56 (3.35)	0.27 (0.20)	0.15 (0.05)	0.20 (0.10)	0.15 (0.02)	0.83 (0.83)
1,2,3,4,7,8,9-HpCDF	0.31 (0.00)	0.57 (0.17)	0.17 (0.04)	0.15 (0.00)	0.15 (0.00)	0.15 (0.00)	0.05 (0.00)
1,2,3,4,6,7,8,9-OCDF	1.88 (0.00)	2.30 (1.85)	0.34 (0.07)	0.29 (0.00)	0.31 (0.04)	0.29 (0.00)	0.05 (0.00)
Total CDD/CDF, pg/g	17.79 (10.67)	75.67 (70.14)	10.51 (8.82)	5.68 (3.88)	14.09 (12.48)	5.69 (4.03)	15.43 (15.23)
CDD/CDF I-TEQ, pg/g	0.89 (0.35)	1.30 (0.46)	0.64 (0.41)	0.40 (0.16)	0.98 (0.80)	0.93 (0.76)	0.82 NR

a Concentrations calculated at non-detects (ND) equal 1/2 the detection limit (results for ND=0 are in parentheses).

Source: Ferrario et al. 1996, 1997; Lorber et al. 1997; Winters et al. 1996

(0.10 ppt), young chickens (0.16 ppt), light fowl (0.03 ppt) and milk (0.07 ppt). The total concentrations of CDDs/CDFs were highest in pork fat (75.67 ppt) and milk (15.43 ppt), and ranged from 5.68 to 14.09 ppt for all other types of foods tested. The TEQ value for CDDs/CDFs combined was highest for pork fat (1.30 ppt), heavy fowl (0.98 ppt), young turkeys (0.93 ppt), and beef fat (0.89 ppt), with lower TEQ values of 0.40–0.82 ppt for young chickens, light fowl, and milk.

CDDs have been found in infant formulas purchased in the United States (Schecter et al. 1989c). The infant formulas were derived from cow's milk or soybeans. In general, both types of infant formula had very low concentrations of CDDs. 2,3,7,8-TCDD and PeCDD were not detected in cow's milk or soybean formula at detection limits ranging from 0.5 to 1.0 ppt. HxCDD was not detected in soybean formula at the same detection limits. Whole and lowfat (2% fat) cow's milk contained total HxCDD at lipid-adjusted concentrations of 3.6 and 3.3 ppt, respectively. Lipid-adjusted levels of HpCDD were found in whole cow's milk formula (6.5 ppt), lowfat (2%) cow's milk formula (8 ppt), and soybean formula (2.3–3.0 ppt). OCDD was the most abundant congener in both cow's milk and soybean formula. Concentrations of OCDD (lipid-adjusted) were as follows: cow's milk formula (15 ppt), low fat (2%) cow's milk formula (21 ppt), and soybean formula (21–36 ppt) (Schecter et al. 1989c).

In comparison, a study by LaFleur et al. (1990) reported the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in whole milk and half and half. These authors also measured the additional exposure that resulted from migration of these compounds from bleached paperboard containers into the milk over various storage periods. The concentrations of 2,3,7,8-TCDD in whole milk ranged from 24 to 25 pg/kg and in half-and-half ranged from 13 to 14 pg/kg. The corresponding concentrations of 2,3,7,8-TCDF ranged from 260 to 280 pg/kg for whole milk and 146 to 195 pg/kg for half and half. These authors also determined the concentration of 2,3,7,8-TCDD and TCDF for cow’s milk obtained directly from a dairy and for milk stored for various time periods in bleached paperboard cartons. On a lipid basis, the concentration of 2,3,7,8-TCDD of control milk obtained directly from the dairy was 0.48 pg/g, and milk stored in paperboard cartons for 24, 48, 120, and 288 hours was 0.95, 1.4, 1.9, and 2.7 pg/g, respectively. The 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in the paperboard carton were 4.3 and 25 ppt, respectively. Concentrations of 2,3,7,8-TCDF in the control milk was not detectable, but increased in milk stored in cartons for 24, 48, 120, and 288 hours to 6.8, 10.2, 20.1, and 35.1 pg/g, respectively. The percent migration of the 2,3,7,8-TCDD ranged from 2 to 6%, while the percentage of migration of the 2,3,7,8-TCDF ranged from 4 to 18% over the same period (LaFleur et al. 1990).

Similar levels of CDD contamination were reported in two European studies. CDDs were detected in 8 samples of cow's milk in Germany at concentrations ranging from 0.2 ppt for 2,3,7,8-TCDD (detection limit 0.2 ppt) to <10 ppt of OCDD (detection limit not significantly higher than blanks) (Beck et al. 1987). In a Swedish study, only 1 of 10 samples of milk held in either glass bottles or paper cartons contained a detectable level of 2,3,7,8-TCDD (0.46 pg/g milk fat; paper carton; detection limit 0.4 pg/g). Other CDDs were also detected (maximum 7.8 pg/g for OCDD) with the highest concentrations associated with milk packaged in paper cartons, indicating that leaching of CDDs from the paper carton into the milk can occur (Rappe et al. 1990).

Fish and Wildlife. A survey of 2,3,7,8-TCDD contamination in benthic (bottom feeding) and predator fish from major U.S. watersheds was conducted for the EPA National Dioxin Study (Kuehl et al. 1989). It was observed that 17 of 90 (19%) samples collected at sites statistically selected by the EPA had detectable levels of 2,3,7,8-TCDD, whereas 95 of 305 (31%) samples from sites chosen by EPA regional laboratories had detectable levels (detection limits 0.5–2 ppt on a wet weight basis). Of the 112 sites where 2,3,7,8-TCDD was detected, 74 samples (67%) were below 5 ppt, 34 samples (32%) were between 5 and 25 ppt, and 4 samples (1%) were above 25 ppt. A subset of samples collected at sites near the discharges from pulp/paper manufacturing facilities (n=28) had a higher frequency of 2,3,7,8-TCDD contamination above 5 ppt (38%). This subset of samples also contained the sample with the highest level of 2,3,7,8-TCDD contamination (85 ppt). Of the 29 samples collected in the Great Lakes region, 23 (79%) of the sites were found to have detectable levels of 2,3,7,8-TCDD. The most highly contaminated sample, with a concentration of 41 ppt, was collected from Lake Ontario near Oswego, NY. Four of 57 (7%) estuarine or coastal sites had detectable 2,3,7,8-TCDD levels in either fish or shellfish. The level of contamination in these 4 samples ranged from 1.08 to 3.5 ppt (Kuehl et al. 1989). In another study, fish sampled downstream from a bleached kraft paper mill were found to contain higher concentrations of CDDs compared with fish sampled upstream of the paper mill (Hodson et al. 1992). TCDD concentrations in the fish ranged from 1.47 pg/g (wet weight basis) in upstream areas to 15.6 pg/g in fish sampled 2 km downstream. Fish sampled 95 km downstream contained only about half the residues (8.87 pg/g TCDD) of those collected immediately downstream of the facility (Hodson et al. 1992).

Travis and Hattemer-Frey (1991) analyzed data collected as part of the National Dioxin Study regarding 2,3,7,8-TCDD concentrations in fish. The TCDD levels measured in fish from lakes and rivers in the United States confirm that 2,3,7,8-TCDD is bioaccumulating in fish and that low-level contamination of fish is widespread (EPA 1987n). The fish survey included 304 urban areas in the vicinity of population

centers or areas with known commercial fishing activity, including sites in the Great Lakes region. The results of this study indicate that only 29% of fish fillets collected at urban sites had detectable concentrations of 2,3,7,8-TCDD (detection limit =1 ppt). The geometric mean for these fillet samples was 0.3 ppt (wet weight basis). Fish samples from the Great Lakes area contained higher concentrations of 2,3,7,8-TCDD than fish from urban areas (e.g., 67 versus 29% contained detectable levels, respectively). In the Great Lakes area, the geometric mean concentrations of 2,3,7,8-TCDD in fish fillets (2.3 ppt) was almost 7 times higher than the concentrations in the fillets from fish collected from urban areas (0.3 ppt). Comparable concentrations of 2,3,7,8-TCDD were detected in bottom-feeding and predator species from the Great Lakes region. Approximately 74% of the fish fillet samples collected from sites near pulp and paper mills contained detectable concentrations of 2,3,7,8-TCDD. The geometric mean concentration for these fillet samples was 0.9 ppt. This geometric mean is 3 times higher than for urban fillet concentrations (0.3 ppt) but is approximately 2 times lower than for TCDD concentrations in fillets from the Great Lakes Region (2.3 ppt).

From 1986 to 1989, the National Study of Chemical Residues in Fish (NSCRF) was conducted by the EPA as a follow-on study to the National Dioxin Study (EPA 1992). The purpose of the NSCRF was to assess the concentrations of 60 toxic pollutants (including CDDs and CDFs) in the tissues of benthic and game fish nationwide. Benthic species were analyzed as whole-body samples, while game species were analyzed as fillet samples and all concentrations were on a wet weight basis. A summary of the prevalence and concentrations of 6 CDDs and 9 CDFs detected at 388 sites surveyed nationwide in the NSCRF is presented in Table 5-8. Four of the CDDs and three of the CDFs analyzed were detected at over 50% (58–89%) of the sites surveyed. The most frequently detected CDD/CDF compounds (1,2,3,4,6,7,8-HpCDD and 2,3,7,8-TCDF) were both found at 89% of the sites. These compounds were also detected at the highest concentrations: 1,2,3,4,6,7,8-HpCDD at 249 ppt and 2,3,7,8-TCDF at 404 ppt (wet weight). The mean concentrations of these 2 compounds were substantially lower at 10.5 and 13.6 ppt, respectively. The CDD (2,3,7,8-TCDD) believed to be the most toxic congener to mammals, was found at 70% of the sites at a maximum concentration of 204 ppt and a mean of 6.8 ppt (wet weight basis). The NSCRF report further shows that pulp and paper mills using chlorine bleach pulp appeared to be the dominant source of the 2,3,7,8-TCDD and 2,3,7,8-TCDF. Fish collected at sites downstream of pulp and paper mills had significantly higher concentrations of 2,3,7,8-TCDD than fish collected near all other source categories. The statistical tests also showed the same result for 2,3,7,8-TCDF, with the exception that fish residue concentrations downstream of Superfund sites also marginally met the statistical criteria. With respect to source categories, the NSCRF data showed that fish collected downstream of pulp and paper mills (using

Table 5-8. Summary of CDDs/CDFs Detected in Fish Tissue as Part of the EPA National Study of Chemical Residues in Fish[a]

Congener	% Sites where detected	Maximum	Mean	Standard deviation	Median
2,3,7,8-TCDD	70	203.6	6.89	19.41	1.38
1,2,3,7,8-PeCDD	54	53.95	2.38	4.34	0.93
1,2,3,4,7,8-HxCDD	32	37.56	1.67	2.39	1.24
1,2,3,6,7,8-HxCDD	69	100.9	4.30	9.25	1.32
1,2,3,7,8,9-HxCDD	38	24.76	1.16	1.74	0.69
1,2,3,4,6,7,8-HpCDD	89	249.1	10.52	25.30	2.83
2,3,7,8-TCDF	89	403.9	13.61	40.11	2.97
1,2,3,7,8-PeCDF	47	120.3	1.71	7.69	0.45
2,3,4,7,8-PeCDF	64	56.37	3.06	6.47	0.75
1,2,3,4,7,8-HxCDF	42	45.33	2.35	4.53	1.42
1,2,3,6,7,8-HxCDF	21	30.86	1.74	2.34	1.42
1,2,3,7,8,9-HxCDF	1	0.96[b]	1.22	0.41	1.38
2,3,4,6,7,8-HxCDF	32	19.3	1.24	1.51	0.98
1,2,3,4,6,7,8-HpCDF	54	58.3	1.91	4.41	0.72
1,2,3,4,7,8,9-HpCDF	4	2.57	1.24	0.33	1.30
EPA-TEQ[c]	NA	213	11.1	23.8	2.80

[a] Concentrations are picograms per gram (pg/g) or parts per trillion (ppt) by wet weight. The mean, median, and standard deviation were calculated using one-half the detection limit for samples which were below the detection limit. In cases where multiple samples were analyzed per site, the value used represents the highest concentration.

[b] Detection limits were higher than the few quantified values for 1,2,3,4,7,8,9-HpCDF and 1,2,3,7,8,9-HxCDF. Maximum values listed are measured values.

[c] This EPA study did not analyze concentrations of octachlorodibenzo-*p*-dioxin or octachlorodibenzofurans in fish tissues; this TEQ value does not include these two compounds.

CDDs = chlorinated dibenzo-*p*-dioxins; CDFs = chlorinated dibenzofurons; EPA = Environmental Protection Agency; HpCDD = heptachlorodibenzo-*p*-dioxin; HpCDF = heptachlorodibenzofuran; HxCDD = hexachlorodibenzo-*p*-dioxin; HxCDF = hexachlorodibenzofuran; NA = not applicable; PeCDD = pentachlorodibenzo-*p*-dioxin; PeCDF = pentachlorodibenzofuran; TCDD = tetrachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran; TEQ=Toxicity equivalency concentration

Source: EPA 1992

chlorine bleaching processes) had the highest median 2,3,7,8-TCDD concentrations (5.66 ppt), compared to the next highest source category, refinery/other industrial sites (1.82 ppt), industrial/urban sites (1.40 ppt), Superfund sites (1.27 ppt), and background sites (0.5 ppt). Source categories with the highest 2,3,7,8-TCDD concentrations in fish also had the highest TEQ values. OCDD and OCDFs were not analyzed in tissue because at the time the NSCRF study was initiated (1986), the TEFs were zero for these compounds. In 1989, TEFs for OCDD and OCDFs were increased to 0.001. Consequently, TEQ values presented in the NSCRF report may be underreported for samples collected at sites with sources of OCDD/OCDFs (e.g., wood preservers) (EPA 1992).

De Vault et al. (1989) collected samples of lake trout and walleye for CDD and CDF analysis from each of the Great Lakes and Lake St. Clair. One of the conclusions of the National Dioxin Study was that fish from the Great Lakes region were among the most severely contaminated in the United States. Fish were analyzed for 8 congeners of CDDs and 10 congeners of CDFs. Total CDD concentrations ranged from 7.2 ng/kg in lake trout from Lake Superior to 64.5 ng/kg in Lake Ontario (wet weight basis). Concentrations of 2,3,7,8-TCDD ranged from 1 ng/kg in lake trout from Lake Superior to 48.9 ng/kg in lake trout from Lake Ontario. The dominant congener in all but Lake Ontario was 1,2,3,7,8-PeCDD at concentrations ranging from 2.3 ng/kg in Lake Superior to 16.7 ng/kg in Lake Michigan. The only other congener that significantly contributed to the total CDD concentration was 1,2,3,6,7,8-HxCDD, which ranged from 1.3 ng/kg in Lake Superior to 10.9 ng/kg in Lake Michigan. Substantial interlake differences exist in the percentage of total CDD contributed by the various congeners. The 2,3,7,8-TCDD congener contributes a relatively small percentage of the total CDD in fish from Lakes Superior, Michigan, and Erie. It is comparatively more important in Lake Huron (32%) and Lake St. Clair (36%) and contributes 76% of the total CDD in Lake Ontario. The results of this study support the widespread contamination of the Great Lakes ecosystem and clearly show that both the concentration of individual congeners and the congener composition of total CDDs in Great Lakes fish vary significantly between lakes and in Lake Michigan between sites. The authors suggest that these differences may be associated with different sources and loadings of these compounds to each of the Great Lakes (De Vault et al. 1989). This is confirmed by the analysis of sources of CDDs in the Great Lakes which appear to be both from atmospheric deposition and industrial point sources (Hebert et al. 1994).

In another study, CDDs and CDFs were measured in four species of salmonids (coho salmon, lake trout, rainbow trout, and brown trout) collected from Lake Ontario (Niimi and Oliver 1989a). Total CDD concentrations ranged from 46 to 290 ng/kg (ppt) in whole fish and 60–366 ng/kg in muscle composite

samples. Levels of 2,3,7,8-TCDD in whole fish ranged from 60–20 ppt (wet weight basis). This represented 60% of total TCDDs and 10% of total CDDs. The HxCDD congener group was most dominant in all fish species and represented approximately 39% of the total CDD concentrations. High concentrations of OCDD were also detected in coho salmon (160 ng/kg whole fish and 280 ng/kg muscle) and lake trout (89 ng/kg whole fish and 28 ng/kg muscle) but not in brown or rainbow trout. The authors could not explain this difference; however, OCDD is typically the CDD present at the highest concentrations in Lake Ontario water, suspended sediments, and sediments. Although the total CDF concentrations were 75% lower than the total CDD concentrations, the levels of 2,3,7,8-TCDF (11–20 ppt) were comparable to levels of 2,3,7,8-TCDD (6–20 ppt). Results of another study by the same authors found that TEQ values for PCB concentrations in Lake Ontario salmonids were several fold higher than TEQ values for CDDs and CDFs in the same fish species (Niimi and Oliver 1989b).

Background concentrations of CDDs in fish were measured in the Mississippi River and Lake Orono in Elk River, Minnesota, a semi-rural location (Reed et al. 1990). No major industrial or commercial activity occurred in the area at the time of the study, and the survey was conducted as a baseline study prior to the operation of the Elk River Electric Generating Station (powered by refuse-derived fuel). None of the fish collected contained measurable amounts of 2,3,7,8-TCDD; however, one of the composites from the Mississippi River contained 3.9 ppt of total TCDD (wet weight basis). Detection limits ranged from 0.28 ppt to 6.6 ppt on a congener-specific and sample-specific basis and were not individually reported for each result. OCDD was the most abundant congener (average 59 ppt, range 56–62 ppt), followed in decreasing order by total HpCDD (average 19.3, range 15–22 ppt), total HxCDD (average 6.87 ppt, range 2.3–11 ppt), and total PeCDD (average 3.9 ppt, range 3.5–4.5 ppt) (Reed et al. 1990). Lake Orono showed the same pattern, with OCDD being the most abundant congener (average 39 ppt, range 35–43 ppt), followed by total HpCDD (average 10.5, range 10–11 ppt), and total HxCDD (3.0 ppt). PeCDDs were not detected in the Lake Orono samples (Reed et al. 1990).

Contamination of the Spring River in southwest Missouri by 2,3,7,8-TCDD is believed to have resulted from several well defined point-source waste disposal sites (Crunkilton et al. 1987). Analysis of 31 fish samples (11 different fish species) collected from 1981 to 1983 demonstrated a rapid decline in 2,3,7,8-TCDD concentrations in fish at increasing distances both upstream and downstream from the area of contamination. Mean concentrations of 2,3,7,8-TCDD 0.5 km downstream from the area of contamination were 38 ppt in whole fish and 20 ppt in fish fillets (wet weight basis). Mean concentrations in fish

caught more than 14 km downstream were below 4 ppt in both whole fish and fillet samples (Crunkilton et al. 1987).

Fish samples (butterfish, flounder, hake, and herring) collected in 1984 from the Atlantic Ocean off Long Branch, NJ, contained no detectable levels of 2,3,7,8-TCDD (detection limit <10 pg/g) (wet weight basis) (Firestone et al. 1986). Cod caught in the northwest Atlantic in November 1990 did not have detectable levels of any CDDs in their muscles or ovaries, although 5 of 10 liver samples had OCDD at a mean concentration of 0.8 ppt and TCDD was found in 3 of 10 samples at 0.1 ppt (Hellou and Payne 1993). A 4-year study of marine and freshwater fish and other edible aquatic organisms taken from Canadian waters that received effluents from pulp and paper mills indicated that 2,3,7,8-TCDD was the most prominent CDD found in the fish regardless of the tissue sampled or sampling location. The maximum 2,3,7,8-TCDD concentration detected in the edible organisms sampled was for crab hepatopancreas tissue (>500 pg/g) (wet weight basis). Whole fish samples also contained greater CDD concentrations than fillet samples (Whittle et al. 1993).

Several studies have been conducted to monitor 2,3,7,8-TCDD concentrations in fish and shellfish in northern New Jersey in the vicinity of a pesticide manufacturing site that allegedly released an estimated 4–8 kg of 2,3,7,8-TCDD over a 20-year period (Bopp et al. 1991). Samples of striped bass, blue crabs, and lobsters collected from Newark Bay and the New York Bight (marine waters directly offshore from New York Harbor) all contained high concentrations (up to 6,200 ppt) (wet weight basis) of 2,3,7,8-substituted TCDD, PeCDD, and CDFs (Rappe et al. 1991). Concentrations of HxCDD and HpCDD ranged from <0.1 to 220.7 ppt and <0.7 to 244.9 ppt, respectively. The concentrations of 2,3,7,8-TCDD in these marine organisms were higher than any other New Jersey samples and represented the highest concentrations of 2,3,7,8-TCDD reported for aquatic species. The two crustaceans sampled in the study had similar congener patterns; they all contained both a large number and large amounts of CDD and CDF congeners in addition to the 2,3,7,8-substituted chlorinated compounds. In contrast, the striped bass samples contained primarily the 2,3,7,8-chlorine-substituted congeners. The concentrations of 2,3,7,8-TCDD in crab hepatopancreas tissue ranged from 3,700 to 6,200 ppt and from 100 to 120 ppt in crab meat. Concentrations of 2,3,7,8-TCDD were lower in the lobster, ranging from 250 to 610 ppt in the hepatopancreas and 5 to 6 ppt in the meat. Concentrations of 2,3,7,8-TCDD in striped bass muscle tissue ranged from 84 to 730 ppt. In this study, the crustacean samples all contained very complex ion curves for the TCDDs showing 10 major and 5 minor peaks, while the striped bass samples primarily contained the 2,3,7,8-TCDD isomer and a few other isomers. With respect to the PeCDDs, the crustacean samples

contained 5–6 peaks including the 1,2,3,7,8-PeCDD (100 ppt in hepatopancreas and 1–2 ppt in meat), while the major isomer found in the striped bass was 1,2,3,7,8-PeCDD (5–10 ppt). Regarding the HxCDDs, the crustacean samples contained 3 major peaks one of which was 1,2,3,6,7,8-HxCDD (100–300 ppt in the hepatopancreas); while the striped bass samples contained concentrations <1 ppt. The HpCDD congeners (1,2,3,4,6,7,9- and 1,2,3,4,6,7,8-) were detected in crustacean hepatopancreas tissue ranging from 31.7 to 411.9 ppt, while meat samples contained 0.00-8.5 ppt. Striped bass tissue samples contained 4–11.4 ppt. Concentrations of OCDD ranged from 50.5 to 94.6 ppt in crustacean hepatopancreas tissues, 6.3–78.8 ppt in meat samples, while concentrations in striped bass ranged from 5.1–49.5 ppt (Rappe et al. 1991).

Cai et al. (1994) analyzed blue crab tissue (hepatopancreas and muscle) from Newark Bay and some adjacent areas of the New York Bight for 2,3,7,8-TCDDs and 2,3,7,8-TCDF and several other 2,3,7,8-substituted CDDs/CDFs. These authors found 2,3,7,8-TCDD concentrations in hepatopancreas tissue to be 10–20 times higher than muscle concentrations. Lipid content of the hepatopancreas is 6–9% as compared to 1% for muscle tissue. The highest concentration of CDDs/CDFs was detected in crabs collected at the station closest to the pesticide production facility. Crabs also had higher CDDs/CDF concentrations in September after feeding all summer than crabs collected in June. Concentrations of 2,3,7,8-TCDD up to 1 ppb were detected in some crabs collected from Newark Bay (detection limit 0.5–1 ppt). Hauge et al. (1994) conducted further studies of blue crabs and lobsters from three distinct fisheries in the Hudson-Raritan estuary. The Ambrose fishery includes Raritan and Sandy Hook Bays and extends to a 7-nautical-mile radius from Ambrose Light near the entrance to New York Harbor. The Alongshore fishery is a box-shaped area extending from Long Branch, NJ, south to Point Pleasant, NJ, and then extending off-shore approximately 25 nautical miles. The Offshore fishery extends eastward from the 50-fathom line to the 100-fathom line approximately 100 miles seaward to the edge of the continental shelf. Combined muscle/hepatopancreas samples of blue crabs from Raritan Bay and the lower Hudson River had a mean 2,3,7,8-TCDD concentration of 71.5 pg/g (range not detected to 260 pg/g) and a mean 2,3,7,8,-TCDF concentration of 67.1 pg/g (range not detected to 110 pg/g). The mean total TEQ concentration was 78.2 pg/g (ppt) (wet weight basis). Both the mean 2,3,7,8-TCDD concentration and the mean TEQ values exceeded the FDA guidelines for "no consumption" (>50 ppt) (see Chapter 7). FDA “no consumption” guidelines advise consumers that fish and shellfish should not be consumed when CDD concentrations exceed 50 ppt. 2,3,7,8-TCDD and TCDF were detected in 53 and 67% of crabs, respectively. Levels of 2,3,7,8-TCDD in muscle and hepatopancreas of lobsters were similar in animals from the Ambrose and Alongshore fisheries, with the mean concentration in both areas ranging from 34 to

41 pg/g of 2,3,7,8-TCDD/TCDF. The mean total TEQ values were 38.5 pg/g (Ambrose fishery) and 44.4 pg/g (Alongshore fishery). Mean 2,3,7,8-TCDD/TCDF levels thus exceeded the FDA "safe consumption" level (#25 ppt), but did not exceed the FDA "no consumption" level (>50 ppt). None of the lobsters from the Offshore fishery contained detectable TCDD/TCDF. Analysis of separate muscle and hepatopancreas tissues from individual lobsters yielded detectable concentrations of both contaminants only in the hepatopancreas (<26–410 pg/g for 2,3,7,8-TCDD and <33–380 pg/g for 2,3,7,8-TCDF). Concentrations in lobster muscle tissues were all below detection limits of 6–20 pg/g for 2,3,7,8-TCDD and 10–25 pg/g for 2,3,7,8-TCDF.

Concentrations of CDDs/CDFs were also evaluated in a bivalve mollusc, the soft-shelled clam *(Mya arenaria)* in Newark Bay, Arthur Kill, and Raritan Bay (Brown et al. 1994). Clams from Newark Bay contained 11–20 ppt TCDD, 3.5–5 ppt TCDF, and 13–25 ppt TEQ; those from Arthur Kill contained 4.8–7.7 ppt TCDD, 3.1–5.1 ppt TCDF, and 6.8–11 ppt TEQ; and those from Raritan Bay contained 0.5–1.1 ppt TCDD, 2–4.6 ppt TCDF, and 1.2–2.1 ppt TEQ (wet weight basis). The maximum TEQ concentration of the Newark Bay clams (25 ppt) approached the upper limit of the FDA "safe consumption" level of #25 ppt. The FDA believes that consumption of fish or shellfish with CDD concentrations #25 ppt should not result in any serious health effects. Concentrations decreased with increasing distance from the suspected pesticide plant site near Newark Bay. The authors also showed that the clams could eliminate TCDD and TCDF when they were removed to clean water sites. The half-lives of the TCDD, TCDF, and TEQ were calculated to be 45, 111, and 66 days, respectively.

Concentrations of CDDs and CDFs were also reported in wood ducks (*Aix sponsa*), species of migratory waterfowl collected near Bayou Meto, Arkansas (White and Hoffman 1995). The EPA identified a former 2,4,5-T chemical manufacturing plant as the source of the contamination and subsequently listed the areas on the NPL of hazardous waste sites in 1982. Residues in wood duck eggs based on 2,3,7,8-TCDD (TEQs) ranged up to 611 ppt, and the egg arithmetic means were 90-fold higher at the site nearest the point source discharge compared to the reference site. The State of Arkansas has issued a wildlife consumption advisory for wood ducks in the Bayou Meto area (EPA 1998).

CDDs were determined in pooled samples of ringed seal *(Phoca hispida)* blubber, beluga whale *(Delphinapterus leucas)* blubber, and polar bear *(Ursus maritimus)* liver and fat collected from several areas throughout the Canadian north (Norstrom et al. 1990). All seal samples and all but one polar bear sample had detectable levels of 2,3,7,8-TCDD (wet weight) ranging from 2 to 37 ppt, but 2,3,7,8-TCDD was not

found in beluga blubber (<2 ppt). All seal samples and one of the three beluga whale samples contained 2,3,7,8-TCDF (2-7 ppt), but 2,3,7,8-TCDF was not found in polar bear samples. OCDD concentrations in seal blubber and polar bear samples ranged from not detected (<8 ppt) to 43 ppt. No biomagnification of TCDD and OCDD occurred from seal to bear fat. The highest concentrations of 2,3,7,8-TCDD and OCDD in seals and bears were found in the central Canadian Arctic Archipelago, and the lowest concentrations were found in the Hudson Bay area. The reason for higher concentrations of 2,3,7,8-TCDD and OCDD in the Arctic than in sub-Arctic areas is thought to be transpolar movement of aerosols from combustion-related sources originating in Eurasia (Norstrom et al. 1990). CDDs and CDFs were determined in caribou tissue samples from 7 herds across the Canadian Arctic (Hebert et al. 1996). In contrast to marine mammals, concentrations for caribou were extremely low, sub-ng/kg (lipid basis), for all congeners except OCDD and 1,2,3,7,8-PeCDD in one herd. OCDD was found in most of the samples at concentrations ranging from < 0.2 ng/kg in fat to 4.7 ng/kg in adipose tissue. The one pooled liver sample analyzed from the Yukon had an OCDD concentration of 11 ng/kg (lipid basis). 2,3,7,8-TCDD was detected in adipose tissue samples of two herds in the eastern Canadian Arctic at levels of 0.73 and 0.14 ng/kg, but was not detected in tissue samples from other herds at detections limits as low as 0.03 ng/kg (lipid basis). CDF levels were sub-ng/kg in all cases. TEQs were dominated by non-ortho substituted PCBs in all cases, and ranged from 0.33 ng/kg to 3.29 ng/kg in adipose tissue. The authors concluded that caribou tissues are therefore less contaminated than tissues from marine mammals.

Consumer products.

Cigarettes and Cigarette Smoke. CDDs have been detected in cigarettes and cigarette smoke. In a recent study, Lofroth and Zebuhr (1992) detected CDD/CDF concentrations in both mainstream (collected directly on a glass fiber filter) and sidestream smoke (emitted into an acrylic box and then collected on a glass fiber filter) from a single brand of commercially available Swedish cigarettes. These authors reported that the mainstream smoke from 20 cigarettes contained about 18 pg TEQ (1 pg TEQ per cigarette), while sidestream smoke contained 39 pg TEQ (2 pg TEQ per cigarette). No particular isomer contributed more than 20% to the total TEQ value. Most isomers were not present at concentrations above the detection limits (0.3–1.3 pg), with the exception of 1,2,3,4,6,7,8-HpCDD (6.8 pg), 1,2,3,4,6,7,8-HpCDF (4 pg), and OCDD (7.3 pg). An earlier study that used low-resolution mass spectrometry for analysis of CDDs in cigarette smoke obtained by a continuous smoking process (all cigarette tobacco gave rise to mainstream smoke) found that HpCDD was the most abundant homologue detected, accounting for >90% of the total CDDs (Muto and Takizawa 1989).

Paper Products. CDDs are formed during pulp bleaching, and as a result they have been found in many different types of paper products. 2,3,7,8-Substituted CDDs were determined in different samples of coffee-filter paper (Beck et al. 1988b, 1989d). 2,3,7,8-TCDD was the most abundant congener detected at a mean concentration of 3.85 ppt (range 1.6–7.3 ppt). OCDD was detected at a mean concentration of 2.05 ppt (range 0.7–3.5 ppt). PeCDDs, HxCDDs, and HpCDDs were identified at concentrations ranging from 0.03 to 0.7 ppt. In an earlier study, HxCDD was the most abundant homologue detected in coffee filters (2.1 ppt) and 2,3,7,8-TCDD was found at concentrations of 1 ppt (Beck et al. 1988b). Coffee brewed without filters did not contain any detectable CDDs; however, coffee brewed with one filter showed leaching of TCDDs from the paper into the coffee. Carryover (leaching) rates were 25% for 2,3,7,8-TCDD, indicating that people who drink coffee brewed with paper filters containing 5 ppt 2,3,7,8-TCDD may ingest small quantities (<5 pg) of 2,3,7,8-TCDD per day (Beck et al. 1989d). Hashimoto et al. (1992) also analyzed CDD/CDF concentrations in coffee-filter paper available in Japan. These authors reported mean TEQ values of 0.89 pg/g (ppt) (range 0.042–3.6 pg/g) for chlorine-bleached filters, 0.13 pg/g (range 0.079–0.18 pg/g) for oxygen-bleached filters, and 0.009 pg/g (range 0.00038–0.017 pg/g) for unbleached filters. Chlorine-bleached filters also gave the highest mean TCDD and TCDF elutions (range <0.043–2.1 and <0.081–6 pg/g, respectively). Oxygen-chlorinated filters gave considerably lower elutions (range <0.085–0.14 and 0.23–0.26 pg/g, respectively), while unbleached filters produced elutions near the detection limits (<0.094 pg/g). Approximately one-third of the total CDD/CDF contamination was eluted from the filter paper into the coffee during brewing; however, almost the same amount was eluted from the filters with hot water. This leaching rate of approximately 30% agrees with that obtained by Beck et al. (1989d). The elution ratio was almost constant for all CDD/CDF congeners and isomers. Using the maximum TEQ value of 3.6 pg/g paper and the minimum TEQ value of 0.00038 pg/g paper, the TEQ values for one cup of coffee were calculated to range from 0.000015 pg to 1.4 pg. The authors suggest that any potential health risk from CDD/CDF exposure from coffee-filter paper is small and can be further reduced by rinsing the filter prior to brewing the coffee.

CDDs (TCDD, PeCDD, HxCDD, HpCDD, and OCDD) are also present in newsprint, facial (cosmetic) tissue, and recycled paper at levels ranging from <0.4 to 335 ppt (Beck et al. 1988b). OCDD was the most abundant congener detected in newsprint (37 ppt). HxCDD was the most abundant homologue detected in cosmetic tissue (79 ppt) and recycled paper (335 ppt). 2,3,7,8-TCDD was found at lower concentrations in cosmetic tissue (1.1 ppt), and recycled paper (0.6 ppt) (Beck et al. 1988b). Another study of CDDs in bleached and unbleached consumer paper products showed that the highest levels of 2,3,7,8-TCDD were found in bleached coffee filters (5 ppt), unbleached coffee filters (2.0 ppt), bleached shopping bags

(1.3 ppt), and cigarette paper (1.4 ppt) (Wiberg et al. 1989). Concentrations were found to be lower in the unbleached products than in the corresponding bleached products (Wiberg et al. 1989). 2,3,7,8-TCDD has been measured in tea bags at concentrations ranging from not detected (0.36 ppt) to 4.79 ppt (Sullivan and Stanford 1990).

The use of newsprint for cow bedding was examined to determine if CDDs in the newsprint would find their way into the cow's milk as a result of the cow's ingesting its bedding. Although HxCDD and OCDD were detected in the milk, TCDD was not detected (detection limit 0.5 ppt) (Shane et al. 1993).

Ryan et al. (1992) analyzed the concentrations of CDDs/CDFs in Canadian bleached-paper milk containers from 1988 to 1989 and examined the resulting concentrations transferred to the milk. Milk-carton paper manufactured prior to 1989 tested positive for 2,3,7,8-TCDF and 2,3,7,8-TCDD, with levels on a TEQ basis varying between 1.4 and 55 ng/kg of paper. Bleached milk-carton paper produced after mid-1989 tested negative for these compounds at a limit of detection of 1 ng/kg paper. Storage of 3 types of milk in the pre-1989 low- and high-level cartons resulted in the transfer of the TCDD/TCDF into the milk, most of which occurred within the first 7 days. The TCDD/TCDF transfer varied between 3 and 25%, with whole and 2% fat milk accumulating about twice the concentrations of skim milk. On the basis of these results, milk stored for up to 14 days at 5 EC in currently produced bleached-paper containers with less than 1 ng TEQ/kg of paper would not contain any detectable CDDs/CDFs (<0.005 ng TEQ/kg milk).

An FDA study of the migration of TCDD from paper products that come in contact with food found that TCDD was present in all paper products at concentrations ranging from 0.5 ppt for coated paper trays to 13 ppt for coated paper cups (average 2–8.5 ppt). Migration of TCDD from the paper into the food ranged from below detectable limits for coated juice cartons to 24% for coffee filters. Most CDDs migrated in the range of 4–8%. The TEQ estimated concentration values ranged from 1.5 ppt for coffee filters to 140 ppt for paper plates (Cramer et al. 1991).

LeBel et al. (1992) analyzed a wide variety of paper products purchased from retail stores in Canada in 1988 and 1991 for TCDD/TCDF through OCDD/OCDF. The congeners exhibiting the highest concentrations in most paper products were 2,3,7,8-TCDF, OCDD, and 2,3,7,8-TCDD. With respect to the TEQ values, the mean TEQ for disposable diapers increased from 1.4 to 2.0 pg/g from 1988 to 1991. The TEQ values decreased during the same period for facial tissues (5.2–4.0 pg/g), paper plates (6.4–2.2 pg/g), paper cups (22.7–10.5 pg/g), and coffee filters (3.7–0.1 pg/g). The CDD/CDF concentrations found in

coffee filters in 1988 were similar to concentrations reported in the United States (Cramer et al. 1991) and in Germany (Beck et al. 1988b). The authors cautioned that, given the small number of both paper products and samples analyzed, these data do not permit them to draw any conclusions regarding trends in CDDs/CDF levels in paper products.

Dyes and Pigments. Malisch (1994) reported the presence of CDDs/CDFs in colored candle wax produced with the dye pigment Violet 23, which is derived from chloranil. The three candle samples with the highest contamination contained 1.8, 1.4, and 0.8 ng TEQ/kg (ppt). The author also noted that candles of the same color could have highly different CDD/CDF concentrations based on the composition of dye pigments used in the manufacturing process.

Three pigments used in fabric dyeing that are derived from chloranil include the dioxazine pigments Violet 23 and Direct Blue 106 and 108 (Williams et al. 1992). Concentrations of the congeners OCDD and OCDF predominated in the pigment Blue 106 and ranged from 18,066 to 41,953 ng/g (ppb) for OCDD and 1,006–12,463 ng/g (ppb) for OCDF. Pigment Blue 108 contained much lower concentrations of CDDs/CDFs, although OCDD and OCDF were also the predominant congeners detected at 23 and 11 ng/g, respectively. Violet 23 contained higher CDD/CDF concentrations than Direct Blue 108 but lower concentrations than Direct Blue 106. OCDD concentrations ranged from 806 to 11,022 ng/g (ppb), while OCDF concentrations ranged from 125 to 3,749 ng/g (ppb). The TEQ values for Direct Blue 106, Direct Blue 108, and Violet 23 were 35.4, 0.1, and 9.1 ng/g (ppb), respectively.

Textile Products. A recent study has identified sources of CDDs/CDFs found in textiles. Horstmann and McLachlan (1994a) detected CDD/CDF concentrations in new textile products ranging from less that 50 pg/g to as high as 290,000 pg/g. The authors believe that textile finishing processes are not the source of the high CDD/CDF concentrations because of the randomness of the textiles with high concentrations. Since PCP is still being used in developing countries, especially for purposes of preserving cotton during sea transport, the authors hypothesize that this is a likely source.

Dry Cleaning Fluid Residues. Chemical analysis of dry cleaning solvent residues collected in Germany prior to 1993 indicated that residues from machines using perchloroethylene contained an average concentration of 256 ppb CDD/CDF, with 2,3,7,8-TCDD being detected in 21 of 28 samples; however, the HpCDD and OCDD congeners comprised between 90 and 95% of the CDDs/CDFs found (Towara et al. 1992). Horstmann and McLachlan (1994b) detected CDD/CDF residues in used dry cleaning fluid and

concluded that the source of the CDDs/CDFs residues in the dry cleaning fluid were introduced by dry cleaning new, unwashed textiles that had been treated with pentachlorophenol (PCP).

Motor Vehicle Exhaust. CDDs have also been identified in automobile exhaust emissions (Marklund et al. 1987, 1990). 2,3,7,8-TCDD was found in car exhaust from 4 Swedish cars running on leaded gasoline at levels ranging from <0.05 to 0.3 ng/24.8 km (0.002–0.01 ng/km) running cycle. PeCDD was also found in the exhaust of cars running on leaded gasoline at levels ranging from 6 to 98 ng/24.8 km (0.24–3.95 ng/km). No CDDs were found in samples where unleaded gasoline was used at detection limits of 0.05 ng (2,3,7,8-TCDD) and 0.3 ng (PeCDD) (Marklund et al. 1987). Another study of exhaust emissions from cars running on leaded and unleaded gasoline found total HpCDD concentrations ranging from not detected to 0.482 ng/km and OCDD concentrations ranging from not detected to <0.510 ng/km for the cars running on leaded gasoline (Bingham et al. 1989). HpCDD was not detected in car exhaust emissions from a single car running on unleaded gasoline. OCDD concentrations ranged from not detected to <0.110 ng/km (Bingham et al. 1989). Most recently, however, Cirnies-Ross et al. (1996) reported that using copper in diesel fuels to reduce soot generation in engines was accomplished at the expense of increasing CDD/CDF during combustion. These authors reported that using the copper doped fuel significantly increased CDD/CDF particulate formation from < 20 ng/L TEQ in normal fuel to almost 60 ng/L TEQ in doped fuel at an engine output of 1 kW. The increased CDD/CDF particulate formation was most striking at low engine output (1 kW).

From the research conducted on CDD emissions from vehicles running on leaded and unleaded gasoline, it is clear that CDD emissions are typically less in cars running on unleaded gasoline. It should be noted however, that because the use of leaded gasoline is no longer permitted in the vast majority of domestic automobiles in the United States, this source of CDD emissions to the air should have been significantly reduced in recent years (EPA 1996a).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

5.5.1 General Population

Currently, consumption of food (including human milk) is by far the most important pathway for exposure to CDDs for the general population representing over 90% of the total daily intake. Other pathways of exposure include inhalation of CDDs from municipal, medical, and industrial waste incinerators and other

incineration and combustion processes (~2% of the daily intake), and ingestion of drinking water(<0.1% of the daily intake) (Travis and Hattermer-Frey 1987; Schaum et al.1994).

Foods. Food is the major source (>90%) of human exposure to CDDs (Beck et al. 1989a; Hattemer-Frey and Travis 1989; Liem and van Zorge 1995; Rappe 1992; Schaum et al. 1994; Schecter et al. 1994d, 1994e, 1996a). An estimate of the daily intake of 2,3,7,8-TCDD by adults in the general U.S. population from ingestion of contaminated food items and drinking water and inhalation of ambient air is given in Table 5-9. The average daily adult intake of 2,3,7,8-TCDD estimated by the model was 47 pg/day (Hattemer-Frey and Travis 1989) with a lower bound daily intake of 8 pg/day and an upper bound daily intake of 300 pg/day. Food, especially meat, and dairy products, accounted for 98% of the total daily intake of 2,3,7,8-TCDD. Hattemer-Frey and Travis (1989) estimated that the average daily intake of 2,3,7,8-TCDD for an adult in the United States from meat alone was 23 pg/day, accounting for 50% of the total daily intake of 2,3,7,8-TCDD from food sources. The average daily intakes of 2,3,7,8-TCDD from milk, produce, and fish were 13 pg/day (27%), 5 pg/day (11%), and 5 pg/day (10%), respectively of the total daily intake in the United States (Hattemer-Frey and Travis 1989). However, for certain subpopulations (recreational and subsistence fishers), fish consumption may be a more important source of CDDs. The maximum daily intake of 2,3,7,8-TCDD for residents of the Great Lakes region who regularly consume fish from the Great Lakes was estimated to range from 390 to 8,400 pg/day (EPA 1985a). Inhalation of ambient air and ingestion of water are not major pathways of human exposure, accounting for only 2% (1 pg/day) and <0.01% (6.5×10^{-3} pg/day), respectively, of the total daily intake of 2,3,7,8-TCDD (Hattemer-Frey and Travis 1989). The percentage of daily intake of 2,3,7,8-TCDD estimated by Hattemer-Frey and Travis (1989) from each exposure pathway agrees closely with more recent estimates made by Schaum et al. (1994) for intakes of total CDDs/CDFs (Table 5-10). However, quantitatively, the estimates differ by a factor of 2–3 because Hattemer-Frey and Travis (1989) considered only 2,3,7,8-TCDD, while Schaum et al. (1994) based their estimates on all CDDs and CDFs.

Based on their congener-specific analysis of 18 food samples collected in Binghamton, New York, Schecter et al. (1994d), estimated the U.S. mean daily exposure to CDD equivalents for an adult (65 kg body weight) to range from 18 to 192 pg TEQs depending on how not-detected values were treated. This is equal to a daily adult intake of CDDs/CDFs ranging from 0.3 to 3.0 pg TEQs/kg body weight. These authors reported that total CDDs ranged from 0.35 to 2.91 ppt (wet weight) in fish, from 0.6 to 59.3 ppt in meat products, and from 0.6 to 14 ppt in dairy products. The total CDD/CDF TEQ value ranged from 0.023 to 0.13 ppt for fish, 0.03 to 1.5 for meat products, and 0.04 to 0.7 for dairy products. The authors

Table 5-9. Estimated Average Daily Intake of 2,3,7,8-TCDD by the General U.S. Population

Source/pathway	Daily intake (pg/day)	Percentage of total daily intake
Ambient sources (total)	**1.01**	**2**
Air/Inhalation	1	2
Water/Ingestion	6.5×10^{-3}	<.01
Soil/Ingestion	—	—
Food sources (total)/ Ingestion	**46**	**98**
Produce (fruits and vegetables)	5	11
Milk	13	27
Meat	23	50
Fish	5	10
Total intake	**47**	**100**

Source: Travis and Hattemer-Frey 1989

2,3,7,8-TCDD = 2,3,7,8-tetrachlorodibenzo-*p*-dioxin

Table 5-10. Estimated Daily Background Exposure to CDDs/CDFs in the General U.S. Population

Source	Daily intake (pg TEQ/day)	Percentage of total daily intake
Ambient Sources (total)	**3**	**2.5%**
Air	2.2	1.8
Water	0.008	0.01
Soil	0.8	0.7
Food (total)	**116**	**97%**
Produce (fruits and vegetables)	ND	ND
Milk and Milk Products	42	35
Milk	18	15
Cheese	24	20
Meat/Meat Products/ Eggs	66.1	55
Pork	12	10
Beef	37	30.8
Chicken	13	11
Eggs	4.1	3.4
Fish and Fish Oil	7.8	6.6
Total Exposure	**120**	**100%**

Source: Schaum et al. 1994

CDD = chlorinated dibenzo-*p*-dioxins; CDF = chlorinated dibenzofurans; ND = no data

reported that a vegetarian diet (vegan diet with no consumption of dairy products) might have health advantages by lowering daily intakes to only 2% of the level estimated for persons consuming fish, meat, and dairy products (Schecter et al. 1994d, 1984e). An ovo-lacto vegetarian diet that contains eggs and dairy products would not achieve this same reduction level. More recently, these same authors estimated the U.S. mean daily exposure to CDD equivalents based on an expanded analysis of 100 food samples collected in supermarkets in Binghamton, New York; Chicago, IL; Louisville, Kentucky; Atlanta, Georgia; and San Diego, California (Schecter et al. 1996a). For 1995, these authors report that the estimated U.S. mean daily exposure to CDDs/CDFs TEQs for an adult (65 kg body weight) ranges from 34 to 167 pg TEQs. This is equivalent to a daily adult intake of CDDs/CDFs ranging from 0.52 to 2.57 pg TEQs/kg body weight. If PCB TEQs are also considered (where TEF values are available), the daily adult intake ranges from 1.16 to 3.57 pg TEQ/kg body weight/day. A more recent survey of CDDs/CDFs in total diet food samples in Canada was conducted by Ryan et al. (1997). These authors found, through analysis of more than 100 food samples collected from commercial outlets in 1992 and 1993, that the total TEQ intake for CDDs/ CDFs was about 0.8 pg TEQs/kg/day. If all dioxin-like PCBs were also included, this TEQ value rose to approximately 1.2 pg TEQs/kg/day.

In 1995, Schecter and Li (1997) conducted a congener-specific analysis of CDDs, CDFs, and dioxin-like PCBs in US fast foods. These authors reported TEQ values from 0.03–0.28 pg/g wet weight for McDonald's Big Mac, 0.03–0.29 pg/g for Pizza Huts personal pan supreme pizza with all toppings, 0.01–0.49 pg/g for Kentucky Fried Chicken 3 piece original recipe meal, and 0.3–0.31 pg/g for Haagen-Daz chocolate-chocolate chip ice cream. Daily TEQ consumption per kilogram body weight assuming a 65-kg adult, from one serving of each of the fast foods tested ranged between 0.046–1.556 pg/kg. This same value in a 20-kg child (6-year-old) ranged from 0.15 to 5.05 pg/kg. A child on average consumes three times more TEQs on a per kg/body weight basis as compared to adults eating any one of the fast foods tested.

Studies conducted in other industrialized countries have reported similar values to those obtained for the United States. Estimated daily intakes of CDDs and CDFs from various foods were calculated in a Canadian study of foods domestically produced in Canada or imported from the United States (Birmingham et al. 1989). Based on contamination levels (CDDs and CDFs) in samples of meats, eggs, fruits, and vegetables from the United States and Canada, a total daily intake of 1.52 pg TEQs/kg body weight was calculated (for a 60-kg adult). The foods that contributed the most exposure to CDDs/CDFs TEQs were milk, eggs, and beef. Approximately one-half (0.81 pg TEQs/kg) was contributed by milk products. Eggs

and beef were also estimated to make substantial contributions (0.28 and 0.27 pg TEQs/kg, respectively). The total contribution from these animal products to the daily dietary intake is 1.5 pg TEQ/kg body weight (99% of the total). Plant products (fruits, vegetables, and wheat products) contribute only 0.068 pg TEQ/kg body weight/day (1% of the total). The authors also estimated that consumption of freshwater fish was 0.28 pg TEQ/day, thus the total daily intake of CDDs/CDFs amounted to 1.8 pg TEQ/kg body weight (Birmingham et al. 1989).

In a German study where 22 samples of different foodstuffs (cow's milk, butter, pork, beef, lamb, chicken, eggs, and fish) were analyzed for CDDs/CDFs, the total average daily intake of 2,3,7,8-TCDD via food was 0.35 pg/kg/day and for total CDDs/CDFs was 1.3 pg TEQs/kg body weight/day for a 70-kg adult (Beck et al. 1989a). Meat, milk and other dairy products, and fish were the most important food groups contributing 17.9, 26.6, and 38.6 pg TEQ/day, respectively, to the body burden. Eggs, vegetable oil, vegetables, and fruits contributed 3.1, 0.3, 2.4, and 1.3 pg TEQ/day, respectively, to the body burden (Beck et al. 1989a).

In general, vegetation contamination from airborne sources of CDDs results in more substantial exposures to grazing animals due to the proportionally higher accumulation from foliar (leaf) deposition as compared to root uptake (Travis and Hattemer-Frey 1987). OCDD is the CDD contaminant most concentrated by plants. This supports the contention that atmospheric deposition is the primary mechanism by which plants become contaminated, as OCDD is not readily available for root uptake or translocation in plants (Hulster and Marschner 1993; Muller et al. 1993). The concentration of 2,3,7,8-TCDD (due to root uptake and foliar deposition) on vegetation consumed by cows was estimated to be 0.11 ng/kg (97% due to foliar deposition). The estimated total concentration on exposed produce and vegetation consumed by humans was 0.02 ng/kg (67% resulting from foliar deposition) (Travis and Hattemer-Frey 1987). A model has been developed that estimated the CDD content in cow's milk based on emissions from a nearby municipal solid-waste incinerator. The model includes three components that predict atmospheric transport and deposition, soil and grass concentrations, and uptake and bioavailability from fodder to cows. Results indicate that models can be used to estimate CDD contamination in foods (Lorber et al. 1994; Slob and Jaarsveld 1993).

Municipal and industrial incinerators and other combustion sources. Combustion processes are widely recognized as a source of CDDs/CDFs. Using a model, Hattemer-Frey and Travis (1989) estimated a total daily intake of CDD/CDF of 3×10^{-4} ng TEQs/day associated with exposure to a typical,

state-of-the-art municipal solid-waste (MSW) incinerator, assuming a CDD/CDF emission rate based on the geometric mean from 11 proposed MSW facilities. Daily intakes of CDD/CDF in TEQs associated with exposure to a typical state-of-the-art municipal waste incinerator were estimated to be $1.3x10^{-4}$ ng/day from inhalation, $1.1x10^{-4}$ ng/day from total ingestion, $5.7x10^{-5}$ ng/day for mother's milk and $2.2x10^{-6}$ ng/day from dermal absorption. This total daily intake value (3×10^{-4} ng TEQs/day) was 160 times lower than the estimated total daily background intake from all sources of CDDs (0.047 ng/day) to which the general U.S. population is exposed. Thus, the authors concluded that MSW incinerators will not substantially increase human exposure to CDDs/CDFs above normal background levels (Hattemer-Frey and Travis 1989). Table 5-11 shows estimated average daily intakes of CDD/CDF TEQs from various exposure pathways. Fries and Paustenbach (1990) evaluated the effects of 2,3,7,8-TCDD from incinerator emissions to humans. These authors also concluded that airborne emissions of CDDs/CDFs from modern waste incinerators that are equipped with appropriate air pollution devices should not pose a significant health hazard via inhalation of CDD contaminated particles or via contamination of foods regardless of the incinerator location. Hattemer-Frey and Travis (1989) focused on ideal state-of-the-art incinerators. In a more recent analysis, Travis and Hattemer-Frey (1991) estimated that the total daily intake of CDDs/CDFs (TEQs) by a maximally exposed individual living near a modern municipal solid waste incinerator was 0.7 pg/day (0.9% of total daily intake) and 92.8 pg/day (99.1%of total daily intake) was from all other background exposures. These estimates are supported by recent data of Schecter et al. (1995) who found that workers who operate municipal waste incinerators have blood levels of TEQs which do not differ significantly from background levels.

The presence of CDDs in cigarette smoke is also of importance with respect to inhalation exposure since cigarette smoke is inhaled directly into the lungs. Daily exposure to CDDs by smoking 20 cigarettes was estimated to be 18 TEQ pg/day equivalent to a daily intake of 0.26 pg/kg body weight/day (for a 70-kg adult) (Lofroth and Zebuhr 1992).

Consumer products. The presence of CDDs in a variety of consumer products ranging from plastic packaging to colored candle wax, and from textiles to air filters for home-heating systems suggests that CDDs are virtually ubiquitous in the environment (Beck et al. 1989d; Berry et al. 1993; Horstmann and McLachlan 1994; Malisch 1994; Ryan et al. 1992). 2,3,7,8-TCDD and 2,3,7,8-TCDF have been found in many paper products, including coffee-filter paper, although present day paper products now contain less than 1 ng/kg TEQ. Under the preconditions of using 4 small coffee-filter papers (4×1 g) per day containing 5 ppt 2,3,7,8-TCDD and 23 ppt 2,3,7,8-TCDF, which leaches into coffee, a daily exposure of 5 pg

Table 5-11. Estimated Average Daily Intake of TEQs Associated with Exposure to a State-of-the-Art Municipal Waste Incinerator

Exposure pathway	Daily intake (ng/TEQ/day)	Percentage of total intake
Inhalation	1.3×10^{-4}	43
Total ingestion	1.1×10^{-4}	37
Mother's milk	5.7×10^{-5}	19
Dermal absorption	2.2×10^{-6}	1
Total intake	3.0×10^{-4}	100

Source: Hattemer-Frey and Travis 1989

TEQs = toxicity equivalencies of dioxins and dibenzofurans

2,3,7,8-TCDD and 32 pg 2,3,7,8-TCDF or nearly 10 pg TEQs (for these two compounds combined) were calculated for a coffee drinker consuming all the coffee (this is a worst-case assumption that an individual would consume all the coffee brewed) (Beck et al. 1989d). TCDD, PeCDDs, HxCDD, HpCDD, and OCDD were found in all samples derived from consumer products (including plastic packaging, clothes dryer lint, vacuum cleaner dust, room and car air filters, and furnace filter dust), and bleached and unbleached paper products tested. In general, the more highly chlorinated congener groups (HxCDD, HpCDD and OCDD) exhibited the highest concentrations. The highest levels of most congeners were found in home-furnace filter dust, which contained HxCDD, HpCDD and OCDD at concentrations up to 135 ppt, 9,990 ppt, and 24,600 ppt, respectively (Berry et al. 1993). Car air filters displayed a different CDD profile than the other products with the highest concentration detected for TCDD (2,080 ppt), PeCDD (1,320 ppt) and HxCDD (1,320 ppt). TEQ values for CDD/CDF were highest for the home furnace filter (170 ppt), car air filter (84 ppt), and room air filter (29 ppt).

Adipose tissue residues. The general population of the United States is continuously exposed to small amounts of CDDs, as exemplified by the fact that all human adipose tissue samples contain CDDs (Orban et al. 1994; Patterson et al. 1986a; Ryan et al. 1986; Schecter et al. 1986b; Stanley 1986; Stanley et al. 1986). Results of the National Human Adipose Tissue Survey (NHATS) conducted in 1982, which estimated the general population exposure to toxic organic chemicals, showed that 2,3,7,8-TCDD was detected in 35 of 46 (76%) composite samples with an average lipid-adjusted concentration of 6.2±3.3 ppt (Stanley 1986; Stanley et al. 1986). The average concentration of the other CDD compounds ranged from 43.5 ppt for PeCDD (detected in 91% of the composites) to 694 ppt for OCDD (detected in 100% of the composites). The congener distributions found in adipose tissue are similar to those found in human milk (i.e., OCDD was the most abundant and 2,3,7,8-TCDD the least abundant congener). The analysis of 46 composite adipose samples verified the prevalence of the 2,3,7,8-substituted tetra- through octa CDDs in the U.S. population (Stanley 1986; Stanley et al. 1986). The number of adipose samples in each composite was defined based on differences in age, gender, race, and regional affiliation of the individuals from whom the specimens were collected. The results also suggested that adipose tissue concentrations tended to increase with age for the congeners tested, with the exception of PeCDD. The NHATS study also showed regional differences in CDD concentrations in adipose tissue, with the greatest exposure occurring in the East North Central region of the United States (i.e., Ohio, Michigan, Indiana, Illinois, and Wisconsin). Exposure was also relatively high in the mid-Atlantic and East South Central regions (Phillips and Birchard 1991).

Results of the more recent 1987 NHATS Study were summarized by Orban et al. (1994). Human adipose samples from autopsy cases were obtained through a network of pathologists to provide a representative sample of the general U.S. population. NHATS samples collected during 1987 were analyzed for 7 CDDs and 10 CDFs and the results are summarized in Table 5-12. Data were evaluated by census region, age group, sex, and racial group. The average concentration of 2,3,7,8-TCDD in adipose tissue in the U.S. population was estimated to be 5.38 pg/g (±6%). The 1987 survey data clearly show that nearly all of the CDD/CDF congeners increased with the age of the donor (i.e., the highest concentrations occur in the 45+ age group and the lowest concentrations occur in the 0–14 age group). On a regional basis, only the average concentration of 2,3,4,7,8-PeCDF was statistically different in the Northeast (13.7 pg/g) compared to the national average (9.7 pg/g). Orban et al. (1994) also compared NHATS 1987 data to the NHATS 1982 data. Because of slight differences in study design, the congeners that were most comparable between the two surveys were 2,3,7,8-TCDD and 2,3,7,8,-OCDD. Statistical analysis of the two survey data sets revealed no significant differences between the national average concentration of 2,3,7,8, TCDD determined in 1982 and 1987. There were also no significant differences in the profiles with respect to census region, sex, and race. With respect to age, however, there was a significant difference; the 1987 NHATS data demonstrated that the concentration of 2,3,7,8-TCDD consistently increased with the age of the donor. The average concentration of 2,3,7,8-TCDD in the 1987 survey increased from 1.98 pg/g in the 0–14-year-old group, to 4.37 pg/g in the 15–44-year-old group, to 9.4 pg/g in the 45+-year-old group. The average concentration of OCDD in the 1982 survey was 768 pg/g (±79.7 standard error) as compared to 724 pg/g (± standard error 28.6) in the 1987 study.

Analysis of human adipose tissue from 35 autopsy cases from Georgia and Utah found 2,3,7,8-TCDD in all of the samples at a concentration range (whole-weight) of 2.7–19 ppt (Patterson et al. 1986b). The geometric mean value for 2,3,7,8-TCDD in these samples on a whole-weight basis was 7.1 ppt. The geometric mean value for 2,3,7,8-TCDD in 31 of these samples on a lipid basis was 9.6 ppt. The histories of exposure to 2,3,7,8-TCDD were not known for any of the autopsy cases (Patterson et al. 1986b).

Blood residues. CDDs/CDFs were measured in the blood (lipid basis) of 10 individuals in Germany with no prior CDD exposure (Päpke et al. 1989b). OCDD was the most abundant congener present (mean 610.8 ppt; range 439–889 ppt), followed by 1,2,3,4,6,7,8-HpCDD (mean 88.2 ppt; range 30-142 ppt), HxCDD (mean 75.7 ppt; range 52–99.8 ppt), 1,2,3,7,8-PeCDD (mean 16.5 ppt; range 5.6–39 ppt), and 2,3,7,8-TCDD (mean 4 ppt; range <1.5–9.1 ppt). Mean blood levels of CDFs ranged from 24 to 46.3 ppt. Detection limits for 2,3,7,8-TCDD were 1–4 ppt (extractable lipids) which corresponds to 0.005–0.02 ppt

Table 5-12. Dioxins and Dibenzofurans in Adipose Tissue of the General U.S. Population[a]

	Concentration (pg/g, lipid basis)[b]		
Compound	Minimum	Median	Maximum
2,3,7,8-TCDD	<0.980[b]	6.54	15.1
2,3,7,8-TCDF	0.893	1.89	3.88
1,2,3,7,8-PeCDD	<2.44	10.2	24.4
1,2,3,7,8-PeCDF	<0.066[c]	0.249	1.42
2,3,4,7,8-PeCDF	<0.264[c]	9.21	29.2
1,2,3,7,8,9-HxCDD	<3.86[c]	11.5	22.0
1,2,3,7,8,9-HxCDF	<0.290[c]	0.341	1.98
1,2,3,4,7,8/1,2,3,6,7,8-HxCDD	13.3	76.1	174.00
1,2,3,4,7,8-HxCDF	<3.11[c]	7.30	17.0
1,2,3,6,7,8-HxCDF	<0.556[c]	5.03	14.3
2,3,4,6,7,8-HxCDF	<0.377[c]	0.479	2.49
1,2,3,4,6,7,8-HpCDD	20.9	11.00	230.0
1,2,3,4,6,7,8-HpCDF	<1.15[c]	17.7	32.5
1,2,3,4,7,8,9-HpCDF	0.731[c]	0.715	1.74
OCDD	152.0	838.0	1630.0
OCDF	<0.680[c]	1.19	13.2

[a] Values represent analysis of 48 composite samples collected from 865 individuals in the general population.
[b] Not detected concentrations were replaced by one-half the limit of detection.
[c] The minimum concentration is less than the minimum reported limit of detection.

HpCDD = heptachlorodibenzo-*p*-dioxin; HpCDF = heptachlorodibenzofuran; HxCDD = hexachlorodibenzo-*p*-dioxin; HxCDF = hexachlorodibenzofuran; OCDD = octachlorodibenzo-*p*-dioxin; OCDF = octachlorodibenzofuran; PeCDD = pentachlorodibenzo-*p*-dioxin; PeCDF = pentachlorodibenzofuran; TCDD = tetrachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran

Source: Orban et al. 1994

for whole blood with a lipid content of 0.5% (Päpke et al. 1989b). These authors reported that blood levels of CDDs/CDFs from persons with no known exposure to CDDs corresponded to those levels measured in adipose tissue of unexposed individuals. Needham et al. (1996) reported reference range data for CDDs and CDFs in blood of individuals who presumably had not been exposed occupationally to these compounds. The range of means in ppt (lipid basis) in blood for 7 CDDs, 11 CDFs, and 4 PCBs are summarized in Table 5-13. OCDD was the most abundant congener present (range of means 560–1,000 ppt), followed by 1,2,3,4,6,7,8-HpCDD (range of means, 80.3–230 ppt), 1,2,3,6,7,8-HxCDD (range of means, 45–85 ppt), 1,2,3,7,8-PeCDD (range of means, 6.6–32 ppt), and 2,3,7,8-TCDD (range of means, 3.2–10.1 ppt). Mean blood levels of CDFs ranged from not detected to 27 ppt. The TEQ range in mean blood levels was 13.7–41.39 ppt for all CDDs and 15.1–58.0 ppt for CDDs/CDFs.

Tepper et al. (1997) compared serum levels of CDD and CDF concentrations in 16 community residents who had no occupational exposure to these compounds. OCDD was the most abundant congener present (range, 285–1,489 ppt), followed by 1,2,3,4,6,7,8-HpCDD (range, 64.1–115 ppt), 1,2,3,6,7,8-HxCDD (range, 48.3–101 ppt), 1,2,3,7,8-PeCDD (range, 2–7.8 ppt), and 2,3,7,8-TCDD (range, 1.5–3.5 ppt). Mean blood levels of CDFs ranged from 0.8 to 31.3 ppt. The mean in TEQs was 13.5 ppt (range, 9.5–19.1) for all CDDs, 5.0 ppt (range, 3.4–8.8 ppt) for all CDFs, and 19.1 ppt (range, 12.9–25.9 ppt) for CDD/CDFs.

Most recently, Michalek et al. (1998) measured levels of 2,3,7,8-TCDD in 1,302 unexposed Air Force Vietnam-era veterans. These veterans served as controls in the 20-year epidemiologic study of Air Force veterans of Operation Ranch Hand, the unit responsible for aerial spraying of Agent Orange in Vietnam. These authors reported mean 2,3,7,8-TCDD concentrations in blood of 4.32±2.53 ppt for the control group. The 99th percentile of the distribution was less than or equal to 10.4 ppt.

Needham et al. (1991) also showed that human adipose tissue concentrations of CDDs may be correlated with blood serum levels after adjusting for total lipid content. On a lipid basis, total CDD/CDFs are higher in blood than adipose tissue. Partitioning is not identical in these tissues; 2,3,7,8-TCDD levels are almost identical in blood and adipose tissues, but OCDD levels are higher in blood. However, the presence of OCDD at levels of 5,000–10,000 pg/person when concentrations in food are generally in the low pg/g level suggests that the contribution of food to the OCDD body burden in humans requires further study (Rappe 1993).

Table 5-13. Reference Range Levels and TEQs of CDDs, CDFs, and Coplanar PCBs in Whole Blood (Lipid Basis) for the General Population

	Compound	Range of means (ppt)	Reference range (ppt)	TEQ (ppt)
CDDs	2,3,7,8-TCDD	3.2–10.1	ND–38	3.2–10.1
	1,2,3,7,8-PeCDD	6.6–32	ND–180	3.3–16
	1,2,3,4,7,8-HxCDD	6.3–13	3.1–58	0.63–1.3
	1,2,3,6,7,8-HxCDD	45–85	17–494	4.5–8.5
	1,2,3,7,8,9-HxCDD	7.1–21.9	3.5–51	0.71–2.19
	1,2,3,4,6,7,8-HpCDD	80.3–230	ND–1260	0.8–2.3
	OCDD	560–1,000	64–2,550	0.56–1
CDD Total TEQ[a]		13.7–41.4		
CDFs	2,3,7,8-TCDF	1.1–9	ND–32	0.11–0.9
	1,2,3,7,8-PeCDF	ND	ND	–
	2,3,4,7,8-PeCDF	5–27	ND–77	0.25–13.5
	1,2,3,4,7,8-HxCDF	4.5–11	1.7–28	0.45–1.1
	1,2,3,6,7,8-HxCDF	4.5–8.5	1.8–18	0.45–8.5
	1,2,3,7,8,9-HxCDF	ND	ND	–
	2,3,4,6,7,8-HxCDF	ND	ND	–
	1,2,3,4,6,7,8-HpCDF	8.7–22.8	ND–55	0.087–0.23
	1,2,3,4,7,8,9-HpCDF	ND	ND	–
	OCDF	ND	ND	–
CDF Total TEQ		1.4–16.6[a]		
PCBs	3,3',4,4'-TCB	11.7	ND–27.9	0.00585
	3,4,4',5-TCB	10.5	1.5–21.3	0
	3,3',4,4',5-PCB	135	14.6–371	13.5
	3,3',4,4',5,5'-HxCB	69	29.5–174	0.69
PCB Total TEQ		14.2[b]		

[a] TEQ values calculated by multiplying range of means values by EPA toxic equivalency value.
[b] TEQ calculated by multiplying range of means value by 0.0005, 0, 0.1, and 0.01 for 3,3',4,4'-TCB, 3,4,4',5-TCB, 3,3',4,4',5-PCB, 3,3',4,4',5,5'-HxCB, respectively.

CDDs = chlorinated dibenzo-*p*-dioxins; CDFs = chlorinated dibenzofurons; HpCDD = heptachlorodibenzo-*p*-dioxin; HpCDF = heptachlorodibenzofuran; HxCB = hexachlorobiphenyl; HxCDD = hexachlorodibenzo-*p*-dioxin; HxCDF = hexachlorodibenzofuran; ND = not detected; OCDD = octachlorodibenzo-*p*-dioxin; OCDF = octachlorodibenzofuran; PCB = polychlorinated biphenyl; PeCDD = pentachlorodibenzo-*p*-dioxin; PeCDF = pentachlorodibenzofuran; TCB = tetrachlorobiphenyl; TCDD = tetrachlorodibenzo-*p*-dioxin; TCDF = tetrachlorodibenzofuran; TEQ=Toxicity equivalency concentration.

Source: Needham et al. 1996

5.5.2 Occupational Exposure

Occupational exposure to CDDs occurs primarily through inhalation and dermal contact of fire fighters and cleanup workers involved with transformers containing PCBs and polychlorobenzenes; in workers involved in incineration operations; in workers in metal reclamation facilities, and in workers producing and handling pesticides, hexachlorophene, trichlorophenol, or other chlorinated compounds (e.g., pentachlorophenol) that may contain small impurities of 2,3,7,8-TCDD or other CDDs (Päpke et al. 1992). In addition, these authors reported that the CDD/CDF homologue profiles in whole blood of workers engaged in a variety of different chemical processes or in occupational accidents exhibited distinct CDD/CDF patterns (Päpke et al. 1992).

A dust sample collected from the ambient atmosphere of a municipal incineration plant in Europe over a 6-day period was analyzed for CDDs. The concentrations of 2,3,7,8-substituted congeners ranged from 0.9 ppb (2,3,7,8-TCDD) to 310 ppb (1,2,3,4,6,7,8-HpCDD) (Tong et al. 1989a). These results indicate that the ambient atmosphere of a municipal incinerator can be contaminated by CDDs by means of fly ash and possibly other incineration products; thus, municipal incinerator workers are at risk of exposure to CDDs (Tong et al. 1989a). Blood analysis of 10 workers at a municipal solid-waste incinerator in Germany showed elevated CDD levels of 4 workers whose work was associated with exposure to fly ash and slag (Päpke et al. 1993). In several individuals studied, the higher chlorinated CDDs/CDFs especially the 2,3,7,8-substituted HxCDD, HpCDD, and OCDD congeners showed slightly elevated levels.

Compared with background 2,3,7,8-TCDD levels (3.6 ppt), workers involved in trichlorophenol production had elevated 2,3,7,8-TCDD blood levels, with a mean concentration of 332 ppt (Päpke et al. 1992). PCP manufacturing resulted in the greatest increases for workers with respect to all congeners, with OCDD blood levels of approximately 300,000 ppt. PeCDF, HxCDF, and HpCDF levels in the blood were elevated in workers at a metals reclamation plant (Päpke et al. 1992). Workers exposed to CDD as a result of an industrial accident had mean 2,3,7,8-TCDD blood levels of 53 ppt almost 36 years after the incident (Päpke et al. 1992). In one documented case, a U.S. domestic agricultural worker was exposed to 2,3,7,8-TCDD during spraying of 2,4,5-T herbicide on pasture land and hay ground. A sample of the herbicide that he used contained 7.7 ppb 2,3,7,8-TCDD. 2,3,7,8-TCDD levels measured in the worker's adipose tissue 5 years post-exposure were 72 ppt (whole weight) or 77 ppt (lipid basis) (Tong et al. 1989b). Thirty-two years after an industrial accident in a chemical plant manufacturing trichlorophenol, the average lipid-adjusted concentration of 2,3,7,8-TCDD in the adipose tissue of exposed workers who

developed symptoms (chloracne and other illnesses) was 49 ppt (range of 11-141 ppt) (Schecter and Ryan 1988).

In a recent study by Tepper et al. (1997), serum levels of CDDs and CDFs were measured in pulp and paper mill workers in the United States. These authors reported that serum levels of CDDs and CDFs among 46 long-term workers at a pulp and paper mill were not appreciably different among three exposure groups studied (community residents, low-exposure-potential worker group, and high-exposure-potential worker group). Serum CDD TEQs were 13.5 ppt (range, 9.5–19.1 ppt), 15.9 ppt (range, 6.5–31.8 ppt), and 13.3 ppt (range, 7.5–24.9 ppt) respectively. Total TEQ for both CDDs and CDFs were similar for the three groups at 19.1 ppt, 21.2 ppt and 18.1 ppt, respectively. Serum levels of CDDs and CDFs in this study were within the range previously reported for persons with no known occupational exposure.

A series of adipose tissue samples collected from one exposed individual, as well as surgical and autopsy specimens from four control individuals, was analyzed for CDDs (TCDD, PeCDD, HxCDD, HpCDD, and OCDD) (Schecter et al. 1985a). All specimens were obtained from persons residing in urban or rural areas of upstate New York during 1983 or 1984. The worker who had been exposed to soot containing PCBs, CDFs, and small amounts of CDDS from the CDD/ CDF-contaminated Binghamton State Office Building in New York, had a total CDD concentration (whole-weight basis) of 1,015 ppt, whereas the average total CDD concentration for the controls was 765 ppt. Mean concentrations were highest for OCDD among all the CDD congener groups in both the controls (585 ppt) and the exposed person (690 ppt). 2,3,7,8-TCDD concentrations were lowest in both groups, an average of 6.3 ppt for the controls and 11.6 ppt for the exposed person. Intermediate levels were found for PeCDD (7.5–13.8 ppt), HxCDD (6.8–64.2 ppt), and HpCDD (2.6–119 ppt) in the control groups. Intermediate levels were also found in the exposed individual for PeCDD (15 ppt), HxCDD (7.3–72.6), and HpCDD (9.6–209 ppt) (Schecter et al. 1985a).

An occupational study of workers exposed to CDDs at a Missouri chemical plant from 1968 to 1972 found a mean 2,3,7,8-TCDD concentration of 390 ppt in the adipose tissue of 4 exposed workers measured 13–17 years post-exposure. The chemical plant made 2,4,5-trichlorophenol (2,4,5-TCP), which was used as a feedstock to produce butyl esters of 2,4,5-trichlorophenoxyacetic acid (2,4,5-T esters) and hexachlorophene between 1968 and 1972. The 2,3,7,8-TCDD was generated as an unintended contaminant during the production of 2,4,5-TCP. Consequently, workers involved in these processes were potentially exposed to 2,3,7,8-TCDD. The mean concentration of 2,3,7,8-TCDD found in these workers was 45 times higher than the mean of 8.7 ppt reported for 7 unexposed Missouri residents (Patterson et al. 1989a).

Another group that is occupationally exposed to CDDs includes analytical chemists involved in the synthesis of CDDs for research purposes or those involved in analysis of environmental samples contaminated with CDDs. Schecter et al. (1994b) reported that a chemist who had been involved in synthesizing 2,3,7,8-TCDD in 1956 at a university research laboratory had subsequently developed chloracne, headaches, backaches, and severe leg pains when he walked. During 1990–91, approximately 35 years after the chemist's initial exposure, blood CDDs analysis was performed to determine whether residual CDD levels still remained. Schecter et al. (1994b) reported that blood TCDD levels (on a blood-lipid basis) were 20 ppt as compared to 3–5 ppt measured in a control population of 100 individuals. Analytical laboratory personnel who are involved in analyzing CDD-contaminated samples also may be exposed to higher levels of CDD contamination than the general population (Hesso et al. 1992; Oliver 1975).

In a study conducted by NIOSH, serum levels of 2,3,7,8-TCDD were measured in 27 U.S. chemical workers previously exposed to CDDs-contaminated products (Fingerhut et al. 1989). The workers were employed at two U.S. facilities that produced 2,4,5-TCP and 2,4,5-T between 1951 and 1972. Serum levels of 2,3,7,8-TCDD were also measured in 19 unexposed controls. A mean serum 2,3,7,8-TCDD level of 208.2 ppt found in the exposed workers exceeded (by more than 25 times) the mean background level of 8.2 ppt found in the controls who did not produce these chemicals (Fingerhut et al. 1989).

Workers who are involved with incineration operations may be exposed to levels of CDDs that are higher than background levels to which the general population is exposed. Schecter et al. (1991b) measured CDD and CDF blood levels on a lipid basis in pooled blood samples from a group of 56 New York City incinerator workers and 14 controls. The levels of 11 of the 18 CDD/CDF congeners measured were increased in the incinerator workers as compared to the controls. CDD levels in incinerator workers were 48, 17, 27, 30, and 31% higher for 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, 1,2,3,4,6,7,8-HpCDD, and OCDD, respectively. Only 2,3,7,8-TCDD and 1,2,3,4,7,8-HxCDD were lower in incinerator workers' blood than in controls (5 and 15% lower, respectively). Overall, the total CDD/CDF level in workers' blood was, 1,007.2 ppt (lipid basis) as compared to 747.3 ppt for the controls (Schecter et al. 1991b). In the past, workers involved in the production or use of hexachlorophene, trichlorophenol, 2,4,5-T, and other compounds that are no longer used were also exposed to 2,3,7,8-TCDD. Workers in pulp and paper mills also have the potential for 2,3,7,8-TCDD exposure because of the occurrence of 2,3,7,8-TCDD in bleached kraft paper-making processes (Clement et al. 1989; Kuehl et al. 1987a; Tepper et al. 1997); although exposure to this source has probably declined since

1990 with the implementation of bleach plant modifications at many pulp and paper mills (NCASI 1993). Workers in the sawmill industry who handle treated lumber may be exposed to chlorophenols, particularly PCP; consequently, they may be exposed to higher levels of the more highly chlorinated CDDs (Kalliokoski and Kauppinen 1990). Workers employed at sites of improper chemical waste disposal (trichlorophenol, hexachlorophene, 2,4,5-T) have a greater potential for exposure to 2,3,7,8-TCDD via inhalation or via oral or dermal contact than the general population.

A current estimate of the number of workers in the United States that are potentially exposed to CDDs is not available. At-risk worker populations include incinerator personnel, those involved in production or use of chlorinated compounds containing CDD contamination (e.g., hexachlorophene, PCP, 2,4,5-trichlorophenol, and 2,4-D), analytical research chemists, and workers at chemical waste disposal sites, electrical utility workers, and firefighters.

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in Section 2.6, 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, and 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; they put things in their mouths; they may ingest inappropriate things such as dirt or paint chips; they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

Children are primarily exposed to CDDs in the same manner as adults in the general population (i.e., via consumption of foods contaminated with small amounts of CDDs, particularly meat, milk and other dairy products, and fish). Children that are at additional risk of exposure primarily through dietary habits, include: infants and young children who are breast-fed; children of recreational and subsistence fishers, who typically consume larger amounts of locally caught fish and shellfish than the general population;

children of subsistence hunters, particularly those in the high latitudes, who typically consume large amounts of locally caught game especially marine mammals; and children of subsistence farmers living in areas contaminated with CDDs (either by waste incinerators or the use of CDD- contaminated sewage on their land) who exclusively consume their own farm-raised beef and dairy products (see Section 5.7).

The human fetus is exposed to CDDs/CDFs through transplacental transfer from the mother. Schecter et al. (1990a) reported 2,3,7,8-TCDD concentrations in liver tissue of three still-born infants ranging from 0.03 to 0.18 ppt (whole weight basis) and 1.3 to 4.3 ppt (lipid weight basis). Schecter et al. (1990a) also reported CDD/CDF concentrations in liver tissue of three stillborn infants ranging from 2.1 to 4.92 ppt (whole weight basis) and 98 to 104 ppt (lipid weight basis). The TEQ for CDD/CDFs combined ranged from 0.14 to 0.49 ppt (whole weight basis) and 6.4 to 12 ppt (lipid weight basis). In a more recent study, Schecter et al. (1996e) reported TEQs for CDDs/CDFs in placental material ranging from 8.4 to 17.6 ppt (lipid basis). In a pooled sample of fetal tissue (8–14 weeks), the TEQ was 5.3 ppt (lipid basis). Concentrations of 2,3,7,8-TCDD in adipose tissue and liver were also reported by Kreutzer et al. (1997) for stillborns at levels between 0.2 and 0.8 ppt and 0.3 to 0.7 ppt, respectively. Kreutzer et al. (1997) developed a pharmacokinetic model for 2,3,7,8-TCDD that predicted a decrease in body burdens during the first year for non-breast-fed infants and this was supported by empirical data (see Section 2.3.4.4, Transfer of CDDs Through the Placenta and Breast Milk).

In addition to transplacental transfer, CDDs and CDFs have been found in human milk (Fürst et al. 1992; Ryan et al. 1993a; Schecter and Gasiewicz 1987b; Schecter et al. 1986a, 1989d, 1989e, 1989g, 1991a); human milk is thus a potential source of CDDs for nursing infants and children (see Section 5.5). In Binghamton, New York, and Los Angeles, California, human breast milk was found to contain almost identical levels of detectable CDDs on a lipid basis probably because food consumption and sources are similar across the United States (Schecter et al. 1989e). Mean values of two pooled samples (n=42) from both cities showed that OCDD was the most abundant congener present (233 ppt), followed in decreasing order by total HxCDD (42.65 ppt), 1,2,3,4,6,7,8-HpCDD (42 ppt), 1,2,3,6,7,8-HxCDD (30.5 ppt), 1,2,3,7,8-PeCDD (6.7 ppt), 1,2,3,7,8,9-HxCDD (6.2 ppt), 1,2,3,4,7,8-HxCDD (4.95 ppt), and 2,3,7,8-TCDD (3.3 ppt). The total CDDs value was reported as 327 ppt. The TEQ for CDDs/CDFs, but not PCBs in breast milk in the United States was 17 ppt (Schecter et al. 1989e). Between 1986 and 1987, concentrations of CDDs found in breast milk sampled from Canadian women ranged from 2.2 ng/kg (ppt) (lipid basis) for TCDDs to 173 ppt for OCDD. In addition, the combined CDD/CDF mean TEQ of 15.6 ppt (lipid basis) declined from a TEQ of 24.7 ppt measured in 1981–1982 (Ryan et al. 1993a).

CDD/CDF concentrations also have been measured in breast milk in several foreign studies. Concentrations of CDDs/CDFs were also measured in preserved breast milk from 505 persons (both primiparas and multiparas) in Japan from 1978 to 1984 (Ogaki et al. 1987). OCDD was a major component in primiparas' milk (789 ppt) and multiparas' milk (518 ppt) (Ogaki et al. 1987). For the primiparas, average concentrations found for HpCDD, HxCDD, PeCDD, total TCDD, and 2,3,7,8-TCDD were 150, 76, 15, 37, and 13 ppt, respectively. The average CDD concentrations found in the milk of multiparas were generally lower than the average concentrations found in the milk of primiparas. For the multiparas, average concentrations for HpCDD, HxCDD, PeCDD, and TCDD (not 2,3,7,8) were 75, 56, 11, and 19 ppt, respectively (Ogaki et al. 1987). A similar study in Germany between 1984 and 1991 found mothers nursing their second child had 20% less CDD TEQs in their milk than did primigravidae (Fürst et al. 1992). CDD concentrations in human milk can be directly correlated with the age of the mother and the amount of animal (but not vegetable) fat and protein consumed, suggesting that meat, milk and other dairy products, and fish are the major sources of CDD intake (Pluim et al. 1993a). The fact the CDD concentrations in milk fat were significantly related to age is in agreement with the results of Stanley et al. (1986) and Orban et al. (1994) who reported a strong correlation between age group and CDD levels in adipose tissue in the general U.S. population. The positive correlation can be expected because of the long half-life of CDDs in humans (7–11.3 years) (Pirke et al. 1989; Wolfe et al. 1994).

Estimated daily intakes of CDD/CDF TEQs by nursing infants in the United States have been reported by Schecter and Gasiewicz (1987a). The daily intake by nursing infants in the United States was estimated to be 83.1 pg TEQs/kg body weight/day. To determine this daily intake, various assumptions were made regarding infant body weight (10 kg), duration of nursing, average amount of milk consumed, and gastrointestinal absorption. It was also assumed that breast milk was the only source of CDDs while the infant was nursing during the first year of life. From results of earlier studies that determined the concentrations of CDDs/CDFs in human breast milk in the United States (Schecter et al. 1989e) and in cow’s milk and soybean-derived infant formula sold in the United States (Schecter el al. 1989c) (see Section 5.4.4), Schecter et al. (1994e) estimated slightly lower intakes of 35–53 pg TEQ/kg of body weight/day for infants (7.3 kg) that were breast-fed within the first year of life as compared to 0.07–0.16 pg TEQ/kg of body weight for infants who were fed soy formula.

Exposure of infants and young children to CDDs may be very high because of their relatively high consumption of milk, including breast milk (ECETOC 1992). Schecter et al. (1994e) evaluated the intake of CDDs/CDFs from human breast milk and estimated that high levels reported for breast milk in the

United States (. 17 ppt TEQ on a lipid basis) contribute 35–53 pg TEQ/kg of body weight per day to the nursing infant in its first year of life (Schecter et al. 1989e). The CDD concentrations in cow's milk and soy-based formula were much lower than in breast milk (327 ppt) (Schecter et al. 1991a). The following concentrations for CDDs (on a lipid basis) were reported: cow's milk (25.1 ppt), 2% cow's milk (32.3 ppt), Similact™ infant formula (39 ppt), Isomil™ infant formula (23.3 ppt), and Prosobee™ infant formula (42.7 ppt) (Schecter et al. 1989c). The TEQ values for cow's milk and soy-based infant formula were also much lower than for human breast milk (. 17 ppt). The corresponding TEQ values for CDDs/CDFs (on a lipid basis) were reported: cow's milk (2.1 ppt), 2% lowfat cow's milk (0.79 ppt), Similac™ infant formula (0.08 ppt), Isomil™ infant formula (0.05 ppt), and Prosobee™ infant formula (0.127 ppt) (Schecter et al. 1989c). Schecter and Gasiewicz (1987a, 1987b) calculated TEQ values for CDDs/CDFs in human milk in two populations in Vietnam and in the general population in the United States. These authors reported mean values during the 1980s of 1.04 pg TEQ/g (whole milk basis) for the United States (maximum 4.72 pg TEQ/g), a mean of 1.11 pg TEQ/g for South Vietnamese (maximum value 4.38 pg TEQ/g) exposed to Agent Orange sprayed between 1962 and 1970, and a mean of 0.065 pg TEQ/g (maximum value 0.18 pg TEQ/g) for a North Vietnamese population that was not exposed to Agent Orange. These authors concluded that some infants in the United States (whose mothers had CDD milk concentrations in the upper range of measured values) were being exposed to mean concentrations comparable to levels observed in the South Vietnamese population exposed to Agent Orange (Schecter and Gasiewicz 1987a, 1987b).

The highest exposure to CDD-contaminated breast milk reported was associated with the widespread use of Agent Orange as a defoliant during the Vietnam War. Human milk specimens from Ho Chi Minh City and Song Be Province in South Vietnam had lower 2,3,7,8-TCDD values in the late 1980s (7.1 and 17 ppt lipid basis) (TEQ values of 18.5 and 31.7 ppt), respectively, than they did in the 1970s when Agent Orange spraying occurred (Schecter et al. 1989e). A 1970 mean value for 2,3,7,8-TCDD in human milk in South Vietnam was reported to be 484.9 ppt (range, not detectable to 1,450 ppt) (Baughman and Meselson 1973; Schecter et al. 1986a). These values serve as reference values for the highest levels of 2,3,7,8-TCDD documented in human milk (Schecter et al. 1989e). Estimated daily intakes of TEQs by nursing infants from Vietnam have been reported (Schecter and Gasiewicz 1987a). The estimated daily intake by nursing infants in southern Vietnam in 1970 was 908 pg TEQs/kg body weight/day, whereas the daily intakes in southern and northern Vietnam in 1984 were 88.7 and 5.1 pg TEQs/kg body weight/day, respectively. Analysis of 9 milk samples from individuals living in northern Vietnam showed no detectable concentrations of 2,3,7,8-TCDD (detection limit 2 ppt) (Schecter and Gasiewicz 1987a). To determine

these daily intakes, various assumptions were made regarding infant weight, duration of nursing, average amount of milk consumed, and gastrointestinal absorption. It was also assumed that breast milk was the only lifetime source of exposure to CDDs during the first year of life. In another study, Tarkowski and Yrjanheikki (1989) evaluated the health risks associated with human milk. These authors concluded that levels of CDD/CDFs in breast milk did not present a health risk to infants and children and that there was no justification for limiting breast-feeding. However, these authors believed there was a need for primary prevention of CDD/CDF exposure in humans. Because of the relatively short period of intake and the accepted benefits of breast-feeding, the World Health Organization did not recommend limitations on breast-feeding at the levels of background exposures to CDDs and CDFs (WHO 1991). More recently, Pohl and Hibbs (1996) reviewed recent studies indicative of a possible link between development of subtle health effects in children and their exposure to CDDs and CDFs from maternal milk. It is the ATSDR position that for background exposures, the benefits of breast feeding outweigh any potential risk associated with exposure. For higher CDDs levels in breast milk, the safety of breast-feeding may be of concern in some cases.

Two recent studies have looked at ways to reduce CDD exposure in breast-fed infants. Koppe (1995) reported that exposure before and after birth to CDDs and PCBs has given rise to subtle abnormalities (disturbed cognitive development and delayed motor development) in approximately 10% of newborns in the Netherlands. This author examined possibilities of reducing this exposure by influencing the diet of the lactating mother. Mobilization of fatty acids from adipose tissue will cause release of stored CDDs which will then be secreted in breast milk. Two maternal diets were tested for their ability to reduce concentrations of CDDs in human milk. One diet was a low fat/high carbohydrate/low CDD diet, while the second was a high fat/low carbohydrate/low CDD diet. Despite significant changes in fatty acid profiles of the milk, no significant changes in CDD concentrations in breast milk were observed. The author concluded that short-term dietary measures will not reduce CDDs in breast milk. A lowering of CDD intake must occur years before the woman becomes pregnant. An important food source for the women is cow's milk and other dairy products and these are responsible for about half of the daily exposure CDDs and PCBs in women in the Netherlands, so levels of the compounds in dairy foods must be lowered. In addition, the author believes that a lowering of CDD concentrations in fish is also necessary. Based on the results of his dietary study, Koppe (1995) reported that daily dietary intake of CDDs during lactation represents only 14% of the daily secretion of CDD in breast milk, while 86% was derived from CDDs stored in adipose tissue. Thus, reducing dietary intake of CDDs during lactation would only reduce CDDs in milk by 14%. Schlaud et al. (1995) also reported that to reduce organochlorine residue levels including

CDDs in human breast milk in the short-term, nursing mothers should be advised not to try to reduce their body weight until after lactation. These authors reported statistically significant positive associations between breast milk contamination and average dietary fat intake per week (P=0.001) and proximity of residence to hazardous waste sites (P<0.05) for CDDs. These authors believe that public promotion of a lower dietary fat intake may reduce the lifetime accumulation of CDDs in human fatty tissues and in the long-term, resulting in lower concentrations in breast milk as well.

In addition to exposure to CDDs through consumption of breast milk, cow's milk, and soy-based infant formula, older children can be exposed through dietary practices similar to those of adults in the general population (see Section 5.4.4). One study has looked at the exposure that might occur in a 6-year-old child who consumes "fast foods." In 1995, Schecter and Li (1997) conducted a congener-specific analysis of CDDs, CDFs, and dioxin-like PCBs in U.S. fast foods. These authors reported a CDD/CDF TEQ value, depending on the treatment of not detected congeners, from 0.03–0.28 pg/g wet weight for one McDonald's Big Mac, 0.03–0.29 pg/g for one Pizza Hut personal pan pizza supreme with all toppings, 0.01–0.49 pg/g for one Kentucky Fried Chicken 3-piece original recipe meal, and 0.3–0.31 pg/g for one Haagen-Daz chocolate-chocolate chip ice cream. The daily intake from one serving of each of the fast foods tested, assuming a 20-kg child (6 years old), ranged between 0.15 and 5.05 pg TEQ/kg body weight. These authors calculated that, on average, a child (6 years old) consumes 3 times more TEQs on a per kg/body weight basis than an adult eating any one of the fast foods tested.

As a result of the transfer of CDDs through the placenta to the fetus, by breast milk to infants and young children, and by lifelong dietary intakes from the consumption of meat, milk and dairy products, and fish, CDDs are found to be widespread in the adipose tissue of members of the general population (Orban et al. 1994). Human adipose samples from the recent 1987 NHATS Study provide a representative sample of CDD body burden in the general U.S. population (see Section 5.5.1). The average concentration of 2,3,7,8-TCDD in the U.S. population was estimated to be 5.38 pg/g (±6%). The 1987 survey data clearly show, however, that nearly all of the CDD/CDF congeners in adipose tissue increased with the age of the donor (i.e., the highest concentrations occur in the 45+ age group and the lowest concentrations occur in children in the 0–14 age group). The average concentration of 2,3,7,8-TCDD in the 1987 survey increased from 1.98 pg/g in the 0–14-year-old group, to 4.37 pg/g in the 15–44-year-old group, to 9.4 pg/g in the 45+-year-old group.

Children may be exposed to CDDs through a variety of lifestyle practices of their parents or of their own. For example, CDD/CDF concentrations have been reported in cigarette smoke (Lofroth and Zebuhr 1992; Muto and Takizawa 1989) (see Section 5.4.4). Young children and infants may be exposure to CDDs indirectly by inhalation of room air contaminated from cigarette smoking of their parents. In addition, older children and teenagers, may be directly exposed if they become smokers themselves. Malisch (1994) reported that some colored candle wax produced with certain dye pigments contained CDDs/CDFs. By burning these candles, CDDs could be released into room air and be an additional source of inhalation exposure for children.

Children may also be exposed to CDDs by dermal contact with some new, unwashed clothing, particularly those manufactured in some developing countries or from fabric shipped from developing countries where pentachlorophenol (PCP) is used for preserving cotton fabrics during sea transport (Horstmann and McLachlan 1994). Exposures can be reduced by washing new clothes prior to wearing.

Children could potentially be exposed to CDDs at home from a variety of incineration sources. For example, if their parents routinely burn domestic garbage containing scrap wood treated with PCP (Chiu et al. 1983) or untreated wood (Clement et al. 1985), old pesticide containers that may have contained 2,4,5 T or 2,4-D or Silvex (Arthur and Frea 1989), or polyvinylchloride (PVC) pipes or other plastics items (Lustenhouwer et al. 1980), or extensively use a wood stove (Clement et al. 1985), children may be exposed to higher levels of CDDs in outdoor and/or indoor air. Time spent in a garage where cars or trucks are being repaired and the engines are running, exposes children and teenagers to exhaust products and engine soot that may also contain CDDs (Bingham et al. 1989; Cirnies-Ross et al. 1996).

Although there are many studies on the effects of CDDs on adults that receive occupational exposures (Fingerhut et al. 1989; Hesso et al. 1992; Patterson et al. 1989a; Schecter et al. 1985a, 1994b; Tepper et al. 1997), no information was located on the potential for workers in the United States to bring CDDs home on their clothing or shoes, thus contaminating other family members, including children. It is conceivable, however, that because CDDs are present in a variety of diverse occupational settings (see Section 5.5.2 Occupational Exposures), that poor occupational hygiene could result in CDDs being brought home and contaminating domestic dwellings.

Children in populations with potentially high exposure living in the vicinity of former or current production sites where CDDs are released as by-products, (e.g., incinerators, other waste disposal facilities, and

hazardous waste sites) may be exposed to CDDs by several pathways (see Section 5.7). Children may be exposed to CDDs in CDD-contaminated soils. Dermal absorption from contaminated soil, however, is likely to be inefficient (Poiger and Schlatter 1980; Shu et al. 1988; Weber et al. 1991c). Young children are potentially exposed to CDDs because of their tendency, through hand-to-mouth activity, to ingest soils (pica) that may be contaminated with CDDs (see Section 5.7 for further details) (Fries and Paustenbach 1990; Kimbrough et al. 1984; Paustenbach et al. 1992; Pohl et al. 1995). La Goy (1987) estimated the following average soil ingestion rates for children: age 0–1 years old, 50 mg/day (maximum 250 mg/day); 1–6 years old, 100 mg/day (maximum 500 mg/day); 6–11 years old, 50 mg/day (maximum 250 mg/day); and over 11 years old, 50 mg/day (maximum 100 mg/day). If children ingest between 50 and 100 mg of soil per day (LaGoy 1987) and the soil they ingest contains 1 pg/g (1 ppt) of CDDs, a child may be exposed to 0.05-0.1 pg of CDDs per day by this pathway alone (see Section 5.7).

Children in high risk populations include children of recreational or subsistence fishers, children of subsistence hunters particularly those that consume tissues of marine mammals, and children of subsistence farmers that consume meat, milk and/or dairy products from their own farm raised animals (see Section 5.7 for further details). For example, Native American and other subsistence fishing communities may be at greater health risks from CDDs in fish and children in these population often consume larger amounts of fish than adult members of the general population (CRITFC 1994; Mott 1995). Children of recreational and subsistence fishers who routinely consume locally caught fish from CDD-contaminated waterbodies can be exposed to higher CDD concentrations than children who consume similar or larger amounts of commercially marketed fish from a variety of sources (Ebert et al. 1996; EPA 1995c; Mott 1995). The exposure to CDDs will also be highest among children who regularly eat fish as compared to those who only occasionally or never eat fish. Several recent studies have documented the higher fish consumption rates among subsistence fishers some of which are Native American populations (CRITFC 1994; Nobmann et al. 1992; Wolfe and Walker 1987). A study of fish consumption patterns among the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River Basin in Washington and Oregon (CRITFC 1994) found that the consumption rate for these Native American children (5 years and younger) from these four tribes was 19.6 g/day (a consumption rate over 3 times higher than that for adults in the general population (6.5 g/day).

This increased exposure has been demonstrated by serum CDD levels, which are found to be several times higher in people who regularly eat fish as compared to those who occasionally or never eat fish (Anderson et al. 1998; Svensson et al. 1991) (see Sections 5.5 and 5.9). In addition, this same situation also applied

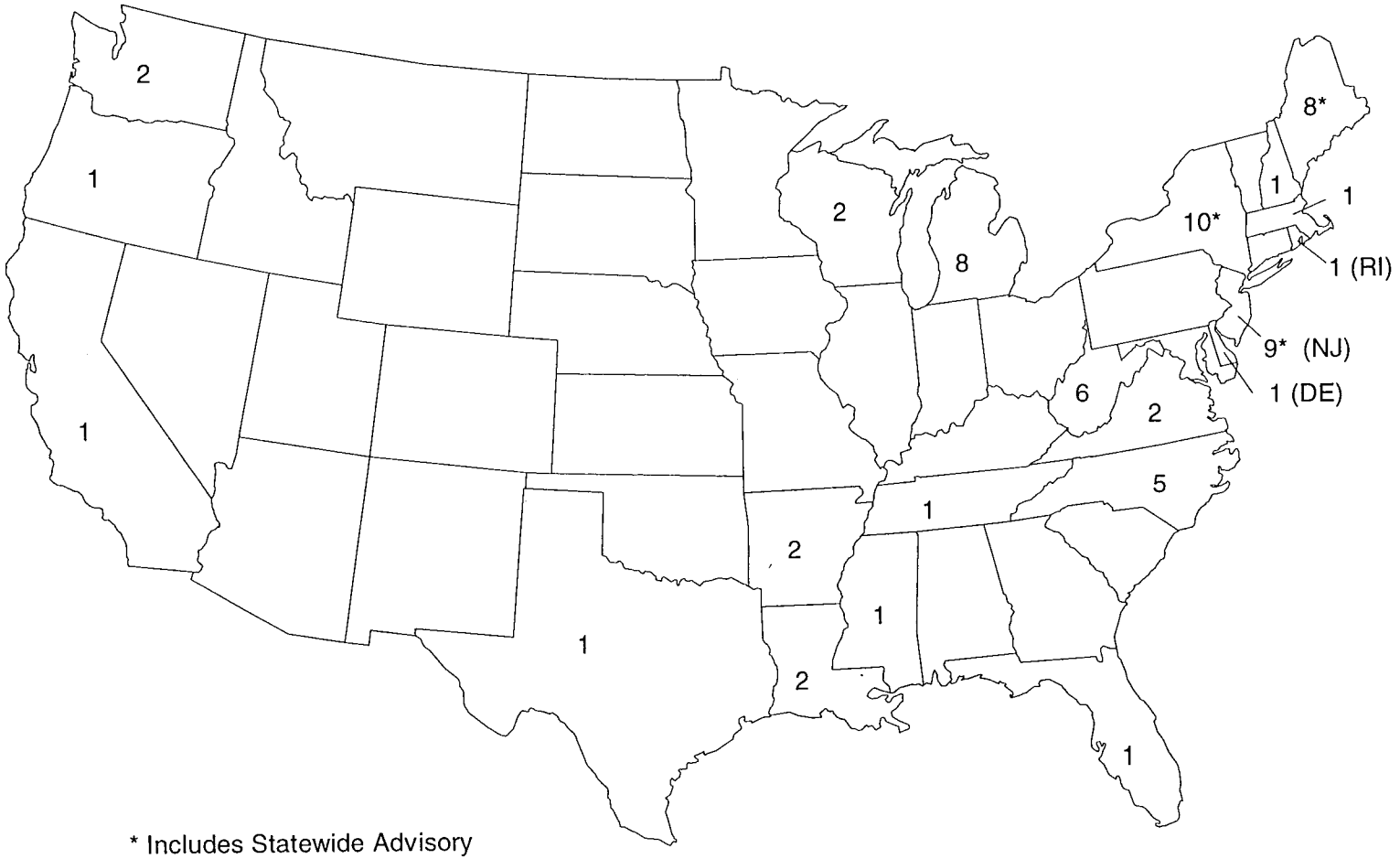
for consumption of wildlife, specifically marine mammals (Ayotte et al. 1997; Dewailly et al. 1992). Similar dietary situations exist for children of subsistence hunters that tend to consume tissues of marine mammals and children of subsistence farmers that consume beef, milk and other dairy products from their own farm raised animals. In the case of subsistence fishers, subsistence hunters, and subsistence farmers, all three populations share one problem, that the source of their fish, meat, and/or milk and other dairy products, is typically restricted to a localized area, and if these food sources are contaminated with CDDs, adults and children in these populations will be exposed to higher levels of CDDs than members of the general population (see Section 5.7 for additional details on these populations at risk).

In order to reduce exposure from consumption of CDD-contaminated fish and wildlife, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish, shellfish, and wildlife species from certain waterbodies where CDD concentrations in tissues of these species exceed the human health level of concern (EPA 1995c) (see Section 5.7 for additional information). Recreational and subsistence fishers typically consume larger quantities of fish and shellfish than the general population and frequently fish the same waterbodies routinely. Because of this, children living in these populations are at greater risk of exposure to CDDs and other chemical contaminants if the waters they fish are contaminated. Currently, 66 advisories have been issued by 21 states restricting the consumption of CDD-contaminated fish and shellfish (EPA 1998b) and one state Arkansas also has issued a consumption advisory for wood ducks, a species of migratory waterfowl. Three states (New Jersey, New York and Maine) also have statewide advisories for CDDs in their marine waters (EPA 1998a). The number of waterbodies under advisory for CDD in each state is shown in Figure 5-8.

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

In addition to individuals who are occupationally exposed to CDDs (Section 5.5), there are several groups within the general population with potentially high exposures (higher-than-background-normal levels) to CDDs. Historically, populations that have been exposed to higher-than-normal background levels of CDDs in the air, water, soil, and/or food have included those who were exposed to 2,3,7,8-TCDD as a result of industrial accidents (e.g., Nitro, West Virginia; and Seveso, Italy) and those exposed through environmental contamination (e.g., Times Beach, MO; Binghamton, NY; Love Canal, NY; Newark, NJ; and Vietnam) (Kahn et al. 1988; Schecter 1985; Schecter and Tiernan 1985; Schecter et al. 1987a, 1989a; Umbriet et al. 1986a, 1986b; Zook and Rappe 1994).

Figure 5-8. National Listing of Fish and Wildlife Consumption Advisories for Dioxins



* Includes Statewide Advisory

Source: EPA 1998b

Currently, individuals living in proximity to sites where CDDs are produced as chemical by-products or sites where CDD-contaminated chemicals are disposed, individuals living near municipal and industrial incinerators, and individuals living near one of the 110 NPL hazardous waste sites where CDDs have been detected in some environmental media (HazDat 1998) are at risk of receiving potentially higher-than-normal background levels of exposure. Other populations at risk of exposure primarily through dietary habits, include recreational and subsistence fishers who typically consume larger amounts of locally caught fish and shellfish than the general population, subsistence hunters, particularly those in the high latitudes, who typically consume large amounts of locally caught game including marine mammals; and subsistence farmers and their families living in areas contaminated with CDDs who exclusively consume their own farm-raised beef and dairy products.

Individuals exposed through industrial accidents or environmental contamination. Very extensive residential contamination by 2,3,7,8-TCDD occurred in Seveso, Italy, when a 2,4,5-TCP reactor exploded in 1976 (Mocarelli et al. 1991). The contaminated area was divided into three zones based on the concentration of 2,3,7,8-TCDD in the soil. Families in zone A, the most heavily contaminated area based on soil 2,3,7,8-TCDD levels, were evacuated within 20 days of the explosion and measures were taken to minimize exposure of residents in nearby zones. A recent analysis of 19 blood samples from residents of zone A, which were collected and stored shortly after the accident, showed serum lipid levels of 2,3,7,8-TCDD that ranged from 828 to 56,000 ppt. These serum lipid levels are among the highest ever reported for humans (Mocarelli et al. 1991).

In a study conducted in Missouri, 2,3,7,8-TCDD was measured in the adipose tissue of 39 volunteers with a history of residential, recreational, or occupational exposure (14 years post-exposure) and in 57 persons in a control group (Patterson et al. 1986a). Based on questionnaire responses, the eligible exposed group for this study consisted of people who were exposed either to areas with 2,3,7,8-TCDD concentrations in soil between 20 and 100 ppb for 2 or more years or to 2,3,7,8-TCDD concentrations >100 ppb for at least 6 months. Persons who met these criteria were classified as having one of three types of exposure: residential (either living in close proximity to areas with 2,3,7,8-TCDD-contaminated soil or having evidence of contamination inside the home), recreational (riding or caring for horses in 2,3,7,8-TCDD-contaminated stable arenas at least one time per week), or occupational (working either in a hexachlorophene production facility or at truck terminals where the grounds had been sprayed with 2,3,7,8-TCDD-contaminated waste oil). All study participants had detectable levels of 2,3,7,8-TCDD in their adipose tissue, but the group with known previous exposures had significantly higher levels than

controls. Nineteen (49%) of the 39 exposed persons had levels higher than the highest 2,3,7,8-TCDD concentration (20.2 ppt) detected in the 57 controls. Six (15%) of the 39 exposed persons had 2,3,7,8-TCDD concentrations >100 ppt. Five of the 6 values >100 ppt were from persons exposed to 2,3,7,8-TCDD during the production of hexachlorophene. The other high value (577 ppt) was found in a man exposed to 2,3,7,8-TCDD while horseback riding in a contaminated arena. 2,3,7,8-TCDD concentrations measured in the occupational group (average 136.2 ppt; range 3.5–750 ppt) were, in general, higher than those in the residential group (average 21.1 ppt; range 2.8–59.1 ppt), the recreational group (average 90.8 ppt; range 5.0–577 ppt), and the control group (average 7.4 ppt; range 1.4–20.2 ppt) (Patterson et al. 1986a).

2,3,7,8-TCDD has been detected at concentrations ranging from 20 to 173 ppt in adipose tissue from 3 Vietnam veterans reported to have been heavily exposed to Agent Orange (Gross et al. 1984). Except for these few men, however, 2,3,7,8-TCDD concentrations in American Vietnam and non-Vietnam veterans were nearly identical with mean serum levels of approximately 4 ppt (CDC 1988). Concentrations of 2,3,7,8-TCDD in the controls (those who never served in Vietnam) ranged from not detected (4 ppt) to 20 ppt. The veterans had served in Vietnam in 1967 and 1968 in areas where Agent Orange had been heavily used (CDC 1988). In another study, 2,3,7,8-TCDD was detected in adipose tissue of 14 Vietnam veterans and 3 control patients at levels ranging from not detected (2–13 ppt) to 15 ppt. No significant differences in the tissue levels of Vietnam veterans and the controls were found in this study (Weerasinghe et al. 1986). Air Force personnel associated with Operation Ranch Hand (spraying of Agent Orange) in Vietnam from 1962 to 1971 had serum CDD levels up to 10 ppt (521 persons). A correlation was found between CDD concentrations and increased body fat (USAF 1991). The median half-life of 2,3,7,8-TCDD in 36 veterans was estimated to be 7.1 years (Pirke et al. 1989). In 1987, many of the exposed Air Force personnel had serum CDD concentrations >50 ppt and several had concentrations exceeding 300 ppt (CDC 1987). Wolfe et al. (1994) reported a half-life value of 11.3 years for Air Force personnel involved in Operation Ranch Hand. Using individuals from two rice oil poisoning episodes (Yusho [Japan] and Yu-Cheng [Taiwan]), Ryan et al. (1993a) have shown that the elimination of related CDFs is not constant, but variable, with faster clearance at higher doses followed by a slowing down in the rate of loss as body burden decreases. By analogy, the same may be true for CDDs. It is also likely that individual congeners or those with the same degree of chlorination are excreted from the body at rates that differ from those estimated for 2,3,7,8-TCDD. Because the rate of clearance is not constant, uncertainty in determining the half-life measurement may result, especially for estimates of the changing body burden of total CDDs measured as TEQs.

Individuals living in proximity to production or disposal sites. Individuals living in the vicinity of former or current production sites where CDDs are released as by-products, (such as incinerators, coal-fired electric generating facilities, other waste disposal facilities, and hazardous waste sites) may be exposed to CDDs from several exposure pathways. CDDs have been detected in soil at 94 of the 126 sites where they have been detected in some environmental media (HazDat 1998).

Children and adults may receive higher CDD exposures from dermal contact if they play or work with CDD-contaminated soils. Several studies have examined the bioavailability of 2,3,4,7-TCDD for uptake by dermal exposure. In a study using *in vitro* human skin tissue, 2,3,7,8-TCDD did not readily penetrate into the human skin and the vehicle of exposure played an important role in the dermal penetration (Weber et al. 1991c). These authors reported that when the exposure vehicle was acetone, the maximum 2,3,7,8-TCDD penetration into the *in vitro* human stratum corneum (30–45% of the dose) was reached within 100 minutes, with a tendency to decrease after 1,000 minutes. Using mineral oil as the exposure vehicle, absorption of 2,3,7,8-TCDD leveled off at 10% of the dose, and it took more than 300 minutes to reach the maximum. The data suggest that the rates of absorption of 2,3,7,8-TCDD into *in vitro* human skin are moderate (worst-case scenario) to low when the 2,3,7,8-TCDD is applied in acetone; when applied in mineral oil, the adsorption rate was further reduced. Shu et al. (1988) reported that in rats, dermal absorption of 2,3,7,8-TCDD in a soil vehicle was only 1% of the administered dose. Similarly, Poiger and Schlatter (1980) reported that in rats, dermal absorption of 2,3,7,8-TCDD was almost eliminated when soil or activated carbon was used as vehicles. These data support the original Kimbrough et al. (1984) risk assessment of a contaminated site in which the authors estimated that the additional lifetime uptake of TCDD from soil above background uptake will consist of 95% from soil ingestion, 3% from dermal exposure to soil (assuming 1% dermal absorption), and 2% from inhalation of soil particles.

Children and adults also may receive potentially higher oral exposures from ingestion of CDD-contaminated soils from their unwashed hands while playing or working in CDD-contaminated areas (Fries and Paustenbach 1990; Kimbrough et al. 1984; Paustenbach et al. 1992; Pohl et al. 1995). Bioavailability is an integral factor in the estimation of the internal dose (or dose at the target tissue) of the chemical. Like dermal absorption, gastrointestinal absorption of 2,3,7,8-TCDD and related compounds is variable, incomplete, and congener- and vehicle-specific (see Section 2.3.1). More lipid soluble congeners, such as 2,3,7,8-TCDF, are almost completely absorbed, while the extremely insoluble OCDD is poorly absorbed. However, laboratory data suggest that there are no major interspecific differences in the gastrointestinal absorption of CDDs and CDFs. Results from animal studies indicate that bioavailability of 2,3,7,8-TCDD

from soil varies between sites because CDDs bind tightly to soil, and increasingly so with the passage of time and clay content of the soil (Gough 1991; Umbreit et al. 1986a;1986b). Therefore, 2,3,7,8-TCDD soil concentrations alone may not be indicative of the potential for human health hazard from contaminated soils, and site-specific evaluation may be essential. In their risk assessments, Kimbrough et al. (1984) assumed 30% bioavailability from ingestion of soil, but they point out that animal studies with contaminated Missouri soil indicated absorption as high as 30 to 50% (McConnell et al. 1984). Pohl et al. (1995) assumed 40% bioavailability of 2,3,7,8-TCDD from soil. In contrast, Paustenbach et al. (1986) assumed only 10–30% bioavailability. However, unless toxicokinetic studies that use soil samples from the specific site are available, it is difficult to speculate on how much 2,3,7,8-TCDD as well as other CDDs will be bioavailable. ATSDR's policy on CDDs contaminated soils is in Appendix B of this profile.

Individuals may also receive higher doses from routine consumption of CDD-contaminated fish from local waters receiving runoff or leachates from the waste site (Paustenbach et al. 1992). CDDs have been detected in fish collected at 12 of the 126 NPL sites where they have been detected in some environmental media (HazDat 1998).

Lastly, individuals living near incinerator or hazardous waste sites may inhale vapors or particulates contaminated with CDDs from ambient outdoor air. This, however, would be a relatively minor exposure pathway as only about 50% of all particles are of inhalable size (<10μm) (Fries and Paustenbach 1990; Paustenbach et al. 1992). These authors concluded that since uptake of 2,3,7,8-TCDD from foods will be approximately 500–1,000-fold greater than that due to inhalation, that inhalation exposure was a relatively insignificant exposure pathway even for individuals living in proximity to an incinerator.

Recreational and subsistence fishers. In general, concentrations of CDDs in sport fish and shellfish from CDD-contaminated waters can be at least an order of magnitude higher than in commercial fish and shellfish purchased in a supermarket (see Section 5.4.4). Since CDDs have been found in fish from contaminated lakes and streams (Crunkilton et al. 1987; De Vault et al. 1989; EPA 1987n, 1992; Kuehl et al. 1989, 1994; Niimi and Oliver 1989a, 1989b; Reed et al. 1990) and fish and shellfish from estuarine waters (Bopp et al. 1991; Brown et al. 1994; Cai et al. 1994; Hauge et al. 1994; Rappe et al. 1991), populations that consume large quantities of fish or shellfish from these contaminated waters are also likely to have higher exposures to CDDs, although the method of preparation (edible fillets versus skin-on fillets or whole fish) and cooked versus raw consumption may substantially reduce the amount of CDDs ingested (Paustenbach et al. 1992; Schecter et al. 1996c).

Recreational (sport) and subsistence fishers (including some native American peoples) who consume locally caught fish from CDD-contaminated waterbodies can be exposed to higher CDD concentrations than individuals who consume similar or larger amounts of commercially marketed fish from a variety of sources (Ebert et al. 1996; EPA 1995c). The exposure to CDDs will also be highest among people who regularly eat large amounts of fish as compared to those who only occasionally or never eat fish. This increased exposure has been demonstrated by serum CDD levels to be several times higher in people who regularly eat fish as compared to those who occasionally or never eat fish (Anderson et al. 1998; Svensson et al. 1991).

Several studies suggests there is a correlation between consumption of CDD-contaminated fish and/or marine mammal tissues and elevated levels of CDDs in blood (Anderson 1998; Ayotte et al. 1998; Svensson et al. 1991). Svensson et al. (1991) reported elevated blood levels of CDDs in Swedish fish consumers living near the Baltic Sea. Three distinct groups of consumers were studied: individuals who did not consume any fish, moderate fish consumers (consumption rate of 220–500 g of fish/week [31–71 g/day]), and high fish consumers (consumption rate of 700–1,750 g of fish/week [100–250 g/day]). The highest fish consuming group was composed of fishermen or workers in the fishing industry who consumed primarily salmon (30–90 pg TEQ/g) and herring (8–18 pg TEQ/g) from the Baltic Sea. The TEQ blood level was found to average 63.5 pg TEQ/g lipid for the high consumption group, 25.8 pg TEQ/g lipid for the moderate consumption group, and 17.5 pg TEQ/g lipid for the nonfish-eating group. With respect to 2,3,7,8-TCDD blood levels, the mean blood level detected was 1.8 pg/g (lipid basis) for individuals that consumed no fish, 2.5 pg/g for the moderate consumers, and 8.0 pg/g for the high consumer group. It should be noted that even in those individuals who consumed no fish, detectable levels of CDDs were present in their blood. This indicates that other food sources (e.g., meat, milk, and other dairy products) are likely to be important contributors to the total body burden of CDDs (Rappe 1992).

Recently, Anderson et al. (1998) completed a preliminary study of the levels of 8 CDDs, 10 CDFs, 36 PCBs, and 11other persistent organochlorine pesticides in human serum samples from Great Lakes sport fish consumers. Overall, the 31 fishers on average consumed 49 Great Lakes sport fish meals per year, for a mean of 33 years. This is in contrast to the general population in the Great Lakes basin that typically consumes 6 meals of Great Lakes sport fish per year. A summary of the distribution of CDDs is provided in Table 5-14. CDD congeners detected most often were 1,2,3,4,6,7,8-HpCDD (31 detects), OCDD (31 detects), 1,2,3,6,7,8-HxCDD (30 detects), 2,3,7,8-TCDD (25 detects) and 1,2,3,7,8-PeCDD (20 detects). The overall mean concentration for 2,3,7,8-TCDD was 6.6 ppt. Total CDD concentrations were highest

Table 5-14. Mean and Range (ppt) of Serum CDD (Lipid Adjusted)

Dioxin congener	All subjects (n=3)	Lake Michigan (n=9)	Lake Huron (n=11)	Lake Erie (n=11)	Comparison group[a]
2,3,7,8-TCDD[b]	6.6 (ND–17.2)	4.7 (ND–7.9)	10.5 (4.4–17.2)	4.3 (ND–9.0)	2.8 (0.3–8.9)
1,2,3,7,8-PeCDD[b]	10.4 (ND–31.5)	9.8 (ND–23.7)	16 (ND–31.5)	5.8 (ND–12.3)	6.6 (0.6–14.1)
1,2,3,4,7,8-HxCDD	8.4 (ND–22.7)	11.4 (ND–16.3)	8.4 (2.1–22.7)	6.6 (ND–16.6)	9.0 (0.9–121)
1,2,3,6,7,8-HxCDD	126 (71.9–228)	120 (71.9–190)	142 (88.7–228)	115 (85.1–150)	70.8 (24.8–160)
1,2,3,7,8,9-HxCDD	7.0 (ND–22.8)	8.7 (ND–22.8)	6.5 (ND–16.1)	5.8 (ND–13)	9.4 (0.9–25.8)
1,2,3,4,6,7,8-HpCDD[b]	134 (34.9–314)	144 (72.5–204)	163 (86.7–314)	95.9 (34.9–179)	124 (29.1–358)
1,2,3,4,6,7,9-HpCDD[c]	[c]	ND	ND	[c]	4.4 (1.0–29.1)
OCDD	777 (297–1,869)	793 (409–1,587)	919 (371–1,869)	623 (297–981)	971 (286–2,710)
Dioxin total (ppt)	1,062 (453–2,410)	1,087 (615–2,017)	1,259 (729–2,410)	844 (453–1,286)	1,198[d]
Dioxin EPA TEQs[b]	27.5 (8.2–58.7)	25.9 (13.8–38.3)	36 (18.5–58.7)	20.7 (8.2–31.0)	15.5[d]

[a] Unexposed sample residing in Jacksonville, Arkansas (n=70)
[b] Three Great Lakes subgroups are statistically different ($p<0.03$)
[c] One observation detected
[d] Range not available

CDDs = chlorinated dibenzo-*p*-dioxins; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; ND = none detected; OCDD = octachlorinated dibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQ=toxicity equivalency concentration.

Source: Anderson et al. 1998

for Lake Huron fish consumers (1,259 ppt), intermediate for Lake Michigan consumers (1,087 ppt) and lowest for Lake Erie consumers (844 ppt). The comparison group serving as a control included individuals residing in Arkansas and had a total CDD serum concentration of 1,198 ppt. With respect to the TEQ values for CDDs, the pattern among Great Lakes fish consumers was similar to that for total CDD consumers with TEQs for Lake Huron fish consumers of 36 ppt, Lake Michigan consumers of 25.9 ppt, and Lake Erie consumers of 20.7 ppt. The TEQ values for the three Great Lakes sport fish consumer groups were statistically different ($p<0.03$). Although the comparison population had CDD concentrations within the range of the Lake Michigan and Lake Huron fish consumers, the TEQ value for CDDs for this population was the lowest of the four groups at 15.5 ppt. The authors concluded that Great Lakes anglers who are life-long frequent consumers of sport fish represent a subpopulation with the potential for significant exposure to CDDs as well as CDFs and PCBs. The levels of CDDs, CDFs, and PCBs found in sportfish and human tissue residues were above those in the general population.

Ayotte et al. (1998) measured concentrations of CDDs/CDFs and PCBs in plasma of adult Inuit living in Arctic Quebec, Canada. The Inuit consume large amounts of fish and marine mammal tissue. The mean concentration of 2,3,7,8-TCDD was 8.4 ppt (range 2.5 to 36.0 ppt) in the Inuit population and < 2 ppt (range <2) for the control population in Southern Quebec. The TEQ values for all CDDs/CDFs was 39.6 ppt (range 17.1 to 81.8 ppt) in the Inuit population and 14.6 ppt (range 11.5 to 18.9 ppt) for the control population. When PCBs and CDDs/CDFs are considered together, the mean TEQ values for all dioxin-like compounds was 184.2 ppt in the Inuit population (range 55.8 to 446.7 ppt) and 26.1 ppt (range 20.1 to 31.7 ppt) for the control population.

Several recent studies have documented the higher fish consumption rates among subsistence fishers some of which are Native American populations. In a study of Alaskan subsistence economies, Wolf and Walker (1987) reported daily fish consumption rates ranging from 6 to 1,536 g/day, with an average consumption rate of 304 g/day. This average consumption rate for subsistence fishers is more than 46 times higher than the mean fish consumption rate of 6.5 g/day estimated for the general population (EPA 1995c). In a study of 11 Alaskan communities, Nobmann et al. (1992) reported an average daily fish consumption rate of 109 g/day. This is more than 16.8 times higher than the mean fish consumption rate of 6.5 g/day estimated for the general population (EPA 1995c). A recent study of fish consumption patterns among the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River Basin in Washington and Oregon (CRITFC 1994) found that adults in these 4 tribes consume an average of 58.7 g/day of fish and the 95th percentile of fishers consume 170 g/day of fish. This mean consumption rate is more than nine times

higher than the mean fish consumption rate estimated for the general population (EPA 1995c). Fitzgerald et al. (1995) conducted a study to establish patterns of fish consumption of nursing Mohawk Indians residing in the vicinity of three hazardous waste sites located near Akwesasne, NY, compared to a control population of Caucasian women in New York state. The dietary data showed that there was a significant past prevalence of local fish consumption among Mohawk mothers (23.5 meals per year) more than a year before pregnancy as compared to the controls (14.1 meals per year).

In order to reduce CDD exposure from consumption of CDD-contaminated fish and shellfish, consumption advisories are issued by states recommending that individuals restrict their consumption of specific fish and shellfish species from certain waterbodies where CDD concentrations in fish and shellfish tissues exceed the human health level of concern. This level of concern is set by individual state agencies, but many states use the FDA tolerance levels of >25 ppt, but <50 ppt to advise consumers to restrict consumption and levels >50 ppt to issue advisories recommending no consumption of contaminated fish and shellfish (see Table 7-1). These values are used despite the fact that they were designed to protect consumers from the health risks associated with consumption of fish and shellfish that are shipped in interstate commerce and are purchased in commercial markets. In 1995, the EPA Office of Water issued guidance to states on sampling and analysis procedures to use in assessing the health risks from locally caught fish and shellfish. The risk assessment method proposed by EPA was specifically designed to assist states in developing fish consumption advisories for recreational and subsistence fishers (EPA 1995c). These two groups within the general population consume larger quantities of fish and shellfish than the general population and frequently fish the same waterbodies routinely. Because of this, these populations are at greater risk of exposure to CDDs and other chemical contaminants if the waters they fish are contaminated. The EPA advises states to use a screening value of $7x10^{-7}$ ppm (0.7 ppt) of total TEQ value (wet weight) in fillets for the general population as a criteria to evaluate their fishable waterbodies (EPA 1995c). Currently, 66 advisories have been issued by 21 states restricting the consumption of CDD-contaminated fish and shellfish (EPA 1998b). Three states (New Jersey, New York, and Maine) also have statewide fish advisories in effect for their marine coastal waters. In addition, the State of Arkansas also has issued a wildlife advisory for wood ducks, a species of waterfowl in Bayou Meto, a site contaminated by a point source discharge (White and Hoffman 1995). The number of waterbodies under advisory for CDD in each state is shown in Figure 5-8.

Subsistence hunters. Native American populations such as the Inuit of Alaska and other subsistence hunters (particularly those living in high latitude areas of the United States) may have higher exposure to

CDDs in marine mammals, but not terrestrial mammals (i.e., caribou). CDDs have been detected in seal and polar bear fat and liver tissues (Norstrom et al. 1990). Recently, Ayotte et al. (1997) reported that in 20 pooled samples of Inuit blood, the TEQ concentration supplied by CDDs/CDFs (combined) was 39.6 ppt as compared to 14.6 ppt in 3 pooled samples of blood from individuals in the general population in Southern Quebec. The mean 2,3,7,8-TCDD concentrations were 8.4 ppt for Inuit and <2 ppt for members of the general population. When planar PCBs, mono- and di- ortho PCBs were added, the mean TEQs were 184.2 ppt and 26.1 ppt, respectively, for Inuit and members of the general population in Southern Quebec. In a related study, Dewailly et al. (1992) found that breast milk of these Inuit was more contaminated by CDDs, CDFs, and PCBs than milk of women in Southern Quebec. For CDDs and CDFs, differences were less impressive than for PCBs. However, mean OCDD concentrations were 292 ppt versus 132 ppt and 2,3,7,8-TCDD concentrations were 6.2 ppt versus 2.3 ppt for Inuit women and women in Southern Quebec, respectively. Concentrations of OCDD and 2,3,7,8-TCDD were 2 to 3 times greater among the native Inuit population that consumed large amounts of fish and marine mammal tissue. No data were located specifically for CDD concentrations in the adipose tissue, blood, or breast milk of native American populations in the United States. However, by analogy to CDDs in Canadian Inuit populations, it is anticipated that CDD concentrations in these tissues are likely to be higher among individuals who routinely consume large quantities of wild game species, especially marine mammal than among members of the general population.

In a study of subsistence economies in the State of Alaska, Wolfe and Walker (1987) reported that total annual per capita consumption of wild game species (including land mammals, marine mammals, and fish) ranged from 10 to 1,498 pounds (median harvest of 252 pounds) as compared to 222 pounds of meat, fish, and poultry consumed each year by individuals in the western United States. In the 1980s, the 98 Alaskan subsistence communities surveyed harvested wild game at levels from one-half to 4 times the U.S mean. The average daily per capita consumption was 0.67 pounds for fish and 0.23 pounds for land mammals based on all 98 communities, and 0.2 pounds for marine mammals based on the 41 coastal communities surveyed. Land mammals consumed in these communities included moose, caribou, deer, black bear, snowshoe and tundra hare, beaver, and porcupine; while marine mammals included seal, walrus and whales. Subsistence hunters and their families are a population at potentially higher risk of CDD exposure when the wild game species particularly marine mammals they consume are contaminated with CDDs. It should be noted that concentrations of CDDs/CDFs were recently determined in caribou tissue samples from 7 herds across the Canadian Arctic (Hebert et al. 1996). In contrast to levels of 2,3,7,8-TCDD found in marine mammals which ranged from 2 to 37 ng/kg (ppt wet weight) (Norstrom et al. 1990),

concentrations of 2,3,7,8-TCDD in caribou were extremely low, sub ng/kg (lipid basis). 2,3,7,8-TCDD was detected in adipose tissue samples of two herds in the eastern Canadian Arctic at levels of 0.73 and 0.14 ng/kg, but was not detected in tissue samples from other herds at detections limits as low as 0.03 ng/kg (lipid basis). CDFs were detected at sub-ng/kg levels in all cases. TEQs were dominated by non-ortho substituted PCBs in all cases, and ranged from 0.33 ng/kg to 3.3 ng/kg in adipose tissue. The authors concluded that caribou tissues are therefore less contaminated than tissues from marine mammals.

Subsistence farmers that consume their own farm-reared meat and dairy products.

Subsistence farmers and their families living on farms where CDD concentrations may be high who exclusively eat meat and dairy products produced on their own farms may be exposed to higher levels of CDD in these foods than the general population. Grazing cattle in farming areas downwind of municipal or industrial incinerators where CDDs may be deposited as particulates on soil or forage crops or grazing cattle in areas with CDD-contaminated soil or soil amended with municipal sewage sludges or paper mill sludges contaminated with CDDs may result in higher CDD tissue residues in the animals (EPA 1991b; Fries and Paustenbach 1990). In an evaluation of potential transmission of 2,3,7,8-TCDD from incinerator emissions to humans via foods, Fries and Paustenbach (1990) determined that the amount of 2,3,7,8-TCDD accumulated in soil from airborne emissions was less important that the amount deposited in forage. These authors further concluded that the airborne emissions of CDDs/CDFs from modern waste incinerators that have appropriate air pollution devices should not pose a significant health hazard regardless of the incinerator location. The authors, however, acknowledged that it would be desirable to measure 2,3,7,8-TCDD in soil and crops around existing facilities to better evaluate their assessment results, although they felt it was likely that concentrations would be too low to reliably quantify. More recently, Fürst et al. (1993) reported that soil levels up to 30 pg TEQ/g dry matter did not result in elevated CDD concentrations in cow's milk. However, these authors did show increasing concentrations of CDDs in grass resulted in slightly higher CDD concentrations in cow's milk. These authors, like Fries and Paustenbach (1990), believe that the pathway of air to grass to cow is more important than the pathway of soil to grass to cow.

In a European survey of cow's milk samples collected on dairy farms, elevated CDD/CDF milk concentrations were used to pinpoint the existence of certain "hotspots" of CDD contamination. These "hotspots" were generally found near CDD/CDF emitting sources, such as cable waste incinerators or metal refining industries (Beck et al. 1990; Liem et al. 1991; Rappe et al. 1987). Riss et al. (1990) analyzed blood from one farmer in a CDD/CDF-contaminated location in proximity to a metal reclamation

plant who consumed milk produced from his own farm, and found 2,3,7,8-TCDD blood levels (55 pg/g on a lipid basis) above expected background levels.

Exposure to 2,3,7,8-TCDD from land application of municipal sewage sludge or paper mill sludge also can occur through the dietary pathway if people consume food grown or animals grazed on sludge-amended lands (EPA 1991b). Wild et al. (1993), using a human exposure assessment model, predicted that if all the produce consumed by a human is derived from agricultural land to which sewage sludge is applied at a rate of 10 tons/hectare and 0.5 ng/kg dry weight concentration, this would increase exposure to 2,3,7,8-TCDD by 0.0332 ng/day or 39% over background conditions. This scenario assumes that the poultry, eggs, and fish CDD concentrations are unaffected by the sludge application. Most recently, McLachlan et al. (1994) reported that the prolonged use of sewage sludge as a soil amendment on English farms under some conditions can lead to an increase in the concentrations of CDDs/CDFs in both the soil and in cow's milk. Subsistence farmers and their families are a population at potentially higher risk of CDD exposure because meat and dairy products are substantial sources of CDDs in the U.S. diet (Schecter et al. 1994e, 1996a).

5.8 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 CDDs is available. Where adequate information is not available, ATSDR, in conjunction with the 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 CDDs.

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.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. The physical and chemical properties of 2,3,7,8-TCDD are sufficiently characterized to predict the environmental fate of 2,3,7,8-TCDD (IARC 1977; Sax and Lewis 1987; Schroy et al. 1985; Shiu et al. 1988). Of all the CDDs, 2,3,7,8-TCDD has been the compound most studied. Not all isomers within each homologous class have been equally well studied for many of the physical and chemical properties. Information on physical and chemical properties of certain congeners (particularly 1,2,3,7,8,-PeCDD and 1,2,3,6,7,8-HxCDDs) would be helpful in better understanding the different fate and transport pathways of the homologous groups.

Production, Import/Export, Use, Release, and Disposal. CDDs are not manufactured commercially in the United States except on a laboratory scale for use in chemical and toxicological research (Cambridge Isotope Laboratories 1995). They are produced as undesired by-products during the manufacture of chlorophenols (e.g., PCP and 2,4,5-trichlorophenol) and during combustion processes (IARC 1977; NTP 1989; Podoll et al. 1986). CDDs are ubiquitous in the environment and have been found at low levels (ppt or lower) in air, water, soil, sediment, and foods. Current disposal methods are efficient and are subject to EPA and state regulations.

According to the Emergency Planning and Community Right-To-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1994, became available in May of 1996. This database will be updated yearly and should provide a list of industrial production facilities and emissions. However, there are no TRI data for CDDs since CDD releases are not required to be reported (EPA 1995g).

Environmental Fate. CDDs are subject to atmospheric transport and both wet and dry deposition (Kieatiwong et al. 1990). They are partitioned to air, water, sediment, and soil, and they accumulate in both aquatic and terrestrial biota. CDDs can volatilize to the atmosphere from water and soil surfaces. They adsorb strongly to soils and are not likely to leach into groundwater (Eduljee 1987b). In the aquatic environment, CDDs partition to sediment or suspended particulates. TCDD, HpCDD, and OCDD are subject to photolysis in air, water, and soil (Plimmer et al. 1973). 2,3,7,8-TCDD is biodegraded very slowly in soil and thus is likely to persist in the soil. A better understanding of environmental behavior of CDDs is needed with respect to the importance of vapor-phase versus particulate transport, the

environmental behavior of different congeners, and the significance of processes that reintroduce CDDs into the atmosphere after deposition. Information regarding the degradation of other congeners, specifically OCDD, and their degradation products in water, sediment, and soil would be useful in evaluating the various pathways of human exposure.

Bioavailability from Environmental Media. Toxicokinetic data in humans regarding adsorption of CDDs following oral and dermal exposure are very limited (Poiger and Schlatter 1986). CDDs can be absorbed following oral exposure in both humans and animals (Birnbaum and Couture 1988; Fries and Marrow 1975; Koshakji et al. 1984; Norback et al. 1975; Olson et al. 1980b; Piper et al. 1973; Poiger and Schlatter 1980). The more highly chlorinated CDD congeners are absorbed to a lesser extent than 2,3,7,8-TCDD (Koshakji et al. 1984). Also, limited information is available on the bioavailability from fly ash (Van den Berg et al. 1983, 1985). 2,3,7,8-TCDD can be adsorbed following dermal contact (Banks and Birnbaum 1991; Poiger and Schlatter 1980; Shu et al. 1988); however, dermal absorption of 2,3,7,8-TCDD from soil is very low (Shu et al. 1988). More information is needed regarding oral and dermal exposure to determine the bioavailability of CDDs from food, water, and soil. Additional information is needed to examine the discrepancy noted in the mass balance from CDDs ingested from foods and eliminated in feces. For inhalation exposure, information on the bioavailability from fly ash and sediments would be useful. Information is also needed on the selective uptake of the 2,3,7,8-substituted CDD congeners.

Food Chain Bioaccumulation. CDDs are bioconcentrated in aquatic organisms, plants, and terrestrial animals. Shellfish (including crustaceans and bivalve mollusks) appear to accumulate CDDs nonselectively to relatively high concentrations in their tissues (Bopp et al. 1991; Brown et al. 1994; Cai et al. 1994; Conacher et al. 1993; Hauge et al. 1994; Rappe et al. 1991). In contrast, finfish appear to selectively accumulate primarily 2,3,7,8-TCDD and other 2,3,7,8-substituted isomers in their tissues (Rappe et al. 1991). Information from a larger number of species on the retention of 2,3,7,8-substituted CDD congeners and general information on retention and distribution of other CDDs would be useful in better understanding both aquatic and terrestrial food chains.

Exposure Levels in Environmental Media. CDDs have been detected in air, water, soil, sediment, plant material, and foods. Environmental monitoring studies show that the higher chlorinated CDDs are usually the ones most commonly found in environmental samples (Christmann et al. 1989b; Clement et al. 1985, 1989; Pereira et al. 1985; Reed et al. 1990; Tashiro et al. 1989a; Tiernan et al. 1989b). Current

monitoring studies are needed to determine CDD levels in media surrounding hazardous waste sites. Using a model, the total average daily intake of 2,3,7,8-TCDD (by air, water, and food) for the general population was estimated to be 0.05 ng/day (range 0.008–0.3 ng/day) (Travis and Hattemer-Frey 1987). Schecter et al. (1994d, 1994e, 1996a) and Schecter and Li (1997) have provided current information on CDD exposures from food. Food consumption accounts for over 90% of background human exposure to 2,3,7,8-TCDD and other CDDs/CDFs in the general U.S. population (Hattemer-Frey and Travis 1989; Schaum et al. 1994). The average daily intake by nursing infants in the United States has been estimated to be 83 pg TEQs/kg (Schecter and Gasiewicz 1987a, 1987b).

Reliable monitoring data for the levels of CDDs in contaminated media at hazardous waste sites are needed so that the information obtained on levels of CDDs in the environment can be used in combination with the known body burdens of CDDs to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. CDDs/CDFs have been found in blood (Fingerhut et al. 1989; Needham et al. 1991; Päpke et al. 1989b, 1992, 1993), adipose tissue (Orban et al. 1994; Patterson et al. 1986a; Ryan et al. 1986; Schecter et al. 1986b; Stanley 1986; Stanley et al. 1986), and breast milk of both the general population and workers exposed through industrial accidents or environmental contamination (Fürst et al. 1992; Pluim et al. 1993a; Ryan et al. 1993b; Schecter and Gasiewicz 1987b; Schecter and Tiernan 1985a; Schecter et al. 1986a, 1986b; 1989e). Levels of 2,3,7,8-TCDD as well as other CDDs are generally higher in occupationally exposed individuals or those individuals exposed through industrial accidents or environmental contamination (Kahn et al. 1988; Schecter et al. 1986b; Schecter and Tiernan 1985; Schecter et al. 1987a; Umbriet et al. 1986a, 1986b). CDDs have also been detected in breast milk and blood of Canadian populations of native Inuit that consume large amounts of fish and marine mammals (Ayotte et al. 1997; Dewailly et al. 1992). Additional biological monitoring data are needed, however, for those U.S. populations surrounding hazardous waste sites or municipal, medical, or industrial incinerators, for urban versus rural exposures, and for other potentially exposed populations including subsistence fishers and hunters (Liem et al. 1991; Startin et al. 1989; Wuthe et al. 1993). Information on tissue levels in the general population worldwide are for the most part lacking (Schecter et al. 1991a). As they are identified, exposed populations should be evaluated to characterize exposure levels and health effects. This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. Children in the general population are exposed to CDDs primarily through dietary exposures *in utero* via placental blood and in newborn infants via breast-feeding. Despite the fact that studies on the concentrations of CDDs in human breast milk have been conducted in various other countries, there is a need to determine the levels of CDDs in human milk in the United States. Additional exposure studies also are needed to determine whether dietary modifications in mothers can reduce total CDD exposures in newborns and whether dietary modifications of the infant can also reduce lifetime exposure. For children in populations with potentially high exposure to CDDs, the primary exposure pathway is through their diet; however, additional exposure to CDDs via consumption of contaminated groundwater, contaminated soil, and dermal exposure to contaminated soil may increase their exposure levels. Studies of workers in various industrial settings that are exposed to CDDs (i.e., elevated CDD levels in adipose or blood serum) should be conducted to determine whether CDDs are routinely brought home by these workers on their clothing and shoes to assess in order to determine whether this is an important exposure route for children.

Schecter and Li (1997) have calculated weight-adjusted intakes of CDDs derived from consumption of four types of fast foods for 6-year-old children. Additional information on dietary intake of CDDs from other types of foods should be conducted for various age groups of children to help identify the magnitude and sources of dietary exposure during childhood. Studies to verify these calculations would be helpful in assessing health risks to children.

The primary childhood specific means to decrease exposure to CDDs involves placing the infant on a cow's milk or soy-based baby formula and on maintenance of children on a long-term diet that is lower in animal fats (meat, dairy products, and fish) and higher in grains, fruits, and vegetables. It should be noted however, that because of the relatively short period of intake and the accepted benefits of breast-feeding that maintenance of children on long-term diet low in animal fat would likely be more beneficial in decreasing total lifetime CDD body burdens than cessation of breast-feeding. Additional means of reducing CDD exposures also should be investigated.

Exposure Registries. Approximately 250 members were enrolled in the 2,3,7,8-TCDD Subregistry of the National Exposure Registry in 1991 (ATSDR 1996). These individuals were chosen because they participated in one or more of the Missouri Dioxin Health Studies and were reportedly exposed to CDDs at one of the four Times Beach, MO area CDD sites. Data collected for each member of the Dioxin Subregistry include demographic information, smoking and occupational histories, and self-reported

responses to 25 general health status questions. The data files for the Subregistry are established at the time baseline data are collected. A follow-up survey is conducted 1 year after baseline data collection, and surveys are, in most cases, conducted at 2-year intervals after that to update the files. For the Dioxin Subregistry, all interviews are conducted by means of computer-assisted telephone interviewing. Subregistry members will be questioned yearly about their health over the previous year. This activity is carried out by ATSDR. The data will become part of public-user data files maintained by ATSDR. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to the exposure to this compound.

The Air Force maintains an exposure registry of about 1,200 personnel previously involved in the spraying of Agent Orange (USAF 1991). The Air Force Health Study (AFHS) is an epidemiologic investigation of the association between occupational exposure to Agent Orange (and its CDD contaminants) and long-term adverse health effects experienced by Air Force personnel who served in Operation Ranch Hand units in Vietnam from 1962 to 1971 and sprayed Agent Orange from fixed winged aircraft. A comparison group, which was formed from Air Force veterans, is used as the unexposed cohort. Evaluations were performed in 1982, 1985, and 1987. In the 1987 examination, 1,670 participants were involved. Health outcomes were evaluated with respect to serum CDD levels. Additional evaluations are planned for 1997 and 2002.

5.8.2 Ongoing Studies

The EPA is currently conducting a reassessment of the risk from exposure to CDDs, and other chlorinated dioxin-like compounds such as CDFs and PCBs. This reassessment involves a literature reevaluation of existing studies and new laboratory studies addressing health and ecological risks from exposure to these compounds (LaFleur et al. 1990; Rappe 1992; Schecter et al. 1994d). Currently, the EPA dioxin reassessment document is undergoing final review.

The National Institute of Environment Health Sciences and the Centers for Disease Control and Prevention are measuring levels of CDDs, CDFs, PCBs and other chemicals in blood of members of the general U.S. population as part of the NHANES program.

In addition, an international “Dioxin” research conference meets annually to discuss developments regarding these environmental contaminants. The proceedings of this international symposium on CDDs and related compounds are published annually in extended abstract form and frequently in a proceedings

issue of the journal Chemosphere and are an extensive source for papers on a wide variety of environmental and health issues related to CDDs and dioxin-like compounds.

A search of Federal Research in Progress (FEDRIP 1998) identified numerous research studies that are currently being conducted that may fill some of the data needs discussed in Section 5.7.1 (see Table 5-15).

Table 5-15. Ongoing Studies on Environmental Fate and Treatment of CDDs

Investigator	Affiliation	Title	Sponsor
Bunge AL	Colorado School of Mines Golden, CO	Dermal Adsorption from Soils—Evaluation and Prediction	National Institute of Environmental Health Sciences
Cooper K	Rutgers University New Brunswick, NJ	Effects of Polychlorinated: Dioxins, Furans, and PCBs on Aquatic Animals and Humans	U.S. Department of Agriculture
Dasinger AM	Geophex, Ltd. Raleigh, NC	Development of a Two State Bioremediation Technology for Dioxin Contaminated Soils	U.S. Air Force
Eskenazi B	University of California, Berkeley CA	Endometriosis and Dioxin Exposure in Females of Seveso	National Institute of Environmental Health Sciences
Fynes G	British Coal Corporation: Coal Research Establishment Stoke Orchard, Cheltenham, England	Emissions of Environmental Concern from Coal Utilization	European Coal and Steel Community (ECSC); British Coal Corporation; Department of Trade and Industry
Hodko D	Lynntech, Inc. College Station, TX	In Situ Degradation of Dioxins by Chemical Oxidation	U.S. Air Force
Hites RA	Indiana University, School of Public and Environmental Affairs Bloomington, IN	Toxic Organic Compounds from Energy Production	U.S. Department of Energy
Hites RA	Indiana University, School of Public and Environmental Affairs Bloomington, IN	A Global Mass Balance for Chlorinated Dioxins and Dibenzofurans	National Science Foundation
James MO	University of Florida Gainesville, FL	Bioavailability of Superfund Chemicals	National Institute of Environmental Health Sciences
Kang HK	Department of Veterans Affairs, Medical Center Washington, DC	Army Chemical Corp Vietnam Veterans Health Study	Department of Veterans Affairs,
Newsted JL	University of Massachusetts Forestry and Wildlife Management Amherst, MA	The Evaluation of Halogenated Aromatic Hydrocarbons in Fish from the Connecticut River Basin, MA	U.S. Department of Agriculture
Shaub W	Solid Waste Association of North Carolina Silver Spring, MD	Mercury and Dioxin in Waste Streams	U.S. Department of Energy
Wolff MS	Mount Sinai School of Medicine of CUNY New York, NY	Core–Exposure Assessment Providing chemical analyses for PAH and TCDD, compounds that are environmentally important	U.S. Department of Health and Human Services

Table 5-15. Ongoing Studies on Environmental Fate and Treatment of CDDs (continued)

Investigator	Affiliation	Title	Sponsor
Zauderer B	Coal Tech Corp Merion Station, PA	Control of Dioxin Emissions from Waste Fuel Combustion by Cofiring with Coal	U.S. Department of Energy
Zimbeck W	Technology Assessment & Transfer, Inc. Annapolis, MD	Experimental Evaluation and Scientific Study of Treatment Technologies for Dioxin Contaminated Soil	U.S. Air Force
Not identified	Xenobiotic Detection Systems, Inc., Durham, NC	Validation of Serum Bioassay for Dioxin-like Toxicants	U.S. Department of Health and Human Services HHS
Not identified	Hybrizyme Corp. Raleigh, NC	New Test Method for Dioxins in Human and Animal Samples	
Not identified	A. Ahlstroem Osakeyhtioe, Karhula, Finland	Processing and Flue Gases and Ashes from CFB Combustion of Municipal Waste to Control Dioxine Concentrations	Ministry of Trade and Industry, Helsinki (Finland)
Not identified	Kuopio University, Department of Environmental Sciences Kuopio, Finland	Release of Chlorinated Hydrocardons in Refuse Incineration	Imatran Voiman Saeaetioe, Helsinki (Finland)
Not identified	Technical Research Centre of Finland, Combustion and Thermal Engineering Lab Jyvaeskylae (Finland)	Emissions from Fluidized Bed Combustion of Waste	Ministry of Trade and Industry, Helsinki (Finland)
Not identified	Kuopion University, Department of Environmental Sciences Kuopion University	Formation of Chlorinated Hydrocarbons in Combustion of Waste Materials	Ministry of Trade and Industry, Helsinki (Finland)
Not identified	AEA Technology	Coalite Works: Soil Survey and Emission Monitoring	Her Majesty's Inspectorate of Pollution
Not identified	Environmental Protection Agency (EPA), Acurex Environmental Corporation, Research Triangle Park, NC	The Effect of Coal Sulfur on Dioxin Formation	