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Levels of Non-*ortho*-Subsituted (Coplanar), Mono- and Di-*ortho*-Substituted Polychlorinated Biphenyls, Dibenzo-*p*-Dioxins, and Dibenzofurans in Human Serum and Adipose Tissue

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We have measured non-*ortho*-substituted (coplanar) polychlorinated biphenyl (PCB) levels as well as polychlorinated dibenzo-*p*-dioxin (PCDD) and polychlorinated dibenzofuran (PCDF) levels in human adipose tissue and serum collected in Atlanta, Georgia. The results show that the concentrations of the coplanar PCBs can be more than an order of magnitude higher than the concentrations of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin. Our measurements in pooled serum collected in 1982, 1988, and 1989 show a decrease in coplanar PCB levels from 1982 to 1989. We found that the pattern of relative amounts of coplanar PCBs in adipose tissue varied greatly from person to person unlike the PCDD and PCDF patterns, which were more nearly the same. Age was significantly correlated with the concentrations of 2,3,7,8-TCDD,3,3′4,4′-PCB, 3,3′,4,4′,5-PCB, and 3,3′4,4′,5,5′-PCB in adipose tissue. We also measured levels of the mono- and di-*ortho* chlorine-substituted PCBs in human serum. The levels for some of these PCB congeners were three orders of magnitude higher than the coplanar PCBs, PCDDs, and PCDFs. We used the international toxicity equivalency factors (TEFs) for PCDDs and PCDFs and the TEFs proposed by Safe for PCBs to calculate the 2,3,7,8-TCDD equivalents. Four PCBs (3,3′,4,4′,5-; 2,3′,4,4′,5-; 2,3,3′,4,4′-; 2,3,3′,4,4′,5-) make a larger contribution than 2,3,7,8-TCDD, while four other PCBs (3,3′,4,4′5,5′-; 2,2′,3,4,4′,5′-; 2,2′,4,4′,5,5′-; 2,2′,3,4,4′,5,5′-) make nearly the same contribution as 2,3,7,8-TCDD. The mono-*ortho*-chlorine-substituted 2,3′,4,4′,5-PCB, however, is the major contributor to the total 2,3,7,8-TCDD equivalents in general population samples from the United States, Sweden, and Japan. The PCDDs are the second most significant contributor in the U.S. samples, whereas the coplanar PCBs are the second most significant contributor in samples from Sweden and Japan.

Introduction

Polychlorinated biphenyl (PCBs) were produced in the United States under the trade name Aroclor and in Japan under the name Kanechlor. A variety of Aroclor and Kanechlor mixtures, which contain varying amounts of the 209 possible PCB congeners, were produced. Because of the widespread use of these mixtures, varying amounts of individual PCB congeners have been found in almost every area of the global ecosystem (*1,2*). The recent development of isomer-specific analytical methods (*3*) for PCBs has allowed researchers to examine PCB mixtures for the non-*ortho*-chlorine-substituted biphenyls (coplanar PCBs) that are clearly the most toxic members of this class of compounds (*4*).

A large part of Aroclor toxicity in animals has been associated with the coplanar PCBs, which are present in the mixtures in small amounts (*3*). Investigators have reported (*5–7*) that 3,3′,4′-tetrachlorobiphenyl [IUPAC number PCB-77 (*8*)], 3,3′, 4,4′5-pentachlorobiphenyl (PCB-126), and 3,3′,4,4′,5,5′-hexachlorobiphenyl (PCB-169) produce the following toxic and biological effects in some species of animals: body weight loss, thymic atrophy, dermal disorder, hepatic damage, teratogenicity, reproductive toxicity, immunotoxicity, high binding affinity to hepatic cytosolic receptor protein (Ah receptor), and high induction potency of 3-methylcholanthrene-type (3-MC-type) hepatic microsomal enzymes. A high level of induction of 3-MC-type hepatic drug metabolizing enzymes in rat hepatoma cell lines (*9*)

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has also been attributed to mono-*ortho*-chlorine-substituted PCBs 2,3,3',4,4'-(PCB-105), 2,3',4,4',5-(PCB-118), and 2,3,3',4,4',5-(PCB-156).

Kannan et al. (*3*) found varying amounts of the coplanar PCBs in Aroclor and Kanechlor mixtures (Table 1). The highest concentration in each mixture was PCB-77, followed by PCB-126 and trace amounts of PCB-169. Japanese researchers have since found these PCBs in terrestrial and marine animals such as dogs, cats, fish, porpoises, dolphins, and whales (*10–12*). Examples of the levels reported (*13–19*) are given in Table 2. In general, the same relative order of concentrations of the coplanar PCBs as are found in Aroclor and Kanechlor mixtures are found in these animals (PCB-77 > 126 > 169).

Tanabe et al. (*10*) have found clear positive correlations between concentrations of total PCBs and each of the three coplanar PCBs in all mammal samples analyzed (r=0.81–0.96 over a concentration range of five orders of magnitude). These authors concluded that the sources of coplanar PCB contamination to the environment are principally from commercial PCB preparations. Tanabe et al. (*10*) also reported that in fish samples, 95% of the coplanar PCBs were PCB-77, 5% were PCB-126, and less than 1% were PCB-169 (this pattern is the same as in commercial PCB mixtures). Marine and terrestrial mammals, on the other hand, had a lower percentage of PCB-77, and the bioconcentration of PCBs-126 and 169 was obvious. The authors suggested that the concentrations of coplanar PCBs in humans and animals suggest that the order of biodegradability is PCB-77 > PCB-126 > PCB-169 (*10*). This order of metabolic stability is consistent with findings in studies of animals (*20,21*) and humans (*22*).

Recent studies by Tanabe et al. (*10,11,14,,23,24*), Kannan et al. (*12,25*), Miyata et al. (*26*), and Kashimoto et al. (*27*) have found levels of PCB-77, PCB-126, and PCB-169 up to several orders of magnitude higher than the levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) in human adipose tissue samples from Japan. Noren et al. (*28*) reported high levels of these congeners in pooled mothers' milk from Sweden; Patterson et al. found them in pooled serum (*29*) and adipose tissue (*30*) from the United States, and Beck et al. (*31*) reported finding PCB-77 in adipose tissue and mothers' milk from Germany. We have examined additional human adipose tissues and serum from Atlanta, Georgia, to determine the distribution of the coplanar PCBs, PCDDs, and PCDFs in these two lipid stores of the human body.

Table 1. Coplanar PCB concentrations (ppm) in Aroclor and Kanechlor mixtures reported by Kannan et al. (*3*).

	PCB-77(%)[a]	PCB-126	PCB-169
Aroclor 1242	5150 (0.5)	19.9	ND
1248	6060 (0.6)	62.3	ND
1254	601 (0.06)	46.0	0.66
1260	256 (0.03)	8.4	ND
Kanechlor 300	4440 (0.44)	18.9	0.09
400	8500 (0.85)	89.3	0.57
500	1530 (0.15)	50.0	1.16
600	738 (0.07)	8.6	Trace

ND, not detected.
[a] Percentage of the PCB mixture.

Table 2. Reported coplaner PCB levels (ppt)

	PCB-77	PCB-126	PCB-169	Reference
Lake Michigan lake trout	3,500	1,500	NR	(*13*)
Killer whale	48,000	3,700	7,700	(*14*)
Baltic herring	2,000–4,000	1,000–3,000	100–400	(*15*)
Baltic herring and cod liver oil	NR	20-50	NR	(*16*)
Baltic (Finnish) fish	70-900	30–300	NR	(*17*)
Pike muscle (Lake Kyrksjon)	77,000	6,500	420	(*18*)
Striped bass (New York)	2-68,000	700–7,000	ND	(*19*)

Abbreviations: NR, not reported; ND, not detected.

Experimental

The laboratory procedures we used to analyze polychlorinated dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), and PCBs in adipose tissue and serum have been previously described in detail (*29,32–37*) and only a brief description will be given here.

Sample Cleanup and Quantification

We spiked the adipose tissue (*32,33,36*) or serum (*29,34,35,37*) samples with carbon-13-labeled PCDDs, PCDFs, and coplanar PCBs and in a separate analysis with carbon-13-labeled nonplanar PCBs. The separate analysis eliminated a previous problem with high recoveries of carbon-13-labeled PCB-77 (*29*). The samples were then extracted with organic solvents, and the solvent extracts were processed through a five-column cleanup procedure developed by Smith et al. (*38*) and modified and semiautomated (*33,39*) by us for PCDD, PCDF, and PCB analyses. The final extracts were quantified by high-resolution gas chromatography/isotope-dilution high-resolution mass spectrometry for the PCDDs and PCDFs and by high-resolution gas chromatography/isotope-dilution high-and low-resolution mass spectrometry for the PCBs.

Sample Procurement

Pooled Human Serum. Serum collected by the Centers for Disease Control (CDC) blood bank in Atlanta, Georgia, was pooled and aliquotted into 100-g samples and stored at -60°C until analysis.

Collection of Adipose Tissues. We collected adipose tissue samples at autopsy from the abdominal wall of men and women who died suddenly in Atlanta, Georgia, in 1984 or 1986. The samples were taken according to a specific protocol designed to ensure that the samples were not cross-contaminated or laboratory contaminated. The dead persons were from the general population in Atlanta, Georgia.

Results and Discussion

The levels that we measured for coplanar PCB-77, PCB-126, PCB-169, 3,4,4'5,-PCB (PCB-81), mono- and di-*ortho*-PCBs, PCDDs, and PCDFs in pooled human serum and in adipose tissues collected in Atlanta, Georgia, are given in Tables 3–5. We measured 2,3,7,8-TCDD and coplanar PCB levels in 28 adipose tissue samples collected either in 1984 or 1986. The results given in Table 3 indicate that the concentrations of the coplanar PCBs

Table 3. Measured levels[a] of 2,3,7,8-TCDD and non-*ortho* substituted PCBs in human adipose tissue collected from the general population in Atlanta, Georgia.

Person no.	Age	Race	Sex	Sample date	2,3,7,8-TCDD	PCB-77	PCB-126	PCB-169	PCB-81
1	39	B	F	1984	3.1	2.8	66.7	36.7	NM
2	68	B	F	1984	22.2	11.7	371	174	NM
3	57	W	F	1984	12.5	11.9	299	56.7	NM
4	35	W	F	1984	9.0	9.6	139	44.5	NM
5	76	B	F	1986	38.0	16.7	263	118	NM
6	35	W	F	1986	4.6	4.3	22.0	48.2	NM
7	55	W	F	1986	8.3	20.8	106	96.5	NM
8	30	W	F	1986	9.2	NR	70.3	91.0	NM
9	48	W	M	1984	3.0	3.0	14.6	34.2	NM
10	69	W	M	1984	8.5	ND	103	154	NM
11	19	B	M	1986	3.0	9.4	33.7	69.9	NM
12	25	B	M	1986	1.6	6.4	25.8	62.2	NM
13	28	W	M	1986	2.6	4.0	18.0	34.5	NM
14	27	W	M	1986	3.2	2.6	39.4	41.8	NM
15	51	B	M	1986	6.8	7.8	61.5	112	NM
16	71	W	M	1984	19.4	11.3	121	66.4	10.9
17	75	B	F	1984	13.6	23.6	170	42.0	10.0
18	19	B	M	1984	10.2	5.7	52.7	67.7	1.5
19	67	B	F	1984	13.2	19.1	149	59.5	8.0
20	56	B	F	1984	9.4	11.3	201	36.4	13.5
21	67	B	M	1984	8.4	27.9	190	62.4	21.3
22	78	B	F	1984	24.3	9.1	146	109	8.9
23	63	W	F	1984	9.2	15.9	159	29.5	13.3
24	46	W	F	1984	11.6	25.0	195	49.0	19.3
25	53	B	M	1984	13.8	19.4	206	84.0	7.2
26	28	B	M	1984	10.6	19.3	192	35.6	13.6
27	48	B	M	1984	7.8	10.9	240	61.6	6.5
28	45	W	M	1984	5.3	6.9	128	54.7	2.6
Men	Mean concentration				7.4	9.6	102	67.1	9.1
	Range				1.6–	ND–	14.6–	34.2–	1.5–
	Mean age, 42.7 years				19.4	27.9	240	154	21.3
Women[b]	Mean concentration				11.6	13.8	161	67.1	12.2
	Range				3.1–	2.8–	22.0–	29.5–	8.0–
	Mean age, 54.1 years				24.3	25.0	371	1748	19.3
Women[c]	Mean concentration				13.4	14.0	168	70.8	12.2
	Range				3.1–	2.8–	22.0–	29.5–	8.0–
	Mean age, 55.7 years				38.0	25.0	371	174	19.3
All Data	Mean concentration				10.4	11.7	135	69.0	10.5
	Range				1.6–	ND–	14.6–	29.5–	1.5–
	Mean age, 49.2 years				38.0	27.9	371	174	21.3

Abbreviations B, black; W, white; F, female; M, male; NM, not measured; ND, not detected; NR, not reported.

[a]Levels are in parts-per-trillion on a lipid-adjusted bases.

[b]Excluding person 5, whose levels were obviously above normal U.S. background levels (*40*)

[c] Including person 5.

Table 4. Measured levels[a] of PCDDs, PCDFs, and non-*ortho* substituted PCBs in human adipose tissue collected from the general population in Atlanta, Georgia.

	Person no.[b]					
Compound	5	6	7	11	12	Mean[c]
2,3,7,8-TCDD	38.0	4.6	8.3	3.0	1.6	4.4
1,2,3,7,8-PeCDD	38	8.5	12	16	10	11.6
1,2,3,4,7,8-HxCDD	18	3.7	5.3	NR	6.4	5.1
1,2,3,6,7,8-HxCDD	246	84	91	121	80.9	94.2
1,2,3,7,8,9-HxCDD	36	15	18	22	12.5	16.9
1,2,3,4,6,7,8-HpCDD	335	NR	48	NR	63.2	55.6
OCDD	2050	NR	396	NR	495	446
2,3,7,8-TCDF	0.8	0.7	1.8	1.1	0.7	1.1
2,3,4,7,8-PeCDF	13	3.3	4.3	3.9	3.4	3.7
1,2,3,,4,7,8-HxCDF	12	3.4	3.2	4.1	3.9	3.7
1,2,3,6,7,8-HxCDF	24	4.0	5.8	8.3	5.1	5.8
1,2,3,4,6,7,8-HpCDF	47.3	NR	8.9	NR	15.2	12.0
3,3',4,4'-PCB	16.7	4.3	20.8	9.4	6.4	10.2
3,3',4,4',5'-PCB	263	22.0	106	33.7	25.8	46.9
3,3',4,4',5,5'-PCB	118	48.2	96.5	69.9	62.2	69.2

Abbreviations: CDD, chlorinated dibenzodioxin; CDF, chlorinated dibenzofuran; T, tetra; Pe, penta; Hx, hexa; Hp, hepta; O, Octa; NR, not reported.

[a]Levels are in parts-per-trillion on a lipid-adjusted basis.

[b]These persons are the same as those descibed in Table 3.

[c]This mean concentration excludes person 5, whose levels were above normal U.S. background levels (*40*).

can be more than an order of magnitude higher than the concentrations of 2,3,7,8-TCDD in human adipose tissue samples from the general population. In addition to measuring the coplanar PCBs, we measured the levels of all the PCDDs and PCDFs (Table 4) in the tissue of 5 of the 15 persons whose data are in Table 3. The mean levels of the PCDDs, PCDFs, and coplanar PCBs for these five are in Table 4. Data on subject 5 were excluded from the calculation of the means since her levels were obviously elevated above normal U.S. background levels (*40*). We also measured PCB levels in pooled serum that was collected in Atlanta, Georgia, (Table 5). The coplanar PCB levels were similar in the 1988 and 1989 pooled samples, whereas the level of PCB-77 was three to six times higher in the 1982 pooled sample. The levels of PCB-126 and 169 were about twice as high in the 1982 pooled sample. These data are consistent with a decrease in human exposure to PCBs and a shorter half-life in humans for PCB-77 than for PCBs-126 and 169.

The only data reported in the scientific literature on levels of PCDDs, PCDFs, and PCBs with which we can compare our results are given in Table 6. These data should be compared with caution, however, because the U.S. (*29*), Japanese (*10,25–27*), and Swedish (*28,41*) samples represent people with different background exposures and were not collected to represent a certain population group. In addition, individual PCB congeners (*42,43*) as well as total PCBs (*44–46*) have been shown to be positively associated with age; differences between the sexes have also been noted (*43,46*). The studies in Table 6 represent cohorts with different mean ages and varying male-to-female ratios.

Toxic equivalency factors (TEFs) have been developed for the various congeners of PCDDs, PCDFs, and PCBs (*4,47*). The TEF scheme compares the *in vivo* and *in vitro* responses of some species of animals to various congeners relative to that of 2,3,7,8-TCDD (Table 7). In deriving the TEFs (*44*), data from the following effects were used in descending order of priority: results of long-term carcinogenicity studies; data from reproductive studies; results of subchronic experiments that measure Ah receptor-mediated responses such as thymic atrophy, body weight loss, and immunotoxicity; acute toxicity studies; and *in vivo* or *in vitro* biochemical responses such as enzyme induction or receptor binding (*4*). A complete set of data that included long-term carcinogenicity studies was available only for 2,3,7,8-TCDD, and therefore the TEFs assigned to other congeners were subjective assessments made by using the above priorities as a guide (*4*).

The 2,3,7,8-TCDD equivalents have been calculated for data in all studies available for comparison and are given in Table 6 along with the percentage of the total 2,3,7,8-TCDD equivalents for PCDDs, PCDFs, and coplanar PCBs. Figure 1 is a comparison of studies that measured all three compound classes in each

Table 5. Measured levels of 2,3,7,8-TCDD, non-*ortho*, mono-*ortho*, and di-*ortho*PCBs in pooled human serum.[a]

	Serum pool 1[b] (100 g samples)			Serum pool 2[d] (50 g samples)			Serum pool 3[e] (10 g samples)		
	n[c]	Mean ± SD	CV	n[c]	Mean ± SD	CV	n	Mean ± SD	CV
2,3,7,8-TCDD	101	0.159 ± 0.0241	15.1	84	0.0165 ± 0.0027	16.7	—	NM	—
3,3',4,4'(77)	3	0.481 ± 0.0762	15.8	6	0.251 ±0.0453	18	3	1.38 ± 0.15	10.8
3,3',4,4',5(126)	3	0.183 ± 0.0203	11.1	6	0.135 ± 0.0233	17	3	0.281 ± 0.0577	19.5
3,3',4,4',5,5'(169)	3	0.151 ± 0.0103	6.8	6	0.192 ± 0.0491	25.5	2	0.282	—
2,3,3',4,4'(105)	4	33.2 ± 19.2	5.3	—	NM	—	—	NM	—
2,3',4,4',5(118)	4	366 ± 1.8	5.4	—	NM	—	—	NM	—
2,3,3',4,4',5(156)	—	NM	—	—	NM	—	—	NM	—
2,2',3,4,4',5'(138)	4	583 ± 33.0	5.7	—	NM	—	—	NM	—
2,2',4,4',5,5'(153)	4	690 ± 60.4	8.7	—	NM	—	—	NM	—
2,2',3,4,4',5,5'(180)	4	466 ± 24.0	5.2	—	NM	—	—	NM	—
Total PCBs	4	3,100 ± 270	8.7	—	NM	—	—	NM	—

Abbreviations: NM, not measured; CV, coefficient of variability.
[a]Levels in parts per trillion on a whole-weight basis.
[b]Pooled human serum (total lipid = 475.3 mg/dL) collected in 1988 from more than 240 donors. This pool has been spiked to a higher level with 2,3,7,8-TCDD (see Table 6 for levels on a lipid-adjusted basis).
[c]The number of repeat analyses on the same pool.
[d]This pool is of unspiked, normal human serum collected in 1989 from more than 200 donors.
[e]This pool is of unspiked, normal human serum collected in 1982 from more than 200 donors.

Table 6. Reported levels and 2,3,7,8-TCDD equivalents of PCDDs, PCDFs, and PCBs in human adipose tissue, mothers' milk, and serum.[a]

	Japan			Sweden		USA		
	Adipose tissue, wet/weight (*10,25*)[b]	Adipose tissue, wet/weight (*26*)[c]	Adipose tissue, lipid adjusted (*27*)[d]	Breast milk, lipid adjusted (*28*)[e]	Breast milk, lipid adjusted (*41*)[f]	Serum, lipid adjusted (*29*)[g]	Adipose tissue, lipid adjusted (this study)[h]	Adipose tissue, lipid adjusted[i]
2,3,7,8-TCDD	9.6 (9.6)	1.2 (1.2)	5.5 (5.5)	3 (3)	1.9 (1.9)	3.6 (3.6)	4.4 (4.4)	10.4 (10.4)
1,2,3,7,8-PeCDD	15.9 (7.95)	11.9 (5.95)	12.3 (6.15)	7 (3.5)	3.4 (1.7)	NR	11.6 (5.8)	NR
1,2,3,4,7,8-HxCDD	8.1 (0.81)	—[j]	—[j]	—[j]	—[j]	NR	5.1 (0.51)	NR
1,2,3,6,7,8-HxCDD	NR	68 (6.8)[j]	79.5 (7.95)[j]	38 (3.8)[j]	18.5 (1.85)[j]	NR	94.2 (9.42)[j]	NR
1,2,3,7,8,9-HxCDD	NR	—[j]	—[j]	—[j]	4.2 (0.42)	NR	16.9 (1.69)	NR
1,2,3,4,6,7,8-HpCDD	NR	28.5 (0.28)	62.1 (0.62)	57 (0.57)	37.3 (0.37)	NR	55.6 (0.56)	NR
OCDD	251 (0.25)	830 (0.83)	1,327 (1.33)	268 (0.27)	173 (0.173)	NR	446 (0.45)	NR
Total	285 (18.6)	940 (15.1)	1,486 (21.6)	373 (11.1)	238 (6.4)	—	634 (22.8)	—
% Total of TEq[k]	24		13.8	33.7	22.5	—	66.9	—
2,3,7,8-TCDF	9.4 (0.94)	NR	3.6 (0.36)	2 (0.2)	1.3 (0.13)	NR	1.1 (0.11)	NR
2,3,4,7,8-PeCDF	29.1 (14.6)	NR	23.2 (11.6)	17 (8.5)	7.1 (3.55)	NR	3.7 (1.85)	NR
1,2,3,4,7,8-HxCDF	14.9 (1.49)	NR	j	j	2.8 (0.28)	NR	3.7 (0.37)	NR
1,2,3,6,7,8-HxCDF	14.9 (1.49)	NR	26.3 (2.63)[g]	7 (0.7)[j]	2.2 (0.22)	NR	5.8 (0.58)	NR
1,2,3,4,6,7,8-HpCDF	NR	NR	11.3 (0.11)	8 (0.08)	4.3 (0.043)	NR	12.0 (0.12)	NR
OCDF	NR	NR	5.8 (0.01)	2 (0.002)	1.7 (0.002)	NR	NR	NR
Total	68.3 (18.6)	—	70.2 (14.7)	36 (9.5)	19.4 (4.2)	—	26.3 (3.03)	—
% Total of TEq[k]	24	—	9.4	28.7	14.8	—	8.9	—
PCB 77	348 (3.48)	39 (0.39)	173 (1.73)	27 (0.27)	20.7 (0.21)	104 (1.04)	10.2 (0.10)	11.7 (0.12)
PCB 126	324 (32.4)	250 (25)	1,002 (100.2)	98 (9.8)	152 (15.2)	39.5 (3.95)	46.9 (4.69)	135 (13.5)
PCB 169	89.5 (4.48)	132 (6.6)	356 (17.8)	47 (2.35)	49.2 (2.46)	32.6 (1.63)	69.2 (3.46)	69.0 (3.45)
Total	762 (40.4)	421 (32)	1,531 (120)	172 (12.4)	222 (17.9)	176 (6.6)	126 (8.25)	181 (13.6)
% Total of TEq[k]	52	—	76.7	37.6	62.7	—	24.2	—
PCB 118 (ppb)	91 (91)	NR	NR	25.4 (25.4)	NR	79 (79)	NR	NR
PCB 105 (ppb)	25 (25)	NR	NR	6.5 (6.5)	NR	7.2 (7.2)	NR	NR
PCB 156 (ppb)	20 (20)	NR	NR	14.3 (14.3)	NR	NR	NR	NR
PCB 138 (ppb)	200 (4)	NR	NR	116 (2.32)	NR	126 (2.52)	NR	NR
PCB 153 (ppb)	NR	NR	NR	151 (3.02)	NR	149 (2.98)	NR	NR
PCB 180 (ppb)	NR	NR	NR	64.4 (1.29)	NR	101 (2.02)	NR	NR

Abbreviations: CDD, chlorinated dibenzodioxin; CDF, chlorinated dibenzofuran; T, tetra; Pe, penta, Hx, hexa; Hp, hepta; O, Octa; NR, not reported.
[a]Levels in parts per trillion [values in parenthesis are 2,3,7,8-TCDD equivalents (*4,47*) see Table 7].
[b]Tissue collected in 1985 from 12 cancer patients in Matsuyama, Japan.
[c]Tissues collected in 1986 from two persons who died suddenly in Osaka City, Japan.
[d]Tissues collected in 1986–1987 from 39 persons who died suddenly in Osaka, Nara, and Okinawa prefectures in Japan.
[e]Pooled milk collected in 1988–1989 from Stockholm, Sweden.
[f]Pooled breast milk collected in 1988 from Sweden.
[g]Pooled serum collected in 1988 from Atlanta, Georgia (see Table 5).
[h]Data from the present study. Tissues collected in 1986 from four persons who died suddenly in Atlanta, Georgia (data from Table 4).
[i]Tissues collected in 1984 and 1986 in 28 persons who died suddenly in Atlanta, Georgia (data from Table 3).
[j]These congeners reported as a sum of HxCDDs or HxCDFs.
[k]Percent of the total 2378-TCDD equivalents for PCDDs, PCDFs, and coplaner PCBs.

Table 7. Toxicity equivalency factors (TEFs) for PCDDs (47), PCDFs (47), and PCBs (4).

Congener	TEF
2,3,7,8-TCDD	1.0
1,2,3,7,8-PeCDD	0.5
1,2,3,4,7,8-HxCDD	0.1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.001
2,3,7,8-TCDF	0.1
2,3,4,7,8-PeCDF	0.5
1,2,3,7,8-PeCDF	0.05[a]
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[a]
1,2,3,4,7,8,9-HpCDF	0.01[a]
OCDF	0.001
3,3',4,4'-PCB (77)	0.01
3,3',4,4',5-PCB (126)	0.1
3,3'4,4',5,5'-PCB (169)	0.05
Mono-*ortho*-substituted PCBs	0.001[b]
Di-*ortho*-substituted PCBs	0.00002[c]

Abbreviations: CDD, chlorinated dibenzodioxin; CDF, chlorinated dibenzofuran; T, tetra; Pe, penta; Hx, hexa; Hp, hepta; O, octa.

[a]Safe has proposed 0.1 (4).

[b]A highly conservative value according to Safe (4).

[c]Recommended as a preliminary value until more data can be acquired for these PCBs (4).

sample for each person. The reported levels in adipose tissue collected in 1985 and 1986 from Japan show that most of the 2,3,7,8-TCDD equivalents are due to the coplanar PCBs. Pooled mothers' milk collected by Noren (28) in 1988 and 1989 in Stockholm, Sweden, has nearly equal percentages of 2,3,7,8-TCDD equivalents due to PCDDs, PCDFs, and coplanar PCBs. The majority of the 2,3,7,8-TCDD equivalents in adipose tissue collected in 1986 from Atlanta, Georgia, are due to the PCDDs (67%), whereas the coplanar PCBs account for the second highest percentage (24%). Measurements in pooled mother's milk by Thornburg (21) collected in 1988 from Sweden indicate that 63% of the 2,3,7,8-TCDD equivalents are due to the coplanar PCBs. The differences between our results in adipose tissue (Figure 1) and the results from the studies in Sweden and Japan could be due to several factors: *a*) different matrixes; *b*) different mean ages; *c*) different sample collection years from different countries [the data of Patterson et al. (48) and Stanley et al. (49) indicate that PCDD and PCDF levels may be decreasing in U.S. serum and adipose tissue, and the data of Noren et al. (28) show that levels of PCDDs, PCDFs, and coplanar PCBs decreased from 1972 to 1985 in pooled mothers' milk in Sweden]; *d*) our adipose tissue levels were measured in men and women, but the mothers' milk levels were measured in women who may have been breastfeeding infants when the samples were collected [breastfeeding may have altered the normal PCDD, PCDF, and PCB patterns]; and *e*) different background exposures to PCDDs, PCDFs, and PCBs in the United States, Japan, and Sweden.

The levels and 2,3,7,8-TCDD equivalents of 2,3,7,8-TCDD and coplanar PCBs that we found in the adipose tissue of men and women from Atlanta, Georgia, are compared in Table 8 to those reported by Tanabe (24) and Kanman (25). In the U.S. samples, women had higher levels of each analyte, whereas men had higher levels in the Japanese samples. The number of samples in both of these studies is, however, too small to make statistically significant statements concerning the observed differences.

The patterns of coplanar PCB levels in adipose tissue also appear to be different for men and women in the Atlanta samples

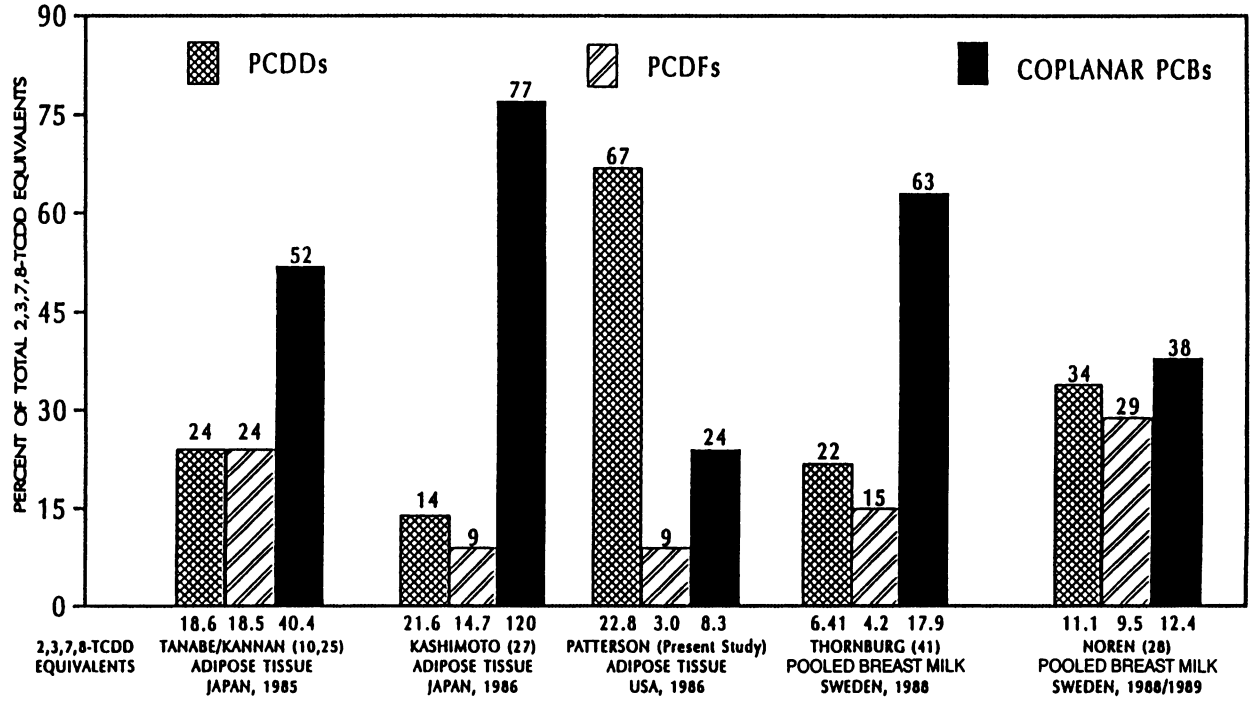

FIGURE 1. Percentage of total 2,3,7,8-TCDD equivalents for general population samples.

Table 8. Reported mean levels and 2,3,7,8-TCDD equivalents[a] of the 2,3,7,8-TCDD and coplanar PCBs in adipose tissue collected in 1985 in Matsuyama, Japan, and in 1986 in Atlanta, Georgia.

	Present study		Tanabe (*24*) and Kannan (*25*)	
	14 Men (mean age=43)	13 Women (mean age=54)	7 men (mean age=59)	5 Women (mean age=59)
2,3,7,8-TCDD	7.4 (7.4)	11.6 (11.6)	11 (11)	7 (7)
PCB-77	9.6 (0.096)	13.8 (0.14)	400 (4)	270 (2.7)
PCB-126	102 (10.2)	161 (16.1)	400 (40)	240 (24)
PCB-169	67.1 (3.35)	67.1 (3.35)	120 (6)	53 (2.6)

[a]Levels in parts per trillion. Values in parenthesis are 2,3,7,8-TCDD equivalents based on data in Safe (*4*) and NATO (*47*).

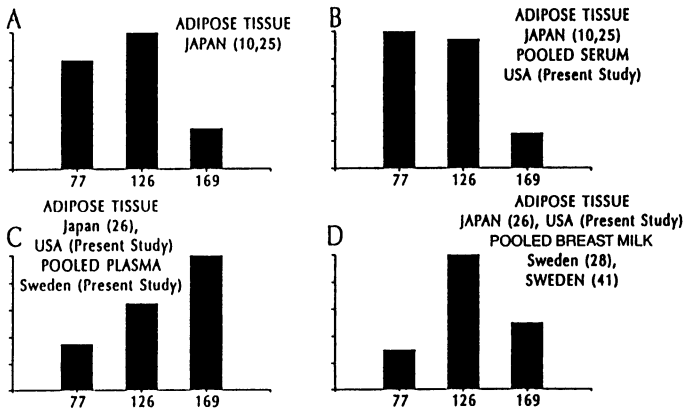

FIGURE 2. Coplanar PCB patterns in human samples.

(Table 3). The level of PCB-126 is the highest of the three coplanar PCBs in 12 of the 14 women and PCB-169 is the highest in 8 of the 14 men (the number of individual samples is too small to establish a definite pattern difference). Within a group of normal population samples, the reported pattern of PCDDs and PCDFs is ususally the same. The absolute levels may vary from person to person, but the relative amounts of the various congeners are generally the same (for example, see Table 4).

This similarity of pattern is not true for the coplanar PCBs. The various patterns of the coplanar PCBs that have been reported are shown in Figure 2. Tanabe and Kannan (*10,25*) found patterns *A* and *B* (Fig. 2) in adipose tissue from Matsuyama, Japan, but Miyata et al. (*26*) found patterns *C* and *D* (Fig. 2) in adipose tissue from Osaka City, Japan. We also found patterns *C* and *D* (Fig. 2) in adipose tissue and pattern *B* (Fig. 2) in serum from Atlanta, Georgia. We measured coplaner PCB levels in a pooled plasma sample that was collected from Sweden as part of a World Health Organization (WHO)-sponsored laboratory round-robin study of PCDDs, PCDFs, and PCBs (*50*). We observed pattern C (Fig. 2) in this pooled plasma sample. Pooled mothers' milk collected in Sweden (*28,41*) exhibited pattern D (Fig. 2). These variable patterns may be caused by different dietary habits (N. Kannan, personal communication), by different metabolic elimination rates, or by different routes of exposure.

We have calculated Pearson correlation coefficients and probability values (Table 9) for the coplaner PCB and 2378-TCDD data from Table 3. There are statistically significant correlations

Table 9. Pearson correlation coefficients between concentration of individual coplanar PCB congeners and 2,3,7,8-TCDD and age in adipose tissue obtained at autopsy from 28 persons from Atlanta, Georgia.

	2,3,7,8-TCDD	PCB-77	PCB-126	PCB-169	Age
2,3,7,8-TCDD	1.000	—	—	—	0.667 (0.0001)
PCB-77	0.365 (0.061)[a]	1.000	—	—	0.409 (0.034)
PCB-126	0.647 (0.0002)	0.540 (0.0037)	1.000	—	0.579 (0.0013)
PCB-169	0.511 (0.0054)	-0.054 (0.788)	0.334 (0.082)	1.000	0.411 (0.030)

[a]Probability values in parenthesis.

between the levels of 2,3,7,8-TCDD and PCB-126 and -169, as well as between PCB-77/126. Age was significantly correlated with concentration for 2,3,7,8-TCDD, PCB-77, PCB-126, and PCB-169 (Table 9). Luotamo et al. (*51*) found good correlations in paired serum and adipose tissue specimens only for the bioaccumulating, slowly metabolized PCB congeners. The lack of a good correlation between PCB-77/169 and PCB-126/169 in our study is consistent with Luotamo's findings. Our serum data (Table 5) for 1982 and 1988/1989 suggest a shorter half-life for PCB-77 relative to PCB-126 and 169 and is consistent with a study in mice that showed that PCB-126 was more slowly metabolized than PCB-77 (*52*). The bioconcentration of PCB-126 and 169 is also clearly seen by comparing the relative levels in the human exposure sources (Aroclors and Kanechlors, Table 1) to the relative amounts found in human tissues (Table 6).

In Figure 3, we compare the levels of mono- and di-*ortho*-substituted PCBs in pooled serum samples taken in Atlanta in 1988 (*29*) with the levels in serum samples taken in 1986 from 200 Wisconsin consumers of sport fish (*53*); serum samples taken in 1988 from 19 Columbia, Missouri, residents (*54*); pooled serum taken in 1990 from Finland (*55*); and pooled serum taken in 1990 from Quebec, Canada (*56*). The relative amounts of the various PCB congeners were similar in our pooled serum and the Columbia, Missouri, residents(*54*), as well as in pooled serum from Canada (*56*). The mean levels for the three major di-*ortho*-substituted PCBs [2,2',3,4,4',5'-(PCB-138); 2,2',4,4',5,5'-(PCB-153); 2,2'3,4,4',5,5'-(PCB-180)] and the major mono-*ortho*-substituted PCB-118 were higher by a factor of about two in the Wisconsin samples than in the Atlanta pooled serum. The differences in PCB levels may be due to the high fish consumption among persons who provided the Wisconsin samples (*53*). Fiore et al. (*57*) have found statistically significant positive Spearman correlations between sport-caught fish meals and PCB serum levels. Humphrey (*58*) has reported that, among Michigan residents, increased fish consumption correlated positively with increased PCB concentrations in serum.

Figure 4 shows the results of six studies with data on mothers' milk, adipose tissue, and lipid-adjusted serum levels of mono- and di-*ortho*-substituted PCBs. Although the absolute amounts of the individual PCB congeners vary among studies, the relative distribution patterns of the PCB congeners within each study are similar. In addition, the di-*ortho*-substituted PCB levels are higher than the mono-*ortho*-substituted levels, which are in turn higher than the coplanar PCB levels (Fig. 4, Table 6).

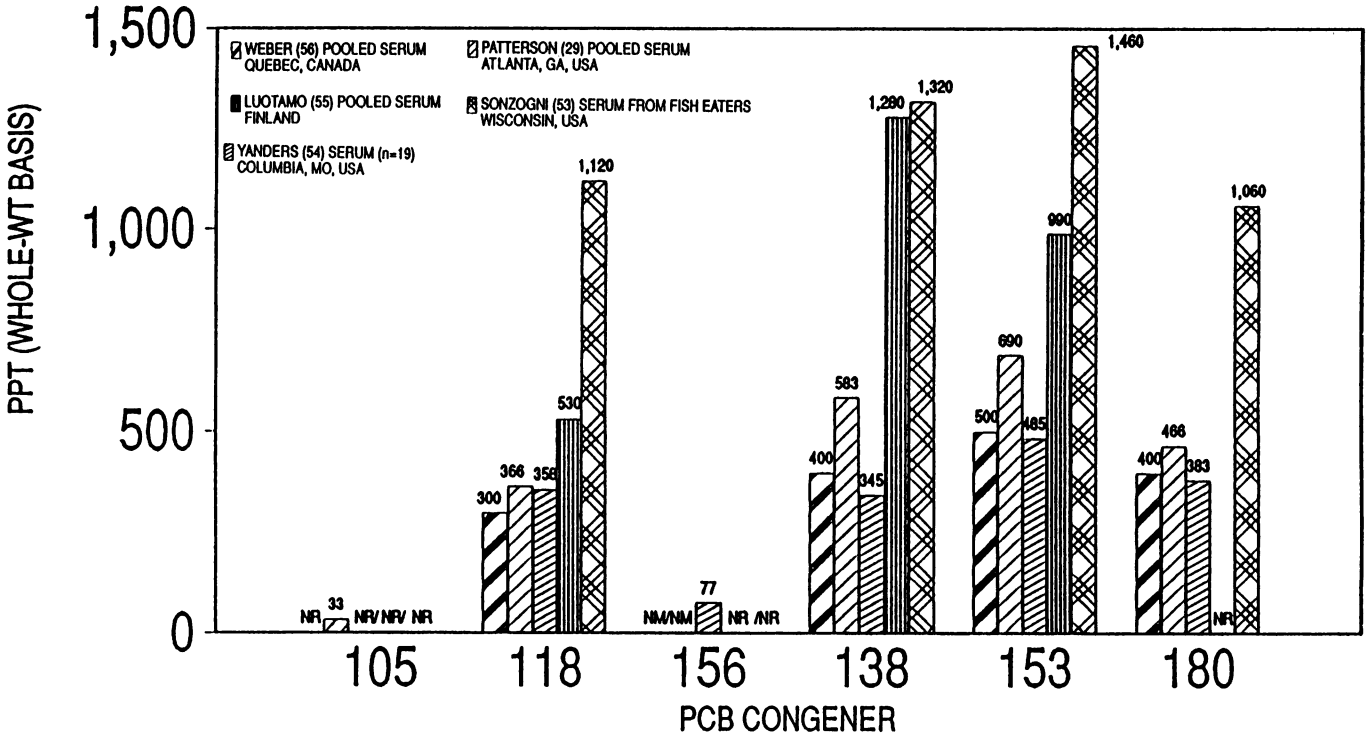

FIGURE 3. Mono- and di-*ortho*-substituted PCBs in human serum.

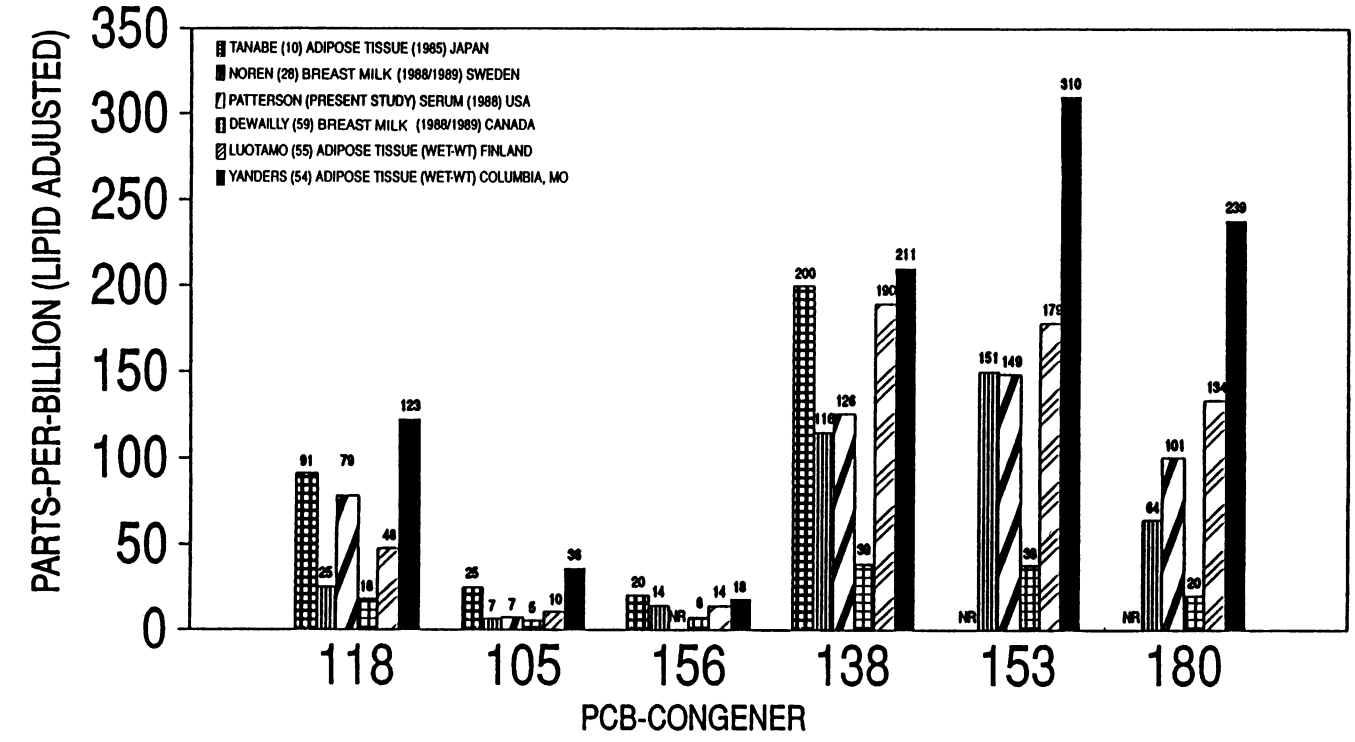

FIGURE 4. Mono-and di-*ortho*-substituted PCBs in human serum, breast milk, and adipose tissue from general population samples.

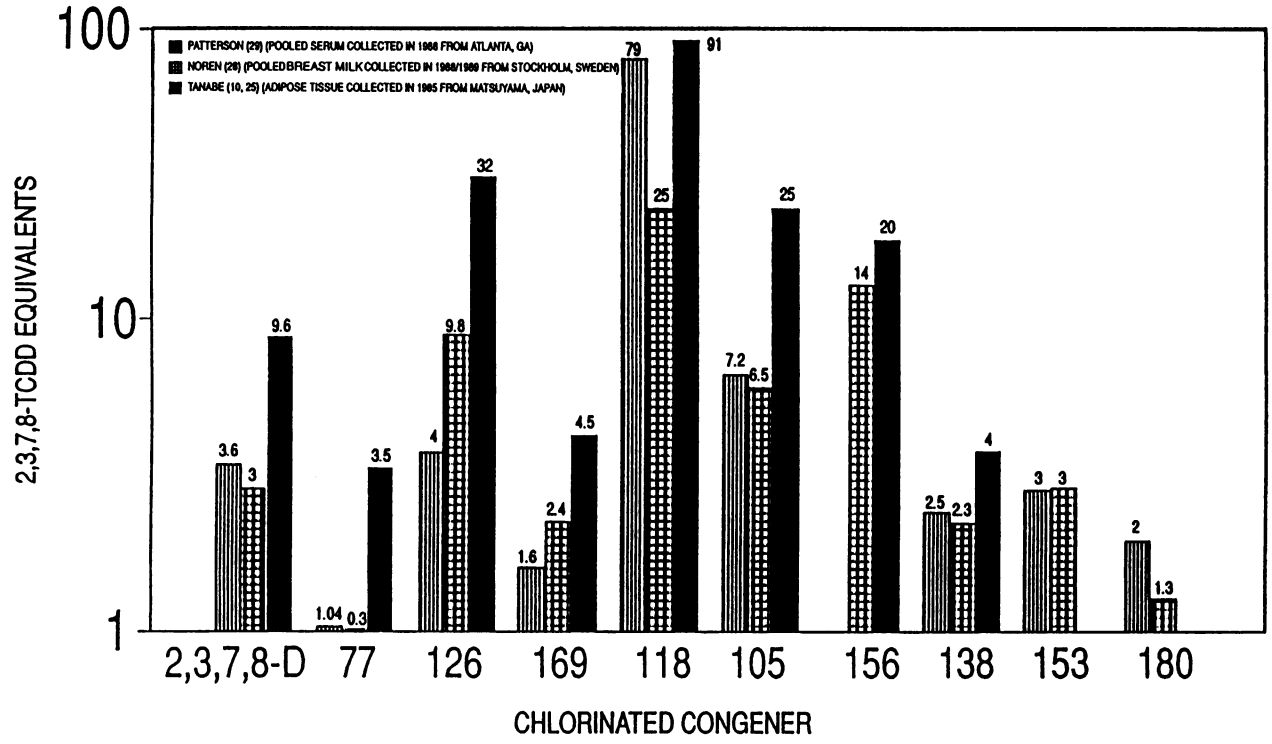

FIGURE 5. 2,3,7,8-TCDD equivalents for 2,3,7,8-TCDD, coplanar PCBs, and mono-and di-*ortho* substituted PCBs from general population samples.

Table 10. Total 2,3,7,8-TCDD equivalents in various countries for PCDDs, PCDFs, coplanar PCBs, and mono- and di-*ortho* substituted PCBs.

	United States (prensent study)	Sweden (*28*)	Japan (*10*)
PCBs-118,105,156	86.2 (68)[a]	46.2 (54)	136 (62)
PCBs-138,153,180	7.5 (6)	6.6 (8)	4 (2)
PCBs-77,126,169	6.6 (5)	12.4 (14)	40.4 (19)
PCDDs	22.8 (18)	11.1 (13)	18.6 (8)
PCDFs	3.0 (2)	9.5 (11)	18.6 (8)

[a]Percent of the total 2,3,7,8-TCDD equivalents in parentheses.

The 2,3,7,8-TCDD equivalents for 2,3,7,8-TCDD, coplanar PCBs, mono- and di-*ortho*-substituted PCBs from serum (*29*), mothers' milk (*28*), and adipose tissue (*10,25*) are compared in Figure 5. Clearly, each of the coplanar PCBs, and mono- and di-*ortho*-substituted PCBs makes a major contribution to the total 2,3,7,8-TCDD equivalents in these general population samples. In fact, PCBs-126, 118, 105, and 156 all make a larger contribution than 2,3,7,8-TCDD, while PCBs-169, 138, 153, and 180 make nearly the same contribution as 2,3,7,8-TCDD. The major contributors, however, to the total 2,3,7,8-TCDD equivalents in samples from the United States, Sweden, and Japan (Table 10) are the mono-*ortho*-chlorine-substituted PCBs 105, 118, and 156 (within this group, the PCB 118 was by far the major contributor: see Fig. 5). The coplanar PCBs were the second most important contributor in the Swedish and Japanese samples, whereas the PCDDs were the second most important in the U.S. samples.

Undoubtedly, more and more measurements of PCBs in environmental and biological specimens will be done by congener-specific methods. In the United States, however, regulatory agencies such as the Food and Drug Adminisration, Environmental Protection Agency, and Department of Agriculture have established guidelines and analytical methods (based on packed-column gas chromatography) for total PCBs, not for individual congeners (*60,61*). In Europe, PCB levels are frequently regulated on the basis of the concentration of five or six PCBs that are normally found at the highest relative levels. Certainly, more comparisons, such as those performed by Burse et al. (*62*), need to be made between results of packed-column and capillary-column methods. More specifically, however, future work will be based on methods that involve capillary-column gas chromatography/mass spectrometry and allow identification and quantification of coplanar PCBs and other PCBs that, although minor in concentration, greatly contribute to the 2,3,7,8-TCDD equivalents. For human studies, we need more work on exposure assessment, half-lives, partitioning within the body's compartments, and assessments of any adverse health effects that these coplanar PCBs alone or in combination with other environmental contaminants may cause.

Use of trade names is for identification only and does not constitute endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

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