PCDD/PCDF: Human Background Data for Germany, a 10-Year Experience

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This paper gives an overview of the development of the environmental or background exposure of humans to polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in Germany. To determine the background exposure, adipose tissue, human milk, or blood can be used. The good comparability of the matrices analyzed is demonstrated. The daily consumption of low-level contaminated food, mainly of animal origin, leads to the accumulation of PCDDs/PCDFs in the human adipose reservoir. The influence of factors such as various eating habits, severe weight loss, age, and nursing (women only) on the human body burden is discussed. Because of decreasing emission of PCDDs/PCDFs into the environment, a decline of these components in humans could be observed over a time span of 10 years. — *Environ Health Perspect* 106(Suppl 2):723–731 (1998). *http://ehpnet1.niehs.nih.gov/docs/1998/Suppl-2/723-731papke/abstract.html*

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Introduction

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are unwanted by-products in a variety of industrial and thermal processes. These two classes of lipophilic and persistent contaminants have been present in the environment for a long time. They were not only identified in historical soil and herb samples collected in England from 1840 until the present (*1*), they were even determined in soil samples from a Roman brickworks along the Lower Rhine near Dormagen (*2*), dating back to about 50 B.C. However, their levels in the environment increased significantly with the beginning of the industrial chlorine industry in this century. Because of their many sources, PCDDs and PCDFs are ubiquitously distributed. The degree of chlorination of the tricyclic components varies between 1 and 8 atoms per molecule. The overall number of dioxins and furans is 75 and 135, respectively.

In humans, only the isomeres with 2,3,7,8-substitution are found, totaling seven dioxins and 10 furans. Humans may become contaminated with PCDD/PCDF through environmental (background), occupational, or accidental exposure. In this overview only environmental exposure will be discussed.

It is generally agreed that for the normal population, food represents the main route of environmental exposure to PCDDs/PCDFs. Usually more than 90% of the total daily intake of these contaminants derives from food.

In contrast, exposure via other routes, such as inhalation and ingestion of particles from air, ingestion of contaminated soil, and dermal absorption, normally contributes less than 10% of daily intake. Because humans are at the high end of the food chain, it becomes obvious that human tissue may contain relatively high amounts of xenobiotics such as PCDDs/PCDFs. Because of the lipophilic nature of these two classes of environmental contaminants, foodstuffs of animal origin are of special importance.

Daily Dietary Intake of PCDDs/PCDFs for Adults

Because food is considered the major source for human exposure to PCDDs/PCDFs, food surveys should give valuable information about the exposure situation. Such surveys performed in Canada, Germany, the United States, the United Kingdom, and the Netherlands show median daily intakes of 1 to 2 pg international toxicity equivalents (NATO–CCMS) (I-TEQ)/kg body weight (bw).

For Germany, three investigations on the daily dietary intake of dioxins have been performed, by Beck et al. (*3*), Fürst et al. (*4*), and the Bund/Länder Working Group on Dioxins (*5*). Because the results of these studies are very similar, only the results of the latest survey are demonstrated here. The mean daily dietary intake of PCDD/PCDF is presented in Figure 1.

At a total intake of approximately 130 pg I-TEQ/day/person, about one-third originates from milk and milk products, one-third from meat, meat products, and eggs, and one-quarter from fish and fish products. The remaining 10% is distributed among bread and cereals, vegetables, and ready-to-serve meals. The total intake/kilogram body weight was calculated at 1.7 pg I-TEQ. These calculations are based on dietary habits of the German population and include analyses of selected food samples. The estimations contain possible uncertainties such as incomplete knowledge of PCDD/PCDF concentrations in special food items and are based in some cases on a small number of analyzed samples.

More reliable data on the actual individual PCDD/PCDF intake via food can be obtained from studies with the so-called duplicate method. Grün et al. (*6*) performed a duplicate study in an area influenced by emissions from a metal reclamation plant that had been closed for 3 years before the study began.

The persons involved in this study—seven women and seven men—had to collect a second portion of the meals they consumed (so-called duplicates) for analytical purposes. To record different diet habits, the samples were taken on 7 consecutive days in spring, summer, fall, and winter of 1994. The contribution of both the self-produced foodstuffs and food originating from the contaminated area could not be exactly estimated but was relatively small compared to food amounts originating from the nationwide supply.

The results of this investigation are shown in Figure 2.

The mean daily PCDD/PCDF intake of the women and men involved for the whole period of the study was calculated at 54 pg I-TEQ/day (range: 10–152) and 69 pg

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Abbreviations used: bw, body weight; HpCDD, heptaCDD; HpCDF, heptaCDF; HxCDD, hexaCDD; HxCDF, hexaCDF; I-TEQ, international toxicity equivalents (NATO–CCMS); OCDD, octaCDD; OCDF, octaCDF; PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; PeCDF, pentaCDF; TCDD, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin.

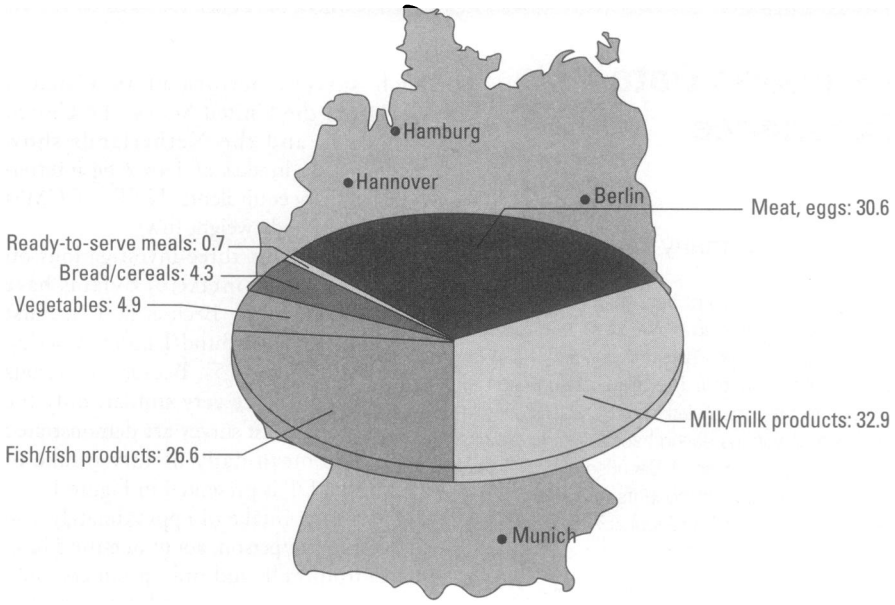

Figure 1. Mean daily intake (127 pg I-TEQ/day; 1.7 pg I-TEQ/day/kg) of PCDDs/PCDFs of humans in Germany via food.

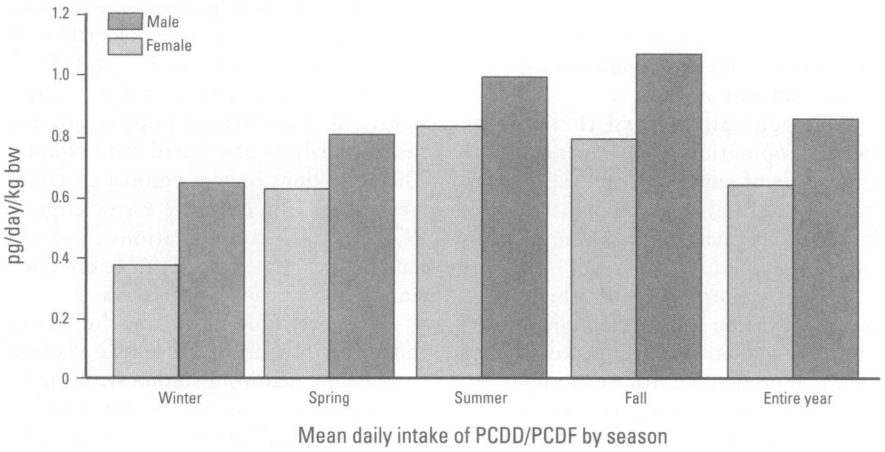

Figure 2. Duplicate study for PCDD/PCDF intake via food in a contaminated area.

I-TEQ/day (range: 19–176), expressed in picograms I-TEQ/kilogram body weight/day at 0.79 and 0.91, respectively. The intake range for women and men was reported at 0.13 to 3.04 and at 0.19 to 2.23 pg/kg/day, respectively. Similar results were found for the same year by Schrey et al. (*7*).

The mean daily dioxin intake in the investigated area amounted to less than 1 pg I-TEQ/kg bw. The intake limit of 1 to 10 pg I-TEQ/kg bw—the aim is 1 pg—set by the German Federal Health and Environmental Offices was not exceeded in this study.

Daily Dietary Intake of PCDD/PCDFs for Infants

The studies mentioned above are relevant only for adults. The situation for nursed infants is quite different. Because of the importance of mothers' milk for infants, the contamination of mother's milk is of public concern. In 1984 the first dioxin measurements were reported for Germany. Until now, more than 2000 samples of mother's milk have been analyzed in Germany by different groups. In Table 1 the intake situation of nursed infants is shown by results from Fürst et al. (*8*).

The basis for this calculation is the daily consumption of 800 ml milk with a lipid content of 3%. This would result in a median daily intake of 77 pg I-TEQ/kg body weight for a 5-kg baby. Consequently the average daily PCDD/PCDF intake for a breast-fed baby is approximately 50 times higher than the average daily PCDD/PCDF intake for an adult.

Table 1. I-TEQ values in mothers' milk and daily intake by infants, North Rhine-Westphalia, 1994.

	Medium	Minimum	Maximum
Mothers' milk,[a] pg/g lipid based	16.1	4.9	30.3
Daily intake			
total pg	386	118	727
pg/kg bw[b]/day	77	24	145

[a] $n = 50$; milk amount, 800 ml; lipid content, 3%. [b] bw, 5 kg.

PCDD/PCDF Determination in Different Human Matrices

As demonstrated, the intake of between less than 0.5 and 3 pg I-TEQ/kg bw via food results in environmental or background contamination of adults. To recognize human background contamination it is possible to analyze adipose tissue, milk, or blood. Blood is easy to obtain from humans and is therefore the preferred tissue to be investigated.

In the first years of dioxin analysis of human tissues, adipose tissue was used almost exclusively for exposure estimation. Because of the low lipid content of blood—about 0.5%—it was not possible to analyze background blood with adequate detection limits. Increasing sensitivity of mass spectrometers opened the possibility of analyzing serum or whole blood.

The correlation between serum and adipose tissue to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) was demonstrated by Patterson et al. (*9*), who analyzed paired serum and adipose tissue samples from 50 persons. The high correlation between adipose tissue and serum TCDD levels found indicates that serum TCDD is a valid measurement of TCDD body burden concentration.

Schecter et al. (*10*) showed a good comparability of further isomeres between whole blood and adipose tissue originating from the same persons. The ratios between both matrices are, for most of the congeners, close to 1.

To demonstrate the correlation of PCDDs/PCDFs between human blood and milk (Figure 3) the 1994 data from Fürst et al. (*11*) for milk are compared with the data of an age-matched German background group from 1994, studied by Päpke et al. (*12*). The I-TEQ value for both materials is similar, whereas the hepta (Hp)CDDs/HpCDFs and the octa(O)CDDs/OCDFs are somewhat higher in the blood.

Before reporting on the development of the background contamination in

Germany, a typical profile of dioxins and furans in human blood should be discussed. In Figure 4A the concentrations are true to scale. The pattern is dominated by OCDD. To give a better impression of the congeners with lower concentration the axis in Figure 4B is adequately expanded. As visible here, pentaCDF (PeCDF) participates because of an international toxicity frequency factor of 0.5, with about 40% on the I-TEQ.

Time Trend of Human PCDD/PCDF Background Data

The first human background data for Germany were published by Fürst et al. (*11*) in human milk and by Beck et al. (*13*) in adipose tissue. The I-TEQ mean for 20 adipose tissue samples from 1986 was 56 pg/g lipids. The first blood data for Germany originated from 10 individuals with no known exposure; the 1988 data were published by Päpke et al. (*14*). The value was found at a mean of 45.8 pg I-TEQ/g blood lipids.

The reference group for Germany was extended with 102 persons for 1989, for whom the mean value was 40.8 pg I-TEQ/g lipids. Between 1991 and 1992 we observed that in an increasing number of cases the levels in individuals without known exposure were lower than the reference data from 1989. As a result further blood data for background contamination with PCDDs/PCDFs have been published (*15–17*).

In Figure 5A all PCDD/PCDF background data—expressed in I-TEQ—are presented for a time span of 10 years. Values have decreased with time. For 1996 we present background data collected in January to September 1996. The mean I-TEQ was calculated at 16.1 pg/g blood lipids originating from 139 individuals.

The time trend for PCDD/PCDF in humans was first observed in German mothers' milk by Fürst et al. (*18*) in 1992. In Figure 5B this trend is demonstrated in both human milk and blood. Between the end of the 1980s and 1996, the reduction ranged between 50 and 70%. Similar effects have been observed in the Netherlands, Denmark, the United States, and the United Kingdom.

These results seem to indicate that efforts to reduce emissions in the industry have notable effects.

The time trend effect was also shown by Hagenmaier and Walczok (*19*) in sediment cores from Lake Constance located in

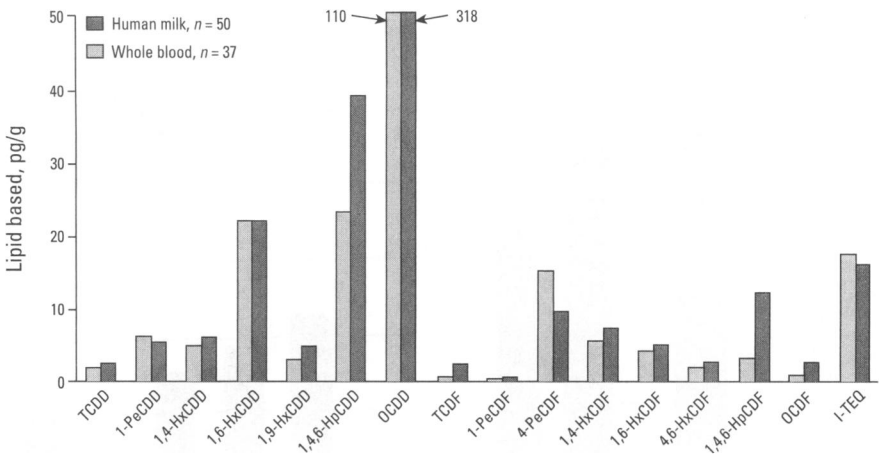

Figure 3. Comparison of PCDDs/PCDFs in human milk (*11*) and whole blood (*12*).

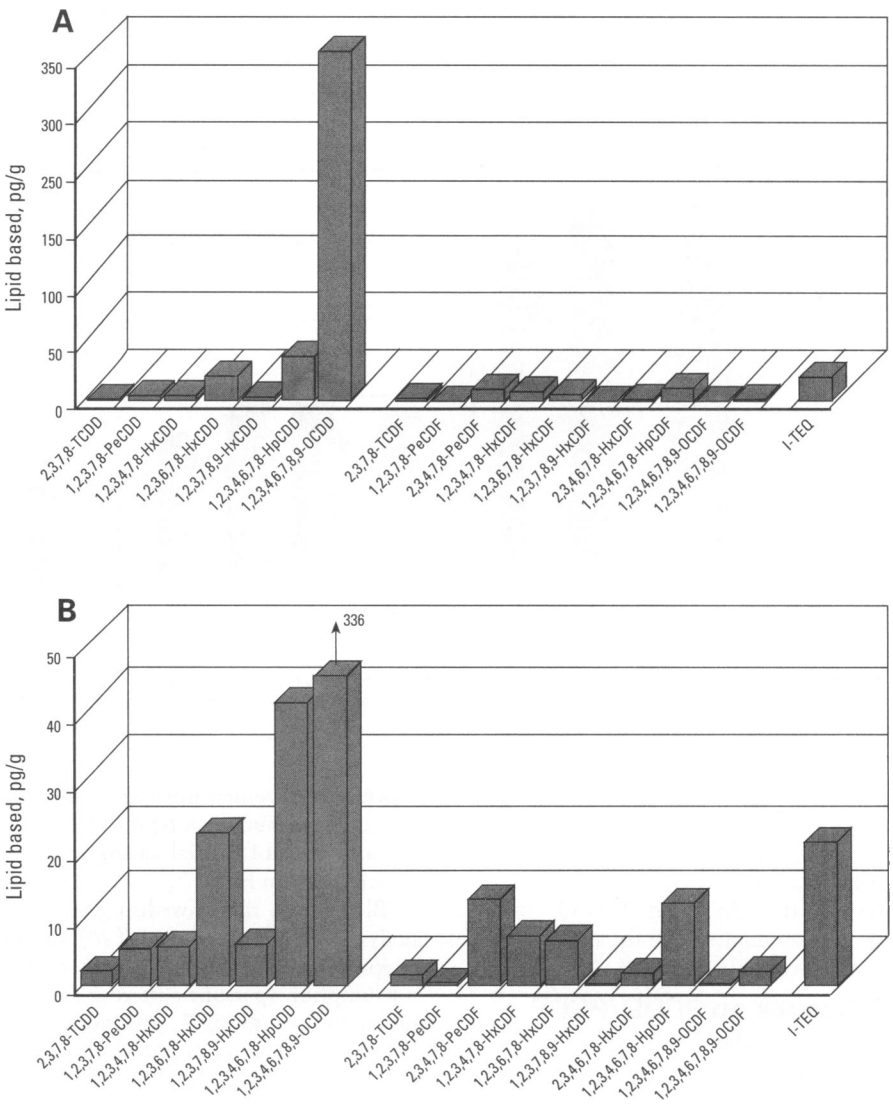

Figure 4. Typical PCDD/PCDF pattern in human blood, background, 1994, median. (*A*) True scale; (*B*) expanded scale; $n = 134$.

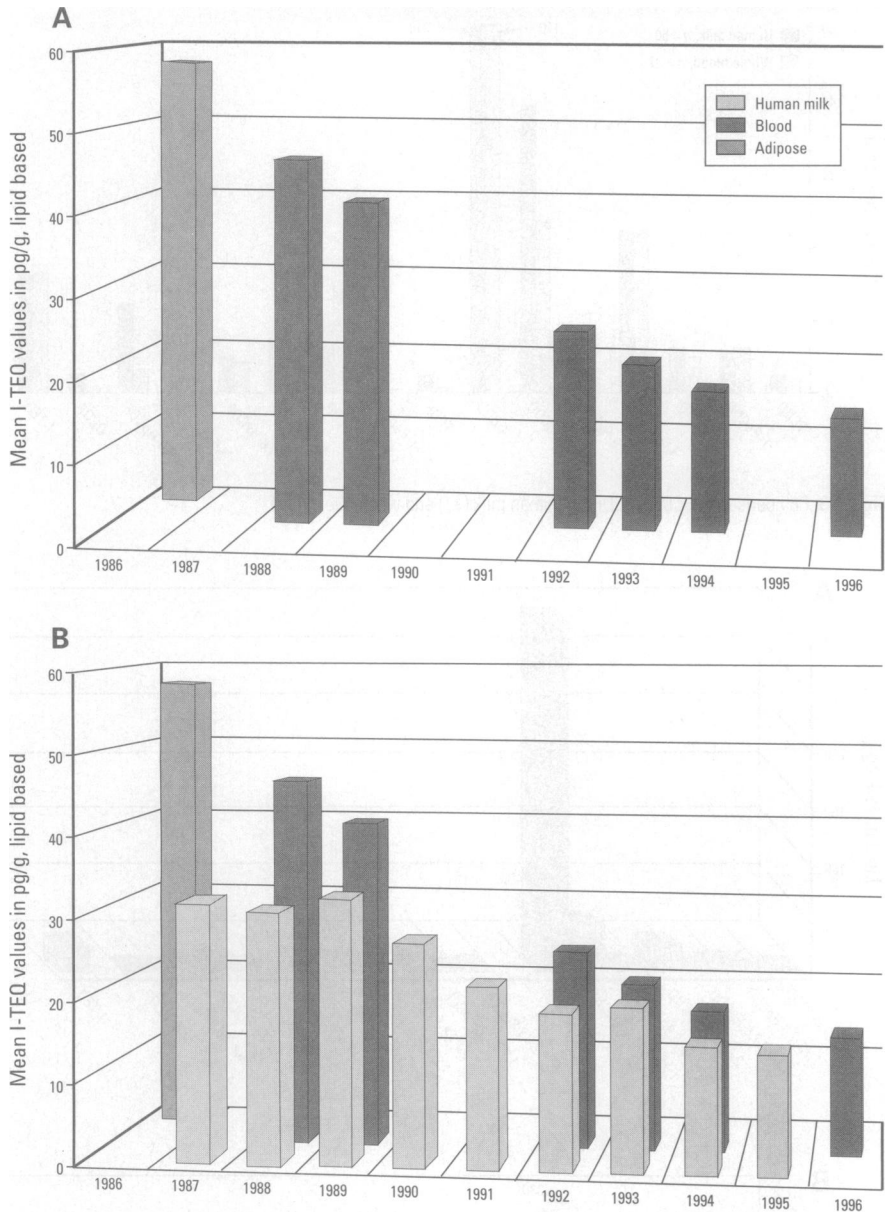

Figure 5. Time trend of PCDD/PCDF in humans in adipose tissue (*5*), blood (*12,14,17*), and mothers' milk (*8,11,18*). 1986, $n=20$; 1988, $n=10$; 1989, n = 102; 1992, $n=44$; 1993, $n=70$; 1994, $n=134$; 1996, $n=139$. (*A*) Blood; (*B*) blood and mothers' milk.

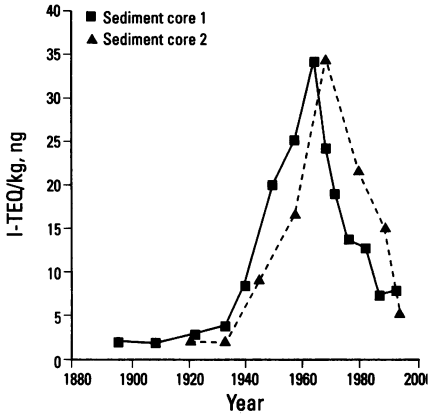

Figure 6. Time trend for PCDDs/PCDFs in sediment cores of Lake Constance, 1880 to present.

Southern Germany as well (Figure 6). With the beginning of the chlorine industry in the 1930s TEQ levels increased significantly, culminating in a maximum level in the 1960s. The I-TEQ values in the latest samples from the mid-1990s are reduced by more than 80%.

Influence on PCDD/PCDF Body Burden

The human body burden can be influenced by a number of factors, including eating habits; severe weight loss (e.g., in cancer patients), which leads to higher values; age dependency; women nursing; and babies nursed or not nursed. A typical example of the influence of special eating habits is demonstrated in Figure 7.

Blood from three Swedish groups was analyzed by Svensson et al. (*20*) and by Rappe et al. (*21*): Group 1, with no fish consumption (persons suffering from an allergy to fish); group 2, with normal fish consumption (about 50 g/day); and group 3, with high fish consumption (> 100 g/day). Group 3 had a body burden, calculated as I-TEQ, approximately three times higher than group 2. Group 1, however, had only slightly lower blood levels of PCDDs/PCDFs than group 2. The dominant congener among the tetra- and pentachlorinated congeners was 2,3,4,7,8-PeCDF. The difference among the three groups was also highest for this particular congener, which is the major congener in fish from the Baltic Sea. The mean PCDD/PCDF values for groups 1, 2, and 3 were 17.5, 25.8, and 63.5 pg I-TEQ/g lipids, respectively.

Another example of elevated human dietary levels was demonstrated by Wuthe et al. (*22*) in so-called egg/chicken consumers. We analyzed blood samples originating from persons who had a high consumption of highly polluted eggs and poultry and/or vegetables originating from contaminated kitchen gardens. In Figure 8A the levels of one of the egg consumers—resident 56—are compared with those of an adequate background group ($n=102$, sampled mainly in 1989). There are pronounced differences for HpCDD and OCDD as well as PeCDF and hexaCDF (HxCDF) in the blood of a resident of the contaminated area. The relationship between the exposure material and the human data is shown in Figure 8B. The diagram presents concentration profiles in soil, eggs, and chickens. The dioxin pathway can be followed in Figure 8B, leading from the polluted soil via eggs and chickens to the consumer.

Different PCDD/PCDF values resulting from varying dietary habits may be expected for vegetarians and nonvegetarians. Figure 9 presents the results of a study by Welge et al. (*23*) concerning these kinds of eating habits. Blood of 24 vegetarians and 24 nonvegetarians was analyzed. It was expected that the mean

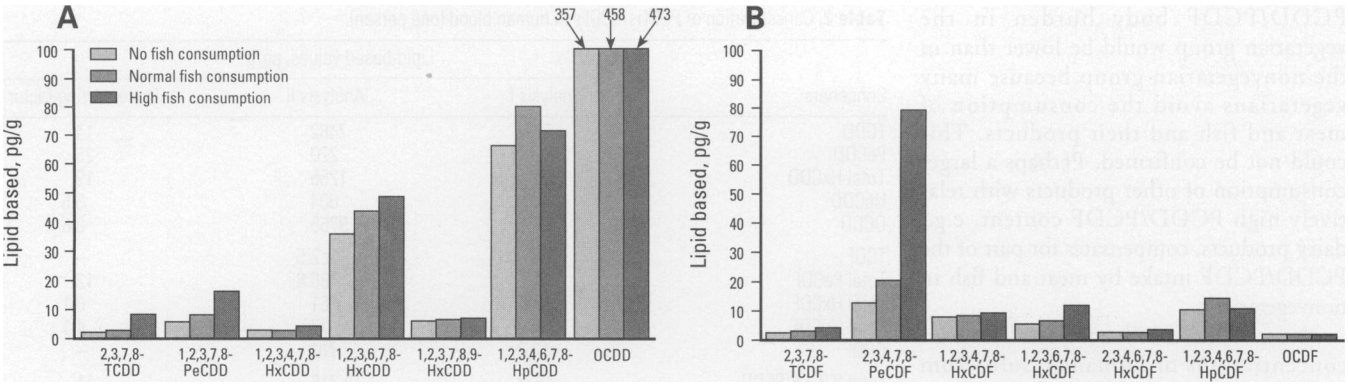

Figure 7. PCDDs/PCDFs in humans, dependency on fish consumption.

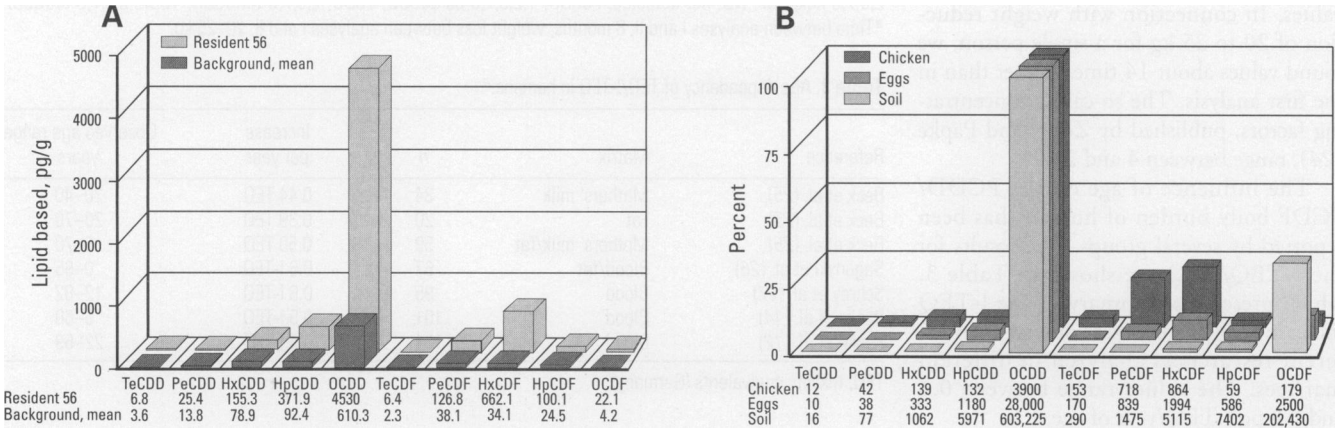

Figure 8. Influence of PCDD/PCDF-contaminated eggs and poultry on human blood levels. (*A*) Comparison between egg consumption and background levels, resident 56 (city in South Germany); (*B*) exposure material.

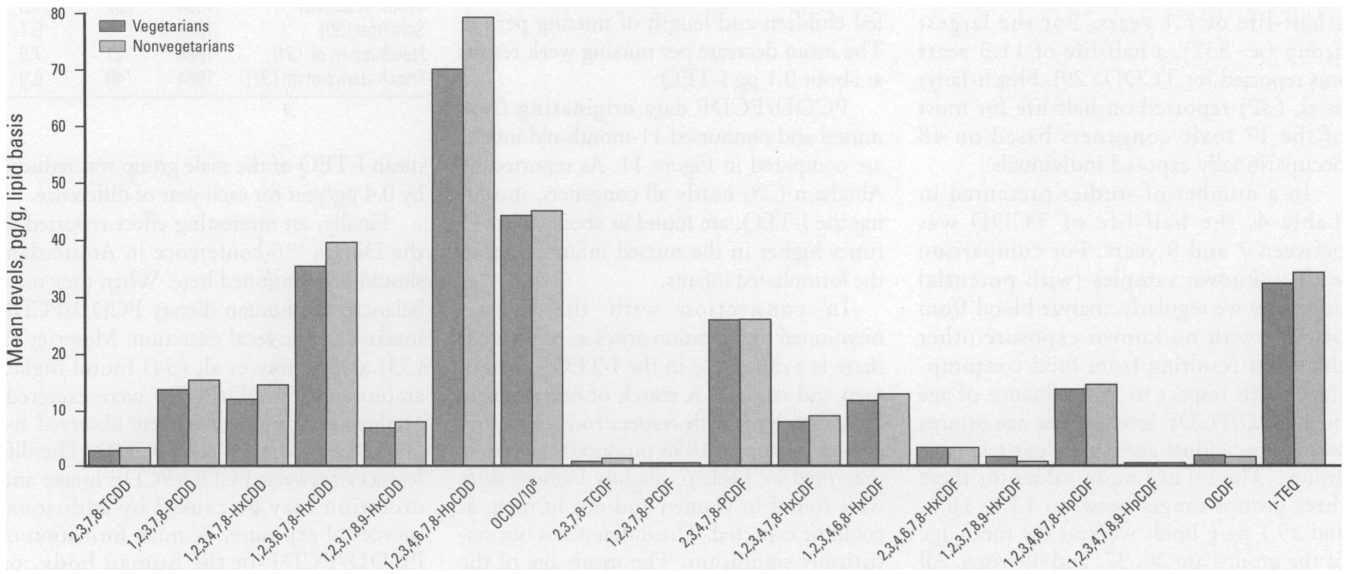

Figure 9. Comparison of PCDD/PCDF values in vegetarians and nonvegetarians. [a]OCDD value divided by 10.

PCDD/PCDF body burden in the vegetarian group would be lower than in the nonvegetarian group because many vegetarians avoid the consumption of meat and fish and their products. This could not be confirmed. Perhaps a larger consumption of other products with relatively high PCDD/PCDF content, e.g., dairy products, compensates for part of the PCDD/PCDF intake by meat and fish in nonvegetarians.

A pronounced influence on the dioxin concentrations of humans results from weight reduction, as demonstrated in Table 2. We analyzed blood samples from some cancer patients a few months before they died. A second analysis, post mortem, resulted in much higher PCDD/PCDF values. In connection with weight reduction of 20 to 25 kg for a single person, we found values about 14 times higher than in the first analysis. The so-called concentrating factors, published by Zober and Päpke (*24*), range between 4 and 25.

The influence of age on the PCDD/PCDF body burden of humans has been reported by several groups. The results for the I-TEQ/TEQ are shown in Table 3, which presents a summary of the I-TEQ increase in picograms/year observed by different researchers analyzed in different matrices. The values range between 0.4 and 0.8 pg I-TEQ/year of age.

For most of the PCDDs/PCDFs, long half-lives have been observed in humans. The half-life of TCDD has been studied most comprehensively. The first results for this compound, in 36 individuals, were reported by Pirkle et al. (*28*). He observed a half-life of 7.1 years. For the largest group ($n = 337$), a half-life of 11.3 years was reported for TCDD (*29*). Flesch-Janys et al. (*32*) reported on half-life for most of the 17 toxic congeners based on 48 occupationally exposed individuals.

In a number of studies presented in Table 4, the half-life of TCDD was between 7 and 8 years. For comparison with unknown samples (with potential exposure) we regularly analyze blood from persons with no known exposure other than that resulting from food consumption. With respect to the influence of age on PCDD/PCDF levels, three age groups serve as individual age-dependent reference groups. The I-TEQ mean values for these three groups ranges between 13.1, 16.3, and 19.1 pg/g lipids whereas the mean age of the groups are 26, 37, and 49 years. All PCDD/PCDFs including I-TEQ are represented in Table 5.

Table 2. Concentration of PCDDs/PCDFs in human blood (one person).

Congeners	Lipid-based values, pg/g[a]		
	Analysis I	Analysis II	Concentrating factor
TCDD	518	7482	14
PeCDD	8.7	220	25
Total HxCDD	66	1255	19
HpCDD	110	604	5.5
OCDD	1135	9755	8.6
TCDF	2.6	<2.5	–
Total PeCDF	39.3	486.8	12
Total HxCDF	42.7	261	6.1
Total HpCDF	19	77	4.1
OCDF	<2.3	ND	–
Total 2,3,7,8-PCDD	1838	19,316	11
Total 2,3,7,8-PCDF	105	826	8
Total 2,3,7,8-PCDD/PCDF	1943	20,142	10
I-TEQ	555	8002	14

Abbreviations: HpCDD, 1,2,3,4,6,7,8-heptaCDD; HpCDF, 2,3,7,8-hepta CDF; HxCDD, 2,3,7,8-hexaCDD; HxCDF, 2,3,7,8-hexaCDF; ND, not detected; PeCDD, 1,2,3,7,8-pentaCDD; TCDD, 2,3,7,8-tetraCDD; TCDF, 2,3,7,8-tetraCDF.
[a]Time between analyses I and II, 8 months; weight loss between analyses I and II, 20–25 kg.

Table 3. Age dependency of TEQ/I-TEQ in humans.[a]

Reference	Matrix	*n*	Increase per year	Observed age range, years
Beck et al. (*25*)	Mothers' milk	34	0.44 TEQ	20–40
Beck et al. (*25*)	Fat	20	0.39 TEQ	20–70
Beck et al. (*25*)	Mothers' milk/fat	59	0.50 TEQ	0–70
Sagunski et al. (*26*)	Blood/fat	67	0.6 I-TEQ	0–65
Schrey et al. (*27*)	Blood	95	0.8 I-TEQ	12–82
Päpke et al. (*14*)	Blood	101	0.5 I-TEQ	6–60
Päpke et al. (*12*)	Blood	134	0.4 I-TEQ	22–69

TEQ, toxicity equivalents (Germany).

Important factors that influence the dioxin levels in mothers' milk are the total length of the nursing period and the number of breast-fed children (Figure 10). As reported by Fürst et al. (*18*), the levels decrease with increasing number of breast-fed children and length of nursing period. The mean decrease per nursing week results at about 0.1 pg I-TEQ.

PCDD/PCDF data originating from nursed and nonnursed 11-month-old infants are compared in Figure 11. As reported by Abraham (*28*) nearly all congeners, including the I-TEQ, are found at about 10 to 15 times higher in the nursed infants than in the formula-fed infants.

In connection with the factors mentioned the question arises as to whether there is a difference in the I-TEQ values of men and women. A search of our database for all analyses with respect to background contamination in 1996 produced the results presented in Table 6. Slightly higher values were found in women and not in men, as could be expected. The difference is not statistically significant. The mean age of the male group is 7 years older than that of the female group. Taking this into account, the mean I-TEQ of the male group was reduced by 0.4 pg/year for each year of difference.

Table 4. Comparison of estimated half-lives for TCDD in different studies

Reference	Year	*n*	Half-life, years
Pirkle et al. (*28*)	1989	36	7.1
Wolfe et al. (*29*)	1994	337	11.3
Schlatter (*30*)	1991	1	9.1
Needham et al. (*31*)	1994	27	7.8
Flesch-Janys et al. (*32*)	1994	48	6.9

Finally, an interesting effect reported at the Dioxin '96 conference in Amsterdam should be mentioned here. When they mass balanced the human dietary PCDD/PCDF intake and the fecal excretion, Moser et al. (*33*) and Schrey et al. (*34*) found higher amounts of PCDD/PCDF were excreted. Highest excretion levels were observed for HxCDD, HpCDD, and OCDD. The differences between PCDD/PCDF intake and excretion may be caused by additional sources of exposure, de novo formation of PCDD/PCDF in the human body, or reduction of the body burden as a consequence of decreasing PCDD/PCDF intake.

Table 5. PCDD/PCDF concentrations in human blood, background, January to September 1996, Germany.

	All age groups, 18–71 years, $n=139$				Age group 1, 18–30 years, $n=47$				Age group 2, 31–42 years, $n=48$				Age group 3, 43–71 years, $n=44$			
Age group, samples, no.	Mean	Med	Min	95th %	Mean	Med	Min	95th %	Mean	Med	Min	95th %	Mean	Med	Min	95th %
Age	37.0	37.0	18.0	55.0	26.1	27.0	18.0	30.0	36.6	37.0	31.0	42.0	49.1	47.5	43.0	57.0
Chemical																
2,3,7,8-TCDD	2.3	2.2	ND	4.3	2.1	1.9	1.0	3.7	2.2	2.2	ND	3.6	2.8	2.6	ND	4.8
1,2,3,7,8-PeCDD	5.9	5.6	1.7	9.9	4.8	4.1	1.7	8.0	5.9	5.6	2.5	9.3	7.0	7.0	3.6	10.3
1,2,3,4,7,8-HxCDD	5.7	5.2	2.0	9.9	5.0	4.6	2.0	9.1	5.7	5.5	2.3	9.2	6.5	6.6	2.8	10.0
1,2,3,6,7,8-HxCDD	22.6	20.1	3.7	40.4	16.8	15.9	3.7	30.1	24.4	23.5	8.6	40.0	26.7	25.7	6.8	44.4
1,2,3,7,8,9-HxCDD	3.8	3.6	1.5	6.2	3.6	3.4	1.8	6.0	3.9	3.6	1.5	6.4	3.9	3.7	1.9	6.1
Total HxCDD	32.1	29.3	8.4	55.0	25.4	24.3	8.4	44.7	34.0	33.1	16.7	54.1	37.1	35.8	14.9	56.1
1,2,3,4,6,7,8-HpCDD	33.0	29.9	9.5	60.2	34.0	35.1	9.8	54.2	33.2	29.3	12.4	63.0	31.5	28.6	9.5	60.2
1,2,3,4,6,7,8,9-OCDD	293	271	106	525	287	272	108	506	310	296	114	549.3	279.6	240	106	500
2,3,7,8-TCDF	1.2	1.2	0.5	1.8	1.2	1.2	0.5	1.7	1.3	1.2	0.5	1.8	1.2	1.1	0.5	1.9
1,2,3,7,8-PeCDF	0.6	0.5	ND	1.0	0.6	0.5	0.5	0.7	0.6	0.5	0.5	1.0	0.5	0.5	ND	1.0
2,3,4,7,8-PeCDF	10.9	9.8	3.2	20.0	8.2	7.8	3.2	13.5	10.9	10.0	4.8	18.8	13.8	13.9	5.2	20.3
Total PeCDF	11.1	10.2	3.8	20.0	8.4	7.9	3.8	14.0	11.2	10.2	4.8	19.8	14.0	14.4	5.2	20.8
1,2,3,4,7,8-HxCDF	6.3	6.0	2.6	10.3	5.8	5.4	2.6	8.9	6.4	6.1	2.8	9.5	6.9	6.8	3.0	10.3
1,2,3,6,7,8-HxCDF	4.7	4.5	2.0	8.3	4.1	4.0	2.2	6.2	4.8	4.6	2.0	8.3	5.3	5.2	2.2	8.7
1,2,3,7,8,9-HxCDF	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
2,3,4,6,7,8-HxCDF	2.4	2.4	0.5	3.8	2.4	2.4	1.4	3.7	2.4	2.4	0.5	4.0	2.5	2.5	1.0	3.7
Total HxCDF	13.5	13.0	6.1	20.9	12.3	11.9	6.5	16.5	13.5	13.0	6.1	20.0	14.7	14.2	6.3	22.1
1,2,3,4,6,7,8-HpCDF	8.1	7.2	4.1	13.4	9.2	9.6	4.2	13.8	7.8	7.1	4.7	12.5	7.1	6.8	4.1	10.9
1,2,3,4,7,8,9-HpCDF	0.9	0.8	0.5	1.6	1.0	1.0	0.7	1.4	1.0	0.8	0.8	1.8	0.8	0.8	0.5	1.0
Total HpCDF	8.4	7.6	4.1	13.8	9.7	10.2	4.2	13.9	8.0	7.6	4.7	12.5	7.3	7.0	4.1	10.9
1,2,3,4,6,7,8,9-OCDF	2.4	2.5	1.5	2.5	2.5	2.5	1.8	2.5	2.4	2.5	1.6	2.5	2.4	2.5	1.5	2.5
Total PCDD	366.1	337.2	152.4	618.9	353.7	330.9	152.8	591.7	385.8	351.2	163.6	686.3	357.9	330.6	152.4	606.8
Total PCDF	36.5	35.0	19.7	55.8	34.0	34.4	19.9	48.1	36.4	33.7	19.7	56.8	39.4	38.6	19.7	55.9
Total PCDD/PCDF	402.6	374.0	177.9	653.8	387.7	367.5	177.9	628.8	422.2	385.1	188.2	733.7	397.3	363.7	185.3	653.7
I-TEQ (NATO–CCMS)	16.1	15.2	7.3	26.7	13.1	11.9	7.32	20.4	16.3	15.3	7.9	25.9	19.1	18.4	10.1	29.6
TEQ (BGA/UBA)	9.4	8.8	4.7	14.7	8.0	7.7	4.73	12.0	9.5	9.1	5.0	14.4	10.8	10.6	6.2	15.7

Abbreviations: BGA/UBA, German; Med, median; Min, minimal value; NATO–CCMS, International; ND, not detected; 95th %, 95th percentile. Values in pg/g (ppt), lipid based.

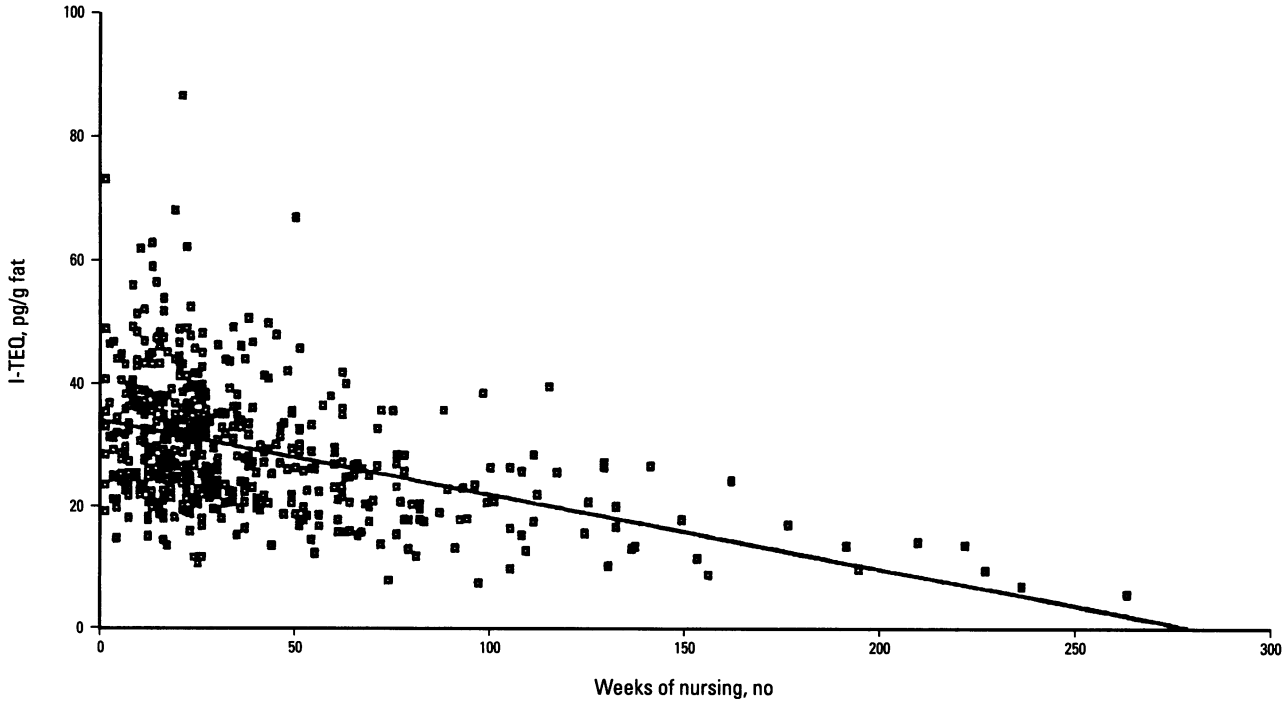

Figure 10. Influence of nursing time, weeks, on picogram/gram adipose tissue, I-TEQ; $n=524$. Reproduced from Fürst et al. (*18*), with permission of Elsevier Science.

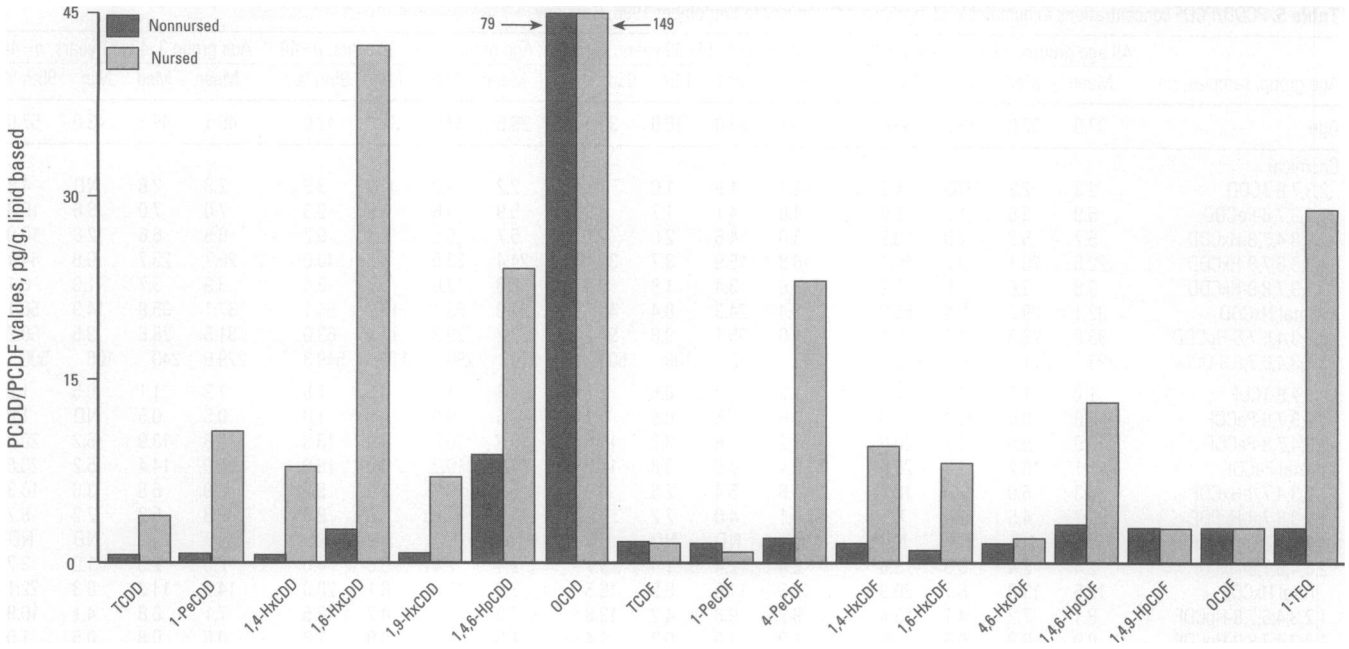

Figure 11. Comparison of PCDD/PCDF values in a nursed and nonnursed infant.

Table 6. Comparison of PCDDs/PCDFs in males and females, background contamination, 1996.

	Blood lipids, pg I-TEQ/g					
	n	Mean age	Mean	Median	5th %	95th %
Male	113	38.6	16.01[a]	14.8	9.1	26.9
Female	29	31.6	16.8	17.4	10.3	26.7

[a]When using the age correction the mean value reduces to 13.2 (7 years, 0.4 pg/year).

Conclusions

A number of conclusions may be drawn from the study presented here:

- The daily consumption of low-level contaminated food leads to the accumulation of PCDDs/PCDFs in human adipose tissue.
- The dominant sources of dioxins in food are fish, beef, and milk, and corresponding dietary products.
- The daily dioxin intake of breast-fed infants is, for the nursing period, 50-fold higher than in adults.
- High consumption of contaminated food may result in a distinct difference in PCDD/PCDF levels compared to normal consumption habits.
- The PCDD/PCDF concentrations based on lipids in human adipose tissue, blood, and milk are very similar.
- The PCDD/PCDF background levels in humans, observed in milk and blood, show a distinct decline over an observed period of 10 years.
- Despite declining PCDD/PCDF trend, the exposure of babies during the breast-feeding period is still a matter of concern and justifies taking measures to reduce PCDD/PCDF emissions into the environment.

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