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Presence of PCDDs and PCDFs in Food for Human Consumption: A Review

B. Jiménez, M. J. González & L. M. Hernández

Instituto de Química Orgánica General (CSIC),
C/ Juan de la Cierva 3, 28006, Madrid, Spain

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ABSTRACT

*Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in human fat and milk samples arise from a variety of sources. It is generally assumed that food intake is mainly responsible for the presence of these contaminants in man. Data on levels in food samples are, however, relatively rare. This review reports the levels found in various foodstuffs which are easily obtained in daily life. From the data, it is clear that most animal foodstuffs have already been contaminated by these contaminants, even though in very small amounts. However, the plant products are free of most PCDDs and PCDFs. It is difficult to evaluate the different sources of contamination of food, but municipal solid waste incinerators and chlorophenol industries are likely to be the most important sources.*

INTRODUCTION

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are two series of tricyclic, almost planar, aromatic compounds that exhibit similar physical, chemical and biological properties. These contaminants are important environmental chemicals among which 2,3,7,8-TCDD (2,3,7,8-tetrachlorodibenzo-*p*-dioxin) has been the focus of many studies because of its extraordinary toxicity to animals (Reggiani, 1982).

Human contamination by PCDDs and PCDFs may occur occupationally, accidentally, from dietary intake, or from environmental exposure to

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air, water or soil (Rappe *et al.*, 1982). The persistent and bioaccumulative properties of these chemicals suggest that these pollutants may enter into the food chain and contaminate the human diet.

Recent reports (Ryan *et al.*, 1985*a,b*; Graham *et al.*, 1985; Rappe *et al.*, 1987) on 2,3,7,8-TCDD and a variety of other PCDDs and PCDFs in adipose tissue and human milk from Canadian, European and US residents indicate widespread low level exposure to these chemicals. Data reported in Table 1 substantiates this observation. All the isomers found in these samples belong to the twelve toxic 2,3,7,8-substituted Cl_4, Cl_5 and Cl_6 congeners.

PCDDs and PCDFs can enter into the environment and subsequently into the diet in a variety of ways, e.g., as contaminants in the production and use of chlorophenol-based chemical products, as products of combustion and as components of solid, liquid and airborne wastes and emissions (Rappe *et al.*, 1982; Liberti & Brocco, 1982).

Previous investigations (Rappe *et al.*, 1986) indicate that it is especially important to identify the source or sources of the toxic 2,3,7,8-substituted

TABLE 1

Levels of PCDDs and PCDFs in Human Samples in ppt (Rappe *et al.*, 1986)

	Adipose N. Sweden n = *31*		*Milk N. Sweden* n = *4*		*Milk Germany* n = *5*	
	Mean	*Range*	*Mean*	*Range*	*Mean*	*Range*
2378-TCDD	3	0–9	0·6	T–2·3	1·9	1·3–3·3
12378-PeCDD	10	3–24	6·5	3·5–13·8	12·6	9–18
123478-HxCDD	ND		2·5	0·8–3·6		
123678-HxCDD	15	3–55	19	12–23	23·4	15–28
123789-HxCDD	4	3–5	6·3	3·9–9·0		
1234678-HpCDD	97	12–380	59·5	38–86	72·8	48–89
OCDD	414	90–763	302	197–484	434	168–623
2378-TCDF	3·9	0·3–11	4·2	2·2–8·7	5·4	4·0–8·0
23478-PeCDF	54	9–89	21·3	7–53	36·4	24–54
123478-HxCDF	6	1–15	4·7	2·7–8·9		
123678-HxCDE	5	1–13	3·4	1·9–6·8	26	13–36
234678-HxCDF	2	1–7	1·4	0·8–2·6		
1234678-HpCDF	11	1–49	7·4	4·4–12·0	9·2	4–12
OCDF	4		3·2	ND–11·0	2·4	1–4

TCDD, tetrachlorodibenzo-*p*-dioxin;
HxCDD, hexachlorodibenzo-*p*-dioxin;
OCDD, octachlorodibenzo-*p*-dioxin;
PeCDF, pentachlorodibenzo-furano;
HpCDF, heptachlorodibenzo-furan;
PeCDD, pentachlorodibenzo-*p*-dioxin;
HpCDD, heptachlorodibenzo-*p*-dioxin;
TCDF, tetrachlorodibenzo-furan;
HxCDF, hexachlorodibenzo-furan;
OCDF, octachlorodibenzo-furan.

PCDDs and PCDFs found in biological samples. Analyses of fish and dairy products strongly indicate that the major exposure is via food (Nygren *et al.*, 1986*a*).

The purpose of current work is to establish the levels of PCDDs and PCDFs in different foodstuffs and to identify the major sources which contaminate human diet.

For this study, we will consider four different food groups: fish, meat, vegetables and dairy products.

Fish

PCDDs and PCDFs are known to occur in aquatic systems and accumulate in aquatic organisms so that fish and shellfish might be a significant dietary source for these compounds in humans.

Reports from Canada demonstrate the presence of 2,3,7,8-TCDD in Great Lakes' fish. These reports coincide with others which establish contamination of fish in several US rivers, notably the Titabawassee River in Michigan (Rossiers, 1987).

Analyses of skinless fillets from fish sampled from inland waters located near sites involved in the manufacture, use or disposal of polychlorinated

TABLE 2
Levels of PCDDs and PCDFs in Fish Samples from the Baltic Sea (pg/g) (Rappe *et al.*, 1987*a*)

	Salmon Ume River, 1985	*Salmon Ume River, 1985*	*Herring Karlskrona, 1983*	*Herring Luleå, 1983*
2,3,7,8-Tetra-CDF	29	12	5·5	3·0
2,3,7,8-Tetra-CDD	1·9	1·3	<0·3	<0·6
1,2,3,7,8-/1,2,3,4,8-Penta-CDF	6·9	3·3	1·4	0·9
2,3,4,7,8-Penta-CDF	49·0	23·0	6·8	8·8
1,2,3,7,8-Penta-CDD	8·8	4·3	1·1	4·7
1,2,3,4,7,8-/1,2,3,4,7,9-Hexa-CDF	1·1	0·7	0·4	0·3
1,2,3,6,7,8-Hexa-CDF	1·3	0·8	0·4	0·3
1,2,3,7,8,9-Hexa-CDF	ND	ND	0·4	0·2
2,3,4,6,7,8-Hexa-CDF	1·1	0·6	0·4	0·2
1,2,3,4,7,8-Hexa-CDD	ND	0·4	0·2	ND
1,2,3,6,7,8-Hexa-CDD	4·6	2·3	1·3	8·1
1,2,3,7,8,9-Hexa-CDD	ND	ND	ND	ND
Total Hepta-CDFs	ND	2·7	0·8	ND
Total Hepta-CDDs	ND	ND	ND	ND
OCDF	ND	1·0	ND	ND
OCDD	ND	ND	ND	ND

ND < 0·1 pg/g.

phenols have revealed TCDD concentrations ranging from 1 to 40 ppt (pg/g) (O'Keefe *et al.*, 1983; Ryan *et al.*, 1984).

An interesting observation is that in the majority of the aquatic samples, only the toxic 2,3,7,8-substituted congeners are found (Rappe *et al*, 1987*a*). In their studies, in which 2,3,7,8-TCDF (2,3,7,8-tetrachlorodibenzo-furan) concentrations in aquatic sediments and fish living in the area of sediments were compared, Stalling (1983) detected small amounts of 2,3,7,8-TCDF in sediment but larger amounts in fish. On the other hand, very little OCDF (octachlorodibenzo-furan) was found in fish, while larger amounts were in the sediment. From the Table 2 (Rappe *et al.*, 1987*a*) this observation is clear. It can be observed that the TCDDs/Fs (Tetrachlorodibenzo-*p*-dioxins and tetrachlorodibenzo-furans) ranging from 1·9 to 29 ppt and PeCDDs/Fs (Pentachlorodibenzo-*p*-dioxins and Pentachlorodibenzo-furans) ranging from 3·3 to 49 ppt exhibit the highest values, while the highest chlorinated types exhibit the lowest values (from ND to 1 ppt). However, Table 3 (Ogaki *et al.*, 1987), shows that crustaceans seem to be an exception to this general behaviour. In shellfish, TCDDs are the major components but all congeners can be found.

More recent data (Niimi & Oliver, 1989) reveal levels ranging from 46 to 290 ng/kg in whole fish and 60 to 366 ng/kg in muscle composite samples from Lake Ontario. Total furan levels are about 25% lower than total dioxin concentrations.

A study in yellow perch and similar studies in rainbow trout (Kleeman *et al.*, 1986*a*,*b*) demonstrates the greater persistence of TCDD in fish than in mammalian species. A possible reason for this difference is that fish metabolize TCDD more slowly than mammals. Therefore, high concentrations in fish cause high exposure concentration of environmental chemicals for fish predator animals and man (Bruggeman *et al.*, 1981).

TABLE 3
Levels of PCDDs in Shellfish in Japan (Ogaki *et al.*, 1987)

Foodstuffs	*TCDD (ppt)*	*PenCDD (ppt)*	*HexCDD (ppt)*	*HepCDD (ppt)*	*OCDD (ppt)*	*Total (ppt)*
Shellfish						
short-necked clam	11	ND	8·3	6·7	5·8	32
short-necked clam	19	ND	1·7	7·1	2·3	28
short-necked clam	53	3·2	ND	8·4	1·5	66
corbicula	360	33	4·6	17	30	450
oyster	39	7·3	4·9	5·3	4·8	61
oyster	42	6·8	5·3	4·3	2·9	61

The values are expressed as a whole base.
ND = not detectable (< 1 ppt).

TABLE 4
Exposure of TCDD Equivalents (Eadon, 1983) for a 55-kg Person or a 5-kg Baby (Rappe *et al.*, 1986, 1987*a*)

Exposure	*pg kg^{-1} Body weight day^{-1}*
Inhalation	0·02
Milk (1 litre day^{-1})	0·5–5
Salmon (100 g week^{-1})	20
Breast milk (850 ml day^{-1})	20–200

In a study where Rappe *et al.* (1987*b*) assessed the exposure via food, it is clear (see Table 4) that exposure by inhalation is marginal compared to exposure via food, especially fish and other foodstuffs from the aquatic environment.

If we compare data from fish with other samples of animal origin (Table 5), we see that fish samples exhibit the highest levels (e.g. the TCDD-EQTS (EPA) values for herring are 21·3 and 39·7 for cod), so it can be concluded that fish is the most important source of dietary intake in man.

Meat

Beck *et al.* (1987*a*) recently showed that food containing animal fat is an important contributory factor (see Table 5). Most available data are for chicken, pork and beef.

Because of the known association of PCDDs and PCDFs with PCPs (pentachlorophenols) and the possibility that food-producing animals are exposed to these compounds through treated wood, Ryan *et al.* (1985*c*) decided to investigate the degree and scope of contamination in food samples from across Canada. Their data are the first to document the presence of these chemicals in poultry and pork samples taken from the general food supply. Here, PCDDs and PCDFs values averaging between 25 and 100 ppt of higher chlorinated congeners were found in about half the 144 samples analyzed with an isomeric pattern similar to that found in PCP. From this information it is believed that the source of PCDDs and PCDFs in the chicken and pork samples is PCP. Table 6 lists the isomeric composition of PCDDs and PCDFs in samples of pork and chicken. It is clear that chicken tissues contain more isomers of PCDDs and PCDFs than pork tissues.

This observation is also clear from Table 5: chicken values are higher than pork for PCDDs and PCDFs. Data for chicken and pork seem to show the tendency that PeCDDs are the lowest while HxCDDs (Hexachlorodibenzo-*p*-dioxins) are the highest.

TABLE 5
PCDD and PCDF Concentrations in Samples of Animal Origin (ppt, Fat Weight Basis) (Beck *et al.*, 1987*a*)

Congener[a]	*Cow's milk*	*Butter*	*Pork*	*Cattle*	*Sheep*	*Chicken*	*Eggs*	*Herring*	*Cod*	*Redfish*
2,3,7,8-TCDF	0·7	0·15	0·11	0·3	0·6	2·1	1·1	57	98	78
2,3,7,8-TCDD	0·2	0·08	0·03	0·6	0·01	0·3	0·2	4·7	23	2·8
1,2,3,7,8-PeCDF	0·2	0·09	0·01	0·01	0·01	0·01	0·6	16	48	31
2,3,4,7,8-PeCDF	1·4	0·45	0·08	1·5	0·9	1·5	0·8	29	3·1	25
1,2,3,7,8-PeCDD	0·7	0·41	0·12	0·8	0·5	0·7	0·4	12	1·3	6·5
1,2,3,4,7,8-HxCDF	0·9	0·43	0·15	0·8	0·9	0·6	0·4	3·0	6·9	3·5
1,2,3,6,7,8-HxCDF	0·8	0·44	0·07	0·6	1·2	0·4	0·3	4·2	13	6·0
2,3,4,6,7,8-HxCDF	0·7	0·31	0·05	1·3	1·5	0·3	1·7	3·6	8·2	7·2
1,2,3,4,7,8-HxCDD	0·3	0·15	0·21	0·6	0·3	0·5	1·3	1·2	0·01	0·5
1,2,3,6,7,8-HxCDD	1·1	0·95	0·29	1·9	1·5	2·8	1·4	5·8	17	8·4
1,2,3,7,8,9-HxCDD	0·4	0·26	0·06	0·6	0·4	0·6	0·5	1·0	5·2	1·3
1,2,3,4,6,7,8-HpCDF	0·5	0·34	1·1	2·2	8·1	0·8	0·6	1·6	10	1·5
1,2,3,4,6,7,8-HpCDD	2	1·5	2·1	18	15	6·0	0·4	3·6	10	3·0
OCDF	1	0·25	0·41	0·2	0·3	0·6	0·2	1·4	2·1	0·3
OCDD	10	3·4	19	25	68	52	12	19	83	11
TCDD-Eqts (FHO)	0·92	0·47	0·20	1·66	1·05	1·37	1·07	18·1	43·4	19·6
TCDD-Eqts (EPA)	0·86	0·43	0·14	1·31	0·52	1·16	0·80	21·3	39·7	20·0

[a] See Table 1 for abbreviations used.

TABLE 6
Isomeric Composition of Chlorinated Dibenzo-*p*-Dioxins (D) and Chlorinated Dibenzofurans (F) in Five Samples in ppt (Ryan *et al.*, 1985)

Isomer	*Sample no. (description)*				
	1 (*chicken fat*)	*2* (*chicken liver*[a])	*3* (*pork fat*)	*4* (*pork liver*)	*5* (*wood*[b])
1,3,4,6,7,8-HxCDF	6·2	8·3			74
1,2,4,6,8,9-HxCDF	11·0	20·6			540
1,2,3,6,7,8-HxCDF	2·9	6·6		26	
Total HxCDF	20·1	35·5	*c*	26	614
1,2,4,6,7,9-HxCDD	4·3	25·5			105
1,2,3,6,7,9-HxCDD	18·7	23·1			216
1,2,3,6,7,8-HxCDD	31·9	75·3	4·5	54	197
1,2,3,7,8,9-HxCDD	2·0	25·0			42
Total HxCDD	56·9	148·9	4·5	54	560
1,2,3,4,6,7,8-HpCDF	14·6	43·4		3 020	990
1,2,3,4,6,8,9-HpCDF	19·6	25·0			4 790
1,2,3,4,7,8,9-HpCDF				131	190
Total HpCDF	34·2	68·4		3 151	5 970
1,2,3,4,6,7,9-HpCDD	21	28	8	26	1 600
1,2,3,4,6,7,8-HpCDD	69	351	71	1 370	2 270
Total HpCDD	90	379	79	1 396	3 870
OCDF		20		5 100	3 470
OCDD	186	447	428	10 100	9 070

[a] Also contained 1,2,3,7,8-D or 1,2,3,6,7-D at 5·0 ppt and 1,2,4,6,8-F at 8·5 ppt.
[b] Values in ppb.
[c] Not detected.
See Table 1 for abbreviations used.

Nygren *et al.* (1986*b*) analyzed bovine samples and identified the same 2,3,7,8-substituted PCDDs and PCDFs as were found in the aquatic samples. However, the levels were lower and normally close to the detection limit. This indicates that the background levels are higher in the aquatic environment than in the terrestrial.

Different values for chicken, pork and beef samples are given in Table 7. From the data it is concluded that chicken exhibits the highest values, followed by pork and beef. It is also notable that chicken exhibits high values for HxCDDs in all cases.

Vegetables and fruit

In general, there is little bioaccumulation of dioxins and furans in plants, so that available data from levels in plants are scarce. According to reviews by

TABLE 7
Levels of PCDDs and PCDFs in Chicken, Pork and Beef Samples in ppt

Sample	*Ref.*[a]	∑*TCDD*	∑*TCDF*	∑*peCDD*	∑*PeCDF*	∑*HxCDD*	∑*HxCDF*	∑*HpCDD*	∑*HpCDF*	*OCDD*	*OCDF*
Chicken fat	1	—	—	—	—	56·9	20·1	90	34·2	186	—
	2	41	—	23	—	420	—	83	—	29	—
Chicken liver	1	—	—	—	—	148·9	35·5	379	68·4	447	20
	2	7·3	—	N.D.	—	25	—	7·0	—	3·5	—
Chicken	3	5·4	—	1·3	—	19·7	—	7·5	—	<4·0	—
Pork fat	1	—	—	—	—	4·5	N.D.	79	—	428	—
	2	16	—	N.D.	—	45	—	2·6	—	5·7	—
Cattle fat	4	12	—	N.D.	—	1·5	—	3·3	—	17	—
Cattle liver	4	3·8	—	N.D.	—	2·4	—	28	—	24	—
Beef	3	1·7	—	—	—	—	—	—	—	<4·5	—

[a] 1, Ryan *et al.* (1985*c*); 2, Ogaki *et al.* (1987); 3, Ono *et al.* (1987); 4, Ogaki *et al.* (1985).

Esposito *et al.* (1980) and Norris (1981), 0·15% of TCDD and D_2CDD (Dichlorodibenzo-*p*-dioxin) applied to soil was found in above-ground plant portions of oats and soybeans. Furthermore, surfactant applied to leaves was not translocated to other parts of the plant. This occurrence indicates that plant products might not have these contaminants or very low levels.

A study from Davies (1988) reveals that every analyzed fruit composite contained all the polychlorinated dibenzodioxin and dibenzofuran species, except TCDD. In contrast, the leafy vegetable composites contained mainly HpCDDs (Heptachlorodibenzo-*p*-dioxins) and OCDD and HpCDFs (Heptachlorodibenzo-furans) and OCDF. This is the only report which documents a high estimated intake of PCDDs and PCDFs from fruit and the least from the leafy vegetable.

Table 8 reveals that, within the different food groups, food that contains animal fat is an important contributory factor whereas food of plant origin is of minor importance (e.g. the values for total PCDDs are 1·6 pg/g in vegetable in contrast to the highest values for pork with 16·1 and chicken with 33·9). It can be observed that vegetables have the lowest levels. This conclusion is supported by studies published by Beck *et al.* (1987*b*).

Dairy products

Dairy products may also be an important source of exposure. Rappe *et al.* (1987*c*) have reported on levels of PCDDs and PCDFs in milk from cows that have been grazing at various distances from incinerator plants in Switzerland. All the milk samples analyzed showed the presence of PCDFs and PCDDs. The results are given in Table 9. It is interesting to note that all the identified isomers belonged to the group with chlorine in positions 2, 3, 7, and 8.

It is thus apparent that dairy products may be an important source of human exposure to this group of compounds.

Based on a mean daily consumption of about 30 g milk fat (milk, cheese, butter, curds, etc.) for adults, 6 pg of 2,3,7,8-TCDD is estimated to be a mean daily intake originating from dairy products (Beck *et al.*, 1987).

On the other hand, the levels found in mother's milk (Rappe *et al.*, 1986; 87*c*) might be of especial interest from a toxicological point of view. An animal of 5 kg body weight, consuming 850 ml of milk a day, receives a dose of 2,3,7,8-TCDD of about 5 kg/pg body weight per day, which is much higher than the virtually safe dose discussed by several authors (Nygren *et al.*, 1986*b*).

It is interesting to note the close similarity in the isomeric pattern between cow milk and human milk (Rappe *et al.*, 1987*c*).

TABLE 8
Concentrations (pg/g on wet weight basis)[a] of PCDDs and PCDFs in Foods (Ono *et al.*, 1987)

	Vegetable	*Oil*	*Rice and wheat*	*Fish*	*Beef*	*Pork*	*Chicken*	*Egg*
$^{13}C_{12}$-2,3,7,8-T_4CDD Rec. (%)	57·3	45·4	61·9	66·5	73·5	64·2	78·1	79·4
1,3,6,8-T_4CDD	0·8	<0·2	0·4	1·0	1·2	0·8	3·9	2·3
1,3,7,9-T_4CDD	0·7	<0·2	0·3	0·8	0·5	0·5	1·5	1·0
Total T_4CDD	1·5	—	0·7	1·8	1·7	1·3	5·4	3·3
1,2,3,6,8-P_5CDD	0·1	<0·1	<0·1	<0·3	<0·3	<0·3	1·3	0·9
Total P_5CDD	0·1	—	—	—	—	—	1·3	0·9
1,2,4,6,7,9-/1,2,4,6,8,9-1,2,3,4,6,8-H_6CDD	<0·1	<0·5	<0·3	<1·1	<0·8	<1·2	1·6	2·3
1,2,3,6,7,9-/1,2,3,6,8,9-H_6CDD	<0·1	<0·5	<0·3	<1·1	<0·8	<1·2	7·3	6·2
1,2,3,6,7,8-H_6CDD	<0·1	<0·5	<0·3	<1·1	<0·8	<1·2	8·1	5·7
1,2,3,7,8,9-H_6CDD	<0·1	<0·5	<0·3	<1·1	<0·8	<1·2	2·7	1·8
Total H_6CDD	—	—	—	—	—	—	19·7	16·0

1,2,3,4,6,7,9-H_7CDD	<0·2	Tr. (0·8)	<0·6	<2·2	<1·4	<1·4	2·9	2·9
1,2,3,4,6,7,8-H_7CDD	<0·2	Tr. (0·9)	<0·6	<2·2	<1·4	2·9	4·6	4·6
Total H_7CDD	—	—	—	—	—	2·9	7·5	7·5
O_8CDD	<0·3	4·4	<1·3	<5·0	<4·5	11·9	<4·0	<4·0
Total PCDDs	1·6	6·1	0·7	1·8	1·7	16·1	33·9	27·7
$^{13}C_{12}$-2,3,7,8-T_4CDF Rec. (%)	64·2	37·7	65·5	76·8	67·4	61·6	75·1	77·7
2,3,7,8-T_4CDF	<0·02	<0·1	<0·05	1·1	<0·2	<0·2	<0·3	<0·2
Total T_4CDF	—	—	—	1·1	—	—	—	—
2,3,4,8,9-P_5CDF	<0·03	<0·1	0·2	<0·2	<0·6	<0·3	<0·6	<0·3
Total P_5CDF	—	—	0·2	—	—	—	—	—
Total PCDFs	—	—	0·2	1·1	—	—	—	—

[a] The values were not corrected by recoveries of internal standards.

TABLE 9
PCDF and PCDD Content of Swiss Cow Milk from Various Locations[a] (Rappe *et al.*, 1987*c*)

Compound	*Blank*	*Bern*	*Bowil*	*Bowil*	*Hunzenschwil*	*Suhr*	*Rheinfelden*
Recovery of [^{13}C]-2,3,7,8-tetra-CDF, %	65	64	57	57	63	57	51
Recovery of [^{13}C]-2,3,7,8-tetra-CDD, %	80	90	100	105	90	78	88
Recovery of [^{13}C]octa-CDD, %	86	59	55	77	72	72	48
2,3,7,8-Tetra-CDF	ND (<0·01)	≤0·028	≤0·035	≤0·021	≤0·022	≤0·032	≤0·028
1,2,3,7,8-Penta-CDF	ND (<0·01)	≤0·020	≤0·022	≤0·021	≤0·020	≤0·036	≤0·032
2,3,4,7,8-Penta-CDF	ND (<0·01)	0·084	0·066	0·069	0·43	0·22	0·23
1,2,3,4,7,8-Hexa-CDF	ND (<0·01)	≤0·020	≤0·026	≤0·017	0·13	0·06	0·084
1,2,3,6,7,8-Hexa-CDF	ND (<0·01)	0·028	≤0·018	≤0·021	0·19	0·095	0·059
2,3,4,6,7,8-Hexa-CDF	ND (<0·01)	≤0·020	≤0·018	ND (<0·02)	0·28	0·12	0·049
1,2,3,4,6,7,8-Hepta-CDF	ND (<0·1)	≤0·12	ND (<0·13)	ND (<0·08)	0·49	0·28	≤0·18
Octa-CDF	ND (<0·1)	≤0·20	ND (<0·13)	ND (<0·09)	ND (<0·16)	ND (<0·21)	≤0·52
2,3,7,8-Tetra-CDD	ND (<0·01)	ND (<0·012)	ND (<0·013)	ND (<0·013)	0·049	0·038	0·021
1,2,3,7,8-Penta-CDD	ND (<0·06)	ND (<0·04)	ND (<0·08)	ND (0·06)	0·25	≤0·086	ND (<0·1)
1,2,3,4,7,8-Hexa-CDD	ND (<0·06)	≤0·068	ND (<0·1)	ND (<0·06)	0·23	0·14	≤0·14
1,2,3,6,7,8-Hexa-CDD	ND (<0·06)	≤0·068	ND (<0·1)	ND (<0·06)	0·29	0·16	≤0·21
1,2,3,7,8,9-Hexa-CDD	ND (<0·06)	≤0·068	ND (<0·1)	ND (<0·06)	0·17	≤0·080	≤0·11
1,2,3,4,6,7,8-Hepta-CDD	ND (<0·05)	≤0·064	≤0·066	≤0·064	0·26	≤0·095	0·42
Octa-CDD	≤0·1	≤0·16	≤0·26	≤0·12	0·28	≤0·16	0·59

[a] Results in ng/kg (ppt), whole milk basis. Note: detection limit values are given in parentheses.

CONCLUSIONS

It is difficult to estimate the average daily intake via total food consumption because of few data. The main available data are from levels in different foodstuffs, although the number of samples is small. Foods with the highest levels are fish, followed by milk, chicken, pork and beef. Fruit and vegetables are free of most PCDDs and PCDFs. Beck *et al.* (1987) calculated the average daily intake via food consumption. Table 10 reveals that, within the different food groups, food which contains animal fat is an important contributory factor. Food of plant origin is of minor importance. This is in agreement with data for PCDDs and PCDFs levels in different food groups.

TABLE 10
Average Daily Intake (pg/g) of 2,3,7,8-TCDD and TCDD Equivalents via Food Consumption (Beck *et al.*, 1987)

Food		*Daily intake (pg/day)*		
Group	*Consumption (g/day)*	*2,3,7,8-TCDD*	*TCDD-equivalents*	
			FHO (FRG)	*EPA (USA)*
Meat Meat products	38 (fat)	7·0	23·5	17·9
Milk Dairy products	33 (fat)	6·2	28·5	26·6
Eggs	3·9 (fat)	0·8	4·2	3·1
Fish Fish products	1·8 (fat)	8·6	33·3	38·6
Vegetable oil[a]	26 (fat)	0·1	0·3	0·3
Vegetables[a]	244	1·2	2·4	2·4
Fruit[a]	130	0·7	1·3	1·3
Total		24·6	93·5	90·2

[a] Calculated with half of the ND values.

REFERENCES

Beck, H., Eckart, K., Mathar, W. & Wittkwoski, R. (1987*a*). PCDD and PCDF body burden from food intake in the Federal Republic of Germany. *VDI-Ber*, **634**, ISS Dioxin, pp. 359–82.

Beck, H., Eckart, K., Kellert, M., Mather, W., Rühl, Ch.-S. & Wittkowski, R. (1987*b*). Levels of PCDFs and PCDDs in samples of human origin and food in the Federal Republic of Germany. *Chemosphere*, **16**, 1977–87.

Bruggeman, W. A., Martrom, L. B. J. M., Kooman, D. & Hutzinger, O. (1981). Accumulation and elimination kinetics of di-, tri-, and tetrachlorobiphenyls by goldfish after dietary and aqueous exposure. *Chemosphere*, **10**, 811–32.

Davies, K. (1988). Concentrations and dietary intake of selected organochlorines, including PCBs, PCDDs and PCDFs in fresh food composites grown in Ontario, Canada. *Chemosphere*, **17**, 263–76.

Esposito, M. P., Tiernan, T. & Dryden, F. E. (1980). Dioxins. US Environmental Protection Agency EPA-600-80-197, pp. 255–6.

Graham, M., Hileman, F., Kirk, D., Wedling, J. & Wilson, J. (1985). Background human exposure to 2,3,7,8-TCDD. *Chemosphere*, **14**, 925–8.

Kleeman, J. M., Olson, J. R., Chen, S. M. & Peterson, R. E. (1986*a*). Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in rainbow trout. *Toxicol. Appl. Pharmacol.*, **83**, 391–401.

Kleeman, J. M., Olson, J. R., Chen, S. M. & Peterson, R. E. (1986*b*). 2,3,7,8-tetrachlorodibenzo-*p*-dioxin metabolism and disposition in yellow perch. *Toxicol. Appl. Pharmacol.*, **83**, 402–11.

Liberti, A. & Brocco, D. (1982). Formation of polychlorodibenzodioxins and polychlorodibenzofurans in urban incinerators emissions. In *Chlorinated Dioxins and Related Compounds. Impact on the Environment*. ed. O. Hutzinger, R. W. Frei, E. Merian & F. Pocchiari. Pergamon Press, Oxford, pp. 245–51.

Niimi, A. J. & Oliver, B. G. (1989). Assessment of relative toxicity of chlorinated dibenzo-*p*-dioxins, dibenzofurans, and biphenyls in Lake Ontario salmonids to mammalian systems using toxic equivalent factors (TEF). *Chemosphere*, **18**, 1413–23.

Norris, L. A. (1981). The movement, persistence and fate of phenoxy herbicides and TCDD in the Forest. *Residue Reviews*, **80**, 65–135.

Nygren, M., Rappe, C., Lindström, G., Hansson, M., Bergqvist, P. A., Marklund, S., Domellöf, L., Hardell, L. & Olsson, M. (1986*a*). *Chlorinated Dioxins and Dibenzofurans in the Total Environment, Vol. III*, ed. C. Rappe, G. Choudhary & L. Keith. Lewis Publishers, Chelsea, MI, pp. 17–34.

Nygren, M., Rappe, C., Lindström, G., Hansson, M., Bergqvist, P. A., Marklund, S., Domellöf, L., Hardell, L. & Olsson, M. (1986*b*). Identification of 2,3,7,8-substituted polychlorinated dioxins and dibenzofurans in environmental and human samples. In *Chlorinated Dioxins and Dibenzofurans in Perspective*, ed. C. Rappe, G. Choudhary & L. Keith. Lewis Publishers, Chelsea MI, pp. 17–34.

Ogaki, J., Takayama, K., Miyata, H. & Kashimoto, T. (1987). Levels of PCDDs and PCDFs in human tissues and various foodstuffs in Japan. *Chemosphere*, **16**, 2047–56.

O'Keefe, P., Meyer, C., Hilker, D., Aldous, K., Jelusturor, B., Dillon, K., Donnelly, R., Horn, E. & Sloan, R. (1983). Analysis of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in Great Lakes fish. *Chemosphere*, **12**, 325–32.

Ono, M., Kashima, Y., Wakimoto, T. & Tatsukama, R. (1987). Daily intake of PCDDs and PCDFs by Japanese through food. *Chemosphere*, **16**, 1823–8.

Rappe, C., Nygren, M., Buser, H.-R. & Kauppinen, T. (1982). Occupational exposure to polychlorinated dioxins and dibenzofurans. In *Chlorinated Dioxins and Related Compounds. Impact on the Environment*, ed. O. Hutzinger, R. W. Frei, E. Merian & F. Pocchiari. Pergamon Press, Oxford, pp. 495–513.

Rappe, C. Nygren, M., Lindström, G. & Hansson, M. (1986). Dioxins and dibenzofurans in biological samples of European origin. *Chemosphere*, **15**, 1635–9.

Rappe, C., Andersson, R., Bergqvist, P.-A. Brohede Ch., Hansson, M., Kjeller, L. O., Lindström, G., Marklund, S., Nygren, M., Swanson, S. E., Tysklind, M. & Wiberg, K. (1987*a*). Overview on environmental fate of chlorinated dioxins and dibenzofurans. Sources, levels and isomeric pattern in various matrices. *Chemospher*, **16**, 1603–18.

Rappe, C. Andersson, R., Bergqvist, P.-A., Brohede, Ch., Hansson, M., Kjeller, L. O., Lindström, G., Marklund, S., Nygren, M., Swanson, S. E., Tysklind, M. & Wiberg, K. (1987*b*). Sources and relative importance of PCDD and PCDF emissions. *Waste Management & Research*, **5**, 225–7.

Rappe, C., Nygren, M., Lindström, G., Buser, H. R., Blaser, O. & Wüthrich, C. (1987*c*). Polychlorinated dibenzofurans and dibenzo-*p*-dioxins and other chlorinated contaminants in cow milk from various locations in Switzerland. *Environ. Sci. & Technol.*, **21**, 964–70.

Reggiani, G. (1982). Toxicology of TCDD and related compounds: Observations in man. In *Chlorinated Dioxins & Related Compounds. Impact on the Environment*, ed. O. Hutzinger, R. W. Frei, E. Merian & R. Pocchiari. Pergamon Press, Oxford, pp. 463–93.

Rossiers, P. E. (1987). National Dioxin Study. In *Solving Hazardous Waste Problems: Learning from Dioxins*, ed. J. H. Exner. ACS Symposium Series 338, Washington, DC, pp. 34–53.

Ryan, J. J., Lau, P.-Y., Pilon, J. C., Lewis, D., McLeod, H. A. & Gervais, A. (1984). Incidence and levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin in Lake Ontario commercial fish. *Environ. Sci. Technol.*, **18**, 719–21.

Ryan, J. J., Lizotte, R. & Lau, B. P.-Y. (1985*a*). Chlorinated dibenzo-*p*-dioxins and chlorinated dibenzofurans in Canadian human adipose tissue. *Chemosphere*, **14**, 697–706.

Ryan, J. J., Schecter, A., Lizotte, R., Wing, F. & Miller, L. (1985*b*). Tissue distribution of dioxins and furans in humans from the general population. *Chemosphere*, **14**, 929–32.

Ryan, J. J., Lizotte, R., Sakuma, T. & Mori, B. (1985*c*). Chlorinated dibenzo-*p*-dioxins, chlorinated dibenzofurans, and pentachlorophenol in Canadian chicken and pork samples. *J. Agric. Food Chem.*, **33**, 1021–6.

Stalling, D. L. (1983). Residues of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in Laurentian Great Lakes fish. In *Human and Environmental Risk of Chlorinated Dioxins and Related Compounds*, ed. R. E. Tucker, A. L. Young & A. P. Gray. Plenum Press, New York, pp. 221–40.