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REVIEW

Levels and congener distributions of PCDDs, PCDFs and dioxin-like PCBs in environmental and human samples: a review

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Abstract The term "dioxins" is often used in a confusing way. In toxicological considerations—and also in the present report—the term is used to designate the PCDDs, the PCDFs and the coplanar ("dioxin-like") PCBs, since these classes of compounds show the same type of toxicity. Because of the large number of congeners, relevant individual congeners are assigned with a toxic equivalency factor (TEF) that relate their toxicity to that of tetrachlorodibenzo-*p*-dioxin (TCDD) (2,3,7,8-TCDD) and are to be evaluated as dioxins. Each concentration of an individual congener in a mixture is multiplied with its TEF, and the resulting TCDD equivalents are added up and expressed as WHO-endorsed toxic equivalents (WHO-TEQ). Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are mainly the by-products of industrial processes (such as metallurgical processing, bleaching of paper pulp, and the manufacturing of some herbicides and pesticides) but they can also result from natural processes like volcanic eruptions and forest fires. Waste incineration, particularly if combustion is incomplete, is among the largest contributors to the release of PCDDs and PCDFs into the environment. Due to their persistence, PCDDs, PCDFs and PCBs are part of the so-called persistent organic pollutants group of compounds that also include some chlorinated pesticides. Since they have a high lipophilicity and resist transformation, they bio-accumulate in animal and human adipose tissues. Consumption of food is considered as the major source of non-occupational human exposure to PCDD/Fs with foodstuffs from animal origin accounting for more than 90% of the human body burden. With meat, dairy, and fish products being the main contributors. The aim of the present review was to summarize experimental data regarding dioxin emissions from contaminated and uncontaminated biological and environmental samples, from the available literature. The information will be presented chronologically with respect to distribution in human milk, serum; food, water, air, soils and sediments.

Keywords Polychlorinated dibenzofurans · Polychlorinated dibenzodioxins · Polychlorinated biphenyls · Human tissue · Soil · Water · Food · Air · Sediments · Occupational exposure

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Introduction

The term "dioxin" refers to a class of structurally and chemically related halogenated aromatic hydrocarbons that includes polychlorinated dibenzodioxins (PCDDs or dioxins), polychlorinated dibenzofurans (PCDFs or furans) and the "dioxin-like" polychlorinated biphenyls (PCBs). Because of their chemistry, dioxins are both toxic and persistent in the environment. Dioxins and furans are included in the UNEP "Dirty Dozen", and Greenpeace describe dioxins as "some of the most dangerous chemicals on earth" (Davy 2004).

Dioxins are unwanted contaminants almost exclusively produced by industrial processes (Lustenhouwer et al. 1980; EPA 2004), including incineration of municipal solid waste (Hylander et al. 2003; Chang et al. 2001, 2004) or medicinal waste (Coutinho et al. 2006), chlorine bleaching of paper and pulp, and the manufacture of some pesticides, herbicides, and fungicides (Chen 2004). Dioxins did not exist prior to industrialization expect in very small amounts

(Czuczwa et al. 1984); they can also result from natural processes like volcanic eruptions and forest fires (SCF 2001; JECFA 2002; Freeman and de Tejada 2002).

Polychlorinated biphenyls on the other hand have been produced commercially for some five decades starting from about 1920, by direct chlorination of biphenyl. They were produced as mixtures; individual congeners were hardly synthesized. The various (commercial) technical PCB-mixtures are characterized by their chlorine content, the brand names of which are known as 'Aroclor' (produced in the USA), 'Clophen' (produced in Germany), 'Phenoclor' (produced in France), 'Fenclor' (produced in Italy), and 'Kanechlor' (produced in Japan). These mixtures were used in a wide range of applications, such as coatings, inks, flame retardants and paints, but its major uses were in electronic appliances, heat-transfer systems, and hydraulic fluids. Due to the persistent nature of PCBs in the environment many countries decided in the 1970s to ban the use of PCBs in open applications. They may, however, still be in use in closed systems such as capacitors and transformers, but this use will decrease over time. Waste disposal, both of households and industrial waste, is the major source of PCB emissions into the environment (ATSDR 2000).

PCDDs and PCDFs are two series of organohalogenated substances, which form a group of 210 different substances and are divided into 135 PCDFs and 75 PCDDs. However, only the isomers presenting chlorine in the 2,3,7,8 positions have been reported to be toxic to exposed organisms (Malisch 2000a; Fueno et al. 2002). This reduces the number of compounds of interest to 17, 7 PCDDs and 10 PCDFs. Nevertheless, not all 2,3,7,8 chlorinated PCDDs/PCDFs present the same toxicity; the 2,3,7,8 tetrachlorodibenzo-*p*-dioxin (TCDD) being the most toxic, was catalogued by the World Health Organization (WHO) as carcinogenic for humans (Abad et al. 2000a).

Because of the large number of congeners, relevant individual congeners are assigned with a toxic equivalency factor (TEF) (Van den Berg et al. 1998). The International Agency for Research on Cancer (IARC) named 2,3,7,8-tetrachlorodibenzo-*p*-dioxins (2,3,7,8-TCDD) as a human carcinogen. Each concentration of an individual congener in a mixture is multiplied with its TEF, and the resulting TCDD equivalents are added up and expressed as WHO-endorsed toxic equivalents (WHO-TEQ) (De Vito and Birnbaum 1995).

At present date, public concern over the adverse health effects of exposure to these toxicants has been enhanced by a number of dioxin contamination incidents involving food and feed. The contamination of milk, butter and meat by the use of contaminated citrus pulp in feedstuffs (Malisch 2000a, b), the Belgium dioxin episode in May 1999 in which a storage tank for animal fat was contaminated with PCBs and dioxins, the 'natural' presence of dioxin in kaolinic clays widely used as agent feed additives (Abad et al. 2000a, b; Malisch 2000a; Rappe and Anderson 2000) or the contamination of chlorine chloride premixtures (Llerena et al. 2001) are some remarkable examples.

The quantitative analysis of dioxin-like compounds should ideally include all chemicals showing the same biological activity and the dioxin-like activity. Two approaches are commonly used. On one hand, chemical methods have a scope restricted to specific target chemicals, as listed in official regulations. Each chemical is affected by a relative toxicity coefficient, the toxic equivalent factor or TEF. The toxic equivalent quantity (TEQ) is the sum of all quantities of toxics weighted by their TEF. On the other hand, biological methods such as chemical-activated luciferase gene expression (CALUX) (Wouwe et al. 2004) or ethoxy-resorufin-*O*-deethylase (EROD) monitor the global toxicity of a given sample (Schroijen et al. 2004; Schwirzer et al. 1998; Van Loco et al. 2004). Discrepancies arise from either the presence of unknowns or the existence of synergic and antagonist effects at the biological level, which modulates the TEF values. The TEF values are in addition largely species dependent (Brenez et al. 2004).

This review article will focus mainly on the human health risk by dietary dioxins and their sources, occurrence in various environmental and biological (human) samples.

Dioxins

Chemical structures and properties

Dioxins, as they are commonly called, are PCDDs and PCDFs are compounds with similar chemical properties. Each compound comprises two benzene rings interconnected by oxygen atoms. In the case of PCDDs, the benzene rings are joined by two oxygen bridges, and in the case of the PCDFs, the benzene rings are connected by a carbon bond and an oxygen bridge. The general formula of the PCDDs, PCDFs and PCBs is shown in Fig. 1.

All PCDDs and PCDFs are organic solids with high melting points and low vapour pressures. They are characterized by extremely low water solubilities, and have a tendency for being strongly adsorbed on surfaces of particulate matter. The water solubility of dioxin and furans decreases and the solubility in organic solvents and fats increase with increasing chlorine content.

Some of the key properties of the dioxins are presented in Table 1 and full physico-chemical properties have been reviewed (Mackay et al. 1992; US EPA-1613 1994). There are 75 PCDDs and 135 PCDFs, each differing in the number and position of the chlorine atoms. Each individual PCDD or PCDF is termed a congener (giving 210 in total),

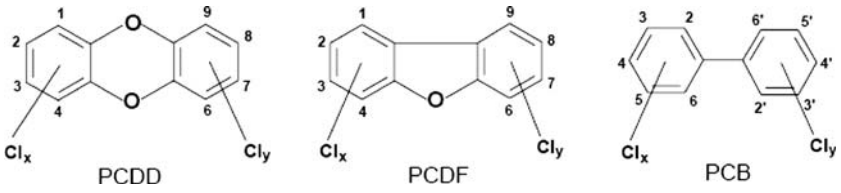

Fig. 1 General formula of polychlorinated dibenzo-*p*-dioxins (*PCDDs*), polychlorinated dibenzofurans (*PCDFs*) and polychlorinated biphenyls (*PCBs*). The possible number of chlorine atoms results in 75 PCDD congeners and 135 PCDF congeners (x = 1–4, y = 0–4), and 209 PCB congeners (x = 1–5, y = 0–5) (Baars et al. 2004)

Table 1 Typical physicochemical properties of PCDD/Fs (after Mackay et al. 1992; US EPA-1613 1994)

Homologue group	Vapour pressure (mmHg at 25°C)	Log K_{ow}	Solubility (mg L^{-1} at 25°C)	Henry's constant
TCDD	8.1×10^{-7}	6.4	3.5×10^{-4}	1.35×10^{-3}
PeCDD	7.3×10^{-10}	6.6	1.2×10^{-4}	1.07×10^{-4}
HxCDD	5.9×10^{-11}	7.3	4.4×10^{-6}	1.83×10^{-3}
HpCDD	3.2×10^{-11}	8.0	2.4×10^{-6}	5.14×10^{-4}
OCDD	8.3×10^{-13}	8.2	7.4×10^{-8}	2.76×10^{-4}
TCDF	2.5×10^{-8}	6.2	4.2×10^{-4}	6.06×10^{-4}
PeCDF	2.7×10^{-9}	6.4	2.4×10^{-4}	2.04×10^{-4}
HxCDF	2.8×10^{-10}	7.0	1.3×10^{-5}	5.87×10^{-4}
HpCDF	9.9×10^{-11}	7.9	1.4×10^{-6}	5.76×10^{-4}
OCDF	3.8×10^{-12}	8.8	1.4×10^{-6}	4.04×10^{-5}

while groups of congeners with the same number of chlorine atoms are called homologues. The number of congeners in each homologue group is shown in Table 2 (Van den Berg et al. 1998; US EPA-1613 1994). The homologue groups are often abbreviated for convenience; for example, tetrachloro CDDs and CDFs (PCDD/Fs with four substituted chlorine atoms) are abbreviated to TCDDs and TCDFs, respectively, while the fully chlorinated octachloro congeners (eight substituted chlorine atoms) are abbreviated to OCDD and OCDF, respectively (Srogi 2007a).

Table 2 Homologues and congeners of PCDDs and PCDFs (Van den Berg et al. 1998; US EPA-1613 1994)

Homologue (abbreviation)	Number of congeners	
	PCDDs	PCDFs
Monochloro (M)	2	4
Dichloro (D)	10	16
Trichloro (Tr)	14	28
Tetrachloro (T)	22	38
Pentachloro (Pe)	14	28
Hexachloro (Hx)	10	16
Heptachloro (Hp)	2	4
Octachloro (O)	1	1
Nonachloro		
Decachloro		
Total	75	135

Toxic equivalent schemes

It is widely accepted that the toxicologically active PCDDs and PCDFs exert their effects by a common mechanism involving binding to a cytoplasmic receptor protein called the Ah (aryl hydrocarbon) receptor. Certain PCB congeners also bind to the Ah receptor and can exhibit similar toxicological effects to the 2,3,7,8-substituted PCDDs and PCDFs. Since these compounds are considered to act by a common mechanism, and because they occur as mixtures in the environment, in food, and in human tissues, they are commonly assessed and regulated as a class. However, detailed toxicological information is available only for TCDD. Therefore, the concept of "TCDD equivalents" or "toxic equivalents" has been introduced to enable the assessment of the toxicity of mixtures of these compounds and to enable risk assessments of these mixtures to be carried out. This concept uses the available toxicological and in vitro biological data, and knowledge of structural similarities among the 2,3,7,8-substituted PCDD and PCDF congeners and the 13 "dioxin-like" PCB congeners (hereafter collectively termed "dioxins"), to generate a set of weighting factors or "toxic equivalency factors", each of which expresses the toxicity of a particular congener in terms of an equivalent amount of TCDD. Multiplication of

the concentration of the congener by its TEF gives a TCDD toxic equivalent (TEQ) (Pollitt 1999).

The TEQ concept was first developed in New York by the State Health Department in a series of experiments in response to the need for reentry criteria of an office building contaminated by a mixture of dioxins following an electrical transformer fire (Eadon et al. 1986). The TEQ approach and current values have been adopted internationally as the most appropriate way to estimate the potential health risk of mixtures of dioxins. Table 3 lists these dioxins (Van den Berg et al. 1998).

The report of Liem (1999) revealed that dietary intake is the main route (>90%) for human exposure to these toxicants. In view of this, in 1990, the World Health Organisation (WHO), based on the available data, suggested a tolerable daily intake (TDI) of 10 pg I-TEQ bw to ensure that the human population would not be exposed to levels that could give rise to adverse effects (WHO 1991). However, in 1998 a new reevaluation recommended to further reduce the TDI between 1 and 4 pg TEQ/kg bw (Van Leeuwen et al. 2000a). Meanwhile, wide-ranging efforts and stringent regulations aimed at reducing the dioxin release have been in forced [(EC) No.194/97 1999].

Table 3 Toxic equivalency factors to express the toxicity of mixtures of PCDFs, PCDDs and PCBs in toxin equivalents of 2,3,7,8-TCDD (WHO-TEF) (Van den Berg et al. 1998)

Structure	WHO-TEF
PCDDs and PCDFs	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
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	0.0001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,4,7,8-HxCDF	0.1
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001
Non-ortho PCBs	
3,3′,4,4′-CB(77)	0.0001
3,4,4′,5-CB(81)	0.0001
3,3′,4,4′5-CB(126)	0.1
3,3′,4,4′,5,5′-CB(169)	0.01
Mono-ortho PCBs	
2,3,3′,4,4′-CB(105)	0.0001
2,3,4,4′,5-CB(114)	0.0005
2,3′,4,4′,5-CB(118)	0.0001
2,3,4,4′,5-CB(123)	0.0001
2,3,3′,4,4′,5-CB(156)	0.0005
2,3,3′,4,4′,5-CB(157)	0.0005
2,3′,4,4′,5,5′-CB(167)	0.00001
2,3,3′,4,4′,5,5′-CB(189)	0.0001

Human health risk by dietary dioxins

The toxicology and human health effects of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and related compounds (i.e., the PCDDs and dibenzofurans, or PCDD/Fs) have been the focus of an extraordinary amount of research over the past 30 years (Srogi 2007b). Toxicologically, TCDD presents a range of interesting features, including its high degree of potency for several endpoints, its status as a confirmed animal carcinogen and teratogen, large inter-species and inter-strain variability in responses, and its receptor-based mechanism of toxicity (Hays and Aylward 2003; for e review, see ten Tusscher and Koppe 2004).

Body burdens of dioxins in breast-fed and non-breast-fed individuals

Dioxins are extremely persistent and bioaccumulative (Schecter et al. 2006). The half-life of TCDD in rodents is usually 2–4 weeks but in humans it has been estimated to be of 7–11 years although with wide individual variation. Other dioxins may be eliminated more or less rapidly with as little as a 6-month half-life of elimination estimated for some PCDFs, but 20 years for others.

Thoma et al. (1990) have reported levels of dioxins (PCDD and PCDF congeners only) in adipose tissue from eight infants aged 2–13 months and from 28 adults aged 31–80 years. The levels were lower in infants than in adults for all congeners except OCDF. Beck et al. (1990) measured the levels of dioxins (PCDD and PCDF congeners only) in the adipose tissue of a sudden infant-death baby (9.3 months old) who had been breast-fed for about 80 days. A level of 3.4 ng TEQ/kg body wt was found. In comparison, the levels in two non-breast-fed infants of 3.8 and 4.8 months of age were 2.8 and 2.1 ng TEQ/kg body wt, respectively. This cannot be explained by low absorption of dioxins in the breast-fed infant, since the bioavailability of dioxins from human milk has been shown to be about 95% (Pluim et al. 1993a). In another work

(Laurent et al. 2002) reported a study of portal absorption of dioxin using three ^{14}C-tagged compounds: ^{14}Cphenanthrene, ^{14}C-benzo[a]pyrene or ^{14}CTCDD (TCCD: 2,3,7,8-tetrachlorodibenzo-*p*-dioxin) in the growing pig. The analysis of portal and arterial blood radioactivity showed that TCDD was absorbed with a maximum concentration at 4–6 h after milk ingestion. Then, the blood radioactivity decreased to reach background levels 24 h after milk ingestion. Main ^{14}C absorption occurred during the 3–6 h time period for ^{14}C-TCDD. These results indicate that TCDD was partly and weakly absorbed.

Pollitt (1999) proposed a number of conclusions:

1. If the assumption is made that the half-life of all dioxin congeners in human milk is identical to that of TCDD (9 years), breast feeding is predicted to result in a higher body burden of dioxins in early life, but not to result in an increased steady-state body burden, compared to that resulting from ingestion of the TDI of 10 pg/kg body wt/day from birth.
2. For congeners with a significantly lower half-life, e.g., 1–3 years, breast feeding will lead to higher body burdens in early life than would have been reached by ingestion of the TDI from birth. However, these peak body burdens will still be below the steady-state body burden achieved by ingestion of 10 pg TCDD/kg body wt/day from birth.
3. The use of the toxic equivalent concept to estimate intakes of mixtures of dioxins is conservative when applied to congeners with half-lives shorter than that of TCDD, since it overestimates the likely accumulation of these congeners in human tissues.
4. The carcinogenicity study on which the TDI is based does not take account of any potential increased susceptibility to dioxins in early life. Otherwise, the TDI appears to accommodate the high intakes of dioxins by breast-fed babies, at least in relation to the end points on which it is based—carcinogenicity, fetotoxicity, and teratogenicity.
5. It is suggested that the toxicological database on TCDD is reexamined to assess whether postnatal development could be adversely affected by the high intakes of dioxins by infants during breast feeding.
6. More work is needed to assess whether breast-fed babies do achieve higher body burdens of dioxins than non-breast-fed babies, as predicted.

Toxic effects of dioxin

Dioxin-type chemicals produce a wide variety of species-specific effects including immunotoxicity, hepatotoxicity, birth defects, endocrine disruption, and the induction of numerous enzymes, most notably that of microsomal cytochrome P4501A1 (CYP1A1) and its associated mono-oxygenase activity, aryl hydrocarbon hydroxylase (for a review, see Diaz-Ferrero et al. 1997). Dioxins exert their effects via high-affinity binding to a specific cellular protein known as the arylhydrocarbon receptor (AhR) (Aoki 2001; Mitrou et al. 2001; Schecter and Gasiewicz 2003). The importance of this receptor is clearly evident from the observations that dioxin-induced symptoms, including organ atrophy and teratogenicity, are not present in AhR-knockout (KO) mice (Fernandez-Salguero et al. 1996). Thus, this receptor plays a crucial role in dioxin toxicity. However, immunosuppression produced by dioxins may occur by mechanism(s) not involving AhR. In connection with this, dioxin-induced changes in protein kinases (Brouwer et al. 1995; Denison and Heath-Pagliuso 1998), phospholipase *c* (Beebe et al. 1990) and low-density lipoprotein receptors (Matsumura et al. 1984) have been suggested to occur via an AhR-independent mechanism. Thus, some forms of dioxin toxicity do not seem to require AhR (Ishida et al. 2005.).

In humans and other vertebrates dioxins have been shown to be the risk factors for cancer; immune deficiency; central and peripheral nervous system pathology; endocrine disruption, including diabetes and thyroid disorders; decreased pulmonary functions and bronchitis; altered serum testosterone level; eyelid pathology, including meibomian gland hypersecretion and hyperpigmented conjunctivae; gum pigmentation; nausea; vomiting; loss of appetite; skin rashes, including, rarely, chloracne or acne caused by chlorine-containing organic chemicals; hypertrichosis; liver damage; elevated serum cholesterol and triglycerides; and enamel hypomineralization of permanent first molars in children.

According to Kociba et al. (1976) chronic exposure to TCDD impairs reproduction. In males, TCDD and its congeners cause decreased spermatogenesis and testicular weight, as well as degeneration of the seminiferous tubules. In females, the morphological lesions observed in the uterus and ovaries during TCDD exposure suggest that the estrus cycle may be suppressed. In addition, under these conditions, plasma progesterone and estrogen concentration have been found to be decreased.

The reader interested by all problems associated with human effects from occupational and epidemiological studies of dioxins may find necessary details in the following works: WHO (1997), CDC (1998), IOM (2001, 2005), ten Tusscher and Koppe (2004).

Exposure to dioxins

Dioxins are persistent, toxic and bio-accumulative (PTB) chemicals and because they can be transported over long distances from the source of emission, they are also per-

sistent organic pollutants (POPs). Combustion is considered a major source of the emissions of PCDDs and PCDFs. Dioxin production is a characteristic of controlled and uncontrolled combustion, e.g. wood (for a review, see Lavric et al. 2004) or pyrolysis process (Srogi 2007a) with strong relevance to health, safety and environmental issues in using biomass for energy (Lavric et al. 2004). Most important dioxin and furan air emission sources shown in Table 4 (Quaß et al. 2000).

Due to their physical properties, the bulk of the PCDD/F is adsorbed in dust and soot particles. These particles are deposited by atmospheric sedimentation (Masunaga et al. 2003) on soil and leafy vegetation such as grass. Depending on the distance from PCDD/F sources, deposition rates can vary considerably, and deposition rates exhibit strong seasonal trends.

In the following sections, main sources of PCDD/Fs in ecosystem include human tissue, food, water, air, soils and sediments will be described.

Human tissue

Breast-milk monitoring programs have been implemented in various countries to assess the importance of human exposure to organohalogen compounds and to predict body burden in the breast-feeding infant. Several public health and environmental benefits result from monitoring breast milk for contaminants. First, breast milk samples offer a convenient and non-invasive means of monitoring humans for the presence of lipophilic compounds. Second, such contaminant data provide an insight into environmental conditions and historical human exposure. Third, these measures complement general environmental monitoring and provide a more accurate assessment of human exposures. Finally, the dose delivered to the nursing infant can be estimated (Hooper et al. 1997). The lipophilicity of dioxin and related chemicals promotes their sequestration in the adipose tissue of the breast and concentration in the milk during lactation (Jensen 1991). As a consequence, breast-fed infants can have daily exposures, 10–20 times higher than the background population (Jödicke et al. 1992; McLachlan 1993).

Yang et al. (2002) determined breast-milk concentration of PCDDs/PCDFs and PCBs in 24 mothers living in Korea, and assessed the maternal body burden based on PCDDs/PCDFs and PCBs concentrations in breast milk and an infant intake rate through breast-feeding based on their concentration in breast milk. PCDDs/PCDFs and PCBs levels in breast milk from primipara mothers were found to be higher than those from multipara mothers. For total PCDDs/PCDFs TEQ level, 2,3,4,7,8-PeCDD was the predominant congener, and the proportion of 2,3,7,8-TCDD was less than 3% of total PCDDs/PCDFs TEQ level. For PCBs TEQ level, PCB-126 was the predominant congener. Maternal body burden levels of PCDDs/PCDFs and PCBs based on their concentrations in breast milk were 268–622 TEQ ng. The daily dioxin intakes of mothers were predicted to be 0.78–2.18 TEQ pg/kg/day for PCDDs/PCDFs and 0.34–0.66 TEQ pg/kg/day for PCBs. For the first year, the body burden of an infant was predicted to be 212 TEQ ng and the daily intake of an infant was predicted to be 85 TEQ pg/kg/day, assuming the mean dioxin-related compounds concentration (27.54 TEQ pg/g fat).

Sasamoto et al. (2006) presented follow-up survey results of PCDDs, PCDFs, and dioxin-like PCBs' concentrations in human breast milk samples from 35 women living in Tokyo from whom samples had been obtained and analyzed to determine the corresponding concentrations in the past. The average concentrations of total PCDDs, PCDFs, dioxin-like PCBs were 8.5, 5.5, 11 TEQ pg/g fat for the first samples

Table 4 Most important dioxin and furan air emission sources (Quaß et al. 2000)

Source type	PCDD/PCDF (g I-TEQ/year)	Emissions	Uncertainty of EF/AR
MSW incineration	1,437–174	Decreasing trend[a]	Low/low
Sinter plants[b]	1,010–115		Medium/low
Residential wood combustion	945	Extent of contaminated wood used uncertain	Medium/high
Clinical waste incineration	816	Few plant data and statistics	High/high
Wood preservation	381	From PCP-treated goods	V.high/v.high
Fires	380		V.high/v.high
Non-ferrous metals	136	Cu, Al, Zn	Medium/low
Road transport	111	Mainly leaded fuel; decreasing trend	Low/low
Total	5545		

I-TEQ international toxic equivalent, *EF* emission factor, *AR* activity rate

[a] Illegal domestic burning of MSW

[b] Sinter plant for recycled materials

and 5.4, 4.0, 6.6 TEQ pg/g fat for the second samples, respectively. The degrees of reduction of total PCDDs and total dioxin-like PCBs were higher than that of total PCDFs because 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF and 3,30,4,40,5-PeCB (#126), which were the predominant congeners among PCDDs, PCDFs, and dioxin-like PCBs, respectively, had different degrees of reduction. Moreover they observed a significant increase of the concentrations of PCDDs, PCDFs, and dioxin-like PCBs in samples from three women in this follow-up survey, and the patterns of increased isomers differed among the three samples. It was conjectured that the increase of the concentrations was due mainly to dietary intake between deliveries. It is important for pregnant women to have a balanced diet to mitigate the exposure of infants to these chemicals.

However, the total dioxin level in Japanese human milk was 250.4 pg/g fat on average (range 116.9–634.1 pg/g fat) for PCDDs + PCDFs + Co-PCBs, and their toxic equivalent was 22.0 pg TEQ/g fat on average (range 7.3–49.7 pg TEQ/g fat). The OCDD level was the highest, accounting for 29.6%. Next came PCB 126.24.3%, then PCB169, 14.1%. As for the contribution to TEF, 2,3,4,7,8-PeCDF accounted for 30.7%, PCB126 accounted for 27.6% and 3,30,4,40,5-PeCDD accounted for 14.3% (Takekuma et al. 2004). According to Hori et al. (1999) the level of total dioxin in human milk in 1973 (Japan) was 1920 pg/g fat (57.1 pg TEQ/g fat) for PCDDs + PCDFs + Co-PCBs, while the level of total dioxin in human milk (1996) was 279.9 pg/g fat (24.1 pg TEQ/g fat) for PCDDs + PCDFs + Co-PCBs. Thus, dioxin levels showed a decreasing tendency from 1973, and it has been reported that the level decreased 85% (toxic equivalent: 58%) during the period of 1973–1996.

According to Paumgartten et al. (2000) levels of PCDD/Fs and PCBs in a pooled sample of breast milk from 40 mothers living in the city of Rio de Janeiro were low compared to concentrations generally found in more industrialized countries (Table 5). They concluded that PCDDs were found at higher concentrations than PCDFs and that, in both cases, concentrations progressively increased from tetra- to octachlorinated congeners except for OCDF. The overall concentration of PCDD/Fs in human milk sample, as expressed by 2,3,7,8-TCDD toxicity equivalents (I-TEQ) calculated according to NATO publications (NATO 1988a, b), was 8.1 pg I-TEq/g milk fat, and 9.7 WHO-TEQ/g milk fat as computed according to WHO reevaluation (van den Berg et al. 1998), respectively. Although total PCDD/Fs and I-TEQ were low in mothers' milk from Rio de Janeiro, the concentrations of the highest chlorinated dioxin congeners, such as HpCDD and OCDD, in this sample tended to be slightly higher than levels generally found in more industrialized countries (WHO 1989, 1996). Recently, concentrations of PCDD/Fs and PCBs were determined in compost from 21 Brazilian municipal solid waste composing plants (Grossi et al. 1998). This study showed that concentrations of PCDD/Fs in Brazilian compost were similar to those found in Germany (Grossi et al. 1998). It was reported that PCBs were below the detection limit of the method in 29 individual samples of breast milk collected in Porto Alegre/Brazil in 1987/1988 (Beretta and Dick 1994). For this sample the PCB congener pattern is similar to that reported from other human milk samples of different origin (WHO 1996). Therefore the total PCB concentration can be estimated to be 0.15 mg/g milk fat by multiplying the sum of the concentrations from PCB-138, PCB-153, and PCB-180

Table 5 PCDD/F concentrations (pg I-TEQ/g fat) in human milk from different countries (after Paumgartten et al., 2000)

Sample/country	I-TEQ (pg/g milk fat)	Reference
Paris/France, 1990	20.1	González et al. (1996)
Madrid/Spain, 1990	13.3	González et al. (1996)
Tarragona/Spain	11.8	Schuhmacher et al. (1999)
Kanado/Czech Republic, 1993	12.1	Bencko et al. (1998)
Uhreské Hradistie/Czech Republic, 1993	18.4	Bencko et al. (1998)
Canada, 1986–1987	15.0	Ryan et al. (1993)
Germany, 1995	16.0	Päpke (1998)
Germany, 1990	31	Alder et al. (1994)
General population/China	2.6	Schecter et al. (1994)
Exposed to pentachlorophenol/China	5.4	Schecter et al. (1994)
Hanoi/Vietnam, 1988	2.1	Schecter et al. (1998)
Southern Vietnam, 1985–1994	5.2–11.0	Schecter et al. (1998)
Several locations, Kazakhstan, 1994	7.0–57.2	Hooper et al. (1998)
Rio de Janeiro/Brazil	8.1	Paumgartten et al. (2000)

(Ballschmiter and Zell 1980) with a factor of 1.6 (Schulte and Malisch 1984).

It should be noted that, a correlation between dioxin content in mothers' milk and thyroid function in the mothers as well as their infants was also reported by Koopman-Esseboom et al. (1994) in The Netherlands, i.e., higher dioxin levels in human milk correlated with lower levels of maternal thyroid hormones, and with higher plasma levels of TSH in the infants in the second week and third month after birth. Nagayama et al. (1997) reported a significant positive correlation between serum TSH and a negative correlation between T4 and estimated total intakes of dioxins and PCB from the breast milk. The levels of serum TSH and T4 were within normal ranges in their report. However, Matsuura et al. (2001a, b) could not find any correlation between serum levels of TSH, FT4 and estimated intake of dioxins. These authors collected breast milk at the second week after delivery and examined serum thyroid functions at 9–14 days of age (Koopman-Esseboom et al. 1994). Dioxin content in breast milk was higher in the early days after delivery than that of later days.

The mean level of the total dioxin in the milk of 95 mothers who smoked was 227.3 pg/g fat (19.8 pg TEQ/g fat), while the mean level of 204 mothers who did not smoke was 261.2 pg/g fat (23.1 pg TEQ/g fat). Most congener levels of the mothers who smoked were lower than the mothers who did not smoke, and the covariance analysis showed that significant differences were found with most congeners (Takekuma et al. 2004). The amount of smoking showed the dioxin levels decreasing with increasing tobacco consumption, supporting Fürst et al. (1992), who reported that mothers who are active, or even passive, smokers contain, on average significantly, lower PCDD/F levels than non-smoking women. While one report (Pluim et al. 1993b) found that smoking habits were not related to dioxin levels, results here showed that the dioxin levels in milk of mothers who smoked were lower than for mothers who did not smoke, and the dioxin levels were generally lower for mothers who were heavy smokers. It is thought that smoking somehow influences the accumulation of dioxin in the body.

It seems clear that it is necessary to consider age, smoking history, lactation in infancy and dietary habits, when dioxin levels are compared among individuals. It is particularly clear that the influence of smoking is significant. Life-style factors, such as smoking, age, and environmental factors, such as area of residence influenced the dioxin levels in human milk (Uehara et al. 2006).

In another work Chen et al. (2004) proposed the evaluation of the serum PCDD/F concentration distribution in residents in the vicinity of a MWI (municipal waste incinerators, Taiwan) and its association with the ambient predicted dioxin exposure. The highest PCDD/F congener level was OCDD, followed by 1,2,3,4,6,7,8-HpCDD, and OCDF; however, 1,2,3,6,7,8-HxCDD was the major contributors to TEQ. Results showed no significant correlations between serum and ambient PCDD/F levels. Residents exposed to the incinerator PCDD/F contamination did not seem to present higher PCDD/F serum concentrations. This might be explained by the fact that ambient exposure was not the single most important contributor to serum concentrations when compared to other sources of exposure such as dietary intake. This study also identified that higher serum concentrations of PCDD/Fs were presented in female groups, and older groups. The variation in serum PCDD/F levels was associated with gender and age, especially for 1,2,3,6,7,8-HxCDD, and OCDD, the two most major congeners contributing to the total PCDD/F levels. Similar patterns were also observed in other studies, especially a German study indicating increased body burden at older ages (Beck et al. 1994; Päpke 1998; Wittsiepe et al. 2000). Deml et al. (1996) also reported that all the congeners of hexa-, hepta- and octa-CDD were higher in women than in men. The above variation might be explained by another study indicating that the increasing half-life of most PCDD/F congeners may be associated with age and gender (Flesch-Janys et al. 1996). In addition, Michalek et al. (1992) also reported that the half-life of 2,3,7,8-TCDD was found with a marginally significant change in the percentage of body fat in 36 members of Operation Ranch Hand—the Air Force unit responsible for the aerial spraying of Agent Orange in Vietnam. Therefore, it may be concluded that old age was likely to show increased accumulation of adipose tissue and decreased metabolism, while the effect was expected to vary with gender (Flesch-Janys et al. 1996).

Dahlgren et al. (2003) reported the results of environmental sampling and modeling in a neighborhood adjacent to a wood processing plant. This plant used creosote and pentachlorophenol (PCP) to treat wood for over 70 years. Between 1999 and 2001, environmental samples were obtained to quantify the level of environmental contamination from the wood-processing plant. Blood from ten residents was measured for chlorinated dioxins and dibenzofurans. Soil sediment samples from drainage ditches and attic/dust samples from nearby residents' homes were tested for PCDD/Fs. The dioxin congeners analysis of the ten residents revealed elevated valued for octachlorodibenzo-*p*-dioxin and heptachlorodibenzo-*p*-dioxin compatible with PCP as the source. The levels of carcinogenic PAHs were higher than background levels and were similar to soil contamination on wood-preserving sites. Wipe sampling in the kitchens of 11 homes revealed that 20 of the 33 samples were positive for octachlorinated dioxins with a mean value of 10.27 ng/m^2. The soil, ditch samples, and positive wipe samples from the homes indicate a possible ongoing

route of exposure to the contaminants in the homes of these residents. Modeled air exposure estimated for the wood-processing waste chemicals indicate some air exposure to combustion products. The estimated air levels for tetrachlorodibenzodiozin in this neighborhood exceeded the recommended levels for these compounds in some states. The quantitative data presented suggest a significant contamination of a neighborhood by wood-processing waste chemicals. These findings suggest the need for more stringent regulations on waste discharges from wood treatment plants.

Food

Food is the main source of polychlorinated dibenzo-*p*-dioxin (PCDD), dibenzofuran (PCDF) and coplanar polychlorinated biphenyl (c-PCB) exposure for human, accounting for 98% of the total intake (for a review, see Fries 1995). It is also known that animal products like meat (Vartiainen and Hallkainen 1994), fish, milk and milk products (Schmid et al. 2003) or bovine adipose tissue and hen's eggs are the main contributors to human dioxin exposure in adults (Vartiainen and Hallkainen 1994; Parzefall 2002; Kim et al. 2004) and contribute largely to the human burden, as PCDD/Fs are bioaccumulated because of their lipophilicity and their low biodegradability (De Fré and Wevers 1998; Liem 1999; Focant et al. 2002; Ryan et al. 1987; Fernandes et al. 2004). Irigaray et al. (2005) on the base of results suggested the risk of a strong increase of 2,3,7,8-TCDD in blood induced by lipolysis for animals or humans previously exposed to this dioxin.

Therefore, dioxin levels in milk can be used as indicators for the actual average local dioxin exposure by atmospheric deposition. In the past, cow's milk has been used for the evaluation of point source emissions (Travis and Hattermer-Frey 1991; Fries et al. 2002).

In a risk assessment of dioxins and dioxin-like PCBs in the diet, the Scientific Committee for Food (SCF) of the European Commission assessed a tolerable weekly intake (TWI) of 14 pg/kg body weight (bw) for these chemicals as toxic equivalents (WHO-TEQ), according to the WHO TEF scheme (European Commission 2001; Van den Berg et al. 1998). Exposure estimates, made by SCF, indicated that a proportion of the European population has a dietary intake of dioxins and dioxin-like PCBs, which is in excess of the TWI.

In accordance with the recommendations of the French Conseil supérieur d'hygiéne publique (1997) (and European Union recommendations), the results of analyses were interpreted according to a non-commercialization threshold of 5 pg TEQ/g of fat (the "target" threshold being of 1 pg TEQ/g of fat). For example, Durand et al. (2000) no concentration were found above the threshold recommended by the French Conseil supérieur d'hygiéne publique (1997) and by the European Union: 5 pg TEQ/g of fat (the maximal observed concentration was 1.75 pg TEQ/g of fat). Thus, the study showed a very low contamination level of the long-life half-skimmed drinking milk far below the target value of 1 pg TEQ/g of fat. The observed results were of the same order of magnitude as results published in the literature for other countries (for example Germany (Blüthgen et al. 1996; Hipplein et al. 1996), UK [MAFF Food Surveillance Information Sheets, various years 100 (January 1997), 107 (June 1997), 120 and 123 (August 1997), 133 and 134 (November 1997), 136 (December 1997), 143 (March 1998)], Finland [Hallikainen and Vartiainen 1998), or Netherlands (Hendriks et al. 1996)].

Although consumption of poultry products contributes 5–10% of the estimated daily intake in industrial countries (Focant et al. 2002), the available literature on transfer of these pollutants from feed to chickens and eggs is limited. However, poultry products have already been highlighted several times in terms of food chain safety, notably in the US in 1997 (Hayward et al. 1999), when ball clay naturally contaminated with high levels of dioxins was added to poultry and fish feed. More recently, in Belgium, contaminated recycled mineral oil was introduced into animal feed, causing a vast economic and political crisis (Bernard et al. 2002). In both accidents, thousands of chickens and eggs were removed from the market in order to reduce exposure risk, resulting in significant economic losses. These events revealed a need to better understand transfer mechanisms of these pollutants from the animal environment to their various compartments, such as eggs, fat or meat. Although incidents of food-chain contamination have occurred many times through consumption of contaminated feedstuffs in commercial chicken farms, few studies have examined the transfer of dioxins and related compounds from commercial feed to chickens. Recently, Maervoet et al. (2004) have studied accumulation and tissue distribution of seven PCBs in chickens. Iben et al. (2003) fed broiler chickens with reasonably low dioxin level feed to determine contamination in edible tissue, and Hoogenboom et al. (2004) have investigated PCB and PCDD/F kinetics in broiler after being fed contaminated feed.

For example, concentrations of PCDDs, PCDFs and Co-PCBs in domestic animal-related samples were summarized in Table 6 (Guruge et al. 2005). Fish oil contained the highest concentration of PCDDs among feed ingredients. Concentrations of PCDDs were three- to tenfold greater than PCDFs in all the feed ingredients. Total PCDDs/PCDFs concentration in fish oil was twofold greater than those in fishmeal and meat and bone meal. The detection frequencies were 100% for 1,2,3,4,6,7,8-HpCDD, OCDD and 1,2,3,4,6,7,8-HpCDF in feed ingredients. These three

Table 6 Mean and range of concentrations (pg g/1 lipid wt) of PCDDs, PCDFs and Co-PCBs in domestic animal related samples (Guruge et al. 2005)

	Fish oil	Fish meal	Meat and bone meal	Cattle feed	Chicken feed	Pig feed	Chicken fat	Pig fat
Number of samples	5	7	5	1	2	2	5	3
Lipid (%)	100	9.5	11	4.8	5.1	3.6	77	78
2,3,7,8-TeCDD	0.6	0.05	0.034	<0.02	<0.02	<0.02	<0.02	<0.02
	(<0.02–1.2)	(<0.02–0.36)	(<0.02–0.17)		<0.02	<0.02	<0.02	<0.02
1,2,3,7,8-PeCDD	0.9	0.13	<0.02	<0.02	<0.02	<0.02	0.23	0.3
	(0.66–1.1)	(<0.02–0.70)	<0.02		<0.02	<0.02	(<0.02–0.92)	(<0.02–0.87)
1,2,3,4,7,8-HxCDD	0.63	0.10	0.077	0.26	<0.02	<0.02	<0.02	<0.02
	(<0.02–1.2)	(<0.02–0.39)	(<0.02–0.23)		<0.02	<0.02	<0.02	<0.02
1,2,3,6,7,8-HxCDD	0.89	0.12	0.11	0.19	<0.02	<0.02	0.43	0.22
	(<0.02–1.4)	(<0.02–0.53)	(<0.02–0.52)		<0.02	<0.02	(<0.02–1.7)	(<0.02–0.65)
1,2,3,7,8,9-HxCDD	<0.02	0.09	0.13	0.29	<0.02	0.22	<0.02	<0.02
	<0.02	(<0.02–0.43)	(<0.02–0.40)		<0.02	(<0.02–0.44)	<0.02	<0.02
1,2,3,4,6,7,8-HpCDD	5.2	2.5	2.4	2.5	1.9	3	3.9	<0.02
	(3.6–7.8)	(0.62–5.5)	(0.70–5.5)		(1.4–2.3)	(2.6–3.3)	(1.5–9.1)	<0.02
OCDD	39	19	19	9.5	9.5	15	5.4	11
	(31–55)	(2.9–45)	(3.8–59)		(6.0–1.3)	(9.1–21)	(<0.02–14)	(7.4–14)
2,3,7,8-TeCDF	2.9	0.51	0.03	<0.02	0.18	<0.02	3.2	0.27
	(1.6–3.6)	(0.12–0.94)	(<0.02–0.13)		(0.10–0.26)	<0.02	(2.1–4.6)	(<0.02–0.81)
1,2,3,7,8-PeCDF	1.5	0.07	<0.02	<0.02	0.17	<0.02	<0.02	<0.02
	(<0.02–2.4)	(<0.02–0.27)	<0.02		(<0.02–0.34)	<0.02	<0.02	<0.02
2,3,4,7,8-PeCDF	2.0	0.35	0.08	<0.02	0.07	0.12	1.6	0.47
	2.0	(<0.02–0.69)	(<0.02–0.22)		(<0.02–0.14)	(<0.02–0.24)	(<0.02–3.4)	(<0.02–0.75)
1,2,3,4,7,8-HxCDF	0.50	0.11	0.039	0.21	0.05	<0.02	0.75	0.3
	(<0.02–0.89)	(<0.02–0.37)	(<0.02–0.19)		(<0.02–0.10)	<0.02	(<0.02–1.7)	(<0.02–0.91)
1,2,3,6,7,8-HxCDF	0.52	0.10	0.1	<0.02	0.08	<0.02	0.53	0.14
	(<0.02–0.88)	(<0.02–0.39)	(<0.02–0.49)		(<0.02–0.16)	<0.02	(<0.02–1.6)	(<0.02–0.43)
1,2,3,7,8,9-HxCDF	<0.02	<0.02	<0.02	<0.02	0.19	<0.02	<0.02	<0.02
	<0.02	<0.02	<0.02		(<0.02–0.38)	<0.02	<0.02	<0.02
2,3,4,6,7,8-HxCDF	0.78	0.08	0.24	<0.02	0.15	<0.02	0.61	<0.02
	(<0.02–1.5)	(<0.02–0.38)	(<0.02–1.2)		(<0.02–0.29)	<0.02	(<0.02–1.3)	<0.02
1,2,3,4,6,7,8-HpCDF	3.7	0.94	1.2	1.5	0.93	1.7	2.3	0.22
	(2.7–5.1)	(0.58–1.4)	(0.59–2.5)		(0.59–1.3)	(0.96–2.5)	(0.55–6.8)	(<0.02–0.66)
1,2,3,4,7,8,9-HpCDF	<0.02	<0.02	0.26	<0.02	<0.02	<0.02	<0.02	<0.02
	<0.02	<0.02	(<0.02–1.3)		<0.02	<0.02	<0.02	<0.02
OCDF	2.5	0.18	1.35	1.3	1.5	1.4	<0.02	<0.02
	(<0.02–4.2)	(0.02–0.86)	(<0.02–3.9)		(0.71–2.3)	(<0.02–2.9)	<0.02	<0.02
3,4,4′,5-TeCB(81)	9.4	1.3	0.38	0.11	0.17	0.33	3.4	0.16
	(5.9–12)	(0.25–3.0)	(0.10–0.60)		(0.15–0.18)	(0.20–0.46)	(1.7–6.0)	(<0.07–0.35)
3,3′,4,4′-TeCB(77)	130	24	6.5	1.4	1.7	2.5	40	2.5
	(75–170)	(8.7–46)	(3.5–10)		(0.97–2.5)	(2.2–2.9)	(19–60)	(1.6–2.8)
3,3′,4,4′5-PeCB(126)	76	10	1.3	0.08	0.42	0.71	8.4	0.45
	(64–95)	(2.2–19)	(0.64–2.7)		(0.37–0.48)	(0.46–0.97)	(3.7–14)	(<0.07–0.70)
3,3′,4,4′,5,5′-HxCB(169)	18	3.7	0.39	<0.07	0.01	0.15	0.97	0.5
	(<0.07–31)	(0.26–19)	(0.04–0.66)		(0.07–0.13)	(0.15–0.16)	(0.89–1.9)	(<0.07–0.76)
2,3,3′,4,4′-PeCB(105)	3000	360	91	4.9	34	17	380	17
	(1,800–4,000)	(33–750)	(5.3–400)		(5.1–63)	(14–20)	(140–740)	(<0.07–33)

Table 6 continued

	Fish oil	Fish meal	Meat and bone meal	Cattle feed	Chicken feed	Pig feed	Chicken fat	Pig fat
2,3,4,4′,5-PeCB(114)	230	23	7.6	0.97	2.5	2.2	34	7.6
	(150–290)	(2.0–55)	(0.80–32)		(0.38–4.6)	(2.0–2.3)	(20–54)	(5.6–9.3)
2,3′,4,4′,5-PeCB(118)	9200	1000	340	11	110	51	1300	160
	(5,600–12,000)	(86–2,200)	(16–1,500)		(16–190)	(37–65)	(400–2,500)	(130–180)
2,3,4,4′,5-PeCB(123)	1500	110	10	1.6	11	4.9	75	6.5
	(320–2,400)	(7.8–460)	(1.2–39)		(0.78–21)	(3.3–4.0)	(47–130)	(4.1–10)
2,3,3′,4,4′,5-HxCB(156)	1100	140	27	1.1	6.6	6.3	76	43
	(690–1,500)	(10–280)	(1.8–110)		(1.9–11)	(3.9–8.6)	(30–140)	(41–47)
2,3,3′,4,4′,5-HxCB(157)	300	34	7.4	0.22	1.9	1.7	20	11
	(200–390)	(3.0–69)	(0.50–31)		(0.62–3.3)	(0.78–2.6)	(8.5–35)	(11–13)
2,3′,4,4′,5,5′-HxCB(167)	930	170	47	0.41	4.8	8.7	170	58
	(640–1,300)	(21–380)	(1.1–210)		(3.0–6.7)	(2.0–15)	(20–350)	(28–82)
2,3,3′,4,4′,5,5′-HpCB(189)	140	16	1.9	0.25	0.62	1.4	7.7	5.1
	(82–200)	(1.5–32)	(0.21–5.3)		(0.29–0.96)	(0.63–2.1)	(4.0–14)	(4.6–5.8)

congeners were commonly found in various fish and farm animals (Fries 1995; Scortichini et al. 2001; Guruge and Tanabe 2004) suggesting that their contribution to animal-originated feed ingredients were greater when compared to other PCDD/DF congeners. 2,3,7,8-TeCDF was found in all the fish oil and fishmeal samples while being found only in one imported meat and bone meal sample. The total concentrations of PCDDs and PCDFs in chicken feed (11 and 3.3 pg/g lipid wt) were similar to those in pig feed (18 and 3.2 pg/g lipid wt). Compared to PCDFs, four to sixfold higher PCDD concentrations were found in mixed feeds. Most of PCDDs congeners were not detected (<0.02 pg/g lipid wt) in chicken feeds. Detection frequency was 100% for 1,2,3,4,6,7,8-HpCDD and OCDD, while most of other PCDDs and PCDFs were not detected in pig feed. This specific accumulation of PCDDs and PCDFs was identical to those earlier reported data for mixed animal feeds (McLachlan and Richter 1998; Scortichini et al. 2001).

The PCDF concentration in chicken fat (9.0 pg g lipid wt) was severalfolds higher than that of pig fat (1.4 pg/g lipid wt), while PCDDs concentration was similar. Detection frequency was 100% for congeners 1,2,3,4,6,7,8-HpCDD, OCDD and 1,2,3,4,6,7,8-HpCDF in chicken fat. Only congener OCDD was detected in all the pig fat samples. The chickens and pigs were approximately 6 months old when sampled. It was noticed that dioxin concentration in pig fat was decreased while unchanged in chicken fat during their later period of growth.

The sum of concentrations of 12 coplanar PCBs in fish oil was 17,000 pg/g lipid wt (Table 6) (Guruge et al. 2005). The fish oil accumulated 9- and 30-fold greater coplanar PCBs than those from fish meal and meat and bone meal, respectively. Fish oil is being strongly promoted as an aquaculture additive to improve the nutritional value to human in Europe (Jacobs et al. 2002). Therefore, these oils could contribute greatly to the contamination of farm-animals. Jacobs et al. (2002) noted that variation in oil sources and processing procedures could be result in inter batch differences in contamination levels in the final products. Guruge et al. (2005) also observed different concentrations of contaminants between imported and locally produced feed ingredients. Congener 118 accounted for the major proportion of the total coplanar PCB concentrations in all the samples. A similar bioaccumulation feature was observed in farmed fish food web (Jacobs et al. 2002) where congener 118 was predominated. Congener 77 was foremost throughout the domestic animal food web for non-ortho PCBs.

Twelve chickens raised according to commercial standards were fed a diet containing about 30 ng TEQ/kg for 10 weeks (Pirard and Pauw 2005). Persistent pollutants were introduced into the poultry feed via recycled oil to mimic contamination conditions closely resembling those occurring during the Belgian crisis 5 years ago. Absorption of congeners with the same chlorination degree did not seem to depend on the substitution, demonstrating that unlike for cows, no preferential absorption for 2,3,7,8-substituted compounds could be observed for chickens. As already observed, absorption decreased with increasing number of chlorines and was not linearly dependent on the octanol/water partition coefficient. On the other hand, no real differences were observed in the absorption of c-PCBs with regard to degree of chlorination. When monitored during the course of experiment, concentrations of PCDD/

Fs and c-PCBs in excreta reached an apparent steady state after 5 weeks. Only 2,3,7,8-substituted dioxins or furans were found in tissues and eggs. All organs showed the same congener profile and similar lipid-normalized concentration, except for the liver. Bioconcentration factors were evaluated, highlighting that the liver preferentially retained highly chlorinated congeners. No depletion of dioxin and PCB concentration was observed after 8 and 14 weeks of control diet, but high inter-individual variation occurs.

According to Kim et al. (2001) concentrations of PCDDs in hamburgers (0–20 fg/g) were detected in lower levels than in fried chickens (16.92–252.00 fg/g). Specially fried chickens show the high contents of 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD that have high TEQ factors (TEQ, 1.0). The TEQ levels of PCDDs in hamburger were lower than in fried chicken. Total TEQ level of PCDD in fried chicken was 47.45 times higher than in hamburger.

In USA, Scheter et al. (1995) reported values from 0.10 to 5.17 pg I-TEQ/g, Fiedler et al. (1997a) presented mean values in chicken samples of 0.7 ± 0.06 with a maximum of 0.78 and a minimum of 0.61, and Ferrario and Byrne (2000) mentioned values about 1.3 pg I-TEQ/g. Fürst et al. (1990) reported poultry concentrations of 1.4 and 2.3 pg I-TEQ/g in Germany. In Canada the concentration of PCDDs/PCDFs in poultry samples was 2.6 pg I-TEQ/g (Fürst et al. 1991) and Theelen et al. (1993) reported a concentration around 1.7 pg I-TEQ/g in The Netherlands.

Kiviranta et al. (2004) have measured the concentrations of PCDD/F and PCBs in ten market baskets consisting of almost 4,000 individual food samples representing 228 different food items, and also in the total diet basket. Lower bound concentrations of PCDD/Fs ranged between 0.0057 and 5.6 pg/g fresh weight in the market baskets and the corresponding values for PCBs from 39 to 25.000 pg/g. The fish basket contributed most to the concentrations of dioxins and PCBs, in which the lower bound range was from 0.82 to 850 pg/g. These authors also assessed the average daily intakes of these substances by the Finnish adult population. The average daily intake of sum of PCDD/Fs and PCBs as WHO toxic equivalents was assessed to be 115 pg, which was 1.5 pg WHO-TEQ/kg body weight using an average mean weight of 76 kg for the general population in Finland. The contribution of fish to the intake of PCDD/Fs was between 94 and 72%, depending on whether lower or upper bound concentrations were used. With respect to PCBs, the contribution of fish was 80%. Table 7 (Kiviranta et al. 2004) provides an overview of the average daily dietary intakes of dioxin- and PCB TEQs of adult populations from a number of countries. In addition, the food groups that contribute most to the intake of dioxins are resented. It is a difficult task to compare the results of intake estimations between countries because there are notable differences in the analytical methods, e.g., upper bound versus lower bound concentrations used and set of TEFs utilized. There are differences between studies in collection methods and number of foods analyzed, and differences in the means to study food consumption. The daily intake of dioxins ranged between 29 pg I-TEQ in Norway (SCOOP 2000) and 104 pg WHO-PCDD/F-TEQ in the USA (Schecter et al. 2001a), and of PCBs from 31 pg WHO-PCB-TEQ in Sweden (Lind et al. 2002) to 110 PCB-TEQ in Norway. The recent Finnish TEQ estimates of daily intakes (46–61 pg in dioxins and 51–60 in PCBs) were within these ranges reported from other countries. The Finnish daily intake of WHO-PCDD/F-TEQ together with WHO-PCB-TEQ per bw was 1.5 pg/kg bw in this study which is at the lower end of the tolerable daily-intake (TDI) range set by WHO, 1–4 pg TEQ/kg bw (Van Leeuwen and Younes 2000b). None of the reported daily intakes in Table 7 (Kiviranta et al. 2004) exceeded the WHO TDI upper range value. The TWI of TEQs in Finland was 10.5 pg WHO-TEQ/kg bw, which is also below the highest recommended TWI value of 14 pg WHO-TEQ/kg bw given by EU (2001). In the future, analyses using distributional information for consumption data are needed in order to assess the percentage of Finns exceeding the TWI.

The levels in milk are strongly correlated with the fat content of the milk (Noren 1988) and influenced by the concentration of adipose tissue. Infants are exposed to PCDDs, PCDFs, and dioxin-like PCBs prenatally and via breast milk (Päpke 1998; Schecter 1998; Schecter et al. 1998; Wang et al. 2004). In the most industrialized countries, concentrations of PCDD/Fs and other organochlorine compounds have been regularly monitored in human milk and a rather large database on the general population contamination is currently available.

In many countries, breast-milk samples have been used as a suitable source of material for examining the level of human exposure to these compounds. Moreover, breast milk is the main conduit for discharging these compounds from the human body, and it is known that the levels of these compounds in human breast milk from mothers nursing their second child are lower than those from mothers breast-feeding their first child (Fürst et al. 1989; Kiviranta et al. 1998).

In the another work (Lai et al. 2004), 100 (from Hong Kong) and 48 (from Guangzhou) breast milk extracts were collected to determine the levels of dioxin-like compounds, of which 65% and 68 of the samples, respectively, were found to contain detectable dioxin-like activities using the H4IIE cell EROD screening assay. The mean EROD-TEQ values of the 65 samples from Hong Kong ranged from 58.1 to 96.5 pg/g of milk fat while the 32 samples from Guangzhou showed mean values of 98.8–202.1 pg/g of

Table 7 Average daily intakes TEQs, PCB, TEQs as pg and (pg/kg bw), and contributions of different ford groups to the dioxin exposure (after Kiviranta et al. 2004)

Country, study period	Daily intake, pg, (pg/kg bw)					Contribution of foods from dioxins (%)					Reference
	I-TEQ	$WHO_{PCDD/F}$-TEQ	PCB-TEQ	WHO_{PCB}-TEQ	Method[a]	Dairy	Meat, poultry	Eggs	Fish	Other[b]	
Finland, 1999	55 (0.72)	58 (0.76)	60 (0.79)	56 (0.74)	0	2	2[c]		94	2	Kiviranta et al. (2004)
Finland, 1999	58 (0.76)	60 (0.79)	60 (0.79)	56 (0.74)	LOQ	14	5[c]		72	9	Kiviranta et al. (2004)
Finland, 1999	46 (0.61)		53 (0.70)		0	8	7	2	82	1	Kiviranta et al. (2001)
Finland, 1999	61 (1.01)		51 (0.84)		LOQ	16	6	4	63	11	SCOOP (2000)
Japan, 2000		82 (1.64)		79 (1.59)	0.5 × LOQ	2	12[c]		71	15	Tsutsumi et al. (2001)
Norway, 1997	29		110		LOQ	22	14	12	46	6	SCOOP (2000)
Korea, 1999	30 (0.51)				Unknown	1	4	5	39	51	Kim et al. (2000)
Belgium, 2001		65			0	30	31		39		Focant et al. (2002)
Sweden, 1999		44 (0.62)		31 (0.43)	0.5 × LOQ	19	15	1	36	51	Lind et al. (2002)
Sweden, 1999	68 (1.06)		63 (0.85)		LOQ	19	31	2	34	14	SCOOP (2000)
Italy, 1996	45 (0.74)				0.5 × LOQ	26	32	7	35	-	SCOOP (2000)
Spain, 2000	78	95			0.5 × LOQ	27	13	2	30	28	Llobet et al. (2003)
China, 2000		72			Unknown	16	35	21	28		Wu et al. (2002)
France, 1999	97 (1.45)				LOQ	33	13	2	26	26	SCOOP (2000)
Germany, 1998	51 (0.73)				0.5 × LOQ	39	30	11	11	9	SCOOP (2000)
The Netherlands, 1999		45 (0.65)		46 (0.85)	0	24	21	5	10	40	Freijer et al. (2001)
The Netherlands, 1991	82		81		LOQ	39	20	4	2	35	SCOOP (2000)
The United Kingdom, 2001		(0.4)		(0.5)	LOQ	44	18	1	6	31	FSA, report 38/03 (2003)
The United Kingdom, 1992	88 (1.26)		57 (0.81)		LOQ	25	20	4	6	45	SCOOP (2000)
USA, 1995	29	104 (1.66)		42 (0.67)	0.5 × LOQ	29	30	7	6	28	Schecter et al. (2001)

[a] Method of denoting concentrations of unquantified congeners in intake calculations: 0 = lower bound, 0.5 × LOQ = medium bound, LOQ = upper bound

[b] Other = e.g. cereals and cereal products, vegetables, fruit, vegetable fats and oils

[c] Includes meat, poultry, and eggs

milk fat. The remaining samples (35% of those from Hong Kong and 32% of those from Guangzhou) showed negative responses in the EROD screening assay. This might be attributable to the detection limit of the assay method, or dioxin-like compounds may truly have been absent in the samples. In comparing the EROD-TEQ values for the different age groups between the two cities, there were no significant differences ($P < 0.05$). However, the mean and median EROD-TEQ values for the Guangzhou population were in general higher than those for the Hong Kong group (Table 8). In other countries, the detectable dioxin concentrations, in terms of chemical-TEQ (C-TEQ), have been 9.6–35 pg/g fat (PCDD/PCDF) in Sweden (Glynn et al. 2001), 9.9–48.5 pg/g fat (PCDD/PCDF/CoPCB) in Japan (Nakagawa et al. 1999), 16–40.2 pg/g fat (PCDD/PCDF) in the Republic of Uzbekistan (Ataniyazova et al. 2001), 21–53 pg/g fat (PCDD/PCDF) in agricultural regions of southern Kazakhstan (Hooper et al. 1999), and5.9–17.1 pg/g fat (PCDD/PCDF) in Spain (Schuhmacher et al. 1999). LaKind et al. (2001) reported a review of worldwide-data on C-TEQs (PCDD/PCDF) in breast milk. During the years 1970–1996, the worldwide-reported C-TEQ values were in the range of 3.1–484 pg/g fat. The highest value was reported in Vietnam in 1970, mainly due to the spraying of Agent Orange during the Vietnam War. Despite the results indicated above, EROD-TEQ and CTEQ analyses have particular pros and cons, and thus caution should be taken in when interpreting the data. It was understandable that EROD-TEQ detected the interaction of all AhR agonists, including both identified and unknown species. On the contrary, the C-TEQ approach could not detect all AhR agonists and thus by itself is incomplete. Chemical analysis indicated the type of contaminants that could be transferred to newborns during breast feeding; however, this is not indicative of the biological or toxicological consequences of their exposure. In addition, because different studies adopt a variety of methods, different C-TEQ data are not always comparable. Although there was a very good correlation between EROD-TEQ and C-TEQ, it has been reported that using rat primary hepatocyte culture, EROD-TEQ has produced data two to fivefold higher than the calculated C-TEQ (Schmitz et al. 1995; Schrenk et al. 1991; Till et al. 1997). Hence, if the data of the present study were divided by a factor of 2 or 5, the recalculated mean levels in our region would be in the range of 29.1–101.1 or 11.62–40.2 pg/g fat, respectively. These levels would be within the range of contamination reported and were comparable to those of other countries.

According to Schmid et al. (2003) concentrations of PCDD/F in milk from farms near point sources (0.63 ± 0.26 ng I-TEQ/kg milk fat) were slightly but significantly higher in than milk from remote areas (0.36 ± 0.09 ng I-TEQ/kg milk fat). Consumer milk collected at the processing plants had intermediary levels (0.51 ± 0.19 ng I-TEQ/kg milk fat). Also in 1998, Malisch (1998) detected an increase of dioxin levels in milk and traced this back to the use of contaminated citrus pulp from Brazil. The pulp had been mixed with contaminated lime, being a waste product from a PVC production plant. The incident also had a major impact in the Netherlands where milk levels increased up to threefold. Since the contamination was only discovered after several months, most of the products had been consumed. Furthermore, the contamination could spread through the recycling of contaminated slaughterhouse offal (Hoogenboom et al. 2004). Also, the levels of PCDD/Fs in a pooled sample of breast milk were determined by Paumgartten et al. (2000). All samples, from 40 mothers living in the urban area of Rio de Janeiro County (Brazil), were collected between 4 and 6 weeks after delivery. The results showed a dioxin equivalent concentration of 8.1 pg I-TEq/g milk fat.

In 1994, UK scientists (Ahlborg et al. 1994) showed that the upper bound dietary intake of dioxins by the average adult consumer was estimated as 2.4 pg TEQ/kg body wt/day or 144 pg TEQ/day for a 60-kg adult (the upper bound estimate is calculated using the assumption that where the levels of the individual congeners are below the limit of detection, they are present at the limit of detection).

In another work (Abad et al. 2002) dioxin content in the 19 milk samples (Spain) analyzed ranged from 0.09 to 0.90 pg I-TEQ/g milk fat with a median of 0.35 pg I-TEQ/g fat and an average value of 0.36 pg I-TEQ/g fat. These

Table 8 TEQ values (pg/g milk fat) of human breast milk collected from Hong Kong and Guangzhou (Lai et al. 2004)

Age (years)	Number		TEQ (pg/g fat), mean ± SD		TEQ (pg/g fat), median	
	HK	GZ	HK	GZ	HK	GZ
21–25	5	5	58.1 ± 31.1	115.8 ± 79.6	56.1	94.8
26–30	17	17	96.5 ± 56.6	202.1 ± 217.8	100.5	112.3
31–35	36	6	83.2 ± 77	98.8 ± 49.8	51.7	82.8
33–34	4	4	92.1 ± 88.7	135.7 ± 58.2	53.8	138
35–36	3	–	71.75 ± 20.38	–	62	–

HK Hong Kong, *GZ* Guangzhou

values were below the background levels (between 1.3 and 2.47 pg I-TEQ/g fat) determined in other sites from Spain (Ramos et al. 1997). However, the values increased from 0.37 to 2.22 when co-PCBs are considered, having a median value of 0.795 pg I-TEQ/g and an average of 1.015 pg I-TEQ/g. The results expressed in WHO-TEQ ranged from 0.11 to 1.08 pg/g fat milk (average and median of 0.43 and 0.41 pg WHO-TEQ/g fat, respectively) and 0.398–2.402 pg/g fat milk including co-PCBs (average and median of 1.078 and 0.859 pg WHO-TEQ/g fat). In general, the dioxin contamination of the milk samples studied was low and in the range of French (Vindel et al. 1999; Durand et al. 2000) and German average (Mayer 1995; Malisch et al. 1999) or some particular sites in USA (Fiedler et al. 1997b). While the 2,3,4,7,8-PeCDF was the major contributor (40%) in Bavarian samples (Mayer 1995), 1,2,3,7,8-PeCDD and 1,2,3,6,7,8-HxCDD contributed mainly to the total I-TEQ (25 and 22%, respectively) in samples from USA (Fiedler et al. 1997a). In the study (Abad et al. 2002), the 2,3,4,7,8-PeCDF was the major contributor in Spain samples (approx. 30%), followed by 1,2,3,7,8-PeCDD with approx. 18%. So far, all samples analyzed presented dioxin content below the limit of 5 pg I-TEQ/g fat established for its commercialization in the European countries and below the limit of 3 pg WHO-TEQ/g proposed in the EC Regulation draft.

According to Schmid et al. (2003), the PCDD/F levels in Swiss consumer milk (pooled milk from industrial milk processing plants) were 0.51 ± 0.19 ng I-TEQ/kg milk fat. This level was only slightly above those determined in milk from rural/alpine regions with an average PCDD/F content of 0.36 ± 0.093 ng I-TEQ/kg milk fat. Milk collected from the proximity of potential and former point sources had PCDD/F levels of 0.63 ± 0.26 ng I-TEQ/kg milk fat, which was slightly but significantly elevated compared to milk from remote areas: the results of a two-sample Wilcoxon rank-sum test indicate that the medians of the two datasets are statistically different ($P = 0.0054$). These levels were well in line with the most recent national average PCDD/F levels in countries of the European Union being in a range of 0.32–2.1 ng I-TEQ/kg milk fat (European Commission Health and Consumer Protection Directorate-General, 2000). Based on the average level in milk from industrial processors (0.59 ng WHO-TEQ/kg milk fat) and an intake of total dairy fat of 44.2 g/adult/day (Schlotke and Sieber 1998) the respective contribution of dairy products to the daily intake of PCDD/F is 0.4 pg WHO-TEQ/kg bw in Switzerland. This estimate which includes only the PCDD/F exploits 40% of the lower end of the range of the tolerable daily intake of PCDD/F and dioxin-like PCBs defined by WHO (1–4 pg WHO-TEQ/kg bw) (WHO 1998).

It also published that PCB levels have been significantly correlated with age, body mass index (BMI), male versus female gender, and the frequency of GLSCF (Great Lakes sport-caught fish) consumption (Hanrahan et al. 1999). Total dioxin, furan, and coplanar PCB TEQs have been higher in men than in women GL fish eaters (Falk et al. 1999). PCBs have been associated with decreased levels of thyroxine in men and women and decreased levels of sex-hormone-binding globulin and sex-hormone-binding globulin-bound testosterone in men (Persky et al. 2001), and maternal PCB exposure has been associated with a decreased sex ratio. Turyk et al. (2006) have found that noncoplanar PCBs were higher in GLSCF consumers than in a referent population from the same geographic area, were associated with GLSCF consumption, and varied significantly by GL. Lower chlorinated dioxin and furan TEQs, and coplanar PCB TEQs were positively associated with noncoplanar PCBs but were not associated with GLSCF consumption independent of PCB level. Highly chlorinated dioxin and furan congener TEQs were not significantly associated with noncoplanar PCBs or GLSCF consumption, suggesting that participants were acquiring some of these TEQs from a source other than GLSCF. In epidemiologic studies, it may be important to include populations with high and low organochlorine levels and to consider the effects of individual congeners or groups of congeners on health outcomes. Also the findings of of other authors' studies (Falk et al. 1999) indicate that fish consumption varied with the gender among the Lake Huron subgroup. Body burden levels of dioxin, furan, and coplanar PCB total TEQs varied with the gender and lake subgroup as well. Serum levels of total dioxin TEQ also varied by lake; the Lake Huron subgroup had a significantly higher median level than the Lake Michigan subgroup. These preliminary data also demonstrated that consumption of lake trout and salmon significantly predicted serum log (total coplanar PCB) levels. In addition, lake trout consumption significantly predicted log (total furan) levels. GL sport fish consumption was not significantly correlated with total dioxin levels.

Studies of Beck et al. (1989b) and Fürst et al. (1990) indicated that dioxin levels of fish or shellfish were higher than for the other food groups, and generally, the Japanese tend to consume large amounts of fish and shellfish compared with Westerners. In the report of Toyoda (1999), the dietary daily intake of PCDDs, PCDFs, and Co-PCBs as TEQs from fish and shellfish in Japan accounted for 62.4% of the total intake. It is probable that the high intake of fish and shellfish is deeply involved in the accumulation of dioxin among the Japanese (Takekuma et al. 2004).

The levels of PCDDs/PCDFs determined in the nine butter samples were very low. The findings ranged between 0.27 and 0.65 pg I-TEQ/g fat butter (with an average and median values of 0.47 and 0.46 pg I-TEQ/g fat, respectively). The major contribution to the total I-

TEQ were 2,3,4,7,8-PeCDF (38%) followed by 2,3,7,8-TCDD and 1,2,3,7,8-PeCDD with approx.15% each one. Similarly as milk samples, the I-TEQ values increased from 0.72 to 1.54 pg/g when co-PCBs are considered (average and median values of 1.05 and 0.97 pg I-TEQ/g fat). The values expressed in pg WHO-TEQ varied from 0.32 to 0.73 pg/g fat (average of 0.54 and a median of 0.53 pg/g fat) and between 0.76 and 1.63 pg/g fat when co-PCBs were included (average and median values of 1.12 and 1.06 pg/g fat). These results were consistent with the data reported by Fiedler et al. (1997a) or Defour et al. (1997) despite the fact that the values were slightly lower.

The patterns of dioxins and dioxin-like chemicals reflect their sources. To a specialist the measured dioxin congener patterns in blood or other tissues can be as informative as an electrocardiogram to a cardiologist. Table 9 shows patterns in patients from different dioxin exposures. The first is an American with massive PCP exposure (Ryan et al. 1987). Primarily higher chlorinated (with 5–8 chlorines) dioxins and PCDFs are noted compared to the background level of the general American population (Schecter et al. 1990). The second shows blood from an Agent Orange-exposed Vietnamese with marked elevation of TCDD, the characteristic dioxin of Agent Orange (Schecter et al. 2001b). The third shows blood from a Japanese municipal solid waste incinerator worker and primarily demonstrates elevated PCDFs compared to the general Japanese population (Schecter et al. 1999). While the congener patterns differ, the total dioxin TEQ is elevated in all three of these cases.

Table 9 Comparison of human tissue levels and toxic equivalents of dioxins and dibenzofurans from different exposures (after Schecter et al. 2006)

Level (pg/g or ppt, lipid)	Fat (USA)		Blood (Vietnam)		Blood (Japan)	
	General population[a]	PCP-exposed person[b]	Pooled Vietnamese blood[c]	Agent Orange exposed[c]	General population[d]	Incinerator worker[d]
2,3,7,8-Tetra-CDD	3.6	33	2.2	101	2.6	6.4
1,2,3,7,8-Penta-CDD	6.6	70	3.5	6.1	8.6	60
1,2,3,4,7,8-Hexa-CDD	8	698	3.5	6.4	0.4	7.7
1,2,3,6,7,8-Hexa-CDD			7.7	16.5	0.4	14.5
1,2,3,7,8,9-Hexa-CDD	61.2	346	2.4	5.4	0.9	10.6
1,2,3,4,6,7,8-Hepta-CDD	NA	15,260	15.4	37	0.4	3.1
OCDD	794	128,913	114	212	0.1	0.1
2,3,7,8-Tetra-CDF	1.3	ND (4.3)	1	0.9	0.6	0.2
1,2,3,7,8-Penta-CDF	NA	NA	0.5	0.5	0.2	0.7
2,3,4,7,8-Penta-CDF	5.6	50	6.8	3.1	7.3	122
1,2,3,4,7,8-Hexa-CDF	6.4	174	10.1	7.8	1.1	27.8
1,2,3,6,7,8-Hexa-CDF	5		7.8	4	0.8	51
1,2,3,7,8,9-Hexa-CDF	NA	NA	0.5	0.5	0.1	34.4
2,3,4,6,7,8-Hexa-CDF	1.4	37	2.1	1.5	0.4	5
1,2,3,4,6,7,8-Hepta-CDF	95	6021	8.6	10.4	0.1	15.4
1,2,3,4,7,8,9-Hepta-CDF	NA	787	0.8	0.9	0	1.1
OCDF	NA	15,348	2.5	2.5	0	0
TEQ (pg/g or ppt, lipid)						
2,3,7,8-TCDD	3.6	33	2.2	101	26	6.4
PCDD	14	374	5	7	11	96
PCDF	5.2	202	5.8	3	11	1,365
Total TEQ	22.8	609	13	111	24.6	1,467

ND not detected, with detection limit; *NA* not analyzed; *PCP* pentachlorophenol

[a] Schecter et al. (1990)

[b] Ryan et al. (1987)

[c] Schecter et al. (2001a)

[d] Schecter et al. (1999)

Water

The US EPA has set the allowable concentration of 2,3,7,8-tetraCDD in drinking water from 0.13 to as low as 0.0013 pg/L based on estimated human cancer risks (tumor incidence risk: 0.13 pg/L for 10^{-5}, 0.0013 pg/L for 10^{-7}), respectively (US EPA 1984). The maximum contaminant level (MCL), based on the tolerable daily intake (TDI) of 10 pg TEQ/kg/body weight/day, as well as the maximum contaminant level goal (MCLG), have been set at 30 pg TEQ/L and 0 pg TEQ/L, respectively (US EPA 2001).

PCDD/Fs, and co-PCBs' analyses in raw and treated water throughout Japan were implemented to identify the concentration and homologue patterns of dioxins before and after the water treatment process (Kim et al. 2002). In 40 surface water and 5 ground water treatment plants, the dioxin-removing efficiency and the extent of influence chlorination has on dioxins' increase in drinking water were also studied. Raw water and treated water were sampled twice—during summer and winter. The mean concentration in raw water and treated water of dioxins was 56.45 pg/L (0.15 pg WHO-TEQ/L) and 4.24 pg/L (0.019 pg WHO-TEQ/L), respectively. Location of water treatment plants not only significantly influenced the concentration level of dioxins but also resulted in different homologue patterns of dioxins. Levels of dioxins in ground water were much less than that of surface water in both raw and treated water. This study showed that most dioxin congeners were well removed (87% removal efficiency) by water treatment. However, in some water treatment plants, the level of TeCDFs (pg WHO-TEQ/L) increased as a result of chlorination. This result is in agreement with that of a previous result and most of dioxins and dioxin-like compounds can be removed by drinking water treatment such as coagulation, sedimentation and filtration (Smirnov et al. 1996). Congener distributions of PCDD/Fs and co-PCBs for raw water are shown in Table 10. As expected, concentration in ground water, compared to total average concentration, was low, 3.48 pg/L (6.2% of total dioxins), whereas the concentration in surface water was much higher, 63.07 pg/L. The average dioxin concentration in ground water is about four times lower than that of the 25 sampling sites reported in 1999 (Tokuda 1999). The average concentration in surface water was lower than that in Germany and England (Götz et al. 1994).

Table 10 Congener distribution of PCDD/Fs and co-PCBs in raw water (after Kim et al. 2002)

Congener	Surface water[a] (pg/L)	Ground water[a] (pg/L)	Total average[b] (pg/L)	Percentage[c](%)	Total average[b] (pg-TEQ/L)	Percentage[c](%)
TeCDDs	10.97	0.66	9.83	17.41	0.0083	5.63
PeCDDs	1.81	0.14	1.62	2.87	0.0294	19.94
HxCDDs	1.20	0.05	1.07	1.90	0.0201	13.64
HpCDDs	3.64	0.03	3.24	5.74	0.0153	10.38
OCDD	26.71	0.11	23.75	42.07	0.0024	1.63
Total PCDD[d]	44.23	0.99	39.51	69.99	0.0755	51.22
TeCDFs	1.23	0.13	1.10	1.95	0.0090	6.11
PeCDFs	0.77	0.07	0.69	1.22	0.0240	16.28
HxCDFs	0.86	0.04	0.77	1.36	0.0246	16.69
HpCDFs	0.99	0.01	0.88	1.56	0.0048	3.25
OCDF	0.88	ND	0.79	1.40	0.0001	0.07
Total PCDF[d]	4.73	0.25	4.23	7.49	0.0625	42.40
Non-*ortho* PCBs	1.04	0.10	0.93	1.65	0.0078	5.29
Mono-*ortho* PCBs	12.98	2.15	11.78	20.87	0.0016	1.09
Total Co PCB[d]	14.02	2.25	12.71	22.52	0.0094	6.38
Total dioxins[e]	63.07	3.49	56.45	100.00	0.1474	100.00

[a] Surface water (pg/L) (resp. ground water (pg/L) are the average dioxin concentrations at 40 surface water plants (resp. five ground water plants)

[b] Total average (pg/L) (resp. total average (pg-TEQ/L) are the average dioxin concentration at 45 water plants

[c] Percentage (%) means the ratio of homologues to total dioxins

[d] Total PCDDs (resp. total PCDFs, resp. total Co-PCBs) are the sum of tetra to octra CDD (resp. sum of tetra to octa CDF and resp. sum of non-ortho PCBs and mono-ortho PCBs)

[e] Total dioxins are the sum of total PCDDs, total PCDFs and total Co-PCBs

Air

It should be noted that monitoring of dioxins plays an important role in public and sanitary decisions. In particular, the presence and trend of these pollutants in the atmosphere has been the subject of many environmental studies performed all over the world (Abad et al. 2004). For instance, Fiedler et al. (2000) reported compiled data from Germany in 1993. The levels in rural areas ranged from 25 to 70 fg I-TEQ/m^3, whereas those in urban areas varied between 70 and 350 fg I-TEQ/m^3, and levels close to source oscillated between 350 and 1,600 fg I-TEQ/m^3. Previously, concentrations over 1,068 ambient air samples from some sites were characterized in several cases by higher concentrations and larger ranges. Stenhouse et al. (1998) reported PCDD/PCDF levels in ambient air in Slovakia collected from 15 sampling locations with maximum levels, expressed in geometrical means, between 40 and 130 fg I-TEQ/m^3 (n = 113). Bolt and de Jong (1993) reported levels of PCDD/Fs from The Netherlands. Background levels between 10 and 15 fg I-TEQ/m^3 were determined, whereas levels in air around a municipal waste incinerator ranged from 15 ± 5 to 125 ± 25 fg I-TEQ/m^3 in the deposition area. The US EPA reported the results after 2 years of the implementation of the National Dioxin Air Monitoring Network (NDAMN). Values in samples collected in rural areas and national parks were not higher than 25 fg WHO98-TEQDF/m^3 (Cleverly et al. 2000, 2001). Sin et al. (2002) reported the results of 27 samples collected in six locations in Hong Kong. Levels of PCDDs/PCDFs ranging from 30 to 430 fg I-TEQ/m^3 were determined in winter, whereas concentrations from 18 to 25 fg I-TEQ/m^3 were calculated in summertime, which also reflects the potential influence of the season parameters affecting the dioxin assessment in the ambient air.

Abad et al. (2004) reported the results of an assessment of dioxin levels in ambient air in samples collected in the four provinces of Catalonia (Spain). The study includes compiled data of more than 133 samples collected in 28 different sites (rural, urban, suburban and industrial) between 1994 and 2002. The levels revealed a variable content of PCDDs/PCDFs depending both on the area and the contamination source. Thus, concentrations from 16 to 954 fg I-TEQ/Nm^3, with a mean value of 180 fg I-TEQ/Nm^3, were determined in industrial areas. The levels found in urban and suburban sites varied from 10 to 357 fg I-TEQ/Nm^3, with a mean value of 80 fg I-TEQ/Nm^3. The lowest concentrations were found in rural areas, ranging from 5 to 125 fg I-TEQ/Nm^3, with a mean value of 42 fg I-TEQ/Nm^3. These results were comparable to those reported in other works (Fiedler et al. 2000; Bolt and de Jong 1993; Cleverly et al. 2001).

As part of the project, levels of samples collected in parallel using two different samplers, a total suspended particulate (TSP) sampler and PM_{10} sampler, were compared. The results of 11 different campaigns indicated that both methods are comparable and no significant differences were determined (Table 11) (Abad et al. 2004).

Chang et al. (2004) measured PCDD/F concentrations in tunnel air and vehicle exhaust. The results indicate that the tunnel air had a PCDD/F TEQ concentration of about two times as high as that of outside air (47.3 and 57.1 fg-I-TEQ/m^3 for tunnel air vs. 37.1 fg-I-TEQ/m^3 and 23.3 fg-I-TEQ/m^3 for outside air, respectively). This provides the direct evidence that PCDD/F compounds are emitted from the combustion processes in gasoline- and diesel-fueled engines. According to the tunnel study, the emission factors ranged from 5.83 to 59.2 pg I-TEQ/km for gasoline vehicles and 23.32 to 236.65 pg I-TEQ/km of diesel vehicles. This indicates that the dioxin emission factor in Taiwan is lower than that measured in USA, Norway and Germany (Table 12). When the speed of the diesel vehicle was set at 40 kmph, the dioxin concentration emitted from diesel vehicle was 278 pg/m^3 (6.27 pg-I-TEQ/m^3) from tailpipe testing. However, when the diesel vehicle was idled, the dioxin concentration increased greatly to 4,078 pg/m^3 (41.9 pg-I-TEQ/m^3). From the results of tunnel air sampling, the PCDDyFs emission from automobiles in Taiwan was estimated as 3.69 g I-TEQ per year.

Table 11 Comparison of individual 2,3,7,8-PCDDs/PCDFs determined by TSP and PM_{10} samplers (Abad et al. 2004)

Compounds	Concentration (fg/Nm^3) TSP sampler	Concentration (fg/Nm^3) PM 10 sampler
2,3,7,8-TDCF	54.70	47.44
1,2,3,7,8-PeCDF	11.02	9.07
2,3,4,7,8-PeCDF	22.43	20.59
1,2,3,4,7,8-HxCDF	54.56	56.41
1,2,3,6,7,8-HxCDF	23.81	21.92
2,3,4,6,7,8-HxCDF	30.39	30.49
1,2,3,7,8,9-HxCDF	1.37	1.31
1,2,3,4,6,7,8-HpCDF	123.19	115.45
1,2,3,4,7,8,9-HpCDF	12.80	13.94
OCDF	118.94	105.77
2,3,7,8-TCDD	2.33	1.89
1,2,3,7,8-PeCDD	6.52	6.52
1,2,3,4,7,8-HxCDD	6.97	6.08
1,2,3,6,7,8-HxCDD	14.24	17.67
1,2,3,7,8,9-HxCDD	24.65	21.99
1,2,3,4,6,7,8-HpCDD	201.66	177.97
OCDD	645.36	492.29

Table 12 Dioxin emission factors from vehicles in different countries (after Chang et al. 2004)

Study and year	Country	Sampling	Vehicles	EF1 (pg I-TEQ/km)	EF2 (pg I-TEQ/L)
CARB (1987)	USA	Tailpipe	Leaded	203	1,794
			Diesel	380	5,904
			Diesel	4,900	27,440
Marklund et al. (1987)	Sweden	Tailpipe	Unleaded	<13	–
			Leaded	20–220	–
Bingham et al. (1989)	New Zealand	Tailpipe	Leaded	15–39	–
Marklund et al. (1990)	Sweden	Tailpipe	Unleaded	0.36	–
Hagenmaier et al. (1990)	Germany	Tailpipe	Unleaded	5.1	50.7
			Unleaded	0.7	7.2
			Leaded	108.3	1,083
			Diesel	2.4	23.6
			Diesel	35	70
Oehme et al. (1991)	Norway	Tunnel	Leaded/unleaded	28–520	–
			Diesel	720–9,500	–
Wevers et al. (1992)	Belgium	Tunnel	Leaded	1641	–
			Unleaded	10	–
			Diesel	35.7	–
Hagenmaier et al. (1995)	Germany	Tailpipe	Diesel	–	10
Buhler and Greiner (1996)	Germany	Tailpipe	Diesel	14	77
Gertler et al. (1998)	USA	Tunnel	Diesel	29	–
Ryan and Gullet (2000)	USA	Tailpipe	Diesel	29–106	–
Chang et al. (2004)	Taiwan	Tunnel	Unleaded	22.9	229.3
			Diesel	91.7	550.4

Soil

According to Lohmann and Jones (1998), PCDD/F concentrations for the total sum of TEQ are typically as follows: remote <10 fg I-TEQ m^{-3}); rural ~20–50 fg I-TEQ m^{-3}); and urban/industrial ~100–400 fg I-TEQ m^{-3}. Concentrations measured in Lisbon are comparable to those found in rural and uncontaminated urban areas: 83% of PCDD/PCDF concentrations in this region range from 10 to 100 fg I-TEQ m^{-3}. On the other hand, in Porto 77% of samples collected are in the 40 to 400 fg I-TEQ m^{-3} range, approximately four times higher than the levels for the Lisbon region. Levels measured in Porto are consistent with data published for Barcelona, on the NE coast of the Iberian Peninsula, where the reported maximum concentrations were in the range of 600 to 800 fg I-TEQ m^{-3} (Abad et al. 2004). Several studies performed in different airsheds (Hippelein et al. 1996; Fiedler et al. 1997a) have shown that atmospheric levels of PCDD/PCDF follow a typical seasonal variation characterized by higher concentrations during winter when compared with typical summer concentrations. This seasonal pattern can be explained by the intensification of the operation of diverse combustion sources during winter as well as by the more frequent presence of thermal inversion layers at the surface level during winter. These inversions cause a significant increase of atmospheric concentrations when pollutants are emitted at low levels below the thermal inversion layer.

According to Coutinho et al. (2006) interpretation of temporal trends of atmospheric dioxin levels in the region of Porto (Portugal) shows the contribution that medical waste incinerators, without any air pollution control devices, might have in the deterioration of air quality in urban areas. Prior to the winter of 2001–2002, winter levels in Porto were approximately three to four times higher than summer levels. Afterwards, it is possible to observe a significant decrease of mean concentrations of atmospheric PCDD/F. The mean value (37 fg I-TEQ m^{-3}) and concentration range (13–42 fg I-TEQ m^{-3}) for Summer 2002 are the lowest recorded in this region, followed by Summer 2003 (mean 50 fg I-TEQ m^{-3}, range 9.8–172 fg I-TEQ m^{-3}). The decrease of atmospheric concentrations of PCDD/F was more evident during winter time: PCDD/F levels showed a reduction by a factor of 2, from average levels typically above 300 fg I-TEQ m^{-3} to values between 150 and 200 fg I-TEQ m^{-3}.

A major seasonal pollution source of atmospheric PCDD/F levels in the region may also be the household

wood burning for heating in winter. Burning woods at low temperatures, with low burning efficiency, plus burning of wood treated with chlorinated phenols (added as a preservative) woods may increase PCDD/PCDF emissions (Dyke et al. 1997).

Sediments

A potential natural formation mechanism for chlorinated organic compounds is biochemical synthesis. Living organisms are capable of synthesizing a variety of halogenated compounds (Gribble 1994). Subsequent biochemical (enzymatic) and chemical reactions may lead to the transformation of suitable precursors into more complex structures. Enzymatic and photochemical formation of PCDD/Fs from precursors has been demonstrated under laboratory conditions (Svenson et al. 1989; Vollmuth et al. 1994). Evidence for natural formation of di- to tetrachlorinated dibenzo-*p*-dioxins and dibenzofurans in forest soils has also been presented (Hoeksta et al. 2000).

Isosaari et al. (2002) reported that PCB sum concentrations in sediments (Lake Ahmasjärvi, Finland) ranged from 50 to 2,540 ng/kg dw (Table 13). They concluded that the PCB sum concentrations in sediments were in the same range as those measured in lake sediments from northern Finland (3.0–5.6 ng/kg dw) (Vartiainen et al. 1997), southern Finland (sum of 8 congeners 5–20 ng/kg dw) (Lampi et al. 1992) and Canada (2.4–39 ng/kg dw) (Muir et al. 1996). The cleanest subsamples taken from Lake Ahmasjärvi were as clean as the lake sediments from Antarctica (Fuoco et al. 1994).

In other studies, PCB concentrations in the old sediment deposits have been below detection limits (Muir et al. 1996; Vartiainen et al. 1997). The production of PCBs on an industrial scale started in 1929 (Bernes 1998), and detectable concentrations typically appear only after the 1940s. It is possible that ambient air contaminates sediment samples with lower chlorinated PCBs, especially. Therefore, more data are needed to validate the presence of PCBs in deposits that are thousands of years old. Isosaari et al. (2002) reported that PCDD/F sum concentrations at the depths of 112–400 cm were 4.91–59.2 ng/kg dw (1.99–8.44 ng/kg dw of 2378-substituted PCDD/Fs). Historical dioxin background in about 8,000-year old sediment cores from an inland sea in Japan was 52 ng/kg dw for 1234679-HpCDD and 320 ng/kg dw for OCDD (Hashimoto et al. 1990). Other clearly pre-industrial data are available on tissue samples of Eskimos who were exposed to incomplete combustion products in their lives about 400 years ago. In these tissues, the levels of the studied tetra- to hexa-CDD/Fs were below detection limits (Schecter et al. 1988).

Their results (Isosaari et al. 2002) support the theory of natural formation of PCDD/Fs. However, it must be noted that the originally formed PCDD/F profile might have become transformed on the way to the sink or in the sink, resulting in a previously addressed discrepancy between PCDD/F homologue patterns of sources and sinks (Duarte-Davidson et al. 1997; Wagrowski and Hites 2000). The mechanisms of wet and dry deposition of PCDD/Fs and decomposition of the lower chlorinated congeners in the atmosphere contribute to a selective enrichment of OCDD in soils and sediments (Koester and Hites 1992). Overall concentrations may also increase. Hypotheses have been proposed to show how PCDD/Fs would accumulate in soil if there were no losses (Duarte-Davidson et al. 1997). Local and temporal changes in PCDD/F accumulation rate,

Table 13 A summary of PCDD/F and PCB sum concentrations (ng/kg dw), (Isosaari et al. (2002)

Depth (cm)	Age[a] (a)	Sum of PCBs[b]	Sum of PCDD/Fs	Sum of 2,3,7,8-PCDD/Fs	WHO-TEQ[c]
0–11	0–311	380	84.3	35.0	1.18
11–23	311–622	554	40.7	6.63	0.56
23–34	622–932	2,530	32.7	11.1	1.70
68–79	1,864–2,175	674	28.6	4.02	0.80
112–124	3,107–3,418	630	19.4	4.07	0.38
124–135	3,418–3,728	750	32.8	8.44	1.62
146–157	4,039–4,350	1,090	59.2	7.09	0.88
227–239	6,214–6,525	1,030	11.8	4.91	1.00
310–321	7,930–7,991	32.0	4.91	2.60	0.41
390–400	8,370–8,425	150	5.72	1.99	0.23

[a] Calibrated age in years before 1999

[b] Sum of 25 mainly di-*ortho* PCB congeners (IUPAC 18, 28, 33, 47, 49, 51, 52, 60, 66, 74, 99, 101, 110, 122, 128, 138, 141, 153, 170, 180, 183, 187, 194, 206, 209), 8 mono-*ortho* congeners (IUPAC 105, 114, 118, 123, 156, 167, 189), and 3 non-*ortho* congeners (IUPAC 77, 126, 169)

[c] WHO-TEQ based on toxicity equivalent factors (TEFs) for PCDD/Fs

and the resulting concentrations in sediments, could be associated with the extent of physical and biological turbulence (for a review, see Fletcher and McKay 1993). Thus, the usefulness of a sediment core as evidence regarding which congeners have been formed and to what extent, and when, is only of a descriptive nature.

Occupational exposure

Some human subpopulations are at risk of either continuous or intermittent exposure to relatively high levels of PCDFs and PCDDs, which may result in adverse health effects. Workers engaged in the production, use, or destruction of materials containing these chemicals or their precursors may be subject to such risks (Beck et al. 1989a; Päpke et al. 1992). Dermal and inhalation are exposure routes particularly relevant to production and usage of agricultural and industrial products. In the recent past, discrete exposures to high levels of these compounds have occurred through industrial accidents (e.g., Seveso, Italy in 1976) and improper disposal of industrial waste (e.g., Times Beach, Missouri, 1982). Subsistence and recreational fishermen (Svensson et al. 1991) may have rich dietary exposure to these compounds, due to their bioconcentration in fish; and subsistent farmers living near point sources of contamination, such as incinerators, may experience similar risks of dietary exposure (Wevers et al. 1993). Airborne fly ash particles emitted from incineration combustion are of respiratory size (Stevens and Swackhamer 1989; Fiedler 1996) and could deposit in the airways of the lung. Airborne dust particulates and volatile TCDD are also a concern for pulmonary absorption (Goldfarb and Harrad 1991). The major contaminant-adsorbing surfaces of soil consist of organic matter and clay and these are predominant in the respirable fraction of soil (Morrill et al. 1982). Even though relatively low ambient levels of PCDDs and PCDFs are found in air, soil, and sediment, these compounds because of their stable and persistent lipophilic nature bioconcentrate in the food chain. Most of the apparent exposure of the general human population to PCDDs and PCDFs occurs through dietary consumption (Winters et al. 1994), with daily human exposure to TCDD through ingestion estimated to be 0.1–0.3 pg TCDD/kg/day (Fürst et al. 1991).

For example, in Table 14 the emission factors calculated from the measurements are presented. The highest emission factor value is calculated for plants: 7, 9 and 12 (foundries of cast iron, secondary aluminium production—aluminium scrap melting—electric furnace and secondary aluminium production —aluminium scrap and cans melting). Emission factors obtained for iron ore sintering

Table 14 Emission factors estimated during the measurement program (Grochowalski et al. 2006)

Type of activity	Emission factors µg I-TEQ/mg of product	Emission factors µg I-TEQ/mg of product (Inventory of Dioxin and Furan Releases in Poland, Report 2002)
Iron and steel metallurgy plants		
Iron ore sintering plant	1.47, 1.10	5
Primary Iron production-Blast Furnace (BF)	0.01	0.03–0.13
Primary and secondary steel production-Basic Oxygen Furnace (BOF)	0.020	0.5–10
Iron casting-hot air cupola (good APCs)[a]	0.06, 4.11	0.03–10
Iron casting-gas rotary kiln	0.02	4.3
Secondary steel production, steel scrap melting-electric arc furnaces	0.62, 0.02[b]	5
Steel casting-electric arc furnace	0.03	1.0
Non-ferrous metallurgy plants		
Primary copper production from concentrate with H_2SO_4 production; copper slag recycling	0.005, 0.004, 0.002	0.01
Copper scrap melting	0.007	50
Secondary aluminium production, aluminium scrap and cans melting	8.65, 3.05, 1.69, 0.34	150
Primary zinc production	0.12	0.15–2.4
Zinc casting	0.02	Not data available

[a] Air pollution control systems

[b] Only secondary off gas was measured in the plants

indicate that the previous data (5 μg I-TEQ/t) have been overestimated.

It also has been shown for primary iron (Blast Furnace), as well as for primary and secondary steel production. That indicates that secondary aluminium production is the most significant dioxin source if calculated as an emission factor value; however, iron ore sintering plants are operated in much higher product mass efficiency and hence this process is the major source to release dioxins, PCB and HCB pollution to the environment. The level of dioxin emission from secondary aluminium production depends on the raw material composition and the applied APCS (Iron casting-hot air cupola). The default emission factor proposed to apply previously was 150 μg I-TEQ/t of aluminium with an uncertainty range of 50–450 μg I-TEQ/t. The Standardized Toolkit (UNEP 2003) does not provide any default emission factors for aluminium casting. As pure ingots are used for the production, the emission factors are estimated to be considerably lower than the electric furnace (EF) for secondary aluminium production. The toolkit applies an EF of 1 μg I-TEQ/t for zinc and zinc casting in furnaces without air pollution control systems (APCS) and 0.3 for casting of zinc alloys. The data obtained from the European Dioxin Inventory (Quaß 1997) applies emission factors for secondary zinc in the range of 0.15–2.4 μg I-TEQ/t.

Conclusion

In numerous investigations it was shown that food is the main route of non-occupational human exposure to polychlorinated dibenzo-*p*-dioxins (PCDDs) and PCDFs. It is also known that animal products like meat, fish, milk and milk products are largely contributing to the human burden as PCDD/Fs are bioaccumulated because of their lipophilicity and their low biodegradability. Also, PCDDs and PCDFs can be formed as unwanted by-products by many anthropogenic processes and their presence in the atmosphere stems from several industrial activities which include, for instance, a variety of thermal processes such as waste management plants, cement kiln plants, sintering plants and other diffuse sources Furthermore, once released into the atmosphere, these toxicants can be transported far away from their original sources, and as a result, their presence can be determined in remote areas. In this sense, great efforts to increase the knowledge about these pollutants have been taken and stringent regulations aiming to protect public health have already been established.

Thus, monitoring of dioxins plays an important role in public and sanitary decisions. In particular, the presence and trend of these pollutants in the atmosphere, food, soil and human samples have been the subject of many environmental studies performed all over the world (for a review, see Parzefall 2002).

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