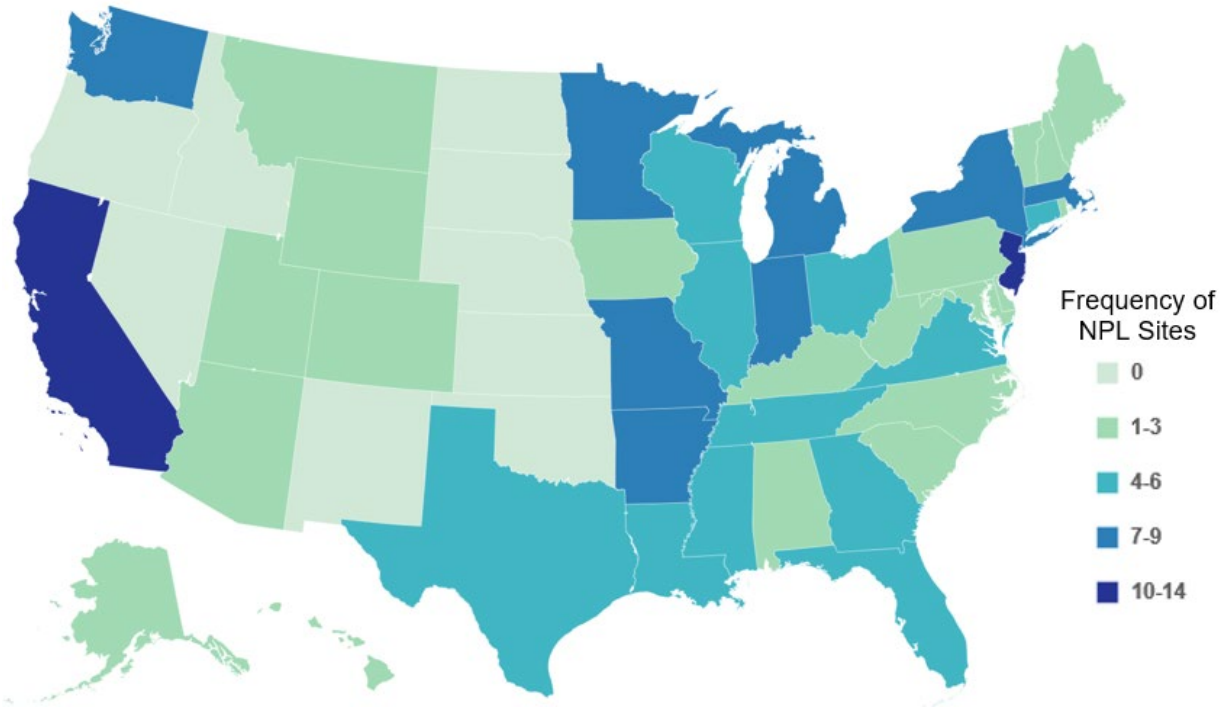
CHAPTER 5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

CDDs have been identified in at least 179 of the 1,867 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (ATSDR 2022). However, the number of sites in which CDDs have been evaluated is not known. The number of sites in each state is shown in Figure 5-1. Of these sites, 177 are located within the United States, and 2 are located in Puerto Rico (not shown).

Figure 5-1. Number of NPL Sites with Chlorinated Dibenzo-*p*-Dioxin (CDD) Contamination



Source: ATSDR 2022

- Ingestion of food items containing CDDs is the primary exposure pathway for the general population.
- Inhalation of ambient air, as well as ingestion of drinking water, are minor routes of human exposure to CDDs; however, inhalation exposure can be a major source in specific locations, near specific industrial sites. Exposure can also occur from certain consumer products.
- The lower chlorinated CDDs are semi-volatile; however, the tetra-, penta-, hexa-, and octa-congeners are considered nonvolatile.

- The lower chlorinated CDDs degrade in the atmosphere by reaction with atmospheric oxidants in a matter of days; however, the higher chlorinated congeners are more persistent and subject to long-range transport. Dioxins have also a high partitioning ratio to ambient particulate matter and particulates released from emission sources.

- Direct photolysis of CDDs is an important degradation process; however, biodegradation occurs slowly, especially for the higher chlorinated CDDs and they are considered persistent in the environment.

- CDDs have large soil adsorption coefficients and possess low mobility in soil surfaces. CDDs bioconcentrate in aquatic organisms.

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). 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). A more detailed discussion of the relative toxicities of the different CDD congeners is provided in Section 2.1.

CDDs in the environment are often measured and studied in conjunction with CDFs, and further information on these substances can be found in the ATSDR Toxicological Profile for CDFs (ATSDR 2023). 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 elicit a number of similar toxicological and biochemical responses in animals. 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); 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). EPA has developed procedures for estimating risks associated with exposures to mixtures of CDDs and CDFs in environmental matrices (EPA 1989). This approach is based on the assignment of 2,3,7,8-TCDD TEFs to CDD/CDF congeners or homologues in complex mixtures. The rationale behind the use of TEFs is explained in Section 2.1. 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 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 2.1).

Source-specific regulations, improvements in source technology, advancements in pollution control technologies, and voluntary actions of U.S. industries (such as metal smelting) to reduce or prevent dioxin releases have decreased the amount of CDDs and CDFs emitted to the environment over the past several decades (EPA 2006). It is currently estimated that nearly 90% of all U.S. total dioxin emissions arise from landfill fires, forest and brush fires, and backyard burning (Dwyer and Themelis 2015). The 2012 dioxin emissions from 53 U.S. waste-to-energy (WTE) power plants that combusted a total of 27.4 million metric tons emitted 3.4 g TEQ and represented only 0.54% of the controlled industrial dioxin emissions.

CDDs occurred 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 byproduct formed in the manufacture of 2,4,5-TCP (Arthur and Frea 1989). 2,4,5-TCP was used to produce the bactericide, hexachlorophene, and the chlorophenoxy herbicide, 2,4,5-T. Trichlorophenol-based herbicides were used extensively for weed control on crops, rangelands, roadways, rights-of-way, etc. Various formulations of 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 (Wolfe et al. 1985). Agent Orange was a 1:1 mixture of 2,4-D and 2,4,5-T. Hexachlorophene use has been restricted by the U.S. Food and Drug Administration (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. 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 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, but its uses are scheduled for cancellation by February 28, 2027 (EPA 2021).

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, 1986a, 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 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. 1989b, 1989e, 1991a, 1994d; Tiernan et al. 1989). HpCDD and OCDD are the most common CDDs found in environmental samples (Christmann et al. 1989; Clement et al. 1985, 1989; Pereira et al. 1985; Reed et al. 1990; Tashiro et al. 1989a; Tiernan et al. 1989).

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.

The detection of CDDs in blood, adipose tissue, human milk, and other tissue samples from the general population indicates universal exposure to CDDs from environmental sources (CDC 2024a, 2024b; 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. 1994a). The major source (>90%) of exposure for the general population, however, is primarily associated with meat, dairy products, and fish (Beck et al. 1989a; FDA 2006; Schaum et al. 1994; Schecter et al. 1994a, 1994d, 1996a). CDDs are transferred through the placenta to the fetus, by human 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, phenoxy herbicides, hexachlorophene, PCP, 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. 1991b). 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 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).

5.2 PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL

5.2.1 Production

CDDs are not manufactured commercially in the United States except on a small scale for use in chemical and toxicological research. CDDs are unique among the large number of organochlorine compounds of environmental interest in that they were never intentionally produced as desired commercial end products (Zook and Rappe 1994). Typically, CDDs are unintentionally produced during various uncontrolled chemical reactions involving the use of chlorine (EPA 1990a) and during various combustion and incineration processes (Zook and Rappe 1994). CDDs are also produced as undesired byproducts during the manufacture of chlorinated phenols such as PCP, 2,4,5-TCP, and related chemicals, and during incineration of chlorinated wastes (IARC 1977; NTP 1989; Podoll et al. 1986). By far, the greatest unintentional production of CDDs occurs via various combustion and incineration processes including all forms of waste incineration (municipal, industrial, and medical), many types of metal production (iron,

steel, magnesium, nickel, lead, and aluminum), and fossil fuel and wood combustion (Czuczwa and Hites 1986a, 1986b; Oehme et al. 1987, 1989; Zook and Rappe 1994).

In general, there are two conventional methods for the preparation of CDDs for research purposes: condensation of a polychlorophenol and direct halogenation of the parent dibenzo-*p*-dioxin or a monochloro- derivative. For example, 2,3,7,8-TCDD is generally synthesized by the condensation of two molecules of 2,4,5-TCP in the presence of a base at high temperatures or by chlorination of dibenzo-*p*-dioxin in chloroform in the presence of iodine and ferric chloride (EPA 1987b; IARC 1977). Other methods of 2,3,7,8-TCDD synthesis include the following: pyrolysis of sodium α-(2,4,5-trichloro-phenoxy) propionate at 500 EC for 5 hours; reaction of dichlorocatechol salts with *o*-chlorobenzene by refluxing in alkaline dimethyl sulfoxide; ultraviolet (UV) irradiation of CDDs of high chlorine content; Ullman reaction of chlorinated phenolates at 180–400 EC; pyrolysis of chlorinated phenolates and chlorinated phenols; and heating 1,2,4-trichloro-5-nitrobenzene and 4,5-dichlorocatechol in the presence of a base (EPA 1984; IARC 1977).

1,2,3,4-TCDD has been prepared by refluxing a mixture of catechol, potassium carbonate, pentachloronitrobenzene, and acetone in nitrogen (IARC 1977).

DCDD can be synthesized by two methods. In the first method, 2-bromo-4-chlorophenol and potassium hydroxide are dissolved in methanol and evaporated to dryness. The residue is then mixed with bis(2-ethoxyethyl) ether, ethylene diacetate, and a copper catalyst; and then heated, cooled, and eluted from a chromatographic column with chloroform. This residue is evaporated and then sublimed. DCDD can also be synthesized by heating the potassium salt of 2,4-dichlorophenol in the presence of copper powder in a vacuum sublimation apparatus (IARC 1977).

1,2,4,6,7,9-HxCDD has been made by heating the potassium salt of 2,3,5,6-tetrachlorophenol with powdered copper and potassium carbonate in a vacuum sublimation apparatus (IARC 1977). 1,2,3,4,7,8-HxCDD has been prepared by mixing 1,2,3,4-TCDD, ferric chloride, chloroform, and a crystal of iodine and then adding a solution of chlorine in carbon tetrachloride (IARC 1977).

OCDD has been synthesized by the following methods: irradiation of aqueous solutions of CDD-free sodium PCP with UV light; heating the potassium salt of PCP; heating PCP in the presence of an initiator, such as chlorine, bromine, iodine, or 2,3,4,4,5,6-hexachloro-2,5-cyclohexadienone; and heating

hexachlorocyclohexadienone in an atmosphere of carbon dioxide for 30 minutes (Crosby and Wong 1976; EPA 1984; IARC 1977).

Table 5-1 summarizes information on companies that reported the production, import, or use of dioxin-like substances, including CDDs, and the range of maximum amounts that are stored onsite for the Toxics Release Inventory (TRI) in 2021 (TRI21 2022). This is a special category in the Toxics Release Inventory (TRI) and includes 17 CDDs and CDFs. TRI data should be used with caution since only certain types of industrial facilities are required to report. This is not an exhaustive list.

Table 5-1. Facilities that Produce, Process, or Use Dioxin and Dioxin-like Compounds

State[a]	Number of facilities	Minimum amount on site in pounds[b]	Maximum amount on site in pounds[b]	Activities and uses[c]
AK	5	0	0.99	1, 5
AL	45	0	99,999	1, 5, 8, 12, 13, 14
AR	20	0	99,999	1, 5, 12, 13, 14
AZ	13	0	99	1, 5, 6, 13
CA	27	0	99	1, 2, 5, 11, 12, 13, 14
CO	13	0	9.99	1, 4, 5, 13
CT	1	0	0.10	1, 5
DE	1	0.10	0.99	1, 13, 14
FL	21	0	9.99	1, 5, 12, 13, 14
GA	30	0	99,999	1, 2, 5, 12, 13, 14
GU	1	0.10	0.99	1, 5
HI	4	0	0.10	1, 5
IA	18	0	9,999	1, 5, 13, 14
ID	3	0	9,999	1, 5, 12, 14
IL	19	0	99	1, 5, 12, 13, 14
IN	28	0	99	1, 5, 12, 13
KS	7	0	0.99	1, 5, 12
KY	29	0	9,999	1, 5, 13, 14
LA	46	0	99,999	1, 5, 10, 12, 13, 14
MD	5	0	9.99	1, 5, 14
ME	3	0	0.99	1, 5, 9
MI	18	0	9,999	1, 2, 5, 12, 13, 14
MN	19	0	999,999	1, 5, 12, 13, 14
MO	25	0	9.99	1, 2, 5, 12, 13, 14
MS	19	0	99,999	1, 5, 8, 13, 14
MT	5	0	9.99	1, 5
NC	23	0	99,999	1, 5, 8, 12, 13, 14

Table 5-1. Facilities that Produce, Process, or Use Dioxin and Dioxin-like Compounds

State[a]	Number of facilities	Minimum amount on site in pounds[b]	Maximum amount on site in pounds[b]	Activities and uses[c]
ND	12	0	0.99	1, 5, 12, 13
NE	12	0	99,999	1, 5, 13, 14
NJ	6	0	9.99	1, 5, 13, 14
NM	3	0	0.10	1, 5, 13
NV	5	0	999,999	1, 5, 13, 14
NY	15	0	9,999	1, 5, 12, 13, 14
OH	31	0	999	1, 3, 5, 13, 14
OK	13	0	99	1, 4, 5, 13, 14
OR	11	0	999,999	1, 5, 14
PA	26	0	9.99	1, 2, 5, 12, 13, 14
PR	1	0	0.10	1, 5
SC	24	0	9.99	1, 5, 12, 13, 14
SD	3	0	99	1, 5
TN	24	0	99	1, 5, 8, 12, 13, 14
TX	66	0	9,999	1, 2, 3, 4, 5, 7, 12, 13, 14
UT	17	0	99,999	1, 3, 4, 5, 9, 12, 13, 14
VA	13	0	9.99	1, 5, 7, 13, 14
VI	2	0	0.10	1, 5
WA	22	0	99,999	1, 2, 5, 6, 13, 14
WI	26	0	99,999	1, 2, 5, 12, 13, 14
WV	15	0	99	1, 5, 12, 13
WY	9	0	9.99	1, 5, 13, 14

[a]Post office state abbreviations used.
[b]Amounts on site reported by facilities in each state.
[c]Activities/uses:

1. Produce
2. Import
3. Used Processing
4. Sale/Distribution
5. Byproduct
6. Reactant
7. Formulation Component
8. Article Component
9. Repackaging
10. Chemical Processing Aid
11. Manufacture Aid
12. Ancillary
13. Manufacture Impurity
14. Process Impurity

The specific chemicals of this category are Chemical Abstracts Service (CAS) numbers 67562-39-4 (1,2,3,4,6,7,8-heptachlorodibenzofuran), 55673-89-7 (1,2,3,4,7,8,9-heptachlorodibenzofuran), 70648-26-9 (1,2,3,4,7,8-hexachlorodibenzofuran), 57117-44-9 (1,2,3,6,7,8-hexachlorodibenzofuran), 72918-21-9 (1,2,3,7,8,9-hexachlorodibenzofuran), 60851-34-5 (2,3,4,6,7,8-hexachlorodibenzofuran), 39227-28-6 (1,2,3,4,7,8-hexachlorodibenzo-*p*-dioxin), 57653-85-7 (1,2,3,6,7,8-hexachlorodibenzo-*p*-dioxin), 19408-74-3 (1,2,3,7,8,9-hexachlorodibenzo-*p*-dioxin), 35822-46-9 (1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin), 39001-02-0 (1,2,3,4,6,7,8,9-octachlorodibenzofuran), 3268-87-9 (1,2,3,4,6,7,8,9-octachlorodibenzo-*p*-dioxin), 57117-41-6 (1,2,3,7,8- pentachlorodibenzofuran), 57117-31-4 (2,3,4,7,8-pentachlorodibenzofuran), 40321-76-4 (1,2,3,7,8- pentachlorodibenzo-*p*-dioxin), 51207-31-9 (2,3,7,8-tetrachlorodibenzofuran), and 1746-01-6 (2,3,7,8-tetrachlorodibenzo-*p*-dioxin).

Source: TRI21 2022 (Data are from 2021)

5.2.2 Import/Export

CDDs are not imported into the United States (NTP 1989). There were no data located pertaining to the export of any CDD for research purposes.

5.2.3 Use

The only reported use of CDDs/CDFs is as research chemicals (NTP 1989). A large, diversified group of researchers use various CDDs in studies of toxicology, environmental fate, transformation, and transport, and in residue analysis of a variety of contaminated media. The immunotoxic properties of CDDs have also been used in studies evaluating other nontoxic AhR ligands as possible treatments of autoimmune diseases. CDDs have been tested for use in flame-proofing polymers such as polyesters and against insects and wood-destroying fungi; however, there are no data reporting commercial production or use for these purposes (IARC 1977).

5.2.4 Disposal

The 1986 estimates on the degree of TCDD contamination in the environment indicated that approximately 500,000 tons of soil and sediment in the United States were contaminated with 2,3,7,8-TCDD (U.S. Congress 1991). The development of treatment technologies for CDD-contaminated soils and wastes needed to address unique problems associated with CDDs; for example, they are insoluble in water, only slightly soluble in organic solvents, have a strong affinity for adsorption on organic matter, and are biologically and environmentally stable (U.S. Congress 1991). In order to meet the clean-up standards established for CDDs, the treatment system must be capable of removing the CDDs from the contaminated matrix (U.S. Congress 1991). Several treatment or disposal methods for CDDs and CDD-contaminated materials have been investigated, including land disposal, thermal destruction, and chemical and biological degradation.

Land disposal of CDD-containing wastes is prohibited unless the dioxin-containing waste is contaminated soil and debris resulting from a response action taken under Section 104 or 106 of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) or a corrective action taken under Subtitle C of RCRA (EPA 1986b, 1988). The Toxic Substances Control Act (TSCA) regulates the use, disposal, and distribution in commerce of process wastewater treatment sludges

intended for land application from pulp and paper mills employing chlorine or chlorine derivative-based bleaching processes (EPA 1991a, 1991b). Also, under the Marine Protection Research and Sanctuaries Act (MPRSA), ocean dumping of CDD-containing wastes is prohibited except when only trace amounts are present (EPA 1977). EPA is responsible for designating and managing ocean dumping sites under the MPRSA for all types of materials. EPA's published ocean dumping regulations appear at 40 CFR Parts 220–229 (EPA 2024). Brief summaries of amendments to this law are available (Congressional Research Service 2016).

Thermal destruction technologies offer the most straightforward approach to treating or disposing of CDD-contaminated materials because under the appropriate conditions, the breakdown of the CDDs is assured (U.S. Congress 1991; WHO 2023). The thermal treatment technologies that are used to treat waste containing hazardous or toxic constituents and that have demonstrated potential use toward the treatment of CDD-contaminated waste include rotary kiln incineration, liquid injection incineration, fluidized-bed incineration, advanced electric reactor (AER), infrared incineration, plasma arc pyrolysis incineration, supercritical water oxidation, and *in situ* vitrification (U.S. Congress 1991). In addition to kiln incinerators, the technologies that have been field-tested for treating CDD-contaminated media under EPA's Superfund Innovative Technology Evaluation (SITE) program include dechlorination, stabilization, and *in situ* vitrification (U.S. Congress 1991). Kulkarni et al. (2008) discusses disposal and remediation technologies of dioxins.

Incineration, involving the high-temperature oxidation of CDD molecules, is the most extensively tested method for disposal of CDDs. CDDs such as TCDD, PeCDD, and HxCDD are classified by EPA as Principal Organic Hazardous Constituents (POHCs). Destruction of compounds with the potential to form dioxins are required to be incinerated under conditions that achieve a destruction and removal efficiency of 99.9999% (EPA 1990b; Sedman and Esparza 1991). Proper incineration of dioxin-contaminated material is the best available method of preventing and controlling exposure to dioxins (WHO 2023). Incineration can also destroy PCB-based waste oils. The incineration process requires temperatures >850°C. For the destruction of large amounts of contaminated material, temperatures of ≥1,000°C are required (WHO 2023).

Kulkarni et al. (2008) discussed treatment and remediation technologies used for dioxins emitted from flue gases. These technologies include particulate matter collection, scrubbers and electrostatic precipitators, sorbent or flow injection processes, fluidized bed processes, and electron irradiation. Waste incineration plants commonly employ filters equipped with activated charcoal or fixed bed activated

carbon filters to reduce emissions of dioxin-like substances. Selective catalytic reduction for NO_x reduction combined with an oxidation catalyst are an effective technology to destroy dioxins. Various methodologies exist to treat contaminated fly ash such as thermal treatment, chemical reactions, non-thermal plasma technology, UV irradiation, hydrothermal treatment, and supercritical water oxidation.

Since the early 1970s, several chemical methods have been investigated for the degradation of CDDs. Treatment of CDD-contaminated materials with alkali polyethylene glycolate (APEG) reagents at hazardous waste sites has been demonstrated to successfully destroy CDDs in liquid wastes and to be viable even under difficult circumstances. This method involves the reaction of potassium hydroxide with polyethylene glycol to form an alkoxide that reacts with one of the chlorine atoms on the CDD to produce an ether and potassium chloride. Bioassays indicate that the byproducts produced by treating 2,3,7,8-TCDD with APEG reagents do not bioaccumulate or bioconcentrate, do not cause mutagenicity, and are far less toxic than 2,3,7,8-TCDD (Klee 1988). Cleavage of the ether linkages with the formation of halophenols may be achieved by treatment with strong acids or quaternary ammonium salts, but the dibenzodioxin nucleus is resistant to chemical attack. Oku et al. (1995) investigated the dechlorination of polychlorinated CDDs and polychlorinated CDFs using a modified alkali-metal hydroxide method. The destruction reagent, prepared by dissolving either potassium hydroxide or sodium hydroxide in 1,3-dimethyl-2-imidazolidinone (DMI) destroyed all components, regardless of the difference in the number of chlorine atoms or isomers of CDDs and CDFs (Oku et al. 1995). The efficiency of the methods was evaluated under varying conditions; in the presence and absence of water, at 90 and 50 EC, for 0.5 and 5 hours. Although the degree of CDD destruction (99.95–99.80%) was less than that for CDFs (99.99–99.98%), overall, the investigators considered the DMI reagent to be more useful than the polyethylene glycols because of its stability under strongly basic conditions and its efficiency in the presence of water (Oku et al. 1995).

CDDs/CDFs can be destroyed by dechlorination of the compounds by UV light most efficiently in the presence of hydrogen donors. The most commonly used hydrogen donor is isopropyl alcohol (des Rosiers 1983). TCDD-contaminated soil was decontaminated by UV treatment of the soil in the presence of olive oil emulsion as a hydrogen donor. A total reduction in excess of 60% was observed after 48 hours of irradiation. Photocatalytic degradation of dioxins using semi-conductor films like TiO_2, ZnO, CdS, and Fe_2O_3 is possible (Kulkarni et al. 2008).

Dougherty et al. (1993) conducted a theoretical analysis of a proposed *in situ* method for decontaminating soil by photodegradation. Up to 87% of TCDD in the soil can be degraded by this process (McPeters and

Overcash 1993). Because of its extremely low water solubility and volatility, TCDD is a very persistent soil contaminant. With the method, based on the physical properties that facilitate photolysis of TCDD by sunlight, an organic solvent mixture (2:1 w/w) of tetradecane and 1-butanol is applied to the contaminated soil (Dougherty et al. 1993). The controlling factors in TCDD photodegradation are desorption of the compound from the soil, the transport mechanism to the soil surface, and the availability of sunlight. As the solvents remove the tightly bound TCDD from the soil, convective upward movements of the compound are caused by the evaporation of the solvent (Dougherty et al. 1993; Zhong et al. 1993). The effectiveness of the process also depends on a balance between the convective movement and sunlight availability for degradation (Dougherty et al. 1993). Modeling conducted by Zhong et al. (1993) identified and quantified the controlling factors governing the TCDD photodegradation process. Following the concentration variation of TCDD in the top 2 mm of soil through sunlight/night cycles over an exposure period of 15 days, the model showed that during the daytime of the first few days, there is little accumulation of TCDD as the losses due to photodegradation were almost equal to the convective flux in magnitude but with different signs. Although the losses due to photodegradation drop to zero at night, the convective flux effected a build-up of TCDD. The losses due to photodegradation held steady while the convective movements decreased as evaporation slowed down (Zhong et al. 1993). A balance between the build-up of TCDD concentration at night and the drop in concentration during the day did not occur until the 11^{th} day of exposure (Zhong et al. 1993).

Hilarides et al. (1994) investigated degradation of TCDD in the presence of surfactants. Their results indicated that radiolytic destruction of TCDD using γ radiation can be achieved. Greater than 92% of the TCDD was destroyed in soils amended with 100 ppb TCDD, 25% water, and 2% nonionic surfactant using ^{60}Co at high radiation doses (800 kGy or 80 Mrad). The use of ^{60}Co as a source avoids the temperature increases and power requirements of other sources of ionizing radiation such as an electron beam. It is also better suited for soil application because of its greater penetration depths (Hilarides et al. 1994).

Biotreatment systems that use microorganisms for degradation of refractory organopollutants, like CDDs, have also been considered. *Phanerochaete chrysosporium*, a white rot fungus, has shown the ability to slowly degrade 2,3,7,8-TCDD in the laboratory (Bumpus et al. 1985; Des Rosiers 1986). The ability of this fungus to metabolize 2,3,7,8-TCDD is thought to be related to its extracellular lignin degrading enzyme system (Bumpus et al. 1985; Des Rosiers 1986).

5.3 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report (EPA 2022). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ ≥10 full-time employees; if their facility's North American Industry Classification System (NAICS) codes is covered under EPCRA Section 313 or is a federal facility; and if their facility manufactures (defined to include importing) or processes any TRI chemical in excess of 25,000 pounds, or otherwise uses any TRI chemical in excess of 10,000 pounds, in a calendar year (EPA 2022).

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. Incineration/combustion processes are the most important sources of CDDs to the environment (EPA 2006; 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; EPA 2006; Thoma 1988; Zook and Rappe 1994).

5.3.1 Air

Estimated releases of 1,067 g (~2.35 pounds [<1 metric ton]) of dioxin compounds, including CDDs, to the atmosphere from 799 domestic manufacturing and processing facilities in 2021 accounted for about <1% of the estimated total environmental releases from facilities required to report to the TRI (TRI21 2022). These releases are summarized in Table 5-2.

For reporting purposes in the TRI, dioxin-like substances releases are reported in grams per year.

Table 5-2. Releases to the Environment from Facilities that Produce, Process, or Use Dioxin and Dioxin-like Compounds[a]

	Reported amounts released in grams per year[b]								
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
AL	45	22	110	0	5,186	0	5,280	38	5,319
AK	5	2	0	0	0	0	2	0	2
AZ	13	24	0	0	55	0	24	55	79
AR	20	15	14	0	22	2	47	6	53
CA	27	4	4	0	70	0	26	52	78
CO	13	14	0	0	0	0	14	0	14
CT	1	0	0	0	0	0	0	0	0
DE	1	0	0	0	0	0	0	0	0
FL	21	12	5	0	9	0	24	3	27
GA	30	17	24	0	20	0	61	1	61
HI	4	0	0	0	1	0	0	1	1
ID	3	26	0	0	1,920	0	1,947	0	1,947
IL	19	11	0	0	11	0	11	11	22
IN	28	43	0	0	344	0	124	263	387
IA	18	20	0	0	41	0	20	41	61
KS	7	5	0	0	0	0	5	0	5
KY	29	61	90	0	14,605	3	170	14,589	14,758
LA	46	41	74	0	331	215	256	404	661
ME	3	3	0	0	2	0	3	2	5
MD	5	2	0	0	0	0	2	0	2
MI	16	15	3	0	819	0	730	107	838
MN	19	80	1	0	81	0	81	81	162
MS	19	11	20	0	1,368	0	1,399	0	1,399
MO	25	25	0	0	0	0	25	0	25
MT	5	7	0	0	2	0	7	2	8
NE	12	4	1	0	0	0	5	0	5
NV	5	3	0	0	2	0	3	2	5
NJ	6	1	0	0	25	0	1	25	27
NM	3	3	0	0	0	0	3	0	3
NY	15	8	1	0	3	10	10	12	22
NC	23	85	8	0	8	0	100	0	101
ND	11	14	0	0	0	0	14	0	14
OH	31	26	1	0	755	0	752	31	782
OK	13	6	0	0	50	0	14	42	56
OR	11	2	0	0	7	0	4	5	9
PA	26	12	0	0	0	0	12	0	12
SC	24	12	5	0	0	0	17	0	17

Table 5-2. Releases to the Environment from Facilities that Produce, Process, or Use Dioxin and Dioxin-like Compounds[a]

	Reported amounts released in grams per year[b]								
							Total release		
State[c]	RF[d]	Air[e]	Water[f]	UI[g]	Land[h]	Other[i]	On-site[j]	Off-site[k]	On- and off-site
SD	3	11	0	0	0	0	11	0	11
TN	24	27	5	0	1,986	0	1,994	23	2,017
TX	65	281	917	209	36,247	0	8,310	29,345	37,655
UT	17	33	0	0	7,508	0	7,537	4	7,541
VA	13	6	2	0	16	0	10	14	24
WA	22	8	7	0	103	0	15	103	118
WV	15	13	3	0	30	0	16	30	46
WI	25	30	0	0	464	0	30	464	494
WY	9	17	0	0	0	0	17	0	17
GU	1	1	0	0	0	0	1	0	1
PR	1	2	0	0	0	0	2	0	2
VI	2	0	0	0	0	0	0	0	0
Total	799	1,067	1,295	209	72,093	230	29,136	45,756	74,893

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

RF = reporting facilities; UI = underground injection

Source: TRI21 2022 (Data are from 2021)

The key sources of CDD releases to air are from anthropogenic combustion processes and the production and use of chemicals contaminated with CDDs. In 2006, EPA published a report summarizing dioxin-like compound releases in the United States for 1987, 1995, and 2000 (EPA 2006). Quantitative results of the inventory are expressed in terms of grams TEQ. The annual releases to the U.S. environment over the 3 reference years were reported as 13,965 g TEQ in 1987, 3,444 g TEQ in 1995, and 1,422 g TEQ in 2000. This indicates that between 1987 and 2000, there was approximately a 90% reduction in the

releases of dioxin-like compounds to the environment of the United States from all known combined sources. For years 1987 and 1995, the leading source of emissions to the U.S. environment was municipal waste combustion; however, because of technology improvements, it dropped to the fourth ranked source by 2000. Burning of domestic refuse in backyard burn barrels remained fairly constant over the years, but in 2000, it emerged as the largest source of dioxin emissions to the U.S. environment (EPA 2006). In the 1980s, bleached chlorine pulp and paper mills were a significant source of emissions but were relatively minor by 2000 due to changes in bleaching practices. The top five sources of dioxin-like compound releases to the atmosphere in 2000 were reported as backyard barrel burning of refuse (498.5 g TEQ), medical waste incineration (378 g TEQ), municipal wastewater treatment sludge applied to land and incinerated (89.7 g TEQ), municipal waste combustion (83.8 g TEQ), and coal fired utility boilers for electric generating plants (69.5 g TEQ). The report concluded that reductions observed over this temporal period were attributed to source-specific regulations, improvements in source technology, advancements in the pollution control technologies specific to controlling dioxin discharges and releases, and the voluntary actions of U.S. industries to reduce or prevent dioxin releases. Dwyer and Themelis (2015) performed a similar analysis of emissions to the atmosphere for 2012 and concluded that nearly 90% of all U.S. total dioxin emissions arise from landfill fires, forest and brush fires, and backyard burning. It is likely that the train derailment and subsequent fire that occurred in February 2023 in East Palestine, Ohio, released CDDs and CDFs to the nearby atmosphere (EPA 2023); however, no studies are available that report atmospheric emissions of dioxins, and most early air sampling tests focused on levels of volatile organic compounds not CDDs. Full reports of EPA ordered testing are available at: https://www.epa.gov/east-palestine-oh-train-derailment/data-validation-reports.

CDDs are known trace contaminants of certain chlorinated industrial chemicals like chlorophenols (Buser 1987). CDDs can inadvertently form as byproducts during the manufacture of chlorophenols.

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 disease is caused by the larval form of the *Schistosoma* parasite is released by freshwater snails. A major contaminant of commercial PCP was identified as OCDD, which was shown to be present at concentrations between 500 and 1,500 mg/kg (ppm) (Dobbs and Grant 1979; Miller et al. 1989a). PCP is currently registered as a restricted-use pesticide for use as a wood preservative; however, EPA has scheduled a cancellation of all pesticide products containing PCP by February 28, 2027 (EPA 2021).

2,3,7,8-TCDD forms during the manufacture of 2,4,5-TCP. 2,4,5-TCP had 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-T. 2,4,5-T, in turn, was used in the production of a wide variety of herbicides including Silvex (2-[2,4,5-trichlorophenoxy]propionic acid) and Agent Orange (Hutzinger et al. 1985). 2,3,7,8-TCDD was an unintended contaminant of hexachlorophene, which was once used as a disinfectant, and contained <15 µg/kg (ppb) 2,3,7,8-TCDD (IARC 1977; Sine 1990). 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 contained CDDs (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 1987b). 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 1987b; 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-D (Josephson 1983). These herbicides were used extensively in silviculture for control of deciduous trees in conifer forests before their use was

discontinued (EPA 1987b). The use of Silvex, an herbicide closely related to 2,4,5-T, was discontinued in the United States in 1984 (Sine 1990).

5.3.2 Water

Estimated releases of 1,295 g (~2.85 pounds [<1 metric ton]) of dioxin compounds including CDDs to surface water from 799 domestic manufacturing and processing facilities in 2021, accounted for about <1% of the estimated total environmental releases from facilities required to report to the TRI (TRI21 2022). This estimate includes releases to wastewater treatment and publicly owned treatment works (POTWs). These releases are summarized in Table 5-2.

CDDs can enter water by a number of different mechanisms including urban runoff, combined sewer overflows (CSOs), and direct discharge by industrial facilities and 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; Pereira et al. 1985; Shear et al. 1996). Direct application or drift of 2,4,5-T or Silvex into water 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.

CDDs/CDFs, specifically 2,3,7,8-TCDD and 2,3,7,8-TCDF, were also present in effluent and sludges from pulp and paper mills that employed the bleached kraft process (Clement et al. 1989; EPA 1991a; Swanson et al. 1988). 2,3,7,8-TCDD was detected in seven of nine 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 wastewaters from four of five paper mills at levels ranging from not detected (<0.006 ppt) to 3.6 ppt (Amendola et al. 1989). Changes in the commercial bleaching process have significantly reduced the levels of CDDs/CDFs in paper products. The use of chlorine dioxide rather than elemental chlorine in the bleaching procedure essentially eliminates the formation of 2,3,7,8-TCDD and 2,3,7,8-TCDF in finished products and effluents (Axegård 2019).

5.3.3 Soil

Estimated releases of 72,093 g (~159 pounds [<1 metric ton]) of dioxin compounds including CDDs to soil from 799 domestic manufacturing and processing facilities in 2021, accounted for about 96% of the estimated total environmental releases from facilities required to report to the TRI (TRI21 2022). An

additional 230 g (~0.46 pounds [<1 metric ton]), constituting about <1% of the total environmental emissions, were released via underground injection (TRI21 2022). These releases are summarized in Table 5-2.

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 1991a). Atmospheric fall-out of CDD-laden particulates and gases appears to be the predominant source of CDDs to soil (Hutzinger et al. 1985).

In February of 2023, a large train derailment occurred in East Palestine, Ohio. The derailment and subsequent fire released CDDs, CDFs, and many other chemicals into nearby soils (EPA 2023; NTSB 2023). Monitoring data from this event are discussed in Section 5.5.3.

The commercial production of trichlorophenol, as well as various derivative products such as 2,4,5-T and other biocides, yielded large quantities of waste products containing substantial concentrations of CDDs; however, these substances are no longer used in the United States. 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 Times Beach, Missouri, which had been heavily contaminated, were 4.4–317 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; Mocarelli et al. 1988, 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 three 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 (Mocarelli et al. 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).

5.4 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, and 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.

5.4.1 Transport and Partitioning

Air. 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 (EPA 1991). However, because of the very low vapor pressure of CDDs, the amount present in the vapor phase generally is low 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 vapor pressure and atmospheric temperature (EPA 1991). Congeners with a 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-duration category.

Gas-particle partitioning of CDDs/CDFs and PCBs was studied in flue gases emitted from two municipal solid waste incinerators located in China. Total CDD/CDFs concentrations in the flue gas ranged from 0.75 to 15 ng m^{-3}, while in the particulate phase, they ranged from 0.14 to 8.1 ng m^{-3} (Han et al. 2017). Lee et al. (2018) studied the vapor-phase particulate-phase monitoring of CDD/CDFs in Taiwan. Since Taiwan is located mostly in the subtropical zone, with higher average temperatures than the United States, many of the CDD/CDFs were observed in the vapor phase. A study on ambient air in southern China found that, in general, during winter months, particulate-phase CDDs increased in fractions, but decreased in the summer months due to the increasing temperature (Tang et al. 2017). Additionally, higher chlorinated CDDs were associated with the particulate phase, while lower chlorinated congeners were predominantly in the vapor phase. Bi et al. (2020) found the total concentration of 17 CDD/CDFs in $PM_{2.5}$ (particles with aerodynamic diameter <2.5 μm) to range from 3.14 to 37.07 pg/m^3 in an industrial area of China.

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; Welsch-pausch et al. 1995). Precipitation (rain, sleet, snow) is very effective in removing particle-bound CDDs from the atmosphere (EPA 1991; Koester and Hites 1992a). Table 5-3 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 5-3 also summarizes the percentages of the congener groups scavenged as particles in rain rather than as dissolved solutes in rain. Total rain scavenging ratios were 10,000–150,000; HpCDDs and OCDD (the congeners most strongly associated with particulates) were the congeners scavenged most efficiently (EPA 1991; Koester and Hites 1992a).

Table 5-3. Rain Scavenging Ratios (RS) and Percent Washout Due to Particulates (%W) for CDDs and CDFs in Ambient Air in Two Midwest Cities

	Bloomington, Indiana		Indianapolis, Indiana	
Congener group	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

[a]Rarely detected; no calculations performed.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; HpCDD = heptachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

Sources: EPA 1991; Koester and Hites 1992a

Water. Volatilization from water surfaces may be an important environmental fate process for the lower chlorinated congeners but will be significantly slower for the higher chlorinated substances because these substances are more likely to adsorb to suspended solids and sediment in the water column, which attenuates the rate of volatilization. The estimated volatilization half-lives for a MCDD were about 15 hours from a model river and 12 days from a model lake estimated using the EPA software, Estimation Programs Interface Suite™ (EPI Suite™) (EPA 2012b). The estimated volatilization half-lives for OCDD were approximately 8 and 93 days from a model river and lake respectively; however, this does not account for adsorption to suspended particles and sediment, which will slow the rate of volatilization.

Experimentally measured bioconcentration factors (BCFs) for selected CDD congeners in various aquatic species are summarized in Table 5-4. 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 and including 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).

Table 5-4. 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
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 (*P. promelas*)	2,3,7,8-TCDD	71	Water	128,000	Cook et al. 1991

Table 5-4. Bioconcentration Factors (BCFs) for Aquatic Organisms

Organism	Congener	Exposure period (days)	Media	BCF	References
Fathead minnow (*P. promelas*)	2,3,7,8-TCDD		Water/ sediment	2,500	Tsushimoto et al. 1982
	2,3,7,8-TCDD		Water/ sediment	5,800	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

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

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 5-4). 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. 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 of 1,400–5,840 and 34–2,226 have been reported for fish exposed to 1,3,6,8-TCDD and OCDD, respectively (Muir et al. 1986a, 1986b). 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; the study authors suggested 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.

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). Estimated BAFs for MCDD through OCDD calculated using EPI Suite™ (EPA 2012b) are provided in Table 5-5.

Table 5-5. Estimated Upper Trophic Bioaccumulation Factors (BAFs) for MCDD Through OCDD

Congener	Log BAF
MCDD	2.9
DCDD	3.3
TrCDD	3.9
TCDD	6.1
PeCDD	5.7
HxCDD	4.7
HpCDD	4.8
OCDD	4.6

DCDD = dichlorodibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; MCDD = monochlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TrCDD = trichlorodibenzo-*p*-dioxin

Source: EPA 2012b

Several studies have examined 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.

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 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.

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).

Freshwater aquatic invertebrates have been shown to bioaccumulate CDDs/CDFs through water, sediment, and food pathways (Isensee 1978; Muir et al. 1985; Yockim et al. 1978). The range in

experimentally determined BCF values for freshwater invertebrates is presented in Table 5-4. 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. 1985). 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. 1985).

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 mollusks) 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 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 5-4) (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 accumulate 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 (BMFs, 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 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 were 5.3–20, 3.2–9.4, 10–20, 3.6–11, and 7.8–16 pg TEQ/g, respectively, for double-crested cormorant eggs, and 8.2–22, 3.3–6.4, 8.7–17, 2.4–6.0, and 9.7–21 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 were 5.7 pg TEQ/g 2,3,7,8-TCDD, 11 pg

TEQ/g 1,2,3,7,8-PeCDD, 2 pg TEQ/g 1,2,3,4,7,8-HxCDD, 11 pg TEQ/g 1,2,3,6,7,8-HxCDD, 1.3 pg TEQ/g 1,2,3,7,8,9-HxCDD, 3.8 pg TEQ/g 1,2,3,4,6,7,8-HpCDD, and 5.3 pg TEQ/g OCDD in eggs. 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 EROD activity in the areas of highest contamination. This indicates that the induction of CYP1A1 has occurred, and that the AhR-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 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, they have similar life histories, and the two colonies are in close proximity. 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 study authors suspected 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.

Sediment and Soil. 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 were 7.39–7.58 (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).

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 4 years since the Dioxin Research Facility was established (Yanders et al. 1989).

Estimated log K_{oc} values for MCDD through OCDD calculated using EPI Suite™ (EPA 2012b) are provided in Table 5-6. The first method reports the estimation using a molecular connectivity index (MCI) method and the second value is an estimation using a correlation with the log K_{ow}.

Table 5-6. Estimated Log K_{oc} for MCDD through OCDD

CDD	Log K_{oc} (MCI)	Log K_{oc} (K_{ow} QSAR)
MCDD	4.4	4.1
DCDD	4.6	4.9

Table 5-6. Estimated Log K_{oc} for MCDD through OCDD

CDD	Log K_{oc} (MCI)	Log K_{oc} (K_{ow} QSAR)
TrCDD	5.2	4.5
TCDD	5.4	4.8
PeCDD	5.6	4.7
HxCDD	5.8	5.6
HpCDD	6.1	5.5
OCDD	6.3	5.6

CDD = chlorinated dibenzo-*p*-dioxin; DCDD = dichlorodibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; MCDD = monochlorodibenzo-*p*-dioxin; MCI = molecular connectivity index; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; QSAR = quantitative structure-activity relationship; TCDD = tetrachlorodibenzo-*p*-dioxin; TrCDD = trichlorodibenzo-*p*-dioxin

Source: EPA 2012b

Other Media. 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 (Facchetti 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 (Facchetti et al. 1986). Using two soils with differing organic matter content, it was shown that for both zucchini and pumpkin, uptake of CDDs by the root and translocation to the shoots and fruit were important mechanisms and may explain why fruits in the *Cucurbita* genus tend to have higher levels of CDDs than other fruits (Hülster et al. 1994). Inui et al. (2008, 2011) also studied the uptake of CDDs in three different zucchini cultivars and found accumulation to be significantly higher in the black beauty and gold rush variety as compared to the patty green cultivar.

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.

Müller et al. (1993) studied transfer pathways of CDD/CDFs to fruit. The study 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 were 1–4 ng/kg (fresh weight) and were 4–8 times higher in the peel than in the pulp. The study authors suggested that airborne CDDs/CDFs are a major source of contamination of fruits grown in contaminated soil. Müller 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 3 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 that 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 profile 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 study 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 study 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.

5.4.2 Transformation and Degradation

Air. CDDs slowly degrade in the atmosphere by reacting with photochemically produced hydroxyl radicals. Using the gas-phase hydroxyl radical reaction rate constants and an average 12-hour daytime hydroxyl radical concentration of 1.5×10^6 molecules cm^{-3}, the atmospheric vapor phase lifetimes of CDDs are estimated to range from about 0.5 days for MCDD to 9.6 days for OCDD, with TCDD having a lifetime of 0.8–2 days (Atkinson 1991). Particulate-phase CDDs have been shown to have much longer atmospheric half-lives as compared to the vapor phase CDDs (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 and slower in winter).

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 study 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 (1992b) 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 study 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).

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 UV 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, Choudhry 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 ranged 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). The study 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 were reported as 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 and 318.6 nm were 72.6 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-lives of 2,3,7,8-TCDD in isooctane were 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 was observed after 20 hours of exposure in 1-octanol (Stehl et al. 1973). Irradiation of 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.

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 were 153–1,388 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 were 121–560 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 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 (EPA 1982; Miller et al. 1987).

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. 1989b). 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. 1989b).

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 study 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). 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 12, 35, or 54 weeks (Philippi et al. 1982). Results showed the occurrence of a polar metabolite of [^{14}C]-2,3,7,8-TCDD, which 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°C 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 study 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 study 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.

5.5 LEVELS 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. Concentrations of CDDs in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on 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 and that every measurement is accompanied with a certain analytical error.

Table 5-7 shows the lowest limit of detections that are achieved by analytical analysis in environmental media. An overview summary of the range of concentrations detected in environmental media is presented in Table 5-8.

Table 5-7. Lowest Limit of Detection Based on Standards[a]

Media	Detection limit	Reference
Air	0.0003 fg/m^3 (2,3,7,8-TCDD)[b]	Friedman et al. 2012
Drinking water	10 pg/L (ppq) (2,3,7,8-TCDD)[c]	EPA 2007a (Method 8290)
Surface water and groundwater	0.3 fg/L (2,3,7,8-TCDD)	Friedman et al. 2012
Soil	0.2 pg/g (ppt) (2,3,7,8-TCDD)	Nestrick et al. 1986
Sediment	1 pg/g (ppt) (2,3,7,8-TCDD)[c]	EPA 2007a (Method 8290)
Whole blood	1.25 pg/L (ppq) (2,3,7,8-TCDD); 3.8 pg/g lipid basis	CDC 2024a; Patterson et al. 1987

[a]Detection limits based on using appropriate sample mass/volume, preparation and analytics. These limits may not be possible in all situations.
[b]Detection limits in air are dependent upon the sampling time/sampling volume. Typical detection limits are in the pg/m^3 range; however, this study had extended sampling times and large volume collections (>150 m^3) ensuring very low detection limits.
[c]The detection limits and quantitation levels in this method are usually dependent on the level of interferences rather than instrumental limitations.

ppq = parts per quadrillion; ppt = parts per trillion; TCDD = tetrachlorodibenzo-*p*-dioxin

Table 5-8. Summary of Environmental Levels of Chlorinated Dibenzo-*p*-Dioxins

Media	Low	High	For more information
Outdoor air (pg/m^3)	<LOD	24	Section 5.5.1
Indoor air (pg/m^3)	<LOD	131.5	Section 5.5.1
Surface water (pg/L)	<LOD	20	Section 5.5.2
Groundwater (pg/L)	<LOD	3,900,000	Section 5.5.2
Drinking water (pg/L)	<LOD	230	Section 5.5.2
Food (pg/g)	<LOD	65	Section 5.5.4
Soil (pg/g)	<LOD	$2x10^9$	Section 5.5.3

LOD = limit of detection

Detections of CDDs in air, water, and soil at NPL sites are summarized in Table 5-9.

Table 5-9. Chlorinated Dibenzo-*p*-Dioxin Levels in Water, Soil, and Air of National Priorities List (NPL) Sites

Medium	Median[a]	Geometric mean[a]	Geometric standard deviation[a]	Number of quantitative measurements	NPL sites
2,3,7,8-TCDD					
Water (ppb)	7.05×10^{-4}	6.37×10^{-4}	401	8	5
Soil (ppb)	6.2	12.5	49.4	95	56
Air (ppbv)			No data		
TCDD[b]					
Water (ppb)			No data		
Soil (ppb)	3.5	6.25	35.1	21	11
Air (ppbv)			No data		
1,2,3,7,8-PeCDD					
Water (ppb)			No data		
Soil (ppb)	1.1	0.369	58.3	5	4
Air (ppbv)			No data		
PeCDD[b]					
Water (ppb)			No data		
Soil (ppb)	3.1	2.97	29.1	17	10
Air (ppbv)			No data		
1,2,3,4,7,8-HxCDD					
Water (ppb)			No data		
Soil (ppb)	4	1.56	38.4	7	6
Air (ppbv)			No data		
1,2,3,6,7,8-HxCDD					
Water (ppb)			No data		
Soil (ppb)	26	4.14	74.5	5	5
Air (ppbv)			No data		
1,2,3,7,8,9-HxCDD					
Water (ppb)			No data		
Soil (ppb)	10.4	3.55	38.6	6	5
Air (ppbv)			No data		
HxCDD[b]					
Water (ppb)			No data		
Soil (ppb)	7.8	10.6	26	28	17
Air (ppbv)			No data		
1,2,3,4,6,7,8-HpCDD					
Water (ppb)			No data		
Soil (ppb)	22.6	36.4	56.7	10	9
Air (ppbv)			No data		

Table 5-9. Chlorinated Dibenzo-*p*-Dioxin Levels in Water, Soil, and Air of National Priorities List (NPL) Sites

Medium	Median[a]	Geometric mean[a]	Geometric standard deviation[a]	Number of quantitative measurements	NPL sites
HpCDD[b]					
Water (ppb)	2.5	2.62	1,380	7	4
Soil (ppb)	4.9	10.6	45.4	36	20
Air (ppbv)			No data		
OCDD					
Water (ppb)	4.57	2.63	2,660	8	5
Soil (ppb)	21	77.7	64.2	47	29
Air (ppbv)			No data		

[a]Concentrations found in ATSDR site documents from 1981 to 2022 for 1,868 NPL sites (ATSDR 2022). Pathways do not necessarily involve exposure or levels of concern.
[b]Refers to summation of the other isomers in the homologues instead of the specified isomer.

5.5.1 Air

The National Dioxin Air Monitoring Network (NDAMN) was established by the EPA in 1998 to determine background air concentrations of CDDs, CDFs, and dioxin-like PCBs in the United States (EPA 2013). Congener-specific data from June 1998 through November 2004 at 34 NDAMN stations (4 urban stations, 23 rural stations, and 7 remote stations) throughout the United States are shown in Table 5-10. Large sampling times and large volumes of collected air guaranteed low detection limits and a high detection frequency. The maximum concentration of 23,953 fg/m^3 (23.953 pg/m^3) was observed for OCDD.

Table 5-10. Congener-specific Monitoring Data from the National Dioxin Air Monitoring Network 1998–2004

Congener	Detection frequency (%)	Mean (fg/m^3)	SD (fg/m^3)
2,3,7,8-TCDD	85	0.6	1.2
1,2,3,7,8-PeCDD	89	3.1	5.9
1,2,3,4,7,8-HxCDD	94	4.2	10.4
1,2,3,6,7,8-HxCDD	97	7.3	15.3
1,2,3,7,8,9-HxCDD	96	7.2	15.3
1,2,3,4,6,7,8-HpCDD	100	102.3	234.6

Table 5-10. Congener-specific Monitoring Data from the National Dioxin Air Monitoring Network 1998–2004

Congener	Detection frequency (%)	Mean (fg/m^3)	SD (fg/m^3)
OCDD	100	352.8	973.4

HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; SD = standard deviation; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: EPA 2013

High levels of CDDs and CDFs were predicted to have arisen following the terrorist attacks at the World Trade Center (WTC) complex in New York City on September 11, 2001 (Rayne et al. 2005). Predicted gas-phase concentrations in Manhattan 6 weeks after the attack were estimated to be as high as 822 fg/m^3 (0.822 pg/m^3) for 2,3,7,8-TCDD at Church and Warren Streets. This location also had the highest predicted combined CDF/CDD TEQ of 2,730 fg/m^3 (2.730 pg/m^3).

Monitoring data in the vicinity of the Passaic River and Newark Bay New Jersey from May 2008 to August 2009, measured vapor-phase concentrations of mono- to octaCDDs (Friedman et al. 2012). Lower chlorinated congeners (2,7-/2,8-DCDD) were detected and likely resulted from photochemical conversion of triclosan in Newark Bay. The highest concentration of these congeners was about 7 pg/m^3. 2,4,7-TrCDD was also detected in atmospheric samples at levels up to 1 pg/m^3. Other higher chlorinated congeners were not detected in vapor-phase air samples.

Lin et al. (2010) studied atmospheric levels of CDDs and CDFs in the air of Taiwan in the vicinity of water treatment facilities. Average atmospheric levels in pg/m^3 were as follows: 2,3,7,8-TCDD, 0.009; 1,2,3,7,8-PeCDD, 0.043; 1,2,3,4,7,8-HxCDD, 0.062; 1,2,3,6,7,8-HxCDD, 0.144; 1,2,3,7,8,9-HxCDD, 0.112; 1,2,3,4,6,7,8-HpCDD, 1.86; and OCDD, 6.06. Levels were consistently higher in the spring as compared to summer, fall, and winter months.

As part of the Stockholm Convention on Persistent Organic Pollutants (POPs) Global Monitoring Plan (GMP), a study was conducted between 2017 and 2019 to monitor dioxin-like POPs in developing countries (Abad et al. 2022). The results were expressed as TEQ and included 195 measurements from 42 developing nations. The findings indicated that there was a noticeable downward trend for CDD/CDF TEQs only in Latin American nations and that the highest levels were determined to be in Asian nations. Results from a GMP study conducted in Brazil showed that mass concentrations of CDDs/CDFs in Sao Paulo declined approximately 50% from 0.0265 to 0.0133 pg/m^3 from 2010 to 2015 (Hu et al. 2019).

Concentrations of individual CDDs were typically <0.01 pg/m^3. Similar monitoring studies were conducted in a rural area of Mexico (Sinaloa) from 2016 to 2018 as part of the GMP (Valenzuela et al. 2022). Ten CDFs and seven CDD congeners were monitored in the ambient air. The predominant CDDs detected were 1,2,3,4,6,7,8,9-OCDD with concentrations of 0.154–0.164 pg/m^3 and 1,2,3,4,6,7,8-HpCDD with concentrations of 0.044–0.048 pg/m^3.

Table 5-11 provides additional air concentrations of CDDs in indoor air, outdoor air, and over oceans.

Table 5-11. Concentrations of CDDs in Ambient Indoor and Outdoor Air in North America and Oceans

Site	Sampling year	CDD	Concentration (pg/m^3)	Reference
Binghampton, New York	1985	2,3,7,8-TCDD	0.23–0.47	Smith et al. 1986
Binghampton, New York	1985	Total TCDD	1.0–1.3	Smith et al. 1986
Chicago, Illinois (outdoor)	2004–2007	ΣCDDs/CDFs	1.3±0.10	Venier et al. 2009
Eagle Harbour, Michigan (outdoor)	2004–2007	ΣCDDs/CDFs	0.12±0.013	Venier et al. 2009
Sturgeon Point, New York	2004–2007	ΣCDDs/CDFs	0.74±0.083	Venier et al. 2009
Sleeping Bear Dunes, (outdoor) Michigan	2004–2007	ΣCDDs/CDFs	0.40±0.093	Venier et al. 2009
Calcasieu Parish, Louisiana outdoor)	2001–2002	ΣCDDs/CDFs	0.0027–0.0924	Gibbs et al. 2003
Minneapolis-St. Paul (outdoor, winter)	1988	2,3,7,8-TCDD	0.015–0.019	Reed et al. 1990
Minneapolis-St. Paul (outdoor, winter)	1988	Total HpCDD	0.5–4.1	Reed et al. 1990
Minneapolis-St. Paul (outdoor, winter)	1988	OCDD	0.74–8.2	Reed et al. 1990
Minneapolis-St. Paul (outdoor, summer)	1988	Total HpCDD	0.204–0.246	Reed et al. 1990
Minneapolis-St. Paul (outdoor, summer)	1988	OCDD	0.018–0.024	Reed et al. 1990
Bloomington, Iowa	1985–1988	Total TCDD	0.0013 (vapor) 0.0002 (particulate)	Eitzer and Hites 1989b
Bloomington, Iowa	1985–1988	Total PeCDD	0.026 (vapor) 0.013 (particulate)	Eitzer and Hites 1989b
Bloomington, Iowa	1985–1988	Total HxCDD	0.033 (vapor) 0.115 (particulate)	Eitzer and Hites 1989b

Table 5-11. Concentrations of CDDs in Ambient Indoor and Outdoor Air in North America and Oceans

Site	Sampling year	CDD	Concentration (pg/m^3)	Reference
Bloomington, Iowa	1985–1988	Total HpCDD	0.0058 (vapor) 0.065 (particulate)	Eitzer and Hites 1989b
Joint Base Balad in Iraq	2007	2,3,7,8-TCDD	0.06 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	1,2,3,7,8-PeCDD	0.15 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	1,2,3,4,7,8-HxCDD	0.12 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	1,2,3,6,7,8-HxCDD	0.26 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	1,2,3,7,8,9-HxCDD	0.48 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	1,2,3,4,6,7,8-HpCDD	1.27 (average all sites)	Masiol et al. 2016
Joint Base Balad in Iraq	2007	OCDD	1.43 (average all sites)	Masiol et al. 2016
North Atlantic	2010–2011	ΣCDDs[a]	0.011 (gas) 0.0095 (aerosol)	Morales et al. 2014
South Atlantic	2010–2011	ΣCDDs[a]	0.013 (gas) 0.040 (aerosol)	Morales et al. 2014
Indian Ocean	2010–2011	ΣCDDs[a]	0.012 (gas) 0.023 (aerosol)	Morales et al. 2014
South Pacific	2010–2011	ΣCDDs[a]	0.0065 (gas) 0.010 (aerosol)	Morales et al. 2014
North Pacific	2010–2011	ΣCDDs[a]	0.012 (gas) 0.0081 (aerosol)	Morales et al. 2014
Global	2010–2011	ΣCDDs[a]	0.011 (gas) 0.020 (aerosol)	Morales et al. 2014

[a]Sum of TCDD, PeCDD, HxCDD, HpCDD, and OCDD congeners.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorodibenzofuran; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

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. 1989). The wood-preserving formulation contained PCP, which was 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. 1989).

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 CDDs/CDFs were 1.46–4.27 pg/m^3, while measured outdoor air concentrations were 0.61–78.97 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 tetrachlorobenzenes 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. 1986). 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. 1986).

Background levels of CDDs 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 were 0.005–0.065 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 were 0.005–0.01 pg/m^3 (0.0004–0.0007 ppq). Wintertime CDD concentrations were greater than those observed for summertime. The study 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 were approximately 0.5–4.1 pg/m^3 (0.029–0.236 ppq) and 0.74–8.2 pg/m^3 (0.039–0.436 ppq), respectively (Reed et al. 1990). Average summertime ambient air concentrations of HpCDD and OCDD were approximately 0.204–0.246 pg/m^3 (0.011–0.014 ppq) and 0.018–0.024 pg/m^3 (0.001–0.0013 ppq), respectively (Reed et al. 1990). The study authors found that, in general, the more highly chlorinated congeners were present at higher concentrations than the less chlorinated congeners.

A long-term study (1985–1988) of CDDs in the ambient atmosphere of Bloomington, Indiana (a suburban area), was carried out to provide baseline 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 showed 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 [$5.3x10^{-5}$ 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 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).

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 seven sampling sessions at a mean concentration of 1.140 pg/m^3. OCDD also was found at all seven 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 three 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. 1989). 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 polyvinyl chloride (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 m 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. 1989).

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, and 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).

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 particulate-phase urban air with OCDD being the most prevalent congener. Concentrations of all CDDs are highest in the air near industrial areas or other point sources such as open burn pits. 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.

5.5.2 Water

The Water Quality Portal is a tool of publicly available water-quality data from the U.S. Geological Survey (USGS), EPA, and over 400 state, federal, tribal, and local agencies. Data from 2020–2021 showed that no CDD congeners were detected in either surface water or groundwater measurements (WQP 2022). Typically, surface water levels of CDDs are near or below detection limits unless there is a nearby emission source.

Khairy and Lohmann (2020a, 2020b) measured levels of CDDs and CDFs in porewater at four locations in the lower Passaic River, New Jersey. Due to industrial activities, this area is historically known for its contamination with PCBs and CDDs/CDFs. The data from this study are summarized in Table 5-12. Porewater concentrations of CDDs (pg/L) at four locations of the lower Passaic River were obtained during four sampling periods.

Table 5-12. Porewater Concentrations of CDDs (pg/L) at Four Locations of the Lower Passaic River Obtained During Four Sampling Periods

Congener	06/2015–08/2015	08/2015–10/2015	10/2015–12/2015	12/2015–02/2016
River Bank Park, Lower Passaic River				
2-MCDD	<LOD	<LOD	<LOD	<LOD
2,7-/2,8-DCDD	5.36	4.92	4.24	4.87
1,2,4-TrCDD	0.21	0.27	0.26	0.29
1,3,6,8-TCDD	0.12	0.10	0.13	0.16
1,3,7,8-TCDD	<LOD	<LOD	<LOD	<LOD
2,3,7,8-TCDD	0.14	0.14	0.11	0.11
1,2,8,9-TCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4,7-PeCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4,7,8-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,6,7,8-HxCDD	0.03	0.03	0.04	0.03
1,2,3,7,8,9-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4, 6,7,8-HpCDD	0.05	0.05	0.07	0.07
1,2,3,4,6,7,8,9-OCDD	0.11	0.11	0.09	0.10

Table 5-12. Porewater Concentrations of CDDs (pg/L) at Four Locations of the Lower Passaic River Obtained During Four Sampling Periods

Congener	06/2015–08/2015	08/2015–10/2015	10/2015–12/2015	12/2015–02/2016
Bridge Street, Passaic River				
2-MCDD	<LOD	<LOD	NA	NA
2,7-/2,8-DCDD	4.341	4.589	NA	NA
1,2,4-TrCDD	0.315	0.307	NA	NA
1,3,6,8-TCDD	0.098	0.096	NA	NA
1,3,7,8-TCDD	0.042	0.031	NA	NA
2,3,7,8-TCDD	0.150	0.161	NA	NA
1,2,8,9-TCDD	<LOD	<LOD	NA	NA
1,2,3,4,7-PeCDD	<LOD	<LOD	NA	NA
1,2,3,4,7,8-HxCDD	<LOD	<LOD	NA	NA
1,2,3,6,7,8-HxCDD	<LOD	<LOD	NA	NA
1,2,3,7,8,9-HxCDD	<LOD	<LOD	NA	NA
1,2,3,4,6,7,8-HpCDD	0.026	0.020	NA	NA
1,2,3,4,6,7,8,9-OCDD	0.017	0.019	NA	NA
Doremus Street, Passaic River				
2-MCDD	<LOD	<LOD	<LOD	<LOD
2,7-/2,8-DCDD	6.38	5.74	5.47	4.68
1,2,4-TrCDD	0.234	0.262	0.277	0.230
1,3,6,8-TCDD	0.113	0.108	0.134	0.135
1,3,7,8-TCDD	<LOD	<LOD	<LOD	<LOD
2,3,7,8-TCDD	0.146	0.156	0.139	0.115
1,2,8,9-TCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4,7,8-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,6,7,8-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,7,8,9-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4,6,7,8-HpCDD	0.023	0.031	0.028	0.032
1,2,3,4,6,7,8,9-OCDD	0.020	0.024	0.024	0.026
Passaic Ave, Passaic River				
2-MCDD	<LOD	<LOD	<LOD	<LOD
2,7-/2,8-DCDD	8.36	7.48	5.90	5.93
1,2,4-TrCDD	0.28	0.23	0.19	0.23
1,3,6,8-TCDD	0.14	0.13	0.10	0.09
1,3,7,8-TCDD	0.07	0.07	0.07	0.07
2,3,7,8-TCDD	0.15	0.15	0.12	0.11
1,2,8,9-TCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,4,7,8-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,6,7,8-HxCDD	<LOD	<LOD	<LOD	<LOD
1,2,3,7,8,9-HxCDD	<LOD	<LOD	<LOD	<LOD

Table 5-12. Porewater Concentrations of CDDs (pg/L) at Four Locations of the Lower Passaic River Obtained During Four Sampling Periods

Congener	06/2015–08/2015	08/2015–10/2015	10/2015–12/2015	12/2015–02/2016
1,2,3,4,6,7,8-HpCDD	0.05	0.05	0.04	0.03
1,2,3,4,6,7,8,9-OCDD	0.03	0.03	0.03	0.03

CDD = chlorinated dibenzo-*p*-dioxin; DCDD = dichlorodibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection; MCDD = monochlorodibenzo-*p*-dioxin; NA = not applicable; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TrCDD = trichlorodibenzo-*p*-dioxin

Source: Khairy and Lohmann 2020b

A second monitoring study in the vicinity of the Passaic River and Newark Bay New Jersey from May 2008 to August 2009, measured mono- to octaCDD congeners in surface and bottom waters (Friedman et al. 2012). Measured concentrations were generally low with the highest measured concentration observed for the 2,7-/2,8-DCDD congeners, which were ≤20 pg/L. Dissolved concentrations for most congeners did not vary between location, depth, or sampling period. The maximum 2,3,7,8-TCDD concentration was 0.023 pg/L and OCDD was never detected. Previous monitoring results from the late 1990s to early 2000s observed levels of 2,3,7,8-TCDD ranging from 0.036 to 0.120 pg/L and OCDD concentrations ranging from 0.200 to 0.350 pg/L.

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 (pg/L). OCDD concentrations were found in three samples in the 60–1,200 ppq (pg/L) range. Lower concentrations of HpCDD (70 ppq [pg/L]) were also found (Tashiro et al. 1989a). Precipitation samples were also collected in 1987–1988 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 two of the urban precipitation samples at concentrations of 33 and 15 ppq (pg/L) (Tashiro et al. 1989b). Rain collected at Bloomington, Indiana, 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 0.1–220 pg/L. Total TCDD was detected in only 3 of 28 samples at concentrations <9 pg/L (EPA 1991c).

Lin et al. (2010) studied concentrations of CDDs and CDFs in drinking water in Taiwan to better understand how atmospheric deposition influence these concentrations. Tap water levels (averaged at three different plants) in pg/L were as follows: 2,3,7,8-TCDD, 0.0001–0.005; 1,2,3,7,8-PeCDD, 0.0002–0.0006; 1,2,3,7,8-PeCDD, 0.0001–0.0006; 1,2,3,7,8-PeCDD, 0.0002–0.0013; 1,2,3,7,8,9-HxCDD, 0.0002–0.0010; 1,2,3,4,6,7,8-HpCDD, 0.0022- 0.0088; and OCDD, 0.0139–0.0416. The study authors found tap water levels for total CDDs/CDFs to be approximately 55% less than levels in source water and that atmospheric deposition to uncovered water treatment facilities likely increased the levels in finished water.

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, New York, facility contained concentrations of TCDDs (1.7 ppq [pg/L]) as well as concentrations of TCDFs to OCDFs (18, 27, 85, 210, and 230 ppq [pg/L], 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.

Groundwater in the vicinity of an abandoned wood treatment facility was sampled from monitoring wells constructed at depths of 6.1–30.5 m 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 m were 61, 1,500, and 3,900 ppt, (61,000, 1,500,000, and 3,900,00 pg/L), respectively. The study authors noted that the high concentrations of CDDs in the sample from a depth of 6.1 m 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 m) contained HxCDD, HpCDD, and OCDD at concentration ranges of not detected to 21 ppt (21,000 pg/L), not detected to 34 ppt (34,000 pg/L), and not detected to 539 ppt (539,000 pg/L), respectively (Pereira et al. 1985).

In conclusion, CDDs are rarely detected in drinking water at ppq levels or higher. Raw water samples generally have higher concentrations of CDDs than finished drinking water samples because conventional water treatment processes remove the CDDs along with the particulates from raw water. In groundwater

samples collected near industrial sites, CDDs have been detected at concentrations up to 3,900 ppt (3,900,000 pg/L).

5.5.3 Sediment and Soil

Following the train derailment and subsequent fire that occurred February 3, 2023, in East Palestine, Ohio, testing began on soil samples collected in the affected area at various sampling depths. Sampling data from March of 2023 showed soil levels of CDD congeners often >1,000 ppt. Comprehensive data are available from the EPA website: https://www.epa.gov/east-palestine-oh-train-derailment/epa-residential-commercial-and-agricultural-soil-sampling#summary. Table 5-13 shows residential, commercial, and agricultural soil sampling data collected by Norfolk Southern for a surface soil (depth 0.0–0.1 feet) on March 12, 2023 (EPA 2023).

Table 5-13. CDD Levels in a Soil Sample Taken from a Sampling Location in East Palestine, Ohio, March 12, 2023

CDD congener	Soil levels ppt (pg/g)
1,2,3,4,6,7,8-HpCDD	2,600
1,2,3,4,7,8-HxCDD	37
1,2,3,6,7,8-HxCDD	99
1,2,3,7,8,9-HxCDD	62
1,2,3,7,8-PeCDD	17
2,3,7,8-TCDD	2.3
OCDD	27,000

CDD = chlorinated dibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection; NA = not applicable; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: EPA 2023

As part of a National Dioxin Study, EPA conducted a 2-year nationwide monitoring program to assess the extent of 2,3,7,8-TCDD contamination (EPA 1987c). 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 (2×10^9 ppt). 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 >1 ppt with one detection at 6 ppb (6,000 ppt). Two-thirds of all detections at the Tier 5 sites were <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 1987c).

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 were 62–640 ppt, while levels of total HxCDD were 12–99 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 2 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 5-14) (Nestrick et al. 1986). Many of the samples were taken within 1 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, not

detected to 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 OCDF 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 5-14. 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (2,3,7,8-TCDD) Levels Measured in Soil Samples Collected in 1984 from Industrialized Areas of U.S. Cities

Sample location	2,3,7,8-TCDD (ppt)
Lansing, Michigan	3 (0.7)[a] ND (0.8)
Gaylord, Michigan	ND (0.2)
Detroit, Michigan	3.6 (0.7) 2.1 (0.4)
Chicago, Illinois	9.4
Middletown, Ohio	ND (0.3) ND (0.3)
Barberton, Ohio	5.6
Akron, Ohio	6.3
Nashville, Tennessee	0.8 (0.3)
Pittsburgh, Pennsylvania	2.6 (0.5)
Marcus Hook, Pennsylvania	0.4 (0.3)
Philadelphia, Pennsylvania	0.9 (0.3)
Clifton Heights, Pennsylvania	ND (0.4)
Brooklyn, New York	2.6 (0.4)
South Charleston, West Virginia	ND (0.4)

Table 5-14. 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (2,3,7,8-TCDD) Levels Measured in Soil Samples Collected in 1984 from Industrialized Areas of U.S. Cities

Sample location	2,3,7,8-TCDD (ppt)
Arlington, Virginia	ND (0.4)
Newport News, Virginia	0.4 (0.3)

[a]Values in parentheses show the detection limit, 2.5 times noise, when the experimental result is <10 times the measured detection limit.

ND = not detected; ppt = parts per trillion

Source: Nestrick et al. 1986

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.

Levels of CDD congeners were monitored in sediment at four locations in the lower Passaic River, New Jersey during a monitoring study conducted in July 2015 (Khairy and Lohmann 2020a, 2020b). Due to industrial activities, this area is historically known for its contamination with PCBs and CDDs/CDFs. Ranges of values in ppt (pg/g) were reported as: 2-MCDD <LOD–2.0; 2,7,2,8-DCDD 38.5–308; 1,2,4-TtrCDD 5.3–29.0; 1,3,6,8-TCDD 1.9–39.2; 1,3,7,8-TCDD, 2.2–8.9; 2,3,7,8-TCDD 43.7–170.7; 1,2,3,4,7-PeCDD 0.9–2.8; 1,2,3,4,7,8-HxCDD 6.9–18.0; 1,2,3,6,7,8-HxCDD <LOD–38.2; 1,2,3,7,8,9-HxCDD 3.1–6.6; 1,2,3,4,6,7,8-HpCDD 92.0–229.3; and 1,2,3,4,6,7,8,9-OCDD 1,100.1–2,792.1.

Sediment samples collected in 1985–1986 from estuarine areas (Passaic River and Newark Bay), near a Newark, New Jersey, 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. 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).

Ehrlich et al. (1994) identified the relative contributions of various sources of CDDs/CDFs to deposited sediments of Newark Bay using polytopic vector analysis, a multivariate statistical technique, and monitoring data collected from 1991 to 1993 at 62 sampling locations. The study 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. 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 1987c). 2,3,7,8-TCDD sediment concentrations ranged from below the detection limit (22 ppt) to 21,000 ppt, whereas OCDD concentrations were 3.1–42,000 ppt, although other sources of OCDD were thought to contribute to the elevated levels of OCDD (Bopp et al. 1991; Wenning et al. 1992). Maximum levels of CDDs from this monitoring study conducted from December 1991 to March 1993 are approximately an order of magnitude greater than the levels reported by Khairy and Lohmann (2020a, 2020b) during a monitoring study conducted in 2015.

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 study authors suggested 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 of 560–4,800 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 TCDFs (range of geometric mean, 2.4–6,223 ppt) and OCDD (range of geometric mean, 12–250 ppt) congeners, 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.

Biosolids obtained from wastewater or sewage treatment facilities are applied to agricultural lands to add nutrients to the soils used for commercial farming applications. CDDs were detected in biosolids collected in 32 U.S. states and the District of Columbia from 94 wastewater treatment plants by the EPA in its 2001 national sewage sludge survey (EPA 2007b). Minimum levels of CDDs ranged from about 0.1 (2,3,7,8-TCDD) to 1 ng/kg (OCDD).

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).

5.5.4 Other Media

Foods. The FDA 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 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 1983–1984, respectively, contained 1,2,3,4,6,7,8-HpCDD concentrations of 21–588 ppt and OCDD concentrations of 80–1,610 ppt. These residues were attributed to local PCP contamination problems in these areas (Firestone et al. 1986). Milk samples contaminated with PCP at levels of 0.01–0.05 μg/g PCP contained no detectable CDDs.

The most recent FDA market basket analysis for CDDs and CDFs was the 2004 study in which more than 200 different food types were collected and analyzed for 17 different CDD or CDF congeners obtained from commercial supermarkets located in Boston, Massachusetts; Syracuse, New York; and Pittsburgh, Pennsylvania (FDA 2006). The entire data set for the years 2000–2004 may be obtained from the FDA website at: https://www.fda.gov/food/process-contaminants-food/dioxin-analysis-resultsexposure-estimates. The highest detected level was for OCDD (65 pg/g) in a sample of liver (beef/calf), which is orders of magnitude lower than OCDD residues in beef livers from previous surveys. The entire data output for any given year is too large to be reproduced in this document; however, results for food items

collected in the 2004 market basket survey in which there was a specific detected amount are provided in Table 5-15 (FDA 2007).

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Liver (beef/calf), pan-cooked with oil	1,2,3,4,6,7,8-HpCDD	6.8	0.006
Beef roast, chuck, oven-roasted	1,2,3,4,6,7,8-HpCDD	5.1	0.003
Frankfurter (beef/pork), boiled	1,2,3,4,6,7,8-HpCDD	2.9	0.003
Beef, ground, regular, pan-cooked	1,2,3,4,6,7,8-HpCDD	2.3	0.002
Mushrooms, raw	1,2,3,4,6,7,8-HpCDD	2	0.009
Salami, luncheon-meat type (not hard)	1,2,3,4,6,7,8-HpCDD	1.3	0.001
Catfish, pan-cooked with oil	1,2,3,4,6,7,8-HpCDD	1.3	0.005
Meatloaf, beef, homemade	1,2,3,4,6,7,8-HpCDD	1.2	0.001
Butter, regular (salted)	1,2,3,4,6,7,8-HpCDD	1.2	0.003
Beef steak, loin/sirloin, broiled	1,2,3,4,6,7,8-HpCDD	1	0.001
Burrito with beef, beans, and cheese, from Mexican carry-out	1,2,3,4,6,7,8-HpCDD	0.92	0.001
Chili con carne with beans, canned	1,2,3,4,6,7,8-HpCDD	0.86	0.002
Cream cheese	1,2,3,4,6,7,8-HpCDD	0.78	0.0043
Cheese, cheddar, natural (sharp/mild)	1,2,3,4,6,7,8-HpCDD	0.75	0.002
Baby food, vegetables and beef	1,2,3,4,6,7,8-HpCDD	0.74	0.003
Pork sausage (link/patty), oven-cooked	1,2,3,4,6,7,8-HpCDD	0.68	0.002
Quarter-pound hamburger on bun, fast-food	1,2,3,4,6,7,8-HpCDD	0.61	0.001
Quarter-pound cheeseburger on bun, fast-food	1,2,3,4,6,7,8-HpCDD	0.52	0.003
Cheese, Swiss, natural	1,2,3,4,6,7,8-HpCDD	0.49	0.003
Green beans, canned	1,2,3,4,6,7,8-HpCDD	0.47	0.006
Margarine, regular (salted)	1,2,3,4,6,7,8-HpCDD	0.47	0.002
Cheese, American, processed	1,2,3,4,6,7,8-HpCDD	0.43	0.002
Baby food, turkey and rice	1,2,3,4,6,7,8-HpCDD	0.43	0.003
Taco/tostada with beef and cheese, from Mexican carry-out	1,2,3,4,6,7,8-HpCDD	0.42	0.004

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Bologna (beef/pork)	1,2,3,4,6,7,8-HpCDD	0.41	0.001
Spaghetti with meat sauce, homemade	1,2,3,4,6,7,8-HpCDD	0.4	0.001
Chicken with vegetables in sauce, from Chinese carry-out	1,2,3,4,6,7,8-HpCDD	0.4	0.001
Refried beans, canned	1,2,3,4,6,7,8-HpCDD	0.39	0.001
Green beans, fresh/frozen, boiled	1,2,3,4,6,7,8-HpCDD	0.38	0.003
Lettuce, leaf, raw	1,2,3,4,6,7,8-HpCDD	0.38	0.002
Baby food, chicken noodle dinner	1,2,3,4,6,7,8-HpCDD	0.36	0.004
Carrot, baby, raw	1,2,3,4,6,7,8-HpCDD	0.33	0.002
Vegetable oil	1,2,3,4,6,7,8-HpCDD	0.31	0.008
Sour cream	1,2,3,4,6,7,8-HpCDD	0.3	0.001
Pizza, cheese and pepperoni, regular crust, from pizza carry-out	1,2,3,4,6,7,8-HpCDD	0.29	0.002
Spinach, fresh/frozen, boiled	1,2,3,4,6,7,8-HpCDD	0.28	0.009
Potato, mashed, prepared from fresh	1,2,3,4,6,7,8-HpCDD	0.26	0.001
Broccoli, fresh/frozen, boiled	1,2,3,4,6,7,8-HpCDD	0.25	0.003
Summer squash, fresh/frozen, boiled	1,2,3,4,6,7,8-HpCDD	0.23	0.004
Beets, canned	1,2,3,4,6,7,8-HpCDD	0.23	0.001
Beef and vegetable stew, canned	1,2,3,4,6,7,8-HpCDD	0.22	0.005
Mayonnaise, regular, bottled	1,2,3,4,6,7,8-HpCDD	0.21	0.003
Corn, canned	1,2,3,4,6,7,8-HpCDD	0.2	0.001
Eggs, scrambled with oil	1,2,3,4,6,7,8-HpCDD	0.19	0.002
Ice cream, regular, vanilla	1,2,3,4,6,7,8-HpCDD	0.19	0.001
Sour cream dip, any flavor	1,2,3,4,6,7,8-HpCDD	0.19	0.001
Salad dressing, creamy/buttermilk type, regular	1,2,3,4,6,7,8-HpCDD	0.19	0.001
Sweet roll/Danish pastry	1,2,3,4,6,7,8-HpCDD	0.18	0.002
Baby food, cereal, barley, dry, prepared with water	1,2,3,4,6,7,8-HpCDD	0.18	0.002
Half and half cream	1,2,3,4,6,7,8-HpCDD	0.17	0.001
Baby food, mixed vegetables	1,2,3,4,6,7,8-HpCDD	0.17	0.005
Sweet potatoes, canned	1,2,3,4,6,7,8-HpCDD	0.17	0.001
Milk shake, chocolate, fast-food	1,2,3,4,6,7,8-HpCDD	0.16	0.001

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Eggs, boiled	1,2,3,4,6,7,8-HpCDD	0.16	0.001
Sandwich cookies with creme filling	1,2,3,4,6,7,8-HpCDD	0.16	0.004
Candy bar, milk chocolate, plain	1,2,3,4,6,7,8-HpCDD	0.16	0.001
Pork chop, pan-cooked with oil	1,2,3,4,6,7,8-HpCDD	0.14	0.002
Peanut butter, creamy	1,2,3,4,6,7,8-HpCDD	0.14	0.002
Cornbread, homemade	1,2,3,4,6,7,8-HpCDD	0.14	0.003
Pumpkin pie, fresh/frozen	1,2,3,4,6,7,8-HpCDD	0.14	0.004
Mixed vegetables, frozen, boiled	1,2,3,4,6,7,8-HpCDD	0.14	0.001
Doughnut, cake-type, any flavor	1,2,3,4,6,7,8-HpCDD	0.14	0.003
White beans, dry, boiled	1,2,3,4,6,7,8-HpCDD	0.14	0.002
Brownie	1,2,3,4,6,7,8-HpCDD	0.13	0.005
Pork and beans, canned	1,2,3,4,6,7,8-HpCDD	0.12	0.003
Ice cream, light, vanilla	1,2,3,4,6,7,8-HpCDD	0.099	0.001
Baby food, macaroni, tomato, and beef	1,2,3,4,6,7,8-HpCDD	0.096	0.001
Soup, Oriental noodles (ramen noodles), prepared with water	1,2,3,4,6,7,8-HpCDD	0.087	0.006
Milk, whole, fluid	1,2,3,4,6,7,8-HpCDD	0.084	0.001
Sugar cookies	1,2,3,4,6,7,8-HpCDD	0.082	0.004
Chocolate chip cookies	1,2,3,4,6,7,8-HpCDD	0.081	0.002
Potato chips	1,2,3,4,6,7,8-HpCDD	0.077	0.004
Infant formula, milk-based, high iron, ready to feed	1,2,3,4,6,7,8-HpCDD	0.071	0.001
Soup, bean with bacon/pork, canned, condensed, prepared with water	1,2,3,4,6,7,8-HpCDD	0.057	0.002
Bread, whole wheat	1,2,3,4,6,7,8-HpCDD	0.035	0.001
Bread, white, enriched	1,2,3,4,6,7,8-HpCDD	0.032	0.002
Frankfurter (beef/pork), boiled	1,2,3,6,7,8-HxCDD	0.69	0.002
Beef roast, chuck, oven-roasted	1,2,3,6,7,8-HxCDD	0.59	0.003
Butter, regular (salted)	1,2,3,6,7,8-HxCDD	0.51	0.003
Beef, ground, regular, pan-cooked	1,2,3,6,7,8-HxCDD	0.34	0.002
Cream cheese	1,2,3,6,7,8-HxCDD	0.32	0.0021
Mushrooms, raw	1,2,3,6,7,8-HxCDD	0.27	0.009
Meatloaf, beef, homemade	1,2,3,6,7,8-HxCDD	0.26	0.001

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Beef steak, loin/sirloin, broiled	1,2,3,6,7,8-HxCDD	0.247	0.001
Cheese, cheddar, natural (sharp/mild)	1,2,3,6,7,8-HxCDD	0.23	0.002
Liver (beef/calf), pan-cooked with oil	1,2,3,6,7,8-HxCDD	0.22	0.002
Salami, luncheon-meat type (not hard)	1,2,3,6,7,8-HxCDD	0.22	0.001
Catfish, pan-cooked with oil	1,2,3,6,7,8-HxCDD	0.21	0.003
Cheese, Swiss, natural	1,2,3,6,7,8-HxCDD	0.19	0.003
Burrito with beef, beans, and cheese, from Mexican carry-out	1,2,3,6,7,8-HxCDD	0.19	0.003
Chili con carne with beans, canned	1,2,3,6,7,8-HxCDD	0.16	0.002
Quarter-pound hamburger on bun, fast-food	1,2,3,6,7,8-HxCDD	0.16	0.001
Cheese, American, processed	1,2,3,6,7,8-HxCDD	0.15	0.003
Quarter-pound cheeseburger on bun, fast-food	1,2,3,6,7,8-HxCDD	0.15	0.003
Taco/tostada with beef and cheese, from Mexican carry-out	1,2,3,6,7,8-HxCDD	0.14	0.005
Sour cream	1,2,3,6,7,8-HxCDD	0.13	0.001
Baby food, vegetables and beef	1,2,3,6,7,8-HxCDD	0.09	0.004
Baby food, beef and noodles/beef stroganoff	1,2,3,6,7,8-HxCDD	0.088	0.004
Pizza, cheese and pepperoni, regular crust, from pizza carry-out	1,2,3,6,7,8-HxCDD	0.084	0.003
Ice cream, regular, vanilla	1,2,3,6,7,8-HxCDD	0.071	0.001
Lasagna with meat, frozen, heated	1,2,3,6,7,8-HxCDD	0.071	0.003
Potato, mashed, prepared from fresh	1,2,3,6,7,8-HxCDD	0.07	0.002
Sour cream dip, any flavor	1,2,3,6,7,8-HxCDD	0.062	0.004
Beef stroganoff with noodles, homemade	1,2,3,6,7,8-HxCDD	0.059	0.003
Half and half cream	1,2,3,6,7,8-HxCDD	0.057	0.001
Beef and vegetable stew, canned	1,2,3,6,7,8-HxCDD	0.056	0.002
Pork sausage (link/patty), oven-cooked	1,2,3,6,7,8-HxCDD	0.054	0.002

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Milk shake, chocolate, fast-food	1,2,3,6,7,8-HxCDD	0.053	0.001
Lamb chop, pan-cooked with oil	1,2,3,6,7,8-HxCDD	0.053	0.003
Bologna (beef/pork)	1,2,3,6,7,8-HxCDD	0.05	0.001
Spaghetti with meat sauce, homemade	1,2,3,6,7,8-HxCDD	0.049	0.001
Candy bar, milk chocolate, plain	1,2,3,6,7,8-HxCDD	0.046	0.001
Ice cream, light, vanilla	1,2,3,6,7,8-HxCDD	0.04	0.001
Eggs, boiled	1,2,3,6,7,8-HxCDD	0.035	0.003
Milk, whole, fluid	1,2,3,6,7,8-HxCDD	0.034	0.001
Eggs, scrambled with oil	1,2,3,6,7,8-HxCDD	0.034	0.003
Pumpkin pie, fresh/frozen	1,2,3,6,7,8-HxCDD	0.034	0.004
Lettuce, leaf, raw	1,2,3,6,7,8-HxCDD	0.033	0.002
Baby food, beef and broth/gravy	1,2,3,6,7,8-HxCDD	0.032	0.002
Chicken with vegetables in sauce, from Chinese carry-out	1,2,3,6,7,8-HxCDD	0.027	0.002
Brown gravy, canned or bottled	1,2,3,6,7,8-HxCDD	0.027	0.003
Vegetable oil	1,2,3,6,7,8-HxCDD	0.025	0.004
Green beans, fresh/frozen, boiled	1,2,3,6,7,8-HxCDD	0.024	0.005
Cornbread, homemade	1,2,3,6,7,8-HxCDD	0.02	0.003
Infant formula, milk-based, high iron, ready to feed	1,2,3,6,7,8-HxCDD	0.02	0.001
Cottage cheese, creamed, low fat (2% milk fat)	1,2,3,6,7,8-HxCDD	0.02	0.003
Pork chop, pan-cooked with oil	1,2,3,6,7,8-HxCDD	0.018	0.001
Beets, canned	1,2,3,6,7,8-HxCDD	0.018	0.003
Carrot, baby, raw	1,2,3,6,7,8-HxCDD	0.017	0.001
Margarine, regular (salted)	1,2,3,6,7,8-HxCDD	0.014	0.004
Beef with vegetables in sauce, from Chinese carry-out	1,2,3,6,7,8-HxCDD	0.014	0.005
Corn, canned	1,2,3,6,7,8-HxCDD	0.012	0.001
Mixed vegetables, frozen, boiled	1,2,3,6,7,8-HxCDD	0.01	0.002
Popcorn, microwave, butter-flavored	1,2,3,6,7,8-HxCDD	0.005	0.001
Milk, low fat (2%), fluid	1,2,3,6,7,8-HxCDD	0.002	0.022

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Beef roast, chuck, oven-roasted	1,2,3,7,8,9-HxCDD	0.2	0.003
Frankfurter (beef/pork), boiled	1,2,3,7,8,9-HxCDD	0.15	0.002
Liver (beef/calf), pan-cooked with oil	1,2,3,7,8,9-HxCDD	0.14	0.002
Catfish, pan-cooked with oil	1,2,3,7,8,9-HxCDD	0.14	0.003
Butter, regular (salted)	1,2,3,7,8,9-HxCDD	0.12	0.003
Beef, ground, regular, pan-cooked	1,2,3,7,8,9-HxCDD	0.089	0.002
Cream cheese	1,2,3,7,8,9-HxCDD	0.081	0.0021
Cheese, cheddar, natural (sharp/mild)	1,2,3,7,8,9-HxCDD	0.075	0.004
Meatloaf, beef, homemade	1,2,3,7,8,9-HxCDD	0.069	0.001
Cheese, Swiss, natural	1,2,3,7,8,9-HxCDD	0.062	0.003
Beef steak, loin/sirloin, broiled	1,2,3,7,8,9-HxCDD	0.055	0.001
Cheese, American, processed	1,2,3,7,8,9-HxCDD	0.047	0.006
Salami, luncheon-meat type (not hard)	1,2,3,7,8,9-HxCDD	0.044	0.001
Burrito with beef, beans, and cheese, from Mexican carry-out	1,2,3,7,8,9-HxCDD	0.043	0.003
Sour cream	1,2,3,7,8,9-HxCDD	0.037	0.001
Chili con carne with beans, canned	1,2,3,7,8,9-HxCDD	0.034	0.002
Quarter-pound hamburger on bun, fast-food	1,2,3,7,8,9-HxCDD	0.03	0.001
Sweet roll/Danish pastry	1,2,3,7,8,9-HxCDD	0.029	0.004
Ice cream, regular, vanilla	1,2,3,7,8,9-HxCDD	0.024	0.001
Potato, mashed, prepared from fresh	1,2,3,7,8,9-HxCDD	0.024	0.002
Sour cream dip, any flavor	1,2,3,7,8,9-HxCDD	0.023	0.002
Baby food, beef and noodles/beef stroganoff	1,2,3,7,8,9-HxCDD	0.022	0.002
Candy bar, milk chocolate, plain	1,2,3,7,8,9-HxCDD	0.021	0.001
Half and half cream	1,2,3,7,8,9-HxCDD	0.019	0.001
Spaghetti with meat sauce, homemade	1,2,3,7,8,9-HxCDD	0.018	0.001
Eggs, scrambled with oil	1,2,3,7,8,9-HxCDD	0.017	0.001
Lettuce, leaf, raw	1,2,3,7,8,9-HxCDD	0.014	0.003
Ice cream, light, vanilla	1,2,3,7,8,9-HxCDD	0.012	0.001

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Infant formula, milk-based, high iron, ready to feed	1,2,3,7,8,9-HxCDD	0.009	0.001
Milk, whole, fluid	1,2,3,7,8,9-HxCDD	0.008	0.001
Beef roast, chuck, oven-roasted	1,2,3,7,8-PeCDD	0.1	0.004
Cream cheese	1,2,3,7,8-PeCDD	0.091	0.0009
Frankfurter (beef/pork), boiled	1,2,3,7,8-PeCDD	0.089	0.004
Cheese, cheddar, natural (sharp/mild)	1,2,3,7,8-PeCDD	0.066	0.003
Beef, ground, regular, pan-cooked	1,2,3,7,8-PeCDD	0.052	0.004
Meatloaf, beef, homemade	1,2,3,7,8-PeCDD	0.043	0.001
Beef steak, loin/sirloin, broiled	1,2,3,7,8-PeCDD	0.038	0.003
Sour cream	1,2,3,7,8-PeCDD	0.033	0.001
Salami, luncheon-meat type (not hard)	1,2,3,7,8-PeCDD	0.031	0.001
Quarter-pound hamburger on bun, fast-food	1,2,3,7,8-PeCDD	0.029	0.001
Liver (beef/calf), pan-cooked with oil	1,2,3,7,8-PeCDD	0.027	0.003
Chicken potpie, frozen, heated	1,2,3,7,8-PeCDD	0.021	0.001
Burrito with beef, beans, and cheese, from Mexican carry-out	1,2,3,7,8-PeCDD	0.021	0.005
Chicken breast, oven-roasted (skin removed)	1,2,3,7,8-PeCDD	0.018	0.004
Chili con carne with beans, canned	1,2,3,7,8-PeCDD	0.016	0.004
Candy bar, milk chocolate, plain	1,2,3,7,8-PeCDD	0.016	0.001
Ice cream, regular, vanilla	1,2,3,7,8-PeCDD	0.015	0.001
Milk shake, chocolate, fast-food	1,2,3,7,8-PeCDD	0.013	0.001
Half and half cream	1,2,3,7,8-PeCDD	0.013	0.001
Syrup, pancake	1,2,3,7,8-PeCDD	0.01	0.007
Ice cream, light, vanilla	1,2,3,7,8-PeCDD	0.01	0.001
Milk, whole, fluid	1,2,3,7,8-PeCDD	0.009	0.001
Catfish, pan-cooked with oil	2,3,7,8-TCDD	0.026	0.001
Cream cheese	2,3,7,8-TCDD	0.021	0.0025
Salmon, steaks/fillets, baked	2,3,7,8-TCDD	0.021	0.001
Baby food, chicken noodle dinner	2,3,7,8-TCDD	0.015	0.007

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Cheese, cheddar, natural (sharp/mild)	2,3,7,8-TCDD	0.013	0.001
Beef roast, chuck, oven-roasted	2,3,7,8-TCDD	0.011	0.002
Meatloaf, beef, homemade	2,3,7,8-TCDD	0.008	0.001
Sour cream	2,3,7,8-TCDD	0.008	0.001
Ice cream, regular, vanilla	2,3,7,8-TCDD	0.004	0.001
Half and half cream	2,3,7,8-TCDD	0.003	0.001
Milk shake, chocolate, fast-food	2,3,7,8-TCDD	0.002	0.001
Liver (beef/calf), pan-cooked with oil	OCDD	65	0.013
Mushrooms, raw	OCDD	36	0.016
Catfish, pan-cooked with oil	OCDD	12	0.011
Beef roast, chuck, oven-roasted	OCDD	10	0.005
Pork sausage (link/patty), oven-cooked	OCDD	7.3	0.007
Frankfurter (beef/pork), boiled	OCDD	5.6	0.003
Vegetable oil	OCDD	5.5	0.007
Beef, ground, regular, pan-cooked	OCDD	4.9	0.003
Margarine, regular (salted)	OCDD	4.2	0.007
Lettuce, leaf, raw	OCDD	3.7	0.003
Salami, luncheon-meat type (not hard)	OCDD	3.5	0.002
Mayonnaise, regular, bottled	OCDD	3.2	0.004
Peanut butter, creamy	OCDD	2.6	0.004
Spinach, fresh/frozen, boiled	OCDD	2.5	0.015
Baby food, chicken noodle dinner	OCDD	2.5	0.006
Burrito with beef, beans, and cheese, from Mexican carry-out	OCDD	2.4	0.003
Bologna (beef/pork)	OCDD	2.3	0.002
Baby food, vegetables and beef	OCDD	2.3	0.006
Salad dressing, creamy/buttermilk type, regular	OCDD	2.3	0.005
Chili con carne with beans, canned	OCDD	2.1	0.003

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Baby food, cereal, barley, dry, prepared with water	OCDD	2.1	0.003
Baby food, mixed vegetables	OCDD	2	0.007
Meatloaf, beef, homemade	OCDD	1.9	0.001
Baby food, turkey and rice	OCDD	1.7	0.009
Green beans, canned	OCDD	1.6	0.012
Crackers, butter-type	OCDD	1.6	0.002
Butter, regular (salted)	OCDD	1.5	0.003
Sandwich cookies with creme filling	OCDD	1.5	0.006
Brownie	OCDD	1.5	0.008
Chicken with vegetables in sauce, from Chinese carry-out	OCDD	1.5	0.003
Beef steak, loin/sirloin, broiled	OCDD	1.4	0.002
Soup, Oriental noodles (ramen noodles), prepared with water	OCDD	1.4	0.007
Green beans, fresh/frozen, boiled	OCDD	1.3	0.004
Candy bar, milk chocolate, plain	OCDD	1.3	0.001
Refried beans, canned	OCDD	1.3	0.002
Cornbread, homemade	OCDD	1.1	0.004
Carrot, baby, raw	OCDD	1.1	0.003
Pork chop, pan-cooked with oil	OCDD	1	0.002
Baby food, cereal, oatmeal with fruit, prepared with water	OCDD	1	0.004
Broccoli, fresh/frozen, boiled	OCDD	0.97	0.005
Sweet roll/Danish pastry	OCDD	0.9	0.003
Eggs, scrambled with oil	OCDD	0.89	0.003
Pumpkin pie, fresh/frozen	OCDD	0.89	0.005
Fish sticks or patty, frozen, oven-cooked	OCDD	0.87	0.002
Pizza, cheese and pepperoni, regular crust, from pizza carry-out	OCDD	0.85	0.002
Cream cheese	OCDD	0.82	0.014
Sugar cookies	OCDD	0.81	0.005
Cheese, cheddar, natural (sharp/mild)	OCDD	0.78	0.002
Infant formula, soy-based, ready to feed	OCDD	0.78	0.001

Table 5-15. Detections of CDD Congeners in Food Items from the 2004 FDA Market Basket Survey

Product description	Congener description	Amount found (pg/g [ppt])	LOD (pg/g [ppt])
Fried rice, meatless, from Chinese carry-out	OCDD	0.77	0.003
Beets, canned	OCDD	0.74	0.002
Sunflower seeds (shelled), roasted, salted	OCDD	0.74	0.002
Fish sandwich on bun, fast-food	OCDD	0.72	0.002
Eggs, boiled	OCDD	0.65	0.002
Corn, canned	OCDD	0.64	0.001
Potato, mashed, prepared from fresh	OCDD	0.57	0.002
Baby food, macaroni, tomato, and beef	OCDD	0.56	0.004
Sweet potatoes, canned	OCDD	0.54	0.002
Mixed vegetables, frozen, boiled	OCDD	0.53	0.002
Pepper, sweet, green, raw	OCDD	0.5	0.003
Ham, cured (not canned), baked	OCDD	0.47	0.003
Pork and beans, canned	OCDD	0.45	0.005
Coleslaw, mayonnaise-type, from grocery/deli	OCDD	0.41	0.001
Soup, bean with bacon/pork, canned, cond, prepared with water	OCDD	0.39	0.003
Milk, low fat (2%), fluid	OCDD	0.37	0.003
Beef and vegetable stew, canned	OCDD	0.37	0.004
Candy bar, chocolate, nougat, and nuts	OCDD	0.33	0.001
Milk shake, chocolate, fast-food	OCDD	0.31	0.002

CDD = chlorinated dibenzo-*p*-dioxin; FDA = U.S. Food and Drug Administration; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection; PeCDD = pentachlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: FDA 2007

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) were 0.35–2.91 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 5-16. The TEQs for both the CDDs and CDFs on a wet weight basis for these food samples were 0.02–1.5 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.

Table 5-16. Dioxins, Dibenzofurans, and Dioxin Toxic Equivalencies (TEQs) in U.S. Foods (ppt or pg/g, 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
Cooked 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

CDDs = chlorinated dibenzo-*p*-dioxin; CDFs = chlorinated dibenzofuran

Source: Schecter et al. 1994d

The EPA and 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 5-17. 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 (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.

Table 5-17. Overall National Averages of the Concentrations (ppt or pg/g) of CDDs 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-TCDD	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)
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 one-half the detection limit (results for ND=0 are in parentheses).

CDD = chlorinated dibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; NR = not reported; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQ = toxic equivalency

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

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 low fat (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), low fat (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).

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. The study 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 were 24–25 pg/kg in whole milk and 13–14 pg/kg in half-and-half. The corresponding concentrations of 2,3,7,8-TCDF were 260–280 pg/kg for whole milk and 146–195 pg/kg for half-and-half. The study 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 was 2–6%, while the percentage of migration of the 2,3,7,8-TCDF was 4–18% 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. 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 (pg/g) in lake trout from Lake Superior to 64.5 ng/kg (pg/g) in Lake Ontario (wet weight basis). Concentrations of 2,3,7,8-TCDD ranged from 1 ng/kg (pg/g) in lake trout

from Lake Superior to 48.9 ng/kg (pg/g) 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 (pg/g) in Lake Superior to 16.7 ng/kg (pg/g) 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 (pg/g) in Lake Superior to 10.9 ng/kg (pg/g) in Lake Michigan. Substantial inter-lake 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 study authors suggested 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).

More recent data suggest that levels of CDDs in fish from the Great Lakes is decreasing, as emissions have declined over the previous decades. A study conducted on dioxin-like substances in fish of the Great Lakes has shown that there has generally been a large decline in CDD/CDF levels in fish since the 1970s (Gandhi et al. 2019). CDD/CDF levels declined between 1989 and 2013 in lake trout from Lakes Ontario, Huron, and Superior by 91, 78, and 73%, respectively, but increased in Lake Whitefish obtained from Lake Erie by 138%. Using an expanded set of data, from the literature, it was shown the TEQ levels in trout from Lake Ontario decreased from 64 to 2.3 pg/g, which is approximately a 96% decrease. The results of this study on 30 types of fish show overall declining levels of CDD/CDF in fish but local/ regional concerns at some locations in the Great Lakes still exist. Pagano et al. (2018) collected monitoring data for CDDs/CDFs in fish from the Great Lakes from 2004 to 2014. The results of this study as well as other recent monitoring data for some congener specific CDDs are summarized in Table 5-18.

Table 5-18. Levels of CDDs in Fish and Other Aquatic Organisms

Species	Sampling area	CDD	Concentration (pg/g [ppt [wet weight]])	Reference
Walleye	Lake Erie	2,3,7,8-TCDD	0.76 (average) 0.88 (maximum)	Pagano et al. 2018
Walleye	Lake Erie	2,3,7,8-TCDD	0.30 (average) 0.4 (maximum)	Pagano et al. 2018
Lake trout	Lake Erie	2,3,7,8-TCDD	0.60–0.73 0.66 (average)	Pagano et al. 2018
Lake trout	Lake Huron	2,3,7,8-TCDD	1.32–3.32 2.35 (average)	Pagano et al. 2018
Lake trout	Lake Michigan	2,3,7,8-TCDD	0.40–1.27 0.72 (average)	Pagano et al. 2018
Lake trout	Lake Ontario	2,3,7,8-TCDD	2.70–13.5 5.76 (average)	Pagano et al. 2018
Lake trout	Lake Superior	2,3,7,8-TCDD	0.35–0.69 0.49 (average)	Pagano et al. 2018
Lake trout	Lake Superior	2,3,7,8-TCDD	0.17-–0.40 0.28 (average)	Pagano et al. 2018
Lake trout	Lake Champlain	2,3,7,8-TCDD	0.422–1.291 (0.724 average)	Pagano and Garner 2020
White sucker	Lake Superior (St. Louis River area)	Total TCDD	<LOD–1.25	Hoffman et al. 2020
White sucker	Lake Superior (St. Louis River area)	Total PeCDD	<LOD–2.14	Hoffman et al. 2020
White sucker	Lake Superior (St. Louis River area)	Total HxCDD	<LOD–3.94	Hoffman et al. 2020
White sucker	Lake Superior (St. Louis River area)	Total HpCDD	<LOD–3.12	Hoffman et al. 2020
Roach	Rybnicki, Poland	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 OCDD	0.005–0.034 <0.004–0.045 <0.002–0.005 0.007–0.040 <0.002–0.006 <0.010–0.033 <0.024–0.033	Mikolajczyk et al. 2022a, 2022b
Bream	Rybnicki, Poland	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 OCDD	0.021–0.277 0.035–0.490 0.016–0.243 0.038–0.481 0.007–0.068 <0.048–0.381 0.035–0.537	Mikolajczyk et al. 2022a, 2022b

Table 5-18. Levels of CDDs in Fish and Other Aquatic Organisms

Species	Sampling area	CDD	Concentration (pg/g [ppt [wet weight]])	Reference
Roach	Maróz Poland	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 OCDD	0.003–0.011 0.005–0.014 <0.002–0.009 0.002–0.009 <0.002–0.005 <0.010–0.087 0.024–0.258	Mikolajczyk et al. 2022a, 2022b
Bream	Maróz Poland	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 OCDD	0.005–0.019 0.008–0.060 0.003–0.026 0.020–0.060 0.003–0.009 0.016–0.060 0.029–0.081	Mikolajczyk et al. 2022a, 2022b
Pike	Maróz Poland	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 OCDD	0.003–0.014 0.006–0.021 <0.002 0.002–0.013 <0.002 0.010–0.025 <0.029–0.066	Mikolajczyk et al. 2022a, 2022b
Pike	Lipczyno Wielkie Poland	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 OCDD	0.003–0.029 0.005–0.020 0.002–0.007 0.002–0.033 <0.002–0.005 <0.010–0.016 0.042–0.048	Mikolajczyk et al. 2022a, 2022b
Bream	Łańskie, Poland	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 OCDD	0.004–0.076 0.019–0.151 0.003–0.056 0.014–0.130 0.004– 0.010 0.029–0.114 0.028–0.097	Mikolajczyk et al. 2022a, 2022b

CDD = chlorinated dibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin
LOD = limit of detection; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin;
TCDD = tetrachlorodibenzo-*p*-dioxin

Data from the Water Quality Portal for 2020–2021 indicated that there were 94 instances of CDDs detected out of 315 fish samples tested. The maximum concentrations were observed for 1,2,3,7,8-PeCDD with values of 30–200 ng/kg (pg/g) obtained from channel catfish, carp, and largemouth bass from McKeller Lake and Nonconnah creek in Tennessee (WQP 2022).

Khairy and Lohmann (2020a, 2020b), measured levels of CDDs and CDFs in four benthic species (i.e., mud crabs, tube worms, clams, and shrimp) collected from the lower Passaic River at different sampling locations. The results for several CDD congeners are provided in Table 5-19.

Table 5-19. Concentrations of CDDs (pg/g Lipid [ppt]) in Benthic Species Collected from the Lower Passaic River

Congener	Range of values in benthic organisms collected at four different locations
2-MCDD	<LOD
2,7/2,8-DCDD	2,750–4,420
1,2,4-TrCDD	<LOD–745
1,3,6,8-TCDD	<LOD–287
1,3,7,8-TCDD	<LOD–1,028
2,3,7,8-TCDD	653–1,969
1,2,8,9-TCDD	<LOD
1,2,3,4,7-PeCDD	<LOD
1,2,3,4,7,8-HxCDD	<LOD
1,2,3,6,7,8-HxCDD	<LOD
1,2,3,7,8,9-HxCDD	<LOD–898
1,2,3,4,6,7,8-HpCDD	484–2,057
OCDD	2,028–8,991

CDD = chlorinated dibenzo-*p*-dioxin; DCDD = dichlorodibenzo-*p*-dioxin; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin LOD = limit of detection; MCDD = monochlorodibenzo-*p*-dioxin; OCDD = octachlorodibenzo-*p*-dioxin; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TrCDD = trichlorodibenzo-*p*-dioxin

Source: Khairy and Lohmann 2020b

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 (pg/g) on a wet weight basis). Of the 112 sites where 2,3,7,8-TCDD was detected, 74 samples (67%) were <5 ppt (pg/g), 34 samples (32%) were between 5 and 25 ppt (pg/g), and 4 samples (1%) were >25 ppt (pg/g). 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 (pg/g)). 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 (pg/g), was collected from Lake Ontario near

Oswego, New York. Four of 57 (7%) estuarine or coastal sites had detectable 2,3,7,8-TCDD levels in either fish or shellfish. The levels of contamination in these four samples were 1.08–3.5 ppt (pg/g) (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 1987c). 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 [pg/g]). 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 [pg/g]) was almost 7 times higher than the concentrations in the fillets from fish collected from urban areas (0.3 ppt [pg/g]). 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 (pg/g). This geometric mean is 3 times higher than for urban fillet concentrations (0.3 ppt [pg/g]) 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 5-20. 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 (pg/g) and 2,3,7,8-TCDF at 404 ppt (pg/g) (wet weight). 2,3,7,8-TCDD was found at 70% of the sites at a maximum concentration of 204 ppt (pg/g) and a mean of 6.8 ppt (wet weight basis). The NSCRF report further shows that pulp and paper mills that previously used 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. With respect to source categories, the NSCRF data showed that fish collected downstream of pulp and paper mills (using chlorine bleaching processes) had the highest median 2,3,7,8-TCDD concentrations (5.66 ppt [pg/g]), compared to the next highest source category, refinery/other industrial sites (1.82 ppt [pg/g]), industrial/urban sites (1.40 ppt [pg/g]), Superfund sites (1.27 ppt [pg/g]), 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/OCDF (e.g., wood preservers) (EPA 1992).

Table 5-20. Summary of CDDs Detected in Fish Tissue as Part of the EPA National Study of Chemical Residues in Fish[a]

Congener	Percent of 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

[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 that were below the detection limit. In cases where multiple samples were analyzed per site, the value used represents the highest concentration.

CDD = chlorinated dibenzo-*p*-dioxin; EPA = U.S. Environmental Protection Agency; HpCDD = heptachlorodibenzo-*p*-dioxin; HxCDD = hexachlorodibenzo-*p*-dioxin; NA = not applicable; PeCDD = pentachlorodibenzo-*p*-dioxin; TCDD = tetrachlorodibenzo-*p*-dioxin; TEQ = toxic equivalency

Source: EPA 1992

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 (pg/g) of total TCDD (wet weight basis). Detection limits ranged from 0.28 to 6.6 ppt (pg/g) on a congener- 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 (pg/g)), 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 (pg/g)), and total PeCDD (average 3.9 ppt, range 3.5–4.5 ppt [pg/g]) (Reed et al. 1990). Lake Orono showed the same pattern, with OCDD being the most abundant congener (average 39 ppt, range 35–43 ppt [pg/g]), followed by total HpCDD (average 10.5, range 10–11 ppt [pg/g]), and total HxCDD (3.0 ppt [pg/g]). 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 (pg/g) in whole fish and 20 ppt (pg/g) in fish fillets (wet weight basis). Mean concentrations in fish caught more than 14 km downstream were <4 ppt (pg/g) 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, New Jersey, 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 (pg/g) and TCDD was found in 3 of 10 samples at 0.1 ppt (pg/g) (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 [pg/g]) (wet weight basis) of 2,3,7,8-substituted TCDD, PeCDD, and CDFs (Rappe et al. 1991). Concentrations of HxCDD and HpCDD were <0.1–220.7 and <0.7–244.9 ppt (pg/g), 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. Concentrations of 2,3,7,8-TCDD in tissue were 3,700–6,200 ppt (pg/g) in crab hepatopancreas and 100–120 ppt (pg/g) 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 from 5 to 6 ppt (pg/g) in the meat. Concentrations of 2,3,7,8-TCDD in striped bass muscle tissue were 84–730 ppt (pg/g). 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 1,2,3,7,8-PeCDD, while the major isomer found in the striped bass was 1,2,3,7,8-PeCDD (5–10 ppt [pg/g]). Regarding the HxCDDs, the crustacean samples contained three major peaks, one of which was 1,2,3,6,7,8-HxCDD (100–300 ppt (pg/g) 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 (pg/g), while meat samples contained 0.00–8.5 ppt (pg/g). Striped bass tissue samples contained 4–11.4 ppt (pg/g). Concentrations of OCDD were 50.5–94.6 ppt in crustacean hepatopancreas tissues and 6.3–78.8 ppt (pg/g) in meat samples, while concentrations in striped bass were 5.1–49.5 ppt (pg/g) (Rappe et al. 1991).

Concentrations of CDDs/CDFs were also evaluated in a bivalve mollusk, 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 (pg/g) TCDD, 3.5–5 ppt (pg/g) TCDF, and 13–25 ppt (pg/g) TEQ; those from Arthur

Kill contained 4.8–7.7 ppt (pg/g) TCDD, 3.1–5.1 ppt (pg/g) TCDF, and 6.8–11 ppt (pg/g) TEQ; and those from Raritan Bay contained 0.5–1.1 ppt (pg/g) TCDD, 2–4.6 ppt (pg/g) TCDF, and 1.2–2.1 ppt (pg/g) TEQ (wet weight basis). Concentrations decreased with increasing distance from the suspected pesticide plant site near Newark Bay. The study 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.

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 [pg/g]). OCDD concentrations in seal blubber and polar bear samples ranged from not detected (<8 ppt [pg/g]) to 43 ppt (pg/g). No biomagnification of TCDD or 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 seven 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).

Consumer Products

Cigarettes and cigarette smoke. CDDs have been detected in cigarettes and cigarette smoke. 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. The study 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 can be formed during pulp bleaching, and as a result, they have been found in many different types of paper products that previously employed elemental chlorine in the bleaching process. 2,3,7,8-Substituted CDDs were determined in different samples of coffee-filter paper (Beck et al. 1988, 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 of 0.03–0.7 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. 1988). 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. 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).

Changes in the commercial bleaching process have significantly reduced the levels of CDDs/CDFs in paper products. The use of chlorine dioxide rather than elemental chlorine in the bleaching procedure essentially eliminates the formation of 2,3,7,8-TCDD and 2,3,7,8-TCDF in finished products (Axegård 2019). Almost all new paper mills use elemental chlorine-free bleaching and other techniques such as oxygen delignification, which reduce the amount of lignin in the pulp and thus lower the need for bleaching chemicals (Axegård 2019). Moreover, the elimination of unchlorinated dioxin containing precursers that were found in some mineral oils formerly used in the paper milling process has also lowered the formation of CDDs/CDFs 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 study 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 were 18,066–41,953 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 were 806–11,022 ng/g (ppb), while OCDF concentrations were 125–3,749 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 study has identified sources of CDDs/CDFs found in textiles. Horstmann and McLachlan (1994) detected CDD/CDF concentrations in new textile products ranging from <50 pg/g to as high as 290,000 pg/g. The study 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 was still being used in developing countries at the time the study was conducted, especially for purposes of preserving cotton during sea transport, the study authors hypothesized 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 (1994) detected CDD/CDF residues in used dry-cleaning fluid and concluded that the source of the CDD/CDF residues in the dry-cleaning fluid were introduced by dry-cleaning new, unwashed textiles that had been treated with PCP.

Motor vehicle exhaust. CDDs have been identified in automobile exhaust emissions (Marklund et al. 1987, 1990). 2,3,7,8-TCDD was found in car exhaust from four Swedish cars running on leaded gasoline

at levels of <0.05–0.3 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 of 6–98 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).

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 (EPA 1996a).

5.6 GENERAL POPULATION EXPOSURE

Consumption of food (including human milk) is by far the most important pathway for exposure to CDDs for the general population, representing >90% of the total daily intake (Beck et al. 1989a; Hattemer-Frey and Travis 1989; Liem and van Zorge 1995; Rappe 1992; Schaum et al. 1994; Schecter et al. 1994a, 1994d, 1996a). 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) (Schaum et al. 1994; Travis and Hattemer-Frey 1987).

The U.S. Department of Agriculture (USDA) Food Safety and Inspection Service (FSIS) examined levels of dioxin like compounds measured in FSIS-regulated meat and poultry (Dearfield et al. 2013). Several different exposure scenarios based upon EPA derived actual consumption pattern scenarios and recommended consumption guidelines were considered given the amount of beef or poultry consumed by a specific age group. They concluded that a typical U.S. adult daily exposure of dioxin-like substances in FSIS-regulated products is below the EPA-established RfD. The mean dioxin exposure from beef products based upon U.S. consumption rates is provided in Table 5-21.

Table 5-21. Mean Dioxin Exposure from Beef, Based on Beef Consumption

Demographic	Amount of beef consumed g/kg per day	Mean DLC[a] pg-TEQ/kg-body weight per day (non-lean beef)[b]	Mean DLC pg-TEQ/kg-body weight per day lean beef)
Whole population	0.77	0.098	0.031
Birth to 1 year	0.34	0.043	0.014
1–2 years	1.38	0.175	0.056
3–5 years	1.42	0.180	0.058
6–12 years	1.11	0.141	0.045
13–19 years	0.83	0.105	0.034
20–49 years	0.73	0.093	0.030
Females 13–49 years	0.60	0.076	0.024
≥50 years	0.58	0.074	0.024

[a]Non-lean beef 19.24% fat; lean beef 6.16% fat.
[b]DLC = dioxin-like compounds, includes 17 CDDs/CDFs and 4 non-ortho PCBs.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; PCB = polychlorinated biphenyl; TEQ = toxic equivalency

Source: Dearfield et al. 2013

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 5-22. Since levels of CDDs and CDFs have declined in environmental media, including food items, as emissions have been reduced, these estimated intakes are likely higher than current intakes. 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 be 390–8,400 pg/day (EPA 1985); however, levels of CDDs and CDFs in fish in the Great Lakes have dropped dramatically since the time of this study (Gandhi et al. 2019). For example, the 40-year trend of five major CDD/CDF congeners including 2,3,7,8-TCDD in lake trout from

Lake Ontario were shown to decrease approximately 96% from the late 1970s to 2013 (Gandhi et al. 2019). 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% ($6.5x10^{-3}$ 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 estimates made by Schaum et al. (1994) for intakes of total CDDs/CDFs (Table 5-23). 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. Lorber et al. (2009) estimated a decrease in dietary exposure to 17 CDD/CDFs of approximately 33% from the mid-1990s to the early 2000s using data from food samples collected between 2001 and 2004 by the FDA.

Table 5-22. Estimated Average Daily Intake of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (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	$6.5x10^{-3}$	<0.01
Soil/ingestion	–	–
Food sources (total)/ingestion	46	98
Produce	5	11
Milk	13	27
Meat	23	50
Fish	5	10
Total intake	47	100

Source: Hattemer-Frey and Travis 1989

Table 5-23. Estimated Daily Background Exposure to Chlorinated Dibenzo-*p*-Dioxins (CDDs) and Chlorinated Dibenzofurans in the General U.S. Population

Source	Daily intake (pg/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	–	–
Milk and milk products	42	35
Milk	18	15
Cheese	24	20

Table 5-23. Estimated Daily Background Exposure to Chlorinated Dibenzo-*p*-Dioxins (CDDs) and Chlorinated Dibenzofurans in the General U.S. Population

Source	Daily intake (pg/day)	Percentage of total daily intake
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

The FDA calculated exposure to CDDs/CDFs based upon data from its 2001–2004 Total Diet Study in which commercially sold food items are collected from different regions of the country and analyzed for specific CDD and CDF congeners (FDA 2006). The dietary exposure estimates from these data are provided in Table 5-24.

Table 5-24. Dietary CDD/CDF Exposure Estimate (pg WHO-TEQ/kg Body Weight/Month) by Food Category from TDS Foods Collected in 2001–2004

Group	Dairy and mixtures	Eggs and mixtures	Fats, oils, mixtures	Fish and mixtures	Fruits, vegetables and mixtures	Meat and mixtures	Poultry and mixtures	Other foods and mixtures	Total
All groups	1.5	0.1	0.2	0.9	0.5	4.5	0.2	1.8	9.6
Infants 6–11 months infants	6.0	0.3	0.1	0.2	1.5	2.4	0.6	1.7	12.8
Children 2 years	7.4	0.5	0.3	0.9	1.3	9.2	0.4	3.5	23.5
Children 6 years	5.0	0.2	0.3	0.8	0.7	7.5	0.3	3.6	18.5
Children 10 years	3.4	0.2	0.1	0.5	0.5	5.4	0.3	2.7	13.1
Females 14–16 years	1.2	0.1	0.1	0.6	0.3	3.5	0.2	1.9	7.8
Males 14–16 years	1.9	0.1	0.1	0.6	0.3	4.6	0.2	2.9	10.7
Women 25–30 years	1.0	0.1	0.1	0.8	0.3	2.6	0.2	1.5	6.6

Table 5-24. Dietary CDD/CDF Exposure Estimate (pg WHO-TEQ/kg Body Weight/Month) by Food Category from TDS Foods Collected in 2001–2004

Group	Dairy and mixtures	Eggs and mixtures	Fats, oils, mixtures	Fish and mixtures	Fruits, vegetables and mixtures	Meat and mixtures	Poultry and mixtures	Other foods and mixtures	Total
Men 25–30 years	1.0	0.2	0.1	0.4	0.3	4.3	0.2	1.9	8.4
Women 40–45 years	0.8	0.1	0.1	0.7	0.4	3.1	0.2	1.2	6.6
Men 40–45 years	1.1	0.1	0.1	0.7	0.4	4.1	0.3	1.2	7.9
Women 60–65 years	0.7	0.1	0.1	1.1	0.4	2.5	0.2	0.7	5.7
Men 60–65 years	0.8	0.2	0.2	1.1	0.4	3.7	0.2	0.9	7.4
Women >70 years	0.8	0.1	0.1	0.9	0.5	2.3	0.2	0.7	5.6
Men >70 years	1.1	0.1	0.2	1.0	0.5	3.1	0.2	0.8	7.1

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; TDS = Total Diet Study; TEQ = toxic equivalency; WHO = World Health Organization

Source: FDA 2006

The National Academy of Science (NAS) has also estimated dioxin (CDD and CDF congeners) intake from meat, poultry, and fish for various age and demographic groups using a subset of data from the FDA's Total Diet Study; these estimates, for consumers of high and low amounts of animal products, are presented in Table 5-25 (NAS 2003).

Table 5-25. Estimated Intake of CDDs and CDFs from Meat, Poultry, and Fish

	TEQ intake[a] (pg/kg body weight/day)
Males and females, 1–5 years of age (not breastfeeding)	1.76–1.26
Males and females, 6–11 years of age	1.14–0.77
Males, 12–19 years of age	0.89–0.47
Males, ≥20 years of age	0.69–0.40
Females, 12–19 years of age, not pregnant or lactating	0.69–0.47
Females, ≥20 years of age, not pregnant or lactating	0.59–0.38

Table 5-25. Estimated Intake of CDDs and CDFs from Meat, Poultry, and Fish

	TEQ intake[a] (pg/kg body weight/day)
Females, pregnant or lactating	0.65–0.54
Males and females, ≥1 years of age, including pregnant or lactating	0.78–0.64

[a]Includes CDD and CDF congeners only; range represents average intake for consumers of high and low (<3 ounces) intakes of meat, poultry, and fish. TEQs calculated using 0.5 (LOD) for non-detects.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; LOD = limit of detection; TEQ = toxic equivalency

Source: NAS 2003

Based on their congener-specific analysis of 18 food samples collected in Binghamton, New York, Schecter et al. (1994d) estimated that the U.S. mean daily exposure to CDD equivalents for an adult (65 kg body weight) were 18–192 pg TEQs, depending on how not-detected values were treated. This is equal to a daily adult intake of CDDs/CDFs of 0.3–3.0 pg TEQs/kg body weight. The study authors reported that total CDDs were 0.35–2.91 ppt (wet weight) in fish, 0.6–59.3 ppt in meat products, and 0.6–14 ppt in dairy products. The total CDD/CDF TEQ values were 0.023–0.13 ppt for fish, 0.03–1.5 for meat products, and 0.04–0.7 for dairy products. The study authors 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. 1994a, 1994d). An ovo-lacto vegetarian diet that contains eggs and dairy products would not achieve this same reduction level. 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, Illinois; Louisville, Kentucky; Atlanta, Georgia; and San Diego, California (Schecter et al. 1996a). For 1995, the study authors reported that the estimated U.S. mean daily exposure to CDDs/CDFs TEQs for an adult (65 kg body weight) ranged from 34 to 167 pg TEQs. This is equivalent to a daily adult intake of CDDs/CDFs of 0.52–2.57 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 follow-up to this study was published in 2001, in which 110 food items were purchased from the same locations (Schecter et al. 2001). The study collected 12 different types of foods from 4 categories: meat, fish, dairy, and milk. Levels of CDDs ranged from below the detection limits to 59.2 pg/g for an OCDD in a sample of butter. For adult men aged 20–79 years, the estimated total TEQ intake per day was calculated as 2.4 pg/kg body weight. A survey of CDDs/CDFs in total diet food samples in Canada was conducted by Ryan et al. (1997). The study 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.

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 CDDs/CDFs of $3x10^{-4}$ 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 CDDs/CDFs 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 ($3x10^{-4}$ 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 study authors concluded that MSW incinerators will not substantially increase human exposure to CDDs/CDFs above normal background levels (Hattemer-Frey and Travis 1989). Table 5-26 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. The study 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 later 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 data of Schecter et al. (1995) who found that workers who operate municipal waste incinerators have blood levels of TEQs that do not differ significantly from background levels.

Table 5-26. Estimated Average Daily Intake of TEQs Associated with Exposure to a Typical State-of-the-Art Municipal Waste Incinerator

Exposure pathway	Daily intake (ng/TEQ/day)	Percentage of total intake
Inhalation	$1.3x10^{-4}$	43
Total ingestion	$1.1x10^{-4}$	37
Mother's milk	$5.7x10^{-5}$	19

Table 5-26. Estimated Average Daily Intake of TEQs Associated with Exposure to a Typical State-of-the-Art Municipal Waste Incinerator

Exposure pathway	Daily intake (ng/TEQ/day)	Percentage of total intake
Dermal absorption	$2.2x10^{-6}$	1
Total intake	$3.0x10^{-4}$	100

TEQ = toxic equivalency

Source: Hattemer-Frey and Travis 1989

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).

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. 1989c; 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 <1 ng/kg TEQ and changes in the milling process have drastically reduced the levels of CDDs/CDFs in these products. 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 (EPA 1986a; Orban et al. 1994; Patterson et al. 1986a; Ryan et al. 1986; Schecter et al. 1986b; Stanley et al. 1986). Results of the 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 (EPA 1986a; 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 congener and 2,3,7,8-TCDD was the least abundant). The analysis of 46 composite adipose samples verified the prevalence of the 2,3,7,8-substituted tetra- through octaCDDs in the U.S. population (EPA 1986a; 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 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 5-27. 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-year-old age group and the lowest concentrations occur in the 0–14-year-old age group). 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 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 pg/g) in the 1987 study.

Table 5-27. Chlorinated Dioxins and Dibenzofurans in Adipose Tissue of the General U.S. Population

	Concentration (pg/g, lipid basis)[a]		
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
1,2,3,7,8,9-HxCDD	<3.86[b]	11.5	22.0
1,2,3,4,7,8/1,2,3,6,7,8-HxCDD	13.3	76.1	174.0

Table 5-27. Chlorinated Dioxins and Dibenzofurans in Adipose Tissue of the General U.S. Population

	Concentration (pg/g, lipid basis)[a]		
Compound	Minimum	Median	Maximum
1,2,3,4,6,7,8-HpCDD	20.9	110.0	230.0
OCDD	152.0	838.0	1,630.0

[a]Not detected concentrations were replaced by one-half the limit of detection.
[b]The minimum concentration is less than the minimum reported limit of detection.

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

Source: Orban et al. 1994

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).

The levels of select CDD congeners were measured in blood samples collected as part of the NHANES. 1,2,3,4,6,7,8-HpCDD, 1,2,3,4,7,8-HxCDD, 1,2,3,6,7,8-HxCDD, 1,2,3,7,8,9-HxCDD, OCDD, 1,2,3,7,8 PeCDD, and 2,3,7,8-TCDD levels for survey years 1999–2000, 2001–2002, and 2003–2004 are presented in the National Report on Human Exposures to Environmental Chemicals (CDC 2024a). These data are summarized in Tables 5-28–5-34. Weighted arithmetic means and unadjusted standard errors of pooled serum concentrations from 2005 to 2012 survey years are also available (CDC 2024b) (Tables 5-35–5-41).

Table 5-28. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	58.2 (<LOD–63.6)	86.0 (75.5–96.7)	112 (101–131)
2001–2002	39.0 (33.7–45.0)	40.2 (34.9–46.9)	68.7 (56.7–82.2)	115 (88.2–138)	147 (126–181)
2003–2004	25.3 (23.4–27.3)	24.9 (22.8–26.9)	42.5 (38.8–48.1)	70.4 (62.7–80.1)	91.3 (73.5–117)
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	63.6 (<LOD–75.6)
2001–2002	ND	ND	ND	ND	ND
2003–2004	16.7 (15.1–18.4)	16.4 (15.1–18.3)	23.6 (21.5–25.8)	33.4 (28.6–36.8)	46.7 (34.5–78.1)
≥20 years					
1999–2000	NC	<LOD	62.0 (57.1–66.7)	92.9 (81.2–101)	120 (102–139)
2001–2002	39.0 (33.7–45.0)	40.2 (34.9–46.9)	68.7 (56.7–82.2)	115 (88.2–138)	147 (126–181)
2003–2004	26.8 (24.6–29.2)	27.3 (24.6–29.0)	45.6 (41.3–53.2)	73.7 (64.1–88.6)	95.0 (76.1–126)
Gender					
Males					
1999–2000	NC	<LOD	<LOD	73.6 (69.0–80.8)	94.7 (83.1–103)
2001–2002	36.6 (31.7–42.3)	39.0 (33.3–42.6)	62.1 (49.7–75.0)	102 (75.8–132)	138 (103–169)
2003–2004	24.2 (21.7–27.0)	23.2 (21.1–25.6)	40.6 (35.3–46.9)	64.2 (58.8–73.7)	85.0 (65.8–113)
Females					
1999–2000	NC	<LOD	62.7 (<LOD–69.1)	102 (86.0–118)	131 (111–164)
2001–2002	41.2 (34.9–48.7)	43.6 (35.3–52.4)	76.0 (59.5–90.1)	125 (93.4–150)	158 (130–191)
2003–2004	26.3 (24.4–28.3)	26.8 (24.3–28.3)	44.4 (41.1–50.2)	76.1 (65.3–89.1)	95.7 (80.7–128)
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	61.4 (<LOD–69.0)	97.7 (82.8–111)	132 (108–159)
2001–2002	39.6 (35.7–43.9)	39.7 (33.6–47.4)	64.0 (55.8–74.7)	107 (82.4–128)	149 (111–171)
2003–2004	25.8 (22.6–29.4)	26.1 (20.9–30.9)	41.9 (36.7–44.7)	61.0 (49.7–71.9)	80.1 (65.0–89.1)

Table 5-28. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	58.1 (<LOD–71.1)	95.0 (75.1–110)	125 (102–183)
2001–2002	43.7 (35.4–54.0)	42.8 (32.2–59.8)	80.6 (60.9–106)	134 (101–166))	167 (130–230
2003–2004	25.8 (22.6–29.4)	23.7 (20.7–27.1)	41.2 (32.6–56.4)	69.2 (54.6–115)	115 (67.1–164)
Non-Hispanic whites					
1999–2000	NC	<LOD	59.0 (<LOD–64.8)	84.9 (72.0–97.0)	106 (96.7–122)
2001–2002	39.3 (33.0–46.8)	40.5 (34.0–50.1)	71.0 (56.3–87.5)	117 (87.1–147)	147 (125–186)
2003–2004	25.0 (22.6–27.7)	24.6 (22.3–27.4)	42.6 (39.4–48.5)	73.5 (60.4–86.7	93.7 (71.6–127)

HpCDD = heptachlorodibenzo-*p*-dioxin; LOD = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 55.9, 10.3, and 13.0 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002.

Source: CDC 2024a

Table 5-29. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
2001–2002	NC	<LOD	<LOD	10.7 (<LOD–13.9)	14.9 (11.7–20.0)
2003–2004		<LOD	<LOD	<LOD	<LOD
Age group					
12–19 years					
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
≥20 years					
2001–2002	NC	<LOD	<LOD	10.7 (<LOD–13.9)	14.9 (11.7–20.0)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Gender					
Males					
2001–2002	NC	<LOD	<LOD	10.9 (<LOD–14.3)	14.7 (11.5–17.6)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Females					
2001–2002	NC	<LOD	<LOD	10.7 (<LOD–14.1)	15.6 (11.1–23.0)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Race/ethnicity					
Mexican Americans					
2001–2002	NC	<LOD	<LOD	<LOD	9.20 (<LOD–11.8
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Non-Hispanic blacks					
2001–2002	NC	<LOD	<LOD	13.9 (<LOD–17.6)	18.3 (13.9–23.0)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD

Table 5-29. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic whites					
2001–2002	NC	<LOD	<LOD	11.3 (<LOD–14.4)	15.1 (12.0–20.5)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD

HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection (LODs for survey years 2001–2002 and 2003–2004 were 9.0 and 11.9 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002.

Source: CDC 2024a

Table 5-30. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	32.6 (28.3–38.2)	56.9 (47.4–67.3)	74.0 (68.3–82.4)
2001–2002	34.6 (29.6–40.6)	39.2 (32.7–44.7)	60.8 (50.3–74.2)	95.2 (76.2–120)	128 (99.4–153)
2003–2004	17.2 (15.7–18.9)	20.0 (17.8–22.9)	36.5 (32.2–40.0)	53.0 (48.1–59.6)	68.5 (59.6–74.9)
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	26.7 (20.2–29.6)
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	16.1 (14.3–18.1)	19.4 (16.4–27.7)
≥20 years					
1999–2000	NC	<LOD	36.2 (31.5–40.7)	62.8 (53.6–69.1)	75.6 (70.5–84.2)
2001–2002	34.6 (29.6–40.6)	39.2 (32.7–44.7)	60.8 (50.3–74.2)	95.2 (76.2–120))	128 (99.4–153
2003–2004	19.7 (17.8–21.8)	23.8 (20.7–26.4)	39.3 (35.4–42.2)	56.6 (49.7–63.8)	70.8 (60.7–82.2)
Gender					
Males					
1999–2000	NC	<LOD	31.5 (23.7–38.2)	55.0 (45.7–64.2)	71.3 (59.4–79.4)
2001–2002	34.1 (28.3–41.1)	38.9 (32.1–44.7)	61.9 (50.0–79.5)	94.9 (70.8–131)	130 (88.5–181)
2003–2004	17.5 (15.5–19.8)	19.8 (17.8–21.6)	35.5 (29.8–40.3)	52.9 (45.4–63.2)	70.2 (57.5–88.7)
Females					
1999–2000	NC	<LOD	34.9 (29.1–39.7)	61.2 (51.0–69.2)	74.9 (68.4–92.2)
2001–2002	35.1 (29.9–41.2)	40.1 (32.4–46.3)	59.8 (49.8–72.3)	97.6 (77.1–114))	126 (108–142
2003–2004	16.9 (15.3–18.6)	20.5 (17.8–24.6)	36.9 (33.2–41.0)	53.6 (48.3–59.6)	65.6 (60.0–73.4)
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	21.3 (<LOD–27.6)	43.3 (34.1–52.3)	58.0 (49.5–64.8)
2001–2002	18.3 (15.6–21.4)	21.2 (19.4–25.0)	31.9 (27.5–40.3)	51.5 (40.3–69.9)	68.3 (48.0–111)
2003–2004			21.1 (16.3–26.5)	32.2 (24.5–47.4)	43.0 (31.5–65.3)

Table 5-30. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	31.9 (26.6–41.2)	56.7 (44.9–74.6)	81.6 (72.2–91.7)
2001–2002	38.9 (33.6–45.0)	40.3 (33.5–47.3)	63.5 (54.6–76.9)	93.9 (78.7–133))	136 (92.6–185
2003–2004	16.2 (12.9–20.4)	18.1 (14.4–21.6)	34.9 (28.4–42.9)	54.5 (44.4–69.4)	74.0 (54.3–122)
Non-Hispanic whites					
1999–2000	NC	<LOD	35.5 (29.7–40.0)	60.9 (51.4–68.3)	74.3 (68.3–83.0)
2001–2002	37.8 (31.5–45.4)	42.8 (33.9–51.2)	65.0 (52.3–82.9)	99.6 (78.4–130)	131 (103–165)
2003–2004	18.7 (17.0–20.6)	22.9 (19.9–26.2)	38.0 (35.2–41.5)	56.6 (48.7–63.8)	69.0 (60.6–74.9)

HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 20.1, 9.1, and 12.3 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002

Source: CDC 2024a

Table 5-31. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	12.5 (10.5–15.3)	17.0 (14.3–20.0)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
≥20 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	12.5 (10.5–15.3)	17.0 (14.3–20.0)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Gender					
Males					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002				12.1 (<LOD–14.8)	15.1 (12.9–18.5)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Females					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002				13.0 (10.7–16.8)	18.3 (15.7–21.1)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	9.60 (<LOD–11.6)	12.2 (<LOD–20.6)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD

Table 5-31. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	14.6 (11.2–20.0)	19.9 (14.6–23.9)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
Non-Hispanic whites					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	12.9 (9.90–15.9)	17.3 (14.7–20.6)
2003–2004	NC	<LOD	<LOD	<LOD	<LOD

HxCDD = hexachlorodibenzo-*p*-dioxin; LOD = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 20.3, 9.3, and 12.3 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002

Source: CDC 2024a

Table 5-32. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	406 (359–453)	674 (597–767)	913 (787–1,010)
2001–2002	346 (<LOD–394)	333 (<LOD–402)	573 (498–668)	944 (780–1,090)	1,260 (998–1,610)
2003–2004	NC	<LOD	336 (283–389)	582 (490–658)	767 (645–913)
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	421 (363–517)
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	244 (<LOD–330)	352 (264–458)
≥20 years					
1999–2000	NC	<LOD	445 (389–496)	710 (624–802))	948 (822–1,080
2001–2002	346 (<LOD–394)	333 (<LOD–402)	573 (498–668)	944 (780–1,090)	1260 (998–1,610)
2003–2004	220 (<LOD–244)	223 (<LOD–243)	358 (297–421)	597 (502–719)	794 (665–978)
Gender					
Males					
1999–2000	NC	<LOD	<LOD	517 (447–580)	704 (563–838)
2001–2002	NC	<LOD	442 (346–579)	767 (593–968)	1,030 (837–1,240)
2003–2004	NC	<LOD	270 (244–320)	457 (377–559)	668 (501–856)
Females					
1999–2000	NC	<LOD	504 (422–579)	802 (674–928)	1,010 (928–1,180)
2001–2002	410 (356–472)	405 (335–502)	647 (574–751)	1,020 (858–1,360)	1,450 (1,060–1,780)
2003–2004	235 (<LOD–256)	238 (225–248)	402 (321–486)	640 (551–749)	829 (675–1,020)
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	418 (365–502)	703 (610–873)	940 (737–1,230)
2001–2002	NC	<LOD	432 (394–545)	755 (578–1,220)	1,150 (696–1,640)
2003–2004	NC	<LOD	296 (225–356)	452 (363–540)	588 (417–861)

Table 5-32. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	444 (371–519)	741 (566–983)	1,120 (799–1,560)
2001–2002	421 (352–503)	420 (339–509)	682 (537–907)	1,110 (956–1,520)	1,640 (1,130–1,900)
2003–2004	NC	<LOD	345 (276–455)	642 (513–883)	926 (636–1,310)
Non-Hispanic whites					
1999–2000	NC	<LOD	391 (333–452)	625 (562–754)	861 (676–1,010)
2001–2002	349 (<LOD–409)	335 (<LOD–421)	574 (496–679)	945 (764–1,170)	1,290 (972–1,660)
2003–2004	NC	<LOD	343 (282–403)	585 (464–674)	758 (635–922)

LODs = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 329.0, 319.0, and 218.0 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002; OCDD = octachlorodibenzo-*p*-dioxin

Source: CDC 2024a

Table 5-33. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	11.3 (9.30–13.6)	15.8 (13.3–19.8)
2003–2004	NC	<LOD	6.10 (5.50–6.80)	9.00 (8.30–9.70)	11.0 (9.90–12.2)
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	<LOD	4.80 (<LOD–5.90)
≥20 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	11.3 (9.30–13.6)	15.8 (13.3–19.8)
2003–2004	NC	<LOD	6.60 (5.90–7.20)	9.30 (8.60–10.1)	11.3 (10.1–12.7)
Gender					
Males					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	10.8 (9.10–13.3)	14.5 (11.7–19.4)
2003–2004	NC	<LOD	5.90 (5.30–6.40)	8.90 (7.90–9.60)	11.0 (9.60–12.7)
Females					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	6.10 (<LOD–7.80)	11.8 (9.40–14.3)	16.6 (13.7–20.8)
2003–2004	NC	<LOD	6.50 (5.70–7.20)	9.10 (8.30–10.1)	11.0 (10.0–12.2)
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	8.70 (<LOD–12.7)
2003–2004	NC	<LOD	<LOD	6.50 (5.20–7.90)	7.80 (6.70–9.20)

Table 5-33. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	7.70 (<LOD–9.30)	13.9 (9.60–18.4)	18.4 (14.2–24.0)
2003–2004	NC	<LOD	6.40 (5.30–8.20)	9.90 (8.50–13.4)	14.4 (9.60–20.1)
Non-Hispanic whites					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	11.7 (9.50–14.3)	16.7 (13.6–20.2)
2003–2004	NC	<LOD	6.50 (5.80–7.10)	9.30 (8.60–10.0)	11.1 (10.1–12.2)

LOD = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 14.2, 6.0, and 4.5 pg/g lipid, respectively); NC = not calculated (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002; PeCDD = pentachlorodibenzo-*p*-dioxin

Source: CDC 2024a

Table 5-34. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Total					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	<LOD
2003–2004	NC	<LOD	<LOD	4.10 (<LOD–4.40)	5.20 (4.30–5.80)
Age group					
12–19 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	ND	ND	ND	ND	ND
2003–2004	NC	<LOD	<LOD	<LOD	<LOD
≥20 years					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	<LOD
2003–2004	NC	<LOD	<LOD	4.30 (3.90–4.60)	5.30 (4.50–6.10)
Gender					
Males					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	<LOD
2003–2004	NC	<LOD	<LOD	<LOD	4.60 (3.80–5.30)
Females					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	6.40 (<LOD–9.20)
2003–2004	NC	<LOD	<LOD	4.40 (4.00–4.90)	5.50 (4.50–6.60)
Race/ethnicity					
Mexican Americans					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	<LOD
2003–2004	NC	<LOD	<LOD	<LOD	3.80 (<LOD–5.50)

Table 5-34. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population

Survey years	Geometric mean (95% confidence limit)	Selected percentiles (95% confidence limit)			
		50th	75th	90th	95th
Non-Hispanic blacks					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	7.50 (<LOD–10.0)
2003–2004	NC	<LOD	<LOD	4.50 (<LOD–6.10)	6.20 (4.40–10.3)
Non-Hispanic whites					
1999–2000	NC	<LOD	<LOD	<LOD	<LOD
2001–2002	NC	<LOD	<LOD	<LOD	<LOD
2003–2004	NC	<LOD	<LOD	4.10 (<LOD–4.50)	5.20 (4.30–5.90)

LOD = limit of detection (LODs for survey years 1999–2000, 2001–2002, and 2003–2004 were 112.1, 5.8, and 3.8 pg/g lipid, respectively); NC = not calculated; (proportion of results below LOD was too high to provide a valid result); ND = data not collected for this age group for survey years 2001–2002; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: CDC 2024a

Table 5-35. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	19.2	1.6	9
			2007–2008	14.5	0.3	6
			2009–2010	13.2	0.5	10
			2011–2012	NC		6
		20–39	2005–2006	19.4	1.4	12
			2007–2008	16.9	1.6	15
			2009–2010	17.1	1	17
			2011–2012	24.3	3.6	12
		40–59	2005–2006	36.1	4.6	12
			2007–2008	21.0	1.7	15
			2009–2010	24.1	2.5	17
			2011–2012	23.0	2.7	12
		≥60	2005–2006	47.8	4.3	15
			2007–2008	36.5	2.1	23
			2009–2010	30.1	1.6	21
			2011–2012	37.1	1.4	12
	Female	12–19	2005–2006	15.0	1	10
			2007–2008	13.0	0.8	7
			2009–2010	9.94	0.9	8
			2011–2012	NC		5
		20–39	2005–2006	20.5	2.1	13
			2007–2008	17.3	0.8	12
			2009–2010	15.1	2.6	18
			2011–2012	15.8	1	13
		40–59	2005–2006	32.2	2.9	13
			2007–2008	22.2	1.8	17
			2009–2010	19.0	0.9	17
			2011–2012	25.0	2.3	11

Table 5-35. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	49.8	3	17
			2007–2008	41.6	3.8	19
			2009–2010	30.8	2.8	14
			2011–2012	49.4	6.2	14
Non-Hispanic blacks	Male	12–19	2005–2006	17.2	0.7	13
			2007–2008	12.2	0.8	6
			2009–2010	8.75	0.59	6
			2011–2012	NC		7
		20–39	2005–2006	16.1	1	6
			2007–2008	16.0	1	6
			2009–2010	11.6	1.5	7
			2011–2012	15.7	1.3	9
		40–59	2005–2006	23.4	1.6	5
			2007–2008	20.8	1.3	6
			2009–2010	17.6	2.6	7
			2011–2012	21.7	3.2	7
		≥60	2005–2006	44.9	5.7	5
			2007–2008	28.9	4.7	8
			2009–2010	24.4	1.6	9
			2011–2012	30.1	3.3	9
	Female	12–19	2005–2006	15.9	1.7	14
			2007–2008	11.0	0.9	4
			2009–2010	8.67	0.72	6
			2011–2012	15.2	3.7	6
		20–39	2005–2006	20.8	1.6	7
			2007–2008	19.9	1.4	7
			2009–2010	13.6	1.9	7
			2011–2012	17.3	2.1	8

Table 5-35. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	36.3	4.3	7
			2007–2008	28.1	1.8	6
			2009–2010	21.9	4.1	7
			2011–2012	33.2	3.7	8
		≥60	2005–2006	77.4	12.5	5
			2007–2008	55.1	5.3	6
			2009–2010	51.0	5.4	7
			2011–2012	50.9	5.6	7
Mexican-American	Male	12–19	2005–2006	15.3	1.1	11
			2007–2008	12.8	1	6
			2009–2010	12.5	0.9	8
			2011–2012	14.1	1.2	5
		20–39	2005–2006	26.0	2.6	9
			2007–2008	18.0	1.2	9
			2009–2010	19.9	2.1	8
			2011–2012	23.0	3.7	4
		40–59	2005–2006	34.5	3.6	4
			2007–2008	29.0	2	6
			2009–2010	29.5	2.2	7
			2011–2012	38.2	4	3
		≥60	2005–2006	40.3	5	4
			2007–2008	28.2	3.2	5
			2009–2010	46.0	7.2	5
			2011–2012	40.2	1.2	2
	Female	12–19	2005–2006	13.2	0.7	16
			2007–2008	11.2	2.3	5
			2009–2010	7.94	0.68	7
			2011–2012	NC		4

Table 5-35. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	25.1	1.9	9
			2007–2008	23.0	2.6	7
			2009–2010	14.0	2.2	10
			2011–2012	17.0	1.9	3
		40–59	2005–2006	41.8	4.3	6
			2007–2008	30.9	3	5
			2009–2010	39.2	4.3	9
			2011–2012	29.9	1.6	3
		≥60	2005–2006	59.1	6.9	3
			2007–2008	53.6	2.3	5
			2009–2010	68.0	7.7	6
			2011–2012	46.5	8.3	3
All Hispanic	Male	12–19	2009–2010	12.4	0.8	11
			2011–2012	14.5	1.1	7
		20–39	2009–2010	16.9	1.9	13
			2011–2012	19.2	2.6	8
		40–59	2009–2010	2.3	2.3	12
			2011–2012	27.7	5.2	6
		≥60	2009–2010	6.3	6.3	8
			2011–2012	30.6	4.3	6
	Female	12–19	2009–2010	8.23	0.56	10
			2011–2012	13.1	1.7	7
		20–39	2009–2010	14.1	1.6	14
			2011–2012	15.6	1.2	7
		40–59	2009–2010	33.3	3.6	14
			2011–2012	24.4	2.3	7
		≥60	2009–2010	53.5	6.9	11
			2011–2012	43.7	4.4	7

Table 5-35. Serum 1,2,3,4,6,7,8-HpCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	27.4	5.9	3
		20–39		30.1	3.9	6
		40–49		26.1	4.7	5
		≥60		37.4	3.4	4
	Female	12–19	2011–2012	19.5	4.3	3
		20–39		34.0	6.5	5
		40–49		50.9	7.5	6
		≥60		33.4	4.6	3

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, and 2011–2012 were 1.8, 0.62, 6.36, and 13.0 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.

NC = not calculated (portion of results below limit of detection was too high to provide a valid result); HpCDD = heptachlorodibenzo-*p*-dioxin

Source: CDC 2024b

Table 5-36. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	NC	NC	9
			2007–2008	1.11	0.23	6
			2009–2010	1.08	0.06	10
			2011–2012	1.26	0.07	6
		20–39	2005–2006	1.44	0.35	12
			2007–2008	1.38	0.24	15
			2009–2010	1.79	0.1	17
			2011–2012	2.10	0.16	12
		40–59	2005–2006	4.47	0.42	12
			2007–2008	2.95	0.16	15
			2009–2010	2.84	0.2	17
			2011–2012	3.08	0.34	12
		≥60	2005–2006	7.15	0.76	15
			2007–2008	5.65	0.48	23
			2009–2010	4.51	0/19	20
			2011–2012	5.20	0.33	12
	Female	12–19	2005–2006	NC	NC	10
			2007–2008	NC	NC	7
			2009–2010	0.767	0.094	8
			2011–2012	0.79	0.064	5
		20–39	2005–2006	NC	NC	16
			2007–2008	1.10	0.18	12
			2009–2010	1.19	0.09	18
			2011–2012	1.49	0.13	12
		40–59	2005–2006	3.59	0.2	13
			2007–2008	2.45	0.21	17
			2009–2010	2.42	0.11	17
			2011–2012	2.86	0.21	11

Table 5-36. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	6.40	0.33	17
			2007–2008	5.71	0.43	20
			2009–2010	4.93	0.38	22
			2011–2012	6.44	0.61	14
Non-Hispanic blacks	Male	12–19	2005–2006	NC	NC	13
			2007–2008	NC	NC	6
			2009–2010	0.705	0.137	5
			2011–2012	1.37	0.26	7
		20–39	2005–2006	1.56	0.33	6
			2007–2008	1.06[d]	0.35	6
			2009–2010	1.17	0.15	7
			2011–2012	1.88	0.08	9
		40–59	2005–2006	2.78	0.37	5
			2007–2008	2.93	0.16	6
			2009–2010	2.34	0.22	7
			2011–2012	3.05	0.35	7
		≥60	2005–2006	6.65	0.19	5
			2007–2008	4.83	0.75	8
			2009–2010	3.85	0.34	9
			2011–2012	5.40	0.36	9
	Female	12–19	2005–2006	NC	NC	14
			2007–2008	NC	NC	4
			2009–2010	0.800	0.097	6
			2011–2012	0.81	0.159	5
		20–39	2005–2006	1.87	0.24	7
			2007–2008	0.972[d]	0.312	7
			2009–2010	1.23	0.13	7
			2011–2012	1.21	0.09	8

Table 5-36. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	3.83	0.44	7
			2007–2008	3.34	0.39	6
			2009–2010	2.49	0.36	7
			2011–2012	3.81	0.43	8
		≥60	2005–2006	12.7	1.7	5
			2007–2008	9.88	1.5	6
			2009–2010	6.79	0.82	7
			2011–2012	8.22	0.83	7
Mexican-American	Male	12–19	2005–2006	NC	NC	11
			2007–2008	NC	NC	6
			2009–2010	1.01	0.1	8
			2011–2012	1.08	0.07	5
		20–39	2005–2006	NC	NC	9
			2007–2008	1.44	0.19	9
			2009–2010	1.44	0.17	8
			2011–2012	2.08	0.14	4
		40–59	2005–2006	2.65[d]	0.93	4
			2007–2008	2.91	0.18	6
			2009–2010	2.66	0.13	7
			2011–2012	3.72	0.39	3
		≥60	2005–2006	4.96	0.17	4
			2007–2008	4.94	0.19	5
			2009–2010	5.68	0/82	5
			2011–2012	4.39	1.17	2
	Female	12–19	2005–2006	NC	NC	16
			2007–2008	NC	NC	5
			2009–2010	0.544	0.043	7
			2011–2012	NC		4

Table 5-36. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	NC	NC	9
			2007–2008	1.47	0.25	7
			2009–2010	0.944	0.133	10
			2011–2012	1.51	0.35	3
		40–59	2005–2006	3.64	0.8	6
			2007–2008	3.03	0.4	5
			2009–2010	2.76	0.18	9
			2011–2012	2.65	0.18	3
		≥60	2005–2006	7.30	0.67	3
			2007–2008	6.26	0.61	5
			2009–2010	6.95	0.96	6
			2011–2012	5.61	0.76	3
All Hispanics	Male	12–19	2009–2010	0.954	0.081	11
			2011–2012	1.03	0.007	7
		20–39	2009–2010	1.30	0.14	13
			2011–2012	1.80	0.15	8
		40–59	2009–2010	2.41	0.15	12
			2011–2012	2.78	0.5	6
		≥60	2009–2010	4.75	0.68	8
			2011–2012	3.90	0.47	6
	Female	12–19	2009–2010	0.573	0.036	10
			2011–2012	0.74	0.147	7
		20–39	2009–2010	0.979	0.096	14
			2011–2012	1.24	0.22	6
		40–59	2009–2010	2.46	0.19	14
			2011–2012	2.30	0.18	7
		≥60	2009–2010	5.85	0.71	11
			2011–2012	5.14	0.4	7

Table 5-36. Serum 1,2,3,4,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	1.23	0.14	3
		20–39		1.72	0.2	6
		40–59		2.22	0.41	6
		≥60		2.46	0.57	4
	Female	12–19	2011–2012	0.70	0.21	2
		20–39		1.70	0.27	6
		40–59		2.33	0.24	6
		≥60		4.42[d]	1.41	3

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, and 2011–2012 were 0.14, 0.26, 0.4, and 0.4 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Unadjusted standard error of the mean estimate is >30%.

HxCDD = hexachlorodibenzo-*p*-dioxin; NC = not calculated (portion of results below limit of detection was too high to provide a valid result)

Source: CDC 2024b

Table 5-37. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	10.5	0.8	9
			2007–2008	9.78	0.47	6
			2009–2010	7.25	0.47	10
			2011–2012	7.52	0.76	6
		20–39	2005–2006	14.7	1.4	12
			2007–2008	14.2	0.6	15
			2009–2010	11.9	0.6	17
			2011–2012	13.2	1.1	12
		40–59	2005–2006	31.8	3.4	12
			2007–2008	23.0	1	15
			2009–2010	23.4	1.5	17
			2011–2012	19.9	2	11
		≥60	2005–2006	46.6	3.4	15
			2007–2008	40.5	2.6	23
			2009–2010	36.5	2.1	20
			2011–2012	41.8	2.5	11
	Female	12–19	2005–2006	6.76	0.95	10
			2007–2008	6.86	0.36	7
			2009–2010	5.69	0.61	8
			2011–2012	5.83	057	5
		20–39	2005–2006	13.1	1.4	16
			2007–2008	12.5	0.8	12
			2009–2010	10.8	1.1	18
			2011–2012	10.2	0.7	13
		40–59	2005–2006	25.6	1.2	13
			2007–2008	23.7	0.9	17
			2009–2010	19.5	0.9	17
			2011–2012	20.8	1.4	7

Table 5-37. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	47.4	3.5	17
			2007–2008	45.7	2.8	20
			2009–2010	40.8	2.8	22
			2011–2012	48.6	4.3	13
Non-Hispanic blacks	Male	12–19	2005–2006	9.16	0.4	13
			2007–2008	7.77	0.76	6
			2009–2010	6.71	0.71	6
			2011–2012	7.76	0.9	7
		20–39	2005–2006	12.7	0.8	6
			2007–2008	13.4	1.1	6
			2009–2010	8.65	1.1	7
			2011–2012	10.2	0.5	9
		40–59	2005–2006	20.4	1.3	5
			2007–2008	23.6	2.3	6
			2009–2010	18.2	1.8	7
			2011–2012	20.8	1.4	7
		≥60	2005–2006	45.5	6.1	5
			2007–2008	36.3	4.1	8
			2009–2010	31.0	2.1	9
			2011–2012	39.0	2.9	8
	Female	12–19	2005–2006	5.50	0.65	14
			2007–2008	6.07	0.32	4
			2009–2010	4.70	0.41	6
			2011–2012	NC		6
		20–39	2005–2006	12.2	0.8	7
			2007–2008	13.7	1.2	7
			2009–2010	9.23	0.94	7
			2011–2012	9.38	1.32	7

Table 5-37. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	25.1	2.6	7
			2007–2008	25.8	1.3	6
			2009–2010	19.8	1.8	7
			2011–2012	23.9	1.8	7
		≥60	2005–2006	69.4	8	5
			2007–2008	54.2	4.7	6
			2009–2010	49.8	5.3	7
			2011–2012	49.1	3.3	7
Mexican-American	Male	12–19	2005–2006	4.24	1.01	11
			2007–2008	7.12	0.78	6
			2009–2010	6.08	0.48	8
			2011–2012	6.46	1.38	4
		20–39	2005–2006	9.78	0.77	9
			2007–2008	10.4	0.4	9
			2009–2010	8.73	0.74	8
			2011–2012	10.2	0.9	4
		40–59	2005–2006	18.9	2.8	4
			2007–2008	19.4	1.2	6
			2009–2010	19.0	1.1	7
			2011–2012	20.8	1.7	3
		≥60	2005–2006	34.5	2.4	4
			2007–2008	32.0	1.7	5
			2009–2010	38.3	5.7	5
			2011–2012	39.0	2.9	8
	Female	12–19	2005–2006	3.62	0.73	16
			2007–2008	4.15	1.24	5
			2009–2010	3.95	0.2	7
			2011–2012	NC		4

Table 5-37. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	8.51	0.79	9
			2007–2008	9.61	0.78	7
			2009–2010	6.40	0.97	10
			2011–2012	8.84	1.97	3
		40–59	2005–2006	18.9	1.5	6
			2007–2008	19.5	1.7	5
			2009–2010	18.4	1.4	9
			2011–2012	19.0	1.1	2
		≥60	2005–2006	36.4	3.3	3
			2007–2008	37.0	3.9	5
			2009–2010	43.6	6.2	6
			2011–2012	41.5	3.2	3
All Hispanics	Male	12–19	2009–2010	6.11	0.38	11
			2011–2012	6.59	1.01	6
		20–39	2009–2010	8.06	0.83	13
			2011–2012	9.25	0.63	8
		40–59	2009–2010	16.8	1.1	12
			2011–2012	17.0	2	6
		≥60	2009–2010	32.9	4.4	8
			2011–2012	24.2	1.8	5
	Female	12–19	2009–2010	4.14	0.24	10
			2011–2012	NC		7
		20–39	2009–2010	6.82	0.69	14
			2011–2012	7.61	0.98	7
		40–59	2009–2010	17.3	1	14
			2011–2012	16.7	1.3	6
		≥60	2009–2010	37.9	4.1	11
			2011–2012	37.8	3.8	7

Table 5-37. Serum 1,2,3,6,7,8-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	7.89	0.17	3
		20–39		10.2	1.3	6
		40–59		14.4	1.7	5
		≥60		17.4	1.2	4
	Female	12–19	2011–2012	NC		3
		20–39		9.95	1.24	6
		40–59		16.3	2.7	6
		≥60		25.9[d]	8.9	3

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, and 2011–2012 were 0.09, 0.09, 0.31, and 4.3 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Unadjusted standard error of the mean estimate is >30%

HxCDD = hexachlorodibenzo-*p*-dioxin; NC = not calculated (proportion of results below limit of detection was too high to provide valid result)

Source: CDC 2024b

Table 5-38. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	1.68	0.43	9
			2007–2008	2.18	0.1	6
			2009–2010	1.89	0.07	10
			2011–2012	1.77	0.16	5
		20–39	2005–2006	1.92	0.26	12
			2007–2008	2.33	0.28	15
			2009–2010	2.25	0.1	17
			2011–2012	2.80	0.26	9
		40–59	2005–2006	3.99	0.38	12
			2007–2008	2.99	0.15	15
			2009–2010	3.06	0.17	17
			2011–2012	2.93	.29	11
		≥60	2005–2006	5.48	0.47	15
			2007–2008	4.80	0.3	23
			2009–2010	4.56	0.25	21
			2011–2012	5.35	0.48	10
	Female	12–19	2005–2006	NC	NC	10
			2007–2008	1.64	0.37	7
			2009–2010	1.60	0.21	8
			2011–2012	1.71	0.28	4
		20–39	2005–2006	2.47	0.2	16
			2007–2008	2.29	0.27	12
			2009–2010	2.04	0.21	18
			2011–2012	2.22	0.1	9
		40–59	2005–2006	3.93	0.25	13
			2007–2008	3.29	0.24	17
			2009–2010	2.91	0.14	17
			2011–2012	3.47	0.44	11

Table 5-38. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	6.02	0.36	17
			2007–2008	6.23	0.4	20
			2009–2010	5.59	0.35	22
			2011–2012	6.75	0.65	13
Non-Hispanic blacks	Male	12–19	2005–2006	NC	NC	13
			2007–2008	1.73	0.28	6
			2009–2010	1.49	0.03	5
			2011–2012	2.18	0.31	6
		20–39	2005–2006	2.08	0.11	6
			2007–2008	1.37[d]	0.43	6
			2009–2010	1.52	0.18	7
			2011–2012	1.76	0.1	7
		40–59	2005–2006	2.57	0.18	5
			2007–2008	3.26	0.3	6
			2009–2010	2.36	0.25	7
			2011–2012	3.04	0.53	4
		≥60	2005–2006	5.18	0.91	5
			2007–2008	4.15	0.49	8
			2009–2010	3.46	0.33	9
			2011–2012	4.09	0.32	8
	Female	12–19	2005–2006	NC	NC	14
			2007–2008	NC	NC	4
			2009–2010	1.23	0.14	6
			2011–2012	1.32	0.18	4
		20–39	2005–2006	2.44	0.23	7
			2007–2008	1.92	0.57	7
			2009–2010	1.89	0.18	7
			2011–2012	2.37	0.2	6

Table 5-38. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	3.84	0.36	7
			2007–2008	3.97	0.26	6
			2009–2010	3.17	0.41	7
			2011–2012	5.47	1.04	4
		≥60	2005–2006	9.10	1.09	5
			2007–2008	8.05	0.79	6
			2009–2010	6.54	0.63	7
			2011–2012	6.76	0.74	6
Mexican-American	Male	12–19	2005–2006	NC	NC	11
			2007–2008	NC	NC	6
			2009–2010	1.67	0.09	7
			2011–2012	1.67	0.17	4
		20–39	2005–2006	2.20	0.29	9
			2007–2008	1.57	0.33	9
			2009–2010	1.67	0.15	8
			2011–2012	2.19	0.22	4
		40–59	2005–2006	3.37	0.53	4
			2007–2008	2.47	0.55	6
			2009–2010	2.73	0.18	7
			2011–2012	3.14	0.15	3
		≥60	2005–2006	4.52	0.22	4
			2007–2008	4.66	0.33	5
			2009–2010	5.43	0.74	5
			2011–2012	ND[e]		1
	Female	12–19	2005–2006	NC	NC	16
			2007–2008	1.46	0.43	5
			2009–2010	1.16	0.11	7
			2011–2012	1.09	0.27	4

Table 5-38. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	1.81	0.32	9
			2007–2008	NC	NC	7
			2009–2010	1.64	0.24	10
			2011–2012	1.97	0.45	3
		40–59	2005–2006	4.02	0.32	6
			2007–2008	3.94	0.29	5
			2009–2010	4.04	0.32	9
			2011–2012	3.88	0.34	2
		≥60	2005–2006	6.50	0.96	3
			2007–2008	7.48	0.57	5
			2009–2010	7.73	0.92	6
			2011–2012	7.23	0.57	3
All Hispanics	Male	12–19	2009–2010	1.77	0.12	10
			2011–2012	1.73	0.14	6
		20–39	2009–2010	1.74	0.12	13
			2011–2012	2.03	0.16	7
		40–59	2009–2010	2.50	0.14	12
			2011–2012	2.67	0.23	6
		≥60	2009–2010	4.67	0.6	8
			2011–2012	3.83	0.65	4
	Female	12–19	2009–2010	1.20	0.08	10
			2011–2012	1.21	0.2	6
		20–39	2009–2010	1.70	0.17	14
			2011–2012	2.03	0.22	6
		40–59	2009–2010	3.47	0.3	14
			2011–2012	3.24	0.36	5
		≥60	2009–2010	6.25	0.74	11
			2011–2012	6.26	0.75	7

Table 5-38. Serum 1,2,3,7,8,9-HxCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	1.90	0.22	3
		20–39		2.32	0.22	6
		40–59		2.43	0.27	5
		≥60		2.67	0.18	4
	Female	12–19	2011–2012	1.35	0.1	2
		20–39		2.49	0.3	6
		40–59		3.34	0.37	6
		≥60		4.51	0.33	2

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, and 2011–2012 were 0.07, 0.1, 0.2, and 0.37 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Unadjusted standard error of the mean estimate is >30%.
[e]Weighted arithmetic means and their standard errors are not available for strata consisting of a single pool.

HxCDD = hexachlorodibenzo-*p*-dioxin; NC = not calculated (portion of results below limit of detection was too high to provide a valid result); ND = not determined

Source: CDC 2024b

Table 5-39. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	123	13	9
			2007–2008	114	7	6
			2009–2010	96.4	7.3	10
			2011–2012	NC		4
		20–39	2005–2006	129	8	12
			2007–2008	125	12	15
			2009–2010	109	5	17
			2011–2012	154	30	7
		40–59	2005–2006	238	25	12
			2007–2008	164	10	15
			2009–2010	172	16	17
			2011–2012	142	9	10
		≥60	2005–2006	379	38	15
			2007–2008	306	23	23
			2009–2010	284	18	21
			2011–2012	223	21	9
	Female	12–19	2005–2006	106	9	10
			2007–2008	104	5	7
			2009–2010	83.6	6.2	8
			2011–2012	NC		5
		20–39	2005–2006	179	19	16
			2007–2008	139	7	12
			2009–2010	126	11	18
			2011–2012	107	5	9
		40–59	2005–2006	304	22	13
			2007–2008	222	15	17
			2009–2010	198	10	17
			2011–2012	231	19	9

Table 5-39. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	546	66	17
			2007–2008	435	49	19
			2009–2010	407	31	22
			2011–2012	437	77	12
Non-Hispanic blacks	Male	12–19	2005–2006	142	12	13
			2007–2008	114	10	6
			2009–2010	90.2	4.3	5
			2011–2012	96.9	14.1	6
		20–39	2005–2006	127	9	6
			2007–2008	136	9	6
			2009–2010	102	6	7
			2011–2012	101	12	7
		40–59	2005–2006	200	32	5
			2007–2008	190	16	6
			2009–2010	161	14	7
			2011–2012	155	27	5
		≥60	2005–2006	420	82	5
			2007–2008	314	30	8
			2009–2010	265	11	9
			2011–2012	241	19	8
	Female	12–19	2005–2006	153	22	14
			2007–2008	100	9	4
			2009–2010	81.7	5.6	6
			2011–2012	NC		3
		20–39	2005–2006	188	18	7
			2007–2008	197	23	7
			2009–2010	139	15	7
			2011–2012	122	13	7

Table 5-39. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	381	43	7
			2007–2008	303	22	6
			2009–2010	239	29	7
			2011–2012	338	72	3
		≥60	2005–2006	927	135	5
			2007–2008	660	78	6
			2009–2010	672	133	7
			2011–2012	490	44	6
Mexican-American	Male	12–19	2005–2006	111	9	11
			2007–2008	117	9	6
			2009–2010	98.4	10.6	8
			2011–2012	NC		2
		20–39	2005–2006	180	21	9
			2007–2008	133	9	9
			2009–2010	130	12	8
			2011–2012	112	12	4
		40–59	2005–2006	220	19	4
			2007–2008	217	13	6
			2009–2010	231	32	7
			2011–2012	219	50	2
		≥60	2005–2006	350	46	4
			2007–2008	273	17	5
			2009–2010	413	78	5
			2011–2012	ND[d]		1
	Female	12–19	2005–2006	111	5	16
			2007–2008	110	13	5
			2009–2010	79.3	5.7	6
			2011–2012	NC		3

Table 5-39. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	184	12	9
			2007–2008	174	12	7
			2009–2010	123	19	10
			2011–2012	114	12	4
		40–59	2005–2006	410	60	6
			2007–2008	290	17	5
			2009–2010	336	34	9
			2011–2012	241	2	2
		≥60	2005–2006	540	65	3
			2007–2008	434	33	5
			2009–2010	552	52	6
			2011–2012	ND[d]		1
All Hispanics	Male	12–19	2009–2010	109	11	11
			2011–2012	NC		3
		20–39	2009–2010	128	13	13
			2011–2012	108	7	7
		40–59	2009–2010	200	22	12
			2011–2012	157	31	5
		≥60	2009–2010	361	68	7
			2011–2012	228	49	3
	Female	12–19	2009–2010	78.4	4.7	10
			2011–2012	NC		4
		20–39	2009–2010	121	14	14
			2011–2012	104	11	8
		40–59	2009–2010	286	29	14
			2011–2012	228	37	4
		≥60	2009–2010	458	48	11
			2011–2012	446	94	3

Table 5-39. Serum 1,2,3,4,6,7,8,9-OCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	ND[d]		1
		20–39		174	13	6
		40–59		168	21	5
		≥60		253	22	3
	Female	12–19	2011–2012	135	30	3
		20–39		233	13	4
		40–59		313	20	4
		≥60		389	88	2

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, 2011–2012 were 8.88, 10.1, 33.9, and 92.0 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Weighted arithmetic means and their standard errors are not available for strata consisting of a single pool.

NC = not calculated (proportion of results below limit of detections was too high to provide a valid result); ND = not determined; OCDD = octachlorodibenzo-*p*-dioxin

Source: CDC 2024b

Table 5-40. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	2.55	0.14	9
			2007–2008	2.31	0.29	6
			2009–2010	2.22	0.1	10
			2011–2012	1.69	0.24	6
		20–39	2005–2006	2.49	0.23	12
			2007–2008	2.93	0.28	15
			2009–2010	2.59	0.17	17
			2011–2012	2.36	0.18	12
		40–59	2005–2006	4.48	0.54	12
			2007–2008	3.96	0.13	15
			2009–2010	3.77	0.21	17
			2011–2012	3.36	0.28	12
		≥60	2005–2006	7.26	0.4	15
			2007–2008	7.08	0.76	23
			2009–2010	6.03	0.24	20
			2011–2012	5.77	0.35	12
	Female	12–19	2005–2006	1.55	0.22	10
			2007–2008	1.64	0.33	7
			2009–2010	1.70	0.24	8
			2011–2012	1.53	0.21	5
		20–39	2005–2006	2.57	0.23	16
			2007–2008	2.40	0.27	12
			2009–2010	2.06	0.12	17
			2011–2012	1.75	0.14	13
		40–59	2005–2006	4.25	0.2	13
			2007–2008	4.12	0.16	17
			2009–2010	3.65	0.17	17
			2011–2012	3.29	0.19	11

Table 5-40. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	7.83	0.56	17
			2007–2008	8.09	0.41	20
			2009–2010	7.06	0.48	22
			2011–2012	5.77	0.35	12
Non-Hispanic blacks	Male	12–19	2005–2006	2.34	0.15	13
			2007–2008	2.21	0.42	6
			2009–2010	NC	NC	6
			2011–2012	1.69	0.27	7
		20–39	2005–2006	2.95	0.16	6
			2007–2008	2.94	0.26	6
			2009–2010	2.14	0.29	5
			2011–2012	1.87	0.23	9
		40–59	2005–2006	4.00	0.24	5
			2007–2008	4.41	0.34	6
			2009–2010	2.81	0.31	7
			2011–2012	3.17	0.62	7
		≥60	2005–2006	8.16	1.26	5
			2007–2008	6.56	0.8	8
			2009–2010	5.33	0.38	9
			2011–2012	5.79	0.37	9
	Female	12–19	2005–2006	NC	NC	14
			2007–2008	NC	NC	4
			2009–2010	NC	NC	5
			2011–2012	0.94	0.249	6
		20–39	2005–2006	2.52	0.1	7
			2007–2008	2.61	0.29	7
			2009–2010	2.17	0.19	7
			2011–2012	1.61	0.17	8

Table 5-40. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	4.64	0.44	7
			2007–2008	4.40	0.44	6
			2009–2010	4.05	0.37	7
			2011–2012	3.70	0.4	8
		≥60	2005–2006	12.5	1.7	5
			2007–2008	11.4	1	6
			2009–2010	8.98	0.84	7
			2011–2012	8.50	0.78	7
Mexican-American	Male	12–19	2005–2006	NC	NC	11
			2007–2008	1.84	0.12	6
			2009–2010	1.87	0.31	8
			2011–2012	1.20	0.11	5
		20–39	2005–2006	2.13	0.26	9
			2007–2008	2.48	0.29	9
			2009–2010	2.09	0.17	8
			2011–2012	2.21	0.14	4
		40–59	2005–2006	3.61	0.63	4
			2007–2008	3.96	0.34	6
			2009–2010	3.45	0.19	7
			2011–2012	4.19	0.24	3
		≥60	2005–2006	5.59	0.16	4
			2007–2008	6.07	0.17	5
			2009–2010	6.57	1.05	5
			2011–2012	ND[d]		1
	Female	12–19	2005–2006	1.14	0.16	16
			2007–2008	1.53	0.28	5
			2009–2010	NC	NC	7
			2011–2012	0.74	0.185	4

Table 5-40. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	1.65	0.23	9
			2007–2008	2.81	0.3	7
			2009–2010	NC	NC	10
			2011–2012	1.22[e]	0.58	4
		40–59	2005–2006	3.23	0.62	6
			2007–2008	4.15	0.68	5
			2009–2010	3.51	0.26	9
			2011–2012	3.07	0.52	3
		≥60	2005–2006	6.48	0.82	3
			2007–2008	6.43	0.64	5
			2009–2010	7.45	0.93	6
			2011–2012	6.68	0.44	3
All Hispanics	Male	12–19	2009–2010	1.78	0.25	11
			2011–2012	1.27	0.15	7
		20–39	2009–2010	1.83	0.18	13
			2011–2012	1.84	0.21	8
		40–59	2009–2010	3.06	0.21	11
			2011–2012	3.05	0.61	6
		≥60	2009–2010	6.07	0.84	7
			2011–2012	4.02	0.61	5
	Female	12–19	2009–2010	NC	NC	10
			2011–2012	0.97	0.164	7
		20–39	2009–2010	NC	NC	14
			2011–2012	1.22	0.32	7
		40–59	2009–2010	3.25	0.2	14
			2011–2012	2.61	0.28	7
		≥60	2009–2010	6.22	0.73	11
			2011–2012	6.58	0.5	7

Table 5-40. Serum 1,2,3,7,8-PeCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	1.71	0.36	3
		20–39		2.58	0.29	6
		40–59		3.65	0.56	5
		>60		4.02	0.19	4
	Female	12–19	2011–2012	1.30	0.16	3
		20-39		1.63	0.34	6
		40–59		3.19	0.35	6
		>60		5.26	0.81	3

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, 2011–2012 were 0.51, 1.07, 1.56, and 0.43 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Weighted arithmetic means and their standard errors are not available for strata consisting of a single pool.
[e]Unadjusted standard error of the mean is >30%.

NC = not calculated (portion of results below limit of detection was too high to provide a valid result); ND = not determined; PeCDD = pentachlorodibenzo-*p*-dioxin

Source: CDC 2024b

Table 5-41. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Non-Hispanic whites	Male	12–19	2005–2006	NC	NC	9
			2007–2008	NC	NC	6
			2009–2010	NC	NC	10
			2011–2012	NC	NC	6
		20–39	2005–2006	NC	NC	12
			2007–2008	NC	NC	15
			2009–2010	NC	NC	17
			2011–2012	NC	NC	12
		40–59	2005–2006	1.21	0.16	12
			2007–2008	0.931	0.086	15
			2009–2010	NC	NC	17
			2011–2012	0.88	0.101	12
		≥60	2005–2006	2.34	0.19	15
			2007–2008	2.13	0.22	23
			2009–2010	1.52	0.1	20
			2011–2012	1.54	0.09	12
	Female	12–19	2005–2006	NC	NC	10
			2007–2008	NC	NC	7
			2009–2010	NC	NC	8
			2011–2012	NC	NC	5
		20–39	2005–2006	NC	NC	16
			2007–2008	NC	NC	12
			2009–2010	NC	NC	18
			2011–2012	NC	NC	13
		40–59	2005–2006	1.31	0.19	13
			2007–2008	1.32	0.07	17
			2009–2010	NC	NC	17
			2011–2012	1.10	0.06	11

Table 5-41. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		≥60	2005–2006	2.86	0.21	17
			2007–2008	2.98	0.2	20
			2009–2010	2.07	0.19	22
			2011–2012	2.50	0.22	14
Non-Hispanic blacks	Male	12–19	2005–2006	NC	NC	13
			2007–2008	NC	NC	6
			2009–2010	NC	NC	5
			2011–2012	NC	NC	7
		20–39	2005–2006	NC	NC	6
			2007–2008	NC	NC	6
			2009–2010	NC	NC	7
			2011–2012	NC	NC	9
		40–59	2005–2006	1.26	0.18	5
			2007–2008	1.32	0.9	6
			2009–2010	NC	NC	7
			2011–2012	0.72	0.139	7
		≥60	2005–2006	3.47[d]	1.25	5
			2007–2008	2.04	0.42	8
			2009–2010	1.34	0.13	9
			2011–2012	1.50	0.12	9
	Female	12–19	2005–2006	NC	NC	14
			2007–2008	NC	NC	4
			2009–2010	NC	NC	5
			2011–2012	NC	NC	6
		20–39	2005–2006	0.712	0.117	7
			2007–2008	NC	NC	7
			2009–2010	NC	NC	7
			2011–2012	0.58	0.059	7

Table 5-41. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		40–59	2005–2006	1.81	0.21	7
			2007–2008	1.68	0.15	6
			2009–2010	1.17	0.22	7
			2011–2012	1.12	0.15	8
		≥60	2005–2006	6.40	1.64	5
			2007–2008	4.50	0.46	6
			2009–2010	3.54	0.37	7
			2011–2012	2.93	0.2	8
Mexican-American	Male	12–19	2005–2006	NC	NC	11
			2007–2008	NC	NC	6
			2009–2010	NC	NC	7
			2011–2012	NC	NC	5
		20–39	2005–2006	NC	NC	9
			2007–2008	NC	NC	9
			2009–2010	NC	NC	7
			2011–2012	0.62	0.108	4
		40–59	2005–2006	1.01	0.24	4
			2007–2008	0.984	0.188	6
			2009–2010	NC	NC	7
			2011–2012	1.01	0.09	3
		≥60	2005–2006	1.29	0.25	4
			2007–2008	1.80	0.21	5
			2009–2010	1.95	0.38	5
			2011–2012	ND[e]		1
	Female	12–19	2005–2006	NC	NC	16
			2007–2008	NC	NC	5
			2009–2010	NC	NC	7
			2011–2012	NC	NC	4

Table 5-41. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
		20–39	2005–2006	NC	NC	9
			2007–2008	0.925	0.121	7
			2009–2010	NC	NC	10
			2011–2012	NC	NC	3
		40–59	2005–2006	1.08	0.3	6
			2007–2008	1.03[d]	0.32	5
			2009–2010	NC	NC	9
			2011–2012	0.84	0.189	3
		≥60	2005–2006	2.34	0.3	3
			2007–2008	2.95	0.32	5
			2009–2010	2.30	0.29	6
			2011–2012	1.81	0.07	3
Mexican-American	Male	12–19	2009–2010	NC	NC	9
			2011–2012	NC	NC	7
		20–39	2009–2010	NC	NC	12
			2011–2012	0.54	0.07	8
		40–59	2009–2010	NC	NC	11
			2011–2012	0.83	0.097	6
		≥60	2009–2010	1.82	0.29	7
			2011–2012	1.25	0.2	5
	Female	12–19	2009–2010	NC	NC	10
			2011–2012	NC	NC	7
		20–39	2009–2010	NC	NC	14
			2011–2012	NC	NC	7
		40–59	2009–2010	NC	NC	14
			2011–2012	0.96	0.122	7
		≥60	2009–2010	2.30	0.2	11
			2011–2012	2.25	0.26	7

Table 5-41. Serum 2,3,7,8-TCDD (Lipid Adjusted) Concentrations (pg/g Lipid) for the U.S. Population[a]

Race/ethnicity	Gender	Age (years)	Survey (years)	Weighted arithmetic mean[b]	Unadjusted standard error	Number of pools[c]
Asians	Male	12–19	2011–2012	NC	NC	3
		20–39		0.71	0.081	6
		40–59		1.09	0.18	5
		≥60		1.27	0.09	4
	Female	12–19	2011–2012	NC	NC	3
		20–39		0.77	0.124	6
		40–59		1.07	0.08	6
		≥60		2.03	0.41	3

[a]Limits of detection for survey years 2005–2006, 2007–2008, 2009–2010, 2011–2012 were 0.39, 0.74, 1.1, and 0.45 pg/g lipid, respectively.
[b]Weighted arithmetic means are not comparable to weighted geometric means; unadjusted standard errors do not incorporate survey design effects.
[c]Each pool was composed of serum from eight persons.
[d]Unadjusted standard error of the mean estimate is >30%.
[e]Weighted arithmetic means and their standard errors are not available for strata consisting of a single pool.

NC = not calculated (portion of results below limit of detection was too high to provide a valid result); ND = not determined; TCDD = tetrachlorodibenzo-*p*-dioxin

Source: CDC 2024b

Patterson et al. (2008, 2009) reported the TEQs for dioxin-like compounds (CDDs, CDFs, coplanar PCBs, and mono-ortho-substituted PCBs) for survey years 2001–2002 and 2003–2004; these values are presented in Table 5-42. The blood TEQs of adults for the 2003–2004 monitoring period appear to be lower than levels in 2001–2002. LaKind et al. (2009) examined the temporal changes in serum CDD/CDF in adults for NHANES survey years 1999–2000, 2001–2002, and 2003–2004 (data summarized in Table 5-43) and found no significant change in median (50th percentile) serum CDD/CDF levels from 1999–2000 to 2001–2002; however, there was a significant decrease in CDD/CDF serum concentration in the 2003–2004 survey year. When the participants were divided by age, 56 and 38% decreases in serum CDD/CDF levels were observed for the 2003–2004 survey year in the 12–19 and 20–39-year-olds, respectively, as compared to the 1999–2000 survey year. A slight nonsignificant decrease (6%) was observed for 40–59-year-olds and a slight increase (12%) was observed for 60+-year-olds.

Table 5-42. Blood TEQ Levels[a] for Dioxin-Like Compounds (CDDs, CDFs, and select PCBs) Levels (pg/g Lipid) at 90th and 95th Percentiles by Age Group in NHANES 2001–2002 and 2003–2004 Survey Years

	TEQ for 2001–2002 survey years		TEQ for 2003–2004 survey years	
	90th percentile	95th percentile	90th percentile	95th percentile
Total, ≥12 years	No data[b]	No data	30.9 (28.2–33.9)[c]	37.8 (35.3–43.4)
Total, ≥20 years	41.0 (35.8–47.1)	56.1 (47.6–65.4)	32.5 (29.2–35.7	39.9 (36.6–45.7)
Age group				
12–29 years	No data	No data	12.1 (10.9–13.0)	14.0 (12.4–15.9)
20–39 years	23.0 (19.7–25.2)	26.2 (23.7–32.5)	16.2 (14.5–17.7)	18.7 (16.9–20.1)
40–49 years	35.4 (29.7–44.8)	46.9 (36.4–66.1)	28.2 (23.7–32.6)	32.0 (28.0–45.3)
≥60 years	67.7 (56.4–79.7)	79.7 (68.2–96.3)	49.7 (41.5–58.6)	63.2 (50.9–75.1)

[a]TEQs calculated using WHO 2005 toxic equivalency factors (TEFs).
[b]Data were not collected for this age group in the 2001–2002 survey.
[c]95% confidence interval.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; NHANES = National Health and Nutrition Examination Survey; PCB = polychlorinated biphenyl; TEQ = toxic equivalency; WHO = World Health Organization

Source: Patterson et al. 2008, 2009

Table 5-43. Serum CDD/CDF Concentrations for Mean and Selected Percentiles for the NHANES 1999–2000, 2001–2002, and 2003–2004 Survey Years

	Serum CDD/CDF concentrations (pg TEQ/g lipid)		
Percentile	1999–2000	2001–2002	2003–2004
10	8.36 (8.03–8.71)[a]	7.76 (7.07–8.07)	5.17 (4.92–5.50)
25	10.52 (10.17–10.99)	10.38 (10.17–10.80)	7.29 (6.95–7.68)

Table 5-43. Serum CDD/CDF Concentrations for Mean and Selected Percentiles for the NHANES 1999–2000, 2001–2002, and 2003–2004 Survey Years

	Serum CDD/CDF concentrations (pg TEQ/g lipid)		
Percentile	1999–2000	2001–2002	2003–2004
50	13.46 (12.92–13.81)	13.98 (13.42–14.59)	11.39 (10.60–12.15)
75	17.68 (17.04–18.30)	20.88 (19.57–22.12)	17.71 (16.61–18.65)
95	27.68 (24.90–29.65)	44.45 (40.11–48.79)	30.62 (28.51–32.34)
Arithmetic mean	15.4 (14.68–15.94)	18.05 (17.25–18.78)	13.90 (13.35–14.42)

[a]95% confidence interval.

CDD = chlorinated dibenzo-*p*-dioxin; CDF = chlorinated dibenzofuran; NHANES = National Health and Nutrition Examination Survey; TEQ = toxic equivalency

Source: LaKind et al. 2009b

A review of general population blood levels of CDDs, CDFs, and PCBs from published literature dating from 1989 to 2010 collected across the world is available (Consonni et al. 2012). The study authors reviewed 187 studies with 29,687 subjects from 26 different countries. The study authors noted that significant temporal decreases in TEQs were observed from the studies (1985–2008) for CDDs and CDFs; however, no significant decrease was found for non-ortho-PCBs, notably PCB 126.

Compared with background 2,3,7,8-TCDD levels (3.6 ppt), workers that were formerly involved in 2,4,5-TCP 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. Miniero et al. (2017) examined blood levels of professionally exposed and non-occupationally exposed individuals in metallurgical plants of Brescia, Italy. The lipid-based 2005 World Health Organization (WHO)-TEQ level of non-professionally exposed individuals was 7.94 pg/g lipid. The TEQs for professionally exposed individuals working in ferrous and non-ferrous metallurgic plants were 8.25 and 9.55 pg/g lipid, respectively. A U.S. domestic agricultural worker was exposed to 2,3,7,8-TCDD during spraying of 2,4,5-T herbicide on pastureland and hay ground. A sample of the herbicide that was 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. 1989). 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 11–141 ppt) (Schecter and Ryan 1988). Since 2,4,5-T and 2,4,5-TCP are no longer used in the United States, these are no longer occupational exposure routes for U.S. workers or workers in many

other nations. Additionally, the only PCP manufacturer in North America was scheduled to close a facility in Mexico that produces PCP, as well as a facility in Alabama that formulates and stores registered wood preservative products containing PCP in 2022 (EPA 2021). PCP is scheduled to have all active registrations phased out in the United States by 2027 (EPA 2021).

In a study by Tepper et al. (1997), serum levels of CDDs and CDFs were measured in pulp and paper mill workers in the United States. The study 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, 21.2, 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 of 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, with averages 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).

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)

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

The recent train derailment and fire that followed in East Palestine Ohio, indicates that residents of this community and nearby may be potentially exposed to higher levels of CDDs and CDFs than other populations. Data collection is ongoing and will likely occur for years, so no long-term studies exist at this time; however, monitoring data show very high levels of CDDs in some environmental media such as soil (EPA 2023). Workers in industries that manufacture or use chemicals contaminated with CDDs such as PCP are one segment of the population at risk for higher exposure; however, PCP is being phased out by the EPA. Persons working in the hazardous waste industry or first responders to incidents where CDDs and CDFs may have been released (e.g., World Trade Center first responders) will be exposed to higher levels than the general population. Although production of PCBs ceased in the United States over 40 years ago, the use of PCBs is still authorized in transformers and other electrical equipment, and accidents involving PCB capacitors and transformers may entail high exposures to CDDs.

Military personnel near open burn pits were potentially exposed to higher levels of CDDs/CDFs than the general population. CDDs/CDFs and other substances were measured in air samples at Joint Base Balad in Iraq in 2007 (Masiol et al. 2016). The major source of CDDs/CDFs in the measured samples arose from the burn pit, which was the largest operating burn-pit on U.S. bases during the Iraq War. The average concentration of OCDD at all the sampling sites was 1.43 pg/m^3, with an average concentration as high as 6.68 pg/m^3 at one of the sampling sites. The next greatest average concentration was observed for 1,2,3,4,6,7,8- HpCDD (1.27 pg/m^3) for all the sampling sites. The average concentration of 2,3,7,8-TCDD at all 10 sampling sites was 0.06 pg/m^3.

A study of firefighters measured urinary CDDs levels before and after responding to a controlled residential fire. The levels of serum 1,2,3,7,8,9-HxCDD were significantly lower post-exposure, as compared to pre-exposure (Mayer et al. 2021a, 2021b). In comparisons to the general population, the serum levels 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 firefighters were significantly lower.

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, Missouri; Binghamton, New York; Love Canal, New York; Newark, New Jersey; and Vietnam) (Kahn et al. 1988; Schecter 1985; Schecter and Tiernan 1985; Schecter et al. 1987a, 1989a; Umbreit et al. 1986a, 1986b; Zook and Rappe 1994). Kahn et al. (2018) collected biomonitoring data from a set of adolescents in 2014–2016 who were exposed to debris from the World Trade Center collapse in 2001 and found that levels of CDDs/CDFs were approximately 7 times greater in these persons than from a control group of unexposed adolescents.

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. An analysis of 20 blood samples from residents of zone A, which were collected and stored shortly after the accident, showed serum lipid levels of 828–56,000 ppt 2,3,7,8-TCDD. These serum lipid levels are among the highest ever reported for humans (Mocarelli et al. 1991).

2,3,7,8-TCDD has been detected at concentrations of 20–173 ppt in adipose tissue from three 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 (Pirkle 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.

It is possible that persons residing near emission sources such as hazardous waste incinerators may have the potential for greater exposure to CDDs than the general population; however, recent studies have suggested that the impact that these facilities create for local populations is low. Nadal et al. (2019) analyzed the temporal trends of total CDDs/CDFs in the plasma of residents living in the vicinity of a hazardous waste incinerator that was constructed in 1998 in Catalonia, Spain. Over a 2-decade period (1998–2018), they reported between a 59 and 80% decrease in plasma CDD/CDF levels for these residents depending upon age and gender. They concluded that these decreases were due to reduced dietary intakes of these substances and that the incinerator did not create measurable risk to the health of the population living in the vicinity of the facility. A comprehensive review of 82 studies regarding the biomonitoring of individuals residing near, or working at, hazardous waste incinerators suggested that there was only a low impact on the internal dose of CDD/CDF levels due to emissions from solid waste incinerators (Campo et al. 2019). Similarly, biomonitoring data of a population near a large waste incinerator located in Turin, Italy showed no significant differences in the serum levels of PCDD/PCDFs, and PCBs measured in the population group residing near the plant after 3 years of operation with respect to a control group (Iamiceli et al. 2021).

Children and adults 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. 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 30–50% (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.

Anderson et al. (1998) completed a preliminary study of the levels of 8 CDDs, 10 CDFs, 36 PCBs, and 11 other 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 six meals of Great Lakes sport fish per year. A summary of the distribution of CDDs is provided in Table 5-44. 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 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 study 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.

Table 5-44. 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)

Table 5-44. 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]
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.

CDD = chlorinated dibenzo-*p*-dioxin; EPA = U.S. Environmental Protection Agency; 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 = toxic equivalency

Source: Anderson et al. 1998

Recent monitoring data in fish from the Great Lakes have shown a large decline in levels of CDDs and CDFs from decades ago, coinciding with declines of atmospheric emissions of dioxin-like substances (Gandhi et al. 2019). However, these monitoring results still show areas in which levels of CDDs and CDFs remain high due to past historical releases.

Ayotte et al. (1997) measured concentrations of CDDs/CDFs and PCBs in plasma of adult Inuits 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 2.5–36.0 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 17.1–81.8 ppt) in the Inuit population and 14.6 ppt (range 11.5–18.9 ppt) for the control population. When PCBs and CDDs/CDFs are considered together, the mean TEQ values for all dioxin-like compounds were 184.2 ppt in the Inuit population (range 55.8–446.7 ppt) and 26.1 ppt (range 20.1–31.7 ppt) for the control population.