POLYCHLORINATED DIBENZOFURANS

1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature and molecular formulae and weights*

Chemical Abstracts Service (CAS) names and synonyms, CAS Registry numbers, molecular formulae and molecular weights for selected polychlorinated dibenzofurans (PCDFs) are presented in **Table 1**. The tetra-, penta-, hexa- and hepta-chlorinated compounds are referred to here as TCDFs, PeCDFs, HxCDFs and HpCDFs, or collectively as, for example, Cl_4–Cl_7 PCDFs or hepta/octa-CDFs.

Table 1. Nomenclature, molecular formulae and molecular weights of selected polychlorinated dibenzofurans

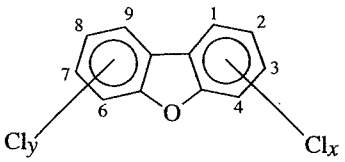
CAS Registry No.	CAS name and synonyms[a]	Molecular formula	Molecular weight
51207-31-9	**2,3,7,8-Tetrachlorodibenzofuran**; F83; TCDF; 2,3,7,8-TCDF; 2,3,7,8-tetra-CDF	$C_{12}H_4Cl_4O$	305.98
57117-41-6	**1,2,3,7,8-Pentachlorodibenzofuran**; F94; 1,2,3,7,8-PeCDF; 1,2,3,7,8-penta-CDF	$C_{12}H_3Cl_5O$	340.42
57117-31-4	**2,3,4,7,8-Pentachlorodibenzofuran**; F114; 2,3,4,7,8-PeCDF; 2,3,4,7,8-PnCDF; 2,3,4,7,8-penta-CDF	$C_{12}H_3Cl_5O$	340.42
70648-26-9	**1,2,3,4,7,8-Hexachlorodibenzofuran**; F118; 1,2,3,4,7,8-HxCDF; 1,2,3,4,7,8-hexa-CDF	$C_{12}H_2Cl_6O$	374.87
57117-44-9	**1,2,3,6,7,8-Hexachlorodibenzofuran**; F121; 1,2,3,6,7,8-HxCDF; 2,3,4,7,8,9-hexachlorodibenzofuran; 1,2,3,6,7,8-hexa-CDF	$C_{12}H_2Cl_6O$	374.87
72918-21-9	**1,2,3,7,8,9-Hexachlorodibenzofuran**; F124; 1,2,3,7,8,9-HxCDF; 1,2,3,7,8,9-hexa-CDF	$C_{12}H_2Cl_6O$	374.87
60851-34-5	**2,3,4,6,7,8-Hexachlorodibenzofuran**; F130; 2,3,4,6,7,8-HxCDF; 2,3,4,6,7,8-hexa-CDF	$C_{12}H_2Cl_6O$	374.87
67562-39-4	**1,2,3,4,6,7,8-Heptachlorodibenzofuran**; F131; 1,2,3,4,6,7,8-HpCDF; 1,2,3,4,6,7,8-hepta-CDF	$C_{12}HCl_7O$	409.31
55673-89-7	**1,2,3,4,7,8,9-Heptachlorodibenzofuran**; F134; 1,2,3,4,7,8,9-HpCDF; 1,2,3,4,7,8,9-hepta-CDF	$C_{12}HCl_7O$	409.31
39001-02-0	**Octachlorodibenzofuran**; F135; OCDF; octa-CDF; perchlorodibenzofuran	$C_{12}Cl_8O$	444.76

[a] Names in bold letters are the Chemical Abstracts Service (CAS) names

1.1.2 *Structural formulae*

The general structure of the PCDFs is shown in **Table 2**. Any or all of the eight hydrogen atoms on dibenzofuran can be replaced with chlorine, giving rise to 135 possible chlorinated dibenzofuran structures. All of the 135 are referred to as congeners (members of a like group) of one another, and congeners having the same number of chlorines are isomers (Clement, 1991).

Table 2. Dibenzofuran structural formula and numbers of chlorinated isomers

Formula	
	
No. of chlorines ($x + y$)	No. of isomers
1	4
2	16
3	28
4	38
5	28
6	16
7	4
8	1
Total	135

1.1.3 *Chemical and physical properties*

Knowledge of basic chemical and physical properties is essential to understanding and modelling environmental transport and fate as well as pharmacokinetic and toxicological behaviour. The most important parameters for the PCDFs appear to be water solubility, vapour pressure, and octanol/water partition coefficient (K_{ow}). The ratio of vapour pressure to water solubility yields the Henry's Law constant for dilute solutions of organic compounds, an index of partitioning for a compound between the vapour and aqueous solution phases (Mackay *et al.*, 1991). Chemical and physical properties of selected PCDFs are presented in **Table 3**.

Limited research has been carried out to determine physical and chemical properties of PCDFs. As with the polychlorinated dibenzo-*para*-dioxins (PCDDs), the tetra- to octachloro congeners with 2,3,7,8-chlorination have received the most attention. Of the large number of possible congeners, only the 2,3,7,8-chlorinated compounds and a few others are available commercially, and preparation and synthesis can be both time-consuming

and difficult. Some of the PCDF congeners have not yet been prepared in pure form. Like the PCDDs, the PCDFs are intentionally prepared only for research purposes.

Table 3. Chemical and physical properties of selected PCDFs[a]

Chemical	Melting point (°C)	Water solubility (mg/L)	Vapour pressure (Pa) at 25 °C	Henry's Law constant (Pa × m^3/mol)[b]	log K_{ow}
2,3,7,8-TCDF	227–228	4.19×10^{-4} at 22.7 °C	2×10^{-6}	1.5	6.53
1,2,3,7,8-PeCDF	225–227		2.3×10^{-7}		6.79
2,3,4,7,8-PeCDF	196–196.5	2.36×10^{-4} at 22.7 °C	3.5×10^{-7}	[0.5]	6.92
1,2,3,4,7,8-HxCDF	225.5–226.5	8.25×10^{-6} at 22.7 °C	3.2×10^{-8}	[1.43]	
1,2,3,6,7,8-HxCDF	232–234	1.77×10^{-5} at 22.7 °C	2.9×10^{-8}	[0.6]	
1,2,3,7,8,9-HxCDF	246–249		2.4×10^{-8}		
2,3,4,6,7,8-HxCDF	239–240		2.6×10^{-8}		
1,2,3,4,6,7,8-HpCDF	236–237	1.35×10^{-6} at 22.7 °C	4.7×10^{-9}	[1.4]	7.92
1,2,3,4,7,8,9-HpCDF	221–223		6.2×10^{-9}		
OCDF	258–260	1.16×10^{-6} at 25 °C	5×10^{-10}	[0.2]	8.78

[a] From Burkhard & Kuehl (1986); Rordorf (1989); Sijm *et al.* (1989); Friesen *et al.* (1990); Mackay *et al.* (1991)

[b] Values in brackets have been calculated by the Working Group.

The concept of toxic equivalency factors (TEFs) was developed by several agencies and national and international organizations (Ahlborg, 1989; Safe, 1990; Ahlborg *et al.*, 1992; Birnbaum & De Vito, 1995) to aid in the interpretation of the complex database and in the evaluation of the risk of exposure to mixtures of structurally related PCDDs and PCDFs. TEF values are derived by evaluating the potency of each PCDD and PCDF isomer relative to that of tetrachlorodibenzo-*para*-dioxin (2,3,7,8-TCDD). TEFs are order-of-magnitude estimates that are based on the evaluation of all available information, including binding to the Ah receptor (see Section 4) and other in-vitro responses as well as in-vivo effects ranging from enzyme induction to tumour formation (Ahlborg *et al.*, 1992; Birnbaum & De Vito, 1995).

The levels of all the individual PCDDs and PCDFs in a mixture may be converted into one value of toxic equivalents (TEQs), as follows:

$$\text{TEQ} = \Sigma \text{ (TEF} \times \text{concentration)}$$

Assignment of relative potencies in a quantitative sense imposes appreciable demands on the experimental data. All congeners must exhibit parallel dose–response curves for the effects studied to be treated as additive. Additivity is an implicit assumption of the TEF concept. Many in-vitro and in-vivo studies have supported the hypothesis that the toxic effects of combinations of PCDDs and PCDFs are additive and have supported the applicability of the TEF concept in practice (Ahlborg *et al.*, 1992a).

Toxicologists have widely adopted the set of TEFs shown in **Table 4** (I-TEFs, also adopted by NATO (North Atlantic Treaty Organization)). Other sets of TEFs have been used in the past (e.g. BGA (German), Nordic, Swiss, Eadon (american)), but TEQs

calculated with these TEFs normally do not differ from those based on I-TEFs by more than a factor of 2 (Ahlborg *et al.*, 1988; Rappe *et al.*, 1993).

Table 4. I-TEFs for 2,3,7,8-substituted PCDFs[a]

Congener	I-TEF
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.001
All other PCDFs	0

[a]From Ahlborg *et al.* (1992a); Rappe *et al.* (1993)
I-TEF, international toxic equivalency factor

1.1.4 *Methods of analysis*

Analysis for PCDFs in the environmental and biological matrices uses essentially the same methods as those developed for PCDDs. In fact, analyses for PCDFs and PCDDs are very frequently performed concurrently. Thus, methods of analysis for both classes of compounds are discussed in the monograph on PCDDs, and the designations A-W used here are those given in Table 5 of the PCDD monograph on page 39.

1.2 Formation and destruction

PCDFs can be formed by a number of different reactions including synthetic chemical, thermal, photochemical and biochemical; analogous pathways can be used for their destruction. PCDFs already present in reservoir sources such as sediments, soil and sewage sludge are significant contributors to current environmental levels.

1.2.1 *Formation of PCDFs*

(*a*) *Chemical reactions*

(i) *Polychlorinated biphenyls (PCBs)*

Mixtures of PCBs (see IARC, 1987d) have been widely used since the 1930s as dielectric fluids in electrical equipment such as cables, transformers and capacitors. They have also been used as non-flammable heat-exchange liquids and as additives to plastics and in cutting oil. The total world production is estimated to exceed 500 000 tonnes

(Rappe *et al.*, 1979a). Primarily due to environmental problems, use of PCBs has now been phased out in most European countries and in many others.

Vos *et al.* (1970) identified PCDFs (tetra- and penta-CDFs) in samples of European PCBs (Phenoclor DP-6 and Clophen A60) but not in a sample of American Aroclor 1260. The toxic effects of these PCBs were found to parallel the levels of PCDFs present. Bowes *et al.* (1975) examined a series of Aroclors as well as the same samples of Aroclor 1260, Phenoclor DP-6 and Clophen A60 previously analysed by Vos *et al.* (1970). They used packed column gas chromatography (GC) with low-resolution mass spectrometry and very few standard compounds and reported that the most abundant PCDFs were PeCDFs (**Table 5**). Using a complete set of PCDF standards and high-resolution GC, Rappe *et al.* (1985a) determined the levels of 2,3,7,8-substituted PCDFs in commercial PCB products (see **Table 6**).

Table 5. Concentrations of PCDFs in PCBs (mg/kg)

PCB[a]	TCDFs	PeCDFs	HxCDFs	Total
Aroclor 1248 (1969)	0.5	1.2	0.3	2.0
Aroclor 1254 (1969)	0.1	0.2	1.4	1.7
Aroclor 1254 (1970)	0.2	0.4	0.9	1.5
Aroclor 1260 (1969)	0.1	0.4	0.5	1.0
Aroclor 1260 (lot AK3)	0.2	0.3	0.3	0.8
Aroclor 1016 (1972)	ND	ND	ND	
Clophen A 60	1.4	5.0	2.2	8.4
Phenoclor DP-6	0.7	10.0	2.9	13.6

From Bowes *et al.* (1975)
ND, not detected at 0.001 mg/kg
[a] Production year in parentheses

(ii) *Chlorophenols*

Chlorophenols (see IARC, 1986b; 1987c) have been used extensively since the 1950s as insecticides, fungicides, mould inhibitors, antiseptics and disinfectants. In 1978, the annual world production was estimated to be approximately 150 000 tonnes (Rappe *et al.*, 1979a). Due to occupational and environmental risks, the use of chlorophenols has now been phased out in most European countries and in a few countries outside Europe. The most important use of 2,4,6-tri-, 2,3,4,6-tetra- and pentachlorophenol (PCP) and their salts is for wood preservation. PCP is also used as a fungicide for slime control in the manufacture of paper pulp and for a variety of other purposes such as in cutting oils and fluids, for tanning leather and in paint, glues and outdoor textiles. **Table 7** summarizes a number of relevant analyses of the levels of PCDFs in commercial chlorophenol formulations (Rappe *et al.*, 1978b).

Buser and Bosshardt (1976) reported the results of a survey of the PCDF and PCDD content of PCP and its sodium salt from commercial sources in Switzerland (analytical method AC). On the basis of the results, the samples could be divided into two groups:

Table 6. Concentrations of PCDFs in commercial PCBs (μg/kg)

PCB type	Tri-CDF	TCDFs		PeCDFs			HxCDFs					HpCDFs
	Total	2378-	Total	12348-/ 12378-	23478-	Total	123479-/ 123478	123678-	123789-	234678-	Total	Total
Pyralene	700	53	630	10	T	35	ND	ND	ND	ND	ND	ND
Aroclor 1254	63	19	1 400	690	490	4 000	2 500	2 100	190	130	10 000	960
Aroclor 1260	10	13	110	48	56	260	500	120	190	27	1 500	1 300
Aroclor 30	500	35	573	14	28	160	50	59	ND	ND	220	T
Aroclor 40	1 300	180	2 600	96	8	1 700	79	68	ND	T	310	ND
Aroclor 50	7 400	3 300	20 000	760	1 100	8 000	700	360	18	98	3 100	75
Clophen A60	770	840	6 900	1 100	990	8 100	1 600	330	170	330	6 800	2 000
Clophen T64	47	23	360	97	122	840	520	390	58	41	2 600	220
Clophen	710	54	1 200	34	30	270	ND	T	ND	ND	T	ND

T, traces; ND, not detected
From Rappe *et al.* (1985a)

a first series with generally low levels (HxCDD, < 1 mg/kg) and a second series with much higher levels (HxCDD, > 1 mg/kg) of PCDDs. Samples with high PCDD levels also had high levels of PCDFs. The ranges of the combined levels of PCDFs were 1–26 mg/kg for the first series of samples and 85–570 mg/kg for the second series of samples. The levels of OCDF were as high as 300 mg/kg and this congener dominated the PCDF content of the samples.

Table 7. Levels of PCDFs in commercial chlorophenols (mg/kg)

	2,4,6-Tri-chlorophenol	2,3,4,6-Tetra-chlorophenol	Pentachlorophenol	
			Sample A	Sample B
TCDFs	1.5	0.5	0.9	≤ 0.4
PeCDFs	17.5	10	4	40
HxCDFs	36	70	32	90
HpCDFs	4.8	70	120	400
OCDF	< 1	10	130	260

From Rappe *et al.* (1978b); Rappe & Buser (1981)

(iii) *Pulp bleaching*

During the 1950s, free chlorine gas was introduced for the bleaching of pulp in pulp and paper mills. In 1986–87, it was first reported that bleaching pulp using free chlorine gas produced 2,3,7,8-TCDF (Rappe *et al.*, 1987a). A survey performed in 1987 in the United States showed that the concentrations of 2,3,7,8-TCDF in bleached pulp ranged from undetectable (at a detection limit of 1.2 ng/kg) up to 330 ng/kg, with a median concentration of 50 ng/kg and a mean of 93 ng/kg (Gillespie & Gellman, 1989).

New technology has been developed for pulp bleaching using chlorine dioxide (ECF, elemental chlorine free) or non-chlorinated reagents (TCF, total chlorine free). 2,3,7,8-TCDF at a level of about 0.2 ng/kg was found in ECF- and TCF-bleached pulp (Rappe & Wågman, 1995).

(iv) *Production of chlorine*

Chlorine is produced primarily by the electrolysis of brine. The annual world production in 1995 was estimated to be 40 million tonnes (Fauvarque, 1996). Rappe *et al.* (1991) reported that residues from the production of chlorine using the chloralkali process were highly contaminated with PCDFs. The problem is particularly associated with the use of graphite electrodes in the process. The graphite electrode process was for a long time the dominant technology and is still very widespread. Chlorine butter, a by-product from this process, was already identified as a chloracnegen 100 years ago. Only a few samples from chloralkali plants have been analysed, primarily from landfills. Total PCDF concentrations of 0.6–0.7 mg/kg have been found. The pattern of PCDFs is dominated by the 2,3,7,8-substituted tetra- to hepta-CDFs, resulting in a nordic TEQ value of 10–30 µg/kg (see **Table 8**).

Table 8. Levels of PCDFs (ng/kg) in three samples of electrode sludge from chloralkali plants

	Sample 1	Sample 2	Sample 3
2,3,7,8-TCDF	26 000	56 000	57 000
Total TCDFs	64 000	150 000	140 000
1,2,3,4,8-/1,2,3,7,8-PeCDF	25 000	55 000	56 000
2,3,7,8-PeCDF	12 000	25 000	24 000
Total PeCDFs	75 000	240 000	240 000
1,2,3,4,7,9-/1,2,3,7,8-HxCDF	32 000	71 000	73 000
1,2,3,6,7,8-HxCDF	7 000	16 000	15 000
Total HxCDFs	68 000	140 000	140 000
1,2,3,4,6,7,8-HpCDF	9 100	19 000	19 000
1,2,3,4,7,8,9-HpCDF	8 100	19 000	20 000
Total HpCDFs	24 000	53 000	54 000
OCDF	31 000	76 000	71 000
TEQ (nordic)	13 000	28 000	28 000

From Rappe *et al.* (1991)

(v) *Production of vinyl chloride*

Stringer *et al.* (1995) reported the analyses of three samples of residues from the production of vinyl chloride (see IARC, 1987e), an intermediate in the production of polyvinyl chloride (PVC) (analytical method ACS). In all samples, the concentrations of PCDFs were much higher than those of PCDDs and the higher chlorinated 2,3,7,8-substituted congeners constituted a substantial proportion (see **Table 9**).

(b) Thermal reactions

(i) *Incineration of municipal waste*

Olie *et al.* (1977) reported the occurrence of PCDDs and PCDFs in fly ash from three municipal incinerators in the Netherlands. Their results indicated only the presence of PCDFs, without isomer identification or quantification. Buser *et al.* (1978a) quantified PCDDs and PCDFs in fly ash from a municipal incinerator and an industrial heating facility in Switzerland. In the former, the level of total PCDFs was 0.1 mg/kg. In the industrial incinerator fly ash, the level was 0.3 mg/kg.

In 1986, a working group of experts convened by the World Health Organization Regional Office for Europe (WHO/EURO, 1987) reviewed the available data on emissions of PCDDs from municipal solid-waste incinerators. They concluded that, because of their high thermal stability, PCDFs were destroyed only after adequate residence times (> 2 s) at temperatures above 800 °C. Total emissions of PCDFs from tests on municipal solid-waste incinerators were reported to range between a few and several thousand ng/m^3 dry gas at 10% carbon dioxide. The WHO working group prepared a table giving a range of estimated isomer-specific emissions for those isomers of major concern with respect to municipal solid-waste incinerators operating under various conditions (**Table 10**).

Table 9. Concentrations of PCDFs in still bottoms and residues from vinyl chloride production (μg/kg)

	Sample 1[a]	Sample 2[b]	Sample 3[c]
2,3,7,8-TCDF	0.91	680	0.44
Total TCDF	15	20 600	6
1,2,3,7,8-PeCDF	9.5	975	1.8
2,3,4,7,8-PeCDF	1.6	1 050	0.58
Total PeCDFs	65	45 300	11
1,2,3,4,7,8-HxCDF	110	10 100	11
1,2,3,6,7,8-HxCDF	24	9 760	2.4
1,2,3,7,8,9-HxCDF	9.5	21 800	1.3
2,3,4,6,7,8-HxCDF	3.1	930	0.89
Total HxCDFs	300	63 700	27
1,2,3,4,6,7,8-HpCDF	250	13 400	38
1,2,3,4,7,8,9-HpCDF	51	1 340	6
Total HpDFs	450	16 600	58
OCDF	390	43 500	650
I-TEQ	20	6 370	3.9

From Stringer *et al.* (1995)

[a] Sample 1 is a process waste including, but not limited to, distillation residues, heavy ends, tars and reactor clean-out wastes, from the production of certain chlorinated aliphatic hydrocarbons by free radical catalysed processes.

[b] Sample 2 is a waste from heavy ends from the distillation of ethylene in ethylene dichloride production.

[c] Sample 3 is a waste from heavy ends from the distillation of vinyl chloride in vinyl chloride monomer production.

The emissions tabulated in column 1 are those which the working group considered to be achievable in modern, highly controlled and carefully operated plants in use in 1986. The results given in column 1 are not representative of emissions that might be expected from such plants during start-up or during occasional abnormal conditions. Emission levels listed in column 2 were considered by the working group to be indicative of the upper limit of emissions from modern municipal solid-waste incinerators. These plants might experience such emissions during start-up or during occasional abnormal conditions, although some of the data reviewed have shown that the figures in column 2 should not be considered to be an absolute maximum. However, most plants existing in 1986, if carefully operated, will have had PCDF emissions in the range between columns 1 and 2. The highest values for municipal solid-waste incinerators (column 3) were obtained by multiplying the values in column 2 by a factor of 5. Emission data that were reported to the working group from all tests and under all circumstances were no greater than these values. Generally, these emission levels are associated with irregular or unstable operating conditions, high moisture content of the municipal solid waste or low combustion or afterburner temperatures. Of special importance is the observation that the major contributor to the TEQ was 2,3,4,7,8-PeCDF.

Table 10. Estimated range of emissions of PCDFs from municipal solid-waste (MSW) and municipal sewage sludge (MSS) incinerators

	Emissions from MSW combustion (ng/m^3)				Emissions from MSS combustion (ng/m^3)
	Achievable with modern plants with no gas cleaning	Maximum from average operation	High emissions[a]	Achievable with modern plants with gas cleaning[b]	Most probably highest emissions
	(1)	(2)	(3)	(4)	(5)
2,3,7,8-TCDF	0.9	10	50	0.1	0.9
1,2,3,7,8-/1,2,3,4,8-PeCDF	2.3	52	260	0.2	2.3
2,3,4,7,8-PeCDF	2.0	40	200	0.2	2.0
1,2,3,4,7,8-/1,2,3,4,7,9-HxCDF	1.1	48	240	0.1	1.1
1,2,3,6,7,8-HxCDF	1.3	40	200	0.1	1.3
1,2,3,7,8,9-HxCDF	0.06	52	260	–	0.06
2,3,4,6,7,8-HxCDF	2.0	36	180	0.2	2.0

From WHO/EURO (1987) excepted when noted

[a] Values obtained by multiplying values in column 2 by a factor of 5

[b] Adapted from ECETOC (1992)

During the second half of the 1980s and 1990, regulatory agencies in several countries, such as Germany, the Netherlands and Sweden, announced strict regulations for municipal solid-waste incinerators. The European Union value is 0.1 ng TEQ/m^3 (European Union, 1994) (see column 4, **Table 10**). This directive has resulted in the introduction of modern air pollution control devices and, together with improved burning conditions, has led to a decrease in PCDF emissions from municipal solid-waste incinerators, which had been considered to be major sources.

(ii) *Incineration of sewage sludge*

Sludge from municipal waste-water treatment plants may be incinerated after being dehydrated. The WHO working group in 1986 reviewed the available data from municipal sewage sludge incinerators and found that PCDD and PCDF emissions from this type of plant were generally lower than emissions from municipal solid-waste incinerators (see Table 10, column 5) (WHO/EURO, 1987).

(iii) *Incineration of hospital waste*

Doyle *et al.* (1985) claimed that the incomplete combustion of certain hospital wastes containing halogenated compounds could produce high emissions of PCDFs. They found the mean levels of total PCDFs to be 156 ng/m^3, but no isomer-specific data were available. Data cited by the United States Environmental Protection Agency indicate that flue gas emissions from hospital waste incinerators are in the range of 10–100 ng I-TEQ/m^3, higher than the levels achievable with modern municipal solid-waste incinerators (United States Environmental Protection Agency, 1994; Thomas & Spiro, 1995). [The Working Group noted that, due to smaller emission volumes, the overall emissions from hospital waste incinerators are generally lower than those from the municipal solid-waste incinerators.]

(iv) *Incineration of polyvinyl chloride (PVC)*

The extent to which PCDFs are formed during the combustion of PVC is a controversial issue. However, the incineration conditions appear to be quite important. On the basis of laboratory experiments, Christmann *et al.* (1989a) considered PVC to be an important source of PCDFs. However, experiments performed in incinerators showed the effect of PVC on the formation of PCDFs to be minimal (Frankenhaeuser *et al.*, 1993; Wikström *et al.*, 1996).

(v) *Combustion of wood*

Schatowitz *et al.* (1994) studied PCDD/PCDF emissions from small-scale laboratory studies of combustion of wood and household waste (analytical method ABS). The results (in ng I-TEQ/m^3) are summarized in **Table 11**. Data on PCDDs and PCDFs are not separated.

(vi) *Automobile emissions*

Hagenmaier *et al.* (1990) reported on the emissions of PCDFs and the lower chlorinated congeners and brominated analogues in automobile emissions from four representative experiments using leaded gasoline, unleaded gasoline with or without catalytic

converters and diesel fuel (analytical method ABS). The results are summarized in **Table 12**.

Table 11. PCDD/PCDF emissions from wood and household waste combustion

Fuel	Furnace	PCDD/PCDFs (ng I-TEQ/m^3)
Beech wood sticks	Fireplace	0.064
Beech wood sticks	Stick wood boiler	0.019–0.034
Wood chips	Automatic chip furnace	0.066–0.214
Uncoated chipboard	Automatic chip furnace	0.024–0.076
Waste wood chips	Automatic chip furnace	2.70–14.42
Household waste	Household stove, closed	114.4

From Schatowitz *et al.* (1994)

Table 12. PCDFs from automobile emissions (pg/m^3)

	Leaded gasoline	Unleaded gasoline	Unleaded gasoline with catalytic converter	Diesel engine
TCDFs	6 628	110	6.8	3.9
2,3,7,8-TCDF	201.4	8.5	0.55	1.04
PeCDFs	1 514	180	6.9	2.0
1,2,3,7,8-PeCDF	141.2	8.6	0.43	< 0.26
2,3,4,7,8-PeCDF	58.4	4.0	0.31	0.36
HxCDFs	824	73	4.3	1.3
1,2,3,4,7,8-HxCDF	111.8	8.1	0.62	0.33
1,2,3,6,7,8-HxCDF	111.8	4.1	0.81	0.34
1,2,3,7,8,9-HxCDF	< 10.0	7.3	0.02	< 0.26
2,3,4,6,7,8-HxCDF	35.7	10.5	0.59	< 0.26
HpCDFs	737	92	3.5	4.1
1,2,3,4,6,7,8-HpCDF	529.2	5.4	2.11	2.07
1,2,3,4,7,8,9-HpCDF	< 10.0	3.2	< 0.02	0.43
OCDF	30	23	3.6	4.8
Total tetra- to octaCDFs	**9 733**	**478**	**25.1**	**16.1**
I-TEQ pg/m^3	141.5	9.8	0.93	1.20
I-TEQ pg/L fuel	1 083.3	50.7	7.20	23.60

From Hagenmaier *et al.* (1990)

(vii) *Metal production*

Steel mills and the manufacturing of iron and steel are considered to be major sources of PCDDs and PCDFs in the environment. The concentrations of PCDFs are much higher than the concentrations of PCDDs in these samples (Rappe, 1994). Jager (1993)

reported on the contamination of scrap samples and I-TEQ levels in flue gas emissions from German steel mills (see **Table 13**) (analytical method ABS).

Table 13. PCDD/PCDF contamination of flue gas, clean gas and flue dust from a steel mill

Proportion of non-metals in scrap added	Flue gas (ng I-TEQ/m^3)	Clean gas (ng I-TEQ/m^3)	Flue dust (ng I-TEQ/g)
Low	0.9	0.02	4.8
Average	1.7	0.04	5.1
High	2.7	0.06	6.0

From Jager (1993)

(viii) *Accidents with electrical equipment containing PCBs*

Buser *et al.* (1978b) reported that pyrolysis of PCBs could cause formation of PCDFs and they issued a warning for this risk. Such an accident occurred in 1981 in the State Office building in Binghamton, NY (United States), when a transformer in the basement exploded and contaminated soot was spread throughout the building (O'Keefe *et al.*, 1985; Schecter, 1986). In the 1980s, following similar accidents involving both transformers and capacitors, people were evacuated from houses and workplaces in the United States, Canada, Sweden, Finland and France (Rappe *et al.*, 1985b; Hryhorczuk *et al.*, 1986; Rappe *et al.*, 1986b). In many cases, analyses showed elevated concentrations of PCDFs in the soot (as high as 2168 mg/kg; the major constituents being 2,3,6,7-/2,3,7,8-TCDFs, 2,3,4,7,8-PeCDF and 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDFs) (Rappe *et al.*, 1985b).

(*c*) *Photochemical reactions*

The photochemical dechlorination of OCDF on soil has been studied by Tysklind *et al.* (1992). Dechlorination occurs preferentially in the lateral (2,3,7,8) positions. Consequently, no 2,3,7,8-substituted PCDFs could be identified in the dechlorination products. Similar results were obtained by Friesen *et al.* (1996) for the photochemical degradation of 2,3,4,7,8-PeCDF, where no 2,3,7,8-TCDF was found.

(*d*) *Biochemical reactions*

Öberg *et al.* (1990) showed that chlorinated phenols could be transformed *in vitro* to PCDFs by peroxidase-catalysed oxidation.

1.2.2 *Destruction of PCDFs*

Although PCDFs are considered to be very stable, they can undergo a series of chemical degradation reactions. Peterson and Milicic (1992) and Oku *et al.* (1995) reported the degradation of a series of PCDFs using a mixture of potassium hydroxide in polyethylene glycol or sodium or potassium hydroxide in 1,3-dimethyl-2-imidazolidinone.

Vollmuth and Niessner (1995) reported no significant degradation of PCDFs by ultraviolet radiation, ozone or a combination of the two.

Thermal degradation of PCDFs occurs at temperatures above 800 °C and at residence times of longer than 2 s (WHO/EURO, 1987), but the conditions required for thermal degradation are matrix-dependent.

Adriaens *et al.* (1995) reported on the biologically mediated reductive dechlorination of 2,3,4,7,8-PeCDF in sediments using inocula derived from contaminated environments.

1.3 Occurrence

All tissue concentrations reported in this section are lipid-based (as ng/kg fat), unless otherwise stated.

1.3.1 *Occupational and accidental exposures to PCDFs*

(*a*) *Occupational exposures*

(i) *Exposure during production of PCBs*

Although several cohorts of PCB workers have been studied, no study has taken PCDFs into account.

(ii) *Exposure during production of TCP and 2,4,5-T*

In the Boehringer-Ingelheim plant in Hamburg manufacturing a range of herbicides, Flesch-Janys *et al.* (1996a) studied 48 workers (45 men and 3 women) (see also monograph on PCDD, p. 55). The blood concentrations of PCDFs in these workers are given in **Table 14**.

Table 14. Concentrations of PCDFs (ng/kg fat) in blood of workers at a German herbicide plant (Boehringer-Ingelheim plant)

		First blood sample[b]		Last blood sample[c]	
	No.[a]	Median	Range	Median	Range
2,3,4,7,8-PeCDF	5	105.9	76.4–406.7	71.3	47.9–108
1,2,3,4,7,8-HxCDF	42	116.7	37.5–1035	61.5	21.9–489.4
1,2,3,6,7,8-HxCDF	31	50.4	28.5–374	30.2	13.6–205.9
2,3,4,6,7,8-HxCDF	6	16.3	10.1–38	8.8	6.1–14.8
1,2,3,4,6,7,8-HpCDF	22	123.1	47.3–1028	45.8	24.7–243
1,2,3,4,7,8,9-HpCDF	6	14.8	3.6–26	3	1.6–5.4

From Flesch-Janys *et al.* (1996a)

[a] Number of persons whose levels exceeded upper background concentrations at all points in time

[b] Mean, 5.4 years after end of employment

[c] Mean, 5.6 years after the first blood sample

(iii) *Exposure during handling and spraying of 2,4,5-T*

Professional pesticide applicators involved in ground-level spraying of 2,4,5-T in New Zealand are claimed to be the group most heavily exposed to agricultural use of 2,4,5-T in the world (Smith *et al.*, 1992a). Many of the applicators sprayed for more than six months per year and some were spraying for more than 20 years. Measurements of PCDFs in blood serum of nine of these workers are given in **Table 15** (see also monograph on PCDDs, p. 57).

Table 15. Levels of PCDFs in serum of nine 2,4,5-T applicators and nine matched control subjects in New Zealand

Congener	Average level (ng/kg fat ± SE)[a]		Ratio[b]
	Applicator	Matched control	
2,3,7,8-TCDF	1.6 ± 0.3	1.7 ± 0.3	0.9
1,2,3,7,8-PeCDF	< 2.1 ± 0.2	< 2.0 ± 0.2	1.1
2,3,4,7,8-PeCDF	8.0 ± 0.9	7.4 ± 0.8	1.1
1,2,3,4,7,8-HxCDF	5.4 ± 0.3	5.1 ± 0.5	1.1
1,2,3,6,7,8-HxCDF	5.5 ± 0.4	5.6 ± 0.6	1.0
1,2,3,7,8,9-HxCDF	< 0.8 ± 0.1	< 0.8 ± 0.1	1.0
2,3,4,6,7,8-HxCDF[c]	< 1.1 ± 0.4	< 1.7 ± 0.2	1.1
1,2,3,4,6,7,8-HpCDF	14.2 ± 0.7	16.0 ± 2.3	0.9
1,2,3,4,7,8,9-HpCDF[c]	< 1.6 ± 0.1	< 1.9 ± 0.3	0.8

From Smith *et al.* (1992a)

[a] Values are adjusted for total lipids in serum.

[b] Ratio, average for applicators/average for matched control subjects

[c] A number of positive signals were below the limit of quantification.

Military personnel in Viet Nam: Nygren *et al.* (1988) analysed adipose tissue and blood samples collected 15–20 years after military service from 27 men, 10 of whom were heavily exposed during their service in Viet Nam, 10 of whom had marginal exposure during service and served as Viet Nam controls and seven veterans who did not serve in Viet Nam and were used as 'era' controls. The results for PCDFs levels are summarized in **Table 16** (see also Section 1.3.1(*a*)(ii) of the monograph on PCDDs).

(iv) *Exposure at incinerators*

Studies of workers at incinerators have not found elevated tissue levels of PCDFs. Rappe *et al.* (1992), Päpke *et al.* (1993a) and Böske *et al.* (1995) found only elevated tissue levels of PCDDs.

(v) *Metal production and recycling*

Triebig *et al.* (1996) analysed the concentrations of PCDFs in the blood of 76 workers in a non-iron recycling plant in the south-western part of Germany. The results were compared with those from a group of 102 controls. Elevated concentrations of PCDFs

Table 16. Concentrations of PCDFs in blood plasma of exposed and unexposed groups of United States Army veterans (ng/kg fat)

Isomers	Arithmetic means						Geometric means		
	Exposed Viet Nam veterans		Viet Nam controls		Era controls[a]		Exposed Viet Nam veterans	Viet Nam controls	Era controls
	Mean	SEM[b]	Mean	SEM	Mean	SEM	Mean	Mean	Mean
2,3,7,8-TCDF	4.0	2.1	3.6	0.9	3.4	1.7	2.3	2.1	1.0
2,3,4,7,8-PeCDF	14.0	2.5	15.2	2.4	19.2	5.9	11.9	13.4	15.2
1,2,3,4,7,8-HxCDF[c]	13.5	1.5	9.3	1.7	11.5	3.0	12.8	7.6	9.2
1,2,3,6,7,8-HxCDF	9.1	1.3	5.4	1.3	7.5	2.6	8.2	3.7	5.7
2,3,4,6,7,8-HxCDF	2.9	0.7	1.9	0.4	1.7	1.7	2.3	1.7	1.5
1,2,3,4,6,7,8-HpCDF	27.2	4.4	20.5	3.6	19.6	2.6	24.6	13.3	16.6

From Nygren *et al.* (1988)

[a] Era controls are veterans who served outside Viet Nam during the period of the Viet Nam conflict.

[b] SEM, standard error of mean

[c] 1,2,3,4,7,8-/1,2,3,4,7,9-HxCDF

were found in the exposed group (up to 1138 ng/kg), around six times above the maximal blood concentrations in controls. Elevated levels were found particularly for 2,3,4,7,8-PeCDF and for HxCDFs (analytical method ACS). In another study, Bergschicker *et al.* (1994) found elevated concentrations of the same PCDFs in a group of 34 workers employed in a primary and secondary copper smelter (analytical method ACS).

Menzel *et al.* (1996) reported elevated blood concentrations of PCDDs/PCDFs in a group of 14 workers employed in welding and cutting of metals (welders: median, 29.9; burners: median, 46.7; referents, 28.3 ng I-TEQ/kg).

Hansson *et al.* (1995) reported a significant increase in Cl_5–Cl_8 PCDFs in the blood of nine workers from a magnesium plant in Norway. They analysed blood samples from workers employed at the plant for 10–36 years and from a control group of nine non-production workers. OCDF was the congener with the highest exposure levels (see **Table 17**).

Table 17. Mean concentrations of PCDFs (expressed in ng/kg fat) in blood plasma from metal workers and controls

PCDF	Controls (n = 9)		Workers (n = 9)		p value[a]
	Mean	Range	Mean	Range	
2,3,7,8-TCDF	2.9	1.2–4.7	3.5	1.5–6.1	0.5
1,2,3,7,8-PeCDF	2.2	< 1–3	5.4	< 1–11	0.06
2,3,4,7,8-PeCDF	20	14–29	51	19–170	0.02
1,2,3,4,7,8-HxCDF	22	5.9–94	59	23–150	0.01
1,2,3,6,7,8-HxCDF	12	5.7–31	59	19–190	0.002
2,3,4,6,7,8-HxCDF	3.4	< 1–6.8	5.4	2–9.6	0.09
1,2,3,4,6,7,8-HpCDF	14	4.7–32	85	21–150	0.0007
1,2,3,4,7,8,9-HpCDF	< 2		6.9	< 2–16	0.0005
OCDF	7.5	< 3–18	216	17–560	0.0005
I-TEQ	25	17–42	60	27–190	0.007

From Hansson *et al.* (1995)

[a] Mann-Whitney U test for comparison between groups (two-tailed)

(vi) *Exposure during production of chlorine gas*

Svensson *et al.* (1993) reported that in a small cohort of workers at a chloralkali plant in Sweden, handling sludge from graphite electrodes had caused exposure to 2,3,7,8-substituted PCDFs (2,3,4,7,8-PeCDF and HxCDFs). However, exposure to contaminated soil did not result in elevated concentrations of PCDFs (analytical method ACS).

(vii) *Exposure in bleached pulp mills*

Rosenberg *et al.* (1995) analysed 34 blood samples from workers at a pulp mill and 14 controls. They found no statistically significant differences in lipid-adjusted concentrations of PCDFs between the two groups.

(viii) *Exposure during production of PVC*

Hansson *et al.* (1997) reported a weak correlation between length of employment in production of PVC (or vinyl chloride monomer) and concentrations of PCDFs in the blood, especially 1,2,3,4,7,8- and 1,2,3,6,7,8-HxCDFs and 1,2,3,4,6,7,8-HpCDF.

(*b*) *Accidental exposure*

Repeated heating of PCBs in the presence of oxygen can result in formation of PCDFs, polychlorinated quaterphenyls (PCQs) and other compounds. In Japan and Taiwan, the use of PCBs as heat exchangers during the deodorizing of cooking oil resulted in contamination of cooking oil, presumably when the processing machines leaked. These incidents caused thousands of cases of PCB/PCDF poisoning when the contaminated oil was distributed and consumed.

(i) Yusho *incident, Japan, 1968*

A mass poisoning, called the *yusho* ('oil disease') incident, occurred in western Japan in 1968. The disease was caused by ingestion of a specific brand of rice oil that was contaminated not only with PCBs but also with PCDFs, PCQs and other substances. About 2000 affected individuals (Chen *et al.*, 1985a) were identified (1870 had been registered by 31 May 1990), primarily in the Fukuoka and Nagasaki prefectures on the island of Kyushu (Ikeda & Yoshimura, 1996). Although *yusho* patients had ingested more than 40 different PCDF congeners, Rappe *et al.* (1979b) found that only a few congeners had been retained by the patients. Most of the retained congeners were found to have the 2, 3, 7 and 8 positions chlorinated. The missing congeners, apparently metabolized and excreted, were those with two vicinal hydrogenated carbon atoms in at least one of the rings. Masuda (1994) determined concentrations of the major PCDF congeners identified in tissues and blood at different sampling times (**see Table 18**). The highest concentrations were found for 2,3,4,7,8-PeCDF and 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDFs. Five years after exposure ended, the mean concentrations of PCBs in the adipose tissue, liver and blood of *yusho* cases were 1.9 mg/kg (ppm), 0.08 mg/kg and 6.7 μg/kg, respectively (Masuda *et al.*, 1985), which were about twice the levels in the control group. Levels of PCDFs in adipose tissue ranged from 6 to 13 μg/kg (Masuda *et al.*, 1985). Sixteen years after exposure, the mean level of PCQs in adipose tissue of *yusho* cases was 207 μg/kg, approximately 100 times the level in Japanese controls (Kashimoto *et al.*, 1985).

(ii) Yucheng *incident, Taiwan, 1979*

In 1979, 11 years after the Japanese *yusho* incident, a similar incident occurred in central Taiwan. About 2000 persons were identified as *yucheng* patients, primarily from Taichung and Changhwa counties (Chen & Hites, 1983; Chen *et al.*, 1985a). Oil samples were found to be contaminated with PCBs, PCDFs and PCQs, like the *yusho* oil. However, the average chlorination level seemed to be higher in the Taiwanese oil than in

Table 18. Concentrations of PCDF congeners in tissues of *yusho* patients

Year of sampling	Tissue	PCBs (mg/kg)	PCDFs (μg/kg wet weight)			
			2,3,7,8-TCDF	2,3,4,7,8-PeCDF	1,2,3,4,7,8-/1,2,3,6,7,8-HxCDF	1,2,3,4,6,7,8-HpCDF
1969	Liver	1.4	0.3	6.9	2.6	
1969	Liver	0.2	0.02	1.2	0.3	
	Adipose	2.8	0.3	5.7	1.7	
1972	Liver	0.03	< 0.01	0.3	0.03	
	Adipose	4.3	ND	0.8	0.2	
1975	Adipose	0.2	ND	0.1	0.5	
1977	Liver	0.06	ND	1.49	5.31	1.39
	Adipose	3.0	0.002	1.45	1.99	0.22
	Lung	0.016	0.002	0.365	0.41	0.05
1985	Uterus	0.005	ND	0.026	0.031	ND
1982	Comedo	0.2	ND	0.36	0.39	0.1
1986	Adipose[a]	2.2	0.003	1.4	0.51	
1986	Adipose[b]	2.3	0.028	0.77	0.66	0.036
	Blood[b]	0.0085	0.0025	0.0025	0.0004	
	Control subject					
1986	Adipose[c]	1.1	0.007	0.02	0.02	0.0009
	Blood[c]	0.0033	0.00007	0.00006	0.0009	

From Masuda (1994)

[a] Average of seven patients

[b] Average of six patients

[c] Average of three controls

the Japanese oil. The major PCDF was 1,2,3,4,7,8-HxCDF. Chen & Hites (1983) analysed tissue samples from a deceased patient (see **Table 19**). PCDFs in the blood of 10 patients were analysed by GC with negative chemical ionization–mass spectrometry. The blood samples were collected 9–27 months after the onset of poisoning. The total PCDFs in the blood of 10 patients ranged from 0.02 to 0.20 μg/kg, the major components being 1,2,3,4,7,8-HxCDF and 2,3,4,7,8-PeCDF. Minor amounts of 1,2,3,4,6,7,8-HpCDF and 1,2,4,7,8-PeCDF were also found (Chen *et al.*, 1985b). In *yucheng* patients, within the first year of exposure, mean serum PCB, PCDF and PCQ levels for 15 cases were 60 mg/kg (range, 4–188 mg/kg, 0.14 μg/kg (range, < 0.005–0.27 μg/kg) and 19.3 μg/kg (range, 0.9–63.8 μg/kg), respectively (Kashimoto *et al.*, 1985). Analysis of PCB levels in 1980–81 in 165 cases (mean, 38 μg/kg; range, 10–720 μg/kg) (Rogan, 1989) and in 1985 in 32 cases (mean, 15.4 μg/kg; range, 0.6–86.8 μg/kg) (Lundgren *et al.*, 1988) suggested that some PCBs were being eliminated. [The Working Group noted that it was not clear from the reports if the samples were drawn from distinctly different individuals or included some of the same individuals.]

Table 19. Concentrations of PCDF congeners in the tissues of a deceased patient with *yucheng* in Taiwan

Tissue	Level of PCDF congener (μg/kg)		
	1,2,4,7,8-PeCDF	2,3,4,7,8-PeCDF	1,2,3,4,7,8-HxCDF
Liver	3.4	6.3	25.4
Intestinal fat	0.9	4.0	7.8
Bronchus	0.4	1.8	3.2
Large intestine	0.3	1.2	2.3
Heart	0.2	0.8	1.4
Stomach	0.05	0.23	0.4
Small intestine	0.05	0.21	0.34
Kidney	0.04	0.18	0.32
Lung	0.01	0.06	0.15
Brain	0.01	0.06	0.15
Spleen	0.01	0.08	0.1

From Chen & Hites (1983)

(*iii*) *PCB explosions and fires*

After the accident at Binghamton, NY (United States), in 1981 (see Section 1.2.1(*b*)(viii), 74.7 ng/kg 2,3,4,7,8-PeCDF, 149 ng/kg 1,2,3,4,7,8-HxCDF, 39.3 ng/kg 1,2,3,4,6,7,8-HpCDF and 25.9 ng/kg 1,2,3,4,7,8,9-HpCDF were found in the adipose tissue of an exposed person (Schecter *et al.*, 1985a). After an accident in Reims, France, in 1985, < 4 ng/kg 2,3,4,7,8-PeCDF, < 2 ng/kg 1,2,3,4,7,8-HxCDF, < 14 ng/kg total HpCDFs as well as < 12 ng/kg total HpCDDs and < 23 ng/kg OCDD were found in the blood of 6 exposed persons (Rappe *et al.*, 1986b).

1.3.2 *Environmental occurrence* (see also Appendix 2)

Most of the analytical data on environmental levels of PCDFs are from studies measuring PCDDs and PCDFs, often reported as a total I-TEQ (see the monograph on PCDDs as well as Appendix 1). However, in some studies, separate data on PCDFs were reported.

(*a*) *Air* (see Appendix 2, Table 1)

In a baseline study on PCDDs and PCDFs in ambient air (Eitzer & Hites, 1989), 55 samples were taken between 1985 and 1987 at three sites in Bloomington, IN (United States). A set of samples was also taken in the Trout Lake, WI, area, a much more rural area than Bloomington. There was consistency in the isomer pattern within a group of isomers, but overall levels of the various groups had somewhat more variation. Some of this variation was related to the atmospheric temperature; for example, more of the lower chlorinated PCDFs were found in the vapour phase at higher temperatures. The TCDF

distribution between vapour and particulate phases at various temperatures (with a detection limit of 1 fg/m^3 (femtogram = 10^{-15} g)) was as follows: at 3 °C, 50% vapour phase and 50% particulate phase; at 12 °C, 80% vapour phase and 20% particulate phase; and at 26 °C, > 95% vapour phase and < 5% particulate phase.

Airborne concentrations of PCDDs and PCDFs in office buildings and in ambient outdoor air in Boston, MA (United States), were measured by Kominsky and Kwoka (1989). Twelve of the 16 samples were collected inside the buildings and four samples were collected at the ambient air intake plenums of the buildings. PCDFs were generally not detected, except for three samples that showed detectable concentrations of TCDFs and PeCDFs. Two of these samples (one inside and one ambient air) contained 2,3,7,8-TCDF. The I-TEQs for the two samples containing 2,3,7,8-TCDF were 0.34 and 0.20 pg/m^3, respectively.

(*b*) *Water* (see Appendix 2, Table 2)

Muir *et al.* (1995) analysed water and other matrices downstream from a bleached kraft pulp mill on the Athabasca River (Alberta, Canada) in 1992. The 'dissolved phase' and the suspended particulates from centrifuged samples were analysed for 41 PCDDs/-PCDFs ranging from mono- to octa-chlorinated congeners. Most PCDD congeners (including 2,3,7,8-TCDD) were undetectable (< 0.1 pg/L) in the centrifugate; however, concentrations of 2,3,7,8-TCDF were above the detection limits at the 1 km site (Weldwood; 0.1 pg/L) and at 48 km (Emerson Lake; 0.09 pg/L). In 1993, 2,3,7,8-TCDF was not detected in the dissolved phase (< 0.1 pg/L) either 1 or 19 km downstream.

In a survey conducted in 1986 for PCDDs/PCDFs and other pollutants in finished water systems throughout New York State (United States), two TCDFs were found at concentrations of 1 pg/L in one finished drinking-water (Meyer *et al.*, 1989). Except for a trace of OCDF detected in one location, no other PCDD or PCDF was detected in any of the 19 other community water systems surveyed.

(*c*) *Soil* (see Appendix 2, Table 3; also Appendix 1, Tables 4, 5 and 8)

Topsoil samples were collected at six typical locations in Flanders, Belgium, including potential PCDD/PCDF source areas, and analysed [but not reported] isomer-specifically for PCDDs and PCDFs (Van Cleuvenbergen *et al.*, 1993). Concentrations in the 0–3-cm soil fraction, averaged per location, ranged between 2.1 ng/kg at a rural location and 8.9 ng/kg (both as I-TEQ) in an industrialized area. Generally, PCDFs made up 70 ± 6% of the I-TEQ, whereas they accounted for 34 ± 6% of the sum of the concentrations of the seventeen 2,3,7,8-substituted congeners.

At the end of 1990, high levels of PCDFs and PCDDs (10–100 µg I-TEQ/kg) were detected in the surface gravel of playgrounds and sports fields in Germany during routine monitoring. The source of this contamination was identified as a fine-grained copper slag which originated from a former copper smelter at Marsberg, Germany. This material had been used as a cover layer due to its red colour (*Kieselrot*), its reduced dust formation compared to other gravels and its quick drying after rain. The PCDDs/PCDFs in the copper slag were formed as by-products of a chlorinating roasting process when up to 8% sodium chloride and coal were added to a copper slag from an old mining process.

About 800 000 tonnes of red copper slag was produced by this process, and a large quantity was used. *Kieselrot* contains a large number of highly chlorinated aromatic compounds as its main organic components. It shows an unusual congener profile for PCDDs and PCDFs. The total amount of PCDFs was about one order of magnitude higher than that of PCDDs. Furthermore, the concentrations increased from TCDFs to OCDF by at least one order of magnitude. The levels of OCDF exceeded those of OCDD by more than a factor of 10. These concentration ratios are typical for metallurgical processes and have been found in the emissions from magnesium production. Typical OCDF and I-TEQ values in *Kieselrot* were 6311 and 64.5 μg/kg, respectively (Döring *et al.*, 1992; Theisen *et al.*, 1993).

In connection with analyses of highly contaminated samples related to the production and use of chlorine (e.g., chloralkali electrolysis sludge and chromate sludge) in Sweden, Rappe *et al.* (1991b) also found high PCDF levels in surface soil samples in the vicinity of the production plant. A typical PCDF congener pattern, called the 'chloralkali pattern', was identified in these soil samples.

(*d*) *Food* (see Appendix 2, Table 4)

All of the relevant data on PCDFs in foods derive from studies of both PCDFs and PCDDs. The monograph on PCDDs in this volume should be consulted for general remarks. The data included have been selected to meet a number of criteria, including: relevance to dietary intake rather than environmental monitoring, appropriate detection limits and appropriate analytical methodology. Some exceptions have, however, been made and some data that are available only as summed TEQs have been included where they seem to be of value. Additionally, the results included either were available on a lipid basis or could be so converted using either reported fat contents or reasonable assumptions. Some further results omitted from the tables are discussed in the text below. Certain entries in the Appendix 1 tables (for PCDDs) do not appear in Appendix 2, since PCDFs were either not determined, or not reported separately from the summed TEQ.

Summed TEQ concentrations are included in Tables 9–18 in Appendix 1 and these have, in most cases, been recalculated using I-TEF values and assuming that congeners that were not detected were present at the full value of the detection limit, unless limits of detection were not reported (indicated as 'ND').

Ryan *et al.* (1990) found an average concentration of 73.3 ng/kg 2,3,7,8-TCDF in cow's milk, much higher than in other reports (see Appendix 2, Table 4), and in a subsequent study showed this to be due to migration into milk from bleached paperboard containers (Ryan *et al.*, 1991). In Germany, Beck *et al.* (1990a) also found elevated levels of 2,3,7,8-TCDF in milk from cardboard containers, in the range 1.6–28 ng/kg (mean, 9.6 ng/kg), contrasting with a range of < 0.1–1.4 ng/kg (mean, 0.7 ng/kg) in other milk. The non-toxic 1,2,7,8-TCDF isomer also migrates. This congener is not normally present in milk but was found at a mean concentration of 4.9 ng/kg in milk from cardboard packages (Beck *et al.*, 1990a) and by Ryan *et al.* (1991) at a mean concentration of 80 ng/kg. Similar increases in 2,3,7,8-TCDF levels were found in studies in New Zealand (Buckland *et al.*, 1990b), the United Kingdom (Startin *et al.*, 1990) and the United States (Glidden *et al.*, 1990) and elsewhere. However, in Sweden, Rappe *et al.* (1990b) found

little or no such migration in cardboard-packaged milk from four out of five towns (5 ng/kg in one). Since 1990, the concentrations of PCDFs in pulp products have been substantially reduced. Thus, in samples from the United Kingdom Total Diet Study (Wright & Startin, 1995), a relatively high 2,3,7,8-TCDF level of 6.6 ng/kg was found in milk collected in 1982, together with 1,2,7,8-TCDF clearly indicating chlorine bleaching as the source, while neither congener was detected in milk collected in 1992.

If Ryan's atypical data are excluded, the samples of milk and dairy products from various locations, which are dominated by European samples from the late 1980s and early 1990s, have mean concentrations of individual PCDFs between about 0.2 and 2.8 ng/kg (Appendix 2, Table 4). The ratios between the lowest and highest measurements for different PCDF congeners vary from around 30 to several hundred. [The Working Group noted that data for OCDF should be treated with particular caution; at these concentrations, this congener is especially difficult to determine accurately and the range of reported concentrations is wide.] Apart from OCDF, 2,3,4,7,8-PeCDF tends to have the highest concentration in most European samples, but not in the single analysis presented of milk from the United States (Eitzer, 1995).

Milk produced close to sites associated with contamination from incineration and similar processes can contain relatively high concentrations of PCDFs (**Table 20**).

Analyses of meats and meat products indicate mean concentrations for most 2,3,7,8-chlorinated congeners of between 1 and 4 ng/kg, with 1,2,3,7,8,9-HxCDF and 1,2,3,4,7,8,9-HpCDF generally at lower concentrations. As with PCDDs, samples of animal liver show considerably higher concentrations.

The rather limited data on poultry meat and eggs suggest that concentrations are usually of the same order of magnitude as those seen in other animal products.

In fish, the pattern of concentrations of different congeners tends to be more extreme and more variable. It is unusual for 1,2,3,7,8,9-HxCDF to be detectable, while most of the tetra- and pentachlorinated congeners are present at higher concentrations than in terrestrial animal products. The predominant congener in fatty fish from the Baltic Sea is 2,3,4,7,8-PeCDF (Svensson *et al.*, 1991). 2,3,7,8-TCDF has been reported in retail samples of marine fish species at concentrations around 100 ng/kg (Beck *et al.*, 1989a; Liem *et al.*, 1991a).

1.4 Human tissue measurements (see Table 21)

Most of the analytical data on biological monitoring of PCDFs have been reported in studies in which both PCDDs and PCDFs were measured (see the monograph on PCDDs in this volume; Section 1.4 and Tables 24, 25 and 27).

In some studies, individual data on PCDFs are reported. All concentrations reported in this section are lipid-based (as ng/kg fat), unless otherwise stated.

Table 20. Concentrations of PCDFs in cow's milk from contaminated areas

Reference	Origin	Sample year	No.	PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Riss *et al.* (1990)	Austria, Brixlegg (metal reclamation)	1988	1	7.4	6.3	29.5	26.5	17.1	–	18.8	–	–	–
			1	9	6.5	57.6	26.8	17.8	–	9.4	–	–	–
Rappe *et al.* (1987b)	Switzerland, Hunzenschwil (MSWI)	–		[< 0.49]	[< 0.45]	[9.62]	[2.91]	[4.25]	ND	[6.26]	[11.0]	ND	[< 3.58]
	Rheinfelden (Cl compound manuf.)			[< 0.80]	[< 0.92]	[6.59]	[2.41]	[1.69]	ND	[1.40]	[< 5.16]	ND	[< 14.9]
	Suhr (MSWI)			[< 1.01]	[< 1.14]	[6.94]	[1.89]	[3.00]	ND	[3.79]	[8.83]	ND	[< 6.62]
Startin *et al.* (1990)	UK, incinerator	1989	1	[0.25]	[0.075]	[1.775]	[1.05]	[0.75]	[< 0.525]	[0.65]	[0.575]	[< 0.5]	[< 3.825]
			1	[0.275]	[< 0.6]	[2.625]	[1.25]	[1.025]	[< 0.55]	[0.925]	[< 1.8]	[< 0.85]	[< 3.05]
	UK, urban/industrial	1989	1	[0.525]	[0.3]	[6.1]	[1.375]	[0.9]	[< 0.425]	[0.725]	[0.425]	[< 0.25]	[< 1.95]
			1	[0.45]	[0.35]	[3.875]	[0.875]	[0.55]	[< 0.25]	[0.375]	[< 0.5]	[< 0.5]	[< 4]
Harrison *et al.* (1996)	UK, Derbyshire (Cl compd. Manuf.) (Farm A) (4% fat assumed)	1990	1	[0.5]	[0.5]	[7.75]	[2.75]	[3]	[< 0.25]	[3.25]	[1.5]	[0.25]	[0.75]
Eitzer (1995)	USA, Connecticut, (incineration) (4% fat assumed)	1993	12	[0.325]	[0.0325]	[0.0825]	[0.1725]	[0.0975]	[0.0675]	[0.1725]	[0.8]	[0.185]	[8.5]

Summed I-TEQs are given in the PCDD monograph in this volume.

–, not reported; ND, not detected and limit of detection not reported; [ ], calculated by the Working Group; MSWI, municipal solid-waste incinerator.

Table 21. Concentrations of PCDFs in human samples

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Austria														
Riss *et al.* (1990)	Brixlegg;	(1)	88	CSN	< 14	7.5	839	116	113	–	15	–	–	–
	blood from farmer	(1)			< 10	1.8	119	27.2	30	–	3.2	–	–	–
Canada														
Ryan *et al.* (1985b)	Kingston/Ottawa[a]; adipose	(8)	79–81	BSIW	2.9 ± 1.9 (6 pos.)	–	16.9 ± 21.4 (6 pos.)	–	–	–	–	–	–	–
Ryan *et al.* (1985c)	Adipose			BSIW										
	Québec	(5)	72		–	–	20.5 (4 pos.)		17.0 (1 pos.)	–	–	28.9	–	–
		(10)	76		–	–	21.7		2.8 (8 pos.)	–	–	23.5 (9 pos.)	–	–
	British Columbia	(5)	72		–	–	30.8		59.7	–	–	55.1	–	–
		(10)	76		–	–	18.3		21.8	–	–	38.3	–	–
	Maritimes	(10)	76		–	–	16.3		11.9 (2 pos.)	–	–	25.1	–	–
	Ontario	(6)	76		–	–	10.4		12.7	–	–	28.0	–	–
	Prairies	(10)	76		–	–	14.8		22.4 (6 pos.)	–	–	48.9 (8 pos.)	–	–
	E. Ontario[a]	(10)	80		–	–	18.4 ± 6.3		17.3 ± 6.9	–	–	39.4 ± 19.6	–	–
LeBel *et al.* (1990)	Adipose			BSO										
	Ontario	(76)	84		2.4 ± 2.6 (ND–10.8)	–	31.3 ± 19.1 (3.4–113)		35.6 ± 20.6 (6.1–107.6)	–	3.7 ± 3.1 (ND–15.2)	25.2 ± 15.0 (8.1–113)	–	4.7 ± 5.2 (ND–27.9)
	Kingston[a]	(13)	79–81		3.5	–	39.0		47.5	–	5.9	44.2	–	16.7
	Ottawa[a]	(10)	79–81		3.1	–	27.6		35.0	–	4.4	29.9	–	7.3
Teschke *et al.* (1992)	British Columbia; adipose, residents of forest industry region	(41)	90–91	CSO	2.2 (0.46–7.5)	1.9 (0.22–13)	10 (2.6–27)	11 (1.2–27)	10 (2.9–26)	3.3 (1.4–9.6)	0.80 (0.06–4.2)	19 (7.6–61)	0.60 (0.33–0.89)	3.7 (1.4–7.0)
China														
Ryan *et al.* (1987)	Shanghai; adipose[b]		84	BSOW										
	LC, 58%	(1)			9.7[c]	–	12			< 2		< 2	–	–
	LC, 50%	(1)			7.4[c]	–	5.8			< 2		< 2	–	–
	LC, 73%	(1)			6.4	–	15			< 2		< 2	–	–
	LC, 70%	(1)			2.7	–	12			16		< 2	–	–
	LC, 73%	(1)			< 2.8	–	14			25		< 2	–	–
	LC, 72%	(1)			< 2	–	9.9			18		< 2	–	–
	LC, 76%	(1)			< 1.8	–	14			14		< 2	–	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
China (contd)														
Schecter (1994)	Blood, general population;		92	BSOW										
	15–19 years	(50)			< 4.2	< 1.6	2.7	3.4	2.1	< 1.0	1.9	5.1	< 1.2	< 5.0
	> 40 years	(50)			2.7	< 1.0	2.7	4.7	3.0	< 1.0	2.7	7.7	< 2.3	< 5.0
Finland														
Rosenberg *et al.* (1995)	Plasma, general population; age, 28–60 years	(14)	89–90	BSIW	1.9 (0.4–5.3)	1.2 (0.3–2.7)	29 (7.8–60)	10 (3.8–17)	9.5 (4.4–18)	1.6 (1.4–1.8)	4.8 (2.6–10)	64 (14–136)	< 0.2	–
France														
Huteau *et al.* (1990a)	Paris; adipose; 6 patients, age, 54–82 years	(8)	< 90	BSO	5.7 (7 pos.) (2.4–12)	18.3 (3 pos.) (1–35.4)	104 (8 pos.) (19.3–242)	12.7 (4 pos.) (9.8–16.0)	11.5 (3 pos.) (9.8–14.4)	6 (1 pos.)	–	59.7 (2 pos.) (16.2–103)	–	–
Germany														
Beck *et al.* (1989b)	Hamburg residents; adipose	(20)	86	BSIW	2.5 (0.7–6.0)	0.4 (0.1–1.6)	40 (10–101)	15 (4.8–39)	16 (4.7–47)	–	4.7 (2.1–10)	20 (7.2–35)	–	0.4 (0.1–0.8)
Thoma *et al.* (1990)	Munich residents		< 89	BSO										
	Adipose	(28)			2.5 (0.7–12.8)	35.2 (7.6–93.3)			41.4 (15.8–146.0)			14.2 (3.8–45.6)		4.0 (1.2–13.5)
	Liver	(28)			5.5 (0.9–45.3)	173.7 (36.7–643)			398.5 (40.8–1801)			218.9 (12.2–757)		29.7 (4.3–65.8)
	Infants; adipose	(8)			2.1 (1.0–4.6)	16.1 (5.3–38.7)			10.4 (3.9–23.6)			4.2 (1.9–7.3)		4.8 (2.5–9.1)
Päpke *et al.* (1992)	General population; blood	(102)	89–90	BSOW	2.3 (0.5–6.7)	2.0 (0.5–7.1)	37 (6.3–99)	15.4 (3.6–49.0)	13.3 (2.7–53.0)	1.7 (0.5–9.4)	4.3 (0.5–14.0)	23.4 (4.8–55.0)	1.5 (0.5–4.0)	4.2 (1.0–15.0)
Kieselrot-studie (1991)	General population; blood	(56)	91	BSOW	4.2 (ND–12)	1.4 (ND–4.4)	34.5 (11–91)	13.9 (4.0–34)	15.9 (6.6–33)	0.5 (ND–4.7)	4.5 (ND–7.9)	22.4 (10–66)	0.4 (ND–4.2)	3.5 (ND–71)
Päpke *et al.* (1993b)	General population; blood	(44)	92	BSOW	1.2 (1.2–3.8)	0.4 (ND–2.5)	18.8 (6.8–48.2)	10.9 (4.4–24.5)	7.8 (3.1–20.7)	ND (ND–1.2)	2.9 (ND–9.9)	19.0 (8.5–38.4)	0.4 (ND–2.4)	0.4 (ND–2.4)

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Germany (contd)														
Schrey *et al.* (1992)	General population; blood	(95)	91	BSOW	1.37 (0.16–5.9)	0.64 (0.30–1.1)	34.3 (6.7–110)	11.5 (3.9–29)	16.5 (5.3–42)	–	3.67 (62–7.9)	15.4 (6.8–45)	0.48 (0.28–1.1)	1.10 (0.24–3.3)
Päpke *et al.* (1994b)	General population; blood	(70)	93	BSOW	2.2 (0.5–5.4)	0.7 (0.5–2.8)	15.3 (5.0–42.1)	8.8 (3.9–22.0)	6.5 (2.4–18.8)	ND	2.8 (1.0–5.6)	12.5 (5.1–36.3)	0.9 (0.5–3.1)	3.5 (1.9–6.1)
Päpke *et al.* (1996)	General population; blood	(134)	94	BSOW	1.9 (0.9–4.3)	0.5 (ND–1.8)	12.8 (3.2–41.3)	7.9 (2.5–19.4)	5.8 (1.8–16.3)	ND	2.6 (1.0–6.9)	11.4 (4.0–27.4)	0.6 (ND–2.0)	2.6 (ND–2.0)
	General population; blood		96	BSOW										
	18–71 years	(139)			1.2 (ND–2.0)	0.6 (ND–1.0)	10.9 (3.2–29.7)	6.5 (2.6–14.5)	4.7 (2.0–8.9)	ND	2.4 (0.5–4.8)	8.1 (4.1–18.3)	0.9 (0.5–0.9)	2.4 (1.5–3.2)
	18–30 years	(47)			1.2 (0.5–1.9)	0.6 (0.5–1.9)	8.2 (3.2–14.3)	5.8 (2.6–11.6)	4.1 (2.2–8.5)	ND	2.4 (1.4–4.1)	9.2 (4.2–18.3)	1.0 (0.7–1.6)	2.5 (1.8–2.5)
	31–42 years	(48)			1.3 (0.5–1.8)	0.6 (0.5–1.0)	10.9 (4.8–20.1)	6.4 (2.8–14.5)	4.8 (2.0–8.4)	ND	2.4 (0.5–4.4)	7.8 (4.7–16)	1.0 (0.8–1.8)	2.4 (1.6–3.1)
	43–71 years	(44)			1.2 (0.5–2.0)	0.5 (ND–1.0)	13.8 (5.2–29.7)	6.9 (3.0–13.3)	5.3 (2.2–8.9)	ND	2.5 (1.0–4.8)	7.1 (4.1–15.1)	0.8 (0.5–1.0)	2.4 (1.5–3.2)
Wittspiepe *et al.* (1993)	Marberg, near copper smelter; blood	(56)	91	BSOW	4.9 (ND–12)	1.8 (ND–10)	41.6 (13–240)	24.4 (5.1–120)	35.1 (6.5–280)	1.1 (ND–8.0)	5.6 (0.7–20)	42.1 (7.4–180)	0.5 (ND–3.2)	5.8 (ND–57)
Körner *et al.* (1994)	Mammary tumour tissue	(7)		BSO	3.2 (1.1–7.0)	1.3 (< 2–4.1)	39.3 (19.5–73.8)		33.5 (16.8–52.3)			14.4 (6.1–23.8)	–	8.1 (2.9–13.8)
Wuthe *et al.* (1990)	Near metal reclamation plant; blood	(22)	89	BSOW	4.9 (1.4–9.8)		72.9 (28–228)		108.6 (43.7–411)				48.3 (16–190)	4.3 (1.2–8.8)
Wuthe *et al.* (1993)	One woman		92	BSOW										
	Blood	(1)			1.8		9.7		19.1				15.4	< 4.4
	Milk	(1)			1.0		13.3		16.3				5.7	0.5
Ewers *et al.* (1994)	Allotment gardeners; blood	(21)	92	BSOW	1.1 (0.5–4.1)		32 (23–76)		27 (16–54)				12 (0.6–30)	2.7 (1.3–6.7)

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Germany (contd)														
Beck *et al.* (1994)	Infants, 3–23 months		< 93	BSOW										
	Adipose	(8)			1.1 (< 0.5–3.1)	< 0.5	8.7 (1.6–33)	4.4 (1.0–12)	2.8 (0.7–9.3)	–	1.0 (< 0.5–3.0)	5.2 (1.0–12)	–	< 0.5 (< 0.5–1.2)
	Liver	(8)			–	–	23 (3.3–82)	27 (< 2–107)	30 (2.9–85)	–	5.6 (< 1–14)	50 (< 4–169)	–	9.1 (4.5–15)
	Spleen	(8)			2.1 (< 1–< 10)	2.1 (< 1–< 10)	15 (< 5–68)	7.0 (1.4–28)	5.0 (1.3–16)	–	4.6 (1.0–20)	10 (1.9–31)	–	1.6 (< 1–< 5)
	Thymus	(8)			5.6 (3.2–7.5)	3.4 (< 1–7.5)	14 (6.1–37)	5.1 (2.2–< 15)	5.6 (3.5–< 15)	–	5.0 (2.1–< 15)	8.3 (2.6–19)	–	4.2 (< 1–< 15)
Jödicke *et al.* (1992)	Infant, 3 months		91	BSO										
	Stool	(1)			< 2	< 2	11.0	12.7		–	< 5	30.7	–	< 5
	Mother's milk	(2)			< 0.5–< 0.7	0.4–< 0.7	13.5–17.5	7.1–7.3		–	1.3–1.6	3.8–4.3	–	< 1–< 2
Welge *et al.* (1993)	Blood		92	BSOW										
	Vegetarians	(24)			0.94 (0.16–2.3)	0.64 (0.42–0.89)	25.8 (11–50)	8.1 (4.2–13)	11.8 (5.3–18)	–	3.16 (0.8–5.0)	14.2 (5.6–72)	0.40 (0.3–0.5)	2.0 (0.2–6.7)
	Non-vegetarians	(24)			1.3 (0.36–2.1)	0.66 (0.36–1.1)	25.5 (9.9–80)	9.1 (4.2–20)	12.9 (5.3–33)	–	3.16 (0.66–7.9)	14.6 (8.5–22)	0.38 (0.4–0.77)	1.4 (0.66–3.6)
Abraham *et al.* (1995a)	Mother's blood	(3)	93–94	BSOW	[< 2.2]	[< 1.0]	[8.5]	[4.7]	[3.2]	–	[1.0]	[4.8]	–	[< 2.4]
	Placenta	(3)			[< 2.4]	[< 1.0]	[8.0]	[2.5]	[1.7]	–	[< 1.0]	[2.2]	–	[< 3.0]
	Umbilical cord	(3)			[< 2.0]	[< 1.0]	[4.3]	[2.7]	[1.7]	–	[< 1.0]	[4.3]	–	[< 5.0]
	Meconium	(1)			[< 2.8]	[< 1.0]	[4.3]	[2.2]	[1.8]	–	[1.0]	[4.3]	–	[< 5.0]
Guam														
Schecter *et al.* (1992)	Whole blood	(10)	89	BSOW	3.9	0.5	9.3	6.3	5.4	0.5	1.2	34.1	0.9	6.4
Japan														
Miyata *et al.* (1977)	Omentum	(5)	75	CRO	< 5	ND–45.4				< 5.0		–	–	–
	Liver	(4)			< 5	ND–17.2				< 5.0		–	–	–
	Mamma	(1)			< 5	19.7				< 5.0		–	–	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Japan (contd)														
Ryan (1986)	Adipose		84	BSIW										
	Age 21; LC, 43%	(1)			–		17			20		–	–	–
	Age 33; LC, 37%	(1)			–		51			ND		–	–	–
	Age 46; LC, 67%	(1)			–		31			64		–	–	–
	Age 55; LC, 80%	(1)			–		29			41		–	–	–
	Age 64; LC, 71%	(1)			–		31			49		–	–	–
	Age 70; LC, 56%	(1)			–		36			168		–	–	–
	Mean; LC, 59%				–		33			68		–	–	–
Ono *et al.* (1986)	Cancer patients; adipose[b]	(13)	85	CSI	9 (3–12)	–	25 (4–71)	15 (4–24)	14 (3–28)	–	8 (4–16)	–	–	–
Okagi *et al.* (1987)	Big city; adipose	(1)	85	CRI	6	–	29	14	–	–	–	–	2	8
Hirakawa *et al.* (1991)	Controls; adipose	(8)	< 91	BSI	3 (1–7)	–	21 (8–30)	7 (3–13)	9 (3–20)	–	–	5 (2.8)	–	–
Masuda (1996)	Control; serum		91–92	BSIW	4.7	11.4	0.8	11.9	8.3	–	3.4	0.9	–	–
Netherlands														
van Wijnen *et al.* (1990)	Liver		< 90	BSIW										
	Fetus	(4)			–	–	0.12	0.12	0.10	–	–	0.10	–	–
	Infant not nursed	(1)			–	–	0.12	0.04	0.04	–	–	0.09	–	–
	Infant nursed	(1)			–	–	2.47	2.09	2.74	–	–	5.39	–	–
	Fat													
	Infant not nursed	(1)			–	–	5.20	ND	ND	–	–	1.80	–	–
	Infant nursed	(1)			–	–	14.66	2.61	2.05	–	–	4.43	–	–
	Placenta	(1)			–	–	1.01	0.17	0.12	–	–	0.18	–	–
New Zealand														
Smith *et al.* (1992a)	Control group for 2,4,5-T applicators; serum		88	BSIW	1.7 ± 0.3	< 2.0 ± 0.2	7.4 ± 0.8	5.1 ± 0.5	5.6 ± 0.6	< 0.8 ± 0.1	< 1.7 ± 0.2	16.0 ± 2.3	1.9 ± 0.3	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Norway														
Johansen *et al.* (1996)	Blood		93	BSO										
	Controls	(10)			2.8 (0.6–5.0)	1.8 (ND–10.9)	17.1 (4.9–34)	8.7 (1.9–21)	9.7 (2.3–22)	0.9 (ND–1)	4.3 (1.6–6.7)	18 (2.5–53)	1.0 (ND–0.9)	8.6 (1.3–30)
	Moderate crab intake	(15)			5.1 (ND–12.7)	7.2 (1.5–19.5)	54.0 (17.6–112)	55 (10.8–107)	45 (8.8–90)	3.6 (ND–34)	8.9 (ND–33)	44 (0.1–118)	4.8 (ND–4.6)	13.4 (ND–94)
	High crab intake	(9)			7.2 (1.7–16.5)	13.4 (1.3–35)	102 (52–148)	130 (34–233)	103 (27–217)	2.3 (ND–5.4)	14.3 (3.2–29)	93 (27–201)	26 (ND–5.3)	5.4 (2.1–8.8)
Russian Federation														
Schecter *et al.* (1992)	Whole blood		88–89	BSOW										
	Baikalsk	(8)			3.0	< 1.8	15	13	6.8	< 1.6	2.1	4.6	< 1.0	< 8
	St Petersburg (pool)	(60)			2.3	< 1.0	9.2	8.1	3.9	< 1.0	1.2	6.3	< 2.2	–
Spain														
Jiménez *et al.* (1995)	Madrid, unexposed; serum	(11)	93	BSO	4.7 ± 3.8 (9 pos.) (0.8–11.5)	1.4 ± 1.0 (9 pos.) (0.5–3.4)	7.0 ± 2.1 (10 pos.) (2.5–9.6)	5.8 ± 1.1 (10 pos.) (4.8–8.2)	5.1 ± 0.8 (10 pos.) (3.9–6.5)	1.8 ± 1.7 (5 pos.) (0.1–4.9)	2.6 ± 1.2 (9 pos.) (1.2–5.0)	12.8 ± 3.3 (11 pos.) (7.5–18.0)	5.0 ± 4.2 (9 pos.) (0.9–13)	20.6 ± 11.1 (9 pos.) (0.9–38.6)
Gonzáles *et al.* (1997)	Mataro; blood, 10 pools	(198)	95	BSOW	1.2		6.2			10.6			7.1	2.4
Sweden														
Rappe (1984b)	Background; adipose[b]	(6)	82	CS	3		40			22			50	3
Nygren *et al.* (1986)	Adipose[b]		84	BSIW										
	Unexposed	(18)			4.2 (0.3–11)	–	32 (9–54)	5 (1–6)	4 (1–5)	–	2 (1–4)	10 (1–18)	–	–
	Cancer patients	(17)			3.4 (0.3–7.2)	–	45 (9–87)	6 (1–15)	5 (1–13)	–	2 (1–7)	13 (1–49)	–	–
	Non-cancer patients	(14)			4.6 (0–11)	–	33 (11–65)	5 (2–7)	4 (2–7)	–	2 (1–4)	10 (5–16)	–	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Sweden (contd)														
Rappe (1992)	Blood		90	BSIW										
	No fish consumption				1.5	0.15	12	5.4	4.4	–	2.1	10	–	1.0
	Normal fish consumption				1.8	0.5	20	7.1	5.4	–	2.2	14	–	1.0
	High fish consumption				3.0	1.3	79	8.3	11	–	2.8	10	–	1.0
Svensson *et al.* (1995a)	Blood; pool[d]		95	BSI										
	South of Bothnia													
	Fishermen				4.8	ND	198	ND	ND	ND	5.0	16	–	ND
	Controls				ND	ND	40	5.9	6.8	ND	ND	19	–	ND
	Baltic Proper													
	Fishermen				ND	ND	110	9.7	12	ND	4.0	16	–	ND
	Controls				ND	ND	41	7.0	7.0	ND	2.6	15	–	3.2
	Baltic South													
	Fishermen				4.8	ND	163	19	23	ND	7.3	31	–	ND
	Controls				2.6	ND	59	10	10	ND	ND	20	–	6.8
	West Coast													
	Fishermen	(100)			ND	ND	47	8.0	8.0	ND	3.0	14	–	2.9
	Controls	(98)			ND	ND	42	8.9	9.6	ND	4.8	18	–	3.2
Hardell *et al.* (1995)	Blood													
	Cancer patients	(7)	> 86	BSI	3.3 (0.7–7.2)	1.0 (0.3–1.9)	59 (22–200)	8.6 (3.9–17)	7.9 (2.7–15)	2.0 (1–3)	2.7 (0.5–7)	16 (3–49)	0.8 (0.3–1)	< 3
	Non-cancer patients	(12)	> 86		5.0 (2.4–11.4)	< 1.0	35 (11–65)	5.3 (3–7)	3.8 (2–6)	–	2.1 (1–4)	9.9 (5–16)	< 1.0	–
Switzerland														
Wacker *et al.* (1990)	Background													
	Adipose[b]	(21)		CSO	0.8	3.3	48.5	8.3	7.4	–	8.0	1.2	–	0.4
	Liver[b]	(21)			0.2	2.3	7.3	3.7	4.7	–	1.8	1.6	–	0.2
Taiwan														
Ryan *et al.* (1994)	Control children; seum		91	BSW	< 5	–	19			25		34	–	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
United Kingdom														
Duarte-Davidson *et al.* (1993)	Wales, 5 pools; adipose	(5)	90/91	CSO	< 10	13	24 (20–27)	26 (18–42)	15 (9–27)	–	–	34 (24–49)	–	46 (36–62)
United States														
Ryan *et al.* (1985c)	NY State; adipose	(6)	83/84	CSOW	–	–	14.7 ± 2.5 (10.9–17.0)		28.7 ± 7.2 (15.1–52.8)	–	–	16.4 ± 4.0 (12.5 ± 23.8)	–	–
Ryan *et al.* (1986)	NY State		< 83	BSIW										
	Adipose (mean LC, 67%)	(3)			–	–	13.9 (6.5–17)		32.4 (ND–52)	–	–	12.1 (ND–24)	–	–
	Liver (mean LC, 24%)	(3)			–	–	4.1 (ND–6.9)		9.9 (4.2–17)	–	–	3.3 (ND–7.7)	–	–
	Adrenal (LC, 28 and 25%)	(2)			–	–	4.9–5.5		8.5–11	–	–	3.5–4.5	–	–
	Bone marrow (LC, 26%)	(1)			–	–	4.4		9.4	–	–	2.7	–	–
	Muscle (mean LC, 11%)	(3)			–	–	1.1 (ND–2.3)		2.4 (1.7–3.4)	–	–	ND	–	–
	Kidney (LC, 3.0 and 4.0%)	(2)			–	–	ND		ND	–	–	ND–1.7	–	–
Ryan (1986)	NY State, one man, 22 years old		85	BSIW										
	Adipose (LC, 83%)	(1)			–	–	4.2	–	–	–	–	–	–	–
	Liver (LC, 4.4%)	(1)			–	–	2.6	–	–	–	–	–	–	–
Schecter *et al.* (1986a)	Binghamton; adipose[b]	(1)	83–84	CRO	< 2	–	12.5	11.4	5.6	–	–	16.3	ND	< 20
		(1)			4.1	–	10.9	9.3	5.8	–	–	13.7	ND	< 20
		(1)			< 2	–	17.0	13	8.8	–	–	12.5	19.6	1.2
		(1)			< 2	–	16.5	22.9	15.4	–	–	23.8	20.6	1.5

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
United States (contd)														
Schecter *et al.* (1986a) (contd)	Adipose; LC, 70.6% (46%–88%)	(8)	< 85	BNW	–	–	14.3 (3.1–19.7)		31.3 (15.1–46.9)	–	–	16.5 (12.5–23.8)	–	–
Stanley *et al.* (1986)	US general population; adipose	(46)	82	BSO	9.1 ± 9.6 (< 2–32)	–	27 ± 16 (< 1.8–77)		18 ± 8.3 (2.9–55)	–	–	18 ± 12 (< 10–55)	–	60 ± 110 (< 2–360)
Nygren *et al.* (1988)	Era control; serum	(1)	< 88	BSIW	< 18	–	10	3.1	2.2	–	< 4	13.6	–	–
		(1)			< 0.1	–	16.4	17.6	12.6	–	< 3	23	–	–
		(1)			2.6	–	27	26	19	–	4.8	51	–	–
		(1)			10.4	–	51	10.4	3.6	–	< 1.5	12.6	–	–
		(1)			< 0.4	–	5.3	4.4	2.8	–	< 2	10	–	–
		(1)			0.6	–	10.9	8.5	5.4	–	1.4	10.7	–	–
		(1)			< 2.0	–	14.1	10.3	6.5	–	< 2.5	16.4	–	–
Schecter *et al.* (1989b)	One person		< 89	N										
	Fat[b]				–	–	17		52	–	–	15	–	–
	Abdomen	(1)												
	Subcutaneous	(1)			–	–	17		22	–	–	12	–	–
	Adrenal[b]	(1)			–	–	4.9		8.5	–	–	3.5	–	–
	Bone marrow[b]	(1)			–	–	4.4		9.4	–	–	2.7	–	–
	Liver[b]	(1)			–	–	ND		4.2	–	–	2.1	–	–
	Muscle[b]	(1)			–	–	1.1		2.1	–	–	ND		
	Kidney[b]	(1)			–	–	ND		2.1	–	–	ND		
	Lung[b]	(1)			–	–	ND		ND	–	–	ND		
Schecter *et al.* (1990b; 1991a)	Plasma	(20)	< 90		1.3	–	6.1	6.9	5.4	–	1.2	25.1	–	< 3
	Adipose	(20)	< 90	BSOW	1.6	–	6.8	5.6	3.7	–	1.5	16.4	–	< 1
	Whole blood	(4)	88/89		3.0	–	70.5	22.7	23.5	–	8.2	29	–	–
	Adipose	(4)	88/89		3.9	–	70.8	14.5	17.8	–	5.3	23.3	–	4.2
Kang *et al.* (1991)	Adipose		78	N										
	Viet Nam veterans	(36)			2.9	1.7	23.1	21.5	10.7	1.5	3.8	37.4	2.2	3.6
	Non-Viet Nam veterans	(79)			2.4	1.1	22.2	19.3	9.9	0.9	3.2	32.9	1.9	4.5
	Civilians	(80)			3.3	1.9	23.3	23.2	12.0	0.9	3.6	39.1	2.2	3.4

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
United States (contd)														
Piacitelli *et al.* (1992)	Referents; serum	(79)	87/88	BSIW	1.2	–	11 (3.3–28)	11 (4.2–28)	8.5 (3.7–18)	–	1.5	20 (2 pos.) (8.7–46)	0.8	1.1
Patterson *et al.* (1994)	General population; adipose	(4)	84/86	BSIW	1.1 (0.7–1.8)	–	3.7 (3.3–4.3)	3.7 (3.2–4.1)	5.8 4.0–8.3)	–	–	12.0 (8.9–15)	–	–
Schecter *et al.* (1994b)	Placenta; pool	(14)	< 94	BSOW	1.9	< 1.0	3.6	4.0	2.0	1.7	< 1.0	6.3	< 1	< 5
	Placenta	(1)	< 94		0.5	0.5	6.8	8.6	1.8	0.8	< 0.2	4.6	< 0.6	4
	Blood, pool	(50)	< 94		2.3	1.2	8.8	10.6	6.9	2.8	2.8	19.6	3.1	9.3
	Fetal tissue, 8–14 weeks, pool	(10)	94		1.3	< 0.2	1.1	2.2	1.0	–	1.5	3.1	< 0.9	< 3.2
Schecter *et al.* (1996c)	Adipose	(5)	96	BSNW	0.59	0.45	2.6	4.3	2.3	0.12	1.2	6.5	0.3	0.8
	Blood, before nursing	(5)	96	BSNW	0.87	0.52	3.0	5.9	3.4	ND	1.7	8.9	ND	1.9
	Placenta	(5)	96	BSNW	0.61	0.58	4.0	3.3	1.8	ND	0.53	2.7	0.52	2.2
	Cord blood	(5)	96	BSNW	< 1.2	< 0.7	0.87	1.5	1.5	ND	1.4	3.5	ND	< 5
	Mother's milk	(5)	96	BSNW	0.49	0.37	2.8	3.9	2.4	ND	1.4	5.4	0.53	< 2.9
Schecter *et al.* (1996d)	Blood pool	(100)	96	BSIW	< 2.0	< 1.4	11.1	14.1	7.9	3.5	< 3.7	12.0	< 4	< 5
	Serum pool	(100)	96		< 2.0	< 1.9	9.3	14.0	7.9	4.0	< 4.1	13.9	4.9	< 5
Viet Nam														
Schecter *et al.* (1986b)	N. Viet Nam; adipose (LC, 50%)	(7)	84	BSOW	–	–	9.7		9.3	–	–	4.2	–	–
	S. Viet Nam; adipose (LC, 60%)	(13)			–	–	13.0		31.7	–	–	17.0	–	–
Schecter *et al.* (1986c)	N. Viet Nam; adipose (LC, 56%)	(9)	84	BSOW	–	–	14.7 (ND–29.4)			13.3 (ND–20)		7 (ND–10.7)	–	–
	S. Viet Nam; adipose (LC, 62%)	(15)			–	–	21.0 (4.3–45.5)			58.3 (13.6–166.7)		28.9 (4.2–74.9)	–	–
Schecter *et al.* (1989c)	S. Viet Nam; adipose	(27)	84–88	ABN	7 (ND–17)	–	–	–	–	–	–	–	–	–

Table 21 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDF concentration (ng/kg, lipid-based)									
					TCDF	PeCDF		HxCDF				HpCDF		OCDF
					2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Viet Nam (contd)														
Nguyen *et al.* (1989)	Ho Chi Minh City; adipose[b] (mean LC, 76%)	(9)	84/85	BSNW	–		13 (4.3–23)			49 (14–93)			29 (7.8–72)	–
Huteau *et al.* (1990b)	S. Viet Nam; Adipose	(27)	< 90	BSO	1.9 (20 pos.) (0.8–3.9)	4.1 (10 pos.) (1.0–16.8)	25 (21 pos.) (6.5–67.8)	26.2 (22 pos.) (1.4–121)	26.2 (22 pos.) (1.4–121)	8.9 (14 pos.) (2.0–38.9)	9.2 (2 pos.) (8.6–9.8)	47.3 (17 pos.) (9.5–238)	–	–
Schecter *et al.* (1990c)	Adipose		80s	BSO										
	N. Viet Nam	(10)			1.4	0.6	9.1	4.6	4.0	1.7	ND	8.0	0.3	2.2
	S. Viet Nam	(13)			0.8	0.7	7.4	6.4	5.1	0.8	–	13.2	0.2	1.6
Schecter *et al.* (1990d)	Liver, stillborn infants	(1)	< 89	BSOW	0.5	0.8	2.4	5.6	2.6	< 0.2	0.6	3.9	< 0.2	< 0.3
		(1)			1.2	2.3	2.6	5.7	3.8	< 0.4	0.8	6.6	< 0.5	< 0.7
		(1)			0.9	0.6	2.2	2.5	1.5	< 0.3	0.3	2.8	< 0.3	< 0.5
Schecter *et al.* (1992)	Blood		< 91	BSOW										
	N. Viet Nam; pool	(82)			4.6	1.7	7.6	20.6	11.1	0.5	2.2	46.7	1.9	4.2
	S. Viet Nam; pool	(383)			2.4	2.0	9.3	23.9	14.7	0.8	2.6	42.7	3.8	4.4
Schecter *et al.* (1995)	Blood		91/92	BSOW										
	S. Viet Nam; pool	(433)			2.1	1.8	8.3	21.1	13	0.7	2.3	37.6	3.4	3.9
	Centr. Viet Nam; pool	(183)			2.9	2.2	14.9	67.4	40.0	0.9	3.0	75.7	1.9	5.1

Data presented are means and, if available, ± standard deviation, with range in parentheses, unless otherwise indicated. Levels of congeners not detected at a known detection limit (for example, 4.2 ng/kg) are presented as < 4.2 when detection limit is given.

Explanation for analytical methods: All analyses use high-resolution gas chromatography: B, HRMS; C, LRMS; I, isomer-specific; O, others; N, no information; S, sophisticated clean-up; R, reduced clean-up; W, WHO-accepted laboratory; –, not reported; ND, not detected; LC, lipid content; pos., positive; S, south; N, north; Centr., central; [ ] Calculated by the Working Group

Summed TEQ values for PCDDs/PCDFs in these studies are given in Table 25 of the monograph on PCDDs in this volume.

[a] Overlap between these studies

[b] Concentrations on wet weight-basis

[c] Contained also 5.5 and 4.7 ng/kg 1,2,7,8-TCDF, respectively

[d] 150 fishermen and 150 controls between all groups

1.4.1 *Blood and tissue samples*

(*a*) *Austria*

Samples of milk from cows grazing in the vicinity of a metal reclamation plant showed significantly higher PCDD/PCDF levels than control samples. In the blood of two farmers, an increase in levels of certain PCDD and PCDF isomers was found. The highest PCDF value in one sample was 2,3,4,7,8-PeCDF at 839 ng/kg (Riss *et al.*, 1990).

(*b*) *Canada*

Ryan *et al.* (1985b) reported that some samples of adipose tissue from older subjects (> 60 years old) who had died in Ontario hospitals in 1979–81 contained small (mean, 3 ng/kg) amounts of 2,3,7,8-TCDF and larger amounts of 2,3,4,7,8-PeCDF (mean, 17 ng/kg).

(*c*) *China*

Human adipose tissue from seven patients (four men, three women; mean age, 54 years) undergoing general surgery in Shanghai was analysed by Ryan *et al.* (1987). Compared with data from other countries, the values for most congeners were low. [The presence of 1,2,7,8-TCDF suggests that sample contamination (from paper/pulp products) may explain, in part, the relatively high levels of 2,3,7,8-TCDF.]

(*d*) *Finland*

In conjunction with a study of possible effects of PCDDs and PCDFs on pulp and paper mill workers in Finland (Rosenberg *et al.*, 1995) (see also Section 1.3.1(*a*)(vii)), measurements were made in a comparison group with no known exposure (n = 14; mean age, 41 years). The mean I-TEQ level in blood plasma was 49 ng/kg (range, 20–99 ng/kg) (see monograph on PCDDs in this volume, Section 1.4.1). 2,3,4,7,8-PeCDF represented about one-third of the TEQ.

(*e*) *France*

Measurements of PCDDs and PCDFs in adipose tissue from eight persons living in Paris were reported by Huteau *et al.* (1990a). Most of the 2,3,7,8-substituted isomers were found, in some cases at unexpectedly high values (2,3,7,8-TCDF, 1,2,3,7,8-PeCDF and 2,3,4,7,8-PeCDF). Surprisingly, non-2,3,7,8-substituted isomers were also reported at relatively high values (TCDFs, TCDDs, HpCDFs). [Sample contamination cannot be excluded.]

(*f*) *Germany*

Age-related increases in blood levels of 2,3,4,7,8-PeCDF and the HxCDFs as well as I-TEQ have been reported (Schrey *et al.*, 1992; Sagunski *et al.*, 1993; Päpke *et al.*, 1996). No or very little age-dependence was observed for 2,3,7,8-TCDF, 1,2,3,7,8-PeCDF, HpCDF or OCDF.

The Kieselrotstudie (Wittsiepe *et al.*, 1993) was designed to assess the degree of exposure to PCDDs and PCDFs in 56 persons living in the vicinity of a former copper

smelter located in Marsberg (see Section 1.3.2(*c*)). The median I-TEQ values of the Marsberg subjects (43.2 ng/kg) and a reference group from Steinfurt (43.0 ng/kg) were similar, whereas the mean of the Marsberg group (52.7 ng/kg) was higher than that of the control group (44.4 ng/kg). The individuals of the Marsberg group had significantly higher levels of PeCDFs, HxCDFs and HpCDFs on average than the individuals of the reference group.

Near a metal reclamation plant in Rastatt, Baden-Württemberg, PCDD/PCDF contamination of soil, dust from homes, indoor air and vegetables was investigated in 1987. Blood samples from 22 volunteers living in the vicinity of the plant were analysed for PCDDs/PCDFs. Levels of certain Pe-, Hx- and HpCDF isomers were increased, in a similar pattern to the contamination throughout the area. The increase in PCDD/PCDF levels was attributed to occupational exposure in the case of workers and to food intake in the other cases. For children (four samples), soil and/or dust ingestion may be a pathway of special importance (Wuthe *et al.*, 1990).

No correlation was seen between adipose tissue or liver concentrations and age or sex in 28 subjects aged between 26 and 80 years (Thoma *et al.*, 1989, 1990). Large differences in the concentrations of PCDDs and PCDFs between adipose and liver were demonstrated for most of the isomers. Thoma *et al.* (1990) also reported concentrations of PCDDs and PCDFs in adipose tissue from eight infants (age, 2–12 months). The levels were lower than in adults for nearly all isomers (see **Table 22**).

Table 22. Concentrations of PCDF isomers in adipose and liver tissues from German adults and adipose tissues of infants

Compound	Adult; ratio liver : adipose	Adipose tissue; ratio infant : adult
TCDF	2.20	0.75
PeCDF	4.93	0.36
HxCDF	9.38	0.22
HpCDF	15.43	0.23
OCDF	7.43	1.02

From Thoma *et al.* (1989, 1990)

Background data on PCDDs and PCDFs in human blood from Germany published by Päpke *et al.* (1989b) have been updated since 1991 by various authors (see Table 27 of the monograph on PCDDs in this volume). The results suggest a decrease in PCDD/PCDF blood levels in Germany over the past decade.

(*g*) *Japan*

The first reports of PCDDs/PCDFs in human tissue from the general population were presented by Miyata *et al.* (1977) in connection with the *yusho* poisoning in Japan. Levels of PCDFs (isomers not separated) in the range of 17–45 ng/kg were reported in

four of six fat and in one of four liver biopsy/autopsy samples taken from the general Japanese population. At that time, no TCDFs or HxCDFs were detected.

Kashimoto *et al.* (1985) detected PCBs and PCQs in blood of *yusho* and *yucheng* patients. PCDFs were found only in *yucheng* patients. In 60 unexposed individuals, PCDFs were not detected at a detection limit of 10 ng/kg (in whole blood).

Thirteen samples of human adipose tissue from cancer patients were analysed for tetra- to octa-CDDs and -CDFs (Ono *et al.*, 1986). These compounds were identified in all of the analysed samples. Total PCDF concentrations were in the range of 7–120 ng/kg on a wet weight basis, and 2,3,4,7,8-PeCDF levels ranged from 4 to 71 ng/kg.

(*h*) *Taiwan*

In connection with the determination of blood serum levels of PCDFs (and PCBs) in *yucheng* children perinatally exposed to contaminated rice oil, Ryan *et al.* (1994) analysed a matched control population. The total PCDD/PCDF profile for the two pooled control sera from the matched children were very similar. The mean of two measurements was given, with an I-TEQ of 12.6 ng/kg for PCDFs. The characteristic '*yucheng* isomers', 2,3,4,7,8-PeCDF and 1,2,3,4,7,8-HxCDF, showed levels 10–15 times and 15–25 times, respectively, higher in the exposed children than in the matched controls.

(*i*) *United Kingdom*

PCDD/PCDF background data were measured in pooled human adipose tissue samples from five areas in Wales (Duarte-Davidson *et al.*, 1993). With the exception of OCDF, which was found at unexpectedly high values in all pooled samples (36–62 ng/kg), the concentrations were similar to those in other industrialized countries. 2,3,7,8-TCDD and 2,3,7,8-TCDF were not detected at detection limits of 10 ng/kg.

(*j*) *United States*

Six samples from both biopsy and autopsy fat taken in 1983–84 from New York State residents were analysed (Ryan *et al.*, 1985c). PCDDs and PCDFs were found in all samples with total (Cl_4–Cl_8) PCDD levels about an order of magnitude higher than total (Cl_5–Cl_7) PCDFs. Only the penta-, hexa- and hepta-PCDF congeners were detected, at levels that were of the same order of magnitude (15–29 ng/kg). TCDF and OCDF were absent.

The tissue distribution of PCDDs and PCDFs was studied in three autopsy subjects from the general population of New York State (Ryan, 1986; Ryan *et al.*, 1986). These were the first reports to show that several 2,3,7,8-chlorine substituted PCDDs/PCDFs are present not only in adipose tissues from the general population, but also in all other tissues assayed. The ratios of the PCDD/PCDF congeners to each other were similar in each tissue, with overall levels on a wet weight basis decreasing in the order fat, adrenal, bone marrow, liver, muscle, spleen, kidney and lung. If the levels are expressed on a lipid basis rather than on a wet weight basis, liver had the highest value and the variation between tissues showed only a two- to four-fold difference.

Analysis for Cl_4–Cl_8 PCDDs/PCDFs was performed for 46 adipose tissue samples prepared from the United States Environmental Protection Agency National Human

Adipose Tissue Survey (NHATS) as composites from over 900 specimens to represent the nine United States census divisions and three age groups (0–14, 15–44 and ≥ 45 years) (Stanley *et al.*, 1986). The results demonstrate that PCDDs/PCDFs are prevalent in the general United States population and that differences exist with age. Only means and ranges of all data were reported.

A comparison of PCDD/PCDF levels in whole blood, plasma and adipose tissue was performed by Schecter *et al.* (1994b). There were few differences in PCDD/PCDF levels between blood, plasma and adipose tissue and also between whole blood and adipose tissue when reported on lipid basis. Total PCDDs/PCDFs appeared higher in plasma than in adipose tissue, if reported by actual measurement. Comparing whole blood with adipose tissue, values were more similar.

PCDDs and PCDFs in adipose tissue of United States Viet Nam veterans and controls were determined by Kang *et al.* (1991). The samples were collected in 1978. The geometric mean (± SD) 2,3,7,8-TCDF levels in adipose tissue for Viet Nam veterans, non-Viet Nam veterans and civilian controls were 2.9, 2.4 and 3.3 ng/kg, respectively. The mean levels for all isomers for these groups were not significantly different from each other.

In a study by Schecter *et al.* (1994b), levels of PCDDs and PCDFs in placenta, blood and fetal tissue were measured. The highest I-TEQ values (lipid-based) were found in blood, followed by placenta. The fetal tissue contained approximately one third of the I-TEQ of the adult values.

In a further study of partitioning of PCDDs/PCDFs in human maternal tissues, including blood, milk, adipose tissue and placenta, Schecter *et al.* (1996c) collected samples from five American women (mean age, 21.6 years; range, 21–34 years) residing in upstate New York and undergoing caesarean section deliveries between September 1995 and January 1996. Blood, placenta and fat were collected at the time of delivery. The milk and second blood were collected about four to eight weeks later. The lowest concentrations were found in the cord blood, at about one half of the maternal adipose and blood levels. A reduction in PCDD/PCDF levels was observed in the 'second' blood samples after a breast-feeding period of between four and eight weeks.

PCDD/PCDF levels in two pools of whole blood and serum (n = 100) collected in 1996 were compared with older blood data; a decrease was not clearly shown. Mean age of the blood donors was not specified (Schecter *et al.*, 1996d).

(*k*) *Viet Nam*

Adipose tissue samples from south Viet Nam were compared with those from north Viet Nam (where there was no exposure to Agent Orange) (Schecter *et al.*, 1986b). 2,3,7,8-TCDF was not detectable. Most of the other chlorinated PCDFs were found at higher values in samples from south Viet Nam. A further 27 individual and 10 pooled human adipose tissue specimens, collected from persons in south and north Viet Nam, respectively, were analysed for 2,3,7,8-TCDD and 2,3,7,8-TCDF (Schecter *et al.*, 1989c). The mean values were 19 ng/kg 2,3,7,8-TCDD and 7 ng/kg 2,3,7,8-TCDF in the

samples from persons in the south; no 2,3,7,8-TCDD or 2,3,7,8-TCDF was detected in samples from persons in the north.

PCDD/PCDF levels in 27 adipose tissue samples from south Viet Nam were reported by Huteau *et al.* (1990b). Besides the usual 2,3,7,8-substituted isomers, they found non-2,3,7,8-substituted isomers in many samples. [Sample contamination cannot be excluded.]

In connection with analysis of blood samples from various geographical locations for PCDDs/PCDFs, Schecter *et al.* (1992) reported results for pooled samples from north Viet Nam (two analyses with a total of 82 persons) and south Viet Nam (nine analyses totalling 383 persons). The I-TEQ values for samples from north and south Viet Nam were 15 and 36 ng/kg, respectively.

1.4.2 *Human milk*

There have been a large number of studies of PCDD/PCDF concentrations in human milk. Many of the available results are shown in **Table 23** and summarized in **Table 24**. In terms of the I-TEQ concentrations, PCDFs account for between 17 and 78% of the total in human milk. The discussion in Section 1.4.2 of the monograph on PCDDs in this volume is equally applicable to PCDFs.

1.5 Regulations and guidelines

In Germany, an occupational technical exposure limit value of 50 pg I-TEQ/m^3 in air has been established for PCDDs and PCDFs (Deutsche Forschungsgemeinschaft, 1996).

At present, the regulatory requirements for incinerator emissions vary widely among the countries of the European Union. The European Union (1994) published a 'Council Directive on the incineration of hazardous waste' which would require that "the emission of PCDDs and PCDFs shall be minimized by the most progressive techniques" and which defines 0.1 ng/m^3 as a guide value which should not be exceeded by all average values measured over the sample period of 6–16 h.

Germany and the Netherlands have set daily average limit values of 0.1 ng I-TEQ/m^3 of exhaust gases for PCDDs/PCDFs from industrial waste incinerators, Sweden 0.1–0.5 ng TEQ/m^3, and the United Kingdom 1 ng I-TEQ/m^3 with a goal to reduce PCDD/-PCDF emissions from industrial and municipal waste incinerators to 0.1 ng/m^3 (ECETOC, 1992; Liem & van Zorge, 1995).

In Germany, sewage sludge used as a fertilizer for farmland is not allowed to contain more than 100 ng I-TEQ/kg dry matter (Ordinance on Sewage Sludge, 1992; Liem & van Zorge, 1995).

The Canadian Government has proposed a tolerable daily intake (TDI) value of 10 pg I-TEQ/kg bw per day for PCDDs and PCDFs (Government of Canada, 1993).

In Japan, a limit of 0.5 ng I-TEQ/m^3 2,3,7,8-PCDD/PCDF is recommended for municipal waste incinerators (Liem & van Zorge, 1995).

For milk and milk products, a maximal tolerable concentration for PCDDs/PCDFs of 17.5 ng I-TEQ/kg fat has been set in the United Kingdom. In Germany, PCDDs/PCDFs

Table 23. Concentrations of PCDFs in human milk

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Albania													
WHO (1996)	Librazhd; unpolluted area (WHO criteria)	10	92–93	0.3	0.2	3.7	1.4	1.2	< 0.1	0.8	2.7	0.1	0.3
	Tirana; polluted area (WHO criteria)	10	92–93	0.4	0.3	4.7	1.7	1.5	< 0.1	0.8	1.3	0.1	0.1
Austria													
WHO (1996)	Brixlegg; industrial area (WHO criteria)	13	92–93	0.9	0.3	13.5	3.5	2.6	< 0.1	1.3	4.6	0.1	2
Yrjänheikki (1989)	Tulln (WHO criteria)	51	86–88	3.9	1.3	16.9	5.3	4.8	ND	2.3	8.7	–	15.4
WHO (1996)	Tulln; rural area (WHO criteria)	21	92–93	0.6	0.2	8.5	3.4	2.3	< 0.1	1.2	2.6	0.1	2
Yrjänheikki (1989)	Vienna (WHO criteria)	54	86–88	4.4	1	16.2	4.8	3.6	ND	1.8	6.5	–	18.2
WHO (1996)	Vienna; urban area (WHO criteria)	13	92–93	0.7	0.2	9.2	3.3	2.1	< 0.1	1	4.9	0.1	5.9
Belgium													
Yrjänheikki (1989)	Industrial (WHO criteria)	–	86–88	6.2	2.9	32	14	6.5	1.4	6.6	7.3	–	0.3
	Rural (WHO criteria)	–	86–88	3.3	1.4	35	16	7.6	–	7	12	–	–
	Urban (WHO criteria)	–	86–88	4	1.3	32	13	6.1	3.3	–	2.2	–	5
WHO (1996)	Brabant Wallou (WHO criteria)	8	93	0.5	0.3	20.1	5.2	4.7	< 0.1	2.2	3.2	0.1	0.3
	Brussels (WHO criteria)	6	93	0.6	0.3	22	5.4	4.8	< 0.1	2.4	4.1	0.2	1.2
	Liege (WHO criteria)	20	93	0.7	0.3	26.7	5.8	5.3	0.1	2.1	3.8	0.1	0.3
Cambodia													
Schecter *et al.* (1991b)	Phnom Penh	8		0.52	0.32	1.6	0.74	0.79	< 0.5	0.41	2.2	< 0.5	2.4

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Canada													
WHO 1996	All provinces (WHO criteria)	200	81	4.2	< 1	13		17	< 1	4.3	15	< 1	< 2
	All provinces (WHO criteria)	100	92	1.4	< 1	6.2		8.1	< 1	2.3	9.2	< 1	< 2
Yrjänheikki 1989	British Columbia	23	86–88	2.4	< 1	10.3	5.2	4.3	< 1	2.2	7.6	–	< 2
	Maritimes	19	86–88	8	< 1	6.7	3.5	2.2	< 1	< 1	5.6	–	< 2
	Ontario N & E	32	86–88	2.9	< 1	7.4	3	2.7	< 1	1.5	3.8	–	< 2
	Ontario SW	44	86–88	1.8	< 1	9.1	3.6	2.8	< 1	1.5	5	–	< 2
	Prairies	31	86–88	5.7	< 1	5.6	4.8	4.2	< 1	2	6	–	< 2
	Québec	34	86–88	4	1.7	7.1	4.2	3.5	< 1	1.3	6.2	–	< 2
Dewailly *et al.* (1991)	Québec (rural area)	16	86–88	6.1	–	5.2	3.3	2.3	–	1.1	4.5	–	–
Croatia (Yugoslavia)													
Yrjänheikki (1989)	Krk (WHO criteria)	14	86–88	< 3.1	0.9	11.3	2.6	3	–	1.3	2.1	–	–
	Zagreb (WHO criteria)	41	86–88	< 2	< 0.9	9.7	3.2	2.9	–	1.6	1.9	–	–
WHO (1996)	Krk (WHO criteria)	10	93	0.4	0.2	7.9	2.5	2	< 0.1	0.8	1.7	0.1	0.3
	Zagreb (WHO criteria)	13	93	0.9	0.6	13.5	4	3.5	< 0.1	1.7	2.8	0.1	0.3
Czech Republic													
WHO (1996)	Kladno (WHO criteria)	11	93	0.9	0.4	16.3	5.7	3.8	< 0.1	1.1	3.4	0.1	0.2
	Uherske Hradiste (WHO criteria)	11	93	1.1	0.4	25.5	7.3	4.7	< 0.1	1.8	2.9	0.1	0.2

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Denmark													
Yrjänheikki (1989)	WHO criteria	10	86–88	1.2	–	12.8	7	5	–	1.5	8.5	–	–
	Pool	42	86~	1.2	–	12	5.6	4.4	–	1.5	8.8	–	–
WHO (1996)	7 cities (WHO criteria)	48	93	0.5	0.2	11.1	3.5	3	0.1	1.2	6.1	0.1	0.4
Abraham *et al.*	Faeroe Islands	1	94~	< 0.5	< 0.5	7	3	2.4	–	< 0.5	2.4	–	< 2
(1995b)		1	94~	1.6	< 0.5	6.2	5.4	3.5	–	2.2	5.3	–	< 2
		1	94~	1	< 0 .5	6	6	4.5	–	< 0.5	6.2	–	2.8
		1	94~	< 3	< 3	5.3	< 3	< 3	–	< 3	9.4	–	9.2
	pool	9	94~	0.7	< 0.2	4.2	2.5	1.9	–	0.9	1.6	–	< 0.5
Estonia													
Mussalo-Rauhamaa	Tallinn (primipara)	6	91	0.7	0.2	12.8	4.7	2.5	< 0.1	0.4	1.9	< 0.1	0.9
& Lindström (1995)	Tarto (primipara)	6	91	1.3	0.2	23.8	3.6	2.6	< 0.1	0.8	3.9	< 0.1	1.2
Finland													
Yrjänheikki (1989)	Helsinki (WHO criteria)	38	86–88	0.3	0.2	15	2.6	1	< 0.5	2	8.8	–	1.6
WHO (1996)	Helsinki (WHO criteria)	10	93	1.1	0.5	19	4.5	3.5	0.1	1.5	9.9	0.1	1.9
Yrjänheikki (1989)	Kuopio (WHO criteria)	31	86–88	0.3	0.3	14	2.9	1.3	< 0.5	2.3	12	–	1.9
WHO 1996	Kuopio (WHO criteria)	24	93	0.6	0.3	9.6	2.6	2.1	< 0.1	0.9	6.9	0.1	0.3
France													
González *et al.* (1996)	Paris	15	90	1.8	0.5	16.5			20.4			45	19

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Germany													
Beck *et al.* (1992a)	Mother having 1, 2 or 3 children	728	82–92	ND	ND	28.3	ND	ND	ND	ND	ND	ND	ND
	1 child	34	NR	2.1	1	13	5.1	5.7	–	2.7	5.9	–	0.3
	2 children	23	NR	2.9	1	24	8.6	8.9	–	3.6	9.9	–	1.8
	3 children	34	NR	2.9	1	18	7.4	7.2	–	2.9	7.6	–	0.9
Frommberger (1990)	Baden-Württemberg	490	88–89	4.2	0.3	38	7.9	5.9	–	2.9	6.8	–	1
Beck *et al.* (1987)	Berlin	30		2.5	< 1	20	8.7	7.8	–	3	8.5	–	< 3.1
Beck *et al.* (1989c)	Berlin	35		2.8	1	21	8.6	7.9	–	3.2	8.6	–	3
Yrjänheikki (1989)	Berlin (WHO criteria)	40	86–88	1.4	0.7	22	8.7	7.7	–	2.7	13	–	0.9
WHO (1996)	Berlin (WHO criteria)	10	93	< 0.4	< 0.4	11.9	5.5	4.4	< 0.4	1	3.3	< 0.1	< 0.1
Beck *et al.* (1989c)	Flensburg (Baltic coast)	6		1.6	0.6	25	8.7	8.4	–	2.6	9.3	–	0.3
Fürst *et al.* (1992b)	North-Rhine Westphalia	526	84–91	1.7	0.5	26.7	7.8	6.5	–	3.4	5.5	–	1.4
Yrjänheikki (1989)	North-Rhine Westphalia (WHO criteria)	79	86–88	2.3	0.6	30	8.2	6.7	< 0.5	3.8	5.3	–	7.2
Yrjänheikki (1989)	Oldenburg (WHO criteria)	35	86–88	2.4	0.9	23.7	15.2	15	ND	6.2	12.8	–	6.7
Beck *et al.* (1989c)	Recklinghausen; industrial area	10		1.4	0.7	22	7.7	9.2	–	3.1	13	–	4
Yrjänheikki (1989)	Recklinghausen (WHO criteria)	23	86–88	1.4	0.9	26	8.5	8	ND	3	8.4	–	1.3
Beck *et al.* (1989c)	Rheinfelden (rural area/PCP manuf.)	9		5.8	1.3	24	11	9.7	ND	3.9	12	ND	1.5
Beck *et al.* (1989c)	Weiden; rural area	14		3.3	1.1	22	7.4	7.5	–	3.3	8.7	–	0.7

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Hungary													
Yrjänheikki (1989)	Budapest (WHO criteria)	100	86–88	0.5	< 0.5	5.7	< 2	< 2	–	0.5	3.3	–	6.5
WHO (1996)	Budapest (WHO criteria)	20	93	0.3	0.2	5.9	2.6	2.1	< 0.1	0.8	2.8	0.1	0.2
Yrjänheikki (1989)	Szentes (WHO criteria)	50	86–88	0.7	< 0.5	7.6	< 2	< 2	–	0.4	< 2	ND	7.6
WHO (1996)	Szentes (WHO criteria)	10	93	0.4	0.3	5.6	2.5	2	< 0.1	1	2.7	0.1	0.3
Japan													
Schecter *et al.* (1989d)/Yrjanheikki (1989)	Fukuoka	6	86	3	1.3	26	4.5	3	< 1	2	4	–	< 2
Hirakawa *et al.* (1995)	Fukuoka (primipara)	7	94	2.3	0.6	11.4	4.3	4.5	1.9	2	2	0.2	2.7
Hirakawa *et al.* (1995)	Fukuoka (multipara)	8	94	2	0.6	7.8	3.3	3.2	1.6	1.5	2.1	0.7	3
Hashimoto *et al.* (1995b)	Various locations	26	93-94	1.7	1.6	38	6.5	6.8	1.2	4	4.2	5.9	3.6
Jordan													
Alawi *et al.* (1996b)	Amman; pool	4–6	94	< 3.2	< 3.2	< 3.2	<3.2	< 3.2	< 3.2	< 3.2	9.6	< 3.2	< 32
	Amman; pool		94	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	< 31
	Aqaba; pool		94	< 4.5	4.5	10.1	17.9	< 4.5	< 4.5	< 4.5	< 4.5	< 4.5	< 22
	Irbid; pool		94	8.3	11.1	75.9	161	104	7.4	54.6	391	106	189
	Madaba; pool		94	< 11	16.8	84.1	112	96.1	< 11	< 11	< 11	< 11	< 22
	Zarka; pool		94	< 2.6	2.6	4.4	5.2	4.4	< 2.6	<2.6	< 2.6	<2.6	<17
Kazakhstan													
Petreas *et al.* (1996)	WHO criteria	40	96	1.1	0.77	5.3	2.3	1.9	0.75	1.2	2.4	1	3

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Lithuania													
WHO (1996)	Anykshchiai; rural area (WHO criteria)	12	93	0.8	0.4	10.3	3.8	2.7	< 0.4	1.3	3.4	0.2	0.6
	Palanga; coastal area (WHO criteria)	12	93	1.1	0.4	16.4	4.1	3.2	< 0.2	1.6	1.8	0.1	0.2
	Vilnius; urban area (WHO criteria)	12	93	1.3	0.9	9.1	4	3	< 0.5	1.8	3.8	0.5	0.8
Netherlands													
Liem *et al.* (1995)	Primipara	103	93	0.4	0.2	18	5.2	4.4	–	2.4	6	0.1	0.3
Yrjänheikki (1989)	Rural area (WHO criteria)	13	86–88	3.1	0.8	24	7	6.3	ND	2.6	16	–	0.8
	Urban area (WHO criteria)	13	86–88	2.8	0.7	23	7.1	7.1	ND	ND	ND	–	2.4
WHO (1996)	WHO criteria	17	93	0.3	0.3	17.2	5.1	4.4	< 0.5	2.6	6	< 0.5	0.3
Norway													
Clench-Aas *et al.* (1992)	Hamar; rural area (WHO criteria)	10	85–86	4.1	0.8	11.4	4.6	2.7	0.7	1	5.5	–	1.2
WHO (1996)	Hamar; rural area (WHO criteria)	10	93	1.1	0.4	7.5	2	1.9	< 0.5	1.1	4.3	< 0.6	1.5
Clench-Aas *et al.* (1992)	Skien-Porsgrunn; Mg production (WHO criteria)	10	85–86	4.9	1.3	17.7	7.8	5.3	0.7	1.7	5.6	–	2.5
WHO (1996)	Skien-Porsgrunn; industrial area (WHO criteria)	10	93	1.2	0.5	10.9	4.5	3.7	< 0.5	1.4	5.2	< 0.6	1.3

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Norway (contd)													
Clench-Aas *et al.* (1992)	Tromsø; coastal area (WHO criteria)	11	85-86	4.3	0.8	12.9	3.6	2.6	0.7	0.9	6.2	–	1.1
WHO (1996)	Tromsø (WHO criteria)	10	93	1.8	0.3	7.6	1.9	1.7	< 0.3	1.4	18.7	< 0.5	3.3
New Zealand													
Buckland *et al.* (1990a)	Auckland (WHO criteria)	11	90~	0.8	0.35	4.9		5.9	< 0.6	0.71	6.2	< 0.5	< 6
	Christchurch (WHO criteria)	9	90~	0.74	0.23	5.8		7.7	< 0.9	0.84	7.4	< 0.8	< 6
	N. Canterbury (WHO criteria)	8	90~	0.78	0.2	6.6		8.8	< 0.6	0.91	7.8	< 0.7	< 6
	Northland (WHO criteria)	9	90~	1.1	0.22	4.7		8.6	< 0.7	1.1	7.5	< 0.7	< 8
Pakistan													
Schecter *et al.* (1990e)	Pool	7		1.2	< 4.3	6.5		5.8	< 3.9	1.5	4.3	< 3.5	< 6.6
WHO (1996)	Lahore (WHO criteria)	14	93	< 0.02	< 0.01	2.9	1.3	1.1	< 0.1	0.5	3.9	< 0.02	13.8
Poland													
Yrjänheikki (1989)	WHO criteria	5	86–88	1.7	4.3	15.4	18.6	10	–	5.9	35.1	–	–
Russian Federation													
WHO (1996)	Arkhangelsk	1	93	1.5	0.5	12.9	3.2	2.3	0.1	1	1.9	0.1	0.2
Schecter *et al.* (1990f)	Baikalsk; pool	5	88-89	2.7	1.3	9.6	8.2	3.2	< 0.5	0.6	1.4	< 0.5	0.4
	Irkutsk; pool	4	88-89	6.3	2.3	19	15	5	< 0.5	1.8	2.6	< 0.5	2
	Kachug; pool	4	88-89	2.8	1	7.4	5.7	2.2	< 0.5	0.7	0.6	< 0.5	0.5
WHO (1996)	Karhopol	1	93	0.7	0.2	5	1.4	0.9	< 0.1	0.3	0.8	0.1	0.1
Schecter *et al.* (1990f)	Moscow	1	88-89	1.9	0.4	11	4	2.5	< 0.5	1.1	1.5	< 0.5	0.8
	Novosibirsk; pool	10	88-89	1.7	0.8	8.4	5.4	2.4	< 0.5	0.8	0.7	< 0.5	1.5

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Slovakia													
WHO (1996)	Michalovce (WHO criteria)	10	93	1.1	0.4	21	5.8	3.5	< 0.1	1.1	5.5	0.1	0.2
	Nitra (WHO criteria)	10	93	0.8	0.5	14.5	5.4	4	0.1	1.4	2.7	0.1	0.3
South Africa													
Schecter *et al.* (1990e)	Pool												
	Black	6		0.8	0.3	2	2.4	1.8	0.6	0.6	5.2	0.6	6.1
	White	18		1.5	0.4	5.5	3.4	3.1	ND	1.3	4.7	0.4	2.8
Spain													
WHO (1996)	Bizkaia (WHO criteria)	19	93	0.9	0.4	16.9	5	4	0.1	1.5	3	0.2	0.5
	Gipuzkoa (WHO criteria)	10	93	0.7	0.4	20.9	6	4.7	0.1	2.2	3.1	0.1	0.2
González *et al.* (1996)	Madrid	13	90	1	0.7	0.9			30			7.2	18
Sweden													
Yrjänheikki (1989); Clench-Aas *et al.* (1992)	Borlänge; rural area	10	85–86	3.6	0.8	17	7	3.7	< 1.5	1.3	5.7	–	< 2.5
	Gothenburg; city (WHO criteria)	10	85–86	4.1	–	17.4	5.2	3.7	< 1.5	2.6	11.4	–	< 2.5
	Sundsvall; industrial (WHO criteria)	10	85–86	3.8	–	19.6	4	3.3	< 1.5	2	6.7	–	< 2.5
	Uppsala (MSWI) (WHO criteria)	10	85–86	3.7	–	17.1	5.3	4.4	< 1.5	2.4	12.1	–	< 2.5
Thailand													
Schecter *et al.* (1991b)	Bangkok	10		1.8	0.7	2.6	1.2	0.9	< 0.5	0.6	0.9	< 0.5	0.6

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
United Kingdom													
Wearne *et al.* (1996)	Cambridge (WHO criteria)	20	93–94	0.82	0.47	16	4.4	4	0.09	2.5	4	0.19	0.57
Startin *et al.* (1989)	Glasgow (WHO criteria)	50	87~	0.9	0.3	19	7.2	5	ND	2.3	7.1	–	6.9
Wearne *et al.* (1996)	Glasgow (WHO criteria)	20	93–94	0.78	0.3	15	4.2	3.6	< 0.1	2.2	4	0.15	0.81
Startin *et al.* (1989)	Sutton Coldfield (WHO criteria)	50	87~	1.4	0.5	25	8.3	7.8	ND	3.6	9.5	–	6.8
Wearne *et al.* (1996)	Birmingham (WHO criteria)	20	93–94	1	0.29	14	4.2	3.6	< 0.1	2	2.9	0.13	0.72
Ukraine													
WHO (1996)	Kiev (WHO criteria)												
	Area 1	5	93	0.8	0.6	9.5	7.1	4.4	0.2	1.3	5.7	0.8	1.9
	Area 2	5	93	0.8	0.5	9.9	7.1	4.6	0.2	1.3	4.6	0.7	1
United States													
Schecter *et al.* (1989d; 1994b; 1996a,b)	United States	43	88	2.85	0.45	7.3	5.6	3.2	< 0.75	1.9	4.1	< 1	4.1
Schecter *et al.* (1990e)	Tennessee; pool	9	–	1	< 1.5	4.1		7.8	< 1.4	1.2	8.1	< 2.7	< 5.8

Table 23 (contd)

Reference	Origin	No.	Coll. period	Mean PCDF concentration (ng/kg fat)									
				TCDF	PeCDF		HxCDF				HpCDF		OCDF
				2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Viet Nam													
Schecter *et al.* (1990e)	Binh Long; pool	4		1	1.3	7.1	8.8	6.7	< 1.3	2	13.2	< 3.5	< 7
Schecter *et al.* (1991b); Schecter (1994)	Da Nang	11	85–90	2.2	4.1	17	34	18	< 0.5	10	40	< 0.5	7.4
	Dong Nai	11	85–90	1.6	1	13	19	11	< 0.5	2.1	6.2	< 0.5	0.9
	Hanoi	30	85–90	2	1	6.1	4.2	3.1	< 0.5	1.4	3.4	< 0.5	2.1
	Ho Chi Minh	38	85–90	2.8	1.4	8.1	5.7	3.6	< 0.5	1.6	8	ND	2.6
	Song Be	12	85–90	2	2	8.7	12	7.8	< 0.5	2.7	10	ND	1.8
Schecter *et al.* (1990e)	Tay Ninh; pool	4		1.1	2	10.9		16.3	< 2.4	3.1	14.9	< 5.9	< 14
	Vung Tau	5		2	1.4	9.3	9.5	5.4	0.7	1.6	11.8	< 4.9	< 7

ND, not detected and detection limit not reported; –, not reported
WHO criteria are described in Section 1.4.2 of the monograph on PCDDs in this volume.
Summed TEQ values for PCDDs/PCDFs in these studies are given in Table 29 of the monograph on PCDDs in this volume.

Table 24. Summary of concentrations (ng/kg fat) of PCDFs in human milk (as reported in Table 23)

	TCDF	PeCDF		HxCDF				HpCDF		OCDF
	2378	12378	23478	123478	123678	123789	234678	1234678	1234789	
Mean	2.0	1.1	15	8.4	6.6	1.1	2.6	9.9	3.2	5.7
Minimum	0.3	0.2	0.9	0.74	0.79	0.09	0.3	0.6	0.1	0.1
5th percentile	0.4	0.2	4.2	1.6	1.3	0.1	0.58	1.5	0.1	0.2
25th percentile	0.8	0.3	7.3	3.5	2.7	0.1	1.1	3.3	0.1	0.4
Median	1.4	0.6	12	5.2	4.1	0.5	1.6	5.6	0.1	1.5
75th percentile	2.8	1	19	7.3	6.6	1.4	2.4	8.5	0.4	3.4
95th percentile	5.7	3.9	31	17	16	3.3	6.3	15	5.9	17
Maximum	8.3	17	84	161	104	7.4	55	391	106	189

must not exceed 5 ng I-TEQ/kg milk fat and, in the Netherlands, they must not exceed 6 ng I-TEQ/kg milk and milk product fat (Liem & van Zorge, 1995).