Journal of Toxicology and Environmental Health, Part A, 63:1–18, 2001

1528-7394/01 $12.00 + .00

INTAKE OF DIOXINS AND RELATED COMPOUNDS FROM FOOD IN THE U.S. POPULATION

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*The first U.S. nationwide food sampling with measurement of dioxins, dibenzofurans, and coplanar, mono-*ortho *and di-*ortho *polychlorinated biphenyls (PCBs) is reported in this study. Twelve separate analyses were conducted on 110 food samples divided into pooled lots by category. The samples were purchased in 1995 in supermarkets in Atlanta, GA, Binghamton, NY, Chicago, IL, Louisville, KY, and San Diego, CA. Human milk also was collected to estimate nursing infants' consumption. The food category with highest World Health Organization (WHO) dioxin toxic equivalent (TEQ) concentration was farm-grown freshwater fish fillet with 1.7 pg/g, or parts per trillion (ppt), wet, or whole, weight. The category with the lowest TEQ level was a simulated vegan*

Received 27 July 2000; sent for revision 29 August 2000; accepted 13 September 2000.

Funding for this project was provided by the C.S. Fund, Warsh-Mott Legacy, and the Albert Kunstadter Family Foundation. This article was prepared with the assistance of Amanda Piskac, Research Assistant, and Bindhu Oommen, MPH student at the University of Texas–Houston School of Public Health, Satellite Program at UT Southwestern–Dallas.

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diet, with 0.09 ppt. TEQ concentrations in ocean fish, beef, chicken, pork, sandwich meat, eggs, cheese, and ice cream, as well as human milk, were in the range 0.33 to 0.51 ppt, wet weight. In whole dairy milk TEQ was 0.16 ppt, and in butter 1.1 ppt. Mean daily intake of TEQ for U.S. breast-fed infants during the first year of life was estimated at 42 pg/kg body weight. For children aged 1–11 yr the estimated daily TEQ intake was 6.2 pg/kg body weight. For males and females aged 12–19 yr, the estimated TEQ intake was 3.5 and 2.7 pg/kg body weight, respectively. For adult men and women aged 20–79 yr, estimated mean daily TEQ intakes were 2.4 and 2.2 pg/kg body weight, respectively. Estimated mean daily intake of TEQ declined with age to a low of 1.9 pg/kg body weight at age 80 yr and older. For all ages except 80 yr and over, estimates were higher for males than females. For adults, dioxins, dibenzofurans, and PCBs contributed 42%, 30%, and 28% of dietary TEQ intake, respectively. DDE was also analyzed in the pooled food samples.

Polychlorinated dibenzo-*p*-dioxins (PCDDs, or dioxins), polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs) are synthetic, toxic, and highly persistent chemicals first produced in the 20th century. They are now ubiquitous and enter the general population almost exclusively from ingestion of food (Startin, 1994; Beck et al., 1989; Birmingham et al., 1989; Theelen, 1991; Ministry of Agriculture, Fisheries, and Food, 1992). Periodically, special food contamination occurs. Recently, in Belgium, chickens, eggs, some pigs and cattle, and dairy products were contaminated from feed made with PCB- and dibenzofuran-contaminated animal fat. A large proportion of Belgian food products had to be removed from the market and destroyed (Hileman, 1999). These were the same chemicals that contaminated rice oil in Japan, leading to the Yusho or rice oil incident of 1968 (Masuda et al., 1996).

This report is the first nationwide study of U.S. foods analyzed for dioxins and the closely related compounds, the dibenzofurans and PCBs. One hundred and ten food items were purchased in 5 regions of the United States and combined by food categories in 12 lots for separate dioxin analyses. Along with dioxins and dibenzofurans, three coplanar, three mono-*ortho,* and four di-*ortho* PCB congeners were measured. The average daily dioxin toxic equivalent (TEQ) intake by males and females of different ages was calculated on the basis of the U.S. Department of Agriculture's Continuing Survey of Food Intakes by Individuals, 1989–1991 (USDA et al., 1995). TEQ intake of nursing infants was estimated using results of analyses of human milk samples collected from nursing women in Binghamton, New York (Schecter et al., 1996a). 1,1-Bis(*p*-chlorophenyl)-2,2-dichloroethylene (DDE), a metabolite of dichlorodiphenyltrichloroethane (DDT), also was measured in the purchased food items, and its average daily intake was calculated.

The concept of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) toxic equivalency (TEQ) was developed in order to simplify risk assessment and regulatory control (Eadon et al., 1986). Toxicity of the individual dioxin, dibenzofuran, and dioxinlike PCB congeners is assessed based on evaluation of the data from in vitro and in vivo studies, leading to determination

of dioxin toxic equivalency factors (TEFs). These are order-of-magnitude consensus estimates of dioxinlike toxicity. The TEFs are used to weight the measured levels of the congeners present in a sample in relation to the most toxic dioxin congener, TCDD, which is defined as having a TEF of 1. The measured concentration of each congener is multiplied by the TEF weighting factor. The total dioxinlike toxic equivalency, or TEQ, is the sum of these products.

The 1998 revision of dioxin, dibenzofuran, and PCB TEFs by the World Health Organization (WHO) is used in this study (Van den Berg et al., 1998). It succeeds the "international" dioxin and dibenzofuran TEFs used since 1988 (NATO, 1988a, 1988b).

At the present time, PCBs are found at much higher levels than PCDDs and PCDFs in the food supply and in human tissues, and some PCB congeners also exhibit dioxinlike toxicity. Results of assessments of PCB exposures in general population samples in industrial countries, based on the 1998 WHO TEFs for PCBs (Ahlborg et al., 1994), suggest that TEQ body burden from PCBs is comparable in amount to the TEQ burden from PCDDs and PCDFs combined (Jimenez et al., 1996; Masuda et al., 1996; Päpke et al., 1996; Schecter et al., 1996b; Liem & Theelen, 1997; Alcock et al., 1998; Becher et al., 1998; Schecter & Päpke, 1998). Current consensus dioxin TEFs are used for 12 PCB congeners from the 1998 WHO revision (Table 2, Van den Berg et al., 1998).

METHODS

In 1995 we purchased a total of 110 food items from supermarkets in cities in 5 regions of the United States: the Northeast (Binghamton, NY), the Mid-South (Louisville, KY), the Deep South (Atlanta, GA), the Midwest (Chicago, IL), and the West Coast (San Diego, CA). Purchased items were immediately frozen and shipped to the Midwest Research Institute in Kansas City, MO, a laboratory certified by WHO for analysis of dioxins, dibenzofurans, and PCBs in biological specimens (WHO, 1991). The samples were pooled into 12 separate lots by food category. Each lot was analyzed separately. The 12 food categories included 4 types of meat, 4 types of dairy product, eggs, ocean fish, freshwater fish, and a simulated completely vegetarian, or "vegan," diet, shown in Table 1.

PCDDs and PCDFs previously have been detected primarily in lipids of animal origin, and are usually below detection limits in vegetables and fruits (Startin, 1994; Beck et al., 1989; Birmingham et al., 1989; Theelen, 1991; Ministry of Agriculture, Fisheries, and Food, 1992). Blood dioxin levels in pure vegans have also been found to be very low in comparison with the general population, indicating a lower contribution of these foods to human dioxin body burden (Schecter & Päpke., 1998). For analysis, all 20 samples of fruits, vegetables, legumes, or cereal products were combined into 1 simulated vegan diet sample.

TABLE 1. Pooled Food Samples for Dioxin Analyses Collected in 1995 From Five Regions of the United States

Pooled samples	Foods included for analysis
Meat	
1. Beef	Ground beef: regular, lean, 75% lean, and choice Beefsteak: cubed, bone in hind, and choice rib steak Round stew beef
2. Chicken	Thigh, drumstick, breast, boneless thigh and breast, split fryer
3. Pork	Pork loin, boneless loin chops, boneless chops Pork sausage and link sausage
4. Sandwich meat	Hot dogs, bologna
Fish	
5. Ocean fish, including shellfish	Fresh salmon steak, king salmon steak, true cod filets, ocean perch filets, sea bass, halibut, true cod filets, fresh scrod/cod, fresh/frozen cod filets, tiger prawns
6. Freshwater fish	Catfish steak, catfish filets, lemon pepper catfish, rainbow trout, boneless rainbow trout, perch filets, Lake Superior whitefish, farm-raised fresh salmon steak
Dairy	
7. Butter	Butter, unsalted butter, and salted sweet cream butter
8. Cheese	American slices, brie, hot pepper cheese food, Swiss slices, American cheese food, processed cheese food, natural Muenster, pasteurized cheese food, Cracker Barrel sharp, cheese variety pack
9. Milk	Whole, vitamin D, homogenized, and heavy cream
10. Ice cream	Chocolate-chocolate chip
11. Eggs	Hard boiled eggs
12. Vegan diet	Fruit: Apples, bananas, oranges, nectarines, canned fruit Vegetables: Green beans, salad, frozen mixed vegetables Legumes: Kidney beans, lentils, tofu, peanut butter Cereals: Graham crackers, bread, cereal, macaroni, rice

Preparation for analysis involved solid/liquid extraction of lipids, followed by a multicolumn cleanup using carbon on glass fiber. Measurement and quantification were performed by high-resolution gas chromatography mass-spectrometry (GC/MS) applying an isotope dilution method. Analytic methods have been described previously and are not repeated in detail here (Stanley & Bauer, 1989).

Samples were analyzed for 7 PCDD, 10 PCDF, and 10 PCB congeners, and also for DDE, listed in Table 2. These compounds are among those referred to as (toxic) persistent organic pollutants (POPs), or sometimes as endocrine disrupters.

The recent human milk samples were collected from women in Binghamton, NY, in 1996, immediately frozen, and shipped to Hamburg,

TABLE 2. Measured Levels of PCDDs, PCDFs, PCBs, and DDE in Foods Collected in Five U.S. Regions (pg/g Wet Weight)

Congener	TEF	Beef	Chicken	Pork	Hot dog/ bologna	Ocean fish	Freshwater fish
PCDDs							
2378 TCDD	1	ND (0.025)	ND (0.025)	ND (0.032)	ND (0.025)	0.033	0.149
12378 PnCDD	1	ND (0.125)	ND (0.124)	ND (0.125)	ND (0.125)	ND (0.125)	0.251
123478 HxCDD	0.1	ND (0.182)	ND (0.124)	ND (0.125)	ND (0.125)	ND (0.125)	0.145
123678 HxCDD	0.1	0.646	ND (0.124)	0.166	0.360	ND (0.125)	0.257
123789 HxCDD	0.1	0.137	ND (0.124)	ND (0.125)	ND (0.125)	ND (0.125)	0.198
1234678 HpCDD	0.01	2.74	0.431	1.57	3.99	0.166	1.14
OCDD	0.0001	4.29	1.07	8	25.7	0.45	5.88
PCDFs							
2378 TCDF	0.1	0.064	ND (0.100)	0.181	ND (0.053)	ND (0.165)	0.693
12378 PnCDF	0.05	ND (0.125)	ND (0.124)	ND (0.125)	ND (0.125)	ND (0.125)	ND (0.367)
23478 PnCDF	0.5	ND (0.125)	0.139	ND (0.125)	ND (0.125)	ND (0.125)	0.365
123478 HxCDF	0.1	ND (0.160)	ND (0.124)	ND (0.125)	ND (0.350)	ND (0.153)	ND (0.162)
123678 HxCDF	0.1	ND (0.282)	ND (0.151)	ND (0.593)	ND (0.708)	ND (0.237)	ND (0.962)
234678 HxCDF	0.1	ND (0.125)	ND (0.124)	ND (0.125)	ND (0.245)	ND (0.136)	ND (0.125)
123789 HxCDF	0.1	ND (0.125)	ND (0.155)	ND (0.125)	ND (0.364)	ND (0.206)	ND (0.125)
1234678 HpCDF	0.01	ND (1.420)	ND (0.293)	ND (0.185)	0.889	ND (0.694)	ND (1.5)
1234789 HpCDF	0.01	ND (0.125)	ND (0.125)	ND (0.125)	ND (0.125)	ND (0.125)	ND (0.131)
OCDF	0.0001	ND (0.394)	ND (0.249)	ND (0.299)	0.564	ND (0.249)	ND (0.250)
Coplanar PCBs							
77 33′44′ TPCB	0.0001	3.93	10.7	10.6	2.96	6.19	29.5
126 33′44′5 PnPCB	0.1	0.394	0.383	0.674	0.712	0.834	6.070
169 33′44′55′ HxPCB	0.01	0.116	ND (0.100)	0.144	ND (0.100)	0.197	0.936
Mono-*ortho* PCBs							
105 233′44′ PnPCB	0.0001	ND (250)	78	ND (250)	395	119	599
114 2344′5 PnPCB	0.0005	ND (250)	ND (250)	ND (250)	ND	ND (250)	ND (250)
118 23′44′5 PnPCB	0.0001	94	ND (197)	ND (250)	1060	318	1750

(*Table continues on next page*)

TABLE 2. Measured Levels of PCDDs, PCDFs, PCBs, and DDE in Foods Collected in Five U.S. Regions (pg/g Wet Weight) (*Continued*)

Congener	TEF	Beef	Chicken	Pork	Hot dog/ bologna	Ocean fish	Freshwater fish
Di-*ortho* PCBs							
128 22′33′44′ HxPCB		ND (250)	ND (250)	ND (250)	310	ND (250)	270
138 22′344′5′ HxPCB		ND (250)	40	98	861	431	1810
153 22′44′55′ HxPCB		83	111	72	560	390	1900
180 22′344′55′ HpPCB		ND (260)	230	ND (250)	140	168	607
Total PCDDs		7.81	1.50	9.74	30.05	0.65	8.02
Total PCDFs		0.06	0.14	0.18	1.45	0	1.06
Total coplanar PCBs		4.44	11.08	11.42	3.67	7.22	36.51
Total mono-*ortho* PCBs		94	78	0	1455	437	2349
Total di-*ortho* PCBs		83	381	170	1871	989	4587
DDE pg/g		433	149	206	1090	978	9950

Congener	TEF	Butter	Cheese	Milk	Ice cream	Eggs	Vegetables
PCDDs							
2378 TCDD	1	0.102	ND (0.050)	ND (0.021)	ND (0.038)	ND (0.025)	ND (0.012)
12378 PnCDD	1	ND (0.301)	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)
123478 HxCDD	0.1	ND (0.251)	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)
123678 HxCDD	0.1	0.998	0.309	ND (0.103)	0.278	ND (0.158)	ND (0.062)
123789 HxCDD	0.1	0.386	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)
1234678 HpCDD	0.01	5.86	1.24	0.22	1.07	2.74	ND (0.187)
OCDD	0.0001	59.2	8.38	1.6	6.22	33.1	2.09
PCDFs							
2378 TCDF	0.1	ND (0.050)	ND (0.050)	ND (0.021)	ND (0.038)	0.033	ND (0.012)
12378 PnCDF	0.05	ND (0.251)	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)
23478 PnCDF	0.5	ND (0.251)	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)

123478 HxCDF	0.1	ND (0.474)	ND (0.249)	ND (0.103)	ND (0.302)	ND (0.671)	ND (0.062)
123678 HxCDF	0.1	ND (0.816)	ND (0.249)	ND (0.103)	ND (0.334)	ND (0.769)	ND (0.062)
234678 HxCDF	0.1	ND (0.753)	ND (0.249)	ND (0.103)	ND (0.411)	ND (0.564)	ND (0.062)
123789 HxCDF	0.1	ND (0.251)	ND (0.249)	ND (0.131)	ND (0.387)	ND (0.827)	ND (0.062)
1234678 HpCDF	0.01	0.791	ND (0.249)	ND (0.103)	ND (0.188)	0.277	ND (0.062)
1234789 HpCDF	0.01	ND (0.251)	ND (0.249)	ND (0.103)	ND (0.188)	ND (0.124)	ND (0.062)
OCDF	0.0001	1.22	ND (0.499)	ND (0.206)	ND (0.377)	1.340	ND (0.012)
Coplanar PCBs							
77 33′44′ TPCB	0.0001	2.76	1.04	0.353	0.739	1.49	0.47
126 33′44′5 PnPCB	0.1	3.36	1.04	0.162	0.861	0.286	0.065
169 33′44′55′ HxPCB	0.01	0.39	ND (0.2)	0	ND (0.151)	ND (0.099)	ND (0.050)
Mono-*ortho* PCBs							
105 233′44′ PnPCB	0.0001	219	ND	NQ	NQ	ND	ND
114 2344′5 PnPCB	0.0005	ND	ND	NQ	NQ	ND	ND
118 23′44′5 PnPCB	0.0001	925	244	NQ	NQ	64	15
Di-*ortho* PCBs							
128 22′33′44′ HxPCB		351	ND	NQ	NQ	ND	14
138 22′344′5′ HxPCB		786	147	NQ	NQ	62	87
153 22′44′55′ HxPCB		690	163	NQ	NQ	69	36
180 22′344′55′ HpPCB		246	32	NQ	NQ	16	7
Total PCDDs		66.5	9.93	1.82	7.57	35.84	2.09
Total PCDFs		2.01	0	0	0	1.65	0
Total coplanar PCBs		6.51	2.08	0.52	1.60	1.78	0.54
Total mono-*ortho* PCBs		1144	244	0	0	64	15
Total di-*ortho* PCBs		2073	342	0	0	147	144
DDE pg/g		20700	2780	NQ	2780	674	453

Note. ND, not detected; limit of detection given in parentheses. NQ, not quantifiable due to interference.

Germany, for analysis at ERGO Laboratory. ERGO Laboratory is also certified by WHO for dioxin analyses of biological specimens (WHO, 1991). The human milk samples were analyzed individually for dioxins, dibenzofurans, and three coplanar PCBs. Analytical methods were the same as for purchased food samples and have been described previously for ERGO laboratory (Ball et al., 1989).

Estimates of average daily consumption were derived from the U.S. Department of Agriculture's Continuing Survey of Food Intakes by Individuals, based on data from 15,000 respondents interviewed during 1989–1991 (USDA et al., 1995). Survey data collection was designed to include balanced representations of different regions of the United States, different socioeconomic groups and races, and different seasons. Results were stratified by age and gender.

RESULTS

The results of analyses of the 12 pooled food samples are given in Table 2 as picograms per gram, or parts per trillion (ppt), wet weight, as eaten. TEFs are listed for dioxins, dibenzofurans, and coplanar and mono-*ortho* PCBs. Although di-*ortho* PCBs currently are not believed to exert dioxinlike toxicity (Van den Berg et al., 1998), the nondioxinlike PCB congeners exhibit toxicity by other pathways that do not involve the aryl hydrocarbon (Ah) receptor as does dioxin. The di-*ortho* congeners are prevalent in the environment and in human tissues and can be useful markers for total PCB contamination in human blood (Ballschmitter et al., 1989; Schecter et al., 1994a; Hardell et al., 1996). Their monitoring in human tissues can be useful to estimate patterns and levels of PCB contamination and also potential PCB health consequences (Seegal & Schantz, 1994; Nicholson & Landrigan, 1994).

Wet weight TEQ concentrations for the pooled samples and the mean concentration for human milk are presented in Table 3. Wet weight rather than lipid-based TEQ levels are employed in order to estimate dietary intake. The percentage lipid content for each lot of foods analyzed and the mean lipid levels of the human milk samples are given to facilitate comparison with lipid-adjusted data. As is customary, congeners not detected are usually estimated at half the level of the limit of detection (LOD) in this article. Total TEQs calculated with nondetected (ND) congeners calculated as zero rather than half the LOD are given in the bottom row of the table for comparison. TEQ levels calculated with ND = ½LOD and ND = 0 are represented graphically in Figure 1, A and B, respectively.

Freshwater fish were found to have the highest wet weight dioxin toxicity, with 1.7 ppt TEQ, followed by butter with 1.1 ppt. Ocean fish, meat, poultry, sandwich meats, eggs, cheese, and milk desserts, as well as human milk, were found to have wet weight dioxin TEQ contamination in the range of 0.33 to 0.51 ppt.

TABLE 3. Whole-Weight TEQ Levels of Dioxins, Dibenzofurans, and PCBs in Different Foods and in Human Milk (pg/g) and Lipid Percentages

	Meat				Fish		Dairy						
	Beef	Chicken	Pork	Hot dogs, bologna	Ocean fish	Freshwater fish	Butter	Cheese	Milk	Ice cream	Eggs	Vegan foods	Human milk
Percent lipid	13.1	5.3	9.2	28.6	1.4	4.8	89.1	21.8	2.15	22.0	14.7	2.7	3.70
PCDDs	0.189	0.097	0.124	0.165	0.115	0.472	0.467	0.218	0.079	0.171	0.125	0.047	0.257
PCDFs	0.083	0.107	0.111	0.130	0.083	0.338	0.195	0.123	0.052	0.127	0.184	0.031	0.089
PCDDs + PCDFs	0.272	0.204	0.234	0.294	0.198	0.810	0.662	0.341	0.131	0.298	0.307	0.078	0.346
Coplanar PCBs	0.041	0.040	0.070	0.072	0.086	0.619	0.340	0.105	0.017	0.087	0.029	0.007	0.075
Mono-*ortho* PCBs	0.084	0.090	0.088	0.146	0.106	0.297	0.114	0.024	NQ	NQ	0.006	0.002	
Total PCBs	0.125	0.130	0.158	0.218	0.192	0.916	0.455	0.129			0.036	0.008	
Total TEQ	0.397	0.334	0.392	0.512	0.390	1.726	1.117	0.470	0.160[a]	0.416[a]	0.343	0.086	0.420[c]
Total TEQ, ND = 0	0.163	0.141	0.121	0.304	0.164	1.578	0.823	0.173	0.022[b]	0.142[b]	0.072	0.008	0.417[c]

Note. Except where otherwise stated, nondetects = ½ limit of detection. NQ, not quantifiable due to interference.
[a]Corrected by factor of 1.08 to compensate for missing data.
[b]Corrected by factor of 1.14 to compensate for missing data.
[c]Actual value may be higher. Totals not corrected to compensate for missing PCB data.

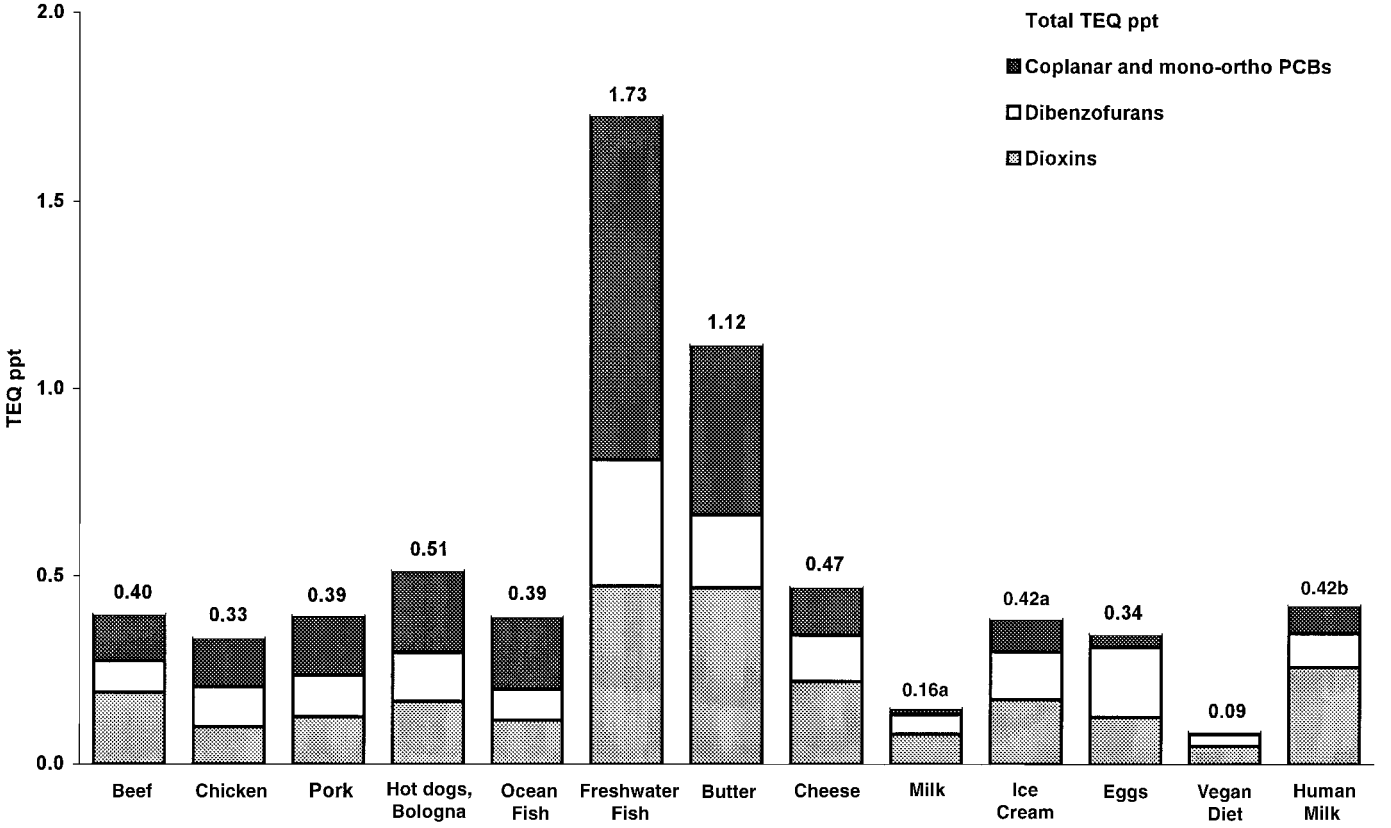

FIGURE 1. Dioxin, dibenzofuran, and PCB TEQs in collected foods and in human milk, pg/g (ppt) wet weight. (A) Nondetects = ½ limit of detection. a, Corrected by factor of 1.08 to compensate for missing PCB data. b, Actual value may be higher. Not corrected to compensate for missing PCB data.

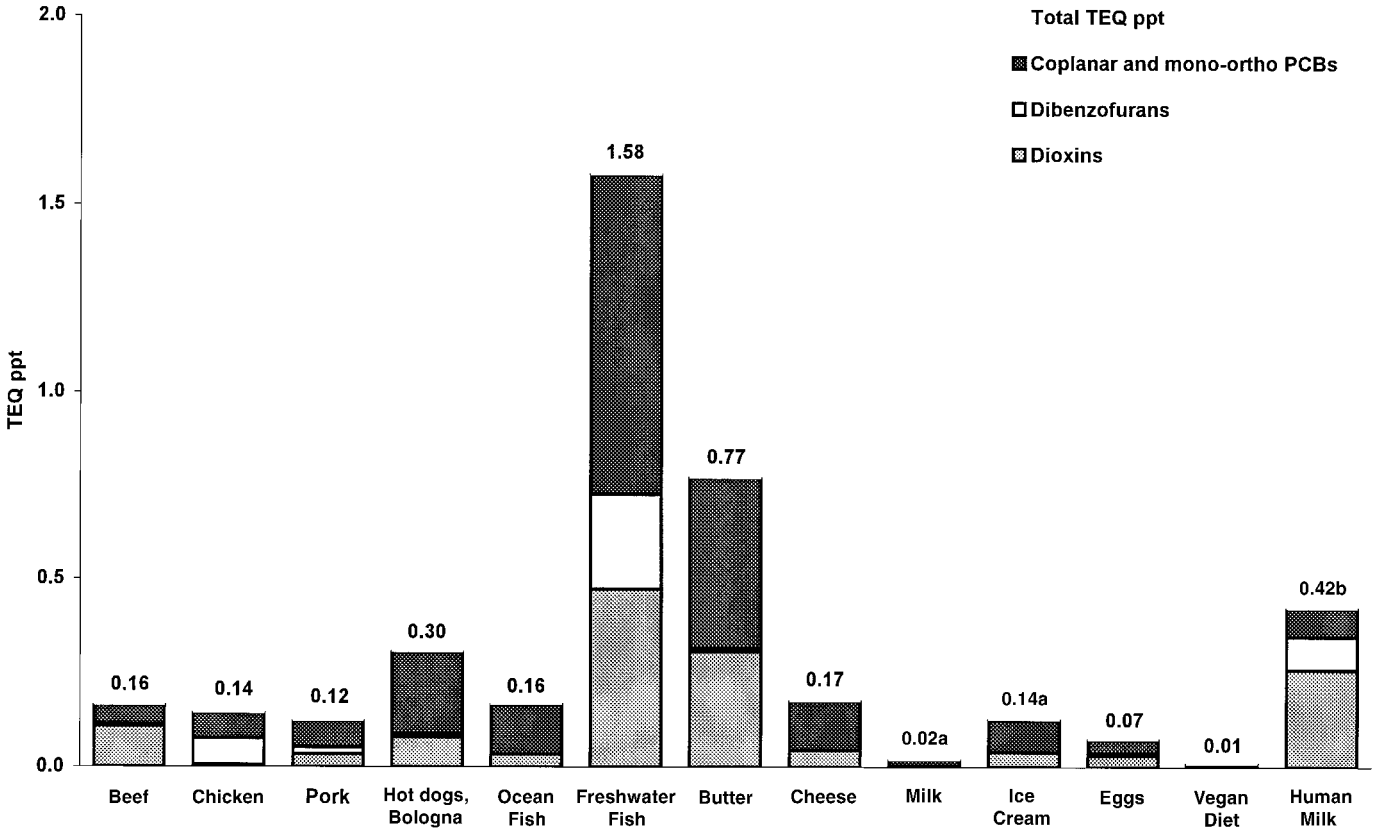

FIGURE 1. (*Continued*) Dioxin, dibenzofuran, and PCB TEQs in collected foods and in human milk, pg/g (ppt) wet weight. (B) Nondetects = 0. a, Corrected by factor of 1.14 to compensate for missing PCB data. b, Actual value may be higher. Not corrected to compensate for missing PCB data.

Estimated average daily TEQ intake in picograms per person for five age groups, along with total daily TEQ intake proportional to body weight, is given in Table 4, with proportional TEQ consumption estimates graphically presented in Figure 2. (TEQ consumption estimates in this report are—with one exception, Table 3, bottom line—based on TEQ levels with ND = ½LOD.) The highest estimated daily TEQ intake, even with absent data for mono-*ortho* PCBs in human milk, is 252 pg/d for breast-feeding infants. Relative to body weight, daily TEQ intake for an infant is 42 pg/kg body weight, several times higher than for children and adults. The high TEQ intake by nursing infants has been recognized previously (Patandin et al., 1997; Schecter & Gasiewicz, 1987; Schecter et al., 1994b). In the age range 1–11 yr, estimated average daily TEQ intake in proportion to body weight is 6.3 pg/kg body weight for boys and a nearly equal 6.1 pg/kg body weight for girls. For ages 12–19 yr, estimated average proportional daily TEQ intake is 3.5 pg/kg body weight for males and 2.7 pg/kg body weight for females. For adult men, aged 20–79 yr, estimated TEQ intake per day is 2.4 pg/kg body weight. For adult women, it is 2.2 pg/kg body weight. Persons aged 80 yr or over have the lowest estimated daily TEQ intake, 1.8 pg/kg body weight for men and 2 pg/kg body weight for women.

For various age groups, percent of TEQ contributed by the major food categories, meat, dairy products, and vegetables, seen in Table 4, is similar for males and females. The share of TEQ contributed by fish is smaller than previous estimates, because fish is consumed in smaller quantities in the United States than in many other countries. The largest share of TEQ is from dairy foods for ages 1–11 and 12–19 yr, followed by meat, then

TABLE 4. Estimated Daily Intake of PCDD, PCDF, and PCB Dioxin TEQ by Food Category, Age, and Gender

Age (yr)	Gender	TEQ: pg/d					Total TEQ (pg/d)	Mean body weight (kg)	TEQ (pg/kg body weight/d)
		Meat	Fish	Dairy	Eggs	Vegetables			
0–1	Male and female			252.0			252	6	42.0
1–11	Male	32.8	4.8	74.8	5.9	25.6	144	23	6.3
	Female	31.8	5.4	71.0	5.2	27.5	141	23	6.1
12–19	Male	61.3	4.8	81.9	6.6	35.9	191	55	3.5
	Female	41.5	4.2	57.7	4.5	25.0	133	50	2.7
20–79	Male	61.7	14.5	49.1	9.5	36.4	171	70	2.4
	Female	38.8	10.8	36.9	5.9	28.5	121	55	2.2
80+	Male	38.9	3.0	38.7	8.6	36.3	126	70	1.8
	Female	25.5	11.4	43.2	4.5	26.6	111	55	2.0

Note. Nondetects = ½ limit of detection.

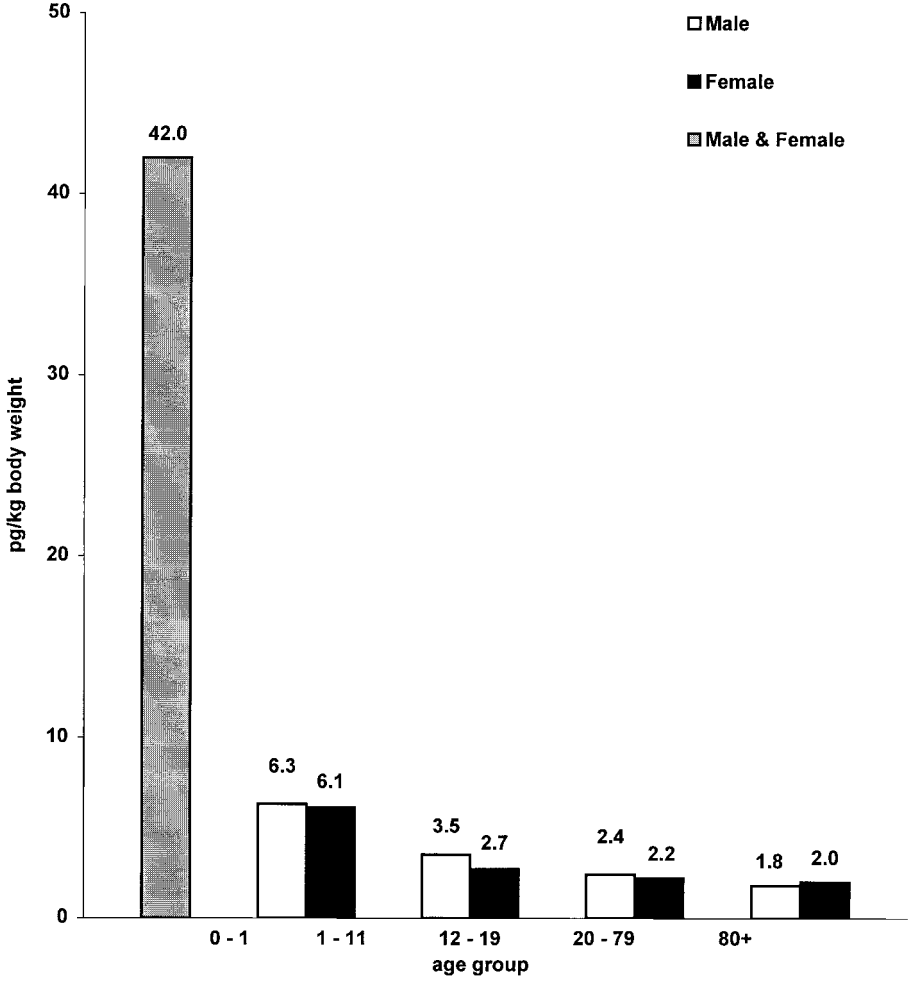

FIGURE 2. Average daily intake of dioxin TEQ by age and sex, pg/kg body weight. a, Nondetects = ½ limit of detection.

vegetables. In our survey, meat contributes the largest share of TEQ in adult diet, followed by dairy foods, then the vegetables. For females 80 yr and over, dairy foods account for the largest share of TEQ intake, then meat and vegetables. For males 80 yr and over, meat, dairy foods, and vegetables all contribute about equally to daily TEQ intake.

Estimated relative contributions of PCDDs, PCDFs, and PCBs to total average daily intake of dioxin TEQ for male and female adults, aged 20–79 yr, are shown in Table 5. PCDDs contribute a slightly larger share of total dietary TEQ intake than do PCDFs or PCBs.

Estimated dietary intakes of DDE (ng/day) are given in Table 6. Vegetables contribute a large amount of DDE.

TABLE 5. Percentage Contributions of PCDDs, PCDFs, and PCBs to Estimated Daily Intake of Dioxin TEQ From Different Food Categories for U.S. Adults Aged 20–79 Years

	Percentage of TEQ intake for food type		
Food category	PCDDs	PCDFs	PCBs
Meat	38	25	37
Fish	28	21	51
Dairy[a]	45	24	31
Eggs	36	53	11
Vegan foods sample	55	36	9
Average	42	30	28

Note. ND = ½ limit of detection.
[a]Based on data for butter and cheese.

DISCUSSION

Nursing infants have a far higher intake of dioxins relative to body weight than do all older age groups. The mean TEQ level in human breast milk included with these results is more than twice that in the dairy milk samples analyzed. A major reason for the markedly higher TEQ intake by infants in the first year of life is their high average daily milk consumption of 100 g/kg body weight (Patandin et al., 1997). The TEQ level in human milk in 1996 is lower than the level found for women living in Binghamton, NY, and Los Angeles, CA, in 1988, 0.43 and 0.64 ppt wet weight, respectively, not including PCBs (Schecter & Gasiewicz, 1987). In this study, mean lipid-based TEQ for dioxins, dibenzofurans, and coplanar PCBs is 11.4 ppt, lower than levels reported recently in human milk for 8 European countries ranging from 20 ppt (Czech Republic) to 51 ppt in the Netherlands (Gladen et al., 1999).

The high proportional intake of TEQ for children aged 1–11 yr relative to adults is due to their consuming almost the same quantity of food,

TABLE 6. Estimated Daily Intake of DDE by Food Category, Age, and Gender (ng/d)

Age (yr)	Gender	Meat	Fish	Dairy	Eggs	Vegetables	Total
1–11	Male	39	19	99	12	152	321
	Female	38	22	102	10	162	334
12–19	Male	73	19	132	13	218	455
	Female	45	17	124	9	155	350
20–79	Male	68	58	120	19	206	471
	Female	40	43	100	12	163	358
80+	Male	44	12	85	17	197	355
	Female	24	46	85	9	160	324

although their average weight is considerably less than that of adults. Similarly, adolescents consume more food relative to body weight than do adults. Adolescent boys, aged 12–19 yr, consume approximately 25% more food relative to body weight than do adolescent girls, leading to higher TEQ intake, 3.5 versus 2.7 pg/kg body weight/d. American men aged 20–79 yr have higher proportional TEQ intake than do women, 2.4 to 2.2 pg/kg body weight/d, because meat and eggs constitute a higher percentage of men's than women's diet, and fruits and vegetables constitute a lower percentage.

In reports published from 1989 to 1992, estimates of mean daily TEQ intake for adults in Germany, England, the Netherlands, and Canada, based on measurement of dioxins and dibenzofurans, but not PCBs, in foods sampled 2–3 yr previously, ranged from 90 to 125 pg (Beck et al., 1989; Birmingham et al., 1989; Theelen, 1991; Ministry of Agriculture, Fisheries, and Food, 1999). In our previous report on U.S. food from the same time, TEQ intake with an average diet was estimated to be 18 to 192 pg/d for adults (Schecter et al., 1994b). Applying the I-TEFs for dioxins and dibenzofurans used in previous studies to results in the present study, mean daily TEQ intake for adults is estimated to be 93 pg, within the range of previous studies. The revised WHO TEFs yield a 14% higher value, 107 pg.

By way of comparison, a Dutch study in 1997 reported a median 135 pg/d TEQ intake for the Dutch population, all ages, based on analyses of foods for dioxins, dibenzofurans, and coplanar PCBs, but not mono-*ortho* PCBs. Its authors used I-TEFs for dioxins and dibenzofurans and the 1994 WHO TEFs for coplanar PCBs to calculate TEQ, and counted nondetected congeners at half the level of detection. PCDDs and PCDFs contributed 65 pg/d to median Dutch population consumption of TEQ, and 70 pg/d were from coplanar PCBs (Liem & Theelen, 1997). Although estimated medians for an entire population cannot be compared directly to means for age groups, it may be noted that our estimated 93 pg/d mean TEQ intake from PCDDs and PCDFs for adults, using I-TEFs, or even 87 pg/d mean TEQ intake for ages 1–11 yr, is higher than the estimated Dutch median intake. American TEQ intake from consumption of coplanar and mono-*ortho* PCBs calculated with the 1994 TEFs would be 38 pg/d for adults, or 35 pg/d for ages 1–11 yr, considerably lower than the Dutch median for coplanar PCBs only.

Consumption of fish is lower in the United States than in some countries, such as Norway, where fish consumption accounts for a larger share of average daily TEQ intake (Becher et al., 1998). The higher lipid content found here in our freshwater fish, 4.8% compared to 1.4% in ocean fish, may account for some of the difference between the types of fish in wet weight dioxin, dibenzofuran, and PCB concentrations. Another factor may be that the freshwater fish analyzed in this study were raised on fish farms, where animal food is commonly used. The inclusion of dioxinlike

PCBs in the analyses for this study may also have increased the vegetable/fruit dioxin TEQ contribution.

The DDT metabolite DDE persists in the U.S. food supply, despite prohibition of DDT in agriculture since 1972. Levels found in the present study are consistent with those reported previously for fast foods. The level found for ice cream, 2780 ppt, is the same. The level of DDE found in chicken in this study, 149 ppt, is only slightly less than the level previously found in Kentucky Fried Chicken, 180 ppt. Beef in the present study was found to have 433 ppt DDE, far less than the 3170 ppt reported previously for a McDonald's Big Mac (Schecter & Lingjun, 1997).

Agreement has not been reached on a "virtually safe," "acceptable daily intake" (A.D.I.), or "risk-specific" daily intake of dioxin TEQ. The standard provisionally adopted by the World Health Organization was 10 pg/kg body weight/d TEQ, and is now 1 to 4 pg/kg body weight/d (Van Leeuwen & Younes, 1998). A target exposure level of 1 pg/kg body weight/d TEQ for the population has been set in Germany and the Netherlands (Liem & Theelen, 1997). The U.S. Environmental Protection Agency has used a level of 0.006 pg/kg/d in the past, and is considering 0.01 pg/kg/d as a new "risk-specific" dose based on one additional cancer case per 1,000,000 people over a 70-yr lifetime (Kociba, 1991; U.S. EPA Dioxin Reassessment Drafts, 1994–1998).

Emissions of these toxic contaminants are believed to have been reduced in some industrialized countries, and there are suggestive data indicating that background levels in human blood and milk in Germany, the Netherlands, and the United States may have declined recently (Schecter & Päpke 1998; Patandin et al., 1997; Schecter et al., 1997, 1989; Päpke, 1998). The efforts of environmental agencies to set and enforce regulations to decrease dioxin formation and spread into the environment should further reduce food contamination. Generally, however, government regulations and enforcement of standards for dioxin levels in food appear not to be in effect in the United States.

Given both the uncertainty about the long-term health effects of various levels of exposure to dioxinlike chemicals and the progress apparently made so far in some European countries in reducing levels of exposure, it seems reasonable to continue periodic surveillance of populations' exposures to dioxins through the food supply.

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