1.0. BACKGROUND AND SUMMARY

1.1. BACKGROUND

This reassessment is comprised of three reports:

Part 1. *Estimating Exposure to Dioxin-Like Compounds* (U.S. EPA, 2000a) (which expanded upon a 1988 draft exposure report titled, *Estimating Exposure to 2,3,7,8-TCDD* [U.S. EPA, 1988]);
Part 2. *Health Assessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds* (U.S. EPA, 1994; U.S. EPA, 2000b); and
Part 3. *Dioxin: Integrated Summary and Risk Characterization for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds* (U.S. EPA, 2000c).

Throughout the remainder of this document, these three parts as a whole will be abbreviated as the Reassessment Documents, and the individual parts will be referred to as the Exposure Reassessment Document, the Health Reassessment Document, and the Risk Characterization. The Exposure Reassessment Document has expanded to three volumes, as discussed below. Volumes 1 and 2 of the Exposure Reassessment Document are summarized in Section 4 of the Risk Characterization.

The process for developing the Reassessment Documents has been open and participatory. Each of the documents has been developed in collaboration with scientists from inside and outside the Federal Government. Each document has undergone extensive internal and external review, including review by EPA's Science Advisory Board (SAB). In September 1994, drafts of each document were made available for public review and comment. This included a 150-day comment period and 11 public meetings around the country to receive oral and written comments. These comments, along with those of the SAB (U.S. EPA, 1995), have been considered in the drafting of this final document. The Dose-Response Chapter of the Health Document underwent peer review in 1997 (U.S. EPA, 1997); an earlier version of the Integrated Summary and Risk Characterization underwent development and review in 1997 and 1998, and comments have been incorporated. In 1998, EPA released a workshop review version of the sources inventory (U.S. EPA, 1998), one of the three volumes of the Exposure Reassessment Document. In addition, as requested by the SAB, a chapter on Toxic Equivalency has been developed and underwent external peer review in parallel with the Integrated Summary and Risk Characterization in July 2000. The November 2000, review by the SAB of the Dose-Response Chapter, the Toxic Equivalency Chapter and the Integrated

Summary and Risk Characterization was the final step in this open and participatory process of reassessment. The full set of background documents and the integrative summary and risk characterization replace the previous dioxin assessments as the scientific basis for EPA decision-making.

The final Exposure Reassessment Document reflects changes made as a result of both review comments and analyses of a variety of other types of information that has come available. These include relevant information obtained from published peer-reviewed literature, EPA program offices, and other Federal agencies. This version of the Exposure Reassessment Document is current in this regard through 2000.

The purpose of the Exposure Reassessment Document is threefold: 1) to inventory the known sources of release of dioxins into the environment, 2) to develop an understanding of dioxins in the environment, including fate and transport properties, environmental and exposure media concentrations, background as well as elevated exposures, and temporal trends in exposure, and 3) provide site-specific procedures for evaluating the incremental exposures due to specific sources of dioxin-like compounds. Following this structure, the Exposure Reassessment Document is presented in three volumes:

Volume 1 - Sources of Dioxin-Like Compounds in the United States

This volume presents a comprehensive review of known sources of environmental releases of dioxin-like compounds in the United States. It includes an inventory of known source activity in terms of estimates of annual releases of dioxin-like compounds into the U.S. environment (i.e., air, water and land). This inventory is specific for two reference years, 1987 and 1995. From these data, it is possible to compare and contrast releases of dioxin-like compounds among the sources and between the reference years.

Volume 2 - Properties, Environmental Levels, and Background Exposures

This volume presents and evaluates information on the physical-chemical properties, environmental fate, environmental and exposure media levels, background and elevated human exposures, and temporal trends of dioxin-like compounds in the U.S. environment during the 20th century.

Volume 3 - Site-Specific Assessment Procedures

This volume presents procedures for evaluating the incremental impact from sources of dioxin release into the environment. The sources covered include contaminated soils, stack emissions, and point discharges into surface water. This volume includes sections on: exposure parameters and exposure scenario development; stack emissions and atmospheric transport modeling; aquatic and

terrestrial fate, and food chain modeling; demonstration of methodologies; and uncertainty evaluations including exercises on sensitivity analysis and model validation, review of Monte Carlo assessments conducted for dioxin-like compounds, and other discussions.

The primary technical resource supporting the development of the inventory of sources of dioxin-like compounds discussed in Volume I (above) is the Database of Sources of Environmental Releases of Dioxin-Like Compounds in the United States (EPA/600/C-01/012. March 2001). This database includes congener-specific CDD and CDF emissions data extracted from original engineering test reports. It has been published independently from the Reassessment and is available on Compact Disk-Read only Memory (CD-ROM), without cost, from EPA's National Service Center for Environmental Publications (NSCEP) in Cincinnati, Ohio (telephone: 1-800-490-9198, or 513-489-8190; fax: 513-489-8695). Summary files from the database will be available for downloading from the Web page of the National Center for Environmental Assessment, www.epa.gov/ncea/dioxin.htm. Instructions on how to order and obtain the CD-ROM will also be available on the Web page.

1.2. DEFINITION OF DIOXIN-LIKE COMPOUNDS

This assessment addresses specific compounds in the following chemical classes: polychlorinated dibenzo-*p*-dioxins (PCDDs or CDDs), polychlorinated dibenzofurans (PCDFs or CDFs), polybrominated dibenzo-*p*-dioxins (PBDDs or BDDs), polybrominated dibenzofurans (PBDFs or BDFs), and polychlorinated biphenyls (PCBs), and describes this subset of chemicals as "dioxin-like." Dioxin-like refers to the fact that these compounds have similar chemical structure, similar physical-chemical properties, and invoke a common battery of toxic responses. Because of their hydrophobic nature and resistance towards metabolism, these chemicals persist and bioaccumulate in fatty tissues of animals and humans. The CDDs include 75 individual compounds; CDFs include 135 different compounds. These individual compounds are referred to technically as congeners. Likewise, the BDDs include 75 different congeners and the BDFs include an additional 135 congeners. Only 7 of the 75 congeners of CDDs, or of BDDs, are thought to have dioxin-like toxicity; these are ones with chlorine/bromine substitutions in, at a minimum, the 2, 3, 7, and 8 positions. Only 10 of the 135 possible congeners of CDFs or of BDFs are thought to have dioxin-like toxicity; these also are ones with substitutions in the 2, 3, 7, and 8 positions. This suggests that 17 individual CDDs/CDFs, and an additional 17 BDDs/BDFs, exhibit dioxin-like toxicity. The database on many of the brominated

compounds regarding dioxin-like activity has been less extensively evaluated, and these compounds have not been explicitly considered in this assessment.

There are 209 PCB congeners. Only 13 of the 209 congeners are thought to have dioxin-like toxicity; these are PCBs with 4 or more lateral chlorines with 1 or no substitution in the ortho position. These compounds are sometimes referred to as coplanar, meaning that they can assume a flat configuration with rings in the same plane. Similarly configured polybrominated biphenyls (PBBs) are likely to have similar properties. However, the database on these compounds with regard to dioxin-like activity has been less extensively evaluated, and these compounds have not been explicitly considered in this assessment. Mixed chlorinated and brominated congeners of dioxins, furans, and biphenyls also exist, increasing the number of compounds potentially considered dioxin-like within the definitions of this assessment. The physical/chemical properties of each congener vary according to the degree and position of chlorine and/or bromine substitution. Very little is known about occurrence and toxicity of the mixed (chlorinated and brominated) dioxin, furan, and biphenyl congeners. Again, these compounds have not been explicitly considered in this assessment. Generally speaking, this assessment focuses on the 17 CDDs/CDFs and a few of the coplanar PCBs that are frequently encountered in source characterization or environmental samples. While recognizing that other "dioxin-like" compounds exist in the chemical classes discussed above (e.g., brominated or chlorinated/brominated congeners) or in other chemical classes (e.g., halogenated naphthalenes or benzenes, azo- or azoxybenzenes), the evaluation of less than two dozen chlorinated congeners is generally considered sufficient to characterize environmental "dioxin."

The chlorinated dibenzodioxins and dibenzofurans are tricyclic aromatic compounds with similar physical and chemical properties. Certain of the PCBs (the so-called coplanar or mono-ortho coplanar congeners) are also structurally and conformationally similar. The most widely studied of this general class of compounds is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). This compound, often called simply "dioxin," represents the reference compound for this class of compounds. The structure of TCDD and several related compounds is shown in Figure 1-1. Although sometimes confusing, the term "dioxin" is often also used to refer to the complex mixtures of TCDD and related compounds emitted from sources, or found in the environment or in biological samples. It can also be used to refer to the total TCDD "equivalents" found in a sample. This concept of toxic equivalency is discussed below.

1.3. TOXIC EQUIVALENCY FACTORS

CDDs, CDFs, and PCBs are commonly found as complex mixtures when detected in environmental media and biological tissues, or when measured as environmental releases from specific sources. Humans are likely to be exposed to variable distributions of CDDs, CDFs, and dioxin-like PCB congeners that vary by source and pathway of exposures. This complicates the human health risk assessment that may be associated with exposures to variable mixtures of dioxin-like compounds. In order to address this problem, the concept of toxic equivalency has been considered and discussed by the scientific community, and TEFs have been developed and introduced to facilitate risk assessment of exposure to these chemical mixtures.

On the most basic level, TEFs compare the potential toxicity of each dioxin-like compound comprising the mixture to the well-studied and understood toxicity of TCDD, the most toxic member of the group. The background and historical perspective regarding this procedure is described in detail in Part II, Chapter 9, Section 9.1, 9.2, and in Agency documents (U.S. EPA, 1987; 1989a,b; 1991). This procedure involves assigning individual TEFs to the 2,3,7,8-substituted CDD/CDF congeners and "dioxin-like" PCBs. To accomplish this, scientists have reviewed the toxicological databases along with considerations of chemical structure, persistence, and resistance to metabolism, and have agreed to ascribe specific, "order of magnitude" TEFs for each dioxin-like congener relative to TCDD, which is assigned a TEF of 1.0. The other congeners have TEF values ranging from 1.0 to 0.00001. Thus, these TEFs are the result of scientific judgment of a panel of experts using all of the available data and are selected to account for uncertainties in the available data and to avoid underestimating risk. In this sense, they can be described as

$$TEQ \cong \sum_{i=n}\left(Congener_i \times TEF_i\right) + \left(Congener_j \times TEF_j\right) + \ldots\ldots \left(Congener_n \times TEF_n\right) \quad (1\text{-}1)$$

"public health conservative" values. To apply this TEF concept, the TEF of each congener present in a mixture is multiplied by the respective mass concentration and the products are summed to represent the 2,3,7,8-TCDD Toxic Equivalence (TEQ) of the mixture, as determined by Equation (1-1):

The TEF values for PCDDs and PCDFs were originally adopted by international convention (U.S. EPA, 1989a). Subsequent to the development of the first international TEFs for CDD/CDFs, these values were further reviewed and/or revised and TEFs were also developed for PCBs (Ahlborg et al., 1994; van den Berg et al., 1998). A problem arises in that past and present quantitative exposure and risk assessments may not have clearly identified which of three TEF schemes was used to estimate the TEQ. This reassessment introduces a new uniform TEQ nomenclature that clearly distinguishes between the

different TEF schemes and identifies the congener groups included in specific TEQ calculations. The nomenclature uses the following abbreviations to designate which TEF scheme was used in the TEQ calculation:

1. I-TEQ refers to the International TEF scheme adopted by EPA in 1989 (U.S. EPA, 1989a). See Table 1-1.
2. TEQ-WHO_{94} refers to the 1994 WHO extension of the I-TEF scheme to include 13 dioxin-like PCBs (Ahlborg et al., 1994). See Table 1-2.
3. TEQ-WHO_{98} refers to the 1998 WHO update to the previously established TEFs for dioxins, furans, and dioxin-like PCBs (van den Berg et al., 1998). See Table 1-3.

The nomenclature also uses subscripts to indicate which family of compounds is included in any specific TEQ calculation. Under this convention, the subscript D is used to designate dioxins, the subscript F to designate furans and the subscript P to designate PCBs. As an example, "TEQ_{DF}-WHO_{98}" would be used to describe a mixture for which only dioxin and furan congeners were determined and where the TEQ was calculated using the WHO_{98} scheme. If PCBs had also been determined, the nomenclature would be "TEQ_{DFP}-WHO_{98}." Note that the designations TEQ_{DF}-WHO_{94} and I-TEQ_{DF} are interchangeable, as the TEFs for dioxins and furans are the same in each scheme. Note also that in this document, I-TEQ sometimes appears without the D and F subscripts. This indicates that the TEQ calculation includes both dioxins and furans.

This reassessment recommends that the WHO_{98} TEF scheme be used to assign toxic equivalency to complex environmental mixtures for assessment and regulatory purposes. Sections in the Health Reassessment Document, and summarized in the Risk Characterization, describe the mode(s) of action by which dioxin-like chemicals mediate biochemical and toxicological actions. These data provide the scientific basis for the TEF/TEQ methodology. In its 20-year history, the approach has evolved, and decision criteria supporting the scientific judgment and expert opinion used in assigning TEFs has become more transparent. Numerous states, countries, and several international organizations have evaluated and adopted this approach to evaluating complex mixtures of dioxin and related compounds. It has become the accepted methodology, although the need for research to explore alternative approaches is widely endorsed. Clearly, basing risk on TCDD alone or assuming all chemicals are equally potent to TCDD is inappropriate on the basis of available data. Although uncertainties in the use of the TEF methodology have been identified (which are described in detail in the Health Reassessment Document, Chapter 9, Section 9.5), one must examine the use of this method in the broader context of the need to evaluate the potential public health impact of complex mixtures of

persistent, bioaccumulative chemicals. It can be generally concluded that the use of TEF methodology for evaluating complex mixtures of dioxin-like compounds decreases the overall uncertainties in the risk assessment process as compared to alternative approaches. Use of the latest consensus values for TEFs assures that the most recent scientific information informs this "useful, interim approach" (U.S. EPA, 1989a; Kutz et al., 1990) to dealing with complex environmental mixtures of dioxin-like compounds. As stated by the U.S. EPA Science Advisory Board (U.S. EPA, 1995), "The use of the TEFs as a basis for developing an overall index of public health risk is clearly justifiable, but its practical application depends on the reliability of the TEFs and the availability of representative and reliable exposure data." EPA will continue to work with the international scientific community to update these TEF values to assure that the most up-to-date and reliable data are used in their derivation and to evaluate their use on a periodic basis.

A chemical is assigned a TEF value based on all the available data comparing the chemical to either TCDD or PCB 126. In addition, there are weighting criteria that place more emphasis on chronic and subchronic studies examining toxic endpoints (van den Berg et al., 1998). There is a broad range in the quantity and quality of the data available for individual congeners. For example, the TEF for PCB 126 is based on over 60 in vivo endpoints examining responses as diverse as enzyme induction, developmental toxicity, immunotoxicity, hepatic toxicity, alterations in hormones and tumor promotion, while the TEF for 3,4,4',5-tetrachlorobiphenyl (PCB 81) is based on in vitro CYP1A induction and QSAR calculations. Fortunately, PCB 81 does not significantly contribute to human TEQ exposures. There are 5 congeners that contribute approximately 80% of the total TEQ in humans: 2,3,7,8-TCDD, 1,2,3,7,8-PCDD, 1,2,3,6,7,8-HxCDD, 2,3,4,7,8-PCDF, and PCB 126 (See Part I, Volume 2 and Section 4.4.3 of this document). With the exception of 1,2,3,6,7,8-HxCDD, the TEFs for these chemicals are based on a number of different endpoints from multiple studies performed in different laboratories. The TEF for 1,2,3,6,7,8-HxCDD is based on a two-year bioassay in which rats were exposed to a mixture of 1,2,3,6,7,8-HxCDD and 1,2,3,7,8,9-HxCDD. The TEFs for 2,3,4,7,8-PCDF and PCB 126 are similar to the mean REP value for all in vivo endpoints and are similar to their REPs for tumor promotion. The TEF for 12378-PCDD is based largely on its REP for tumor promotion in rats. From these data, it is clear that the chemicals that contribute approximately 80% to the total human TEQ are well studied and the assigned TEFs provide reasonable estimates of the relative potency of these chemicals. In contrast, while there are some chemicals in the TEF methodology which have minimal data sets to reliably assess their relative potency, these chemicals do not contribute substantially to the human blood TEQ.

The ability of the TEF methodology to predict the biological effects of mixtures containing dioxin-like chemicals has been evaluated in a number of experimental systems. These studies generally demonstrate that the assumption of additivity provides a reasonable estimate of the dioxin-like potential of a mixture (described in the Health Reassessment Document, Chapter 9, Section 9.4). In addition, there are examples of non-additive interactions between dioxins and non-dioxins. Both greater than additive and less than additive interactions have been observed in these studies. In general the non-additive interactions between the dioxins and non-dioxins have been observed at doses that are considerably higher than present background human exposures.

There are a number of natural chemicals that bind and activate the AhR and induce some dioxin-like effects. It has been proposed by some scientists that these chemicals contribute significantly to the total TEQ exposures and that these exposures far out weigh those from PCDDs, PCDFs and PCBs (Safe, 1995). While this hypothesis is intriguing, there are several limitations to these analyses. The in vivo data on the natural aromatic hydrocarbon receptor (AhR) ligands is limited to enzyme induction and a single developmental study. Few, if any, toxicology studies demonstrating clear dioxin-like toxicities have been published. The natural AhR ligands are rapidly metabolized and result in both transient tissue concentrations and transient effects. The natural ligands also have significant biological effects that are independent of the AhR and it is not clear as to the role of the AhR in the biological effects of these chemicals. Clearly this issue requires further research in order to better understand the relative potential health effect of dioxin and related chemicals as compared to natural AhR ligands.

One of the limitations of the use of the TEF methodology in risk assessment of complex environmental mixtures is that the risk from non-dioxin-like chemicals is not evaluated in concert with that of dioxin-like chemicals. Another limition of the TEF methodology is their application to non-biological samples. The fate and distribution of PCDDs, PCDFs and PCBs are not necessarily related to their TEF. Thus, the use of the TEF for non-biological media must be done cautiously. Future approaches to the assessment of environmental mixtures should focus on the development of methods that will allow risks to be predicted when multiple mechanisms are present from a variety of contaminants.

1.4. CONTENTS OF THIS VOLUME

The purpose of this volume is to: (1) summarize information on the physical and chemical properties of dioxin-like compounds; (2) provide an overview of the levels of dioxin-like compounds found in environmental media and food; (3) estimate background

exposures to dioxin-like compounds for the general population of the United States; (4) provide information on the potential for elevated exposures among certain subpopulations of the United States; and (5) summarize the evidence that suggests a downward trend in dioxin-like concentrations in the environment, as well as trends in exposure. These topics are organized in this volume as follows:

Chapter 2 - Physical and Chemical Properties and Fate

This chapter summarizes available information regarding the physical and chemical properties and fate of the dioxin-like compounds. Physical/ chemical properties addressed in Chapter 2 include melting point, water solubility, vapor pressure, Henry's Law constant, octanol/water partition coefficient, organic carbon partition coefficient, and photochemical quantum yield. Fate and transport processes addressed include photolysis, oxidation, hydrolysis, biodegradation, volatilization, and sorption. Biologically-mediated transport properties (i.e., bioconcentration, plant uptake, etc.) are also addressed in this volume. (These properties are also addressed in Volume 3: Site-Specific Assessment Procedures.) These data were compiled from a review of the current scientific literature on dioxin-like compounds.

Chapter 3 - Levels of CDD, CDF, and PCB Congeners

This chapter provides an overview of the concentrations at which dioxin-like compounds have been found in the environment and food based on data presented in the recent published literature. Data are presented for air, soil, sediment, water, and foods. For foods, the general focus is on foods with relatively high fat content (i.e., beef, pork, poultry and eggs, milk and dairy products, fish, and vegetable fats) because these items are most likely to contain dioxins and related compounds. Data from Government-sponsored monitoring studies and studies reported in the peer-reviewed literature are used in this chapter to estimate U.S. background concentrations of dioxin-like compounds in the various environmental media and foods. In order to represent current exposure concentrations, data used for the calculation of background media levels are based on studies published in the late 1980s and 1990s, but primarily in the 1990s. The studies used for the estimation of background concentrations were also chosen on the basis of credibility and representativeness. CDD/CDF profiles for environmental media are also presented in this chapter.

Chapter 4 - Human Exposures to CDD/CDF, and PCB Congeners

This chapter assesses background exposures to the dioxin-like compounds among the general population of the United States. Recent assessments of background exposures cited in the scientific literature are summarized, and background exposure estimates, based on the data presented in Chapter 3, are presented. Data on the concentrations of dioxin-like compounds in human tissue (i.e., adipose tissue, blood, and human breast milk) are also presented. Two methods are used in to estimate background daily intake of dioxin-like compounds. One method estimates background exposures based on pharmacokinetic modeling using the human tissue data. The other derives background exposure estimates from dietary intake and contact with other media containing dioxin-like compounds. The primary focus of this chapter is background exposure among the general population.

Chapter 5 - Potentially Elevated Populations

This chapter focuses on elevated exposures that may occur among the general population from dietary habits such as breast feeding or high rates of fish ingestion, increased environmental levels of dioxin-like compounds from localized sources, or cigarette smoking. This chapter does not, however, address occupational or accidental exposure. Epidemiological studies that have evaluated whether elevated dioxin exposure has occurred to certain workers in the chemical industry, members of the Air Force who worked with Agent Orange, and residents of Seveso, Italy, who were exposed as a result of a pesticide plant explosion are fully discussed in the Epidemiology Chapter of the Dioxin Health Reassessment Document.

Chapter 6 - Temporal Trends

This chapter describes trends in the levels of dioxin-like compounds that have been observed in various environmental media and foods, as well as evidence of downward trends in exposure to dioxin-like compounds in humans. The downward trend in human exposure is supported by a modeling exercise that reconstructs the most likely past doses of dioxin-like compounds contributing to observed body burdens. Reviews of several studies and the modeling exercise are followed by several key observations with regard to temporal trends of dioxin-like compounds.

1.5. SUMMARY OF FINDINGS IN THIS VOLUME

1.5.1. Physical and Chemical Properties and Fate

The physical/chemical properties of individual dioxin congeners vary and the various congeners 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. Therefore, for purposes of environmental fate modeling, it is important to use the individual CDD/CDF and PCB congeners values, rather than TEQs. Estimates of environmental releases are presented in Volume 1. Full congener-specific release rates for most sources are provided in an electronic database which is available as a companion to this document (Database of Sources of Environmental Releases of Dioxin-Like Compounds in the United States (EPA/600/C-01/012). In 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. The following paragraphs provide a summary of the fate of dioxin-like compounds.

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, generally classified as semivolatile, and tend to bioaccumulate in animals. Some evidence has shown that these compounds can degrade in the environment, but in general they are considered 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 either as resuspended soil particles or as vapors. In water, they can be resuspended into the water column from sediments, volatilized out of the surface waters into the atmosphere or become buried in deeper sediments. Immobile sediments appear to serve as permanent sinks for the dioxin-like compounds. Though not always considered an environmental compartment, these compounds are also found in anthropogenic materials (such as PCP) and have the potential to be released from these materials into the broader environment.

Atmospheric transport and deposition of the dioxin-like compounds are a primary means of dispersal of these compounds throughout the environment. The dioxin-like compounds can be 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 with similar properties, and this 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, 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.

1.5.2. Environmental Media and Food Concentrations

Background levels of dioxin-like compounds in various environmental media including food are presented in Table 1-4 in terms of means, variability and sample sizes used to support the background 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 these data were derived from studies that were not designed to estimate national background means. The environmental media concentrations were similar to studies in Western Europe. These data are the best available for comparing with site-specific values. Because of the limited number of locations examined, it is not known if 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 this document (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 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 8 states (CA, OH, GA, NY, PA, OR, MN, WS; 2 samples/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 are 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, 1992) 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 Food and Drug Administration during the mid to latter1990s (Jensen and Bolger, 2000; Jensen et al., 2000). An important aspect of the U.S. Food and Drug Administration (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 over 300 individual samples and good representation of the most highly consumed species. However, the levels of dioxins in fish remain more uncertain than 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 which measured the effects of cooking on the levels of CDDs, CDFs and PCBs in foods (see

Chapter 3, Section 3.7.5). 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 that 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.

Some evidence from Europe suggests that during the 1990s a decline has occurred in concentrations of dioxins and furans in food products, particularly dairy products (see Chapter 6, Section 6.5). For example, the United Kingdom's Ministry of Agriculture, Fisheries, and Food (MAFF) 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, while 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 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 twentieth century (Winters et al., 1998). It was found that meat samples of the 1950s through the 1970s had concentrations that were 2-3 times higher for the CDD/CDF TEQs and about 10 times higher for the PCB TEQs, as compared to current meat concentrations.

1.5.3. Background Exposures

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 which measured dioxin levels in adipose, blood, and human milk, all conducted in North America. The number of people in most of these studies, however,

is relatively small and the participants were not statistically selected in ways that assure their representativeness of the general U.S. adult population. One study, the 1987 National Human Adipose Tissue Survey (NHATS), involved over 800 individuals and provided broad geographic coverage, but 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 trends discussion in 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) summarized levels of CDDs, CDFs, and PCBs in human blood collected during the time period 1995 to 1997. The individuals sampled were all U.S. residents with no known exposures to dioxin other than normal background. The blood was collected from 316 individuals in six different locations with an age range of 20 to 70 years. While the samples in this data set were not collected in a manner that can be considered statistically representative of the national population and 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 the same proportion to the total PCB TEQ as in earlier data, they would increase our 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. A summary of the CDC (2000) data is shown in Table 1-5.

This finding regarding 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 our best estimate of current adult intake, i.e., 66 pg WHO_{98}-TEQ_{DFP}/d. Using this intake in a one-compartment, steady-state pharmacokinetic model yields a tissue level estimate of about 11.2 pg TEQ/g lipid (assumes TEQ_{DFP} has an effective half-life of 7.1 yr, 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 that was observed, than 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 in a person who turned 50 in 2000. Future studies should help address these uncertainties. The National Health and Nutrition Examination Survey (NHANES) began a new national survey in 1999 that will measure blood levels of CDDs, CDFs, and PCBs 126, 77, 169, and 81 in about 1,700 people per year (see http:www.cdc.gov/nchs/nhanes.htm). The survey is conducted at 15 different locations per year and is designed to select individuals statistically representative of the civilian U.S. population in terms of age, race, and ethnicity. These new data should provide a much better basis for estimating national background tissue levels and evaluating trends than the currently available data.

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, air) with contact rates (ingestion, inhalation). Table 1-6 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). Pharmacokinetic (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 yr, 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 that are thought to be higher than current levels (see 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 significant exposure pathways.

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 comes from several analyses of the Bogalusa Heart Study (Cresanta et al., 1988; Nicklas et al., 1993; Nicklas et al., 1995; Nicklas et al., 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 which 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. This is based on the observation that the 95th percentile blood level in the CDC (2000) study was almost twice the mean level.

Intakes of CDD/CDFs and dioxin-like PCBs are over three times higher for a young child as compared to that of an adult, on a body weight basis. This is based on combining age-specific food consumption rate and average food concentrations, as was done above for adult intake estimates (see Table 1-7).

Only four of the 17 toxic CDD/CDF congeners and one 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, 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 indicating that these congeners are also the primary contributors to dietary dose. These five compounds make up about 80% of the total WHO_{98}-TEQ tissue level.

1.5.4. Potentially Highly Exposed Populations or Developmental Stages

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 non-nursing infants and adults.

CDD/CDF contamination incidents involving the commercial food supply have occurred in the United States and other countries. For example, in the United States, contaminated ball clay was used as an anti-caking agent in soybean meal and resulted in elevated dioxin levels in some poultry and catfish. This incident, which occurred in 1998, involved less than 5% of the national poultry production and 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 such incidents have led to disproportionate exposures to populations living near where these incidents have 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 highly exposed local populations.

Elevated exposures associated with the workplace or 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 ppt TCDD. There is no clear evidence that elevated exposures are currently occurring among United States 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 non-nursing infants. Three German studies have compared dioxin levels in infants who have been breast-fed with those who have been formula-fed. All have shown elevations in the concentrations of dioxins in infants being breast-fed. Collectively these studies included 99 infants and found that blood levels (in units of pg TEQ_{DF}-WHO_{98}/g lipid - i.e., dioxin-like PCBs not included) in infants aged 4-12 months were generally more than 20 in nursing infants and less than 5 in formula fed infants. The most comprehensive of these studies was by Abraham et al. (2000) who reported on 80 breast-fed infants. In that study, the median concentration was 25.3 pg/g TEQ_{DF}-WHO_{98}. Six of the nursing infants in the Abraham et al. (2000) study had lipid levels greater than 50 pg/g TEQ_{DF}-WHO_{98} and the maximum was 107 pg/g TEQ_{DF}-WHO_{98}. Five of these six children were from a region where mother's milk was found to be elevated due to regional contamination by a copper

recycling plant. These data suggest that breast-fed infants could have body burdens more than five times higher than formula-fed infants, depending on length of breast-feeding, dioxin concentrations in mother's milk, and other factors.

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 dropped to 22 pg TEQ_{DFP}-WHO_{98}/kg/day after 12 months of nursing. The average intake over one year of nursing was calculated to be 92 pg TEQ_{DFP}-WHO_{98}/kg/day. The cumulative intake for a one year nursing scenario represented about 12% of the total lifetime cumulative intake (see Chapter 5, Section 5.2 for details on these calculations).

The CDC (1997) reported that in 1995, 55% of all babies experience 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 is ideal nutrition and sufficient to support optimal growth and development for 6 months after birth. They 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. Changes in TEQ tissue concentration over time were modeled for a variety of nursing scenarios: formula only, 6 weeks nursing, 6 months nursing, and one year. These scenarios reasonably capture the range of current nursing practice. This modeling effort required using the intake assumptions described earlier and a variety of additional assumptions including: the fraction of the oral dose which is absorbed into the body, changes in body weight over time, and changes in body fat fraction over time. Assumptions were also made about changes in the biological half-life of dioxins as a function of body fat fraction. For the infant, the half-life was less than one year, and during adulthood the half-life increased as the fraction of body fat increased. The short half-life at birth was based on a study by Kreuzer et al. (1997) and the longer half-life during the later years of life, when body fat fraction increased, was based on a model presented in Michalek et al. (1996). The complete set of input values are listed in Chapter 5, Section 5.2.

The modeling results in terms of changes in lipid concentrations and body burdens as a function of age are shown in Figure 1-2. Some key observations include:

- For the 6 and 12 month nursing scenarios, lipid concentrations peaked at around 4 months at about 46 ppt TEQ_{DFP}-WHO_{98}. The formula-fed infants 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.

A sensitivity analysis was performed to test the assumptions about changes in breast milk concentrations during lactation and changes in half-life over time. In this analysis, breast milk concentrations were held steady at 25 pg TEQ_{DFP}-WHO_{98}/g lipid for a 6-month nursing scenario, and the half-life of dioxins in the body remained steady at 7.1 years from birth until 70 years of age. With these two changes, the maximum infant lipid concentration increased from 46 to 70 pg TEQ_{DFP}-WHO_{98}/g lipid. The major impact of a steady half-life assumption, instead of one which increased with increasing body lipid fractions in the aging adult, was that the lipid concentrations stabilized at about 8 pg TEQ_{DFP}-WHO_{98}/g lipid in the adult, instead of rising to 13 pg TEQ_{DFP}-WHO_{98}/g lipid at age 70.

The above analysis indicates that the average annual infant intake resulting from one year of nursing, 92 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, i.e. infant body burdens will not exceed adult body burdens by 92 times. Rather, the modeling suggests that peak infant body burdens are only about 2 times current adult body burdens (46 vs 25 pg TEQ_{DFP}-WHO_{98}/g lipid). The reduced body burden impacts in nursing infants (relative to the intake) is thought to be due to the rapidly expanding infant body weight and lipid volume and the possibly faster elimination rate in infants. Impacts to 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. Most people eat some fish from multiple sources, both fresh and salt water. The estimated dioxin concentrations in these fish and the typical rates of consumption are included in the mean background calculation of exposure. People who consume large quantities of fish at estimated contamination levels may have elevated exposures. These kinds of exposures are addressed within the estimates of variability of background and are not considered to result in highly exposed populations. If individuals obtain their fish from areas where the concentration of dioxin-like chemicals in the fish is elevated, they may constitute a highly exposed subpopulation. Although this scenario seems reasonable, very

little supporting data could be found for such a highly exposed subpopulation in the United States. One study measuring 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. Another study measuring 90 PCB congeners (seven of which were dioxin-like PCBs, although PCB 126 was not measured) in the blood of sport fishers consuming high amounts of fish caught from Lake Michigan (>26 pounds of sport fish/yr) did, however, show significant elevations of PCBs in their blood as compared to a control population (individuals consuming < 6 pounds of sport fish/yr). The average total concentration of PCBs in the blood of these sport fishers was over 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 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 have also documented elevations in the levels of dioxin-like compounds in blood from the families consuming their home products.

1.5.5. Temporal Trends Information

Some general observations can be made about changes in levels of dioxin-like compounds in the environment over time. These are discussed below and summarized in Table 1-8.

Concentrations of CDD/CDFs and PCBs in the U.S. environment were consistently low prior to the 1930s. Then, concentrations rose steadily until about 1970. At that time, the trend reversed and the concentrations began to decline. That trend has continued to the present. The most compelling supportive evidence of this trend for the CDD/Fs and PCBs comes from dated sediment core studies. Sediment concentrations in these studies are generally assumed to be an indicator of the rate of atmospheric deposition. CDD/F and PCB concentrations in sediments began to increase around the 1930s, and continued to increase until about 1970. Decreases began in 1970 and have

continued to the time of the most recent sediment samples (about 1990). Sediment data from 20 U.S. lakes and rivers from seven separate research efforts consistently support this trend. Additionally, sediment studies in lakes located in several European countries have shown similar trends.

It is reasonable to assume that sediment core trends should be driven by a similar trend in emissions to the environment. The period of increase generally matches the time when a variety of industrial activities began rising and the period of decline appears to correspond with growth in pollution abatement. Many of these abatement efforts should have resulted in decreases in dioxin emissions, i.e. elimination of most open burning, particulate controls on combustors, phase out of leaded gas, and bans on PCBs, 2,4,5-T, hexachlorophene, and restrictions on use of pentachlorophenol. Also, the national source inventory of this assessment documented a significant decline in emissions from the late 1980s to the mid-1990s. Further evidence of a decline in CDD/F levels in recent years is emerging from data, primarily from Europe, showing declines in foods and human tissues.

In addition to the congener specific PCB data discussed earlier, a wealth of data on total PCBs and aroclor mixtures exist which also supports these trends. It is reasonable to assume that the trends for dioxin-like PCBs are similar to those for PCBs as a class because the predominant source of dioxin-like PCBs is the general production of PCBs in aroclor mixtures. PCBs were intentionally manufactured in large quantities from 1929 until production was banned in 1977. U.S. production peaked in 1970, with a volume of 39,000 metric tons. Further support is derived from data showing declining levels of total PCBs in Great Lakes sediments and biota during the 1970s and 1980s. These studies indicate, however, that during the 1990s the decline is slowing and may be leveling off.

Past human exposures to dioxins were most likely higher than current estimates. This is supported by a study which applied a non-steady state pharmacokinetic model to data on background U.S. tissue levels of 2,3,7,8-TCDD from the 1970s and 80s. Various possible intake histories (pg/kg-day over time) were tested to see which best-fit the data. An assumption of a constant dose over time resulted in a poor fit to the data. The "best-fit" (statistically derived) to the data was found when the dose, like the sediment core trends, rose through the 60s into the 70s, and declined to low current levels. Some additional support for this finding comes from a limited study of preserved meat samples from several decades in the twentieth century. One sample, from before 1910, showed very low concentrations of dioxins and coplanar PCBs. Thirteen other samples, from the 1940s until the early 1980s, consistently showed elevated levels of all dioxin-like compounds as compared to food surveys conducted during the 1990s.

REFERENCES

Abraham, K.; Papke, O.; Gross, a.; Kordonouri, O.; Wiegand, S.; Wahn, U.; Helge, H. (1998) Time course of PCDD/PCDF/PCB concentrations in breast-feeding mothers and their infants. Chemosphere. 37(9-12):1731-1741.

Ahlborg, V.G.; Becking, G.C.; Birnbaum, L.S.; Brower, A.; Derks, H.J.G.M.; Feeley, M.; Golor, C.; Hanberg, A.; Larsen, J.C.; Liem, A.K.D.; Safe, S.H.; Schlatter, C.; Waern, F.; Younes, M.; Yrankeikki, E. (1994) Toxic equivalency factors for dioxin-like PCBs. Chemosphere. 28(6):1049-1067.

American Academy of Pediatrics. (1997) Breastfeeding and the use of human milk. Pediatrics 100 (6):1035-1039.

CDC. (1997) Vital and Health Statistics. Fertility, Family Planning, and Women's Health: New Data From the 1995 National Survey of Family Growth. National Center for Health Statistics, Centers for Disease Control and Prevention, U.S. Department of Health and Human Services. Series 23, No. 19. May 1997.

CDC. (2000) Personal communication from D. Patterson, CDC, Atlanta, GA to M. Lorber, U.S. EPA, Washington, DC. April, 2000.

Cleverly, D.; Monetti, M.; Phillips, L.; Cramer, P.; Heit, M.; McCarthy, S.; O'Rourke, K.; Stanley, J.; Winters, D. (1996) A time-trends study of the occurrences and levels of CDDs, CDFs and dioxin-like PCBs in sediment cores from 11 geographically distributed lakes in the United States. Organohalogen Compounds. 28:77-82.

Cresanta, JL; Farris, RP; Croft, JB; Frank, GC; Berenson, GS. (1988) Trends in fatty acid intakes of 10-year-old children, 1973-1982. Journal of American Dietetic Association 88:178-184.

Czuczwa, J.M.; McVeety, B.D.; Hites, R.A. (1985) Polychlorinated dibenzo-p-dioxins and dibenzofurans in sediments from Siskiwit Lake, Isle Royale. Chemosphere. 14:623-626.

DeWit, C.A.; Jamberg, U.G.; Asplund, L.T.; Jansson, B.; Olsson, M.; Odsjo, I.L.; Lindstedt, L.L.; Anderrson, O.; Bergek, S.; Hjelt, M.; Rappe, C.; Jansson, A.; Nygren, M. (1994) The Swedish dioxin survey: summary of results from PCDD/F and coplanar PCB analyses in biota. Organohalogen Compounds. 20:47-50.

Frank, G.C.; Webber, L.S.; Farris, R.P.; Berenson, G.S. (1986) Dietary databook: quantifying dietary intakes of infants, children, and adolescents, the Bogalusa heart study, 1973-1983. National Research and Demonstration Center - Arteriosclerosis, Louisiana State University Medical Center, New Orleans, Louisiana.

Fürst, P.; Wilmers, K. (1995) PCDD/F levels in dairy products 1994 versus 1990. Organohalogen Compounds. 26:101-104.

Fürst, P.; Wilmers, K. (1997) Decline of human PCCDD/F intake via food between 1989 and 1996. Organohalogen Compounds. 33:116-121.

Harner, T.; Mackay, D.; Jones, K. (1995) Model of long-term exchange of PCBs between soil and the atmosphere in the southern UK. Environmental Science and Technology. 29:1200-1209.

Hebert, C.E.; Norstrom, R.J.; Simon, M.; Braune, B.M.; Weseloh, D.V.; MacDonald, C.R. (1994) Temporal trends and sources of PCDDs and PCDFs in the Great Lakes: herring gull egg monitoring, 1981-1991. Environmental Science and Technology. 28:1268-1277.

Hiester, E.; Brickman, P.; Böhm, R.; Eynck, P.; Gerlack, A.; Mülder, W.; Ristow, H. (1995) Pronounced decrease of PCDD/PCDF burden in ambient air. Organohalogen Compounds. 24:147-152.

Jensen, E.; Bolger, P.M. (2000) Exposure assessment of dioxins/furans consumed in dairy foods and fish. Submitted for publication in Food Additives and Contaminants.

Jensen, E.; Canady, R.; Bolger, P.M. (2000) Exposure assessment for dioxins and furans in seafood and dairy products in the United States, 1998-99. Organohalogen Compounds. 47:318-321.

Kjeller, L.O.; Jones, K.C.; Johnston, A.E.; Rappe, C. (1991) Increases in the polychlorinated dibenzo-p-dioxin and -furan content of soils and vegetation since the 1840s. Environmental Science and Technology. 25:1619-1627.

Kjeller, L.O.; Jones, K.C.; Johnston, A.E.; Rappe, C. (1996) Evidence for a decline in atmospheric emissions of PCDD/CDFs in the U.K. Environmental Science and Technology. 30:1398-1403.

Korhonen, M.; Mannio, J.; Vartiainen, T. (1995) The levels of PCBs in aquatic biota in Finland. Organohalogen Compounds. 24:453-456.

Kreuzer, PE; Csanady, GyA; Baur, C; Kessler, W; et al. (1997) 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and congeners in infants. A toxicokinetic model of human lifetime body burden by TCDD with special emphasis on its uptake and nutrition. Arch Toxicol 71:383-400.

Kutz, FW; Barnes, DG; Bretthauer, EW; et al. (1990) The International Toxicity Equivalency Factor (I-TEF) method for estimating risks associated with exposures to complex mixtures of dioxins and related compounds. Toxicological and Environmental Chemistry 26:99-109.

Liem, AKD; Atuma, S; Becker, W; Darnerud, PO; et al. (2000) Dietary intake of dioxin and dioxin-like PCBs by the general population of ten European countries. Results of EU-SCOOP Task 3.2.5. (Dioxins). Organohalogen Compounds 48:13-16.

MacDonald, R.W.; Cretney, W.J.; Crewe, N.; Paton, D. (1992) A history of octachlorodibenzo-p-dioxin, 2,3,7,8-tetrachlorodibenzofuran, and 3,3'3,3'-tetrachlorobiphenyl contamination in Howe Sound, British Columbia. Environmental Science and Technology. 26:1544-1550.

Ministry of Agriculture, Fisheries, and Food (MAFF). (1995) Dioxins in food - UK dietary intakes. MAFF Food Surveillance Information Sheet No. 71. United Kingdom, Food Safety Directorate.

Michalek, J; Pirkle, J; Caudill, S; Tripathi, R; et al. (1996) Pharmacokinetics of TCDD in veterans of operation Ranch hand: 10 year follow-up. J of Tox and Env Epi 47: 209-220.

Nicklas, T.A.; Webber, L.S.; Srinivasan, S.R.; Berenson, G.S. (1993) Secular trends in dietary intakes and cardiovascular risk factors in 10-y-old children: the Bogalusa heart study (1973-1988). Amer J Clin Nut 57:930-937.

Nicklas, T.A.; Johnson, C.C.; Meyers, L.; Webber, L.S.; et al. (1995) Eating patterns, nutrient intakes, and alcohol consumption patterns of young adults: the Bogalusa heart study. Med Exercise Nut Health 4:316-324.

Pearson, R.F.; Swackhamer, D.L.; Eisenreich, S.J. (1995) Concentrations , inventories, and accumulations of polychlorinated dibenzo-p-dioxins and dibenzofurans in the sediments of the Great Lakes. Organohalogen Compounds. 24:267-271.

Safe, S. (1995) Human dietary intake of aryl hydrocarbon (Ah) receptor agonists: mass balance estimates of exodioxins and endodioxins and implications for health assessment. Organohalogen Compounds 26:7-13.

Smith, R.M.; O'Keefe, P.; Aldous, K.; Briggs, R.; Hilker, D.; Connor, S. (1992) Measurement of PCDFs and PCDDs in air samples and lake sediments at several locations in upstate New York. Chemosphere. 25:95-98.

Smith, R.M.; O'Keefe, P.W.; Hilker, D.R.; Bush, B.; Connor, S.; Donnelly, R.; Storm, R.; Liddle, M. (1993) The historical record of PCDDs, PCDFs, PAHs, PCBs, and lead in Green Lake, New York - 1860 to 1990. Organohalogen Compounds. 20:215-218.

Smith, R.M.; O'Keefe, P.W.; Hilker, D.; Connor, S.; Posner, E. (1995) Direct and indirect contributions of atmospheric PCDDs and PCDFs to Hudson River national estuarine research reserve sediment cores. Organohalogen Compounds. 24:141-145.

Tan, Y.L.; Kong, A.; Monetti, M.A.; Lau, B.P.Y.; Gubala, C.; Landers, D. (1993) Polycyclic aromatic hydrocarbons and polychlorinated dibenzo-p-dioxins and dibenzofurans in sediment from a sub-Arctic lake in Alaska. Organohalogen Compounds. 309-312.

U.S. Environmental Protection Agency. (1987) Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs). EPA/625/3-87/012.

U.S. Environmental Protection Agency. (1988) Estimating exposure to 2,3,7,8-TCDD. Washington, DC: Office of Health and Environmental Assessment. EPA/600/6-88/005A.

U.S. Environmental Protection Agency. (1989a) Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs) and 1989 update. Washington, DC. Risk Assessment Forum. EPA/625/3-89/016.

U.S. EPA. (1989b) Review of draft documents: a cancer risk-specific dose estimate for 2,3,7,8-TCDD. Washington, DC. EPA Science Advisory Board Ad Hoc Dioxin Panel.

U.S. Environmental Protection Agency. (1991) Workshop report on toxicity equivalency factors for polychlorinated biphenyls congeners. EPA/625/3-91/020.

U.S. Environmental Protection Agency (1992) National study of chemical residues in fish. Washington, DC: Office of Science and Technology. EPA/823-R-02-008.

U.S. Environmental Protection Agency. (1993) Interim report on data and methods for assessment of 2,3,7,8-tTetrachlorodibenzo-p-dioxin risks to aquatic life and associated wildlife. Duluth, MN: Environmental Research Laboratory, Office of Research and Development. EPA/600/R-93/055. March 1993.

U.S. Environmental Protection Agency. (1994) Health assessment for 2,3,7,8-TCDD and related compounds. Public Review Draft. EPA/600/EP-92/001.

U.S. Environmental Protection Agency. (1995) An SAB report: A second look at dioxin. Review of the Office of Research and Development's Reassessment of Dioxin and Dioxin-Like Compounds by the Dioxin Reassessment Review Committee. Science Advisory Board. EPA-SAB-EC-95-021. September 1995.

U. S. Environmental Protection Agency (1997). Chapter 8. Dose-response modeling for 2,3,7,8-TCDD. January 1997 Workshop Review Draft. EPA/600/P-92/001C8.

U.S. Environmental Protection Agency (1998). Inventory of sources of dioxin in the United States. National Center for Environmental Assessment, Office of Research and Development. Workshop Review Draft. EPA/600/P-98/002Aa. April 1998.

U.S. Environmental Protection Agency (2000a). Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 1: Estimating exposure to dioxin-like compounds. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. EPA/600/P-00/001B(b-d).

U.S. Environmental Protection Agency (2000b). Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 2: Health assessment for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. National Center for Environmental Assessment, Office of Research and Development. EPA/600/P-00/001Be.

U.S. Environmental Protection Agency (2000c). Exposure and human health reassessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Part 3: Dioxin: Integrated summary and risk characterization for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. EPA/600/P-00/001Bf.

Van den Berg, M, L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R. van Leeuwen, A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Warn, T. Zacharewski (1998) Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environmental Health Perspectives 106: 775-792.

Vartiainen, T.; Mannio, J.; Korhonen, M.; Kinnunen, K.; Strandman, T. (1995) Levels of PCDDs and PCDFs in dated sediment lake sediments in subarctic Finland. Organohalogen Compounds. 24:369-372.

Winters, D.L.; Anderson, S.; Lorber, M.; et al. (1998) Trends in dioxin and PCB concentrations in meat samples from several decades of the 20th century. Organohalogen Compounds, Volume 38:75-78.

Table 1-1. The TEF Scheme for I-TEQ_{DF}

Dioxin (D) Congener	TEF	Furan (F) Congener	TEF
2,3,7,8-TCDD	1.0	2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDD	0.5	1,2,3,7,8-PeCDF	0.05
1,2,3,4,7,8-HxCDD	0.1	2,3,4,7,8-PeCDF	0.5
1,2,3,6,7,8-HxCDD	0.1	1,2,3,4,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDD	0.1	1,2,3,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDD	0.01	1,2,3,7,8,9-HxCDF	0.1
OCDD	0.001	2,3,4,6,7,8-HxCDF	0.1
		1,2,3,4,6,7,8-HpCDF	0.01
		1,2,3,4,7,8,9-HpCDF	0.01
		OCDF	0.001

Table 1-2. The TEF Scheme for dioxin-like coplanar PCBs, as determined by the World Health Organization in 1994

Chemical Structure	IUPAC Number	TEF
3,3',4,4'-TeCB	PCB-77	0.0005
2,3,3',4,4'-PeCB	PCB-105	0.0001
2,3,4,4',5-PeCB	PCB-114	0.0005
2,3',4,4',5-PeCB	PCB-118	0.0001
2',3,4,4',5-PeCB	PCB-123	0.0001
3,3',4,4',5-PeCB	PCB-126	0.1
2,3,3',4,4',5-HxCB	PCB-156	0.0005
2,3,3',4,4',5'-HxCB	PCB-157	0.0005
2,3',4,4',5,5'-HxCB	PCB-167	0.00001
3,3',4,4',5,5'-HxCB	PCB-169	0.01
2,2',3,3',4,4',5-HpCB	PCB-170	0.0001
2,2',3,4,4',5,5'-HpCB	PCB-180	0.00001
2,3,3',4,4',5,5'-HpCB	PCB-189	0.0001

Table 1-3. The TEF Scheme for TEQ_{DFP}-WHO_{98}

Dioxin Congeners	TEF	Furan Congeners	TEF
2,3,7,8-TCDD	1.0	2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDD	1.0	1,2,3,7,8-PeCDF	0.05
1,2,3,4,7,8-HxCDD	0.1	2,3,4,7,8-PeCDF	0.5
1,2,3,6,7,8-HxCDD	0.1	1,2,3,4,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDD	0.1	1,2,3,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDD	0.01	1,2,3,7,8,9-HxCDF	0.1
OCDD	0.0001	2,3,4,6,7,8-HxCDF	0.1
		1,2,3,4,6,7,8-HpCDF	0.01
		1,2,3,4,7,8,9-HpCDF	0.01
		OCDF	0.0001

Chemical Structure	IUPAC Number	TEF
3,3',4,4'-TeCB	PCB-77	0.0001
3,4,4',5-TCB	PCB-81	0.0001
2,3,3',4,4'-PeCB	PCB-105	0.0001
2,3,4,4',5-PeCB	PCB-114	0.0005
2,3',4,4',5-PeCB	PCB-118	0.0001
2',3,4,4',5-PeCB	PCB-123	0.0001
3,3',4,4',5-PeCB	PCB-126	0.1
2,3,3',4,4',5-HxCB	PCB-156	0.0005
2,3,3',4,4',5'-HxCB	PCB-157	0.0005
2,3',4,4',5,5'-HxCB	PCB-167	0.00001
3,3',4,4',5,5'-HxCB	PCB-169	0.01
2,3,3',4,4',5,5'-HpCB	PCB-189	0.0001

Table 1-4. Summary of North American CDD/CDF and PCB TEQ-WHO_{98} Levels in Environmental Media and Food (whole weight basis; concentrations provided in parenthesis for food products are calculated at ND = 0).

Media	CDD/CDFs[a]	PCBs[a]
Urban Soil, ppt	n=270 9.3 ± 10.2[d] Range = 2 - 21	n=99 2.3[d]
Rural Soil, ppt	n = 354 2.7[d] Range = 0.1 - 6	n=62 0.59[d]
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	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	n=289 1.0 (NA[b])	n = 1 composite of 10 samples plus 6 composites 1.2[c] (NA[b])
Marine Fish and Shellfish, ppt	n=158 0.26 (NA[b])	n = 1 composite of 13 samples plus 5 composites 0.25[e] (NA[b])
Water, ppq	n=236 0.00056 ± 0.00079 (NA[b])	Nab
Milk, ppt (Note: each composite for CDD/F/PCB comprised of 40+ U.S. regional samples)	n=8 composites 0.018 ± 0.0012 (0.017)	n = 8 composites 0.0088 (0.0088)
Dairy, ppt[e]	n = 8 composites 0.12 ± 0.22 (0.12)	n = 8 composites 0.058 (0.058)
Eggs, ppt (Note: each composite for CDD/F data comprised of 24 eggs)	n=15 composites 0.081[c] (0.013)	n = 18 plus 6 composites 0.10[c] (NA[b])
Beef ppt	n=63 0.18 ± 0.11 (0.061) Range = 0.11 - 0.95	n = 63 0.084 (0.084)
Pork, ppt	n=78 0.28 ± 0.28 (0.080) Range = 0.15 - 1.8	n = 78 0.0093 (0.006)
Poultry, ppt	n=78 0.068 ± 0.070 (0.043) Range = 0.03 - 0.43	n = 78 0.026 (0.026)
Vegetable Fats, ppt	n=30 0.056 ± 0.24[d] (NA[b])	n = 5 composites 0.037[c]

[a] Values are the arithmetic mean TEQs, in ppt, 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.

[b] NA = not available; Congener-specific PCB data, and data to calculate TEQ concentrations at ND = 0, are limited.

[c] Standard deviations could not be calculated due to limitations associated with the data (i.e., composite analyses).

[d] TEQ calculated by setting nondetects to zero.

[e] Dairy concentration calculated from milk lipid concentrations and then assuming a fat fraction for dairy.

Table 1-5. Background Serum Levels in the United States 1995 - 1997

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

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

Source: CDC (2000).

Table 1-6. Adult Contact Rates and Background Intakes of Dioxin-like Compounds

Exposure Route	Contact Rate	Dioxins and Furans		Dioxin-like PCBS		Total intake (pg TEQ_{DFP}-WHO_{98}/kg-d)
		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)	
Soil ingestion	50 mg/d	9.3 pg/g	0.0066	2.3	0.0016	0.0082
Soil dermal	12 g/d	9.3 pg/g	0.0016	2.3	0.00034	0.0019
Freshwater fish and shellfish	5.9 g/d	1.0 pg/g	0.084	1.2 pg/g	0.1	0.18
Marine fish and shellfish	9.6 g/d	0.26 pg/g	0.036	0.25 pg/g	0.045	0.070
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	0.71 g/kg-d	0.18 pg/g	0.13	0.084 pg/g	0.060	0.19
Pork	0.22 g/kg-d	0.28 pg/g	0.062	0.012 pg/g	0.0026	0.065
Poultry	0.50 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 pg/g	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	0.0090	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)**

Table 1-7. Variability in Average Daily 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 yr	50	3.3
6-11 yr	54	1.9
12-19 yr	61	1.1
Adult	66	0.94

Table 1-8. Summary of Findings with Regard to Trends in Dioxin Levels in the Environment and in Humans

Finding	Support	Uncertainty
Concentrations of CDD/CDFs in the environment were consistently low for centuries until the 1930s. Then, concentrations rose steadily until about the 1960s, at which point concentrations began to drop. Evidence suggests that the drop in concentrations is continuing to the present.	***Sediment core studies show a trend of rising concentrations in 1930s and 1940s through the 1960s and 1970s and a subsequent decline to the present.*** - 11 lakes/reservoirs in the U.S.: Cleverly et al., 1996; Versar, 1996 - Lake Huron: Czuczwa et al., 1985 - Green Lake, NY: Smith et al., 1992, 1993 - Hudson River: Smith et al., 1995 - Lakes Superior, Michigan, and Ontario: Pearson et al., 1995 - Straight of Georgia, British Columbia: MacDonald et al., 1992 - A remote arctic lake: Tan et al., 1993; Vartiainen et al., 1995	The assumption of nondegradation of CDD/CDFs in sediment cores.
	Analogous trends in environmental loadings - Rise of the manufacture and use of chlorinated phenolic intermediates and products - Banning of leaded gasoline, certain phenoxy herbicides, PCBs - Reductions in pulp and paper mill discharges - EPA National Source Inventory showing 60% reduction in CDD/CDF TEQ emissions between 1987 and 1995 (see Volume 1)	Indirect measure of environmental levels.
	Limited trend for other environmental concentrations - Rises and declines in historical food products in the U.S.: Winters et al., 1998 - Rises and declines in herbage, soil, and air measured in archived samples in UK: Kjeller et al., 1991, 1996; Harner et al., 1995 - Reductions in the past two decades in herring gull eggs in the Great Lakes and the Gulf of St. Lawrence River (Hebert et al., 1994); pike in Sweden (DeWit et al., 1994); pike in Finland (Korhonen et al., 1995); air in Germany (Hiester et al., 1995); German dairy products and human milk between 1990 and 1994 (Fürst and Wilmers, 1995; 1997)	Very few archived environmental measurements to ascertain trends beyond the past decade or so. More recent data showing a decline in trends is limited.
	Suggestive evidence of declines in human body burdens in recent decade - National Human Adipose Tissue Survey: U.S. EPA, 1991 - Ministry of Agriculture, Fisheries, and Food's calculations of declines in dose based on market basket surveys showing reductions in levels in combination with reductions in consumption of key food items: MAFF (1995)	Long half-lives in humans impede responses in body burdens to changes in environmental levels.
Environmental levels of coplanar PCBs began increasing in the 1920s, peaking in the 1970s, and decreasing after that	***PCB production data*** - Rise in the manufacture from the latter 1920s to the early 1970s; complete ban in 1977	Nearly all trends data are specific to PCBs or Aroclors, and not to coplanar PCBs.
	PCB declining environmental levels - Evidence of declines in biota and sediment levels in the Great Lakes	

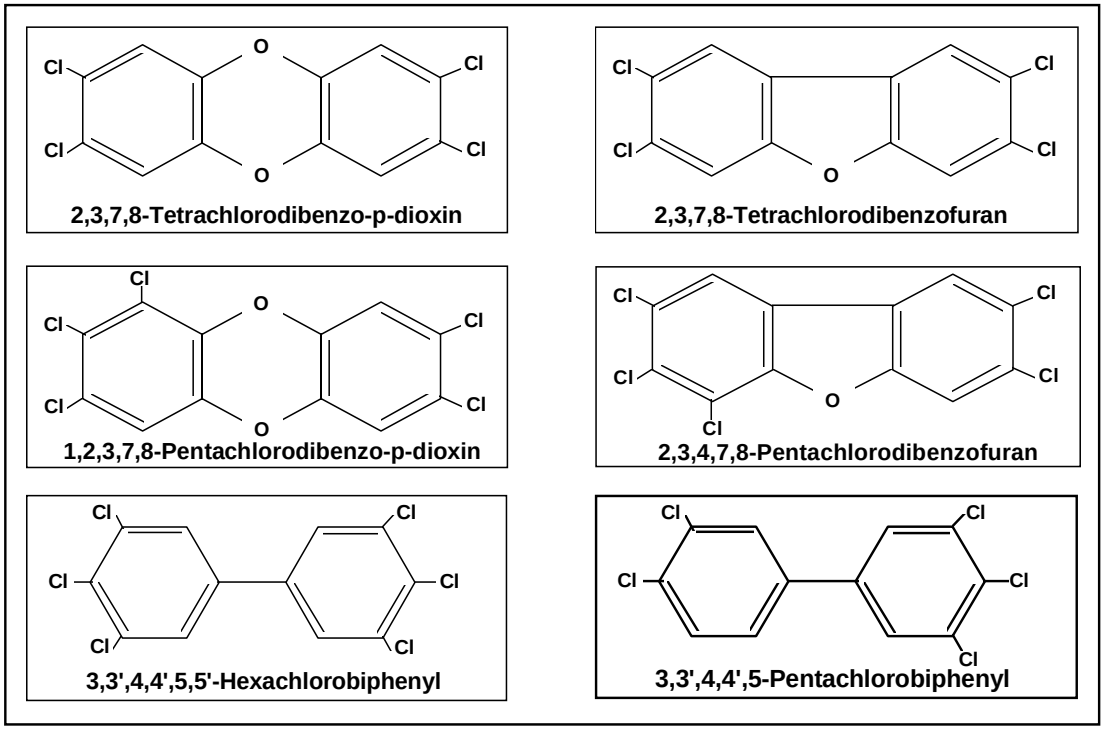
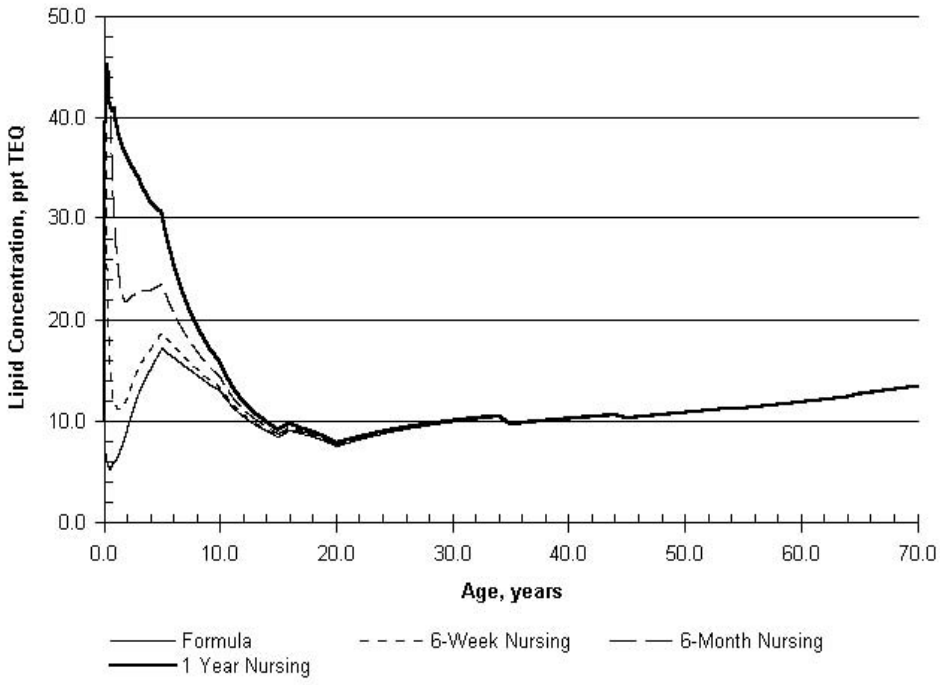


Figure 1-1. Chemical Structure of 2,3,7,8-TCDD and Related Compounds

(a)



(b)

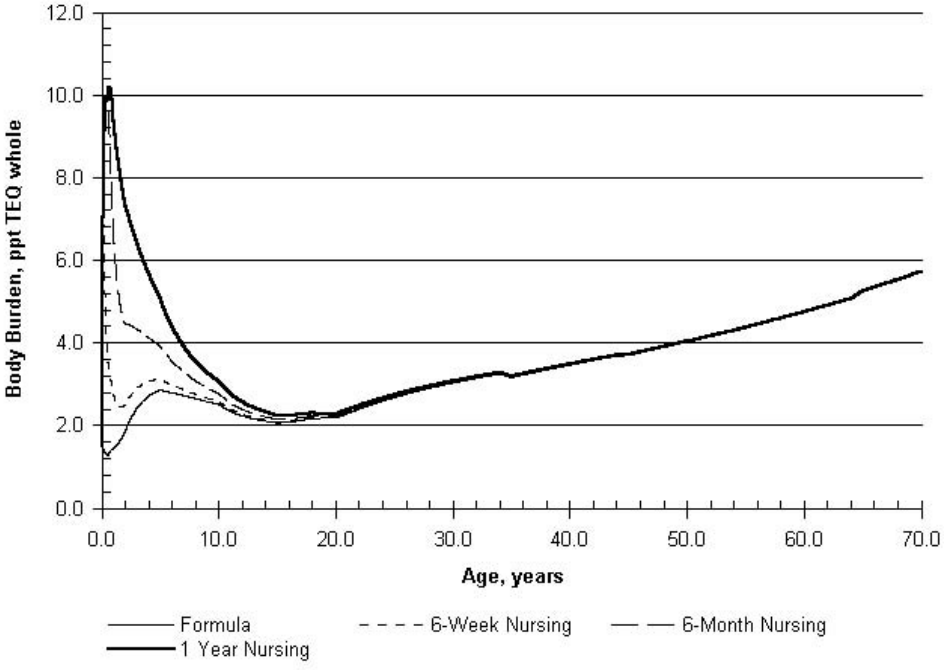


Figure 1-2. Lipid (a) and Body Burden (b) Concentrations in a Hypothethical Female Until Age 70 Under Four Nursing Scenarios: Formula Only, and 6-week, 6-month, and 1 year Nursing