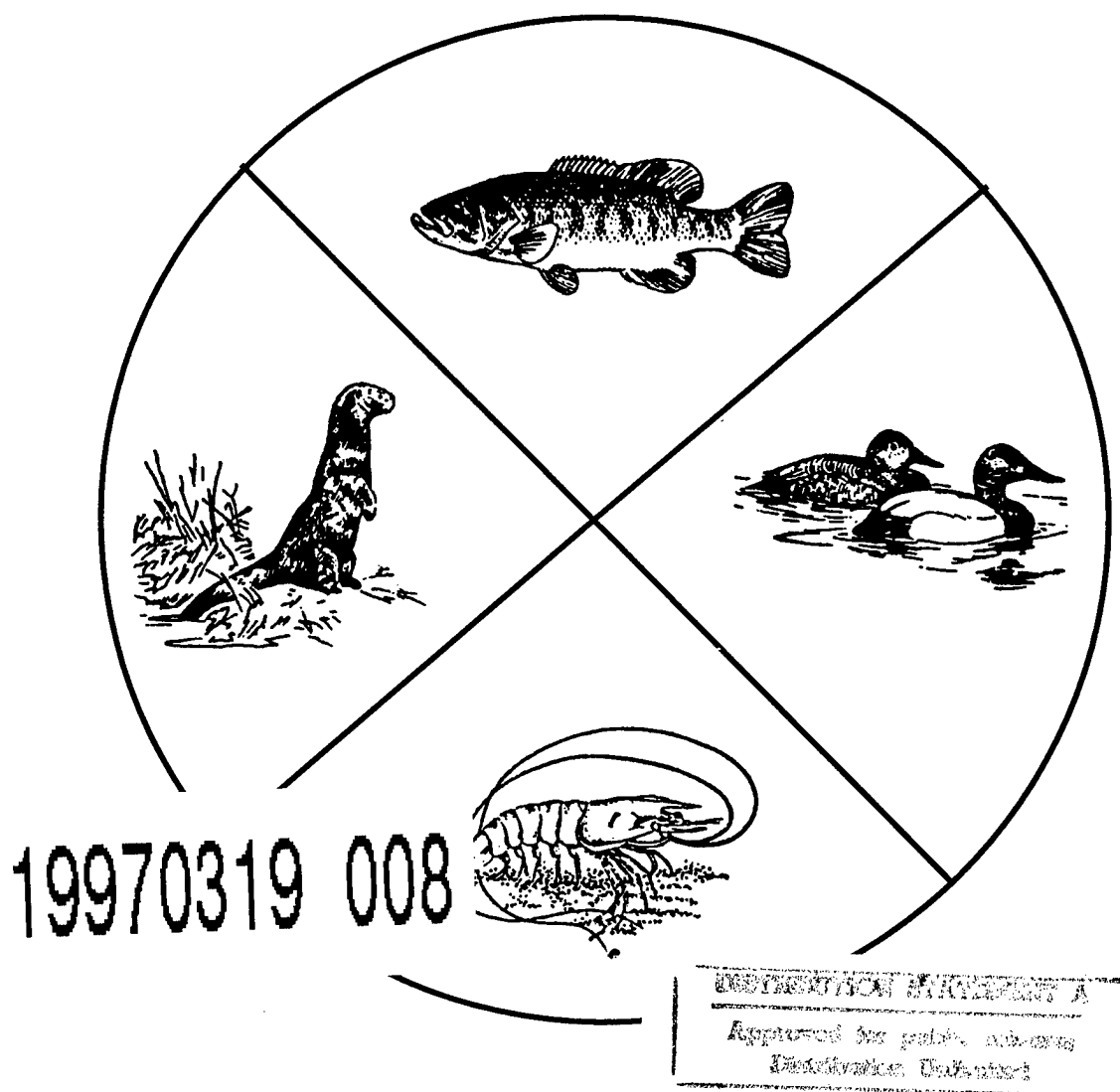


Planar PCB Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review



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Planar PCB Hazards to Fish, Wildlife, and Invertebrates: a Synoptic Review

By

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Abstract. Ecological and toxicological aspects of polychlorinated biphenyls (PCBs) in the environment are reviewed with emphasis on biologically active congeners and fish and wildlife. Subtopics include sources and uses, chemical and biochemical properties, concentrations in field collections, lethal and sublethal effects, and recommendations for the protection of sensitive resources. All production of PCBs in the United States ceased in 1977. Of the 1.2 million tons of PCBs manufactured to date, about 65% are still in use in electrical equipment and 31% in various environmental compartments, and 4% were degraded or incinerated. The 209 PCB congeners and their metabolites show wide differences in biological effects. A significant part of the toxicity associated with commercial PCB mixtures is related to the presence of about 20 planar congeners, i.e., congeners without chlorine substitution in the *ortho* position. Toxic planar congeners, like other PCB congeners, have been detected in virtually all analyzed samples, regardless of collection locale. Planar PCB concentrations were usually highest in samples from near urban areas and in fat and liver tissues, filter-feeding bivalve mollusks, fish-eating birds, and carnivorous marine mammals. Adverse effects of planar PCBs on growth, survival, and reproduction are highly variable because of numerous biotic and abiotic modifiers, including interaction with other chemicals. In general, embryos and juveniles were the most sensitive stages tested to planar PCBs, and the chinook salmon (*Oncorhynchus tshawytscha*), domestic chicken (*Gallus* sp.), mink (*Mustela vison*), rhesus macaque (*Macaca mulatta*), and laboratory white rat (*Rattus* sp.) were among the most sensitive species. For protection of natural resources, most authorities now recommend (1) analyzation of environmental samples for planar and other potentially hazardous congeners; (2) exposure studies with representative species and specific congeners, alone and in combination with other environmental contaminants; (3) clarification of existing structure-induction-metabolism relations; and (4) more research on physiological and biochemical indicators of PCB-stress.

Key Words: Polychlorinated biphenyls, PCBs, planar PCBs, ecotoxicology, fish, wildlife, invertebrates

Polychlorinated biphenyls (PCBs), a group of 209 synthetic halogenated aromatic hydrocarbons, are used extensively in the electricity generating industry as insulating or cooling agents in transformers and capacitors. Because of human activi-

ties and the chemical characteristics of the products, PCBs are now distributed worldwide, and measurable concentrations occur in aquatic organisms and wildlife from North America, Europe, the United Kingdom, and the Atlantic and Pacific Oceans. PCBs elicit a variety of effects including death, birth defects, reproductive failure, liver damage, tumors, and a wasting syndrome. They

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bioaccumulate and biomagnify in the food chain. Legislation has prohibited virtually all uses of PCBs and their manufacture in the United States since 1979; the ban has been accompanied by declines in PCB residues in fishes and wildlife. But the current environmental burden of PCBs in water, sediments, disposal sites, deployed transformers, and other PCB containers—now estimated at more than 374 million kg, much of it localized—continues to represent a potential hazard to associated natural resources (Pa et al. 1980; Eisler 1986; Hansen 1987; Parkinson and Safe 1987; Safe 1987a, 1990, 1994; Huckins et al. 1988; Tanabe 1988; Skaare et al. 1991; Hoffman et al. 1995).

In this report we summarize the recent technical literature on PCB hazards to fishery and wildlife resources, and place special emphasis on the biologically active planar PCBs. This report is part of a continuing series of brief reviews prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service and other requestors.

Sources and Uses

The Monsanto Industrial Chemical Company, the principal domestic manufacturer of PCBs, began PCB production in 1929; commercial mixtures of PCBs were also produced in Western Europe and Japan. PCBs have been used in dielectric fluids; in waxes for metal castings; as heat transfer agents; as plasticizers in paints, coatings, and carbonless copy paper; in cutting oils; in sealants and caulking compounds; and as pesticide extenders (Eisler 1986). The use of PCBs was curtailed in the United States in 1971, and sales were limited to manufacturers of capacitors and transformers; all new uses were banned in 1976 (U.S. National Oceanic and Atmospheric Administration [NOAA] 1991). In 1977, all production of PCBs was halted and no shipments were made after October. Direct and indirect sources of PCB contamination may include aerial transport of combustion products, vaporization from continental and marine areas, current and historic industrial and municipal waste discharges, precipitation, land runoff, concealed dumping, transformer fires, and accidental spills (NOAA 1991; U.S. Environmental Protection Agency [USEPA] 1992a).

The ubiquity of PCBs is indicated by their presence in environmental samples from the polar regions of air, snow, ice, water, and in living

organisms (Norstrom et al. 1988; Hargrave et al. 1989; Larsson et al. 1992; Tanabe et al. 1993). The presence of PCBs in such remote areas suggests the importance of atmospheric transport. The Committee on the Assessment of Polychlorinated Biphenyls in the Environment estimated that 50–80% of the PCBs derived from the United States were now in sediments and waters of the north Atlantic Ocean (National Academy of Sciences [NAS] 1979). Of the estimated total world PCB production of 1.2 million tons to date, about 374,000 tons are now in various portions of the terrestrial, coastal, and open ocean ecospheres (Table 1). Another 783,000 tons are still in use in electrical equipment and other products or deposited in landfills and dumps (Tanabe 1988) and represent a potential source of environmental contamination. An additional 43,000 tons have been degraded or incinerated (Tanabe 1988). Long range atmospheric and oceanic transport seem to be the primary mechanism of global PCB dispersal (Kannan et al. 1989).

Chemical and Biochemical Properties

General

Polychlorinated biphenyls, a highly lipophilic group of global pollutants, consist of 209 congeners (Fig. 1) with widely different toxicity and other biological effects (Kannan et al. 1989). In vertebrates, toxicological effects of PCBs have been related to their ability to induce the cytochrome

Table 1. Estimated PCB loads in the global environment (Tanabe 1988).

Ecosystem	PCB loads (metric tons)
Terrestrial and coastal	
Air	500
River and lakewater	3,500
Seawater	2,400
Soil	2,400
Sediment	130,000
Biota	4,300
Open ocean	
Air	790
Seawater	230,000
Sediment	110
Biota	270
Total (rounded)	374,000

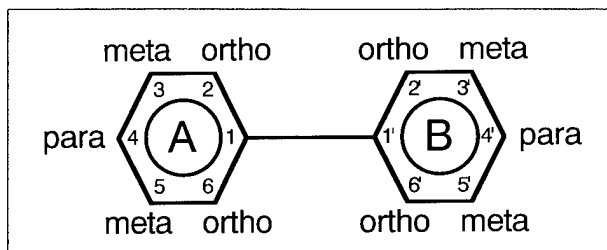


Figure 1. Structure of biphenyl (National Academy of Sciences 1979; U.S. Environmental Protection Agency 1980; Safe 1984, 1994; Eisler 1986). Polychlorinated biphenyls (PCBs) are commercially produced by chlorination of a biphenyl with anhydrous chlorine in the presence of iron filings or ferric chloride as the catalyst. Depending on the conditions under which chlorination occurred, the purified product is a complex mixture of chlorobiphenyls containing 18 to 79% chlorine. Ten possible degrees of chlorination of the biphenyl group produce 10 PCB congener groups: mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decachlorobiphenyl. Within any congener group, a number of positional isomers are possible. For example, the tetrachlorobiphenyl group consists of 30 possible isomers and the pentachlorobiphenyl congener group contains 46 possible isomers. Not all of the 209 possible isomers are likely to be formed during the manufacturing process. In general, the most common PCB isomers formed have either an equal number of chlorine atoms on both rings, or a difference of one chlorine atom between rings. Chlorine substitution is favored at the *ortho* and *para* positions; however, commercial products are complex mixtures of isomers and congeners with no apparent positional preference for halogen substitution.

P450-dependent monooxygenase system (P450). This varies with the degree of chlorination and the arrangement of chlorine atoms on the biphenyl molecule (Skaare et al. 1991). Transformation of PCBs to hydroxylated metabolites by the cytochrome P450 system is the major pathway of PCB metabolism (Sipes and Schnellmann 1987) and occurs mainly in the liver. The rate of cytochrome P450-catalyzed hydroxylation of PCBs decreases as the number of chlorines increases and as the number of unsubstituted adjacent carbon atoms decreases. Some animals metabolize PCBs at different rates, and this is related in part to differences in the basal level of particular isozymes of cytochrome P450 present in the liver. For example, the dog eliminates PCBs more rapidly than other species because it has higher levels of a constitutive isozyme of cytochrome P450 with activity toward the slowly metabolized, bioaccumulated 2,2',4,4',5,5'-hexachlorobiphenyl (Hansen 1987; Sipes and Schnellmann 1987). The reactive arene oxides formed during biotransformation can

bind covalently to tissue macromolecules or conjugate with glutathione. Derivatives of glutathione conjugates and glucuronides of the hydroxylated products are major PCB metabolites. Biotransformation of xenobiotics by cytochrome P450 is not always beneficial to the organism because metabolites can be more toxic or biologically active than the parent compound (Parkinson and Safe 1987). The carcinogenic effect of certain xenobiotics depends on the conversion by cytochrome P450 to a reactive carcinogenic metabolite (Parkinson and Safe 1987). Metabolites and possible degradation pathways of selected PCBs in mammals are presented in detail by Sipes and Schnellmann (1987).

Sedimentation and volatilization are the dominant processes that determine the fate of PCBs in lakes. Both processes remove PCBs from the water, but the relative importance of the transferred amount is influenced by particulate dissolved-phase partitioning that determines the relative size of the particulate pool for sedimentation and the soluble pool for volatilization of PCBs (Millard et al. 1993). High productivity of algae increased the proportion of added PCBs that is absorbed to particulate matter and sedimented. In general, PCB volatilization losses increase under conditions of high mixing and low productivity (Millard et al. 1993).

Physical Properties

Mullin et al. (1984) synthesized and determined the retention times and response factors relative to a reference standard (octachloronaphthalene) of all 209 congeners (Table 2) by using temperature-programmed, high resolution gas chromatography, and electron-capture detection methods (HRGC/ECD). The relative retention times (the ratio of congener retention time/reference standard retention time) of 187 of the 209 congeners differed and permitted HRGC column identification contingent on full or partial separation. Eleven congener pairs had the same retention times and coeluted: 60/56, 70/76, 94/61, 95/80, 133/122, 140/139, 144/135, 145/81, 163/160, 202/171, and 203/196. Of the 209 congeners, 20 can assume a planar configuration (Creaser and Al-Haddad 1989) because of the absence of chlorine substitution in the *ortho* positions (Hong et al. 1992; Fig. 1). Approximately 1% of the non-*ortho*-substituted biphenyl molecules adopt the planar configuration (Safe 1987a). Among the isomers of each homolog, the planar PCBs had the longest retention times. The presence of *ortho*-chloro

Table 2. Polychlorinated biphenyls: structure, retention times, response factors, and octanol-water partition coefficients ($\log K_{ow}$).^a

Isomeric group and PCB number	Structure (chlorine-filled)	Relative retention time ^b	Relative response factor ^c	$\log K_{ow}$ ^d
Monochlorobiphenyls				
1	2	0.0997	0.0251	4.601
2	3	0.1544	0.0393	4.421
3	4	0.1937	0.04	4.401
Dichlorobiphenyls				
4	2,2'	0.2245	0.0374	5.023
5	2,3	0.2785	0.119	— ^e
6	2,3'	0.2709	0.38	5.021
7	2,4	0.2566	0.69	5.15
8	2,4'	0.2783	0.206	5.301
9	2,5	0.257	0.388	5.18
10	2,6	0.2243	0.262	5.311
11	3,3'	0.3238	0.0449	5.343
12	3,4	0.3298	0.179	5.295
13	3,4'	0.3315	0.2	— ^e
14	3,5	0.2373	0.3047	5.404
15	4,4'	0.3387	0.107	5.335
Trichlorobiphenyls				
16	2,2',3	0.3625	0.447	5.311
17	2,2',4	0.3398	0.412	5.761
18	2,2',5	0.3378	0.313	5.551
19	2,2',6	0.3045	0.3037	5.481
20	2,3,3'	0.417	0.7238	5.577
21	2,3,4	0.4135	1.0598	5.517
22	2,3,4'	0.4267	1.0935	5.421
23	2,3,5	0.377	0.5	5.577
24	2,3,6	0.3508	0.793	5.671
25	2,3',4	0.3937	0.5	5.677
26	2,3',5	0.3911	0.603	5.667
27	2,3',6	0.3521	0.495	5.447
28	2,4,4'	0.4031	0.854	5.691
29	2,4,5	0.382	0.6339	5.743
30	2,4,6	0.3165	0.8202	5.504
31	2,4',5	0.4094	0.562	5.677
32	2,4',6	0.3636	0.278	5.751
33	2',3,4	0.4163	0.447	5.572
34	2',3,5	0.3782	0.6092	5.667
35	3,3',4	0.4738	0.3746	5.827
36	3,3',5	0.4375	0.2948	4.151
37	3,4,4'	0.4858	0.58	4.941
38	3,4,5	0.5102	0.722	5.767
39	3,4',5	0.4488	0.347	5.897
Tetrachlorobiphenyls				
40	2,2',3,3'	0.5102	0.722	5.561
41	2,2',3,4	0.499	0.5469	6.111
42	2,2',3,4'	0.487	0.792	5.767
43	2,2',3,5	0.4587	0.503	5.757
44	2,2',3,5'	0.4832	0.524	5.811
45	2,2',3,6	0.4334	0.54	5.537
46	2,2',3,6'	0.445	0.468	5.537
47	2,2',4,4'	0.4639	0.848	6.291

Table 2. *Continued.*

Isomeric group and PCB number	Structure (chlorine-filled)	Relative retention time ^b	Relative response factor ^c	Log K _{ow} ^d
48	2,2',4,5	0.4651	0.556	5.787
49	2,2',4,5'	0.461	0.648	6.221
50	2,2',4,6	0.4007	0.6817	5.637
51	2,2',4,6'	0.4242	0.6	5.637
52	2,2',5,5'	0.4557	0.418	6.091
53	2,2',5,6'	0.4187	0.3606	5.627
54	2,2',6,6'	0.38	0.3643	5.904
55	2,3,3',4	0.5562	0.829	6.117
56	2,3,3',4'	0.5676	0.829	6.117
57	2,3,3',5	0.5515	0.6	6.177
58	2,3,3',5'	0.5267	0.609	6.177
59	2,3,3',6	0.486	0.6	5.957
60	2,3,4,4'	0.5676	1.0164	5.452
61	2,3,4,5	0.5331	1.2227	5.943
62	2,3,4,6	0.4685	1.1478	5.897
63	2,3,4',5	0.529	0.728	6.177
64	2,3,4',6	0.4999	0.607	5.957
65	2,3,5,6	0.4671	0.8408	5.867
66	2,3',4,4'	0.5447	0.646	5.452
67	2,3',4,5	0.5214	0.6	6.207
68	2,3',4,5'	0.504	0.726	6.267
69	2,3',4,6	0.451	0.8024	6.047
70	2,3',4',5	0.5407	0.658	6.231
71	2,3',4',6	0.4989	0.468	5.987
72	2,3',5,5'	0.4984	0.5515	6.267
73	2,3',5',6	0.4554	0.5805	6.047
74	2,4,4',5	0.5341	0.671	6.671
75	2,4,4',6	0.4643	0.6461	6.057
76	2',3,4,5	0.5408	0.5795	6.137
77	3,3',4,4'	0.6295	0.3812	6.523
78	3,3',4,5	0.6024	1.1151	6.357
79	3,3',4,5'	0.5894	0.881	6.427
80	3,3',5,5'	0.5464	0.7278	6.583
81	3,4,4',5	0.6149	0.7159	6.367
Pentachlorobiphenyls				
82	2,2',3,3',4	0.6453	0.773	6.142
83	2,2',3,3',5	0.6029	0.6339	6.267
84	2,2',3,3',6	0.5744	0.386	6.041
85	2,2',3,4,4'	0.6224	0.7396	6.611
86	2,2',3,4,5	0.6105	0.7968	6.204
87	2,2',3,4,5'	0.6175	1.021	6.371
88	2,2',3,4,6	0.5486	0.6892	7.516
89	2,2',3,4,6'	0.5779	0.561	6.077
90	2,2',3,4',5	0.5814	0.611	6.367
91	2,2',3,4',6	0.5549	0.571	6.137
92	2,2',3,5,5'	0.5742	0.5375	6.357
93	2,2',3,5,6	0.5437	0.6676	6.047
94	2,2',3,5,6'	0.5331	0.4514	6.137
95	2,2',3,5',6	0.5464	0.443	6.137
96	2,2',3,6,6	0.5057	0.4308	5.717
97	2,2',3',4,5	0.61	0.631	6.671
98	2,2',3',4,6	0.5415	0.6246	6.137
99	2,2',4,4',5	0.588	0.613	7.211
100	2,2',4,4',6	0.5212	0.5871	6.237
101	2,2',4,5,5'	0.5816	0.668	7.071

Table 2. Continued.

Isomeric group and PCB number	Structure (chlorine-filled)	Relative retention time ^b	Relative response factor ^c	Log K _{ow} ^d
102	2,2',4,5,6'	0.5431	0.4561	6.167
103	2,2',4,5',6	0.5142	0.6068	6.227
104	2,2',4,6,6	0.4757	0.4561	5.817
105	2,3,3',4,4'	0.7049	0.94	6.657
106	2,3,3',4,5	0.668	1.0046	6.647
107	2,3,3',4',5	0.6628	0.8183	6.717
108	2,3,3',4,5'	0.6626	1.0654	6.717
109	2,3,3',4,6	0.6016	0.9625	6.487
110	2,3,3',4',6	0.6314	0.65	6.532
111	2,3,3',5,5'	0.6183	0.6601	6.767
112	2,3,3',5,6	0.5986	0.8286	6.457
113	2,3,3',5',6	0.5862	0.604	6.547
114	2,3,4,4',5	0.6828	1.0261	6.657
115	2,3,4,4',6	0.6171	1.1328	6.497
116	2,3,4,5,6	0.6132	1.3987	6.304
117	2,3,4',5,6	0.615	0.8895	6.467
118	2,3',4,4',5	0.6693	0.87	7.121
119	2,3',4,4',6	0.5968	0.8239	6.587
120	2,3',4,5,5'	0.6256	0.7444	6.797
121	2,3',4,5',6	0.5518	0.7659	6.647
122	2',3,3',4,5	0.6871	0.7247	6.647
123	2',3,4,4',5	0.6658	0.6645	6.747
124	2',3,4,5,5'	0.6584	0.848	6.737
125	2',3,4,5,6'	0.6142	0.556	6.517
126	3,3',4,4',5	0.7512	0.4757	6.897
127	3,3',4,5,5'	0.7078	0.5834	6.957
Hexachlorobiphenyls				
128	2,2',3,3',4,4'	0.7761	1.188	6.961
129	2,2',3,3',4,5	0.7501	0.997	7.321
130	2,2',3,3',4,5'	0.7184	0.952	7.391
131	2,2',3,3',4,6	0.6853	0.8492	6.587
132	2,2',3,3',4,6'	0.7035	0.7303	6.587
133	2,2',3,3',5,5'	0.6871	1.148	6.867
134	2,2',3,3',5,6	0.6796	0.7331	7.304
135	2,2',3,3',5,6'	0.6563	0.7031	7.151
136	2,2',3,3',6,6'	0.6257	0.444	6.511
137	2,2',3,4,4',5	0.7329	1.112	>7.711
138	2,2',3,4,4',5'	0.7403	0.827	7.441
139	2,2',3,4,4',6	0.6707	0.7219	6.677
140	2,2',3,4,4',6'	0.6707	0.6732	6.677
141	2,2',3,4,5,5'	0.720	1.352	7.592
142	2,2',3,4,5,6	0.6848	1.218	6.517
143	2,2',3,4,5,6'	0.6789	0.7088	6.607
144	2,2',3,4,5',6	0.6563	0.8764	6.677
145	2,2',3,4,6,6'	0.6149	0.6789	6.257
146	2,2',3,4',5,5'	0.6955	0.728	6.897
147	2,2',3,4',5,6	0.6608	0.6	6.647
148	2,2',3,4',5,6'	0.6243	0.554	6.737
149	2,2',3,4',5',6	0.6672	0.572	7.281
150	2,2',3,4',6,6'	0.5969	0.5676	6.327
151	2,2',3,5,5',6	0.6499	0.785	6.647
152	2,2',3,5,6,6'	0.6062	0.5235	6.227
153	2,2',4,4',5,5'	0.7036	0.688	7.751
154	2,2',4,4',5,6'	0.6349	0.57	6.767
155	2,2',4,4',6,6'	0.5666	0.586	7.123

Table 2. Continued.

Isomeric group and PCB number	Structure (chlorine-filled)	Relative retention time ^b	Relative response factor ^c	Log K _{ow} ^d
156	2,3,3',4,4',5	0.8105	1.389	7.187
157	2,3,3',4,4',5'	0.8184	1.1965	7.187
158	2,3,3',4,4',6	0.7429	1.132	7.027
159	2,3,3',4,5,5'	0.7655	0.9934	7.247
160	2,3,3',4,5,6	0.7396	1.1914	6.937
161	2,3,3',4,5',6	0.6968	0.9672	7.087
162	2,3,3',4',5,5'	0.7737	1.0322	7.247
163	2,3,3',4',5,6	0.7396	0.9976	6.997
164	2,3,3',4',5',6	0.7399	0.9848	7.027
165	2,3,3',5,5',6	0.692	1.0777	7.057
166	2,3,4,4',5,6	0.7572	1.0421	6.937
167	2,3',4,4',5,5'	0.7814	1.0658	7.277
168	2,3',4,4',5',6	0.7068	0.8375	7.117
169	3,3',4,4',5,5'	0.8625	0.8355	7.427
Heptachlorobiphenyls				
170	2,2',3,3',4,4',5	0.874	0.75	7.277
171	2,2',3,3',4,4',6	0.8089	1.1712	6.704
172	2,2',3,3',4,5,5'	0.8278	1.172	7.337
173	2,2',3,3',4,5,6	0.8152	2.044	7.027
174	2,2',3,3',4,5,6'	0.7965	0.806	7.117
175	2,2',3,3',4,5',6	0.7611	0.381	7.177
176	2,2',3,3',4,6,6'	0.7305	1.0589	6.767
177	2,2',3,3',4',5,6	0.8031	1.0009	7.087
178	2,2',3,3',5,5',6	0.7537	0.621	7.147
179	2,2',3,3',5,6,6'	0.7205	0.8237	6.737
180	2,2',3,4,4',5,5'	0.8362	1.295	7.367
181	2,2',3,4,4',5,6	0.7968	1.6046	7.117
182	2,2',3,4,4',5,6'	0.7653	1.1272	7.207
183	2,2',3,4,4',5',6	0.772	0.976	7.207
184	2,2',3,4,4',6,6'	0.7016	1.0046	6.857
185	2,2',3,4,5,5',6	0.7848	1.437	7.933
186	2,2',3,4,5,6,6'	0.7416	1.2236	6.697
187	2,2',3,4',5,5',6	0.7654	1.122	7.177
188	2,2',3,4',5,6,6'	0.692	0.7337	6.827
189	2,3,3',4,4',5,5'	0.9142	1.5091	7.717
190	2,3,3',4,4',5,6	0.874	1.31	7.467
191	2,3,3',4,4',5',6	0.8447	1.4741	7.557
192	2,3,3',4,5,5',6	0.8269	1.599	7.527
193	2,3,3',4',5,5',6	0.8397	1.4167	7.527
Octachlorobiphenyls				
194	2,2',3,3',4,4',5,5'	0.962	1.868	8.683
195	2,2',3,3',4,4',5,6	0.9321	0.415	7.567
196	2,2',3,3',4,4',5',6	0.8938	1.2321	7.657
197	2,2',3,3',4,4',6,6'	0.8293	0.9522	7.307
198	2,2',3,3',4,5,5',6	0.8845	1.07	7.627
199	2,2',3,3',4,5,6,6'	0.8494	1.1508	7.207
200	2,2',3,3',4,5',6,6'	0.8197	0.369	7.277
201	2,2',3,3',4',5,5',6	0.8875	0.803	7.627
202	2,2',3,3',5,5',6,6'	0.8089	1.165	8.423
203	2,2',3,4,4',5,5',6	0.8938	1.629	7.657
204	2,2',3,4,4',5,6,6'	0.8217	0.8034	7.307
205	2,3,3',4,4',5,5',6	0.9678	1.406	8.007

Table 2. Continued.

Isomeric group and PCB number	Structure (chlorine-filled)	Relative retention time ^b	Relative response factor ^c	Log K _{ow} ^d
Nonachlorobiphenyls				
206	2,2',3,3',4,4',5,5',6	1.0103	1.673	9.143
207	2,2',3,3',4,4',5,6,6'	0.9423	1.3257	7.747
208	2,2',3,3',4,5,5',6,6'	0.932	1.1756	8.164
Decachlorobiphenyl				
209	2,2',3,3',4,4',5,5',6,6'	1.0496	1.139	9.603

^a Ballschmiter and Zell (1980), McDuffie (1981), Bruggeman et al. (1982), Yalkowsky et al. (1983), Mullin et al. (1984), Rapaport and Eisenreich (1984), Shiu and Mackay (1986), Woodburn et al. (1987), Hawker and Connell (1988).

^b Gas chromatography retention time of PCB congener relative to the retention time of the reference standard octachloronaphthalene on a capillary column of SE-54.

^c Gas chromatography peak area response of PCB relative to peak area of 1 ng of octachloronaphthalene.

^d K_{ow} (octanol-water partition coefficient) = C_{ow}/C_{wo}, where C_{ow} is the concentration of the solute in octanol saturated with water, and C_{wo} is the concentration of the solute in water saturated with water.

^e — = no data.

substituents reduces planarity of the rings. However, congeners with 1 or 2 *ortho* chlorines can also assume a planar ring position (Safe 1990). Although peak resolution is greatly improved by substitution of high resolution capillary columns for packed columns, more than one candidate congener may occur at many GC peaks. Using a narrow bore column, Duinker et al. (1988a) found only 17 PCB congeners that were fully separated without potential interference from coeluting congeners.

The transport and fate of PCBs in aquatic systems and their partitioning between sediment, water, and organisms depends largely on sorption reactions. In soils, the sorption and retention of PCB congeners is influenced by the number of chlorine atoms in the molecule, and the more highly-chlorinated PCBs tend to be more strongly bound. Relative sorption capacity and other properties of congeners also depend on PCB configuration. The soil sorption capacities of PCB congeners and their bioconcentration factors were related to octanol-water partition coefficients (Connor 1984) as follows:

$$K_{ow} = C_{ow} / C_{wo}$$

where K_{ow} is the octanol-water partition coefficient, C_{ow} the concentration of the solute in octanol saturated with water, and C_{wo} the concentration of the solute in water saturated with octanol.

K_{ow} values are used to estimate bioconcentration factors (bioaccumulation after uptake from water), soil and sediment organic carbon-water partition coefficients, toxicities, and aqueous solu-

bilities (Woodburn et al. 1987; Hawker and Connell 1988). Techniques for measuring the K_{ow} values include the shake-flask method (Shiu and Mackay 1986), the reverse-phase thin-layer chromatography (Bruggeman et al. 1982), reverse-phase high-performance liquid chromatography (Rapaport and Eisenreich 1984), and the generator column technique (Woodburn et al. 1987; Hawker and Connell 1988), or it may be calculated by an estimation technique based on correlation with properties of compounds with known K_{ow} values. From a strong linear relation calculated between known log K_{ow} values and calculated total surface areas (TSA; correlation coefficient of 0.951 for 30 congeners), Hawker and Connell (1988) estimated K_{ow} values of individual congeners from the calculated TSA. Estimated K_{ow} values (Table 2) were previously unavailable for many PCB congeners, including toxic non-*ortho* substituted congeners found only in relatively small amounts in commercial Aroclors. Reported K_{ow} values of some congeners vary in the literature and may not be comparable when different measuring techniques are used. Reverse-phase thin-layer chromatography (RP TLC) and high-performance liquid chromatography (RP-HPLC) require empirical correction factors. Discrepancies in K_{ow} values between the generator column and RP-HPLC techniques for *ortho* PCBs relative to the other PCBs were resolved with an *ortho* correction factor (Woodburn et al. 1987). Non-planarity of the *ortho*-substituted PCBs may reduce the ability of the solute to interact with the stationary phase in RP-TLC (Bruggeman et al. 1982). Despite these

uncertainties, the K_{ow} is routinely used in hazard evaluation and risk assessment of most organic chemicals.

Toxic Equivalency Factors

A significant part of the toxicity associated with commercial PCB mixtures is related to the presence of the small number of planar congeners. These compounds induce several similar toxic effects in mammals and birds, such as hepatotoxicity, immunotoxicity, and reproductive toxicity (Janz and Metcalfe 1991b; Brunstrom et al. 1995). Planar halogenated aromatic compounds act, in part, by a common mechanism initiated by binding to a cytosolic aryl hydrocarbon receptor. The relative toxicities of planar halogenated hydrocarbons are calculated by expressing their toxicity in relation to 2,3,7,8-TCDD, the most potent compound in this class of chemicals. Toxic equivalency factors (TEFs) are fractional potencies that relate a compound's potency to that of 2,3,7,8-TCDD. The 2,3,7,8-TCDD TEF has been used to estimate the relative toxic potencies of individual planar halogenated hydrocarbons (Safe 1987b; 1990, 1994; Janz and Metcalfe 1991b; Johansen et al. 1993). According to Safe (1990), TEF values should be derived from data on the following effects in descending order of priority: (1) long-term carcinogenicity studies; (2) reproductive studies; (3) subchronic studies that measure Ah receptor-mediated responses, such as thymic atrophy, loss in body weight, and immunotoxicity; (4) acute toxicity studies; and (5) in vivo or in vitro biochemical responses such as enzyme induction and receptor binding. Relative toxic potencies are modified by many variables including age, sex, species, and strain of the animal; the efficiency of the chemical to induce cytochrome P450 and associated monooxygenase enzyme activities, glucuronosyl and glutathione transferases, and other drug-metabolizing enzymes; and the efficiency of the organism to modulate steroid-metabolizing enzymes, induce delta aminolevulinic acid synthetase, inhibit porphyrinogen decarboxylase, decrease Ah receptor binding activity, and alter Vitamin A and thyroid hormone levels (Safe 1990, 1994).

The in vitro induction of the cytochrome P450c-dependent monooxygenases, aryl hydrocarbon hydroxylase (AHH), or ethoxyresorufin O-deethylase (EROD) by 2,3,7,8-TCDD and related halogenated aryl hydrocarbons in rat liver cells was developed as a short term quantitative bioassay for these chemicals; aromatics that do not fit this correlation

are considered as congeners that are readily metabolized in vivo (Safe 1987b). Induction of either AHH or EROD activity in the H4IIE rat hepatoma cell line by PCB, PCDF, and PCDD congeners, either singly or in combination, correlates well with the in vivo toxicity of these compounds to rats (as quoted in Ankley et al. 1991). The proposed mean TEF values range up to 0.1 in non-*ortho* substituted planar PCBs, 0.0005 in mono-*ortho* substituted planar PCBs, and 0.0001 in di-*ortho* substituted planar PCBs (Table 3). Although the concentration of non- and mono-*ortho* substituted PCBs in animal tissues ranges from about 0.01 µg/kg to several micrograms per kg (about 1,000 to 100,000 times lower than the sum of total PCBs), it is significantly higher than the concentration of the highly toxic 2,3,7,8-TCDD and 2,3,4,7,8-pentachloro-dibenzofuran. Accordingly, the non- and mono-*ortho* PCBs—despite their lower toxic potency—often contribute as much or more to the 2,3,7,8-TCDD-like activity than either dioxins or furans (Johansen et al. 1993). However, the overall importance of mono-*ortho* PCBs to the TEF is questioned by Ahlborg et al. (1994), and this must be considered in future risk assessment evaluations.

A note of caution: recent studies revealed that mammal-derived TEFs underestimate the potency of planar PCB mixtures in fish (Newsted et al. 1995). For example, TEFs of non-*ortho* PCB congeners based on mortality of rainbow trout in early life stage are as much as 1,000 times lower than TEFs proposed for human risk assessment (Walker and Peterson 1991, 1994).

Table 3. Proposed toxicity equivalency values (TEF) relative to 2,3,7,8-TCDD of non-*ortho*, mono-*ortho*, and di-*ortho* planar PCBs (Safe 1990, 1994; Ahlborg et al. 1994).

PCB congener	TEF
Non-<i>ortho</i> planar PCBs	
PCB 77	0.0005
PCB 126	0.1
PCB 169	0.01
Mono-<i>ortho</i> planar PCBs	
PCB 105	0.0001
PCB 114	0.0005
PCB 118	0.0001
PCB 123	0.0001
PCB 156	0.0005
PCB 157	0.0005
PCB 167	0.00001
PCB 189	0.0001
Di-<i>ortho</i> planar PCBs	
PCB 170	0.0001
PCB 180	0.00001

Structure-Function Relations

Safe (1984, 1990) described three classes of PCB congeners on the basis of their ability to induce benzo(a)pyrene hydroxylase (also known as aryl hydrocarbon hydroxylase or AHH) and ethoxyresorufin *O*-deethylase (EROD) activities: (1) planar PCBs; (2) mono-*ortho* analogs of the planar PCBs; and (3) di-*ortho* analogs of the planar PCBs. Among the 20 possible planar PCB congeners and their analogs (Fig. 2), the most toxic in rats were PCBs 77, 126, and 169; these three congeners are approximate isostereomers of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) and have similar toxic effects, including induction of 3-methylcholanthrene type drug-metabolizing enzymes, body weight loss, thymic atrophy, dermal disorders, hepatic damage, high binding affinity to hepatic cytosolic receptor proteins, immunotoxicity, reproductive impairment, and teratogenicity (Masse et al. 1986; Parkinson and Safe 1987; Tanabe et al. 1987; Gooch et al. 1989; Kannan et al. 1989; Janz et al. 1992; Himberg 1993; Giesy et al. 1994a). PCBs 77, 126, and 169 have been detected in eggs of terns from Lake Michigan, in marine mammals and humans, and in fish from the Hudson and Ohio Rivers (Huckins et al. 1988). However, they are difficult to detect without proper methods (Tanabe 1988; Schwartz et al. 1993).

The structural characteristics of individual PCB congeners influence their induction of various P450 activities. In mammals, PCB congeners have been characterized as 3-methylcholanthrene-type inducers, phenobarbital-type inducers, or mixed-type inducers of both. AHH and EROD activities (which are preferentially catalyzed by the P450IA gene subfamily) have been induced by planar PCBs in fish and mammals and by some mono- and di-*ortho* analogs of planar PCBs in mammals (Skaare et al. 1991).

The mechanism of toxic action of planar and mono-*ortho* planar PCBs is linked to an interaction with the 2,3,7,8-TCDD (or Ah) receptor protein. But this mechanism does not account for all observed PCB toxicities (Hansen 1987; Safe 1994). Toxic responses unrelated to Ah receptor effects have been reported of PCBs 4, 28, 31, 49, 52, 84, 95, 110, 136, and 153. For example, PCB 153 is less cytotoxic than PCB 169 but is a more effective inhibitor of intercellular communication. PCB 52 caused moderate chick embryotoxicity; however, PCBs 18 and 153 were inactive, and PCBs 84

and 118 were severely toxic but by different mechanisms (Hansen 1987).

Group I planar PCBs are 10 times more toxic and 100 times more effective as inducers of cytochrome P450c dependent monooxygenase and 70 times more effective in competitively displacing 2,3,7,8-TCDD from a rat cytosol receptor protein than Group II planar PCBs (Table 4; Safe 1990; Fig. 2). Mono-*ortho* analogs of the planar PCBs have one substituent in the *ortho* (2 or 2') position; these compounds possess diminished yet significant EROD- or AHH-inducing capacity and also induce P450 forms that are induced by the phenobarbital class of compounds (Gooch et al. 1989). Mono-*ortho* derivatives (PCBs 105, 118, 156, and 189) may be more important in terms of 2,3,7,8-TCDD-like activity and in occurrence (Hansen 1987). Di-*ortho* analogs of the planar PCBs, that is, those with *ortho*-, *meta*-, and *para*-substituents, possess still weaker but significant AHH-inducing activity (Gooch et al. 1989). Certain di-*ortho* derivatives of the 3,3',4,4' pattern (PCBs 128, 138, 153, 170, 180) are significant components of PCB residues; however, PCBs 128, 138, and 170 have reduced 2,3,7,8-TCDD-like effects (Hansen 1987). In rats, several PCBs (105, 114, 118, 123, 126, 156, 157, 169) produced a linear correlation between the EC50 response (in vitro) of AHH induction against the ED50 (in vivo) of body weight loss, thymic atrophy, hepatic AHH, and EROD induction (Safe 1987b). The planar mono- and di-*ortho* derivatives (PCBs 105, 118, 156, 189, 128, 138, 153, 170, 180) are referred to as mixed inducers because they elicit effects similar to coadministration of phenobarbital plus methylcholanthrene (Hansen 1987).

PCB 156, a mixed inducer of microsomal enzymes, significantly increases the incidences of cleft palates by 2,3,7,8-TCDD in rodents (Birnbbaum et al. 1985). Interactions among polychlorinated congeners may range from antagonism to additivity to synergism (Safe 1990), and the toxicity of individual PCBs can be raised by interaction with other PCBs (Table 5).

Quantitation

PCBs are chemically inert, non-polar compounds and relatively stable during collection and storage; however, PCB concentrations in environmental samples vary with different measurement techniques (Kratovich et al. 1984; Huckins et al. 1990b; Lebo et al. 1992; Prest et al. 1992; Bidleman et al. 1993; Litten et al. 1993), with types of Aroclor

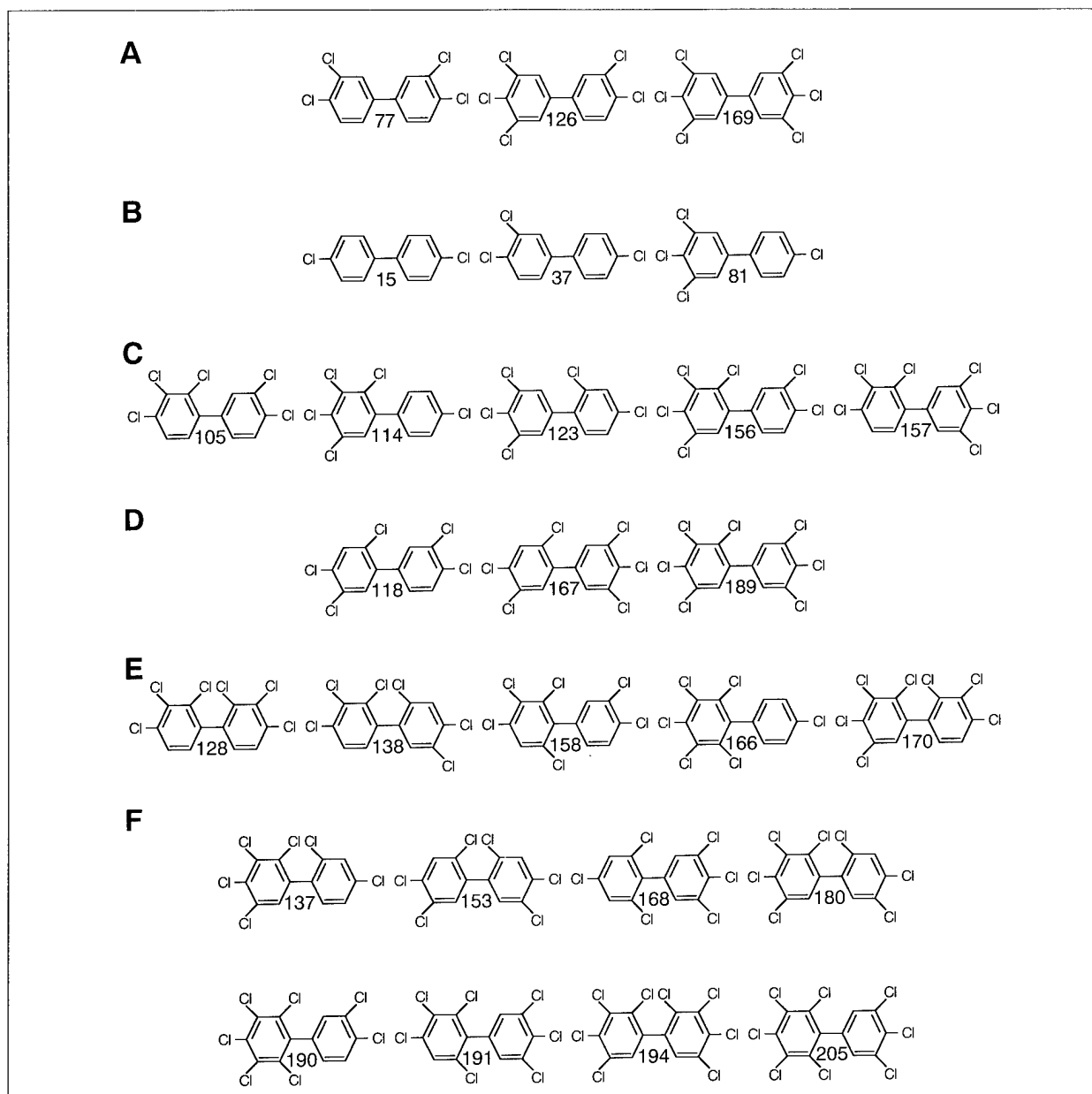


Figure 2. Planar polychlorinated biphenyls and their derivatives (Hansen 1987; Parkinson and Safe 1987; Safe 1987b, 1994; Tanabe et al. 1987; Kannan et al. 1989; de Voogt et al. 1990; Ankley et al. 1991; Sonzogni et al. 1991; Hong et al. 1992; Johansen et al. 1993). The general order of biological activity is: Group I non-*ortho* planar PCBs > Group II non-*ortho* planar PCBs > mono-*ortho* planar PCBs > di-*ortho* planar PCBs. A. Group I. Potent non-*ortho* planar PCBs. This group contains no *ortho*, 2 *para* and at least 2 *meta*-chlorines (PCBs 77, 126, and 169), and are approximate stereoisomers of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD). They are less potent than 2,3,7,8-TCDD but elicit similar biological responses including induction of cytochrome P450 and aryl hydrocarbon hydroxylase (AHH). This group is the most biologically active of all planar PCBs and their derivatives. B. Group II. Less-potent non-*ortho* planar PCBs (PCBs 15, 37, and 81). Group II non-*ortho* planar PCBs, when compared to Group I, were only 0.1 times as toxic, 0.01 times as effective in inducing cytochrome P450c-dependent monooxygenase, and about 0.015 times as effective in competitively displacing 2,3,7,8-TCDD from a cytosol-receptor protein in rat liver. C. Potent mono-*ortho* planar PCBs. The addition of a single *ortho*-chlorine substituent to non-*ortho* planar PCBs 77, 81, 126, and 169 yields 8 derivatives, of which 5 (PCBs 105, 114, 123, 156, 157) were more potent when the *ortho* chlorine was adjacent to a *meta* hydrogen. D. Less-potent mono-*ortho* planar PCBs. The toxicity of non-*ortho* planar PCBs is reduced by the introduction of an *ortho*-chloro substituent, especially when it is adjacent to a *meta*-chlorine (PCBs 118, 167, 189). E. Potent di-*ortho* planar PCBs. The di-*ortho* planar PCBs are less toxic than the mono-*ortho* planar PCBs. At least 5 di-*ortho* derivatives (PCBs 128, 138, 158, 166, 170) compete with 2,3,7,8-TCDD for receptor binding sites in rat liver cytosol to induce cytochrome P450c. F. Less-potent di-*ortho* planar PCBs. These 8 di-*ortho* derivatives (PCBs 137, 153, 168, 180, 190, 191, 194, 205) are less potent than those figured in E.

Table 4. Interactive effects of PCBs on the induction of rat (*Rattus* sp.) liver microsomal cytochrome P450c (Parkinson and Safe 1987).

PCB congener and dose ($\mu\text{mol/kg}$ body weight)	Microsomal enzyme activity (nmoles product/mg protein/min)	
	Benzo (a)pyrene	Ethoxyresorufin O-deethylase
Control, corn oil	0.088	0.277
PCB 153 (300)	0.121	0.530
PCB 126 (0.01)	0.486	3.60
PCB 126 (0.01) plus PCB 153 (300) ^a	0.887	6.03
PCB 169 (125)	0.676	6.89
PCB 169 (125) plus PCB 153 (300) ^b	1.06	10.5

^a administered 7 days prior to treatment with PCB 126^b administered 7 days prior to treatment with PCB 169

standards used for calibration (Table 6), and with oven drying techniques (Table 7). Reports of interlaboratory comparison studies for PCB analysis show wide variations and strongly indicate a need for more rigorous quality control and assurance. In one multilaboratory study (Alford-Stevens 1988)—wherein PCBs were determined in water, soil, and sediments—percent recoveries from analysis of fortified waters averaged 60% in the high concentration sample containing 148 $\mu\text{g/L}$ total PCBs and 55% in the low concentration sample of 37 $\mu\text{g/L}$. No single combination of extraction and cleanup was best for all solid samples. An analytical intercomparison exercise (International Council for the Exploration of the Sea [ICES] 1992) was conducted on solutions containing 10 PCB congeners. Of the 61 evaluated laboratories, a group of 47 laboratories produced between-group standard deviations of 1.10-1.13 by all PCB congeners except PCB 52; a group of 11 laboratories were identified as an outlier group. Only three laboratories were able to quantify PCBs 110 and 77, which coelute in most GC columns. Peak height measurements gave better reproducibility than peak area methods. In

another intercomparison study, the analysis by 11 laboratories of a solution containing 12 pure congeners resulted in a variation of as much as 20% for analysis of a single congener. For the analysis of the same congeners in a fish oil, the coefficient of variation ranged from 17.4 to 132% (66.2% without outliers) and had a median of 47.1% (ICES 1992).

PCBs in biological samples are usually extracted by a Soxhlet column and with a nonpolar solvent such as hexane; the sample is first mixed with sodium sulfate to remove moisture. The extraction of PCBs from sediments was tested with sonication, with two sonications interspersed at a 24-h quiescent interval, with steam distillation, or with Soxhlet extraction (Dunnivant and Elzerman 1988). Comparison of the recoveries of various PCB mixtures from dry and wet sediments by the four techniques and the extraction efficiency of four solvents showed that the best overall recoveries were obtained by Soxhlet extraction and the two sonication procedures. In comparisons of solvent systems of acetone, acetonitrile, acetone-hexane (1+1), and water-acetone-isooctane (5+1.5+1), recoveries of lower chlorinated congeners (dichloro- to tetrachloro-) were usually higher with acetonitrile and recoveries of higher chlorinated congeners (tetrachloro- to heptachloro-) extracted with acetone were superior (Dunnivant and Elzerman 1988). The completeness of extraction from a sample matrix does not seem to discriminate against specific isomers; however, discrimination in the cleanup and fractionation process may occur and must be tested (Duinker et al. 1988b).

With most analytical techniques for the quantification of PCB residue levels, chromatographic separations were used, most frequently electron-capture gas chromatography (GC/ECD). With early methods, selected peaks were used to estimate total PCBs. Low resolution separations were satisfactory when packed columns that produced a pattern of peaks with measured areas were used.

Table 5. Summary of PCB structure-function relations in rats, *Rattus* sp. (Parkinson and Safe 1987).

PCB structure	Cytochrome P450 induction (% of controls)		Relative activity (% of controls)		
	P450c+P450d	P450b+P450e	AHH induction		Receptor binding
			In vivo	In vitro	
Planar PCBs: Group I	1,800-4,100	None	+++	1-100	35-100
Planar PCBs: Group II	1,100-1,500	600-1,400	++	0.03	0.5
Mono- <i>ortho</i> planars	750-2,400	2,600-4,700	++	0.00002-0.3	1.5-6
Di- <i>ortho</i> planars	250-900	1,000-6,300	+	Inactive	<0.3
PCB 153	None	7,300	Inactive	Inactive	<0.3
2,3,7,8-TCDD	3,500	None	+++++	400	2,500

Table 6. PCB congeners in Aroclor 1254 and 1260.^a

Average percent in Aroclor			Average percent in Aroclor			Average percent in Aroclor		
PCB Number	1254	1260	PCB Number	1254	1260	PCB Number	1254	1260
16	— ^b	0.04	90	0.93	0.56	160	—	0.05
17	0.19	0.05	91	0.83	0.07	167	0.21	0.21
18	0.41	0.12	92	1.58	0.59	169	—	0.05
21	—	0.01	95	6.02	2.87	170	0.31	5.36
22	—	0.01	96	0.08	—	171/202	0.05	1.65
24	—	0.01	97	2.55	0.34	172	0.05	0.78
26	—	0.02	99	3.60	0.12	173	0.09	0.21
28	0.25	0.045	100	0.10	0.02	174	0.34	4.68
29	—	0.02	101	7.94	3.82	175	0.05	0.36
31	0.22	0.05	105	3.83	0.07	176	0.32	0.64
33	0.14	0.09	107	0.72	0.03	177	0.21	2.06
37	—	0.04	110	5.85	1.80	178	1.35	1.41
40	0.20	0.03	115	0.30	0.05	180	0.38	8.11
41	0.64	0.20	118	6.39	0.53	183	0.17	2.03
42	—	0.04	119	0.14	—	185	—	2.72
43	—	0.02	122	0.50	0.21	187	0.32	4.24
44	2.03	0.11	123	0.81	—	189	—	0.13
45	—	0.07	128	2.07	0.76	190	0.08	0.79
46	—	0.02	129	0.23	0.66	191	—	0.18
47	0.17	0.11	130	0.63	0.08	193	—	0.57
48	0.14	0.19	131	0.16	0.16	194	—	1.50
49	1.64	0.06	132	1.98	3.69	195	—	0.38
52	5.18	0.41	134	0.49	0.35	196	—	1.90
53	0.09	0.04	135	1.62	2.56	197	—	0.12
56/60	0.56	0.14	136	1.12	1.82	198	—	0.09
63	0.05	—	137	0.25	0.14	199	—	0.82
64	0.45	—	138	3.20	6.31	200	—	0.62
66	0.59	—	141	1.04	2.53	201	0.68	1.95
67	0.09	—	144/135	—	1.5	203	—	2.05
70	3.21	0.12	146	0.83	1.39	205	—	0.13
74	0.78	0.03	149	2.21	7.61	206	—	0.65
82	0.95	0.112	151	1.17	3.08	207	—	0.07
83	0.45	0.04	153	4.26	10.20	208	—	0.17
84	1.95	0.45	156	1.62	0.66	209	—	0.05
85	1.66	0.09	157	—	0.14			
87	3.78	0.61	158	0.77	0.70			
						Total (%)	96.25	105.55

^a Bush et al. (1985), Safe et al. (1985), Schulz et al. (1989), Smith et al. (1990).^b — = no data.

The patterns were compared with known amounts of Aroclor mixtures. If the Aroclor peaks in a sample closely resembled a particular Aroclor reference mixture of known weight, the total area or peak height of the sample PCBs was compared to those of the reference mixture and the weight of calculated sample PCBs (Kaiser et al. 1980). Other investigators used selected peaks to report Aroclor equivalents (Draper et al. 1991; Turle et al. 1991), but these methods are not useful when samples and Aroclor standards are dissimilar. For another procedure, response factors were used for individual Aroclor peaks as determined by GC and GC-MS procedures (Webb and McCall 1973). The sample peaks were compared with peaks from common Aroclors obtained on packed columns; retention time windows for the early, middle, and late-eluting peaks were assigned to 3 Aroclors

(1242, 1254, 1260) based on the presence or absence of specific peaks. The weight of PCB in the sample peak was calculated by multiplying its peak area by the appropriate response factor; all peaks were added to obtain a total weight of PCB. However, packed columns failed to separate and to identify many congeners because several congeners usually eluted under a single peak of identical retention time. These methods do not account for changes in composition from: interfering compounds; congener changes from hydrolysis, photodegradation, and biodegradation; selective evaporation and adsorption of certain isomers; or solubility differences resulting in different partitioning ratios among the various environmental compartments. Some researchers, who used capillary columns, estimated PCB residues on the basis of a relatively simple cleanup and analysis by high

Table 7. Effect of oven drying of sediments on percent loss of selected PCB congeners present at >0.1 mg/kg fresh weight (Bush et al. 1987). Mean loss of these congeners is 56 percent.

PCB number	Percent loss	PCB number	Percent loss
1	65	42	0
4	61	44	0
6	50	46	46
8	50	47	43
9	50	49	47
10	62	51	53
15	100	52	44
16	43	59	50
17	43	60	33
18	57	64	45
19	53	66	50
22	50	70	32
25	40	82	40
26	50	84	50
28	43	94	34
31	33	136	0
40/41	50	151	0

resolution GC-ECD (Duinker et al. 1988b; Maack and Sonzogni 1988; Sericano et al. 1990; Draper et al. 1991) or by HRGC/MS (Porte et al. 1988; Niimi and Oliver 1989; Harrad et al. 1992); others emphasized total PCBs, homolog subgroups, or individual congeners present in substantial amounts in the sample or in commercial mixtures (Maack and Sonzogni 1988; Niimi and Oliver 1989). And still others used total PCBs derived from the sum of homolog subgroup concentrations (Gebhart et al. 1985; Sericano et al. 1990), concentrations of individual subgroups (Gebhart et al. 1985), or the contribution of all congeners in each subgroup (Niimi and Oliver 1989).

Gas chromatography/mass spectrometry in the electron ionization mode (GC/MS-EI) has been used to a more limited extent for routine analysis of PCBs (Alford-Stevens et al. 1985; Kuehl et al. 1991). Some interference with quantification ions can occur with EI when compounds such as PCBs 77 and 110 coelute. GC/MS has also been operated in the negative chemical ionization mode (NCI) for PCB determinations (Guevremont et al. 1987; Swackhammer et al. 1987; Erhardt-Zabik et al. 1990). Increased selectivity is observed in the NCI mode, although interference from other compounds that readily form stable negative ions may be observed. Improvements have also been made in capillary column separations. However, the application of single long, narrow bore capillary columns does not enable investigators to attain full separation of PCB congeners (Ballschmiter and

Zell 1980; Bush et al. 1983; Mullin et al. 1984; Duinker et al. 1988b). Multidimensional GC (MDGC) analysis of PCBs drastically improved congener separations and the analysis of all sample constituents. Schomburg et al. (1985) connected two capillary columns of different polarities in a double oven instrument; the first column was temperature programmed, and the second was operated isothermally. Congeners of Clophen A 50 were separated by valveless flow switching. Using MDGC techniques, Duinker et al. (1988a, 1988b) analyzed PCBs 28, 52, 101, 138, 153, and 180 and positively identified PCBs 52, 101, and 180 as well as coeluting peaks of PCBs 24, 26, 29, 44, 49, 81, 84, 114, 128, 151, 169, 177, 183, 187, 189 and 194. In a following paper, Schulz et al. (1989) used the same MDGC technique to fully resolve all congeners in commercial PCB mixtures of Clophen A30, A40, A50, A60 and in Aroclors 1221, 1016, 1242, 1254, and 1260. A column of SE-54 with ECD was used to monitor the eluate, and a second, more polar column (usually OV-210) with ECD was used to obtain fully resolved single peaks. The procedure required exact timing of cuts based on retention time but fully separated the PCB congeners by gas chromatography for the first time. A total of 132 congeners at concentrations above 0.05% (w/w) were eluted as fully resolved single peaks and measured (Schulz et al. 1989).

Pattern recognition techniques that incorporate statistical methods have been used to determine spatial and temporal patterns in PCB residue data. Stalling et al. (1980, 1987), for example, characterized sample PCB profiles from 105 individual congeners, measured parameters of similarity between sample and standard mixtures, and determined whether the sample residue pattern corresponded to an Aroclor mixture. Modeling environmental samples with the individual congener concentrations provided more accurate estimations of Aroclor profiles than homolog concentrations. Intact commercial Aroclors have been characterized by automated mass spectrometric determination of weight percent distribution by homolog groups (Alford-Stevens 1986). One isomer from each level of chlorination was normally used to calibrate the MS response to all measurable isomers in that group. In another example, Macdonald et al. (1992) applied pattern recognition techniques to assess biomagnification and to characterize source patterns of multicomponent pollutants such as PCBs, PCDDs, and PCDFs in eggs of the herring gull (*Larus argentatus*) from the Great Lakes between 1983 and 1990. Turle et

al. (1991) fitted a linear regression to PCB concentrations in herring gull eggs from the Great Lakes during 1970-85 and from more recent measurements. Aroclor equivalents in eggs were determined with PCB 138 as a single peak estimate for the older data and more recent PCB data as the sum of 41 congeners. The earlier choice of PCB 138 for quantitation seemed fortunate; uptake of heptachloro isomers maintained a stable percentage of total PCB egg residues over time, whereas less chlorinated congeners generally declined and more chlorinated congeners increased over time (Turle et al. 1991).

Methods were developed for routine analysis of AHH-inducing and other PCB congeners in fish by using a comparatively simple gel permeation chromatography (GPC) cleanup and GC/MS-NCI (Schmidt and Hesselberg 1992). Methane was used as the reagent gas for NCI, source temperature and pressure were optimized (1100° C and 0.9 Torr), and fragmentation of the major ions was reduced as much as 10%. A peak area correction factor for interfering ion fragmentation was obtained by instrument calibration with standards and manually applied to coeluting ions as needed (Schmidt and Hesselberg 1992). Additional methods were developed for the analysis of planar congeners. Using a carbon foam adsorbent, Huckins et al. (1978, 1980, 1988) devised procedures for the analysis of toxic non-*ortho*-chloro substituted PCB congeners and trace planar impurities in Aroclors. With carbon-foam chromatography (carbon particles suspended on a polyurethane substrate), concentrations of planar PCB congeners in Aroclors 1016, 1242, 1248, 1254, and 1260 were detected by high resolution electron capture gas chromatography (HRGC/ECD). With current chemical methods for analysis of non-*ortho*-chloro substituted planar PCBs, extraction and a preliminary cleanup, carbon chromatography, and HRGC/ECD or HRGC/MS are generally used (Feltz et al. 1995). The brands of carbon available for isolating planar PCBs include Amoco AX-21 (PX-21), Alltech SK-4, Serva SP-1, and Wako active carbon (Storr-Hansen and Cederberg 1992). All methods shared at least three characteristics: (1) they require some form of adsorption chromatography (usually with carbon) to isolate planar compounds; (2) they include several clean-up steps; and (3) they are complicated.

A semipermeable membrane device (SPMD) with a nonpolar, low density polymeric film is used to separate PCBs from large amounts (20->50 g) of lipids by dialysis in an organic solvent prior to

chemical analysis (Huckins et al. 1990a; Meadows et al. 1993). Liquid-liquid phase partitioning of extracts in organic solvent with sulfuric acid has been used to convert fats and pigments into water-soluble compounds that can be separated and removed from the target analytes with water rinses. Fats may be removed by refluxing or partitioning extracts with alcoholic potassium hydroxide to form water soluble hydrolysis products. For example, Tanabe et al. (1987) and Kannan et al. (1987b) combined alkali digestion, active carbon column chromatography, fuming sulfuric acid clean-up, HRGC/ECD, and HRGC/MS confirmation for the analysis of PCBs 77, 126, and 169 in porpoise blubber. Measurement of these three highly toxic PCBs in Aroclor and Kanechlor mixtures is reported by Kannan et al. (1987a). Creaser and Al-Haddad (1989) separated five non-*ortho* substituted PCBs from a synthetic mixture containing Aroclors, organochlorine and organophosphorus pesticides, dioxins, and dibenzofurans on an HPLC column packing of porous graphite carbon (PGC). Soil samples required prior cleanup to remove coextracted organics, and elution from a multilayered column containing acid, base, and silica was followed by elution from Florisil.

Procedures are available for separating mono- and non-*ortho* chloro PCBs. Hong and Bush (1990) used sulfuric acid cleanup, low-pressure liquid chromatography with activated carbon/silica gel and HRGC/ECD to analyze four non-*ortho* and eight mono-*ortho* substituted PCBs. Haglund et al. (1990) used a non-carbon HPLC column of 2-(1-pyrenyl) ethyldimethylsilylated silica (PYE) column to isolate mono- and non-*ortho* chloro PCBs from tissue samples. Isolation with this column required almost complete prior removal of lipids by sulfuric acid partitioning and subsequent gel permeation chromatography (GPC) with Bio-Beads S-X3 to remove remaining lipids. Ford et al. (1993) adapted an automated dioxin analysis system with programmable pump and valve setup for the sequential processing of non-*ortho* substituted PCBs in five blubber samples. The procedure included ball/mill tissue extraction, preliminary GPC separation and cleanup, silica gel chromatography, and automated separation of non-*ortho* substituted PCBs on AX21 activated carbon/glass fiber with three solvent systems and was followed by GC-ECD and GC/MS-EI analysis with selected ion monitoring (SIM). The application of high resolution capillary column techniques combined with the development of carbon adsorbents for the separation of planar aromatics has become a powerful

tool for the identification and measurement of single congeners in complex PCB mixtures and has facilitated the resolution and more accurate quantification of individual congeners and the correction of the presence of non-PCB interferences. But the introduction of such methods in long-term monitoring programs, where one, several, or all peaks in Aroclor standard mixtures were used to estimate total PCBs, raises the problem of comparing results among the data sets. For such purposes a value for total Aroclor can also be reported (Porte et al. 1988; Turle et al. 1991).

Concentrations in Field Collections

General

An increasing number of reports indicate the widespread presence of toxic planar PCB congeners such as the non-*ortho* substituted planar PCBs 77, 126, and 169. These PCB congeners were detectable in all Finnish food commodities of animal origin sold in Helsinki (Himberg 1993); in all samples of salmon muscle, cod liver, and seal blubber from the Baltic Sea and environs in the 1980s (Koistinen 1990); and at concentrations between 0.03-30 $\mu\text{g/kg}$ fat fresh weight (FW) in a wide variety of vertebrates, including fish, marine mammals, dogs, cats, and humans (Tanabe et al. 1987). These and other planar congeners contribute the majority of the toxic potency to PCB mixtures as judged by their ability to induce AHH and EROD (Kannan et al. 1989; Ankley et al. 1991; Sonzogni et al. 1991). Detection of these toxic residues in field collections from remote areas suggests that planar PCBs are now as widely distributed as other PCB isomers (Tanabe et al. 1987). The clear positive correlation between concentrations of total PCBs and PCBs 77, 126, and 169 in all analyzed mammals suggests that the sources of planar PCB contamination to the environment are mainly commercial PCB formulations (Tanabe et al. 1987).

Nonbiological Materials

Relatively little contamination from PCBs was found in sediments from riverine and pothole wetlands at national wildlife refuges and waterfowl production areas (WPA) in the north central United States in 1980-82. PCBs were above detection levels (20 $\mu\text{g/kg}$) in less than 4% of the sedi-

ments; a similar case was recorded in fish from WPAs (Martin and Hartman 1985). Maximum total PCB concentrations in field collections of non-biological materials were 0.000028 $\mu\text{g/kg}$ in ice, 0.000125 $\mu\text{g/kg}$ in snow, 12.3 $\mu\text{g/m}^3$ in air, 233 $\mu\text{g/L}$ in seawater, 3,860 $\mu\text{g/L}$ in sediment interstitial waters, and 1,800 mg/kg in sediments. Concentrations were comparatively elevated in urban areas, near anthropogenic activities, and at known sites of PCB contamination (Table 8).

Atmospheric transport is a major route in PCB distribution (Swackhammer et al. 1988). Deposition and evaporation studies of 17 PCB congeners in Siskiwit Lake on Isle Royale, Michigan, showed that PCB input fluxes to the lake from rain, snow, and aerosol equalled output fluxes from sedimentation and evaporation. The magnitude of the net vapor flux, calculated by difference and with the assumption that inputs equal outputs, was large and positive for almost all 17 congeners (Swackhammer et al. 1988). Low but measurable PCB concentrations (measured as Aroclor 1254) were found in air, snow, ice, seawater, and sediment in the Arctic Ocean north of Axel Heiberg Island off Canada's northern outer coast (Hargrave et al. 1989). PCB residues were relatively high in melted snow (8-125 pg/L), indicating efficient scavenging from air. The ice island area from which surface seawater samples were collected was well removed from any direct influence of river drainage, and PCB concentrations probably reflect those over much of the Arctic Ocean (Hargrave et al. 1989). PCBs were also monitored in the atmosphere of Ross Island, Antarctica, from March 1988 to January 1990 (Larsson et al. 1992). The geometric mean of total PCBs in air during this period was 15.2 pg/m^3 and the maximum concentration was 12,300 pg/m^3 . The geometric mean during the Antarctic summers of 1988-89 and 1989-90 was 21 pg/m^3 . During one sampling period (16-28 December 1988), PCB levels were about 100 times higher than during any other period, suggesting either irregular, long-range transport of atmospheric pollutants or volatilization of PCBs from a local dumpsite at McMurdo Base on Ross Island. PCB levels did not correlate with seasonal temperature changes, although changes in atmospheric levels were recorded. The absence of seasonal differences may be due to the cold climate and the low vapor pressure of PCBs (Bidleman et al. 1983).

PCB concentrations in sediment cores from Lake Ontario were similar to production and sales data of PCBs in the United States (Eisenreich et al. 1989). Annual PCB accumulation rates in the

Table 8. PCB concentrations in field collections of selected nonbiological materials. Concentrations are in $\mu\text{g/kg}$ fresh weight (FW) or dry weight (DW) unless indicated otherwise.

Material	Concentration ^a	Reference ^b
Air ($\mu\text{g}/\text{m}^3$)		
Antarctica, Ross Island; 1988-90		
PCB 95	Max. 0.000415 FW	1
PCB 101	Max. 0.000574 FW	1
PCB 110	Max. 0.000444 FW	1
PCB 138	Max. 0.00181 FW	1
PCB 149	Max. 0.00161 FW	1
PCB 153	Max. 0.00128 FW	1
Total PCBs	0.015 FW; Max. 12.3 FW	1
Arctic Ocean; 1986-87; total PCBs	<0.00001 FW	2
Ice		
Arctic Ocean; 1986-87; total PCBs	Max. 0.000028 FW	2
Seawater		
Arctic Ocean; 1986-87; total PCBs; water column vs. particulates under ice	Max. 0.00001 FW vs. 2-99 DW	2, 3
Massachusetts; New Bedford Harbor (PCB-contaminated); 1986 total PCBs; bedded phase vs. suspended phase	15 FW vs. 233 FW	4
New York Bight dumpsite; 1970s-1980s; total PCBs	Max. 0.04 FW	5
Sediment interstitial waters		
New Bedford Harbor; 1986; total PCBs	3,860 FW	4
Sediments		
Arctic Ocean; 1986-87; total PCBs	<0.05 DW	2
Canada, Lake Ontario		
Cores; total PCBs		
1940	0-10 FW	6
1966-69	470-880 FW	6
1980	250-290 FW	6
Surficial sediments; 1981		
Total PCBs	(71-1,200) FW	7
Tri-CBs	(11-22) FW	7
Tetra-CBs	(77-200) FW	7
Penta-CBs	(76-180) FW	7
Hexa-CBs	(41-93) FW	7
Hepta-CBs	(20-48) FW	7
Octa-CBs	(11-22) FW	7
Nona-CBs	(2.4-5.7) FW	7
Deca-CB	(5.3-9.4) FW	7
Settling sediments; total PCBs		
1982-83	1,300-1,900 FW	7
1983-84	350-500 FW	7
1984-85	410-680 FW	7
1985-86	80-290 FW	7
Sweden, Eman River; near paper recycling plant		
PCB 77	30.0 DW	8
PCBs 126, 169	Nondetectable	8
Switzerland, Lake Zurich; total PCBs		
1929-34	<0.5 DW	9
1960-65	210 DW	9
1975-80	70 DW	9
United States		
California; total PCBs		
Hunter's Point	40 DW	10
Islais Creek	164 (57-255) DW	11
Long Beach; 1984-89	>200,000 DW	12
Oakland Bay	30-61 DW	10, 11
Palos Verdes; 1984-89	>200,000 DW	12
San Diego Bay; 1984-89	>200,000 DW	12
San Francisco Bay	Max. 1,400 DW	13
San Pablo Bay	11.4 (5.7-17.5) DW	10, 11
San Pedro Bay; 1984-89	>200,000 DW	12
Santa Monica Bay; 1984-89	>200,000 DW	12
Southampton Shoal	12 DW	10
Connecticut; 1984-89; total PCBs		
Connecticut River	>200,000 DW	12
Long Island Sound	>200,000 DW	12
Florida; 1984-89; total PCBs		
Choctawhatchee Bay	>200,000 DW	12

Table 8. Continued.

Material	Concentration ^a	Reference ^b
St. Andrews Bay	>200,000 DW	12
Tampa Bay	>200,000 DW	12
Illinois, Waukegan Harbor; 1978		
PCB 77	1.05 (0.005-27.5) DW	14
PCB 105	0.86 (0.10-131.0) DW	14
Total PCBs	515 (11-13,360) DW	14
Maryland; 1984-89; total PCBs		
Baltimore Harbor	>200,000 DW	12
Upper Chesapeake Bay	>200,000 DW	12
Michigan		
Raisin River; 1983		
PCB 77	0.6 DW	15
PCB 126	0.28 DW	15
PCB 169	0.07 DW	15
Total PCBs	40,000 DW	15
Saginaw River; 1984		
PCB 77	0.017 DW	15
PCB 126	<0.015 DW	15
PCB 169	ND	15
Massachusetts; total PCBs		
Boston Harbor and Buzzards Bay; 1984-89	>200,000 DW	12
New Bedford Harbor; 1986	1,800,000 FW	4
Salem Harbor; 1984-89	>200,000 DW	12
Nebraska; waterfowl production areas; 1980-82; total PCBs	ND	16
New Jersey; Raritan Bay; total PCBs		
1970s	(3-2,035) DW	17
1984-89	>200,000 DW	12
New York		
Hudson-Raritan estuary and Long Island Sound; 1984-89; total PCBs	>200,000 DW	12
Upper Hudson River; anoxic sediments		
PCB 1	200,000 DW	18
PCB 4	160,000 DW	18
PCBs 6/19	40,000 DW	18
PCB 8	33,000 DW	18
PCB 10	75,000 DW	18
PCB 18	7,800 DW	18
PCB 31	12,000 DW	18
PCB 32	12,000 DW	18
PCB 47	12,000 DW	18
PCB 49	21,000 DW	18
PCB 50	9,200 DW	18
PCB 52	30,000 DW	18
PCB 64	11,000 DW	18
PCBs 82/85/110	12,000 DW	18
PCB 92	5,500 DW	18
Others	52,000 DW	18
Ohio, Cuyahoga River; 1984		
PCB 77	0.09 DW	15
PCB 126	0.011 DW	15
PCB 169	<0.01 DW	15
Rhode Island, Narragansett Bay; 1984-89; total PCBs	>200,000 DW	12
South Carolina, Lake Hartwell; total PCBs		
Cores; 1984-87	Max. 153,840 DW	19
Cores; distance from source, in km		
33.2	40,500 DW; Max. 88,500 DW	20
78.2	380 DW; Max. 1,100 DW	20
South Dakota; waterfowl production areas; 1980-82; total PCBs	ND	16
U.S. National Wildlife Refuges; North Central area; 1980-82; total PCBs	ND-1 DW	16
Virginia; Elizabeth River; 1984-89; total PCBs	>200,000 DW	12
Washington; total PCBs		
Elliot Bay; 1984-89	>200,000 DW	12
Puget Sound; 1980s		
Main basin	93 DW	21
Nonurban bays	15-34 DW	21
Urban bays	750 DW	21

Table 8. Continued.

Material	Concentration ^a	Reference ^b
Wisconsin		
Fox River; 1984		
PCB 77	1.4 DW	15
PCBs 126, 169	ND	15
Fox River; 1988		
Total PCBs	22,000-41,450 DW	15, 22
Total AHH-active PCBs	710 DW	15
PCB 77	8.5 DW	15
PCB 105	6 DW	15
PCB 118	17 DW	15
PCB 128	13 DW	15
PCB 138	87 DW	15
PCB 156	2 DW	15
PCB 158	1.5 DW	15
PCB 170	31 DW	15
Green Bay; 1984		
PCB 77	0.94 DW	15
PCB 126	0.024 DW	15
PCB 169	<0.005 DW	15
Lake Pepin; 1983		
PCBs 77, 126, 169	<0.002-0.002 DW	15
Total PCBs	56 DW	15
Menominee River; 1984		
PCB 77	0.1 DW	15
PCB 126	0.04 DW	15
PCB 169	0.003 DW	15
Snow		
Arctic Ocean; 1986-87; total PCBs	Max. 0.000125 FW	2

^a Concentrations are shown as mean, range (in parentheses), maximum (Max.), and nondetectable (ND).

^b 1, Larsson et al. 1992; 2, Hargrave et al. 1989; 3, Hargrave et al. 1992; 4, Burgess et al. 1993; 5, Boehm 1981; 6, Eisenreich et al. 1989; 7, Oliver et al. 1989; 8, Asplund et al. 1990; 9, Buser and Muller 1986; 10, NOAA 1987; 11, Chapman et al. 1986; 12, NOAA 1991; 13, Law and Goerlitz 1974; 14, Huckins et al. 1988; 15, Smith et al. 1990; 16, Martin and Hartman 1985; 17, Stainken and Rollwagen 1979; 18, Rhee et al. 1989; 19, Elzerman et al. 1991; 20, Dunnivant et al. 1989; 21, Ginn and Pastorok 1992; 22, Ankley et al. 1992.

sediments rose from about 2 ng/cm² in 1950 to about 40 ng/cm² in the 1966-69 peak years and declined in 1980 to 10-20 ng/cm²; about 50% of the 1966-69 load was attributed to the upward mixing by oligochaete worms (Eisenreich et al. 1989). Each of several PCB congeners in Lake Ontario sediments contributed 5.7 to 7.9% of the total PCBs (PCBs 66, 110, and 56/60) and others (including PCBs 44, 52, 70/76, 101, and 153) contributed 4.0-4.7% (Oliver and Niimi 1988; Oliver et al. 1989). Surficial sediments of Lake Ontario in 1981-86 contained elevated concentrations of organochlorine compounds, including mirex, chlorobenzenes, octachlorostyrenes, DDT, 2,3,7,8-TCDD, fluorinated aromatic compounds, and PCBs (Oliver et al. 1989). Among the identified congeners, PCBs 60 (5.7%) and 118 (2.6%) are AHH-active (Smith et al. 1990). The frequency of isomers of low chlorination decreased with core depth in sediments, remained fairly stable of hexa isomers, and increased with core depth of the more highly chlorinated congeners. This pattern with

depth reflects the change in use pattern over time to less highly chlorinated PCBs such as Aroclor 1016. The more highly chlorinated congeners that are disproportionately present in deeper sediments may be due to stronger partitioning to sediment and higher hydrophobicity than those of less chlorinated congeners and to the positive correlation with their high octanol-water partition coefficients (Karickhoff 1981). Anaerobic dechlorination was not evident in these sediments. This contradicts the findings of others who maintain that relatively high levels of less-chlorinated PCBs in the bottom (anaerobic) core sections of Hudson River sediments were due to anaerobic dechlorination (Brown et al. 1985). Oliver et al. (1989) concluded that the total mass of PCBs in Lake Ontario sediments was about 50 tons and sufficient to impact Lake Ontario for many years. Lake Michigan sediments, analyzed for 18 planar AHH-active PCBs, total PCBs, 2,3,7,8-TCDD, and 2,3,7,8-tetrachloro-*p*-dibenzofuran (2,3,7,8-TCDF), had elevated levels of PCBs 77, 126, and 169 (Smith et

al. 1990). Results suggest that the contribution of toxic equivalents—that is, the sums of congeners after the raw congener concentrations were normalized by the TEF—was greater from PCBs 77, 126, and 169 than from 2,3,7,8-TCDD and 2,3,7,8-TCDF, even in environments with significant concentrations of dioxins and dibenzofurans (Smith et al. 1990). Sediments from the Waukegan Harbor, Illinois, in 1978 contained weathered mixtures of Aroclors 1242, 1248, and 1254. Total PCB concentrations were variable between stations and ranged from 10.6 mg/kg to 13,360 mg/kg DW; 3,3',4,4'-TCB residues ranged from 5 to 27,500 µg/kg DW, or about 0.16% of the total PCBs; concentrations were higher of 2,3,3',4,4'-PCB (102 to 131,000 µg/kg DW, or 0.66%; Huckins et al. 1988).

PCB homologs and total PCBs in water, sediments, and biota were measured in Hamilton Harbour on Lake Ontario and Wheatley Harbour on Lake Erie. Hamilton Harbour receives inputs from steel mills and an incinerator plant; Wheatley Harbour receives fish-processing plant wastes. Total PCBs in sediments ranged from 608 to 14,185 µg/kg DW in Hamilton Harbour and 166-1,177 µg/kg DW in Wheatley Harbour (Mudroch et al. 1989). The high PCB value in Hamilton Harbour is similar to values reported earlier in Lake Erie sediments (Frank et al. 1977). Concentrations of lower chlorinated homologs were greater in water than sediment in both harbors (Mudroch et al. 1989). Homolog patterns in biota (oligochaetes, snails, isopods, and fish) reflected patterns in sediments but not water. Concentrations of penta- and hexachlorobiphenyls were dominant in the oligochaetes and sediment samples from Wheatley Harbour; concentrations of these homologs also predominated in Hamilton Harbour, but the differences were less pronounced. Particle size distribution of 82-98% silt and clay (63 µm) was similar in both harbors. The concentrations of several metals (Zn, Pb, Cu, and Cr) were much higher in Hamilton Harbour, but their effect on PCB bioaccumulation is unknown (Mudroch et al. 1989).

Contaminated sediments are a major source of PCBs in aquatic environments (Dillon and Burton 1992). PCB discharges prior to 1977 from capacitor manufacturing plants on the Hudson River at Ft. Edward and Harbor Falls, New York (14 kg PCBs daily for 30 years, mostly Aroclors 1242 and 1016), contaminated 306 km of river bed between Hudson Falls and New York Harbor. By 1978, an estimated 63,500 kg of PCBs were in the river bank deposits, 134,000 kg in the upper river, and 91,000 kg in the lower river (Brown et al. 1985; Eisler

1986; Bush et al. 1987; Kennish 1992). PCB concentrations in biota and the water column are largely controlled by PCB concentrations in surficial sediments. Declines in PCB levels in Hudson River sediments corresponded to decreased use of PCBs at local capacitor-manufacturing plants (Brown et al. 1985; Kennish 1992). The stabilization of highly contaminated upper stream banks and reduced PCB releases from bed sediments contributed to lower concentrations in the water column. A model of the fate and accumulation of 7 PCB homologs in the Hudson River estuary showed that 66% of the 270,454 kg of total PCBs discharged into the estuary between 1947 and 1987 had volatilized, 6% was stored in sediments, and the rest was either dredged or lost by boundary transport to the New York Bight and Long Island Sound (Thomann et al. 1991). Total PCBs peaked in the mid-1970s and upstream loading now has a relatively small effect. The upstream PCB load above Troy, New York, accounts for about 20% of the PCB concentrations in striped bass. A steady decline of Aroclors 1016 and 1254 in upstream contribution was projected to the year 2005 and suggests that 95% of 3-6 year old striped bass in the lower Hudson would have residues below the USFDA action level of 2 mg/kg FW by the year 2004 (Thomann et al. 1991).

PCB congener patterns in Hudson River sediments differed from the Aroclors discharged into the river. Sediments had comparatively enriched concentrations of certain lower-chlorinated congeners, especially *ortho*-substituted congeners, and some congeners not usually detected in commercial Aroclor mixtures. Changes in PCB composition of sediments were greater in lower portions of sediment cores than in surficial sediments (Brown et al. 1987). Two dechlorination mechanisms seemed to be operating: *meta*, *para*-dechlorinations with step-wise, selective dechlorination at the *meta* and *para* positions of certain *ortho*-substituted di- through tetra-*ortho* chlorinated biphenyls; and *ortho*, *meta*, *para*-dechlorinations, in which the dechlorinations occur at *ortho*, *meta*, or *para* positions and reactivity is favored by increasing electron affinity and relatively positive reduction potential. Both mechanisms preferentially removed toxic congeners (Brown et al. 1987). Residues of individual PCB congeners in the upstream water were similar to residues in caddisfly larvae from that system (Bush et al. 1985), although residues in the larvae were enriched with lower chlorinated congeners. Because of their relatively high water solubility and low affinity for

sediment, higher concentrations of lower chlorine homologs were expected to preferentially dissolve in water. But PCB profiles of dissolved residues transported downstream in the water samples during low flow season were dominated by only three low chlorinated PCBs; more than half of the residues consisted of 2-chlorobiphenyl and 2,2'- and 2,6' dichlorobiphenyl congeners (Bush et al. 1985).

Anoxic Hudson River sediments contaminated with PCBs and dredged sediments in clay encapsulation were treated to induce or raise anaerobic biodegradation of PCBs (Rhee et al. 1989). Variations in sediment type, bacterial flora, and treatment influenced dechlorination. The addition of biphenyl under anoxic nitrogen was the most effective treatment for raising biodegradation. Unlike the findings of Brown et al. (1987), no evidence was found of accumulation of less chlorinated PCBs from biodegradation of more highly chlorinated congeners, although a faster degradation rate of less chlorinated congeners than the more highly substituted PCBs would have allowed this phenomena to occur unobserved. Hudson River sediments incubated at room temperature (25° C) under a nitrogen atmosphere or incubated with biphenyl enrichment under nitrogen for 7 months decreased significantly in chlorobiphenyl compounds of 33 to 65%. In general, mono- and dichlorobiphenyl congeners from Hudson River sediments were not significantly degraded by the biphenyl, but biphenyl addition significantly decreased the higher chlorinated congeners (Rhee et al. 1989). In earlier studies with the same mixed bacterial culture without biphenyl addition, significant reductions were evident in Hudson River sediments of PCB congeners with as many as three chlorines (Chen et al. 1988).

Sediments in oceans, estuaries, rivers, and lakes concentrate PCBs. Organisms accumulate PCBs by way of the water column, from interstitial sediment waters, and from consumption of contaminated prey by predator species. Coastal sediments with the highest total PCB concentrations between 1984 and 1989 are usually within 20 km of population centers of more than 100,000 people (NOAA 1991) and directly correlate with sediment content of total organic carbon (Karickhoff 1981). Variability in PCB content of sediments is great, and large differences between adjacent stations is not uncommon (NOAA 1988). The approximate percent PCB distribution in sediments by level of chlorination was di-3.9%, tri- 11%, tetra- 20%, penta- 24%, hexa- 21%, hepta-12%, octa- 3.9%, and

nona- 1.3%. The mean PCB concentration in sediments at 233 sites was 39 µg/kg, and concentrations greater than 200,000 µg/kg were considered elevated (NOAA 1988; Table 8). In most regions of New Jersey, for example, PCB contamination of sediments and water column was negligible (Kennish 1992). But PCBs in estuarine and coastal locations of New Jersey showed elevated sediment contamination in the northeastern region—an area that included the Hudson-Raritan estuary and adjacent ocean waters of the New York Bight—where direct discharges from capacitor manufacturing plants on the upper Hudson River, dredged material, and sewage-sludge dumping were the principal sources of PCB contamination. PCB levels in teleosts from the northeastern region in 1986-87 exceeded the USFDA action level of 2,000 µg/kg and were consistent with data from previous years. Action levels were also exceeded in fishes from Camden and from the northern coast regions (Kennish 1992). PCB sediment residues in Raritan Bay in the 1970s averaged 100 µg/kg DW in a range of 3.4 to 2,035 µg/kg (Stainken and Rollwagen 1979). Water column concentrations were usually not detectable but approached 0.04 µg/L in parts of the New York Bight after sewage sludge and dredged material were deposited in the 1970s and early 1980s (Boehm 1981). Sediments near dumpsites in the New York Bight contained as many as 15,000 µg/kg PCBs (MacLeod et al. 1981), and bottom sediments in the upper Hudson River had localized contaminated areas containing more than 50,000 µg/kg (Brown et al. 1985). Nation-wide, PCB loadings in surficial sediments and mussels (*Mytilus edulis*) in the Long Island Sound were greater than in other sites (Robertson et al. 1991).

Surficial stream sediments in the San Francisco estuarine system during 1972 had high residues (350-1,400 µg/kg DW) at several locations (Phillips and Spies 1988); however, residues were highly variable between sites (Law and Goerlitz 1974). In 1984, low to intermediate concentrations of PCBs (9-60 µg/kg DW) were measured at four locations in the San Francisco Bay. Overall, congener profiles resembled Aroclor 1254 and were dominated by pentachlorobiphenyl isomers (NOAA 1987). The patterns of the 11 reported congeners were variable between areas, indicating many sources of different PCB mixtures.

Lake Hartwell in South Carolina received Aroclor 1016 and 1254 discharges for 21 years from a capacitor manufacturing plant (Elzerman et al. 1991). Analysis of sediment cores collected

between 1984 and 1987 showed that samples nearest the source were relatively high in lower chlorinated PCB congeners, and downstream samples were enriched with the higher congeners; in all samples, total PCB concentrations decreased with increasing distance from the point source. The estimated total remaining PCBs was 41,000 kg (Elzerman et al. 1991). The highest PCB concentrations were in subsurface sediments, although PCB levels in surficial sediments were also elevated (Dunnivant et al. 1989). A survey of total PCBs in sediments and invertebrates in major estuaries of South Carolina showed no significant contamination. PCBs were found only in sediments (622 µg/kg) and in oysters (87 µg/kg) from the Wando River near a large ship repair and retrofitting facility; PCB residues in crabs, when present, ranged from 95 to 375 µg/kg (Marcus and Renfrow 1990).

Aquatic Organisms

Marine mammals are the most vulnerable and most probable target organisms to PCBs (Tanabe 1988). The metabolic potential to degrade organochlorine contaminants and therefore accumulate relatively high concentrations of persistent PCBs is lower in marine mammals, particularly in cetaceans, than in terrestrial mammals (Tanabe et al. 1987; Kannan et al. 1989, 1993). Dead harbour porpoises (*Phocoena phocoena*) along the Dutch coast contained 51,350-139,790 µg total PCBs/kg blubber; 35-50% of the total PCBs were PCBs 138 and 153 (Duinker et al. 1988a); a similar pattern was noted in harbour porpoises from Scandinavia in 1987-91 (Kleivane et al. 1995). The intrinsic toxicity of PCBs mainly resulted from the planar PCB congeners that imposed a greater toxic threat to marine mammals than chlorinated dioxins and furans (Tanabe 1988; Tanabe et al. 1989; Daelamans et al. 1993; Falandysz et al. 1994b). The toxic threat of planar PCBs to higher aquatic predators was primarily assessed by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin toxic equivalent analysis, which is based on the induction of aryl-hydrocarbon hydroxylase (AHH) and ethoxyresorufin *O*-deethylase (EROD; Kannan et al. 1989). The concentrations of planar PCBs in marine mammals were higher (in the order of di-*ortho* > mono-*ortho* > non-*ortho* congeners) and significantly higher than the levels of toxic dioxins and furans (Tanabe 1988; Tanabe et al. 1989; Kannan et al. 1989). In particular, the accumulation of PCBs 105 and 126 in carnivorous aquatic mam-

mals is cause for considerable concern (Tanabe et al. 1987, 1989; Kannan et al. 1989; Daelamans et al. 1993; Storr-Hansen and Spliid 1993).

Declines in total PCB levels in blubber of marine mammals between the late 1960s and 1990-92 have been noted worldwide (Muir et al. 1988; Lake et al. 1995a), possibly because of the PCB ban in the mid-1970s. PCB levels in animal tissues will probably not decline in the near future because of the greater quantities of PCBs still in use than the quantity that already escaped into the open environment (Tanabe 1988). Temporal changes of PCBs in remote marine waters are slow and could be attributable to the large PCB load in the marine environment (Loganathan et al. 1990). The geographical distribution of planar PCBs with reference to total PCBs did not vary in terrestrial, coastal, and open ocean mammals, whereas those of dioxins and furans decreased from land to ocean (Tanabe et al. 1989). PCB concentrations—including planar PCBs—in blubber of striped dolphins (*Stenella coeruleoalba*) from the Mediterranean Sea in 1990 are among the highest reported in the literature (Table 9; Kannan et al. 1993). Striped dolphins affected by the western Mediterranean morbillivirus epizootic also contained extremely high concentrations of PCBs (including non- and mono-*ortho* planar congeners) and low immune suppression, suggesting that PCBs were a major factor in this epizootic; however, this needs verification. Planar PCBs in blubber of striped dolphins accounted for about 53% of total PCBs and for virtually all of the potential toxicity. Di-*ortho* planar PCBs accounted for 93.7% of all planar PCB residues in striped dolphins and for 21% of the potential toxicity, mono-*ortho* planars for 6.3% of the residues and 70.8% of the potential toxicity, and non-*ortho* planars for 0.03% of the residues and 8.2% of the potential toxicity (Kannan et al. 1993). Concentrations of PCBs in tissues of adult male striped dolphins of the same age from the Pacific coast of Japan did not change between 1979 and 1986 (Loganathan et al. 1990).

White whales (*Delphinapterus leucas*) of the St. Lawrence estuary were severely contaminated by DDT metabolites and PCBs; the highest residues were in blubber (Masse et al. 1986). Several PCB congeners known to be AHH inducers—including PCBs 138 and 153—were among the major PCB congeners detected in tissues of white whales. Because these compounds are not metabolized and persist indefinitely in tissues of the white whale, the integrated total exposure to these AHH inducers may be significant. Considerable

Table 9. PCB concentrations in field collections of selected aquatic organisms. Concentrations are in micrograms PCBs per kilogram (PPB) fresh weight (FW), dry weight (DW), or lipid weight (LW).

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
Invertebrates		
Bivalve mollusks (<i>Mytilus</i> , <i>Tapes</i> , <i>Ostrea</i> , <i>Crassostrea</i>); Catalonia, Spain, 1989-90; soft parts		
Sum of PCBs 28, 52, 101, 118, 138, 153, 180	1.4-596.0 DW	1
Crabs; southern Norway, 1990-92; hepatopancreas		
PCBs 28, 52, 66, 74, 99, 101, 110, 157	ND	2
PCB 77	Max. 3.4 LW	2
PCB 126	Max. 2.5 LW	2
PCB 169	Max. 1.1 LW	2
PCB 209	Max. 2,600 LW	2
Total of PCBs 105, 118, 128, 138, 149, 153, 156, 170, 180, 187, 194, 206	600-2,050 LW	2
Eastern oyster, <i>Crassostrea virginica</i> ; soft parts; 1990-1991; Galveston Bay, Texas vs. Tampa Bay Florida; maximum values		
PCB 77	2.0 DW vs. 1.5 DW	45
PCB 105	39.0 DW vs. 7.6 DW	45
PCB 118	48.0 DW vs. 36.0 DW	45
PCB 126	2.2 DW vs. 0.3 DW	45
PCB 128	4.4 DW vs. 2.0 DW	45
PCB 138	50.0 DW vs. 8.9 DW	45
PCB 169	0.79 DW vs. 0.28 DW	45
Mayfly, <i>Hexagenia bilineata</i> ; upper Mississippi River, summer 1988; total PCBs (sum of 125 congeners)	210-4,100 DW; 1,200-29,000 LW	46
Mayfly, <i>Hexagenia limbata</i> ; Lake St. Clair, July 1987, sediments vs. whole adults		
PCB 87	1.2 DW vs. 0.7 FW	34
PCB 101	2.9 DW vs. 1.7 FW	34
PCB 118	2.1 DW vs. 0.9 FW	34
PCB 138	2.4 DW vs. ND	34
PCB 153	1.8 DW vs. 0.9 FW	34
PCB 180	0.8 DW vs. 0.6 FW	34
Burrowing mayfly, <i>Hexagenia</i> sp., adults; summer 1987; contaminated sites on Detroit and St. Clair rivers		
PCB 18	Max 7.6 DW	35
PCB 31	Max. 2.8 DW	35
PCB 52	Max. 10.9 DW	35
PCB 66	Max 4.6 DW	35
PCB 87	Max. 6.5 DW	35
PCB 97	Max 3.5 DW	35
PCB 101	Max. 16.6 DW	35
PCB 110	Max. 6.0 DW	35
PCB 118	Max. 13.6 DW	35
PCB 138	Max. 18.1 DW	35
PCB 141	Max. 6.1 DW	35
PCB 153	Max. 23.2 DW	35
PCB 170	Max. 4.8 DW	35
PCB 180	Max. 10.4 DW	35
PCB 182	Max. 7.3 DW	35
PCB 194	Max. 4.7 DW	35
Squid, <i>Illex illecebrosus argentinus</i> ; near Falkland Islands, March 1988; muscle		
PCBs 28, 31, 44, 47, 49, 52, 66, 87, 97, 101, 105, 110, 118, 128, 138, 141, 149, 151, 153, 170, 180, 184, 187, 206	ND (= nondetectable)	3
Common mussel, <i>Mytilus edulis</i> German Bight, 6 locations; 1993; extractable organic matter; spring vs. autumn		
PCB 77	33-50 DW vs. 4-8 DW	49
PCB 81	5-7 DW vs. 2-4 DW	49
PCB 105	45-60 DW vs. 37-130 DW	49
PCB 114	Max. 1.1 DW vs. Max. 0.5 DW	49
PCB 118	75-97 DW vs. 65-520 DW	49
PCB 123	9-150 DW vs. 0.2-4 DW	49
PCB 126	4-15 DW vs. 6-12 DW	49
PCB 138	280-420 DW vs. 83-210 DW	49

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 156	Max. 3.9 DW vs. Max. 1.4 DW	49
PCB 169	0.0-2.1 DW vs. 0.4-4.1 DW	49
PCB 170	0.0-7.9 DW vs. 2.2-4.1 DW	49
PCB 180	18-43 DW vs. 9-23 DW	49
PCB 189	0.5-1.7 DW vs. 0.1-0.4 DW	49
Long Island Sound, New York vs. freshwater mussels (species unknown) from a contaminated site near Troy, New York; soft parts		
Total PCBs	247 DW vs. 2,734 DW	4
Total planar PCBs	11.9 DW vs. 112.9 DW	4
PCB 77	0.4 DW vs. 6.9 DW	4
PCB 81	ND vs. 0.9 DW	4
PCB 105	3.3 DW vs. 27.0 DW	4
PCB 114	ND vs. 4.0 DW	4
PCB 118	8 DW vs. 53 DW	4
PCB 123	ND vs. ND	4
PCB 126	ND vs. 0.6 DW	4
PCB 156	ND vs. 1.8 DW	4
PCB 157	ND vs. 0.6 DW	4
PCB 167	0.2 DW vs. 18.6 DW	4
PCB 169	ND vs. 0.1 DW	4
PCB 189	ND vs. 0.1 DW	4
Fishes		
Bloater, <i>Coregonus hoyi</i> ; whole; Lake Michigan; total PCBs		
1972 vs. 1975	5,700 FW vs. 4,500 FW	5
1978 vs. 1982	3,100 FW vs. 2,100 FW	5
1986	1,600 FW	5
Margined flyingfish, <i>Cypselurus cyanopterus</i> ; near Falkland Islands, March 1988; muscle		
PCBs 28, 31, 44, 47, 49, 52, 66, 87, 97, 101, 105, 110, 128, 141, 151, 170, 194, 206	0.01-0.1 FW	3
PCBs 118, 138, 180, 187	0.14-0.31 FW	3
PCB 153	0.45 FW	3
Fish liver oil; various species; Finland		
PCB 77	2.7 LW	6
PCB 105	30.0 LW	6
PCB 126	0.62 LW	6
PCB 169	0.13 LW	6
Fish muscle; various species sold for human consumption; Finland		
PCB 77	0.006-0.153 FW	6
PCB 105	0.113-2.7 FW	6
PCB 126	0.002-0.035 FW	6
PCB 169	ND-0.012 FW	6
Fish, 14 species, muscle; Wisconsin, 1986-87; total PCBs	1,300 (700-7,000) FW	7
Freshwater fishes; USA; nationwide; whole; adults; total PCBs measured as Aroclors 1248, 1254, and 1260; noncontaminated sites		
1976-77	Max. 70.6 FW	48
1978-79	Max. 92.8 FW	48
1980-81	Max. 11.3 FW	48
1984	Max. 6.7 FW	48
Freshwater fishes; USA; nationwide; whole; 1986-87; mostly contaminated sites		
Median	209 FW	52
Mean	1,890 FW	52
Maximum	124,000 FW	52
Atlantic cod, <i>Gadus morhua</i> Liver; Norway, 1988 PCB 28		
PCB 28	19 (0.5-64) FW	8
PCB 52	31 (0.5-144) FW	8
PCB 101	78 (0.5-533) FW	8
PCB 118	170 (1.1-653) FW	8
PCB 138	283 (1.4-918) FW	8
PCB 153	363 (1.3-1,175) FW	8
PCB 170	28 (0.4-163) FW	8
PCB 180	68 (1.1-391) FW	8

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 209	7 (0.3-35) FW	8
Liver; Norway 1989		
PCB 77	Max. 4.8 LW	9
PCB 105	Max. 39.0 LW	9
PCB 126	Max. 1.0 LW	9
PCB 169	Max. 0.3 LW	9
Muscle; Baltic Sea, 1988		
PCB 77	Max. 4.2 LW	9
PCB 105	Max. 36.0 LW	9
PCBs 126, 169	ND	9
Ictalurids, 4 species (blue catfish, <i>Ictalurus furcatus</i> ; black bullhead, <i>Ameiurus melas</i> ; channel catfish, <i>Ictalurus punctatus</i> ; flathead catfish, <i>Pylodictis</i> <i>olivaris</i>); whole; 1,944 km stretch of Mississippi River; July-August 1987		
Total PCBs	Max. 138 FW; Max. 2,910 LW	10
Tetrachlorobiphenyls	Max 21 FW; Max. 671 LW	10
Pentachlorobiphenyls	Max 56 FW; Max. 1,230 LW	10
Hexachlorobiphenyls	Max 67 FW; Max. 870 LW	10
Heptachlorobiphenyls	Max. 12 FW; Max. 140 LW	10
Dab, <i>Limanda limanda</i>		
German Bight; December 1988-May 1989; total of 35 PCB congeners		
Females		
Liver (mostly PCBs 77, 138, and 153)	1,200-16,200 LW	36
Ovaries (mostly PCBs 77, 138, and 153)	2,600-4,300 LW	36
Males		
Liver	900-24,400 LW	36
Testes (mostly PCBs 15, 13, 52)	1,900-11,800 LW	36
North Sea; January-March 1987; total PCBs		
Liver, Females	1,450 FW	11
Liver, Males	2,000 FW	11
Ovaries	2,600 FW	11
Tilefish, <i>Lopholatilus chamaeleonticeps</i> ; 1981-1982; New Jersey vs. Georges Bank		
Total PCBs		
Gonad	3,373 DW vs. 1,004 DW	12
Liver	4,693 DW vs. 1,002 DW	12
Muscle	435 DW vs. 190 DW	12
Dichlorobiphenyls		
Gonad	196 DW vs. 15 DW	12
Liver	258 DW vs. 42 DW	12
Muscle	9 DW vs. 5 DW	12
Trichlorobiphenyls		
Gonad	164 DW vs. 11 DW	12
Liver	278 DW vs. 35 DW	12
Muscle	11 DW vs. 7 DW	12
Tetrachlorobiphenyls		
Gonad	1,176 DW vs. 192 DW	12
Liver	789 DW vs. 151 DW	12
Muscle	62 DW vs. 25 DW	12
Pentachlorobiphenyls		
Gonad	806 DW vs. 463 DW	12
Liver	1,273 DW vs. 358 DW	12
Hexachlorobiphenyls		
Gonad	664 DW vs. 191 DW	12
Liver	1,290 DW vs. 221 DW	12
Muscle	112 DW vs. 46 DW	12
Heptachlorobiphenyls		
Gonad	158 DW vs. 27 DW	12
Liver	109 DW vs. 27 DW	12
Muscle	14 DW vs. 3 DW	12
Nonochlorobiphenyls		
Gonad	62 DW vs. 6 DW	12
Liver	22 DW vs. 13 DW	12
Muscle	3 DW vs. 0.5 DW	12

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
Decachlorobiphenyl		
Gonad	45 DW vs. 6 DW	12
Liver	24 DW vs. 16 DW	12
Muscle	2 DW vs. 0.7 DW	12
PCB 77		
Gonad	48 DW vs. 19 DW	12
Liver	86 DW vs. 45 DW	12
Muscle	62 DW vs. ND	12
PCB 126		
Gonad	ND vs. 27 DW	12
Liver	214 DW vs. 33 DW	12
Muscle	19 DW vs. 4 DW	12
Argentinian hake, <i>Merluccius merluccius hubbsi</i> ; near Falkland Islands, March 1988; muscle		
PCBs 28, 31, 44, 47, 49, 87, 97, 105, 128, 141, 151, 194, 206	0.01-0.04 FW	3
PCBs 52, 66, 101, 110, 170	0.05-0.1 FW	3
PCBs 118, 138, 149, 153, 180, 187	0.11-0.28 FW	3
Striped bass, <i>Morone saxatilis</i> ; muscle, New York, Long Island Sound, 1985		
Total PCBs	5,500 FW; Max. 15,000 FW	13
PCBs 48, 52, 82, 101, 118, 138, 153	Contributed at least 28% of total PCB concentration	13
New York, various locations		
Total PCBs	1,100-24,100 FW	4
Total planar PCBs	100-2,020 FW	4
PCB 77	Max. 37 FW	4
PCB 81	Max. 5 FW	4
PCB 105	Max. 562 FW	4
PCB 114	Max. 112 FW	4
PCB 118	Max. 779 FW	4
PCB 126	Max. 8 FW	4
PCB 156	Max. 202 FW	4
PCB 157	Max. 68 FW	4
PCB 167	Max. 232 FW	4
PCB 169	Max. <0.1 FW	4
PCB 189	Max. 19 FW	4
Striped mullet, <i>Mugil cephalus</i> ; Japan, May 1976; muscle		
Total PCBs	1,200 (220-3,200) FW	14
PCB 77	2.1 (0.6-4.8) FW	14
PCB 126	0.1 (0.03-0.23) FW	14
PCB 169	0.002 (0.001-0.004) FW	14
Rainbow trout, <i>Oncorhynchus mykiss</i> ; Lake Ontario, 1989; liver vs. muscle		
PCB 77	2.9 FW, 78 LW vs. 3.3 FW, 85 LW	31
PCB 126	0.7 FW, 18 LW vs. 0.9 FW, 22 LW	31
PCB 169	0.3 FW, 8 LW vs. 0.3 FW, 9 LW	31
Red mullet, <i>Mullus barbatus</i> ; Spain, Mediterranean coast, summers 1989, 1990; muscle		
Sum of PCBs 28, 52, 101, 118, 138, 153, 180	21.2 (7.4-33.2) FW	33
Sum of PCBs 18, 31, 44, 97, 99, 105, 110, 128, 134, 146, 149, 151, 170, 174, 177, 183, 187, 194, 201	38.4 (15.5-61.4) FW	33
Chinook salmon, <i>Oncorhynchus tshawytscha</i> ; Lake Michigan, 1986; eggs		
Total PCBs	7,020 FW	32
PCBs 77, 105, 118, 126	0.2-12 FW	32
Fathead minnow, <i>Pimephales promelas</i> ; upper Hudson River, 1985; caged juveniles exposed for 3 to 42 days during the year; whole fish	Regardless of exposure duration or season, the consistently most abundant congeners in caged fish were PCBs 37, 42, 44, 47, 48, 49, 52, 59, 61, 66, 70, 73, 75, 76, 93, 95, and 104	15
European flounder, <i>Platichthys flesus</i> ; Norway, 1988; liver		
PCB 28	7 (0.3-68) FW	8
PCB 52	21 (0.7-609) FW	8
PCB 101	44 (1.1-1,452) FW	8
PCB 118	77 (3.2-1,693) FW	8
PCB 138	90 (1.1-1,960) FW	8

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 153	95 (5.7-2,088) FW	8
PCB 170	7 (0.4-158) FW	8
PCB 180	16 (1.2-296) FW	8
PCB 209	4 (0.4-88) FW	8
Paddlefish, <i>Polyodon spathula</i> ; Ohio River, Kentucky; 1988-89; total PCBs; males vs. females Gonads	16,200 (5,600-23,000) FW vs. 7,300 (50-18,700) FW	16
Red muscle (females only)	4,200 (2,000-6,300) FW	16
White muscle	700(50-3,300) FW vs. 400 (50-1,000) FW	16
Winter flounder, <i>Pleuronectes americanus</i> Long Island Sound, New York, 1984-86; total PCBs Liver	420-2,400 FW	17
Ovaries	30-730 FW	17
Testes	50-190 FW	17
New Bedford Harbor, Massachusetts vs. two control sites in Rhode Island; spring, 1988; liver Planar PCBs		
PCB 77	391 DW vs. 4.6 DW	38
PCB 126	49 DW vs. 1.8 DW	38
PCB 169	3.7 DW vs 0.4 DW	38
Nonplanar PCBs		
PCB 47	2,530 DW vs. 14 DW	38
PCB 52	1,100 DW vs. 6.5 DW	38
PCB 101	2,450 DW vs. 49 DW	38
PCB 105	2,200 DW vs. 92 DW	38
PCB 118	12,800 DW vs. 239 DW	38
PCB 128	1,370 DW vs. 47 DW	38
PCB 138	7,070 DW vs. 286 DW	38
PCB 151	422 DW vs. 19 DW	38
PCB 153	11,000 DW vs. 441 DW	38
PCB 180	1,010 DW vs. 141 DW	38
PCB 194	79 DW vs. 23 DW	38
PCB 195	32 DW vs. 1.4 DW	38
PCB 206	33 DW vs. 33 DW	38
PCB 209	3.1 DW vs. 20.5 DW	38
Total PCBs		
Gravid females	333,000 DW vs. 3,900 DW	38
Spent females	132,000 DW vs. 4,000 DW	38
Ripe males	124,000 DW vs. 13,300 DW	38
Sediments	8,000 DW vs. 500 DW	38
Atlantic salmon, <i>Salmo salar</i> ; Baltic Sea and environs Eggs, 1988		
PCB 77	Max. 28 LW	9
PCB 105	Max. 90 LW	9
PCB 126	Max. 2.4 LW	9
PCB 169	Max. 0.6 LW	9
Muscle, 1985		
Total PCBs	Max. 332 FW	18
PCB 77	Max. 1.1 FW	18
PCBs 126, 169	ND	18
Muscle, 1988		
PCB 77	Max. 34 LW	9
PCB 105	Max. 170 LW	9
PCB 126	Max. 3.8 LW	9
PCB 169	Max. 0.8 LW	9
Brown trout, <i>Salmo trutta</i> ; Catalonia, Spain; isolated mountain lakes; summers 1989, 1990; muscle Sum of PCBs 28, 52, 101, 118, 138, 153, 180	2.5 (1.3-3.8) FW	33
Sum of PCBs 18, 31, 44, 97, 99, 105, 110, 128, 134, 146, 149, 151, 170, 174, 177, 183, 187, 194, 201	4.8 (2.7-7.5) FW	33
Lake trout, <i>Salvelinus namaycush</i> Lake Ontario; total PCBs; whole fish 1977 vs. 1978	4,500 FW vs. 4,800 FW	19
1979 vs. 1980	2,500 FW vs. 3,100 FW	19
1981 vs. 1982	3,400 FW vs. 2,500 FW	19

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
1983 vs. 1984	4,500 FW vs. 4,800 FW	19
1985 vs. 1986	2,500 FW vs. 3,100 FW	19
1987 vs. 1988	3,400 FW vs. 2,500 FW	19
Lake Michigan		
Adult females; total PCBs		
1985	4,900-10,100 FW	47
1986	6,100-6,800 FW	47
1987	3,500-13,900 FW	47
Adult females; 1987; whole fish vs. eggs		
PCB 77	9.7 FW vs. 2.1 FW	47
PCB 126	4.0 FW vs. 0.65 FW	47
PCB 169	<0.4 FW vs. <0.4 FW	47
Marine Mammals		
Giant bottlenosed whale, <i>Berardius bairdii</i> ; blubber		
Japan, July 1985		
Total PCBs	2,300 (1,800-2,800) FW	20,21
Di-ortho planars		
PCB 128	54 (36-70) FW	20,21
PCB 138	460 (260-810) FW	20,21
Mono-ortho planars		
PCB 105	5 (2-8) FW	20,21
PCB 118	47 (15-94) FW	20,21
PCB 156	23 (6-50) FW	20,21
Non-ortho planars		
PCB 77	1.3 (0.7-1.9) FW	20,21
PCB 126	0.35 (0.1-0.57) FW	20,21
PCB 169	0.24 (0.09-0.45) FW	20,21
Northern north Pacific, July 1985		
Total PCBs	2,600 (2,400-2,800) FW	14
PCB 77	1.6 (1.3-1.9) FW	14
PCB 126	0.48 (0.39-0.57) FW	14
PCB 169	0.31 (0.17-0.45) FW	14
Beluga whale, <i>Delphinapterus leucas</i> ; Canada, St. Lawrence estuary; November 1983-December 1984; beach-stranded whales; total PCBs		
Blubber	Max. 72,200 FW	22
Kidney	Max. 10,000 FW	22
Liver	Max. 2,500 FW	22
Lung	Max. 560 FW	22
Milk	Max. 1,720 FW	22
Dolphins and toothed whales; blubber; total PCBs		
Atlantic Ocean, 1980-88; 15 species	130-190,000 FW	23
Indian Ocean, 1980-91; 5 species	520-7,900 FW	23
Pacific Ocean, 1980-86; 7 species	190-40,000 FW	23
Gray seal, <i>Halichoerus grypus</i>		
Baltic Sea and environs; 1981-87; blubber		
PCB 77	Max. 10 LW	9
PCB 105	Max. 180 LW	9
PCB 126	Max. 3 LW	9
PCB 169	Max. 2 LW	9
Nova Scotia, 1984-85; total PCBs; mother vs. pups		
Blood	6,080 LW vs. 6,240 LW	
Blubber	16,200-30,300 LW vs. 8,800-10,400 LW	
Milk (mother)	7,480-10,120 LW	
Serum	10,210 LW vs. 11,840 LW	24
Pacific white-sided dolphin, <i>Lagenorhynchus obliquidens</i> ;		
Japan, 1981; blubber		
Total PCBs	53,00 (40,000-71,00)FW	14
PCB 77	27 (14-38) FW	14
PCB 126	3.8 (3.2-4.4) FW	14
PCB 169	1.2 (0.9-1.4) FW	14
Finless porpoise, <i>Neophocaena phocaenoides</i> ; Japan, July 1985; blubber		
Total PCBs	320,000 FW	20,21
Di-ortho planars		
PCB 128	3,500 FW	20,21
PCB 138	35,000 FW	20,21

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
Mono-ortho planars		
PCB 105	1,200 FW	20,21
PCB 118	11,000 FW	20,21
PCB 156	160 FW	20,21
Non-ortho planars		
PCB 77	14 FW	14,20,21
PCB 126	0.9 FW	14,20,21
PCB 169	0.6 FW	14,20,21
Killer whale, <i>Orcinus orca</i> ; blubber		
Died after 2 years in captivity		
Total PCBs	160,000 FW	14
PCB 77	42.0 FW	14
PCB 126	4.0 FW	14
PCB 169	3.6 FW	14
Pacific coast of Japan; July 1985		
Total PCBs	370,000 (350,000-410,000) FW	20,21
Di-ortho planars		
PCB 128	8,000 (3,200-12,000) FW	20,21
PCB 138	65,000 (24,000-85,000) FW	20,21
Mono-ortho planars		
PCB 105	3,000 (2,300-3,600) FW	20,21
PCB 118	11,000 (6,700-14,000) FW	20,21
PCB 156	1,900 (950-3,100) FW	20,21
Non-ortho planars		
PCB 77	48 (39-55) FW	14,20,21
PCB 126	3.7 (2.4-4.4) FW	14,20,21
PCB 169	7.7 (2.3-12.0) FW	14,20,21
Ringed seal, <i>Pusa hispida</i> ; Norway, March-April, 1990		
Blubber		
PCBs 77, 126, 169	ND-0.19 LW	25
PCBs 66, 110, 149	20-22 LW	25
PCBs 52, 61, 105, 180	40-52 LW	25
PCBs 101, 118, 138	108-188 LW	25
PCBs 153	280 LW	25
Kidney		
PCBs 77, 126, 169	ND	25
PCBs 66, 110, 149, 180	12-14 LW	25
PCBs 52, 61, 105	21-28 LW	25
PCBs 101, 118, 138	49-67 LW	25
PCB 153	95 LW	25
Liver		
PCBs 77, 126, 169	ND-0.78 LW	25
PCBs 61, 66, 105, 110, 149, 180	18-43 LW	25
PCBs 52, 101, 118	57-71 LW	25
PCBs 138, 153	115-188 LW	25
Harbor seal, <i>Phoca vitulina</i> ; northeast coast of USA; 1980-92		
Blubber; 1980 vs. 1990-92		
Total	12,000 (7,300-24,300) FW vs. 6,660 (2,610-11,300) FW	51
PCB 8	ND vs. 0.9 FW	51
PCB 18	26 FW vs. 2 FW	51
PCB 28	99 FW vs. 16 FW	51
PCB 44	105 FW vs. 17 FW	51
PCB 52	661 FW vs. 213 FW	51
PCBs 66 + 95	192 FW vs. 29 FW	51
PCB 77	0.36 FW vs. 0.07 FW	51
PCB 101	897 FW vs. 500 FW	51
PCB 105	205 FW vs. 82 FW	51
PCBs 118 + 149	615 FW vs. 279 FW	51
PCB 126	1.45 FW vs. 0.53 FW	51
PCB 128	459 FW vs. 244 FW	51
PCB 138	2,990 FW vs. 1,650 FW	51
PCB 153	3,040 FW vs. 1,880 FW	51
PCB 169	0.019 FW vs. 0.013 FW	51
PCBs 170 + 190	458 FW vs. 266 FW	51
PCB 180	1,210 FW vs. 713 FW	51
PCB 187	865 FW vs. 601 FW	51

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 195	67 FW vs. 56 FW	51
PCB 206	72 FW vs. 79 FW	51
PCB 209	22 FW vs. 34 FW	51
Liver; 1980 vs. 1990-92		
Total	9,860 (6,290-16,000) FW vs. 6,260 (528-25,300) FW	51
PCB 8	ND vs. 1.2 FW	51
PCB 18	8.2 FW vs. 0.5 FW	51
PCB 28	27 FW vs. 10 FW	51
PCB 44	42 FW vs. 8 FW	51
PCB 52	343 FW vs. 145 FW	51
PCBs 66 + 95	127 FW vs. 9 FW	51
PCB 101	523 FW vs. 371 FW	51
PCB 105	345 FW vs. 26 FW	51
PCBs 118 + 149	470 FW vs. 196 FW	51
PCB 128	385 FW vs. 232 FW	51
PCB 138	2,250 FW vs. 1,550 FW	51
PCB 153	2,040 FW vs. 1,690 FW	51
PCBs 170 + 190	476 FW vs. 253 FW	51
PCB 180	1,160 FW vs. 696 FW	51
PCB 187	1,450 FW vs. 904 FW	51
PCB 195	85 FW vs. 60 FW	51
PCB 206	97 FW vs. 80 FW	51
PCB 209	41 FW vs. 36 FW	51
Common porpoise, <i>Phocoena phocoena</i>		
Found dead along Dutch coast, 1971-81		
Blubber vs. liver		
PCB 44	530 FW vs. 1 FW	26
PCB 49	900 FW vs. 5 FW	26
PCB 52	10,000 FW vs. 80 FW	26
PCB 101	3,200 FW vs. 20 FW	26
PCB 118	8,200 FW vs. 35 FW	26
PCB 138	28,900 FW vs. 280 FW	26
PCB 149	16,700 FW vs. 140 FW	26
PCB 153	32,600 FW vs. 340 FW	26
PCB 172	530 FW vs. 5 FW	26
PCB 174	3,700 FW vs. 40 FW	26
PCB 177	6,200 FW vs. 60 FW	26
PCB 180	4,000 FW vs. 80 FW	26
PCB 183	3,700 FW vs. 50 FW	26
PCB 194	700 FW vs. 10 FW	26
PCB 201	1,200 FW vs. 20 FW	26
PCB 206	360 FW vs. 7 FW	26
PCB 209	40 FW vs. 10 FW	26
Total PCBs (from above congeners)		
Blubber	51,350-139,790 FW	26
Heart	1,410-4,900 FW	26
Kidney	860-3,970 FW	26
Liver	1,190-17,400 FW	26
Muscle	960-3,990 FW	26
Three females found dead in fishing nets; Baltic Sea, 1989-90		
Blubber, maximum concentrations		
PCB 77	3.6 FW	37
PCB 126	1.4 FW	37
PCB 169	2.1 FW	37
PCB 60	15 FW	37
PCB 105	150 FW	37
PCB 118	1,100 FW	37
PCB 156	ND	37
PCB 137	730 FW	37
PCB 138	7,700 FW	37
PCB 153	9,600 FW	37
PCB 170	380 FW	37
PCB 180	1,500 FW	37
PCB 194	84 FW	37
Liver vs. muscle		
PCB 77	0.2 FW vs. ND	37

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 126	0.05 FW vs. ND	37
PCB 169	0.02 FW vs. ND	37
PCB 118	63 FW vs. 120 FW	37
PCB 138	250 FW vs. 350 FW	37
PCB 153	320 FW vs. 500 FW	37
PCB 170	33 FW vs. 64 FW	37
PCB 180	110 FW vs. 220 FW	37
Scandinavia; 1987-91; blubber; males; total of 47 detected PCB congeners	23,300 (3,710-65,260) LW	50
Dall's porpoise, <i>Phocoenoides dallidalli</i> ; northern North Pacific, 1980-85; blubber		
Total PCBs		
Females	1,500 (1,000-2,000) FW	14
Males	12,000 (7,100-18,000) FW	14
All samples	8,600 (1,000-18,000) FW	20,21
Di-ortho planars		
PCB 128	480 (110-1,100) FW	20,21
PCB 138	970 (160-2,000) FW	20,21
Mono-ortho planars		
PCB 105	100 (30-200) FW	20,21
PCB 118	280 (90-450) FW	20,21
PCB 156	11 (5-15) FW	20,21
Non-ortho planars		
PCB 77	2.3 (0.4-3.5) FW	14,20,21
PCB 126	0.16 (0.08-0.25) FW	14,20,21
PCB 169	0.11 (0.04-0.2) FW	14,20,21
Ringed seal, <i>Pusa hispida</i> ; Baltic Sea and environs, 1981-87; blubber		
PCB 77	Max. 9 LW	9
PCB 105	Max. 1,100 LW	9
PCB 126	Max. 4 LW	9
PCB 169	Max. 0.3 LW	9
Striped dolphin, <i>Stenella coeruleoalba</i> ; blubber Japan; 1978-79 vs. 1986; adult males; total PCBs	29,000 (15,000-46,000) FW vs. 28,000 (17,000-38,000) FW	27
Total PCBs		
Alive vs. dead	480,000 FW vs. 340,000 FW	28
Males vs. females	430,000 FW vs. 94,000FW	28
Matures vs. immatures	430,000 FW vs. 380,000 FW	28
Healthy vs. emaciated	480,000 FW vs. 340,000 FW	28
Di-ortho planars		
PCB 137	5,500 FW	
PCB 138	60,000 FW	28
PCB 153	73,000 FW	28
PCB 170	12,000 FW	28
PCB 180	39,000 FW	28
PCB 194	4,000 FW	28
Mono-ortho planars		
PCB 60	160 FW	28
PCB 105	2,000 FW	28
PCB 118	7,900 FW	28
PCB 156	3,000 FW	28
Non-ortho planars		
PCB 77	43 (16-85) FW	28
PCB 126	6.8 (2.4-13.0) FW	28
PCB 169	7.8 (1.9-15.0) FW	28
Goosebeaked whale, <i>Ziphius cavirostris</i> ; Bermuda, 1981; beached		
Blubber vs. liver		
PCB 44	5 FW vs. 0.4 FW	26
PCB 49	6 FW vs. 0.4 FW	26
PCB 52	36 FW vs. 4 FW	26
PCB 101	47 FW vs. 6 FW	26
PCB 118	74 FW vs. 13 FW	26
PCB 138	153 FW vs. 19 FW	26
PCB 149	73 FW vs. 15 FW	26
PCB 153	186 FW vs. 21 FW	26

Table 9. *Continued.*

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
PCB 172	14 FW vs. 2 FW	26
PCB 174	32 FW vs. 6 FW	26
PCB 177	24 FW vs. 5 FW	26
PCB 180	92 FW vs. 11 FW	26
PCB 183	37 FW vs. 6 FW	26
PCB 194	20 FW vs. 3 FW	26
PCB 201	64 FW vs. 10 FW	26
PCB 206	26 FW vs. 5 FW	26
PCB 209	6 FW vs. 0.5 FW	26
Total PCBs (from above congeners)		
Blubber	720-1,450 FW	26
Heart	18 FW	26
Kidney	26-78 FW	26
Liver	98-127 FW	26
Muscle	6-138 FW	26
Integrated Studies		
Freshwater lake near Amsterdam, the Netherlands; near dredged materials discharge site		
Sediments		
PCB 28	1.4 DW	29
PCB 52	2.3 DW	29
PCB 101	4.1 DW	29
PCB 138	13.7 DW	29
PCB 153	7.2 DW	29
PCB 180	3.4 DW	29
Plankton, 2 species vs. zebra mussel, <i>Dreissena polymorpha</i>		
PCB 28	0.2 FW, 68 LW vs. 0.5 FW, 36 LW	29
PCB 52	0.3 FW, 102 LW vs. 0.9 FW, 57 LW	29
PCB 101	0.5 FW, 206 LW vs. 3.8 FW, 220 LW	29
PCB 138	0.7 FW, 259 LW vs. 4.5 FW, 258 LW	29
PCB 153	0.5 FW, 209 LW vs. 5.8 FW, 322 LW	29
PCB 180	0.2 FW, 60 LW vs. 1.2 FW, 75 LW	29
Crustaceans, 3 species vs. European eel, <i>Anguilla</i> sp.		
PCB 28	3 FW, 362 LW vs. 4 FW, 21 LW	29
PCB 52	3 FW, 400 LW vs. 15 FW, 83 LW	29
PCB 101	4 FW, 532 LW vs. 28 FW, 186 LW	29
PCB 138	4 FW, 529 LW vs. 97 FW, 986 LW	29
PCB 153	4 FW, 505 LW vs. 89 FW, 932 LW	29
PCB 180	0.8 FW, 107 LW vs. 41 FW, 436 LW	29
Gulf of Mexico; 1986-87; coastal sediments vs. oyster soft parts		
Di-CBs	0.08-0.6 DW vs. 0.4-1.6 DW	39
Tri-CBs	0.6-2.6 DW vs. 7.6-8.3 DW	39
Tetra-CBs	1.8-11.0 DW vs. 26-38 DW	39
Penta-CBs	3.1-13.4 DW vs. 66-78 DW	39
Hexa-CBs	2.7-15.6 DW vs. 26-42 DW	39
Hepta-CBs	1.3-9.5 DW vs. 5-6 DW	39
Octa-CBs	0.3-2.6 DW vs. 0.4-1.0 DW	39
Nona-CBs	0.06-0.4 DW vs. 0.3-0.4 DW	39
Total PCBs	9.8-55.7 DW vs. 134-1,734 DW	39
Hudson River (upper), New York; 1983; total PCBs; water column vs. fish muscle	0.14 FW vs. 3,800 FW	40
Lake Clear (PCB-contaminated), Canada; 1986-87; vs. Lake Scugog (noncontaminated); total of 19 PCB congeners		
Sediments	571 DW vs. 22 DW	41
Crayfish	73 FW vs. 9 FW	41
Plankton	59 FW vs. ND	41
Fish	90-153 FW vs. 6-27 FW	41
Lake Erie, Hamilton Harbour; 1984; water column vs. sediments		
Tri-CBs	0.04 FW vs. 244 FW	42
Tetra-CBs	0.07 FW vs. 405 FW	42
Penta-CBs	0.07 FW vs. 1,025 FW	42
Hexa-CBs	0.02 FW vs. 982 FW	42
Hepta-CBs	0.01 FW vs. 983 FW	42
Octa-CBs	0.002 vs. 246 FW	42
Total PCBs	0.22 vs. 3,927 FW; Max. 14,185 FW	42

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
Oligochaetes		
Tri-CBs	33 FW	42
Tetra-CBs	39 FW	42
Penta-CBs	54 FW	42
Hexa-CBs	45 FW	42
Hepta-CBs	31 FW	42
Octa-CBs	5 FW	42
Total PCBs	207 FW	42
Lake Ontario, Wheatley Harbour; 1984; sediments vs. oligochaetes		
Tri-CBs	14 FW vs. 22 FW	42
Tetra-CBs	53 FW vs. 40 FW	42
Penta-CBs	133 FW vs. 102 FW	42
Hexa-CBs	141 FW vs. 72 FW	42
Hepta-CBs	69 FW vs. 23 FW	42
Octa-CBs	6 FW vs. 3 FW	42
Puget Sound, Washington; total PCBs		
Sediments	270-380 DW	43
Crab hepatopancreas	32,000 DW	43
English sole (<i>Pleuronectes vetulus</i>); liver	35,000 DW	43
Rainy River, Ontario; 1988; total PCBs		
Mill effluent, water vs. suspended solids	86-334 FW vs. 131-414 FW	44
Fish, two species; upstream vs. downstream	ND vs. 149-229 FW	44
Marine mammals (4 species; blubber) vs. terrestrial mammals (human, dog, cat; adipose or intestinal fat)		
Total PCBs	2,300-370,000 FW vs. 100-2,000 FW	20
Di-ortho planars		
PCB 128	54-8,000 FW vs. 1.2-72 FW	20
PCB 138	460-65,000 FW vs. 6.2-360 FW	20
Mono-ortho planars		
PCB 105	5-3,000 FW vs. 0.6-46 FW	20
PCB 118	47-11,000 FW vs. 2-160 FW	20
PCB 156	11-1,900 FW vs. 1-22 FW	20
Non-ortho planars		
PCB 77	1.3-48.0 FW vs. 0.03-0.37 FW	20
PCB 126	0.16-3.7 FW vs. 0.007-0.33 FW	20
PCB 169	0.11-7.7 FW vs. 0.03-0.09 FW	20
Waukegan Harbor, Illinois, Lake Michigan; August 1978; heavily contaminated by PCBs		
Sediments, samples from 5 locations		
Total PCBs	10,600-6,996,000 DW	30
PCB 77	10-390 DW	30
PCB 105	70-43,400 DW	30
PCBs 126, 169	ND	30
Fish		
White sucker, <i>Catostomus commersoni</i> ; whole		
Total PCBs	41,400 FW	30
PCB 77	50 FW	30
PCB 105	483 FW	30
PCBs 126, 169	ND	30
Black bullhead, <i>Ameiurus melas</i> ; whole		
Total PCBs	49,400 FW	30
PCB 77	89 FW	30
PCB 105	352 FW	30
PCBs 126, 169	ND	30
Largemouth bass, <i>Micropterus salmoides</i> ; offal		
Total PCBs	56,600 FW	30
PCB 77	86 FW	30
PCB 105	290 FW	30
PCBs 126, 169	ND	30
Coho salmon, <i>Oncorhynchus kisutch</i> ; whole		
Total PCBs	2,400 FW	30
PCB 77	2 FW	30
PCB 105	45 FW	30
Yellow perch, <i>Perca flavescens</i> ; whole		
Total PCBs	11,200 FW	30
PCB 77	23 FW	30
PCB 105	80 FW	30

Table 9. Continued.

Taxonomic group, organism, PCB congener, and other variables	Concentration ($\mu\text{g/kg}$) ^a	Reference ^b
White crappie, <i>Pomoxis annularis</i> ; whole		
Total PCBs	40,300 FW	30
PCB 77	24 FW	30
PCB 105	242 FW	30
Black crappie, <i>Pomoxis nigromaculatus</i> ; whole		
Total PCBs	32,200 FW	30
PCB 77	43 FW	30
PCB 105	114 FW	30

^aConcentrations are shown as means, range (in parentheses), maximum (Max.), and nondetectable (ND).

^b1, Galceran et al. 1993; 2, Johansen et al. 1993; 3, de Boer and Wester 1991; 4, Hong et al. 1992; 5, Hesselberg et al. 1990; 6, Himberg 1993; 7, Maack and Sonzogni 1988; 8, Marthinsen et al. 1991; 9, Koistinen 1990; 10, Leiker et al. 1991; 11, Knickmeyer and Steinhart 1989; 12, Steimle et al. 1990; 13, Bush et al. 1990; 14, Tanabe et al. 1987; 15, Jones et al. 1989; 16, Gundersen and Pearson 1992; 17, Greig and Sennefelder 1987; 18, Tarhanen et al. 1989; 19, Borgmann and Whittle 1991; 20, Kannan et al. 1989; 21, Tanabe et al. 1989; 22, Masse et al. 1986; 23, Tanabe et al. 1993; 24, Addison and Brodie 1987; 25, Daelemans et al. 1993; 26, Duinker et al. 1988b; 27, Loganathan et al. 1990; 28, Kannan et al. 1993; 29, Van der Oost et al. 1988; 30, Huckins et al. 1988; 31, Janz et al. 1992; 32, Williams and Giesy 1992; 33, Sanchez et al. 1993; 34, Gobas et al. 1989; 35, Kovats and Ciborowski 1989; 36, Kammann et al. 1993; 37, Falandysz et al. 1994b; 38, Elskus et al. 1994; 39, Sericano et al. 1990; 40, Brown et al. 1985; 41, Macdonald and Metcalfe 1989; 42, Mudroch et al. 1989; 43, Long 1982; 44, Merriman et al. 1991; 45, Sericano et al. 1994; 46, Steingraeber et al. 1994; 47, Mac et al. 1993; 48, Schmitt et al. 1990; 49, Huhnerfuss et al. 1995; 50, Kleivane et al. 1995; 51, Lake et al. 1995a; 52, USEPA 1992a.

variations by sex, age, and lipid content of the tissues in PCB concentrations were observed in white whales. Qualitative and quantitative differences in the PCB profiles of white whales were not solely related to the respective lipid content of the tissues but also to the specific nature of the lipids, which varied from one tissue to the other. The major PCB components in various tissues of white whales were PCBs 52, 99, 129, 137, 141, 153, 165, 180, and 185. Organ-specific retention of some PCB congeners occurs in white whales. PCBs 165 and 179, which were minor in blubber, were more abundant in kidney, liver, and lung. PCB 129 was more abundant in kidney and liver than in lung. Atlantic cod (*Gadus morhua*) composed a minor part of the white whale diet; however, PCB patterns in cod positively correlated with those in white whale tissues, suggesting a common source of intake. PCB congeners 52, 91, 99, 108, 118, 128, 138, 144, 149, 153, 163, and 180 were among the most abundant chlorinated biphenyls in cod liver oil and in the white whale blubber, liver, lung, kidney, and milk (Masse et al. 1986).

In gray seals (*Halichoerus grypus*), PCBs were transferred from blubber to milk by way of the circulatory system. Of the total concentration of PCBs in various tissues of maternal gray seals, PCB 153 accounted for about 30% in blubber, 20% in serum, and 30% in milk; PCB 180 accounted for 20% in blubber, 20% in serum, and 10% in milk; PCB 77 accounted for 20% in serum (Addison and Brodie 1987). In whales (*Ziphius* sp.), the dominant PCB congeners in blubber had 5-8 chlorines;

PCBs 138, 153, 149, 180, and 201 accounted for about 70% of the total PCBs (Duinker et al. 1988b). In the Canadian Arctic food chain of Arctic cod (*Boreogadus saida*) to ringed seal (*Phoca hispida*) to polar bear (*Ursus maritimus*), the total PCB concentrations in $\mu\text{g/kg}$ FW ranged from 4 in cod muscle to 680 in seal blubber to 4,500 in bear fat. The hexachlorobiphenyl PCB 153 accounted for 42% of the total PCBs in bear fat and for only 20% in seal blubber and 7% in cod muscle. Tri- and tetrachlorobiphenyl homologs predominated in cod, penta- and hexachlorobiphenyl congeners predominated in seal blubber, and hexa- and heptachlorobiphenyl congeners in fat of polar bears (Muir et al. 1988).

In comparison to conspecifics from noncontaminated sites, aquatic invertebrates from PCB-contaminated sites contained elevated concentrations of PCBs in tissues (Table 9). Adult aquatic insects are one of the groups considered useful and reliable indicators of PCB contamination (Wood et al. 1987; Kovats and Ciborowski 1989). Mayflies (*Hexagenia* spp.) from Lake St. Clair had PCB concentrations that reflected sediment PCB concentrations (Table 9). Observed mayfly sediment concentration ratios from PCBs linearly correlated with K_{ow} when expressed on a logarithmic basis (Gobas et al. 1989). PCB congener distributions in lake biota showed that no particular trophic level consistently accumulated the highest PCB concentrations and suggest that accumulations were associated with the organism's lipid concentrations (Table 9). A relation was consistent between the

concentration of dissolved PCBs and tissue concentrations in mussels from PCB-contaminated sites, such as New Bedford Harbor, Massachusetts. Uptake of PCB congeners in blue mussels (*Mytilus edulis*) from the dissolved phase of seawater was predictable from the log of the bioconcentration factor and the log K_{ow} of the congeners (Bergen et al. 1993). Eastern oysters (*Crassostrea virginica*) from Galveston Bay, Texas, contained as much as 1,100 µg/kg total PCBs DW soft parts, whereas conspecifics from Tampa Bay, Florida contained only 580 µg/kg DW soft parts; most (54-94%) of the relative toxicity in both groups was due to PCBs 77, 126, and 169 (Sericano et al. 1994).

The partitioning of individual, highly chlorinated PCB congeners with small differences in K_{ow} values may not adequately explain the accumulations in aquatic organisms (van der Oost et al. 1988). Hydrophobic chemicals, such as PCBs, are accumulated as a consequence of chemical partitioning between the water column, the organic phase of sediment, and biotic lipids or from biomagnification, a process reflecting the ratio between uptake rate from food and elimination rate from the organism. Accumulations of six PCB congeners (PCBs 28, 52, 101, 138, 153, 180) in surficial sediments (0-20 cm) and in an aquatic food chain in Lake Nieuwe Meer—a freshwater lake near Amsterdam containing contaminated dredged materials discharged over 30 years—were low in sediments; elevated in carnivores, plankton, mollusks, crustaceans, and eels; and independent of fat content (van der Oost et al. 1988). With concentrations in organisms expressed on the basis of lipid content (C_{org}) and concentrations in sediments expressed on the basis of organic carbon (C_{sed}) the median C_{org}/C_{sed} PCB accumulation patterns in aquatic organisms showed significant differences and indicated that mechanisms other than partitioning were operating. In plankton and mollusks, C_{org}/C_{sed} ratios seemed to be independent of hydrophobicity of PCB congeners. But with ascending trophic level from plankton to mollusks to crustaceans to eels, the median C_{org}/C_{sed} ratios of higher chlorinated congeners (PCBs 138, 153, 180) increased. Differences in accumulations of individual congeners were attributed to (1) increased biomagnification of higher chlorinated congeners because increasing hydrophobicity decreased elimination rates but not uptake efficiency; (2) greater mobility of eels and different feeding habits of eels and crustaceans that impact accumulation patterns because of biomagnification and partitioning; (3) variability in the

time period (limited by lifespan) available in a particular trophic level for equilibration between uptake and clearance; and (4) the tendency of equilibrium to be established at faster rates in less chlorinated congeners. In crustaceans, C_{org}/C_{sed} ratios decreased with decreasing hydrophobicity; the opposite occurred in eels and is attributed to differences in uptake efficiencies and low elimination rates of lower chlorinated congeners in crustaceans (van der Oost et al. 1988).

A correlation exists between the concentration of lipophilic, hydrophobic, chlorinated hydrocarbons in benthic fishes and the concentration of these compounds in sediments. The correlation is affected by the solubility of the contaminants, as reflected by the octanol/water partition coefficient K_{ow} and the carbon content of the sediment (Connor 1984; Breck 1985). Connor (1984) suggested that surface sediments, which change more slowly than the water column, are useful for averaging spatial and temporal contaminant inputs; however, correlations between PCB concentrations in sediment and those in nonbenthic carnivores with limited home ranges are extremely variable.

Concentrations of total PCBs—measured as Aroclors 1248, 1254, and 1260—in adult freshwater fishes in the United States from noncontaminated sites declined between 1976 and 1984 (Table 9), and more than 90% of all analyzed samples contained measurable quantities of PCBs during this period (Schmitt et al. 1990). Total PCB concentrations in domestic freshwater fishes in 1986-87 from contaminated sites were as high as 124,000 µg/kg FW (USEPA 1992a). In general, total PCB concentrations in domestic freshwater fishes sampled between 1976 and 1984 were highest in the industrialized regions of the northeast, the Great Lakes, the upper Mississippi River, and the Ohio River (Schmitt et al. 1990). Phillips and Birchard (1990) reviewed PCB concentrations (as judged by residues of Aroclors 1016, 1221, 1232, 1242, 1248, 1254, and 1260) in sediments and fish tissues in the United States during 1978-87. During 1978-81, total PCB concentration rankings in sediments were highest in the lower Mississippi, Tennessee, South Atlantic-Gulf of Mexico, and lower Colorado regions and lowest in the Great Lakes, Arkansas, mid-Atlantic, Pacific Northwest, and Rio Grande regions. During this same period, PCB concentrations in fish tissues were highest in the Missouri, upper Colorado, California, and the Great Lakes regions and lowest

in the upper Mississippi, New England, Ohio, Pacific Northwest, Tennessee, lower Mississippi, and Rio Grande regions. Sediment PCB rankings during 1982-87 were highest in the Arkansas, California, Ohio, and Missouri regions and lowest in the South Atlantic-Gulf, and Colorado regions. Total PCBs were highest in fish tissues in the upper Mississippi Region during 1982-87. The fish tissue rankings in descending order assigned to 12 regions with sufficient total PCB data were the upper Mississippi and Missouri regions, Ohio, south Atlantic-Gulf, Arkansas, Great Basin, lower Colorado, California, Pacific Northwest, Ohio, upper Colorado, and lower Mississippi. PCB rankings between fish tissues and sediments were not necessarily comparable because high levels in sediment do not necessarily result in high levels in fishes if bioconversion was significant (Phillips and Birchard 1990). Total PCB concentrations in coastal sediments and fish liver in the United States were highest in the Boston Harbor (17.1 mg/kg DW sediment, 10.5 mg/kg in liver of winter flounder, *Pleuronectes americanus*), San Diego Harbor (0.42 mg/kg DW sediment, 19.7 mg/kg in barred sand bass, *Paralbrax nebulifer*), and Elliot Bay, Washington (0.33 mg/kg sediment, 14.7 mg/kg in flathead sole, *Hippoglossoides elassodon*). Trichloro PCBs were in sediments at many sites but did not accumulate in fish livers except in the Boston Harbor. Sediments in the Boston Harbor, western Long Island Sound, and Raritan Bay were contaminated with PCB mixtures that were relatively high in tri- and tetrachlorobiphenyl isomers, although penta- and particularly hexachlorobiphenyls were the dominant isomers at most sediment sites. As expected, levels of hexachlorobiphenyls in fish livers were dominant because of the more persistent and lipophilic characteristics of increasingly chlorinated PCBs (NOAA 1987).

Variations in PCB concentrations in sediment and water in the Great Lakes can largely account for the variability between different bodies of water in fish PCB residues. Other variables include fish lipid content, position of the fish species in the food web, and trophic structure of the food chain. Collectively, these variables explain 72% of the variation in PCB concentrations of 25 species of Great Lakes fishes (Rowan and Rasmussen 1992). Tissue concentrations of PCBs in benthic and lower trophic organisms in lakes can be estimated by assuming equal lipid-normalized concentrations in biota and sediment; however, food chain transport had a greater effect on PCB concentrations in higher trophic levels (Macdonald et al.

1993). Total PCB concentrations in whole bodies of lake fishes were higher among older piscivores and higher with increasing lipid concentration and seemed to reflect exposure conditions at the capture site (Southworth 1990).

Eggs of chinook salmon (*Oncorhynchus tshawytscha*) from Lake Michigan in 1986 contained AHH-active PCB congeners—including PCBs 77, 105, 118, and 126—at concentrations from 0.9 to 262 µg/kg FW; concentrations of these congeners did not correlate with survival (Williams and Giesy 1992). Mortality of chinook salmon eggs was not related to total PCB concentrations as high as 7,020 µg/kg FW (Williams and Giesy 1992). In Lake Ontario, the overall trend in total PCB concentrations in whole lake trout (*Salvelinus namaycush*) between 1977 and 1988 was a gradual decline with a half-time persistence of about 10 years (Borgmann and Whittle 1991). In Lake Michigan, total PCB concentrations declined 64% in bloaters (*Coregonus hoyi*) from 5,700 mg/kg FW in 1972 to 1,600 µg/kg FW in 1986 (Hesselberg et al. 1990). In Lake Superior, the PCB congener fingerprint in eggs of the lake trout differed from that of lake trout eggs of other Great Lakes (Mac et al. 1993). A difference between residue patterns was also identified between eggs and the parent fish, suggesting preferential deposition of congeners other than AHH-active congeners. Concentrations of individual congeners in lake trout have been declining at similar rates in the Great Lakes during a 10-year period (Mac et al. 1993).

Distribution patterns of PCB congeners in water, sediment, and four groups of biota from two lakes in Ontario contaminated by known point sources of PCBs (Lake Clear, Rice Lake) were compared with the congener distribution in Lake Scugog, a relatively clean control lake exposed only to atmospheric inputs of PCBs (Macdonald and Metcalfe 1989). Samples were analyzed for 19 PCBs. Those from Lake Clear had a distribution pattern similar to Aroclor 1254 and dominant concentrations of congeners 87, 101, and 118; this lake was contaminated with a PCB mixture similar to Aroclor 1254 in the mid to late 1970s. The sources to Rice Lake were less clear. Lake Scugog had a higher proportion of less chlorinated PCBs, in agreement with another study of atmospheric deposition to isolated lakes (Swackhammer et al. 1988). Because the sediments contained elevated levels of organic carbon, the sediments were expected to also hold relatively large concentrations of the higher, more hydrophobic PCBs, in accord with previous reports (Karickhoff 1981; Formica et

al. 1988). But this was not the case; subsequent deposition of total and higher chlorinated congeners into the bottom sediments (organic carbon basis) was unexpectedly low. The proportion of higher chlorinated congeners in sediments were also lower than in biota (lipid weight basis) in all three lakes. Because dissolved organic carbon (DOC) increases the solubility of PCBs in water (Gschwend and Wu 1985), the high DOC levels may have caused partitioning of more PCBs into the water and less sorbed onto sediments. The sediments were not efficient at accumulating PCBs, although bottom sediment concentrations were higher in contaminated lakes. Adsorption of PCBs on suspended particles occurred, as anticipated; PCBs on total suspended solids were higher in contaminated lakes (978 $\mu\text{g/kg}$) than in the control lake (49 $\mu\text{g/kg}$) and reflected lake concentrations (Gschwend and Wu 1985). In a related study, Macdonald and Metcalfe (1991) analyzed the concentration and distribution of 19 PCB congeners in biota, sediments, water, and suspended solids of isolated oligotrophic lakes in central Ontario that were contaminated by atmospheric deposition. The range of the total congener concentrations was 1-2 ng/L dissolved in water, 10-50 $\mu\text{g/kg}$ DW in sediment, 5-10 $\mu\text{g/kg}$ FW in lower trophic levels, and 10-30 $\mu\text{g/kg}$ in fishes from upper trophic levels. The high proportion of trichlorobiphenyls previously reported in vapor (Duinker and Bouchertall 1989; Baker and Eisenreich 1990) and hexachlorobiphenyl congeners 153 and 138 in particulate-bound PCBs (Swackhammer et al. 1988; Duinker and Bouchertall 1989; Baker and Eisenreich 1990) were reflected in the four study lakes. PCB concentrations (lipid basis) were higher in teleosts than they were in invertebrate prey organisms.

Winter flounder from the PCB-contaminated harbor in New Bedford, Massachusetts, had grossly elevated concentrations of PCBs in their livers (as high as 333,000 $\mu\text{g/kg}$ DW); concentrations were about 5 times higher than in any other fish sample collected worldwide (Table 9; Elskus et al. 1994). PCB patterns in the New Bedford Harbor showed high agreement between the exposure environment (water and sediments) and ribbed mussels (*Geukensia demissa*) and mummichogs (*Fundulus heteroclitus*); however, agreements with American eels (*Anguilla rostrata*) or grass shrimp (*Palaemonetes pugio*) were poor because, in part, of differential metabolism of PCBs by these species (Lake et al. 1995b). PCB concentrations in four species of catfishes from the Mis-

issippi River and its tributaries in summer 1987 were highest from the Illinois River; the Ohio River at Olmsted; and the Mississippi River at Helena, Arkansas, and Arkansas City, Arkansas. These sites seem to be point sources of PCB pollution because PCB residues in catfishes above and below these sites were lower. Although PCBs were banned in 1978, the elevated levels in catfishes suggests PCB leakage from hazardous waste sites with transformer and hydraulic fluids and flame-resistant plasticizers (Leiker et al. 1991). Findings of high (greater than 4,000 $\mu\text{g/kg}$ FW) total PCB levels in mature roe samples of the paddlefish (*Polyodon spathula*) from the Ohio River warranted warnings of the general public about consuming this domestic caviar (Gundersen and Pearson 1992).

The upper Hudson River was massively contaminated with PCBs from an industrial plant for several decades prior to 1975. All fishing in this section in 1976 was banned because of PCB contamination. The prohibition is still in effect because, in part, of measurable PCB residues in caged fishes from this area (Table 9; Jones et al. 1989). Striped bass (*Morone saxatilis*) collected near Troy and Albany, New York, contained higher concentrations in muscle of PCB 77 (37 $\mu\text{g/kg}$ FW) and PCB 126 (8 $\mu\text{g/kg}$ FW) than conspecifics from other locations in New York (Hong et al. 1992). Almost all (99%) the PCB toxicity in muscle of striped bass was attributed to PCBs 77, 105 (62 $\mu\text{g/kg}$ FW), and 126 (Hong et al. 1992).

The most prominent PCB congeners in muscle from 14 species of Wisconsin fishes in 1986-87 were PCBs 28/31, 66/95, 70/76, 101, 105, 110, 118, 138, 146, 149 and 180 (Maack and Sonzogni 1988). Congeners 105 and 118 were found in the greatest amount in fishes at 1 to 5% of the total PCB concentration of each. Congeners with responses similar to 2,3,7,8-TCDD, that is, the planar PCBs, were seldom present above detection levels. The sum of the individual congeners measured in Wisconsin fish muscle were similar to total recorded PCB values (Maack and Sonzogni 1988). Increased fish consumption by Wisconsin anglers in 1985 positively correlated with increased human serum PCB concentrations (Sonzogni et al. 1991). Human consumers of Wisconsin game fishes (chinook salmon, *Oncorhynchus tshawytscha*; yellow perch, *Perca flavescens*; walleye, *Stizostedion vitreum*) in 1986 contained various PCB congeners in their sera. PCB 153 (78% frequency of occurrence) was present at 1.46 (0.6-7.3) $\mu\text{g/L}$ human serum, PCB 138 (56%) at 1.32 (0.6-6.0) $\mu\text{g/L}$, PCB 180 (42%) at

1.06 (0.6-3.5) $\mu\text{g/L}$, PCB 118 (34%) at 1.12 (0.6-5.7) $\mu\text{g/L}$, PCB 187 (11%) at 0.98 (0.6-2.2) $\mu\text{g/L}$, PCB 170 (5.8%) at 0.86 (0.6-1.4) $\mu\text{g/L}$, PCB 28 (1.2%) at 0.8 $\mu\text{g/L}$, PCB 101 (0.58%) at 0.8 $\mu\text{g/L}$, PCB 70 (0.58%) at 0.7 $\mu\text{g/L}$, and a single planar PCB—PCB 77—(0.58% frequency of occurrence) at 1.3 $\mu\text{g/L}$. PCBs 118, 138, and 180 are potentially most toxic to human consumers, as judged by the concentrations of these congeners in human sera (Sonzogni et al. 1991).

Concentrations of PCBs in female northern pikes (*Esox lucius*) from a Scandinavian lake decreased with increasing age, weight, or body length (Larsson et al. 1993). Seasonal elimination of the lipophilic contaminants in roe—which contained as much as 10 times more fat than muscle and more than 10 times the amount of pollutants than muscle—is the major route of PCB loss. Male northern pikes contained higher concentrations of PCBs than females because of the lower elimination by way of gonadal products; males showed no significant relation between age and PCB burdens in tissues (Larsson et al. 1993). Total PCB levels of 7,700 to 34,000 $\mu\text{g/kg}$ LW in eggs of the Arctic char (*Salvelinus alpinus*) from Lake Geneva, Switzerland, correlated with a mortality rate of 29 to 100% (Gundersen and Pearson 1992).

On the Pacific coast and in adjacent areas of Mexico, data from more than 150 survey and monitoring programs were summarized on contamination of sediments, invertebrates, and fishes (Mearns 1992). PCBs in sediments seem to be reflected in mussels, and PCB residues from mussels collected at harbor entrances remained unchanged or were increasing. The harbors in Los Angeles-Long Beach and San Diego remained contaminated with PCBs, and PCB concentrations in sediments were reflected in fish livers. Waste management seems to have been effective in the Palos Verdes outfall area. Sediments and mussel samples in Palos Verdes from 1974-88 showed decreasing PCB levels that reflected a 100-fold reduction in PCB wastewater emissions during that period. Contamination of the coastal zone declined to levels found 30 and 40 years ago. PCB levels had declined at least one order of magnitude in teleosts and shellfish at offshore sites since the 1970s. Bays and harbors were more contaminated than the open coastal zone and must be monitored more closely; lower detection levels (0.001 to 0.01 mg/kg FW versus the current analytical limits of 0.02 mg/kg FW) were proposed to monitor the effectiveness of current source control programs (Mearns 1992).

PCBs in the Puget Sound, Washington, were measured in sediments, fish livers, and benthic invertebrates. Maximum total PCB concentrations were 2,100 $\mu\text{g/kg}$ DW in sediments near Tacoma and 32,000 $\mu\text{g/kg}$ DW in crab hepatopancreas and 35,000 $\mu\text{g/kg}$ DW in fish liver near Seattle (Long 1981). PCB concentrations in sediments of the Puget Sound in May 1988 positively correlated with PCB concentrations in livers of several species of flatfishes in these sediments (Stein et al. 1992). Increased sediment PCB concentrations also correlated well with increased hepatic AHH and EROD activities and with increases in total hepatic GSH, all of which are acknowledged early indicators of chemical contamination by PCBs and other organic contaminants (Gooch et al. 1989; Stein et al. 1992).

PCB residues in liver of the European flounder (*Platichthys flesus*) were extremely variable, but residues of individual congeners were usually higher in fall, higher in females, and higher in flounders captured inland near a PCB point source (Marthinsen et al. 1991); a similar pattern was documented in the Atlantic cod, (*Gadus morhua*; Marthinsen et al. 1991). In the dab (*Limanda limanda*), a marine flatfish, the accumulation of PCBs 128, 138, and 163 differs significantly by sex (Knickmeyer and Steinhart 1989). Depletion of lipids from the liver of female dabs during ovary maturation is an important excretory pathway for PCBs during spawning (Knickmeyer and Steinhart 1989). PCB levels in liver of dabs were higher in spring than in winter; livers and ovaries were dominated by penta- and hexachlorobiphenyls, but the dominant PCBs in testes were tri- and tetrachlorobiphenyls (Kammann et al. 1993).

The most prominent PCB congeners at 280-323 $\mu\text{g/kg}$ DW in the tilefish (*Lopholatilus chamaeleonticeps*) from Georges Bank in 1981-92 were PCBs 138 and 153 in gonad and liver; at 69-82 $\mu\text{g/kg}$ DW the most prominent PCB congeners in the tilefish from New Jersey during this same period were PCBs 138 and 153 in liver (Steimle et al. 1990). Total PCB concentrations in marine coastal fishes were dominated by the hexachlorobiphenyls (Knickmeyer and Steinhart 1989), but trout from isolated mountain lakes had tri-, tetra-, and pentachlorobiphenyls as the major components of total PCBs (Sanchez et al. 1993).

Reptiles

PCBs accumulate in the fat, testes, and brain of snapping turtles (*Chelydra serpentina*), and

concentrations seem to reflect the lipoprotein solubility of individual congeners (Bryan et al. 1987a; Table 10). With increasing hydrophobicity (increasing K_{ow}) of PCB congeners, accumulations increased in livers of snapping turtles; total liver PCB concentrations in adults increased with increasing age, length, and weight (Hebert et al. 1993). PCB loadings in snapping turtle eggs were not related to the body size of females or to the number of eggs in the clutch (Bishop et al. 1994). However, a positive relation between PCB loadings in liver of adult female snapping turtles and their eggs was significant (Hebert et al. 1993).

PCB 105 may be an important contributor to the toxic burden of snapping turtle populations (Hebert et al. 1993). Eggs of snapping turtles from the Great Lakes had a lower hatch rate and a significantly increased frequency of deformed hatchlings than eggs from a control site, and this seemed to be strongly associated with total PCB concentrations and PCB 105 (Bishop et al. 1991). Of the 5 toxic PCB congeners measured in the yolks, egg whites, and shells of snapping turtle eggs, PCBs 105 and 167 accounted for more than 99% of the total toxicity—as measured by 2,3,7,8-TCDD TEF equivalents—and 95% of the total toxicity resided in the yolk (Bryan et al. 1987b). Large reserves of fat in eggs of the snapping turtle do not seem to protect against toxic PCB congeners from being dispersed into egg components low in fat (Bryan et al. 1987b).

Birds

In general, total PCB concentrations in birds were usually higher in males and in eggs than in livers, in adipose tissues, in fish-eating species, and at PCB-contaminated sites; PCBs 138 and 153 tended to predominate in all samples (Table 11). The change in PCB content in livers of Norwegian raptors between 1965 and 1983 was not significant despite a marked reduction in the use of these compounds (Froslie et al. 1986). When total PCB concentrations declined, for example, in eggs of red-breasted mergansers (*Mergus serrator*) between 1977 and 1990, the relative potency of the mixture of PCBs—as measured by 2,3,7,8-TCDD equivalents—was unchanged (Williams et al. 1995).

Commercial PCB mixtures frequently contain impurities that may contribute to the 2,3,7,8-TCDD toxic equivalency factor; these impurities may include other PCBs, dioxins, dibenzofurans, naphthalenes, diphenyl ethers and toluenes, phe-

noxy and biphenyl anisoles, xanthenes, xanthonenes, anthracenes, and fluorenes (Jones et al. 1993). PCB concentrations in avian tissues sometimes positively correlate with DDE concentrations (Mora et al. 1993). Eggs of peregrine falcons (*Falco peregrinus*) from California, for example, contained measurable quantities of various organochlorine compounds, including dioxins, dibenzofurans, mirex, hexachlorobenzene, and p,p' DDE at 7.1–26.0 mg/kg FW; PCB 126 accounted for 83% of the 2,3,7,8-TCDD equivalents, but its interactions with other detectable organochlorine compounds is largely unknown (Jarman et al. 1993).

There is a relation between PCB uptake and the position of the species in the food chain. In a 3-step central-European oak-forest food chain involving the great tit (*Parus major*), caterpillars (*Tortrix viridana*, *Operophtera brumata*, *Erannis defoliaria*), and leaves of the red oak (*Quercus* sp.), mean concentrations of PCB 153—the most abundant measured congener—rose from about 1 µg/kg DW in leaves to 10 in caterpillars to 170 in bird eggs (Winter and Streit 1992). Older juvenile tits contained 307 µg of PCB 153/kg whole body DW; these birds received PCBs from the mother during egg transfer and from the caterpillar food source during the nesting period. PCBs 101, 138, and 180 were also present in most samples but at lower concentrations than PCB 153. Populations of *Parus major* in this area declined in recent years, and the influence of anthropogenic contaminants may be a factor (Winter and Streit 1992). Fish-eating waterfowl and seabirds had comparatively high total PCB and high planar PCB concentrations in eggs and tissues; waterfowl and seabirds that feed mainly on invertebrates had lower PCB concentrations (Focardi et al. 1988b; Borlakoglu et al. 1990; Gonzalez et al. 1991; Jones et al. 1993). PCB concentrations were higher in adipose tissues of the Arctic tern (*Sterna paradisaea*) than in those of their fish and invertebrate food items (Scharenberg 1991a). PCB concentrations in adipose tissues of cormorants, when compared to their diet of fishes, were 10 to 100 times higher than marine fishes and 100 to 1,000 times higher than freshwater fishes (Scharenberg 1991b). Double-crested cormorants (*Phalacrocorax auritus*) biomagnify total PCBs from their fish diet to their eggs—based on 2,3,7,8-TCDD equivalents—by a factor of 31.3 (Jones et al. 1994). Higher-chlorinated PCBs accumulated in tissues of the herring gull (*Larus argentatus*) to a greater extent than was present in the alewife (*Alosa pseudoharengus*), a primary food item; lower-chlorinated biphenyls, including

Table 10. PCB concentrations in field collections of selected reptiles. Concentrations are in $\mu\text{g/kg}$ (ppb) fresh weight (FW) or lipid weight (LW).

Species, tissue, PCB congener, and other variables	Concentration ^a in $\mu\text{g/kg}$	Reference ^b
American alligator, <i>Alligator mississippiensis</i>;		
total PCBs; Florida		1
Eggs, 1984	80-170 FW; Max. 670 FW	
Muscle, 1985	100-2,100 LW	2
Snapping turtle, <i>Chelydra serpentina</i>		
Canada		
Great Lakes (Ontario and Erie) vs. control site in central Ontario; eggs; sum of PCB congeners 105, 118, 138, 153, 170, and 180		
1986-87	2,600-2,700 FW vs. 80 FW	3
1988-89	300-3,300 FW vs. 30 FW	3
Hamilton Harbour; adults; found dead; 1986-87; fat; sum of PCBs 105, 118, 138, 153, 170, and 180	57,700-72,200 LW	3
Lake Ontario; eggs; 1990		
PCB 52	20(10-40) LW	4
PCB 105	1,700 (500-2,900) LW	4
PCB 118	7,300 (2,100-11,700) LW	4
PCB 138	9,300 (2,000-16,200) LW	4
PCB 153	9,300 (2,500-16,200) LW	4
PCB 180	6,400 LW	4
PCB 194	600 LW	4
Total PCBs	54,300 (13,300-96,400) LW	4
Southern Ontario; adults; 1988-89; muscle; sum of Aroclors 1254 and 1260	7-660 FW; Max. 2,120 FW	5
United States		
Contaminated site (South Glens Falls, New York) vs. noncontaminated site (Columbus, New York); adults; total PCBs		
Brain	82,000 LW vs. 1,000 LW	6
Heart	49,000 LW vs. 600 LW	6
Kidney	48,000 LW vs. 1,200 LW	6
Liver	72,000 LW vs. 1,000 LW	6
Lungs	13,000 LW vs. 400 LW	6
Pancreas	48,000 LW vs. 1,200 LW	6
Testes	100,000 LW vs. 1,600 LW	6
Fat	1,600,000 LW vs. 4,200 LW	6
PCB 66	272,000 LW vs. 200 LW	6
PCB 82	100,000 LW vs. 90 LW	6
PCB 99	166,000 LW vs. 50 LW	6
PCB 105	171,000 LW vs. 160 LW	6
PCB 136	297,000 LW vs. 200 LW	6
PCB 176	152,000 LW vs. 300 LW	6
Upper Hudson River, New York; egg yolk		
PCB 105	700-1,890 FW	7
PCB 118	16-32 FW	7
PCB 120	120-280 FW	7
PCB 167	200-560 FW	7
PCB 189	60-120 FW	7

^a Concentrations are shown as means, range (in parentheses), and maximum (Max.).

^b 1, Heinz et al. 1991; 2, Delany et al. 1988; 3, Bishop et al. 1991; 4, Bishop et al. 1994; 5, Hebert et al. 1993; 6, Bryan et al. 1987a; 7, Bryan et al. 1987b.

the tetra- and penta-CBs, did not biomagnify (Braune and Norstrom 1989).

Declining populations of Caspian terns (*Sterna caspia*)—especially populations nesting in Green Bay and Saginaw Bay between 1986 and 1990—were associated with elevated PCB concentrations in blood; the frequency of developmental

abnormalities and deformities in Caspian tern populations at Saginaw Bay was almost 100 times above that recorded in the same area between 1962 and 1972 (Table 11; Mora et al. 1993). High PCB concentrations in tissues of white-tailed eagles (*Haliaeetus albicilla*) are directly connected to high concentrations in eggs and associated with

Table 11. PCB concentrations in field collections of selected birds. Concentrations are in µg/kg (ppb) fresh weight (FW), dry weight (DW), or lipid weight (LW).

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Northern goshawk , <i>Accipiter gentilis</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	2,000 (<100-1,260,000) FW vs. 12,300 (2,600-53,000 FW)	1
Northern sparrow hawk , <i>Accipiter nisus</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	1,100 (<100-107,000) FW vs. 5,900 (<100-39,000) FW	1
Sharp-shinned hawk , <i>Accipiter striatus</i> ; blood plasma; total PCBs (as Aroclors 1254 and 1260); Great Lakes, 1985-89	80 (10-190) FW	2
Tengmalm's owl , <i>Aegolius funereus</i> ; liver vs. eggs total PCBs; Norway, 1965-83; dead on collection	1,000 (<100-9,400) FW vs. 400 ; (<100-1,300) FW	1
Red-winged blackbird , <i>Agelaius phoeniceus</i> ; eggs (less shell) and chicks (less feathers, feet, beaks, stomach and contents, and wings); total PCBs; Green Bay, Wisconsin, 1989	5,400-8,900 FW	9
Razorbill , <i>Alca torda</i> ; adipose tissue; males vs. females; England, 1988		
PCBs 3, 8, 18	ND vs. ND	3
PCB 28	110 FW vs. ND	3
PCB 52	140 FW vs. ND	3
PCB 101	310 FW vs. 32 FW	3
PCB 118	2,500 FW vs. 500 FW	3
PCB 138	6,500 FW vs. 1,600 FW	3
PCB 153	4,400 FW vs. 1,100 FW	3
PCB 180	2,600 FW vs. 600 FW	3
Total PCBs	16,500 FW vs. 4,100 FW	3
Imperial eagle , <i>Aquila heliaca adalberti</i> ; infertile eggs; Spain, 1986-87		
PCB 101	9-16 FW; Max. 440 FW	4
PCB 118	1-4 FW; Max. 66 FW	4
PCB 138	7-30 FW; Max. 620 FW	4
PCB 153	37-115 FW; Max. 5,300 FW	4
PCB 180	30-110 FW; Max. 4,800 FW	4
Total PCBs	280-820 FW; Max. 28,900 FW	4
Golden eagle , <i>Aquila chrysaetos</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	2,000 (<100-250,000) FW vs. 1,000 (400-5,700) FW	1
Grey heron , <i>Ardea cinerea</i> ; total PCBs (35 congeners); England, 1988-90		
Fat	198,000 FW; 226,000 LW	5
Kidney	3,600 FW; 135,000 LW	5
Liver	2,800 FW; 936,000 LW	5
Muscle	8,100 FW; 139,000 LW	5
Great blue heron , <i>Ardea herodias</i> ; eggs; British Columbia		
PCB 77	0.05-0.2 LW	6
PCB 105	17-31 LW	6
PCB 118	63-116 LW	6
PCB 126	0.1-0.2 LW	6
PCB 169	0.02-0.04 LW	6
Short-eared owl , <i>Asio flammeus</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	300 (<100-46,000) FW vs. 2,100 (2,000-22,000) FW	1
Greater scaup , <i>Aythya marila</i> ; total PCBs; carcass; Detroit River, 1981	11,000 FW	25
Eagle owl , <i>Bubo bubo</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	3,000 (100-550,000) FW vs. 4,000 (2,200-29,000) FW	1
Common buzzard , <i>Buteo buteo</i> ; infertile eggs; total PCBs; Spain, 1985-86	1,650 (1,600-1,700) FW	7
Rough-legged buzzard , <i>Buteo lagopus</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	200 (<100-15,000) FW vs. 800 (200-9,300) FW	1
White stork , <i>Ciconia ciconia</i> ; infertile eggs; total PCBs; Spain, 1985-86	800 (200-2,700) FW	7
Dipper , <i>Cinclus cinclus</i> ; addled eggs; Wales vs. Ireland, 1990-92		
PCB 101	10-20 LW vs. <10 LW	8
PCB 118	10-70 LW vs. 10-1,280 LW	8
PCB 138	10-380 LW vs. 10-80 LW	8
PCB 153	20-530 LW vs. 10-160 LW	8
PCB 170	10-190 LW vs. 10-30 LW	8
PCB 180	10-20 LW vs. 10-40 LW	8

Table 11. Continued.

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Merlin, <i>Falco columbarius</i> ; liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	400(<100-41,000) FW vs. 4,500 (2,400-11,000) FW	1
Peregrine, <i>Falco peregrinus</i> Liver vs. eggs; total PCBs; Norway, 1965-83; dead on collection	120,000 FW vs. 43,000 FW	1
Eggs; California, 1983-88		
PCB 37	0.18 FW	10
PCB 77	0.93 FW	10
PCB 126	1.0 FW	10
PCB 138	750 FW; Max. 2,440 FW	10
PCB 153	1,200 FW; Max. 4,400 FW	10
PCB 169	0.14 FW	10
Total PCBs	4,800 (1,400-13,000) FW	10
Gyr falcon, <i>Falco rusticolus</i> ; liver vs. eggs; total PCBs; Norway 1965-83; dead on collection	5,000 (300-42,000) FW vs. 12,000 FW	1
Kestrel, <i>Falco tinnunculus</i> ; liver vs. eggs; total PCBs; Norway 1965-83	500 (<100-45,000) FW vs. 600 (<100-1,000) FW	1
Chicken, <i>Gallus</i> sp. ; fat vs. feather; total of PCBs 118, 138, 153, and 156; PCB-contaminated area	12,800 LW vs. 200 LW	11
Gull-billed tern, <i>Gelochelidon nilotica</i> ; infertile eggs; Spain, 1988		
PCB 138	Max. 220 FW	12
PCB 153	Max. 90 FW	12
PCB 180	Max. 190 FW	12
White-tailed sea eagle, <i>Haliaeetus albicilla</i> Liver vs. eggs; total PCBs; Norway 1965-83	5,000 (100-180,000) FW vs. 13,900 (4,200-31,000) FW	1
Breast muscle; Poland, 1982-90		
Non-ortho planars		
PCB 77	3-140 FW	13
PCB 126	1-160 FW	13
PCB 169	2-380 FW	13
Mono-ortho planars		
PCB 60	5-760 FW	13
PCB 105	110-9,200 FW	13
PCB 118	290-28,000 FW	13
PCB 156	53-9,200 FW	13
Di-ortho planars		
PCB 128	ND-600 FW	13
PCB 137	50-6,800 FW	13
PCB 138	930-65,000 FW	13
PCB 153	1,100-92,000 FW	13
PCB 170	100-22,000 FW	13
PCB 180	330-61,000 FW	13
PCB 194	20-8,900 FW	13
Booted eagle, <i>Hieraaetus pennatus</i> ; infertile eggs; total PCBs; Spain, 1985-86	1,500 (500-8,400) FW	7
Loggerhead shrike, <i>Lanius ludovicianus</i> ; eggs vs. carcasses; total PCBs; Virginia, 1985-88	940 FW; Max. 1,300 FW vs. <5 FW	14
Herring gull, <i>Larus argentatus</i> Eggs; Great Lakes; total PCBs		
Lake Erie		
1972-74 vs. 1977-80	34,700-130,700 FW vs. 14,600-33,100 FW	26
1983 vs. 1987-88	11,800-14,100 FW vs. 5,600-21,900 FW	26
Lake Huron		
1971-77 vs. 1980-83	43,000-59,500 FW vs. 8,800-10,900 FW	26
1987-88	4,500-11,300 FW	26
Lake Ontario		
1971-72 vs. 1974-77	58,800-143,900 FW vs. 36,200-96,600 FW	26
1980-83 vs. 1987	15,900-27,500 FW vs. 14,400-15,800 FW	26
Lake Superior; 1973-77 vs. 1980-87	37,100-46,400 FW vs. 5,500-11,400 FW	26
Eggs; Great Lakes		
PCB 77	0.6-3.0 LW	6
PCB 105	50-860 LW	6
PCB 118	180-2,550 LW	6
PCB 126	2-10 LW	6
PCB 169	0.2-9.0 LW	6

Table 11. Continued.

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Infertile eggs; Spain, 1988		
PCB 138	Max. 450 FW	12
PCB 153	Max. 400 FW	12
PCB 180	Max. 280 FW	12
Total PCBs	2,000 (1,100-3,600) FW	12
Lake Ontario, 1985		
Adults		
Total PCBs		
Carcass	47,000 FW	15
Diet (fish)	510 FW	15
Liver	12,000 FW	15
Total tetra-chlorobiphenyls (PCBs 56, 60, 66, and 74); carcass vs. liver	1,490 FW vs. 450 FW	15
Total penta-CBs (PCBs 99, 101, 105, 110, and 118); carcass vs. liver	7,440 FW vs. 2,050 FW	15
Total hexa-CBs (PCBs 128, 132, 137, 138, 141, 146, 149, 153); carcass vs. liver	19,900 FW vs. 5,100 FW	15
Total hepta-CBs (PCBs 170, 171, 172, 174, 177, 178, 180, 182, 183, 187, 190, 197); carcass vs. liver	14,400 FW vs. 3,700 FW	15
Total octa-CBs carcass vs. liver	3,400 FW vs. 860 FW	15
Eggs		
Total PCBs	16,000 FW	15
Total tetra-CBs	560 FW	15
Total penta-CBs	2,880 FW	15
Total hexa-CBs	7,250 FW	15
Total hepta-CBs	4,340 FW	15
Total octa-CBs	860 FW	15
Yellow-legged herring gull, <i>Larus cachinnans</i>; eggs; Italy, 1981-86		
Total PCBs (30 congeners)	30,400-56,100 DW	16, 17
PCB 138	4,600-6,800 DW	16
PCB 153	7,800-14,100 DW	16
PCB 180	3,900-7,000 DW	16
Audouin's gull, <i>Larus audouinii</i>; eggs Italy, 1981-86; total PCBs	28,600-45,900 DW	17
Spain, 1988		
Total PCBs	9,000 (4,700-20,500) FW	12
PCB 138	Max. 1,800 FW	12
PCB 153	Max. 1,600 FW	12
PCB 180	Max. 1,300 FW	12
Red-breasted merganser, <i>Mergus serrator</i>; eggs; Michigan; 1977-78 vs. 1990		
Total PCBs	23,000 FW vs. 11,100 FW	28
PCB 77	24 FW vs. 20 FW	28
PCB 81	5 FW vs. 3 FW	28
PCB 126	13 FW vs. 6 FW	28
PCB 169	1.6 FW vs. 0.9 FW	28
PCB 105	454 FW vs. 205 FW	28
PCB 118	847 FW vs. 415 FW	28
PCB 101	246 FW vs. 115 FW	28
PCB 138	1,417 FW vs. 659 FW	28
PCB 153	2,313 FW vs. 1,221 FW	28
PCB 180	641 FW vs. 327 FW	28
Black kite, <i>Milvus migrans</i>; infertile eggs; total PCBs; Spain, 1985-86	2,900 (500-18,700) FW	7
Black-crowned night heron, <i>Nycticorax nycticorax</i>; embryos; total PCBs		
Control site, Virginia	1,130 (240-4,000) FW	18
Cat Island, Green Bay, Wisconsin	9,300 (2,400-53,000) FW	18
San Francisco Bay	900-2,600 (ND-12,000) FW	18
Osprey, <i>Pandion haliaetus</i>; liver vs. eggs; Norway, 1965-83; dead on collection	5,000 (<100-26,000) FW vs. 3,200 (700-8,300) FW	1
Double-crested cormorant, <i>Phalacrocorax auritus</i>; eggs; Great Lakes; total PCBs	900-7,300 FW; reduced hatch at >6,500 FW	27

Table 11. Continued.

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Great cormorant, <i>Phalacrocorax carbo</i>; yolk sac;		
Netherlands		
PCB 105	Max. 25,000 LW	19
PCB 118	Max. 75,000 LW	19
PCB 156	Max. 20,000 LW	19
PCB 157	Max. 10,000 LW	19
PCB 167	Max. 15,000 LW	19
Great cormorant, <i>Phalacrocorax carbo sinensis</i>;		
age <2 years vs. age >2 years; Germany, 1985-86		
Brain		
PCB 28	100 FW vs. 50 FW	20
PCB 52	100 FW vs. 100 FW	20
PCB 101	20 FW vs. 10 FW	20
PCB 138	300 FW vs. 900 FW	20
PCB 153	200 FW vs. 1,500 FW	20
PCB 180	100 FW vs. 600 FW	20
Liver		
PCB 28	200 FW vs. 100 FW	20
PCB 52	80 FW vs. 100 FW	20
PCB 101	100 FW vs. 50 FW	20
PCB 138	800 FW vs. 2,400 FW	20
PCB 153	800 FW vs. 3,700 FW	20
PCB 180	300 FW vs. 1,000 FW	20
Subcutaneous fat		
PCB 28	1,000 FW vs. 1,400 FW	20
PCB 52	400 FW vs. 1,200 FW	20
PCB 101	800 FW vs. 6,100 FW	20
PCB 138	13,000 FW vs. 85,000 FW	20
PCB 153	20,000 FW vs. 93,000 FW	20
PCB 180	5,800 FW vs. 53,000 FW	20
White spoonbill, <i>Platalea leucorodia</i>; infertile eggs;	600 (400-1,300) FW	7
total PCBs; Spain, 1985-86		
Caspian tern, <i>Sterna caspia</i>; blood plasma; total PCBs		
(Aroclors 1242, 1248, 1254, 1260); Great Lakes, 1990		
Green Bay	3,500 (900-13,800) FW	21
Saginaw Bay	2,500 (1,600-3,800) FW	21
Other locations	900-2,100 (400-3,600) FW	21
Forster's tern, <i>Sterna forsteri</i>; whole eggs and chicks;	Max. 5,100-9,500 FW vs. Max. 3,800-	9, 22
total PCBs; Green Bay, Wisconsin; 1988 vs. 1989	8,500 FW	
Common tern, <i>Sterna hirundo</i>; eggs and chicks;		
total PCBs; Green Bay, Wisconsin, 1989	5,000-14,100 FW	9
Arctic tern, <i>Sterna paradisaea</i>; prefledgling carcass;		
German Wadden Sea, 1988; found dead; age 2-14		
days vs. age 15-27 days		
PCB 26	2,800 LW vs. 500 LW	23
PCB 44	200 LW vs. 40 LW	23
PCB 49	200 LW vs. 40 LW	23
PCB 128	2,200 LW vs. 400 LW	23
PCB 138	15,800 LW vs. 4,000 LW	23
PCB 153	25,700 LW vs. 5,000 LW	23
PCB 180	8,600 LW vs. 1,700 LW	23
PCB 183	2,200 LW vs. 300 LW	23
PCB 187	3,300 LW vs. 600 LW	23
PCB 194	700 LW vs. 20 LW	23
Tawny owl, <i>Strix aluco</i>; liver vs. eggs; total PCBs;	700 (<100-70,000) FW vs. 1,100	1
Norway, 1965-83; dead on collection	(300-6,600) FW	
Northern gannet, <i>Morus bassanus</i>; total PCBs		
(35 congeners); England, 1988-90		
Fat	14,600 FW; 18,000 LW	5
Liver	700 FW; 184,000 LW	5
Muscle	2,900 FW; 20,000 LW	5
Tree swallow, <i>Tachycineta bicolor</i>; eggs and chicks;	10,800-13,100 FW	9
total PCBs; Green Bay, Wisconsin, 1989		
Guillemot, <i>Uria aalge</i>; total PCBs (35 congeners);		
England, 1988-90		
Brain	Max. 3,200 FW; Max. 56,000 LW	5
Fat	Max. 450,000 FW; Max. 659,000 LW	5

Table 11. Continued.

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Gizzard and contents	Max. 137,000 FW; Max. 563,000 LW	5
Kidney	Max. 5,900 FW; Max. 321,000 LW	5
Liver	Max. 1,500 FW; Max. 354,000 LW	5
Muscle	Max. 1,100 FW; Max. 67,000 LW	5
Waterfowl, 8 species; eggs; total PCBs; Italy	500-24,000 DW	24

^aConcentrations are shown as means, range (in parentheses), maximum (Max.), and nondetectable (ND).

^b1, Froslic et al. 1986; 2, Elliott and Shutt 1993; 3, Borlakoglu et al. 1991b; 4, Hernandez et al. 1989; 5, Boumphrey et al. 1993; 6, Kennedy et al. 1992; 7, Hernandez et al. 1988; 8, Ormerod and Tyler 1994; 9, Jones et al. 1993; 10, Jarman et al. 1993; 11, Zupancic-Kralj et al. 1992; 12, Gonzalez et al. 1991; 13, Falandysz et al. 1994a; 14, Blumton et al. 1990; 15, Braune and Norstrom 1989; 16, Focardi et al. 1988a; 17, Leonzio et al. 1989; 18, Rattner et al. 1993; 19, van den Berg et al. 1992; 20, Scharenberg 1991b; 21, Mora et al. 1993; 22, Ankley et al. 1993; 23, Scharenberg 1991a; 24, Focardi et al. 1988b; 25, Smith et al. 1985; 26, Turle et al. 1991; 27, Jones et al. 1994; 28, Williams et al. 1995.

eggshell thinning and low reproductive success (Falandysz et al. 1994a). A total lack of reproduction among white-tailed sea eagles in the coastal area of the southwestern Baltic Sea in the 1960s and 1970s may be related, in part, to high concentrations of PCBs 105, 118, 126, and 156 in tissues of adult eagles. It is noteworthy that concentrations of planar PCBs in adult white-tailed sea eagles were among the highest reported in wildlife and that total PCB concentrations in this species were similar to those reported in dead eagles from Sweden and Finland in the 1960s and 1970s (Falandysz et al. 1994a).

PCB 153 is the most widespread PCB in the environment because it is easily stored and retained in adipose tissue; PCB 153 was the main PCB congener in eggs of eight examined species of Italian waterfowl and accounted for 11.4-21.2% of the total PCB concentration (Focardi et al. 1988b). Infertile eggs of the endangered imperial eagle (*Aquila heliaca adalberti*) contained as much as 28.9 mg total PCBs/kg FW; PCB 153 constituted 13.5% of the total PCB loading, PCB 180 13%, PCB 138 3.2%, PCB 101 3.2%, and PCB 118 0.7% (Hernandez et al. 1989). In the endangered Audouin's gull (*Larus audouinii*), most (62%) of the total PCB burden consisted of PCBs 153, 138, 170, and 180; other important congeners were PCBs 118, 194, and 203, and each contributed about 5% (Leonzio et al. 1989). PCBs 138, 153, and 180 comprised more than 50% of the total PCB burden in eggs of the yellow-legged herring gull (*Larus cachinnans*); a similar case is made for eggs of other species of marine birds (Focardi et al. 1988a). PCBs 138, 153, and 180 were also dominant in tissues of most birds collected in Great Britain between 1988 and 1990, although total PCB concentrations ranged from 0.02 to 105 mg/kg FW and also differed considerably in different tissues from individual birds

(Boumphrey et al. 1993). PCBs 138 and 153 were the most prominent congeners in eggs of 3 species of gulls collected in Spain during 1988, accounting for 10.5% and 8.7%, respectively, of the total PCB burden; other important congeners were PCBs 180 (7.5%), 170 (3.2%), 101 (1.9%), 151 (1.1%) and 194 (0.9%; Gonzalez et al. 1991).

PCB signatures in bird eggs are not constant. Eggs of the dipper (*Cinclus*) from Wales and Ireland were dominated by PCB 118 in 1990, PCB 170 in 1991, and PCB 153 in 1992; 6 congeners accounted for 26-35% of the total PCBs in Welsh eggs and for 10-26% of the total in eggs from Ireland (Ormerod and Tyler 1992, 1994). In tissues of birds in Great Britain, the mono-*ortho* congeners—PCBs 105 and 118—made a high contribution (70%) to the TEF, whereas the non-*ortho* congeners (PCBs 77, 126, 169) contributed 20%, and the di-*ortho* congeners (PCBs 138, 153, 180) contributed 10% (Boumphrey et al. 1993). Young of all avian species sampled in Wisconsin accumulated PCBs 77, 105, 126, and 169. Chicks of Forster's terns (*Sterna forsteri*) had daily uptakes of 15 µg total PCBs, 0.07 µg PCB 77, 0.2 µg PCB 105, 0.006 µg for PCB 126, and 0.00014 µg PCB 169 (Ankley et al. 1993).

Concentrations of mono-*ortho* PCBs in yolk-sacs of cormorants ranged from 10 to 250 mg/kg LW; high PCB residues in yolk were associated with increased cytochrome P450 and EROD activities and decreased thyroid hormone activity (van den Berg et al. 1992). Embryos of the black-crowned night-heron (*Nycticorax nycticorax*) with the greatest burdens of total PCBs had increased cytochrome P450-associated monooxygenase activities and cytochrome P450 proteins, which suggests that cytochrome P450 may be a useful biomarker of exposure to some PCB mixtures (Rattner et al. 1993). The absence of

established thresholds for P450 induction indicates more research is needed (Rattner et al. 1994) to make this a useful technique for evaluating PCB exposure.

Mammals

The highest total PCB concentrations recorded in terrestrial mammalian wildlife occurred in fat and liver tissues of species collected near urban areas; di-*ortho* congeners were the major contributors to PCB tissue burdens (Table 12). Atmospheric transport of PCBs governed uptake in terrestrial mammalian herbivores and predators; for example, PCB residues in tissues of voles and shrews in the Scandinavian peninsula directly correlated with fallout loadings (Larsson et al. 1990). An increase in atmospheric deposition of PCBs increased PCB burdens in plants, herbivores, and predators of the herbivores. But herbivores and predators differentially metabolized PCBs, raising concentrations of highly-chlorinated congeners in predators and concentrations of the more easily metabolized low-chlorinated PCBs in herbivores (Larsson et al. 1990).

Populations of mink (*Mustela vison*) declined in many areas of the world, and the declines were linked to exposures to synthetic halogenated hydrocarbons (Giesy et al. 1994b). In the Great Lakes region, mink density is lower along the shores of the Great Lakes and their tributaries where mink have access to fishes from the Great Lakes. Tissue PCB concentrations and their dioxin TEFs were considered critical in the hazard assessment of PCBs. Mink that consumed fishes below dams in Michigan were 10 to 20 times more likely to suffer PCB damage than mink consuming fishes from above the dams, as judged by the elevated concentrations of total PCBs and dioxin TEFs in fishes from below the dams (Giesy et al. 1994b). European polecats (*Mustela putorius*) collected in the Netherlands between 1985 and 1990 had PCB patterns that were independent of diet and seemed to be controlled by anal gland secretions containing elevated PCB residues (Leonards et al. 1994). Juvenile polecats contained higher PCB concentrations than adult males and females, and this is attributed to an increased elimination of PCBs by adults through anal gland secretions. In all examined polecat tissues, PCB 126 accounted for 63 to 98% of the 2,3,7,8-TCDD toxic equivalents (Leonards et al. 1994).

The use of PCBs in Germany was prohibited in 1989. From 1983 to 1991, the body fat of red foxes

(*Vulpes*) in Germany showed a reduction in the mean concentration of highly-chlorinated PCBs (PCBs 138, 153, and 180) but an increase in the lower-chlorinated congeners (PCBs 24, 49, and 52). These findings suggest a trend toward a reduction of environmental contamination with highly-chlorinated biphenyls since 1983, perhaps as a consequence of metabolic degradation, whereas contamination with lower-chlorinated biphenyls from diverse sources is increasing (Georgii et al. 1994). Low-chlorinated congeners that are metabolized via reactive intermediates must be evaluated because they show weak tumor-initiating properties (Georgii et al. 1994).

Populations of bats in Europe have been declining, and PCBs together with pesticides and wood preservatives are the suspected main causes of the decline (Fernandez et al. 1993). Three species of bats collected in Spain in 1988-90 contained only a few dominant PCB congeners; PCBs 138, 153, and 180 accounted for about 80% of the total PCB burden in whole bats (Fernandez et al. 1993). But the most abundant PCB congeners in brain and liver of European otters (*Lutra*) were in the descending order of PCBs 163, 153, 138, and 170, each constituting at least 10% of the total PCB burden (Mason and Ratford 1994).

The PCB composition in tissues of polar bears (*Ursus maritimus*) suggest that polar bears—unlike other mammals—can readily metabolize PCB congeners with unsubstituted *para* positions and unsubstituted adjacent *ortho-meta* positions (Norheim et al. 1992). Six PCB congeners (PCBs 99, 138, 153, 170, 180, 194)—all with a minimum 2,2',4,4'-chlorine substitution—accounted for about 99% of the total PCB content in liver and 87% in fat; PCB 153 accounted for 37% of the total PCB loading in liver and 30% in fat. The PCB congener pattern in polar bear liver and adipose tissue is similar and seems to be independent of sex, age, nutritional status, collection locale, and PCB body burden (Norheim et al. 1992).

Lethal and Sublethal Effects

General

In all tested organisms, PCBs—especially PCBs with 2,3,7,8-TCDD-like activity—adversely affected patterns of survival, reproduction, growth, metabolism, and accumulation. Common manifestations of PCB exposure in animals include hepatotoxicity (hepatomegaly, necrosis),

Table 12. PCB concentrations in field collections of selected mammals. Concentrations are in $\mu\text{g/kg}$ (ppb) fresh weight (FW) or lipid weight (LW).

Species, tissue, PCB congener, and other variables	Concentration ^a in $\mu\text{g/kg}$	Reference ^b
Bats; whole body less wings, feet, and head;		
total PCBs; Spain, 1988-90		
Schreiber's bat, <i>Miniopterus schreibersi</i>	760 LW	1
Common pipistrelle, <i>Pipistrellus pipistrellus</i>	1,290 LW	1
Greater horseshoe bat, <i>Rhinolophus ferrumequinum</i>	480 LW	1
All species		
Madrid vs. other locations	2,980 LW vs. <560 LW	1
Immatures vs. adults	1,940 LW vs. 800 LW	1
Domestic dog, <i>Canis familiaris</i>; muscle fat;		
Germany, 1987		
PCB 28	<1 LW	2
PCB 49	5 (6-8) LW	2
PCB 52	<1 LW	2
PCB 101	3 (ND-9) LW	2
PCB 138	11 (3-25) LW	2
PCB 153	22 (7-58) LW	2
PCB 180	47 (6-153) LW	2
Human, <i>Homo sapiens</i>;		
Adipose tissue; United States; 1982 vs. 1986		
Tetra-CBs	16 FW vs. 56 FW	7
Penta-CBs	78 FW vs. 135 FW	7
Hexa-CBs	176 FW vs. 314 FW	7
Hepta-CBs	85 FW vs. 125 FW	7
Total PCBs	407 FW vs. 672 FW	7
Maternal milk; New York State, 1988-90; maximum values		
PCB 77	1.3 LW	3
PCB 81	1.1 LW	3
PCB 105	6.7 LW	3
PCB 114	1.3 LW	3
PCB 118	8.7 LW	3
PCB 123	6.2 LW	3
PCB 126	3.6 LW	3
PCB 156	3.2 LW	3
PCB 157	9.4 LW	3
PCB 167	8.8 LW	3
PCB 169	ND	3
PCB 189	4.7 LW	3
Total PCBs	52.9 (3.4-179) LW; 1.5 FW	3
European otter, <i>Lutra lutra</i>; total PCBs		
Ireland; brain vs. liver	4,700 (1,200-14,400) LW vs. 42,800 (18,500-92,100) LW	4
Denmark; liver	58,000 (22,000-104,000) LW	4
England; liver	51,000 (2,000-190,000) LW	4
European polecat, <i>Mustela putorius</i>; the		
Netherlands, 1985-90		
Anal gland		
Total PCBs	32,000 (5,000-104,000) LW	5
Non-ortho PCBs	6.9 LW	5
Di-ortho PCBs	28,000 (4,000-88,000) LW	5
Mono-ortho PCBs	3,700 (700-13,000) LW	5
Fat (mesenteric)		
Total PCBs	51,000 (1,000-370,000) LW	5
Non-ortho PCBs	2.7 LW	5
Di-ortho PCBs	49,000 (2,000-350,000) LW	5
Mono-ortho PCBs	1,800 (100-11,000) LW	5
Kidney		
Total PCBs	22,000 (2,000-114,000) LW	5
Non-ortho PCBs	7.6 LW	5
Di-ortho PCBs	20,000 (1,000-107,000) LW	5
Mono-ortho PCBs	1,100 (200-4,200) LW	5
Liver		
Total PCBs	48,000 (4,000-260,000) LW	5
Non-ortho PCBs	5.5 LW	5
Di-ortho PCBs	45,000 (4,000-248,000) LW	5
Mono-ortho PCBs	1,200 (300-3,400) LW	5

Table 12. *Continued.*

Species, tissue, PCB congener, and other variables	Concentration ^a in µg/kg	Reference ^b
Muscle		
Total PCBs	28,000 (3,000-150,000) LW	5
Non-ortho PCBs	2.8 LW	5
Di-ortho PCBs	26,000 (3,000-140,000) LW	5
Mono-ortho PCBs	1,300 (200-5,700) LW	5
Polar bear , <i>Ursus maritimus</i> ; total PCBs; Norway, 1978-89; liver vs. fat		
Adults	13,000 FW vs. 31,000 FW	6
Juveniles	12,000 FW vs. 15,000 FW	6
Red fox , <i>Vulpes vulpes</i> ; muscle fat; Germany, 1983 vs. 1991; maximum values		
PCB 28	160 LW vs. 50 LW	2
PCB 49	<1 LW vs. 30 LW	2
PCB 52	130 LW vs. 50 LW	2
PCB 101	240 LW vs. 90 LW	2
PCB 138	2,720 LW vs. 310 LW	2
PCB 153	4,300 LW vs. 1,000 LW	2
PCB 180	7,890 LW vs. 2,080 LW	2

^aConcentrations are shown as means, range (in parentheses), maximum (Max.), and nondetectable (ND).

^b1, Fernandez et al. 1993; 2, Georgii et al. 1994; 3, Hong et al. 1994; 4, Mason and Ratford 1994; 5, Leonards et al. 1994; 6, Norheim et al. 1992; 7, USEPA 1994.

immunotoxicity (atrophy of lymphoid tissues, suppressed antibody responses), neurotoxicity (impaired behavior and development, catecholamine alterations), increased abortion, low birth weight, embryoletality, teratogenicity, gastrointestinal ulceration and necrosis, bronchitis, dermal toxicity (chloracne, edema, hyperplasia), weak mutagenicity at high doses, and preneoplastic changes at low doses (Hansen 1987). At concentrations above a threshold, PCBs are potent promoters of hepatic carcinogenesis in laboratory rodents; however, there is no clear evidence of carcinogenicity of PCBs to human and animal populations from natural exposure (Hayes 1987). Induction of hepatic microsomal enzymes is one of the earliest and most sensitive responses to PCBs (Hansen 1987).

PCB-induced toxicity patterns are highly variable. Variability, as discussed later, is attributed in part to differences between species and strains in ability to metabolize PCBs and in primary sites of action; in the age, growth rate, biomass, and lipid content of the species; in dose rate, duration of exposure, route of administration, and tested congeners; in physicochemical characteristics of the habitat during exposure; and in PCB interactions with other PCBs, other organochlorine compounds, and heavy metals. Chinook salmon (*Oncorhynchus tshawytscha*) had decreased hatch when eggs contained the equivalent of 0.1 µg 2,3,7,8-TCDD/kg fresh weight (FW); domestic chickens (*Gallus sp.*) had decreased survival and

increased developmental abnormalities when embryos had 20 µg PCB 77/kg FW; mink (*Mustela vison*) had reduced growth when fed 100 µg Aroclor 1254/kg BW daily and reduced survival at 50 µg PCB 169/kg diet; rhesus macaques (*Macaca mulatta*) had reproductive impairment when fed more than 8 µg Aroclor 1016/kg BW daily; and rats (*Rattus sp.*) had reduced litter sizes and survival when given 10 µg PCB 126/kg BW daily during gestation.

Aquatic Organisms

PCBs influence patterns of survival, reproduction, growth, enzyme activities, and accumulation in representative aquatic organisms (Table 13). Some PCB congeners at laboratory concentrations that were several orders of magnitude higher than those encountered under field conditions killed 47 to 83% of tested freshwater fishes and invertebrates in 24-48 h; however, most PCB tested congeners produced negligible mortality under these conditions (Dillon and Burton 1992; Table 13). Mortality increased when PCB 133 or PCB 177 concentrations in whole guppies (*Poecilia reticulata*) exceeded 1 µmole/g, equivalent to more than 200 mg PCB/kg whole body FW or about 4,000 mg PCB/kg on a lipid weight basis (Opperhuizen and Schrap 1988). PCBs—especially those with TCDD-type activity—adversely affect reproductive success of spawning female chinook salmon. Chinook salmon eggs that contained total PCB

Table 13. Effects of PCBs on representative aquatic organisms.

Taxonomic group, organism, PCB congener, dose, and other variables	Effect	Reference ^a
Invertebrates		
Daphnid, <i>Daphnia magna</i> ; early life stages		
PCB 1; 710 µg/L	LC50 (24h)	
PCB 2; 430 µg/L	LC50 (24h)	
PCB 3; 420 µg/L	LC50 (24h)	
PCB 18; 86 µg/L	47% dead in 48 h	
PCBs 28 (1.5 µg/L), 52 (74 µg/L), 77 (0.3 µg/L), 101 (1.2 µg/L), 116 (2.8 µg/L), 128 (0.4 µg/L), 153 (1.3 µg/L), 171 (1.7 µg/L), 194 (3.0 µg/L)	Negligible mortality; 87-100% survival in 48 h	1
PCB 47; 30 µg/L	LC50 (24h)	1
Zebra mussel, <i>Dreissena polymorpha</i>		
Fed alga (<i>Chlorella</i> sp.) containing 500 mg PCB 77/kg for 32 days	Maximum tissue concentration of 3.4 mg PCB 77/kg fresh weight soft parts reached in about 14 days; concentration dropped to about 2 mg/kg after 32 days	2
Fed <i>Chlorella</i> containing 500 mg PCB 169/kg for 50 days, then clean <i>Chlorella</i> diet for 45 days	Maximum tissue concentration of 3.7 mg PCB 169/kg soft parts reached in 10 days, then decline during exposure to equilibrium level of about 1 mg/kg soft parts. After 25 days of clearance soft parts contained 0.1 mg/kg fresh weight	2
Amphipod, <i>Gammarus pseudolimnaeus</i>		
PCBs 8, 15, 32, 155, 101; initial water concentra- tions of 70-210 µg/L	LC50 (96h)	1
Mysid, <i>Mysis relicta</i> ; exposed to radiolabeled PCB 153 for 6 h at 4°C then transferred to clean water for 26 days	Bioconcentration factor from water of 442,230; calculated half-time persistence of 220 day	10
Amphipod, <i>Pontoporeia hoyi</i> ; exposed to radiolabeled PCB 153 for 6 h at 4°C then transferred to clean water for 26 days	Bioconcentration factor from water of 101,660; calculated half-time persistence of 50 days	10
Vertebrates		
Rainbow trout, <i>Oncorhynchus mykiss</i>		
Eggs injected 24 to 50 h after fertilization with graded doses of various PCBs		
PCB 4; >24,200 µg/kg egg fresh weight (FW)	LD50, embryos	13
PCB 28; >24,300 µg/kg egg FW	LD50 embryos	13
PCB 52; >30,400 µg/kg egg FW	LD50, embryos	13
PCB 77 0.578 µg/kg egg FW	At age 35 days, sac-fry contained 78% of original dose	13
1,348 (1,064-1,621) µg/kg egg FW	LD50, embryos; blue-sac syndrome	13, 14
PCB 81; 549 µg/kg egg FW	LD50, embryos; blue-sac syndrome	13
PCB 105 67 µg/kg egg FW	At sac-fry stage 78% of original dose remained	13
>6,970 µg/kg egg FW	LD50, embryos	14
PCB 118 1,330 µg/kg egg FW	About 81% of original dose present in sac-fry	13
>6,970->57,400 µg/kg egg FW	LD50, embryos	13, 14
PCB 126 74 (44-83) µg/kg egg FW	LD50, embryos	13, 14
1,203 µg/kg egg FW	About 88% recovered from embryos	13
PCBs 128, 138, and 156; >115,000 µg/kg egg FW	LD50, embryos	13
PCB 153; >6,200 µg/kg FW	LD50, embryos	14
PCB 169; 7,110 (5,630-8,090) µg/kg egg FW	LD50, embryos; blue-sac syndrome	13
PCB 170; >41,100 µg/kg egg FW	LD50, embryos	13
Single intraperitoneal (ip) injection; 134 µg/kg BW of PCB 77; or 5.8 µg/kg BW of PCB 126; or 93.7 µg/kg BW of PCB 169; subadults	50% increase in liver EROD activity	12
Single ip injection of 1.0 mg PCB 77/kg BW; livers analyzed 13 days postinjection; subadults	Compared to controls, livers were elevated in levels of cytochrome P-450 (2X), ethoxycoumarin-O-deethylase (10X), and	11

Table 13. Continued.

Taxonomic group, organism, PCB congener, dose, and other variables	Effect	Reference ^a
Injected ip with 2,3,7,8-TCDD at 0.00037, 0.0022 or 0.0045 µmol/kg BW, or with PCB 77 at 0.2, 1.1, or 2.2 µmol/kg BW; subadults	ethoxyresorufin-O-deethylase (50X) 50% induction of AHH (arylhydrocarbon hydroxylase) activity in liver microsomes of sexually immature trout at 0.002 µmol/kg for TCDD and 0.37 µmol/kg for PCB 77	3
Single ip injection of mixture of 0.00018 µmol TCDD/kg BW plus 0.1 µmol/kg PCB 77; or mixture of 0.0011 µmol TCDD/kg BW plus 0.6 µmol PCB 77/kg BW; subadults	Mixtures of TCDD and PCB 77 produced greater than additive AHH responses	3
Single ip injection of mixture of 0.0022 µmol TCDD/kg BW plus 1.1 µmol PCB 77/kg BW; subadults	AHH induction response was less than additive	3
Single ip injection of PCB 77, PCB 126, or 2,3,7,8-TCDD. Liver AHH activity measured 3 days later; subadults	50% AHH induction occurred at 0.005 µmol/kg BW for TCDD, 1.0 for PCB 77 and 2.2 for PCB 126. PCB 77 was 1/200 as effective in inducing AHH activity in liver as TCDD, and PCB 126 was 1/500 as effective	4
Single ip injection of 30 mg PCB 118/kg BW to immatures. Fish killed 4 days later and liver analyzed	Liver EROD (7-ethoxyresorufin O-deethylase) activity was 5.6 times higher than controls; AHH activity was 2.7 times higher than controls	5
Fingerlings were injected ip with ¹⁴ C-labeled PCB mixture at 0.3, 1, 3, 10, and 30 mg PCB/kg BW; tissues sampled up to 70 days postinjection.	At 3 days postinjection high doses of 10 and 30 mg PCB/kg BW caused elevation of liver microsomal monooxygenase activity when maximum tissue concentrations, in mg total PCB/kg FW, were 55 in bile, 12 in blood, 8 in muscle, and 8 in liver. Elevated hepatic microsomal monooxygenase activity with muscle and liver PCB concentrations of >0.3 mg/kg FW, but not 0.25 mg/kg FW	6
Fathead minnow, <i>Pimephales promelas</i> ; fry		
PCB 18; 86 µg/L	83% dead in 48h	1
PCBs 28 (1.5 µg/L), 52 (74 µg/L), 77 (0.3 µg/L), 101 (1.2 µg/L), 116 (2.8 µg/L), 128 (0.4 µg/L), 153 (1.3 µg/L), 171 (1.7 µg/L), 194 (3.0 µg/L)	Negligible mortality; 97-100% survival in 48h	1
Guppy, <i>Poecilia reticulata</i> ; adult males		
Fed diets containing 6-150 mg PCB 133 or PCB 197/kg diet for 191-247 days, or 550-1,400 mg/kg diet for 65 days	At 6-150 mg/kg diet, uptake efficiency was near 50%; at higher dietary loadings uptake efficiency decreased to 25%. No deaths in controls or at low dietary dosages; 13-25% mortality at 550-1,400 mg/kg diet. Prior to death, guppies were sluggish, uncoordinated, and darkly-colored; fish that died during exposure contained >0.7 µmol PCB/g FW	7
Fed PCB 133 at 550 mg/kg diet FW or PCB 197 at 530 mg/kg diet FW for 65 days, followed by a clean diet for 89 days, then reexposure to the contaminated diet for another 37 days	No PCB clearance during initial exposure; significant elimination of both PCBs when fed a clean diet. During reexposure, uptake efficiency for both PCBs was significantly higher than during the initial exposure.	8
Scup, <i>Stenotomus chrysops</i>		
Injected intraperitoneally with 1,5, or 10 mg/kg BW of PCBs 77, 105, 118, 128, or 138 and examined for increases in ethoxyresorufin-O-deethylase (EROD) activity, immunodetectable cytochrome P450E (the EROD catalyst in scup) and in vitro translatable mRNA for P450E	Monooxygenase parameters were significantly induced only by PCB 77; translatable mRNA for P450E was induced at all doses; EROD activity and P450E were decreased at the 5 and 10 mg/kg BW doses; a positive correlation was established between PCB 77 residues in liver and decreased EROD activity at the higher doses	9

^a1, Dillon and Burton 1992; 2, Brieger and Hunter 1993; 3, Janz and Metcalfe 1991b; 4, Janz and Metcalfe 1991a; 5, Skaare et al. 1991; 6, Melancon et al. 1989; 7, Schrap and Oppenhuizen 1988; 8, Oppenhuizen and Schrap 1988; 9, Gooch et al. 1989; 10, Evans and Landrum 1989; 11, Tyle et al. 1991; 12, Newsted et al. 1995; 13, Zabel et al. 1995; 14, Walker and Peterson 1991.

concentrations equivalent to 0.1 μg 2,3,7,8-TCDD equivalents/kg eggs and higher had a dose-dependent decrease in hatching success (Ankley et al. 1991). PCBs also impair the reproductive capacities of marine mammals (Kannan et al. 1993).

The relation between PCB accumulation by the freshwater alga *Scenedesmus* sp. and the compound's octanol/water partition coefficient (K_{ow}) was measured with 40 PCB compounds in a log K_{ow} range of 4.46 to 8.18, PCB concentrations between 0.03 and 1.1 $\mu\text{g/L}$, and exposures between 20 and 30 days (Swackhammer and Skoglund 1993). The accumulation process was consistent with partitioning from water into cell lipids but was slower than the growth of *Scenedesmus* (i.e., no significant uptake of PCBs from congeners with log $K_{ow} > 5.0$ under conditions of rapid growth or $> \log 7.0$ under conditions of slow growth). Thus, under nonwinter field conditions, many PCB congeners never reached equilibrium concentrations (Swackhammer and Skoglund 1993). Similar results are reported of other species of freshwater algae, including *Selenastrum capricornutum*, *Anabaena* sp., and *Synedra* sp. (Stange and Swackhammer 1994).

Zebra mussels (*Dreissena polymorpha*) accumulated PCB 77 from their diet and from the surrounding lake sediments (Brieger and Hunter 1993). An uptake rate of PCB 77 by zebra mussels followed the descending order of sediment, food, and water. Tissue concentrations in mussels peaked after 10-14 days at 3.4-3.7 mg PCB 77/kg FW soft parts; equilibrium levels of PCB 77 were near 1.0 mg/kg FW. Zebra mussels are more efficient accumulators of PCBs than other bivalve mollusks to which they are attached; accordingly, high densities of zebra mussels probably influence contaminant dynamics (Brieger and Hunter 1993). A freshwater crustacean (*Mysis relicta*) plays an important role in the transfer of PCBs from sediments into the Lake Champlain food web (Lester and McIntosh 1994), and freshwater grazing and shredding benthic invertebrates promote downstream transport of PCB 153 (Sallenave et al. 1994). Marine invertebrates accumulated PCBs 52, 101, 128, 138, 151, 153, 180, 194, 206, and 209 from PCB-contaminated sediments (Pruell et al. 1993). Clams (*Macoma nasuta*) reached a steady state equilibrium in 10 days, but sandworms (*Nereis virens*) took 70-120 days. Clams showed preferential accumulation of lower molecular weight PCB congeners, and this may be due to the comparatively low lipid content in this species. Sandworms and grass shrimp (*Palaemonetes*

pugio) metabolized PCBs 52, 101, and 151 (Pruell et al. 1993).

Golden shiners (*Notemigonus crysoleucas*) rapidly accumulated radiolabeled PCBs from water during 96-h exposure (Karara and McFarland 1992). The uptake rate of PCBs from water was controlled by gill blood-flow rate. About 50% of the accumulated PCBs in shiners was eliminated in 4.9 days, and this is similar to PCB elimination rates in striped bass (*Morone saxatilis*) and channel catfish (*Ictalurus punctatus*; Karara and McFarland 1992). In guppies, residual PCB concentrations increased with increasing duration of exposure; however, steady state concentrations did not occur after dietary exposure for 65 days (Schrapp and Opperhuizen 1988). PCB congeners with *ortho*-chlorine substitutions (PCBs 77, 105, 118, 128, 138) were effective inducers of EROD (7-ethoxyresorufin *O*-deethylase) and AHH (aryl hydrocarbon hydroxylase) activities in marine mammals (Gooch et al. 1989) and freshwater fishes (Janz and Metcalfe 1991a; Skaare et al. 1992) but were ineffective at doses as high as 10 mg/kg BW in scup (*Stenotomus chrysops*), a marine teleost (Gooch et al. 1989). Industrial mixtures of planar and nonplanar PCBs induced AHH in fishes (Skaare et al. 1991). Mixtures of planar PCBs and dioxins, however, produced synergism of AHH activity in fish liver at low doses and antagonism at high doses; the possible antagonistic effects on nonplanar halogenated compounds may further complicate these interactions (Janz and Metcalfe 1991b). Liver is the primary target organ for the induction of cytochrome P450-dependent monooxygenases by PCBs in fishes, and the most frequently examined organ; however, in salmonids, muscle tissue is also suitable for evaluation of hepatic monooxygenase induction, as judged by PCB concentrations in muscle (Janz et al. 1992).

Birds

PCB 126 is among the most toxic of all PCB congeners to birds and the domestic chicken is the most sensitive tested species (Table 14; Hoffman et al. 1995). However, adverse effects of PCBs in birds vary markedly between species and tissues. And birds react differently to different PCB congeners and to PCB-metal mixtures. American kestrels (*Falco sparverius*) differ from Japanese quail (*Coturnix japonica*) after exposure to PCBs 105, 126, and 153; quail accumulated porphyrins in liver but kestrels did not (Elliott et al. 1991).

Table 14. Effects of PCBs on selected birds.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
Mallard, <i>Anas platyrhynchos</i>; PCB 77		
Single injection into egg yolk on day 5 of incubation	No effect on hatching rate; chicks normal	1
0.1 mg/kg egg fresh weight (FW)	No effect on embryo survival and no gross abnormalities	2
5.0 mg/kg egg FW		
Greylag goose, <i>Anser anser</i>		
PCB 77; single injection into egg yolk on day 5 of incubation; 1.0 mg/kg egg FW	No effect on survival or development	2
Goldeneye, <i>Bucephala clangula</i>		
PCB 77; single injection into egg yolk; 1.0 mg/kg egg FW	33% hatch vs. 52% hatch in controls; chicks normal	1
Northern bobwhite, <i>Colinus virginianus</i>; PCB126;	LD50 through hatching	16
single injection of egg with 0.04-0.07 mg/kg FW		
Japanese quail, <i>Coturnix japonica</i>		
Adults given a single oral dose of either PCB 47 at 87.6 mg/kg body weight (BW), PCB 77 at 87.6 mg/kg BW, or Aroclor 1242 at 100 to 500 mg/kg BW. Quail were killed 48 h postdosing and liver and intestine analyzed for porphyrins and cytochrome P450 monooxygenases	All compounds caused a significant increase in EROD activity and porphyrin content in small intestine and liver, and a significant increase in hepatic P450 content	3
Adults fed diets for 30 days containing 100 mg cadmium/kg diet, 100 mg Aroclor 1260/kg diet, or mixture of 100 mg cadmium plus 100 mg Aroclor 1260/kg	Quail fed the mixture diet had 3 times more PCBs in muscle than the Aroclor group alone, and 70 times more PCBs than controls. The most dominant PCB congeners were 138, 153, 170, and 180	4
American kestrel, <i>Falco sparverius</i>		
Aroclor 1248; fed diets containing 3 mg/kg ration for 6 months; carcasses and eggs analyzed for total PCBs (Aroclors 1248, 1254, and 1260)	Carcasses of adults had 18.5 mg total PCBs/kg FW (vs. 3.3 in controls); eggs had 5.6 mg/kg FW vs 1.5 in controls; shell thickness of eggs reduced 5%	6
Fed diets for 4 weeks containing daily doses equivalent to 3 mg PCB 105/kg BW, 0.05 mg 126/kg BW, or 4.0 mg PCB 153/kg BW. Birds were killed 3 days after final treatment and livers analyzed for porphyrins, PCB residues, and activities of ethoxyresorufin O-deethylase (EROD), aminopyrine N-demethylase (APND), and aldrin epoxidase (AE)	PCB 105 caused significant APND induction; liver had 182.0 mg PCB 105/kg FW vs. 2.4 in corn oil controls. PCB 126 induced hepatic EROD and AE; liver had 3.3 mg PCB 126/kg FW. PCB 153 induced APND and AE; liver contained 119.0 mg PCB 153/kg FW. For all dose groups, liver weights and liver porphyrin levels were normal	6
Nestlings given daily doses of PCBs 77, 105, or 126 for 10 days		
PCB 77; 1.0 mg/kg BW daily	Liver contained 892 µg PCB 77/kg FW; onset of liver necrosis	16
PCB 105; 4.0 mg/kg BW daily	Liver contained 1,677 µg PCB 105/kg FW; liver necrosis	16
PCB 126		
50 µg/kg BW daily	Liver contained 158 (68-563) µg PCB126/kg FW; pronounced liver enlargement; lymphoid depletion of spleen	16
250 µg/kg BW daily	Liver had 380 µg PCB 126/kg FW	16
1.0 mg/kg BW daily	Liver had 1.1 (0.6-4.5) mg PCB 126/kg BW; decreased body weight	16
Egg injected through air cell with 70-100 µg PCB 126/kg FW	LD50 through hatching	16
Domestic chicken, <i>Gallus sp.</i>		
PCB 77; single dose injected into egg yolk on day 4 of fertilization		
0.0006 mg/kg egg FW	50% increase in AHH activity after 48 h	7
0.004 mg/kg egg FW	60% hatch vs. 88% in controls	1, 8
0.010 mg/kg egg FW	17% dead in 18 days; reduced thymus weight	7
0.020 mg/kg egg FW	70-100% dead by day 18; treated embryos had increased frequency of liver lesions, subcutaneous edema, shortened beaks, and microphthalmia	1, 2, 8
0.050 mg/kg egg FW	All dead within 18 days	7
0.100 mg/kg egg FW	Zero hatch; all dead	8
PCB 77; single dose injected into air sac on day 3 of incubation; 0.005 mg/kg egg FW	Whole liver homogenates had increases of 300-fold in 7-ethoxycoumarin O-deethylase	15

Table 14. Continued.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
	and 75-fold in AHH activities between days 5 and 10	
PCB 77; single dose injected into air sac on day 13 of incubation		
0.045 mg/kg egg FW	Reduction by 50% in lymphoid cells in bursa of Fabricius	9
0.20-0.30 mg/kg egg FW	30-fold increase in AHH activity; bursa almost devoid of lymphoid cells	9
PCBs 77, 136, 153, 169; 17-day old embryos; single injection at 0.5-30,000 nmol/egg; examined for liver histopathology and induction of mixed function oxidases after 24 h	Significant alterations occurred at 0.5 nmol PCB 77/egg (about 0.003 mg/kg egg), at 5 nmol PCB 169/egg, and between 500 and 5,000 nmol PCB 153/egg. PCB 136 had no effect on liver pathology at the highest dose tested of 30,000 nmol/egg	10
Single injection of eggs of PCBs 105 (2.2 mg/kg FW), 126 (0.0006-0.0031 mg/kg FW), 157 (1.5-2.0 mg/kg FW), or 169 (0.17 mg/kg FW)	LD50 through hatching	16
PCBs 128, 136, 153, 155, and 169; each was fed in diets to 1-day-old chicks for 21 days at 400 mg/kg ration		
PCB 128	Decrease in weight gain; moderate liver pathology; gross accumulations of hepatic porphyrins and increased delta-aminolevulinic acid synthetase activity	11, 12
PCB 136	Decrease in weight gain; comparatively small increase in liver weight	11
PCB 153	Decrease in weight gain; mild liver pathology	11
PCB 155	Largest increase in liver weight; significant liver pathology	11
PCB 169	All dead in 11 days; thymic involution and edema; gross accumulations of uroporphyrins in liver	11, 12
Herring gull, <i>Larus argentatus</i>		
PCB 77; single injection into egg yolk on day 5 of incubation; 1.0 mg/kg egg FW	No effect on survival or development	2
Black-headed gull, <i>Larus ridibundus</i>		
PCB 77; single injection into egg yolk of 1.0 mg/kg egg FW	No effect on hatching rate; chicks normal	1
Wild turkey, <i>Meleagris gallopavo</i>		
PCB 77; single injection into air sac of 5-day-old embryos		
0.006 mg/kg egg FW	50% increase in AHH activity after 48 h	7
1.0 mg/kg egg FW	60% dead in 24 days	7
Ring-necked pheasant, <i>Phasianus colchicus</i>		
PCB 77; single injection into egg yolk		
0.1 mg/kg egg FW	No decrease in hatching rate; chicks normal	1
1.0 mg/kg egg FW	All embryos died	1
Common eider, <i>Somateria mollissima</i>; single injection into yolk on day 5 of incubation		
PCB 77; 1.0 mg/kg egg FW	No effect on survival	13
PCB 126; 0.1 mg/kg egg FW	35% dead on day 24 of incubation vs. 20% dead in controls	13
Common tern, <i>Sterna hirundo</i>; PCB 126; single egg injection with 0.045 mg/kg FW	35% embryo mortality through hatching	16
Ringed-turtle dove, <i>Streptopelia risoria</i>; fed iodine-deficient diet		
PCB 77; given 20 mg/kg BW 3 times over a 28-day period	Thyroid hyperplasia caused by low iodine diet was not enhanced by PCB 77	14
PCB 77; single dose of 60 mg/kg BW	Thyroid hyperplasia reversed within 7 days	14

^a1, Brunstrom and Reutergardh 1986; 2, Brunstrom 1988; 3, Miranda et al. 1987; 4, Leonzio et al. 1992; 5, Lowe and Stendell 1991; 6, Elliott et al. 1991; 7, Brunstrom and Lund 1988; 8, Brunstrom and Darnerud 1983; 9, Nikolaidis et al. 1988; 10, Rifkind et al. 1984; 11, McKinney et al. 1976; 12, Goldstein et al. 1976; 13, Brunstrom et al. 1990; 14, Spear and Moon 1985; 15, Brunstrom 1986; 16, Hoffman et al. 1995.

Japanese quail dosed with PCBs 47 or 77 showed marked differences between the abilities of the small intestines and liver to metabolize porphyrin and the induction of cytochrome P450 isozymes and associated monooxygenases (Miranda et al. 1987). Pure hexachlorobiphenyls (HCBs) caused uroporphyrin accumulations and increased delta-aminolevulinic acid synthetase activity in chicken livers, and some HCBs significantly increased cytochrome P450 and *p*-nitrophenol glucuronyl transferase when given in the feed for 3 weeks at 400 mg/kg ration; however, PCBs 128 and 169 caused grosser accumulations of hepatic porphyrins than PCBs 138, 155, and 156 (Goldstein et al. 1976, 1977). PCBs 77, 136, 153, and 159 produced acute histopathological changes in chick embryo livers and selectively induced cytochrome P448-mediated mixed function oxidases; the degree of histopathologic change produced by each of the tested PCBs positively correlated with the degree of P448 inhibition (Rifkind et al. 1984). Interactions of metals with PCBs are not well documented, although some studies with Japanese quail showed that cadmium raises PCB uptake from the diet. In one study, cadmium fed at high dietary levels of 100 mg/kg ration interfered with high levels of dietary PCBs (100 mg/kg ration). In that study, muscle of treated quails had increased loadings of congeners chlorinated in the 2,4,5 position (such as PCBs 138, 153, 170, and 180), and these are comparatively toxic and resistant to metabolic degradation (Leonzio et al. 1992). More research is needed on variables known to modify PCB uptake, retention, translocation, and toxicity.

Embryos of the domestic chicken (*Gallus* sp.) are unusually sensitive to PCB 77. Mortality and a high incidence of developmental abnormalities—including microphthalmia, beak deformities, edema, and retarded growth—were recorded in chicks at 0.02 mg PCB 77/kg egg FW, but no deaths or abnormalities were recorded in embryos of ducks, pheasants, and gulls at 0.1-1.0 mg PCB 77/kg egg FW (Table 14; Brunstrom and Lund 1988). Chicken embryos were 20 to 100 times more sensitive to PCB 77 than embryos of the wild turkey (*Meleagris gallopavo*), and this sensitivity emphasizes the uncertainties of applying toxicity data from one species of bird to predict toxic effects in other avian species (Brunstrom 1988; Brunstrom and Lund 1988). Differences in sensitivity of birds to PCB 77 may be related to differences in metabolism and in the formation of toxic metabolites and to the increased availability of Ah receptors in chicks (Brunstrom and Reutergardh 1986).

For example, Ah receptors were detected in livers of 7-day-old chicken embryos but not in livers of 9-day-old turkey embryos (Brunstrom and Lund 1988).

Birds and mammals exposed to PCBs frequently react differently. PCBs generally elicit large-colloid goiters in birds; these goiters are inherently different from hyperplastic goiters produced in mammals exposed to PCBs (Spear and Moon 1985). Fish-eating seabirds, such as the razorbill (*Alca torda*), can rapidly metabolize PCB congeners that have at least one pair of adjacent unsubstituted *meta-para* combinations in the biphenyl moiety (Borlakoglu et al. 1991b). Razorbills metabolized 4-chlorobiphenyl to 4-chloro-4'-hydroxybiphenyl; however, mice (*Mus* sp.) metabolized the same compound 15 times faster. PCB-exposed razorbills and rock doves (*Columba livia*) had similar concentrations of cytochrome P450 and glutathione-S-transferase enzymes, but concentrations were significantly higher in rats (*Rattus* sp.) than either avian species (Borlakoglu et al. 1991b). The relative potency of tested PCBs—as measured by EROD activity of microsomal liver enzymes—in fertile eggs of the domestic chicken were 0.02 for PCB 77, and less than 0.001 for PCB 169 (Bosveld et al. 1992); these values differ somewhat from those proposed for mammals of 0.0005 for PCB 77, and 0.01 for PCB 169 (Safe 1990). Lymphoid development in the bursa of Fabricius of the avian embryo is inhibited by TCDD-like congeners. PCB 77, for example, affects the immune system by interacting with the Ah receptor, causing inhibition of lymphoid development in the mammalian thymus and in the avian bursa of Fabricius (Nikolaides et al. 1988). More research seems needed on the relation of avian Ah receptors to natural physiological processes.

Mammals

Deleterious effects were significant on growth, survival, reproduction, or metabolism from chronic exposures of sensitive species of tested rodents, primates, and mustelids to daily concentrations as low as 0.008 mg/kg BW of Aroclor 1016, 0.01-0.02 mg/kg BW of PCB 126, 0.01-0.05 mg/kg diet or 0.1 mg/kg BW of PCB 169, 0.09 mg/kg BW of Aroclor 1246, 0.1 mg/kg BW of Aroclor 1254, and 0.3-1.0 mg/kg diet or 0.6 mg/kg BW of PCB 77 (Table 15). Several PCBs had negligible adverse effects on tested mammals during chronic daily exposures to doses of at least 5 mg/kg ration or 5 mg/kg BW, specifically, PCBs 4, 15, 47, 52, 80, 136, 153, 155,

Table 15. Effects of PCBs on selected mammals.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
Cotton top marmoset monkey, <i>Callithrix jacchus</i>		
PCB 77; adult females orally dosed with 0.1, 1.0, or 3.0 mg/kg body weight (BW) twice weekly for 18-28 weeks	Severe toxicity in the 3.0 mg/kg group that included body weight loss, hair loss, abnormal nail growth, scaly skin, anemia, elevated blood triglyceride and cholesterol levels, and tissue histopathology. Toxicity was less severe in the 1.0 mg/kg group and minor in the low-dose group	1
Rhesus macaque, <i>Macaca mulatta</i>		
Aroclor 1016		
>0.008 mg/kg BW daily via the diet	Adverse effects on reproduction	2
>0.028 mg/kg BW daily	Adverse effects on growth	2
Aroclor 1248; >0.09 mg/kg BW daily	Adverse effects on growth	2
Aroclor 1254; females were fed 0, 5, 20, 40, or 80 µg/kg BW daily for 6 years. During this time females were bred with non-dosed males. Resultant offspring were nursed for 22 weeks and fed no additional PCBs until they were necropsied at age 120 weeks	Total PCB concentrations in all tissues of adult females increased with increasing dosage; highest levels were in adipose tissue (Max. 141mg/kg fresh weight [FW]; Max. 171 mg/kg lipid weight [LW]) and lowest in brain (1.1 mg/kg FW; 12.9 mg/kg LW). PCB concentrations were higher in infants from dosed dams than those nursed by controls; higher in females having a stillborn infant than those with a viable infant; and higher in those in poor health. The PCB distribution pattern in tissues from a dosed mother/infant pair was different; more hepta-chlorobiphenyls were found in the infant than in the dam	28
PCB 52		
Fed diets containing 3.0-5.0 mg/kg for 180-200 days	No clinical effects or pathologic lesions	3, 4
PCB 77		
Fed diets containing 0.3-3.0 mg/kg ration for 1 to 6 months	Dose- and time-dependent increase in chloracne; weight loss; death; and histopathology of sebaceous glands, thymus, and gastric mucosa	3
Fed diet containing 1.0 mg/kg ration for 38 days	Adverse effects on survival	4
Immature males fed diets containing 1.0 or 3.0 mg/kg ration	All were moribund in 7-14 weeks; abnormal gastric histology	5
Adult females received a total of 9 intragastric doses 20-40 days postconception; total doses were 0.6 or 3.15 mg/kg BW	All animals in both dose groups survived, but all aborted	4
Adult females given a single intravenous injection of 0.6 mg/kg BW; blood measured 1 h to 42 days postinjection	As a percentage of the total dose administered, PCB 77 concentrations in blood fell from 4.4% at 1 h to 0.14% at 42 days. About 60% of the total dose was excreted in feces, and 10% in urine	18
PCBs 136, 153, or 155; each fed in diet at 15-65 mg/kg ration for 63-122 days	No discernable deleterious effects	4
PCB 169; fed diet containing 400.0 mg/kg ration for 40 days	Concentrations in body fat increased steadily during exposure; recovery was protracted and incomplete during a 6-month observation period	4
Mouse, <i>Mus</i> sp.		
Aroclor 1254		
>1.3 mg/kg BW daily via diet	Adverse effects on reproduction	2
Single intraperitoneal injection of 500 mg/kg BW (a dose that promoted nitrosamine-initiated lung and liver tumors). The amounts of the 9 congeners that made up >90% of the PCBs present 1 day after treatment (PCBs 99, 105, 118, 128, 138, 153, 156, 170, 180) were quantified in liver, lung, and whole body for 112 days after dosing	In carcass fat, net PCB loss was attributed to metabolic loss of PCBs 105 and 138. In lung, all congeners except 153 were retained and decreased only as a function of dilution due to growth. In liver, all congeners were retained and 105 was enriched. Total PCBs in carcass averaged 80.8 mg/kg BW 24 h after treatment and about 10 mg/kg BW after 16 weeks; similar trends occurred in liver and lung. Tb 1/2 for PCB 153 was 81-101 days	6
PCB 15; pregnant mice given daily doses of 16, 32, or 64 mg/kg BW on days 6-15 of gestation and	Toxic to dams at 64 mg/kg BW daily, but no embryotoxicity	7

Table 15. Continued.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
killed on day 18		
PCB 77		
Pregnant mice given 1, 2, 4, 8, 16, 32, or 64 mg/kg BW daily on days 6-15 of gestation and killed on day 18	Dose-dependent increase in incidence of malformed fetuses—especially cleft palate and hydronephrosis—at 4 mg/kg BW daily and higher. At 16 mg/kg BW and higher, dams had dose-dependent increase in weight loss, frequency of vaginal bleeding, and other evidence of abortion	7
Pregnant mice given single intravenous injection of 3.5 mg/kg BW of ¹⁴ C-labeled PCB 77 on days 4-17 of gestation and killed 4-96 h later	Radioactivity levels were elevated in uterine fluid and in fetuses in late gestation. No unmetabolized PCB 77 was detected in fetuses but PCB 77 metabolites (including 3,3',4,4'-tetrachloro-2-biphenylol, and methylsulphonyl-tetrachloro-biphenyl) were found in fetuses in late gestation	8
Strain C57BL/R _{ij} given single intraperitoneal injection		
15 mg/kg BW	30-40% reduction of retinol and retinyl palmitate concentrations in liver within 2-4 days	9
17 mg/kg BW	50% reduction in hepatic retinyl palmitate	9
32 mg/kg BW	50% reduction in hepatic retinol	9
Strain DBA/2; single intraperitoneal injection of 729 mg/kg BW	No reduction in hepatic retinoids	9
Single oral dose of 25, 50, or 100 mg/kg BW given to Ah-responsive pregnant mice on gestation day 11, 12, or 13; mice killed on day 18 of gestation	Dose-dependent increase in embryo deaths, resorption of the conceptus, and frequency of developmental abnormalities (cleft palate, dilated kidney pelvis, thymus hypoplasia). ED50 for cleft palate was about 100 mg PCB 77/kg BW	10
Dams received oral dose of 32 mg/kg BW daily on days 10-16 of gestation	Permanent motor dysfunction in weaning mice characterized by swift circling movements, restlessness, and hyperkinesia; spinal and cranial nerve roots abnormal	11
Ah-responsive and Ah-nonresponsive strains; females, age 10 weeks; given single intraperitoneal injection of 50 mg/kg BW; killed after 7 days	Both groups had increased body weight, increased blood EROD activity, decreased plasma retinol levels, and increased plasma total thyroid hormone levels. The Ah-responsive group also had increased hepatic pentoxyresorufin-O-deethylase activity, increased liver cytochrome P450 activity, and increased liver weight	12
PCB 80; pregnant mice given 64 mg/kg BW daily on days 6-15 of gestation and killed on day 18	No effect on maternal or developmental toxicity	7
PCB 169; pregnant mice given daily doses of 0.1, 1, 2, 4, 8, or 16 mg/kg BW by gavage on days 6-15 of gestation; mice killed on day 18 and dams evaluated for reproductive health and fetuses were examined on day 19 for malformations	All dams survived all treatments; some lost weight during pregnancy at 8 mg/kg BW and higher. Incidence of malformed fetuses increased from 0.9% in controls, to 3.6-4.3% in the 0.1-1.0 mg/kg groups to 37% in the 4 mg/kg group to 61-66% in the 8-16 mg/kg groups. Fetal deaths increased at daily doses of 4 mg/kg BW and higher, and abortions increased in the 8 and 16 mg/kg groups. Fetal liver discolored at 1 mg/kg BW daily	13
Mink, <i>Mustela vison</i>		
Aroclor 1254		
0.1 mg/kg BW daily	Adverse effects on growth	2
0.115 mg/kg BW daily (1.64 mg daily)	Adverse effects on reproduction	2, 16
PCB 47; subadult females given daily intraperitoneal injections of 50 mg/kg BW for 3 days and killed 7 days after last dose	No evidence of illness or pathology. PCB 47 residues, in mg/kg FW, were 389 in fat and 38 in liver	14, 15
PCB 77; subadult females given daily intraperitoneal injections of 50 mg/kg BW for 3 days and killed 7 days after last dose	Severe anorexia, diarrhea, and melena. Significant histopathology of mucosa of the small intestine. PCB residues, in mg/kg FW, were 139 in fat and 16 in liver	14, 15

Table 15. Continued.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
PCBs 136 and 167; each fed to adult females in the diet at 5 mg/kg ration for 3 months	No adverse effects on reproduction	16
PCB 169; fed in diet at concentrations of 0.01-0.5 mg/kg ration for 135 days		
0.01 mg/kg diet	No deaths; some weight loss and liver enlargement	17
0.05 mg/kg diet	50% mortality in 135 days	17
0.1 mg/kg diet	50% dead in 75 days	17
0.5 mg/kg diet	All dead within 73 days	17
Laboratory white rat, <i>Rattus</i> sp.		
Aroclor 1254		
Dams and resultant pups fed diets containing 0, 3, 30, or 300 mg Aroclor 1254/kg ration from conception to weaning	Most congeners that accumulated in pup tissues were concentrated in the dam's milk when compared to the feed. PCB uptake by pups was greater during lactation than during gestation. Congeners that accounted for 46% of the brain PCB content and also accumulated in a dose-dependent manner were PCBs 105, 138, 141, 153, 168, 178, 179, and 186	19
0.25 mg/kg BW daily via the diet	Adverse effects on reproduction	2
PCB 4; young males given intraperitoneal injections of 50 mg/kg BW daily for 3 days and killed 4 days after last injection	Negligible morphological effects on liver when compared to PCBs 15, 52, and 77 groups dosed at same regimens	20
PCB 28; dams dosed on days 10 to 16 of gestation by gavage with 8 or 32 mg/kg BW daily; offspring tested in mazes at age 12 to 16 weeks	Birth weight lower in high dose group; female pups—but not males—in the high dose group had learning deficits	27
PCB 47; subadult females given 3 daily injections of 50 mg/kg BW and killed 7 days after last injection	No clinical signs of illness and no significant gross or microscopic lesions. PCB 47 residues, in mg/kg FW, were 747 in fat and 28 in liver	14, 15
PCB 77		
Young adults given single intravenous injection of 0.6 mg/kg BW. Blood and tissue concentrations analyzed 0.5 h-7 days postinjection	In males (females), adipose tissue 4 h postinjection contained 23% (12%) of the total dose administered, skin 14% (16%), liver 6.8% (8.8%), muscle 6.7% (16.2%), and blood 4.1% (4.0%); after 7 days, adipose tissue contained 2.7% (7.4%) and other tissues 0.5-2.2% (1.0-3.4%)	18
Pregnant rats dosed orally on days 6-18 of gestation with 1, 3, or 10 mg/kg BW daily; fetuses were examined on day 19 for developmental abnormalities	A dose-dependent increase in mortality, intestinal histopathology, and external malformations, and decrease in fetal size and length of tibias. A daily dose of 3 mg/kg BW significantly affected fetus growth, bone development, and survival	21
Single intraperitoneal injection of 15 mg/kg BW	Significant reduction in liver and heart retinol and in liver retinylester concentrations	22
Single intraperitoneal injection of 50 mg/kg BW	Metabolites in feces included 5-hydroxy-3,4,3',4'-tetra-chlorobiphenyl, 4-hydroxy-3,5,3',4'-tetra-chlorobiphenyl, and a dihydroxy- and monohydroxy trichlorophenyl. After 5 days, unchanged PCB 77 in feces accounted for 0.8% of the initial dose, but hydroxylated metabolites constituted about 32%	23
Subadult females given 3 daily intraperitoneal injections of 50 mg/kg BW and killed 7 days after last injection	No signs of illness or histopathology. PCB 77 residues, in mg/kg FW, were 148 in fat and 138 in liver	14, 15
Adults injected intra-peritoneally for 3 days at 80 mg/kg BW daily then killed 24 h after last injection	Heme destruction in the P448-containing reconstituted monooxygenase system; reactive epoxide, or possibly nonepoxide, intermediate metabolites may participate in cytochrome P448 destruction	24
PCB 118; dams dosed on days 10 to 16 of gestation by gavage with 4 or 16 mg/kg BW daily; offspring tested in mazes at age 12-16 weeks	Birth weight lower in high-dose group; female offspring, but not males, in high-dose group had learning deficits	27

Table 15. Continued.

Species, PCB congener, dose, and other variables	Effect	Reference ^a
PCB 126; dams given 0.01 or 0.02 mg/kg BW by gavage every second day from days 9-19 of gestation	Reduction in litter size, body weight, and survival of sucklings; delayed spontaneous movement and neuromuscular maturation. Dams and pups had reduced body weight, and increased cytochrome P4501A1 activity	25
PCB 153		
Dams dosed on days 10 to 16 by gavage with 16 or 64 mg/kg BW daily; offspring tested on mazes at age 12 to 16 weeks	Female offspring from high-dose group had learning deficits; males were not affected	27
Immature female pups given 8, 11, 25, 51, or 59 mg/kg BW on days 20 and 21; killed on day 22	Increased uterine weight at 25 and 51 mg/kg BW, but not in other groups	26

^a 1, van den Berg et al. 1988; 2, Golub et al. 1991; 3, McNulty et al. 1980; 4, McNulty 1985; 5, Becker and McNulty 1984; 6, Anderson et al. 1993; 7, Marks et al. 1989; 8, Darnerud et al. 1986; 9, Brouwer et al. 1985; 10, d'Argy et al. 1987; 11, Chou et al. 1979; 12, Murk et al. 1991; 13, Marks et al. 1981; 14, Gillette et al. 1987a; 15, Gillette et al. 1987b; 16, Kihlstrom et al. 1992; 17, Aulerich et al. 1987; 18, Abdel-Hamid et al. 1981; 19, Shain et al. 1986; 20, Hansell and Ecobichon 1974; 21, Wardell et al. 1982; 22, Brouwer et al. 1988; 23, Yoshimura et al. 1987; 24, Shimada and Sawabe 1983; 25, Bernhoft et al. 1994; 26, Li et al. 1994; 27, Schantz et al. 1995; 28, Mes et al. 1995.

and 167 (Table 15). Although subhuman primates seem to be more sensitive to reproductive and other adverse effects of PCBs than humans, no clear and convincing evidence associated PCB exposures with human cancers and reproductive problems (Kimbrough 1995). At present, no meaningful reproductive problems have been identified in female capacitor workers and no carcinogenicity was evident in humans having more than 1.0 mg total PCBs/L serum or more than 400 mg total PCBs/kg adipose fat (Kimbrough 1995).

Mink (*Mustela vison*) is among the most sensitive mammals to PCB toxicity (Aulerich et al. 1987; Edqvist et al. 1992; Table 15). Reproductive failure, especially fetal death and resorption, of PCB-fed mink is well documented (Backlin and Bergman 1992; Bergman et al. 1992b; Edqvist et al. 1992; Madej et al. 1992). The mechanisms of intra-uterine death of mink fetuses after PCB exposure are not fully understood (Backlin and Bergman 1992), although planar PCB congeners seem to be implicated (Hakansson et al. 1992). Studies showed that EROD and AHH activities were maximally induced in adult mink by PCB fractions containing non-*ortho* or mono-*ortho* chlorobiphenyls and that mink kits are about 10 times more sensitive to P450-inducers than adults (Brunstrom 1992; Kihlstrom et al. 1992). PCB-dosed mink also show altered blood chemistry (Edqvist et al. 1992), abnormal liver metabolism and histology (Edqvist et al. 1992; Bergman et al. 1992b), raised cortisol excretion (Madej et al.

1992), enhanced EROD activity (Brunstrom 1992), and altered metabolism of Vitamin A (Hakansson et al. 1992). Selective retention of certain PCBs and their hydroxylated metabolites occurred in mink muscle after 3 months of a diet containing a PCB mixture (Bergman et al. 1992a). Retention of PCBs 99, 105, 118, 138, 153, 156, and 180 was high in muscle of mink on the diet. Not retained and presumably metabolized were PCBs 44, 49, 52, 91, 92, 95, 97, 107, 132, 149, and 174 (Bergman et al. 1992a).

Estrogen receptors bind to many compounds other than natural estrogen. Several organochlorine compounds, including certain PCBs, reportedly act as estrogen mimics (Hileman 1994). Many estrogen mimics are persistent, lipid soluble compounds that are defined by their ability to stimulate the proliferation of cells in the uterus of the mouse (*Mus* spp.); in males, these mimics may also inhibit sperm production and testes growth. PCBs and their metabolites may be estrogenic to wildlife, although the evidence is not conclusive (Hileman 1994). PCB mixtures and pure individual chlorobiphenyls with a significant degree of *ortho*-substitution have elevated estrogenic activity; Aroclor 1221, for example, rich in *ortho*-chlorobiphenyl, has estrogenic activity in female rats (Korach et al. 1988). Hydroxylated metabolites of PCBs also show estrogenic hormonal activity (Korach et al. 1988; Li et al. 1994). Hydroxylation of PCBs occurs in amphibians and teleosts but at a much slower rate than that of mammals (Safe et

al. 1976). Hydroxylated metabolites of PCB 3 include 4'-chloro-4-biphenylol, 4'-chloro-3,4-biphenyldiol, and 4'-chlor-3-methoxy-4-biphenylol; PCB 15 gave 4,4'-dichloro-3-biphenylol; and Aroclor 1254 yielded mono-, di-, and tri-chlorophenylols (Safe et al. 1976). PCB 77 is detoxified by metabolic hydroxylation to hydroxy biphenyl metabolites (Borlakoglu et al. 1991a). Hydroxylated PCB metabolites may be more toxic than the parent product because of their (1) estrogenic properties (Yoshimura et al. 1987); (2) tendency to accumulate in the fetus (Darnerud et al. 1986), and (3) interference with thyroxine metabolism (Brouwer et al. 1990). The ability of hydroxylated PCB metabolites to bind to the uterine estrogen receptor in rats was in increasing order of effectiveness 4-hydroxy 3,5,4'-trichlorobiphenyl, 4,4'-dihydroxy 3,5,3',5'-tetrachlorobiphenyl, 4-hydroxy 2-chlorobiphenyl, 4-hydroxy 4'-chlorobiphenyl, 4,4'-dihydroxy 2',3',5',6'-tetrachlorobiphenyl, 4,4'-dihydroxybiphenyl, and 4-hydroxybiphenyl; PCB compounds that demonstrated appreciable receptor binding activity were also active in stimulating uterine weight increases (Korach et al. 1988). More research seems needed on the estrogenic properties of hydroxylated PCB metabolites.

Long-term neurobehavioral changes were reported in children, monkeys, and rodents exposed to commercial PCB mixtures during fetal and neonatal development (Schantz et al. 1995). Perinatal exposure of rats (*Rattus* sp.) to *ortho*-substituted PCBs (PCBs 28, 118, 153) can cause long-lasting deficits in learning in females; males were not affected (Table 15).

In comparison to primates and mustelids, rodents are only moderately sensitive to intoxication by most PCBs (Abdel-Hamid et al. 1981). In rodents, PCBs 77 and 169 cause effects that are characteristic of the dioxins and furans, including P450 induction, porphyrin accumulations, and atrophy of the lymphatic organs (Abdel-Hamid et al. 1981), as well as teratogenicity in mice (Darnerud et al. 1986). Exposure of rats to PCB 126 in utero and through lactation produced fetotoxic effects, delayed physical maturation, and induced liver xenobiotic metabolizing enzymes without causing neurobehavioral effects (Bernhoft et al. 1994). In mice, PCBs 77, 126, and 169 were teratogenic in a high percentage of the fetuses from treated dams but without apparent effect on the dams (Marks et al. 1981). PCB 77, for example, was toxic to the conceptus at dose levels below those toxic to the dam when administered to pregnant CD-1 mice on

days 6-15 of gestation (Marks et al. 1989). The predominant PCB-induced fetal malformations in mice were cleft palate and hydronephrosis (Marks et al. 1981). PCB 77 also interferes with retinyl ester hydrolase (REH), the enzyme responsible for the hydrolysis of vitamin A into free retinol, lowering levels of vitamin A in liver and decreasing serum concentrations of REH and retinol (Mercier et al. 1990).

Toxicokinetics of PCBs in rodents were altered when administered in mixtures (de Jongh et al. 1992). PCBs 153, 156, and 169 produced biphasic elimination patterns in mice when administered in combinations but single phase elimination when administered alone; elimination of all PCBs was more rapid after coadministration. Mixtures of PCBs 153 and 156 raised EROD activity and lengthened retention of each congener in liver; however, a mixture of PCBs 153 and 169 lowered EROD activity (de Jongh et al. 1992). Selected PCBs of low acute toxicity may increase the toxicity of compounds such as 2,3,7,8-TCDD (Birnbaum et al. 1985). Thus, PCBs 153 or 157 at sublethal dosages (20-80 mg/kg BW) did not produce cleft palate deformities in mouse embryos. But a mixture of PCB 157 and 2,3,7,8-TCDD produced a 10-fold increase in the incidence of palate deformities that were expected of 2,3,7,8-TCDD alone; palate deformities did not increase with a mixture of PCB 153 and 2,3,7,8-TCDD. The widespread environmental occurrence of PCB-PCDD and PCB-PCDF combinations suggests a need for further evaluation of the mechanism of this interaction (Birnbaum et al. 1985).

Intraspecific variability to PCBs was high, especially between genetically inbred AHH-responsive and AHH-nonresponsive strains of mice (Robertson et al. 1984). In mice, PCB 77 was metabolized and excreted more rapidly than 2,3,7,8-TCDD (d'Argy et al. 1987); in rats, PCB 77 was excreted more rapidly than PCB 47 (Shimada and Sawabe 1984). PCBs that lack adjacent hydrogen atoms in at least one of the rings are enriched in rat tissues, indicating that accumulation exceeds elimination by metabolism and excretion. PCBs with a tendency to accumulate were non-*ortho* and mono-*ortho* substituted congeners; however, PCBs with *meta-para* unsubstituted carbon atoms in at least one ring were not enriched in tissues (Borlakoglu et al. 1991a). Uptake and retention of individual PCB congeners in rats are related to properties associated with K_{ow} and high chlorination, especially in the tetra- and penta-chlorobiphenyls

(Shain et al. 1986). The highly chlorinated hexa- and octa-chlorobiphenyls produced morphological changes in rats comparable to those produced by DDT, Aroclor 1254, and Aroclor 1260 (Hansell and Ecobichon 1974).

Recommendations

Proposed PCB criteria for the protection of natural resources are predicated on total PCBs and selected Aroclor compounds (Table 16) and offer minimal insight into PCB toxicokinetics. Most authorities now agree that future PCB risk assessments require (1) analysis of non-*ortho* PCBs and selected mono-*ortho* PCBs; (2) exposure studies of individual species to specific congeners alone or in combination with other compounds, including other PCB congeners, dioxins, and dibenzofurans; (3) clarification of existing structure-induction relations, and (4) more refined analytical techniques (Eisler 1986; Maack and Sonzogni 1988; Tanabe 1988; Gooch et al. 1989; Hernandez et al. 1989; Kannan et al. 1989; Tanabe et al. 1989; Borlakoglu et al. 1990; Hebert et al. 1993; Giesy et al. 1994a; Safe 1994; Walker and Peterson 1994). Marked differences between species in their abilities to metabolize specific PCB congeners must be considered in toxicity testing. Foxes and dogs, for example, in contrast to monkeys and rats, can degrade the otherwise highly persistent PCB 153 because they possess an unusual cytochrome P450 isoenzyme that metabolizes PCB 153 (Georgii et al. 1994). Also, low-chlorinated congeners that are metabolized via reactive intermediates must be critically evaluated because they show weak tumor-initiating properties (Georgii et al. 1994).

To complement PCB chemical residue analyses, the rat hepatoma cell bioassay was useful for assessing the toxic potency of PCBs in extracts from environmental samples (Tillitt et al. 1991). This *in vitro* bioassay of cytochrome P450IA1 catalytic activity in the H4IIE cells in response to planar halogenated hydrocarbons was considered accurate and precise. Comparison of the responses of the H4IIE cells was calibrated against their responses to 2,3,7,8-TCDD (Tillitt et al. 1991). EROD (ethoxyresorufin-*O*-deethylase) and porphyria induction measurements also potentially complement PCB chemical residue analyses and have been used to determine the toxic potencies of complex mixtures of PCBs and other halogenated aromatic hydrocarbons extracted from wildlife tis-

sues (Kennedy et al. 1992). In one case, extracts of PCB-contaminated eggs of herring gulls (*Larus argentatus*) from the Great Lakes and of great blue herons (*Ardea herodias*) from British Columbia induced EROD and porphyria in primary cultures of chicken embryo hepatocytes (Kennedy et al. 1992). Hepatic cytochrome P450-associated monooxygenases and cytochrome P450 proteins in embryos of the black-crowned night-heron (*Nycticorax nycticorax*) were associated with concentrations of total PCBs and 11 PCB congeners that express toxicity through the Ah receptor, and also should be considered as biomarkers for assessing PCB contamination of wetlands (Rattner et al. 1994).

The interpretation of PCB residue data is challenging from several perspectives, as judged by analysis of eggs of Forster's terns (*Sterna forsteri*) from Wisconsin (Schwartz and Stalling 1991): (1) data from a single analysis frequently contained measurable concentrations of 100 to 150 PCB congeners; (2) a single sample was not sufficient to understand the environmental distribution of PCBs; (3) source profiles of PCB inputs into the environment were poorly characterized; (4) PCB congeners in the original polluting material often merged with congeners from other sources; and (5) the contaminant mixture may have been altered by metabolism and subsequent partition into multiple environmental compartments that may be further changed by weathering or degradation. To understand these processes and to correlate residue profiles with specific toxic responses required congener-specific methods of analysis and complex statistical techniques (principal component analysis). Using these techniques, it was established that eggs of Forster's terns of two colonies differed significantly in PCB composition (Schwartz and Stalling 1991). Similar techniques were used to identify various PCB-contaminated populations of harbor seals (*Pusa vitulina*) in Denmark (Storr-Hansen and Spliid 1993).

Selected congeners should be quantified in human foodstuffs and tissues, as determined from a survey of PCB congener frequency in commercial formulations, environmental and biological samples and human tissues, and a consideration of the relative toxicity and persistence of the congeners (Jones 1988). PCBs 28, 74, 77, 99, 105, 118, 126, 128, 138, 153, 156, 169, 170, 179, and 180 reportedly account for more than 70% of the total PCB burden in any sample and should be quantified. Additionally, PCBs 8, 37, 44, 49, 52, 60, 66, 70, 82, 87, 101, 114, 158, 166, 183, 187, and 189 should be

Table 16. Proposed PCB criteria for the protection of natural resources and human health.

Resource, criterion, and other variables	Effective concentration	Reference ^a
Aquatic life protection, total PCBs		
Fish		
Diet	<500 µg/kg fresh weight (FW)	1
Eggs	<300 µg/kg FW	1
Whole body	<400 µg/kg FW	1
Marine mammals, blubber	<70 mg/kg FW	2
Medium		
Freshwater		
Acute	<2.0 µg/L	6
Chronic	<0.014 µg/L	1, 6
Saltwater		
Acute, most species	<10.0 µg/L	6
Chronic, sensitive species	<0.03 µg/L	6
Filter-feeding shellfish	<0.006 µg/L	1
Birds, total PCBs		
Brain	<300 mg/kg FW	3
Mammals		
Aroclor 1016		
Rhesus macaque	<0.008- <0.028 mg/kg body weight (BW) daily	4
Aroclor 1248		
Rhesus macaque	<0.009 mg/kg BW daily	4
Aroclor 1254		
Mink	<0.1- <0.115 mg/kg BW daily	4
Mouse	<1.3 mg/kg BW daily	4
Rat	<0.25 mg/kg BW daily	4
Human health protection, total PCBs (unless indicated otherwise)		
Air		
Aroclor 1242	<1.0 mg/m ³	6
Aroclor 1254	<0.5 mg/m ³	6
Reduced risk from cancer	<7.7 mg/kg BW daily	6
Drinking water		
Child	<1.0 µg/L	6
Adult	<4 µg/L	6
Fish, edible portion		
Canada	<2 mg/kg FW	5
United States	<2 mg/kg FW	6
New York State	<2 µg/kg FW	5

^a1, Eisler 1986; 2, Norheim et al. 1992; 3, Bryan et al. 1987a; 4, Golub et al. 1991; 5, Hebert et al. 1993; 6, USEPA 1992b.

considered for quantification because of their reported occurrence or toxicity. Some PCBs are particularly prevalent in aquatic animals, especially PCBs 95, 101, 110, 118, 138, 149, 153, 180, and 187; also detected in aquatic biota and reported as important components were PCBs 26, 52, 66, 70, 99, 105, 132, 151, 170, 177, 201, and 206.

In the Netherlands, maximum PCB limits in fishes as dietary items for human health protection are now derived from the sum of PCBs 28, 95, 101, 138, 149, 153, and 180. From a toxicological viewpoint, other congeners may be more important. These have been identified (on the basis of ability to induce AHH) as the most toxic planars

(PCBs 15, 37, 77, 81, 126, and 169), the mono-*ortho* analogues of the planar PCBs (PCBs 105, 114, 123, 156, and 189), and the di-*ortho* analogues (PCBs 128, 138, 158, 166, and 170). Of these compounds, PCBs 37, 105, 114, 128, 138, 156, and 158 occur in human tissues and PCBs 15, 77, 81, 123, 126, 166, and 169 do not occur (Jones 1988).

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