POLYCHLORINATED DIBENZO-*para*-DIOXINS

These substances were considered by previous working groups, in February 1977 (IARC, 1977a) and March 1987 (IARC, 1987a). Since that time, new data have become available and these have been incorporated in the monograph and taken into consideration in the evaluation.

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 dibenzo-*para*-dioxin and selected polychlorinated dibenzo-*para*-dioxins (PCDDs) are presented in **Table 1**. The tetra-, penta, hexa- and hepta-chlorinated compounds are referred to here as TCDDs, PeCDDs, HxCDDs and HpCDDs or collectively as, for example, Cl_4–Cl_7 CDDs or hepta/octa-CDDs.

1.1.2 *Structural formulae*

The general structure of the PCDDs is shown in **Table 2**. Any or all of the eight hydrogen atoms of dibenzo-*para*-dioxin can be replaced with chlorine, giving rise to 75 possible chlorinated dibenzo-*para*-dioxin structures. All of the 75 are referred to as congeners (members of a like group) of one another and congeners having the same number of chlorines are isomers. The term 'dioxins' has been widely used to refer to the PCDDs and often the polychlorinated dibenzofurans (PCDFs) as well (Liem & van Zorge, 1995), although it is technically incorrect (Clement, 1991) and is not so used in this monograph.

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 PCDDs 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 PCDDs are presented in **Table 3**.

Table 1. Nomenclature, molecular formulae, and molecular weights of dibenzodioxin and chlorinated derivatives

CAS Reg. No. (Deleted CAS Nos)	CAS name and synonyms[a]	Molecular formula	Molecular weight
262-23-4	**Dibenzo[*b*,*e*][1,4]dioxin**; Dibenzodioxin; dibenzo-[1,4]dioxin; dibenzo-*para*-dioxin; diphenylene dioxide; oxanthrene; phenodioxin; DD	$C_{12}H_8O_2$	184.2
33857-26-0	**2,7-Dichlorodibenzo[*b*,*e*][1,4]dioxin**; 2,7-dichloro-dibenzo-*para*-dioxin; 2,7-DCDD; 2,7-dichlorodibenzo-dioxins; 2,7-diCDD	$C_{12}H_6Cl_2O_2$	253.0
1746-01-6 (56795-67-6)	**2,3,7,8-Tetrachlorodibenzo[*b*,*e*][1,4]dioxin**; D48; dioxin; TCDBD; TCDD; 2,3,7,8-TCDD; 2,3,7,8-tetra-chlorodibenzo-1,4-dioxin; 2,3,7,8-tetrachlorodibenzo-*para*-dioxin; 2,3,7,8-tetraCDD	$C_{12}H_4Cl_4O_2$	321.98
40321-76-4	**1,2,3,7,8-Pentachlorodibenzo[*b*,*e*][1,4]dioxin**; D54; 1,2,3,7,8-PeCDD; 1,2,3,7,8-PnCDD; 1,2,3,7,8-penta-chlorodibenzo-*para*-dioxin; 1,2,3,7,8-pentachloro-dibenzodioxin; 2,3,4,7,8-pentachlorodibenzo-*para*-dioxin; 2,3,4,7,8-pentachlorodibenzodioxin; 1,2,3,7,8-pentaCDD	$C_{12}H_3Cl_5O_2$	356.42
39227-28-6	**1,2,3,4,7,8-Hexachlorodibenzo[*b*,*e*][1,4]dioxin**; D66; 1,2,3,4,7,8-hexachlorodibenzodioxin; 1,2,3,4,7,8-hexa-chlorodibenzo-*para*-dioxin; 1,2,3,4,7,8-hexachloro-dibenzo[1,4]dioxin; 1,2,3,4,7,8-HxCDD; 1,2,3,4,7,8-hexaCDD	$C_{12}H_2Cl_6O_2$	390.87
57653-85-7	**1,2,3,6,7,8-Hexachlorodibenzo[*b*,*e*][1,4]dioxin**; D67; 1,2,3,6,7,8-hexachlorodibenzodioxin; 1,2,3,6,7,8-hexa-chlorodibenzo-*para*-dioxin; 1,2,3,6,7,8-hexachloro-dibenzo[1,4]dioxin; 1,2,3,6,7,8-HxCDD; 1,2,3,6,7,8-hexaCDD	$C_{12}H_2Cl_6O_2$	390.87
19408-74-3	**1,2,3,7,8,9-Hexachlorodibenzo[*b*,*e*][1,4]dioxin**; D70; 1,2,3,7,8,9-hexachlorodibenzodioxin; 1,2,3,7,8,9-hexa-chlorodibenzo-*para*-dioxin; 1,2,3,7,8,9-hexachloro-dibenzo[1,4]dioxin; 1,2,3,7,8,9-HxCDD; 1,2,3,7,8,9-hexaCDD	$C_{12}H_2Cl_6O_2$	390.87
35822-46-9	**1,2,3,4,6,7,8-Heptachlorodibenzo[*b*,*e*][1,4]dioxin**; D73; 1,2,3,4,6,7,8-heptachlorodibenzodioxin; heptachloro-dibenzo-*para*-dioxin; 1,2,3,4,6,7,8-heptachlorodibenzo-*para*-dioxin; 1,2,3,4,6,7,8-heptachlorodibenzo[1,4]dioxin; 1,2,3,4,6,7,8-HpCDD; 1,2,3,4,6,7,8-heptaCDD	$C_{12}HCl_7O_2$	425.31
3268-87-9	**Octachlorodibenzo[*b*,*e*][1,4]dioxin**; D75; OCDD; octa-chlorodibenzo-*para*-dioxin; 1,2,3,4,6,7,8,9-octachloro-dibenzo-*para*-dioxin; 1,2,3,4,6,7,8,9-octachlorodibenzo-[1,4]dioxin; octaCDD	$C_{12}Cl_8O_2$	460.76

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

Table 2. Dibenzo-*para*-dioxin structural formula and numbers of chlorinated isomers

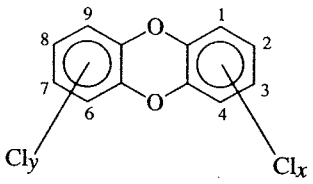
Formula	
	
No. of chlorines ($x + y$)	No. of isomers
1	2
2	10
3	14
4	22
5	14
6	10
7	2
8	1
Total	75

Table 3. Chemical and physical properties of dibenzo-*para*-dioxin and selected chlorinated derivatives[a]

Chemical	Melting point (°C)	Water solubility (mg/L) at 25 °C	Vapour pressure (Pa) at 25 °C	Henry's Law constant (Pa × m^3/mol)	log K_{ow}
Dibenzo-*para*-dioxin	122–123	0.87	5.5×10^{-2}	11.70	4.30
2,7-DCDD	209–210	3.75×10^{-3}	1.2×10^{-4}	8.10	5.75
2,3,7,8-TCDD	305–306	1.93×10^{-5}	2.0×10^{-7}	3.34	6.80
1,2,3,7,8-PeCDD	240–241		5.8×10^{-8}		6.64
1,2,3,4,7,8-HxCDD	273–275	4.42×10^{-6}	5.1×10^{-9}	1.08	7.80
1,2,3,6,7,8-HxCDD	285–286		4.8×10^{-9}		
1,2,3,7,8,9-HxCDD	243–244		6.5×10^{-9}		
1,2,3,4,6,7,8-HpCDD	264–265	2.40×10^{-6}	7.5×10^{-10}	1.27	8.00
OCDD	325–326	0.74×10^{-7}	1.1×10^{-10}	0.68	8.20

[a] From Rordorf (1987); Sijm *et al.* (1989); Mackay *et al.* (1991)

Limited research has been carried out to determine physical and chemical properties of PCDDs. The tetra- through octa-chloro congeners with 2,3,7,8-chlorination (sometimes referred to as laterally substituted PCDDs) have received the most attention, with 2,3,7,8-TCDD being the most intensively studied compound. Of the large number of possible congeners, only the 2,3,7,8-chlorinated compounds and a few others are available commercially, and synthesis and separation can be both time-consuming and difficult. Some of the PCDD congeners have not yet been prepared in pure form. The PCDDs are intentionally prepared only for research purposes.

The concept of toxic equivalency factors (TEFs) was developed by several agencies and national and international organizations (Ahlborg *et al.*, 1988; Safe, 1990; Ahlborg *et al.*, 1992a) to aid 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 2,3,7,8-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 4.3.1) and other in-vitro responses as well as in-vivo effects ranging from enzyme induction to tumour formation (Ahlborg *et al.*, 1992a).

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

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

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 PCDDs[a]

Congener	I-TEF
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	0.5
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.001
All other PCDDs	0

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

1.1.4 *Methods of analysis*

The analysis of environmental and biological samples for PCDDs and PCDFs has presented a major challenge for analytical chemists and has catalysed the development of new and improved methods and equipment with applications to many other problems in environmental chemistry. The challenge in PCDD/PCDF analysis arises from several factors. First, these compounds are usually present at very low levels in environmental samples, requiring methods with detection limits several orders of magnitude lower than those for other environmental contaminants. In addition, the toxicities of the various PCDD/PCDF congeners differ dramatically, so that for a proper risk assessment, individual PCDD and PCDF isomers must be selectively determined, often in the presence of other isomers and congeners. Interference between congeners and with other chlorinated compounds in the matrix can cause serious analytical difficulties. The widespread occurrence of these compounds in the environment has required the development of methods for many different media and sample types.

Progress in the analytical chemistry of the PCDDs and PCDFs over the past 30 years has been remarkable (Buser, 1991; Clement, 1991; de Jong & Liem, 1993). In the 1960s and early 1970s, the principal method of analysis was packed column gas chromatography (GC) with electron capture detection (ECD), and detection limits were in the parts-per-million (ppm; μg/g or mg/kg) to parts-per-billion (ppb; ng/g or μg/kg) range. Most work during this period focused on the determination of levels of 2,3,7,8-TCDD as a contaminant in herbicides and other industrial chemicals, and PCDDs and PCDFs were frequently not detected in environmental samples due to the lack of sensitivity and specificity of the methods.

Beginning in the 1970s, mass spectrometry (MS) was used, first by direct injection of samples and extracts, to reduce interferences and confirm the presence of 2,3,7,8-TCDD (Baughman & Meselson, 1973a,b). Soon, the advantages of MS as a detection method with GC were recognized, and GC-MS became the analytical method of choice for PCDDs and PCDFs in environmental and biological samples (Hass & Friesen, 1979; Rappe, 1984a; Crummett *et al.*, 1986). Advances in sample clean-up procedures, gas chromatographic separations with fused silica capillary columns and GC-MS systems in the 1970s and 1980s and the availability of appropriate calibration standards have made it possible now to identify and quantify individually all of the 2,3,7,8-substituted PCDDs and PCDFs at ng/kg concentrations or lower in most matrices (Clement & Tosine, 1988; Buser, 1991; Clement, 1991). However, these methods for ultra-trace analysis remain complex and difficult, and considerable variability still exists among laboratories in their capabilities and skill in performing these analyses (Stephens *et al.*, 1992).

Guidelines and methods of analysis have been proposed or established by a number of international and national governmental organizations and agencies. Examples include IARC Scientific Publications No. 108 (Rappe *et al.*, 1991a), recommendations from a workshop convened by the Community Bureau of Reference of the Commission of the European Union (Maier *et al.*, 1994) and United States Environmental Protection Agency Methods (United States Environmental Protection Agency, 1986, 1995, 1996a).

(a) General considerations

The recognition by toxicologists of the extreme toxicity and biological activity of some PCDD/PCDF congeners has generated the requirement for highly sensitive and specific analytical methods. Method development has been directed primarily at the quantitative determination of the seven PCDDs and 10 PCDFs with chlorine at the 2, 3, 7 and 8 positions on the aromatic rings (see **Table 2** in this monograph as well as Table 2 in PCDF monograph). The remaining 68 PCDDs and 125 PCDFs can seriously interfere with the determination of the 2,3,7,8-substituted congeners. In addition, the pattern of congeners in an environmental sample can provide clues as to the source of the PCDDs/-PCDFs. The larger number of isomers with four or five chlorines (see **Table 1**) makes isomer-specific analysis more difficult in the tetra- and penta-substituted series (Rappe, 1984a; Buser, 1991; Clement, 1991).

Ultra-trace PCDD/PCDF analyses can require sample enrichment by a few thousand-fold to a million-fold or more before GC-MS determination. Co-extracted, interfering compounds may be present in the sample at much higher levels than the PCDDs and PCDFs, necessitating sophisticated matrix-specific clean-up techniques as well as highly selective separation and detection methods (Clement & Tosine, 1988; Buser, 1991; de Jong & Liem, 1993).

Two different strategies have been applied in PCDD/PCDF analyses. In the first approach, the objective is to recover all PCDDs and PCDFs in a single fraction by a containment-enrichment procedure, and then to analyse this fraction for all congeners by high-resolution GC-MS. In this approach, congener distribution patterns can be obtained that may help to identify sources of the PCDDs/PCDFs. The second approach focuses on separation of isomers during sample preparation and purification (e.g., by including reversed-phase and normal-phase high-performance liquid chromatography (HPLC) steps in the clean-up procedures) and results in multiple fractions that may be analysed for one or more congeners. This approach has often been applied to the determination of a specific congener, such as 2,3,7,8-TCDD (Rappe, 1984a; Buser, 1991).

Analyte standards for calibration are essential in ultra-trace analysis, and by the mid-1980s an adequate set of labelled and unlabelled PCDDs and PCDFs had become commercially available. Although not all of the 210 PCDD and PCDF congeners are available, all of the 2,3,7,8-substituted congeners can be purchased in crystalline form or in solution. These congeners are also available fully ^{13}C-labelled, and some are available fully labelled with ^{37}Cl. Analysis of environmental and biological samples requires the use of these standards for method development, to monitor recoveries and for isotope-dilution or other GC-MS analyses. It would be desirable to have available natural-matrix certified reference materials to verify spiked-sample recoveries based on calibration standards, but few are currently available (Rappe, 1984a; Clement & Tosine, 1988; Alvarez, 1991; Clement, 1991; Schimmel *et al.*, 1994; Maier *et al.*, 1995).

PCDD and PCDF determinations are frequently required for incinerator emissions (flue gases, fly ash, bottom ash and aqueous effluents), soils, sediments and sludges, air (vapour and particulates), water, biological samples of all types and chemical products. At present, typical detection limits of methods used for biological samples are in the

1 ng/kg range; lower detection limits are possible for some media, such as ambient air (in the low femtogram [10^{-15} g]/m^3 range) and drinking-water (as low as 0.01 pg/L), and higher detection limits (µg/kg range) are often adequate for chemical products (Buser, 1991; de Jong & Liem, 1993).

The analysis of PCDDs and PCDFs in environmental and biological samples can be considered to proceed in five stages, each of which must be carefully controlled and optimized to ensure reliable data: (1) sampling, (2) extraction, (3) clean-up, (4) separation and (5) quantification. Each of these stages is discussed briefly in the following sections. Reviews and guidelines have been published for various environmental and biological matrices (Buser, 1991; Maier *et al.*, 1994). The following comments are based on these reviews and the other references cited. In the subsequent text and tables, methods are indicated by the system presented in **Table 5**. In the text, this information is given as '(analytical method...)', using these abbreviations.

Table 5. Abbreviations for descriptions of analytical methods[a]

A	HRGC (high-resolution gas chromatography)
a	LRGC (low-resolution gas chromatography)
B	HRMS (high-resolution mass spectrometry)
C	LRMS (low-resolution mass spectrometry)
I	Isomer-specific polar column, e.g., SP 2330/31
O	Other than isomer-specific nonpolar column, e.g., DB-5
N	No information
S	Sophisticated clean-up, e.g., multicolumn, use of all ^{13}C-labelled 2,3,7,8-substituted standards
R	Reduced clean-up
W	WHO-accepted laboratory. The laboratory has fulfilled the requirements for interlaboratory studies of determination of PCDDs/PCDFs in biological material, organized by WHO (Stephens *et al.*, 1992; WHO, 1996).

[a]Descriptions usually have four elements: gas-chromatographic resolution (A or a), mass-spectrometric resolution (B or C), isomer-specificity of GC column (I or O) and clean-up (S or R). Any one or more of these may have no information (N).

(*b*) *Sampling*

The sampling protocols for PCDD and PCDF analyses depend on the type of sample, the level of PCDDs/PCDFs and potential interferences in the sample, the detection limit of the method, the requirements of the analysis, and the specific situations encountered. As with most ultra-trace analyses, sample size, homogeneity, storage and handling and the avoidance of contamination are important considerations.

Samples should be protected from light and heat. Although PCDDs and PCDFs are generally quite stable, they are prone to photolysis, especially in solution. The less chlo-

rinated congeners tend to photolyse more rapidly than those with more chlorine substituents (ECETOC, 1992).

Contamination of samples is a serious problem. Potential sources of contamination include sampling equipment and containers, solvents and reagents, adsorbents, glassware, other samples and even laboratory tissue wipes, floor-cleaning solutions and cigarette smoke (Albro, 1979; Patterson *et al.*, 1990).

Samples to characterize incinerator emissions may include grab samples of fly ash (from electrostatic precipitators) and bottom ash (or slag), aqueous effluents from gas-scrubbing equipment and flue gases and particulates. Stack sampling is carried out isokinetically, collecting particulates on filters (e.g., glass fibres) and volatiles by cooling and trapping in impingers or on adsorbent resins. Collection efficiencies are checked by introducing isotope-labelled PCDDs and PCDFs into the sampling train (Ozvacic, 1986; ECETOC, 1992; United States Environmental Protection Agency, 1995). For measurement of flue gases of municipal waste incinerators operating with emissions below 0.1 ng I-TEQ/m^3 (European Union Directive; see Section 1.5), certain modifications in the sampling and analytical procedures are necessary, as described by Ball and Düwel (1996) and Bröker (1996).

PCDD and PCDF levels in air (particles and gases) are normally determined by collecting high-volume samples (up to 1000 m^3 over a 24-h period) on glass fibre/polyurethane foam filters (Tondeur *et al.*, 1991). For samples of, say, 1500 m^3 collected over several days, the filters should be spiked before sampling with ^{13}C-labelled standards to demonstrate possible sampling losses (Tysklind *et al.*, 1993).

PCDDs and PCDFs have very low solubilities in water, with solubility decreasing with increasing number of chlorine substituents. However, they tend to adsorb strongly onto fine particles suspended in water, so water samples must include suspended particulates and cannot be subsampled after collection unless they are first micro-filtered and the water and fine particulates are analysed separately. Small to medium sample volumes (1–5 L) can be extracted directly, but larger volumes needed for ultra-trace analysis are preconcentrated on sorbent resins (e.g., XAD-2) or a polyurethane filter (Ryan, 1991; Luksemburg, 1991).

Soils and sediments are sampled with core samplers at the surface (top 5–10 cm) and sometimes at lower depths. Often, several core samples are pooled for composite analysis (500–1000 g), based on a sampling grid for investigation of soil contamination in an area. Samples are air-dried, sieved (to remove debris), mixed and homogenized before extraction (Kleopfer *et al.*, 1985; Solch *et al.*, 1985; de Jong *et al.*, 1993; Fortunati *et al.*, 1994).

Biological samples should be deep frozen (–20 °C or lower) until analysed to prevent enzymatic or microbiological alterations. Choice of sampling procedures depends on the tissue and species; for example, usually only small samples of human fat are available, whereas larger samples of ecological species (e.g., fish) or foods normally can be obtained. Tissue samples may be ground with anhydrous sodium sulfate or silica gel and homogenized before extraction (Stanley *et al.*, 1985; Norstrom & Simon, 1991; Patterson *et al.*, 1991; Olsson, 1994).

In discussing problems associated with the variation in biological samples, Bignert *et al.* (1994) noted that large numbers of samples are generally required to define spatial or temporal trends or differences between various ecological matrices with respect to the concentrations of PCDDs and PCDFs.

(*c*) *Extraction*

^{13}C-Labelled internal standards are added before extraction and clean-up to determine recoveries and to allow correction for losses during work-up. Homogenized samples are sometimes digested to destroy the sample matrix and free any trapped PCDDs/PCDFs before extraction (e.g., hydrochloric acid treatment of fly ash and some soil/sediment samples). However, alkaline saponification at elevated temperatures (which has sometimes been used with fatty samples) is not recommended, as it can cause decomposition of PCDDs and PCDFs (Albro, 1979; Ryan *et al.*, 1989a). For chlorophenols and chlorophenoxyacetic acid herbicides, initial separation of the (neutral) PCDDs and PCDFs is often accomplished by partitioning with alkali (Buser, 1991; United States Environmental Protection Agency, 1996a).

Extraction procedures and solvents vary widely, depending on the type of sample and method of clean-up and analysis. Extraction of PCDDs/PCDFs into solvent may be accomplished by simple dissolution, shaking, blending, ultrasonic treatment or Soxhlet extraction. For example, fats and oils may be dissolved directly in dichloromethane, and water samples may be extracted with dichloromethane, toluene or hexane. Particulates filtered from water or resin-sorbed water samples may require Soxhlet extraction with toluene or benzene. Incinerator flue gases (adsorbed on polyurethane foam) and fly or bottom ash are usually Soxhlet-extracted with toluene or benzene. For soils, sediments and sludges, sequential Soxhlet extractions with different and/or mixed solvents (acetone/hexane, toluene, benzene, dichloromethane) are sometimes required.

Many different extraction procedures and solvents have been used with biological samples, such as fish and animal tissues, human tissues and vegetation. Multi-laboratory comparison studies with fish tissues and porcine fat, human blood and adipose tissue and human and cow's milk suggest that acceptable recoveries can be obtained with a variety of extraction methods (Clement & Tosine, 1988; Patterson *et al.*, 1990; Stephens *et al.*, 1992; Schimmel *et al.*, 1994).

Supercritical fluid extraction (SFE) using carbon dioxide is a new technique, which has been used for extraction of PCDDs and PCDFs. Alexandrou *et al.* (1992) have used it for extraction of solid samples such as fly ash and paper pulp, and also for aqueous matrices such as pulp mill effluents. More recently van Bavel *et al.* (1996) have demonstrated the use of SFE in the extraction of biological samples, including human adipose tissue.

(*d*) *Clean-up*

The objective of the clean-up procedures is to purify and prepare the extract for final separation and quantification. Such procedures remove co-extracted compounds that may interfere in the GC-MS analysis. The extent of clean-up required is determined by the

analytical objectives (number of congeners to be quantified), the matrix and the sophistication of the GC-MS system.

Clean-up is normally accomplished by column chromatography through a series of columns, sometimes followed by HPLC. Pretreatment to remove large quantities of co-extractives may be necessary for some samples. Pretreatment may include acid or base washing; elution through multilayer columns containing silica gel impregnated with sulfuric acid, sodium hydroxide and silver nitrate (de Jong *et al.*, 1993); or gel permeation chromatography to remove lipids and other compounds of high molecular weight (Norstrom & Simon, 1991).

Column chromatography of extracts on alumina removes chlorinated benzenes, polychlorinated biphenyls (PCBs) and terphenyls, and higher chlorinated diphenyl ethers in a first fraction (2% dichloromethane in *n*-hexane); PCDDs and PCDFs are then recovered from the column with 50% dichloromethane in *n*-hexane. This treatment also removes the polychlorinated 2-phenoxyphenols (predioxins) that can undergo thermal ring closure in the gas chromatograph to form PCDDs (Buser, 1991).

Since the mid-1980s, a two-column procedure (Smith *et al.*, 1984a) has come into extensive use. Extracts are first chromatographed on activated carbon with dichloromethane to separate planar compounds (including PCDDs and PCDFs, which are retained) from non-planar compounds. The planar compounds are removed from the carbon column by reverse elution with toluene and then chromatographed on alumina to separate the PCDDs and PCDFs from other planar contaminants (e.g., non-*ortho*-substituted PCBs, polychloronaphthalenes). The method has been adapted and validated for complex biological samples in a semi-automated format (with additional clean-up steps) (Patterson *et al.*, 1990; Turner *et al.*, 1991).

HPLC also has found extensive application in PCDD/PCDF analysis in recent years (Clement & Tosine, 1988). It has been used principally for isomer-specific quantification of 2,3,7,8-TCDD and/or the other toxic PCDD/PCDF congeners. It is also used to supplement other clean-up methods for difficult samples.

Bergqvist *et al.* (1993) have described the use of a polyethylene semipermeable membrane in a nondestructive method for the reduction of lipid in analyses of PCDDs and PCDFs in environmental biological samples. This allows the analysis of samples with total size of 500 g.

Many other clean-up methods have been reported for environmental and biological matrices. The extent of clean-up required for a given sample depends on a number of factors, as noted above. Even with elaborate clean-up procedures, the final fractions may still contain chlorinated contaminants that need to be considered in the separation and quantification steps.

(*e*) *Separation*

The final separation of PCDDs and PCDFs from residual contaminants and into the individual congeners is almost invariably performed by high-resolution GC (HRGC). In the 1960s and 1970s, GC with packed columns (low-resolution GC (LRGC)) allowed separation into groups of isomers (e.g., separation of the TCDDs as a group from the

PeCDDs), but the resolution was inadequate for separation of the isomers within a group (e.g., separation of the 22 isomeric TCDDs). In the mid-1970s, glass capillary columns were first used with PCDDs and PCDFs and were found to offer much better separation of isomers and improved sensitivity. The special skills required to prepare these columns, however, initially restricted their use to a limited number of laboratories. Thus, the commercial development of prepared glass capillary columns, especially the introduction of flexible, stable, reproducible fused silica columns around 1980, was one of the most important advances in environmental PCDD/PCDF analysis (Clement & Tosine, 1988; Clement, 1991).

The HRGC columns used for PCDD/PCDF analysis range from 15 to 60 m in length with inner diameters in the range of 0.22–0.35 mm. They are often referred to as wall-coated open-tubular (WCOT) columns, as the inner wall of the column is uniformly coated with a thin film (0.15–0.25 μm) of a silicone (stationary phase) that accomplishes the separations. Many different stationary phases have been used in PCDD/PCDF analyses. In general, non-polar stationary phases (e.g., alkyl/aryl siloxanes) efficiently separate PCDD/PCDF mixtures into groups with the same numbers of chlorines (all TCDDs and TCDFs, all PeCDDs and PeCDFs, etc.), while polar stationary phases (e.g., cyanosilicones) distinguish between the isomers within a group. Frequently, separate analyses using more than one column are required to ensure adequate separation of congeners, for example, a short to medium-length, non-polar (SE-54 or DB-5) column and a longer polar (Silar 10C or SP 2330) column (Clement & Tosine, 1988). A workshop convened by the Community Bureau of Reference of the Commission of the European Union recently recommended the use of a single, nonpolar column (e.g., DB-5) for samples containing only 2,3,7,8-substituted PCDDs and PCDFs, and both a non-polar and a polar column (e.g., SP 2331, CPSIL 88) for samples which also contain PCDDs/-PCDFs with chlorine at other positions (Maier *et al.*, 1994). Since higher terrestrial vertebrates tend to accumulate selectively the 2,3,7,8-substituted congeners, satisfactory analyses of biological samples from farm animals and humans are often achieved on non-polar columns alone (de Jong *et al.*, 1993).

Gas chromatographic retention times are compared with those of authentic standards of the various PCDD and PCDF isomers (^{13}C-labelled or unlabelled) for tentative isomer identification. Confirmation of identity and quantification are accomplished by mass spectrometry.

(*f*) *Quantification*

Although a few early studies used GC with ECD to quantify PCDDs and PCDFs, modern practice depends almost exclusively on mass spectrometric detection with selected ion monitoring (SIM) of two or more ions from the isotopic group of molecular ions. This allows the use of stable-isotope-labelled internal standards, provides selectivity against coextracted endogenous compounds and many other contaminants and allows congeners with different degrees of chlorination to be quantified separately (Clement & Tosine, 1988). Monitoring of the exact masses of the ions at high mass spectometer resolution provides additional selectivity (Lamparski *et al.*, 1991; Tondeur & Beckert, 1991).

The advantages and disadvantages of various mass spectrometric methods for PCDDs and PCDFs have been extensively reviewed and discussed in recent years (Clement & Tosine, 1988; Buser, 1991; de Jong & Liem, 1993) and are therefore not reviewed here. Either low-resolution mass spectrometry (LRMS) or high-resolution mass spectrometry (HRMS) can provide adequate data, if it has been demonstrated by the analyst that 'the entire analytical procedure exhibits a sufficient sensitivity and specificity with regard to concentration levels of the PCDD/F and the matrix composition' (Maier *et al.*, 1994). Most analysts agree that HRMS, although not essential, is preferable. The monitoring of additional fragment ions or use of collision-induced dissociation and tandem MS can provide additional selectivity for confirmation or resolution of specific analytical difficulties but, in practice, are seldom used.

Electron impact ionization is most commonly used for PCDD/PCDF analyses. Negative-ion chemical ionization is very sensitive for the highly chlorinated congeners but is not suitable for lower congeners such as 2,3,7,8-TCDD and may be difficult to use quantitatively because of its marked dependence on operating conditions (Buser, 1991; Maier *et al.*, 1994).

Some differences in the relative abundances of ion fragments from different isomers have been observed (Buser & Rappe, 1978), but do not provide a means of unambiguously differentiating between isomers; this requires chromatographic separation by GC and/or by prior fractionation (Hagenmaier *et al.*, 1986; Lamparski *et al.*, 1991).

Quantification of PCDDs and PCDFs is accomplished by SIM comparisons of the responses for sample components with those of internal standards, usually ^{13}C-labelled 2,3,7,8-substituted PCDDs. Calibration standards are used to determine detector response for the various congeners and to confirm its linearity in the concentration range of the samples. Careful attention to quality control and quality assurance procedures is essential for the successful analysis of PCDDs and PCDFs at ultra-trace levels (Mitchum & Donnelly, 1991).

1.2 Formation and destruction

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

1.2.1 *Formation of PCDDs*

(*a*) *Chemical reactions*

(i) *Chlorophenoxyacetic acid herbicides* (referred to hereafter as phenoxy herbicides)

The phenoxy herbicide 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) (see IARC, 1977b, 1986a, 1987b) was introduced in the late 1940s, and its use was maximal in the 1960s and 1970s. After that time, it was phased out in most European countries and the United States of America. 2,4,5-Trichlorophenol (TCP) (see IARC, 1979a, 1986b, 1987c) is the

key intermediate in the production of 2,4,5-T. PCDDs, primarily 2,3,7,8-TCDD, were formed during the production of TCP from 1,2,4,5-tetrachlorobenzene.

Depending on the temperature control and purification efficiency, levels of 2,3,7,8-TCDD in commercial products vary greatly. For example, levels of 2,3,7,8-TCDD in drums of the herbicide Agent Orange (a 1 : 1 mixture of the *n*-butyl esters of 2,4,5-T and 2,4-dichlorophenoxyacetic acid (2,4-D); see IARC, 1977c) stored in the United States and in the Pacific before 1970 were between 0.02 and 47 mg/kg (analytical method N). Nearly 500 samples were analysed and the mean value was 1.98 mg/kg (Young *et al.*, 1978; Young, 1983). Since Agent Orange was formulated as a 1 : 1 mixture of the butyl esters of 2,4,5-T and 2,4-D, the levels of 2,3,7,8-TCDD in individual preparations of 2,4,5-T manufactured and used in the 1960s could have been as high as 100 mg/kg (Rappe & Buser, 1981).

Rappe *et al.* (1978a) reported that in other samples of Agent Orange (as well as in 2,4,5-T formulations produced in Europe and the United States in the 1950s and 1960s), 2,3,7,8-TCDD was the dominant PCDD/PCDF contaminant. Only minor amounts of other PCDDs were found, primarily lower chlorinated PCDDs, in samples of Agent Orange (analytical method AC). The concentrations of 2,3,7,8-TCDD have been reported for 2,4,5-T formulations used in Scandinavia (**Table 6**) and New Zealand (**Table 7**).

Table 6. Concentrations of 2,3,7,8-TCDD (mg/kg) in Scandinavian 2,4,5-trichlorophenoxyacetic acid and ester formulations

Sample	Source	2,3,7,8-TCDD
2,4,5-T acid	1952, Sweden	1.10
2,4,5-T ester	Unknown, Sweden	0.50
2,4,5-T ester	Unknown, Sweden	< 0.05
2,4,5-T ester	1960, Sweden	0.40
2,4,5-T ester	1962, Finland	0.95
2,4,5-T ester	1966, Finland	0.10
2,4,5-T ester	1967, Finland	< 0.05
2,4,5-T ester	1967, Finland	0.22
2,4,5-T ester	1967, Finland	0.18
2,4,5-T acid	1964, USA	4.8
2,4,5-T acid	1969, USA	6.0

From Rappe *et al.* (1978a); Norström *et al.* (1979); Rappe & Buser (1981)

Table 7. Average concentrations of 2,3,7,8-TCDD (μg/kg) in 2,4,5-T produced in New Zealand

Year	2,3,7,8-TCDD
1971	950
1972	470
1973	47
1974	33
1975	24
1976	27
1977	31
1978	22
1979	13
1980	14
1981	7.3
1982	8.5
1983	5.3
1984	5.9
1985	4.7

From Smith & Pearce (1986)

As a result of governmental regulations, efforts were made by producers during the 1970s to minimize the formation of 2,3,7,8-TCDD during the production of 2,4,5-T. In the 1980s, all producers claimed that their products contained less than 0.1 mg/kg 2,3,7,8-TCDD (Rappe & Buser, 1981). During production, TCP is separated from 2,3,7,8-TCDD by one or two distillations, which results in 2,3,7,8-TCDD being concentrated in the still-bottom residues. Up to 1 g/kg 2,3,7,8-TCDD in such residues has been reported (Kimbrough *et al.*, 1984; analytical method N).

Sixteen samples of 2,4-D esters and amine salts from Canada were analysed for the presence of PCDDs. Eight of nine esters and four of seven amine salts were found to be contaminated, with esters showing significantly higher levels (210–1752 μg/kg) than salts (20–278 μg/kg) (analytical method AC). The TCDD isomer observed was the 1,3,6,8-isomer, verified using a synthetically prepared standard (Cochrane *et al.*, 1982). On the other hand, Schecter *et al.* (1993a) found no 2,3,7,8-TCDD at a detection limit of 0.02 μg/kg in one 2,4-D sample of Russian origin. Higher chlorinated PCDDs were found in this sample at < 1 μg/kg (I-TEQ for PCDDs/PCDFs, 0.2 μg/kg). Although 2,3,7,8-TCDD is not expected to be a contaminant in 2,4-D, Hagenmaier (1986) reported that one German 2,4-D formulation contained 6.8 μg/kg 2,3,7,8-TCDD (analytical method ACS).

No data were available to the Working Group on the analysis of samples of the herbicide 4-chloro-2-methylphenoxyacetic acid (MCPA) for 2,3,7,8-TCDD or other PCDDs. [However, it is not expected that PCDDs would be formed during the production of MCPA, based on the starting materials and production route.]

(ii) *Hexachlorophene*

The bactericide hexachlorophene (see IARC, 1979b) is prepared from TCP. Due to additional purification, the level of 2,3,7,8-TCDD in this product is usually below 0.03 mg/kg (WHO, 1989) (analytical method AC). Ligon and May (1986) reported 4.7 µg/kg 2,3,7,8-TCDD in one hexachlorophene sample; Baughman and Newton (1972) found 3.8 and 0.5 µg/kg in two samples. The use of hexachlorophene in cosmetics has been banned in the European Union (Her Majesty's Stationery Office, 1989).

(iii) *Chlorophenols*

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. Chlorophenols 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). 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 and 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 (Rappe *et al.*, 1978b). **Table 8** summarizes a number of analyses of the levels of PCDDs in commercial chlorophenol formulations.

Table 8. Levels of PCDDs in commercial chlorophenols (mg/kg)

	2,4,6-Tri-chlorophenol	2,3,4,6-Tetra-chlorophenol	Pentachlorophenol	
			Sample A	Sample B
TCDDs	–	0.7	< 0.02	< 0.1
PeCDDs	< 0.3	5.2	< 0.03	< 0.1
HxCDDs	< 0.5	9.6	10	8
HpCDDs	< 0.2	5.6	130	520
OCDD	–	0.7	210	1 380

From Rappe (1979a)

Buser and Bosshardt (1976) reported the results of a survey of the PCDD and PCDF 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: 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 PCDDs and PCDFs were 2–16 and 1–26 mg/kg, respectively, for the first series of samples and 120–500 and 85–570 mg/kg, respectively, for the second series of samples. The levels of OCDD were as high as 370 mg/kg; this congener dominated the PCDD content of the samples.

Miles *et al.* (1985) analysed HxCDDs in PCP samples from five different manufacturers in Canada using an isomer-specific analytical method (analytical method ACI).

The study included both free PCP and sodium salts. Total HxCDDs in PCP samples ranged from 0.66 to 38.5 mg/kg while, in the sodium salts, levels between 1.55 and 16.3 mg/kg were found. The most abundant HxCDD isomer in free PCP was the 1,2,3,6,7,8-isomer; however, in the sodium salts, the 1,2,3,6,7,9- and 1,2,3,6,8,9-HxCDD pair was the most abundant, probably due to different routes of synthesis.

Hagenmaier (1986) reported that 2,3,7,8-TCDD could be detected in PCP formulations from Germany and the United States at concentrations of 0.21–0.56 μg/kg (analytical method ABS). 1,2,3,7,8-PeCDD was found in these formulations at higher concentrations (0.9–18 μg/kg; analytical method ABS) (Hagenmaier & Brunner, 1987).

(iv) *Chlorodiphenyl ether herbicides*

Yamagishi *et al.* (1981) reported on the occurrence of PCDDs and PCDFs in the Japanese commercial diphenyl ether herbicides primarily used in rice fields: 1,3,4-trichloro-2-(4-nitrophenoxy)benzene (CNP), 2,4-dichloro-1-(4-nitrophenoxy)benzene (Nitrofen; NIP; see IARC, 1983) and 2,4-dichloro-1-(3-methoxy-4-nitrophenoxy)-benzene (chlomethoxynil; X-52). The total TCDD content was 140–170 mg/kg in CNP, 0.38 mg/kg in NIP and 0.03 mg/kg in X-52. Very few synthetic standards were used, but the major TCDDs identified were the 1,3,6,8- and 1,3,7,9- isomers, the expected impurities in the starting material 2,4,6-trichlorophenol (2,4,6-TCP). No 2,3,7,8-TCDD was detected in these samples.

(v) *Hexachlorobenzene*

Hexachlorobenzene (see IARC, 1979c) used to be applied for the control of wheat bunt and fungi. Villanueva *et al.* (1974) analysed three commercial hexachlorobenzene preparations and identified OCDD in the range of 0.05–211.9 mg/kg (analytical method C).

(vi) *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-TCDD (Rappe *et al.*, 1987a). A survey performed in 1987 in the United States showed that the concentrations of 2,3,7,8-TCDD in bleached pulp ranged from undetectable (at a detection limit of 1 ng/kg) up to 51 ng/kg, with a median concentration of 4.9 ng/kg and a mean of 13 ng/kg (Gillespie & Gellman, 1989).

New technology has been developed for pulp bleaching using chlorine dioxide (elemental chlorine-free, ECF) or non-chlorinated reagents (total chlorine-free, TCF). No 2,3,7,8-TCDD was found in ECF- or TCF-bleached pulp (detection limit, 0.03 ng/kg) (analytical method ABS) (Rappe & Wågman, 1995).

(vii) *Dyes and pigments*

Williams *et al.* (1992) analysed a series of dioxazine dyes and pigments (Blue 106, Blue 108 and Violet 23) from Canada. OCDD was detected in all samples (analytical method ACS) in the range of 23 μg/kg–42 mg/kg, with the highest concentrations in three samples of Blue 106.

(*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 the presence of up to 17 PCDD peaks, but isomer identification and quantification were not possible due to the lack of synthetic standards. Buser *et al.* (1978a) studied fly ash from a municipal incinerator and an industrial heating facility in Switzerland. In the former, the total level of PCDDs was 0.2 mg/kg. In the industrial incinerator fly ash, the level was 0.6 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, PCDDs were destroyed only after adequate residence times (> 2 s) at temperatures above 800 °C. Total emissions of PCDDs 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 9**).

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 PCDD 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 levels 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 emission of 1,2,3,7,8-PeCDD normally exceeds the emission of 2,3,7,8-TCDD by a factor of 3–10.

During the second half of the 1980s and 1990, regulatory agencies in several countries, such as Sweden, Germany and the Netherlands, announced strict regulations for municipal solid-waste incinerators. The European Union value is 0.1 ng I-TEQ per m^3 (European Union, 1994) (see column 4, **Table 9**). 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 PCDD emissions from municipal solid-waste incinerators, which had been considered to be major sources.

Table 9. Estimated range of emissions of PCDDs 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 (1)	Maximum from average operation (2)	High emissions[a] (3)	Achievable with modern plants with gas cleaning[b] (4)	Most probably highest emissions (5)
2,3,7,8-TCDD	0.1	1.5	7.5	0.01	0.1
1,2,3,7,8-PeCDD	0.3	14	70	0.03	0.3
1,2,3,4,7,8-HxCDD	0.2	31	155	0.02	0.2
1,2,3,6,7,8-HxCDD	0.6	56	280	0.05	0.6
1,2,3,7,8,9-HxCDD	0.4	20	100	0.04	0.4

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)

(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 9**, 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 emission of PCDDs. They found the mean levels of total PCDDs to be 69 ng/m^3, but no isomer-specific data were given. Data cited by the United States Environmental Protection Agency indicate that flue gas emissions from hospital waste incinerators are in the range 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 municipal solid-waste incinerators.]

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

The formation of PCDDs during the combustion of PVC (see IARC, 1979d) 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 PCDDs/PCDFs. However, experiments performed in incinerators showed the effect of PVC on the formation of PCDDs/PCDFs to be minimal (Frankenhaeuser *et al.*, 1993; Wikström *et al.*, 1996).

(v) *Combustion of wood*

Schatowitz *et al.* (1994) measured 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 10**. Data on PCDDs and PCDFs are not separated.

Table 10. PCDD/PCDF emissions from wood and household waste combustion

Fuel	Furnace	(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)

(vi) *Automobile emissions*

Hagenmaier *et al.* (1990a) reported on the automobile exhaust emissions of PCDDs/-PCDFs and the lower chlorinated congeners and brominated analogues. The results of four representative experiments using leaded gasoline, unleaded gasoline with or without catalytic converters, and diesel fuel (analytical method ABS) are summarized in **Table 11**.

Table 11. PCDDs from automobile emissions (pg/m^3)

	Leaded gasoline	Unleaded gasoline	Unleaded gasoline with catalytic converter	Diesel engine
TCDDs	595	84	3.7	1.9
2,3,7,8-TCDD	16.7	0.5	0.21	0.40
PeCDDs	436	93	3.3	1.0
1,2,3,7,8-PeCDD	55.5	3.7	0.21	0.46
HxCDDs	244	59	3.4	1.2
1,2,3,4,7,8-HxCDD	24.5	3.2	0.31	< 0.26
1,2,3,6,7,8-HxCDD	27.1	3.3	0.45	< 0.26
1,2,3,7,8,9-HxCDD	24.1	3.4	0.40	< 0.26
HpCDDs	152	18	5.0	4.5
1,2,3,4,6,7,8-HpCDD	65.7	7.8	1.97	2.24
OCDDs	65	34	21.9	22.4
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.* (1990a)

(*c*) *Photochemical reactions*

It was reported by Nilsson *et al.* (1974) that chlorinated 2-phenoxyphenols (predioxins) could be converted to PCDDs by a photochemical cyclization reaction. More recently, Vollmuth *et al.* (1994) reported on the photochemical dimerization of chlorophenols yielding OCDD. The most important photochemical reaction, however, is photochemical dechlorination resulting in transformation of PCDDs to lower chlorinated compounds. Kieaitwong *et al.* (1990) reported that 2,3,7,8-TCDD could be identified among the products of photochemical dechlorination of OCDD.

(*d*) *Biochemical reactions*

Öberg *et al.* (1990) showed that chlorinated phenols could be transformed *in vitro* to PCDDs by peroxidase-catalysed oxidation. It was also demonstrated, using ^{13}C-labelled PCP, that HpCDDs and OCDDs could be formed in sewage sludge (Öberg *et al.*, 1993).

1.2.2 *Destruction of PCDDs*

Although PCDDs generally are considered to be very stable, they can undergo a series of chemical degradation reactions. Peterson and Milicic (1992) reported the degradation

of a series of PCDDs using a mixture of potassium hydroxide in polyethylene glycol. Oku *et al.* (1995) also reported the successful destruction of PCDDs using sodium or potassium hydroxide in the new solvent 1,3-dimethyl-2-imidazolidinone.

Thermal degradation of PCDDs 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.

Hosoya *et al.* (1995) showed that 2,3,7,8-TCDD can be decomposed successfully by photochemical reactions with a hydrophobic octadecylsilylated-silica gel used as the solid support. Conversely, Vollmuth and Niessner (1995) reported no significant degradation of PCDDs by ultraviolet radiation, ozone or a combination of the two.

Adriaens *et al.* (1995) reported on the biologically mediated reductive dechlorination of HxCDDs 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 PCDDs*

(*a*) *Occupational exposures*

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

Germany: In November 1953, an uncontrolled decomposition reaction occurred in an enclosed TCP production facility of BASF at Ludwigshafen, Germany. 2,3,7,8-TCDD was formed during the reaction and contaminated the autoclave section of the building. Employees were exposed to 2,3,7,8-TCDD contaminant during subsequent clean-up and repair activities (Zober *et al.*, 1990; Zober & Päpke, 1993). Analysis of a sample of adipose tissue collected in 1984 from one of the exposed workers showed a 2,3,7,8-TCDD concentration of 100 ng/kg wet weight adipose tissue (Nygren *et al.*, 1986). Zober and Päpke (1993) later analysed samples of autopsy tissues collected from four cases 35–39 years after the same accident. In all samples, the concentrations of most PCDDs were in the normal range, with the exception of 2,3,7,8-TCDD, the concentrations of which were much higher (see **Table 12**; analytical method ACS). Measurements of the blood concentration of 2,3,7,8-TCDD were performed in 1988 in 138 men whose first contact with PCDDs occurred within one year of the accident (Zober *et al.*, 1994). Current values were extrapolated back to the time of exposure. The geometric mean concentrations ranged from 1118 ng/kg in group with severe chloracne to 148 ng/kg in group with no chloracne. Background blood levels of 2,3,7,8-TCDD in the plant were ≤ 5 ng/kg. Ott *et al.* (1993) reported on the concentrations of 2,3,7,8-TCDD in blood lipids of these workers sampled between 1988 and 1992, 35–39 years after the accident. The geometric mean value was 15.4 ng/kg. [Back-calculation to the time of the accident, using a 2,3,7,8-TCDD half-life of seven years, gives an estimated blood lipid concentration of 480 ng/kg. If the normal German background concentration in 1990 is first subtracted, the estimate is approximately 400 ng/kg.]

Table 12. Post-mortem concentrations of 2,3,7,8-TCDD (ng/kg fat) in autopsy samples from four individuals involved in the 1953 BASF accident

	Blood	Adipose[a]	Liver	Kidney	Brain
Case 1	255	171	178	198	36
Case 2	32	34	28	–	7
Case 3	7482[b]	13 563	13 563	11 195	2457
Case 4	448	648	550	–	–

From Zober & Päpke (1993)

[a] German background concentration, 5 ng/kg

[b] Significant weight loss before death may have resulted in increased levels of 2,3,7,8-TCDD in blood and other tissues, especially in Case 3. Eight months before death, blood concentration for Case 3 was 518 ng/kg (compared with 7482 ng/kg at autopsy); Case 3 lost 20–25 kg in body weight in the eight months before death. Case 2 showed a smaller effect but in the same direction (blood concentration, 17 ng/kg at 5 months before death; 7–10 kg weight loss). Case 4 had a blood level of 590 ng/kg 10 months before death, and no subsequent weight loss was reported. No data were available for Case 1.

Päpke *et al.* (1992) analysed in 1988–91 the blood of 12 workers exposed during production of TCP. Concentrations of 2,3,7,8-TCDD were approximately 100 times higher than those in a group of background controls, while the concentrations of the higher chlorinated PCDDs were lower in exposed workers than in controls, but not significantly. In blood from 20 workers exposed to PCDDs during production of PCP, the concentrations of OCDD, 1,2,3,4,6,7,8-HpCDD and 2,3,7,8-derived HxCDDs were much higher in the exposed workers than in controls (see **Table 13**).

Table 13. Concentrations of PCDDs in human blood (ng/kg fat) during production of chlorophenols in Germany

	TCP (n = 12)	PCP (n = 20)	Control (n = 102)
2,3,7,8-TCDD	331.8	4.5	3.6
1,2,3,7,8-PeCDD	10.7	28.3	13.8
All 2,3,7,8-substituted HxCDDs	34.5	398.8	78.9
1,2,3,4,6,7,8-HpCDD	44.3	2 514.1	92.4
OCDD	428.9	33 191.5	610.3

From Päpke *et al.* (1992)

TCP, 2,4,5-trichlorophenol; PCP, pentachlorophenol

Flesch-Janys *et al.* (1996a) studied 48 workers (45 men and 3 women) from a Boehringer-Ingelheim plant in Hamburg manufacturing a range of herbicides. The blood of these workers was sampled twice (for 43 workers) or three times (for 5 workers). The mean time between the end of employment and the first blood sampling was 5.4 years (median, 2.0 years), and the mean time between first and last blood sampling was 5.6 years (median, 6.3 years) (analytical method AB). The results are given in **Table 14**. In the first sampling, the median concentration of 2,3,7,8-TCDD was 84.1 ng/kg (range, 15.6–300.2 ng/kg). The corresponding values for the second sampling are: median, 48.9 ng/kg (range, 7.7–277.9 ng/kg). More measurements from the Boehringer plant and two other German plants (the BASF plant in Ludwigshafen (this cohort was different from the accident-exposed group) and a Bayer plant in Ingelheim (Becher *et al.*, 1996)) were also performed (summarized in Kogevinas *et al.*, 1997). The 190 subjects from the Boehringer plant showed mean estimated concentrations at the end of employment of 141 ng/kg 2,3,7,8-TCDD (range, 3–2252 ng/kg). In the BASF plant, 20 subjects showed mean 2,3,7,8-TCDD values of 401.7 ng/kg (range, 23–1935 ng/kg), while 19 workers at the Bayer plant had a mean level of 3.2 ng/kg (range, 1.3–6.5 ng/kg).

Table 14. Concentrations of PCDDs (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
2,3,7,8-TCDD	48	84.1	15.6–300.2	48.9	7.7–277.9
1,2,3,7,8-PeCDD	40	51.1	27.2–251.2	35.9	13.2–190.3
1,2,3,4,7,8-HxCDD	41	83.2	25.6–746.9	51.3	18.7–559.7
1,2,3,6,7,8-HxCDD	40	354.7	127.7–2939	255.8	101.6–2493
1,2,3,7,8,9-HxCDD	39	88.2	29.3–680.8	39.5	17.6–288.3
1,2,3,4,6,7,8-HpCDD	26	641.2	310.5–5152	234.5	94.2–1526
OCDD	32	2 526	1356–17 566	1 288.5	842–10 395
TEQ[d]	45	191.9	43.1–767.2	115.3	29.4–500.4

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

[d] German TEQ (total PCDDs/PCDFs)

United States: The largest study in the United States of workers assigned to production of substances contaminated with 2,3,7,8-TCDD was conducted by the United States National Institute for Occupational Safety and Health (Fingerhut *et al.*, 1991a). A more detailed technical report of this study is also available (Fingerhut *et al.*, 1991b). This 12-plant study included the Nitro, WV, plant of Monsanto and the Midland, MI, plant of Dow. The cohort was constructed after a review of personnel records at plants producing chemicals known to be contaminated with 2,3,7,8-TCDD (principally TCP

and 2,4,5-T). The cohort included most workers in the United States likely to have been exposed to 2,3,7,8-TCDD in chemical manufacturing; these were 5000 men with work records showing assignment to a production or maintenance job in a process involving 2,3,7,8-TCDD contamination, as well as an additional 172 men without work history records but known to have been exposed based upon inclusion in a prior cross-sectional medical study by Suskind and Hertzberg (1984) at the Nitro, WV, plant. These latter 172 men and an additional 30 men in the United States National Institute for Occupational Safety and Health study lacked sufficient work history information to permit their inclusion in more detailed analyses by duration of exposure. Follow-up was conducted through 1987. Serum 2,3,7,8-TCDD levels, available for 253 cohort members at two plants (different from the Nitro and Midland plants), measured in 1987 averaged a mean of 233 ng/kg, compared with 7 ng/kg for a group of 79 unexposed workers. The mean level was 418 ng/kg for 119 workers exposed for more than one year. All workers had last been exposed 15–37 years earlier. Extrapolation to the date of last employment of these workers, assuming a 7.1-year half-life for 2,3,7,8-TCDD elimination, indicated a mean serum level at that time of 2000 ng/kg (highest level, 32 000 ng/kg). The correlation between extrapolated serum 2,3,7,8-TCDD levels and duration of exposure was 0.72 (Fingerhut *et al.*, 1991b).

Sweden: Littorin *et al.* (1994) analysed blood from five workers at a factory in Sweden where 2,4-D, MCPA, 2-(4-chloro-2-methylphenoxy)propanoic acid (MCPP) and 2,4,6-trichlorophenol (Saracci *et al.*, 1991) had been formulated, but where production had ceased in 1979, and that of five referents. [2,4,5-T was not produced at this site.] The results are given in **Table 15**. The concentrations of all PCDDs were high, especially that of 1,2,3,7,8-PeCDD, which was unexpectedly found to be higher than that of 2,3,7,8-TCDD. However, a leachate sample from the factory also contained a higher level of 1,2,3,7,8-PeCDD.

Austria: Neuberger *et al.* (1991) reported mean blood levels of 389 ng/kg 2,3,7,8-TCDD (range, 98–659 ng/kg) in TCP and 2,4-D production workers with chloracne exposed about 17 years earlier.

Netherlands: Hooiveld *et al.* (1996a) took blood samples in 1993 from 48 persons occupationally exposed to phenoxy herbicides, chlorophenols (and the contaminant 2,3,7,8-TCDD) in 1955–85 in a chemical factory where an accident occurred in 1963. The geometric mean levels of 2,3,7,8-TCDD were 22.9 ng/kg in men who worked in the main production unit and 87.2 in subjects exposed as a result of the accident.

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

Nygren *et al.* (1986) reported analyses of adipose tissue from 31 persons in Sweden, 13 of whom were exposed to phenoxy herbicides and 18 of whom were unexposed controls. Of the exposed persons, 11 had neoplastic diseases; in controls, six persons had cancer, giving a total of 17 cancer patients and 14 persons without cancer. No difference was found between exposed and unexposed or between cancer patients and persons without cancer (see **Table 16**; analytical method ACS).

Table 15. Blood plasma concentrations of PCDDs in five phenoxy herbicide/chlorophenol production workers and five referents in Sweden

Analyte	Plasma concentration (ng/kg fat)				
	Workers		Referents		*p* value[a]
	Mean[b]	Range	Mean[b]	Range	
PCDDs					
2,3,7,8-TCDD	17	9.1–37	2.0	0.7–3.3	0.008
1,2,3,7,8-PeCDD	22	14–33	6.9	4.0–10	0.008
1,2,3,4,7,8-HxCDD	4.5	2.7–6.9	2.4	0.8–4.4	0.2
1,2,3,6,7,8-HxCDD	43	28–58	28	10–39	0.2
1,2,3,7,8,9-HxCDD	18	8.8–27	7.5	2.0–13	0.1
1,2,3,4,6,7,8-HpCDD	87	33–120	43	7.9–78	0.06
OCDD	750	400–1300	280	110–440	0.02
I-TEQ	56	30–94	21	8.2–34	0.02

From Littorin *et al.* (1994)

[a] Wilcoxon's rank sum test for unpaired samples (two-tail)

[b] Values below the detection limit were set at half that concentration.

In another study in Sweden including 20 non-Hodgkin lymphoma patients and 17 controls, Hardell *et al.* (1996) reported no significant differences in the concentrations of PCDDs and PCDFs in adipose tissue (see also Section 2.2.2(*b*)).

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 2,3,7,8-TCDD in blood serum of nine of these workers (**Table 17**) gave arithmetic means of 53.3 ng/kg for the exposed group and 5.6 ng/kg for a group of matched controls (ratio, 9.5). For all other congeners, the ratio between the levels in the exposed group and the controls was below 1.5.

Military personnel in Viet Nam: The Viet Nam Experience Study is described by its authors as a 'multidimensional assessment of the health of Viet Nam veterans (Centers for Disease Control Vietnam Experience Study, 1988a,b,c,d). This study was designed to examine effects among men who served in the United States armed forces in Viet Nam, where Agent Orange (see Section 1.2.1(*a*)(i)) was widely sprayed as a herbicide and defoliant (Operation Ranch Hand). The study population was a random sample of men who enlisted in the United States Army from 1965 through 1971, whose military occupational status was other than 'duty soldier', who enlisted for a single term with a minimum of 16 weeks' active duty and who were discharged at pay grades of E-1 to E-5. The controls were selected from among veterans enlisting during the same period but whose duty station was the United States, Germany or Korea. Participation involved

Table 16. Concentrations of PCDDs in human adipose tissue (ng/kg wet weight) from persons exposed to phenoxy herbicides in Sweden

Compound	Mean value (n = 31)	Range	Exposed		Unexposed		Cancer patients		Controls (without cancer)	
			Mean (n = 13)	Range	Mean (n = 18)	Range	Mean (n = 17)	Range	Mean (n = 14)	Range
2,3,7,8-TCDD	3	0–9	2	0–9	3	2–6	3	2–9	3	2–6
1,2,3,7,8-PeCDD	10	3–24	6	3–24	9	4–18	9	4–24	9	3–18
1,2,3,6,7,8-HxCDD	15	3–55	19	8–55	12	3–18	18	3–55	12	8–18
1,2,3,7,8,9-HxCDD	4	3–5	5	3–13	4	3–5	4	3–13	4	3–5
1,2,3,4,6,7,8-HpCDD	97	12–380	104	20–380	85	12–176	100	12–380	85	20–168
OCDD	414	90–763	398	90–763	421	98–679	408	90–620	421	182–763

From Nygren *et al.* (1986)

completion of a telephone survey of current and past health status by 7924 veterans who served in Viet Nam and 7364 veterans who served outside Viet Nam. A random subsample of 2940 Viet Nam and 1972 non-Viet Nam veterans participated in the health evaluation component.

Table 17. Levels of PCDDs 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-TCDD	53.3 ± 16.1	5.6 ± 1.1	9.5
1,2,3,7,8-PeCDD	12.4 ± 1.1	8.8 ± 0.7	1.4
1,2,3,4,7,8-HxCDD	6.8 ± 0.5	5.7 ± 0.4	1.2
1,2,3,6,7,8-HxCDD	28.6 ± 5.1	23.3 ± 4.9	1.2
1,2,3,7,8,9-HxCDD	9.9 ± 0.9	8.2 ± 0.6	1.2
1,2,3,4,6,7,8-HpCDD	121.9 ± 28.5	119.4 ± 18.4	1.0
OCDD	788.6 ± 82.3	758.7 ± 92.8	1.0

From Smith *et al.* (1992a)

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

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

Investigators in the Centers for Disease Control Veterans Health Studies (1988; see **Table 18**) studied serum levels of 2,3,7,8-TCDD among 646 Viet Nam veterans who were ground combat troops during 1967–68 in areas heavily sprayed with Agent Orange. Also studied were 97 veterans who did not serve in Viet Nam. The mean serum level was 4 ng/kg for each group, as measured in 1987. There was no correlation between serum levels of 2,3,7,8-TCDD and service in areas of Viet Nam ranked by level of presumed intensity of spraying based on military records, or between serum levels and self-reported levels of exposure to Agent Orange. Only two Viet Nam veterans had levels above 20 ng/kg (25 and 45 ng/kg). This study did not include Viet Nam veterans who served in the Air Force spraying of Agent Orange (the Operation Ranch Hand veterans), nor members of the Army Chemical Corps which also sprayed Agent Orange. There are data indicating that these specific groups (especially Ranch Hand veterans) had significant levels of exposure to PCDDs above background.

One of the largest epidemiological studies of United States military personnel stationed in Viet Nam is being conducted by the United States Air Force. The study population consists of Air Force personnel who served in Operation Ranch Hand units in Viet Nam from 1962 to 1971 and who were employed in the dissemination of Agent Orange through aerial spraying. Comparisons included Air Force personnel who flew or maintained C-130 aircraft in south-east Asia during the same period. The study design includes a series of cross-sectional medical studies conducted at five-year intervals

Table 18. Serum levels of 2,3,7,8-TCDD in United States veterans who served in Viet Nam and elsewhere in 1987

Place of service	No.	Mean ± SD (ng/kg)	Percentile				
			25th	50th	75th	90th	95th
Non-Viet Nam	97	4.1 ± 2.3	2.8	3.8	4.9	7.2	9.2
Viet Nam	646	4.2 ± 2.6	2.8	3.8	5.1	6.8	7.8

From Centers for Disease Control Veterans Health Studies (1988)

beginning with the baseline study in 1982 (1045 exposed, 1224 unexposed). Two follow-up evaluations were conducted in 1985 (1016 exposed, 1293 unexposed) and 1987 (995 exposed, 1299 unexposed). Each cross-sectional study included comprehensive physical and psychological evaluations. In the 1982 baseline and 1985 and 1987 follow-up studies, exposure was based on the comparison of the Ranch Hand group versus the comparison group. An additional analysis estimated the approximate exposure (low, medium or high) for the Ranch Hand group by using historical military data and herbicide procurement and usage records (Roegner *et al.*, 1991).

The Air Force Ranch Hand veterans (Wolfe *et al.*, 1990) (see **Table 19**) tested in 1987 and exposed during 1962–71 (n = 888) had a median of 12.4 ng/kg serum 2,3,7,8-TCDD compared with a median of 4.2 ng/kg for comparison subjects (n = 856) who were Air Force personnel not exposed to herbicides. The highest levels were for non-flying enlisted personnel (n = 407), with a median of 23.6 ng/kg. Air Force Ranch Hand veterans sprayed approximately 88–90% (Thomas & Kang, 1990) of the Agent Orange applied in Viet Nam, with application from airplanes. The remaining 10–12% was sprayed by the Army Chemical Corps which applied the herbicide around the perimeter of bases either from the ground or from helicopters. Kahn *et al.* (1988) studied 2,3,7,8-TCDD serum levels in a sample of 10 heavily exposed Viet Nam veterans and matched controls. The two highest levels were found in Army Chemical Corps personnel (approximately 75 and 180 ng/kg. It should be noted that both Ranch Hand veterans and Army Chemical Corps veterans sprayed other herbicides (Agent White or Agent Blue) besides Agent Orange, although few other herbicides were contaminated with PCDDs (Thomas & Kang, 1990). These data suggest that, although some Ranch Hand subjects were exposed to very high levels of 2,3,7,8-TCDD, most had lower exposures.

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 are summarized in **Table 20**. The only difference in the mean values was for 2,3,7,8-TCDD, for which the arithmetic mean for the heavily exposed group is approximately 8–10 times higher than that for controls. The highest level found was 213 ng/kg. In another report from the same study group, Gochfeld *et al.* (1989)

found a good correlation (r = +0.89) between the concentrations of 2,3,7,8-TCDD in blood and adipose tissue.

Table 19. Serum 2,3,7,8-TCDD levels (ng/kg) in Ranch Hand and control veterans

Stratum	Ranch Hand veterans			Comparison subjects		
	No.	Median	Range	No.	Median	Range
Flying officers (pilot)	247	7.3	0.0–42.6	239	4.7	0.0–18.5
Flying officers (navigator)	63	9.3	1.1–35.9	53	4.5	2.2–7.9
Nonflying officers	19	6.6	3.1–24.9	11	4.3	0.0–6.0
Flying enlisted personnel	152	17.2	0.0–195.5	137	4.0	0.0–12.7
Nonflying enlisted personnel	407	23.6	0.0–617.7	416	3.9	0.0–54.8
All personnel	888	12.4	0.0–617.7	856	4.2	0.0–54.8

From Wolfe *et al.* (1990)

(iii) *Exposure to PCDDs at incinerators*

The levels of PCDDs in blood from 11 workers at a Swedish hazardous waste incinerator fell within the range of the normal background (Rappe *et al.*, 1992). The same result was reported by Bolm-Audorff *et al.* (1994) for 31 workers at three hazardous waste incinerators in Germany.

Böske *et al.* (1995) analysed 37 blood samples of employees at a municipal solid-waste incinerator. They reported that no increased concentrations could be attributed to professional activities by the donors. Similarly, Päpke *et al.* (1993a) found normal I-TEQ values in 10 blood samples from workers employed at municipal solid-waste incinerators in Germany.

In order to determine occupational exposure of employees in three hazardous waste incinerators, 25 workplace air measurements were analysed (Päpke *et al.*, 1994a). The highest concentration measured was 3.79 pg I-TEQ/m^3, corresponding to 7.6% of the German occupational technical exposure limit (TRK) of 50 pg I-TEQ/m^3 (see Section 1.5). All the sampling took place during a normal working day at the plants.

The exposure situation can change quite drastically when repair works are carried out at hazardous waste incinerators and/or during welding, cutting and burning of metals (Menzel *et al.*, 1996), which can lead to exceptionally high concentrations. Outside of hazardous waste incinerators, high concentrations can also occur at demolition sites where steel constructions and machines are burned. The TRK value was exceeded by factors of 2–24 in the cases observed. All air samples were taken by personal sampling.

Table 20. Concentrations of PCDDs 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-TCDD	46.2	19.1	6.6	0.9	4.6	0.9	15.7	5.9	3.9
1,2,3,7,8-PeCDD	13.7	2.5	14.3	2.3	14.4	2.3	11.7	12.9	13.3
1,2,3,4,7,8-HxCDD	15.5	2.7	12.0	2.8	8.8	2.2	13.2	8.5	7.0
1,2,3,6,7,8-HxCDD	124	10.7	108	25.5	96.3	30.3	120	80.6	73.8
1,2,3,7,8,9-HxCDD	21.6	4.0	11.5	2.1	11.1	4.8	16.9	8.7	6.8
1,2,3,4,6,7,8-HpCDD	201	19.2	157	28.2	139	51.1	194	137	108
OCDD	1 582	461	1 118	356	1 120	406	1 204	618	709

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

(iv) *Exposure in paper and pulp mills*

Exposure to PCDDs and PCDFs among workers in paper and pulp mills appears not to be significantly higher than among referents outside the paper industry, even among those workers thought to have the most opportunity for exposure (those exposed to the effluent of the bleaching process).

The United States National Institute for Occupational Safety and Health (Mouradian *et al.*, 1995) conducted air and wipe sampling in a paper mill and measured PCDD and PCDF levels in the sera of 46 workers (14 with low potential exposure, 32 with high potential exposure) and in 16 community residents who served as referents. While some low levels of PCDD and PCDF were detected in the air, there were no differences in serum PCDD and PCDF levels between referents, 'low-exposure' workers and 'high-exposure' workers, all of whom showed normal low levels. For 2,3,7,8-TCDD, the respective medians were 1.8, 1.9 and 1.9 ng/kg, while the median PCDD/PCDF total I-TEQs were 19.1, 21.2 and 18.1 ng/kg for each group, respectively. Regression analyses indicated that occupational exposure was not a significant predictor of serum I-TEQ levels, but consumption of local fish, age and body mass index were predictors. A number of workers had a long history in the plant and would have been expected to have higher serum levels if occupational exposure was significant.

Rosenberg *et al.* (1994) found similar results for workers in a Finnish paper mill. They measured serum levels of PCDDs and PCDFs in 14 workers from the bleaching area, 20 workers from the paper mill and 14 controls who worked in areas of the mill without contact with paper or bleach. There were no significant differences in lipid-adjusted PCDD/PCDF concentrations between the three groups (median PCDD/PCDF total I-TEQs, 52.7, 54.7 and 47.0 ng/kg, respectively) and results for all three groups were similar to background concentrations for men not occupationally exposed. There were some significant differences in levels of 2,3,7,8-TCDD (medians, 4.9, 2.4 and 3.3 ng/kg, respectively) and of 2,3,7,8-TCDF (medians, 2.9, 1.5 and 1.6 ng/kg, respectively), with workers in the bleaching areas having the highest levels. However, levels of these congeners did not differ from background levels for workers not exposed occupationally. The authors indicated that workers ate locally caught fish one to two times a week and that fish consumption may have influenced the results.

(v) *Exposure in steel mills*

PCDDs and PCDFs have been investigated in the work environment in steel mills in Sweden by Antonsson *et al.* (1989). Values for air samples at points close to a furnace, an overhead crane and a crane cabin ranged between 0.80 and 14 pg Nordic TEQ/m^3.

From these results, a daily intake of PCDDs/PCDFs was estimated to be 5–10% of the maximum limit of admissible daily intake (ADI; 35 pg/kg per week).

(b) Population exposure due to industrial accident

In an accident on 10 July 1976 at the ICMESA plant at Seveso, Italy, a runaway reaction led to a blow-out of a TCP production reactor. The chemical cloud that emerged from the reactor entrained nearly 2900 kg of organic matter, including at least 600 kg of

sodium trichlorophenate and an amount of 2,3,7,8-TCDD which is still being evaluated [probably of the order of several kilograms]. The visible part of the cloud rose up to about 50 m, subsequently subsided and fell back to the earth, but was wind-driven over a wide area. Within less than 2 h after the accident, chemicals settled on the ground as far as 6 km south of the factory, or were dispersed by wind streams. Plants, domestic animals and birds were seriously affected, many dying within a few days of the accident [probably due to chemicals other than 2,3,7,8-TCDD]. About 10 days after the accident, 2,3,7,8-TCDD was found in samples of various types collected near the factory (Bertazzi & di Domenico, 1994). As a first step, information on the location of toxic and pathological events was used to draw an approximate diagram of the contaminated area. This was confirmed by chemical monitoring of 2,3,7,8-TCDD in the soil carried out under emergency conditions. Within five weeks of the accident, the area was subdivided into Zones A, B and R in descending order of 2,3,7,8-TCDD contamination and toxicological risk levels. The borderline between Zones A and B was set at average 2,3,7,8-TCDD concentrations in the soil of ≤ 50 μg/m^2; the boundaries between Zones A and B and Zone R were fixed where the average contamination was ≤ 5 μg/m^2. Zone R included the remaining territory where detectable levels of 2,3,7,8-TCDD (formally ≥ 0.75 μg/m^2) were found. Soil levels are further described in Section 1.3.2.

From Zone A, over 730 inhabitants were evacuated. Zones B and R were subjected to area-specific hygiene regulations including prohibition of farming, consumption of local agricultural products and keeping poultry and other animals. All persons residing in these zones at the time of the accident, as well as all the newborn and new residents in the subsequent 10-year period, were considered to have been exposed. Three exposure categories were formed, corresponding to the zone of residence of the subjects at the time of the accident or later entry into the area. As a reference, the population of 11 municipalities surrounding the contaminated area was adopted (Bertazzi *et al.*, 1993).

Mocarelli *et al.* (1990) reported analyses of 19 non-randomly selected samples of blood collected in 1976 from persons living in Zone A (**Table 21**) (analytical method ACS). Of these persons, 10 had chloracne of type 3 or 4 (see Section 4.2.1(*a*)) and nine had no chloracne. The persons with chloracne in general had higher concentrations than persons without chloracne, but high concentrations were also found in the healthy group. The 2,3,7,8-TCDD level of 56 000 ng/kg fat is the highest ever reported.

Recently, blood measurements of 2,3,7,8-TCDD were performed in persons randomly sampled from the most contaminated areas (Zones A and B), and in members of the population adopted as reference in the epidemiological investigations (Landi *et al.*, 1996). Preliminary results were reported. In the six persons from Zone A, the median value was 71.5 ng/kg (extrapolation back to 1976 assuming a half-life of 7.1 years gave a value of 388.7 ng/kg); in the 52 inhabitants in Zone B, the corresponding values were 12.5 ng/kg and 77.6 ng/kg, while in the 52 subjects from the reference population, the median value was 5.5 ng/kg. Women consistently had higher levels of 2,3,7,8-TCDD than men in all three areas.

Table 21. Concentrations of 2,3,7,8-TCDD in the blood[a] of individuals from Zone A, Seveso

Age in 1976	Chloracne type[b]	2,3,7,8-TCDD (ng/kg fat)
4	Type 4	56 000
2	Type 4	27 800
6	Type 4	26 400
4	Type 4	26 000
8	Type 3	17 300
6	Type 4	15 900
11	Type 4	12 100
10	Type 3	7 420
5	Type 3	1 690
16	Type 3	828
15	None	10 400
38	None	9 140
27	None	6 320
71	None	5 560
39	None	4 540
41	None	3 730
55	None	3 050
46	None	2 650
50	None	1 770

From Mocarelli *et al.* (1990)

[a] Blood samples were collected in 1976

[b] Types 3 and 4 are the most severe chloracne

(*c*) *Summary table*

Table 22 summarizes data from the major cohorts of populations highly exposed to 2,3,7,8-TCDD occupationally or as a result of accidents. Estimates of mean or median exposures, or in one case a range of individual exposures, are presented as reported in the cited publications. When tissue concentrations were measured, concentrations at the time of exposure estimated in the cited publications, or back-calculated by the Working Group based on a seven-year half-life, are also given.

1.3.2 *Environmental occurrence*

I-TEQ values are for PCDDs and PCDFs combined.

(*a*) *Air*

PCDD/PCDF levels in air have been reported for many countries during the last 10 years. Due to the enormous amount of information, only a selection of representative data are summarized below. For detailed information, see Appendix 1 (Table 1).

Table 22. Estimated exposures to 2,3,7,8-TCDD (ng/kg blood lipid) of selected study populations

Study	Date(s) or duration of exposure	Date of sampling	No. of workers	Mean measured 2,3,7,8-TCDD blood concentration	Back-calculated to exposure date
Industrial workers					
BASF (Germany) (Ott *et al.*, 1993)	1953 (accident)	1988–92	138	15.4 (geom.)	geom. mean: [~ 400][a] (1008: estimated cumulative concentration at time of exposure in workers with severe chloracne)
Boehringer-Ingelheim (Germany) (Flesch-Janys *et al.*, 1995, 1996a; Kogevinas *et al.*, 1997)	13.1 years Mean of 5.4 years after end of exposure	1985–94	48	84.1 (median)	141 (3–2252) (measured levels for the total cohort)
	Mean of 11.0 years after end of employment		48 (same 48 as above)	48.9 (median)	
USA (Fingerhut *et al.*, 1991a,b)	1987 (15–37 years after employment)		253 (from 2 of 12 plants)	233	~ 2000 (mean) 32 000 (max.)
	> 1 year of exposure		119	418	
Netherlands (Hooiveld *et al.*, 1996a,b)	1955–1985 (factory A)	1993	48	22. 9 (production) (geom.) 87.2 (1963 accident) (geom.)	geom. mean: 286 (17–1160) 1434 (301–3683)
Handling and spraying of 2,4,5-T					
Ranch Hand/ US Viet Nam veterans (Nygren *et al.*, 1988)	Late 1960s	1984–85	9	46.3 (arith.) 15.7 (geom.)	[~ 180][a] [~ 60][a]
Ranch Hand/ US Viet Nam veterans (Wolfe *et al.*, 1990)	Late 1960s	1987	888	12.4 (median)	[~ 50][a]

Table 22 (contd)

Study	Date(s) or duration of exposure	Date of sampling	No. of workers	Mean measured 2,3,7,8-TCDD blood concentration	Back-calculated to exposure date
Handling and spraying of 2,4,5-T (contd)					
New Zealand (Smith *et al.*, 1992a)	1953–88 (mean duration of exposure, 16 years)	1988	9	53.3	~ 300 (in 1970)
Seveso accident					
Mocarelli *et al.* (1990)	1976	1976	19 (zone A)	828–56 000 (range of individuals)	
Landi *et al.* (1996)	1976	[1992–93]	6 (zone A)	61.5 (mean) 71.5 (median)	333.8 388.7
			52 (zone B)	16.8 (mean) 12.5 (median)	111.4 77.6
			52 (outside)	5.3 (mean) 5.5 (median)	

[a] Values in [ ] calculated by the Working Group

(i) *Australia*

Taucher *et al.* (1992) reported PCDD/PCDF data from four sites in Sydney. The levels found in urban areas (0.016–0.062 pg I-TEQ/m^3) were similar to those from the northern hemisphere. There seemed to be a dose similarity with profiles obtained from combustion sources.

(ii) *Austria*

The first systematic approach (Moche & Thanner, 1996a) to monitor ambient air concentrations of PCDDs/PCDFs in Austria was started in 1992. Within and in the vicinity of Vienna, Graz, Linz and Steyregg, 100 samples were taken during a whole year. The values generally ranged in the summer and winter period between 0.022–0.041 and 0.050–0.222 pg I-TEQ/m^3, respectively. The winter PCDD/PCDF values at Graz (south of the city centre on the grounds of a garden nursery) were approximately 0.22 pg I-TEQ/m^3 and were clearly above the levels found at Vienna and Linz.

(iii) *Belgium*

Wevers *et al.* (1992) compared PCDD/PCDF concentrations in air in a vehicle tunnel near Antwerp with local ambient air. The I-TEQ values in tunnel air were two to three times higher than those in ambient (background) air. Wevers *et al.* (1993) also reported values from six sampling locations across Flanders (Belgium) in the neighbourhood of typical emission sources. The values found ranged between 0.018 and 0.379 pg I-TEQ/m^3.

(iv) *Canada*

Measurement of PCDDs/PCDFs in ambient air in south-west Ontario have been presented by Bobet *et al.* (1990) for two sampling stations: downtown Windsor and Walepole Island Indian Reserve. No isomer-specific analyses were reported. Concentrations measured at the Walepole station in 1988 were 4–20 times lower than those in Windsor. In the 13 samples collected in Windsor, the mean level of OCDD was 2.12 pg/m^3 with a maximum of 7.09 pg/m^3. The mean for total HpCDD was 1.19 pg/m^3. At both stations, the majority of the PCDDs detected comprised hepta- and octa-chlorinated congeners. The tetra- and penta-CDDs were not found above detection limit (0.1 pg/m^3) at either station.

Additional data for Ontario were measured in 1988 in Dorset and on Toronto Island by Steer *et al.* (1990a). The highest levels were mainly for OCDD: Toronto Island, 0.3–0.8 pg/m^3; Dorset, 0.1–7 pg/m^3. No other isomer-specific data were reported.

Samples collected in the vicinity of a cement kiln were reported by Reiner *et al.* (1995). The values found were lower than the average values found in air samples taken on Toronto Island. All levels were considerably lower than the Ontario Ministry of Environment and Energy guideline of 5 pg/m^3.

Steer *et al.* (1990b) reported elevated PCDD and PCDF air levels in connection with a fire at a disposal site containing some 14 million tyres. Sampling sites were changed as required to provide samples near the fire (1 km downwind) and samples at the limit of

the evacuation zone (3 km downwind). The highest total I-TEQ of 2.5 pg/m^3 measured represents 50% of the provincial interim guideline of 5 pg/m^3 (annual average).

(v) *Germany*

Bruckmann and Hackhe (1987) reported 21 ambient air measurements in Hamburg. The sampling was performed at sites with wide differences in air quality (e.g., at a dump site, residential area, industrial area, highway tunnel, close to a highway, vicinity of Hamburg). The values were between < 0.1 pg and 2.2 pg/m^3 expressed in German TEQs. The main sources of the PCDDs/PCDFs detected were thermal processes (industry, traffic and probably home heating facilities). Using more sophisticated analytical techniques, Rappe *et al.* (1988) analysed 13 Hamburg samples not only for the 2,3,7,8-substituted isomers but also for all single isomers separated on SP 2331 GC.

Between 1985 and 1986, 18 sites in North-Rhine-Westphalia were sampled to quantify PCDDs/PCDFs in ambient air (Kirschmer, 1987). For the Rhine-Ruhr area, mean values for total PCDDs and PCDFs at 11 sampling sites were 3.2 pg/m^3 and 5.5 pg/m^3, respectively. 2,3,7,8-TCDD was not detected in any sample (detection limit, 0.1 pg/m^3).

Christmann *et al.* (1989b) analysed 22 air samples originating from Berlin, Gelsenkirchen and Recklinghausen. Usually, PCDD/PCDF levels were in the lower pg/m^3 (0.02–0.4 pg German TEQ/m^3). In one indoor air sample, the TEQ value was increased, due to the application of PCP as wood preservative, up to 2.6 pg/m^3.

Measurements in kindergartens with PCP-treated wood have been reported by Päpke *et al.* (1989a). The mean of 15 measurements was 0.696 pg German TEQ/m^3 (range, 0.018–2.46).

In 1990, at six sites located in Hessen, average ambient air PCDD/PCDF concentrations (calculated as I-TEQ) of between 0.048 pg/m^3 (rural reference site) and 0.146 pg/m^3 (industrial sites combined) were determined (König *et al.*, 1993). At five locations, a distinct annual cycle of PCDD/PCDF concentrations was observed, with relatively low concentrations in summer and increasing concentrations towards the winter months. On the other hand, the ratio of total PCDF to total PCDD levels showed a maximum in summer because of a comparatively larger decrease in PCDD towards the summer months. The annual average distribution of PCDD/PCDF homologue groups was similar at all six sites. The contributions of PCDF homologues to the total tetra- to octa-CDD/CDF content decreased with increasing degree of chlorination, whereas that of PCDD homologues increased with increasing degree of chlorination. For each homologous group, the contribution of congeners with a 2,3,7,8-chlorine substitution pattern was similar.

Towara *et al.* (1993) analysed the distribution of airborne PCDDs/PCDFs in relation to particle size. Particle sizes from < 0.15 to > 4.05 μm were grouped into five categories; the patterns of PCDDs/PCDFs detected were very similar for all five categories. Hence, particle-mediated transport and deposition should be similar for all PCDD/PCDFs.

Data on PCDDs/PCDFs in ambient air have recently been reported by Wallenhorst *et al.* (1995) for Baden-Württemberg. In rural areas, total concentrations were 0.015–0.020 pg I-TEQ/m^3. In urban areas, the concentrations were 0.07–0.08 pg I-TEQ/m^3. PCDD/PCDF concentrations in total air decreased from city centre to suburban areas and the surrounding areas.

Annual mean concentrations of PCDDs/PCDFs in the ambient air of four cities in North-Rhine-Westphalia were measured at the same locations in 1987–88 and again in 1993–94 (Hiester *et al.*, 1995). Over this period, the annual average PCDD/PCDF concentrations decreased from 0.130 to 0.040 pg I-TEQ/m^3 in Cologne (69%), from 0.332 to 0.124 pg I-TEQ/m^3 in Duisburg (63%), from 0.204 to 0.076 pg I-TEQ/m^3 in Essen (63%) and from 0.224 to 0.120 pg I-TEQ/m^3 in Dortmund (46%). This decrease can be related to actions taken since 1989.

A similar situation has been shown for Hamburg by Friesel *et al.* (1996). Monitoring of ambient air and deposition demonstrated decreases of about 70% and 20% respectively in PCDD/PCDF concentrations in air (pg I-TEQ/m^3) and deposition flux (pg I-TEQ/m^2 per day). Ambient air concentrations decreased from 0.108 in 1990 and 0.037 in 1993 to 0.036 in 1995; deposition decreased from 16 in 1990 to 11 in 1993 but rose to 13 in 1995. The fact that the reduction in ambient air concentrations has been much greater than the reduction in emissions may be attributed to long-range transport and to smaller diffuse sources not yet identified.

The atmospheric levels of PCDDs/PCDFs in both the gas and particle phase were measured continuously over one year (1992–93) at seven sites on the outskirts of Augsburg (Hippelein *et al.*, 1996; Swerev *et al.*, 1996) and at a rural site 15 km from the city. The PCDD/PCDF levels were about two times higher at the sampling points on the edge of the city than at the remote location. There was a pronounced temporal variability in total air concentrations, levels in winter being nine times higher than those in summer. The gas/particle partitioning was characterized by higher gaseous fraction for more volatile compounds and higher ambient temperatures. It was concluded that the ambient air concentrations of the PCDDs/PCDFs are determined by local and regional emissions, with seasonal (winter) sources contributing the vast majority of the emission fluxes.

Rabl *et al.* (1996) reported PCDD/PCDF measurements in relation to distance of 1.3, 2.0 and 3.3 km from a supposed local major emission source in Bavaria (a municipal waste incinerator at Schwandorf). The PCDD/PCDF concentrations were quite similar at each distance. At emission concentrations of 1–10 pg I-TEQ/m^3 from the incinerator, no influence of the waste incineration plant could be found.

(vi) *Japan*

Isomer-specific analyses of PCDD/PCDFs and other chlorinated components in ambient air have been reported by Sugita *et al.* (1993, 1994). The concentrations of PCDDs/PCDFs were higher in winter than in summer. Ranges were 0.469–1.427 pg I-TEQ/m^3 in summer and 0.294–2.990 pg I-TEQ/m^3 in winter. Coplanar and mono-*ortho* PCBs were present at concentrations about 10 times higher than those of 2,3,7,8-substituted PCDDs/PCDFs. PCDFs accounted for about 70% of the total toxic equivalents, PCDDs for 20% and the sum of both types of PCBs for 10%.

Similar findings have been presented by Kurokawa *et al.* (1994). At three different sites, values were highest in winter. Coplanar PCBs contributed about 11% to the total TEQ. In winter, most of the PCDDs/PCDFs were found in the particle phase.

(vii) *The Netherlands*

van Jaarsveld and Schutter (1993) estimated (air calculation models) emissions of PCDDs/PCDFs to the atmosphere in various parts of north-western Europe on the basis of emission factors combined with production quantities. Atmospheric residence times were calculated for different particle sizes. Small particles, carrying more than 50% of total PCDDs/PCDFs, have residence times of the order of three days, while very large particles (diameter, > 20 μm) reside only a few hours in the atmosphere. Comparison of predicted PCDD/PCDF levels in ambient air with available measurements showed fairly good agreement.

Ambient air measurements of PCDDs/PCDFs in The Netherlands have been performed by Bolt and de Jong (1993). Particulate-bound PCDD/PCDF levels in air around a municipal waste incinerator ranged between 0.015 ± 0.005 and 0.125 ± 0.025 pg I-TEQ/m^3. Congener profiles were very similar in all wind directions and were well correlated with the incinerator emissions.

(viii) *Norway*

Oehme *et al.* (1991) reported on the determination of PCDDs/PCDFs in air at the inlet and outlet of a longitudinally ventilated tunnel with separate tubes for each traffic direction. The varying percentage of heavy-duty vehicles made it possible to differentiate between factors for light-duty vehicles (LDVs) and heavy-duty (diesel) vehicles (HDDVs). Depending on driving conditions, the estimated emission factors were of the order of 0.04–0.5 ng Nordic TEQ/km for LDVs and 0.8–9.5 ng/km for HDDVs. On a volume basis, the values ranged between 0.097 and 0.98 pg Nordic TEQ/m^3. A typical ambient air concentration for central Oslo was 0.04 pg Nordic TEQ/m^3.

First measurements for PCDDs/PCDFs in Arctic air have been reported recently by Schlabach *et al.* (1996). The sampling was performed in spring and summer of 1995 in Spitzbergen. The values of two samples were 0.0023 and 0.0011 pg I-TEQ/m^3.

(ix) *Poland*

The first results for PCDDs/PCDFs in ambient air were published by Grochowalski *et al.* (1995). Two measurements performed in winter 1995 in Cracow — a heavily industrialized city — gave values of 0.95 pg I-TEQ/m^3 (market square) and 11.95 pg I-TEQ/m^3 (crossroads), respectively. OCDD and OCDF were reported at 280 and 220 pg/m^3, respectively.

(x) *Russian Federation*

Khamitov and Maystrenko (1994) reported ambient air levels, sampled in Ufa, of between 0.2 and 0.5 pg I-TEQ/m^3. Levels of isomers were measured by Kruglov *et al.* (1996) in two air samples originating from an industrial city during an accidental oil fire.

(xi) *Slovakia*

As part of the TOCOEN (Toxic Compounds in the Environment) project, Holoubek *et al.* (1991) reported I-TEQ values in ambient air from former Czechoslovakia. The samples — collected in industrial and urban areas in 1990 — showed a wide range of PCDD/PCDF contamination, from none detected to 6.3 pg I-TEQ/m^3.

(xii) *Spain*

Ambient air samples associated with municipal waste incinerators, chemical industry, traffic and urban air ranged between 0.05 and 0.55 pg I-TEQ/m^3. Surprisingly high levels of OCDF (126.8 pg/m^3) were found near a municipal waste incinerator, while OCDD was reported at only 5.7 pg/m^3. Levels of highly chlorinated PCDDs and PCDFs correlated poorly (Abad *et al.*, 1996).

(xiii) *Sweden*

Rappe *et al.* (1989a) analysed nine ambient air samples taken in winter in 1986 in Rörvik and in Gothenburg under various atmospheric conditions. The results demonstrated that the contribution to the total *air* burden in Gothenburg from local sources during periods with good ventilation seems to be of secondary importance. The isomeric patterns of PCDDs/PCDFs among all samples were very similar. There was also a striking similarity in the isomeric pattern for TCDFs and PeCDFs in the particulate samples, automobile exhaust and emissions from municipal waste incinerators. The results strongly indicated that the general background of PCDDs and PCDFs in airborne particulates has its origin in various types of incineration processes, including automobile exhaust.

(xiv) *United Kingdom*

Data on PCDD/PCDF air concentrations have been monitored in three major cities (London, Manchester and Cardiff) and a busy industrial town (Stevenage) from the beginning of 1991 to the end of 1992 (Clayton *et al.*, 1993). Levels of total 2,3,7,8-substituted PCDDs and PCDFs, other isomers and the total I-TEQ for the four sites have been reported. Concentrations of the 2,3,7,8-substituted PCDDs are mainly associated with the HpCDDs and OCDD, the OCDD accounting for over 76% of the total 2,3,7,8-substituted PCDDs. The tetra-, penta- and hexa-chlorinated congeners were generally below detection limits. Higher values were found in winter than in summer. The mean values for about 43 single measurements for each site were between 0.039 and 0.102 pg I-TEQ/m^3.

Dyke and Coleman (1995) reported PCDD/PCDF measurements before, during and after 'bonfire night' at Oxford. Bonfire night (5 November) is an annual event during which it is customary in England to set off fireworks and light bonfires. An increase in PCDD/PCDF concentrations by approximately a factor of four occurred during the period of bonfire night.

(xv) *United States*

The results of a baseline study on PCDDs/PCDFs in the ambient atmosphere have been published (Eitzer & Hites, 1989). Between 1985 and 1987, 55 samples were taken

at three sites in Bloomington, IN, and a set of samples was also taken in the Trout Lake, WI, area, a much more rural area than Bloomington. In ambient air, consistency was seen in the isomer pattern within each group of congeners with the same number of chlorine substituents, but overall levels of the various groups had somewhat more variation. Some of this variation was related to the atmospheric temperatures, with more of the lower chlorinated congeners found in the vapour phase at higher temperatures. No isomer-specific concentrations (except for OCDD) were reported. The mean OCDD values for Bloomington and the Trout Lake area were 0.89 pg/m^3 and 0.16 pg/m^3, respectively.

Smith *et al.* (1989, 1990a,b), analysed downwind and upwind ambient air samples around the industrial area of Niagara Falls, for PCDDs/PCDFs. One location, predominantly downwind of Niagara Falls, showed a variety of patterns and a wide concentration range of PCDDs/PCDFs (0.07–53 pg Eadon TEQ/m^3). The upwind 'control' location results showed lower concentrations of PCDDs and PCDFs and less variable patterns. In nearly all samples, HpCDDs and OCDD were found at ranges from not detected to 5.43 pg/m^3 and from 1.36 to 8.88 pg/m^3, respectively.

PCDDs and PCDFs were measured in ambient air samples collected in Ohio in 1987 by Edgerton *et al.* (1989). No 2,3,7,8-TCDD was detected in any of the samples at a detection limit of less than 0.24 pg/m^3. Using a chemical mass balance model applied to PCDD/PCDF congener group profiles, major potential sources of these compounds to the atmosphere in Ohio were determined to be municipal solid waste and sewage sludge combustion plants.

Hahn *et al.* (1989) reported on pre-operational background ambient air levels of PCDDs/PCDFs around two modern municipal waste incinerators. In addition, workplace air levels inside one facility (Hillsborough County, FL) were measured to evaluate the potential exposure of employees. The levels of PCDDs/PCDFs and heavy metals were similar to ambient levels outside the facility before the facility began operation.

The concentrations of PCDDs and PCDFs in ambient air of several sites in metropolitan Dayton, OH, have been determined (Tiernan *et al.*, 1989). Total PCDDs detected in industrial areas ranged from 1.6 to 11.2 pg/m^3, and the corresponding total PCDFs ranged from 0.62 to 11.7 pg/m^3. No PCDDs or PCDFs were found in rural regions, typical average detection limits being on the order of 0.02–0.17 pg/m^3. Approximately 50% of the total PCDD/PCDFs in the ambient air samples collected consisted of 2,3,7,8-chlorine-substituted congeners.

Airborne concentrations of PCDDs/PCDFs in office buildings and in corresponding ambient air in Boston, MA, were measured by Kominsky and Kwoka (1989). Twelve of the 16 samples were collected inside the buildings and four were collected at the ambient air intake plenums of the buildings. The distribution of total PCDD congeners was quite similar to corresponding surface wipe samples. In all 20 samples, it was possible to detect OCDD.

PCDD/PCDF concentrations in ambient air in winter at Bridgeport, the largest city in Connecticut, have been analysed by Hunt and Maisel (1990). The Connecticut Department of Environmental Protection has proposed an ambient air quality standard for PCDDs/PCDFs expressed in 2,3,7,8-TCDD equivalents on an annualized basis of

1.0 pg/m^3. In order to ensure that this standard is satisfied, it is required that ambient monitoring for each of the 2,3,7,8-substituted PCDD/PCDF isomers be conducted in the vicinity of each municipal solid-waste incinerator on both a pre-operational and post-operational basis. The report described the pre-operational measurements in the autumn and winter of 1987–88 in the vicinity of a municipal solid-waste incinerator. The TCDFs were the major PCDF class found. 1,2,3,4,6,7,8-HCDF was the predominant PCDF of toxicological significance. HxCDDs, HpCDDs and OCDD were the predominant PCDD congeners, and 1,2,3,4,6,7,8-HpCDD the predominant PCDD isomer of toxicological significance. In winter months, PCDDs/PCDFs are almost exclusively particulate-associated. Comparison of pre- and post-operational ambient PCDD/PCDF concentrations in the vicinity of the incinerator during winter showed similar levels and profiles for both periods.

Maisel and Hunt (1990) presented data on PCDDs/PCDFs in a 'typical' ambient air sample from Los Angeles, CA, including isomer-specific concentrations. Ambient PCDD/PCDF burdens for a Connecticut coastal location (n = 27, urban, winter), a southern Californian location (n = 34, urban) and a central Minnesota location (n = 16, rural) were 0.092, 0.091 and 0.021 pg I-TEQ/m^3, respectively.

A comprehensive programme for measurement of airborne toxic agents, designed to establish baseline concentrations of atmospheric PCDDs/PCDFs in the South Coast Air Basin (California), involved nine sampling sessions between December 1987 and March 1989 at eight different locations (Hunt & Maisel, 1992). The PCDD/PCDF congener profiles from most of the sample examined strongly suggested the influence of combustion sources. 1,2,3,4,6,7,8-HpCDD was the most prevalent PCDD after OCDD. The most toxic congener, 2,3,7,8-TCDD, was below the 10–20 fg/m^3 detection limit for most of the ambient air samples.

Ten years after the Binghamton State Office Building transformer incident in 1981 (see PCDF monograph, Section 1.2.1(*b*)(viii)), Schecter and Charles (1991) presented data on PCDDs/PCDFs in the air. In 1981–82, airborne samples contained levels of 352 pg Eadon TEQ/m^3 at the transformer site. In 1986, nine years into clean up, levels of 74 pg/m^3 TEQ were recorded. The guidelines for reoccupancy of the building were reported to be 14 pg TEQ/m^3 for renovation workers and 10 pg TEQ/m^3 for office workers.

The levels of airborne PCDDs/PCDFs in the area of the Columbus Municipal Waste-to-Energy facility in Columbus, OH, were reported by Lorber *et al.* (1996a). This facility, the largest single source of PCDDs/PCDFs identified in the literature (1992, 984 g I-TEQ per year; 1994, 267 g I-TEQ per year), ceased operation in December 1994. Air concentrations in the city were higher in 1994 when the facility was operating than after it shut down: in March and April 1994, the levels were 0.067 and 0.118 pg I-TEQ/m^3, respectively, compared with 0.049 pg I-TEQ/m^3 in June 1995 after the facility had closed.

Also in Ohio, air in the vicinity of the Montgomery County north and south municipal waste incinerators was analysed for PCDD/PCDFs (Riggs *et al.*, 1996). PCDD levels in September 1995 were similar to those in the same area in 1988, but PCDF levels were

much lower. At one site, the TEQ of PCDDs/PCDFs in ambient air was about a factor of 10 lower in 1995 than in 1988.

(*b*) *Water* (see Appendix 1, Table 2)

Because of the complex cycling and partitioning of PCDDs/PCDFs in the aquatic environment and the difficulty of analysis, adequate sampling and analysis methods have become available only over the past decade.

PCDDs and PCDFs are highly lipophilic and thus have an affinity for particulate organic carbon (Webster *et al.*, 1986). Knowledge of their partitioning between the sedimentary, colloidal, dissolved, organic and even vapour phases remains limited, largely because of the difficulty of measuring these compounds in solution.

PCDDs partition between particulate and dissolved fractions. Material that passes through a filter of 0.45 μm or 0.2 μm pore size is commonly defined as dissolved. The major problem with this definition is that colloidal particles (suspended solid particles of < 0.2 μm diameter) and other macromolecules pass through such filters and are included in the 'dissolved' phase. However, colloids and 'dissolved' organics may function as 'small particles' in terms of the mechanisms of PCDD/PCDF uptake by biological systems (Broman *et al.*, 1991, 1992).

(i) *Canada*

A survey of drinking-water supplies in the Province of Ontario was initiated in 1983 to determine the extent of their contamination with PCDDs/PCDFs (Jobb *et al.*, 1990). A total of 49 water supplies were examined. Water supplies in the vicinity of chemical industries and pulp and paper mills were sampled up to 20 times. Detection limits were in the low ppq (pg/L) range for all tetra- to octa-CDDs/CDFs. From 399 raw and treated water samples, only 37 positive results were reported. OCDD was detected in 36 of these samples, at values of 9–175 pg/L. 2,3,7,8-TCDD was not detected in any sample.

Muir *et al.* (1995) analysed water and other matrices downstream from a bleached kraft pulp mill on the Athabasca River (Alberta) in 1992. The 'dissolved phase' and the suspended particulates from continuous centrifuged samples were analysed for 41 PCDDs and PCDFs ranging from mono- to octachloro-substituted congeners. Most PCDD congeners (including 2,3,7,8-TCDD) were undetectable (< 0.1 pg/L) in the centrifugate.

(ii) *Finland*

High concentrations (70–140 μg/L) of total chlorophenols were found in drinking-water and 56 000–190 000 μg/L in ground water close to a sawmill in southern Finland. No data on PCDDs/PCDFs were reported. Due to their presence in commercial chlorinated phenols used, exposure of the population to PCDDs/PCDFs could not be excluded. No increased PCDD/PCDF concentrations were found in milk samples from mothers who had used the contaminated water (Lampi *et al.*, 1990).

(iii) *Germany*

Götz *et al.* (1994) reported an analysis of dissolved and particle-bound PCDDs/-PCDFs in the River Elbe. Samples upstream of Hamburg (Bunthaus) showed total values of 3.15 pg I-TEQ, while downstream in Blankenese values of 1.21 pg I-TEQ were found. More than 98% of the I-TEQ concentration in the water was particle-bound.

(iv) *Japan*

Seawater from Japanese coastal areas has been analysed. Adequate detection limits were achieved by use of a pre-concentration system, applied to 2000 L coastal seawater, resulting in the detection of almost all 2,3,7,8-substituted PCDDs and PCDFs. Values between < 5 and 560 fg/L were found (Matsumura *et al.*, 1994, 1995).

Hashimoto *et al.* (1995a) reported data on two seawater samples taken in 1990 near Matsuyama and Misaki. Only hepta- and octa-CDDs were detected (0.1–2.5 pg/L) but no PCDFs.

Drinking-water samples including home tap-water and well-water collected in 1991 in Shiga and Osaka Prefectures were analysed for PCDDs/PCDFs. The levels were in the low ppq (pg/L) or less in all samples analysed. The total daily intake of PCDDs/PCDFs via drinking-water was estimated to be only 0.00086–0.015% of that via food (Miyata *et al.*, 1992, 1993).

(v) *New Zealand*

A national Organochlorine Programme, including monitoring for PCDDs/PCDFs in water, was initiated in 1995. Samples were collected from 13 rivers at 16 sites during the period January to March 1996. No PCDDs/PCDFs were detected in any of the 16 composite samples. Limits of detection for the tetra-, penta- and hexa-chlorinated congeners were typically 1 pg/L or less. The I-TEQs were in the range of 0.25–2.4 pg/L, with a mean of 0.97 pg/L. The approximate 10-fold range of TEQ values determined for these samples is a result of variation in the detection limits in the analyses rather than any inherent differences in the samples. In calculating TEQs, half the detection limits were taken for non-detectable congeners (Buckland *et al.*, 1996).

(vi) *Russian Federation*

Ignatieva *et al.* (1993) found a level of 8.0 pg/L of total PCDDs in water from the Angara River, the only river that emerges from Lake Baikal.

Data on PCDD/PCDF concentrations in various river water samples were reported by Khamitov and Maystrenko (1995): Belaja River, 1.7–6.0 pg/L; Ufa River, 0.6–1 pg/L ; Inzer River, 1.8 pg/L; Zilim River, 0.2 pg/L. In contrast, drinking-water from the Ufa River was reported to be contaminated in 1990 with 0.5–1.0 pg/L. [It was not apparent from the paper whether the values were I-TEQs or total PCDDs/PCDFs].

Smirnov *et al.* (1996) reported values for PCDDs/PCDFs in the Ufa River at totals of 0.13–0.20 µg/L. 'Emergency' situations, where the concentration of PCDDs/PCDFs in river or tap-water exceeds the 'permissible' level (0.02 ng/L) by 10 to 100 times were stated to occur on a regular basis.

Fedorov (1993) reported on levels of PCDDs/PCDFs in various matrices (soil, sediments, air and water) near or at sites of some Russian chemical plants. In the spring of 1991, the drinking-water at Ufa, after cleaning, was found to be contaminated with 0.14 ng/L 1,2,3,6,7,8-HxCDD. Especially high concentrations of 28–167 pg/L 2,3,7,8-TCDD were measured in April 1992 in samples of Ufa drinking-water from four different water sources.

(vii) *Spain*

Sludge from drinking-water treatment plants was analysed by Rivera *et al.* (1995). Because of the high level of pollution in water from the Llobregat River, a combination of several treatment processes, including prechlorination, activated carbon and post-chlorination, is applied. The sludge from two treatment plants has been reported to be contaminated at levels of 5.59 and 2.74 ng I-TEQ/kg, respectively.

(viii) *Sweden*

Rappe *et al.* (1989b, 1990a) analysed surface and drinking-water with very low detection limits. At very low detection limits, most 2,3,7,8-substituted PCDDs and PCDFs were detected in all samples. 2,3,7,8-TCDD was found in most water samples, but at extremely low levels, between 0.0031 pg/L for river water and 0.0005 pg/L for drinking-water. This is at or below the lower level in the United States Environmental Protection Agency guideline of 0.0013 pg/L for this compound.

(ix) *United States*

Results of a survey conducted in 1986 for PCDDs/PCDFs and other pollutants in finished water systems throughout New York State were described by Meyer *et al.* (1989). The finding of OCDD is unsure, as it has been detected at 1 pg/L in blanks. Except for a trace of OCDF detected in one location, no other PCDDs or PCDFs were detected in any of 19 other community water systems surveyed.

Storm-water samples were collected from two outfalls that discharge into San Francisco Bay, CA, and represent sources of run-off from areas with different dominant land uses. The samples from the outfall located close to Oakland had higher values (mean, 21 pg I-TEQ/L) than samples from the city of Benicia located at the north of San Francisco Bay (mean, 3.5 pg I-TEQ/L) (Paustenbach *et al.*, 1996).

(*c*) *Soil* (see Appendix 1, Table 3)

(i) *Australia*

In 1990, surface soil samples from the Melbourne metropolitan area were analysed for PCDDs/PCDFs and other compounds (Sund *et al.*, 1993). A surface sample from Werribee Farm treatment complex paddock, where cattle graze on land that is used for filtration of sewage, contained the highest concentration (520 ng I-TEQ/kg). Samples of other origins contained between 0.09 and 8.2 ng I-TEQ/kg. Detection limits ranged from 0.08 to 24 ng/kg.

Levels of PCDDs/PCDFs in soil samples from conservation areas following bush fires have been reported by Buckland *et al.* (1994). Samples were collected (2 cm deep)

six weeks after the fires were extinguished. The total PCDD/PCDF levels in the unburned conservation area samples were 1.1, 7.2 and 7.7 μg/kg (3.1, 8.7 and 10.0 ng I-TEQ/kg). The total PCDD/PCDF levels in the burnt conservation area samples were 1.3, 2.0 and 27.8 μg/kg (2.2, 3.0 and 36.8 ng I-TEQ/kg). The total PCDDs/PCDFs in the single Sydney metropolitan area sample was 22.3 μg/kg (42.6 ng I-TEQ/kg). The results of this limited survey indicate that bush fires have not had a major impact on the levels of PCDDs/PCDFs in conservation area soil.

(ii) *Austria*

Around a densely populated industrial urban area (Linz), soils from grassland and forest areas were analysed for PCDDs/PCDFs and PCBs (Weiss *et al.*, 1993, 1994). The concentrations of PCDDs/PCDFs were higher in soils near chemical and/or steel plants. The highest PCDD/PCDF concentration in grassland soil (I-TEQ, 14.4 ng/kg) was measured near a hospital refuse incineration plant.

Riss *et al.* (1990) compared PCDD/PCDF levels in soil, grass, cow's milk (see Section 1.3.2(*d*)(i)), human blood and spruce needles in an area of PCDD/PCDF contamination through emissions from a metal reclamation plant (Brixlegg, Tyrol). Soil concentrations were highest near the plant and in the main wind direction, and decreased in all directions with increasing distance. The highest concentration found corresponds to 420 ng German TEQ/kg. The level of 40 ng/kg TEQ (which has been proposed as an upper limit for agricultural use) was exceeded in an area reaching 600 m from the plant in the main wind directions. Milk samples from farms with meadows in the contaminated area contained TEQ levels of PCDDs/PCDFs ranging from 13.5 to 37.0 ng/kg fat, which were considerably higher than that of 3.6 ng/kg in the control sample from Kossen/Tyrol. About 50% of the TEQ of the contaminated milk was accounted for by 2,3,7,8-TCDD (normally about 10%). Because of the elevated PCDD/PCDF levels in cow's milk, blood plasma from two farmers who drank milk from their own cows was analysed for PCDDs/PCDFs. One had significantly elevated blood levels (see **Table 25**).

Determinations of PCDDs and PCDFs in soil samples from rural, urban and industrial sites in Salzburg state (Boos *et al.*, 1992) gave I-TEQ values generally in the low ng/kg range. Six samples contained PCDDs/PCDFs. Levels exceeding 5 ng German TEQ/kg, above which limit the cultivation of certain vegetables is restricted, but none exceeded 40 ng TEQ/kg, which would exclude the cultivation of any plants. Including in the calculation undetected congeners at 50% of the detection limit would lead to seven samples exceeding the lower limit. The lowest level found was 0.1 ng TEQ/kg.

(iii) *Belgium*

Topsoil samples were collected at six locations in Flanders, and analysed isomer-specifically [not in tables reported] for PCDDs/PCDFs (Van Cleuvenbergen *et al.*, 1993). Concentrations in the 0–3-cm soil fraction, averaged for each location, ranged from 2.1 ng/kg at a rural location to 8.9 ng/g (both as I-TEQ) in an industrialized area.

(iv) *Brazil*

Krauss *et al.* (1995) analysed PCDDs/PCDFs and PCBs in forest soils from Brazil. The I-TEQ values found in four areas are presented in Appendix 1 (Table 4). In the Amazon basin the PCDD/PCDF concentrations of soil were close to the detection limits. In industrial regions around Rio de Janeiro and São Paulo, highly contaminated soils with I-TEQ values between 11 and 654 ng/kg were found.

(v) *Canada*

Soil in the vicinity of a large municipal refuse incinerator was analysed for PCDDs/PCDFs. Fourteen locations, which included three control sites, were sampled in 1983 (McLaughlin *et al.*, 1989; Pearson *et al.*, 1990). In nearly all samples, OCDD was detected. The measurements were not isomer-specific. As expressed by the authors, the results showed that PCDDs/PCDFs emitted from the incinerator (Suaru, Hamilton) since 1973 had not accumulated in surface soil in the vicinity of the plant (McLaughlin *et al.*, 1989). Concentrations of PCDDs/PCDFs in soils near refuse and sewage sludge incinerators and in remote rural and urban locations (no 2,3,7,8-substituted isomers, exclusively OCDD and in some cases OCDF) were measured. In nearly all cases, total hepta-CDD and OCDD were most abundant. No evidence of any source-related airborne deposition of PCDDs/PCDFs in the soil around a sewage sludge incinerator in Scarborough was identified.

In 97 soil samples analysed for PCDDs/PCDFs, no clear connection between emission sources and levels of these components in soil or food was found by Birmingham (1990). Levels, patterns and quantities of I-TEQ in soils from various sources were analysed. The mean and standard deviation for I-TEQ, expressed in ng/kg, were: rural soils (n = 30), 0.4 ± 0.6; urban soils (n = 47), 11.3 ± 21.8; industrial soils (n = 20), 40.8 ± 33.1. Using the worst-case scenario, an infant consuming urban soil containing the mean plus three standard deviations (77 ng I-TEQ/kg) would ingest less than one tenth of the tolerable daily intake (see Section 1.5).

In 1989, soils were sampled in areas of cleared forest in New Brunswick, where 2,4-D/2,4,5-T herbicide was applied in one or more application at 3–10 lbs/acre (3.3–11 kg/ha) between 1956 and 1965. Residues of 2,3,7,8-TCDD were detected at up to 20 ng/kg in the upper 5 cm of soil. Residues were also found at greater depths at lower concentrations at one test site. 2,3,7,8-TCDF was not found in soil samples from sprayed or unsprayed areas (Hallett & Kornelsen, 1992).

(vi) *China*

Wu *et al.* (1995) published results for two soil samples, originating from the Ya-Er lake area where chemical plants which produce chlorine compounds have discharged much waste water into the sea. The two samples contained hexachlorobenzene at levels of 35 and 38 mg/kg, respectively, but only very low levels of PCDDs/PCDFs. Sediment samples originating from the same area contained maximal levels of 136.6 μg OCDD/kg and 797 ng I-TEQ/kg.

(vii) *Czech Republic*

2,3,7,8-TCDD contamination was measured in more than 100 soil samples (0–20 cm depth) from a factory that produced mainly sodium 2,4,5-trichlorophenoxyacetate, sodium pentachlorophenolate, PCP and TCP, starting in 1965 (Zemek & Kocan, 1991). 2,3,7,8-TCDD levels ranged from undetectable to 29.8 μg/kg. Samples from residential and agricultural areas in Neratovice showed levels between undetectable and 100 ng/kg. Soil samples collected at a distance of 50–80 m from the plant contained levels between undetectable and 60 ng/kg 2,3,7,8-TCDD.

(viii) *Finland*

Soils at sites where wood preservatives had been used (Sandell & Tuominen, 1993) contained high levels of PCDDs/PCDFs. The dominant congeners were 1,2,3,6,7,8-HxCDD, 1,2,3,4,6,7,8-HpCDD, OCDD, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 2,3,4,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDF and OCDF. In the surface layers (0–20 cm), I-TEQ values ranging from 1.7–85 μg/kg were found.

Assmuth and Vartiainen (1995) reported on further soil samples from sites where wood preservatives like 2,3,4,6-tetrachlorophenol, PCP and 2,4,6-TCP have been used. High concentrations (maximum value, 90 μg I-TEQ/kg; mean, 19 μg I-TEQ/kg) were found. PCDDs/PCDFs were distributed heterogeneously between soil layers, the concentrations in topsoil samples being generally lower. Concentrations of PCDDs/PCDFs were unrelated to the chlorophenol contents in the soil samples. Hexa-, hepta- and octa-CDFs were the dominant congeners in concentration.

(ix) *Germany*

In soils contaminated with motor oils and re-refined oils, Rotard *et al.* (1987) found PCDDs at μg/kg levels. No PCDFs were detected.

An analytical programme to detect contaminants in soil samples at the site of a herbicide plant in Hamburg that closed in 1984 was presented by Schlesing (1989) and Jürgens and Roth (1989). In total, 2652 soil samples deriving from 196 borings were investigated. The highest value reported (presumably a production residue stored on site) reached at a depth of 2 m was 0.7 mg 2,3,7,8-TCDD/kg. Even at depths of 7 and 9 m, values for 2,3,7,8-TCDD of 391 and 41.8 μg/kg, respectively, were found.

The fate of polychlorinated aromatics was studied in the vicinity of a former copper smelter (Rastatt) and a former cable pyrolysis plant (Maulach) that both closed in 1986. Contamination with PCDDs/PCDFs from stack emissions in samples taken within a radius of 100 m from the source at 0–30 cm in depth reached 29 μg I-TEQ/kg soil (Hagenmaier *et al.*, 1992; She & Hagenmaier, 1996). Comparing homologue and isomer concentrations of samples collected in 1981, 1987 and 1989 (same sites, same depths, same sampling method), no significant difference in either homologue pattern or isomer pattern could be detected. Vertical migration of PCDDs/PCDFs in highly contaminated soil is very slow and more than 90% of the compounds were found in the top 10 cm three years after the sources of emission were closed. Within the limits of analytical accuracy (± 20%), there was no indication of appreciable loss of PCDDs/PCDFs by vertical

migration, evaporation or decomposition over a period of eight years, which underlines the persistence of these compounds in the soil.

The distribution of PCDDs/PCDFs in soil around a hazardous waste incinerator at Schwabach was analysed by Deister and Pommer (1991). The highest values were 20.7 and 4.4 ng German TEQ/kg (350 m from the chimney) at depths of 0–2 cm and 0–30 cm, respectively.

Unger and Prinz (1991) found that levels of PCDD/PCDF contamination of soils decreased with increasing distance from a road. The highest values were 23–44.8 ng I-TEQ/kg between 0.1 and 1 m from the road. The number of cars per day on a certain road influenced the PCDD/PCDF levels found.

The finding of PCDDs and especially PCDFs in surface gravel of playgrounds and sports fields (*Kieselrot*) is described in Section 1.3.2(*c*) of the monograph on PCDFs in this volume (see also **Table 25** in this monograph).

Concurrence of farmers' and municipal interests has led to the widespread use of sewage sludge fertilization in West Germany. In 1988, 608 thousand tonnes (dry weight) of sewage sludge, 25% of the total German production, was spread on 360 000 hectares of farmland (McLachlan & Hutzinger, 1990). A survey of 43 samples from 28 German waste-water treatment plants found PCDD/PCDF concentrations between 28 and 1560 ng German TEQ/kg dry weight (Hagenmaier *et al.*, 1985). This compares with the German recommendation for unlimited agricultural use of soil of 5 ng TEQ/kg. McLachlan and Reissinger (1990) analysed soil samples from north-eastern Bavaria with different sludge fertilization histories. The relationship observed between the length of sewage sludge use and PCDD/PCDF concentrations showed clearly that PCDDs/PCDFs accumulate in the soil. The TEQ level was 4.5 times higher in soil that had been fertilized for the previous 10 years than in soil that had had no sludge fertilization. It was 11 times higher in field soil and 18 times higher in meadow soil that had been fertilized for 30 years. The homologue pattern of the fertilized soils lay between that of unfertilized soil and that of the sludge. Similar results on the influence of sewage sludge on PCDD/PCDF levels in soil have been found by Hembrock-Heger (1990) and Albrecht *et al.* (1993).

On the other hand, as observed also in air and food, PCDD/PCDF levels in sewage sludge are generally declining. Ilic *et al.* (1994) found mean I-TEQ values in sewage sludge of 39 ng/kg (dry matter) in 1991 and 28 ng/kg (dry matter) in 1992.

Fürst *et al.* (1993) studied PCDDs/PCDFs in cow's milk in relation to their levels in grass and soil. The contamination levels in soil did not influence the PCDD/PCDF levels in cow's milk, as had been suspected. Even soil levels of up to 30 ng I-TEQ/kg were not associated with elevated PCDD/PCDF levels in cow's milk. However, increasing levels in grass were associated with slightly higher levels in milk.

Levels of PCDDs/PCDFs in soil and atmospheric deposition in an agricultural area in the south-east of Hamburg, adjacent to an industrial area, were measured (Sievers *et al.*, 1993). Soil samples were collected at 62 sites at a maximum depth of 15 cm. The PCDD/PCDF contents ranged from 1.7 to 684 ng I-TEQ/kg with a median of 18 ng/kg. Elevated soil levels were observed in the vicinity of a former chemical plant (up to

159 ng I-TEQ/kg) and along the banks of tributaries of the River Elbe (up to 684 ng I-TEQ/kg), where until the 1950s the land was regularly flooded during winter time.

Rotard *et al.* (1994) examined background levels of PCDDs/PCDFs in soils in Germany at sites outside industrial and urban regions. Elevated levels were found in the topsoil layers of forest. PCDD/PCDF levels found in ploughland and grassland samples were considerably lower, with means of 2 ng I-TEQ/kg. The isomer profiles for air and forest soil samples showed striking coincidence.

Little is known about PCDD/PCDF contamination of soils in the former German Democratic Republic. Kujawa *et al.* (1995) analysed 49 soil samples of rural origin. Only total I-TEQ levels of PCDDs/PCDFs were reported. The authors compared data for samples originating from the western and eastern parts of Germany (see Appendix 1, Table 5).

(x) *Italy*

Following the Seveso accident in 1976, soil levels of 2,3,7,8-TCDD were determined in the Zones A, B and R established around the ICMESA plant (see Section 1.3.1(*b*)). Since 2,3,7,8-TCDD concentrations detected in Zone A ranged over more than four orders of magnitude (from < 0.75 μg/m^2 to > 20 mg/m^2), the zone was broken down into subzones A_1–A_8, each characterized by a somewhat lower range of 2,3,7,8-TCDD levels (di Domenico & Zapponi, 1986). Mean concentrations of 2,3,7,8-TCDD in the soil in September 1976 in Zones A_1–A_8 were 1600, 2500, 130, 260, 120, 91, 12 and < 5 μg/m^2, respectively. The mean concentrations in Zones B and R in 1976–77 were 3.4 and ~0.5 μg/m^2, respectively. Tolerable limits for land (soil), housing interiors, equipment and other matrices and items were set by regional law. The risk areas — namely Zones A, B and R — were defined by taking into account the 2,3,7,8-TCDD levels detected predominantly in the 7-cm topsoil layer, and therefore the 'surface density' unit μg/m^2 was extensively used, the unit surface being defined as a 1-m square with a 7-cm thickness. 2,3,7,8-TCDD surface densities were converted to the more common ng/kg concentration units by multiplying by an average factor of 8. The following limits were obtained for the different risk areas and matrices (Bertazzi & di Domenico, 1994): farmable land, < 0.75 μg/m^2 (< 6 ng/kg); non-farmable land, ≤ 5 μg/m^2 (≤ 40 ng/kg); limit of evacuation, > 50 μg/m^2 (> 400 ng/kg); and, for comparison, outdoor surfaces of buildings, ≤ 0.75 μg/m^2; indoor surfaces of buildings, ≤ 0.01 μg/m^2.

di Domenico *et al.* (1993a) reported on the occurrence of PCDDs/PCDFs and PCBs in the general environment in Italy. Sampling was carried out in five regions, at or slightly above sea level (n = 10), at an altitude of 800–1300 m (n = 11) and in caves normally not visited by the general public (n = 6). All sampling sites were far from an urban or industrial setting, at least 5 km from towns or villages, and the soil was collected from top to 7-cm depth. PCDD/PCDF levels were between 0.10 and 4.3 ng I-TEQ/kg in open areas and between 0.057 and 0.12 ng I-TEQ/kg in samples from caves.

(xi) *Japan*

Very few data have been reported on Japanese soils. Nakamura *et al.* (1994) found PCDD/PCDF levels in soils of 271 and 49.6 ng I-TEQ/kg (two agricultural fields) and 42.4 ng I-TEQ/kg (urban field).

(xii) *Jordan*

Alawi *et al.* (1996a) described the concentration of PCDDs/PCDFs in the Jordanian environment in a preliminary study on a municipal landfill site with open combustion near Amman. Six samples were collected from locations distributed over the area of the landfill. The concentrations measured in soil samples ranged from 8.2 to 1470 ng German TEQ/kg dry weight.

(xiii) *The Netherlands*

van Wijnen *et al.* (1992) measured PCDD/PCDF levels in 20 soil samples collected from areas in the vicinity of Amsterdam where it was known that small-scale (illegal) incineration of scrap wire and scrap cars might have resulted in contamination with PCDDs/PCDFs. At certain spots, the illegal incineration resulted in strongly increased soil levels of PCDDs/PCDFs, ranging between 60 and 98 000 ng I-TEQ/kg dry matter. Nine samples had PCDD/PCDF levels (far) above the so-called 'level of concern' of 1000 ng/kg dry matter proposed by Kimbrough *et al.* (1984) for soil contamination with 2,3,7,8-TCDD in Times Beach, Missouri (USA).

At the Volgermeerpolder hazardous waste site, 10 years after all dumping activities ceased, the concentrations of the most toxic PCDD/PCDF congeners in topsoil and eel determined in 1994 fell within the same range as was found in 1981–84, indicating that the contamination circumstances had remained basically unaltered (Heida *et al.*, 1995).

(xiv) *Russian Federation*

Soil samples taken near plants where products such as chlorophenol, TCP and 2,4-D had been produced had 2,3,7,8-TCDD levels in the range of 900–40 000 ng/kg (Pervunina *et al.*, 1992).

Some information on ecological problems in Russia caused by PCDD/PCDF emissions from other chemical industry facilities is available (Fedorov, 1993). In 1990, at a chemical fertilizer plant in Chapaevsk, the soil near the section for PCP production contained 18.7 µg 2,3,7,8-TCDD/kg. It should be noted that the major PCDD/PCDF isomers formed in PCP production are OCDD, OCDF and HpCDDs. Soil originating from Chapaevsk farming areas (1991–92) contained between 0.2 and 68 ng 2,3,7,8-TCDD/kg. In Ufa, close to a TCP production plant, values of between 8000 and 40 000 ng 2,3,7,8-TCDD/kg have been reported.

In the Bashkortostan Republic, the main sources of environmental contamination with PCDDs/PCDFs are chemical plants in Ufa and Sterlitamak, which have manufactured organochlorine products including the pesticides 2,4,5-T and 2,4-D for more than 40 years. Soil samples taken in urban areas of Ufa and Sterlitamak, had PCDD/PCDF concentrations of 1–20 ng I-TEQ/kg, which did not exceed the norms for Russia. High concentrations of PCDDs/PCDFs were detected in industrial zones not far from chemical

and oil/chemical plants of Ufa, ranging between 280 and 980 ng I-TEQ/kg. In the majority of farm regions, PCDD/PCDF content in soil was reported to be between 0.01 and 0.13 ng I-TEQ/kg and did not exceed the permissible level of 10 ng/kg (Khamitov & Maystrenko, 1995). [It was not apparent from the paper whether the values reported were I-TEQs or total PCDD/PCDFs.]

(xv) *Spain*

Near a clinical waste incinerator in Madrid, PCDD/PCDF levels in soil samples (0–5 cm) from 16 sites indicated slight contamination by these pollutants (Gonzáles *et al.*, 1994; Jiménez *et al.*, 1996a). The highest levels were found at points located between 400 and 1200 m from the incinerator but there was no relation between PCDD/-PCDF levels and the prevailing wind direction. The analytical data for PCDDs/PCDFs and the distribution of the PCDD/PCDF homologues and the 2,3,7,8-substituted congeners failed to reveal whether this plant was the only source responsible for the soil contamination detected.

Schuhmacher *et al.* (1996) analysed soil samples in the vicinity of a municipal solid-waste incinerator in Tarragona. The highest PCDD/PCDF level (0.84 ng I-TEQ/kg) was found at a distance of 750 m from the incinerator.

(xvi) *Sweden*

PCDDs/PCDFs in soil and digested sewage sludge from Stockholm were analysed (Broman *et al.*, 1990). The contribution of PCDDs/PCDFs from sewage sludge to the total soil level was compared with the contribution from two non-point emission sources, i.e., road traffic and the urban area of the city of Stockholm. The data reported (based on the Nordic TEF model) are not based on dry matter but on organic weight, making it difficult to compare the data obtained with other published data. The mean concentration in four sludge samples was 79 ng TEQ/kg organic weight. Soil samples taken close to major roads varied between 13 and 49 ng TEQ/kg organic weight and soil samples which were not taken close to major roads varied between 9 and 32 ng TEQ/kg organic weight. The results indicate that both traffic and the urban area influence PCDD/PCDF concentrations in arable soil. Fertilization with sludge (1 tonne dry weight/hectare and year) raised the initial soil concentration of PCDDs/PCDFs in the fields by approximately 2–3%.

(xvii) *Switzerland*

Soil samples were collected at 33 sites in the northern part of Switzerland (Rheinfelden to Wallbach) (Gälli *et al.*, 1992). Concentrations of PCDDs/PCDFs in the topsoil ranged from 0.7 to 26.8 ng German TEQ/kg. Eighty per cent of the samples had concentrations < 5 ng TEQ/kg, slightly above the background level of ~ 1 ng TEQ/kg in rural areas.

(xviii) *Taiwan*

Since 1966, waste electric wires and/or magnetic cards have been incinerated directly on site during reclamation of metals, especially copper, silver and gold, in Taiwan. Surface soil samples from six sites at which these open incinerations took place were

analysed for PCDDs/PCDFs (Huang *et al.*, 1992). All samples were contaminated, with total PCDD levels ranging from undetectable to 540 ng/kg and total PCDF levels of 1.8–310 ng/kg. Only the samples from the incineration sites with waste electric wire were heavily polluted.

Soong and Ling (1996) found exceptionally high PCDD/PCDF levels in soil samples collected from a PCP manufacturing facility located in the southern part of Taiwan (maximum, 1357 μg I-TEQ/kg) five years after operation had ceased.

(xix) *United Kingdom*

PCDD/PCDF levels in archived soil samples collected from the same semi-rural plot in south-east England between 1846 and the present were determined by Kjeller *et al.* (1990, 1991). Atmospheric deposition is known to have been the only source of PCDDs/PCDFs to the site. PCDDs/PCDFs were present in all the samples. Generally, the total PCDD/PCDF concentration began to increase in the early twentieth century from about 30 ng/kg in the 1850s to the 1890s to about 90 ng/kg in the 1980s.

A comprehensive study of British soils to measure the background contamination with PCDDs/PCDFs was performed by Creaser *et al.* (1989), who analysed 77 topsoil samples (0–5 cm) from points of a 50 km grid covering England, Wales and Scotland. Mean concentrations for a reduced data-set were in the range of 9.4 ng/kg for total TCDDs to 191 ng/kg for OCDD.

In a further study by Creaser *et al.* (1990), soil samples from five British cities (London, Birmingham, Leeds, Sheffield and Port Talbot) were analysed for PCDDs/-PCDFs. The mean levels were significantly higher than those from rural and semi-urban locations. The concentrations of the lower PCDD and PCDF congener groups show the greatest increase. By principal component analysis, it was deduced that combustion processes, such as coal burning and municipal waste incineration, are the main sources of PCDDs/PCDFs in these soils.

Stenhouse and Badsha (1990) presented baseline concentrations for PCDDs/PCDFs and PCBs around a site proposed for a chemical waste incinerator near Doncaster. The values were between 3 and 20 ng I-TEQ/kg, indicating that the area had relatively low contamination with these components and may be comparable with a rural environment.

Biomass burning is known to constitute a highly dispersed source of PCDDs/PCDFs (Levine, 1992). Walsh *et al.* (1994) monitored soils before and after straw field fires and assessed the degree and nature of formation/destruction and transformation processes occurring during biomass burning. PCDD/PCDF concentrations of post-burn soils showed a slight relative reduction with respect to the corresponding pre-burn soils at three burnings. This reduction may be attributed to the high temperature achieved during the burn, leading to vaporization and destruction of PCDDs/PCDFs in the surface layer of soil. The overall I-TEQ for PCDDs/PCDFs decreased after the straw fire, with a decrease in concentration of 2,3,7,8-TCDD and an increase in that of OCDD.

Foxall and Lovett (1994) analysed soil samples from South Wales, Pantec district, where the major industries used to be coal mining, iron and steel production, aluminium smelting and glass manufacturing. Current industries involve steel rolling, automotive

engineering, pharmaceuticals and others. Forty-two samples from 32 sites were analysed by eight national or international laboratories. The total PCDD/PCDF levels show considerable variation between sampling sites, with a mean of 66 ng I-TEQ/kg and a median of 10.5 ng I-TEQ/kg (range, 2.5–1745 ng I-TEQ/kg). The maximal concentration in soil was found at a site near a chemical water incinerator.

PCDD/PCDF contamination on land around a chemical waste incinerator has been described (Holmes *et al.*, 1995; Sandalls *et al.*, 1996). After 1991 during a routine surveillance, high PCDD/PCDF concentrations were detected in the milk of cows grazing within 2 km of a chemical factory in Bolsover, Derbyshire, which had produced 2,4,5-TCP. As a follow-up, PCDDs/PCDFs were measured in soil samples taken up to 5 km from the factory. The samples were taken from 46 sites where the soil had remained undisturbed for many years. As in flue gas at the incinerator, total TCDD was always found to be prominent in soil and was selected as a marker for assessment purposes. Within 1 km of the factory, the concentration of total TCDDs in soil was up to 9400 ng/kg, and even 4–5 km away several hundred ng/kg were found. The authors concluded that the special distribution pattern of PCDDs on surrounding land implicated the chemical factory as the likely source since the soil samples showed a congener ratio pattern not resembling that found in a United Kingdom background survey.

(xx) *United States*

In 1971, 29 kg of 2,3,7,8-TCDD-contaminated chemical sludge were mixed with waste oils and used as a dust suppressant at sites in 10 counties of Missouri. Kimbrough *et al.* (1977) described an epidemiological and laboratory investigation of a poisoning outbreak that involved three riding arenas and killed 57 horses and numerous other animals. The outbreak was traced to the spraying of the arenas with the contaminated oil containing TCP, PCBs and hexachloroxanthene. The 2,3,7,8-TCDD levels in the soil samples ranged up to 33 mg/kg. The presence of hexachloroxanthene in most of the contaminated sites, although at levels varying considerably from site to site and also within sites, implicates a hexachlorophene producer as the source (Kleopfer *et al.*, 1985).

Nestrick *et al.* (1986) measured levels of 2,3,7,8-TCDD in samples taken at various sites at a major chemical plant in Midland, MI, in the city of Midland and in other industrialized areas in the United States. Within the chemical plant, certain areas had localized elevated levels (above 5 µg/kg) 2,3,7,8-TCDD in the surface soil (Appendix 1, Table 6). In the zone immediately surrounding the Midland plant, most 2,3,7,8-TCDD soil levels were below 1000 ng/kg, but many times higher than those found in other industrialized urban areas, suggesting that the chemical plant was a primary source of 2,3,7,8-TCDD found in the immediate environment. The data for other US cities are summarized in Appendix 1 (Table 7).

Reed *et al.* (1990) measured PCDD/PCDF levels at Elk River, Minnesota, before operation of an electric generating station by powered refuse-derived fuel. The area was semi-rural without industry. The soil data reflect generally low background concentrations of PCDDs/PCDFs, with surprisingly high values for OCDD (ranging from 340 to 3300 ng/kg).

Concentrations of PCDDs/PCDFs in 36 soil samples from eight counties in southern Mississippi were reported by Rappe *et al.* (1995) and Fiedler *et al.* (1995). The selected sampling sites were not directly influenced by human activities, such as heavy traffic or dust. Controlled burning is a common practice in southern Mississippi, and potential sites were not excluded when traces of former fires could be seen. The sampling depth was 5 cm. Most Cl_4–Cl_8 PCDDs/PCDFs were detected in all samples. 2,3,7,8-TCDD was identified in 17 of the 36 samples at a detection limit of 0.02–0.05 ng/kg dry weight. The highest concentration was 22.6 ng I-TEQ/kg in a sample from Perry County. In some cases, surprisingly high values for OCDD were found (13–15 µg/kg) (see Appendix 1, Table 8). The source of these high levels is not known but the authors suggested a non-anthropogenic origin.

The Columbus Municipal Waste-to-Energy facility in Columbus, OH, began operation in 1983. In 1992 it was reported to be the largest known single source of PCDD/-PCDF emissions. Emission was reduced from 984 g I-TEQ/year in 1992 to 267 g/year in 1994. Soil was monitored for PCDDs/PCDFs at the plant site, directly off-site in the predominant wind direction (four samples), at 14 sites in the city of Columbus and 28 miles away from Columbus in a rural setting (Lorber *et al.*, 1996b). The high PCDD/PCDF levels in the four samples taken close to the facility (average, 356 ng I-TEQ/kg) suggest direct soil contamination due to the operation of the plant (stack emission and/or on site ash handling). The congener profile seen in the stack emission was very similar to those observed in the soil samples taken at the plant. The background soil concentrations of 1–2 ng I-TEQ/kg are consistent with other measurements in North America and elsewhere.

(xxi) *Viet Nam*

During the Second Indo-China War, the south of Viet Nam was sprayed with herbicides contaminated with 2,3,7,8-TCDD (see Section 1.3.1(*a*)(ii)). Matsuda *et al.* (1994) measured the persisting levels of 2,3,7,8-TCDD in soils. To assess background concentration in soil, five samples were collected in Hanoi, where the herbicides had not been sprayed and no 2,3,7,8-TCDD has been found. Contrary to expectation, 2,3,7,8-TCDD was detected in only 20 out of 106 south Vietnamese samples analysed. The levels of 2,3,7,8-TCDD detected in 14/54 samples from Tay Ninh Province, South Viet Nam, ranged between 1.2 and 38.5 ng/kg dry weight (mean, 14.0 ng/kg). The results suggest that most of the sprayed material has been leached and ultimately drained towards the sea and/or to the soil subsurface during the rainy season.

(*d*) *Food*

It is estimated that intake from food consumption accounts for well over 90% of the body-burden of PCDDs and PCDFs in the general human population (Gilman *et al.*, 1991; Travis & Hattemer-Frey, 1991).

Over the last decade a number of studies of foods have appeared, but several are of only limited usefulness because of low sensitivity. The results from Canadian studies of meat collected in 1980 were published by Ryan *et al.* (1985a), but the lower chlorinated congeners were not detected. Firestone *et al.* (1986) published the results of analyses for

the higher chlorinated PCDDs in various foods collected in the United States by the Food and Drug Administration in a five-year period beginning in 1979; lower chlorinated PCDDs including 2,3,7,8-TCDD were not determined. Stanley and Bauer (1989) analysed composites of selected foods collected from San Francisco and Los Angeles, CA, including salt-water fish, freshwater fish, beef chicken, pork, cow's milk and eggs. The analytical methods and quality control were rigorous but the limits of detection were somewhat higher than those obtained in some other recent studies and few measurable residues were found. Birmingham *et al.* (1989) analysed samples of beef, pork, chicken, eggs, milk, apples, peaches, potatoes, tomatoes and wheat from Ontario (Canada). Some samples were found to contain detectable concentrations of the higher chlorinated congeners but no Cl_4 or Cl_5 PCDDs or PCDFs were detected. The data from this study are available only on a whole sample rather than a lipid-adjusted basis.

Studies by Beck *et al.* (1987, 1989a) of foods available in Berlin, Germany, were based on analysis of 12 random purchases of foods (chicken, eggs, butter, pork, red fish, cod, herring, vegetable oil, cauliflower, lettuce, cherries and apples) and of eight samples of cow's milk. The analysis used high-resolution mass spectrometry and achieved very high sensitivity, so that most of the congeners with a 2,3,7,8-substitution pattern were detected in most of the samples. Fürst *et al.* (1990) concentrated on fatty foods and foods of animal origin in a study involving over 100 individual samples from the North-Rhine Westphalia area of Germany, but with slightly lower sensitivity. Fürst *et al.* (1992a) subsequently reported further analysis of milk and dairy products which made use of more sensitive high-resolution mass spectrometry, but detailed congener-specific results were not published.

A comprehensive assessment of Dutch foodstuffs was reported by Liem *et al.* (1991a,b). The sampling scheme was designed after considering the individual consumption data of approximately 6000 individuals from 2200 families over a two-day period. Animal fat and liver from six different types of animal were collected from slaughter houses, and cereal products, cow's milk, dairy products, meat products, nuts, eggs, fish and game from retail sources. In each of these groups, duplicate collection of samples was carried out in each of four Dutch regions and proportional pooling was performed to provide a pair of duplicate samples for each group and region, and one national composite sample from the four regional pools. Analysis was by high-resolution mass spectrometry and achieved high sensitivity. Liem *at al.* (1991c) also reported the analysis of 200 samples of cow's milk obtained from the vicinity of municipal solid-waste incinerators and other potential PCDD/PCDF sources in the Netherlands.

Results are available from a study of composite samples from the UK Total Diet Study (TDS) (Ministry of Agriculture, Fisheries and Food, 1995; Wright & Startin, 1995; Wearne *et al.*, 1996; see **Table 23**). In this scheme, retail samples of 115 specified food items are purchased at two-week intervals from different locations in the United Kingdom, prepared as for consumption, then combined into one of 20 food groups in proportions representing the relative importance of the retail foods in the average British diet. Concentrations of PCDDs and PCDFs were determined in archived TDS samples of fatty foods and bread collected in 1982 and 1992. For each food group, the material

analysed was a composite of samples from all 24 locations included in the TDS that year. Fruit, vegetables and other non-fatty foods were not analysed. The determinations used high-resolution mass spectrometry and met appropriate acceptance criteria (Ambidge *et al.*, 1990).

Table 23. Concentrations of PCDDs/PCDFs (ng I-TEQ/kg whole food) in Total Diet Study samples collected in 1982 and 1992 in the United Kingdom

Food group	PCDDs/PCDFs (ng I-TEQ/kg)	
	1982	1992
Bread	0.02	0.03
Other cereals	0.13	0.17
Carcass meat	0.49	0.13
Offal	1.6	0.59
Meat products	0.32	0.08
Poultry	0.50	0.13
Fish	0.41	0.21
Oils and fats	1.3	0.20
Eggs	0.92	0.17
Milk	0.16	0.06
Milk products	1.2	0.16

From Wearne *et al.* (1996)

Several studies of food items from Viet Nam, Russian Federation and the United States have been reported by Schecter *et al.* (1989a, 1990a, 1994a), who worked with various analytical laboratories of good repute. Most of the data relate to individual samples. In the study of food in the United States, 18 samples of dairy products, meat and fish from a supermarket in upstate New York were analysed (Schecter *et al.*, 1994a). A more recent study (Schecter *et al.*, 1996a) of 100 food items combined by type has not been fully reported, but led to estimates of intake close to those of the earlier study. A more comprehensive investigation of concentrations in beef in the United States has been reported by Ferrario *et al.* (1996).

Several studies have been conducted of foods in the Japanese diet, but some doubt must be attached to the results. Ono *et al.* (1987) reported the analysis of vegetables, cooking oils, cereals, fish, pork, beef, poultry and eggs collected in Matsuyame in 1986. In contrast to other studies, the dominant congeners in the fish and animal samples were generally not those with 2,3,7,8-substitution, suggesting that the results do not reflect biologically incurred residues but some other form of contamination. OCDD (which is normally the congener of highest concentration) was not consistently found and it is possible that the alkaline saponification procedure used for some samples resulted in dechlorination of OCDD and distortion of the congener profile. Other studies by

Takizawa and Muto (1987) and by Ogaki *et al.* (1987) also employed alkaline saponification and the reports do not include congener-specific results.

Relevant data are summarized in Appendix 1 (Tables 9–18). The data included have been selected to meet a number of criteria, including relevance to dietary intake rather than environmental monitoring, and adequate detection limits and appropriate analytical methodology. Some exceptions have, however, been made and some data that are available only as summed I-TEQs have been included where they seem to be of value. Additionally, the results included were either available on a lipid basis or could be so converted using either reported fat contents or reasonable assumptions. Some additional results omitted from the tables are discussed below. All concentrations quoted are lipid-adjusted unless otherwise stated.

Most of the summed I-TEQ concentrations included in the tables have been re-calculated using I-TEF values and assuming that congeners that were not detected were present at the full value of the detection limit. Obviously, however, this was not possible where detection limits were not reported (indicated as 'ND') or where the original author's calculations were used.

(i) *Background exposure*

Vegetables

Studies on uptake from soil, which have been reviewed (Kew *et al.*, 1989), are not entirely consistent but it is generally accepted that systemic uptake through the roots and translocation within plants is virtually absent in most species. Some members of the *Cucurbitaceae* family, however, have been shown to take up PCDDs and PCDFs from soil, leading to a uniform concentration of about 20 ng I-TEQ/kg dry mass in the above-ground parts of plants grown in soil containing 148 ng I-TEQ/kg (Hülster *et al.*, 1994).

Apart from localized contamination, PCDD and PCDF concentrations in fruit and vegetables have usually been found to be immeasurably small. In the survey of foodstuffs available in West Berlin (Beck *et al.*, 1989a), five vegetable samples were analysed (including cauliflower, lettuce, cherries and apples) and PCDDs and PCDFs were not found, subject to a detection limit of 0.01 ng/kg for each isomer. Similarly PCDDs and PCDFs were not detected to any significant extent in vegetable samples from the Total Diet Survey schemes in the United Kingdom (Ministry of Agriculture, Fisheries and Food, 1992) or in Canada (Birmingham *et al.*, 1989).

A number of studies have dealt with vegetable oils. Beck *et al.* (1989a), Fürst *et al.* (1990) and Liem *et al.* (1991a,b) have all reported that the concentrations of PCDDs and PCDFs were below the limit of detection, apart from some low levels of the hepta- and octa-chlorinated congeners.

Cow's milk

The detection of PCDDs and PCDFs in cow's milk was first reported by Rappe *et al.* (1987b) in samples from Switzerland. In retail milk from Bern and Bowil (a location remote from potential PCDD sources), several PCDDs were identified although concen-

trations were close to the detection limit. The congeners found were exclusively 2,3,7,8-substituted.

Data from this and subsequent reports on levels of PCDDs in milk are given in Appendix 1 (Table 9; summarized in Table 10).

Data on levels in cow's milk from various locations are dominated by European samples from the late 1980s and early 1990s. The data show a mean concentration of 2.3 ng I-TEQ/kg with a range of 0.26–10.0 ng I-TEQ/kg. Concentrations of 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 1,2,3,4,7,8-HxCDD are all within a range of 0.13–2.3 with means of about 0.6–0.7 ng/kg. Levels of 1,2,3,6,7,8-HxCDD are somewhat greater, with a mean of 2.0 ng/kg and range of 0.3–8.9 ng/kg. [The concentration of 1,2,3,7,8,9-HxCDD found in Spanish milk (Ramos *et al.*, 1996) is dubious; the other results give a mean of 0.58 ng/kg.] Measured concentrations of HpCDD and OCDD are frequently unreliable due to the confounding influence of laboratory contamination and other analytical difficulties, but are greater than those of the Cl_4–Cl_6 congeners.

The data of Fürst *et al.* (1993) (see Section 1.3.2(*c*)(ix)) imply that the pathway air → grass → cow is more important than the pathway soil → grass → cow. The carry-over factors for PCDD/PCDF congeners between grass and milk differ significantly. While 2,3,7,8-TCDD showed the highest carry-over factor, OCDD was accumulated less by a factor of almost 40.

In addition to the tabulated data, Stanley and Bauer (1989) reported the analysis of eight composite samples from the United States, but most PCDDs and PCDFs were not detected and the detection limits were slightly higher than typical background concentrations found in other studies. LaFleur *et al.* (1990) reported 0.002 ng 2,3,7,8-TCDD/kg in milk used in a study of migration of PCDDs from milk cartons, but did not analyse higher congeners. Glidden *et al.* (1990) have also reported that these congeners were not detectable in milk that had not been packaged in paperboard cartons.

Dairy products

Reported concentrations of PCDDs and PCDFs in dairy products are given in Appendix 1 (Table 11) and Appendix 2 (Table 4) and, as expected, are similar to those in milk when expressed on a fat basis. In milk products, the mean summed I-TEQ concentration for PCDDs together with PCDFs (calculated assuming non-detected congeners to be present at the full value of the detection limit) is about 2.4 ng I-TEQ/kg (range, 0.8 to 8 ng I-TEQ/kg fat). [The Working Group noted that this is a conservative estimate; the true concentrations may be lower in some cases but most of the studies considered achieved excellent sensitivity, and other assumptions would lead to fairly small changes in the mean.] As with milk, data from UK Total Diet Survey samples show a considerable decrease from 1982 to 1992 (Wright & Startin, 1995; Wearne *et al.*, 1996).

Meat

Reported data for various meats and meat products, shown in Appendix 1 (Table 12; summarized in Table 13), indicate a mean concentration of 2,3,7,8-TCDD which is also about 0.5 ng/kg fat, but with a range of about two orders of magnitude. Concentrations of

the other congeners tend to be a little higher than in milk — between 1.5 and 5 ng/kg for PeCDD and HxCDD isomers, 62 ng/kg for HpCDDs and 350 ng/kg for OCDD. Ranges are again rather large and are widest for OCDD (a factor of 250). In TEQ terms, the average concentration is about 6.5 ng I-TEQ/kg.

Beck *et al.* (1989a) found concentrations in the range of 1.65–2.59 ng I-TEQ/kg for beef, lamb and chicken but a much lower concentration (0.28 ng I-TEQ/kg) in pork. Similar results were reported by Fürst *et al.* (1990); concentrations in beef, lamb, chicken and in canned meat were in the range 2.4–3.7 ng I-TEQ/kg while PCDDs and PCDFs other than OCDD were not detected in pork. Fürst found a rather higher average concentration of 7.7 ng I-TEQ/kg in veal, that is presumably due to the high early-life input from milk.

Similar concentrations were found in beef, mutton and chicken from the Netherlands where the range was 1.6–1.8 ng I-TEQ/kg (Liem *et al.*, 1991b). Again a rather lower level of 0.42 ng I-TEQ/kg was found in pork. This study also included horse and goat fat, in which higher concentrations of 14 and 4.2 ng I-TEQ/kg, respectively, were found. Liver from these animals was also analysed and the concentrations, on a fat basis, were 2–10-fold higher than in the corresponding animal fat.

The limited number of reported measurements of levels in animal liver from food distribution channels show a tendency to higher concentrations in I-TEQ terms (6–60 ng I-TEQ/kg). Concentrations of HpCDD and OCDD especially are relatively large, with maxima approaching 1 μg/kg for the former and exceeding 4 μg/kg for the latter congener.

LaFleur *et al.* (1990) examined samples of canned corned beef hash, ground beef, beef hot dogs and ground pork available in the United States for 2,3,7,8-TCDD and 2,3,7,8-TCDF. The former was found in all the beef samples at concentrations between 0.03 and 0.35 ng/kg but was not detected in pork.

Schecter *et al.* (1994a) more recently reported on a number of individual samples of retail meat products from the United States. The concentrations in four different samples of beef and beef products spanned a wide range from 0.04 to 1.5 ng I-TEQ/kg on a whole sample basis. Cooked ham contained 0.03 ng I-TEQ/kg, while a single pork chop contained 0.26 ng I-TEQ/kg and a sample of lamb sirloin 0.41 ng I-TEQ/kg. A very low level of 0.04 ng I-TEQ/kg was found in a sample of chicken.

Concentrations in samples from widely separated locations in the Soviet Union ranged from 0.2–6 ng I-TEQ/kg in beef, pork and sausage (see Appendix 1, Table 12).

Poultry

The rather limited data (Appendix 1, Table 14) on poultry meat suggest typical concentrations of the same order of magnitude as those in other animal products, apart from the rather greater concentrations found in two samples from Viet Nam (Schechter *et al.*, 1989a). The UK Total Diet Survey (Wright & Startin, 1995) shows a marked decrease between 1982 and 1992.

Eggs

Although the 2,3,7,8-substituted PCDD/PCDF congeners predominate in eggs, other congeners are observed to a greater extent than in other animal-derived foods. There have been relatively few studies of contamination in eggs at the retail level, but the limited data in Appendix 1 (Table 15) show reasonable agreement between samples from the Netherlands, Germany, Spain and the United Kingdom (1992 Total Diet Survey sample). In terms of I-TEQ concentrations for PCDDs and PCDFs, the British results show a decrease from 1982 to 1992 of nearly a factor of 5.

Earlier results of 0.22 and 0.16 ng I-TEQ/kg whole egg found in two egg composites from the UK Total Diet Survey from 1988 (Ministry of Agriculture, Fisheries and Food, 1992) and an average of 0.2 ng TEQ/kg whole egg in samples from Norway (Faerden, 1991) are also consistent, assuming a fat content of 10%.

In the United States, the data of Stephens *et al.* (1995) point to a similar background level in eggs from chickens fed on commercial formulations to that in Europe, whereas in eggs from free-range birds with access to moderately contaminated soils, concentrations were as much as 100-fold higher than in commercial eggs.

In the Canadian data, only higher chlorinated congeners were detected in eggs, but an average concentration of 0.59 ng I-TEQ/kg was used for dietary intake calculations (Birmingham *et al.*, 1989).

Two chicken's eggs from Viet Nam had concentrations of 0.55 and 1.62 ng I-TEQ/kg whole egg while PCDDs and PCDFs were not detected in a single sample of duck eggs (Olie *et al.*, 1989).

Lovett *et al.* (1996) reported 1.2 ng I-TEQ/kg fresh mass in chicken eggs from rural sites in Wales, 0.6 ng I-TEQ/kg in bantam hen eggs and 0.7 ng I-TEQ/kg in duck eggs. [Assuming 10% as a typical fat content, these seem much too high.]

Fish

Although PCDDs and PCDFs are usually present in aquatic systems only at very low levels, bioaccumulation of the 2,3,7,8-substituted congeners can result in significant concentrations in fish. As with animals, the 2,3,7,8-substituted congeners dominate the congener pattern found in fish, although this is not true of crustaceans and shellfish (Oehme *et al.*, 1989). Since different species occupy quite different trophic positions, large differences in PCDD and PCDF concentrations are to be expected.

Data from studies of food fish are shown in Appendix 1 (Table 16; summarized in Table 17). Average concentrations of 2,3,7,8-TCDD and of 1,2,3,7,8-PeCDD are about an order of magnitude greater than in foods of animal origin.

A study of cod and herring from the seas around Sweden has been reported (Bergqvist *et al.*, 1989). Herring from the Baltic were found to have PCDD/PCDF concentrations in the range of 6.7–9.0 ng I-TEQ/kg wet weight, while considerably lower concentrations of 1.8–3.4 ng I-TEQ/kg were found in fish from the west coast of Sweden. de Wit *et al.* (1990) also demonstrated higher levels in fish from the Baltic Sea.

Takayama *et al.* (1991) reported data on coastal and marketing fish from Japan. The means for these two groups were 0.87 and 0.33 ng I-TEQ/kg, respectively, on a wet weight basis.

A number of studies have dealt with contamination of fish in the North American Great Lakes and rivers in the Great Lakes Basin (Harless *et al.*, 1982; O'Keefe *et al.*, 1983; Ryan *et al.*, 1983; Stalling *et al.*, 1983; Fehringer *et al.*, 1985), which have been among the most severely contaminated in the United States. An extensive survey of 2,3,7,8-TCDD in fish from inland waters in the United States has also been conducted (Kuehl *et al.*, 1989) and over 25% of all samples were found to be contaminated at or above the detection limit, which varied between 0.5 and 2.0 ng/kg. Concentrations in excess of 5.0 ng/kg were found in 10% of samples and the highest level was 85 ng/kg. Samples collected near sites of discharge from pulp and paper mills had a higher frequency of 2,3,7,8-TCDD contamination than other samples. More recently, Firestone *et al.* (1996) summarized the results of monitoring for 2,3,7,8-TCDD in the edible portion of fish and shellfish from various United States waterways since 1979. Analyses of 1623 test samples indicated that 2,3,7,8-TCDD residues in fish and shellfish were not widespread but rather were localized in areas near waste sites, chlorophenol manufacturers and pulp and paper mills. The levels in aquatic species from these sites have been declining steadily. No 2,3,7,8-TCDD (limit of detection and confirmation, 1–2 ng/kg) has been found in recent years in aquatic species from most Atlantic, Pacific and Gulf of Mexico sites and Great Lakes other than Lake Ontario and Saginaw Bay (Lake Huron).

There are rather fewer data on retail samples of the major food species and the ranges of reported results are wider than for animal products. Two recent studies of composite samples of sea fish, formulated to represent national average dietary habits in the United Kingdom and the Netherlands, showed considerable differences for all congeners (see Appendix 1, Table 16) (Liem *et al.*, 1991b; Ministry of Agriculture, Fisheries and Food, 1995). The available data lead to averages of about 5 ng/kg for 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 1,2,3,6,7,8-HxCDD, with lower concentrations of 1,2,3,4,7,8-HxCDD and 1,2,3,7,8,9-HxCDD and rather higher ones of HpCDDs and OCDD. The mean total concentration of 25 ng I-TEQ/kg has a larger contribution from PCDFs than that for animal products (see Appendix 1, Table 17).

In addition to the tabulated results, data from the United Kingdom from eight retail samples, including plaice, mackerel, herring, cod, skate and coley, gave a mean of 0.74 ng/kg and a range of 0.15–1.84 ng TEQ/kg on a wet weight basis (Startin *et al.*, 1990), but fat contents were not determined.

In contrast to the extensive North American measurements on fish caught in specific locations, there is little information documenting the levels in the general food supply there, apart from the results reported by Schecter *et al.* (1994a) who found 0.02 and 0.03 ng I-TEQ/kg in haddock fillets, 0.023 ng I-TEQ/kg in a cod fillet and in a perch fillet, and 0.13 ng I-TEQ/kg in crunchy haddock. Fat contents were not measured. These levels are rather lower than those typical in Europe but relate to a very restricted sampling base.

The very limited reported data on fish oils (used, for example, as dietary supplements) are consistent with the lipid-based concentrations reported in fish.

Other foods

Table 18 in Appendix 1 gives PCDD concentrations that have been found in some other commodities and products. Bread was included in the UK TDS as it is a staple item in most people's diet and also because fat is usually used in preparing the dough. Many of the congeners were not detected but detection limits varied so that the upper-bound total I-TEQ concentration appears to increase in the more recent pooled sample. [The summations are almost certainly a considerable overestimate of true concentrations.]

Various studies of cooked foods and prepared dishes have been reported; concentrations of PCDDs found were broadly similar to those in individual commodities.

[The Working Group noted that the available data on food show that average PCDD/PCDF concentrations in animal fats consumed in the diet in different industrialized regions are similar, even for different continents.]

Data from a UK study (Wearne *et al.*, 1996) in which a comparison was made of composites of various fatty food composites taken from Total Diet Study (TDS) samples from 1982 and 1992 show a considerable decrease in PCDD/PCDF levels over this decade (**Table 23**). Results obtained separately on a small number of individual TDS samples from 1988, while less robust, support this finding (Ministry of Agriculture, Fisheries and Food, 1995).

Fürst and Wilmers (1995) have reported a decrease of nearly 25% in PCDD/PCDF levels in cow's milk and milk products collected from all 30 dairies in North Rhine–Westphalia in Germany in 1994 compared to 1990.

(ii) *Foods from contaminated areas*

Vegetables

The outer surface of root crops can obviously become contaminated by soil contact; low concentrations of PCDDs and PCDFs have been measured in root crops such as carrots and potatoes grown in contaminated soils but were largely absent if the vegetables were peeled (Facchetti *et al.*, 1986; Hülster & Marschener, 1993).

Surface-borne PCDD and PCDF contamination of foliage and fruits may include contributions from direct deposition of airborne particulates and from absorption of vapour-phase contaminants from the air, including those which are attributable to evaporation from the soil (Reischl *et al.*, 1989). In apples and pears grown on highly contaminated soil, Müller *et al.* (1993) found total concentrations of PCDDs and PCDFs (including non-2,3,7,8-substituted congeners) in the range of 1–4 ng/kg. Peeling removed most of the PCDD and PCDF contamination, although washing was not effective.

In studies of field-grown vegetables, measurable amounts of PCDDs/PCDFs have generally been found only in areas where a specific contamination problem was known to exist. Concentrations of 2,3,7,8-TCDD of 100 ng/kg were detected in the peel of fruits grown on soils contaminated in the Seveso incident, but not in the flesh (Wipf *et al.*, 1982). Investigations in the vicinity of a wire reclamation incinerator showed contami-

nation of leaf vegetables at concentrations of 5–10 ng I-TEQ/kg and rather less in fruits (Prinz *et al.*, 1990).

Cows' milk

A number of studies have demonstrated the localized influence of incinerators and other sources on PCDD/PCDF concentrations in cow's milk (**Table 24**). Thus, Rappe *et al.* (1987b) found between about 8 and 12 ng I-TEQ/kg in the milk of individual cows grazing near municipal solid-waste incinerators and a chlorinated chemical production site in Switzerland.

More recently, in surveillance around incinerators in the Netherlands, levels up to 13.5 ng I-TEQ/kg in cow's milk were found (Liem *et al.*, 1991c). The highest PCDD concentrations were usually found within about 2 km of the source.

Schmid and Schlatter (1992) analysed milk from sites near waste incineration, metal recycling and other industrial facilities in Switzerland, where the PCDD/PCDF levels were reported to be two- to four-fold greater than in commercial samples.

In the United Kingdom, Startin *et al.* (1990) found between 3 and 6.2 ng I-TEQ/kg for PCDDs/PCDFs in milk from farms near an incinerator and close to a densely populated and industrialized area, compared with the mean for rural background areas of 1.1 ng I-TEQ/kg. In 1990, concentrations of 40 and 42 ng I-TEQ/kg were found in milk from two farms near Bolsover in Derbyshire, while milk from 30 other farms in the region contained an average of 4.3 ng I-TEQ/kg (range, 1.8–12.5) (Harrison *et al.*, 1996). In contrast to the results discussed above where the proportions of different congeners were fairly similar to those in background samples, milk from Bolsover showed a distinctive pattern dominated by 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD and 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDD.

Riss *et al.* (1990) investigated contamination caused by a metal reclamation plant at Brixlegg in Austria and found PCDD/PCDF levels in two samples of cow's milk giving 55 and 69 ng I-TEQ/kg.

(iii) *Human intake levels from food*

Birmingham *et al.* (1989) estimated the daily intake of PCDDs and PCDFs from food for Canadian adults to be 92 pg I-TEQ, the main contributors being milk and dairy products, beef and eggs. Beck *et al.* (1989a) and Fürst *et al.* (1990) estimated West German exposure to be 93.5 and 85 pg German TEQ/day, respectively. Both groups concluded that intake was derived about equally from milk, meat and fish.

The estimates above were derived by multiplying average concentration in foods and average food consumption statistics. An alternative approach using a database of food consumption data for 5898 individuals was applied by Theelen *et al.* (1993) to estimate intakes in the Netherlands. This showed a median adult intake of about 70 pg I-TEQ/day.

In the United Kingdom, both approaches have been applied to estimating intakes in 1982 and 1992 (Wearne *et al.*, 1996). Using the average consumption method, estimated intakes of PCDDs and PCDFs were [240 pg TEQ/day] in 1982 and [69 pg TEQ/day] in 1992. Based on seven-day consumption records for over 2000 adults, mean intake

Table 24. Concentrations of PCDDs reported in cow's milk from contaminated areas

Reference	Origin	Sample year	No.	PCDD concentration (ng/kg fat)[a]							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ[b] PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Riss *et al.* (1990)	Austria, Tyrol, Brixlegg (metal reclamation)	1988	1	17.8	24.5	3.7	11.2	9.4	5.6	NR	[54.6]
			1	18.5	25.3	5.4	14.5	3.6	17.1	NR	[69.1]
Rappe *et al.* (1987b)	Switzerland, Hunzenschwil (SE from MSWI)	NR	1	[1.1]	[5.59]	[5.15]	[6.49]	[3.8]	[5.82]	[6.26]	[11.8]
	Switzerland, Rheinfelden (Cl compound manuf.)	NR	1	[0.60]	[< 2.87]	[< 4.01]	[< 6.02]	[< 3.15]	[12.0]	[16.9]	[7.53]
	Switzerland, Suhr (SW from MSWI)	NR	1	[1.2]	[< 2.71]	[4.42]	[5.05]	[< 2.52]	[< 3.0]	[< 5.05]	[8.38]
Startin *et al.* (1990)	UK, incinerator	1989	1	[0.85]	[1.2]	0	[2.58]	[0.83]	[1.7]	[6.45]	[3.04]
			1	[0.9]	[1.95]	0	[2.15]	[< 1.78]	[1.73]	[7.75]	[4.07]
	UK, urban/industrial	1989	1	[2.03]	[1.05]	0	[1.58]	[0.4]	[1.63]	[8.05]	[6.24]
			1	[1.08]	[0.48]	0	[0.6]	[< 0.75]	[4.15]	[6.4]	[3.71]
Harrison *et al.* (1996)	UK, Derbyshire (Cl compound manuf.) (Farm A) (4% fat assumed)	1990	1	[24.5]	[18.75]		[45]		[13.3]	[4.5]	[15]	[44.7]
Eitzer (1995)	USA, Connecticut (incineration) (4% fat assumed)	1993	12	[0.38]	[0.17]	[0.65]	[0.58]	[0.23]	[3]	[42.5]	[0.82]

NR, not reported; MSWI, municipal solid-waste incinerator

[a] When concentrations were given by the authors on whole milk basis, they have been recalculated on lipid basis by the Working Group.

[b] Summed TEQ concentrations recalculated by the Working Group where possible assuming congeners that were not detected were present at the full value of the limit of detection

estimates were made of 250 pg TEQ/day in 1982 and 88 pg TEQ/day in 1992. The decrease in intake was attributed partly to the decrease in the concentration of PCDDs and PCDFs (on a fat basis) in foods, and partly to changes in dietary habits and a decrease in the average fat content of the foods consumed between 1982 and 1992.

1.4 Human tissue measurements (see **Table 25**)

This section considers exclusively populations without exposure to PCDDs through occupation or industrial accidents.

PCDDs and PCDFs are found ubiquitously in human tissues. The concentrations in humans are higher in industrialized countries than in non-industrialized countries, now being about 15 ng I-TEQ/kg lipid and normally below 10 ng I-TEQ/kg lipid, respectively. These values are several orders of magnitude lower than those observed in accidentally and/or occupationally exposed individuals. In general, no significant differences in tissue levels have been found between people living in urban and rural areas. Extreme consumption of certain foods or normal consumption of highly contaminated foods may result in higher body burden, but only in some special circumstances does the increase exceed a factor of about 5.

Human milk is both a useful matrix for biological monitoring and an important food. Data on levels of PCDDs in human milk are therefore presented in some detail in a separate table, organized by country and discussed separately (see Section 1.4.2).

With a very few exceptions, only 2,3,7,8-substituted congeners are found in human tissue samples. A general observation for human background contamination is that OCDD is the most abundant isomer, followed by the 2,3,7,8-substituted hepta- and hexachloro-congeners. 2,3,7,8-TCDD is normally less abundant than PeCDD.

All values reported in this section are given on a lipid content basis. The concentrations are reported in µg/kg extractable fat (if not expressed otherwise).

1.4.1 *Blood and tissue samples*

(*a*) *Austria*

For comparison with an exposed group of 2,4,5-T production workers, Neuberger *et al.* (1991) reported 2,3,7,8-TCDD blood concentrations in men with low or no occupational exposure to 2,3,7,8-TCDD. The range and median for the group were < 5–23 and 13 ng/kg, respectively.

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 in the same area, increased levels of certain isomers were found. The highest value (one sample) was found for 1,2,3,7,8-PeCDD at 780 ng/kg (Riss *et al.*, 1990).

(*b*) *Canada*

Ryan *et al.* (1985b) reported that adipose tissue from 23 older subjects (> 60 years old) who had died in Ontario hospitals in 1979–81 contained an average of 11 ng/kg 2,3,7,8-TCDD.

Table 25. Concentrations of PCDDs in human samples from the general population

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Austria												
Neuberger *et al.* (1991)	Blood		90	BSI								
	Occup. physicians	(2)		(med.)	16 (8–24)	–	–	–	–	–	–	
	MWI plant											
	External workers	(11)			15 (< 5–23)	–	–	–	–	–	–	
	External referents	(6)			13 (< 5–23)	–	–	–	–	–	–	
Riss *et al.* (1990)	Brixlegg; blood from	(1)	88	CSN	55.0	780	40	412	ND	32.5	–	
	farmer	(1)			13.1	92.4	6.4	242	ND	116	–	
Canada												
Ryan *et al.* (1985b)	Adipose;		79–81	BSIW								
	Kingston[a]	(13)			12.4 ± 5.8	–	–	–	–	–	–	
	Ottawa[a]	(10)			8.6 ± 4.4	–	–	–	–	–	–	
Ryan *et al.* (1985c)	Adipose;			BSIW								
	Québec	(5)	72		ND[b]	12.5 (4 pos.)	–	42.6	–	83.6	756	
		(10)	76		5.4 (5 pos.)	11.6	–	63.1	–	70.7	628	
	British Columbia	(5)	72		10.7	21.7	–	180	–	444	1355	
		(10)	76		7.5 (3 pos.)	11.6	–	117	–	160	1304	
	Maritimes	(10)	76		5.3 (7 pos.)	8.3	–	64.0	–	82.1	572	
	Ontario	(6)	76		6.1 (5 pos.)	6.1	–	40.3	–	116	528	
	Prairies	(10)	76		12.7 (1 pos.)	13.3	–	97.9	–	247	843	
	E. Ontario[a]	(10)	80		10.0 ± 4.9	13.2 ± 4.0	–	90.5 ± 38.9	–	116 ± 41.8	611 ± 226	
LeBel *et al.* (1990)	Adipose;			BSO								
	Ontario	(76)	84		11.2 ± 7.8 (1.4–49.1)	23.7 ± 11.5 (3.4–65.7)	– –	172.4 ± 74.1 (31.2–533.8)	22.1 ± 9.2 (6.9–53.2)	231.7 ± 181 (69.5–1242)	1037 ± 712 (194–5024)	65.9 ± 31.5 (10.9–184)
	Kingston[a]	(13)	79–81	BSO	19.5	27.5	–	212.3	31.7	342.3	1627	88.3
	Ottawa[a]	(10)			8.7	22.0	–	178.5	20.9	244.9	1154	61.6
Teschke *et al.* (1992)	British Columbia; adipose, residents of forest industry region	(41)	90–91	CSO	4.2 (1.8–9.2)	14 (4.1–26)	15 (2.8–33)	137 (33–313)	17 (6.0–39)	136 (42–300)	500 (67–1333)	29.1 (8.4–56.4)

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
China												
Ryan *et al.* (1987)	Shanghai; Adipose		84	BSOW[c]								
	LC, 58%	(1)			< 2	< 2		< 2		< 2	< 10	
	LC, 50%	(1)			< 2	< 2		< 2		< 2	< 10	
	LC, 73%	(1)			< 2	< 2		< 2		< 2	70	
	LC, 70%	(1)			< 2	5.3		15		18	122	
	LC, 73%	(1)			< 2	< 2		9.6		27[d]	373	
	LC, 72%	(1)			< 2	< 2		9.5		< 2	63	
	LC, 76%	(1)			< 2	6.5		19		< 2	59	
Schecter (1994)	Blood, general population;		92	BSOW (pool)								
	Age 15–19 y	(50)			< 1.2	1.6	1.8	4.3	1.7	11.6	104.1	4.8
	Age over 40 y	(50)			< 1.2	3.1	3.8	4.9	2.6	17.5	117.0	5.7
Finland												
Rosenberg *et al.* (1995)	Plasma, general population; Age 41 (28–60)		89–90	BSIW	4.1 (1.3–10)	17 (7.0–45)	4 (1.8–6.1)	150 (87–216)	12 (5.8–26)	132 (36–317)	804 (369–1745)	49 (20–99)
France												
Huteau *et al.* (1990a)	Paris; adipose tissue	(8)	< 90	BSO	10.3 (6 pos.) (2.9–23)	9.8 (1 pos.)	5.7 (2 pos.) (4.9–6.4)	46.7 (7 pos.) (28.6–61.6)	11.0 (2 pos.) (8.6–13.3)	164 (7 pos.) (80.4–232)	624 (8 pos.) (362–887)	
Germany												
Beck *et al.* (1989b)	Hamburg; adipose	(20)	86	BSIW	7.2 (1.5–1.8)	21 (8.8–48)	19 (8.7–29)	89 (35–129)	12 (5.8–20)	101 (39–216)	591 (212–1061)	56 (18–122)
Thoma *et al.* (1990)	Munich		< 89	BSO								
	Adipose	(28)			8.0 (2.6–18)	16.4 (7.7–40.4)		94.7 (35.7–178.2)		107 (35.1–246)	373 (117–789)	
	Liver	(28)			16.4 (1.0–88.9)	20.1 (7.3–58.7)		166.8 (56.4–615.1)		1002 (95.7–3463)	4416 (473–15259)	
	Adipose, infants	(8)			3.0 (< 1–7.0)	5.4 (1.4–13.1)		25.5 (4.5–68.8)		27.1 (11.8–51.1)	104.9 (55.3–180)	

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Germany (contd)												
Schecter *et al.* (1991a)	Whole blood	(4)	< 90		4.0	17.5		109	9.8	185	761	69
	Adipose	(4)	< 90		5.1	21.5		101	8.1	153	653	69
Päpke *et al.* (1992)	General population; blood	(102)	89–90	BSOW	3.6 (0.6–9.1)	13.8 (2.1–39.0)	1.9 (1.0–33.0)	54.6 (15.0–124)	10.6 (0.5–71.0)	92.4 (19.0–280)	610 (145–1524)	40.8 (11.6–93.5)
Kieselrotstudie (1991)	General population; blood	(56)	91	BSOW	4.5 (ND–12)	17.3 (6.7–43)	16.9 (3.6–38)	54.5 (18–110)	11.4 (4.7–23)	98.3 (30–210)	565 (180–1100)	44.4 (16.9–98)
Päpke *et al.* (1993b)	General population; blood	(44)	92	BSOW	3.7 (1.0–8.8)	8.3 (2.8–20.8)	10.2 (3.6–19.4)	35.5 (7.5–99.0)	5.9 (1.8–15.8)	56.7 (16.7–159	462 (126–1267)	26.0 (12–61)
Schrey *et al.* (1992)	General population; blood	(95)	91	BSOW	4.62 (1.2–12)	18.0 (5.6–44)	16.3 (3.9–38)	45.9 (12–110)	9.26 (2.9–22)	87.2 (21–210)	446 (140–950)	42.7 (11.2–114)
Päpke *et al.* (1994b)	General population; blood	(70)	93	BSOW	3.2 (0.5–8.7)	7.2 (3.5–16.0)	7.9 (3.4–21.8)	28.9 (9.5–71.4)	5.4 (0.5–13.8)	46.9 (13.6–143)	389 (99.9–945)	21.7 (10.3–48.8)
Päpke *et al.* (1996)	General population; blood	(134)	94	BSOW	2.9 (1.0–7.8)	6.3 (1.6–15.4)	6.9 (ND–22)	26.7 (5.3–62.2)	4.9 (1.3–11.9)	45.3 (8.6–115.8)	370 (90.3–949)	19.1 (5.2–43.9)
			97	BSOW								
	Age 18–71 y	(139)			2.3 (ND–4.9)	5.9 (1.7–12.1)	5.7 (2.0–15.7)	22.6 (3.7–60.3)	3.8 (1.5–11.1)	33.0 9.5–93.5)	293 106–664)	16.1 (7.3–33.6)
	Age 18–30 y	(47)			2.1 (1.0–4.3)	4.8 (1.7–9.8)	5.0 (2.0–15.7)	16.8 (3.7–33.6)	3.6 (1.8–6.7)	34.0 (9.8–56.7)	278 (108–530)	131 (7.3–13.1)
	Age 31–42 y	(48)			2.2 (ND–4.4)	5.9 (2.5–11)	5.7 (2.3 (10.5)	24.4 (8.6–42.5)	3.9 8.6–42.5)	33.2 12.4–79.4)	310 (114–597)	16.3 (7.9–22.3)
	Age 43–71 y	(44)			2.8 (ND–4.9)	7.0 (3.6–12.1)	6.5 (2.8–11.4)	26.7 (6.8–60.3)	3.9 (1.9–11.1)	31.5 (9.5–93.5)	280 (106–664)	19.1 (10.1–336)
Wittsiepe *et al.* (1993)	Marsberg; vicinity of copper smelter, blood	(56)	91	BSOW	4.6 (ND–12)	19.4 (6.7–80)	16.3 (5.3–51)	57.9 (19–110)	11 (3.4–31)	93.2 (18–248)	666 (120–1770)	52.7 (22.1–231)
Körner *et al.* (1994)	Mammary tumour tissue	(7)		BSO	7.8 (5.1–11.7)	16.5 (11.4–26.2)		86.5 (41–121)		128 (72–183)	608 (186–1361)	50.1 (27.4–76.0)
Wuthe *et al.* (1990)	Metal reclamation plant neighbourhood; blood	(22)	89	BSOW	3.4 (1.3–6.2)	12.4 (2.9–21)		59.2 (29.7–118)		83.5 (23–238)	506 (176–2126)	31.0 (16.1–80.4)

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Germany (contd)												
Wuthe *et al.* (1993)	One woman; blood	(1)	92	BSOW	2.2	5.7		31.3		41.1	229	
Ewers *et al.* (1994)	Allotment gardeners; blood	(21)	92	BSOW	5.8 (2.4–14)	17 (11–26)		69 (37–110)		83 (20–150)	390 (270–680)	44.3 (29.2–81.1)
Beck *et al.* (1994)	Infants, 3–23 months		< 93	BSOW								
	Adipose tissue	(8)			1.1 (< 0.2–3.9)	4.5 (0.4–14)	2.9 (0.3–9.2)	14 (2.1–37)	3.4 (0.8–9.5)	18 (5.1–57)	114 (43–341)	11 (2.1–36)
	Liver	(8)			1.4 (< 1–4.6)	5.0 (< 1–14)	6.5 (< 2–17)	19 (3.0–54)	5.7 (< 2–16)	115 (20–396)	1221 (375–2916)	28 (4.7–88)
	Spleen	(8)			2.2 (< 1–< 10)	5.5 (1.1–20)	11 (1.6–50)	24 (< 5–113)	4.9 (0.8–18)	76 (16–236)	166 (107–281)	20 (4.3–77)
	Thymus	(8)			3.4 (< 1–7.5)	4.4 (<2 –< 15)	5.6 (2.4–< 15)	15 (7.5–24)	4.6 (1.5–< 15)	60 (19–155)	782 (446–1500)	19 (8.4–39)
	Brain	(8)			–	–	–	–	–	–	–	< 1
Jödicke *et al.* (1992)	Infant, 3 months; stool	(1)	91	BSO	< 2	< 5	< 5	36.7	< 5	152	1367	13.6
	Mother's milk	(2)			1.0–2.1	4.6–5.3	3.8–4.2	25.4–31.4	2.8–3.0	26.7–29.7	104–118	14.6–18.6
Welge *et al.* (1993)	Blood		92	BSOW								
	Vegetarians	(24)			3.4 (1.2–5.4)	14.1 (6.5–25)	12.3 (4.6–23)	36.0 (17–66)	6.8 (3.7–12)	70.2 (32–120)	447 (180–1100)	32.6 (14.6–52.9)
	Non-vegetarians	(24)			3.6 (1.2–11)	15.5 (5.8–43)	14.7 (5.4–36)	39.9 (18–110)	8.3 (2.9–22)	80.0 (24–160)	456 (150–950)	34.3 (14.3–98.0)
Abraham *et al.* (1995a)	Mother's blood	(3)	93–94	BSOW	[1.6]	[4.5]	[4.2]	[18.3]	[3.1]	[26.2]	[367]	[12.4]
	Placenta	(3)			[2.7]	[4.5]	[2.5]	[8.7]	[1.7]	[11.7]	[114]	[11.1]
	Umbilical cord	(3)			[< 1.0]	[2.6]	[2.6]	[8.9]	[1.8]	[9.0]	[93.6]	[6.5]
	Meconium	(1)			[1.4]	[3.2]	[2.5]	[9.9]	[2.2]	[12.2]	[152]	[7.7]
Guam												
Schecter *et al.* (1992)	Guam Island; whole blood	(10)	89	BSOW	2.6	14.7	8.3	62.1	15.5	163	749	28

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Japan												
Ryan (1986)	Adipose;		84	BSIW								
	Age 21 y; LC, 43%	(1)			ND	–		70		–	560	
	Age 33 y; LC, 37%	(1)			ND	–		146		–	650	
	Age 46 y; LC, 67%	(1)			9.7	–		90		–	1600	
	Age 55 y; LC, 80%	(1)			5.5	–		61		–	2400	
	Age 64 y; LC, 71%	(1)			3.2	–		66		–	860	
	Age 70 y; LC, 56%	(1)			8.0	–		84		–	2100	
	Mean; LC, 59%				6.6	–		86		–	1360	
Ono *et al.* (1986)	Cancer patients; adipose	(13)	85	CSI[c]	9 (6–18)	15 (3–36)	8 (5–14)	70 (26–220)	12 (4–44)	77 (29–180)	230 (25–1100)	
Ogaki *et al.* (1987)	Adipose;		< 84	CRI								
	Big city	(9)			13 (2.7–33)	22 (1.6–45)		130 (64–290)		190 (27–840)	2000 (130–10000)	
	Rural town	(3)			8.1 (6.4–11)	23 (15–30)		34 (20–58)		27 (13–40)	660 (160–1400)	
		(5)			15 (9.3–25)	18 (12–32)		77 (40–160)		58 (27–87)	250 (160–390)	
Hirakawa *et al.* (1991)	Controls; adipose	(8)	< 91	BSI	3 (1–5)	14 (4–18)	–	70 (21–130)	–	–	563 (180–1330)	17[c] (5–24)
Muto *et al.* (1991)	Cancer patients;		84–86	CSI[c]								
	Lung	(5)			2	9.3	4.1	3.8	6.2	112	249	
	Liver	(5)			2.2	31.5	8.1	31.6	4.9	78.1	165	
	Kidney	(5)			1.7	11.5	1.2	18.8	3.7	12.4	17.3	
	Pancreas	(5)			1.2	37.4	123	62.7	16.8	25.2	39.9	
	Spleen	(5)			ND	84.0	11.4	67.5	1.2	113	243	
	Gonad	(5)			2.7	25.3	5.7	36.7	1.0	23.2	110	
	Gall-bladder	(5)			ND	11.0	ND	1.5	ND	90.3	132	
	Muscle	(5)			ND	17.0	6.2	18.0	1.5	304	344	
Masuda (1996)	Control; serum		91–92	BSIW	3.1	[9.2]	[4.3]	[38.8]	[8.3]	[46]	[1140]	[26]

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Netherlands												
van Wijnen *et al.* (1990)	Fetus; liver	(4)	< 90	BSIW	0.23	0.09	0.09	0.29	0.07	1.03	8.8	
	Infant not nursed											
	Liver	(1)			0.03	0.07	0.04	0.16	0.03	0.29	3.87	
	Fat	(1)			3.4	2.13	ND	7.07	ND	12.3	87.4	
	Infant nursed											
	Liver	(1)			0.29	0.58	0.49	2.77	0.52	6.84	67.9	
	Fat	(1)			3.98	6.93	3.18	29.98	2.95	10.0	105.1	
	Placenta	(1)			0.26	0.53	0.21	0.42	0.08	0.59	4.22	
New Zealand												
Smith *et al.* (1992a)	Control group for 2,4,5-T applicators; serum		88	BSIW	5.6 ± 1.1	8.8 ± 0.7	5.7 ± 0.4	23.3 ± 4.9	8.2 ± 0.6	119 ± 18.4	759 ± 93	
Norway												
Johansen *et al.* (1996)	Blood		93	BSO								
	Controls	(10)			3.6 (0.2–7.0)	5.9 (0.5–10.6)	2.4 (0.9–3.4)	14.7 (2.7–24.5)	4.3 (0.6–7.3)	54.1 (10–179)	478 (52–951)	2.1[f]
	Moderate crab intake	(15)			7.7 (3–13.6)	17.3 (6.9–34.8)	8.0 (ND–30.1)	27.6 (13.1–48.2)	8.6 (ND–43.5)	45.5 (21–77)	336 (157–440)	60.8[f]
	High crab intake	(9)			11.0 (6.3–22.4)	28.3 (15.4–45)	10.8 (4.0–17.4)	39.1 (16.9–63.7)	9.9 (5.9–20.9)	33.3 (17–64)	267 (104–363)	109.6[f]
Russian Federation												
Schecter *et al.* (1992)	Whole blood		88–89	BSOW								
	Baikalsk	(8)		(pool)	3.7	4.7	4.7	6.3	2.0	9.6	57	18
	St Petersburg	(60)			4.5	9.3	2.1	8.5	2.4	14	89	17
Spain												
Jiménez *et al.* (1995)	Madrid, unexposed; serum	(11)	93	BSO	1.52 ± 1.19 (0.61–3.9)	4.09 ± 0.91 (2.25–5.71)	2.75 ± 1.18 (1.55–5.10)	32.6 ± 12.4 (18.8–57.2)	5.81 ± 2.67 (2.01–11.4)	71.5 ± 34.7 (32.5–137.2)	397 ± 174 (117–690)	
Gonzaléz *et al.* (1997)	Mataro; blood, 10 pools	(198)	95	BSOW	1.6	4.9		39.4		70.1	484	13.3

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Sweden												
Rappe (1984b)	Background; adipose	(6)	82	CS[c]	1.5	12		11		73	240	
Nygren *et al.* (1986)	Adipose;		84	BSIW[c]								
	Unexposed	(18)			3 (2–6)	9 (4–18)		12 (3–18)	4 (3–5)	85 (12–176)	421 (98–679)	
	Cancer patients	(17)			3 (2–9)	9 (4–24)		18 (3–55)	4 (3–13)	100 (12–380)	408 (90–620)	
	Non-cancer patients	(14)			3 (2–6)	9 (3–18)		12 (8–18)	4 (3–5)	85 (20–168)	421 (182–763)	
Rappe (1992)	Blood;		90	BSIW								
	No fish consumption				1.8	5.7	2.8	35	5.7	56	357	17.5
	Normal fish consumption				2.5	7.6	3.0	43	6.0	80	458	25.8
	High fish consumption				8.0	16	3.9	48	6.5	71	473	63.5
Svensson *et al.* (1995a)	Blood pool[a]			BSI								
	Sea of Bothnia											
	Fishermen				27	35	6.2	66	12	84	551	154[f]
	Controls				6.1	12	4.2	38	6.6	67	574	40[f]
	Baltic proper											
	Fishermen				6.9	20	4.6	44	ND	58	351	80[f]
	Controls				4.0	10	3.4	32	5.7	58	364	37[f]
	Baltic south											
	Fishermen				17	33	8.8	75	ND	110	585	131[f]
	Controls				6.4	14	5.8	53	8.5	100	605	54[f]
	West coast											
	Fishermen	100			5.7	11	4.9	36	6.0	54	367	42[f]
	Controls	88			4.0	13	ND	34	ND	85	553	39[f]
Hardell *et al.* (1995)	Blood		> 86	BSI								
	Cancer patients	(7)			10.1 (< 0.4–36)	24 (9–57)	1.2 (0.9–2.6)	45.3 (13–130)	6.3 (2.6–13)	118 (9–380)	510 (154–1600)	64.7[2,f] (19.9–187)
	Non-cancer patients	(12)			2.8 (< 0.4–6)	9.6 (< 0.4–19)	< 1	12.2 (8–18)	3.7 (3–5)	85 (32–168)	413 (247–672)	29.7[f] (12.9–53.4)

Table 25 (contd)

Reference	Origin; sample description (and no.)	Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Switzerland											
Wacker *et al.* (1990)	Background		CSO[c]								
	Adipose (21)			–	24.1	15.3	144.3	–	195	1161	
	Liver (21)			–	1.1	1.1	10.5	–	81	491	
Taiwan											
Ryan *et al.* (1994)	Control children; serum	91	BSW	3.0	7.3		23		52	612	22.7
United Kingdom											
Duarte-Davidson *et al.* (1993)	Wales, 5 pools; adipose	90–91	CSO	< 10	23 (21–24)	37 (29–47)	182 (160–210)	28 (22–210)	154 (120–230)	816 (590–1100)	57
United States											
Ryan *et al.* (1985c)	New York State; adipose (6)	83–84	CSOW	6.4 ± 1.6 (3.7–8.3)	9.7 ± 2.4 (7.5–13.8)	–	57.8 ± 6.5 (46.2–64.2)	–	95.2 ± 29.2 (39.4–119)	585 ± 98 (428–695)	
Ryan *et al.* (1986)	New York State;	< 83	BSIW[c]								
	Adipose (3) (mean LC, 67%)			5.4 (3.7–8.4)	7.4 (7.8–11)	–	73.7 (61–130)	–	95 (53–120)	528 (201–700)	
	Liver (3) (mean LC, 24%)			2.4 (ND–4.6)	2.3 (ND–4.5)	–	36.8 (6.5–56)	–	34 (22–44)	250 (180–350)	
	Adrenal (2) (LC, 28.25%)			3.8–3.7	3.1–4.8	–	35–39	–	36–55	210–600	
	Bone marrow (1) (LC, 26%)			ND	12	–	30	–	48	540	
	Muscle (3) (mean LC, 11%)			< 2.5 (ND–2.5)	1.3 (1.2–1.5)	–	11.6 (5.8–21)	–	11.7 (5–16)	122 (76–170)	
	Spleen (2) (LC, 1.8–1.7%)			(ND–1.3)	(1.1–11)	–	(1.5–1.9)	–	(4.7–13)	(20–46)	
	Kidney (2) (LC, 3.0–4.0%)			ND	ND	–	(2.5–4.0)	–	(5.2–13)	(31–39)	
	Lung (LC, 2.2%) (1)			ND	ND	–	1.4	–	2.9	21	
Gross *et al.* (1984)	Adipose	84	BNN								
	Controls (11)			5.6 (ND–14)	–	–	–	–	–	–	
	Air Force scientists (3)			5 (4–6)	–	–	–	–	–	–	

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
United States (contd)												
Ryan (1986)	New York State;		85	BSIW[c]								
	Adipose											
	6 months(LC, 75%)				ND	–		4.5		–	130	
	22 y (LC, 83%)				2.2	–		74		–	920	
	Liver											
	6 months (LC, 4.0%)				ND	–		ND		–	22	
	22 y (LC, 4.4%)				ND	–		21		–	420	
Graham *et al.* (1985)	Background; adipose	(8)	< 84	CSI	5.4 (1.8–10)	–	–	–	–	–	–	
		(3)			7.7 (2–14)	10.3 (4–16)	–	–	–	–	–	
Schecter *et al.* (1986a)	Binghamton; adipose		< 84	CRO[c]								
		(1)			8.3	13.8	–	46.2	7.4	95.8	534	
		(1)			7.2	10.3	–	54.5	7.5	39.4	593	
		(1)			6.0	8.2	–	60.3	7.4	119	695	
		(1)			3.7	7.5	–	60.4	6.8	93.1	586	
	LC, 70.6% (46–88)	(8)		BNW	7.2 (1.4–17.7)	11.1 (5.2–25.2)	–	95.9 (46.2–355)	–	164 (53–691)	707 (214–1931)	
Patterson *et al.* (1986a)	Georgia and Utah; adipose		< 86	BSIW								
	Black women, 60.6 years old	(7)			12.2	–	–	–	–	–	–	
	Black men, 55.9 years old	(7)			10.4	–	–	–	–	–	–	
	White women, 52.2 years old	(8)			10.9	–	–	–	–	–	–	
	White men, 54.8 years old	(9)			8.8	–	–	–	–	–	–	
Stanley *et al.* (1986)	General population; adipose	(46)	82	BSO	5.0 ± 2.8 (< 1–10)	32 ± 38 (< 1–180)		72 ± 70 (7.9–330)		87 ± 78 (< 23–390)	560 ± 290 (64–1250)	
Patterson *et al.* (1986b)	Missouri; adipose	(57)	86	BSIW	7.4 (1.4–20.2)	–	–	–	–	–	–	

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
United States (contd)												
Centers for Disease Control Veterans Health Studies (1988)	Non-Viet Nam veterans; serum	(97)	87	BSIW	4.1 ± 2.3 (ND–15)	–	–	–	–	–	–	
Nygren *et al.* (1988)	Era control; serum	(1)	< 88	BSIW	1.5	9.0	< 4	36	< 4	59	84	
		(1)			7.3	15	11.6	191	< 5	59	675	
		(1)			7.1	26	17.6	231	38	428	3246	
		(1)			2.4	16	3.6	47	4.0	188	468	
		(1)			2.2	6.9	4.7	36	6.0	65	1747	
		(1)			5.5	14	8.7	64	10.1	72	1052	
		(1)			6.0	13.5	13.2	69	15	105	566	
Andrews *et al.* (1989)	Missouri controls		86	BSIW								
	Adipose											
	Men	(51)			6.8 ± 4.1 (ND–20)	–	–	–	–	–	–	
	Women	(77)			7.2 ± 4.0 (1.4–20.2)	–	–	–	–	–	–	
Fingerhut *et al.* (1989)	Referents; serum	(19)	< 89	BSIW	8.2 (3.7–17.1)	–	–	–	–	–	–	
Schecter *et al.* (1989b)	One patient[c]		< 89	N								
	Abdominal fat (LC, 75%)	(1)			5.7	7.8	–	64	–	110	680	
	Subcutaneous fat (LC, 75%)	(1)			6.0	8.2	–	60	–	120	700	
	Adrenal fat (LC, 28%)	(1)			3.8	3.1	–	35	–	55	600	
	Bone marrow (LC, 26%)	(1)			ND	12	–	30	–	48	540	
	Liver (LC, 6%)	(1)			ND	ND	–	6.5	–	22	220	
	Muscle (LC, 9%)	(1)			ND	1.2	–	7.9	–	14	170	
	Spleen (LC, 1.8%)	(1)			ND	11	–	1.9	–	13	46	
	Kidney (LC, 3.0%)	(1)			ND	ND	–	2.5	–	5.2	31	
	Lung (LC, 2.2%)	(1)			ND	ND	–	1.4	–	2.9	21	

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
United States (contd)												
Wendling *et al.* (1990a)	Faeces; laboratory workers		< 90	BSO								
	Pool				0.61 ± 0.09	–	–	–	–	–	–	
	Individual sample				0.74	–	–	–	–	–	–	
Schecter *et al.* (1990b; 1991a)	US veterans		< 90	BSOW								
	Adipose	(20)			6.9	7.7		59.3	6.3	82.5	429	24
	Plasma	(20)			5.7	7.1		56.0	8.5	107.9	843	23
Kang *et al.* (1991)	Adipose		78	N								
	Viet Nam veterans	(36)			13.4	20.6		170.4	19.4	276	1262	
	Non-Viet Nam veterans	(79)			12.5	18.3		152.9	17.2	245	1109	
	Civilians	(80)			15.8	18.3		165.1	17.9	300	1393	
Piacitelli *et al.* (1992)	Referents; serum	(79)	87–88	BSIW	7 (2–20)	12 (3.5–51)	13 (3.2–58)	84 (17–183)	13 (3.6–33)	160 (39–460)	1010 (480–2300)	
Patterson *et al.* (1994)	Atlanta; adipose		84–86	BSIW								
	All data	(28)			10.4 (1.6–38)	–	–	–	–	–	–	
	Men	(14)			7.4 (1.6–19.4)	–	–	–	–	–	–	
	Women	(14)			11.6 (3.1–24.3)	–	–	–	–	–	–	
	General population	(4)			4.4 (1.6–8.3)	11.6 (8.5–16)	5.1 (3 pos.) (3.7–6.4)	94.2 (81–121)	16.9 (12.5–22)	56 (2 pos.) (48–63)	446 (2 pos.) (396–495)	
Schecter *et al.* (1994b)	Placenta, pooled	(14)	< 94	BSOW	2.4	4.0	2.4	15.9	3.2	36.2	282	10.1
	Placenta	(1)			2.0	9.5	6.4	11.2	5.3	47.7	236	14.4
	Blood, pool	(50)			3.8	9.3	9.8	72.1	11.9	118.6	793.9	27.0
Schecter *et al.* (1996b)	Fetal tissue, 8–14 weeks, pool	(10)	94		1.4	2.0	2.3	8.9	1.7	22.9	98.8	5.3

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
United States (contd)												
Schecter *et al.* (1996c)	Adipose	(5)	96	BSNW	1.3	2.8	3.3	22.3	3.1	45.4	214	8.5
	Blood, before nursing	(5)			1.7	3.2	4.0	19.9	4.2	57.9	374	9.9
	Placenta	(5)			2.7	4.5	2.5	10.2	2.6	21.5	103	9.4
	Cord blood	(5)			1.3	1.3	1.6	10.5	2.2	24.1	95.8	4.7
	Mother's milk	(5)			1.4	2.5	3.0	20.1	3.5	34.0	104	8.1
	Blood after 4–8 weeks of nursing	(5)			1.5	2.6	3.2	18.7	4.1	45.2	226	8.3
Schecter *et al.* (1996d)	Blood, pool	(100)	96	BSIW	4.3	8.7	9.7	63.7	7.8	102	781	27.1
	Serum, pool	(100)			4.2	9.8	10.6	67.9	10.7	117	878	27.6
Viet Nam												
Schecter *et al.* (1986b)	Adipose		84	BSOW[c]					–			
	North Viet Nam; LC, 50%	(7)			< 2	< 2	–	4.6		19.0	36.1	
	South Viet Nam; LC, 60%	(13)			22.1 (10 pos.)	9.9	–	46.7	–	105	514	
Schecter *et al.* (1986c)	Adipose		84	BSOW								
	North Viet Nam; LC, 56%	(9)			< 2	3.8 (1 pos.)		11.4 (6 pos.) (ND–23.9)	–	28.8 (6 pos.) (ND–56.3)	104 (8 pos.) (ND–205)	
	South Viet Nam; LC, 62%	(15)			27.9 (12 pos.) (ND–103)	15.4 (14 pos.) (ND–43.3)		99.8 (22.6–347)		178 (13.7–710)	1326 (141–3410)	
Schecter *et al.* (1989c)	Adipose		84–88	BN								
	North Viet Nam; pool	(10)			< 2	–	–	–	–	–	–	
	South Viet Nam	(27)			19 (16 pos.) (ND–36)	–	–	–	–	–	–	
Nguyen *et al.* (1989)	Ho Chi Minh City; adipose; mean LC, 76%	(9)	84–85	BSNW	23 (4–103)	12 (4.5–27)		89 (23–261)		272 (77–710)	2114 (25–4284)	
Hoang *et al.* (1989)	Adipose		< 89	N								
	North Viet Nam	(11)			1.3[a] (1 pos.)	–	–	–	–	–	–	
	South Viet Nam	(44)			20.5 (40 pos.)	–	–	–	–	–	–	
Huteau *et al.* (1990b)	South Viet Nam; adipose	(27)	< 90	BRO	16.2 (24 pos.) (1.5–129)	17.5 (4 pos.) (9.7–34.6)	11.7 (16 pos.) (4.5–23.5)	56.1 (23 pos.) (9.5–157)	12.7 (22 pos.) (2.4–48.5)	95.5 (23 pos.) (8.4–303)	569 (25 pos.) (35–2113)	

Table 25 (contd)

Reference	Origin; sample description (and no.)		Coll. period	Anal. meth.	PCDD concentration (ng/kg fat)							
					TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
					2378	12378	123478	123678	123789	1234678		
Viet Nam (contd)												
Schecter *et al.* (1990c)	Adipose		80s	BSO								
	North Viet Nam	(10)			1.4	2.5	1.4	6.0	1.2	30.1	230	
	South Viet Nam	(13)			6.7	6.4	5.0	27.6	6.1	69.2	470	
Schecter *et al.* (1990d)	Liver, stillborn infants	(1)	< 89	BSOW	4.3	7.8	4.5	5.1	2.9	15	64	12
		(1)			3.5	8.3	3.8	6.1	2.3	11	65	12
		(1)			1.3	3.8	4.1	4.6	3.9	22	58	6.4
Schecter *et al.* (1992)	Whole blood; pool		< 91	BSOW								
	North Viet Nam	(82)			2.2	4.1	3.7	13.4	4.8	25.5	132	15
	South Viet Nam	(383)			14.6	9.1	7.5	33.8	9.4	87.3	696	36
Schecter *et al.* (1995)	Blood		91–92	BSOW								
	South Viet Nam	(433)			12.9	8.0	6.6	29.9	8.3	77.2	616	31.3
	Central Viet Nam	(183)			13.2	16.3	13.0	46.2	13.4	78.1	751	50
	North Viet Nam	(82)			2.2	4.1	3.7	13.4	4.8	25.5	132	15.3
Le *et al.* (1995)	Blood; pool			BSOW								
	North Viet Nam	(133)	91	pool	2.7	–	–	–	–	–	–	15.2
	Military region I	(315)	91–92		12.8	–	–	–	–	–	–	56.4
	Military region II	(176)	91–92		4.2	–	–	–	–	–	–	30.6
	Military region III	(1443)	91–92		12.1	–	–	–	–	–	–	34.5
	Military region IV	(569)	91–92		8.05	–	–	–	–	–	–	27.4

Data presented are arithmetic 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, high-resolution mass spectrometry; C, low-resolution mass spectrometry; 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; +, contains 50% of detection limit; LC, lipid content; pos., positive; S, south; N, north; C, control; [ ] Calculated by the Working Group

[a] Overlap between these studies

[b] Detection limit, > 2–3 ng/kg

[c] WW, weight-based

[d] Contained also 22 ng/kg 1,2,3,4,6,7,9-HpCDD

[e] PCDD-I-TEQ only

[f] Nordic TEQ

[g] 50 fishermen and 150 controls for the 3 groups

[h] Mean level, ND are included at 1 ng/kg (half the detection limit)

Ryan *et al.* (1985c) analysed 46 and 10 adipose tissue samples originating from people who had died accidentally in 1976 and 1972, respectively, in Canada for various reasons, as well as 10 others from deceased hospital patients. Total PCDD levels were about an order of magnitude higher than total PCDFs. PCDD levels increased with increasing chlorination from 2,3,7,8-TCDD (average, 5–13 ng/kg) to OCDD (average, 528–1355 ng/kg). Only the penta-, hexa- and hepta-CDF congeners were detected at these levels (10–60 ng/kg); TCDF and OCDF were not detected.

Human adipose tissue samples obtained during autopsies in five Canadian municipalities within the Great Lakes basin (Cornwall, London, St Catherine's, Welland, Windsor) were analysed for PCDDs and PCDFs (LeBel *et al.*, 1990). The mean congener levels for male and female donors in each municipality were similar to those previously reported. The ages of the 40 men ranged from 29 to 83 years (mean, 63 years) and that of the 36 women from 12 to 88 years (mean, 69 years). No significant differences in congener levels between male and female donors or between municipalities were detected. However, in four cases, the values for women were 15–45% higher than the corresponding values for men. Additionally, a positive correlation with age was observed for the levels of several congeners as well as for the I-TEQ.

In connection with the forestry industry, there has been concern that by-products of pulp and paper production and chlorophenol fungicides used in sawmills may result in residents being environmentally or occupationally exposed to PCDDs/PCDFs. To examine this possibility, PCDD and PCDF levels were measured in the adipose tissue of 41 British Columbians selected to match the age and sex distribution of the exposed population. The group consisted of 18 men and 23 women. The mean age of the subjects was 45 years (range, 18–77 years) and the mean weight was 96 kg (range, 50–193 kg). The mean of the 41 samples was 29.1 ng I-TEQ/kg (range, 8.4–56.4). The highest 2,3,7,8-TCDD level measured was 9.2 ng/kg (lipid-based) (Teschke *et al.*, 1992).

Ryan (1986) examined the relationship between the PCDD/PCDF level and age (between 14 and 76 years) in 46 Canadian individuals.

(*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 were low. At a detection limit of 2.0 ng/kg (based on wet weight), no 2,3,7,8-TCDD was found.

In connection with a study to examine the exposure of agricultural workers to PCP (for snail control) and PCDDs/PCDFs, Schecter (1994) reported data on two pooled age-matched control groups. For the two groups from the general population, very low blood concentrations of PCDDs/PCDFs were found at 4.8 ng I-TEQ/kg (age, 15–19 years) and 5.7 ng/kg (over 40 years), respectively. These are consistent with values found in mothers' milk (see Section 1.4(*b*)).

(*d*) *Finland*

In a study of pulp and paper mill workers in Finland (Rosenberg *et al.*, 1995), a comparison group with no known exposure was analysed. This control group consisted of 14 persons with a mean age of 41 years (range, 28–60 years). The mean total I-TEQ level in plasma was 49 ng/kg (range, 20–99 ng/kg) (see Section 1.3.1(*a*)(iv)).

(*e*) *France*

The levels 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*

The first German data on background levels of PCDDs in human adipose tissue were reported by Beck *et al.* (1989b). Concentrations in 20 unexposed individuals (mean age, 50 years) were used for comparison in a study of 45 occupationally exposed employees at a chemical plant in Hamburg. The values for the comparison group ranged between 18 and 122 ng I-TEQ/kg (mean, 56 ng/kg).

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 tissues were demonstrated for most of the isomers (see **Table 26**). 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.

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**). The results suggest a decrease in PCDD/PCDF blood levels in Germany over the past decade.

Age-related increases in blood levels of most of the PCDD congeners and the I-TEQ have been reported (Sagunski *et al.*, 1993; Schrey *et al.*, 1992; Päpke, 1996).

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 monograph on PCDFs in this volume; Section 1.3.2(*c*)). The copper smelter was in operation until 1945. In 1991, high levels of PCDDs/PCDFs were found in materials from the slag dumps (10–100 µg I-TEQ/kg). The median I-TEQ values of the Marsberg group (43.2 ng/kg blood lipid) and the reference group from Steinfurt (43.0 ng/kg blood lipid) 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).

Near a metal reclamation plant in Baden-Württemberg, Rastatt, 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 and 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 traced 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).

Table 26. Concentrations of PCDD isomers in adipose and liver tissues of German adults and adipose tissue of infants

Compound	Adult; ratio liver : adipose	Adipose tissue; ratio infant : adult
TCDD	2.05	0.55
PeCDD	1.22	0.30
HxCDD	1.76	0.23
HpCDD	9.39	0.23
OCDD	11.83	0.19

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

Table 27. Time trend in background data on concentrations of PCDDs/PCDFs in human blood

Reference	Collection year	No.	Mean I-TEQ ng/kg, lipid-based
Päpke *et al.* (1989b)	1988	10	[45.8]
Päpke *et al.* (1992)	1989–90	102	40.8
Kieselrotstudie (1991)	1991	56	44.4
Päpke *et al.* (1993b)	1992	44	26.0
Schrey *et al.* (1992)	1991	95[a]	42.7
Päpke *et al.* (1994b)	1993	70	21.7
Päpke *et al.* (1996)	1994	134	19.1
Päpke *et al.* (1996)	1996	139	16.1

[a]Contains 56 samples from Kieselrotstudie (1991)
[Calculated by the Working Group]

In Rheinfelden in southern Germany, soil concentrations of PCDDs/PCDFs of up to approximately 1000 ng German TEQ/kg were found. The source of contamination of the soils was identified as residues from a PCP production process. Locally produced food, such as eggs, chicken and vegetables, with high PCDD/PCDF levels were reported to be an important source of elevated human levels. Good agreement between human blood and milk concentrations was demonstrated in one woman (Wuthe *et al.*, 1993).

PCDD/PCDF levels were determined in the venous blood of 21 allotment gardeners from Duisburg (Ewers *et al.*, 1994). Soil analysis showed elevated levels of PCDDs/-PCDFs in garden soil (range, 16.4–77.6 ng I-TEQ/kg). Vegetable plants also had

elevated levels (up to 65.6 ng I-TEQ/kg). The mean I-TEQ of 44.3 ng/kg (range, 29.2–81.1 ng/kg) in blood fat of the gardeners was within the range of a control group.

In seven breast cancer patients without known occupational exposure to PCDDs/-PCDFs, the levels of PCDD/PCDF congeners in mammary carcinoma tissue were not elevated above the concentrations in tumour-free tissue (Körner *et al.*, 1994).

PCDD/PCDF levels in various organs from infants were measured by Beck *et al.* (1990, 1994). Adipose tissue, liver, spleen, thymus and brain from eight infants (age, 3–23 months) who had mostly died from sudden infant death syndrome were analysed. Five of the infants had been breast-fed exclusively for between 21 and 91 days. The lipid-based PCDD/PCDF levels in liver, thymus and spleen were higher than those in adipose tissue. Very often the concentrations were below the detection limits. The lowest levels were found in brain.

Elimination of PCDDs/PCDFs through the faeces was studied in a three-month-old breast-fed infant (Jödicke *et al.*, 1992). Little excretion of Cl_4–Cl_6 congeners was found. The data suggest that more than 90% had been absorbed by the infant. However, 1,2,3,4,6,7,8-HpCDD as well as OCDD were found to be highly concentrated in stool fat (by a factor of 5–12 over the concentration in milk fat).

(*g*) *Guam*

Schecter *et al.* (1992) reported a mean PCDD/PCDF level of 28 ng I-TEQ/kg in blood samples from 10 residents of the Pacific island of Guam. This was suggested to be due to consumption of food mainly imported from the United States and Japan. [A local source cannot be excluded.]

(*h*) *Japan*

In adipose tissue from six individuals 20–70 years old in Japan collected in 1984 (Ryan, 1986), the mean 2,3,7,8-TCDD concentration was 6.6 ng/kg and that of OCDD 1360 ng/kg.

Thirteen samples of human adipose tissue from cancer patients were analysed for Cl_4–Cl_8 PCDDs and PCDFs (Ono *et al.*, 1986). These compounds were identified in all of the analysed samples. 2,3,7,8-TCDD concentrations ranged from 6 to 18 ng/kg.

In a large study, more than 500 human milk and 17 adipose tissue samples were analysed for PCDDs (Ogaki *et al.*, 1987). Adipose tissues from inhabitants of a large city and its suburbs had higher levels of PCDDs that those of rural towns.

Muto *et al.* (1991) investigated the tissue distribution of 2,3,7,8-substituted PCDDs in humans who died of cancer. The values for OCDD were the highest in each organ and tissue. All data were wet weight-based, and no lipid concentrations for the tissues were reported.

(*i*) *The Netherlands*

Intake and faecal excretion of PCDDs/PCDFs in breast-fed infants at a range of ages were studied (Pluim *et al.*, 1993a). In three totally breast-fed infants, the amount of PCDDs/PCDFs consumed via breast milk and excreted in the stools was measured at the

ages of four, eight and 12 weeks. Intake was high, especially at the age of four weeks (mean, 257 pg/kg bw). A strong decline was observed in the second month, mainly due to a reduction in PCDD/PCDF concentrations in whole breast milk as a result of reduced fat content. With the exception of OCDD, faecal excretion of the congeners was below 5% of their intake, indicating a bioavailability of more than 95% from breast milk. No influence of age on faecal excretion of PCDDs/PCDFs during the first three months of life was observed.

(j) New Zealand

In a study to determine whether blood serum levels of 2,3,7,8-TCDD in a group of professional 2,4,5-T applicators in New Zealand were greater than those of a matched control group not involved in 2,4,5-T spraying, the mean level of 2,3,7,8-TCDD in the control group was 5.6 ng/kg ± 1.1 (standard error) (Smith *et al.*, 1992a) (see Section 1.3.1(*a*)(ii) and Table 17).

(k) Norway

PCDDs/PCDFs have been analysed in human blood in relation to consumption of crabs from a contaminated fjord area in Norway (Johansen *et al.*, 1996). The analyses were performed in three different groups: reference, moderate crab intake and high crab intake (age between 40 and 54 years). A significant increase in blood levels of many PCDD and PCDF congeners was found in crab consumers; the differences were greatest for several of the PCDFs that are characteristic of the contamination of marine biota in the fjord caused by a magnesium-producing plant. Almost all subjects in the high-intake group exceeded the tolerable weekly intake of 35 pg 2,3,7,8-TCDD/kg bw per week proposed by the Nordic Expert Group (see Section 1.5).

(l) Poland

Changes over time in PCDD/PCDF levels in the adipose tissue of a person with long-term exposure to PCP were studied (Górski *et al.*, 1984). The analyses, performed by GC-ECD, yielded half-lives of 1,2,3,6,7,8-HxCDD, 3.5 years; 1,2,3,4,6,7,8-HpCDD, 3.2 years; OCDD, 5.7 years; 1,2,3,4,6,7,8-HpCDF, < 1.7 years; OCDF, 1.8 years.

(m) Russian Federation

Schecter *et al.* (1992) found PCDD/PCDF concentrations in pooled blood samples from Baikalsk (eastern Siberia) and St Petersburg to be 18 and 17 ng I-TEQ/kg, respectively. Levels of OCDD were very low (57 and 89 ng/kg, respectively).

(n) Spain

Jiménez *et al.* (1995) measured the blood levels of PCDDs and PCDFs from 11 individuals (age, 19–55 years) living in Madrid. The mean total level of PCDDs was 515.3 ng/kg, of which 1.5 ng/kg was 2,3,7,8-TCDD.

(*o*) *Sweden*

After Rappe *et al.* (1983) found elevated PCDD/PCDF levels in the blood of occupationally exposed workers (chlorophenol workers, textile workers), samples from the general population in Sweden were analysed. Levels of all higher chlorinated (Cl_4–Cl_8) PCDDs and PCDFs in adipose tissue were measured (Rappe, 1984b). The samples originated from patients undergoing cancer or gall bladder surgery. It was concluded that the low ng/kg levels for tetra- to octa-CDDs/CDFs represented typical contamination of the general population.

In a continuation of this study (Nygren *et al.*, 1986; Rappe *et al.*, 1986a), PCDD/-PCDF patterns in adipose tissue from a group of 17 cancer patients and a group of 14 controls were compared. No difference was seen in the pattern of congeners between the two groups.

In order to study the influence of diet on the body burden of PCDDs/PCDFs, blood from three different Swedish groups was analysed (Svensson *et al.*, 1991; Rappe, 1992): Group 1, with no fish consumption (persons suffering from an allergy); Group 2, with normal fish consumption (around 50 g/day); and Group 3, with high fish consumption (> 100 g/day). Group 3 had a body burden, calculated as TEQ, approximately three times higher than Group 2. Group 1, however, had only slightly lower blood levels of PCDDs/PCDFs than Group 2. The dominant congener among the tetra- and penta-chlorinated congeners was 2,3,4,7,8-PeCDF. The difference between the three groups was also highest for this particular congener, which is also the major congener in fish from the Baltic Sea (see Section 1.3.2(*d*)). The mean PCDD/PCDF values for Groups 1, 2 and 3 were 17.5, 25.8 and 63.5 ng I-TEQ/kg, respectively.

This study was extended to cover the main fishing areas in Sweden (Svensson *et al.*, 1995a) and assessed dietary habits and exposure to selenium, persistent organochlorine compounds, including PCDDs/PCDFs, methylmercury and methylamines among Swedish fishermen. The interview data showed that 250 fishermen ate almost twice as much fish as the 250 referents from the general population. Fishermen from the Baltic Coast ate more fatty fish than fishermen from the Atlantic Coast, and they also had higher blood levels of persistent organochlorine compounds such as PCDDs/PCDFs than both the Atlantic Coast fishermen and the referents.

Measurements of PCDD/PCDF concentrations in adipose tissue from seven patients with malignant lymphoproliferative diseases and 12 surgical patients without malignant disease showed significantly higher concentrations of 1,2,3,7,8-PeCDD, 1,2,3,6,7,8-HxCDD and 2,3,4,7,8-PeCDF in the cases with lymphoproliferative diseases than in the patients in the other group (Hardell *et al.*, 1995). Nordic TEQ values also were significantly higher in the first group (mean, 64.7 ng/kg; range, 19.9–187) than in the second group (mean, 29.7 ng/kg; range, 12.9–53.4).

(*p*) *Switzerland*

Wacker *et al.* (1990) analysed liver and adipose tissue samples of 21 Swiss inhabitants (age, 15–85 years; mean, 47 years) for PCDDs/PCDFs. All values were

reported on a wet weight basis. **Table 28** shows the concentration ratios between adipose tissue and liver. No correlation between age or weight and tissue levels was detected.

Table 28. Concentration ratios (wet weight-based) of PCDDs in adipose and liver tissue of Swiss inhabitants

Congener	Adipose : liver	SD
1,2,3,7,8-PeCDD	21.1	6.6
1,2,3,4,7,8-HxCDD	14.6	6.8
1,2,3,6,7,8-HxCDD	13.8	6.1
1,2,3,4,6,7,8-HpCDD	2.4	0.9
OCDD	2.4	0.8

From Wacker *et al.* (1990)

(q) United Kingdom

PCDD/PCDF background levels 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.

(r) 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. PCDD levels increased with increasing chlorination from tetra- (mean, 6.4 ng/kg) to octa-CDD (mean, 585 ng/kg). Only the penta-, hexa- and hepta-PCDF congeners were detected, at levels that were of the same order of magnitude. 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 *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.

Schecter *et al.* (1986a) reported PCDD/PCDF levels in adipose tissue of eight control samples from Binghamton, NY. All values were based on wet weight. The 2,3,7,8-TCDD levels ranged between 1.4 and 17.7 ng/kg. HpCDDs and OCDD were found at

mean values of 164 and 707 ng/kg, respectively, with maximum values of 691 and 1931 ng/kg, respectively.

Patterson *et al.* (1986a) reported 2,3,7,8-TCDD levels in 61 adipose tissue samples from 35 autopsy cases from Georgia and Utah. The geometric mean for these samples on a wet-weight basis for 2,3,7,8-TCDD was 7.1 ng/kg. The geometric mean of values for 2,3,7,8-TCDD in 31 of these samples on a lipid basis was 9.6 ng/kg. On the basis of the wet-weight adipose concentrations, the authors concluded that concentrations of 2,3,7,8-TCDD increased with age in both sexes. There was no significant difference in the concentration of 2,3,7,8-TCDD between blacks and whites, and women had a slightly higher (2 ng/kg) mean concentration than men.

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 $\geq$ 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.

Stanley *et al.* (1990) also found a decrease in 2,3,7,8-TCDD with time in a subset of the NHATS adipose tissue samples (age group, 15–44 years) collected between 1971 and 1987 (see **Table 29**).

Table 29. Differences in 2,3,7,8-TCDD levels in adipose tissues by year of sample collection in the United States

2,3,7,8-TCDD (ng/kg lipid)	Collection year	Remark
~ 18	1971–73	Individual specimen
~ 14.5	1974–76	Individual specimen
~ 10.5	1977–79	Individual specimen
~ 9.5	1980–82	Individual specimen
~ 8	1982	Composite specimen
~ 4	1987	Composite specimen

Adapted by the Working Group from Stanley *et al.* (1990)

In an interim report, Patterson *et al.* (1986b) described high concentrations of 2,3,7,8-TCDD in adipose tissue from persons (mean age, 52.6 years) exposed recreationally, residentially and occupationally in a highly contaminated area in Missouri, compared with 57 controls. The geometric and arithmetic means were 6.4 and 7.4 ng/kg (wet weight-based), respectively, with a range of 1.4–20.2 ng/kg.

The Missouri study control group (Patterson *et al.*, 1986b) was expanded in 1989 by 128 persons with no known exposure (Andrews *et al.*, 1989): adipose 2,3,7,8-TCDD

levels ranged from non-detectable to 20.2 ng/kg, with 95% of the levels at 16.6 ng/kg or below.

Patterson *et al.* (1994) analysed adipose samples originating from Atlanta, GA, in 1984–86. They found mean 2,3,7,8-TCDD concentrations for men and women of 7.4 ng/kg and 11.6 ng/kg, respectively. Further adipose tissue samples were analysed for all isomers.

Piacitelli *et al.* (1992) compared serum 2,3,7,8-TCDD levels in 280 chemical plant workers with the levels in 99 unexposed referents. The mean serum level for the control group was 7 ng/kg (lipid-based).

A comparison of PCDD/PCDF levels in whole blood, plasma and adipose tissue was performed by Schecter *et al.* (1990b) and Päpke *et al.* (1992). There were few differences in PCDD/PCDF levels between blood plasma and adipose and also between whole blood and adipose tissue when reported on lipid basis. The difference was most striking for OCDD between plasma and adipose tissue, with a ratio of 2 : 1. Total PCDDs/PCDFs appeared higher in plasma than in adipose tissue, if reported by actual measurement, because OCDD is usually the most abundant congener. Comparing whole blood with adipose tissue, the values were more similar. When TEQs were used, however, the values were almost identical in the two series. Wendling *et al.* (1990a) found 2,3,7,8-TCDD levels in samples of faeces from laboratory workers of between 0.61 and 0.74 ng/kg, dry weight-based. The fat content of the faeces was not measured. With some assumptions, a faecal fat 2,3,7,8-TCDD concentration of 3.7 ng/kg was estimated.

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-TCDD levels in adipose tissue for Viet Nam veterans, non-Viet Nam veterans and civilian controls were 11.7 (± 1.7), 10.9 (± 1.7) and 12.4 (± 1.9) ng/kg on a lipid weight basis, respectively. The mean levels for these groups were not significantly different from each other.

The effect of fasting on blood PCDD/PCDF levels was tested in 13 Viet Nam veterans (Hansson *et al.*, 1989). Although 2,3,7,8-TCDD levels increased slightly, for no congener including 2,3,7,8-TCDD was the change statistically significant.

In connection with analysis of blood samples from various geographical locations for PCDDs/PCDFs, Schecter *et al.* (1992) reported a total I-TEQ of 41 ng/kg (lipid-based) in a pooled blood sample (n = 100) from the United States. [The Working Group noted that the dates of collection of these samples were not given.]

In a study by Schecter *et al.* (1994b; 1996b), levels of PCDDs/PCDFs in placenta, blood and fetal tissue were measured. The highest semen levels, reported on wet weight, were found for OCDD, OCDF and HpCDF. 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. The mean age of the blood donors was not specified (Schecter *et al.*, 1996d).

(*s*) *Viet Nam*

2,3,7,8-TCDD was not detected in seven adipose tissue samples from north Viet Nam (no exposure to Agent Orange) but was found in 10/13 samples from south Viet Nam (mean level, 34 (range, 9–103) ng/kg). Most of the other chlorinated PCDDs and PCDFs were found in samples from south Viet Nam (Schecter *et al.*, 1986b). 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. Differences in 2,3,7,8-TCDD body burden continued to be substantial between the populations of south and north Viet Nam.

In 1984–85, adipose tissues were taken from nine patients at a hospital in south Viet Nam (Nguyen *et al.*, 1989) and analysed for PCDDs/PCDFs. Eight patients had detectable levels of 2,3,7,8-TCDD ranging from 4 to 103 ng/kg, with a mean of 23 ng/kg.

Effects of geographical conditions and other variables on the distribution of 2,3,7,8-TCDD levels in adipose tissues from Viet Nam have been studied (Hoang *et al.*, 1989). No 2,3,7,8-TCDD was detected in 10/11 samples from the north (the positive sample contained 5 ng/kg). In the south, 2,3,7,8-TCDD was not detected in 4/44 samples and the mean level was 20.5 ng/kg in the four positive samples.

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 2,3,7,8-TCDD level and I-TEQ value for samples from north and south Viet Nam were 2.2 and 14.6 ng/kg and 15 and 36 ng I-TEQ/kg, respectively.

PCDD levels in persons living at various localities in Viet Nam were summarized (Le *et al.*, 1995) using data from pooled blood samples. Each sample was obtained from 30–100 adults over 40 years old, who had been living for more than five years in a given area. As from 1992, some samples were taken from younger age groups between 18–40 years. The results of 43 analyses (containing blood of 2722 individuals) indicated

that most of the samples from the south had higher levels of 2,3,7,8-TCDD as well as I-TEQ than the ones from the north.

In a study of the persistence of elevated PCDD levels in human tissues from Viet Nam, Schecter *et al.* (1995) performed 160 analyses on tissues from 3243 persons. 2,3,7,8-TCDD levels of up to 103 ng/kg were found in adipose tissue collected in the 1980s. Pooled blood collected from south Viet Nam in 1991–92 also showed 2,3,7,8-TCDD levels up to 33 ng/kg, whereas tissues from north Viet Nam had 2,3,7,8-TCDD levels at or below 2.9 ng/kg.

1.4.2 *Human milk*

There have been a large number of studies of PCDD concentrations in human milk. Many of the results are shown in **Table 30** and summarized in **Table 31**.

Only 2,3,7,8-substituted congeners have been found in human milk. The concentrations of the individual congeners present have been shown to be significantly correlated (Van den Berg *et al.*, 1986a; Beck *et al.*, 1987). Several studies have involved analysis of quite large numbers of individual samples without pooling, and these results show inter-individual variation of about a factor of 5 to 10 for most congeners (Beck *et al.*, 1989c; Frommberger, 1990; Dewailly *et al.*, 1991; Fürst *et al.*, 1992b; Hashimoto *et al.*, 1995b; Liem *et al.*, 1995).

Fürst *et al.* (1989, 1992b) and Beck *et al.* (1992) have both shown, by statistical analysis of their results, that the concentrations of PCDDs in human milk decrease as the period of breast-feeding increases, and decrease significantly for successive breast-fed children. Fürst *et al.* (1992b) found the summed I-TEQ concentration of PCDDs and PCDFs for the third breast-feeding period to be about 75% of that for the first, while Beck *et al.* (1992) reported a corresponding reduction to 57%. In Japan, Hirakawa *et al.* (1995) found that a small group of multipara donors had an average summed I-TEQ level about 10% lower than that for primipara donors.

Beck *et al.* (1992) found that mean PCDD and PCDF levels in six mothers on a vegetarian diet were slightly lower than the mean of a larger control group. Pluim *et al.* (1993b) concluded that there was a direct relationship between the amount of animal fat consumed and the PCDD/PCDF concentrations in breast milk. Short-term dietary measures to reduce PCDD/PCDF intake have been found, however, to have no effect on human milk concentrations (Pluim *et al.*, 1994a).

Many of the data included in **Table 30** were obtained in studies coordinated by the World Health Organization European Centre for Environment and Health and involved the following selection criteria (Yrjänheikki, 1989; WHO, 1996):

- donors should be primiparae;
- both mother and child should be apparently healthy, and the pregnancy should have been normal;
- the mother should be breast-feeding one child only (i.e., no twins);
- mothers who had resided outside the area for more than six months during the last five years should be excluded;
- only mothers who were exclusively breast-feeding should be included.

Table 30. Concentrations of PCDDs in human milk

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ[a] PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
WHO (1996)	Albania, Librazhd; pool; unpolluted area (WHO criteria)	10	1992–93	0.4	1	0.6	4.1	1.1	6.3	21.6	3.8
WHO (1996)	Albania, Tirana; pool; polluted area (WHO criteria)	10	1993–93	0.6	1.3	0.6	4.8	1.1	7	23.6	4.8
WHO (1996)	Austria, Brixlegg; pool; industrial area (WHO criteria)	13	1992–93	2.2	4.5	2.5	12.3	2.3	14.1	82.3	14
Yrjänheikki (1989)	Austria, Tulln; pool (WHO criteria)	51	1986–88	2.7	5.2	17.2		6.1	72.9	141	[18.7]
WHO (1996)	Austria, Tulln; pool; rural area (WHO criteria)	21	1992–93	2	3.5	2.2	13.2	3	18.6	110	10.9
Yrjänheikki (1989)	Austria, Vienna; pool (WHO criteria)	54	1986–88	2.9	4.4	14		4	46.5	159	[17.2]
WHO (1996)	Austria, Vienna; pool; urban area (WHO criteria)	13	1992–93	1.3	3.4	2.1	14	2.7	27.4	150	10.7
Yrjänheikki (1989)	Belgium; pool; industrial area (WHO criteria)	–	1986–88	10.2	10.7	5.8	28	7.7	52	283	[40.2]
Yrjänheikki (1989)	Belgium; pool; rural area (WHO criteria)	–	1986–88	–	10.5	7.9	39	9.3	121	555	[33.7]
Yrjänheikki (1989)	Belgium; pool; urban area (WHO criteria)	–	1986–88	9.1	9.6	7	30	9.6	88	517	[38.7]
WHO (1996)	Belgium, Brabant Wallou; pool (WHO criteria)	8	1992–93	2.5	6.9	4.1	22.3	4.8	31.5	152	20.8
WHO (1996)	Belgium, Brussels; pool (WHO criteria)	6	1992–93	3.4	8	5.2	27.2	27.6	55.6	310	26.6
WHO (1996)	Belgium, Liege; pool (WHO criteria)	20	1992–93	3.1	9.1	5.6	31.2	5.5	30.3	191	27.1

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Schecter *et al.* (1991b)	Cambodia, Phnom Penh	8	–	0.49	1.6	0.6	3.4	1.1	11	59	3.1
WHO (1996)	Canada (all provinces); pool	200	1981	3.4	8.9		84	18	94	361	28.6
WHO (1996)	Canada (all provinces); pool	100	1992	2.1	6.2		37	7.2	33	138	14.5
Yrjänheikki (1989)	Canada, British Columbia; pool	23	1986–88	3.4	10.2	6.3	63	3.1	82	160	[23.5]
Yrjänheikki (1989)	Canada, Maritimes; pool	19	1986–88	2.5	5.9	5.1	35	4.8	63	148	[15.7]
Yrjänheikki (1989)	Canada, Ontario N&E; pool	32	1986–88	2.2	6.5	5.2	46	6.6	68	143	[17]
Yrjänheikki (1989)	Canada, Ontario SW; pool	44	1986–88	2.2	7.4	6.9	42	5.7	69	137	[17.9]
Yrjänheikki (1989)	Canada, Prairies; pool	31	1986–88	2.7	8.1	7.6	54	8.7	75	143	19.4
Yrjänheikki (1989)	Canada, Québec; pool	34	1986–88	2.8	8.1	5.6	41	6.8	73	152	[18.2]
Dewailly *et al.* (1991)	Canada, Québec; pool; rural regions	16	1988–89	2.3	4.8		35	6.4	41	132	[13.4]
Yrjänheikki (1989)	Croatia, Krk; pool (WHO criteria)	14	1986–88	1.6	3		15.1	4.2	19.8	101	[12]
Yrjänheikki (1989)	Croatia, Zagreb; pool (WHO criteria)	41	1986–88	1.9	2.4		20.6	4.7	14.6	90	[11.7]
WHO (1996)	Croatia, Krk; pool (WHO criteria)	10	1992–93	1.2	2.4	1.9	8	2.3	17	84.3	8.4

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
WHO (1996)	Croatia, Zagreb; pool (WHO criteria)	13	1992–93	2	3.6	3.3	10.1	2.6	21.5	99.4	13.5
WHO (1996)	Czech R, Kladno; pool (WHO criteria)	11	1992–93	0.9	1.9	1.1	5.2	1.6	8.9	39.8	12.1
WHO (1996)	Czech R, Uherske Hradiste; pool (WHO criteria)	11	1992–93	1.3	3	1.5	7.3	2.1	11.3	40.3	18.4
Yrjänheikki (1989)	Denmark; pool (WHO criteria)	10	1986–88	2.3	5.5	–	34	4.9	51	157	[17.5]
Yrjänheikki (1989)	Denmark; pool (WHO criteria)	42	1986–88	2.1	6.2	–	40	4.7	47	210	[17.7]
WHO (1996)	Denmark; 7 cities; pool (WHO criteria)	48	1992–93	1.7	5.6	7.7	25.9	5.1	26	141	15.2
Mussalo-Rauhamaa & Lindström (1995)	Estonia, Tallinn (primipara)	6	1991	3.3	3	1.6	6.6	3.5	10	111	13.5
Mussalo-Rauhamaa & Lindström (1995)	Estonia, Tarto (primipara)	6	1991	4.1	5.6	2.2	7.9	4.3	13.5	147	21.4
Abraham *et al.* (1995b)	Faeroe Islands	1	1994~	0.9	3.7	4.5	13	1.9	9.8	45	9
Abraham *et al.* (1995b)	Faeroe Islands	1	1994~	1	4	4	17	2.1	19	114	10
Abraham *et al.* (1995b)	Faeroe Islands	1	1994~	< 1	5.2	3.5	16	2	9.3	75	[10.2]
Abraham *et al.* (1995b)	Faeroe Islands	1	1994~	< 3	5	3.2	12	< 3	22	63	[11.7]

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Abraham *et al.* (1995b)	Faeroe Islands; pool	9	1994~	0.8	2.9	3.3	11	1.4	9.2	64	6.7
Yrjänheikki (1989)	Finland, Helsinki; pool (WHO criteria)	38	1986–88	2	5.6	< 0.5	36	7	49	154	18
WHO (1996)	Finland, Helsinki; pool (WHO criteria)	10	1992–93	2.3	6.9	2.6	34.7	5.9	50.1	238	21.5
Yrjänheikki (1989)	Finland, Kuopio; pool (WHO criteria)	31	1986–88	1.8	5.6	< 0.5	22	5.8	28	113	[15.7]
WHO (1996)	Finland, Kuopio; pool (WHO criteria)	24	1992–93	1.2	3.9	1.4	24.5	3.7	29.2	131	12
González *et al.* (1996)	France, Paris	15	1990	2.4	6.6		25.9		56	290	20.1
Beck *et al.* (1992a)	Germany (primipara)	34	–	4.3	13	10	48	9.7	56	292	33
Beck *et al.* (1992a)	Germany (multipara)	23	–	3.1	10	9.6	39	7.9	44	252	25.6
Beck *et al.* (1992a)	Germany (multipara)	6	–	2.3	7.8	6.6	28	5.8	32	187	18.9
Frommberger (1990)	Germany, Baden-Württemberg	490	1988–89	4.2	10	9.2	32	6.6	61	503	[36.2]
Beck *et al.* (1987)	Germany, Berlin	30	–	3.4	15	12	59	11	61	530	[32.6]
Beck *et al.* (1989c)	Germany, Berlin	35	–	3.5	15	12	57	11	60	500	[33]
Yrjänheikki (1989)	Germany, Berlin; pool (WHO criteria)	40	1986–88	3.3	14	12	57	10	44	210	[32.1]

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
WHO (1996)	Germany, Berlin; pool (WHO criteria)	10	1992–93	2.2	7.1	6.3	21.6	5.1	23.4	164	16.5
Beck *et al.* (1989c)	Germany, Flensburg (Baltic coast)	6	–	4.1	12	7.3	46	8.3	41	200	[31.6]
Fürst *et al.* (1989)	Germany, North Rhine Westphalia	189	1985–88	2.9	9.9	7.6	31.2	6.6	44.7	195	[27.5]
Fürst *et al.* (1992b)	Germany, North Rhine Westphalia	526	1986–91	3.2	10.1	8.4	35.8	6.4	41.2	208	29.3
Yrjänheikki (1989)	Germany, North Rhine Westphalia; pool; primipara (WHO criteria)	79	1986–88	2.7	11.8	8.6	35.2	7.5	50.6	224	[31.8]
Yrjänheikki (1989)	Germany, Oldenburg; pool (WHO criteria)	35	1986–88	3.3	7.2	9.6	40	8.8	49.8	255	[35.4]
Beck *et al.* (1989c)	Germany, Recklinghausen (industrial area)	10	–	3.9	12	13	46	8.3	45	260	[30.6]
Yrjänheikki (1989)	Germany, Recklinghausen; pool (WHO criteria)	23	1986–88	3.8	14	9.7	45	7.6	42	170	32.8
Beck *et al.* (1989c)	Germany, Rheinfelden; pool; rural area/PCP manufacture	9	–	4.5	16	13	54	12	79	441	[36.9]
Beck *et al.* (1989c); Yrjänheikki (1989)	Germany, Weiden; rural area	14	1986–88	3.7	12	9.3	44	9.6	60	264	30.1
Wuthe *et al.* (1993)	Germany, one woman	1	1992	2.1	5.9		39.3		39.5	199	–

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Yrjänheikki (1989)	Hungary, Budapest; (WHO criteria)	100	1986–88	3.4	1	3.2	8.2	1	60	210	[9.36]
WHO (1996)	Hungary, Budapest; pool (WHO criteria)	20	1992–93	1.7	2.8	4.3	8.3	2.1	25.6	129	8.5
Yrjänheikki (1989)	Hungary, Szentes; pool (WHO criteria)	50	1986–88	3.7	1.4	5.2	12.8	1.6	63	271	[11.7]
WHO (1996)	Hungary, Szentes; pool (WHO criteria)	10	1992–93	1.3	2.6	3.1	7.8	2	31.1	149	7.8
Yrjanheikki (1989)	Japan, Fukuoka; pool	6	1986	[2.1]	[4.6]	[4]	[30]	[5.6]	[62]	[975]	[24.3]
Hirakawa *et al.* (1995)	Japan, Fukuoka; multipara	8	1994	1.2	5	2.6	18.9	3.6	31.3	195	[11.8]
Hirakawa *et al.* (1995)	Japan, Fukuoka; primipara	7	1994	2	8.9	4.7	32.3	6.9	29.8	174	[18.6]
Hashimoto *et al.* (1995b)	Japan, various locations	26	1993–94	2.7	12	3.5	57	10	21	160	[37]
Alawi *et al.* (1996b)	Jordan, Amman; pool	4 to 6	1994	< 3.2	< 3.2	< 3.2	< 3.2	< 3.2	3.2	64.6	[9.38]
Alawi *et al.* (1996b)	Jordan, Amman; pool	4 to 6	1994	< 6.3	< 6.3	< 6.3	< 6.3	< 6.3	15.7	56.1	[18.3]
Alawi *et al.* (1996b)	Jordan, Aqaba; pool	4 to 6	1994	7.8	< 4.5	< 4.5	19	< 4.5	14.5	46.9	[22]
Alawi *et al.* (1996b)	Jordan, Irbid; pool	4 to 6	1994	3.7	21.3	< 3.7	56.5	< 3.7	47.2	75.8	[98.5]

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Alawi *et al.* (1996b)	Jordan, Madaba; pool	4 to 6	1994	< 11	55	11	11	11	96	147	[110]
Alawi *et al.* (1996b)	Jordan, Zarka; pool	4 to 6	1994	< 2.6	6.1	< 2.6	9.6	< 2.6	14	29	[11.4]
Petreas *et al.* (1996)	Kazakstan (WHO criteria)	40	< 1996	13.6	4.45	1.15	4.01	1.14	10	112	20.1
WHO (1996)	Lithuania, Anykshchiai; pool; rural area (WHO criteria)	12	1992–93	5.5	3.6	3.6	3.8	1.2	5.7	32.7	14.4
WHO (1996)	Lithuania, Palanga; pool; coastal area (WHO criteria)	12	1992–93	4.8	3.4	1.9	4.5	1.4	5.1	21.3	16.6
WHO (1996)	Lithuania, Vilnius; pool; urban area (WHO criteria)	12	1992–93	5.4	2.8	1.8	4.7	1.5	7.2	39.5	13.3
Liem *et al.* (1995)	Netherlands (primipara)	103	1993	3.1	8.1	8.6	37.1	6.9	44.9	295	23.5
Yrjänheikki (1989)	Netherlands; pool; urban area (WHO criteria)	13	1986–88	5.2	18	10	75	11	112	627	39.6
Yrjänheikki (1989)	Netherlands; pool; rural area (WHO criteria)	13	1986–88	5.4	17	10	72	7.6	82	545	37.4
Pluim *et al.* (1992, 1993c, 1994)	Netherlands, Amsterdam			3.8	10.6	1.3	49.1	6.5	54.3	297.5	–
Koopman-Esseboom *et al.* (1994)	Netherlands, Rotterdam/Groningen			4.0	10.6	8.7	47.4	6.7	63.2	799.6	–
WHO (1996)	Netherlands; pool (WHO criteria)	17	1992–93	2.9	7.6	7.9	35.6	6.8	46.1	324	22.4

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Tuinstra *et al.* (1995)	Netherlands, Groningen/Rotterdam; collected at 10 and 42th day after delivery	168	–	–	–	–	–	–	–	–	30
Yrjänheikki (1989)	Norway, Hamar; pool; rural area (WHO criteria)	10	1985–86	2.5	4.7		18.8	4.9	40.3	150	[14.8]
WHO (1996)	Norway, Hamar; pool; rural area (WHO criteria)	10	1992–93	1.8	2.8	1.6	9.2	2.4	17.4	116.7	9.3
Clench-Aas *et al.* (1992)	Norway, Skien-Porsgrunn; Mg production (WHO criteria)	10	1985–86	2.7	5.0		20.3	3.2	36.6	156	[19.2]
WHO (1996)	Norway, Skien-Porsgrunn; pool; industrial area (WHO criteria)	10	1992–93	2.1	3.8	1.9	10.7	3.5	19.1	112	12.5
Clench-Aas *et al.* (1992)	Norway, Tromsø; pool; coastal area (WHO criteria)	11	1985–86	2.9	4.7		19.2	4.7	36	155	15.9
WHO (1996)	Norway, Tromsø; pool; coastal area (WHO criteria)	10	1992–93	2.4	3.1	1.4	9.3	2.4	15.7	86.9	10.1
Buckland *et al.* (1990a)	New Zealand, Auckland (WHO criteria)	11	< 1990	4.6	6		26	5.1	53	200	15
Buckland *et al.* (1990a)	New Zealand, Christchurch (WHO criteria)	9	< 1990	5.7	8.2		34	6.3	51	240	19
Buckland *et al.* (1990a)	New Zealand, N. Canterbury (WHO criteria)	8	< 1990	5.6	8.4		38	7.2	53	220	19
Buckland *et al.* (1990a)	New Zealand, Northland (WHO criteria)	9	< 1990	4.7	6.8		39	5.5	51	180	17
Schecter *et al.* (1990e)	Pakistan; pool	7	–	3.3	5.2		15.3	4.8	40.7	180	12.6

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Yrjänheikki (1989)	Poland; pool (WHO criteria)	5	1986–88	3.6	6	3	11	6.5	30.9	250	[21.1]
WHO (1996)	Russian Federation, Arkhangelsk; pool	1	1993	4.7	4	3.8	5.6	1.3	7.9	35.9	15.2
Schecter *et al.* (1990f)	Russian Federation, Baikalsk; pool	5	1988–89	2	2.7	0.9	4	0.7	5	30	10.4
Schecter *et al.* (1990f)	Russian Federation, Irkutsk; pool	4	1988–89	1.9	3.6	1.6	6.1	2	6.9	48	17.3
Schecter *et al.* (1990f)	Russian Federation, Kachug	4	1988–89	2.5	2.2	1	4.4	1.1	6	33	9.29
WHO (1996)	Russian Federation, Karhopol; pool	1	1992–93	1.9	1.6	0.7	1.9	0.5	1.5	9	5.9
Polder *et al.* (1996)	Russian Federation, Kola Peninsula	30	1992–93	–	–	–	–	–	–	–	15.8
Schecter *et al.* (1990f)	Russian Federation, Moscow	1	1988–89	8.7	6.3	4	14	1.8	16	88	20.6
Schecter *et al.* (1990f)	Russian Federation, Novosibirsk; pool	10	1988–89	3.4	3.9	2.2	6.6	1.2	11	68	11.9
WHO (1996)	Slovakia, Michalovce; pool (WHO criteria)	10	1992–93	1.3	2.4	1.5	5.5	1.3	7.2	30.2	15.1
WHO (1996)	Slovakia, Nitra; pool (WHO criteria)	10	1992–93	1.5	2.7	1.8	7.8	2	13.6	57.6	12.6
Schecter *et al.* (1990e)	South Africa; Black; pool	6	–	1.2	3.9		21.5	6.1	51.1	196	8.3

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Schecter *et al.* (1990e)	South Africa; White; pool	18	–	1.7	5.8		26.5	6.9	60.7	254	12.6
WHO (1996)	Spain, Bizkaia; pool (WHO criteria)	19	1992–93	2	6	3.1	33.4	5.8	32.6	158	19.4
WHO (1996)	Spain, Gipuzkoa; pool (WHO criteria)	10	1992–93	2.3	7.7	4.2	53.5	9.3	59.6	164	25.5
González *et al.* (1996)	Spain, Madrid	13	1990	1.2	6.7		53.6		46	234	[14.3]
Yrjanheikki (1989); Clench-Aas *et al.* (1992)	Sweden, Borlänge; pool; rural area (WHO criteria)	10	1985–86	2.8	6.5		26.5	6.1	41.8	184	[20.1]
Yrjanheikki (1989)	Sweden, Gothenburg; pool; urban area (WHO criteria)	10	1985–86	3.2	7.5		39	6.2	67.3	237	22.8
Yrjanheikki (1989)	Sweden, Sundsvall; pool; industrial area (WHO criteria)	10	1985–86	3.3	7.8		28.1	7.1	52.2	209	22.6
Yrjanheikki (1989)	Sweden, Uppsala; pool; MSWI (WHO criteria)	10	1985–86	2.9	7.2		38.9	8.2	72.1	255	22.4
Schecter *et al.* (1991b)	Thailand, Bangkok	10	–	0.3	1.1	0.5	1.1	0.7	10	68	3
Wearne *et al.* (1996)	UK, Cambridge (WHO criteria)	20	1993–94	3.7	9.9	11	32	7.5	47	190	24
Startin *et al.* (1989)	UK, Glasgow (WHO criteria)	50	1987~	4.6	12		57	6.5	65	271	[29]

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Wearne *et al.* (1996)	UK, Glasgow (WHO criteria)	20	1993–94	3.1	8.6	9.6	27	6.4	40	170	21
Startin *et al.* (1989)	UK, Sutton Coldfield (WHO criteria)	50	1987~	6.5	14	67		10	76	303	37
Wearne *et al.* (1996)	UK, Birmingham (WHO criteria)	20	1993	3.5	9	9.2	27	6.5	31	130	21
WHO (1996)	Ukraine, Kiev; pool (WHO criteria)	5	1992–93	2.8	2.4	1.9	3.9	1.4	6.6	40.2	11.0
WHO (1996)	Ukraine, Kiev; pool (WHO criteria)	5	1992–93	4.8	2.7	1.6	3.5	1.1	3.6	24.4	13.3
Schecter *et al.* (1989d)	USA	42	1988	3.3	6.7	4.95	30.5	6.2	42	233	[16.6]
Schecter *et al.* (1990e)	USA, Tennessee; pool	9	–	2.5	6.8	38.2		9.6	59.6	234	14.6
Schecter *et al.* (1990e)	Viet Nam, Binh Long; pool	4	NR	2.8	6.9	17		4.4	37.9	146	14.5
Schecter *et al.* (1991b)	Viet Nam, Da Nang	11	1985–90	5.6	15	5.1	22	11	55	292	34
Schecter *et al.* (1991b)	Viet Nam, Dong Nai	11	1985–90	10	7.2	2.1	10	4	28	119	26
Schecter *et al.* (1991b)	Viet Nam, Hanoi	30	1985–90	2.1	2.9	1.8	5.2	1.8	11.5	78.3	9
Schecter *et al.* (1989d)	Viet Nam, Ho Chi Minh	38	1985–90	7.1	6	2.9	15	4.2	36	231	18.5

Table 30 (contd)

Reference	Origin	No.	Coll. period	Concentration (ng/kg fat)							
				TCDD	PeCDD	HxCDD			HpCDD	OCDD	I-TEQ PCDD/PCDF
				2378	12378	123478	123678	123789	1234678		
Schecter *et al.* (1989d)	Viet Nam, Song Be	12	1985–90	17	8.2	6.6	18	6	36	185	31.7
Schecter *et al.* (1990e)	Viet Nam, Tay Ninh; pool	4	NR	5.7	14.1		43.8	10.9	88	415	[28.6]
Schecter *et al.* (1990e)	Viet Nam, Vung Tau; pool	5	NR	6.2	10.5		15.1	6	52.9	181	21.9
Schechter *et al.* (1995)	South Viet Nam; analytical method N	1	1970	1832	–	–	–	–	–	–	
		1	1970	1465	–	–	–	–	–	–	
		1	1970	732	–	–	–	–	–	–	
		1	1970	366	–	–	–	–	–	–	
		1	1970	333	–	–	–	–	–	–	
		1	1973	266	–	–	–	–	–	–	
		1	1973	280	–	–	–	–	–	–	
		1	1973	133	–	–	–	–	–	–	
		2	1985–88	5	–	–	–	–	–	–	
		2	1985–88	11	–	–	–	–	–	–	
		30	1985–88	2.1	–	–	–	–	–	–	

–, not reported; ND: not detected and limit of detection not reported

[a] Summed TEQ concentrations calculated by the Working Group [ ] where possible assuming congeners that were not detected were present at the full value of the limit of detection or as given by the authors when they correspond to the value calculated by the Working Group.

Table 31. Summary of concentrations (ng/kg fat) of PCDDs in human milk (as reported in Table 30)

	TCDD	PeCDD	HxCDD			HpCDD	OCDD	Total TEQ (PCDDs/ PCDFs)
	2378	12378	123478	123678	123789	1234678		
Mean	3.4	7.2	5.0	26	5.4	39	180	20
Minimum	0.3	1	0.5	1.1	0	1.5	9	3.1
5th percentile	1.03	1.71	0.83	3.9	1.1	6.17	30.11	8.16
25th percentile	2	3.68	1.9	10.25	2.3	15.93	89.5	12.35
Median	2.85	6.15	4	22.15	5.5	38.95	155	17.9
75th percentile	3.73	8.9	7.6	37.78	7.1	53.5	233.25	24.9
95th percentile	7.70	15	11.35	57	11	82	501.35	37.15
Maximum	17	55	13	84	27.6	121	975	110

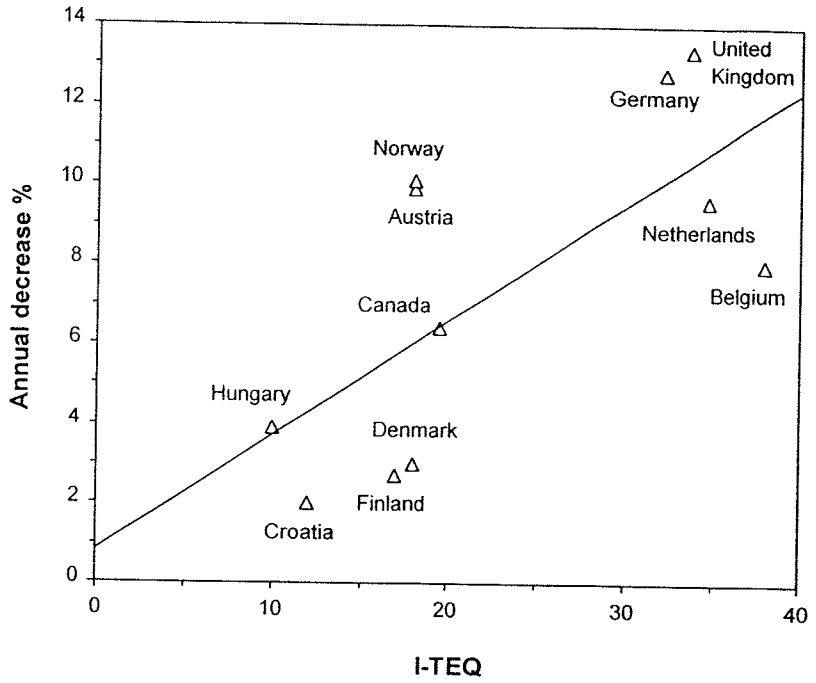
By involving only primiparous women, these criteria may lead to slight overestimation of average concentrations in milk, but the results provide a good basis for comparing different regions and variations over time. Laboratories generating data have also participated in an extended series of quality assurance intercomparisons (Rappe *et al.*, 1989c; Yrjänheikki, 1989; Stephens *et al.*, 1992; Carlé *et al.*, 1995).

Most of the regional comparisons within countries have shown no significant differences between donor cohorts from rural and industrial areas. Koopman-Esseboom *et al.* (1994a) reported significantly higher levels of a number of PCDDs and PCDFs in women living in industrialized areas of the Netherlands, compared with women living in rural areas. Rather larger differences were found by Alawi *et al.* (1996b) for different samples from Jordan, but variable detection limits and a high incidence of non-detection make interpretation uncertain. Pioneering work by Baughman, R. showed elevated 2,3,7,8-TCDD levels as high as 1450 ng/kg in milk lipid (Schecter, 1994) collected from south Viet Nam in 1970, while Agent Orange spraying was still occurring or had only just ceased. More recent samples from Viet Nam, analysed by Schecter *et al.* (1989d,e, 1990e,g, 1991b, 1995), have not shown any exceptional concentrations.

There is evidence of differences between certain parts of the world, and of a decrease over time. Samples taken in the second half of the 1980s from the industrialized countries of western Europe contribute most of the upper-quartile data in **Table 31**. Relatively low average levels have been found in samples from Albania, Cambodia, Croatia, the Faeroe Islands, Hungary, Pakistan and Thailand.

There is now clear evidence of a decrease in PCDD/PCDF levels in human milk over time in almost every region for which suitable data exist (Alder *et al.*, 1994; Liem *et al.*, 1995; WHO, 1996). The WHO (1996) study also shows that the highest rates of decrease, nearly 14% per annum, have been in the areas with the highest initial concentrations (**Figure 1**). These data imply that a substantial reduction in intake of PCDDs and PCDFs has occurred in recent years.

Figure 1. Annual percentage decrease of PCDD/PCDF levels in human milk for 11 countries as a function of the dioxin levels in 1988



From WHO (1996)

1.5 Regulations and guidelines

In Germany, an occupational technical exposure limit (TRK) value of 50 pg I-TEQ/m^3 in air has been established for PCDDs and PCDFs. 2,3,7,8-TCDD is classified in Germany as a III A2 compound (shown to be clearly carcinogenic only in animal studies but under conditions indicative of carcinogenic potential at the workplace) (Deutsche Forschungsgemeinschaft, 1996).

The United States Occupational Safety and Health Administration (OSHA) has not set a permissible exposure level (PEL) for the PCDDs. The United States National Institute for Occupational Safety and Health (NIOSH) has not set a recommended exposure level (REL) for PCDDs, but states that 2,3,7,8-TCDD should be treated as a potential occupational carcinogen and occupational exposures should be limited to the lowest feasible concentration (United States National Institute for Occupational Safety and Health, 1994). The American Conference of Governmental Industrial Hygienists (1995) has not set a threshold limit value (TLV) for the PCDDs.

The United States Environmental Protection Agency (1996b) has established a final drinking water standard for 2,3,7,8-TCDD. The maximum contaminant level (MCL) (the maximum permissible level of a contaminant in water delivered to any user of a public water system) is set at 3×10^{-8} mg/L (30 pg/L).

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

A Nordic tolerable weekly intake of 0–35 pg 2,3,7,8-TCDD/kg bw was proposed (Ahlborg *et al.*, 1988). A TDI of 1 pg dioxin-like compounds/kg bw per day has been

recommended in the Netherlands (Health Council for the Netherlands, 1996) and one of 5 pg/kg bw per day has been established in Sweden (Ahlborg *et al.*, 1988). A TDI of 10 pg PCDDs/PCDFs per kg bw per day from food has been recommended by WHO/EURO (WHO, 1991; Ahlborg *et al.*, 1992b) as well as by the Ministry of Health and Welfare of the Japanese Government (Kurokawa, 1997).

At present, the regulatory requirements for incinerator emissions vary widely among the countries of the European Union. The European Commission (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 to 16 h.

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

In Japan, a limit of 0.5 ng I-TEQ/m^3 2,3,7,8-TCDD/TCDF is recommended for municipal waste incinerators (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 European Union has specified that 2,3,7,8-TCDD must not form part of the composition of cosmetic products (European Commission, 1992).

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