4. EXPOSURE CHARACTERIZATION

This section summarizes key findings developed in the exposure portion of the Agency's dioxin reassessment. These findings are developed in the companion document entitled *Part I: Estimating Exposure to Dioxin-Like Compounds*, which is divided into three volumes: (1) Sources of Dioxin in the United States, (2) Properties, Environmental Levels, and Background Exposures, and (3) Site-Specific Assessment Procedures. Readers are encouraged to examine the more detailed companion document for further information on the topics covered here and to see complete literature citations. The characterization discussion provides cross-references to help readers find the relevant portions of the companion document.

This discussion is organized as follows: (1) sources, (2) fate, (3) environmental media and food concentrations, (4) background exposures, (5) potentially highly exposed populations, and (6) trends. The key findings are presented in italics.

4.1. SOURCES (Cross-reference: Part I, Volume 1: Sources of Dioxin-Like Compounds in the United States)

CDD/CDFs have never been intentionally produced other than on a laboratory-scale basis for use in scientific analysis. Rather, they have been generated as unintended by-products in trace quantities in various combustion, industrial, and biological processes. PCBs, on the other hand, were commercially produced in large quantities, but they are no longer commercially produced in the United States. EPA has classified sources of dioxin-like compounds into five broad categories:

1. *Combustion Sources.* CDD/CDFs are formed in most combustion systems, which can include waste incineration (such as municipal solid waste, sewage sludge, medical waste, and hazardous wastes), burning of various fuels (such as coal, wood, and petroleum products), other high temperature sources (such as cement kilns), and poorly or uncontrolled combustion sources (such as forest fires, building fires, and open burning of wastes). Some evidence exists that very small amounts of dioxin-like PCBs are produced during combustion, but they appear to be a small fraction of the total TEQs emitted.

2. *Metals Smelting, Refining, and Processing Sources.* CDD/CDFs can be formed during various types of primary and secondary metals operations, including iron ore sintering, steel production, and scrap metal recovery.

3. *Chemical Manufacturing.* CDD/CDFs can be formed as by-products from the manufacture of chlorine-bleached wood pulp, chlorinated phenols (e.g., pentachlorophenol, or PCP), PCBs, phenoxy herbicides (e.g., 2,4,5-T), and chlorinated aliphatic compounds (e.g., ethylene dichloride).

4. *Biological and Photochemical Processes.* Recent studies suggest that CDD/CDFs can be formed under certain environmental conditions (e.g., composting) from the action of microorganisms on chlorinated phenolic compounds. Similarly, CDD/CDFs have been reported to be formed during photolysis of highly chlorinated phenols.

5. *Reservoir Sources.* Reservoirs are materials or places that contain previously formed CDD/CDFs or dioxin-like PCBs and have the potential for redistribution and circulation of these compounds into the environment. Potential reservoirs include soils, sediments, biota, water, and some anthropogenic materials. Reservoirs become sources when they have releases to the circulating environment.

The development of national estimates of annual environmental releases to air, water, and land is complicated by the fact that only a few facilities in most industrial sectors have been evaluated for CDD/CDF emissions. Thus, an extrapolation is needed to estimate national emissions. The extrapolation method involves deriving an estimate of emissions per unit of activity (i.e., an emission factor) at the tested facilities and multiplying this by the total activity level in the untested facilities.

In order to convey the level of uncertainty in both the measure of activity and the emission factor, EPA developed a qualitative confidence rating scheme. The confidence rating scheme, presented in Table 4-1, uses qualitative criteria to assign a high, medium, or low confidence rating to the emission factor and activity level for those source categories for which emission estimates can be reliably quantified. The overall "confidence rating" assigned to a quantified emission estimate was determined by the confidence ratings assigned to the corresponding "activity level" and "emission factor." If the lowest rating assigned to either the activity level or the emission factor terms is "high," then the category rating assigned to the

emission estimate is high (also referred to as "A"). If the lowest rating assigned to either the activity level or emission factor terms is "medium," then the category rating assigned to the emission estimate is medium (also referred to as "B"). If the lowest rating assigned to either the activity level or emission factor terms is "low," then the category rating assigned to the emission estimate is low (also referred to as "C").

For many source categories, either the emission factor information or the activity level information were inadequate to support development of reliable quantitative release estimates for one or more media. For some of these source categories, sufficient information was available to make preliminary estimates of environmental releases of CDD/CDFs or dioxin-like PCBs; however, the confidence in the activity level estimates or emission factor estimates was so low that the estimates cannot be included in the sum of quantified emissions from sources with confidence ratings of A, B, or C. These estimates were given an overall confidence class rating of D. For other sources, some information exists suggesting that they may release dioxin-like compounds; however, the available data were judged to be insufficient for developing any quantitative emission estimate. These estimates were given an overall confidence class rating of E.

4.1.1. Inventory of Releases

This dioxin reassessment has produced an "inventory" of sources of environmental releases of dioxin-like compounds for the United States (Table 4-2). The inventory was developed by considering all sources identified in the published technical and scientific literature and by the incorporation of results from numerous individual emissions test reports of individual industrial and combustion source facilities. In order to be representative of the United States, data generated from U.S. sources of information were always given first priority for developing emission estimates. Data from other countries were used for making estimates in only a few source categories where foreign technologies were judged similar to those found in the United States and the U.S. data were judged to be inadequate. The inventory is limited to sources whose releases can be reliably quantified (i.e., those with confidence ratings of A, B, or C, as defined above). As discussed below, this document does provide preliminary estimates of releases from Class D sources, but they are presented separately from the inventory.

The inventory presents the environmental releases in terms of two reference years: 1987 and 1995. The year 1987 was selected primarily because little empirical data existed for making source-specific emission estimates prior to this time; 1995 represents the latest year that could reasonably be addressed within the timetable for producing the rest of this document. EPA

expects to conduct periodic revisions and updates to the source inventory in the future to track changes in environmental releases over time.

Figure 4-1 displays the emission estimates to air for sources included in the inventory and shows how the emission factors and activity levels were combined to generate emission estimates. Figure 4-2 compares the annual mean I-TEQ emission estimates to air for the two reference years (1987 and 1995).

The following conclusions are made for sources of dioxin-like compounds included in the inventory:

- *EPA's best estimates of releases of CDD/CDFs to air, water, and land from reasonably quantifiable sources were approximately 3300 g TEQ_{DF}-WHO_{98} (3000 g I-TEQ) in 1995 and 14,000 g TEQ_{DF}-WHO_{98} (12,800 g I-TEQ) in 1987.* This finding is derived directly from Table 4-2.

- *The inventory indicates that, between 1987 and 1995, there was approximately a 76% decrease in total environmental releases of CDDs/CDFs from known sources in the United States.* EPA is currently evaluating source releases for the year 2000. Preliminary indications support the observation of a continued reduction in total environmental releases from 1995 levels. The inventory updated for the year 2000 will undergo scientific peer review.

- *The environmental releases of CDD/CDFs in the United States occur from a wide variety of sources, but they are dominated by releases to the air from combustion sources.* The current (1995) inventory indicates that emissions from combustion sources are more than an order of magnitude greater than emissions from the sum of emissions from all other categories. Approximately 70% of all quantifiable environmental releases were contributed by air emissions from just three source categories in 1995: municipal waste incinerators (representing 38% of total environmental releases); backyard burning of refuse in barrels (19%); and medical waste incinerators (14%).

- *The decrease in estimated releases of CDD/CDFs between 1987 and 1995 (approximately 76%) was due primarily to reductions in air emissions from municipal and medical waste incinerators, and further reductions are anticipated.*

For both categories, these emission reductions have occurred from a combination of improved combustion and emission controls and from the closing of a number of facilities. EPA's regulatory programs estimate that full compliance with recently promulgated regulations should result in further reductions in emissions from the 1995 levels of more than 1800 I-TEQ. These reductions will occur in the following source types: municipal waste combustors, medical waste incinerators, and various facilities that burn hazardous waste (see Part I, Volume 1, for further details about these reductions). No federal regulations are in place or currently under development for limiting dioxin emissions from backyard burning of refuse in barrels. A number of states have general restrictions on the practice of backyard trash burning.

- *Insufficient data are available to comprehensively estimate point source releases of dioxin-like compounds to water.* Sound estimates of releases to water are available only for chlorine bleached pulp and paper mills (356 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} for 1987 and 20 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} for 1995) and the manufacture of ethylene dichloride (EDC)/vinyl chloride monomer (VCM) (< 1 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} in 1995). Other releases to water bodies that cannot be quantified on the basis of existing data include effluents from publicly owned treatment works (POTW) and most industrial/commercial sources. EPA's Office of Water estimates that when full compliance with limitations on effluent discharges of CDD/CDF from chlorine bleached pulp and paper mills is achieved, annual emissions will be reduced to 5 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98}.

- *Based on the available information, the inventory includes only a limited set of activities that result in direct environmental releases to land.* Total releases to land quantified in the national inventory are estimated at 110 g TEQ_{DF}-WHO_{98} in 1995 and are principally from municipal wastewater treatment sludge (76.6 g) and the use of 2,4-D (28.9 g). Not included in the inventory's definition of an environmental release is the disposal of sludge and ashes into approved landfills.

- *Significant amounts of dioxin-like compounds produced annually are not considered environmental releases and, therefore, are not included in the national inventory.* Examples include dioxin-like compounds generated internal to a process but destroyed before release, waste streams that are disposed of in approved landfills and

are therefore outside the definition of annual environmental releases, and products that contain dioxin-like compounds but for which environmental releases, if any, cannot be estimated.

The procedures and results of the U.S. inventory may have underestimated releases from contemporary sources. A number of investigators have suggested that national inventories may underestimate emissions because of the possibility of unknown sources. This claim has been supported with mass balance analyses that suggest that deposition exceeds emissions (Rappe, 1991; Harrad and Jones, 1992; Bruzy and Hites, 1995); however, the uncertainty, in both the emissions and deposition estimates for the United States prevents the use of this approach for reliably evaluating the issue.

A variety of other arguments indicate that the inventory could underestimate emissions of dioxin-like compounds:

- A number of sources lacked sufficient data to include in the inventory but there were limited evidence indicating that these sources can emit CDD/CDFs. These sources are listed in Tables 4-3 and 4-4 and include various components of the metals industries, such as electric arc furnaces and foundries and uncontrolled or minimally controlled combustion practices (e.g., accidental fires at landfills).

- The possibility remains that truly unknown sources exist. Many of the sources that are well-accepted today were discovered only in the past 10 years. For example, CDD/CDFs were found unexpectedly in the wastewater effluent from bleached pulp and paper mills in the mid 1980s. Ore sintering is now listed as one of the leading sources of CDD/CDF emissions in Germany, but it was not recognized as a source until the early 1990s.

4.1.2. General Source Observations

For any given time period, releases from both contemporary formation sources and reservoir sources determine the overall amount of the dioxin-like compounds that are being released to the open and circulating environment. Because existing information is incomplete with regard to quantifying contributions from contemporary and reservoir sources, it is not currently possible to estimate the total magnitude of release for dioxin-like compounds from all

sources into the U.S. environment. For example, in terms of 1995 releases from reasonably quantifiable sources, this document estimates releases of 3300 g TEQ_{DF}-WHO_{98} (3000 g I-TEQ_{DF}) for contemporary formation sources and 2900 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} for reservoir sources.

In addition, there remain a number of unquantifiable and poorly quantified sources. No quantitative release estimates can be made for agricultural burning or for most CDD/CDF reservoirs or for any dioxin-like PCB reservoirs. The preliminary 1995 estimate of releases from poorly characterized contemporary formation sources is 1400 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98}. The preliminary release estimates for contemporary formation sources and reservoir sources are presented in Table 4-2. Table 4-3 lists all the sources that have been reported to release dioxin-like compounds but cannot be characterized on even a preliminary basis.

Additional observations and conclusions about all sources of dioxin-like compounds are summarized below:

- *The contribution of dioxin-like compounds to waterways from nonpoint source reservoirs is likely to be greater than the contribution from point sources.* Current data are only sufficient to support preliminary estimates of nonpoint source contributions of dioxin-like compounds to water (i.e., from urban storm water runoff and rural soil erosion). These estimates suggest that, on a nationwide basis, total nonpoint releases are significantly larger than point source releases.

- *Current emissions of CDD/CDFs to the U.S. environment result principally from anthropogenic activities.* Evidence that supports this finding includes matches in time of rise of environmental levels with time when general industrial activity began rising rapidly (see trend discussion in Part I, Volume 2, Chapter 6), the lack of any identified large natural sources, and observations of higher CDD/CDF body burdens in industrialized versus less industrialized countries (see discussion on human tissue levels in Part I, Volume 2, Chapter 4).

- *Although chlorine is an essential component for the formation of CDD/CDFs in combustion systems, the empirical evidence indicates that for commercial-scale incinerators, chlorine levels in feed are not the dominant controlling factor for rates of CDD/CDF stack emissions.* Important factors that can affect the rate of CDD/CDF formation include the overall combustion efficiency, post-combustion flue gas

temperatures and residence times, and the availability of surface catalytic sites to support CDD/CDF synthesis. Data from bench-, pilot- and commercial-scale combustors indicate that CDD/CDF formation can occur by a number of mechanisms. Some of these data, primarily from laboratory and pilot-scale combustors, have shown direct correlation between chlorine content in fuels and rates of CDD/CDF formation. Other data, primarily from commercial-scale combustors, show little relation between availability of chlorine in feeds and rates of CDD/CDF formation.

- The conclusion that chlorine in feed is not a strong determinant of CDD/CDF emissions applies to the overall population of commercial-scale combustors. For any individual commercial-scale combustor, circumstances may exist in which changes in chlorine content of feed could affect CDD/CDF emissions. For uncontrolled combustion, such as open burning of household waste, the chlorine content of the waste may play a more significant role in rates of CDD/CDF formation and release than is observed at commercial-scale combustors. The full discussion on this issue is presented in Part I, Volume 1, Chapter 2.

- *Dioxins are present in some ball clays, but insufficient data are available to estimate whether environmental releases occur during mining and use.* Recent studies in the United States and Europe have measured dioxins (principally CDDs) in some ball clays and other related clays. As discussed in Part I, Volume 1, Chapter 13, it is likely that the dioxin present in ball clay is of a natural origin. Ball clay is principally used in the manufacture of ceramics, which involves firing the clay in high-temperature kilns. This activity may cause some portion of the CDDs contained in the clay to be released into the air, but emission tests have not yet been conducted that would allow characterizing these releases.

- *Data are available to estimate the amounts of CDD/CDFs contained in only a limited number of commercial products.* No systematic survey has been conducted to determine levels of dioxin-like compounds in commercial products. The available data do, however, allow estimates to be made of the amounts of dioxin-like compounds in bleached pulp (40 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} in 1995), POTW sludge used in fertilizers (3.5 g I-TEQ_{DF} or 2.6 g TEQ_{DF}-WHO_{98} in 1995), pentachlorophenol-treated wood (8400 g I-TEQ_{DF} or 4800 g TEQ_{DF}-WHO_{98} in 1995),

dioxazine dyes and pigments (< 1 g I-TEQ_{DF} or TEQ_{DF}-WHO_{98} in 1995), and 2,4-D (18.4 g I-TEQ_{DF} or 28.9 g TEQ_{DF}-WHO_{98} in 1995).

- *No significant release of newly formed dioxin-like PCBs is occurring in the United States.* Unlike CDD/CDFs, PCBs were intentionally manufactured in the United States in large quantities from 1929 until production ceased in 1977. Although it has been demonstrated that small quantities of coplanar PCBs can be produced during waste combustion, no strong evidence exists that the dioxin-like PCBs make a significant contribution to TEQ releases during combustion. The occurrences of dioxin-like PCBs in the U.S. environment most likely reflect past releases associated with PCB production, use, and disposal. Further support for this finding is based on observations of reductions since the 1980s in PCBs in Great Lakes sediment and other areas.

- *It is unlikely that the emission rates of CDD/CDFs from known sources correlate proportionally with general population exposures.* Although the inventory shows the relative contribution of various sources to total emissions, it cannot be assumed that these sources make the same relative contributions to human exposure. It is quite possible that the major sources of dioxin in food (see the discussion in Part I, Volume 2, Chapter 2, indicating that diet is the dominant exposure pathway for humans) may not be those sources that represent the largest fractions of current total emissions in the United States. It is important to consider the geographic locations of sources relative to the areas from which much of the beef, pork, milk, and fish come. That is, many of the agricultural areas that produce dietary animal fats are not located near or directly downwind of the major sources of dioxin and related compounds.

- *The contribution of reservoir sources to human exposure may be significant.* Several factors support this finding:
  1. Because the magnitude of releases from current sources of newly formed PCBs are most likely negligible, human exposure to the dioxin-like PCBs is thought to be derived almost completely from reservoir sources. Key pathways involve releases from both soils and sediments to both aquatic and terrestrial food chains. As discussed in Part I, Volume 2, Chapter 4, one-third of general population

TEQ_{DFP} exposure is due to PCBs. Thus, at least one-third of the overall risk from dioxin-like compounds comes from reservoir sources.

2. CDD/CDF releases from soil via soil erosion and runoff to waterways may be significant. These releases appear to be greater than releases to water from the primary sources included in the inventory. CDD/CDFs in waterways can bioaccumulate in fish, leading to human exposure via their consumption. As discussed in Part I, Volume 2, Chapter 4, fish consumption makes up about one-fifth of the total general population CDD/CDF TEQ exposure. This suggests that a significant portion of the CDD/CDF TEQ exposure could be due to releases from the soil reservoir. It is not known, however, how much of the soil erosion and runoff represents recently deposited CDD/CDFs from primary sources or longer-term accumulation. Much of the eroded soil comes from tilled agricultural lands, which would include a mix of CDD/CDFs from various deposition times. The age of CDD/CDFs in urban runoff is less clear.

3. Potentially, soil reservoirs could have vapor and particulate releases that deposit on plants and enter the terrestrial food chain. The magnitude of this contribution, however, is unknown.

Collectively, these three factors suggest that reservoirs are a significant source of current background TEQ exposure, perhaps contributing half or more of the total.

4.2. ENVIRONMENTAL FATE (Cross-reference: Part I, Volume 2, Chapter 2)

The estimates of environmental releases are presented above in terms of TEQs. This is done for convenience in presenting summary information and to facilitate comparisons across sources. For purposes of environmental fate modeling, however, it is important to use the individual CDD/CDF and PCB congeners values rather than TEQs because the physical/chemical properties of individual dioxin congeners vary and will behave differently in the environment. For example, the relative mix of congeners released from a stack cannot be assumed to remain constant during transport through the atmosphere and deposition to various media. The full congener-specific release rates for most sources are given in an electronic database that is available as a companion to this document (U.S. EPA, 1998) Database of Sources of Environmental Releases of Dioxin-Like Compounds in the United States. EPA/600/P-98/002Ab.

In Part I, Volume 3, site-specific procedures are provided for estimating the impact of emissions on local populations, and this section emphasizes that congener specific emission values should be used in modeling their environmental fate. Finally, it is important to recognize that this document does not use source release estimates to generate background population intake/risk estimates; rather, these estimates are derived primarily from food levels and consumption rates.

Dioxin-like compounds are widely distributed in the environment as a result of a number of physical and biological processes. The dioxin-like compounds are essentially insoluble in water, they are generally classified as semivolatile, and they tend to bioaccumulate in animals. Some evidence has shown that these compounds can degrade in the environment, but in general they are considered to be very persistent and relatively immobile in soils and sediments. These compounds are transported through the atmosphere as vapors or attached to airborne particulates and can be deposited on soils, plants, or other surfaces (by wet or dry deposition). The dioxin-like compounds enter water bodies primarily via direct deposition from the atmosphere or by surface runoff and erosion. From soils, these compounds can reenter the atmosphere as either resuspended soil particles or vapors. In water, they can be resuspended into the water column from sediments, they can be volatilized out of the surface waters into the atmosphere, or, they can become buried in deeper sediments. Immobile sediments appear to serve as permanent sinks for the dioxin-like compounds. Although anthropogenic materials (such as PCP) are not always considered an environmental compartment, dioxin-like compounds are also found in such materials, and from there they have the potential to be released into the broader environment.

Atmospheric transport and deposition of the dioxin-like compounds are a primary means of their dispersal throughout the environment. The dioxin-like compounds have been measured in wet and dry deposition in most locations, including remote areas. Numerous studies have shown that they are commonly found in soils throughout the world. Industrialized countries tend to show similar elevated concentrations in soil, and detectable levels have been found in nonindustrialized countries. The only satisfactory explanation available for this distribution is air transport and deposition. Finally, by analogy these compounds would be expected to behave similarly to other compounds that have similar properties, and this postulated mechanism of global distribution is becoming widely accepted for a variety of persistent organic compounds.

The two primary pathways for the dioxin-like compounds to enter the ecological food chains and human diet are air-to-plant-to-animal and water/sediment-to-fish. Vegetation receives these compounds via atmospheric deposition in the vapor and particle phases. The compounds are retained on plant surfaces and bioaccumulated in the fatty tissues of animals that

feed on these plants. Vapor phase transfers onto vegetation have been experimentally shown to dominate the air-to-plant pathway for the dioxin-like compounds, particularly for the lower chlorinated congeners. In the aquatic food chain, dioxins enter water systems via direct discharge or deposition and runoff from watersheds. Fish accumulate these compounds through their direct contact with water, suspended particles, and bottom sediments and through their consumption of aquatic organisms.

Although these two pathways are thought to normally dominate contribution to the commercial food supply, others can also be important. Elevated dioxin levels in cattle resulting from animal contact with PCP-treated wood have been documented by the U.S. Department of Agriculture. Animal feed contamination episodes have led to elevations of dioxins in poultry in the United States, milk in Germany, and meat/dairy products in Belgium (see Part I, Volume 2, Chapter 5).

4.3. ENVIRONMENTAL MEDIA AND FOOD CONCENTRATIONS (Cross-reference: Part I, Volume 2, Chapter 3)

Background levels of dioxin-like compounds in various environmental media, including food, are presented in Table 4-4 in terms of means, variability, and sample sizes used to support the estimates. Estimates for background levels of dioxin-like compounds in environmental media are based on a variety of studies conducted at different locations in North America. Of the studies available for this compilation, only those conducted in locations representing "background" were selected. The amount and representativeness of the data vary, but in general they were derived from studies that were not designed to estimate national background means. The environmental media concentrations were similar to those in studies from Western Europe. These data are the best available for comparisons with site-specific values. Because of the limited number of locations examined, it is not known whether these estimates adequately capture the full national variability. As new data are collected, these ranges are likely to be expanded and refined. The limited data on dioxin-like PCBs in environmental media are summarized in Part I, Volume 2, Chapter 3.

Estimates for levels of dioxin-like compounds in food are based on data from a variety of studies conducted in North America. Beef, pork, and poultry estimates were derived from statistically based national surveys. Milk estimates were derived from a survey of a nationwide milk sampling network. Dairy estimates were derived from milk fat concentrations, coupled with appropriate assumptions for the amount of milk fat in dairy products. The background egg concentrations were based on an analysis of 15 egg samples collected from retail stores in eight

states (CA, OH, GA, NY, PA, OR, MN, WS; two samples per state except one in OR), where each sample was a composite of 24 individual eggs (i.e., 15 samples represented 360 eggs). The fish data, as discussed below, were derived from multiple studies, with samples collected both directly from water bodies and from retail outlets. All fish concentrations were expressed on the basis of fresh weight in edible tissue. As with other environmental media, food levels found in the United States were similar to levels found in Europe.

The procedure to evaluate background fish exposures emphasizes the use of both species-specific consumption rates and species-specific concentrations. EPA's national bioaccumulation study (U.S. EPA, 1992b) provides some species-specific information on freshwater/estuarine fish caught in the wild at various locations in the United States. Additional species-specific data on store-bought fish are available from studies conducted by the U.S. Food and Drug Administration (FDA) during the mid to latter 1990s (Jensen and Bolger, 2000; Jensen et al., 2000). An important aspect of the FDA studies is that they include data on store-bought catfish, tuna, shellfish, and salmon, which are some of the most highly consumed species. Accordingly, the data used to characterize CDD/CDF fish levels are much improved over previous estimates, with more than 300 individual samples and good representation of the most highly consumed species. However, the levels of dioxins in fish remain more uncertain than those in the other foods.

The compilation of data from different studies still lacks the geographic coverage and statistical power of the other food surveys. The EPA and FDA studies did not address dioxin-like PCBs; rather, these are based on a much smaller data set derived from the open literature. Also, the estimates of dioxin intake resulting from fish consumption do not include consumption of fish oils. Currently, insufficient data are available to support estimates of dioxin intake from direct fish oil consumption.

The general population dioxin intake calculations used in this document are a function of both consumption rate and dioxin concentration in food. The concentration data used in this document were measured in raw foods; therefore, if cooking significantly alters the dioxin concentration in consumed portions it must be accounted for in estimating dioxin intake.

This issue has been examined in a number of studies that measured the effects of cooking on the levels of CDDs, CDFs, and PCBs in foods (see Part I, Volume 2, Chapter 3). These studies have a range of results, depending on food type and cooking method. Most of the cooking experiments suggested that cooking reduces the total amount of dioxins in food but causes relatively little change in its concentration.

Although some cooking experiments have shown increases and others have shown decreases in dioxin concentrations, the relative prevalence of these impacts have not been

established. Therefore, given that most experiments show little change and others show change in both directions, the most reasonable assumption that can be made from the existing data is that dioxin concentration in uncooked food is a reasonable surrogate for dioxin concentration in cooked food. Although cooking in general does not reduce dioxin concentration in food, some specific food preparation practices can be adopted that can reduce dioxin intake by significantly reducing overall animal fat consumption. For example, carefully trimming fat from meat, removing skin from chicken and fish, and avoiding cooking in animal fats should reduce both animal fat and dioxin intake.

Some evidence from Europe suggests that during the 1990s a decline occurred in concentrations of dioxins and furans in food products, particularly dairy products (see Part I, Volume 2, Chapter 6). For example, the United Kingdom's Ministry of Agriculture, Fisheries, and Food collected milk samples in 1990 and again from similar locations in 1995. In 1990, the I-TEQ_{DF} ranged from 1.1 to 3.3 ppt, whereas the 1995 I-TEQ_{DF} ranged from 0.7 to 1.4. In Germany, a sampling of 120 dairy products in 1994 found I-TEQ_{DF} concentrations that were 25% lower than those in a similar sampling program in 1990. Liem et al. (2000) reports on a European cooperative study coordinated by the National Institute of Public Health and the Environment in the Netherlands and the Swedish National Food Administration. Ten countries supplied data on food concentrations, food consumption patterns, and other data used to evaluate exposure to dioxins in Europe. Some of the data suggested reductions in concentrations over time, but the available information was insufficient to draw general conclusions.

No systematic study of temporal trends in dioxin levels in food has been conducted in the United States. Although not statistically based, one U.S. study examined dioxin levels in 14 preserved food samples from various decades in the 20th century (Winters et al., 1998). It was found that meat samples of the 1950s through the 1970s had concentrations that were two-three times higher for the CDD/CDF TEQs and about 10 times higher for the PCB TEQs, as compared to current meat concentrations.

The food data and associated exposure estimates presented here reflect a mid-1990's time frame. New studies underway now or recently completed could be used in future updates to this report to make exposure estimates for a new reference year, such as 2000. The following studies on dioxin levels in food were not completed in time to be included in this document and should be considered in future updates:

- The milk levels used in Tables 4-4 and 4-6 are based on a study by Lorber et al. (1998) where milk samples were collected in 1996. A very similar milk survey was

conducted by Schaum et al. (2003) involving the collection and analysis of TEQ_{DFP} in cow milk samples from 45 dairy plants in July of 2000 and again in January 2001. This study reported TEQ_{DFP} levels in whole milk which were about half the levels found by Lorber et al. (1998). Follow-up work by Schuda et al. (2004), which addressed CDD/Fs only, allowed estimation of 2000/2001 TEQ_{DF} milk levels on a lipid basis. This approach showed similar TEQ_{DF} levels in milk lipid, or perhaps a slight decrease, when comparing CDD/F TEQs in the two sampling times (0.71 pg TEQDF/g lipid in 2000/2001 compared to 0.82 pg TEQDF/g lipid in 1996).

- USDA is currently conducting a nationwide survey of dioxin levels in beef, pork and poultry. Samples were collected in 2002 and 2003 and data analysis is now underway. The survey design and data analysis are structured in a similar way to the earlier USDA surveys used in this report and should allow for trend analysis.

- The Institute of Medicine of the National Academies published a review of dioxin levels in foods in 2003 (Institute of Medicine of the National Academies, 2003). This document presents policy options for reducing dietary exposure to dioxins in food and related research recommendations. Appendix B of the Institute of Medicine's report summarizes FDA's Total Diet Survey of dioxin levels in food collected in 2001. A wide variety of foods were sampled including dairy products, eggs, meats, fish, fruits, vegetables and fats/oils.

The food consumption rates used here are based primarily on USDA's 1994-1996 Continuing Survey of Food Intakes by Individuals. As new USDA survey data come available, these should be incorporated into future updates of this report.

4.4. BACKGROUND EXPOSURES (Cross-reference: Part I, Volume 2, Chapter 4)

4.4.1. Tissue Levels

The average CDD/CDF/PCB tissue level for the general adult U.S. population appears to be declining, and the best estimate of current (late 1990s) levels is 25 ppt (TEQ_{DFP}-WHO_{98}, lipid basis).

The tissue samples collected in North America in the late 1980s and early 1990s showed an average TEQ_{DFP}-WHO_{98} level of about 55 pg/g lipid. This finding is supported by a number of studies—all conducted in North America—that measured dioxin levels in adipose, blood, and

human milk. However, the number of participants in most of these studies was relatively small and they were not statistically selected in ways that ensure their representativeness of the general U.S. adult population. One study, the 1987 National Human Adipose Tissue Survey, involved more than 800 individuals and provided broad geographic coverage, but it did not address coplanar PCBs. Similar tissue levels of these compounds have been measured in Europe and Japan during similar time periods.

Because dioxin levels in the environment have been declining since the 1970s (see the trends discussion in Part I, Volume 2, Chapter 6), it is reasonable to expect that levels in food, human intake, and, ultimately, human tissue have also declined over this period. The changes in tissue levels are likely to lag the decline seen in environmental levels, and the changes in tissue levels cannot be assumed to occur proportionally with declines in environmental levels.

CDC (2000) summarizes levels of CDDs, CDFs, and PCBs in human blood collected between 1995 to 1997 from 316 U.S. residents (ages 20–70 years). The individuals sampled had no known exposures to dioxin other than normal background. Although the samples in this data set were not collected in a manner that can be considered statistically representative of the national population and they lack wide geographic coverage, they are judged to provide a better indication of current tissue levels in the United States than the earlier data.

PCBs 105, 118, and 156 are missing from the blood data for the comparison populations reported by CDC (2000). These congeners account for 62% of the total PCB TEQ estimated in the early 1990s. Assuming that the missing congeners from the CDC study data contribute in the same proportion to the total PCB TEQ as in earlier data, they would increase the estimate of current body burdens by another 3.3 pg TEQ/g lipid, for a total PCB TEQ of 5.3 pg/g lipid and a total of 25.4 pg TEQ_{DFP}-WHO_{98}/g lipid (i.e., the TEQ_{DF}-WHO_{98} concentration was 20.1 pg/g lipid, and the TEQ_{P}-WHO_{98} concentration was estimated at 5.3 pg/g lipid). A summary of the CDC (2000) data is shown in Table 4-5.

A portion of the CDC blood data were plotted as a function of age. This plot, shown in Figure 4-3, indicates that blood levels generally increase with age, as does the variability in blood levels.

The calculation of a current tissue level of 25.4 pg/g lipid TEQ_{DFP}-WHO_{98} is further supported by the observation that this mean tissue level is consistent with the best estimate of current adult intake, 66 pg TEQ_{DFP}-WHO_{98}/d. Using this intake in a one-compartment, steady-state pharmacokinetic model yields a tissue level estimate of about 11.3 pg TEQ_{DFP}/g lipid (assumes TEQ_{DFP} has an effective half-life of 7.1 years, 80% of ingested dioxin is absorbed into the body, and lipid weight is 25% of the adult assumed body weight of 70 kg, or 17.5 kg).

Because intake rates appear to have declined in recent years, and steady-state is not likely to have been achieved, it is reasonable to observe higher measured tissue levels, such as the 25.4 pg TEQ/g lipid, than those predicted by the model.

Characterizing national background levels of dioxins in tissues is uncertain because the current data cannot be considered statistically representative of the general population. It is also complicated by the fact that tissue levels are a function of both age and birth year. Because intake levels have varied over time, the accumulation of dioxins in a person who turned 50 years old in 1990 is different than that in a person who turned 50 in 2000. As discussed in Part I, Volume 2, Chapter 6, exposure to dioxin-like compounds peaked during the 1960s, with declining exposures since then. Therefore, a person born in 1910 will see a rise in body levels that peaks at 50 to 70 years old. At the other end of the spectrum, a person born in 1970 will experience a higher body concentration very early in life, with declining levels in later years.

A pharmacokinetic (PK) modeling framework was developed to study trends in population body burdens of CDDs/CDFs throughout the 20th century and into the 21st century (Lorber, 2002). It was assumed that individuals within a population were exposed to doses rising from 0.50 pg WHO_{98}-TEQ_{DF}/kg-day during the 1940s to about 6.5 pg WHO_{98}-TEQ_{DF}/kg-day by the late 1960s, down to 1.0 pg WHO_{98}-TEQ_{DF}/kg-day by 1980, and finally to 0.50 pg WHO_{98}-TEQ_{DF}/kg-day by 2000, remaining constant at that level into the 21st century. It was found that a modeled population tissue level distribution will vary, depending on the year the modeled population is sampled. The results of this analysis are presented in Figure 4-4, which shows modeled population tissue level distributions for four years. An "age trend" is seen in the figure for modeled populations sampled in 1985 and 1995, as was seen in the CDC monitoring study of actual blood measurements of WHO_{98}-TEQ_{DFP} (see Fig. 4-3). Figure 4-4 also suggests that this age trend will disappear in the 21st century and that the CDD/CDF tissue level will drop below 10 ppt TEQ_{DF}-WHO_{98} lipid basis by 2030.

Monitoring studies which are currently underway should help determine whether the decline in body burdens has been continuing into the 21st century, as suggested by modeling. Results from the National Health and Nutrition Examination Survey of 1999-2000 (NHANES 1999-2000) were recently made available (CDC, 2003). NHANES 1999-2000 included data on dioxin-like compounds in the blood of 1921 sampled individuals, aged 12 and higher, and sampled from numerous locations around the country. These compounds included the 17 dioxin and furan congeners, as well as PCB congeners 126, 77, 169, and 81.

The current estimate of background body burden is based on 6 different studies totaling 316 individuals around the country which measured concentrations of these compounds in

populations characterized as "background" (CDC, 2000). Often these populations were selected the "background" population for studies which targeted other potentially exposed populations. The dates of these surveys, as noted above, were from about 1995 to 1997. In addition to being more recent, the NHANES 1999-2000 sampled population was much larger, but perhaps most importantly, NHANES was statistically designed to be representative of U.S. background after several years of data collection while the merged population from the 6 studies was not.

However, the amount of blood serum available for individual measurements in NHANES 1999-2000 was too small to be able to detect and characterize current levels of dioxin like compounds in the population. A large majority of the measurements were nondetects. For this reason, an effort is underway to pool remaining, available individual samples from NHANES and measure them for dioxin-like compounds, which would provide an updated measure of average concentrations of these compounds in the blood of U.S. citizens (ages 12 and greater, circa 1999-2000, and with all other delimiters relevant to the pooled samples, of course).

4.4.2. Intake Estimates

Adult daily intakes of CDD/CDFs and dioxin-like PCBs are estimated to average 43 and 23 pg TEQ_{DFP}-WHO_{98}/day, respectively, for a total intake of 66 pg/day TEQ_{DFP}-WHO_{98}. Daily intake is estimated by combining exposure media concentrations (food, soil, and air) with contact rates (ingestion, inhalation). Table 4-6 summarizes the media concentrations, contact rates, and resulting intake estimates.

The intake estimate is supported by an extensive database on food consumption rates and estimates of dioxin-like compounds in food (as discussed above). PK modeling provides further support for the intake estimates. Applying a simple steady-state PK model to an adult average blood level of 25 ppt TEQ_{DFP}-WHO_{98} (on a lipid basis) yields a daily intake of 146 pg TEQ_{DFP}-WHO_{98}/day (assumes TEQ_{DFP} has an effective half-life of 7.1 years, 80% of ingested dioxin is absorbed into the body, and lipid weight is 25% of the adult assumed body weight of 70 kg, or 17.5 kg). This PK-modeled CDD/CDF/PCB intake estimate is about 2.2 times higher than the direct intake estimate of 66 pg TEQ_{DFP}-WHO_{98}/day. This difference is to be expected with this application of a simple steady-state PK model to current average adipose tissue concentrations. Current adult tissue levels reflect intakes from past exposure levels, which are thought to be higher than current levels (Lorber, 2002; also in Part I, Volume 2, Chapter 6). Because the direction and magnitude of the difference in intake estimates between the two approaches are understood, the PK-derived value is judged supportive of the pathway-derived estimate. It

should be recognized, however, that the pathway-derived value will underestimate exposure if it has failed to capture all the significant exposure pathways.

4.4.3. Variability in Intake Levels

CDD/CDF and dioxin-like PCB intakes for the general population may extend to levels at least three times higher than the mean. Variability in general population exposure is primarily the result of the differences in dietary choices that individuals make. These are differences in both quantity and types of food consumed. An increased background exposure can result from either a diet that favors consumption of foods high in dioxin content or a diet that is disproportionately high in overall consumption of animal fats.

The best data available to determine the variability of total fat consumption come from several analyses of the Bogalusa Heart Study (Cresanta et al., 1988; Nicklas et al., 1993, 1995, Nicklas, 1995; Frank et al., 1986). These data show that the 95th percentile of total fat consumption is about twice the mean and the 99th percentile is approximately three times the mean. For a diet that has a broad distribution of animal fats (as does the typical U.S. diet), this same distribution can be assumed for dioxin intake.

Although body burden data cannot be assumed to be perfectly representative of current intakes (because they reflect past exposures as well as current ones), they also provide some support for this finding, based on the observation that the 95th percentile blood level in the CDC (2000) study was almost twice the mean level.

Intakes of CDDs/CDFs and dioxin-like PCBs are more than three times higher for a young child than for an adult, on a body-weight basis. This figure is based on combining age-specific food consumption rate and average food concentrations, as was done above for adult intake estimates (see Table 4-7).

Only 4 of the 17 toxic CDD/CDF congeners and 1 of the 11 toxic PCBs account for most of the toxicity in human tissue concentrations: 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 1,2,3,6,7,8-HxCDD, and 2,3,4,7,8-PCDF and PCB 126. This finding is derived directly from the data described earlier on human tissue levels and is supported by intake estimations that indicate that these congeners are also the primary contributors to dietary dose. These five compounds make up about 80% of the total TEQ_{DFP}-WHO_{98} tissue level.

4.5. POTENTIALLY HIGHLY EXPOSED POPULATIONS OR DEVELOPMENTAL STAGES (Cross-reference: Part I, Volume 2, Chapter 5)

As discussed earlier, background exposures to dioxin-like compounds may extend to levels at least three times higher than the mean. This upper range is assumed to result from the normal variability of diet and human behaviors. Exposures from local elevated sources or exposures resulting from unique diets would be in addition to this background variability. Such elevated exposures may occur in small segments of the population, such as individuals living near discrete local sources. Nursing infants represent a special case: for a limited portion of their lives, these individuals may have elevated exposures on a body-weight basis when compared with nonnursing infants and adults.

Dioxin contamination incidents involving the commercial food supply have occurred in the United States and in other countries. For example, in the United States, contaminated ball clay was used as an anticaking agent in soybean meal, which resulted in elevated dioxin levels in some poultry and catfish. This incident, which occurred in 1998, involved a small faction of the national poultry production, and the use of contaminated ball clay has since been eliminated. Elevated dioxin levels have also been observed in a few beef and dairy animals, where the contamination was associated with contact with pentachlorophenol-treated wood. Evidence of this kind of elevated exposure was not detected in the national beef survey. Consequently, its occurrence is likely to be low, but it has not been determined.

These incidents may have led to small increases in dioxin exposure to the general population. However, it is unlikely that they have led to disproportionate exposures to populations living near where they occurred because in the United States meat and dairy products are highly distributed on a national scale. If contamination events were to occur in foods that are predominantly distributed on a local or regional scale, then such events could lead to more highly exposed local populations (see Part I, Volume 2, Chapter 5).

Elevated exposures associated with the workplace or with industrial accidents have also been documented. U.S. workers in certain segments of the chemical industry had elevated levels of TCDD exposure, with some tissue measurements in the thousands of part per trillion TCDD. There is no clear evidence that elevated exposures are currently occurring among U.S. workers. Documented examples of past exposures for other groups include certain Air Force personnel exposed to Agent Orange during the Vietnam War and people exposed as a result of industrial accidents in Europe and Asia.

Consumption of breast milk by nursing infants leads to higher levels of exposure and higher body burdens of dioxins during early years of life as compared with those of nonnursing infants (Part I, Volume 2, Chapter 5).

Kreuzer et al. (1997) and Abraham et al. (1994, 1995, 1998, 2000) compared dioxin levels in infants who were breast-fed with those who were formula-fed. All the studies showed elevations in the concentrations of dioxins in the breast-fed infants. Collectively, these studies included more than 100 infants, and they found that blood levels in infants aged 4-12 months were generally higher than 20 pg TEQ_{DF}-WHO_{98}/g lipid in nursing infants and lower than 5 pg TEQ_{DF}-WHO_{98}/g lipid in formula fed infants. Limited data suggest a similar difference for dioxin-like PCBs. Abraham et al. (1995) reported that at 11 months a breast-fed infant had a concentration of 31.4 pg TEQ_{P}-WHO_{98}/g lipid, compared to 2.5 pg TEQ_{P}-WHO_{98}/g lipid for the formula-fed infant.

U.S. dioxin intakes from nursing were calculated using time-dependent values for breast milk concentrations, consumption rates, and body weights. These calculations estimated an intake immediately after birth of 242 pg TEQ_{DFP}-WHO_{98}/kg/day. This level dropped to 18 pg TEQ_{DFP}-WHO_{98}/kg/day after 12 months of nursing. The average intake over 1-year of nursing was calculated to be 87 pg TEQ_{DFP}-WHO_{98}/kg/day. The cumulative intake for a 1 year nursing scenario represented about 13% of the total lifetime cumulative intake (see Lorber and Phillips, 2002, and Part I, Volume 2, Chapter 5, for details on these calculations).

CDC (1997) reported that in 1995, 55% of all babies experienced some breast-feeding, with about half of those breast-feeding beyond 5 months. The average duration of breast-feeding was 28.7 weeks. In a policy statement, the American Academy of Pediatrics (1997) stated that exclusive breast feeding provides ideal nutrition and is sufficient to support optimal growth and development for 6 months after birth. It recommended that breast-feeding continue for at least 12 months and thereafter for as long as mutually desired.

To better evaluate the impact of nursing on infants, changes in body burden were calculated using a one-compartment, first-order pharmacokinetic model (Lorber and Phillips, 2002). First, the model was validated using data from Abraham et al. (1998). Dioxin and furan concentrations for six mother/infant pairs were provided, including two breast milk measurements while the mother was feeding her infant and a blood measurement for the infant at about 1 year. These mothers' milk concentrations were used as the independent source term for the model, and the infant blood concentrations served as dependent model prediction. Other required parameters included the infant's body weight and lipid fraction over time (assigned average male and female infant values), absorption fraction (assigned a constant value of 0.80),

and, most importantly, an assumption of a rapid dissipation rate of TEQs in the infant (half-life < 1 year) during the early months of life. This dissipation rate was developed by Kreuzer et al. (1997), and it contrasts the more typical 7-year half-life found in adults for TCDD.

The average observed infant concentration was 24 pg TEQ_{DF}-WHO_{98}/g lipid, compared to a predicted concentration of 26 pg TEQ_{DF}-WHO_{98}/g lipid. The observed high and low concentrations were 5 and 44 pg TEQ_{DF}-WHO_{98}/g lipid, compared to predicted high and low concentrations in these infants of 10 and 36 pg TEQ_{DF}-WHO_{98}/g lipid. When the model was rerun at a higher TEQ dissipation rate of 7 years, the average predicted concentration rose to 39 pg TEQ_{DF}-WHO_{98}/g lipid. This demonstrated the appropriateness and importance of the assignment of a rapid dissipation rate of TEQs in infants.

This framework was used to evaluate various nursing scenarios: formula only and 6 weeks, 6 months, 1 year, and 2 years nursing. These scenarios reasonably capture the range of current nursing practices. This modeling effort required using the intake assumptions described earlier—242 pg TEQ_{DFP}-WHO_{98}/kg/day at birth and an average of 87 pg TEQ_{DFP}-WHO_{98}/kg/day over a year of breast-feeding—and other parameters noted above including the fraction of the oral dose that is absorbed into the body, changes in body weight over time, and changes in body fat fraction over time. For the infant, the half-life was less than 1 year, and during adulthood the half-life increased as the fraction of body fat increased. The longer half-life during the later years of life was based on a model presented in Michalek et al. (1996). The complete set of input values is listed in Lorber and Phillips (2002) as well as in Part I, Volume 2, Chapter 5.

The modeling results in terms of changes in lipid concentrations and body burdens as a function of age are shown in Figure 4-5**.** Some key observations include:

- For the 6-month, 1-year, and 2-year nursing scenarios, lipid concentrations peaked at around 9 weeks at 44 ppt TEQ_{DFP}-WHO_{98}. For the formula-fed infants they peaked at less than 10 ppt after the first year.

- In all four scenarios, the lipid concentrations merged at about 10 years of age at a concentration of about 13 ppt TEQ_{DFP}-WHO_{98}. Lipid and body burdens declined slightly from age 10 to about age 20 and then rose gradually through adulthood. This rise was due to the increase in half-life with age. At age 70, the modeled lipid and body burden concentrations were 13 ppt TEQ_{DFP}-WHO_{98} lipid and 5 ppt TEQ_{DFP}-WHO_{98} whole body weight.

- Breast-feeding leads to higher total lifetime exposures to TEQs as compared to formula feeding. Using an AUC approach, 70-year cumulative lifetime exposures were evaluated. The results suggest that breast-feeding added between 3% (for the 6-week breast-feeding scenario) and 18% (for the 2-year scenario) more accumulated exposure to TEQs as compared to formula-feeding.

The above analysis indicates that the average annual infant intake resulting from 1 year of nursing, 87 pg TEQ_{DFP}-WHO_{98}/kg/day, significantly exceeds the currently estimated adult intake of 1 pg TEQ_{DFP}-WHO_{98}/kg/day. The impact of nursing on infant body burdens, however, is much less, that is, infant body burdens will not exceed adult body burdens by 87 times. Rather, the modeling suggests that peak infant body burdens are only about two times the current adult body burdens (44 vs. 25 pg TEQ_{DFP}-WHO_{98}/g lipid). The reduced impact on body burden levels in nursing infants (relative to the intake) is due to the rapidly expanding infant body weight and lipid volume, and the faster elimination rate in infants. Body burden levels in nursing infants should decline in the future if, as discussed earlier, general population exposures decline.

Consumption of fish, meat, or dairy products containing elevated levels of dioxins and dioxin-like PCBs can lead to elevated exposures in comparison with the general population. The above discussion identified the general population distribution as extending up to roughly three times the mean. Most people will have exposures within this range even if they have unusual diets in terms of meat and dairy products. This is because (1) most people eat food from multiple sources, which tends to average out the contamination levels, and (2) meat and dairy products have similar dioxin levels, so substitution of one type of meat for another should not have a great impact on total exposure. Clearly, elevated exposures are possible in unusual situations, such as when an individual consumes large quantities of meat or dairy products that have significantly increased dioxin levels.

Elevated exposures resulting from fish consumption can occur in different situations. Concentrations in freshwater fish are significantly greater than in meat and dairy products; therefore, individuals who consume large quantities of freshwater fish at background contamination levels may have intakes higher than the general population distribution. A simple scenario was devised to evaluate this hypothesis. Through a review of the literature, EPA (U.S. EPA, 1997) concluded that a range of consumption of 59 to 170 g/day describes subsistence fish consumption behavior. These consumption rates were adopted to characterize the range of exposures in this scenario. Further, it is assumed that freshwater fish is the primary source of protein, that is, no meat or eggs are consumed. Assuming that all other exposure pathways stay

the same and using background exposure media concentrations, adult daily intake in this subsistence fisher scenario is calculated to range from 2.2 to 5.7 pg TEQ_{DFP}-WHO_{98}/kg-day. These intakes are about two to six times higher than the adult general population mean daily intake of 0.93 pg TEQ_{DFP}-WHO_{98}/kg-day. If subsistence fishers obtain their fish from areas where the concentration of dioxin-like chemicals in the fish is elevated, their exposure could be higher. Although this scenario appears reasonable, no clearly supportive data could be found to confirm that such highly exposed subpopulations exist in the United States.

One study that measured dioxin-like compounds in the blood of sport fishers in the Great Lakes area showed elevations over mean background but within the range of normal variability. However, another study that measured 90 PCB congeners (seven of which were dioxin-like PCBs, although PCB 126 was not measured) in the blood of sport fishers who consume high amounts of fish caught from Lake Michigan (> 26 pounds of sport fish per year) did find significant elevations of PCBs in their blood as compared to a control population (individuals consuming < 6 pounds of sport fish per year). The average total concentration of PCBs in the blood of the sport fishers was more than three times higher than that of the control population. Similarly, elevated levels of coplanar PCBs have been measured in the blood of fishers on the north shore of the Gulf of the St. Lawrence River who consume large amounts of seafood. Elevated CDD/CDF levels in human blood have been measured in Baltic fishermen. For further details on these studies see Part I, Volume 2, Chapter 5.

High exposures to dioxin-like compounds as a result of consuming meat and dairy products would most likely occur in situations where individuals consume large quantities of these foods and the level of these compounds is elevated. Most people eat meat and dairy products from multiple sources, and even if large quantities are consumed they are not likely to have unusually high exposures. Individuals who raise their own livestock for basic subsistence have the potential for higher exposures if local levels of dioxin-like compounds are high. One study in the United States showed elevated levels in chicken eggs near a contaminated soil site. European studies at several sites have shown elevated CDD/CDF levels in milk and other animal products near combustion sources, and some of these studies have also documented elevations in the levels of dioxin-like compounds in blood from families who consume their own home products.

Table 4-1. Confidence rating scheme

Confidence category	Confidence rating	Activity level estimate	Emission factor estimate
Categories/media for which emissions can be reasonably quantified			
A	High	Derived from comprehensive survey	Derived from comprehensive survey
B	Medium	Based on estimates of average plant activity level and number of plants or limited survey	Derived from testing at a limited but reasonable number of facilities believed to be representative of source category
C	Low	Based on data judged possibly nonrepresentative.	Derived from testing at only a few, possibly nonrepresentative facilities or from similar source categories
Categories/media for which emissions cannot be reasonably quantified			
D	Preliminary estimate	Based on extremely limited data, judged to be clearly nonrepresentative.	Based on extremely limited data, judged to be clearly nonrepresentative.
E	Not quantified	No data.	(1) Argument based on theory but no data (2) Data indicating dioxin formation but not in a form that allows developing an emission factor

Table 4-2. Inventory of environmental releases (grams/year) of TEQ_{DF}-WHO_{98} in the United States

Emission source category	Confidence rating[a] reference year 1995				Confidence rating[a] reference year 1987		
	A	B	C	D	A	B	C
Releases (g TEQ/yr) to air							
Waste incineration							
Municipal waste incineration		1250				8877	
Hazardous waste incineration		5.8				5	
Boilers/industrial furnaces			0.39				0.78
Medical waste/pathological incineration			488				2590
Crematoria			9.1[b]				5.5[b]
Sewage sludge incineration		14.8				6.1	
Tire combustion			0.11				0.11
Pulp and paper mill sludge incinerators[c]							
Power/energy generation							
Vehicle fuel combustion							
- leaded[d]			2				37.5
- unleaded			5.6				3.6
- diesel			33.5				27.8
Wood combustion							
- residential			62.8[b]				89.6[b]
- industrial		27.6				26.4	
Coal combustion							
- utility boilers		60.1				50.8	
- residential				30			
- commercial/Industrial				40			
Oil combustion							
- industrial/utility			10.7				17.8
- residential				6			
Other high temperature sources							
Cement kilns (hazardous waste burning)			156.1				117.8
Lightweight aggregate kilns burning hazardous waste			3.3[b]				2.4[b]
Cement kilns (nonhazardous waste burning)			17.8				13.7
Petroleum refining catalyst regeneration			2.21				2.24

Table 4-2. Inventory of environmental releases (grams/year) of TEQ_{DF}-WHO_{98} in the United States (continued)

Emission source category	Confidence rating[a] reference year 1995				Confidence rating[a] reference year 1987		
	A	B	C	D	A	B	C
Releases (g TEQ/yr) to air (continued)							
Other high temperature sources (continued)							
Cigarette combustion			0.8				1
Carbon reactivation furnaces			0.08[b]				0.06[b]
Kraft recovery boilers		2.3				2	
Combustion of landfill gas				7			
Biogas combustion				< 1			
Minimally controlled or uncontrolled combustion[e]							
Backyard barrel burning[f]			628				604
Landfill fires				1000			
Accidental fires (structural)				< 20			
Accidental fires (vehicles)				30			
Forest and brush fires				200			
Metallurgical processes							
Ferrous metal smelting/refining							
- sintering plants		28					32.7
- electric arc furnaces				40			
- foundries				20			
Nonferrous metal smelting/refining							
- primary copper		< 0.5[b]				< 0.5[b]	
- secondary aluminum			29.1				16.3
- secondary copper			271				983
- secondary lead		1.72				1.29	
- primary magnesium				15			
Coke production				7			
Drum and barrel reclamation			0.08				0.08
Chemical manufacturing/processing sources							
Ethylene dichloride/vinyl chloride		11.2[b]					
TOTAL RELEASES TO AIR[g]	3125				13515		

Table 4-2. Inventory of environmental releases (grams/year) of TEQ_{DF}-WHO_{98} in the United States (continued)

Emission source category	Confidence rating[a] reference year 1995 A	B	C	D	Confidence rating[a] reference year 1987 A	B	C
Releases (g TEQ/yr) to water							
Chemical manufacturing/ processing sources Bleached chemical wood pulp and paper mills	19.5				356		
POTW (municipal) wastewater				10			
Ethylene dichloride/vinyl chloride		0.43[b]					
Reservoir sources Urban runoff to surface water				190			
Rural soil erosion to surface water				2700			
TOTAL RELEASES TO WATER[g]	19.93				356		
Releases (g TEQ/yr) to land							
Chemical manufacturing/ processing sources Bleached chemical wood pulp and paper mill sludge	1.4				14.1		
Ethylene dichloride/vinyl chloride		0.73[b]					
Municipal wastewater treatment sludge	76.6				76.6		
Commercially marketed sewage sludge	2.6				2.6		
2,4-Dichlorophenoxy acetic acid	28.9				33.4		
TOTAL RELEASES TO LAND[g]	110.23				126.7		
OVERALL RELEASES (g/yr) TO THE OPEN AND CIRCULATING ENVIRONMENT	**3255 (SUM OF COLUMNS A, B, C)**				**13,998 (SUM OF COLUMNS A, B, C)**		

[a] The most reliable estimates of environmental releases are those sources within Categories A, B, and C, which are defined as:

A = Characterization of the Source Category judged to be **Adequate for Quantitative Estimation** with **High Confidence** in the **Emission Factor** and **High Confidence** in **Activity Level**.

Table 4-2. Inventory of environmental releases (grams/year) of TEQ_{DF}-WHO_{98} in the United States (continued)

B = Characterization of the Source Category judged to be **Adequate for Quantitative Estimation** with **Medium Confidence** in the **Emission Factor** and at least **Medium Confidence** in **Activity Level**.

C = Characterization of the Source Category judged to be **Adequate for Quantitative Estimation** with **Low Confidence** in either the **Emission Factor** and/or the **Activity Level**.

D = **Preliminary Indication** of the Potential Magnitude of I-TEQ_{DF} Emissions from "Unquantified" (i.e., Category D) Sources in Reference Year 1995. **Based on extremely limited data, judged to be clearly nonrepresentative**.

[b] Congener-specific emissions data were not available; the I-TEQ estimate was used as a surrogate for the TEQ_{DF}-WHO_{98} emissions estimate.

[c] Included within estimate for Wood Combustion - industrial.

[d] Leaded fuel production and the manufacture of motor vehicle engines requiring leaded fuel for highway use have been prohibited in the United States. (See Section 4.1 for details.)

[e] This refers to conventional pollutant control, not dioxin emissions control. Very few of the sources listed in this inventory control specifically for CDD/CDF emissions.

[f] This term refers to the burning of residential waste in barrels.

[g] TOTAL reflects only the total of the estimates made in this report.

Table 4-3. Sources that are currently unquantifiable (Category E)[a]

Category	Unquantified sources
Combustion sources	Uncontrolled combustion of PCBs Agricultural burning
Metal smelting and refining	Primary aluminum Primary nickel
Chemical manufacturing	Mono- to tetrachlorophenols Pentachlorophenol Chlorobenzenes Chlorobiphenyls (leaks/spills) Dioxazine dyes and pigments 2,4-Dichlorophenoxy acetic acid Tall oil-based liquid soaps
Biological and photochemical processes	Composting
Reservoir sources	Air Sediments Water Biota PCP-treated wood

[a] There exist no or insufficient data characterizing environmental releases from these sources. Therefore, it is currently not possible to arrive at an estimate of annual environmental releases.

Table 4-4. Summary of North American CDD/CDF and PCB TEQ-WHO$_{98}$ levels in environmental media and food[a]

Media	CDD/CDFs[b]	PCBs[b]
Urban soil, ppt	n= 270 9.3 ± 10.2 Range = 2–21	n = 99 2.3
Rural soil, ppt	n = 354 2.7 Range = 0.11–5.7	n = 62 0.59
Sediment, ppt	n=11 5.3 ± 5.8 Range = <1–20	n = 11 0.53 ± 0.69
Urban air, pg/m^3	n=106 0.12 ± 0.094 Range = 0.03–0.2	n=53 0.0009
Rural air, pg/m^3	n=60 0.013 Range = 0.004–0.02	n=53 0.00071
Freshwater fish and shellfish, ppt[c]	n=222 1.0 (NA[d])	n = 1 composite of 10 samples plus 6 composites 1.2[e] (NA[d])
Marine fish and shellfish, ppt[c]	n=158 0.26 (NA[d])	n = 1 composite of 13 samples plus 5 composites 0.25 (NA[d])
Water, ppq	n=236 0.00056 ± 0.00079 (NA[d])	NA[d]
Milk, ppt (Note: each composite for CDD/F/PCB comprised of 40+ U.S. regional samples)	n=8 composites 0.018[e]	n = 8 composites 0.0088[e]
Dairy, ppt[f]	n = 8 composites 0.12[e]	n = 8 composites 0.058[e]
Eggs, ppt (Note: each composite for CDD/F data comprised of 24 eggs)	n=15 composites 0.081[e]	n = 18 plus 6 composites 0.10[e] (NA[d])

Table 4-4. Summary of North American CDD/CDF and PCB TEQ-WHO_{98} levels in environmental media and food (continued)

Media	CDD/CDFs[b]	PCBs[b]
Beef ppt	n=63 0.18 ± 0.11 Range = 0.11–0.95	n = 63 0.084
Pork, ppt	n=78 0.28 ± 0.28 Range = 0.15–1.8	n = 78 0.012
Poultry, ppt	n=78 0.068 ± 0.070 Range = 0.03–0.43	n = 78 0.026
Vegetable fats, ppt	n=30 0.056 ± 0.24[g] (NA[d])	n = 5 composites 0.037[e]

[a] Whole-weight basis; concentrations provided in parenthesis for food products are calculated at ND = 0.
[b] Values are the arithmetic mean TEQs and standard deviations. Nondetects were set to one-half the limit of detection, except for soil and CDD/CDFs in vegetable fats for which nondetects were set to zero.
[c] The TEQ_{df} fish concentrations reported here are species-specific ingestion rate weighted averages.
[d] NA = not available; congener-specific PCB data and data to calculate TEQ concentrations at ND = 0 are limited.
[e] Standard deviations could not be calculated due to limitations associated with the data (i.e., composite analyses).
[f] TEQ calculated by setting nondetects to zero.
[g] Dairy concentration calculated from milk lipid concentrations and then assuming a fat fraction for dairy.

Table 4-5. Background serum levels in the United States 1995–1997

Value	TEQ_{DFP}-WHO_{98} (pg/g lipid)	2,3,7,8-TCDD (pg/g lipid)
Median	18.7	1.9
Mean	22.1[a]	2.1
95th Percentile	38.8	4.2

[a] After adjusting to account for missing PCBs, the mean is 25.4 pg/g lipid.

Source: CDC, 2000

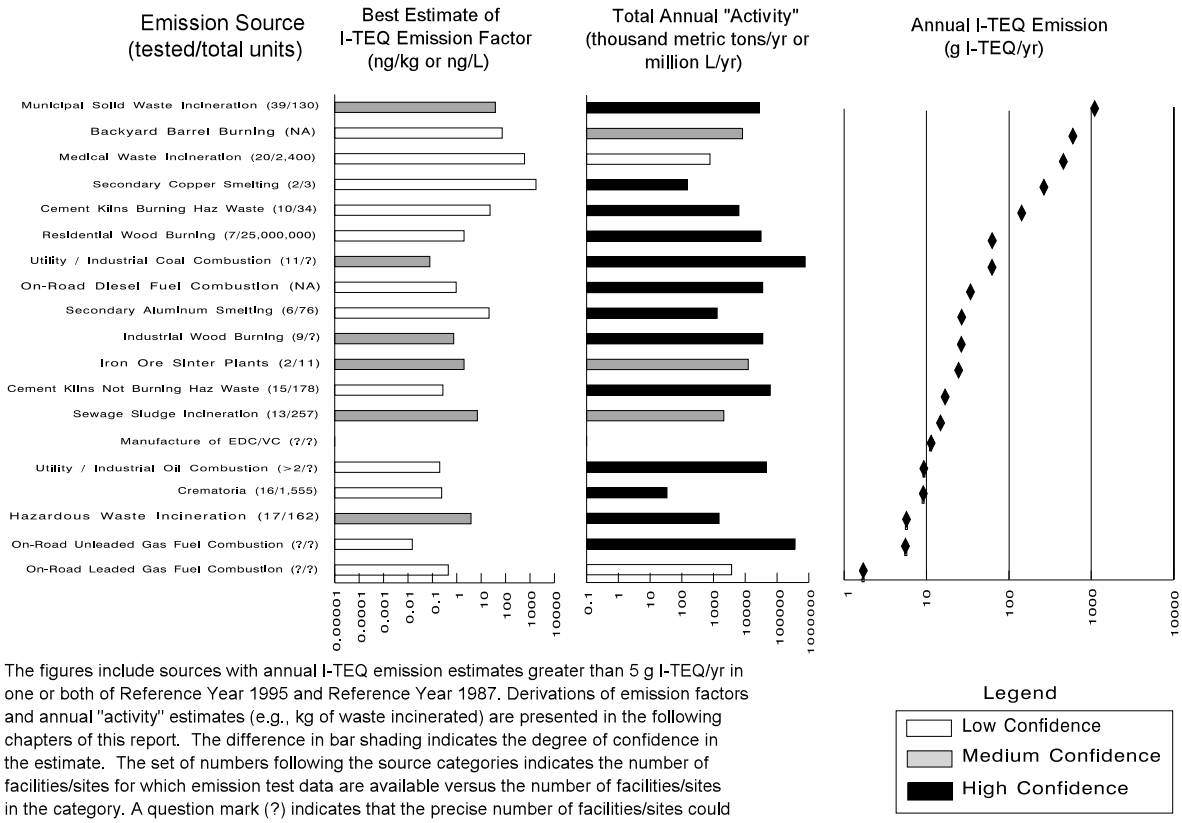
Table 4-6. Adult contact rates and background intakes of dioxin-like compounds

Exposure route	Contact rate	Dioxins and furans		Dioxin-like PCBS		Total
		Concentration TEQ_{DF}-WHO_{98}	Intake (pg TEQ_{DF}-WHO_{98}/kg-d)	Concentration TEQ_{P}-WHO_{98}	Intake (pg TEQ_{P}-WHO_{98}/kg-d)	Intake (pg TEQ_{DFP}-WHO_{98} /kg-d)
Soil ingestion	50 mg/d	9.3 pg/g	0.0066	2.3 ppt	0.0016	0.0082
Soil dermal	12 g/d	9.3 pg/g	0.0016	2.3 ppt	0.00039	0.002
Freshwater fish and shellfish[a]	5.9 g/d	1.0 pg/g	0.084	1.2 pg/g	0.1	0.18
Marine fish and shellfish[a]	9.6 g/d	0.26 pg/g	0.036	0.25 pg/g	0.034	0.07
Inhalation	13.3 m^3/d	0.12 pg/m^3	0.023	NA	NA	0.023
Milk	175 g/d	0.018 pg/g	0.045	0.0088 pg/g	0.022	0.067
Dairy	55 g/d	0.12 pg/g	0.094	0.058 pg/g	0.046	0.14
Eggs	0.24 g/kg-d	0.081 pg/g	0.019	0.10 pg/g	0.024	0.043
Beef	0.67 g/kg-d	0.18 pg/g	0.13	0.084 pg/g	0.06	0.19
Pork	0.22 g/kg-d	0.28 pg/g	0.062	0.012 pg/g	0.0026	0.065
Poultry	0.5 g/kg-d	0.068 pg/g	0.034	0.026 pg/g	0.013	0.047
Other meats	0.35 g/kg-d	0.18 ppt	0.062	0.041 pg/g	0.014	0.076
Vegetable fat	17 g/d	0.056 pg/g	0.014	0.037 pg/g	0.009	0.023
Water	1.4 L/d	0.0005 pg/L	0.000011	NA	NA	0.000011
Total			**0.61 (43 pg/d)**		**0.33 (23 pg/d)**	**0.94 (66 pg/d)**

[a] The TEQ_{df} fish concentrations reported here are species-specific ingestion rate weighted averages.

Table 4-7. Variability in average daily toxic equivalent (TEQ) intake as a function of age

Age range	Intake, mass basis pg TEQ_{DFP}-WHO_{98}/d	Intake, body weight basis pg TEQ_{DFP}-WHO_{98}/kg-d
1–5 years	50	3.3
6–11 years	54	1.8
12–19 years	61	1.1
Adult	66	0.9



The figures include sources with annual I-TEQ emission estimates greater than 5 g I-TEQ/yr in one or both of Reference Year 1995 and Reference Year 1987. Derivations of emission factors and annual "activity" estimates (e.g., kg of waste incinerated) are presented in the following chapters of this report. The difference in bar shading indicates the degree of confidence in the estimate. The set of numbers following the source categories indicates the number of facilities/sites for which emission test data are available versus the number of facilities/sites in the category. A question mark (?) indicates that the precise number of facilities/sites could not be estimated.

Figure 4-1. Estimated CDD/CDF I-TEQ emissions to air from combustion sources in the United States, 1995.

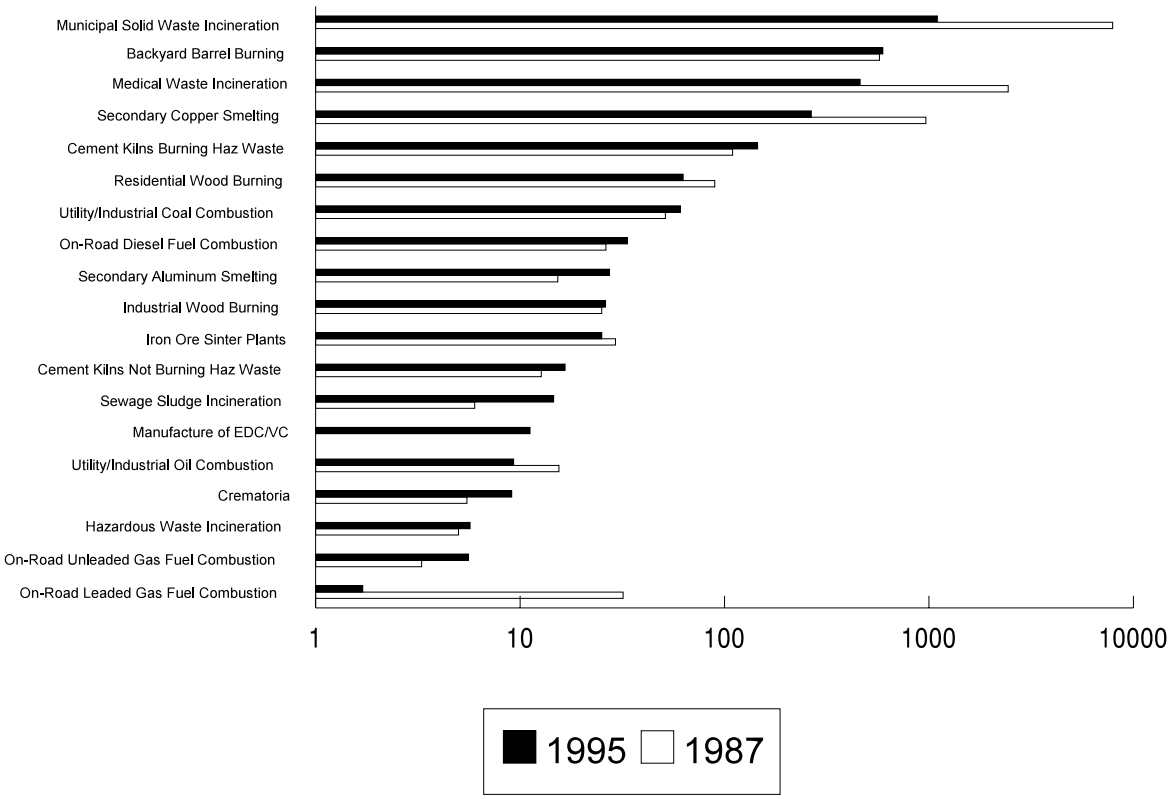


Figure 4-2. Comparison of estimates of annual I-TEQ emissions to air (grams I-TEQ/yr) for reference years 1987 and 1995.

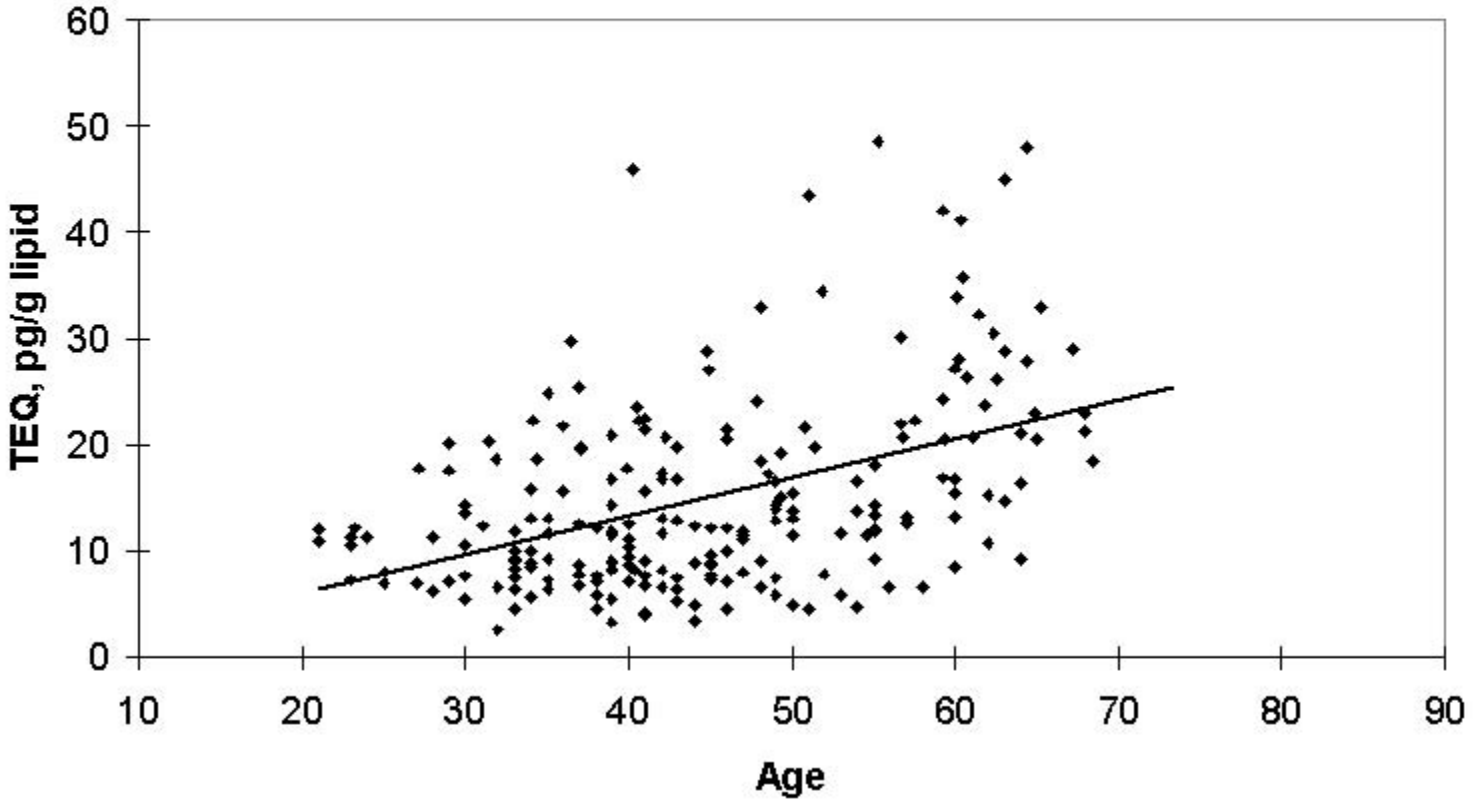


Figure 4-3. Blood levels (I-TEQ for CDD/CDF + WHO_{94}) versus age of a subset of participants in the CDC (2000).

Source: ATSDR, 1999b

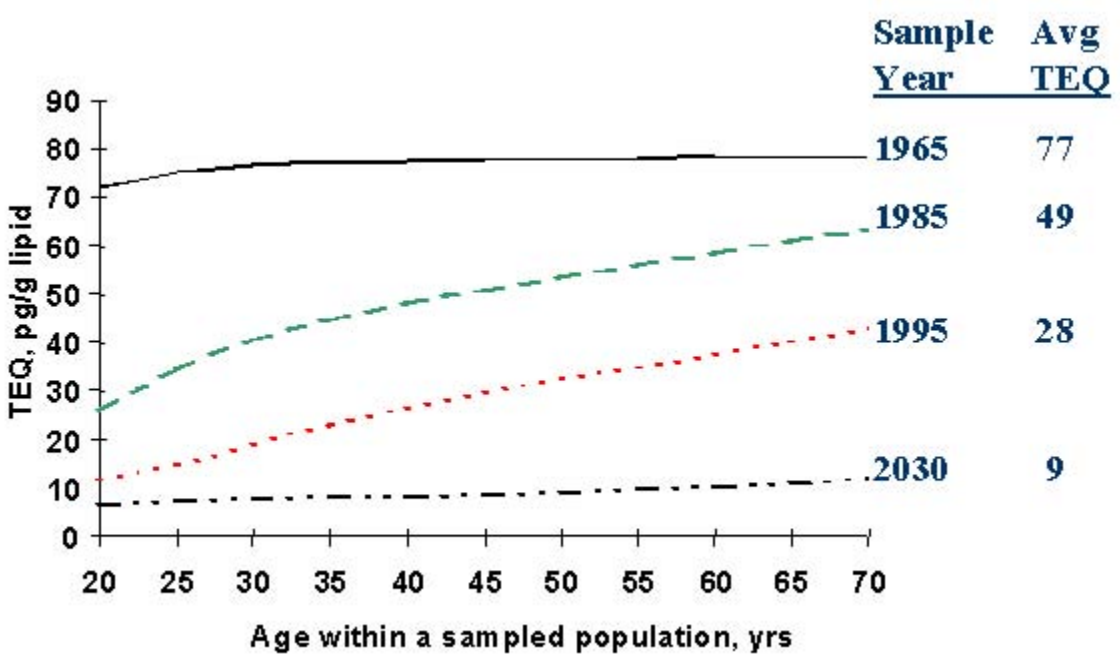
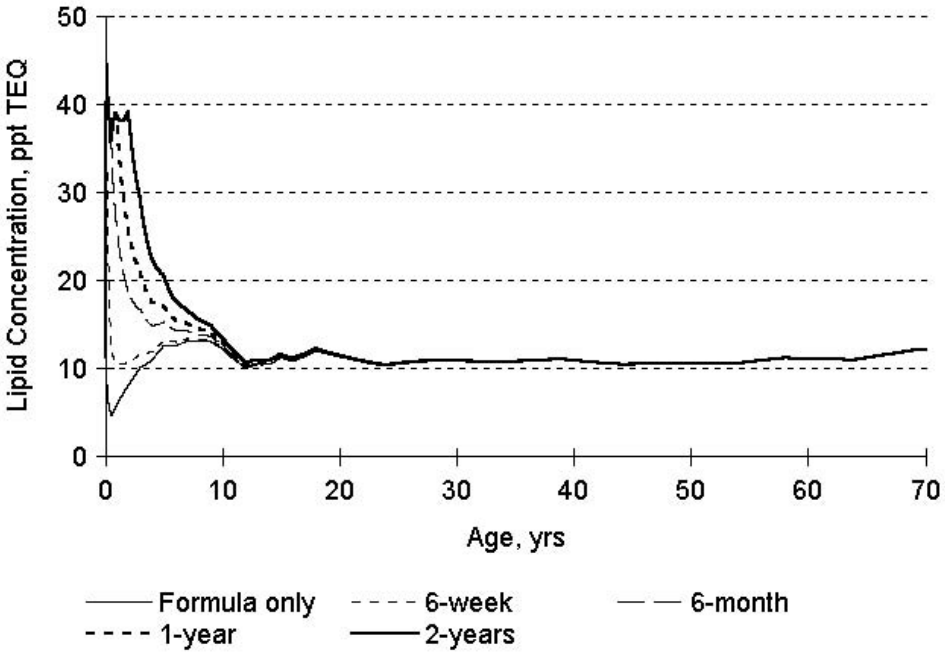


Figure 4-4. Predicted distributions and average TEQ_{DF} - WHO_{98} concentrations within an adult population for four years: 1965, 1985, 1995, and 2030. (CDD/CDFs only, not PCBs).

Source: Adapted from Lorber, 2002

(A)



(B)

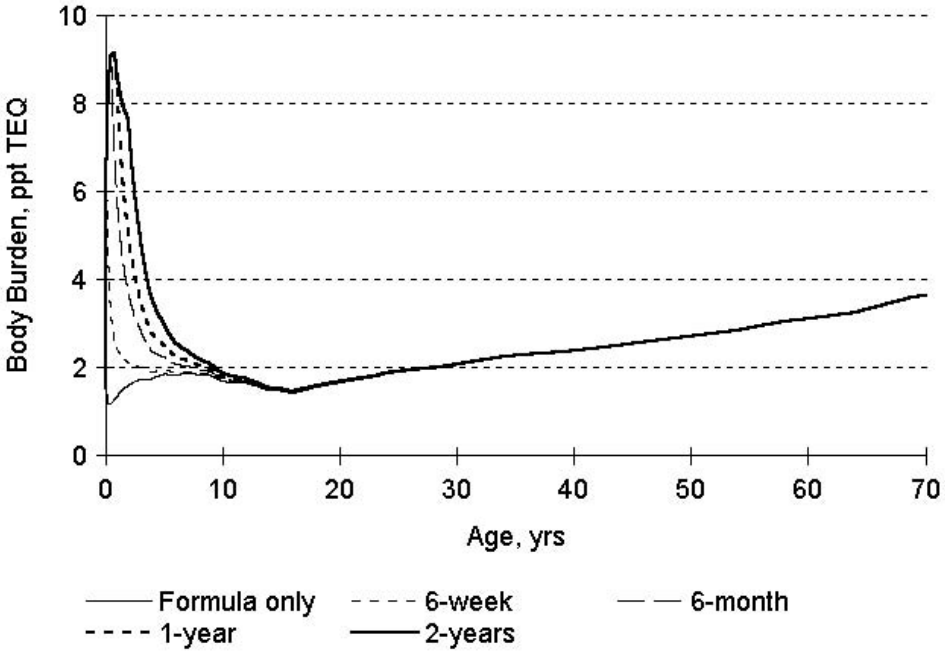


Figure 4-5. Demonstration of the model for evaluating impacts on lipid concentrations (A) and body burdens (B) of infants resulting from various nursing scenarios during a lifetime.