4. HUMAN EXPOSURES TO CDD, CDF, AND PCB CONGENERS

4.1. INTRODUCTION

The purpose of this chapter is to assess background exposures to the dioxin-like compounds. Recent assessments of background exposures cited in the scientific literature are summarized, and background exposure estimates based on the data presented in this report are presented. Two methods have been used in this chapter to estimate background daily intake of dioxin-like compounds. One method estimates background exposures based on pharmacokinetic modeling using body burden data. The other derives background exposure estimates from dietary intake and contact with other media containing dioxin-like compounds. These two approaches provide comparable estimates of daily TEQ-WHO_{98} intake of dioxin-like compounds.

The primary focus of this chapter is background exposure among the general population. The general population consist of people who are exposed to background levels of dioxin-like compounds in soil and air. Most of their exposure comes from the commercial food supply and they do not have significant occupational exposure. People outside the general population are those living in areas with elevated soil or air levels, or whose dietary exposure is strongly influenced by food outside the commercial food supply (i.e., nursing infants, sports or subsistence fishermen, etc.).

The term "background," as applied to exposure, can be used to represent different concepts. Two common definitions are (1) the level of exposure that would occur in an area without known point sources of the contaminant of concern or (2) the average level of exposure occurring in an area whether sources are present or not. For the purposes of this document, "background" is defined as suggested in the first definition above. To the extent possible, background exposures estimated in this chapter are based on monitoring data obtained from sites removed from known contaminant sources (i.e., food data representative of the general food supply) and body burden data from nonoccupationally exposed members of the general population. Most of the data are based on studies published in the late 1980s and 1990s, but primarily the 1990s. These data are considered to be the most useful for describing background exposure levels.

Chapter 5 also includes information on potentially elevated exposures. It describes the potential for elevated exposures among subpopulations such as nursing infants, sport

and subsistence fishermen, cigarette smokers, and individuals living in areas that may be affected by localized sources of dioxin-like compounds.

4.2. LEVELS OF DIOXIN-LIKE COMPOUNDS IN HUMAN TISSUE

4.2.1. Adipose Tissue and Blood Studies from the 1980s and Early 1990s

The most extensive U.S. study of CDD/CDF body burdens is the National Human Adipose Tissue Survey (NHATS) (U.S. EPA, 1991a). NHATS was designed to estimate national population average levels of CDD/CDFs. The survey analyzed for CDD/CDFs in 48 human tissue samples that were composited from 865 samples. Each composite contained an average of 18 specimens. These samples were collected during 1987 from autopsied cadavers and surgical patients. The sample compositing prevents use of these data to examine the distribution of CDD/CDF levels in tissue among individuals. Also, not all 48 composites were used for all congeners in the statistical analysis of the data because some components did not meet the data quality objectives of the study. However, the study results allowed conclusions to be made in the following areas:

- National Averages - The national population averages for all TEQ congeners were estimated as listed in Table 4-1. Nondetects were treated as half the detection limit for averaging purposes. As shown in this table, all congeners except some CDFs, had a very low frequency of nondetects. Thus, the overall TEQ estimate is not sensitive to how nondetects were treated in the averaging.

- Age Effects - Tissue concentrations of CDD/CDFs were found to increase with age (Orban et al., 1994) (Table 4-2).

- Geographic Effects - In general, the average CDD/CDF tissue concentrations appeared fairly uniform geographically. Only one TEQ congener was found to have a significant difference among geographic regions of the country. This compound, 2,3,4,7,8-PeCDF, was found at the lowest level in the West (4.49 pg/g) and the highest in the Northeast (13.7 pg/g).

- Race Effects - No significant difference in CDD/CDF tissue concentrations was found on the basis of race (Table 4-2).

- Sex Effects - No significant difference in CDD/CDF tissue concentrations was found between males and females (Table 4-2).

- Temporal Trends - The 1987 survey showed decreases in tissue concentrations relative to the 1982 survey for all congeners. However, it is not known whether these declines were due to improvements in the analytical methods or actual reductions in body burden levels. The percent reductions among individual congeners varied from 9 percent to 96 percent.

Patterson et al. (1994) provided additional information on levels of dioxin-like compounds in human tissue. Human adipose from 28 individuals was collected. The individuals studied were ones who died suddenly in the Atlanta area during 1984 or 1986. Their ages ranged from 19 to 78 years and averaged 49 years. 2,3,7,8-TCDD levels varied with the upper end of the range equaling between three and four times the mean concentration. The tissue data are summarized in Table 4-3. This table shows that the mean PCB levels generally exceeded the mean 2,3,7,8-TCDD level and PCB-126 exceeded the 2,3,7,8-TCDD level by over an order of magnitude. The mean TEQ levels for these dioxin-like PCBs summed to about 14 ppt on a lipid basis (using either TEF_P-WHO_{94}s or TEF_P-WHO_{98}s). A complete CDD/CDF congener analysis was conducted on tissues of four of the individuals, resulting in an average of 26 ppt I-TEQ_{DF} (31 ppt TEQ_{DF}-WHO_{98}) on a lipid basis. These tissue samples were also analyzed for PCBs 77, 126, and 169. The lipid-based TEQ_P-WHO_{94} levels for these dioxin-like PCBs summed to 5.4 ppt. Thus, PCBs 77, 126, and 169 contributed between 15 and 20 percent of the total CDD/CDF and PCB TEQs. Patterson et al. (1994) also studied serum collected by the CDC blood bank in Atlanta during 1982, 1988, and 1989. These samples were pooled from over 200 donors. The average levels for 2,3,7,8-TCDD and PCBs are summarized in Table 4-4 in units of ppt on a whole weight basis. The serum data appear to indicate a decrease in exposure to PCBs from 1982 to 1988/1989. The lipid-based TEQ_P-WHO_{94} for the 1988 sample was 14 ppt based on PCBs 77, 126, 160, 105, 118, and 180. In general, the Patterson et al. (1994) data suggest that the dioxin-like PCBs can contribute significantly to body burdens of dioxin-like compounds. The data suggest that the dioxin-like PCBs can increase the total background body burden to over 40 ppt of total TEQ_{DFP}-WHO_{94}. This conclusion is uncertain because the people studied by Patterson et al. (1994) may not be representative of the overall U.S. population.

Schecter et al. (1993) reported on the comparisons of congener-specific measurements of CDDs, CDFs, and dioxin-like PCBs (77, 105, 118, 126, 156, 169, 170, and 180) in whole blood samples of four individuals with known exposures to that of the

general population. In this comparison, the analytical results of separate 450 mL blood samples collected from 50 Michigan residents, and a pooled blood sample from 5 donors at a blood bank in Missouri were used as the control group. Two of the exposed individuals were pulp and paper plant workers with potential exposure to dioxins, and the other two were Michigan residents who had elevated blood PCB levels from consuming contaminated fish. It was found that the control group and the pulp and paper mill workers who had no known exposures to PCBs had relatively high levels of coplanar, mono-ortho, and di-ortho PCBs in their whole blood. On average, the Michigan and Missouri control samples showed mean I-TEQ_{DF} concentrations of 27 ppt and 24 ppt (TEQ_{DF}-WHO_{98}s were 31 ppt and 26 ppt), respectively. These same samples showed TEQ_{P}-WHO_{94} mean concentrations of 17 ppt for the Michigan controls, and 10 ppt for Missouri controls.

Cole et al. (1995) reported on CDD/CDFs and PCBs in 132 serum samples (pooled to 14) from Ontario Great Lakes anglers and control populations. Based on a preliminary survey, anglers from the communities of Cornwall and Mississauga, Canada, were categorized based on the numbers, species, and locations of fish caught and kept for consumption, and on data reflecting the contaminant levels for the fish in these areas. Individuals categorized as having the highest and lowest potential for having elevated body burdens of CDD/CDFs and PCBs were selected for biological sampling. Individuals who did not consume fish served as controls. Study participants were further categorized by age (i.e., $<$38 years, 38-50 years, and $>$50 years). The results indicated that mean CDD/CDF TEQ levels were similar for both eaters and noneaters of Great Lakes' fish in these communities. I-TEQ_{DF}s ranged from 20.8 to 41.2 ppt for fish eaters and 24.7 to 36.8 ppt for noneaters. In general, mean I-TEQ_{DF}s increased with age (Table 4-5). PCBs 77, 126, and 169 were also evaluated in the serum samples collected from Cornwall residents. TEQ_{P}-WHO_{94}s ranged from 2.6 to 17.3 ppt for fish eaters and noneaters combined. Because no statistical differences were observed between fish eaters and noneaters, the data from this study were assumed to represent background exposures and were included in the background tissue level calculations in this chapter.

Schecter et al. (1989a) provided data on PCB levels in adipose samples from three patients from North America with no known chemical exposure history. The mean TEQ_{P}-WHO_{94} level based on PCBs 118, 105, 156, and 180 was 12.2 ppt on a lipid basis (the

TEQ_P-WHO_{98}, recalculated using TEF_P-WHO_{98}s, was 11.5 ppt on a lipid basis). Williams and LeBel (1991) reported on the mean residue levels of PCBs 126 and 169 in 62 adipose tissue samples collected in Canada during 1984. The mean lipid-based TEQ_P for these samples was estimated to be 28 ppt based on TEF_P-WHO_{94} or TEF_P-WHO_{98}s for PCBs.

Kang et al. (1997) reported on the levels of PCBs 77, 126, and 169 in human serum collected from white male paper mill workers (n = 46), as well as residents (n = 16) of a northeastern U.S. community. PCB 77 was not detected in any samples, but PCBs 126 and 169 were detected in most samples. The mean lipid-based concentrations of the two congeners (i.e., PCB 126 and 169) were 25 ppt and 31 ppt, respectively, for paper mill workers, and 18 ppt and 27 ppt, respectively, for community residents. Using TEF_P-WHO_{94}s for these PCBs (PCB 126 - 0.1, PCB 169 - 0.01), the relative contribution of these PCBs to the total CDD/CDF/PCB TEQ (using I-TEF_{DF}s for CDD/CDFs) for all study participants was approximately 10 percent. Kang et al. (1997) also observed that age, body mass index, and consumption of locally caught fish were significant predictors of coplanar PCB concentrations in human serum.

The levels of dioxin-like compounds found in human tissue/blood appear similar in Europe and North America. Schecter (1991) compared levels of dioxin-like compounds found in blood among people from U.S. (pooled samples from 100 subjects) and Germany (85 subjects). Although mean levels of individual congeners differed by as much as a factor of two between the two populations, the total I-TEQ_{DF} averaged 42 ppt in the German subjects and 41 ppt in the pooled U.S. samples. Using TEF_{DF}-WHO_{98}s, these TEQ_{DF}-WHO_{98} concentrations would be 49 ppt and 50 ppt, respectively. In later papers, Schecter et al. (1992a; 1994a) reported human blood levels for the general population from various countries. These data are presented in Table 4-6. Schecter (1991) reported adipose tissue levels in various countries, as summarized in Table 4-7. The adipose tissue data show more variation between countries, but also involved much fewer samples, reducing confidence in the accuracy of the mean.

Gonzalez et al. (1993) reported that the levels and patterns of CDD/CDFs in the adipose tissue obtained from the general population of Madrid, Spain, were similar to those of other industrialized countries. A total of 17 adipose tissue samples were collected from male and female patients ranging in age from 48 to 89 years. The lipid-based mean I-TEQ_{DF} was 42 ppt (46 ppt using TEF_{DF}-WHO_{98}s) and the mean level of

2,3,7,8-TCDD was 3.28 ppt. CDDs were found to be higher than CDFs in these samples with the higher-chlorinated CDDs accounting for the highest portion of the total CDD/CDFs (Table 4-8). The mean lipid-based I-TEQ_{DF} concentration in the blood of 11 individuals from Madrid, Spain, was 15.7 ppt (Jimenez et al., 1995). The higher-chlorinated CDDs (i.e., HpCDD and OCDD) were the dominant congeners observed in these samples.

Schumacher et al. (1999a and 1999b) conducted two studies to analyze background concentrations of CDD/CDFs in blood and adipose tissue from individuals from Tarragona, Spain. In the first study (Schumacher et al., 1999a), blood plasma samples were collected from 20 nonoccupationally exposed subjects living near an area where a hazardous waste incinerator is being constructed. The reported mean blood lipid CDD/CDF concentration was 27.0 ppt I-TEQ_{DF} with a range of 14.8 to 48.9 ppt. The maximum TEQ_{DF} value observed in this study was approximately 1.7 times the mean. CDD/CDF TEQs were higher in women (e.g., 27.7 ppt) than in men (e.g., 25.2 ppt). The results, however, were not statistically significant. Schumacher et al. (1999b) conducted a second study on adipose tissues of 15 autopsied subjects. The arithmetic mean I-TEQ_{DF} was 30.98 ppt (range of 13.4 to 69.4 ppt). The maximum I-TEQ_{DF} value observed in this study was approximately 2.2 times the mean. Unlike their previous study, I-TEQ_{DF}s were statistically higher ($p < 00.1$) in the fat of women (mean value: 45 ppt) than in men (mean value: 24 ppt). Levels of CDD/CDFs were higher for those people that lived in industrialized areas than the residents who lived in the city, but this difference was not statistically significant.

Beck et al. (1994) reported on levels of CDD/CDFs in adipose tissue from 20 males (mean age-50 years) from Germany. I-TEQ_{DF}s ranged from 18 ppt to 122 ppt with a mean of 56 ppt (using TEF_{DF}-WHO_{98}s, the mean TEQ_{DF} would be 65 ppt), on a fat weight basis. The I-TEQ_{DF} maximum concentration in this study was approximately 2.4 times the mean. Beck et al. (1994) also reported on CDD/CDF levels in various organs of the body. In comparison to adipose tissue, the concentrations of CDD/CDFs in brain and placental tissue were found to be low. Accumulation of CDD/CDFs was not found to occur in the thymus, spleen, and liver, based on whole weight concentrations. Schecter et al. (1994a) also reported on I-TEQ_{DF} levels in organs of two autopsy patients from New York. The highest concentrations of CDD/CDFs were found in adipose tissue (28 ppt I-TEQ_{DF}),

adrenal tissue (14 ppt I-TEQ_{DF}), and liver (12 ppt I-TEQ_{DF}), on a whole weight basis. Lower concentrations were observed in spleen (4.6 ppt I-TEQ_{DF}), muscle (2.4 ppt I-TEQ_{DF}), and kidney (0.8 ppt I-TEQ_{DF}). Schecter et al. (1994b) reported PCB levels for these two autopsy patients. Total PCBs in adipose tissue were 280.7 ppb on a wet weight basis and 344.2 ppb on a lipid weight basis.

Beck et al. (1994) also observed that CDD/CDF tissue levels were dependent on the age of the individual. I-TEQ_{DF} concentrations in infants ranged from 2.1 pg/g to 22 pg/g on a lipid basis. 2,3,7,8-TCDD was found to increase at a rate of 0.12 pg/g fat per year, and I-TEQs increased at a rate of 0.77 pg/g fat per year. Schecter et al. (1995a) measured levels of CDD/CDFs in human fetal tissue (N = 10) at 8 to 14 weeks gestational age and observed an average of 5 pg I-TEQ_{DF}/g on a lipid basis. Stillborn liver (N = 3) concentrations averaged 10 pg I-TEQ_{DF}/g on a lipid basis. These levels are considerably lower than those observed in adult tissues (Schecter et al., 1995a). Päpke et al. (1996) also observed that I-TEQ_{DF} levels in human tissues were age dependent. Whole blood samples collected in 1994 indicated that I-TEQ_{DF} concentrations increased with increasing age. Similar age effects were noted for PCBs 77, 126, and 169 (Päpke et al., 1996).

Wuthe et al. (1995) studied body burdens of CDD/CDFs among children in Germany. Three study groups were evaluated: blood from 11 nonexposed children, age 9 to 15 years; adipose and liver tissue from 20 stillborn or otherwise deceased infants, age 0 to 44 weeks, some of whom had been breast-fed; and pooled blood from 10-year-olds from 3 different regions. The total I-TEQ_{DF} concentration for the first study group (i.e., blood from 11 children between the ages of 9 and 15 years) was 10.7 ppt. Based on the other study groups, the authors made the following conclusions: (1) because CDD/CDFs were found in stillborns, a diaplacental transfer of these compounds occurred; (2) breast feeding has an impact on CDD/CDF concentrations (i.e., the mean I-TEQ_{DF} concentration was 12.7 ppt for breast-fed infants and 3.6 ppt for formula-fed infants); and (3) body burdens of CDD/CDFs are lower among children than adults.

Lanting et al. (1998) examined PCBs in adipose tissue, liver, and brain from nine stillborns at varying gestational ages. Of the four PCB congeners examined, only PCB 118 was dioxin-like. The median levels reported for PCB 118 were 20 ppt for adipose tissue, 17 ppt for the liver, and 6 ppt for the brain. The results of the study indicated that there was a significant relationship (correlation coefficient = 0.98; $p < 0.01$) between adipose

tissue concentrations and liver concentrations. Correlation between the levels of PCB congeners in these tissues and gestational age of the infants were not significant; correlation coefficients varied between 0.22 and 0.47.

Kruezer et al. (1997) reported CDD/CDF concentrations from lipids of adipose tissue and livers from cadavers (3 stillborns and 17 infants aged 0.43 to 44 weeks old who died from sudden infant death syndrome). I-TEQ_{DF} lipid-based concentrations were in the range of 1.55 to 29.63 ppt for adipose tissue (n = 20) and 2.05 to 57.73 ppt (n = 19) for liver. TCDD concentrations in lipids of breast-fed infants were higher compared to nonbreast-fed infants.

Nagayama et al. (1995) studied the effect of birth order on the body burdens of CDD/CDFs and PCBs among 50 healthy Japanese women. The concentrations of these dioxin-like compounds in blood were found to be significantly higher among first-born women than among other women. No relationship was found between the method by which these women were fed (i.e., breast-fed, formula-fed, or mix between breast milk and formula) and the blood concentrations of CDD/CDFs and PCBs.

Human breast tissue has also been analyzed for dioxin-like PCBs (Dahl et al., 1994; Petreas et al., 1998). Dahl et al. (1994) examined breast tissue collected from 16 women seeking hospital care for breast tumors in Sweden. PCB levels were observed to increase with age. Based on PCBs 105, 114, 118, 156, 157, 170, 180, and 189, the mean total TEQ_P-WHO_{98} for these samples was 40 ppt. Petreas et al. (1998) studied human breast adipose tissue collected from women undergoing breast surgery at Stanford University in California to determine CDD/CDF and PCB levels. Of the 17 CDD/CDF congeners, only OCDD, HpCDD, HxCDD, and PeCDF were observed to be above the limit of detection. I-TEQ_{DF} lipid-based concentrations, using one-half LOD for non-detects, ranged from 6 ppt to 78 ppt with a mean of 17.8 ppt (n = 62). Based on only the four detected congeners, the I-TEQ_{DF} concentration ranged from 5 ppt to 42 ppt with a mean of 12.6 ppt (the maximum I-TEQ_{DF} value is 3.3 times higher than the mean). Lipid-based PCB levels ranged from 451 ppb to 3,830 ppb with a mean of 1,120 ppb, based on PCBs 153/132, 180, 74, 138, 182/187, 170, 196/203, 194, 199, 156, 118, 206, 183, 99/113, 177, 28, 105/127, 128/162, 157, and 101 (n = 61). The maximum concentration is 3.4 times the mean. Lipid-based TEQ_P-WHO_{94} levels for coplanar PCBs 77, 126, and 169 ranged from 7 ppt to 110 ppt with a mean of 38 ppt (the maximum TEQ_P-WHO_{94} is 2.9 times higher than

the mean). The most prevalent PCB congeners included PCBs 153/132, 180, 74, 138, 182/187, and 170, which, when summed, contributed over 50 percent of the total PCB measure.

Iida et al. (1999) analyzed blood samples from 50 young (i.e., approximately 20 years of age) Japanese women for dioxin-like compounds. The women were described as "normal subjects" who had not yet had children, and the samples were collected in 1993 and 1994. The range if I-TEQ_{DF}s was 7.3 pt to 28.0 ppt with a mean of 16.4 ppt (the maximum value is 1.7 times higher than the mean). The range of TEQ_{P}-WHO_{94}s (based on PCBs 77, 126, and 169) was 1 ppt to 10 ppt with a mean of 4.9 ppt. The total TEQ_{DFP}-WHO_{94} was 21 ppt and the maximum value was 37 ppt. This maximum value is 1.8 times higher than the mean.

4.2.2. Breast Milk Studies from the 1980s and Early 1990s

Schecter et al. (1989b; 1992b) reported that in a study of 42 U.S. women, the average I-TEQ_{DF} was 16 ppt (20 ppt of TEQ_{DF}-WHO_{98}) (3.3 ppt of 2,3,7,8-TCDD) in the lipid portion of breast milk. Schecter et al. (1989b) also reported a total I-TEQ_{DF} of 27 ppt (TEQ_{DF}-WHO_{98} = 31 ppt) for human milk collected in Germany (n = 185). A much larger study in Germany (n = 526) showed an average of 29 ppt of I-TEQ_{DF} (TEQ_{DF}-WHO_{98} = 34 ppt) in lipid portion of breast milk (Fürst et al., 1994). Bates et al. (1994) analyzed breast milk samples from 38 women in New Zealand and reported mean lipid-based I-TEQ_{DF}s of 16.5 ppt for urban women and 18.1 ppt for rural women (average I-TEQ_{DF} = 17.2 ppt; average TEQ_{DF}-WHO_{98} = 21 ppt). The age of the mother was found to be positively correlated with the concentration of CDD/CDFs in breast milk. Beck et al. (1994) reported a mean I-TEQ_{DF} of 30 ppt (TEQ_{DF}-WHO_{98} = 35 ppt) in the milk fat based on 112 human milk samples from Germany. The congeners that contributed the most to the total I-TEQ_{DF} were 2,3,4,7,8-PeCDF (35 percent), total HxCDD (22 percent), and 1,2,3,7,8-PeCDD (21 percent). Beck et al. (1994) observed that CDD/CDF levels decreased with the number of children and the duration of breast feeding, but increased with the age of the mother. Beck et al. (1994) also compared the adipose tissue levels of breast-fed and bottle-fed infants who had died of sudden infant death syndrome. The breast-fed infants had higher tissue levels (5.4 to 22 pg/g fat; n = 4) than the bottle-fed infants (2.1 to 4.4 pg/g fat; n = 2).

Hirakawa et al. (1995) studied differences in CDD/CDF levels in human milk collected from primipara and multipara Japanese women. Human milk samples were taken from seven primiparas and eight multiparas between the ages of 22 and 40 years and analyzed for CDD/CDFs and dioxin-like PCBs. Total lipid-based TEQ concentrations were 34.6 ppt for the primiparas and 30.7 for multiparas, using I-TEF_{DF}s for CDD/CDFs and TEF_P-WHO_{94} for PCBs. Significant differences were observed between the concentrations of 2,3,7,8-TCDD; 1,2,3,7,8-PeCDD; 1,2,3,6,7,8-HxCDD; 1,2,3,7,8,9-HxCDD; 2,3,4,7,8-PeCDF; and 1,2,3,6,7,8-HxCDF in primipara and multipara women. The concentrations of these congeners varied by a factor ranging from 1.3 to 1.8 for the two study groups (Table 4-9). The mean I-TEQ_{DF} plus three standard deviations indicates that the high-end CDD/CDF concentration is approximately 2 times higher than the mean.

Van Cleuvenbergen et al. (1994) observed lipid-based I-TEQ_{DF} levels in human milk ranging from 27 to 43 ppt with a mean of 34 ppt (TEQ_{DF}-WHO_{98} = 40 ppt), based on samples from 9 women living in Belgium in 1992. The maximum I-TEQ_{DF} concentration observed in this study was approximately 1.3 times higher than the mean. OCDD and 1,2,3,4,6,7,8-HpCDD accounted for the highest proportion of total CDD/CDFs, but 2,3,4,7,8-PeCDF accounted for the largest proportion of the total CDD/CDF I-TEQ_{DF} (i.e., approximately 45 percent (Table 4-10)). Similar I-TEQ_{DF} levels have been observed in other countries. Schecter et al. (1989c) collected human milk samples from southern Japan in 1986. The mean lipid-based total I-TEQ_{DF} for two composites, containing three samples each, was 26 ppt. Based on data from Startin et al. (1989), the mean lipid-based I-TEQ_{DF} for a pool of 80 human milk samples from the United Kingdom was 33 ppt (TEQ_{DF}-WHO_{98} was 39 ppt).

Pluim et al. (1994a) studied the influence of short-term dietary changes in fats and carbohydrate intake on CDD/CDF concentrations in human milk. Two different diets were administered to two groups of lactating women in The Netherlands. Sixteen women had a low-fat/high-carbohydrate/low-dioxin diet, and 18 women had a high-fat/low-carbohydrate/low-dioxin diet for 5 consecutive days. At the end of this dietary regimen, milk samples were collected and analyzed for CDD/CDFs. No significant differences between CDD/CDF levels were observed. The mean I-TEQ_{DF} values for mothers using the low-fat/high-carbohydrate/low-dioxin diet were 30.2 ppt and 30.0 ppt before and after the test period, and the mean I-TEQ_{DF} values for the mothers using the high-fat/low-

carbohydrate/low-dioxin diet were 24.4 ppt and 24.0 ppt before and after the test period. Pluim et al. (1994a) concluded that short-term dietary changes were not an effective means of reducing dioxin concentrations in human milk. In another study, Pluim et al. (1994b) measured the levels of CDD/CDFs in breastmilk as part of a study to evaluate relationships between neonatal CDD/CDF exposure via breastmilk and potential physiological effects. CDD/CDFs were measured in the breastmilk of 35 Dutch mothers when their nursing infants were 11 weeks of age. The mean lipid-based I-TEQ_{DF} level in these breastmilk samples was 28.1 ppt (TEQ_{DF}-WHO_{98} = 33.5 ppt).

In 1994 and 1996, Hooper et al. (1998) monitored levels of CDD/CDFs in breast milk samples collected in Kazakstan, a country of the former Soviet Union. The mean reported CDD/CDF levels ranged from 7.2 to 57 ppt I-TEQ_{DF}. The detection limit for the sampling was 1 ppt, and only levels above the detection limit were reported. Approximately 92 breast milk samples were collected in both of these years. The range and mean values of individual and composite samples were similar by region and ethnicity. In addition, this study found that CDD/CDF levels were significantly higher in breast milk samples collected from rural sites (mean 46 ppt I-TEQ_{DF}, n = 23) than from a nonrural site (mean 11 ppt I-TEQ_{DF}, n = 32). Hooper et al. (1998) did not identify the reason for the higher CDD/CDF concentrations in samples from rural women. Several postulations include the high use of a pesticide (Hexachlorocyclohexane) in Kazakstan, the Kazakstan diet may include more contaminated fish from the Ural River, and consumption of cottonseed oil and kefir (a beverage of fermented cow's milk), which has been shown to have high dioxin levels. Consumption of cottonseed oil and kefir is more common in the rural areas than in urban areas.

Recently, Liem et al. (1996) reported on the results of the second round of a human breast milk study conducted by the World Health Organization (WHO). Human milk samples were collected from women in 19 countries during 1992/93 and analyzed for CDDs, CDFs, and PCBs (i.e., non-ortho 77, 126, 169; mono-ortho 105, 118; markers 28, 52, 101, 138, 153, 180). The results were compared to the results of the first round of sampling that occurred among 11 countries in 1987/88 to evaluate trends in exposure to dioxin-like compounds. Based on the 1992/93 results of pooled human milk samples, lipid-based I-TEQ_{DF} concentrations ranged from 3.8 pg/g for the Librazhd area of Albania to 27.1 pg/g for the Liege area of Belgium (Table 4-11). Overall, significantly lower I-TEQ_{DF}s

and PCBs were observed in Albania, Hungary, and Pakistan (Table 4-11). The highest I-TEQ_{DF} levels were observed in Belgium and The Netherlands (Table 4-11), and the highest TEQ_P-WHO_{94} levels were observed in Canada's Hudson Bay region and in regions of the Czech and Slovak Republics. An analysis of individual samples from The Netherlands and Denmark indicated a high level of variability among individuals (i.e., levels varied by a factor of 3 to 5). Comparison of the 1992/93 data to the 1987/88 data indicated that the levels of CDD/CDFs and marker PCBs in breast milk have declined in some countries with concentrations decreasing up to 50 percent in some areas (Table 4-12). Liem et al. (1996) estimated an overall annual decrease in CDD/CDFs of 7.2 percent over the 5-year time period evaluated.

Vartiainen et al. (1997) reported CDD/CDF and PCB levels in the human milk of 167 women collected in 1987 from an urban area and a rural area in Finland. The average CDD/CDF levels were significantly higher ($p < 0.001$) in the urban area (26.3 pg I-TEQ_{DF}/g fat; $n = 47$) than in the rural area (20.1 pg I-TEQ_{DF}/g fat; $n = 37$) for all primiparae individuals. Similarly, the total PCB concentrations were higher ($p < 0.01$) among urban primiparae (496 ng/g fat; 36.8 pg TEQ_P-WHO_{94}/g; n-47) than among rural primiparae (396 ng/g fat/ 26.3 pg TEQ_P-WHO_{94}/g; $n = 37$). The CDD/CDF and PCB levels in the milk of these women decreased with the increasing number of children breast-fed by them. Vartiainen et al. (1997) estimated that a woman's third child would be exposed to about 70 percent of the CDD/CDF and PCB levels that her first-born child was exposed to, and the eighth to tenth child would be exposed to only about 20 percent of the levels of the first-born. In addition, Vartiainen et al. (1997) observed a possible correlation between average I-TEQ_{DF} levels and total PCB concentrations (correlation coefficient (R) was 0.84 for the urban area and 0.71 for the rural area).

Kiviranta et al. (1999) coordinated a study from 1992-1994, which was designed as a follow-up of the Vartianen et al. (1997) study, measuring CDD/CDF and PCB levels in human milk in Finland. One round of 20 samples focused on urban areas (Helsinki, Finland) and the second round of 64 samples focused on rural areas (Koupio, Finland, and surroundings). Samples were divided into groups based on the number of children the mother has nursed. The groups included women who have had 1, 2, 3, 4, 6, or 13 children. The average CDD/CDF levels reported were 13.6 pg I-TEQ_{DF}/g fat for rural areas and 19.9 pg I-TEQ_{DF}/g fat for urban areas for all primiparae women. The average total

PCB concentrations were 198 pg/g fat from rural areas, and 296 pg/g fat for urban areas. The conclusions of the Kiviranta et al. (1999) study were identical to the Vartianen et al. (1997) study. The differences between the breast milk I-TEQ_{DF}s and PCB concentrations for rural and urban women remain and I-TEQ_{DF}s and PCB concentrations in breast milk also decreased proportionally when women had two or more children. It was also evident that there was a marked decrease in I-TEQ_{DF} and PCB levels when comparing to the values reported in 1992-1994 to those in 1987.

Tuinstra et al. (1994) evaluated the CDD/CDF and dioxin-like PCB content of human milk from The Netherlands. Samples were collected 10 and 42 days after delivery from about 200 mothers. Based on these data, the mean total I-TEQ_{DF} was 31 ppt (TEQ_{DF}-WHO_{98} = 36 ppt) (Tuinstra et al., 1994), and the mean TEQ_P-WHO_{94} for PCBs 77, 126, 169, 105, 118, 156, 170, and 180 was 36 ppt (TEQ_P-WHO_{98} = 31 ppt) (Tuinstra et al., 1994; Koopman-Esseboom et al., 1994).

Similar estimates of the dioxin-like PCB content of human milk have been obtained for North America and Europe. Hong et al. (1992) analyzed human milk samples from upstate New York for PCBs 77, 105, 114, 118, 123, 126, 156, 157, 167, 169, and 189. PCB 118 accounted for the highest proportion of the total PCB concentration. The mean lipid-based TEQ_P-WHO_{94} and TEQ_P-WHO_{98} for these samples was 13 ppt. The total TEQ_P-WHO_{94} for 96 pooled human milk samples from Canada was also 13 ppt (TEQ_P-WHO_{98} = 10 ppt) (Dewailly et al., 1994). She et al. (1995) analyzed 12 human milk samples for PCBs 77, 118, 105, 126, 156, 169, 170, and 180. The total TEQ_P-WHO_{94} for these samples was 16 ppt (TEQ_P-WHO_{98} = 14 ppt).

For European countries, the lipid-based TEQ_P-WHO_{94} levels were 22 ppt (TEQ_P-WHO_{98} = 18 ppt), based on 1990/91 data for PCBs 118, 156, 170, and 180 from 68 German women (Georgii et al., 1995) and 32 ppt (TEQ_P-WHO_{98} = 30 ppt), based on data for PCBs 77, 126,169, 105, 118, 114, 156, 170, and 180 from 28 Norwegian mothers (Johansen et al., 1994). Noren et al. (1990) and Noren and Lunden (1991) analyzed human milk samples from Sweden in 1989 (n = 2) and in every 4 years between 1972 and 1988/89, respectively. Total TEQ_P-WHO_{94}s based on Noren et al. (1990) were 29 ppt (TEQ_P-WHO_{98} = 27 ppt) (PCBs 118, 105, 156, 180, 77, 126, and 169). Noren and Lunden (1991) observed that the concentrations of PCBs in human milk declined between 1972 and 1984/85, but that the 1988/89 samples had similar concentrations as the

1984/85 samples. Based on the 1988/89 sampling period, the total TEQ_P-WHO_{94} was 19 ppt (TEQ_P-WHO_{98} = 18 ppt) based on PCBs 105, 156, 180, 77, 1216, and 169 (n = >100).

Van der Velde et al. (1994) compared the levels of PCBs 77, 126, and 169 in cow's milk and human milk from The Netherlands. The concentrations of these compounds were found to be higher in human milk than in cow's milk collected from a background location (Table 4-13). Based on these data, the total TEQ_P-WHO_{94} and TEQ_P-WHO_{98} for human milk was 9.4 ppt for these three dioxin-like PCBs.

Abraham et al. (1998) measured CDD/CDF and coplanar PCBs in blood of four mothers before and after delivery and during lactation. Abraham et al. (1998) also examined their breast milk and their infants blood for concentrations of CDD/CDF and coplanar PCBs. CDD/CDF and coplanar PCBs were also quantified in the cord blood, meconium, and transit stool. Table 4-14 presents a summary of the TEQ_{DF}s of mothers' milk and blood, and infants' blood. For two of the mothers (mother 1 and mother 2), the data were associated with their second delivery, and data were also available for their first-born infants at the age of 11 to 12 months. Mother 3 was the only subject that did not fully breastfeed her infant for at least 17 weeks. The results of this study suggest that CDD/CDF and coplanar PCB TEQs in the blood of the second infants were only about half as much as in the first born children (at the same age). This is likely a result of reductions in CDD/CDF concentrations in breast milk as a result of previous lactation. In addition, the infant that was not fully breast-fed had a lower l-TEQ_{DF} concentration in the blood than the fully breast-fed infants. Lipid-based CDD/CDF concentrations in the infants' tissues appeared to increase during the 11 months after birth, based on the comparison of infants' blood CDD/CDF concentrations at 11 months and CDD/CDF concentrations in cord blood concentrations.

Schecter et al. (1998) analyzed blood and milk from a mother that nursed twin babies over a 38-month period. In this study, a woman gave birth to twins on December 15, 1992. Blood and milk samples were taken each month starting in February 1993 and ending in September 1995. Overall, CDD levels in milk decreased from 309 ppt to 173 ppt, CDF levels dropped from 21 ppt to 9 ppt, and total coplanar PCB levels decreased from 151 to 21 ppt during that time period. Schecter et al. (1998) estimated that the mother reduced her dioxin body burden from 310 to 96 ng TEQ_{DFP}-WHO_{98}, or

approximately 69 percent during that time period. Overall, the CDD/CDF/PCB concentrations in the maternal whole blood dropped from 698 ppt to 262 ppt in lipids during that time period. The twins' consumption of CDD/CDF and coplanar PCBs from breast feeding was estimated to be approximately 115 ng TEQ_{DFP}-WHO_{98} per twin.

The levels of dioxin-like compounds in human breast milk can be predicted on the basis of the estimated dioxin intake by the mother. Such procedures have been developed by Smith (1987) and Sullivan et al. (1991). The approach by Smith assumes that the concentration in breast milk fat is the same as in maternal fat and can be calculated as:

$$C_{milk\ fat} = \frac{m\ h\ f_1}{0.693\ f_2} \qquad \text{(Eqn. 4-1)}$$

where:

$C_{milk\ fat}$ = Concentration in maternal milk (pg/kg of milk fat);
m = Average maternal intake of dioxin (pg/kg of body weight/day);
h = Half-life of dioxin in adults (days);
f_1 = Proportion of ingested dioxin that is stored in fat; and
f_2 = Proportion of mother's weight that is fat (kg maternal fat/kg total body weight).

This steady-state model assumes that the contaminant levels in maternal fat remain constant. Though not described here, Smith (1987) also presents more complex approaches that account for changes in maternal fat levels during breast feeding. The model developed by Sullivan et al. (1991) is a variation of the models proposed by Smith (1987). The Sullivan model considers changes in maternal fat levels and predicts chemical concentrations in milk fat as a function of time after breast feeding begins. The model proposed by Smith assumes that infant fat concentration at birth is zero; whereas, Sullivan assumes that the infant fat concentration at birth is equal to the mother's fat concentration.

Flesch-Janys et al. (1996) estimated the half-life of 2,3,7,8-TCDD in humans to be approximately 7 years. For the purpose of this preliminary analysis, it is assumed that a 7-year half-life applies to all of the dioxin-like compounds. Smith (1987) suggests values of 0.9 for f_1 and 0.3 for f_2. Using these assumptions and a background exposure level of

1 to 3 pg of TEQ_{DFP}-WHO_{98}/kg-d (derived from diet analysis, see Section 4.4.2 and previous assessments of background exposure), the concentration of dioxin-like compounds in breast milk fat is predicted to be about 10 to 30 ppt of TEQ, which is slightly lower than the measured values.

Uncertainty is introduced into this estimate by the assumption that the assumed half-life rate and partitioning factors apply to all the dioxin related compounds. Although these properties are likely to be similar among the various congeners, some variation is expected. It is unknown whether the net effect of these uncertainties would lead to over or under estimates of dose. However, the simple model appears to provide reasonable predictions of background levels found in breast milk and was judged adequate for purposes of a preliminary analysis. For detailed assessments, readers should consider using the more complex models and developing chemical-specific property estimates.

Travis et al. (1988) presented an alternative approach to estimating breast milk contaminant levels. They proposed a biotransfer approach:

$$C_m = B_m I \qquad \text{(Eqn. 4-2)}$$

where:

C_m = Contaminant concentration in breast milk fat (mg/kg);
B_m = Biotransfer factor for breast milk fat (d/kg); and
I = Maternal intake of contaminant (mg/d).

Travis et al. (1988) also argued that the biotransfer factor is primarily a function of the octanol-water partition coefficient (K_{ow}) and developed the following geometric mean regression:

$$B_m = 6.2 * 10^{-4} K_{ow} \qquad \text{(Eqn. 4-3)}$$

This regression was derived from data on six lipophilic compounds (log K_{ow} range: 5.16 to 6.5), but did not include any dioxins or furans. Assuming a log K_{ow} of 6.6 for 2,3,7,8-TCDD, a B_m of 3,700 d/kg is predicted. Combining this value with a maternal intake of 6

pg/d, a breast milk concentration on a fat basis of 22 ppt is predicted. This prediction is about 7 times higher than what has been measured for TCDD in breast milk in the United States. Thus, this approach appears to overpredict TCDD levels while the approach suggested by Smith (1987) appears to underpredict total TEQ levels.

4.2.3. The Blood Studies of the CDC Collaboration (1995-1997)

The Centers for Disease Control (CDC) has compiled data on blood concentrations of dioxins, furans, and coplanar PCBs from individuals in the United States with no known exposures to dioxins (CDC, 2000). These data come from site-specific studies (with permission from principle investigators in those studies), and CDC has provided the laboratory analyses of all the blood samples. All the samples were collected between 1995 and 1997. There are a total of 316 individuals included in their compilation from six locations: 1) Manchester, Missouri (n = 61), 2) Times Beach, Missouri (n = 67), 3) Jacksonville, Arkansas (n = 57), 4) Oregon (n = 9), 5) Wisconsin (n = 93), and 6) North Carolina (n = 29). CDC is preparing manuscripts for peer literature publication of statistical summaries and interpretations of this data. They have provided EPA with an overall statistical summary of the congener-specific and overall TEQ results from this compilation (Patterson, 2000), and those results will be described shortly. EPA judges these data to be the best representation of current background concentrations of dioxin-like compounds in the blood of US citizens, for these reasons: 1) all individuals were evaluated by the CDC analysis group as appropriately representing US background conditions and EPA concurs with this evaluation - that is, all individuals were judged to be exposed only through background exposures, including inhalation of background ambient air (i.e., not impacted by nearby high dioxin stack emitters), consumption of animal food products not known or expected to be contaminated, no occupational exposures, and so on, 2) the blood was analyzed using a consistent, high resolution, mass spectrometry state-of-the-art protocol (Patterson and Turner, 1997) which included 4 dioxin-like coplanar PCBs, 3) the data represent a wide range of adult ages, from 20 to over 70 years of age, and 4) the sampling was of relatively recent origin - 1995 to 1997, more recent than other studies reviewed in this chapter. Prior to describing this overall profile, information on four of the six study sites have been made available to EPA, and these will be described first.

With the assistance of the Agency for Toxic Substances and Disease Registry, the Missouri Department of Health (MDOH, 1999) conducted an exposure study to evaluate the potential impact of incinerating contaminated soil from Times Beach. Approximately 265,000 tons of soil and other materials containing 2,3,7,8-TCDD from 27 eastern Missouri sites were burned at the Times Beach Superfund site during the period March 17, 1996 through June 20, 1997. MDOH (1999) undertook a study to evaluate the impact of emissions from this incineration. Their approach was to take blood samples from a target and a comparison population before, during, and after the incineration, and evaluate the differences in blood levels of dioxin-like compounds between the populations and over time. MDOH (1999) selected a target population based on air dispersion and deposition modeling. This population resided within a 4-kilometer radius of the incinerator. A comparison population from Manchester was located about 16 kilometers from the incinerator. From a list of over 650 individuals from both populations, totals of 76 and 74 individuals were selected from the target and comparison groups, respectively, for blood sampling. These selections considered demography, whether or not a woman was pregnant or breast feeding (neither was selected), and other critical factors. Blood samples were taken from all participants in September 1995, July 1996, and June 1997, and questionnaires were administered each time. Mean concentrations of each of 15 dioxin and furan congeners, and 4 coplanar PCB congeners were determined assuming non-detects were equal to one-half the detection limit. These detection limits, on a lipid basis, were: 0.8 ppt for the tetra- and penta-CDD congeners and the tetra- through octa-CDF congeners, 1.2 ppt for the hexa- through hepta-CDD congeners, 3.8 ppt for the coplanar PCB congeners, and 15.4 ppt for OCDD. Concentrations for two hexa-CDD congeners, 1,2,3,4,7,8-HxCDD and 1,2,3,6,7,8-HxCDD, and one hexa-CDF congener, 1,2,3,7,8,9-HxCDF, were not reported, and concentrations of one hepta-CDD congener which is not assigned a TEF value, 1,2,3,4,6,7,9-HpCDD, was reported. The mean concentrations for each congener for each testing period and study group, is shown in Table 4-15. Further details on this study can be found in MDOH (1999).

The CDC compilation included only the data from 1997. For that year, 67 of the 76 individuals from Times Beach had available measurements for their compilation, and 61 of the 74 individuals from the comparison site, Manchester, had available measurements.

MDOH (1999) concluded that there was no statistically significant differences between the target and comparison groups for all the analytes measured except for PCB 126, which was slightly higher in the comparison group. MDOH (1999) concluded that the values measured were some of the lowest values ever recorded on a human population. As seen in Table 4-15, the TEQ_{DFP}-WHO_{98} for the target group was 11.7 ppt while for the comparison group it was 12.6 ppt (averaged over all sampling dates). However, the actual TEQ concentrations would be higher than these since this study did not report on measurements for the three congeners noted earlier. Other data suggest that the hexa-CDD congeners not reported on in this study, mainly 1,2,3,6,7,8-HxCDD, comprise in the range of one-fourth to one-third of the total body burden of TEQ. MDOH (1999) also observed that there appeared to be a decrease in concentrations from pre- to post-incineration for most analytes. Of all factors examined through questionnaires, only two appeared to be important for dioxin body burdens: smoking and age. Combining both populations, the average TEQ for participants living in homes with cigarette smokers as 12.8 ppt (I-TEQ_{DF} + TEQ_{P}-WHO_{94}), compared to 9.4 ppt (I-TEQ_{DF} + TEQ_{P}-WHO_{94}) in homes that do not have smokers. No age-specific results were presented in MDOH (1999), but a Pearson correlation of 0.525 for average TEQ concentration (statistical significance <0.001, two-tailed) was found for age. The average age of participants in both populations was about 43 years.

The Arkansas Department of Health (ADH) and the Agency for Toxic Substances and Disease Registry (ATSDR) cooperated on the design and implementation of a study to evaluate the exposure of individuals to dioxin-like compounds and other contaminants manufactured and then disposed of through incineration at the Vertac/Hercules Superfund Site (abbreviated the Vertac Site) in Jacksonville, Arkansas (ADH, 1995). The site had been used from the 1950s to manufacture herbicides such as 2,4,-D, 2,4,5-T, and 2,4,5-TP. It had changed hands several times until being abandoned by Vertac in 1987. Incineration occurred between 1992 and 1994. One component of the study was to sample and then analyze blood from three target groups of individuals: 1) residents living near the Site for more than 15 years as of 1991 - 72 individuals recruited, 2) residents living between 1 and 5 years as of 1991 - 36 recruited, and 3) residents living in a comparison area - 72 recruited; 71 participated. The comparison area chosen was in Mabelvale, Arkansas, a demographically similar community approximately 25 miles south

of Jacksonville. Study participants ranged in age from 18 to 65 years old. The average age of the comparison group at the first sampling in 1991 was 40 years. Blood samples were taken in March, 1991, and participants also filled out an extensive questionnaire at that time. Subsets of individuals from all three populations were sampled once again in 1994 and 1995 after the incineration had been completed.

The CDC compilation used only the data from 1995 in their compilation. This data set included individuals who lived both in Jacksonville and in Mabelville - most of the individuals followed into 1995 lived in Jacksonville. The number of individuals sampled in 1995 included in the CDC compilation is 57.

The 1991 and 1994 sampling were described in a draft report released by the Arkansas Department of Health for public comment in 1995 (ADH, 1995). This report has never been finalized. However, the blood data has been available and even used by one researcher citing results from the Mabelville population sampled in 1991 as a comparison group to his own study of dioxin-like compounds in the blood of a Great Lakes sport-fishing population (Anderson et al., 1998). Individual results that are summarized here have been provided to EPA via personal communication (Cranmer, 1996). The data supplied for each dioxin-like congener was either: identified as a quantified concentration (in serum, on a lipid basis), identified as "not detected" (ND), or identified as "not reported"(NR). Detection limits were not specified. Therefore, for purposes of the calculation of means, non-detects were assumed equal to zero. Measurements identified as NR were not included in the calculation of means.

Table 4-16 summarizes the results from the comparison population only. This table shows the results for the entire set of 71 individuals sampled in 1991. It also shows the results for subsets of these individuals that were sampled in 1994 and 1995. For comparison, the 1991 means for these same subsets are also provided. Unlike the target population of the Times Beach study described earlier, there appeared to be measurable impacts on the blood levels of dioxin-like compounds in the target populations at Vertac, as evidenced by the 1991 sampling. However, these impacts have not been tied directly to activities at Vertac. For example, in groups 1 (15 years residence near the site) and 2 (between 1 and 5 years residence), the mean lipid-based concentrations of 2,3,7,8-TCDD were 8.5 and 4.2 ppt, while the mean for the background population was 2.5 ppt. The high means for groups 1 and 2 were driven by a small number of very high concentrations

(the three high concentrations from group 1 were 29.7, 84.9, and 94.8 ppt). However, if these high values are excluded, the overall concentrations from these groups are still higher than for the comparison group. The average TEQ_{DFP}-WHO_{98} from the comparison population in 1991 was 25.2 ppt. The select group of 18 individuals who were targeted for resampling in 1994 were individuals whose lipid-based concentration of 2,3,7,8-TCDD ranged from 2 to 5 ppt. Table 4-16 suggests that the average blood TEQ_{DFP}-WHO_{98} level for this group decreased between 1991 and 1994, from 26.8 to 22.6 ppt. However, when evaluating the average CDD/CDF/PCB concentration of the 14 individuals resampled in 1995 (a further subset of the 18 who provided samples in 1994), there appears to be little evidence of a decline in TEQ_{DFP}-WHO_{98}. The TEQ_{DFP}-WHO_{98} concentrations were 25.0 ppt in 1991 and 24.0 ppt in 1995 for this group. As with other studies, ADH (1995) also reported on an important age effect - the levels of dioxins and furans increased with age.

Grassman et al. (1999) developed a method to evaluate inter-individual variation in dioxin responsiveness among humans. Specifically, they developed a system that measures dioxin-responsive biomarkers in peripheral blood lymphocytes challenged *in vitro* with 10 nM TCDD during cell culture. Grassman et al. (1999) evaluated the capabilities of this method by obtaining blood samples from 3 populations widely variable in the magnitude and duration of their exposure to dioxin. One was a group of plant workers in a German chemical manufacturing plant, one was comprised of men, women, and children living in the vicinity of Seveso, Italy, during the accidental release of 2,3,7,8-TCDD in 1976, and the third was comprised of adult North Carolina volunteers, with no known occupational or unusual exposures to dioxin. This third group is comprised of 29 individuals, with ages ranging from 21 to 52 years, mean of 34.5 years, and it is the results from their analyses that are considered here as a U.S. background population. Grassman et al. (1999) reported that their average lipid-based TEQ_{DFP}-WHO_{94} was 14.2 ppt. Results of the study comparing the three study groups are reported in Grassman et al. (1999).

The North Carolina participants were sampled in 1996. EPA was provided the congener specific data for the 29 individuals of this study (Masten, 2000). Average congener concentrations from this group are provided in Table 4-17. Interferences were found in the analysis for 1,2,3,6,7,8-HxCDD, so this congener was not reported for any of the individuals, and TEQs were calculated without this congener. Other body burden data

suggests that this congener could comprise in the range of one-fourth to one-third of the body burden of TEQ_{DFP}, so the overall TEQ for this population is underestimated. A small number of additional measurements from other congeners were not reported, and these were not considered in the generation of mean congener values. The mean values were calculated by assuming that non-detects were equal to one-half the detection limit. With this procedure, the lipid-based TEQ_{DFP}-WHO_{98} was calculated to be 15.0 ppt. Assuming that non-detects are equal to zero would not change these results by much; the lipid-based TEQ_{DFP}-WHO_{98} in this case was calculated as 13.0 ppt.

The CDC compilation includes these same data from the 29 North Carolina individuals. The congener profile for the overall compilation done by CDC is shown in Table 4-18. These averages were derived assuming non-detects were equal to ½ the detection limit. These average congener concentrations were derived only using data from the overall set where these congeners were reported. As noted in the above discussions, there were some studies where congeners were not reported, such as 1,2,3,6,7,8-HxCDD. Therefore, the number of observations that went into calculating overall averages for each congener was less than or equal to the total number of individuals (n = 316) in the study. These congener profiles were not used to generate TEQ concentrations for the overall data base. Instead, Patterson (2000) supplied statistical results for the TEQ_{DFP}-WHO_{98} concentrations that were generated using substitution methods for each individual included who had "not reported" (NR) for some of the congeners. Each time a congener was NR in an individual's congener profile, the average concentration from other individuals in the same study set was substituted for the individual who had the missing data. When that congener was missing from an entire study set, then the average for that congener from all other data sets where it was reported was substituted for all individuals in the data set with the missing congener. With these substitution techniques, every individual included in the overall data base had a complete set of congener results including quantified concentrations, non-detects with known detection limits, and substituted values. Then, each individual's TEQ_{DFP}-WHO_{98} lipid-based concentration was derived (assuming non-detects equal ½ detection limit), and from these TEQs, means and percentiles were generated. By this discussion, it should be clear that one cannot derive the TEQ concentrations in Table 4-18 from the congener profiles in Table 4-18, although they will be close.

As seen in Table 4-18, the average lipid-based TEQ_{DFP}-WHO_{98} concentration was 22.1 ppt. It was found that the substituting ND = ½ LOD did not influence the TEQ results. At ND = 0, the average TEQ concentration was only 1 ppt lower at 21.1 ppt TEQ_{DFP}-$WHO_{98.}$ However, this TEQ_{DFP}-WHO_{98} concentration included only 4 of the 12 coplanar PCB congeners. The overall compilation of literature data on coplanar PCB concentrations in human tissues, other than this CDC compilation, shown later in this chapter in Table 4-21, includes data on 11 of the dioxin-like coplanar PCBs. That data suggests a weighted mean TEQ_P-WHO_{98} concentration in blood of 15.6 ppt TEQ_P-WHO_{98}, of which these four congeners comprise 5.9 ppt. Therefore, the congeners missing from the CDC data base account for 62% [(15.6-5.9)/15.6 * 100%] of the total PCB TEQ estimated in the early 1990's for blood. From the congener profile in Table 4-18, it is calculated that the 4 PCB congeners add about 2.0 ppt TEQ to the overall mean concentration of 22.1 ppt. 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 the estimate of current PCB blood concentrations by another 3.3 ppt TEQ_P-WHO_{98} lipid for a total PCB TEQ of 5.3 pg/g lipid and a total TEQ_{DFP}-WHO_{98} of 25.4 ppt lipid. This will be the TEQ lipid concentration assumed to represent current background conditions in the United States.

4.2.4. Additional Recent Tissue Studies

Petreas, et al. (2000) reported on the analysis of breast adipose tissue samples for the seventeen dioxin-like CDD/F congeners. Samples were taken in 1998 from women in San Francisco area hospitals undergoing breast surgery for suspected breast cancer. l-TEQ_{DF} concentrations were reported for 45 of these women who were found to be cancer-free. The range of l-TEQ_{DF} concentrations found in this study population was 10 to 60 ppt lipid-basis, with a median concentration of 19 ppt. This was calculated assuming non-detects were equal to ½ the detection limit. When assuming non-detects were equal to zero, this dropped slightly to 16 ppt l-TEQ_{DF}. When recalculating TEQs using the WHO_{98} TEF scheme, Petreas et al. (2000) found the concentrations to increase by 2-3 ppt. These concentrations compare well to the mean concentration of WHO_{98}-TEQ_{DF} of approximately 21.6 ppt lipid-basis found in the 316 samples of the CDC compilation (Patterson, 2000) reported on earlier. These results were compared to a set of 17 adipose

samples from other women patients undergoing surgeries for other reasons 10 years earlier in 1988. From 17 samples, the range was similar at 13 to 63 I-TEQ_{DF}, but the median was higher at 27.3 pg/g I-TEQ_{DF} lipid-basis. Other analyses by Petreas demonstrate the apparent downward trend in body burdens in these adipose tissues.

4.2.5. Summary of Human Tissue Levels

Tables 4-19 and 4-20 present summaries of the TEQ_{DF} concentrations in human tissues from North America, and Europe and Japan, respectively, as reported in the literature. In general, these data represent studies conducted in the late 1980s and early 1990s. These data on human adipose tissue, blood, and breast milk indicate that mean tissue concentrations of CDD/CDFs ranged from 20 to 50 ppt TEQ_{DF}-WHO_{98} on a lipid basis, with a midpoint of 35 ppt TEQ_{DF}-WHO_{98} during that time period. The mean TEQ_{DF}-WHO_{98} from the U.S. studies was 32.7 ppt, and the mean from the European and Japanese studies was 41.0 ppt. The assumption is made here that levels in all three tissues are similar (on a lipid basis) and that levels in all of these tissues can be considered representative of overall body burden. Van den Berg et al. (1994) reported that (on a lipid basis) the serum-to-blood tissue ratio for 2,3,7,8-TCDD is approximately one and this ratio increases with higher chlorinated CDD/CDFs. Van den Berg et al. (1994) also compared lipid-based concentrations for all CDD/CDF congeners reported in human milk, blood, and adipose, and concluded that the levels are strikingly similar across tissues.

It should be noted that all available human tissue studies have uncertainties that prevented a precise, statistically-based estimate of the national mean. Except for NHATS, the number of people in the available studies of CDD/CDFs in human tissues is relatively small, and participants are not selected in a statistically based manner. Other biases may have also been present in NHATS, as well as in other studies. Thus, it is uncertain how representative these data were of the general population.

Tables 4-21 and 4-22 present summaries of PCB TEQ concentrations in human tissues from North America and Europe, respectively, based on data from the 1980s and early 1990s. The average tissue level of dioxin-like PCBs for the general U.S. population was probably within the range of 10 to 30 ppt TEQ_{P}-WHO_{98} on a lipid basis, with a midpoint of about 20 ppt. The mean TEQ_{P}-WHO_{98} from these U.S. studies was 16.7 ppt. The mean from the European studies was 31.9 ppt. This indicates that on a TEQ_{P}-WHO_{98},

PCB levels were between one-half and two-thirds that of CDD/CDFs. Inclusion of dioxin-like PCBs raised the estimate of U.S. human tissue levels to approximately 30 to 70 ppt TEQ_{DFP}-WHO_{98} (midpoint = 55 ppt) for the late 1980s and early 1990s.

As discussed above, the representativeness of these PCB studies for the general population is unknown. The toxic equivalency factors for PCBs are not as well established as the CDD/CDFs and increase uncertainty in these estimates. Uncertainty is also increased by the high background levels of PCBs found in many laboratories, which can create analytical difficulties. In addition, not all studies presented data for the same set of PCB congeners. Therefore, studies were combined to calculate a total TEQ_P-WHO_{98} based on all PCB congeners for which TEF_P-WHO_{98}s have been established. Total TEQ_P-WHO_{98}s were calculated by summing weighted mean TEQ_P-WHO_{98} concentrations (based on one or more studies) for each toxic PCB congener.

The CDC data base includes 316 individuals from 6 sites in the time frame of 1995-1997. These data form the basis of the estimates of current background tissue levels in the United States. The mean TEQ tissue level from the study data alone is 22.1 ppt TEQ_{DFP}-WHO_{98}. Because this concentration does not include important dioxin-like PCB congeners, this average has been increased to 25.4 ppt TEQ_P-WHO_{98} using information from earlier studies of dioxin-like PCBs in blood. This concentration will be used to represent current background conditions in the United States. This use includes an overall conclusion for body burdens of dioxin-like compounds in this chapter, as well as an assumption for mother's milk concentration in an evaluation of the impacts of nursing on infants in Chapter 5.

It is important to note that the 95th percentile concentration from this study data base is 38.8 ppt TEQ_P-WHO_{98}, which is nearly twice the mean of 22.1 ppt TEQ_P-WHO_{98} from this study. Later in this chapter, variation in background dose is investigated using data on dietary consumption of fats. Using statistical surveys on food consumption, it was found that the 95th percentile of fat consumption was about twice the mean (and the 99th percentile is about 3 times the mean). Knowing that dioxins are transmitted primarily through consumption of dietary fat, this result from the CDC blood compilation is consistent with the dietary result; the 95th percentile consumption of dietary fat appears to lead to the 95th percentile in body burden of dioxin-like compounds.

A portion of the CDC blood data were plotted as a function of age. This plot is shown in Figure 4-1. This figure was generated as part of a site-specific study conducted by the Agency for Toxic Substances and Disease Registry at Mossville, Louisiana (ATSDR, 1999). The data shown in Figure 4-1 encompass the control population that was to be compared against measurements in Mossville. This comparison population is a subset of the full CDC (2000) population. Figure 4-1 shows that blood levels generally increase with age, and also that the variability in blood levels increase with age. An age trend such as this one has been observed in other studies, such as the NHATS tissue data described earlier (U.S. EPA, 1991a).

4.2.6. Body Burden Profiles

The profiles for CDD/CDF concentrations in human adipose tissue, blood, and human milk are presented in Figure 4-2 and Table 4-23 based on the literature studies from the 1980s and early 1990s. These profiles were generated by calculating the ratio of the mean concentrations of the 2,3,7,8-substituted congeners to total concentration of 2,3,7,8-substituted CDD/CDFs when nondetects were set to one-half the detection limit. In addition, it should be noted that some studies (i.e., adipose tissue - Schecter, 1991 and U.S. EPA, 1991a; blood - Schecter et al., 1994a and Cole et al., 1995) reported total 2,3,7,8-substituted HxCDD/F and HpCDD/F concentrations instead of reporting concentrations for the individual HxCDD/F and HpCDD/F congeners. Thus, in order to provide a complete profile based on all 17 of the 2,3,7,8-substituted congeners, the concentrations of total HxCDDs, HxCDFs, HpCDDs, and HpCDFs from these studies were apportioned among the individual HxCDD/F and HpCDD/F congeners based on the ratios of individual congeners to total HxCDD/Fs and HpCDD/Fs reported in studies providing data for the individual 2,3,7,8-substituted HxCDD/F and HpCDD/F congeners (i.e., adipose tissue - Patterson et al., 1994; blood - Schecter et al., 1993). The profiles generated for these three body tissues appear to be similar. In general, higher-chlorinated CDDs dominate with OCDD accounting for over 65 percent of the total 2,3,7,8-substituted CDD/CDFs. CDFs account for a relatively small portion of the total 2,3,7,8-substituted CDD/CDFs.

The profile of 2,3,7,8-CDD/CDF congeners in human blood from the more recent (i.e., 1995-1997) CDC blood data set was also generated. This profile is shown in Figure

4-3 and is based on the data in Table 4-18. The profile is similar to that generated from earlier human tissue data (Figure 4-2).

4.3. INTAKE ESTIMATES BASED ON TISSUE LEVELS AND PHARMACOKINETIC MODELING

4.3.1. Steady State Approach

Examination of human tissue data provides a way to estimate exposures of humans to CDD/CDFs. Average daily intake of CDD/CDFs may be estimated using human tissue data and pharmocokinetic modeling as follows:

$$D = \left[\left(\frac{\ln 2}{t_{1/2}}\right) V * CF_1 (C) CF_2\right] / (A) \qquad \text{(Eqn. 4-4)}$$

where:

D	=	Daily intake of CDD/CDF (pg/day);
$T_{1/2}$	=	Half-life of CDD/CDF (years);
V	=	Volume of body fat (kg);
C	=	Concentration of CDD/CDF in tissue (pg/g)
CF_1	=	Conversion factor (1,000 g/kg);
CF_2	=	Conversion factor (year/365 days); and
A	=	Fraction of dose that is absorbed.

The level of 2,3,7,8-TCDD found in human adipose tissue averages about 5.5 ppt in the United States based on data from a variety of studies from the 1980s and mid 1990s, and 2.1 pt based on the CDC data set. These values may be used to estimate the associated exposure levels using a simple pharmacokinetic model that back calculates the dose needed to achieve the observed tissue levels under the assumption of steady-state exposure/dose, as given above. (See Equation 4-4.) This model requires an estimate of the fraction of the dose that is absorbed, the elimination rate constant, and body fat volume.

A complete summary of the literature on gastrointestinal, dermal, transpulmonary, and parenteral absorption is provided in Part II - Health Assessment of 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin and Related Compounds, Chapter 1 - Disposition and Pharmacokinetics. The summaries there pertaining to oral absorption justify the selection of 0.8 as an absorption fraction for dioxin TEQs in simple exercises conducted in this section on pharmacokinetic modeling. Most of the gastrointestinal absorption research has been conducted on 2,3,7,8-TCDD and laboratory animals. Results suggest that 2,3,7,8-TCDD is absorbed at a rate greater than 50% in oil or in diet, with several studies reporting average absorption at 70% or more: Rose, et al. (1976) found an average of 84% in rats where the vehicle was a mixture of acetone and corn oil; Piper, et al. (1973) found an average of 70% on rats with the same vehicle; Diliberto et al. (1996) reported 88% in rats in a vehicle of vegetable oil, ethanol, and water; and Olson, et al (1980) reported 70% in hamsters in a vehicle of olive oil. Similar and even higher absorption was found for 2,3,7,8-TCDF, 1,2,3,7,8-PCDD, 2,3,4,7,8-PCDF, and 3,3',4,4'-TCB. Lower absorption at 2 to 15% was found for OCDD (Birnbaum and Couture, 1988), but since background TEQ doses are dominated by the lower chlorinated congeners, the low absorption of OCDD may be less critical. In limited studies and evaluations of oral absorption on humans, it is concluded that the more soluble congeners, such as 2,3,7,8-TCDF are almost completely absorbed, whereas the extremely insoluble OCDD is poorly absorbed. In one experiment, Poiger and Schlatter (1986) found that >87% of the oral dose of TCDD in corn oil in a 42 year-old man was absorbed from the gastrointestinal tract. Like some of the experiments on rats, the amount absorbed in some cases was dose dependent, with lower absorptions at higher doses. Again, low absorption at high doses is less critical for the current exercises, which focus on low background dose of TEQs.

Flesch-Janys et al. (1996) estimated the half-life of 2,3,7,8-TCDD (and other CDD/CDFs) based on blood levels of a group of occupationally exposed individuals. The median half-life for 2,3,7,8-TCDD (n=48) was estimated to be 7.2 years. Half-lives for other CDD/CDF congeners ranged from 3.0 to 19.6 years. Van der Molen et al. (1998) estimated the elimination rate constant of 2,3,7,8-TCDD using data on the TCDD blood lipid levels of Vietnam veterans who had been involved in the spraying of Agent Orange. The Van der Molen et al. (1998) model predicted half-lives ranging from 5.5 years in

young adults to 11 years in elderly men. The model accounted for age-dependent body composition, and age- and time-dependent background intake.

Ryan et al. (1997) reported the elimination rate constant of 2,3,7,8-TCDD by back calculating from the levels in 1992 and 1996 blood samples collected from six of the 2,4,5-trichlorophenoxyacetic (2,4,5-T) workers in Russia. The elimination rate constants of four of the six samples ranged from 6.9 to 17 years (6.9, 9.7, 9.7, and 17, respectively), while those of two of the six samples were incalculable. Ryan et al. (1997) stated that these four values were in the range reported by other investigators. However, no supporting references were provided. Due to the large variability of values, the small sample sizes (one single value for each sample), and a potential inconsistency in sample analysis (samples were analyzed by two different laboratories at two different times), there is uncertainty in these values. Therefore, these values require further consideration.

Based on available data, the elimination rate constant (i.e., half-life) for 2,3,7,8-TCDD was assumed to be about 7.1 years, and the fat volume was assumed to be 17.5 kg (i.e., 70 kg body weight * 0.25 fat) which yielded a background TCDD dose of about 32 pg/day using the TCDD tissue estimate from the 1980s to mid 1990s (5.5 ppt), and 12 pg/day (0.18 pg/kg/day) using the TCDD tissue concentration from the CDC data set (2.1 ppt). These estimates agree well with the background exposure estimates (to 2,3,7,8-TCDD only) of 35 pg/day by Travis and Hattemer-Frey (1991) and 25 pg/day by Fürst et al. (1991), but are somewhat higher than the current background exposure estimate of 5.6 pg/day from this assessment (see Section 4.4.2), as derived using typical media levels and contact rates. Using the current CDD/CDF TEQ body burden data presented in Table 4-18 and the pharmacokinetic model presented in Equation 4-4, the average daily intake of total CDD/CDFs is estimated to be 126 pg TEQ_{DF}-WHO_{98}/day. This estimate assumes a half-life of TEQ_{DF}-WHO_{98}s in the body of 7.1 years, a fat volume of 17.5 kg, a concentration in the body fat of 21.6 ppt (i.e., the approximate mean TEQ concentration for CDD/CDFs only, as calculated from the data in Table 4-18), and steady-state conditions. This value is also three times higher than the current background exposure estimate of 41 pg TEQ_{DF}-WHO_{98}/day from this assessment, as derived using typical media levels and contact rates. If PCBs are included in this exercise (i.e., using the current TEQ_{DFP}-WHO_{98} background tissue concentration of 25.4 ppt) the estimated TEQ_{DFP}-WHO_{98} dose would be 146 pg/day. This estimate is approximately 2.2 times higher than

the direct estimate from the dietary data of 65 pg TEQ_{DFP}-WHO_{98}/day. Because this model was originally developed for use with 2,3,7,8-TCDD, the effect of using it to model CDD/CDFs introduces uncertainty into these estimated values.

An important uncertainty in the modeling exercise described above was the assumption that the half-life estimate for 2,3,7,8-TCDD (7.1 yr) would apply to TEQ_{DF}-WHO_{98}s. Thus, the same pharmacokinetic model was applied to average human tissue levels for each congener, using half-lives that are specific to each congener, and then summing the estimated intakes for each congener. This approach yielded an estimated intake of 87 pg TEQ_{DF}-WHO_{98}/day (Table 4-24). This value is approximately 2 times higher that the current background estimate of 41 pg TEQ_{DF}-WHO_{98}/day.

Another, perhaps more important, uncertainty in using this approach to estimate current dose is that the dose is assumed to be constant over time. If, in fact, the dose which has resulted in current average body burden were constant over the past several decades, than use of this steady-state PK model would provide quite reasonable estimates of current dose. The only uncertainty in this case (beside the simplistic nature of it being a one-compartment PK model) is the use of 7.1 years as the half-life (as described above). If the dose regime instead was characterized by very low doses in the middle of the twentieth century only to rise significantly in the latter part of the century, than this model would, by definition, provide an underestimate of current dose. If, on the other hand, doses were very much higher in the mid-portions of the twentieth century only to drop towards the end of the century, than this steady state model would, by definition again, provide an overestimate of the current dose. The steady-state model only provides an average dose over time that could account for a given body burden - it obviously doesn't address the possibility of changes in dose over time. As will be described in the next section, there is a very large amount of evidence suggesting that doses were higher in the mid-decades of the twentieth century, and may be significantly higher, as compared to the latter decades. The tissue levels representing the "current average body burdens" included a significant number of individuals living in this middle decades of the twentieth century. This being the case, it is concluded that the steady state approach will overestimate current dose.

4.3.2. Non-Steady State Approach

Chapter 6 describes evidence supporting temporal trends in CDD/CDF/PCB concentrations in environmental media, foods, and associated doses. It appears that the levels of dioxin-like compounds have increased in the environment starting from the 1930s through the 1960s, and loadings began to decline perhaps starting in the 1970s to the present. Recent evidence collected on animal food products in the United States (Winters, et al., 1998), combined with body burden data, provide evidence that human exposures to dioxins may have followed the same trends. (See Chapter 6.)

Pinsky and Lorber (1998) used a non-steady state approach to reconstruct the pattern of past exposure and estimate current exposure to 2,3,7,8-TCDD, using a simple pharmacokinetic model that included a time-varying TCDD dose. A first order, one-compartment PK model was used to compute an individual's body lipids TCDD concentration through time. Key inputs for that model include: (1) a time-varying dose of TCDD (expressed in units of pg/kg-day), (2) a fraction of dose absorbed into the body lipid compartment (assumed to be constant), (3) the volume of the body lipid compartment (assumed to be time varying), and (4) a rate of TCDD loss from the lipid compartment (modeled as a function of the percent of body fat). In order to calculate the rate of TCDD loss, a model of how body lipid volumes vary over time, in addition to a model of how overall body weight varied over time, was required.

In this modeling exercise, all inputs were fixed, except the time-varying dose of TCDD. Using Bayesian statistical approaches, the non-steady state dose was "calibrated" to best-fit a set of data on TCDD concentration in body lipids from the 1970s to the 1990s. The results of this exercise indicated that the dose appears to have increased from the 1940s through the 1960s, and began to drop through the 1970s, with a baseline level being reached by the 1980s. The results suggest that TCDD exposures may have been 20 times higher during the 1960s than the 1980s. Over a 10-year peak period in the 1960s and early 1970s, daily exposures could have been as high as 1.5 to 2.0 pg/kg-day, possibly dropping as low as 0.10 pg/kg-day (7 pg/day) and less into the 1980s. This estimate of current dose of 7 pg 2,3,7,8-TCDD/day is quite similar to the estimate of 6.1 pg 2,3,7,8-TCDD/day made using typical media levels and contact rates. In another test, Pinsky and Lorber (1998) used the same modeling structure to test the steady state assumption by forcing the dose to be constant over time. In that test, Pinsky

and Lorber (1998) solved for a 'best-fit' dose of 0.35 pg/kg-day. This is higher than the 1980s calibrated 'current dose' of 0.10 pg/kg-day, derived by allowing the dose to vary over time. As described in the previous section, if much higher doses of dioxin occurred in the middle part of the twentieth century, than a steady state model will provide an overestimate of current dose; in other words, this Pinsky and Lorber (1998) result is to be expected. In addition, the steady-dose 'best-fit' solution provided a significantly poorer fit to the data as compared to the non-steady dose solution, providing even more evidence that doses have not been steady during the twentieth century. (See Chapter 6 for a complete description of this modeling approach.)

4.4. INTAKE ESTIMATES BASED ON EXPOSURE MODELING

4.4.1. Previous Assessments of Background Exposures

Several researchers have published quantitative assessments of human exposures to CDDs and CDFs. Some of the more recent assessments are discussed below (Travis and Hattemer-Frey, 1991; Fürst et al.,1990; Fürst et al., 1991; Henry et al., 1992; Theelen, 1991; Schuhmacher et al., 1997; Gilman and Newhook, 1991; Schrey et al., 1995; MAFF, 1995; and Jacobs and Mobbs, 1997; Himberg, 1993; and Liem et al. 2000a, 2000b). It is generally concluded by these researchers that dietary intake is the primary pathway of human exposure to CDDs and CDFs. Over 90 percent of human exposure occur through the diet, with foods from animal origins being the predominant sources.

Travis and Hattemer-Frey (1991) estimated that the average daily intake of 2,3,7,8-TCDD by the general population of the United States is 34.8 pg/day. Ingestion exposures were estimated by multiplying the concentration of 2,3,7,8-TCDD in beef, milk, produce, fish, eggs, and water (estimated using the Fugacity Food Chain model) times the average U.S. adult consumption values for these products reported by Yang and Nelson (1986). The calculations assume that 100 percent of the 2,3,7,8-TCDD ingested are absorbed through the gut. Intake via inhalation was estimated by multiplying the concentration in air times the amount of air inhaled per day (20 m^3) assuming that 100 percent of inhaled 2,3,7,8-TCDD are absorbed through the lung. The results of their assessment, summarized in Table 4-25, indicate that foods from animal origins comprise

95 percent of the estimated total daily exposure. These foods include milk and dairy products, beef, fish, and eggs. Exposure resulting from consumption of vegetables and other produce was estimated to account for 3.4 percent of the total intake. Exposure from ingestion of water, ingestion of soil, and inhalation of air together accounted for about 1 percent of the total daily intake.

Fürst et al. (1990) estimated human exposure to CDD/CDFs based on the analysis of 107 food samples collected in the Federal Republic of Germany. The average daily I-TEQ_{DF} intake was estimated to be 85 pg/person/day or 1.2 pg/kg body weight/day. Fürst et al. (1990) concluded that foods of animal origin contribute significantly to the human body burden of CDD/CDFs. In a subsequent study, Fürst et al. (1991) assessed human exposure to CDDs and CDFs from foods using data from more than 300 randomly selected food samples and food consumption data reflective of consumption habits of the German population. These authors estimated that the German population's average daily intake of CDDs and CDFs from food is 158 pg I-TEQ_{DF} per person of which 25 pg is 2,3,7,8-TCDD. Dairy products, meat and meat products (primarily beef), and fish and fish products each contribute about 32 to 36 percent of the daily intake of I-TEQ_{DF}. Based on the levels of CDD/CDFs observed in human samples, the average daily intake via food was estimated to be in the range of 1 to 3 pg I-TEQ_{DF}/kg body weight.

Henry et al. (1992) of the U.S. Food and Drug Administration estimated the average exposure to the U.S. population from 2,3,7,8-TCDD through the food supply using the following assumptions: (1) all dairy products have background lipid 2,3,7,8-TCDD levels equivalent to those found in milk and half-and-half, i.e., about 55 ppq (whole dairy food levels were estimated using percent fat in each food); (2) levels averaging 35 ppq in beef tissue are present in all meat products; (3) ocean fish with tissue levels equal to half of the detection limit (about 0.5 ppt) are the sole fish source in the diet; (4) average food consumption figures (total-sample-basis) available from nationally representative data bases were used for frequency of eating (Market Research Corporation of America's (MRCA) Menu Census VI (1977-78)) and for serving sizes (U.S. Department of Agriculture's 1977-78 National Food Consumption Survey). The concentration assumptions used in the Henry et al. (1992) study were based on previously published data. For example, most of the food data were based on La Fleur et al. (1990), and the fish data were based on U.S. EPA (1992). These studies are described in Sections 3.7.2

and 3.6.1, respectively. FDA's estimates of 2,3,7,8-TCDD intake were derived by multiplying the food dioxin levels by the average amounts of food consumed per day. The results of the FDA assessment, summarized in Table 4-26, indicate an average daily exposure of 15.9 pg/day of 2,3,7,8-TCDD of which 4 percent are due to dairy and milk products, 41 percent are due to meats, and 54 percent are due to ocean fish.

Theelen (1991), of The Netherlands National Institute of Public Health and Environmental Protection, estimated the average daily intake of 2,3,7,8-TCDD and total I-TEQ_{DF} by residents of The Netherlands for various possible routes of exposure. The results, summarized in Table 4-27, indicate an average intake of 20 pg/day of 2,3,7,8-TCDD and 115 pg/day of total I-TEQ_{DF} from food and 0.08 pg/day (2,3,7,8-TCDD) and 3.2 pg/day (I-TEQ_{DF}) from combined direct air and soil exposure. Milk and dairy products make up about one-third of the total daily exposure. Animal fat in meat, poultry, and fish (i.e., fish oil) also contribute about one-third. Fish consumption represents 18.5 percent of total daily exposure. In a later study, Theelen et al. (1993) reported a median daily intake for adults of 1 pg I-TEQ_{DF}/kg body weight, and a 95th percentile rate of 2 pg I-TEQ_{DF}/kg body weight. These values were based on CDD/CDF residue levels in food products and food consumption survey data.

Becher et al. (1998) estimated dietary intake of CDD/CDFs and dioxin-like PCBs in the Norwegian population. Average food consumption data obtained from the 1992-1994 Norwegian consumer survey of 4,033 households was analyzed in conjunction with measured CDD/CDF and dioxin-like PCB concentrations in basic foodstuffs to determine dietary intake. Becher et al. (1998) investigated pooled samples from 20 to 25 seafood samples and 10 to 15 samples of other foodstuffs. Average CDD/CDF dietary intake ranged from 71 to 85 pg I-TEQ_{DF}/day and average PCB dietary intake ranged from 86 to 106 pg TEQ_{P}-WHO_{94}/day. Fish and fish products constituted the largest contribution to the dietary intake of CDD/CDFs and PCBs. PCBs contributed more to the total dioxin related toxicity (i.e.,TEQ_{DFP}-WHO_{98}) than CDD/CDFs in the following food groups: milk, meat, eggs, and cod liver oil; while CDD/CDFs were the higher contributor in the fats food group.

Buckland et al. (1998) estimated dietary intake of CDD/CDFs and PCBs in the population of New Zealand. The estimate was based on 19 food group composites from 51 individual food samples purchased from retail outlets in four major cities and one

provincial center. Estimated dietary intake was calculated based on two typical diets, an average exposure diet of an adult male and a high-end exposure diet of an adolescent male. Total dietary levels of all CDD/CDF congeners ranged from 14.5 to 30.6 pg I-TEQ_{DF}/day (whole weight). Total dietary PCB levels ranged from 12.2 to 22.7 pg TEQ_P-WHO_{94} (whole weight). Total PCB concentration was based on levels of the following PCBs: 28, 31, 52, 77, 101, 99, 123, 118, 114, 105, 126, 153, 138, 167, 156, 157, 169, 187, 183, 180, 170, 189, 202, 194, and 206. These calculations were made by setting concentrations less than the LOD at one-half the LOD. Vegetable fats/oils, cereals, cooked potatoes and hot chips, and processed meats constituted the largest contribution to I-TEQ_{DF}s in the adolescent diet. Butter, processed meats, and milk constituted the largest contribution of TEQ_P-WHO_{94}s to the adolescent diet. The authors noted that while it is difficult to compare these total dietary TEQ results to countries with different dietary patterns, the results appear to indicate that estimated dietary intake of CDD/CDFs and PCBs is lower in New Zealand than in other countries that have conducted similar studies (e.g., USA, UK, Spain, The Netherlands, Federal Republic of Germany, and Norway).

Schuhmacher et al. (1997) and Domingo et al. (1999) estimated dietary intake of CDD/CDFs based on the analysis of 35 food samples from local supermarkets in Catalonia, Spain. Most of the results are in agreement with the recent data reported elsewhere; however, the levels in whole milk, vegetables, lentils and beans, and cereals are higher than those reported in previous studies. The average intake per adult was estimated as 210 pg I-TEQ_{DF}/day. The contributions from vegetables and cereals were relatively high (8.13 percent and 23.09 percent, respectively, of total intake) compared to previous studies where the vegetable and cereal contributions are almost negligible. The high contributions may be explained by high consumption of these foods in the Mediterranean diet. Schuhmacher et al. (1997) stated that since the Mediterranean diet is typical throughout most Spanish regions, the results reported could be a representative of the dietary intake of CDD/CDFs in Spain.

Gilman and Newhook (1991), of the Canadian Department of National Health and Welfare and the Ontario Ministry of the Environment, respectively, estimated an average lifetime daily intake of 140 to 290 pg of I-TEQ_{DF} for the typical Canadian. Their results, summarized in Table 4-28, indicate that between 94 and 96 percent of the estimated

intake are from food sources. No breakdown of intake by food type was provided in the report.

Schrey et al. (1995) estimated dietary intake of CDD/CDFs using the duplicate method. A total of 14 food samples that were duplicates of the food eaten by seven German men and seven German women (age 24-64 years) were collected and analyzed for CDD/CDFs. The 3-day sampling period included both weekdays and weekends. All samples contained detectable levels of 2,3,7,8-substituted CDD/CDFs, but OCDD had the highest concentrations. Daily intake was estimated to range from 3.3 to 14 pg/day (0.026 to 0.26 pg/kg-day) for 2,3,7,8-TCDD and 23 to 96 pg/day (0.18 to 1.7 pg/kg-day) for I-TEQ_{DF}s. These values are slightly lower than those observed in earlier German studies conducted by Beck et al. (1991), even though the dietary intake of fat was similar.

Recently, the United Kingdom's Ministry of Agriculture, Fisheries, and Food (MAFF, 1995) analyzed Total Diet Study samples collected during 1982 and 1992 for CDD/CDFs to analyze trends in dioxin intake over recent years. Samples of 11 food groups collected from 24 locations in the United Kingdom were analyzed. The average intake of dioxins from each food group was calculated by multiplying the CDD/CDF residue concentration in the food group by the average daily intake of the food based on data from the United Kingdom's National Food Survey. Average daily intake of I-TEQ_{DF}s was estimated to be 240 pg/day in 1982 and 69 pg/day in 1992 (Table 4-29). These values represent upper bound exposures because I-TEQ_{DF}s were calculated by setting nondetects to the limit of detection. Based on these results, the authors concluded that the relative contributions of the various food groups to total dioxin intake in the United Kingdom have changed over the years. In the most recent study, the proportion of total exposure attributable to cereal products increased, while exposures from fats, oils, and milk products decreased.

Jacobs and Mobbs (1997) conducted a reassessment of human dietary exposure to CDD/CDFs in the UK. Based on the data of the UK Total Diet Survey (TDS) in 1992, the levels of CDD/CDFs in 11 fat-containing food groups were recalculated. Instead of using the food consumption data from the UK's National Food Survey as in the MAFF study, Jacobs and Mobbs (1997) obtained individual dietary intake data from three other surveys that included adults, children (aged 1.5 to 4.5 years), and infants (aged 6 to 12 months) in the UK. Combining the dietary intake data with the data of CDD/CDF levels in foods, Jacobs and Mobbs (1997) reported an adult daily dietary intake of CDD/CDF as 175.5 pg

I-TEQ_{DF}/day (2.93 pg TEQ/kg/day), a value that is more than twice that estimated by MAFF (1995). The levels for young children ranged from 54.19 pg I-TEQ_{DF}/kg/day at 6 months of age, to 0.25 pg TEQ_{DF}/kg/day at 4.5 years of age. It should be noted that for infants (under 1 year of age), breast milk is the largest contributing source. Estimation of the cumulative dietary I-TEQ_{DF} intake indicated that the levels peak sharply between age 0 and 1 year at about 80 pg I-TEQ_{DF}/kg/day, decrease until 10 years of age, and then rise to about 15 pg TEQDF/kg/day at 22 years of age.

Dioxin-like PCBs can also contribute to TEQ exposures. Himberg (1993) evaluated exposures to dioxin-like PCBs 77, 15, 126, and 169 in Finnish foods. Based on fish, beef, pork, poultry, and inner organs, total TEQ_P-WHO_{94} intake was estimated to be 118 pg/day, calculated using TEQ_P-WHO_{94} concentrations in foods and consumption data from Finland's 1990 household survey. PCB congeners 105 and 126 contributed the most to total TEQ_P-WHO_{94} intake. Intake of PCBs in fish products accounted for the greatest proportion (i.e., approximately 70 percent) of the total TEQ_P-WHO_{94} intake from these foods.

Currado and Harrad (1997) measured air concentrations of PCBs from 9 different indoor environments, including two laboratories, two offices, and five residential houses in the United Kingdom (UK). The results indicated that the total PCB levels found in indoor air (1.4 to 19.1 ng/m^3, mean = 7.1 ng/m^3) were between 2 and 19 times higher than the levels in outdoor air (0.77 to 0.87 ng/m^3, mean = 0.82 ng/m^3). Currado and Harrad (1997) also calculated the daily human intake of PCBs via inhalation. The estimate ranged from 36.9 to 176.5 ng/person/day (mean = 103.5 ng/person/day), and represented between 10 and 33 percent of overall exposure to PCBs for a typical UK individual with a 340 ng/day dietary intake of PCBs (estimated by the UK Ministry for Agriculture, Fisheries and Food (MAFF) in 1992). Currado and Harrad (1997) suggested that, compared to the dietary intake of 340 ng/person/day of PCBs, inhalation of indoor air might be a significant pathway for PCB exposure. It should be noted that the study did not focus on dioxin-like PCBs; only concentrations of four dioxin-like PCB congeners were reported for indoor and outdoor areas.

Liem et al. (2000a) reported 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, including Belgium, Denmark,

Finland, France, Germany, Italy, The Netherlands, Norway, Sweden, and the United Kingdom, delivered available data on the occurrence of PCDDs, PCDFs, and dioxin-like PCBs in food products and human milk. When available, these countries also delivered data on the consumption of these foods and other data on the dietary exposures of the general populations of these countries. Consumption data were combined with concentration data to arrive at exposure doses in pg TEQ_{DFP}-WHO_{94}. Concentrations and doses expressed in terms of the more recent WHO 1998 TEF scheme are generally higher than these earlier TEF schemes, by about 5-10%, in American food and environmental media. Some countries also provided consumption data representative of the 95th, or 97.5, percentile of the population. These consumption data were combined with mean concentration data from the countries to evaluate higher end exposures of the general population. Liem et al. (2000) concluded that data were reasonably available for dioxins and furans, but limited for the dioxin-like PCBs.

Based on the short summary of this effort in Liem et al. (2000a), it appears that trends in European CDD/F food concentrations and exposures are consistent with those from the United States, although dioxin-like PCB concentrations may be somewhat higher in Europe. National average concentrations of CDD/Fs in eggs, fats and oils, meat products, and milk products are generally less than 1 up to 2-3 pg/g fat, I-TEQ_{DF} basis. Concentrations in fruits, vegetables and cereals were found to be generally close to the limits of detection. Some of the data suggested reductions in concentrations over time, but the available information was insufficient to draw general conclusions. Limited data on dioxin-like PCBs suggest average TEQ_P-WHO_{94} that are between 1 and 2 times higher than I-TEQ_{DF} concentrations in all food products, more so in fish. PCB TEQ concentrations in fish were between 0.25 and 10-20 pg/g fat WHO_{94}-TEQ_P. In contrast, in the United States, TEQ concentrations of dioxin-like PCBs are roughly comparable, if not lower, than TEQ concentrations of CDD/Fs in foods of terrestrial origin Some data on dioxin-like PCBs in fish suggest higher concentrations than CDD/Fs, particularly from a recent study of several fish species from the Great Lakes (Kolic et al., 2000a).

Given the limitations of the available data, Liem et al. (2000a) reported that for eight countries and for the period after 1995, the average adult dietary intakes of CDD/Fs ranged between 29 and 97 pg I-TEQ/day. This compares well to the estimate of 45 pg TEQ_{DF}-WHO_{98}/day developed in this assessment. The upper percentiles estimates of

dietary exposures, where 95 and 97.5% of consumption rates were combined with average concentrations, was 2-3 times the mean intake. This analysis was only available from data on consumption supplied by the Netherlands and the United Kingdom. This also compares well to evaluations done in this assessment based on dietary fat (and total diet) intake, which suggest that the TEQ intakes at the 95th (2 standard deviations above the mean) and 99th (3 standard deviations above the mean) percentiles would be 2 and 3 times the mean intakes, respectively.

Their limited data on dioxin-like PCBs suggest perhaps more of an impact than developed in this assessment. Their average daily intake estimates ranged between 48 and 110 pg TEQ_P-WHO_{94}, compared to the 25 pg TEQ_P-WHO_{94}/day calculated for the US in this evaluation. For countries where data were available for both CDD/Fs and PCBs, the dioxin-like PCBs contributed between a roughly equal amount (Finland, Netherlands, Sweden, United Kingdom) to approximately 4 times (Norway) the TEQ contributions of the CDD/Fs.

Other findings of interest include:

1) Based on concentrations in foods taken in the 1970s and 1980s, calculated doses were much higher, ranging from 127 to 314 pg I-TEQ/day.

2) Similar to findings in this assessment, the highest dietary contributions were made by milk and dairy products (between 16 and 39% of total TEQ intakes), meat and meat products (6-32%), and fish and fish products (2-63%).

3) The intake to breast-fed children was estimated to be between 1 and 2 orders of magnitude higher than adults, on a body weight basis. This is similar to the finding in this assessment that the average infant dose of a year's worth of breast-feeding would be about 77 pg TEQ_{DFP}-WHO_{98}/kg-day.

According to Liem et al. (2000b), "Countries that started to implement measures to reduce dioxin emissions in the late 1980s, such as The Netherlands, United Kingdom, and Germany, clearly show decreasing PCDD/PCDF and PCB levels in food and consequently a

significantly lower dietary intake of these compounds by almost a factor of 2 with the past 7 years".

As reported in Section 3.7.1, CDD/CDFs can migrate from bleached paper packaging and paper food-contact articles to foods. Some investigators have included this pathway in estimates of background exposure. U.S. EPA (1990) estimated that I-TEQ_{DF} intake due to leaching from paper products into food from paper packaging was in the range of 5.5 to 12.7 pg/d. Henry et al. (1992) estimated that daily intake of 2,3,7,8-TCDD due to migration from paper to food could amount to 12 pg/d, almost as much as the daily intake from unaffected food of 16 pg/d. (See Table 4-26.) As shown in Table 4-27, Theelen (1991) estimated that out of a total of about 120 pg of I-TEQ_{DF}/d, 9 pg of I-TEQ_{DF}/d could be due to migration from paper. These estimates are based on levels in paper before recent changes in industry practices that are expected to substantially reduce dioxin levels in paper. As discussed in Section 3.7.1, these reductions are expected to have significantly lowered the CDD/CDF levels currently found in food due to any leaching of dioxin-like compounds from paper.

Horstmann and McLachlan (1994) measured CDD/CDF levels in human skin using an adhesive tape stripping method. Skin samples of the stratum corneum were collected from the backs of eight volunteers of varying age and sex. Two additional layers of increasing depth were collected from five people. All showed a decrease in CDD/CDF levels with depth. The concentration in the first layer ranged from 1,000 to 7,800 pg/g on a total CDD/CDF basis. The second layer was an average of 43 percent lower, and the third layer was an average of 33 percent lower. OCDD was the dominant congener in all three layers. Also, non-2,3,7,8 substituted congeners were identified, congeners which are not normally present in human tissue. In addition, samples of the epidermis and subcutis were analyzed. These analyses indicated that levels of the non-2,3,7,8 substituted congeners were much higher in the stratum corneum than in the epidermis, and none were identified in the subcutis. The authors argue that because these congeners could not be transported from inside the body to the stratum corneum, the CDD/CDF in the stratum corneum must originate from external sources. Horstmann and McLachlan (1994) hypothesized that textiles could be the source of skin contamination. Thirty-five new textiles, primarily cotton products, were analyzed and found to have a total CDD/CDF

level that was generally less than 50 ng/kg; however, several colored T-shirts had high levels, with concentration up to 290,000 pg/g. The homolog patterns in the textiles were similar to the patterns found in the skin. Experiments were then conducted measuring the CDD/CDF levels in human skin before and after wearing T-shirts. Significant increases in CDD/CDF levels in the skin occurred after wearing the highly contaminated shirts for 1-2 weeks, and significant decreases in CDD/CDF levels in the skin occurred after wearing the uncontaminated shirts for 1-2 weeks. This work strongly suggests that dermal exposure to textiles may be contributing to background exposures to CDD/CDFs. Horstmann and McLachlan (1994) comment that although the levels of most CDD/CDF congeners in humans can be explained on the basis of diet, the origins of OCDD in humans is less clear. Because OCDD was found to be the dominant congener in textiles and skin, they speculate that the human body burden of this congener may result from dermal absorption. Horstman and McLachlan (1994) further discuss that human scale (stratum corneum) contributes to house dust and could lead to exposure via inhalation.

Klasmeier et al. (1999) further studied the transfer of CDD/CDFs from textiles to human skin. Spatial variability, variability among individuals, and the percent transfer from different cotton textiles was examined. Spatial variability in transfer to the skin was measured by placing 7 and 10 cm^2 patches of contaminated and uncontaminated (for background determination) textiles on the upper back of human volunteers for 8 hours. The four samples collected from the outermost layers of the skin of the back of 12 volunteers contained similar concentrations of all detected congeners. The results indicated that the skin surface properties determining the transfer of CDD/CDFs from cotton textiles to the stratum corneum of the human back did not vary. An additional volunteer wore a similarly contaminated cotton t-shirt for 72 hours. The mean percent transfer for the 72 hour exposure was 1.6 to 2.5 times higher than for the 8-hour exposure.

Matsueda et al. (1995) measured CDD/CDFs and PCBs in skin lipids from the faces of eight Japanese men between the ages of 21 and 73 years. Skin lipids were collected in the morning before washing the face, using facial wipes containing 70 percent alcohol. The I-TEQ_{DF} concentrations in the samples ranged from 8.8 ppt to 22.3 ppt with a mean of 15.3 ppt. I-TEQ_{P}-WHO_{94} concentrations ranged from 7.3 ppt to 22.5 ppt. Matsueda et

al. (1995) also collected serum samples from these same subjects. Blood I-TEQ_{DF}s ranged from 13.5 ppt to 36.5 ppt, and TEQ_{P}-WHO_{94} ranged from 7.1 ppt to 22.7 ppt.

4.4.2. Updated Assessment of Background Exposures on the Basis of Media Levels and Contact Rates

Background exposures to CDD/CDFs and dioxin-like PCBs in North America were estimated using: (1) the arithmetic mean TEQ_{DFP}-WHO_{98} levels in environmental media and food from Table 3-61; (2) the standard contact rates for ingestion of soil, water, and food, and inhalation of ambient air; and (3) the appropriate unit conversion factors. The general equation used to estimate background exposures is as follows:

$$\text{Intake (pg WHO98-TEQ/kg-day)} = \frac{\text{Daily Contact Rate x Concentration x Unit Conversion Factors}}{\text{Body Weight}} \qquad \text{(Eqn. 4-5)}$$

where:

Contact Rate = inhalation or ingestion rate (m^3/day, mg/day, L/day, or g/day); and

Concentration = residue level in media of concern (pg/m^3, ppt, or ppq).

These background exposure estimates represent administered doses and not absorbed doses.

The estimated exposures and assumptions made for adults concerning ingestion or contact rates are presented in Table 4-30 for CDD/CDFs and Table 4-31 for PCBs. Standard intake rates representative of the adult general population were used. The background exposure estimates reported here do not account for individuals with higher consumption rates of a specific food group (e.g., subsistence fishermen, cigarette smokers, and individuals with exposures from localized impacts--these are discussed in Chapter 5). The estimates are assumed to represent typical (i.e., "central tendency") U.S. background exposures, and do not account for these types of variations in the population as a result of differences in intake rates of the various food groups. Average contact rates for ingestion of soil, water, beef, pork, poultry, other meats, and eggs, and inhalation were derived from the revised Exposure Factors Handbook (U.S. EPA, 1997). The intake

rate for other meats represents total meat intake minus the intake rates for beef, pork, and poultry. Other meats could include lamb, game, etc. It should be noted that the concentration of dioxin-like compounds in other meats was assumed to be similar to that observed in beef, pork, and poultry, because data were not available for these other meats. Thus, the TEQ_{DFP}-WHO_{98} for other meats was estimated as the average of TEQ_{DFP}-WHO_{98} concentrations for beef, pork, and poultry. Mean fish ingestion rates were derived from U.S. EPA (2000). Contact rates for milk, dairy, and vegetable fats were derived from USDA (1995). The contact rate for dermal contact with soil was calculated as the skin surface area that contacts the soil (cm^2/day) x the soil adherence rate (mg/cm^2) x the dermal absorption fraction for CDD/CDFs (0.03) (U.S. EPA, 1999). The age-specific surface areas and adherence factors were based on data and estimation methods recommended in U.S. EPA (1997) and U.S. EPA (1999) for adult and child residents. The soil ingestion rates used here are those recommended by U.S. EPA (1997). Soil ingestion occurs commonly among children during activities such as mouthing of toys and other objects, nonsanitary eating habits, and inadvertent hand-to-mouth transfers. In addition to normal soil ingestion activities, some individuals exhibit behavior known as pica which involves intentional soil ingestion. Soil ingestion rates associated with pica are probably much higher. Some limited data suggest rates as high as 5 to 10 g/day for deliberate soil ingestion rates for pica children. The current Exposure Factors Handbook (U.S. EPA, 1997) suggests a central tendency value for non-pica children of 100 mg/day. To a lesser extent, soil ingestion also occurs among adults from activities such as hand-to-mouth transfer when eating sandwiches or smoking, and other inadvertent ingestion of soil, such as that in household dust. Data on soil ingestion are even more scarce for adults. Based on limited data, a central tendency value of 50 mg/day is suggested by U.S. EPA (1997), which is used here.

It should be noted that the contact rates used in this assessment for some food products (e.g., meats) are lower than those used in an earlier 1994 draft (U.S. EPA, 1994) of this document. The values in the earlier draft were based on the average of food disappearance rates and intake rates. The intake rates were 1-day diary data derived from the 1987/1988 USDA National Food Consumption Survey (NFCS) (USDA, 1995). This type of survey is considered to be the best indicator of food consumption patterns, and statistical designs used by USDA optimized the ability to correctly account for factors

such as seasonality, geography, age of recipients, and other factors. The intake data derived from the 1987/1988 USDA NFCS in the 1994 draft used assumptions to allocate meat mixtures among the various meat groups. These assumptions were required because the meat consumption rates available at the time did not account for meats consumed as mixtures. These assumptions over-estimated intake for the various individual meat groups because it was assumed that intake rates for the NFCS meat mixture category included intake of foods made up of mixed meat items only. For example, it was assumed that meat mixtures were made up of 40 percent beef, 17 percent pork, 32 percent poultry, and 11 percent fish. However, meat mixtures actually included food items that had dietary components (i.e., grains, vegetables, etc.) other than meats. Therefore, the individual meats accounted for a much smaller fraction of the mixtures than assumed in 1994. "Disappearance rates" are derived as the total amount of food that disappears (i.e., is used) from the U.S. commercial food supply divided by the number of people in the U.S., corrected for removal of bone and fat, food that goes into pet foods, and food that is imported (USDA, 1993). These rates are expected to overestimate average daily intakes because they do not account for uneaten portions, spoilage, or waste. In 1994, EPA used the USDA's report on Food Consumption, Prices, and Expenditures between 1970 and 1992 (USDA, 1993) to derive disappearance rates for each food type.

The intake data used in this current assessment are derived from a newer set of USDA intake data. EPA recently conducted a statistical analysis of the USDA food data from the 1989-1991 Continuing Survey of Food Intake among Individuals (CSFII) for inclusion in the Exposure Factors Handbook (U.S. EPA, 1997). The USDA CSFII is a 3-day survey that provides national data on the amount of food eaten by individuals over the survey period. During 1989 through 1991, over 15,000 individuals participated in the CSFII (USDA, 1995). Using a stratified sampling technique, individuals of all ages living in selected households in the 48 coterminous states and Washington, D.C., were surveyed. Individuals provided 3 consecutive days of data, including a personal interview on the first day followed by 2-day dietary records. The survey uses a statistical sampling technique designed to ensure that all seasons, geographic regions of the U.S., and demographic and sociodemographic groups are represented (USDA, 1995). EPA's analysis of the CSFII data tabulated intake rates for the major food groups, as well as individual food items. The

analysis allocated intake of meat mixtures among the various meat groups and other applicable food groups according to the percentages provided by USDA (1995), as described in U.S. EPA (1997). For example, according to USDA (1995), meat mixtures contained 20 percent beef, 2 percent pork, and 8 percent poultry. Intake of other food groups (i.e., grains, vegetables, etc.) accounted for the balance of meat mixture intake. These meat mixture fractions are considerably lower than those assumed for the 1994 draft.

As an example of the difference in the 1994 and the current food consumption rates, the 1994 pork consumption rate, 47 g/day, was derived as the average of the disappearance rate of 62 g/day and the intake rate of 32 g/day. (The intake rate of 32 g/day was estimated as the intake rate for pork of 14 g/day plus an assumed 17 percent of meat mixtures.) The resulting value, 47 g/day, is higher than the intake rate used in this current draft, 0.22 g/kg-day, or approximately 15 g/day assuming a 70 kg adult. Other differences are also significant: 77 g/day beef (1994) versus 50 g/day (currently), 68 g/day poultry (1994) versus 35 g/day, and 67/251 dairy/milk g/day (1994) vs. 55/175 g/day (currently). Also, contact rates for some of the other media are lower (e.g., soil), based on the revised Exposure Factors Handbook (U.S. EPA, 1997).

Another reason that current estimates of exposure are lower than in the 1994 document is that the estimated TEQ_{DF}-WHO_{98} concentrations for several food items (i.e., beef, pork, poultry, milk, and dairy) are also lower in this assessment than in the earlier (1994) draft. Estimates in the earlier draft were based on limited data sets for these foods, whereas the current assessment uses data from the more recent statistically-based national analyses of several food categories, as described in Chapter 3. Some of the older studies had nondetectable congener concentrations and higher detection limits than the newer studies, resulting in higher TEQ concentrations. For example, the beef concentration assumed in the 1994 assessment was 0.48 pg I-TEQ/g whole weight basis, while the current estimate is 0.18 pg TEQ_{DF}-WHO_{98}/g whole; poultry was 0.19 pg I-TEQ/g, while here it is 0.068 pg TEQ_{DF}-WHO_{98}/g. Table 4-32 compares the contact rates, TEQ concentrations, and background exposure estimates from the 1994 draft and this assessment. It should be noted that the previous draft estimated I-TEQ_{DF}s, while TEQ_{DF}-WHO_{98}s are used in the current assessment.

Background exposure levels are also presented for Germany, based on data from Fürst et al. (1990; 1991). The current total background TEQ_{DF}-WHO_{98} exposure shown in Table 4-33 is approximately 43 pg/day for North America. Based on Fürst et al. (1990; 1991), the estimated total CDD/CDF I-TEQ background exposure from food consumption for Germany is 79 pg/day (Table 4-33). However, it should be noted that the estimated background level for the United States and Germany are based on limited data, and exposure to all food groups was not considered. Also, the addition of TEQs for multiple pathways presumes that individuals are exposed by all pathways, and assumes that the fraction absorbed into the body is the same for all ingestion and inhalation pathways (i.e., 100 percent absorption in the gut and lungs is assumed). The dermal absorption pathway assumes that 3 percent of the CDD/CDFs in soil that adheres to the skin surface is dermally absorbed. The following sections present observations about CDD/CDF exposures in North America, comparisons between exposure estimates from this and previous studies, and comparisons between North American and European exposures to CDD/CDFs.

Based on the data presented in this report, the adult general population total background TEQ_{DF}-WHO_{98} exposure for North America was estimated to be 0.61 pg/kg-day (or 43 pg/day assuming a 70 kg adult), for all media combined. Exposure to 2,3,7,8-TCDD accounts for approximately 13 percent (5.5 pg/day) of the total TEQ exposure. Estimated exposures based on total TEQ_{DF}-WHO_{98} from the various exposure pathways are presented in Figure 4-4. The highest exposures were estimated to occur via ingestion of CDD/CDFs in fish and shellfish (0.12 pg/kg-day) and beef (0.13 pg/kg-day), which accounted for about 20 and 21 percent of the total TEQ_{DF}-WHO_{98} exposure, respectively. The ingestion of foods accounted for approximately 95 percent of the total TEQ_{DF}-WHO_{98} exposure. Exposure to CDD/CDFs via ingestion of water appears to be very low. Exposure via inhalation, soil ingestion, and dermal contact with soil are 0.023 pg/kg-day, 0.0063 pg/kg-day, and 0.0015 pg/kg-day, respectively. These exposures account for approximately 5.0 percent of the total CDD/CDF TEQ exposure in North America.

Adult general population TEQ_{P}-WHO_{98} exposure for North America was estimated to be 0.33 pg/kg-day (or approximately 23 pg/day, assuming a 70 kg adult), for all foods combined. This estimate is based on data on dioxin-like PCBs for food items and soil; PCB congener data were not available for urban air or water. For CDD/CDFs, these

environmental media accounted for about 3.8 percent of the overall TEQ_{DF}-WHO_{98} exposure. Assuming that these media account for a similar percentage of dioxin-like PCB exposure, total PCB exposure would be approximately 0.34 pg/kg-day (i.e., 0.33 pg/kg-day x 1.038). Thus, TEQ_{P}-WHO_{98} exposures from PCBs are approximately three quarters the TEQ_{DF}-WHO_{98} exposures from CDD/CDFs.

4.4.3. Assessment of Background Exposures Among Children

Exposures among other age groups of the U.S. population were also estimated using the same media TEQ-WHO_{98} concentrations that were used to estimate adult exposures. However, age-specific contact rates and body weights were used. These values were derived from data presented in U.S. EPA (1997) and USDA (1995). Background exposures were estimated for three age groups (i.e., 1-5 years, 6-11 years, and 12-19 years). Table 4-34 compares the contact rates and estimated CDD/CDF exposures for these age groups to adult contact rates and exposures. Table 4-35 makes similar comparisons for TEQ_{P}-WHO_{98}s. As shown in these tables, the dose per unit body weight (pg/kg/day) decreases with increasing age, but the daily dose (pg/day) increases with age. On a pg/kg-day basis, adult TEQ_{DF}-WHO_{98} doses were 3.6 times lower than those of 1 to 5 year old children and 2.1 times lower than those of 6 to 11 year old children. Likewise, for PCBs, TEQ_{P}-WHO_{98} adult doses were 3.3 times lower for 1 to 5 year old children and 1.8 times lower for 6 to 11 year olds. Table 4-36 presents the percentage contribution of each environmental media and food group to total TEQ dose for each age group. Figure 4-5 depicts these percentages for CDD/CDFs, grouped as meat/fish/eggs, dairy, and other, for the four age groups.

Milk and dairy products accounted for approximately 56 percent of the total TEQ_{DF}-WHO_{98} and TEQ_{P}-WHO_{98} exposures in 1 to 5 year old children, but only approximately 22 percent in adults. In contrast, meat and fish intake accounted for a much smaller portion of total exposure in 1 to 5 year olds, and a higher portion in adults.

Patandin et al. (1999) observed similar results using data for adults and children in The Netherlands. Data on CDD/CDF and PCB residues in foods were combined with food consumption data for various age groups to model dietary intake of dioxin-like compounds in the following age groups: 1 to 5 years, 6 to 10 years, 10 to 15 years, 16 to 20 years, and 20 to 25 years. The doses, on a body weight basis, were higher than those

estimated for the United Sates population, but the ratio of adult to child doses were similar to those described above. For example, Patandin et al. (1999) estimated a daily TEQ_{DFP}-WHO_{94} dose of 6.5 pg/kg-day for male children, age 1 to 5 years; 3.9 pg/kg-day for male children, age 6 to 10 years; and 2.4 pg/kg-day for adults, age 20 to 25 years. The adult value is 2.7 and 1.6 times lower than the values for 1 to 5 year old males and 6 to 10 years old males, respectively. Patandin et al. (1999) also reported on the contributions of various food group to total dietary intake of CDD/CDF/PCBs for various age groups. The results are consistent with those described above for the U.S. population.

4.4.4. Variability in Intake Estimates

The background adult daily intake values presented in Tables 4-30 and 4-31 are representative of mean exposures among the adult general population because they are based on mean TEQ_{DF}-WHO_{98} and TEQ_{P}-WHO_{98} concentrations and mean contact rates. They do not account for individuals with higher contact rates for foods or environmental media, or individuals who may be exposed to higher concentrations of dioxin-like compounds such as those affected by localized contamination.

Exposures to dioxin-like compounds were estimated as the product of media concentrations of CDD/CDF/PCBs times contact rates for these media with food ingestion accounting for the vast majority of the dose. Assuming that, over the long-term, all individuals in the general population are exposed to the mean TEQ_{DFP}-WHO_{98} media concentrations, variability among this population can be assessed by evaluating variations in contact rates. The assumption that long-term media concentrations to which the general population are exposed are represented by mean values is reasonable if temporarily elevated concentrations are offset by lower concentrations during other time periods, and if no regional trends are assumed (e.g., foods with varying CDD/CDF/PCB concentrations are equally distributed in the market place). Also, because food intake accounts for such a large percentage of the total dose, variations in long-term average food contact rates (i.e., ingestion rates) are likely to have the greatest impact on long-term average dose.

Some sense of the variability in general population exposures to TEQ_{DFP}-WHO_{98} can be gained by evaluating either the variability in fat intake among the general population (i.e., because fatty foods account for a high percentage of total exposure), or by

evaluating the variability of specific dietary components (i.e., food groups of the total diet). Published data on the variability in fat intake among the general population are somewhat limited. However, Cresanta et al. (1988), Nicklas et al. (1993), and Frank et al. (1986) analyzed dietary fat intake data as part of the Bogalusa heart study. The Bogalusa study "is an epidemiologic investigation of cardiovascular risk-factor variables and environmental determinants in a population that began 20 years ago" (Nicklas et al., 1995). Among other things, the study collected fat intake data for children, adolescents, and young adults. According to Nicklas (1995), "the diets of children in the Bogalusa study are similar to those reported in national studies of children." Thus, these data are useful in evaluating the variability in fat intake among the general population for the purposes of evaluating variability in exposure for dioxin-like compounds among this group. Based on data for 6 month old to 17 year old individuals during 1973 to 1982, maximum total fat intakes are 2.5 to 5 times higher than mean fat intakes. Maximum animal fat intakes for this group are 3 to 7.6 times mean animal fat intakes (Frank et al., 1986). Based on the mean total fat intake plus three standard deviations for 10-year old children during 1992 to 1994 and young adults (i.e., 19 to 28 years) during 1988 to 1990, upper-range fat intake is between two to three times that of mean intake (Nicklas et al., 1993; Nicklas et al., 1995). (Three standard deviations around the mean should represent approximately 99 percent of the population.) These data are presented in Table 4-37. Based on the assumption that variability in intake is the key contributing factor to variability in exposure to dioxin-like compounds, and that the fat intake data from these studies is representative of the general population of the United States, upper-range exposures to dioxin-like compounds would be expected to be two to three times higher than the mean background exposures estimated in this chapter.

Block (1992) and Norris (1997) estimated dietary fat intake among the adult general population using data from National Health Interview Surveys (NHIS) conducted by the National Center for Health Statistics (NCHS). Block (1992) used data for 20,143 men and women, ages 18 to 80+ years, from the survey. The mean and standard deviation fat intakes from this analysis are presented in Table 4-38. Assuming that the mean value plus three standard deviations represents the upper end of the range of fat intake, maximum fat intake is approximately two to three times higher than the mean. Norris (1997) used data for 10,827 men and women from the 1992 NHIS. The mean fat intake

was 64.4 g/day and the standard deviation was estimated to be 41.6 g/day. Using the same assumption as stated above (mean plus three standard deviations), the upper end of the range of fat intake wold be 189.2 g/day. This value is 2.9 times higher than the mean. Thus, these data from a nationally representative sample of adults are consistent with the data for children and young adults from the Bogulusa study. This variability is also supported by the ranges of tissue CDD/CDF/PCB levels, as described in Section 4.2. These data show that maximum tissue levels of dioxin-like compounds are typically two to three times the mean values.

Another way to assess variability in CDD/CDF/PCB background doses among the general U.S. population is to evaluate variability in total dietary intake and the contribution of specific dietary components to total dietary intake. Recently, EPA conducted an analysis of USDA's 1994-1996 CSFII data set to estimate total dietary intake as well as the contribution of the major food groups (i.e., total dairy, total fish, total meats, total fats, eggs, etc.) to the total diet. Intake data from this analysis were used in conjunction with average CDD/CDF/PCB concentrations in foods to evaluate variability in background dose of dioxin-like compounds.

The procedure used to evaluate variability in CDD/CDF/PCB doses from total dietary intakes derived from the CSFII was developed as follows. First, estimates of "total dietary intake" for individuals in the CSFII were determined as the sum of all food intakes reported by the individuals included in the survey. For purposes of this exercise, specific food items reported by each individual in the CSFII were grouped into classes, including total dairy, total meats, total fish, total vegetables, total eggs, and total fats. Once these total dietary intakes were compiled, CSFII survey adult individuals were ranked from lowest to highest based on total dietary intake, and intake rates at specific percentiles, such as the 50th or 90th percentile were examined. From these percentiles, subsets were defined including a "central" group of adults, which were those in the 45-55th percentile of total intake, and an upper percentile group of adults, which were defined as those above the 90^{th} percentile of total intake. For the purposes of evaluating variability in CDD/CDF/PCB doses that extend above the average doses reported in this chapter, intake rates for the upper percentile group of adults was of interest. To calculate upper percentile doses of CDD/CDF/PCBs, point estimates of the intake rates for each of the major food groups were calculated as the mean intake rate for the individuals within the upper percentile of

total food intake (i.e., above the 90th percentile). As noted above, these intake rates represented intakes for major food groups only (e.g., total meats) and not specific food items (e.g., beef, pork, poultry). Therefore, to complete this exercise, it was necessary to convert the intake rates of the major food groups to intake rates for the categories of individual foods for which CDD/CDF/PCB concentration data were available. To do so, it was assumed that the proportions of individual foods (e.g., beef, pork, poultry, and other meats) making up a food group (e.g., total meats) were the same for the upper percentile groups as for the average background individual assessed in Tables 4-30 and 4-31. Finally, average concentrations of CDD/CDF/PCBs in the various individual food items (as shown in Tables 4-30 and 4-31) were combined with the upper percentile intakes rates for individual food items to arrive at the doses to an upper percentile adult. The results for this exercise for the "upper percentile" intake rates are shown in Tables 4-39 and 4-40, which also include the average non-food exposures associated with soil, water, and air. As shown in Table 4-39 the estimated TEQ_{DF}-WHO_{98} dose among adults in the "upper percentile" of total food intake is 1.1 pg/kg/day or 77 pg day. This dose is 1.8 times higher than the mean TEQ_{DF}-WHO_{98} dose estimated in Table 4-30. The estimated TEQ_{P}-WHO_{98} dose for "upper percentile" adults is 0.65 pg/kg-day or 45 pg/day (Table 4-40). This dose is 1.9 times higher the mean dose estimated in Table 4-31.

The variability in current dose of about 2 to 3 times above the mean is similar to the range of tissue CDD/CDF/PCB levels, as described in Section 4.2. These data show that maximum tissue levels of dioxin-like compounds are typically two to three times the mean values. However, it was also discussed that important factors such as the age of the individual and their past history of exposure also contributed to variability in tissue levels, perhaps more so than their current dose. Therefore, this variability in tissue data, while similar to the variability in intakes based on the dietary data discussed here, should not be considered as important supportive evidence to a finding that elevated intakes of dioxin-like compounds range up to 3 times higher than the average dose. Also of note is that the 1994 Dioxin Reassessment documents developed an estimate of variability of intake of between 3 and 7 times the mean intake rates. This variability estimate was based on statistical extrapolations from a relatively small study measuring CDD/CDFs in blood. The new variability estimates presented here are considered more strongly

supported because they are based on larger studies, do not involve extrapolations, and more directly reflect consumption.

4.4.5. Comparison of Previous North American Studies to This Study

Previous studies of CDD/CDF exposures in North America were presented in Section 4.4.1 of this report. These studies reported CDD/CDF exposures based on the most toxic congener, 2,3,7,8-TCDD, and not on the total TEQ_{DF} value for all congeners combined. For the purposes of comparison, mean background levels of 2,3,7,8-TCDD in North America from this assessment were used to calculate exposure via various pathways. Background exposures were calculated using background environmental levels of 2,3,7,8-TCDD, standard contact rates, and appropriate unit conversion factors, as described previously. Total 2,3,7,8-TCDD exposure among adults for all pathways combined was 5.5 pg/day for the current assessment compared to 15.9 and 34.8 pg/day for the two previous studies of 2,3,7,8-TCDD exposure in North America (Henry et al., 1992; and Travis and Hattemer-Frey, 1991). Figure 4-6 depicts the comparisons of the percent contribution of various exposure pathways to total exposure to 2,3,7,8-TCDD for the current assessment and for previous North American studies. Figure 4-6 indicates that exposure via ingestion of meats accounted for a large portion of the exposure in all three studies. However, fish accounted for a higher percentage, and dairy products accounted for a lower percentage of the total 2,3,7,8-TCDD exposure in the Henry et al. (1992) study and in the current assessment than in the Travis and Hattemer-Frey (1991) study. These differences reflect differences in assumptions for food ingestion rates as well as in TCDD levels. All three studies indicate that beef, dairy products, and fish comprise over 93 percent of the total exposure. Because of the data base weaknesses noted earlier, it is not known if these differences can be considered significant.

European CDD/CDF exposure studies may also be compared to the exposures estimated in U.S. reports and in the current assessment. Comparisons may be made based on the 2,3,7,8-TCDD congener or on total TEQ_{DF} exposures (Table 4-41). Adult general population exposures to 2,3,7,8-TCDD in North America range from 5.5 pg/day to 34.8 pg/day based on the current assessment and two other U.S. studies. These values are comparable to the 2,3,7,8-TCDD exposures reported in Germany and The Netherlands by Fürst et al. (1991) and Theelen (1991). Fürst et al. (1991) reported an estimated

2,3,7,8-TCDD exposure of 25 pg/day based on ingestion of dairy products, meat, and fish; Theelen (1991) reported an estimate of 20 pg/day based on dairy, meat, poultry, and fish intake. Total TEQ_{DF} background exposure estimates for North America range from approximately 43 pg TEQ_{DF}-WHO_{98}/day for the current assessment to 140 to 290 pg I-TEQ_{DF}/day based on Gilman and Newhook's (1991) Canadian study. For Europe, total I-TEQ_{DF} exposure estimates range from 79 pg/day based on Fürst et al. (1990) to 158 pg/day based on Fürst et al. (1991).

4.4.6. Relative Contribution of Exposure Pathways to Total Intake

Figure 4-7 depicts the contributions of various exposure pathways to total background TEQ exposures for North America, Germany, the United Kingdom, and The Netherlands based on data from the current assessment (Fürst et al., 1990; MAFF, 1995; and Theelen, 1991). For all three geographic regions, over 90 percent of the exposures were attributed to ingestion of CDD/CDFs in foods. For the United States and Germany, intake of meat, fish, and eggs account for over 60 percent of the daily exposure, while milk and dairy consumption account for less than 30 percent, and soil ingestion, inhalation, etc. account for less than 7 percent of the total exposure. For The Netherlands and the United Kingdom, the meat/fish/eggs group accounts for somewhat less of the total intake, while milk/dairy and the "other" category account for more of the exposure. In particular, approximately 30 percent of the total exposure came from breads and cereals in the United Kingdom. These food groups were not evaluated in the United States estimates.

Based on the data presented in Figure 4-7, it is reasonable to expect that the CDD/CDF body burden in vegetarians would be lower than the body burden in nonvegetarians because vegetarians avoid the consumption of meat and fish and their derivative products. Welge et al. (1993) tested this hypothesis by comparing the CDD/CDF levels in the blood of 24 German vegetarians with the blood levels of 24 nonvegetarians, matched for age, sex, body weight, and height. With the exception of two individuals, all vegetarians had practiced a diet without meat and fish for at least 3 years. The CDD/CDF levels in the vegetarian group ranged from 14.64 to 52.85 pg I-TEQ_{DF}/g (lipid basis) with a mean of 32.60 pg I-TEQ_{DF}/g. In the nonvegetarian group, the CDD/CDF levels ranged from 14.26 to 97.98 pg I-TEQ_{DF}/g (lipid basis) with a mean of

34.32 pg I-TEQ_{DF}/g. There was no significant difference (α = 0.05) between the vegetarian and nonvegetarian group in the mean levels of any of the 2,3,7,8-substituted congeners, in the total CDD levels, in the total CDF levels, in the total CDD/CDF levels, or in the total I-TEQ_{DF} levels (each on a lipid and on a whole weight basis). Welge et al. (1993) suggested several reasons why no differences were found. First, all tested vegetarians had at one time been nonvegetarians. The higher levels of exposure during this nonvegetarian period coupled with the long biological half-life of CDD/CDFs may be responsible for the apparent similarity in body burdens using blood as the measure of body burden. Second, the vegetarians may have a higher level of consumption of dairy products than the nonvegetarians and thus have a similar CDD/CDF exposure even without consumption of fish and meat.

Schecter and Papke (1998) collected blood samples from two individuals (one male and one female) who had been vegans for over 20 years and analyzed them for CDD/CDFs and coplanar PCBs. These individuals were strict vegetarians, consuming no milk, cheese, eggs, or other animal products. Total CDD/CDF and PCB concentrations, as well as I-TEQ_{DF} and TEQ_{P}-WHO_{94} concentrations among these vegans were compared to the levels in two pooled samples from 100 men and 100 women from the general population. Total concentrations of CDD/CDF/PCBs were 244 ppt and 330 ppt for male and female vegans, respectively. These values were considerably lower than those observed in pooled samples from the general population; 643 ppt and 906 ppt for male and female subjects, respectively. Likewise, the TEQ_{DFP}-WHO_{94} concentrations were lower among the vegans (4.4 ppt and 8.7 ppt for males and females, respectively) than the general population (24.2 ppt and 29.3 ppt for males and females, respectively). Both the total concentrations and TEQ levels of CDD/CDFs and PCBs were higher in the samples collected from those taken from males.

4.4.7. Geographical Contributions to Dietary Exposure

As indicated in the previous sections, dietary intake appears to be the primary pathway of human exposure to dioxin-like compounds. Over 90 percent of the background dose is obtained through the diet, with foods of animal origin being the predominant sources. Aside from some episodes of localized contamination that may result in elevated exposures among individuals who consume foods from contaminated

areas (see Chapter 5), the general population of the United States is assumed to consume foods, over the long-term, that contain average background concentrations of dioxin-like compounds, resulting in background exposures that are similar across all regions of the United States. Except for some of the more perishable foods (i.e., milk and eggs) most foods are widely distributed in commerce. Thus, the general population of the United States may consume foods from a wide variety of geographic locations. In addition, the concentrations of foods grown in the various geographic regions may not vary widely. The national studies of beef, pork, and poultry, conducted jointly by EPA and USDA (Winters et al., 1996a; Winters et al., 1996b; Lorber et al. 1997; Ferrario et al., 1997), indicated that there was little variation in the concentrations of dioxin-like concentrations, based on geographic location. The milk study (Lorber et al., 1998) suggested the possibility of a geographic trend, with CDD/CDF concentrations being somewhat higher in the southeastern United States than in the southwestern United States.

Based on the distribution of foods in commerce, and the similarities of concentrations in many foods, variations in dietary exposure on the basis of geography would not be likely to be significant and the general population would be expected, over the long term, to be exposed to similar concentrations of dioxin-like concentrations in foods. However, the total amount of dioxin-like compounds entering the food supply may vary geographically because of the predominance of certain types of food production in certain regions of the country. For example, food such as pork is produced primarily in the northern midwest and some areas on the southeastern part of the United States; whereas poultry is produced primarily in the southeast.

The purpose of this section is to present the results of a study of the geographic variability of dioxin production as indicated by variability in production of animal fats. EPA conducted an analysis to determine the geographic origin (within the 48 contiguous United States) of several food groups that are likely to contain dioxin-like compounds (e.g., meats and dairy products). Cattle, chicken, and hog producer sales figures from the 1997 Census of Agriculture (USDA, 1997), enumerated by county, were converted to an equivalent dioxin TEQ using data in Putnam and Allshouse (1999). The 1997 food disappearance data for beef, pork, and chicken in this reference were used to convert the USDA production data, expressed in units of individual animals sold, to grams of animal fat entering the food chain. Food disappearance is the total supply at the start of the

year, plus imports, minus exports and shipments to U.S. territories, minus stock at the end of the year. It therefore includes all food eaten in the home, wasted by spoilage in the home, lost in preparation, or left uneaten on the plate. The food disappearance data were expressed as a boneless weight assuming a standard conversion factor for each animal type (Putnam and Allshouse, 1999). This total weight was converted to dioxin TEQs using CDD/CDF/PCB concentration values from the EPA meat/milk surveys and WHO_{98} TEFs. The total dioxin value was then divided by the total number of animals to yield ng TEQ per animal. This value was multiplied by the county-level USDA data to yield ng TEQ per year for every county.

Production figures for dairy products are not included in the Census of Agriculture, but the number of dairy cows is provided for each county. State-level data on milk fat production were apportioned among each state's counties on the basis of the number of dairy cows in each county. This approach assumes that all milk cows in a given state are equally productive. In a similar way, where only state-level egg production data are available, county values were calculated by apportioning the state-level data among the counties on the basis of the number of layers and pullets in each county. Examples of the county-level production data are shown in Figures 4-8 and 4-9 for pork and dairy products, respectively. Similar maps were produced for the other products (i.e., beef, poultry, and eggs) and for the total TEQ_{DF}-WHO_{98} over all five products. It should be noted that the geographic variability in this analysis is based on variability in food production only, and not in the concentration of dioxin-like compounds in the foods. Thus, it does not indicate that the concentrations of dioxin-like compounds are higher in some regions than in others. Instead it indicates that the production of dioxin-containing foods is higher in some regions than in others. The relative contributions of the five food products included in this study compare favorably with EPA's current estimates of total TEQ_{DF}-WHO_{98} dose based on 1989-91 CSFII food intake data (Figure 4-10).

This analysis may be useful, in conjunction with source analyses, in identifying important food production areas where dioxin-like compounds are also being released. To that end, major contributors to the total dioxin TEQ for the 48 contiguous states were identified. The 3,048 counties in the database were sorted in descending order and divided into four groups, with each group encompassing 25 percent of the 48-state total. The resulting map (Figure 4-11) shows that the top 65 counties account for 25 percent of

the total TEQ. The second, third, and fourth quartiles encompass 212, 498, and 2,303 counties, respectively. Assuming that the dominant pathway resulting in dioxin exposure for domestic meat and dairy animals is air deposition onto feed crops, it necessarily follows that the dioxin sources that dominate general population exposure have to be those sources that dominate ambient air concentrations in the areas flagged by this analysis. Future work is aimed at identifying these dioxin sources.

4.4.8. Contribution of CDD/CDF Congeners to Background Dose and Body Tissue Concentration

The purpose of this section is to evaluate the contribution of individual congeners to background dose and tissue concentrations. This section also evaluates whether the congeners that are the primary contributors to dietary dose are consistent with those that dominate the body burden. Section 4.4.2 derived a background dose of approximately 1 pg TEQ_{DF}-WHO_{98}/kg-day, which included doses of 0.61 pg TEQ_{DF}-WHO_{98}/kg-day for CDD/Fs and 0.33 pg TEQ_{DF}-WHO_{98}/kg-day for coplanar PCBs. These doses were calculated assuming average exposure media concentrations and contact rates for several pathways. Food consumption made up most of this total dose, with the food consumption pathways of beef, pork, chicken, fresh fish, marine fish, dairy, and milk totaling 0.90 pg TEQ_{DFP}-WHO_{98}/kg-day. This exercise will focus on these pathways alone. Section 4.2 examined body tissue concentrations of the dioxin-like congeners. The average TEQ_{DF}-WHO_{98} lipid concentration in blood was calculated at 21.6 pg TEQ_{DF}-WHO_{98}/g. For dioxin-like PCBs, the average lipid concentration in blood was 2.0 TEQ_{P}-WHO_{98}/g. These data were based on the CDC blood data, as described previously, and represent recent body burdens. It should be noted, however, that PCB data were only available for four congeners (i.e., PCBs 77, 81, 126, and 169). The exercise in this section determines the percentage TEQ_{DFP}-WHO_{98} contribution of each toxic CDD, CDF, and dioxin-like PCB congener to the daily total background dose of TEQ_{DFP}-WHO_{98} s. It also determines the percentage TEQ_{DFP}-WHO_{98} contribution of each toxic congener to the body tissue TEQ_{DFP}-WHO_{98} concentrations. The exercise concludes with a comparison of the two sets of percentages.

The following general rules were applied in developing the information for this exercise:

1) *The food surveys used to calculate average concentrations for background dose calculation of TEQ-WHO_{98}s were also used to calculate TEQ-WHO_{98} congener profiles, when possible.*

For all food groups except two, the same data used in calculating background doses were used in this analysis. The total TEQ_{DF}-WHO_{98} for these data are summarized in Table 3-59 and the total TEQ_{P}-WHO_{98} are summarized in Table 3-60. For freshwater and marine fish, it was not possible to derive a CDD/CDF congener profile using the same data as that used to calculate a background dose because the individual congener concentrations were not provided in the core reference. Thus, data from Schecter et al. (1995b) were used. These data represent a sampling of 10 freshwater fish from supermarkets. For marine fish, data from Fiedler et al. (1997) were used.

2) *Average concentration profiles for food were calculated assuming non-detects are equal to one-half detection, which was the same procedure for calculating body tissue concentration profiles.*

This was the assumption used to calculate the background dose. However, it should be noted that this could be problematic for some data, specifically when the detection limits were high. The determination of the food concentration profiles in Chapter 3 was accomplished assuming nondetects were equal to zero for this reason.

3) *When more than one survey was used to determine the average representative concentration profile in food or body tissue concentration, all samples were pooled and assumed equally weighted for dioxins. However, for coplanar PCBs, the data in the literature studies were developed by compositing methods that did not allow for the calculation of weighted averages. Because of this, one concentration per study was derived for each congener, and then the average concentration was assumed to be the average over the number of studies.*

Since many of the studies reporting CDD/CDF concentrations, particularly the food studies, were grab sample studies, it seems most reasonable to simply treat all samples equally. Also, mean food concentrations, for purposes of background dose derivation of the CDD/CDFs, were calculated giving all samples equal weight. Therefore, the determination of the representative profiles was made consistent with the dose

calculation. The background dose calculation for the dioxin-like PCBs was done slightly differently. In these cases, two of the principal studies, Mes and Weber (1989) and Mes et al. (1991) composited several samples. In one study, nondetected congeners on composite samples were set to one-half the detection limits for calculating mean congener concentrations. However, in the other study, mean congener concentrations were based on positive composites only. Thus, there was no simple method for calculating a weighted mean for these studies.

4) *For the dioxin-like PCBs, not all the studies evaluated the same coplanar congeners. This occurred in both the food data and the tissue data. Therefore, this analysis is incomplete with regard to estimating the full dose of dioxin-like PCBs as well as the percentage of dose/body tissue* TEQ_P-WHO_{98} *that can be attributed to each congener. This appears to be an issue for two of the dioxin-like PCBs. However, inclusion of the full information of these two congeners will unlikely change the important qualitative finding in the dioxin-like PCB analysis - that PCB 126 dominates both tissue and body burden concentration.*

There are 11 dioxin-like PCBs with some dioxin-like toxicity, based on the TEQ_P-WHO_{98} scheme (Younes, 1998). Using the TEF_P-WHO_{98} scheme, 13 PCB congeners were considered to have dioxin-like toxicity. The CDC data set included data for only four of the dioxin-like PCBs for human tissues (i.e., PCBs 77, 81, 126, and 169). There were no reported concentrations in food for two of the congeners, PCBs 123 and 167. PCB 114 had some impact on total tissue concentrations and was included in some of the food survey data. However, this congener was not included in the USDA/EPA national studies on pork, beef, poultry, and milk. Other food studies also measured PCB 189, which was not included in the USDA/EPA studies, but contributed an insignificant amount to coplanar TEQ_P-WHO_{98} concentration, so its exclusion in the USDA/EPA studies was not critical. The net effect for exclusion of PCB 114 in these food groups is that the contribution of PCB 126 to TEQ_P-WHO_{98} was overestimated while the contribution from PCB 114 was underestimated. Likewise for human tissues, the contribution of PCB 126 to TEQ_P-WHO_{98} is likely overestimated.

Further details on the procedures used in the forward dose calculations and the body tissue concentrations are presented below.

4.4.8.1. *Background Dose*

Approximately 90 percent of the background daily TEQ_{DFP}-WHO_{98} dose is derived from the following foods: freshwater fish, marine fish, milk, dairy, beef, pork, and poultry. For CDDs/CDFs/PCBs, the total daily dose from these pathways is estimated to be 58 pg TEQ_{DFP}-WHO_{98}/day. The remaining dose comes from: soil ingestion, marine shellfish ingestion, inhalation, water ingestion, egg ingestion, and vegetable fat ingestion. For ease of calculation, this exercise focuses on the higher contributing food groups rather than on all routes of exposure. Further, when calculating the percentage of the total TEQ_{DFP}-WHO_{98} dose which can be attributed to each congener, it is assumed that the 58 pg TEQ_{DFP}-WHO_{98}/d represents 100 percent of the daily dose. For ease of understanding, the CDD/CDF and PCBs are tabulated separately in the tables and figures. The TEQ_{DF}-WHO_{98} dose from CDDs/CDFs is 38 pg/day and the dose for coplanar PCBs is 20 pg TEQ_{P}-WHO_{98}/day. The congener contributions from the dietary intake calculation is characterized in terms of the percentage each congener contributes to the TEQ_{DFP}-WHO_{98}. This will be compared to the congener contributions to body burdens, which are also compiled on an individual percentage basis.

The procedure for doing the dietary intake calculations is described in the following four steps:

1. **Determine the representative congener concentrations in the food product.** These were determined as the average concentrations of the individual congeners from available survey data, given the rules stated above. Based on the way in which the data were reported in the literature, the basis for food concentrations was either on a lipid basis or on a whole weight basis. Most of the CDD/CDF food data were reported on a lipid basis, while most of the coplanar PCB data were reported in the literature on a whole food basis. Although the basis for the food concentrations is important for calculating a dose because the concentration data must be consistent with the intake data (i.e., if concentrations are reported on a whole weight basis, whole weight intake rates must be used), it was not important for calculating the fractional contribution of each congener to the total TEQ since the same values would be calculated using either lipid-based or whole weight concentrations. Therefore, lipid-based CDD/CDF concentrations were used for all foods, and whole weight PCB concentrations were used for all foods.

2. **Determine the toxic equivalent concentrations in the food product.** These were easily determined as the product of the average congener concentration and the appropriate TEF.

3. **Determine the TEQ-WHO_{98} congener profiles as the fractional contribution of each congener to total TEQ-WHO_{98} concentration.** This was determined as the ratio of the toxic equivalent concentration of each congener to the total TEQ-WHO_{98}.

4. **Determine the TEQ-WHO_{98} congener profile of the dietary dose by multiplying the TEQ-WHO_{98} fractional contribution of each congener by food intake rate for that food product.** A multiplication of each food product's overall TEQ-WHO_{98} concentration, in pg/g, and the corresponding food consumption rate, in g/day, gives the pg TEQ-WHO_{98} consumed per day by that food product. Further multiplication of this pg TEQ-WHO_{98}/day and each congener's fractional contribution gives the pg TEQ-WHO_{98}/day contributed by each congener. The representative food TEQ-WHO_{98} concentrations described in Section 4.4.2 to determine background dose were expressed on a whole weight basis, to be consistent with the consumption rates of the food products, which were also on a whole weight basis. The whole concentration, in pg TEQ_{DF}-WHO_{98}/g for CDD/CDFs and TEQ_{P}-WHO_{98}/g for coplanar PCBs for all food products were: beef - 0.18 pg/g CDD/CDFs and 0.084 pg/g PCBs; pork - 0.28 pg/g CDD/Fs and 0.012 pg/g PCBs; poultry - 0.068 pg/g CDDs/CDFs and 0.026 pg/g PCBs; dairy - 0.12 pg/g CDD/CDFs and 0.058 pg/g PCBs; milk - 0.018 pg/g CDDs/CDFs and 0.0088 pg/g PCBs, freshwater fish - 1.2 pg/g CDD/CDFs and 1.2 pg/g PCBs; and marine fish - 0.36 pg/g CDD/Fs and 0.25 pg/g PCBs. The consumption rates for this exercise were expressed in g/day, which were calculated using the g/kg-day consumption rates given in Section 4.4.2 multiplied by a 70 kg adult: beef - 49.7 g/day, pork - 15.4 g/day, poultry - 35 g/day, dairy - 55 g/day, milk - 175 g/day, freshwater fish - 5.9 g/day, and marine fish - 9.6 g/day.

The results of this four-step procedure are demonstrated in Table 4-42 for the beef consumption pathway for CDDs/CDFs. Tables 4-43 and 4-44 show the average congener concentrations of CDDs/CDFs and PCBs, respectively, derived for the food groups, the total TEQ-WHO_{98} concentration for each food group from this profile, and the TEQ-WHO_{98} percentage contributions for each congener and food group. Tables 4-45 and 4-46 show

the final results of this exercise for CDDs/CDFs and PCBs, respectively. Results suggest that 72 percent of the total TEQ_{DF}-WHO_{98} background dose of CDDs/CDFs comes from four congeners: 1,2,3,7,8-PCDD (33 percent), 2,3,4,7,8-PCDF (17 percent), 2,3,7,8-TCDD (10 percent), and 1,2,3,6,7,8-HxCDF (12 percent). PCB 126 comprises 61 percent of the TEQ_P-WHO_{98} dose of dioxin-like PCBs. When adding the doses of the CDDs/CDFs to the coplanar PCBs, PCB 126 and 1,2,3,7,8-PCDD are the largest contributors at 21 percent, followed by the three CDD/CDF congeners at 2,3,4,7,8-PCDF (11 percent), 2,3,7,8-TCDD (7 percent), and 1,2,3,6,7,8-HxCDD (8 percent).

4.4.8.2. *Background Tissue Concentrations*

For the purposes of this exercise, the tissue concentrations from the CDC studies reported in Table 4-18 were used. For coplanar PCBs, the main issue was that data for only four PCB congeners were included (i.e., PCBs 77, 81, 126, and 169). As a result of the exclusion of the other PCBs, their percent contribution to the TEQ tissue concentration could not be calculated.

Once the concentrations were derived, the TEQ-WHO_{98} contributions of individual congeners to the total TEQ-WHO_{98} were derived in a manner similar to the food results. Tables 4-47 and 4-48 show the final results of this exercise for CDDs/CDFs and coplanar PCBs, respectively, giving the derived actual congener concentrations, and the percentage contribution to TEQ-WHO_{98} for each congener. The studies used in this exercise are the same as those used in Section 4.2.3 to estimate recent (i.e., 1990s) body burden levels.

Table 4-47 indicates that four congeners contribute 82 percent of CDD/CDF TEQ_{DF}-WHO_{98}: 1,2,3,6,7,8-HxCDD (34 percent), 1,2,3,7,8-PCDD (24 percent), 2,3,4,7,8-PCDF (14 percent), and 2,3,7,8-TCDD (10 percent). These are the same four congeners contributing the most to background dose. From Table 4-48, it is seen that PCB 126 overwhelms all other congeners, and for all tissue types. PCB 126 comprises 90 percent of the dose of dioxin-like PCBs. Figures 4-12 and 4-13 compare the fractional TEQ-WHO_{98} contributions of each congener to the total TEQ-WHO_{98} background dose of CDD/CDFs (Figure 4-12) and coplanar PCBs (Figure 4-13), to the TEQ-WHO_{98} contributions of each congener to average body tissue TEQ-WHO_{98} concentration of CDD/CDFs (Figure 4-12) and coplanar PCBs (Figure 4-13). The match between the

highest contributors is noteworthy from this figure, as is the lack of contribution from other congeners. Some key observations that can be gleaned from this exercise include:

1) As noted, five congeners dominate the TEQ-WHO_{98} body burden as well as the TEQ dose. These are, 1,2,3,7,8-PCDD, 1,2,3,6,7,8-HxCDD, 2,3,4,7,8-PCDF, and 2,3,7,8-TCDD from the CDD/CDFs, and PCB 126 from the coplanar PCBs.

2) For the four dominant CDD/CDF congeners combined, the body burden had a higher TEQ-WHO_{98} contribution than the food: contributions from the four congeners to body burden TEQ-WHO_{98} equaled 82 percent while for food they equaled 72 percent.

3) While 2,3,7,8-TCDD has been the focus of past exposure and health studies, it would appear that the other CDD/CDF congeners found to be high contributors in this exercise may also be important from an exposure and health standpoint.

4.5. Comparison of Assessment Approaches and Best Estimates of Intake

Two approaches were used in this chapter to estimate background exposures to dioxin-like compounds among the general population of the Unites States. The first approach used pharmacokinetic modeling to calculate a dose from tissue concentrations. This was done using either a steady state or non-steady state approach. Using the steady state approach, the TEQ_{DF}-WHO_{98} dose was estimated to be 126 pg/day, when the half life for TCDD (i.e., 7.1 years) was assumed to apply to the total TEQ, and 87 pg/day, when congener specific half-lives were used. PCB doses could not be estimated in this way because of the lack of congener-specific half-life information. The advantage of modeling doses from tissue concentrations is that all pathways of exposure are accounted for. However, because the half-lives of dioxin-like compounds in the body are relatively long (i.e., 7.1 years for TCDD), modeled doses may reflect the cumulative effect of previous doses and not current doses. This was demonstrated by a non-steady state model used to reconstruct past doses of 2,3,7,8-TCDD. The results of the modeling exercise indicated that current doses would be expected to be less than past doses. Assuming that these results would apply to all dioxin-like congeners, and not just 2,3,7,8-TCDD, the current total TEQ_{DF}-WHO_{98} dose would be expected to be somewhat lower than 88 pg/day, as estimated using the steady state approach.

The second approach used for estimating background doses to dioxin-like compounds was to evaluate dioxin-like compounds in various dietary components (i.e.,

meats, dairy products, fish, etc.) and environmental media (i.e., air, soil, water) to which humans are exposed. By combining TEQ_{DFP}-WHO_{98} concentrations in foods and these media with the contact rates (i.e., ingestion, inhalation, dermal contact rates) for these foods and media, CDD/CDF and PCB doses were calculated. Using this approach, the daily TEQ_{DF}-WHO_{98} dose was estimated to be 43 pg/day and the TEQ_{P}-WHO_{98} was estimated to be 23 pg/day. The advantage of using this approach is that, if current media concentrations and intake estimates are used, the estimated doses should reflect current exposures. In this analysis, the most recent data on the concentrations of dioxin-like compounds in beef, pork, poultry, milk, and vegetable oil, collected by EPA, have been used. Recent data from the published literature have also been used for freshwater and marine fish and shellfish. Likewise, intake rates are based on EPA's recently published *Exposure Factors Handbook* (U.S. EPA, 1997)which presented data from USDA's 1989-1991 Continuing Survey of Food Intake Among Individuals (USDA, 1995) (a more recent USDA data set has been released since the *Exposure Factors Handbook* was published, but EPA has not yet completed its analysis of these data), and the most current data for establishing contact rates for other media. It should be noted, however, that the dose component approach may underestimate current doses if important pathways of exposure are not accounted for in the component analysis. For example, in this assessment, fruits and vegetables have not been considered as significant contributors to the overall dose. Data for the concentrations of dioxin-like compounds in fruits and vegetables are limited, but it expected that the concentrations would be lower in these foods than in fatty foods such as meat, fish and dairy products. Thus, a fruit and vegetable component has not been included in this analysis. If fruits and vegetables actually account for a more significant portion of the exposure than expected, the dose estimated here may be lower than that experienced by the general population of the United States. Other uncertainties introduced by this approach include the use of soil ingestion rates that may or may not account for all types of inadvertent soil ingestion (e.g., outdoor soil, household dust), the lack of PCB residue data for soils and air, and non-representative sampling data for air. For example, the adult soil ingestion rate cited in the *Exposure Factors Handbook* (U.S. EPA, 1997) is based on a limited data set, but is used as a reasonable surrogate for all forms of soil ingestion. The accuracy of this assumption is difficult to assess; however, because soil ingestion accounts for a small percentage of the overall dose, this uncertainty

is not expected to significantly affect one's confidence in the dose estimates. Likewise, the lack of PCB soil and air data, and the non-representative nature of the CDD/CDF air data would be expected to have little effect on the overall dose estimate, because these pathways account for a small percentage of the overall dose.

Despite these uncertainties, the dose component approach is believed to provide the best estimate of the mean current background dose to the general U.S. population. Variability was evaluated using dietary fat data, high-end intake rates, and by evaluating variability in body burden. In general, these data indicate that the high-end dose of dioxin-like compounds is likely to be 2 to 3 times higher than the mean.

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Table 4-1. NHATS Mean Adipose Tissue Data (ppt, lipid adjusted)

Congener	Congener Concentration (pg/g)	I-TEQ_{DF} Concentration (pg/g)	TEQ_{DF}-WHO_{98} Concentration (pg/g)	Percent Detected[a]
2,3,7,8-TCDD	5.38	5.38	5.38	97
2,3,7,8-PeCDD	10.7	5.35	10.7	97
2,3,7,8-HxCDD	86.8	8.68	8.68	97
2,3,7,8-HpCDD	110	1.1	1.1	100
OCDD	724	0.72	0.072	100
2,3,7,8-TCDF	1.88	0.19	0.19	100
1,2,3,7,8-PeCDF	0.31	0.016	0.016	14
2,3,4,7,8-PeCDF	9.7	4.85	4.85	95
2,3,7,8-HxCDF	14.2	1.42	1.42	2 to 92
2,3,7,8-HpCDF	16	0.16	0.16	4 to 89
OCDF	2.28	0.002	0.0002	30
TOTAL		27.9	32.6	

[a] Based on analysis of 48 samples composited from 865 samples

Source: U.S. EPA (1991a).

Table 4-2. Estimated Mean I-TEQ_{DF} Concentrations (ppt) in Adipose Tissue for U.S. Subpopulations from the 1987 NHATS

	I-TEQ_{DF} Concentration (ppt)	Percent of Population[a]
Census Regions		
Northeast	31.1	22
North Central	29.7	26
South	26.6	33
West	24.4	19
Age Groups		
0-14 years	9.7	23
15-44 years	24.6	46
45 + years	46.5	31
Race		
Caucasian	26.5	83
Non-Caucasian	35.2	17
Sex		
Male	26.1	49
Female	29.9	51
Total Population	27.9	100

[a] Population percentage based on 1980 U.S. Census.

Source: Orban et al. (1994).

Table 4-3. Human Adipose Tissue Data (ppt, lipid adjusted)

Chemical	Range (ppt)	Mean (ppt)
2,3,7,8-TCDD	1.6 to 38	10.4
PCB 77	Nondetect to 27.9	11.7
PCB 126	14.6 to 371	135
PCB 169	29.5 to 174	69
PCB 81	1.5 to 21.3	10.5

Source: Patterson et al. (1994).

Table 4-4. Mean Levels in Human Serum (ppt, whole weight basis)

Chemical	1982	1988	1989
2,3,7,8-TCDD	Not Measured	0.159	0.0165
PCB 77	1.38	0.481	0.251
PCB 126	0.281	0.183	0.135
PCB 169	0.282	0.151	0.192
PCB 105	Not Measured	33.2	Not Measured
PCB 118	Not Measured	366	Not Measured
PCB 180	Not Measured	466	Not Measured
Total PCBs	Not Measured	3,100	Not Measured

Source: Patterson et al. (1994).

Table 4-5. Mean TEQ Levels in Pooled Serum Samples

	$I\text{-}TEQ_{DF}$ (ppt, lipid basis)	$TEQ_P\text{-}WHO_{94}$ (ppt, lipid basis)
Cornwall		
Sports Fishers		
<38 years, lower	20.8	--
higher	22.2	3.6
38 years, lower	28.4	3.1
higher	31.4	9.5
> 50 years, higher	33.5	17.3
Nonfish Eaters		
<38 years	24.7	2.6
38-50 years	29.8	6.8
>50 years	36.8	9.7
Mississauga		
Sports Fishers		
<38 years	32.4	--
38-50 years	40.1	--
>50 years	41.2	--
Nonfish Eaters		
<38 years	34.0	--
38-50 years	29.1	--
>50 years	34.3	--

Source: Adapted from Cole et al. (1995).

Table 4-6. CDD/CDF Levels in Human Blood from Various Countries

Country	Mean Blood Level (ppt I-TEQ_{DF}, lipid)	Number of Samples
USA	41 (50 ppt TEQ_{DF}-WHO_{98})	100
Germany	42 (49 ppt TEQ_{DF}-WHO_{98})	85
S. Vietnam (Ho Chi Minh)	28	50
S. Vietnam (Dong Nai)	49	33
N. Vietnam (Hanoi)	12	32
Guam	32	10
Soviet Union (St. Petersburg)	17	50
Siberia (Baikalsk)	18	8
Japan	31 (35 ppt TEQ_{DF}-WHO_{98})	50-100

Source: Schecter et al. (1992a; 1994a).

Table 4-7. CDD/CDF Levels in Human Adipose Tissues from Various Countries

Country	Mean Tissue Level (ppt I-TEQ_{DF})	Number of Samples
USA	24 (27 ppt TEQ_{DF}-WHO_{98})	15
Germany	69 (79 ppt TEQ_{DF}-WHO_{98})	4
China	18	7
Japan	38 (43 ppt TEQ_{DF}-WHO_{98})	6
Canada	36 (40 ppt TEQ_{DF}-WHO_{98})	46
S. Vietnam	30	41
N. Vietnam	4	26

Source: Schecter (1991).

Table 4-8. Levels of CDDs and CDFs 2,3,7,8-Substituted Found in Spanish Human Adipose Tissue on Fat Weight Basis in pg/g (ppt). (17 samples)

Isomers	No. of Pos.	Range (pg/g)	Mean (pg/g)	S.D. (pg/g)	I-TEQ_{DF} (pg/g)
2,3,7,8-TCDD	6	ND-13.86	3.28	5.03	3.28
2,3,7,8-TCDF	11	ND-18.52	3.98	5.24	0.39
1,2,3,7,8-PCDF	4	ND-25.87	2.01	6.47	0.02
2,3,4,7,8-PCDF	13	ND-44.77	25.14	15.86	12.7
1,2,3,7,8-PCDD	12	ND-22.57	10.74	8.89	5.37
1,2,3,4,7,8-HCDF	10	ND-83.63	18.77	25.43	1.87
1,2,3,6,7,8-HCDF	10	ND-68.10	14.92	19.05	1.49
2,3,4,6,7,8-HCDF	8	ND-66.31	10.87	21.05	1.87
1,2,3,7,8,9-HCDF	10	ND-76.40	20.63	38.6	2.06
1,2,3,4,7,8-HCDD	5	ND-54.5	6.52	14.62	0.65
1,2,3,6,7,8-HCDD	12	ND-152.4	65.64	54.60	6.56
1,2,3,7,8,9-HCDD	13	ND-41.28	19.9	13.31	1.99
1,2,3,4,6,7,8-HCDF	14	ND-102.2	23.63	25.45	0.23
1,2,3,4,7,8,9-HCDF	6	ND-106.6	9.40	26.55	0.09
1,2,3,4,6,7,8-HCDD	17	60.4-707.4	187.4	146.35	1.87
OCDF	11	ND-293.7	72.30	99.59	0.072
OCDD	17	91-2847.5	1318.1	742.49	1.31
CDDs	17	313.9-3457	1608.3	839.6	21.03
CDFs	17	23.9-649.7	203.4	188.3	20.79
CDDs + CDFs	17	963.7-3604.2	1811.7	813.8	41.8 (TEQ_{DF}-WHO_{98} = 46 pg/g)

ND = Not detected

Source: Gonzalez et al. (1993).

Table 4-9. Concentration of CDDs, CDFs, and PCBs in Human Milk on a Fat Basis (pg/g)

Congener	Primipara (n = 7)		Multipara (n = 8)		Ratio Pri/Multi
	Mean	SD	Mean	SD	
2,3,7,8-TCDD	2.0	0.4	1.2	0.3	1.7***
1,2,3,7,8-PeCDD	8.9	1.7	5.0	2.6	1.8**
1,2,3,4,7,8-HxCDD	4.7	4.3	2.6	1.1	1.8
1,2,3,6,7,8-HxCDD	32.3	8.1	18.9	6.4	1.7**
1,2,3,7,8,9-HxCDD	6.9	2.7	3.6	1.2	1.9**
1,2,3,4,6,7,8-HpCDD	29.8	15.4	31.3	15.6	0.9
OCDD	174.2	137.0	194.6	75.5	0.9
2,3,7,8-TCDF	2.3	0.8	2.0	0.5	1.1
1,2,3,7,8-PeCDF	0.6	0.5	0.6	0.5	1.1
2,3,4,7,8-PeCDF	11.4	1.3	7.8	3.0	1.5*
1,2,3,4,7,8-HxCDF	4.3	0.5	3.3	1.2	1.3
1,2,3,6,7,8-HxCDF	4.5	0.5	3.2	1.3	1.4*
1,2,3,7,8,9-HxCDF	1.9	0.8	1.6	0.4	1.2
2,3,4,6,7,8-HxCDF	2.0	1.3	1.5	0.7	1.3
1,2,3,4,6,7,8-HpCDF	2.0	0.7	2.1	0.5	1.0
1,2,3,4,7,8,9-HpCDF	0.2	0.2	0.7	0.9	0.3
OCDF	2.7	1.3	3.0	1.3	0.9
3,3',4,4'-TeCB	10.4	6.4	13.7	7.3	0.8
3,3',4,4',5-PeCB	134.5	70.7	165.9	87.4	0.8
3,3',4,4',5,5'-HxCB	60.0	33.4	50.1	21.1	1.2
Total CDD	258.7	144.7	257.2	78.9	1.0
Total CDF	27.0	4.2	21.5	5.7	1.3*
Total CDD/CDF	285.7	145.8	278.7	83.5	1.0
Total Dioxin-like PCB	204.9	94.3	229.8	105.9	0.9
I-TEQ_{DF}	32.6	9.6	28.9	8.9	1.1
Fat (%)	4.6	1.8	3.5	0.9	1.3
Age	27.4	3.8	32.1	4.2	0.9

*** $p < 0.01$
** $p < 0.1$
* $p < 0.5$

Source: Hirakawa et al. (1995).

Table 4-10. CDD/CDF Concentrations and I-TEQ_{DF} Levels in Human Milk
(ppt, lipid basis)

Congener	Concentration (pg/g fat)		Toxicity Equivalents (pg/g fat, as I-TEQ_{DF})	
	Mean	Range	Mean	Range
2,3,7,8-T_4CDD	4.2	2.9 - 5.1	4.21	2.92 - 5.06
1,2,3,7,8-P_5CDD	11.9	8.4 - 16.6	5.94	4.18 - 8.30
1,2,3,4,7,8-H_6CDD	7.1	5.0 - 11.0	0.71	0.50 - 1.10
1,2,3,6,7,8-H_6CDD	35.3	27.8 - 45.5	3.53	2.78 - 4.55
1,2,3,7,8,9-H_6CDD	8.0	6.5 - 11.1	0.80	0.65 - 1.11
1,2,3,4,6,7,8-H_7CDD	81.4	40.8 - 142	0.81	0.41 - 1.42
O_8CDD	272	154 - 455	0.27	0.15 - 0.46
2,3,7,8-T_4CDF	1.3	0.7 - 1.9	0.13	0.07 - 0.19
1,2,3,7,8-P_5CDF	0.9	0.5 - 1.8	0.045	0.02 - 0.09
2,3,4,7,8-P_5CDF	31.1	24.7 - 42.6	15.56	12.35 - 21.30
1,2,3,4,7,8-H_6CDF	8.6	6.8 - 11.0	0.86	0.68 - 1.10
1,2,3,6,7,8-H_6CDF	7.8	6.3 - 10.4	0.77	0.63 - 1.04
1,2,3,7,8,9-H_6CDF	0.5	<0.1 - 1.0	0.05	<0.01 - 0.10
2,3,4,6,7,8-H_6CDF	4.9	2.0 - 7.0	0.49	0.20 - 0.70
1,2,3,4,6,7,8-H_7CDF	13.4	5.4 - 30.1	0.13	0.05 - 0.30
1,2,3,4,7,8,9-H_7CDF	5.0	2.5 - 15.0	0.05	0.03 - 0.15
O_8CDF	3.4	1.6 - 7.0	0.0034	0.00 - 0.01
Total CDD/CDFs	497	333 - 715	34.4 (TEQ_{DF}-WHO_{98} = 40 ppt)	27.3 - 43.2

Source: Van Cleuvenbergen et al. (1994).

Table 4-11. CDD/CDF and PCB TEQ Concentrations in Breastmilk from Various Countries and Regions Based on 1992/93 Sampling[a]

Country	Area	Indiv. Samples in Pool	Fat (wt%)	CDD/CDF (pg I-TEQ_{DF}/g)	Non-Ortho PCBs (pg TEQ_P-WHO_{94}/g)	Mono-Ortho PCBs (pg TEQ_P-WHO_{94}/g)	∑ [Marker PCBs] (ng/g)
Albania	Tirana	10	5.84	4.8	1.3	1.1	63
	Librazhd	10	4.72	3.8	1.0	0.7	43-46
Austria	Vienna (urban)	13	4.10	10.7	8.3	3.4	381
	Tulln (rural)	21	3.80	10.9	9.4	3.0	303
	Brixlegg (industrial)	13	3.40	14.0	15.1	3.8	449
Belgium	Brabant Wallou	8	3.79	20.8	3.8	3.6	275-277
	Liege	20	2.98	27.1	1.7	3.1	306-308
	Brussels	6	2.81	26.6	4.0	3.9	260-261
Canada	Maritimes 92	20	2.76	10.8-11.0	2.9	1.2-1.4	86-87
	Québec 92	20	3.06	13.4-13.6	5.1	1.7-1.9	137-138
	Ontario 92	20	3.09	18.1-18.3	5.8	1.8-2.0	128-129
	Prairies 92	20	3.20	14.6-14.8	2.3	0.9-1.1	58-59
	British Columbia 92	20	2.97	15.7-15.8	2.5	1.0-1.2	70-71
	All Provinces 92	100	2.96	14.5-14.6	3.8	1.5-1.7	112-113
	Gaspe	12	3.52	23.2-23.4	9.5	3.2-3.4	220-221
	Basse Côte-Nord	4	3.63	14.6-14.7	19.6	5.7-6.0	559-560
	Ungave Bay	4	3.31	14.3-14.5	9.8	4.3-4.6	576
	Hudson Bay	5	3.26	20.9-21.1	13.3	8.0-8.3	1361
Croatia	Kirk	10	3.80	8.4	3.8	2.2	218-219
	Zagreb	13	3.26	13.5	5.2	2.7	219
Czech	Kladno	11	5.41	12.1	2.5	3.5	532-533
	Uherske Hradiste	11	4.92	18.4	4.1	5.7	1068
Denmark	7 Different Cities	48	3.61	15.2	2.3	2.2	209-210
Finland	Helsinki	10	4.14	21.5	1.9	2.7	189
	Kuopio	24	4.49	12.0	1.0	1.4	133-135
Germany	Berlin	10	5.00	16.5-16.6	9.0	2.7	375
Hungary	Budapest	20	4.97	8.5-8.6	0.8	0.8	61-65
	Scentes	10	4.97	7.8	0.9	0.5	45-47
Netherlands	Whole Country	17	2.73	22.4-22.5	8.8	2.5	253-256
Norway	Tromsø (coastal)	10	2.56-2.70	10.1	16.1	3.4	273
	Hamar (rural)	10	2.51-2.76	9.3	7.4	3.0	265-266
	Skien/Porsgrumm (ind)	10	2.75-3.00	12.5-12.6	6.7	2.9	302
Lithuania	Palanga (coastal)	12	4.00-4.83	16.6	12.8	7.6	361
	Anykshchiai (rural)	12	3.56-4.10	14.4	12.9	7.8	287
	Vilnius City (urban)	12	2.69-2.87	13.3	11.6	8.9	322
Pakistan	Lahore	14	4.31	3.9	1.9	0.4	19-20
Russia	Arkhankelsk	1	5.17	15.2	2.9	5.7	197
	Karhopol	1	3.64	5.9	2.0	2.9	102
Slovak	Michalovce	10	4.77	15.1-15.2	6.4	7.0	1015
	Nitra	10	3.61	12.6	3.6	2.5	489-490
Spain	Bizkaia	19	3.75	19.4	6.7	3.9	461
	Gipuzkoa	10	3.86	25.5	3.8	4.4	452-453
Ukraine	Kiev nr.1	5	3.40	11.0	9.3	5.6	264
	Kiev nr.2	5	3.76	13.3	6.0	5.6	191-192
United Kingdom	Birmingham	20	3.09-3.10	17.9	2.5	1.8	129-131
	Glasgow	23	3.40-3.45	15.2	2.6	1.3	131-133

[a] Results from the second round of WHO-coordinated exposure studies on levels of PCBs, PCDDs, and PCDFs (on fat basis) in human milk. In calculating sums of the six marker PCBs and levels of PCDDs, PCDFs, non-*ortho*, and mono-*ortho* PCBs expressed in TEQ, both data are shown when non-detect values are equal to zero and non-detect values are equal to the limit of detection. If no differences appeared, a single value is presented.

I-TEF_{DF}s used in calculating TEQ_{DF}s for CCD/CDFs; TEF_P-WHO_{94}s used in calculating TEQ_Ps for PCBs.

Source: Liem et al. (1996).

Table 4-12. Comparison of Results from the First and Second Round of WHO-Coordinated Human Milk Study

Country	Area	CDDs and CDFs (pg I-TEQ_{DF}/g)				∑ [Marker PCBs] (ng/g)			
		1987/88[b]	n	1992/93	n	1987/88	n	1992/93	n
Austria	Vienna (urban)	17.1	54	10.7	13			381	13
	Tulln (rural)	18.6	51	10.9	21			303	21
Belgium	Brabant Wallou	33.7		20.8	8	558	12	275	8
	Liege	40.2		27.1	20	609	21	306	20
	Brussels	38.8		26.6	6			260	6
Canada	All Provinces 1981			28.6	200			212	200
	All Provinces 1982			14.5	100			112	100
	Maritimes	15.6	19	10.8	20			86	20
	Québec	18.1	34	13.4	20			137	20
	Ontario[c]	17.6	76	18.1	20			128	20
	Prairies	19.4	31	14.6	20			58	20
	British Columbia	23.0	23	15.7	20			70	20
Croatia	Kirk	12.0	14	8.4	10	500[a]	14	218	10
	Zagreb	11.8	41	13.5	13	450[a]	41	219	13
Denmark	Several Regions/Cities	17.8	42	15.2	48	830[a]	10	209	48
Finland	Helsinki	18.0	38	21.5	10	150	38	189	10
	Kuopio	15.5	31	12.0	24	203	31	133	24
Germany	Berlin	32.0	40	16.5	10			375	10
	North Rhine-Westphalia	31.6	79	20.7[e]		762	143		
Hungary	Budapest	9.1	100	8.5	20			61	20
	Scentes	11.3	50	7.8	10			45	10
Netherlands	Rural Area	37.4	13			416	10		
	Urban Area	39.6	13			392	10		
	All Regions	34.2	10	22.4	17	272	96	253	17
Norway[d]	Tromsø (coastal)	18.9	11	10.1	10	562[a]	10	273 (536[a])	10
	Hamar (rural)	15.0	10	9.3	10	507[a]	10	265 (483[a])	10
	Skien/Porsgrumm (ind)	19.4	10	12.5	10	533[a]	8	302 (468[a])	10
United Kingdom	Birmingham	37.0		17.9	20			129	20
	Glasgow	29.1		15.2	23			131	23

NOTE: Results are expressed on a fat basis. ∑ (marker PCBs) and TEQs are calculated assuming non-detect values are equal to zero.

[a] Analyzed using packed column technique.
[b] Calculated using Nordic TEF-model.
[c] Ontario-1988 denotes proportional mean of two pooled samples analyzed in the first round.
[d] To compare results between first and second round, samples from 1992/93 have been reanalyzed using (old) packed column technique (Becher and Skåre, personal communication).
[e] Dioxin levels in human milk samples from North Rhine-Westphalia collected in 1992 as reported by Fürst[19]).

Source: Liem et al. (1996).

Table 4-13. PCB Concentrations in Cow's Milk and Human Milk from The Netherlands (ppt, lipid basis)

	Cow's Milk (background site)	Human Milk
PCB 77	3.5	13.7
PCB 126	14.4	88.1
PCB 169	2.8	55.2

Source: Van der Velde et al. (1994).

Table 4-14. I-TEQ_{DF}s in Mother's Milk and Blood, and Infant's Blood (ppt)

Time Period	Samples Taken	Mother/Child Pair 1	Mother/Child Pair 2	Mother/Child Pair 3	Mother/Child Pair 4
Before 2nd pregnancy	Mother's blood Milk 1st Infant's blood	12.3 16.3 29.2 (age 11 months	10.5 12.8 37.5 (age 12 months)	NA	NA
At or after birth*	Mother's blood Milk Placenta Cord blood	10.3 11.9 14.5	11.9 15.6 18.5 8.4	13.4 11.8 9.7 4.1	14.5 10.9 24.4 9.1
5 Months after birth	Mother's blood Milk	11.2 11.0	6.0 11.3	No Data	11.1
11 Months after birth	Mother's blood Infant's blood	10.1 10.8 (2nd infant)	5.6 16.0 (2nd infant)	11.5 4.2 (2nd infant)	15.8 23.7 (2nd infant)

NA - Not applicable
* Represents second birth for mothers 1 and 2, and first birth for mothers 3 and 4.

Source: Abraham et al. (1998).

Table 4-15. Mean Concentrations of CDD/CDFs and Coplanar PCB Congeners from the Times Beach Exposure Study

	Target Population (n = 76)					Comparison Population (n = 74)				
	Sep, 1995	July, 1996	June, 1997	Mean	n*	Sep, 1995	July, 1996	June, 1997	Mean	n*
CDD Congeners										
2,3,7,8-TCDD	1.79	1.27	1.23	1.43	66	1.46	1.38	1.23	1.36	61
1,2,3,7,8-PCDD	4.93	4.04	2.95	3.97	67	4.53	4.96	3.45	4.31	60
1,2,3,7,8,9-HxCDD	7.24	5.98	5.15	6.12	64	6.28	7.25	5.47	6.33	59
1,2,3,4,6,7,8-HpCDD	88.0	75.4	60.5	74.6	60	83.7	84.7	64.8	77.8	61
1,2,3,4,6,7,9-HpCDD	0.89	1.06	0.68	0.88	58	0.99	0.93	0.79	0.90	59
OCDD	650.0	542.0	435.0	542.3	64	535.0	512.0	404.0	483.7	46
TEQ_D-WHO_{98}	8.4	6.7	5.3	6.8		7.5	8.0	5.9	7.1	
CDF Congeners										
2,3,7,8-TCDF	0.48	0.56	0.53	0.52	62	0.56	0.54	0.45	0.52	51
1,2,3,7,8-PCDF	0.42	0.46	0.46	0.45	66	0.48	0.49	0.46	0.48	60
2,3,4,7,8-PCDF	5.73	5.00	4.12	4.95	61	5.43	5.82	4.52	5.26	59
1,2,3,4,7,8-HxCDF	7.36	6.40	5.03	6.26	64	6.18	7.24	4.91	6.11	59
1,2,3,6,7,8-HxCDF	6.40	5.07	4.01	5.16	65	5.19	5.86	4.03	5.03	58
2,3,4,67,8-HxCDF	0.46	0.45	0.47	0.46	63	0.77	0.80	0.63	0.73	55
1,2,3,4,6,7,8-HpCDF	14.4	12.1	9.0	11.83	63	11.5	11.7	8.30	10.5	59
1,2,3,4,7,8,9-HpCDF	0.40	0.47	0.42	0.43	65	0.45	0.42	0.40	0.42	58
OCDF	1.13	1.08	0.56	0.92	53	1.22	1.08	1.46	1.25	48
TEQ_F-WHO_{98}	4.5	3.9	3.2	3.9		4.1	4.5	3.4	4.0	
Coplanar PCB Congeners										
77	2.43	1.90	2.52	2.28	63	1.90	2.47	2.22	2.20	59
81	1.97	1.92	1.91	1.93	65	2.13	2.10	2.07	2.10	54
126	9.97	8.80	8.15	8.97	66	12.8	14.2	12.3	13.1	59
169	16.4	14.4	10.8	13.9	63	16.2	16.4	13.1	15.2	59
WHO_{98} TEQ_P	1.2	1.0	0.9	1.0		1.4	1.6	1.4	1.5	

* n = number of individuals with measurements of this congener for all three sampling dates.

Source: MDOH (1999).

Table 4-16. Results of Blood Sampling for the Comparison Population at Vertac in Jacksonville, AK

	1991 Sampling of 71 individuals	1994 Resampling of 18 individuals		1995 Resampling of 14 individuals	
	1991	1991	1994	1991	1995
CDD Congeners					
2,3,7,8-TCDD	2.5	3.0	2.7	3.1	3.3
1,2,3,7,8-PCDD	6.1	6.6	5.7	5.9	5.9
1,2,3,4,7,8-HxCDD	7.7	7.9	12.4	7.4	NR
1,2,3,6,7,8-HxCDD	70.8	70.4	56.0	66.4	68.1
1,2,3,7,8,9-HxCDD	8.6	8.9	7.2	9.8	10.2
1,2,3,4,6,7,8-HpCDD	124.1	115.0	77.2	102.9	81.7
OCDD	970.8	944.7	608.7	690.6	650.9
TEQ_D- WHO_{98}	18.6	19.6	16.8	18.4	17.9
CDF Congeners					
2,3,7,8-TCDF	2.0	0.6	0.2	0.6	0.1
1,2,3,7,8-PCDF	0.1	0.3	0 (ND)	0.2	0 (ND)
2,3,4,7,8-PCDF	5.4	6.4	5.6	5.9	5.6
1,2,3,4,7,8-HxCDF	8.1	8.0	6.8	7.4	6.6
1,2,3,6,7,8-HxCDF	5.0	5.6	4.4	5.1	4.9
1,2,3,7,8,9-HpCDF	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0 (ND)
2,3,4,6,7,8-HxCDF	3.2	4.0	2.6	4.0	2.5
1,2,3,4,6,7,8-HpCDF	19.9	18.0	13.5	18.9	14.6
1,2,3,4,7,8,9-HpCDF	0.1	0.3	0.2	0 (ND)	0 (ND)
OCDF	0.6	0.8	0 (ND)	1.0	0 (ND)
TEQ_F-WHO_{98}	4.7	5.2	4.3	4.9	4.4
Coplanar PCB Congeners					
77	5.9	3.1	0 (ND)	4.4	NR
81	0 (ND)	0 (ND)	0 (ND)	0 (ND)	0.4
126	17.2	17.6	13.2	15.4	15.1
169	16.3	20.8	18.5	18.2	17.9
WHO_{98} TEQ_P	1.9	2.0	1.5	1.7	1.7

Source: ADH (1995) and Cranmer (1996).

Table 4-17. Congener-specific Average Concentrations for 29 North Carolina Adults

	North Carolina Adults, n = 29, sampled in 1996
CDD Congeners	
2,3,7,8-TCDD	2.38
1,2,3,7,8-PCDD	4.51
1,2,3,4,7,8-HxCDD	3.46
1,2,3,7,8,9-HxCDD	3.99
1,2,3,4,6,7,8-HpCDD	54.04
OCDD	391.3
TEQ_D-WHO_{98}	8.22
CDF Congeners	
2,3,7,8-TCDF	1.01
1,2,3,7,8-PCDF	1.16
2,3,4,7,8-PCDF	6.26
1,2,3,4,7,8-HxCDF	5.44
1,2,3,6,7,8-HxCDF	4.67
2,3,4,6,7,8-HxCDF	1.66
1,2,3,7,8,9-HxCDF	1.37
1,2,3,4,6,7,8-HpCDF	11.77
1,2,3,4,7,8,9-HpCDF	1.32
OCDF	2.80
TEQ_F-WHO_{98}	4.74
Coplanar PCB Congeners	
77*	51.00
81*	4.11
126*	17.95
169*	14.95
WHO_{98} TEQ_P	2.00

* PCBs 77 and 81 were not detected in any sample, so the concentrations shown are the average of ½ detection limit for the 29 samples. PCBs 126 and 169 were detected in most of the samples, so the average concentrations calculated at ½ detection limits reported above are very similar to average concentrations calculated at ND = 0.

Source: Masten (2000).

Table 4-18. Results of CDC Compilation of Blood Data from Six Study Sites (all results in pg/g lipid; n = 316)

Congener	Mean	75th Percentile	90th Percentile	95th Percentile
CDD Congeners				
2,3,7,8-TCDD	2.1	2.7	3.5	4.2
1,2,3,7,8-PCDD	5.2	6.5	7.8	9.2
1,2,3,4,7,8-HxCDD	6.2	7.8	10.9	12.0
1,2,3,6,7,8-HxCDD	73.1	87.6	116.9	127.3
1,2,3,7,8,9-HxCDD	7.1	8.8	10.7	12.6
1,2,3,4,6,7,8-HpCDD	79.2	94.9	131.3	161.5
OCDD	664.0	793.6	1084.7	1394.0
CDF Congeners				
2,3,7,8-TCDF	0.7	0.9	1.2	1.5
1,2,3,7,8-PCDF	0.8	1.0	1.4	1.7
2,3,4,7,8-PCDF	6.2	7.5	10.2	12.2
1,2,3,4,7,8-HxCDF	6.5	7.8	10.5	12.2
1,2,3,6,7,8-HxCDF	5.3	6.2	8.4	9.8
1,2,3,7,8,9-HxCDF	0.7	0.8	1.2	1.4
2,3,4,6,7,8-HxCDF	2.2	2.6	3.3	4.0
1,2,3,4,6,7,8-HpCDF	13.2	15.4	21.2	25.8
1,2,3,4,7,8,9-HpCDF	1.3	1.5	2.1	2.6
OCDF	2.1	2.6	3.3	4.0
Coplanar PCB Congeners				
77	31.1	32.6	51.7	72.7
81	3.2	3.9	5.4	6.9
126	18.1	21.8	32.2	45.8
169	19.4	25.1	32.7	37.7
Toxic Equivalent Concentrations for the Entire Data Base*				
TEQ_{DFP}-WHO_{98}	22.1	26.7	33.9	38.8

* This TEQ concentration was derived separately from the congener profile, and cannot be derived from the profile. See text for more detail.

Table 4-19. CDD/CDF Levels in Human Tissues in North America (ppt TEQ_{DF}-WHO_{98}, lipid basis) (late 1980s to early 1990s)

	2,3,7,8-TCDD	1,2,3,7,8-PECDD	Total HXCDD	1,2,3,4,6,7,8-HPCDD	OCDD	2,3,7,8-TCDF	1,2,3,7,8-PECDF	2,3,4,7,8-PECDF	Total HXCDF	1,2,3,4,6,7,8-HPCDF	OCDF	Total TEQ*
ADIPOSE TISSUE												
NHATS, U.S. EPA, 1991a U.S. (n = 865; 48 composites)	5.4	10.7	8.7	1.1	0.072	0.19	0.016	4.9	1.4	0.16	0.0002	32.6
Patterson et al., 1994 U.S. (n = 4)	4.4	11.6	11.6	0.56	0.045	0.11	-	1.9	0.95	0.12	-	31.3
Schecter, 1991 U.S. (n = 15)	6.9	7.7	6.6	0.83	0.043	0.16	-	3.4	1.1	0.16	0.00005	26.8
Schecter, 1991 Canada (n = 46)	7.1	11	9.7	1.5	0.095	-	-	8.5	1.8	0.3	-	40.0
MEAN	6.0	10.3	9.1	1.00	0.064	0.15	0.02	4.7	1.3	0.19	0.0001	32.7
SD	1.1	1.5	1.8	0.35	0.021	0.03	0.00	2.5	0.33	0.07	0.0001	
WEIGHTED MEAN	5.5	10.7	8.7	1.1	0.073	0.19	0.02	5.0	1.4	0.17	0.0002	32.8
BLOOD												
Cole et al., 1995 Canada (n = 132; 14 composites)	4.4	9.9	8.5	1.1	0.053	0.18	-	8.3	3.0	0.12	-	35.8
Schecter et al., 1993 U.S. (n = 5; composite)	3.4	7.0	8.1	1.6	0.12	0.3	0.1	3.5	2.1	0.5	0.001	26.4
Schecter et al., 1993 U.S. (n = 50)	3.8	9.2	9.1	1.2	0.08	0.2	0.1	4.4	2.3	0.23	0.001	30.9
Schecter et al., 1994a U.S. (n = 100)	5.2	21.0	11.2	1.9	0.12	0.31	0.14	6.5	3.3	0.36	0.0004	50.0
MEAN	4.20	11.8	9.3	1.4	0.093	0.27	0.08	5.7	2.7	0.26	0.0008	35.8
SD	0.68	5.4	1.2	0.32	0.028	0.04	0.04	1.9	0.48	0.06	0.0003	
WEIGHTED MEAN	4.5	13.6	9.6	1.4	0.081	0.28	0.11	6.9	3.0	0.28	0.0006	39.8
HUMAN MILK												
Schecter et al., 1989b U.S. (n = 42)	3.3	6.7	4.2	0.42	0.023	0.29	0.023	3.65	1	0.043	0.0004	19.7
ALL TISSUE TYPES												
MEAN	4.9	10.5	8.7	1.1	0.072	0.23	0.06	5.00	1.9	0.20	0.001	32.7
SD	1.3	4.1	2.2	0.45	0.032	0.07	0.04	2.2	0.80	0.09	0.0004	
WEIGHTED MEAN	5.2	11.2	8.8	1.2	0.073	0.21	0.03	5.4	1.8	0.19	0.0003	34.0

* Sum of mean TEQ_{DF}-WHO_{98} concentrations for all congeners.

Table 4-20. CDD/CDF Levels in Human Tissues in Europe and Japan (ppt TEQ_{DF}-WHO_{98}, lipid basis) (ate 1980s to early 1990s)

	2,3,7,8-TCDD	1,2,3,7,8-PeCDD	Total HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDF	Total HxCDF	1,2,3,4,6,7,8-HpCDF	OCDF	Total TEQ*
ADIPOSE TISSUE												
Beck et al., 1994 Germany (n=20)	7.2	21.0	11.9	1.0	0.059	0.25	0.02	20.0	3.6	0.2	0.00004	65.2
Gonzalez et al., 1993 Spain (n=17)	3.3	10.7	9.2	1.9	0.13	0.39	0.02	12.7	7.3	0.32	0.0072	45.9
Schecter, 1991 Germany (n=4)	5.1	21.5	10.9	1.5	0.065	0.39	-	35.4	3.8	0.23	0.00042	78.9
Schecter, 1991 Japan (n=6)	6.6	13.0	8.6	0.69	0.14	0.31	-	6.5	6.9	0.71	-	43.4
MEAN	5.6	16.6	10.2	1.3	0.098	0.34	0.02	18.7	5.4	0.37	0.003	58.4
SD	1.5	4.8	1.32	0.46	0.036	0.06	0.00	10.8	1.7	0.20	0.003	
WEIGHTED MEAN	5.5	16.3	10.4	1.3	0.095	0.32	0.02	17.0	5.4	0.31	0.003	56.6
BLOOD												
Schecter et al., 1992a Germany (n=102)	3.6	13.8	7.6	0.92	0.061	0.23	0.1	18.5	3.5	0.25	0.00042	48.5
Schecter et al., 1992a Japan (n=50-100)	3.2	11.7	6.1	0.59	0.14	0.51	0.038	10.3	2.5	0.13	0.00031	35.1
MEAN	3.4	12.8	6.8	0.76	0.10	0.37	0.07	14.4	3.0	0.19	0.0004	41.8
SD	0.20	1.1	0.78	0.17	0.040	0.14	0.03	4.10	0.49	0.06	0.0001	
WEIGHTED MEAN	3.4	12.9	7.0	0.78	0.095	0.35	0.07	15.0	3.1	0.20	0.0004	42.9

Table 4-20. CDD/CDF Levels in Human Tissues in Europe and Japan (ppt TEQ_{DF} = WHO_{98}, lipid basis) (late 1980s to early 1990s) (continued)

	2,3,7,8-TCDD	1,2,3,7,8-PeCDD	Total HxCDD	1,2,3,4,6,7,8-HpCDD	OCDD	2,3,7,8-TCDF	1,2,3,7,8-PeCDF	2,3,4,7,8-PeCDF	Total HxCDF	1,2,3,4,6,7,8-HpCDF	OCDF	Total TEQ*
HUMAN MILK												
Bates et al., 1994 New Zealand (n=37)	5.1	7.4	4.0	0.52	0.021	0.089	-	2.7	0.85	0.071	-	20.7
Beck et al., 1994 Germany (n=112)	3.6	12.0	6.6	0.51	0.034	0.25	0.05	10	1.9	0.084	0.00016	35.0
Furst et al., 1994 Germany (n=526)	3.2	10.1	5.1	0.41	0.021	0.17	0.025	13.4	1.8	0.055	0.00014	34.2
Pluim et al., 1994b The Netherlands (n=35)	3.8	10.6	5.7	0.54	0.030	0.2	0.01	11.0	1.6	0.061	0.00013	33.5
Schecter et al., 1989b Germany (n=185)	3	9.3	4.6	0.46	0.019	0.2	0.035	12	1.6	0.052	0.00099	31.3
Schecter et al. 1989c Japan (n=6)	4.5	4.6	3.9	0.62	0.098	0.3	0.053	12.8	0.94	0.040	-	27.7
Startin et al., 1989 United Kingdom (n=80)	5.6	13.0	7.0	0.71	0.027	0.12	0.02	11	1.7	0.083	0.00069	39.2
Tuinstra et al., 1994 The Netherlands (n=200)	4.1	11.5	6.2	0.63	0.079	0.09	0.03	11.3	1.7	0.077	0.00013	35.7
Van Cleuvenbergen et al., 1994 Belgium (n=9)	4.2	11.9	5.04	0.81	0.027	0.13	0.045	15.6	2.2	0.18	0.00034	40.1
MEAN	4.2	10.0	5.3	0.58	0.041	0.17	0.04	11.1	1.6	0.08	0.0004	33.1
SD	0.83	3.5	1.1	0.12	0.028	0.07	0.01	3.5	0.43	0.04	0.0003	-
WEIGHTED MEAN	3.6	10.5	5.4	0.50	0.033	0.16	0.03	11.9	1.7	0.06	0.0003	34.0
ALL TISSUE TYPES												
MEAN	4.4	12.1	6.9	0.81	0.066	0.24	0.04	13.7	2.9	0.18	0.001	41.0
SD	1.3	4.2	2.4	0.40	0.043	0.12	0.02	7.3	1.9	0.17	0.002	-
WEIGHTED MEAN	3.6	11.0	5.8	0.56	0.043	0.19	0.04	12.5	2.0	0.09	0.0004	35.8

* Sum of mean TEQ concentrations for all congeners.

Table 4-21. PCB Levels in Human Tissues in North America (ppt TEQ_P-WHO_{98}, lipid basis) (late1980s to early 1990s)

PCB Congeners	77	105	114	118	123	126	156	157	167	169	189	Total TEQ*
ADIPOSE TISSUE												
Mes and Weber, 1989 Canada (n = 1)	0.0003	-	-	-	-	2.0	-	-	-	0.0016	-	2.0
Patterson et al., 1994 U.S. (n = 28)	0.0012	-	-	-	-	13.5	-	-	-	0.69	-	14.2
Schecter el al., 1989a U.S. (n = 3)	-	6.0	-	1.5	-	-	4.0	-	-	-	-	11.5
Williams and LeBel, 1991 Canada (n = 62)	-	-	-	-	-	26.7	-	-	-	1.6	-	28.3
MEAN	0.0008	6.00	-	1.50	-	14.1	4.0	-	-	0.76	-	26.3
SD	0.0004	0.00	-	0.00	-	10.1	0.00	-	-	0.65	-	
WEIGHTED MEAN	0.0012	6.00	-	1.50	-	22.4	4.0	-	-	1.3	-	35.2
BLOOD												
Cole et al., 1995 Canada (n = 7; pooled from 132)	0.013	-	-	-	-	6.9	-	-	-	0.57	-	7.5
Dewailly et al., 1994 Canada (n = 10-57)	-	-	-	2.5 (n = 51)	-	4.8 (n = 10)	-	-	-	0.29 (n = 10)	-	7.6
Kang et al., 1997 U.S. (n = 14-16)	-	-	-	-	-	1.8 (n = 14)	-	-	-	0.27 (n = 16)	-	2.1
Patterson et al., 1994 U.S. (n = 2,3, pooled from 240)	0.010	0.72	-	7.9	-	3.95	-	-	-	0.33	-	12.9
Schecter et al., 1993 U.S. (n = 1, pooled from 5)	0.003	0.32	-	1.1	-	5.0	2.1	-	-	0.3	-	8.9
Schecter et al., 1993 U.S. (n = 50)	0.008	0.69	-	1.6	-	10.4	3.0	-	-	0.46	-	16.2
MEAN	0.009	0.58	-	3.3	-	5.5	2.6	-	-	0.37	-	12.3
SD	0.004	0.18	-	2.7	-	2.7	0.45	-	-	0.11	-	-
WEIGHTED MEAN	0.011	0.71	-	6.1	-	5.5	2.9	-	-	0.41	-	15.6

Table 4-21. PCB Levels in Human Tissues in North America (ppt TEQ_P-WHO_{98}, lipid basis) (late 1980s to early 1990s) (continued)

PCB Congeners	77	105	114	118	123	126	156	157	167	169	189	**Total TEQ***
HUMAN MILK												
Dewailly et al., 1994 Canada (n = 96; pooled to 16)	0.0008	-	-	1.7	-	8.0	-	-	-	0.33	-	10.1
Hong et al., 1992 U.S. (n = 5)	0.034	0.64	0.75	2.6	0.017	5.8	2.2	0.50	0.011	0.58	0.04	13.2
Mes and Weber, 1989 Canada (n = "several" pooled samples)	0.0012	-	-	-	-	5.1	-	-	-	0.006	-	5.1
She et al., 1995 U.S. (n = 12)	0.0007	1.7	-	3.8	-	5.8	2.8	-	-	0.15	-	14.3
MEAN	0.0009	1.7	0.75	2.7	0.02	6.1	2.5	0.50	0.01	0.27	0.04	14.2
SD	0.014	0.53	0.00	0.84	0.00	1.1	0.31	0.00	0.00	0.21	0.00	-
WEIGHTED MEAN	0.002	1.4	0.75	2.0	0.02	7.7	2.7	0.50	0.01	0.32	0.04	15.3
ALL TISSUE TYPES												
MEAN	0.007	1.7	0.75	2.9	0.02	7.7	2.8	0.50	0.01	0.43	0.04	16.7
SD	0.010	2.0	0.00	2.1	0.00	6.3	0.68	0.0	0.00	0.39	0.00	-
WEIGHTED MEAN	0.009	0.79	0.75	5.1	0.02	8.2	2.9	0.50	0.01	0.52	0.04	18.8

* Sum of mean TEQ concentrations for all congeners.

Table 4-22 . PCB Levels in Human Tissues in Europe (ppt TEQ_P-WHO_{98}, lipid basis, using WHO TEFs) (late 1980s to early 1990s)

PCB Congeners	77	105	114	118	123	126	156	157	167	169	189	**Total TEQ***
ADIPOSE TISSUE												
Beck et al., 1989 Germany (n = 7)	0.006	-	-	-	-	-	-	-	-	-	-	0.006
HUMAN MILK												
Beck et al., 1989 Germany (n = 10)	0.0022	-	-	-	-	-	-	-	-	-	-	0.0022
Georgii et al., 1995 Germany (n = 68)	-	-	-	4.4	-	-	13.5	-	-	-	-	17.9
Johansen et al., 1994 Norway (n = 28)	0.046	0.77	2.0	2.6	-	15.6	5.8	0.8	-	1.9	-	29.6
Noren et al., 1990 Sweden (n = 2)	0.0024	1.2	-	2.8	-	12.4	10.1	-	-	0.86	-	27.4
Noren and Lunden, 1991 Sweden (n = 6,7; pooled from 120,140)	0.0027	0.65	-	-	-	9.8	7.2	-	-	0.47	-	18.1
Koopman-Esseboom et al., 1994 The Netherlands (n = 195)	0.002	0.9	-	3.6	-	15.2	10.5	-	-	0.8	-	31.0
Van der Velde et al., 1994 The Netherlands (n = "several")	0.0014	-	-	-	-	8.8	-	-	-	0.55	-	9.4
Dwarka et al., 1995 United Kingdom (n = 193)	-	-	-	2.3	-	-	-	-	-	-	-	2.3
Startin et al., 1989; Duarte-Davidson et al., 1992 United Kingdom (n = 6; pooled from 57)	-	0.99	-	1.8	-	-	-	-	-	-	-	2.8
MEAN	0.009	0.89	2.0	2.9	-	12.4	9.4	0.80	-	0.92	-	29.3
SD	0.016	0.17	0.00	0.86	-	2.8	2.7	0.00	-	0.52	-	-
WEIGHTED MEAN	0.006	0.83	2.0	3.0	-	13.1	9.7	0.80	-	0.76	-	30.2
BREAST TISSUE												
Dahl et al., 1994 Sweden (n = 16)	-	1.1	1.5	5.5	-	-	17.0	2.4	-	-	0.38	27.8
ALL TISSUE TYPES												
MEAN	0.009	0.93	1.7	3.3	-	12.4	10.7	1.6	-	0.92	0.38	31.9
SD	0.015	0.18	0.28	1.2	-	2.8	3.8	0.78	-	0.52	-	

Table 4-23. Weighted Mean CDD/CDF Profiles for Human Tissues from Studies in the 1980s and Early 1990s

2,3,7,8-Substituted CDD/CDFs	Adipose Tissue[a]		Blood[b]		Human Milk[c]	
	Concentration (ppt, lipid)	Fraction of Total 2,3,7,8-substituted CDD/CDFs	Concentration (ppt, lipid)	Fraction of Total 2,3,7,8-substituted CDD/CDFs	Concentration (ppt, lipid)	Fraction of Total 2,3,7,8-substituted CDD/CDFs
2,3,7,8-TCDD	5.49	0.0055	4.54	0.0040	3.30	0.0093
1,2,3,7,8-PeCDD	10.7	0.0107	13.6	0.0119	6.70	0.0188
1,2,3,4,7,8-HxCDD	3.82	0.0038	9.93	0.0086	4.95	0.0139
1,2,3,6,7,8-HxCDD	70.6	0.0711	73.0	0.0636	30.5	0.0856
1,2,3,7,8,9-HxCDD	12.7	0.0128	13.0	0.0113	6.20	0.0174
1,2,3,4,6,7,8-HpCDD	111.4	0.1121	138.1	0.1202	42.0	0.1178
OCDD	725.6	0.7306	811.7	0.7069	233.0	0.6537
2,3,7,8-TCDF	1.89	0.0019	2.77	0.0024	2.85	0.0080
1,2,3,7,8-PeCDF	0.32	0.0003	1.20	0.0010	0.45	0.0013
2,3,4,7,8-PeCDF	12.1	0.0122	13.9	0.0121	7.30	0.0205
1,2,3,4,7,8-HxCDF	5.89	0.0059	12.6	0.0110	5.55	0.0156
1,2,3,6,7,8-HxCDF	9.24	0.0093	8.22	0.0072	3.20	0.0090
1,2,3,7,8,9-HxCDF	--	--	6.93	0.0062	1.85	0.0050
2,3,4,6,7,8-HxCDF	--	--	3.54	0.0031	0.25	0.0007
1,2,3,4,6,7,8-HpCDF	21.6	0.0218	25.2	0.0219	4.00	0.0112
1,2,3,4,7,8,9-HpCDF	--	--	4.27	0.0037	0.25	0.0007
OCDF	1.97	0.0020	5.74	0.0050	4.10	0.0115
TOTAL	993.2	1.0	1,148.2	1.0	356.5	1.0

[a] Based on data from Patterson et al. (1994); Schecter (1991); and U.S. EPA (1991a).
[b] Based on data from Schecter et al. (1993, 1994a), and Cole et al. (1995).
[c] Based on data from Schecter et al. (1992b).

Table 4-24. Estimated Dose Based on Congener-Specific Half-Lives and Adipose Tissue TEQ_{DF}-WHO_{98} Concentrations, and Pharmocokinetic Modeling

	½ Life [d]	Adipose Tissue Conc. (ppt TEQ_{DF}-WHO_{98})	Dose [e,f] (pg/day)
2,3,7,8-TCDD	7.2	2.1	7.8
1,2,3,7,8-PECDD	15.7	5.2	8.8
1,2,3,4,7,8-HXCDD	8.4	0.62	2.0
1,2,3,6,7,8-HXCDD	13.1	7.3	14.8
1,2,3,7,8,9-HXCDD	4.9	0.71	3.9
1,2,3,4,6,7,8-HPCDD	3.7	0.79	5.7
OCDD	6.7	0.066	0.26
2,3,7,8-TCDF	7.2	0.07	0.26
1,2,3,7,8-PECDF	15.7	0.04	0.07
2,3,4,7,8-PECDF	19.6	3.1	4.2
1,2,3,4,7,8-HXCDF	6.2	0.65	2.8
1,2,3,6,7,8-HXCDF	6	0.53	2.4
1,2,3,7,8,9-HXCDF	6	0.070	0.3
2,3,4,6,7,8-HXCDF	5.8	0.22	1.0
1,2,3,4,6,7,8-HPCF	3	0.13	1.2
1,2,3,4,7,8,9-HPCDF	3.2	0.013	0.11
OCDF	6.7	0.00021	0.0008
TOTAL TEQ_{DF}-WHO_{98}		21.6	87

[a] Represents the mean half-life for all 2,3,7,8-substituted congeners in this class.
[b] Half-life for this congener not available; half-life assumed to be the same as for the CDD with the same chlorination pattern.
[c] No half-life data available for this congener; assumed to be the same as for 1,2,3,6,7,8-HxCDF
[d] Half-life data from Flesch-Janys et al. (1996).
[e] Assumes a body fat volume of 17.5 kg.
[f] Dose = [(ln2/T 0.5 yrs)*17.5 kg*Conc. (pg/g)*(1,000 g/kg)*(1 yr/365 days) / (0.8 absorption).

Table 4-25. Predicted Average Daily Intake of 2,3,7,8-TCDD by the General Population of the United States

Media	Predicted Media Concentration[a]	Media Intake (person/day)	Daily Intake of 2,3,7,8-TCDD (pg/day)	Percent of Daily Intake
Inhalation	0.02 (pg/m^3)	20 (m^3)	0.4	1.1
Water	0.003 (pg/L)	1.33 L[b]	0.004	0.01
Soil ingestion	0.96 (ng/kg)	20 mg	0.02	0.05
Food				
Produce	0.06 (ng/kg)	20 g[b]	1.2	3.4
Milk and dairy products				
Beef	0.03 (ng/kg)	266 g[b]	8.0	23.0
Fish	0.20 (ng/kg)	90 g[b]	18.0	51.7
Eggs	0.38 (ng/kg)	18 g[b]	6.7	19.3
	0.01 (ng/kg)	25 g[b]	0.5	1.4
TOTAL			34.8	100

[a] Values predicted by the Fugacity Food Chain model.

[b] Inferred consumption rate calculated by dividing reported daily intake (column 4) by predicted concentration (column 2).

Source: Travis and Hattemer-Frey (1991).

Table 4-26. Predicted Average Daily Intake of 2,3,7,8-TCDD from Foods by the General Population of the United States

Media	2,3,7,8-TCDD Concentration in Food (ng/kg)	Food Intake (g/person/day)	Daily Intake of 2,3,7,8-TCDD (pg/day)	Percent of Daily Intake
Milk	0.0018	108.9	0.20	1.2
Cream	0.0072	2.0	0.01	<0.1
Sour cream	0.010	0.7	0.01	<0.1
Cheese	0.016	19.4	0.31	1.9
Ice cream	0.0055	7.5	0.04	0.3
Butter	0.044	2.6	0.11	0.7
Cottage cheese	0.0021	5.5	0.01	<0.1
Meats	0.035	187	6.55	41.2
Ocean fish	0.500	17.2	8.6	54.1
Coffee	0.0001	363.6	0.04	0.3
Orange juice	0.0002	33.5	0.01	<0.1
TOTAL			15.9	100

Source: Henry et al. (1992).

Table 4-27. Daily Exposure to 2,3,7,8-TCDD and I-TEQ_{DF} from Air, Soil, Food, and Nonfood in The Netherlands

Media	Media Intake (g/person/day)	Daily Intake of 2,3,7,8-TCDD (pg/day)	Daily Intake of I-TEQ_{DF} (pg/day)
Air inhaled	20 m^3 [a]	0.05	2
Air ingested (particulates)	[b]	0.025	1
Soil dermal	150 mg	0.004	0.15
Soil ingested		0.003	0.10
Uptake from air and soil		0.08	3.2
Leafy vegetables	27 g	0.2-2	1.8-7
Pork	15 g fat	0.45	4.2
Beef	5 g fat	3	13
Chicken and eggs	2.5 g fat	0.6	4.8
Milk	8 g fat	3.2	17
Cheese, butter	12.5 g fat	5	26
Sea fish	0.4 g fat	2	14
Freshwater fish	0.4 g fat	4	10
Fish oil	5.5 g	1.1	7.2
Vegetable oil	40 g	NDA	14
Intake from food		19.5-21.3	112-117
Intake from paper food packaging		NDA	9.1
TOTAL INTAKE		19.6-21.4	121-126

[a] Intake rate could not be determined from Theelen (1991).

[b] Assumes exposure of 2,000 cm^2 of skin to 1 mg of soil/cm^2. Soil concentrations assumed to be 7,000 mg I-TEQ_{DF}/kg and 175 mg of 2,3,7,8-TCDD/kg. Dermal absorption of 1 percent assumed.

NDA = No data available.

Source: Theelen (1991).

Table 4-28. Estimated Lifetime Average Daily Exposure of Canadians to Dioxin I-TEQ_{DF}

Media	Daily Intake of Dioxin[a] (I-TEQ_{DF}) (pg/day)		
	Adult A[b]	Adult B[c]	Adult C[d]
Food	132 - 282	291 - 441	132 - 282
Air	3.5	3.5	12
Soil	1.75 - 1.90	1.75 - 1.90	1.75 - 1.90
Water	<0.7 - 3.5	<0.7 - 3.5	<0.7 - 3.5
Consumer Products	<0.7	<0.7	<0.7
Total Estimated Lifetime Intake[e]	140 - 290	300 - 450	150 - 300

[a] These estimates represent the lifetime average daily intake calculated by dividing the total estimated intakes for each life stage (i.e., adult, child, infant, neonate) by the 70-year exposure period. The estimates in this table are based on the upper range of average national values and conservative assumptions that overestimate rather than underestimate exposures. These estimates are only approximations and not absolute values.

[b] Adult a is an average 70-kg adult consuming average amounts of air (20 m^3/day), water (1 liter/day), and soil (20 mg/day). Food intakes based on Nutrition Canada 1977 survey.

[c] Adult B is similar to Adult a except that consumption of fish contaminated with CDDs and CDFs is in excess of current Canadian guidelines.

[d] Adult C is similar to Adult a except that he/she lives in close proximity to an incineration/combustion source.

[e] These estimates have been rounded off because of the uncertainty in the data.

Source: Gilman and Newhook (1991).

Table 4-29. Estimated Upper Bound Dietary Intakes of CDD/CDFs by the Average UK Consumer in 1982 and 1992

Food Group	1982			1992		
	Consumption (kg/person/day) Mean	CDD/CDF Concentration (ng I-TEQ_{DF}/kg fresh weight) Mean	CDD/CDF Intake (pg I-TEQ_{DF}/person/day) Mean	Consumption (kg/person/day) Mean	CDD/CDF Concentration (ng I-TEQ_{DF}/kg fresh weight) Mean	CDD/CDF Intake (pg I-TEQ_{DF}/person/day) Mean
Bread	0.125	0.02	3	0.118	0.03	4
Other Cereal Products	0.105	0.13	14	0.098	0.17	17
Carcass Meat	0.032	0.49	16	0.029	0.13	4
Offals (internal organs)	0.002	1.57	3	0.001	0.59	1
Meat Products	0.048	0.32	15	0.046	0.08	3
Poultry	0.017	0.50	8	0.018	0.13	2
Fish	0.016	0.41	7	0.014	0.21	3
Oils and Fats	0.030	1.26	38	0.031	0.20	6
Eggs	0.024	0.92	22	0.017	0.17	3
Milk	0.303	0.16	48	0.293	0.06	17
Milk Products	0.055	1.20	66	0.056	0.16	9
TOTAL	--	--	240	--	--	69

Note: Estimated total dietary intakes were calculated before rounding.

Source: MAFF (1995).

Table 4-30. Estimated CDD/CDF Mean Background Exposures for Adults in the United States

Media	Conc. TEQ_{DF}-WHO_{98}[a]	Contact Rate[b]	Daily Intake[c] (mg/kg-day)	Daily Intake (pg/kg-day)	% of Total
Soil ingestion	9.3 ppt[e]	50 mg/day	6.6×10^{-12}	6.6×10^{-3}	1.1
Soil dermal contact	9.3 ppt	12 mg/day[f]	1.6×10^{-12}	1.6×10^{-3}	0.3
Freshwater fish and shellfish ingestion	1.0 ppt[i]	5.9 g/day	8.4×10^{-10}	8.4×10^{-2}	13.9
Marine fish and shellfish ingestion	0.26 ppt[i]	9.6 g/day	3.6×10^{-11}	3.6×10^{-2}	5.9
Inhalation	0.12 pg/m^3	13.3 m^3/day	2.3×10^{-11}	2.3×10^{-2}	3.7
Water ingestion	0.00056 ppq	1.4 L/day	1.1×10^{-14}	1.1×10^{-5}	<0.01
Milk ingestion	0.018 ppt	175 g/day	4.5×10^{-11}	4.5×10^{-2}	7.4
Dairy ingestion	0.12 ppt	55 g/day	9.4×10^{-11}	9.4×10^{-2}	15.5
Eggs ingestion	0.081 ppt	0.24 g/kg/day	1.9×10^{-11}	1.9×10^{-2}	3.2
Beef ingestion	0.18 ppt	0.71 g/kg/day	1.3×10^{-10}	1.3×10^{-1}	21.0
Pork ingestion	0.28 ppt	0.22 g/kg/day	6.2×10^{-11}	6.2×10^{-2}	10.1
Poultry ingestion	0.068 ppt	0.50 g/kg/day	3.4×10^{-11}	3.4×10^{-2}	5.6
Other meat ingestion	0.18 ppt[g]	0.35 g/kg/day[h]	6.2×10^{-11}	6.2×10^{-2}	10.1
Vegetable fat ingestion	0.056 ppt[e]	17 g/day	1.4×10^{-11}	1.4×10^{-2}	2.2
	Total		6.1×10^{-10}	6.1×10^{-1} [d]	100.0

[a] Values from Table 3-64.

[b] Values for adult soil ingestion, inhalation, water ingestion, and eggs, beef pork, and poultry ingestion from Exposure Factors Handbook (U.S. EPA, 1997). Contact rates for milk, dairy, and vegetable fats are based on data from USDA (1995). Contact rates for fish from U.S. EPA (2000).

[c] Daily intake (mg/kg-day) = [Contact rate (g/day; m^3/day; L/day; mg/day) x Conc. TEQ x Unit Conversion (soil unit conversion = 10^{-12}, all other media unit conversion = 10^{-9})/Body Weight (kg)] or Contact rate (g/kg-day) x Conc. TEQ x Unit Conversion.

[d] Approximately equivalent to 43 pg/day, assuming an adult body weight of 70 kg.

[e] Calculated by setting nondetects to zero.

[f] Calculated as the surface area of the body that contacts the soil (5,700 cm^2/day) x the rate that soil adheres to the skin (0.07 mg/cm^2) x the fraction of CDD/CDFs absorbed through the skin (0.03); exposure factors based on recommendations in U.S. EPA (1999) for an adult resident, which assumes that the lower legs, forearms, hands, and head are exposed to the soil.

[g] Estimated as the average of beef, pork, and poultry.

[h] Calculated as the total meat intake rate minus the intake rates for beef, pork, and poultry (U.S. EPA, 1997).

[i] This concentration is a species-specific ingestion-weighted average value.

Table 4-31. Estimated Dioxin-Like PCB Mean Background Exposures for Adults in the United States

Media	Conc. WHO98-TEQ[a]	Contact Rate[b]	Daily Intake[c] (mg/kg-day)	Daily Intake (pg/kg-day)	% of Total
Soil ingestion	2.3 ppt[e]	50 mg/day	1.6×10^{-12}	1.6×10^{-3}	0.5
Soil dermal contact	2.3 ppt	12 mg/day[f]	3.9×10^{-13}	3.9×10^{-4}	0.1
Freshwater fish and shellfish ingestion	1.2 ppt	5.9 g/day	1.0×10^{-10}	1.0×10^{-1}	30.9
Marine fish and shellfish ingestion	0.25 ppt	9.6 g/day	3.4×10^{-11}	3.4×10^{-2}	10.5
Inhalation	--	--	--	--	--
Water ingestion	--	--	--	--	--
Milk ingestion	0.0088 ppt	175 g/day	2.2×10^{-11}	2.2×10^{-2}	6.7
Dairy ingestion	0.058 ppt	55 g/day	4.6×10^{-11}	4.6×10^{-2}	13.9
Eggs ingestion	0.10 ppt	0.24 g/kg/day	2.4×10^{-11}	2.4×10^{-2}	7.3
Beef ingestion	0.084 ppt	0.71 g/kg/day	6.0×10^{-11}	6.0×10^{-2}	18.2
Pork ingestion	0.012 ppt	0.22 g/kg/day	2.6×10^{-12}	2.6×10^{-3}	0.8
Poultry ingestion	0.026 ppt	0.50 g/kg/day	1.3×10^{-11}	1.3×10^{-2}	4.0
Other meat ingestion	0.041[g]	0.35 g/kg/day[h]	1.4×10^{-11}	1.4×10^{-2}	4.3
Vegetable fat ingestion	0.037 ppt	17 g/day	9.0×10^{-12}	9.0×10^{-3}	2.7
	Total		3.3×10^{-10}	3.3×10^{-1} [d]	100.0

[a] Values from Table 3-64.

[b] Values for adult soil ingestion, eggs, beef pork, and poultry ingestion from Exposure Factors Handbook (U.S. EPA, 1997). Contact rates for milk, dairy, and vegetable fats are based on data from USDA (1995). Contact rates for fish from U.S. EPA (2000).

[c] Daily intake (mg/kg-day) = [Contact rate (g/day; m^3/day; L/day; mg/day) x Conc. TEQ x Unit Conversion (soil unit conversion = 10^{-12}, all other media unit conversion = 10^{-9})/Body Weight (kg)] or Contact rate (g/kg-day) x Conc. TEQ x Unit Conversion.

[d] Approximately equivalent to 23 pg/day, assuming an adult body weight of 70 kg.

[e] Calculated by setting nondetects to zero.

[f] Calculated as the surface area of the body that contacts the soil (5,700 cm^2/day) x the rate that soil adheres to the skin (0.07 mg/cm^2) x the fraction of CDD/CDFs absorbed through the skin (0.03); exposure factors based on recommendations in U.S. EPA (1999) for an adult resident, which assumes that the lower legs, forearms, hands, and head are exposed to the soil.

[g] Estimated as the average of beef, pork, and poultry.

[h] Calculated as the total meat intake rate minus the intake rates for beef, pork, and poultry (U.S. EPA, 1997).

Table 4-32. Comparison of Adult Contact Rates, TEQ_{DF} Concentrations, and Background Exposure Estimates from the 1994 Draft and Current Version of This Document

Media	Previous I-TEQ_{DF} Concentration	Current TEQ_{DF}-WHO_{98} Concentration	Previous Contact Rate	Current Contact Rate	Previous Daily Intake Rate (pg/kg-day)	Current Daily Intake Rate (pg/kg-day)
Soil Ingestion	8.0 ppt[a]	9.3 ppt[b]	100 mg/day	50 mg/day	1.1×10^{-2}	6.6×10^{-3}
Soil Dermal Contact	--	9.3 ppt	--	12 mg/day	--	1.6×10^{-3}
Freshwater Fish and Shellfish Ingestion	1.2 ppt	1.0 ppt[c]	6.5 g/day	5.9 g/day	1.1×10^{-1}	8.4×10^{-2}
Marine Fish and Shellfish Ingestion	--	0.26 ppt[c]	--	9.6 g/day	--	3.6×10^{-2}
Inhalation	0.095 pg/m^3	0.12 pg/m^3	23 m^3/day	13.3 m^3/day	3.1×10^{-2}	2.3×10^{-2}
Water Ingestion	0.0056 ppq	0.00056 ppq	1.4 L/day	1.4 L/day	1.1×10^{-4}	1.1×10^{-5}
Milk Ingestion	0.07 ppt	0.016 ppt	251 g/day	175 g/day	2.5×10^{-1}	4.5×10^{-2}
Dairy Ingestion	0.36 ppt	0.12 ppt	67 g/day	55 g/day	3.4×10^{-1}	9.4×10^{-2}
Eggs Ingestion	0.14 ppt	0.081 ppt	29 g/day	0.24 g/kg/day	5.8×10^{-2}	1.9×10^{-2}
Beef Ingestion	0.48 ppt	0.18 ppt	77 g/day	0.71 g/kg/day	5.3×10^{-1}	1.3×10^{-1}
Pork Ingestion	0.26 ppt	0.28 ppt	47 g/day	0.22 g/kg/day	1.7×10^{-1}	6.2×10^{-2}
Poultry Ingestion	0.19 ppt	0.068 ppt	68 g/day	0.50 g/kg/day	1.8×10^{-1}	3.4×10^{-2}
Other Meat Ingestion	--	0.18 ppt	--	0.35 g/kg/day	--	6.2×10^{-2}
Vegetable Ingestion	--	0.056 ppt	--	17 g/day	--	1.4×10^{-2}
TOTAL	--	--	--	--	1.7×10^{0} (119 pg/day)	6.1×10^{-1} (43 pg/day)

a Rural/pristine background sites
b Urban background sites
c This concentration is a species-specific ingestion-weighted average value.

Table 4-33. Background Exposures via Consumption of German Food

Food	I-TEQ_{DF}[a] concentration (fat basis)	Intake Rate[b] (g fat/day)	TCDD - Equivalent[a] (pg/day)
Cow's milk	1.35	6.0	8.1
Cheese	0.98	5.2	5.1
Butter	0.66	12	7.9
Beef	1.69	10	16.9
Veal	3.22	0.1	0.3
Pork	<0.4	14	5.6
Chicken	1.41	1	1.4
Canned meat	1.29	2	2.6
Lard	0.47	1.5	0.7
Salad oil	<0.4	5	1
Margarine	<0.4	14	2.8
Fish and Fish Products		1.8	27
Freshwater fish	13.25		
Saltwater fish	16.82		
Fish oil	2.64		
Cod liver oil	13.31		
Total I-TEQ_{DF}			**79.4**

[a] Milk data based on Fürst et al. (1991); other data based on Fürst et al. (1990).
[b] Based on data reported by Fürst et al. (1990).

Table 4-34. Comparison of Contact Rates and Background TEQ_{DF}-WHO_{98} Exposures for Three Age Groups of Children to Adults

Media	TEQ_{DF}-WHO_{98} Concentrations (whole weight)	Age 1-5 Years[a]		Age 6-11 Years[b]		Age 12-19 Years[c]		Adult[d]	
		Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)
Soil Ingestion	9.3 ppt[g]	100 mg/day	6.2×10^{-2}	50 mg/day	1.6×10^{-2}	50 mg/day	8.0×10^{-3}	50 mg/day	6.6×10^{-3}
Soil Dermal Contact	9.3 ppt[g]	2.2 mg/day[e]	1.3×10^{-3}	3.2 mg/day[e]	9.8×10^{-4}	11 mg/day[e]	1.8×10^{-3}	12 mg/day[e]	1.6×10^{-3}
Freshwater Fish and Shellfish Ingestion	1.0 ppt[h]	1.5 g/day[f]	1.0×10^{-1}	1.9 g/day[f]	6.3×10^{-2}	2.3 g/day[f]	4.0×10^{-2}	5.9 g/day	8.4×10^{-2}
Marine Fish and Shellfish Ingestion	0.26 ppt[h]	2.5 g/day[f]	4.3×10^{-2}	3.1 g/day[f]	2.7×10^{-2}	3.7 g/day[f]	1.7×10^{-2}	9.6 g/day	3.6×10^{-2}
Inhalation	0.12 pg/m^3	7.5 m^3/day	6.0×10^{-2}	12 m^3/day	4.8×10^{-2}	14 m^3/day	2.9×10^{-2}	13.3 m^3/day	2.3×10^{-2}
Water Ingestion	0.00056 ppq	0.69 L/day	2.6×10^{-5}	0.79 L/day	1.5×10^{-5}	0.97 L/day	9.4×10^{-6}	1.4 L/day	1.1×10^{-5}
Milk Ingestion	0.018 ppt	348 g/day	4.2×10^{-1}	357 g/day	2.1×10^{-1}	308 g/day	9.6×10^{-2}	175 g/day	4.5×10^{-2}
Dairy Ingestion	0.12 ppt	103 g/day	8.2×10^{-1}	88 g/day	3.5×10^{-1}	77 g/day	1.6×10^{-1}	55 g/day	9.4×10^{-2}
Eggs Ingestion	0.081 ppt	0.75 g/kg/day	6.1×10^{-2}	0.41 g/kg/day	3.3×10^{-2}	0.24 g/kg/day	1.9×10^{-2}	0.24 g/kg/day	1.9×10^{-3}
Beef Ingestion	0.18 ppt	1.4 g/kg/day	2.5×10^{-1}	1.1 g/kg/day	2.0×10^{-1}	0.83 g/kg/day	1.5×10^{-1}	0.67 g/kg/day	1.3×10^{-1}
Pork Ingestion	0.28 ppt	0.48 g/kg/day	1.3×10^{-1}	0.35 g/kg/day	9.8×10^{-2}	0.27 g/kg/day	7.6×10^{-2}	0.22 g/kg/day	6.2×10^{-2}
Poultry Ingestion	0.068 ppt	1.1 g/kg/day	7.5×10^{-2}	0.87 g/kg/day	5.9×10^{-2}	0.56 g/kg/day	3.8×10^{-2}	0.49 g/kg/day	3.4×10^{-2}
Other Meats Ingestion	0.18 ppt	1.1 g/kg/day	1.9×10^{-1}	0.69 g/kg/day	1.2×10^{-1}	0.42 g/kg/day	7.4×10^{-2}	0.35 g/kg/day	6.2×10^{-2}
Vegetable Fat Ingestion	0.056 ppt[g]	4 g/day	1.5×10^{-2}	9 g/day	1.7×10^{-2}	12 g/day	1.2×10^{-2}	17 g/day	1.4×10^{-2}
TOTAL	--	--	2.2×10^{0} (34 pg/day)	--	1.3×10^{0} (37 pg/day)	--	7.2×10^{-1} (42 pg/day)	--	6.1×10^{-1} (43 pg/day)

a 15 kg body weight assumed
b 30 kg body weight assumed
c 58 kg body weight assumed
d 70 kg body weight assumed
e Dermal contact rates based on the calculation: skin surface area contacting soil (cm^2/day) x soil adherence rate (mg/cm^2) x absorption fraction (0.03). Exposure factor values based on recommended data and procedures in U.S. EPA (1999) for adult and child residents. For all ages it was assumed that the head, hands, lower legs, and forearms were exposed to soil. Adherence factors for ages 1-5 years and 6-11 years were calculated using data for children playing in dry soil. For ages 12-19 years and adults, a gardening scenario was assumed. Surface areas were assumed to be 2,400, 3,500, 5,300, and 5,700 cm^2/day for ages 1-5 years, 6-11 years, 12-19 years, and adults, respectively. Adherence factors for these age groups were estimated to be 0.03, 0.03, 0.07, and 0.07 mg/cm^2, respectively.
f Fish intake rates for children based on data in Table 10-46 of EPA's Exposure Factors Handbook (U.S. EPA, 1997). Total fish intake values apportioned among various fish categories based on the proportions for adults.
g Calculated by setting nondetects to zero.
h This concentration is a species-specific ingestion-weighted average value.

NOTE: Contact rates derived from U.S. EPA (1997) except for milk, dairy, and vegetable fats which were derived from USDA (1995). Dairy intake is assumed to be intake of total milk and milk products minus fluid milk intake.

Table 4-35. Comparison of Contact Rates and Background TEQ_P-WHO_{98} Exposures for Three Age Groups of Children to Adults

Media	TEQ_P-WHO_{98} Concentrations (whole weight)	Age 1-5 Years[a]		Age 6-11 Years[b]		Age 12-19 Years[c]		Adult[d]	
		Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)	Contact Rate	Daily Intake (pg/kg-day)
Soil Ingestion	2.3	100 mg/day	1.5×10^{-2}	50 mg/day	3.8×10^{-3}	50 mg/day	2.0×10^{-3}	50 mg/day	1.6×10^{-3}
Soil Dermal	2.3	2.2 mg/day	3.3×10^{-4}	3.2 mg/day	2.4×10^{-4}	11 mg/day	4.4×10^{-4}	12 mg/day	3.9×10^{-4}
Freshwater Fish and Shellfish Ingestion	1.2 ppt	1.5 g/day[e]	1.2×10^{-2}	1.9 g/day[e]	7.6×10^{-2}	2.3 g/day[e]	4.8×10^{-2}	5.9 g/day	1.0×10^{-1}
Marine Fish and Shellfish Ingestion	0.25 ppt	2.5 g/day[e]	4.2×10^{-2}	3.1 g/day[e]	2.6×10^{-2}	3.7 g/day[e]	1.6×10^{-2}	9.6 g/day	3.4×10^{-2}
Inhalation	--	7.5 m^3/day	--	11 m^3/day	--	14 m^3/day	--	13.3 m^3/day	--
Water Ingestion	--	0.7 L/day	--	0.8 L/day	--	1.0 L/day	--	1.4 L/day	--
Milk Ingestion	0.0088 ppt	348 g/day	2.0×10^{-1}	357 g/day	1.1×10^{-1}	308 g/day	4.7×10^{-2}	175 g/day	2.2×10^{-2}
Dairy Ingestion	0.058 ppt	103 g/day	4.0×10^{-1}	88 g/day	1.7×10^{-1}	77 g/day	7.7×10^{-2}	55 g/day	4.6×10^{-2}
Eggs Ingestion	0.10 ppt	0.75 g/kg/day	7.5×10^{-2}	0.41 g/kg/day	4.1×10^{-2}	0.24 g/kg/day	2.4×10^{-2}	0.24 g/kg/day	2.4×10^{-2}
Beef Ingestion	0.084 ppt	1.4 g/kg/day	1.2×10^{-1}	1.1 g/kg/day	9.2×10^{-2}	0.83 g/kg/day	7.0×10^{-2}	0.71 g/kg/day	6.0×10^{-2}
Pork Ingestion	0.012 ppt	0.48 g/kg/day	5.8×10^{-3}	0.35 g/kg/day	4.2×10^{-2}	0.27 g/kg/day	3.2×10^{-3}	0.22 g/kg/day	2.6×10^{-3}
Poultry Ingestion	0.026 ppt	1.1 g/kg/day	2.9×10^{-2}	0.87 g/kg/day	2.3×10^{-2}	0.56 g/kg/day	1.5×10^{-2}	0.50 g/kg/day	1.3×10^{-2}
Other Meats Ingestion	0.041 ppt	1.1 g/kg/day	4.5×10^{-2}	0.69 g/kg/day	2.8×10^{-2}	0.42 g/kg/day	1.7×10^{-2}	0.35 g/kg/day	1.4×10^{-2}
Vegetable Fat Ingestion	0.037 ppt	4 g/day	9.9×10^{-3}	9 g/day	1.1×10^{-2}	12 g/day	7.7×10^{-3}	17 g/day	9.0×10^{-3}
TOTAL	--	--	1.1×10^{0} (16 pg/day)	--	5.8×10^{-1} (17 pg/day)	--	3.3×10^{-1} (19 pg/day)	--	3.3×10^{-1} (23 pg/day)

a 15 kg body weight assumed
b 30 kg body weight assumed
c 58 kg body weight assumed
d 70 kg body weight assumed
e Fish intake rates for children based on data in Table 10-46 of EPA's Exposure Factors Handbook (U.S. EPA, 1997). Total fish intake values apportioned among various fish categories based on the proportions for adults.

NOTE: Contact rates derived from U.S. EPA (1997) except for milk, dairy, and vegetable fats which were derived from USDA (1995). Dairy intake is assumed to be intake of total milk and milk products minus fluid milk intake.

Table 4-36. Percentage TEQ_{DFP}-WHO_{98} Contribution of Each Media to Total Dose by Age Group

Media	CDD/CDFs				PCBs			
	1-5 Years	6-11 Years	12-19 Years	Adult	1-5 Years	6-11 Years	12-19 Years	Adult
Soil Ingestion	2.8	1.2	1.1	1.1	1.4	0.7	0.6	0.5
Soil Dermal Contact	0.06	0.08	0.2	0.3	0.03	0.04	0.1	0.1
Freshwater Fish and Shellfish	4.5	5.1	5.5	13.9	11.3	13.1	14.6	30.9
Marine Fish and Shellfish	1.9	2.2	2.3	5.9	3.9	4.5	4.9	10.5
Inhalation	2.7	3.8	4.0	3.7	--	--	--	--
Water	0.001	0.001	0.001	0.002	--	--	--	--
Milk	18.7	17.2	13.3	7.4	19.2	18.1	14.3	6.7
Dairy	36.8	28.2	22.2	15.5	37.5	29.3	23.6	13.9
Eggs	2.7	2.7	2.7	3.2	7.1	7.1	7.4	7.3
Beef	11.3	15.9	20.8	21.0	11.1	15.9	21.4	18.2
Pork	6.0	7.9	10.5	10.1	0.5	0.7	1.0	0.8
Poultry	3.3	4.7	5.3	5.6	2.7	3.9	4.5	4.0
Other Meat	8.6	9.7	10.3	10.1	4.2	4.8	5.2	4.3
Vegetable Fat	0.7	1.3	1.6	2.2	0.9	1.9	2.3	2.7

Table 4-37. Variability in Fat Intake from the Bogalusa Heart Study

Age (Years)	Total Fat Intake (g) 1973-1982 Data[a]			Animal Fat Intake (g) 1973-1982 Data[a]			Total Fat Intake (g) 1992-1994 Data[b]			Total Fat Intake (g) 1988-1991 Data[c]		
	Mean	Maximum	Max/Mean	Mean	Maximum	Max/Mean	Mean	Mean + 3SD	Mean + 3SD/Mean	Mean	Mean + 3SD	Mean + 3SD/Mean
0.5	37.1	107.6	2.9	18.4	61.1	3.3	--	--	--	--	--	--
1	59.1	152.7	2.6	36.5	127.1	3.5	--	--	--	--	--	--
2	86.7	236.4	2.7	49.5	153.1	3.1	--	--	--	--	--	--
3	91.6	232.5	2.5	50.1	182.6	3.6	--	--	--	--	--	--
4	98.6	584.6	5.9	50.8	242.2	4.8	--	--	--	--	--	--
10	93.2	529.5	5.7	54.1	412.3	7.6	84.6	205.8	2.4	--	--	--
13	107.0	282.2	2.6	56.2	209.6	3.7	--	--	--	--	--	--
15	97.7	251.3	2.6	53.8	182.1	3.4	--	--	--	--	--	--
17	107.8	327.4	3.0	64.4	230.0	3.6	--	--	--	--	--	--
19-28	--	--	--	--	--	--	--	--	--	98.5	290.2	2.9

a Frank et al. (1986)
b Nicklas et al. (1993)
c Nicklas et al. (1995)

Table 4-38. Fat Intake (g/day) Among the Adult U.S. Population, Based on Data from the 1987 NHIS

Age (yrs)	Men				Women			
	N	Mean (g/day)	SD [a] (g/day)	Mean + 35D / Mean	N	Mean (g/day)	SD [a] (g/day)	Mean + 35D / Mean
18-34	3,166	116.5	69.5	2.9	4,296	67.6	32.8	2.5
35-49	2,346	103.6	48.4	2.4	2,923	65.4	43.3	3.0
50-64	1,512	90.2	46.7	2.6	2,092	57.8	27.4	2.4
65-79	1,148	76.0	40.7	2.6	1,926	50.7	26.3	2.6
80+	213	73.8	39.4	2.6	521	50.5	18.3	2.1

a Standard deviation calculated from standard error (SE) as follows: SD = SE x $\sqrt{n}$.

Table 4-39. Estimated CDD/CDF Upper Percentile Background Exposures for Adults in the United States

Media	Conc. TEQ_{DF}-WHO_{98}[a]	Contact Rate[b]	Daily Intake[c] (mg/kg-day)	Daily Intake (pg/kg-day)	% of Total
Soil ingestion	9.3 ppt[e]	100 mg/day	1.3×10^{-11}	1.3×10^{-2}	1.2
Soil dermal contact	9.3 ppt	51.3 mg/day[f]	6.8×10^{-12}	6.8×10^{-3}	0.6
Freshwater fish and shellfish ingestion	1.0 ppt[g]	10.3 g/day	1.5×10^{-10}	1.5×10^{-1}	13.3
Marine fish and shellfish ingestion	0.26 ppt	16.7 g/day	6.2×10^{-11}	6.2×10^{-2}	5.6
Inhalation	0.12 pg/m^3	15.2 m^3/day	2.6×10^{-11}	2.6×10^{-2}	2.4
Water ingestion	0.00056 ppq[g]	2.0 L/day	1.6×10^{-14}	1.6×10^{-5}	<0.01
Milk ingestion	0.018 ppt	421 g/day	1.9×10^{-10}	1.9×10^{-1}	16.8
Dairy ingestion	0.12 ppt	132 g/day	2.3×10^{-10}	2.3×10^{-1}	20.4
Eggs ingestion	0.081 ppt	0.39 g/kg/day	3.2×10^{-11}	3.2×10^{-2}	2.9
Beef ingestion	0.18 ppt	0.93 g/kg/day	1.7×10^{-10}	1.7×10^{-1}	15.1
Pork ingestion	0.28 ppt	0.30 g/kg/day	8.4×10^{-11}	8.4×10^{-2}	7.6
Poultry ingestion	0.068 ppt	0.68 g/kg/day	4.6×10^{-11}	4.6×10^{-2}	4.2
Other meats ingestion	0.18 ppt	0.48 mg/kg/day	8.6×10^{-11}	8.6×10^{-2}	7.8
Vegetable fat ingestion	0.056 ppt[e]	28.8 g/day	2.3×10^{-11}	2.3×10^{-2}	2.1
	Total		1.1×10^{-9}	$1.1 \times 10^{+0}$ [d]	100.0

[a] Values from Table 3-64.

[b] Values for adult soil ingestion based on data in U.S. EPA (1991b). Inhalation rate based on data for males in Exposure Factors Handbook (U.S. EPA, 1997). Water ingestion rate based on high-end value in U.S. EPA (1997). Contact rates for fish, milk, dairy, eggs, meats, and vegetable fats are based on data from an unpublished analysis of USDA's 1994-1996 CSFII data conducted by EPA.

[c] Daily intake (mg/kg-day) = [Contact rate (g/day; m^3/day; L/day; mg/day) x Conc. TEQ x Unit Conversion (soil unit conversion = 10^{-12}, all other media unit conversion = 10^{-9})/Body Weight (kg)] or Contact rate (g/kg-day) x Conc. TEQ x Unit Conversion.

[d] Approximately equivalent to 77 pg/day, assuming an adult body weight of 70 kg.

[e] Calculated by setting nondetects to zero.

[f] Calculated as the surface area of the body that contacts the soil (5,700 cm^2/day) x the rate that soil adheres to the skin (0.30 mg/cm^2) x the fraction of CDD/CDFs absorbed through the skin (0.03); exposure factors based on recommendations in U.S. EPA (1999) for an adult resident, which assumes that the lower legs, forearms, hands, and head are exposed to the soil.

[g] This concentration is a species-specific ingestion-weighted average value.

Table 4-40. Estimated Dioxin-Like PCB Upper Percentile Background Exposures for Adults in the United States

Media	Conc. WHO98-TEQ[a]	Contact Rate[b]	Daily Intake[c] (mg/kg-day)	Daily Intake (pg/kg-day)	% of Total
Soil ingestion	2.3 ppt[e]	100 mg/day	3.3×10^{-12}	3.3×10^{-3}	0.5
Soil dermal contact	2.3 ppt	51.3 mg/day	1.7×10^{-12}	1.7×10^{-3}	11.1
Freshwater fish and shellfish ingestion	1.2 ppt	10.3 g/day	1.8×10^{-10}	1.8×10^{-1}	28.5
Marine fish and shellfish ingestion	0.25 ppt	16.7 g/day	6.0×10^{-11}	6.0×10^{-2}	9.6
Inhalation	--	--	--	--	--
Water ingestion	--	--	--	--	--
Milk ingestion	0.0088 ppt	421 g/day	9.6×10^{-11}	9.6×10^{-2}	15.5
Dairy ingestion	0.058 ppt	132 g/day	1.1×10^{-10}	1.1×10^{-1}	17.6
Eggs ingestion	0.10 ppt	0.39 g/kg/day	3.9×10^{-11}	3.9×10^{-2}	6.3
Beef ingestion	0.084 ppt	0.93 g/kg/day	7.8×10^{-11}	7.8×10^{-2}	12.6
Pork ingestion	0.012 ppt	0.30 g/kg/day	3.6×10^{-12}	3.6×10^{-3}	0.6
Poultry ingestion	0.026 ppt	0.68 g/kg/day	1.8×10^{-11}	1.8×10^{-2}	2.9
Other meats ingestion	0.041 ppt	0.48 mg/kg/day	2.0×10^{-11}	2.0×10^{-2}	3.2
Vegetable fat ingestion	0.037 ppt	28.8 g/day	1.5×10^{-11}	1.5×10^{-2}	2.5
	Total		6.2×10^{-10}	6.2×10^{-1} [d]	100.0

[a] Values from Table 3-64.

[b] Contact rates for fish, milk, dairy, eggs, meats, and vegetable fats are based on data from an unpublished analysis of USDA's 1994-1996 CSFII data conducted by EPA.

[c] Daily intake (mg/kg-day) = [Contact rate (g/day; m^3/day; L/day; mg/day) x Conc. TEQ x Unit Conversion (unit conversion = 10^{-9})/Body Weight (kg)] or Contact rate (g/kg-day) x Conc. TEQ x Unit Conversion.

[d] Approximately equivalent to 43 pg/day, assuming an adult body weight of 70 kg.

[e] Calculated by setting nondetects to zero.

[f] Calculated as the surface area of the body that contacts the soil (5,700 cm^2/day) x the rate that soil adheres to the skin (0.07 mg/cm^2) x the fraction of CDD/CDFs absorbed through the skin (0.03); exposure factors based on recommendations in U.S. EPA (1999) for an adult resident, which assumes that the lower legs, forearms, hands, and head are exposed to the soil.

Table 4-41. Comparisons of Predicted Average Daily Intake of 2,3,7,8-TCDD and Total TEQ_{DF}s

Location	Daily Intake of 2,3,7,8-TCDD (pg/day)	Daily Total TEQ_{DF} intake (pg/day)	Media
United States[a]	34.8	--	beef, milk, produce, fish, eggs, water, inhalation
United States[b]	15.9	--	dairy, meat, fish
North America[c]	5.5	43	dairy, eggs, meat, poultry, fish, inhalation, soil ingestion, soil dermal contact
Canada[d]		140-290	air, water, soil, food
Germany[e]		85 (79)	dairy, meats, fish
Germany[f]	25.0	158	dairy, meat, fish
Netherlands[g]	20.0	121-126	dairy, meat, poultry, fish
United Kingdom[h]	--	69	meat, fish, dairy, poultry, eggs, milk products, breads, and cereals
United Kingdom[i]	--	175.5	meat, fish, dairy, poultry, eggs, milk products, breads, and cereals
Spain[j]	--	210	vegetables, lentils and beans, cereals, fruit, fish, meat, eggs, dairy, milk, and oil

[a] Travis and Hattemer-Frey (1991)
[b] Henry et al. (1992)
[c] Current Assessment; TEF_{DF}-WHO_{98}s used
[d] Gilman and Newhook (1991); I-TEF_{DF}s used
[e] Fürst et al. (1990); value in parentheses is the corrected I-TEQ_{DF} value based on the milk data from Fürst et al. (1991); I-TEF_{DF}s used
[f] Fürst et al. (1991); I-TEF_{DF}s used
[g] Theelen (1991); I-TEF_{DF}s used
[h] MAFF (1995); data from 1992; I-TEF_{DF}s used
[l] Jacobs and Mobbs (1997) I-TEF_{DF}s used
[j] Schuhmacher et al. (1997) and Domingo et al. (1999); I-TEF_{DF}s used

Table 4-42. Example of the Calculation of the Picograms of TEQ_{DF}-WHO_{98} Contributed by Individual CDD/CDF Congeners for the Beef Consumption Pathway

Congener	Average congener concentration, pg/g lipid	Average TEQ_{DF}-WHO_{98} concentration, pg/g lipid	Fraction of TEQ_{DF}-WHO_{98} contributed by each congener[1]	TEQ_{DF}-WHO_{98} contributions to the diet by each congener (pg/day)[2]
2378-TCDD	0.052	0.052	0.049	0.43
12378-PCDD	0.35	0.35	0.33	2.9
123478-HxCDD	0.46	0.064	0.044	0.39
123678-HxCDD	1.4	0.14	0.13	1.2
123789-HxCDD	0.53	0.053	0.050	0.45
1234678-HpCDD	4.5	0.045	0.042	0.38
OCDD	4.8	0.00050	0.00045	0.0040
2378-TCDF	0.030	0.0030	0.0030	0.026
12378-PCDF	0.31	0.016	0.015	0.13
23478-PCDF	0.36	0.18	0.17	1.5
123478-HxCDF	0.55	0.055	0.051	0.46
123678-HxCDF	0.40	0.040	0.038	0.33
234678-HxCDF	0.31	0.031	0.030	0.26
123789-HxCDF	0.39	0.039	0.036	0.33
1234678-HpCDF	1.0	0.01	0.0093	0.084
1234789-HpCDF	0.31	0.0031	0.0030	0.026
OCDF	1.9	0.00019	0.00018	0.0016
TOTAL		1.06	1.00	8.9

[1] This is calculated as the picograms TEQ_{DF}-WHO_{98} contributed by each congener divided by the total TEQ_{DF}-WHO_{98} concentration. For example, the 0.049 for 2,3,7,8-TCDD is calculated as 0.052/1.06.

[2] Picograms contributed by each congener = (0.18 pg/g) (0.71 g/kg/day) (70 kg) (TEQ_{DF}-WHO_{98} fraction), where 0.18 pg/g is whole weight beef concentration as derived in Section 4.4.2, 0.71 g/kg/day is the consumption rate, 70 kg is the average adult body weight, and the TEQ_{DF}-WHO_{98} fraction is shown in the fourth column above, just preceding this final column of results.

Table 4-43. Average Concentrations (not on a TEQ_{DF}-WHO_{98} basis) and the Fraction of TEQ_{DF}-WHO_{98} Contributed by Each CDD/CDF Congener for the Various Food Groups

	Beef		Pork		Chicken		Other Meat		Dairy		Milk		Fresh Fish		Marine Fish	
Congener	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac
2378-TCDD	0.052	0.049	0.10	0.068	0.16	0.21	0.10	0.094	0.070	0.071	0.070	0.071	3.1	0.18	7.1	0.20
12378-PCDD	0.35	0.33	0.45	0.31	0.24	0.32	0.35	0.31	0.32	0.33	0.32	0.33	5.2	0.31	17	0.47
123478-HxCDD	0.46	0.044	0.52	0.035	0.18	0.024	0.39	0.035	0.39	0.040	0.39	0.040	3.0	0.018	9.0	0.025
123678-HxCDD	1.4	0.13	1.1	0.075	0.40	0.053	0.98	0.089	1.9	0.19	1.9	0.19	5.3	0.032	47	0.13
123789-HxCDD	0.53	0.050	0.47	0.032	0.37	0.049	0.46	0.042	0.55	0.056	0.55	0.056	4.1	0.024	13	0.036
1234678-HpCDD	4.5	0.042	10	0.069	1.5	0.020	5.4	0.049	5.0	0.051	5.0	0.051	24	0.014	52	0.014
OCDD	4.8	0.00045	53	0.0036	5.0	0.00066	21	0.0019	4.9	0.00050	4.9	0.00050	120	0.00071	76	0.00021
2378-TCDF	0.030	0.0030	0.090	0.0061	0.29	0.038	0.14	0.012	0.080	0.0082	0.080	0.0082	14	0.083	11	0.031
12378-PCDF	0.31	0.015	0.45	0.015	0.21	0.014	0.33	0.015	0.050	0.0025	0.050	0.0025	3.8	0.011	3.5	0.0049
23478-PCDF	0.36	0.17	0.56	0.19	0.26	0.17	0.39	0.18	0.28	0.14	0.28	0.14	7.6	0.23	5.1	0.071
123478-HxCDF	0.55	0.051	0.98	0.066	0.22	0.029	0.58	0.053	0.39	0.040	0.39	0.040	1.7	0.010	2.5	0.0069
123678-HxCDF	0.40	0.038	0.58	0.039	0.20	0.026	0.39	0.036	0.25	0.025	0.25	0.025	10	0.060	2.1	0.0058
234678-HxCDF	0.31	0.030	0.57	0.039	0.20	0.026	0.36	0.033	0.28	0.029	0.28	0.029	1.3	0.0077	1.2	0.0033
123789-HxCDF	0.39	0.036	0.45	0.031	0.15	0.020	0.33	0.030	0.050	0.0051	0.050	0.0051	1.3	0.0077	0.21	0.00058
1234678-HpCDF	1.0	0.0094	3.6	0.024	0.26	0.0034	1.6	0.015	0.83	0.0085	0.83	0.0085	16	0.0095	2.1	0.00058
1234789-HpCDF	0.31	0.0030	0.57	0.0039	0.17	0.0022	0.35	0.0032	0.050	0.00051	0.050	0.00051	1.4	0.00083	0.22	0.000061
OCDF	1.9	0.00018	2.3	0.0016	0.33	0.000043	1.5	0.00014	0.050	5.1E-6	0.050	5.1E-6	2.6	0.000016	1.8	0.0000050
TEQ_{DF}-WHO_{98}, pg/g	1.1		1.5		0.76		1.1		0.98		0.98		17		36	

Note:
Conc = average concentration, pg/g lipid for all foods;
Frac = fractional contribution of each congener to TEQ concentration

Table 4-44. The Average Concentrations (not on a TEQ_P-WHO_{98} basis) and the Fraction of TEQ_P-WHO_{98} Contributed by Each Dioxin-Like PCB Congener for the Various Food Groups

Congener	Beef		Pork		Chicken		Other Meat		Dairy		Milk		Fresh Fish		Marine Fish	
	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac	Conc	Frac
PCB 77	0.17	0.00020	0.30	0.0025	0.81	0.0031	0.43	0.0010	1.3	0.0022	0.19	0.0022	25	0.021	6.2	0.0025
PCB 105	16	0.019	6.4	0.054	12	0.046	11	0.027	20	0.035	3.1	0.035	350	0.029	160	0.064
PCB 114	--	--	--	--	--	--	--	--	--	--	--	--	170	0.073	74	0.15
PCB 118	76	0.089	18	0.15	51	0.20	48	0.12	82	0.14	12	0.14	1900	0.16	330	0.13
PCB 123	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
PCB 126	0.69	0.81	0.063	0.53	0.17	0.65	0.31	0.75	0.43	0.74	0.065	0.74	5.5	0.47	0.83	0.34
PCB 156	10	0.058	4.1	0.17	3.9	0.075	6.0	0.073	7.2	0.061	1.1	0.061	390	0.17	83	0.17
PCB 157	2.3	0.013	0.97	0.041	0.98	0.019	1.4	0.017	1.7	0.014	0.25	0.14	210	0.089	68	0.14
PCB 167	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
PCB 169	0.12	0.014	0.049	0.042	0.019	0.0073	0.063	0.015	0.060	0.010	0.0090	0.010	0.7	0.0060	0.2	0.0081
PCB 189	--	--	--	--	--	--	--	--	--	--	--	--	33	0.0028	8.0	0.0032
TEQ_{DF}-WHO_{98}, pg/g	0.084	--	0.012	--	0.026	--	0.041	--	0.058	--	0.088	--	1.2	--	0.25	--

Note:

Conc = average concentration, pg/g whole for all foods;
Frac = fraction contribution of each congener to TEQ_P-WHO_{98} concentration;
blank spaces indicate that no information was available on the concentration

Table 4-45. TEQ_{DF}-WHO_{98} Contribution of Each CDD/F Congener to the Daily Dose for Each Group and Overall (pg/day)

Congener	Beef	Pork	Chicken	Other Meat	Dairy	Milk	Fresh fish	Ocean fish	TOTAL	Fraction
2378-TCDD	0.43	0.29	0.50	0.42	0.47	0.22	1.1	0.49	4.0	0.10
12378-PCDD	2.9	1.3	0.75	1.4	2.2	1.0	1.8	1.2	12.6	0.33
123478-HxCDD	0.39	0.15	0.056	0.16	0.26	0.13	0.11	0.062	1.3	0.034
123678-HxCDD	1.2	0.32	0.13	0.39	1.3	0.61	0.19	0.33	4.4	0.12
123789-HxCDD	0.45	0.14	0.12	0.18	0.37	0.18	0.14	0.090	1.7	0.044
1234678-HpCDD	0.38	0.30	0.047	0.22	0.34	0.16	0.084	0.036	1.6	0.041
OCDD	0.0040	0.015	0.0016	0.0084	0.0033	0.0016	0.0042	0.00053	0.039	0.0010
2378-TCDF	0.026	0.026	0.091	0.055	0.054	0.026	0.49	0.076	0.85	0.022
12378-PCDF	0.13	0.066	0.033	0.065	0.017	0.0080	0.067	0.012	0.40	0.010
23478-PCDF	1.5	0.82	0.41	0.79	0.95	0.45	1.3	0.18	6.4	0.17
123478-HxCDF	0.46	0.29	0.069	0.23	0.26	0.13	0.060	0.017	1.5	0.040
123678-HxCDF	0.34	0.17	0.063	0.16	0.17	0.080	0.35	0.015	1.3	0.035
234678-HxCDF	0.26	0.17	0.063	0.14	0.19	0.090	0.046	0.0083	0.97	0.025
123789-HxCDF	0.33	0.13	0.047	0.13	0.034	0.016	0.046	0.015	0.73	0.019
1234678-HpCDF	0.084	0.10	0.0081	0.064	0.056	0.027	0.056	0.015	0.40	0.010
1234789-HpCDF	0.025	0.017	0.0053	0.014	0.0034	0.0016	0.0049	0.00015	0.073	0.0019
OCDF	0.0016	0.00067	0.00010	0.00060	0.000034	0.000016	0.000091	0.000012	0.0031	0.000082
TOTAL	8.9	4.3	2.4	4.4	6.6	3.2	5.9	2.5	38	
Fraction	0.23	0.11	0.062	0.12	0.17	0.082	0.15	0.065		

Note: The total background dose is estimated to be 43 pg/day. The pathways above add to 38 pg/day, or about 90 percent of total. All numbers above were rounded and may not add up perfectly.

Table 4-46. TEQ_P-WHO_{98} Contribution of Each Coplanar PCB Congener to the Daily Dose for Each Group and Overall (pg/day)

Congener	Beef	Pork	Chicken	Other Meat	Dairy	Milk	Fresh fish	Ocean fish	TOTAL	Fraction
PCB 77	0.00083	0.00047	0.0028	0.0010	0.0070	0.0033	0.015	0.0060	0.037	0.0012
PCB 105	0.078	0.010	0.042	0.028	0.11	0.053	0.21	0.15	0.69	0.033
PCB 114	--	--	--	--	--	--	0.51	0.36	0.87	0.042
PCB 118	0.37	0.028	0.18	0.12	0.45	0.22	1.1	0.32	2.8	0.14
PCB 123	--	--	--	--	--	--	--	--	--	--
PCB 126	3.4	0.099	0.59	0.75	2.4	1.1	3.3	0.80	12	0.61
PCB 156	0.24	0.032	0.068	0.073	0.20	0.094	1.2	0.40	2.3	0.11
PCB 157	0.056	0.0076	0.017	0.017	0.046	0.022	0.63	0.33	1.1	0.055
PCB 167	--	--	--	--	--	--	--	--	--	--
PCB 169	0.059	0.0077	0.0066	0.015	0.033	0.016	0.042	0.019	0.20	0.0097
PCB 189	--	--	--	--	--	--	0.020	0.0077	0.030	0.0013
TOTAL	4.2	0.18	0.91	1.0	3.2	1.5	7.1	2.4	20	--
Fraction	0.20	0.0090	0.044	0.049	0.16	0.075	0.35	0.12	--	--

Note: All numbers above were rounded and may not add up perfectly.

Table 4-47. Average CDD/CDF Concentrations in Human Tissue and Fractional Contribution of CDD/CDF Congeners to Total TEQ_{DF}-WHO_{98} Tissue, Based on CDC Blood Data

Congener	Average	
	conc	frac
2378-TCDD	2.1	0.097
12378-PCDD	5.2	0.24
123478-HxCDD	6.2	0.029
123678-HxCDD	73	0.34
123789-HxCDD	7.1	0.034
1234678-HpCDD	79	0.037
OCDD	664	0.0031
2378-TCDF	0.7	0.0033
12378-PCDF	0.8	0.0019
23478-PCDF	6.2	0.14
123478-HxCDF	6.5	0.030
123678-HxCDF	5.3	0.025
234678-HxCDF	0.7	0.010
123789-HxCDF	2.2	0.0032
1234678-HpCDF	13.2	0.0061
1234789-HpCDF	1.3	0.00060
OCDF	2.1	9.7E-6
TEQ_{DF}-WHO_{98}	21.6	

Note:

conc = Actual, not TEQ_{DF}-WHO_{98}, lipid-based concentration profile in pg/g.
frac = Fractional contribution to TEQ_{DF}-WHO_{98} of each congener.

Table 4-48. Average Coplanar PCB Concentrations in Human Tissue and Percentage Contribution of CDD/F Congeners to Total TEQ_P-WHO_{98} Tissue, Based on CDC Blood Data

Congener	Average	
	conc	frac
PCB 77	31	0.0016
PCB 81	3.2	0.00016
PCB 105	--	--
PCB 114	--	--
PCB 118	--	--
PCB 123	--	--
PCB 126	18	0.90
PCB 156	--	--
PCB 157	--	--
PCB 167	--	--
PCB 169	19	0.095
PCB 189	--	--
TEQ_P- WHO_{98}	2.0	--

Note:

conc = Lipid-based concentration profile in pg/g; and
frac = fractional contribution to TEQ_P-WHO_{98} of each congener.

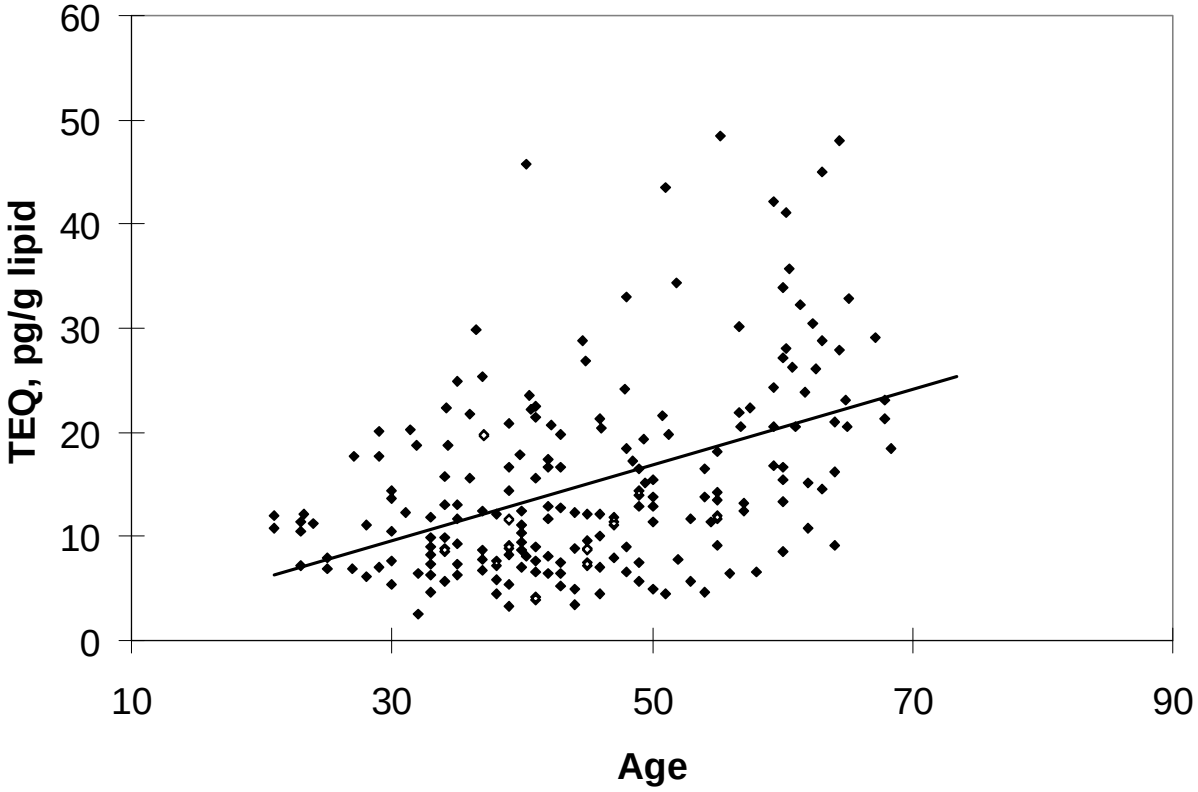


Figure 4-1. TEQ (I-TEQ for CDD/CDF + WHO_{94} for a Subset of Four Dioxin-Like PCBs) Lipid Concentrations for a Comparison Population and the Population of Mossville, Louisiana, as a Function of Age

Source: ATSDR, 1999b.

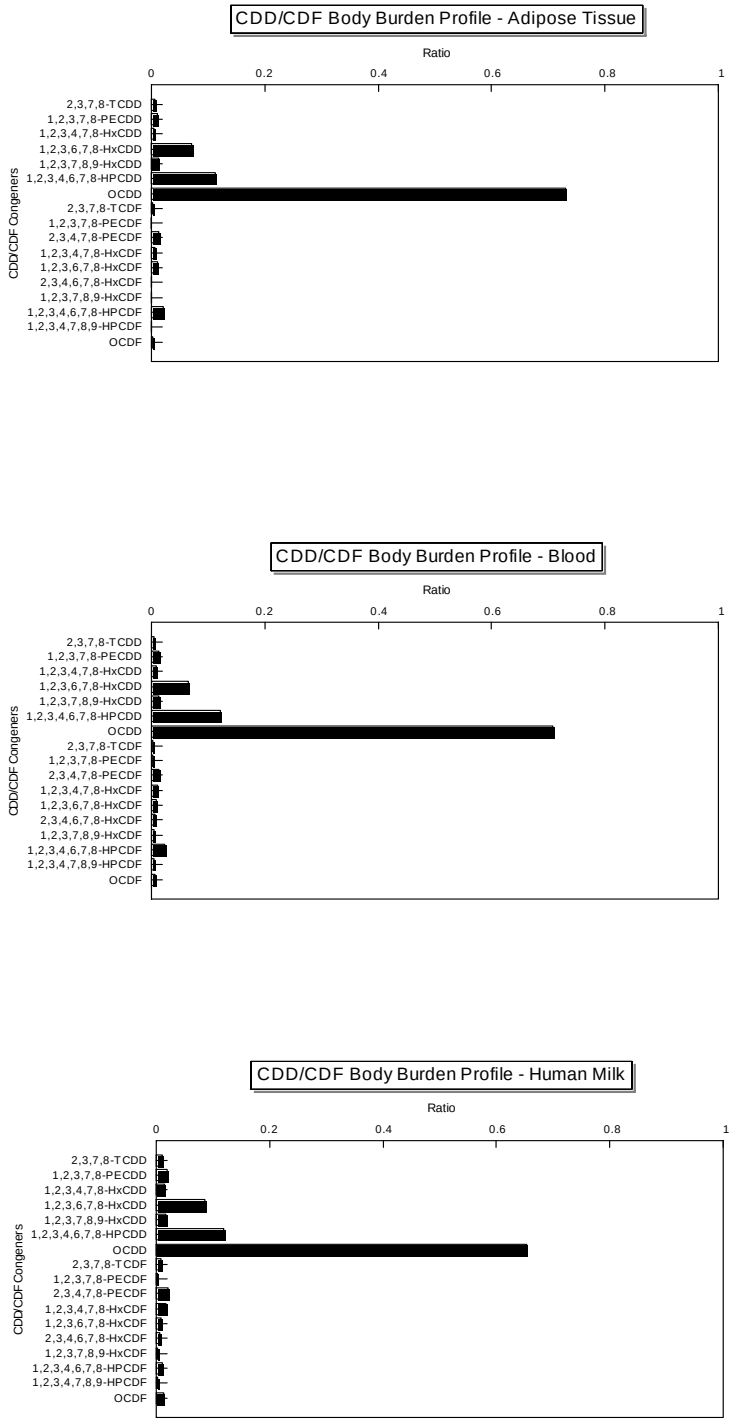


Figure 4-2. CDD/CDF Profiles for Adipose Tissue, Blood and Human Milk Based on Literature Studies from the 1980s to the Early 1990s

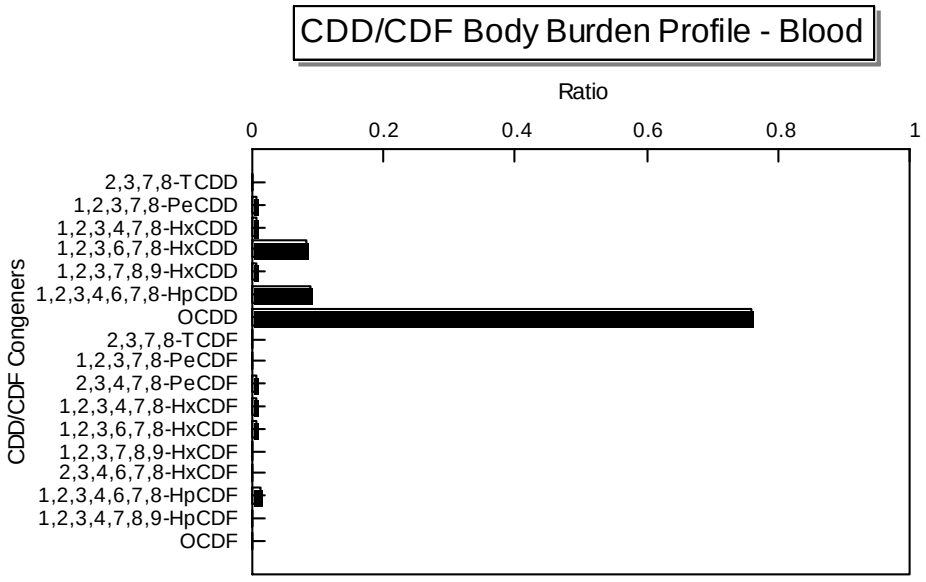


Figure 4-3. Congener Profile for the CDC Blood Data Set (1995-1997)

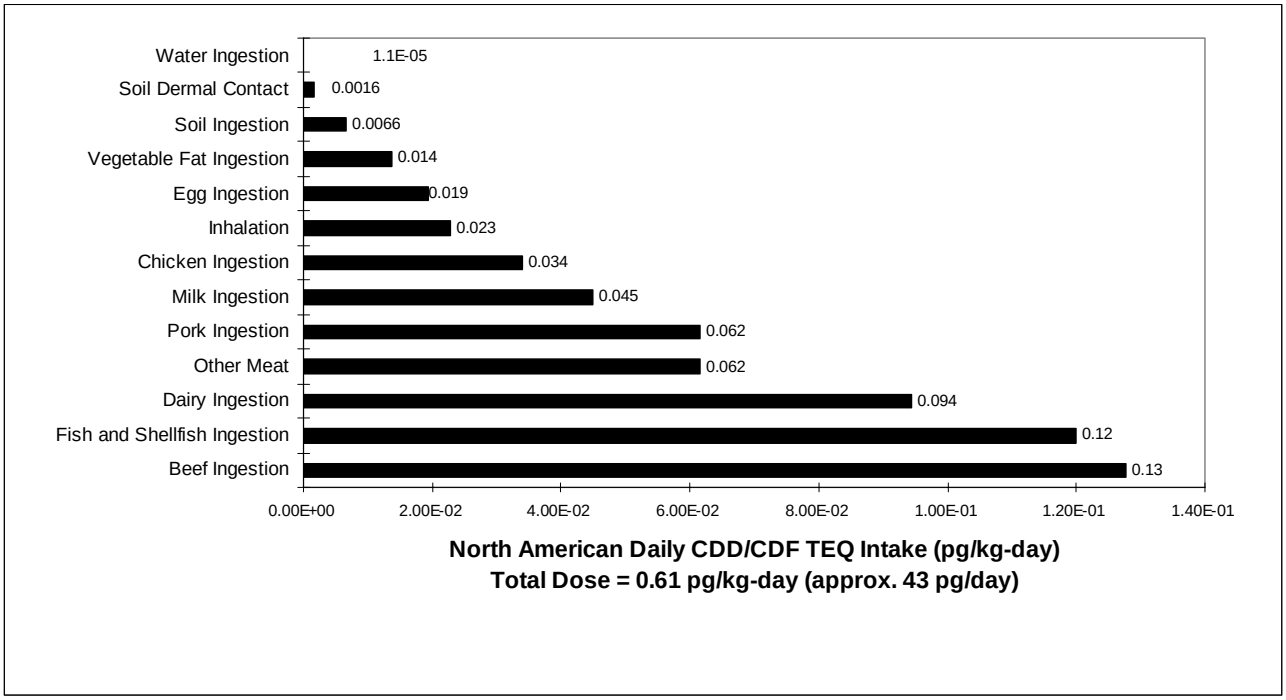
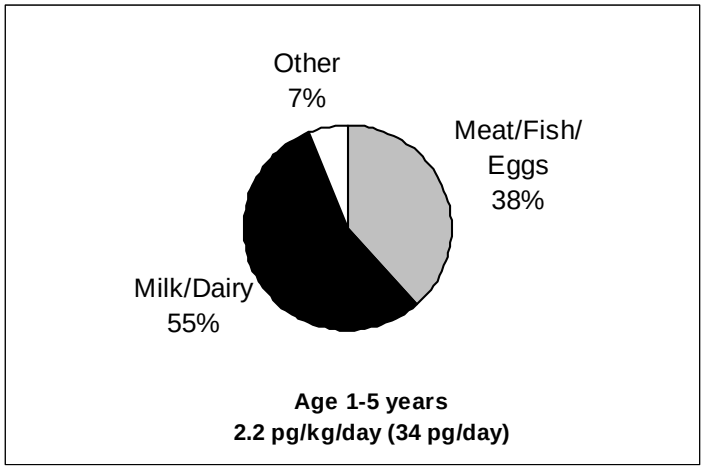
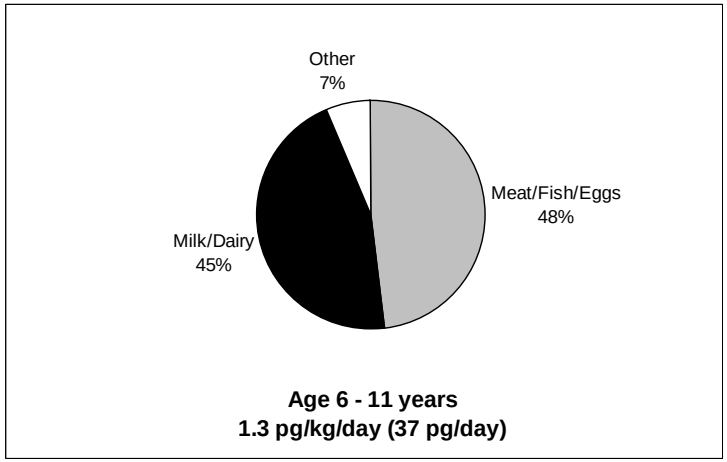
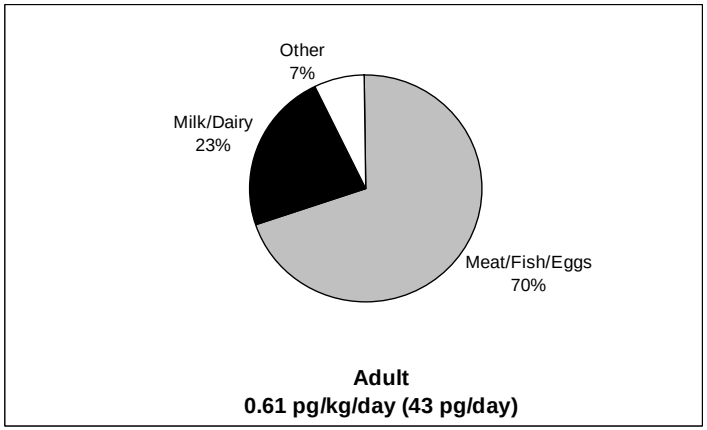
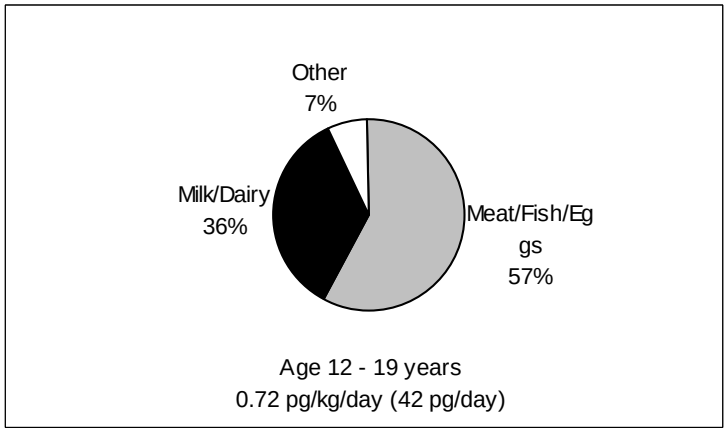


Figure 4-4. Background TEQ_{DF}-WHO_{98} Exposure for North America, by Pathway



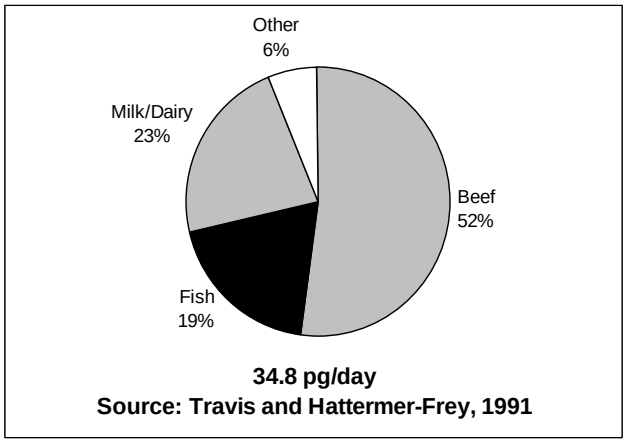


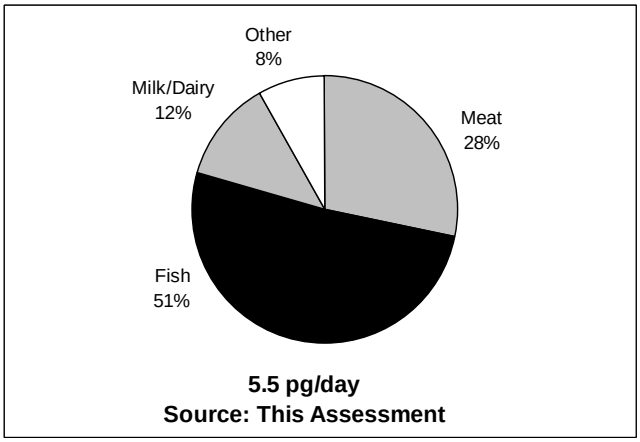


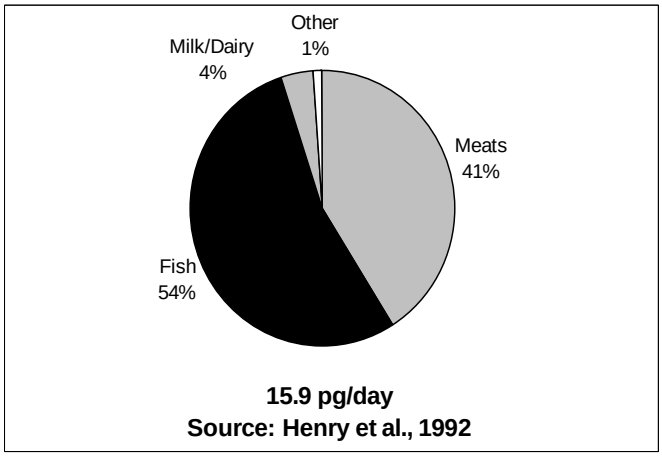


Note: See text for a discussion of the media concentrations and contact rates used to assess dose among these populations.

Figure 4-5. Percent Contribution of Various Media to TEQ_{DF}-WHO_{98} Dose, By Age Group

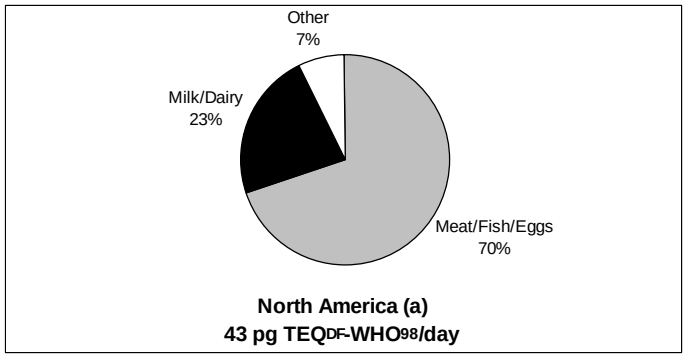


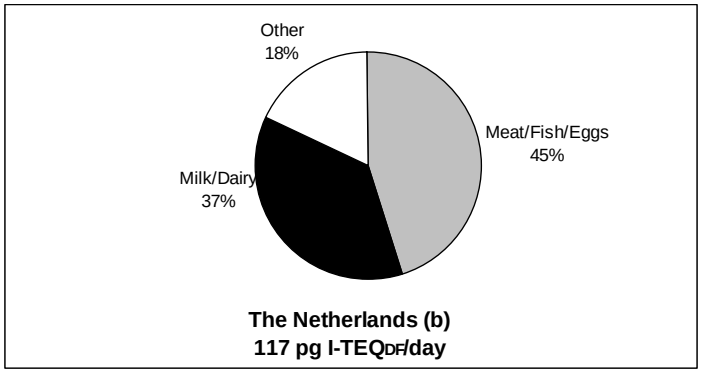


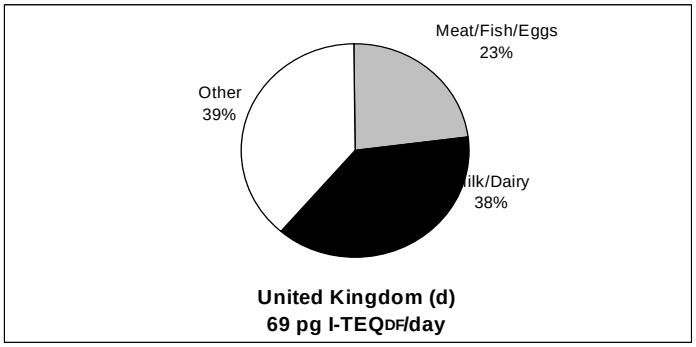


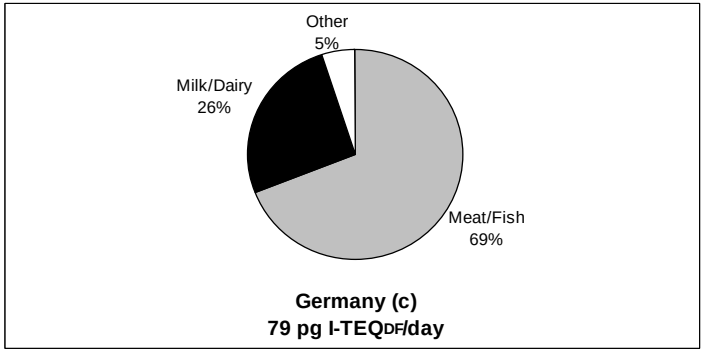
Note: Background exposures are the product of media-specific contact rates and residue concentrations. Reduction in the intake of one food type may not result in CDD/CDF exposure if dietary intake of that food type is replaced by other high CDD/CDF content foods.

Figure 4-6. Contribution of Various Media to 2,3,7,8-TCDD Exposure in North America









Note:
(a) Current assessment. See Table 4-30. Other category includes inhalation (3.7%), soil ingestion (1.1%), soil dermal contact (0.3%), vegetables oils (2.2%), and water (0.002%).
(b) Based on Theelen (1991). See Table 4-27. Other refers to inhalation (2.5%), soil ingestion (0.2%), leafy vegetables (3.4%), and vegetable oil (11.9%).
(c) Based on Furst et al. (1990, 1991). See Table 4-33. Other category includes salad oil (1.3%), and margarine (3.5%).
(d) Based on MAFF (1995). See Table 4-29. Other refers to breads and cereals (30%), and oils and fats (9%).
Percentages rounded to nearest whole number.
Reduction in the intake of one food type may not result in a reduction in CDD/CDF exposure if dietary intake of that food type is replaced by other high CDD/CDF content foods.

Figure 4-7. Comparison of North American and European Background CDD/CDF TEQ Exposures

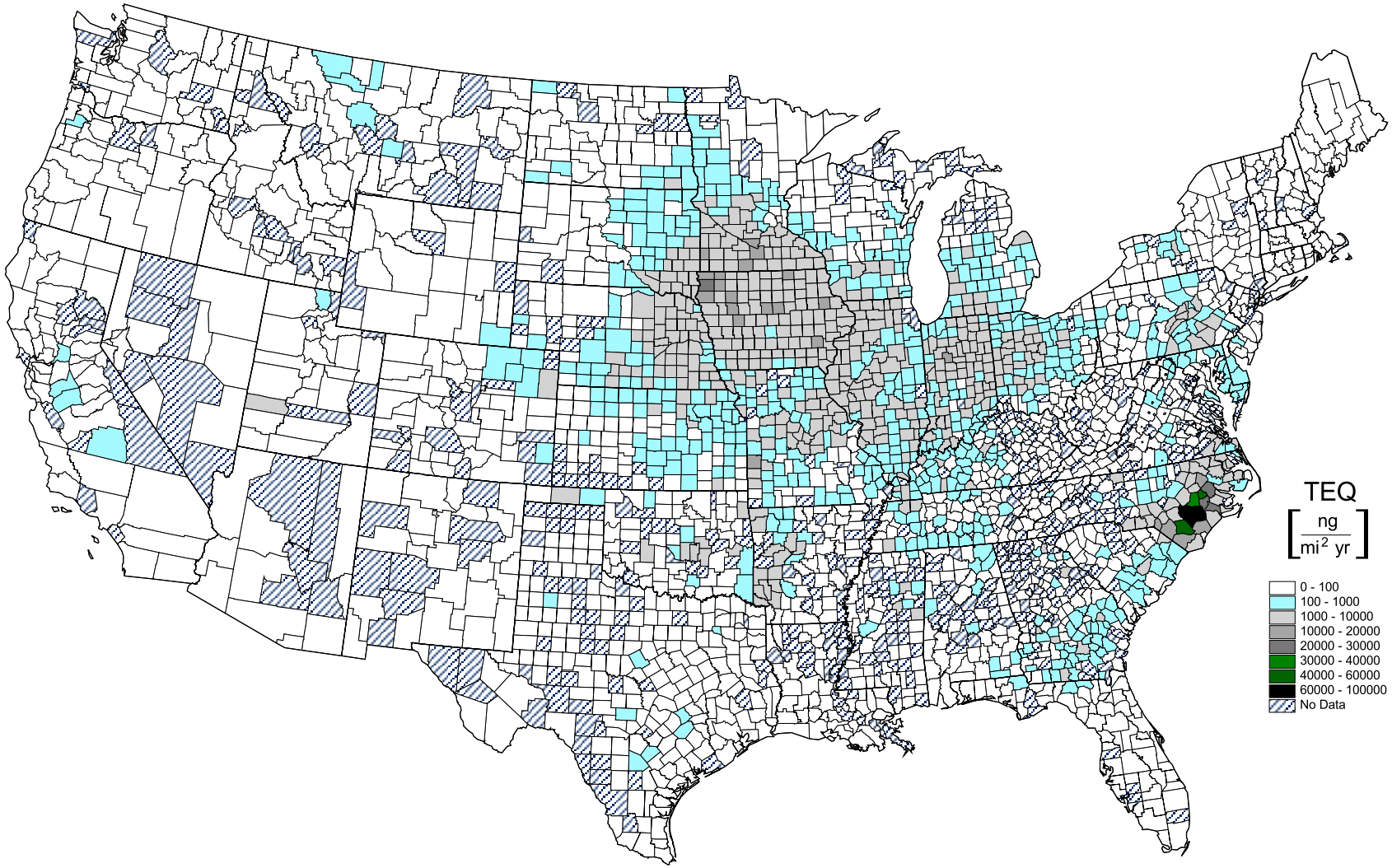


Figure 4-8. TEQ_{DF}-WHO_{98} Derived from Pork Production Data

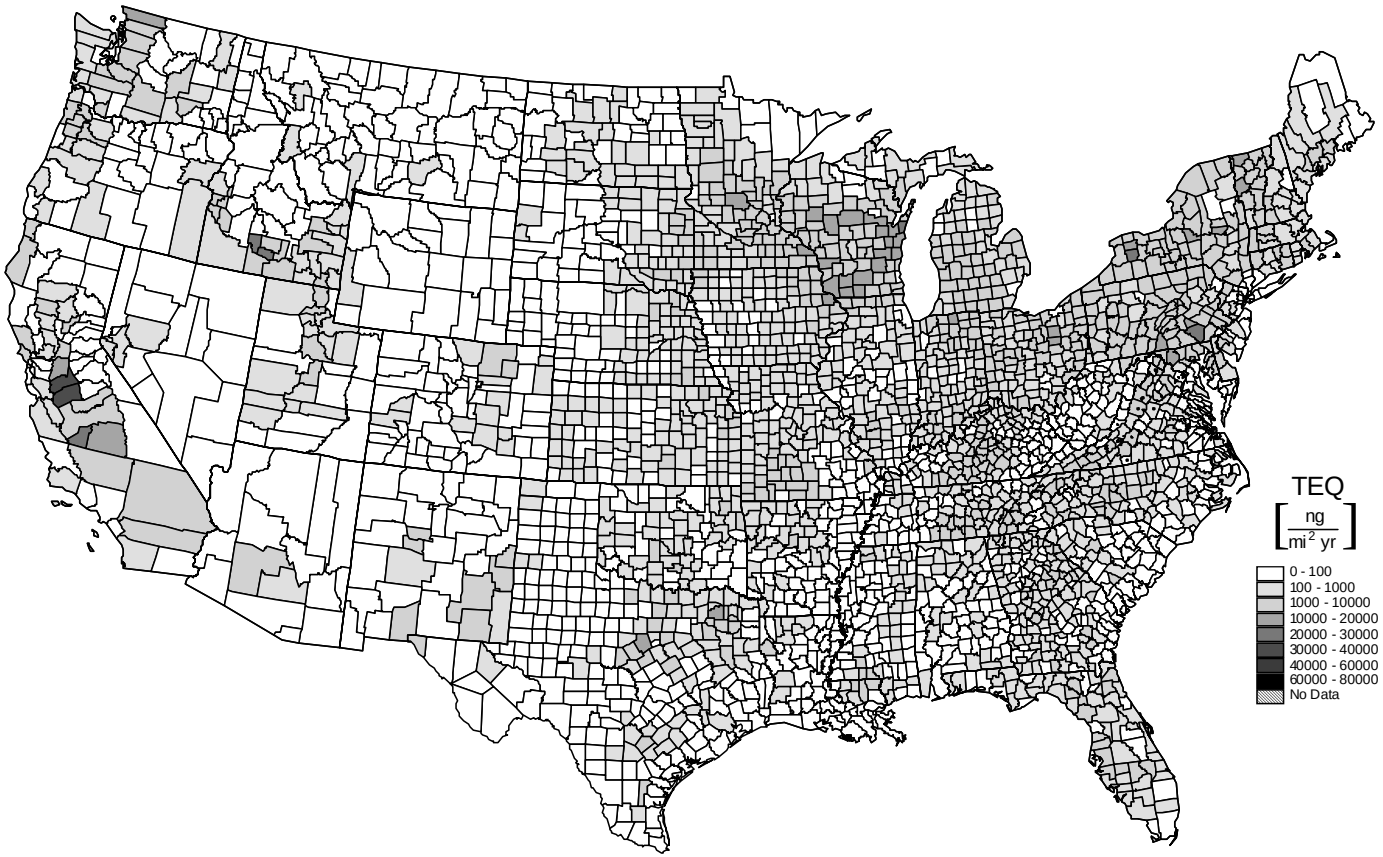


Figure 4-9. TEQ_{DF}-WHO_{98} Derived from Dairy Products Production Data

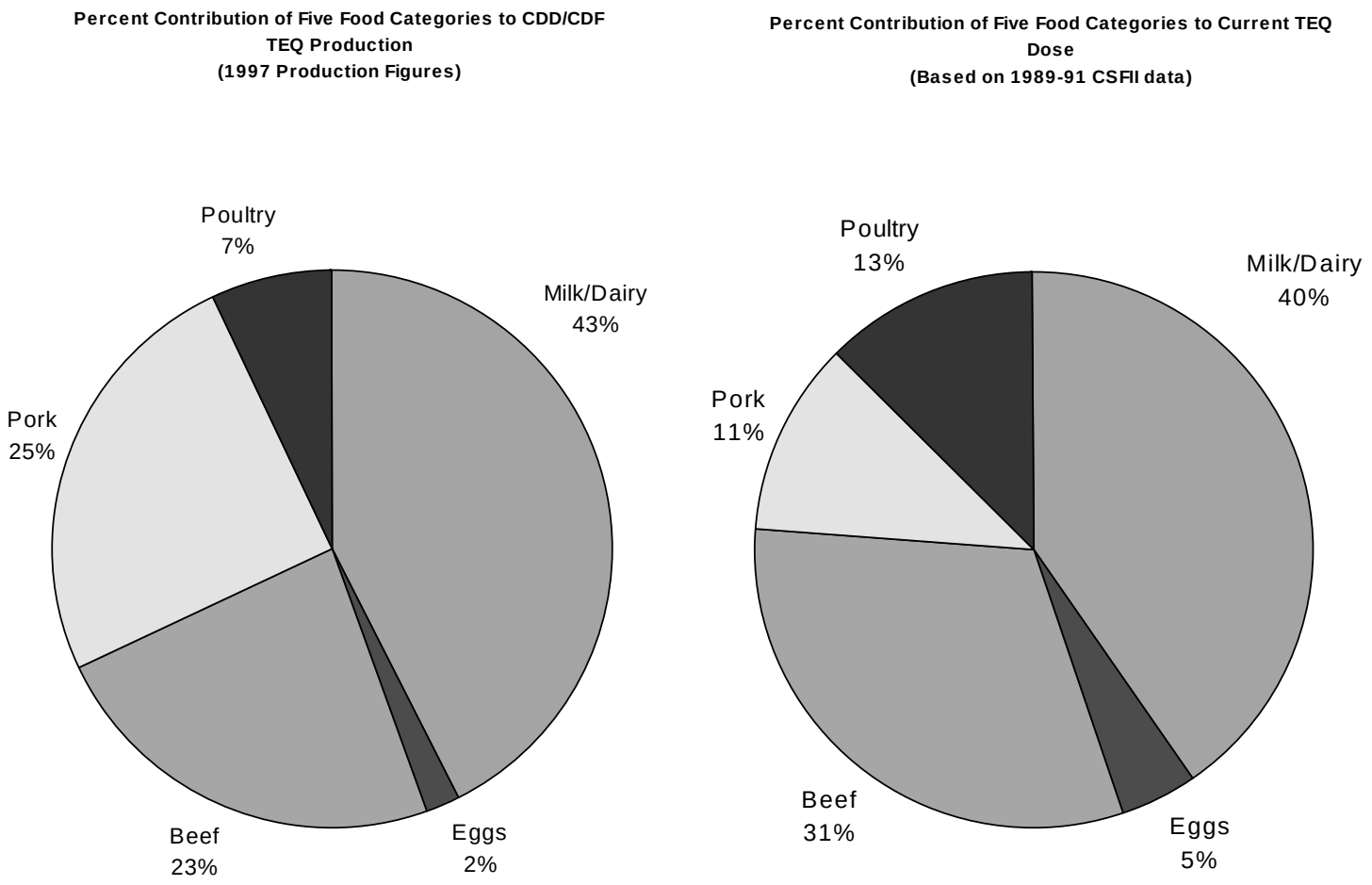


Figure 4-10. Comparison of Food Contributions to TEQ_{DF}-WHO_{98} Production Data and Dose

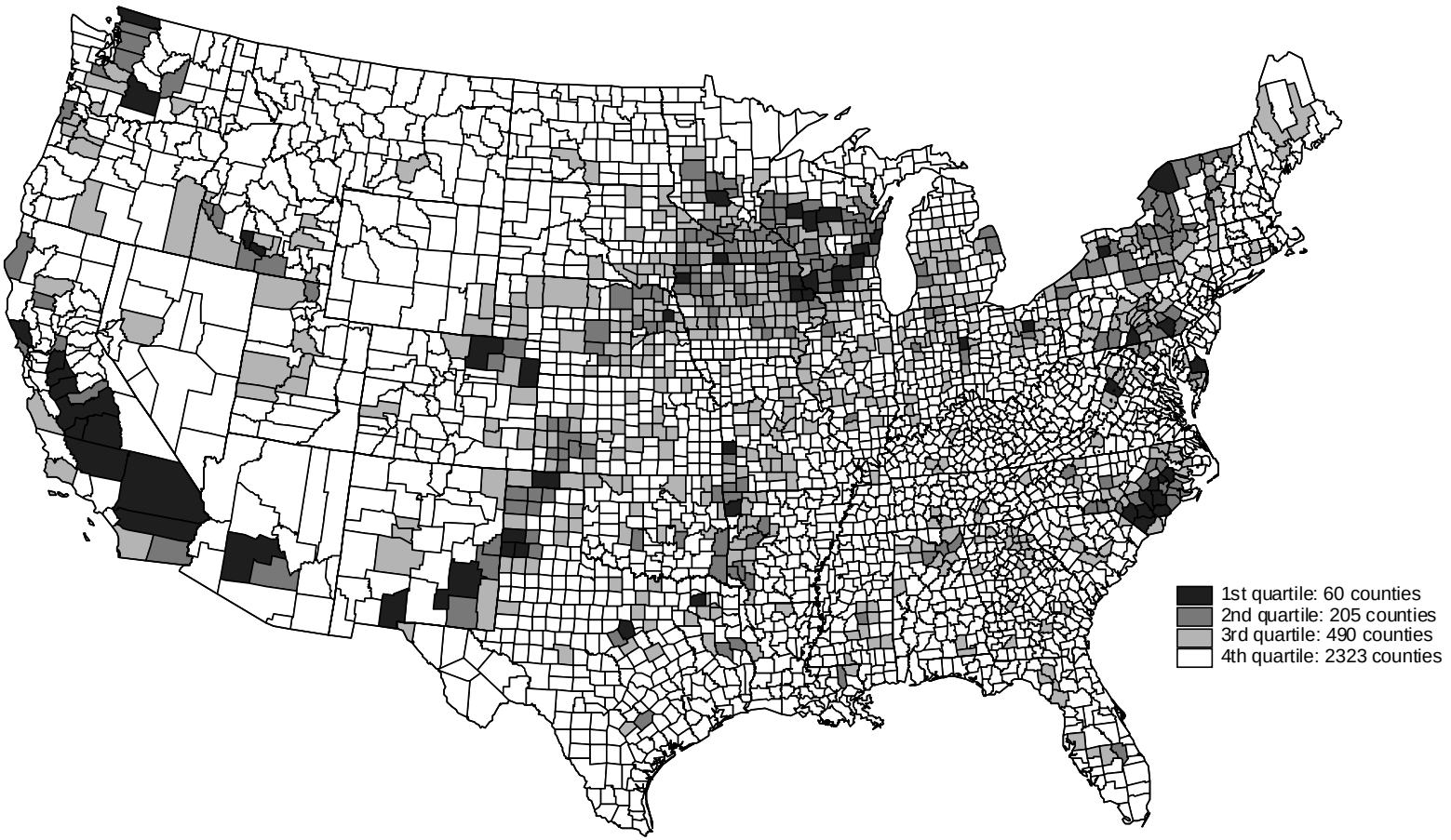


Figure 4-11. Total TEQ Production in Five Food Categories, Categorized by Quartile.

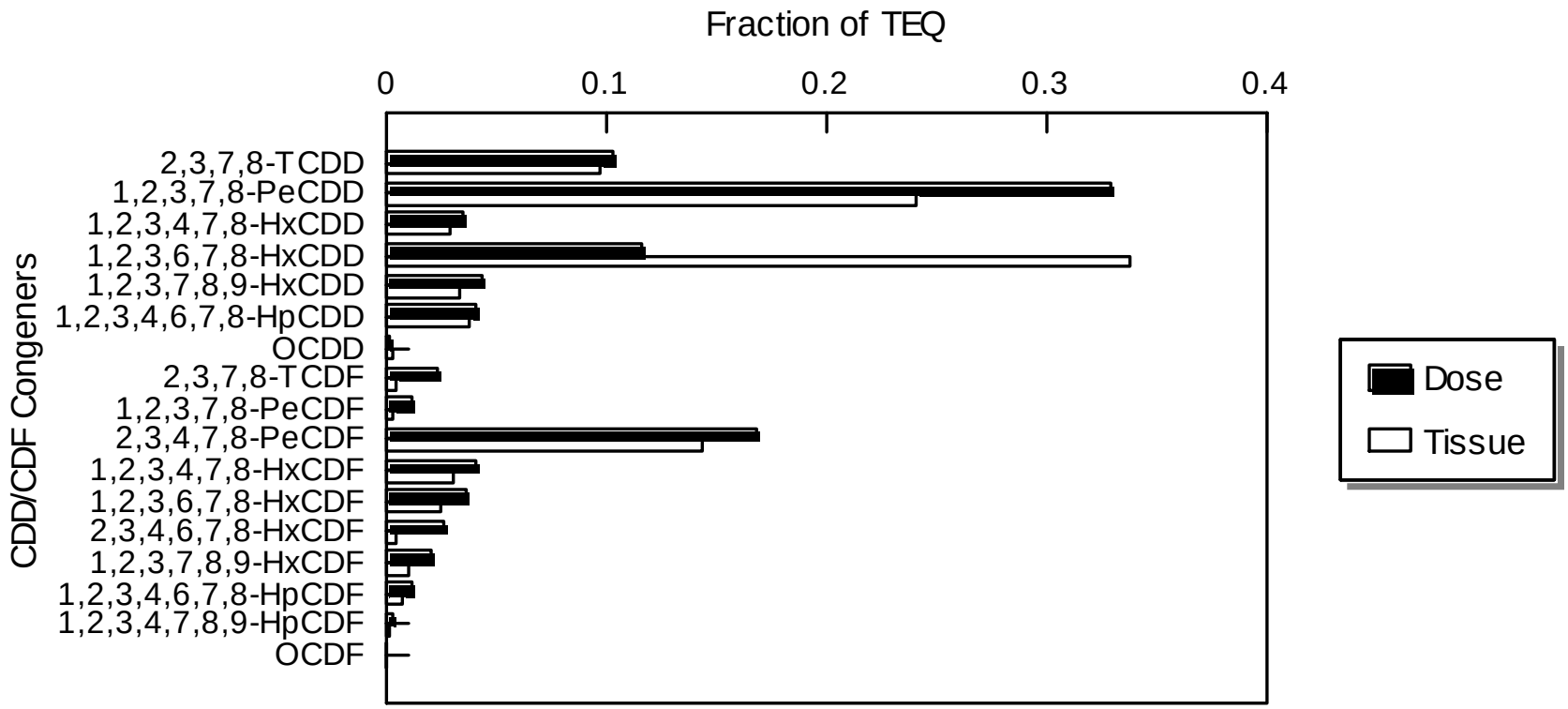


Figure 4-12. Fractions of the Background TEQ Dose and TEQ Tissue Concentration Contributed by Each CDD/CDF Congener

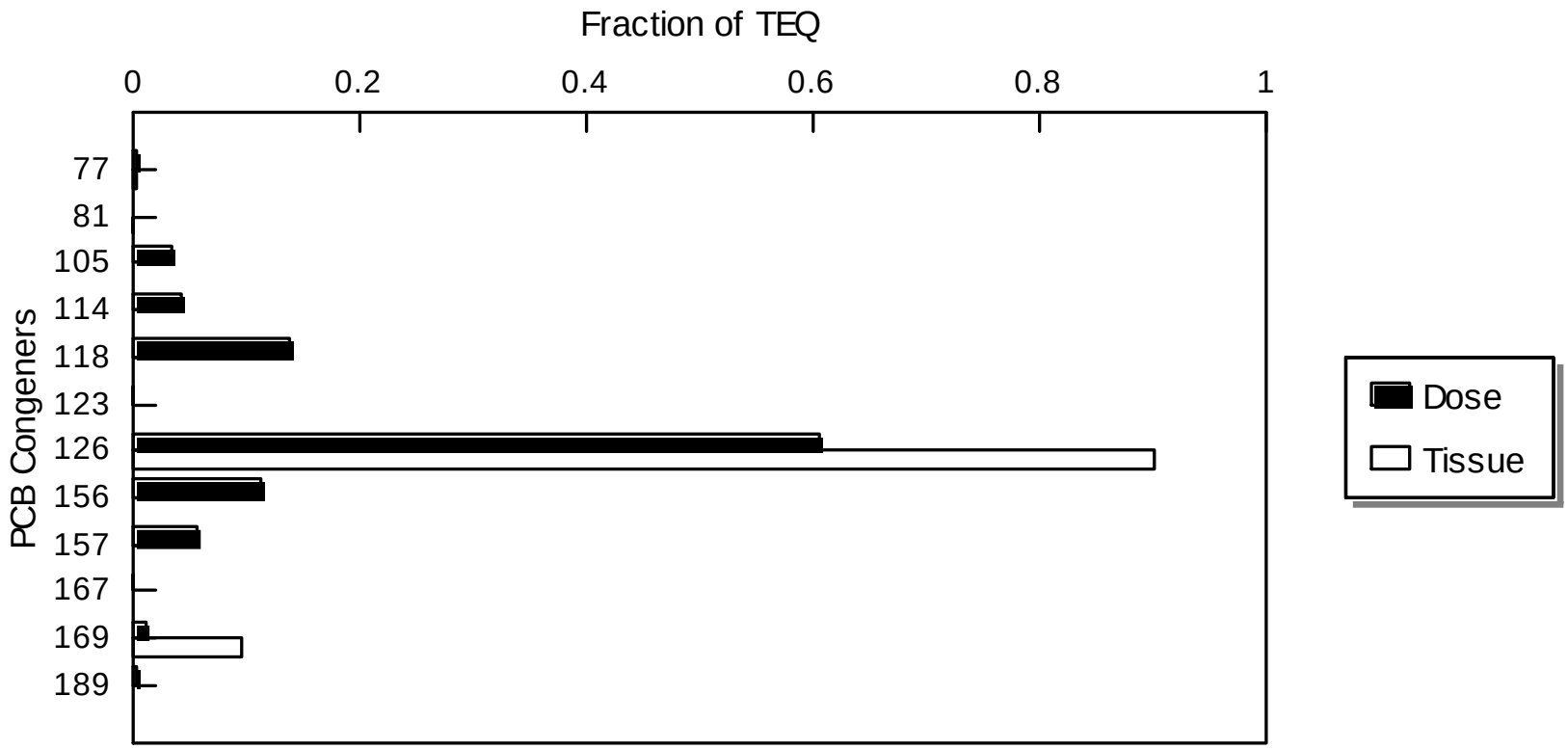


Figure 4-13. Fractions of the Background TEQ Dose and TEQ Tissue Concentration Contributed by Each PCB Congener