IV. ENVIRONMENTAL FATE AND EFFECTS

IV.1. Persistence, Metabolism and Fate

IV.1.1. Persistence

PCBs have a long life in the environment (1-3). The more chlorinated compounds in particular are resistant to metabolism (Appendix E) and persist in soils (305), water (41, 42); sediments (50, 99, 105) and biological tissues (111, 130, 141, 155, 170, 208, 35) for weeks, months or even years. Indirect evidence for their persistence in the environment is provided by their wide distribution, even in remote parts of the earth where PCBs are unlikely to have been used (1-3, 56, 225, Appendix C).

Precise data on the life-time of PCBs after release into the environment are difficult to obtain, because natural systems are open: if a decline in concentrations is observed it is difficult to distinguish degradation from net export. Hom et al. (329) provided almost the only unequivocal data: they found PCBs in dated (varved) sediments in the Santa Barbara basin off southern California. PCBs were first detectable in sediment layers dated to the mid-1940's and their concentration in the overlying sediments increased steadily in parallel with the known increase in PCB usage. This shows that PCBs can last for at least 30 years in these circumstances (anaerobic sediments with no burrowing organisms). More recently Risebrough et al. (56) have identified PCBs in Antarctic snow at depths up to 6 metres below the surface.

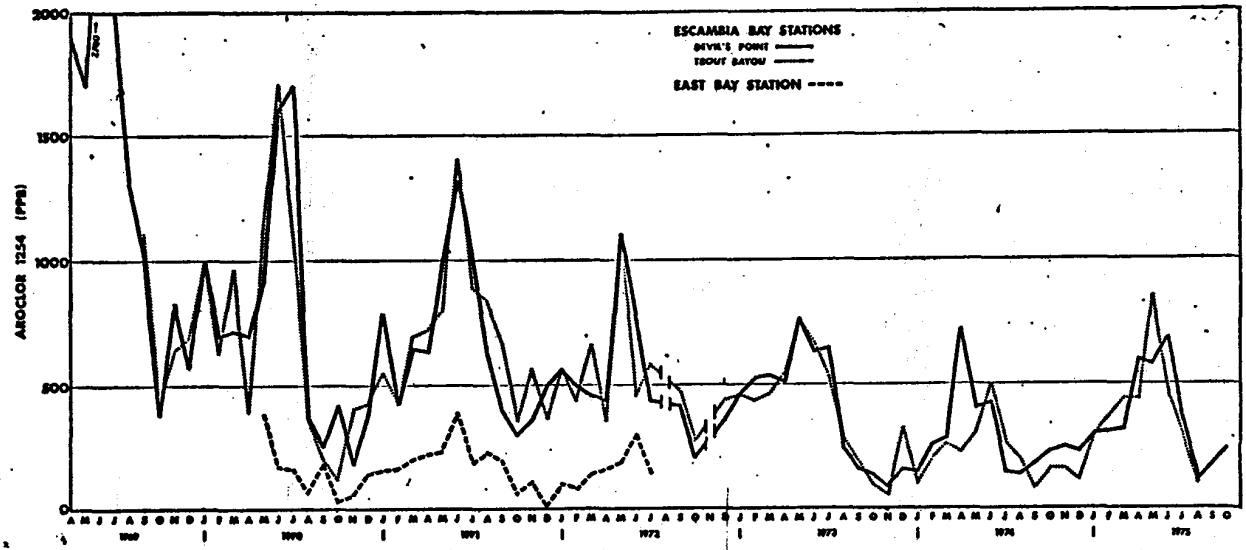
Figure IV.1.1 shows concentrations of PCBs measured in oysters in Escambia Bay, Florida, since 1969 (330). PCBs in Escambia Bay are believed to have originated largely from a single point source, identified and closed in 1969 (99, 330). The residues in oysters in the bay show seasonal fluctuations (related to spawning) but otherwise have declined only slowly over the years since 1969.

All these data indicate that the more chlorinated PCBs have a life-time of years, if not decades, in at least certain compartments of the environment (2, 331).

IV.1.2. Differential Persistence of Lower and Higher Chlorinated Biphenyls.

Nisbet and Sarofim (332, 2) pointed out an anomaly, viz., that most PCBs found in environmental samples consisted of penta- and higher CBs, whereas a substantial fraction of the PCBs released into the environment in the past must have consisted of tetra- and lower CBs. They suggested that the discrepancy was probably due to differential degradation of the lower CBs, ruling out the main alternative explanation (differential mobility) primarily because samples of PCBs from remote areas also consisted primarily of higher PCBs. A critical set of observations was that of Veith and Lee (333, 334), who showed from samples of water and fish from the Milwaukee River that the proportion of lower CBs declined as the water and sediments moved downstream away from the major industrial sources. However, this decline could be explained either as differential volatilization or differential degradation (333).

RESIDUES OF AROCLOR 1254 IN OYSTERS



Attachment 1

Figure IV,1.1 (ref. 330)

The differential persistence and/or bioaccumulation of lower and higher CBs is important in establishing criteria, because it implies differential exposure of human and other target organisms to the various components of the various commercial mixtures. Accordingly the following sections of this document will pay particular attention to differences in environmental behavior between lower and higher CBs. The potential for human exposure to tetra- and lower CBs is of especial importance, because the main difference between Aroclors 1016 and 1242 is the lower proportion of the more persistent penta-CBs in the former (Table II.3.1). In 1972 it was believed that most human exposure was to penta- and higher CBs, but recently more evidence for human exposure to tetra-CBs has been presented (331, 332).

IV.1.3. Metabolism

Studies of the metabolic transformation and degradation of PCBs are summarized in Appendix E. Bacteria are able to metabolize biphenyl and mono- and di-CBs fairly rapidly, but tri- and tetra-CBs are degraded more slowly and penta-CBs hardly at all (Section E.1). Accordingly there is considerable differentiation of PCB mixtures during microbial degradation, as illustrated in Figures E.4 to E.7. Figure IV.1.2 (from ref. 13) shows that even 14 days' incubation with activated sludge had little effect on the higher components (mostly tetra-CBs) in Aroclor 1016.

Aquatic invertebrates and fish are able to metabolize tri-CBs to a substantial degree but have very little ability to metabolize tetra- or penta-CBs to non-polar products (Sections E.2 and E.3).

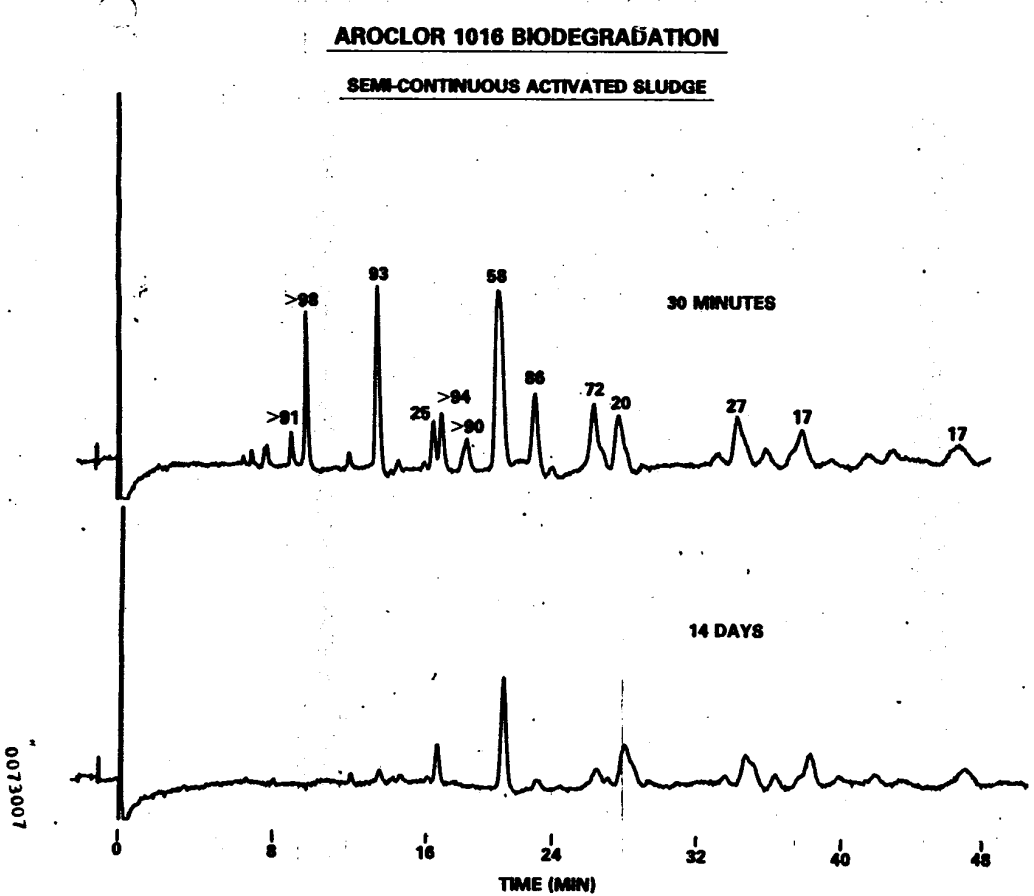


Figure IV.1.2

Birds and mammals can metabolize tetra- and penta-CBs to hydroxy derivatives at varying speeds, but they have very little ability to metabolize hexa- or higher CBs (Sections E.4 and E.5).

The principal mechanism of metabolism appears to be via oxidation to arene oxide (epoxide) intermediates, followed by rearrangement ('NIH shift') to form hydroxy-CBs. Hydroxylation at the 3- or 4-positions is favored (Section E.6). Less important mechanisms of metabolism are direct hydroxylation, dechlorination, and isomerization. There is evidence for metabolic formation of CDFs in chickens and rats (Section E.7). Unidentified non-polar metabolites are stored in invertebrates and fish (Section E.3).

Metabolism does not always represent detoxification, since CDFs are much more toxic and at least one hydroxy-CB is substantially more toxic than the parent compound. The formation of arene oxide intermediates is of much concern because this reactive class of compounds is implicated as causative agents in toxic, carcinogenic, and mutagenic effects (Section E.8).

IV.1.4. Photodegradation

The photochemical properties of PCBs were summarized in Section II.8. Photolysis of PCBs has been studied only under laboratory conditions and it is difficult to use the data to predict rates of degradation in the environment. In the experiment which most nearly simulated environmental conditions (61), there was little overall degradation after 3 weeks in sunlight and the principal net effect was a shift from higher to lower CBs in the mixture (Table II.8.3).

Photodegradation does not necessarily represent detoxification, since lower CBs are formed by dechlorination and small quantities of CDFs are sometimes formed (Sections II.8.2, II.8.3).

IV.1.5. Transport

PCBs are mobile in the environment and may be transported in solution, by motion of suspended sediments, as vapors, on airborne particulates, or in the tissues of mobile animals (2, 12, 332, 333, 47, 50, 52, 53, 54, 56, etc.). They are concentrated at the air/water interface (52, 55) and may be transported across it by several mechanisms, including volatilization, solution, dry fallout of particulates, precipitation in rain and snow, or ejection in spray (Section II.8). Critical measurements are scanty and conflicting (56) and it is still not possible to construct satisfactory models of transport to and from the aquatic environment (56, 331).

Within bodies of water, there is evidence that the transport of PCBs is controlled by the presence and transport of sediments (Section II.6.5). The presence of sediments or particulates inhibits volatilization of PCBs (41, 42) and is strongly associated with their transport (50, 56, 12). In natural waters with high sediment loads it may be reasonable to treat PCBs as passively transported on particulates, but it is not known whether this would be valid for the ocean or for clear lakes. However, it is questionable whether conditions in aquaria without sediments provide good models for natural environments.

IV.1.6. Fate

Theoretically, there are three ultimate fates for PCBs released into the environment: metabolic degradation, photolytic degradation, and deposition in sediments in lakes or the deep ocean (2). All three processes are known to occur, but their relative importance in the natural environment remains to be determined (2, 56, 331).

IV.2. Bio-accumulation and Bio-magnification

IV.2.1. Mechanisms of uptake and accumulation

One of the most important environmental properties of PCBs is their tendency to be "bio-accumulated" by aquatic organisms -- i.e., to be concentrated into their tissues to levels much higher than those in the ambient water (1-3). This property results from the high solubility of PCBs in lipids and their low solubility in water: Metcalf et al. (45) have shown that the degree of bio-accumulation of a chemical in aquatic animals is closely related to its partition coefficient between organic solvents and water (Figure II. 6.6). There is a further tendency for PCBs to be concentrated into the tissues of animals to levels higher than those in their food (1-3): this phenomenon is sometimes referred to as "bio-magnification". Within organisms, PCBs are further concentrated into certain organs, especially the fat (1-3).

The kinetics of organochlorine compounds in animals have recently been reviewed by Moriarty (335). It is necessary to consider bio-accumulation as a multi-stage process: the chemicals are taken into the organism via food, water, or air, circulated through

the organism in the blood, transferred into and out of various organs, and finally metabolized and/or excreted. Each stage of transfer is a dynamic process whose rate depends on the concentrations of the chemicals in the various media and body organs. Under conditions of constant exposure, an organism may eventually reach a quasi-equilibrium, in which the concentrations of the chemical in the tissues are constant. It is then possible to define a storage factor (ppm in organism/ppm in ambient medium). In principle, storage factors should be defined separately for each organ in the body, but in practice it is common to define a single "bio-accumulation factor" (average ppm in whole body/ppm in ambient water). However, where the time required to reach equilibrium is long, as it often is for PCBs, the organism's physiological state may change and a true equilibrium may never be reached (335).

Aquatic plants take up PCBs directly from water, and their bio-accumulation factors reflect partitioning across the cell membranes. Animals may be exposed to PCBs in food, in air, or in water. Aquatic invertebrates and fish have to process so much water in order to breathe that they usually reach quasi-equilibrium with PCB concentrations in water fairly quickly; intake via food is usually only a minor route of intake (336). Accordingly the storage factor (ppm in whole body/ppm in ambient water) is the most useful measure of bio-accumulation. However, for air-breathing animals such as birds and mammals the food is usually the most important route of intake and the storage factor (ppm in whole body/ppm in food) is

the most useful measure: this factor is sometimes referred to as the "bio-magnification factor" (336).

Laboratory experiments on bio-accumulation and bio-magnification are usually highly simplified, involving constant exposure via one route only. The situation in the real world is more complex, since animals are exposed simultaneously via several routes, levels of exposure fluctuate greatly in time and space, and the animals' physiological state changes in response to aging and periodic environmental stresses. Accordingly, while laboratory experiments provide useful information on mechanisms of uptake and excretion, and comparative data for the various components of PCBs, they do not necessarily provide quantitative measures of the degree of bio-accumulation and bio-magnification to be expected in the field. Measurements on wild animals in the field are needed to gauge the extent of bio-accumulation and bio-magnification, and to specify their variability in time and space.

IV.2.2. Bio-accumulation in Aquatic Invertebrates

In laboratory experiments, Sanders and Chandler (98) found that uptake and bio-accumulation of Aroc lor 1254 by some aquatic invertebrates was very rapid. Bio-accumulation factors for various organisms ranged from 2,800 in stone-flies to at least 47,000 in Daphnia magna (Table IV.2.1). In scud (Gammarus psaudolimnaeus), the tri- and tetra-CBs in Aroclor 1254 were differentially accumulated by factors 2-8 times higher than the hexa- and hepta-CBs (Table IV.2.2). Nebeker and Puglisi (93) found bio-magnification factors of 16,000-36,000 for

Table IV.2.1
(from ref. 98)

Biological magnification of ^{36}Cl-labeled Aroclor® 1254 by aquatic invertebrates

Organism	Organisms per sample 1/	Water concentration ppb $\bar{x}$ ± SE 2/	Organism concentration (4 day exposure) ppm $\bar{x}$ ± SE	Magnification factor 3/ 1-day	4-day	7-day	14-day	21-day
Daphnid *Daphnia magna*	60	1.1 ± 0.2	52 ± 2.0	24,700	47,000	-	-	-
Phantom midge *Chaoborus punctipennis*	5	1.3 ± 0.1	30 ± 1.6	22,000	23,000	23,800	24,800	-
Scud *Gammarus pseudolimnaeus*	6	1.6 ± 0.1	39 ± 3.0	17,000	24,000	26,000	27,500	27,000
Mosquito larvae *Culex tarsalis*	10	1.5 ± 0.3	27 ± 2.0	12,600	18,000	20,000	-	-
Glass shrimp *Palaemonetes kadiakensis*	3	1.3 ± 0.1	16 ± 2.6	10,300	12,300	13,700	14,200	16,600
Stonefly *Pteronarcys dorsata*	3	2.8 ± 0.8	7.0 ± 0.30	2,100	2,500	2,800	2,900	2,800
Dobsonfly *Corydalus cornutus*	3	1.1 ± 0.1	5.1 ± 0.23	1,400	4,600	5,700	6,600	6,800
Crayfish *Orconectes nais*	2	1.2 ± 0.1	0.2 ± 0.20	570	1,700	3,400	4,500	5,100

1/ Samples were taken in triplicate.
2/ Samples were taken in triplicate and expressed as mean value ± standard error (P=.05).
3/ Concentration in organism/concentration in water.

Table IV.2.2
(from ref. 98)

Changes in isomer ratios of Aroclor® 1254 residues in scud

Peak No.	Rel. Ret. Time[1]	No. Cl's[2]	Concentration Factor[3] Sample 1	Sample 2	Average	Aroclor® 1254 R.P.A.
1	0.27	3	2.00	2.00	2.00	0.03
2	0.37	4	1.69	1.72	1.71	0.29
3	0.45	4	1.81	1.81	1.81	0.11
4	0.50	4	1.80	2.00	1.90	0.05
5	0.63	5	1.31	1.31	1.31	0.51
6	0.77	5	1.00	1.00	1.00	1.00
7	0.98	5	1.13	1.16	1.15	0.38
8	1.07	5	1.07	1.16	1.12	0.57
9	1.19	6	0.33	0.66	0.49	0.03
10	1.31	6	0.84	0.85	0.85	0.65
11	1.59	6	0.55	0.55	0.55	0.33
12	1.67	6	0.80	0.80	0.80	0.30
13	1.79	7	0.43	0.57	0.50	0.07
14	2.04	6	0.56	0.60	0.58	0.45
15	2.61	6	0.50	0.62	0.56	0.08
16	3.22	7	0.25	0.50	0.37	0.04
17	3.68	7	0.25	0.25	0.25	0.04

[1] Retention time relative to p,p' DDE on GLC column of 2 mm i.d. x 1.8 m 0.3% w/w OV-7 on 80-100 mesh Corning® 110 glass beads; 15 ml/min. flow of nitrogen carrier gas; column operated at 155° C.

[2] Number of chlorine atoms substituted on biphenyl ring determined by gas chromatography-mass spectrometry (9).

[3] Concentration factor = Relative Peak area of sample ÷ Relative Peak areas as Aroclor® 1254.

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the same species of scud when exposed to Aroclor 1242, and of 28,000-108,000 when exposed to Aroclor 1248. In estuarine shrimps, bio-accumulation factors ranged up to 26,000 during chronic exposure to Aroclor 1254 (104: Table IV.2.3). In short-term tests, shrimp accumulated Aroclor 1016 to levels 2-4,000 times higher than those in the ambient water within 96 hours (100). Bio-accumulation factors reported in oysters ranged up to 101,000 (108) and to 165,000 (337).

In a comparative study in a model ecosystem, Metcalf et al. (45) found higher bio-accumulation factors for representative tetra- and penta-CBs than for a representative tri-CB (Tables E.4 and E.5). In snails the bio-accumulation factors (E.M. in Table E.5) recorded after 33 days were 5,800 for the tri-CB, 39,400 for the tetra-CB, and 59,600 for the penta-CB. In mosquito larvae the corresponding factors were 815, 10,600, and 17,300, but these represented only 6 days' exposure. It should be noted, however, that some of the unidentified non-polar metabolites were bio-accumulated even more than the parent CBs (Table E.4). "Unknown I" from the tri-CB, for example, was bio-accumulated 12,000 times in the snail and 8,000 times in the mosquito.

IV.2.3. Bio-accumulation in Fish

In laboratory experiments in flowing sea water, juvenile pinfish (Lagodon rhomboides) and spot (Leiostomus xantherus), both accumulated increasing amounts of Aroclor 1254, up to quasi-equilibrium levels, as duration of exposure increased (111). Spot exposed to 1 ppb Aroclor 1254 for 56 days reached maximum residue levels of

Table IV.2.3

ACCUMULATION OF AROCLOR 1254 IN Palaemonetes pugio WITH TIME

AFTER EXPOSURES TO THE CHEMICAL IN WATER AT THREE CONCENTRATIONS (µg/1)
(Each value represents a composite sample of 10 animals)

Length of Exposure (hr/ days)	Control		0.04		0.09		0.62	
	Body Conc. (mg/kg)	Conc. Factor	Body Conc. (mg/kg)	Conc. Factor	Body Conc. (mg/kg)	Conc. Factor	Body Conc. (mg/kg)	Conc. Factor
0	0.1	*	0.1	*	0.1	*	0.1	*
1	—	*	0.1	*	0.1	*	0.1	*
2	—	*	0.1	*	0.1	*	0.1	*
3	—	*	0.1	*	0.1	*	0.1	*
4	—	*	0.1	*	0.1	*	0.1	*
8	—	*	0.1	*	0.1	*	0.12	190
12	—	*	0.1	*	0.1	*	0.14	230
16	—	*	0.1	*	0.1	*	0.26	420
24 / 1	—	*	0.1	*	0.1	*	0.20	320
36 / 1.5	—	*	0.1	*	0.1	*	0.20	470
48 / 2	—	*	0.1	*	0.10	1100	0.37	600
72 / 3	—	*	0.1	*	0.14	1560	0.58	930
96 / 4	—	*	0.1	*	0.15	1670	0.40	650
154 / 6.5	0.1	*	0.1	1590	0.33	3670	1.28	2060
336 / 14	—	*	0.13	3250	0.43	4780	7.40	11930
504 / 21	0.1	*	0.15	3750	0.45	5000	6.67	10900
672 / 28	0.14	*	0.17	4250	1.57	17400	10.82	17450
840 /35	0.10	*	0.21	5250	0.75	8330	16.48	26580
			PCB - STOPPED					
1176 / 49	0.1	*	0.1	*	0.12	*	3.24	*
1512 / 63	0.15	*	0.1	*	0.13	*	1.64	*

* Magnification factor not calculated.

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37,000 times the water concentration in 14-28 days (Figure IV.2.1), whereas pinfish exposed to Aroclor 1254 accumulated it to 22,000 times the water concentration (111). Thereafter, the concentration of PCB (ppm in tissues) remained roughly constant while the total amount continued to increase as the fish grew. After placing the fish in clean water the PCBs were gradually eliminated, falling by half in about 4 weeks (Figure IV.2.1). In a parallel experiment with pinfish exposed to Aroclor 1016, a bio-accumulation factor of about 20,000 was measured after 42 days (Table IV.2.4, Figure IV.2.1, ref. 100). Thus the bio-accumulation of Aroclors 1016 and 1254 was similar in this fish. However, there was some differential uptake of the components of Aroclor 1016, since the peaks 1-3 in the chromatogram (mono- and di-CBs) were proportionately reduced in the PCBs stored in the fish tissue, whereas the tri- and tetra-CBs were proportionately increased in the fish tissues (Figure IV.2.2, and Table IV.2.5; cf. Figure II.3.3).

DeFoe et al. (113) found that the bio-accumulation factor for adult fathead minnows at 25°C is approximately 120,000 for Aroclor 1248 and 270,000 for Aroclor 1260. Female fathead minnows accumulated about twice as much PCBs as the males but the difference is largely due to the greater amount of lipids in the females. The residues of PCBs in the fish tissues were directly proportional to the water concentration (Figure IV.2.3). The storage factor for fish lipids (ppm in lipids/ppm in water) was between 1 and 2 million (ibid.). Nebeker et al. (112) found that the bio-accumulation factor

BIOACCUMULATION AND DEPURATION OF PCB's BY FISHES

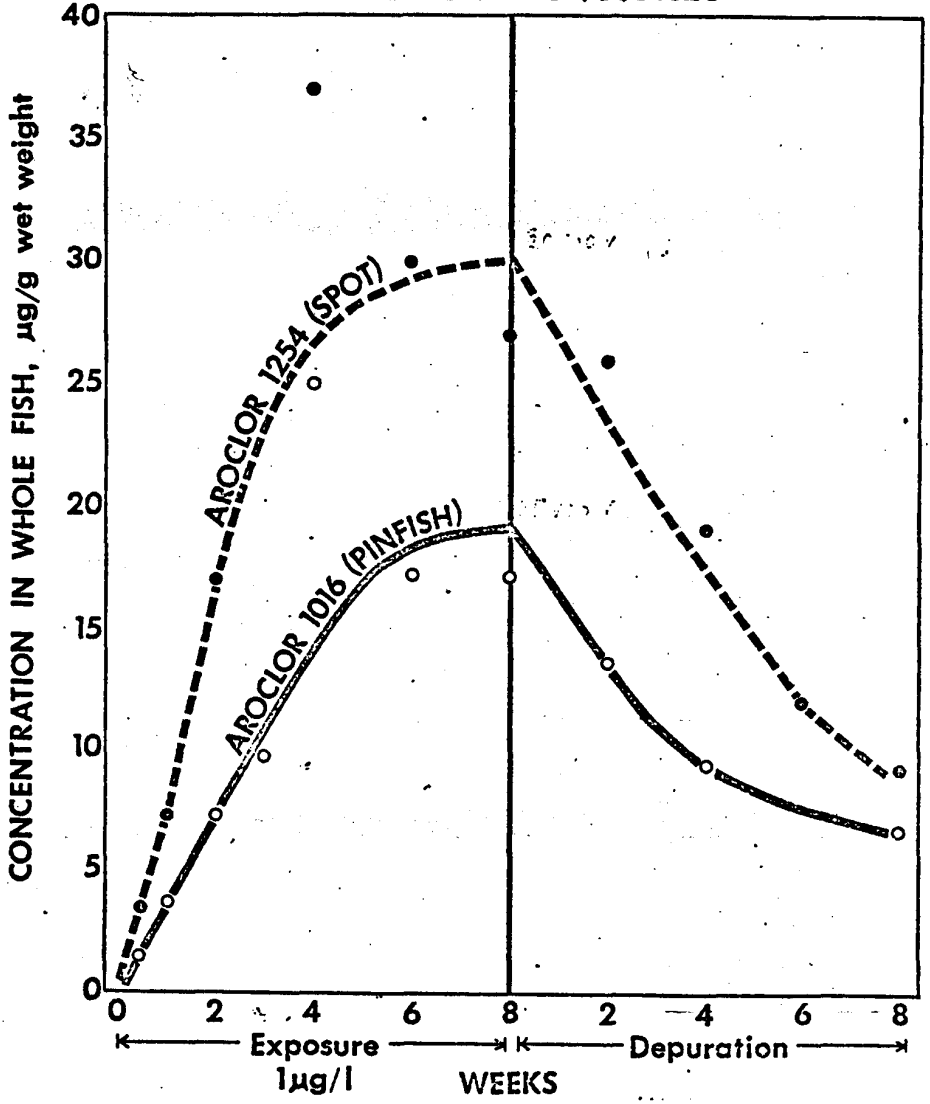

Figure IV.2.1
(from ref. 330)

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Table IV.2.4
(from ref. 100)

TOXICITY AND UPTAKE OF AROCLOR 1016 BY PINFISH (*Lagodon rhomboides*) EXPOSED FOR 42 DAYS IN THREE SEPARATE EXPERIMENTS

Test concentration (μg/liter)		Mortality (%)	Concentration in fish (μg/g, wet weight)		
Nominal	Measured		Flesh	Flesh and skin	Whole fish
Control	ND[a]	36	ND	ND	ND
0.1	ND	16	0.7	0.8	2.4
1.0	0.8	48	5.1	6.3	11
10.0	3.0	38	60	90	166
Control	ND	12	0.5	0.6	0.5
1.0	0.9	16	4.0	6.0	17
3.2	2.5	16	34	39	65
10.0	7.0	28	63	76	170
32.0	13	44[b]	140	180	620
Control	ND	6	ND	ND	ND
10.0	6.8	6	23	49	111
32.0[c]	21	50[b]	30	48	106
100.0[c]	50	50[b]	38	72	205

[a] ND, not detectable: <0.2 μg/liter in water: <0.2 μg/g in tissue.
[b] Mortality significantly greater than in control fish, $\alpha = 0.01$.
[c] Exposure terminated and tissues analyzed when 50% of the fish died: 33 days at 32 μg/liter and 18 days at 100 μg/liter.

Figure IV.2.2 (from ref. 100)

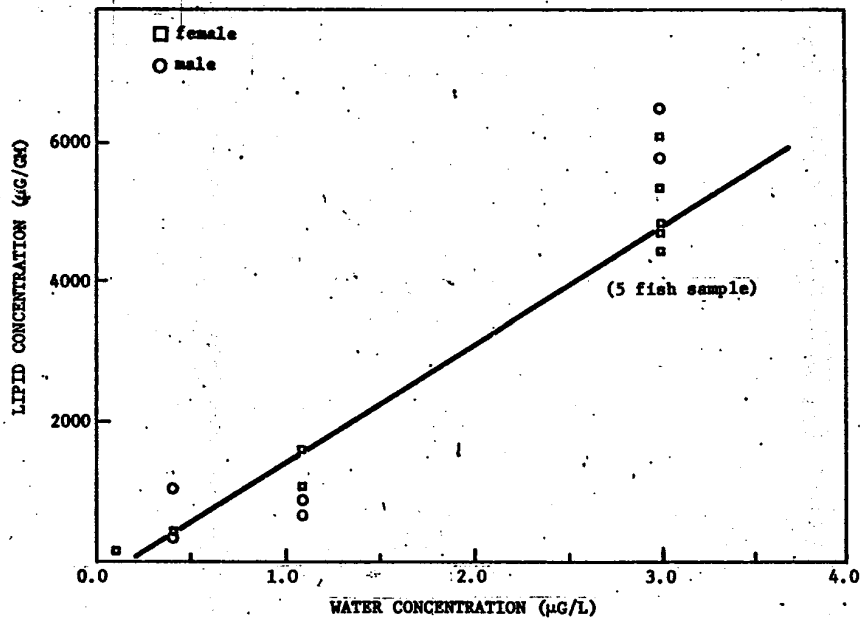
Chromatogram of 9 peaks of Aroclor 1016 reference standard. Operating conditions: gas flow nitrogen 25 ml/min; injection and detector temperature 210°C; oven temperature 190°C; ^3H electron capture detector; 152.4 × 0.32 cm glass column packed with 2% OV-101 on 100–120 Gas Chrom Q.

Table IV.2.5 (from ref. 100)

PERCENTAGES OF THE 9 MEASURED PEAKS FROM CHROMATOGRAMS OF AROCLOR 1016 REFERENCE STANDARDS AND FROM TISSUES OF PINFISH EXPOSED TO 1 μG/LITER OF AROCLOR 1016 FOR 56 DAYS AND THEN HELD IN PCB-FREE WATER FOR 56 DAYS

	Percentage of peak*									Chromatograms
	1	2	3	4	5	6	7	8	9	
Reference standard	12.4	11.3	10.0	23.5	13.3	6.4	8.1	8.8	6.2	8
All tissues through 14 days of exposure	2.4	5.3	1.8	39.6	7.7	10.3	12.1	9.4	11.4	7
All tissues, days 21–56 of exposure	2.5	5.0	2.1	33.2	7.8	11.9	11.3	12.3	15.9	12
All tissues during 56 day depuration	1.0	1.7	0.3	38.2	2.8	15.0	12.4	11.9	16.7	7

* Determined as $\frac{\text{peak height}}{\text{sum of nine peaks}} \times 100$.



Source: DeFoe, D.L., G.D. Veith, and R.W. Carlson, 1975. Effects of Aroclor 1248 and 1260 on the fathead minnow (Pimephales promelas), U.S. Environmental Protection Agency, National Water Quality Laboratory, Duluth, Minnesota.

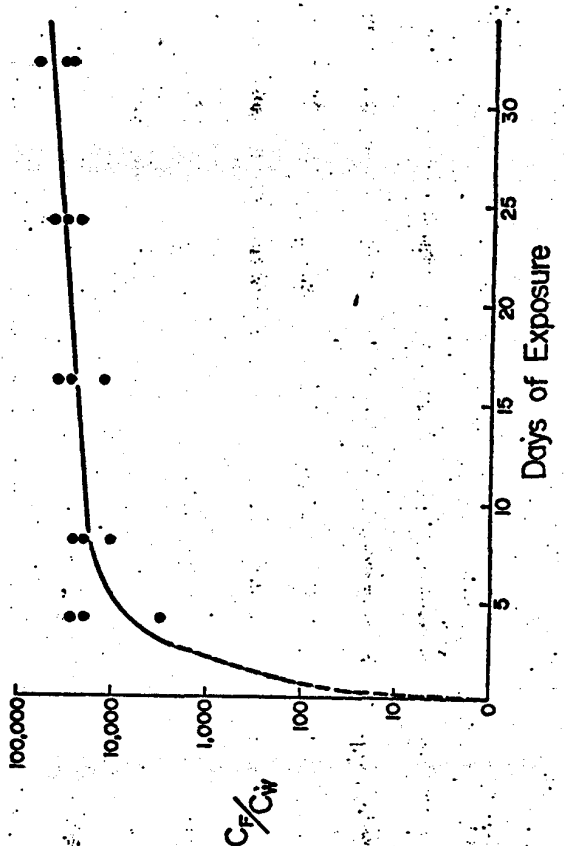
Figure IV.2.3 (ref. 113)
Aroclor 1248 residue in fathead minnows, normalized to lipid concentration in fish.

for Aroclor 1254 in fathead minnows was between 110,000 and 240,000, and that for Aroclor 1242 was between 32,000 and 274,000. Although the results varied somewhat between replications of these experiments there is no evidence that the bio-accumulation factors in this fish increase significantly with the degree of chlorination of the mixture. Veith and Kiwus (338) conducted an experiment with fathead minnows exposed to Aroclor 1016 and found a bio-accumulation factor of about 50,000, but the concentrations in the fish were still rising after 32 days (Figure IV.2.4). This result for Aroclor 1016 falls well within the range observed for the same species for Aroclor 1242, and within a factor of 3 of that observed for Aroclors 1254 and 1248 (112, 113).

Bio-accumulation factors for bluegills exposed to Aroclors 1248 and 1254 were in the range 26,300 to 71,400. No major modification of the PCB isomer ratios were observed in the tissue residues, and no new compounds were identified (95).

Several studies have shown that fish also store PCBs when fed contaminated food while kept in clean water. In these cases the bio-magnification factors were quite modest, because the fish were able to exchange PCBs across their gills with the ambient water; in natural environments both water and food would contain PCBs and fish would take in PCBs via both routes.

Coho salmon (Oncorhynchus kisutch) fed Aroclor 1254 for 240 days at dietary concentrations of 0.4 to 580 ppm (14.5 to 14,500 µg/kg body weight per day) accumulated whole body residues which



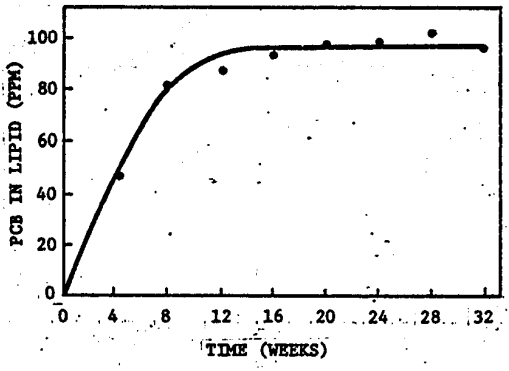
Variation of the Aroclor 1016 bioconcentration factor (C_F/C_W) with time of exposure. The C_F is derived from composites of five fish.

Figure IV.2.4 (ref.338)

were 0.9 to 0.5 times the dietary levels (95). The highest residue value was 300 ppm.

Accumulation and retention of Aroclor 1254 by rainbow trout (Salmo gairdnerii) from the diet followed the same pattern as that for coho salmon (339). The relative concentration of PCB in the lipid fraction of trout increased rapidly during 8 weeks' exposure to 15 ppm Aroclor 1254 in the food, then tended to equilibrate at about 95 ppm (Figure IV.2.5). While the relative amount in the fish reached quasi-equilibrium, the absolute quantities continued to increase as the trout grew (Figure IV.2.6). The distribution of PCB among the tissues was dependent on the lipid content of the various tissues (Table IV.2.6).

The uptake of pure PCB isomers by fish has been studied in three experiments. Gruger et al. (121) fed 3,4,3',4' tetra-CB and two hexa-CBs to juvenile coho salmon at a dietary concentration of 3.3 ppm each (total 10 ppm) for 165 days. The material fed during the first 24 days was retained well in the tissues, but tissue concentrations tended to level off and after 28 days the concentration of the tetra-CB was only about half that of either of the two hexa-CBs (Tables IV.2.7, IV.2.8). The experiment is complicated by the fact that the treated fish lost weight and depleted their lipid reserves, and that equilibrium concentrations in the tissues had not been reached (Table IV.2.7). As in the case of the trout, most of the PCBs were concentrated in the lipid-rich tissues, and the average concentration of PCBs in lipids was similar (about 70 ppm) in all



Source: Lieb, A.J., D.D. Bills and R.O. Sinnhuber, 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (Salmo gairdneri), Journal of Agricultural and Food Chemistry, 22(4): 638-642.

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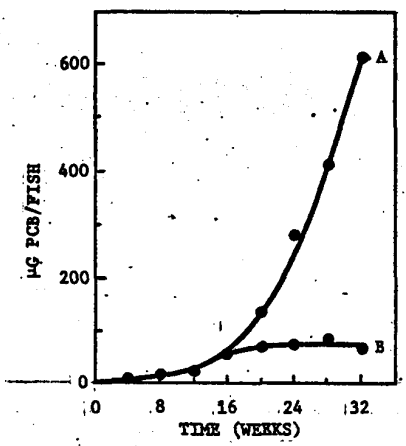
Figure IV.2.5 (ref. 339)
PCB in lipid fraction of rainbow trout after exposure to 15 ppm Aroclor 1254.



Source: Lieb, A.J., D.D. Bills, and R.O. Sinnhuber, 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (Salmo gairdneri), Journal of Agricultural and Food Chemistry, 22(4): 638-642.

Figure IV.2.6 (ref. 339)
Total amount of PCB per fish: (A) fish on diet containing 15 ppm of PCB; (B) fish removed from diet containing 15 ppm of PCB at end of 16 weeks.

Table IV.2.6 DISTRIBUTION OF AROCLOR 1254 IN TISSUE OF RAINBOW TROUT

Tissue	% lipid in tissue	Concn of PCB in lipid, ppm	Concn of PCB in tissue ppm
Visceral adipose	92.8	111	103
Gill	9.7	113	11.3
Muscle	2.7	104	2.8
Stomach	6.5	104	6.8
Liver	3.5	57	2.3
Whole fish	8.5	96	8.2

Source: Lieb, A.J., D.D. Bills, and R.D. Sinnhuber, 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (Salmo gairdneri) Journal of Agricultural and Food Chemistry 22(4): 638-642.

Table IV.2.7 (ref. 121)

ANALYSES OF WHOLE JUVENILE COHO SALMON WHEN FED FOR 24, 53, AND 108 DAYS[1]

Group	Days	Body wt, g	Lipid content, wt%	3,4,3',4', chlorobiphenyl	2,4,5,2',4',5' chlorobiphenyl	2,4,6,2',4',6' chlorobiphenyl	Total
				Wet tissue,[2] µg/g			
Test	24	4.24	7.60	0.49 (47%)	0.73 (70%)	0.66 (63%)	1.88 (60%)
	53	5.26	6.48	0.59 (30%)	0.93 (47%)	0.90 (45%)	2.42 (41%)
	108	3.06	3.8	0.65 (16%)	1.41 (35%)	1.42 (35%)	3.48 (29%)
Control	24	3.79	5.83	0.059	0.090	0.050	0.199
	53	5.42	7.38	0.044	0.047	0.053	0.144
	108	7.87	6.8	0.107	0.023	0.030	0.160

1. Three fish per group. 2. Percent of chlorobiphenyl amount found in fish relative to amount fed is given in parentheses.

Source: Gruger, E.H., Jr., N.L. Karrick, A.I. Davidson and T. Hruby. 1975. Accumulation of 3,4,3',4'-tetrochlorobiphenyl and 2,4,5,2',4',5'-and 2,4,6,2',4',6'-hexachlorobiphenyl in juvenile coho salmon. Environmental Science and Technology 9(2): 121-127.

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Table IV.2.8 (ref. 121)

CONCENTRATIONS OF CHLOROBIPHENYLS AND LIPID CONTENT IN TISSUES OF JUVENILE COHO SALMON FROM TEST AND CONTROL GROUPS

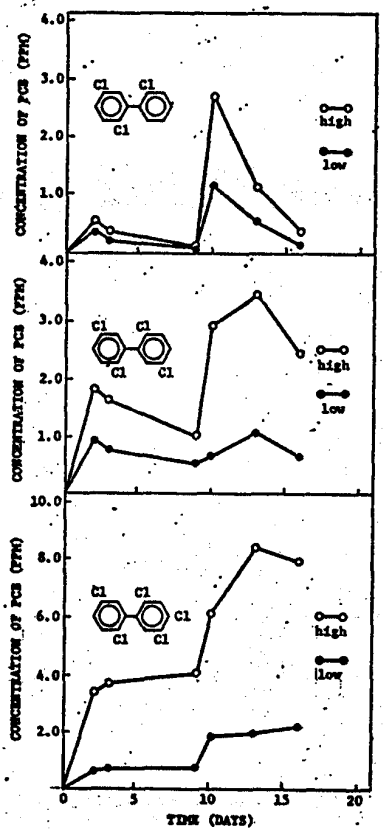
Tissue specimens at 117 days	Lipid content, wt%	3,4,3',4' chlorobiphenyl	2,4,5,2',4',5' chlorobiphenyl	2,4,6,2',4',6' chlorobiphenyl	Total
		Wet tissue, μg/g			
Test group					
Brain	7.1	0.15	0.31	0.38	0.84
Liver	2.9	0.25	0.35	0.50	1.1
White muscle	2.6	0.29	0.63	0.64	1.6
Intestines	4.3	0.30	0.79	0.82	1.9
Stomach and pyloric caneca	3.3	0.43	0.76	0.95	2.1
Spinal column	6.6	0.92	1.4	1.4	3.7
Heart	-	0.98	1.2	1.5	3.7
Lateral line muscle	6.1	0.77	1.8	1.9	4.5
Spleen	-	0.85	2.0	2.1	4.9
Adipose	73.0	10.6	19.3	18.8	48.7
Control group					
Brain	6.6	0.020	0.009	0.017	0.046
Liver	3.9	0.034	0.014	0.037	0.085
White muscle	5.5	0.028	0.015	0.025	0.068
Intestines	6.6	01098	0.032	0.069	0.20
Stomach and pyloric caneca	4.2	0.032	0.014	0.022	0.069
Spinal column	23.7	0.096	0.026	0.032	0.15
Heart	-	0.85	0.068	0.18	1.1
Lateral line muscle	11.4	0.10	0.058	0.077	0.24
Spleen	-	(2.0)	0.13	0.12	(2.3)
Adipose	73.5	0.30	0.19	0.36	0.85
at 165 days					
Test Group					
White muscle		0.086	0.20	0.22	0.51
Brain		0.26	0.37	0.56	1.2

the tissues tested after 117 days (Table IV.2.8). After 48 days' starvation the concentrations of PCBs in the tissues were depleted somewhat (to a total of 33 ppm in adipose), but those in the brain were increased slightly (Table IV.2.8). The tetra-CB was excreted only slightly faster than the hexa-CBs (121).

Sanborn et al. (307) exposed green sunfish to three representative chlorobiphenyls in two pulsed exposures (24 hours' exposure to 1 or 3 ppb 9 days apart). Although the tri-CB was rapidly excreted and was almost eliminated after 15 days, the penta-CB was well retained and there was no evidence of elimination in the 15-day period of study (Figure IV.2.7). The tetra-CB was taken up and retained slightly less well than the penta-CB, but the concentrations in the fish at the end of the 15-day experiment were only 2-3 times smaller than those of the penta-CB, despite the limited exposure time (Figure IV.2.7). Thus the major difference in uptake and retention appeared to be between the tri- and tetra-CBs.

Metcalf et al. (45) exposed mosquito fish to the same three chlorobiphenyls under model ecosystem conditions (exposure via both food and water). Although the exposure to the fish was for only 3 days, they nevertheless accumulated the tri-CB to 6,400 times the water concentration, and the tetra- and penta-CBs to about 12,000 times the water concentration (Table E.5). Thus under these circumstances there was no difference in bio-accumulation between the tetra- and penta-CBs.

To summarise, under laboratory conditions fish bio-accumulate PCBs to concentrations 30,000 to 300,000 times higher than those in



Source: Sanborn, J.R., W.F. Childers, and R.L. Metcalf, 1975. Uptake of three polychlorinated biphenyls, DDT, and DDE by the green sunfish, Lepomis cyanellus, RAF., Bulletin of Environmental Contamination and Toxicology, 13(2): 209-217.

Uptake of twi-, tetra-, and pentachlorobiphenyl by the green sunfish.

Figure IV.2.7

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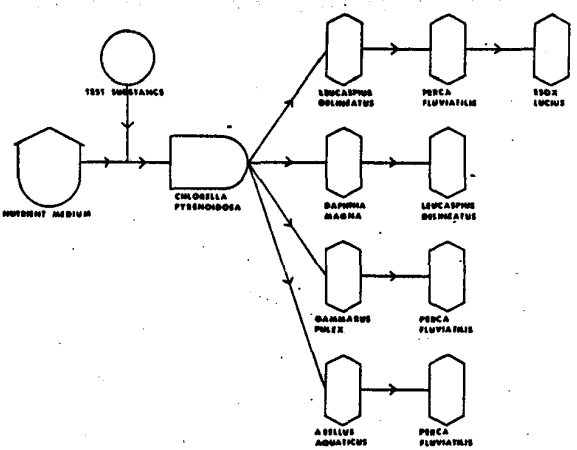
the ambient water (up to 2 million times higher in lipids). There is evidence that mono-, di-, and tri-CBs are poorly retained in fish tissues, but the differences in retention between tetra- and higher CBs are small, no more than a factor of 2-3 in the circumstances of these experiments.

IV.2.4. Bio-accumulation in Model Aquatic Ecosystems

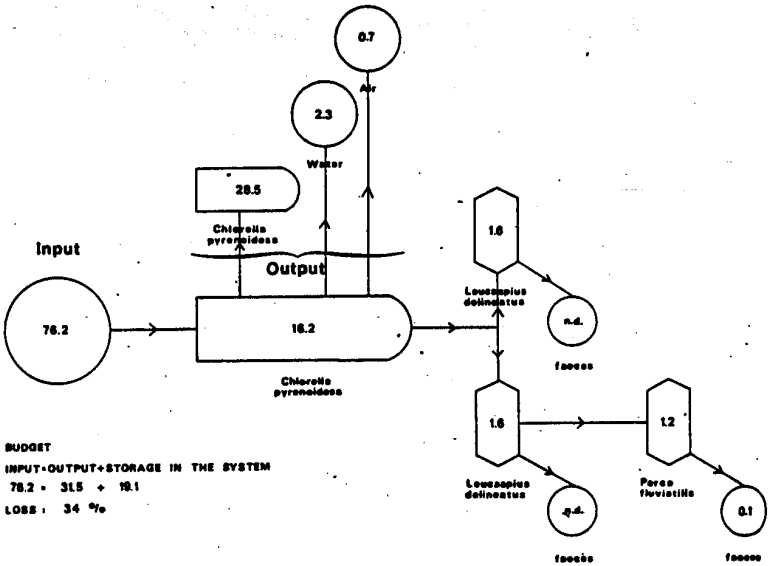
Two studies have been published of the behavior of PCBs in model aquatic ecosystems, designed to simulate the transport of PCBs from water through simple food chains. In one study by Metcalf et al. (45), radiolabelled chlorobiphenyls were applied to a mixed terrestrial-aquatic system and subsequently traced in water and various aquatic organisms. The results have already been summarized in the previous sections and in Appendix E (Tables E.3-E.5). The study showed how PCBs and their metabolites move from the terrestrial to the aquatic environment and are taken up there by aquatic organisms, including plants, insects, fish, and snails. Because the fish were only introduced into the system for the last few days of the experiment, the results do not indicate the full potential for bio-accumulation in food-webs: the figures of 6,000-60,000 for bio-accumulation factors in snails (Table E.5) give the best measure of this potential.

While Metcalf's system is primarily designed to investigate storage and biodegradability (45), a system designed by Södergren was designed to trace the movement of PCBs through a simple food-web (340). Figure IV.2.8 shows the basic design of the system and Figure IV.2.9 shows the results of a test with Clophen A50. Most of



Principle design of the model aquatic ecosystem. The organisms not mentioned in the text constitute alternative food-chains viable in the system, but not included in this study.

Figure IV.2.8
(from ref. 340)



Distribution and budget of Clophen A 50 within the model aquatic ecosystem (µg). n.d. = no substance detected.

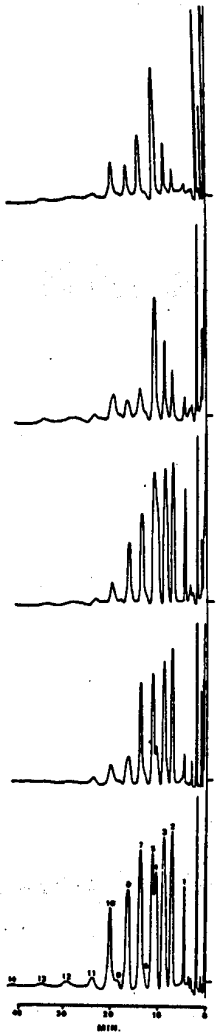
Figure IV.2.9
(from ref. 340)

the transport of the material to the first consumer organism (the cyprinid fish Leucaspius delineatus) took place via the alga Chlorella pyrenoidosa. There were some changes in the relative proportions of the components of the Clophen mixture as they passed through the system (Figure IV.2.10). However, these changes were very minor except in the two predatory fish (the pike Esox lucius and the perch Perca fluviatilis). Even the first component in the Clophen mixture (2,5,2',5'-tetraCB) was passed essentially unchanged through the alga to the first consumer (Figure IV.2.10).

IV.2.5. Storage and Bio-magnification in Birds

Storage and bio-magnification in birds are of interest for two reasons: (a) to indicate the range of residues likely to be ingested by humans eating wild or domestic birds; (b) to indicate the degree to which passage of residues up the food chain can lead to toxic effects in the birds themselves or in their predators.

In a study with chickens (149, 150), laying females fed Aroclor 1248 at 0.5 ppm accumulated 3.1 ppm in their adipose tissues and 0.22 ppm in their eggs after 8 weeks; correspondingly higher levels in tissues and eggs were found at higher feeding levels (Table IV.2.9). Similar results were obtained with Aroclor 1242: hens fed at 5 ppm laid eggs containing 1.7 ppm after 6 weeks (152). However, laying hens may be a poor model for other birds because their continuous egg-laying provides them with an important route of excretion (341) not available to wild birds which lay few eggs. Pheasants given 12.5 mg Aroclor 1254 weekly (equivalent to about 50 ppm in the



Gas chromatograms obtained from various components in a model aquatic ecosystem when testing Clophen A 50.

Figure IV.2.10
(from ref. 340)

Table IV.2.9
(from ref. 150)

—Transfer of PCBs[1] from the laying diet to the adipose tissues and eggs of hens

Diet no.	PCBs in diet	PCBs in adipose tissues			PCBs in eggs				
		1 wk.	4 wks.	8 wks.	1 wk.	4 wks.	6 wks.	7 wks.	8 wks.
	p.p.m.	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*	*p.p.m.*
1 (Diet A)	0	0	0	0	0	0	0	0	0
2	0.5	1.54	2.16	3.10	0.10	0.16	0.21	0.20	0.22
3	1.0	2.21	4.41	6.62	0.19	0.33	0.42	0.45	0.41
4	10.0	10.4	22.7	37.1	1.05	2.21	2.83	3.11	3.06
5	20.0	16.3	54.7	82.7	2.19	4.51	5.37	5.72	7.04

[1] Aroclor 1248, Monsanto. Inc., St. Louis, Mo.

diet) for only 16 weeks accumulated an average of 24 ppm in their whole bodies; this did not decline significantly even after 6 months on a clean diet (141). Mallards fed 25 ppm Aroclor 1254 laid eggs containing 33-56 ppm PCBs (wet weight) (131). Ring doves fed 10 ppm Aroclor 1254 in the diet accumulated 8 ppm in the muscle, 5.5 ppm in the brain, 15.3 ppm in the liver, 736 ppm in the fat, and 4.8 ppm in the eggs (342, 161-163); after starvation the PCBs were mobilized from the fat and the birds died with 293 ppm in the brain (Table IV.2.10). American Kestrels fed 10 ppm Aroclor 1254 in the diet laid eggs with average residues of 225 ppm (dry weight, corresponding to about 30 ppm, wet weight) (164).

A number of workers studying the storage of PCBs in birds have reported that the lower chlorobiphenyls in Aroclor 1254 (tetra-CBs) are retained less well than the higher components (141, 131, 342, etc.). Call *et al.* (343) have provided quantitative data showing that tetra- and penta-CBs are stored at relatively lower levels and hexa- and hepta-CBs at relatively higher levels in Japanese quail than in the Aroclor 1260 with which they were fed.

However, the differential storage of PCBs in birds is not related simply to the degree of chlorination. DeFreitas and Norstrom (27), studying the elimination of components of Aroclor 1254 from pigeons, found that the components which were excreted and/or metabolized most rapidly were those with 2,3,- 3,4-, or 2,3,6- substitutions in at least one ring. Components with 2,4,5-, 2,3,4-, and 2,3,4,5-substitution patterns were not eliminated. Components with 2,5-

Table IV.2.10
(from ref. 161)

Organ levels, ppm, wet weight basis.

	Muscle	Brain	Liver	Fat
Pre-stress (n=4)	8.1±2.3	5.5±1.6	15.3±12.6	736.1±211.6
	(4.1–9.9)	(4.8–8.0)	(3.6–27.5)	(481.2–1069.4)
Post-stress (n=5)	172.9±38.3	293.0±27.6	1118±267	***
	(120.0–227.0)	(254.0–340.2)	(937.3–1688.0)	

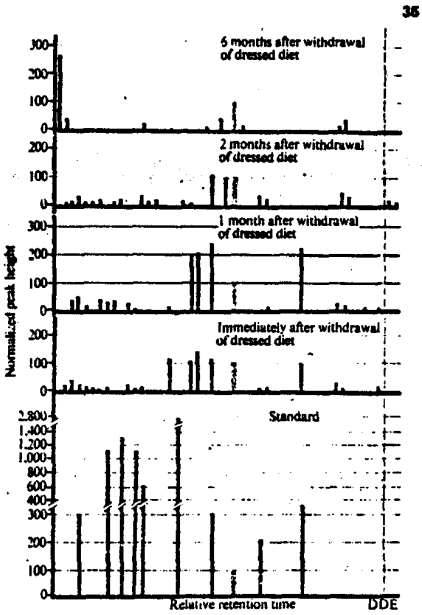
Figures are means, standard deviations, and range.
*** No fat present.

Table IV.2.11
(from ref. 344)

Mean Total PCB Residues (p.p.m.), found in Quail Tissue after Feeding 'Aroclor 1242' and 'Aroclor 1254' at 250 p.p.m. for 20 Days

Tissue	'Aroclor 1242'	'Aroclor 1254'
Liver	17.0±2.6*	28.1±7.9
Heart	3.2±0.7	7.0±1.1
Brain	7.8±1.7	7.7±2.3
Omental fat	123±17	304±31

* All figures are mean of six birds ± s.e.



Gas-liquid chromatographic "spectra", normalized to a common peak (stippled column) of 'Arocior 1242' standard and mean pigeon liver residues following feeding at 500 p.p.m. in diet for 28 days.

Figure IV.2.11
(from ref. 344)

substitutions were eliminated, but much more slowly than those with 2,3-, or 3,4- substitutions. Bush et al. (155) obtained similar results for differential elimination of components of Aroclor 1254 from laying hens, and suggested that 4,4' substitution is more important than number of chlorine atoms in determining the degree of persistence of chlorobiphenyls in hens. This agrees with the findings of Matthews and Anderson (328) in rats.

Finally, Bailey and Bunyan (344) found that although Aroclor 1242 was not accumulated so strongly from the diet by Japanese quail as Aroclor 1254, the difference was only by a factor of 2-2.5 (Table IV.2.11). Although the lower components in Aroclor 1242 (di- and some tri-CBs) were not well retained in pigeon tissues, some tri- and most tetra-CBs were well retained and were only slowly excreted over a 2-6 month period (Figure IV.2.11). Of the components well retained in the pigeon tissues, only one (probably 2,5,3',4'-tetra-CB) is not present in Aroclor 1016 (Figure II.3.3). Thus, although there is substantial differentiation of the components of PCB mixtures as they are taken up, stored, and eliminated by birds, some tetra- and even tri-CBs are reasonably well retained in tissues and secreted into eggs.

Very little information is available on bio-magnification of PCBs by any fish-eating birds, which are of interest as being the species at greatest risk in the environment. However, cormorants (Phalacrocorax carbo) dosed with an average of about 300 ppm Clophen A60 (about 25 mg/kg/day) accumulated 850-2,750 ppm PCB residues in

their whole bodies in 55-125 days, including residues of 10-20,000 ppm in their fat (142). This indicates a somewhat higher degree of bio-magnification than for any of the other birds listed above.

IV.2.6. Storage and Bio-magnification in Mammals

The storage and kinetics of PCBs in mammals are of interest (a) to indicate the range of residues likely to be ingested by humans eating domestic mammals; (b) as models for the storage and kinetics of PCBs in humans themselves. There is an extensive literature on the uptake, kinetics, and elimination of PCBs in mammals, but for this document primary interest is attached to the results of long-term feeding at low levels.

Absorption of PCBs from the gastrointestinal tract appears to be very efficient. Albro and Fishbein (345) fed nine individual chlorobiphenyl isomers ranging from mono- to hexa-CBs to rats and found that 91-99% of the dose was retained in the body. After absorption PCBs are distributed throughout the body: their distribution tends to parallel the lipid content of the tissues, so that PCB storage in the tissues occurs in the following order:

fat > liver > feces > kidney > brain > plasma > urine (346).

The concentration of PCBs in adipose tissue is 10-100 times the concentration found in other tissues, both early after single doses (182) and after prolonged intake (346).

Allen et al. (196) fed six adult female rhesus monkeys 25 ppm of Aroclor 1248 for two months. PCB concentrations in samples of adipose tissue averaged 127 μg/g fat for all animals. At that time, the experimental diet was discontinued. Eight months later the PCB content was 34 μg/g fat. PCBs were transferred across the placenta of one female which gave birth and concentrated at high levels in the fat and adrenals of the infant (Table IV.2.12). In an infant born

to a female 29 months following the discontinuation of PCBs, the levels within the adipose tissue were 3.38 ppm at 4 months of age (69).

Female monkeys given 5.0 ppm PCB in their diets attained maximum levels of PCBs within their adipose tissue at 6 months (141 to 177 ppm adipose tissue). However, it required approximately 14 months on the 2.5 ppm diet for the monkeys to reach similar maximum PCB levels in their adipose tissue (126 to 144 ppm). Males which received 5.0 ppm PCBs attained levels ranging from 128 to 200 µg per g adipose tissue at 14 months. Infants born of mothers exposed to 2.5 and 5.0 ppm PCBs within the diet contained concentrations of PCBs ranging from 1.0 to 4.8 ppm within the skin at birth. While nursing from mothers consuming PCB diets, the infants continued to accumulate the compound. At 3 months the levels within the tissues ranged from 86 to 136 ppm. The concentration of PCBs within the milk ranged from 0.15 to 0.40 ppm. The tissues of the infants which died while nursing PCB-fed mothers contained high levels of PCBs within the thymus, ovaries, brain, kidneys, adrenal glands and pancreas (20-48 ppm tissue). Lower levels were found in the liver, lymph nodes and bone marrow (8-16 ppm) (69).

Table IV.2.12
(from ref. 196)

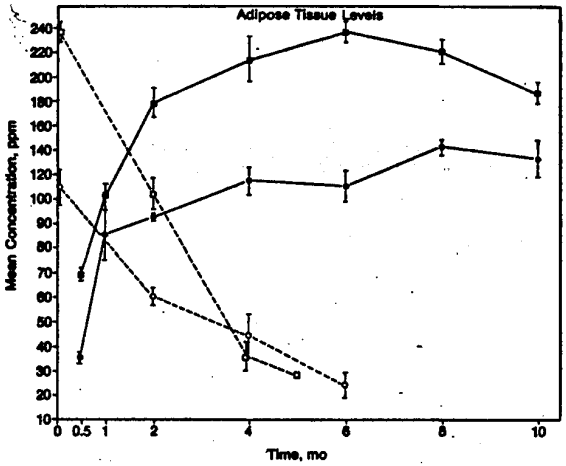
POLYCHLORINATED BIPHENYL CONTENT OF TISSUES OBTAINED FROM MOTHER AND INFANT FOLLOWING CONSUMPTION OF AROCLOR 1248 BY THE MOTHER PRIOR TO PREGNANCY

Organ	Weight (g)	PCB content (μg/g)
Mother		
Liver	—	56.3
Fat	—	50.0
Placenta	—	0.9
Infant		
Fat	—	27.70
Adrenals	0.2	24.40
Liver	11.9	0.01
Muscle	—	0.98
Brain	52.9	0.29
Kidneys	2.8	0.10
Large intestine	—	0.08
Small intestine	—	0.52
Stomach	—	0.55
Lung	6.1	0.21
Skin	—	0.31

Curley et al. (346) fed weanling Sherman rats a diet containing 100 ppm Aroclor 1254 for 58 days and for 240 days. Animals were sacrificed at various intervals. A steady buildup of PCB in all tissues was observed over the 58 day period. The rats stored more PCB in their tissues after 240 days than at the end of 58 days. In experiments at two dose levels (100 ppm and 500 ppm) for 240 days, Curley et al. (346) observed that PCB in fat after 240 days at dietary levels of 100 and 500 ppm were 1,101 and 10,021 ppm, respectively. They did not reach a point of equilibrium storage.

Grant et al. (347) fed male rats a diet containing either 0, 2, 20 or 100 ppm of Aroclor 1254 for 246 days. The residues in the blood, brain, heart, liver and fat were found to be dose related. All components of residues were not metabolized at the same rate.

Burse et al. (208) fed rats diets containing 100 ppm of Aroclor 1242 or Aroclor 1016 for up to ten months. Rats were sacrificed at intervals and tissues analyzed. A roughly steady state concentration of PCBs in adipose tissue was approached in two months and reached in 4-8 months (115 ppm of Aroclor 1242 and 214 ppm of Aroclor 1016) (Figure IV.2.12). Equilibrium levels of Aroclor 1016 and 1242 seem to be reached sooner than equilibrium levels of Aroclor 1254 and the levels of Aroclors 1016 and 1242 were 5-10 times smaller than those reached after continuous feeding with Aroclor 1254 (208, 346). This indicates considerably less bio-magnification of tri- and tetra-CBs than of penta- and hexa-CBs in rats. After the rats were placed on a PCB-free diet the residues of Aroclor 1016 were eliminated somewhat faster than those of Aroclor 1242 (Figure IV.2.12). Comparison



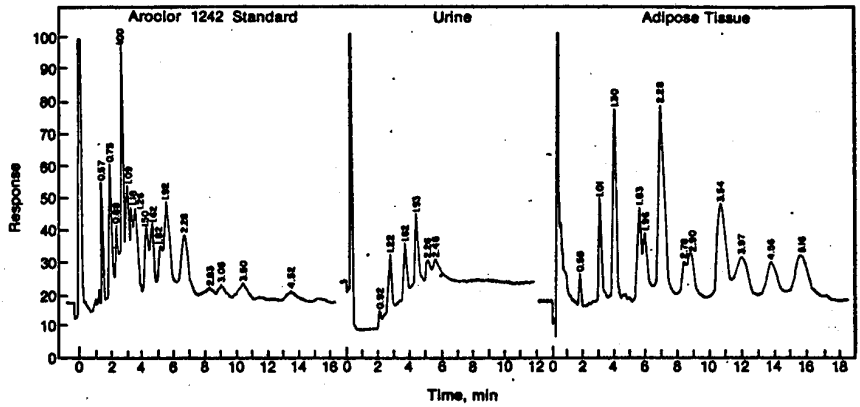
-Residues resulting from a continuous diet of a 100 ppm concentration of Aroclor 1016 (■) or Aroclor 1242 (•) for a period of ten months. Residues following a continuous diet for only six months and recovery for five months for Aroclor 1016 (□) and for six months for Aroclor 1242 (○) are illustrated by the broken lines. Each point represents the mean and standard error (I) of four rats.

Figure IV.2.12
(from ref. 208)

of chromatograms (Figures IV.2.13, IV.2.14) shows that only the later eluting components (penta-, tetra-, and some tri-CBs) of Aroclors 1242 and 1016 were stored in the adipose tissue. The lower components were largely metabolized, as hydroxylated metabolites were found in the urine (348).

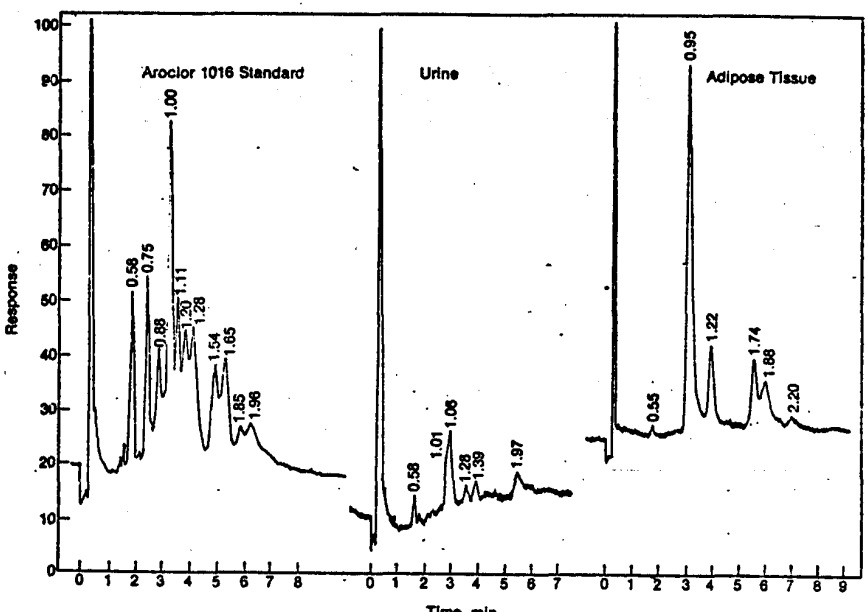
Storage and dynamics of PCBs have also been studied in domestic animals. Hansen et al. (216) fed sows with 20 ppm Aroclor 1242 throughout gestation and nursing and found PCBs in many tissues of the sows and their offspring. PCB levels in the fat and blood of the sows averaged 14 ppm and 0.25 ppm respectively (Table III.9.9). The offspring appeared to have somewhat lower concentrations of PCBs in their fat but higher concentrations in the blood (Figure IV.2.15). As with other mammals, there was considerable differentiation of the components of the Aroclor 1242 mixture (Figure IV.2.15): later eluting components (primarily penta- and tetra-CBs) were retained much better in the fat than the earlier eluting components. One-third to one-half of the PCBs stored in the fat of the sows consisted of components that would not be present in Aroclor 1016.

Fries et al. (349) fed 200 mg of Aroclor 1254 daily (0.4 mg/kg/day, equivalent to 12 ppm in the diet) to nine cows. Residues in the body fat and milk fat built up to 40 and 60 ppm respectively during a 60-day feeding period, but did not reach equilibrium levels in that time (Figure IV.2.16). Following discontinuation of dosing, levels of PCBs in fat and milk fell off steadily, but those in body fat had fallen only to 30 ppm after 60 days. The cows excreted only



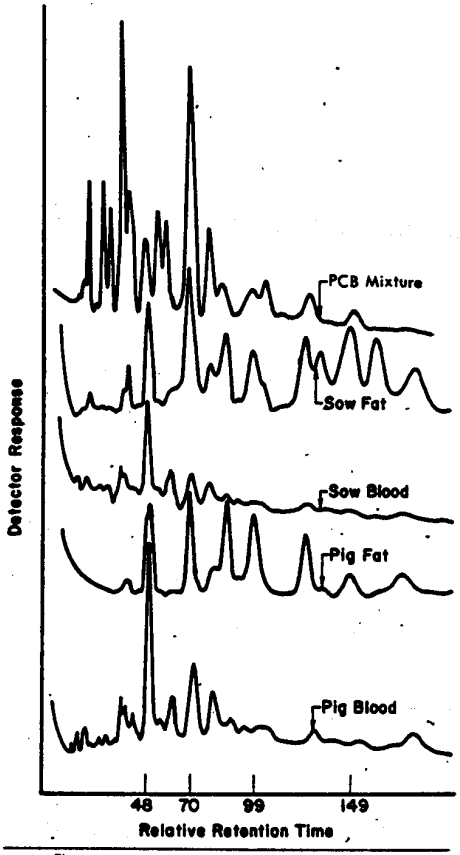
–Chromatograms of Aroclor 1242: 3.66 ng, an extract of urine: 4.5 mg, and adipose tissue: 0.027 mg from a male rat fed a diet containing 100 ppm Aroclor 1242 for eight months.

Figure IV.2.13
(from ref. 208)



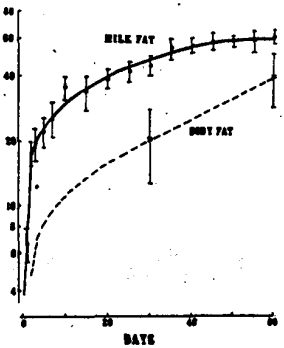
—Chromatograms of Aroclor 1016: 2.84 ng, extract of urine: 5.4 mg, and adipose tissue: 0.008 mg from a male rat fed a diet containing 100 ppm Aroclor 1016 for eight months.

Figure IV.2.14
(from ref. 208)



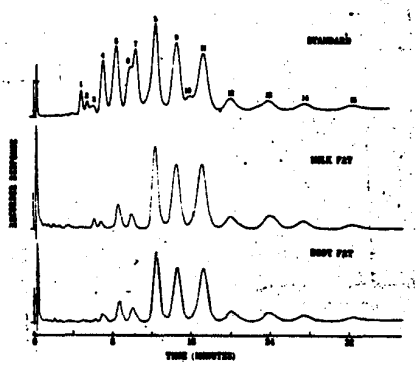
—Electron capture gas chromatographs of PCB mixture in standard and tissue extracts from sows given PCB mixture in the feed and from their offspring. Back-fat samples were diluted 100-fold.

Figure IV.2.15
(from ref. 208)



Concentration of PCB in milk fat and body fat of cows fed 200 mg of PCB per day. Each point is an average of nine cows ± standard deviation. The curve for body fat is extrapolated before 30 days.

Figure IV.2.16 (from ref. 349)



Typical gas chromatograms of an Aroclor 1254 standard, a milk fat residue sample, and a body fat residue sample. Retention times of peaks 1, 2, 4, 6, and 9 were equal to the retention times of the pure compounds 2,5,2',5'-tetrachlorobiphenyl, 2,3,2',5'-tetrachlorobiphenyl, 2,5,3',4'-tetrachlorobiphenyl, 2,3,4,2',5'-pentachlorobiphenyl, and 2,4,5,2',4',5'-hexachlorobiphenyl, respectively.

Figure IV.2.17 (from ref. 349)

20-25% of the daily dose in their milk, implying that even under conditions of continuous milk production they were still far from equilibrium after 60 days. There was differential accumulation of the higher components of the mixture in both body fat and milk (Figure IV.2.17), but peaks 2 and 3 (both tetra-CBs) were as well represented in the milk fat as in the original Aroclor mixture. Jan et al. (404) found that cows excreted substantial amounts of 3,5,3',5'-tetraCB in milk (up to 2 ppm in milk fat after a single oral dose of 0.8 mg/kg).

Frank et al. (405) reported data on body burdens of PCBs in captive harp seals (Pagophilus groenlandicus), as shown in the following table:

Age at Death and time in Captivity (months)	Wt. at Death (kg)	Estimate PCB Intake last 2-4 Months (mg/day)	PCB (mg)	PCB (mg/kg)
12-11	52.0	0.75	163	3.13
15-14	68.0	1.14	143	2.15
24-23	70.0	1.07	613	8.75
27-26	63.8	1.01	659	10.32
38-35	65.0	0.75	785	12.10
52-50	54.0	0.69	697	12.90

The diet of the seals contained an averaga concentration of about 0.2-0.3 ppm PCBs, but over 4 years' captivity the average whole-body concentration built up to 12.9 ppm (~~roughly 50~~ 25-38 ppm in fat).

IV.2.7. Storage and Bio-magnification in Humans

Although extensive surveys have been made of the occurrence of PCBs in human tissues, there is little systematic information on rates of uptake or elimination. PCBs are efficiently absorbed through the gastrointestinal tract: Price et al. (350) measured PCBs in diets, urine and feces of eight preadolescent girls in Virginia, and estimated absorption of the ingested PCBs as 88%. Humans are also exposed via air, water, and dermal contact, but it is generally believed that the major exposure is via the diet (1-3, 331, 351).

The most extensive data on distribution of PCBs in human tissues derive from Japan (35, 25). Fujiwara cited mean levels of 3.1 ppb in blood plasma, 4.7 ppm in body fat, and 50 ppb in milk of persons surveyed in the Kyoto area (25). He also cited a national survey which showed a mean level of about 34 ppb in 1116 samples of human milk. Kuratsuna (35) cited a national survey which reported PCB levels in the range 0.04-1.7 ppm in skin, 0.3-6.4 ppm in fat, and 0.01 to 0.6 in liver (Table III.16.9). Although these figures do not all refer to the same individuals, they suggest a distribution in the body tissues paralleling that of lipids, as in other animals.

Surveys in Canada show that the majority of Canadians have residues of 1-2 ppm in their adipose tissue. Human milk of Ontario residents was found to have average residues of about 1.1 ppm PCBs on a fat basis (342).

Extensive surveys in the United States have shown that the median level of PCBs in human adipose tissue is slightly less than 1 ppm (Table IV.2.13) (353). Less information is available on the occurrence of PCBs in other human tissues. Finklea et al. (354) found mean levels of 2-3 ppb in blood plasma in South Carolina. Measurements of residues in human milk have ranged from not detectable up to 100 ppb; 30 ppb has been suggested as a typical level (331).

The Total Diet Survey of the Food and Drug Administration provides an estimate of the dietary intake of an average person in the United States: in recent years this estimate has been close to 9 µg/person/day (351: Table IV.2.14). The Total Diet Survey is based upon a high consumption diet which includes 4 kg food/day, almost twice that consumed by the "average" man (355). Hence the average concentration of PCBs in the food sampled in the Total Diet Survey is about 2.2 ppb. Comparing this with the median level in adipose tissue from the Human Monitoring Program (353), the storage factor for human fat (ppm in fat/ppm in diet) appears to be at least 400. This is far higher than that recorded for any experimental animal: see Section IV.2.6, where high figures for storage factors in mammalian fat range from 120-180 in seals, through 35-60 in monkeys, 11-20 in rats, and 5 in cows, to 0.7 in pigs. Several explanations are possible for this discrepancy:

Table IV.2.13 (from ref. 353)

Levels of polychlorinated biphenyls in human adipose tissue

Data source	Sample size	Percent nondetected	Percent < 1 ppm	Percent 1-2 ppm	Percent > 2 ppm
Yobs, 1972	688	34.2	33.3	27.3	5.2
FY 1973 Survey	1277	24.5	40.2	29.6	5.5
FY 1974 Survey	1047	9.1	50.6	35.4	4.9

Table IV.2.14 (from ref. 351)

Estimates of daily PCB intakes (total diet study--teenage male)

Fiscal year	Average daily intake of PCB's[a]	
	Total diet (μg/day)	Meat-fish-poultry food class (μg/day)
1971	15.0	9.5
1972	12.6	9.1
1973	13.1	8.7
1974	8.8	8.8
1975 (1st half)	8.7	8.7

[a]Lower limit of quantitative reporting = 0.05 ppm with analytical method employed.

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(i) the dietary intake estimated by the Total Diet Survey may be too low, because of a high frequency of residues near the level of detectability;

(ii) routes other than the diet may give significant exposures;

(iii) the residues stored in fat may reflect much higher exposures in the past;

or (iv) humans may store PCBs much more efficiently than other animals.

Jelinek and Corneliussen considered (i) and attempted to minimize errors by assigning finite residue values to "trace" findings (351). Although other routes may be locally significant, no quantitative data have been provided that suggest intakes greater than a few micrograms per day for a typical person (2, 8, 331). The rapid decline of PCBs observed in the Yusho victims (Table III.16.9) makes it unlikely that present storage levels reflect exposures more than a few years ago. Hence it seems probable that humans do in fact store PCBs more efficiently than other animals. Humans are known to store other chlorinated hydrocarbons, such as dieldrin (335, 356) much more efficiently than other animals. This is of considerable importance for establishing "safe" levels for human intake, because it implies that for a given dietary concentration, humans will have tissue levels up to 10 or more times higher than those of experimental animals.

There is no precise information on the molecular or isomeric constitution of the PCBs stored in human adipose tissue in the

United States, although Kutz and Strassman state that most consist of penta-, hexa- and hepta-CBs (353). A detailed study of a pooled sample of human adipose tissue from Sweden indicated that it contained 4-6% tetra-CBs, 23-25% penta-CBs, 44-46% hexa-CBs, 21-22% hepta-CBs, 4% octa-CBs, and 0.6% deca-CB (Table II.4.1). Musial et al. (357) reported that most samples of human milk analyzed in Canada had PCBs similar in constitution to Aroclor 1254, but that peaks corresponding to Aroclor 1242 (i.e. tetra-CBs) were detected in all samples. Thus it seems likely that tetra-CBs, although minor, are not negligible constituents of the PCBs in human tissues.

IV.2.8. Bio-accumulation and Bio-magnification in Natural Ecosystems

Several writers have commented that bio-accumulation and bio-magnification factors observed in wild animals in the field are often much greater than those measured under controlled conditions in the laboratory. Some examples of this phenomenon as it relates to PCBs are given below.

Escambia Bay, Florida. Hansen (74) and Nimmo (330) have pointed out that bio-accumulation factors estimated for several wild species in Escambia Bay are much greater than those measured in the laboratory (99, 105; see Table IV.2.15). The discrepancies are especially great for crabs ($>$ 230,000 in the bay, versus a maximum of 4,600 in the laboratory) and fish ($>$ 670,000 in the bay, versus a maximum of 37,000 in the laboratory).

Table IV.2.15 (from ref. 330)

BIOACCUMULATION OF AROCLOR 1254 IN ESTUARINE ORGANISMS

	CONCENTRATION FACTORS	
ORGANISM	LABORATORY	ESCAMBIA BAY
OYSTER	165,000	> 100,000
SHRIMP	26,000	> 470,000
CRABS	4,600	> 230,000
FISHES	37,000	> 670,000

Hudson River, New York. In a field experiment, Veith et al. (358) held a number of fish of five species in a live-car (in contact with river water but isolated from sediments) for 14 days in the Hudson River below Fort Edward, N.Y. Whole-body residues of Aroclor 1016 increased to 1.8-3.8 ppm during the 14-day period of exposure. Veith et al. calculated the average concentration of Aroclor 1016 in the river to be 100-170 ppt, and hence estimated bio-accumulation factors in their test fish to be 10-40,000, comparable to those observed in the laboratory (358). However: (a) PCB levels in wild fish in the Hudson River have been up to 100 or more times higher than those observed in the test fish (Appendix C, 359); (b) Veith et al. calculated the PCB concentration in the river from the known discharges and the river flow: adsorption to the sediments is likely to have reduced the actual concentration considerably.

The Great Lakes. Extensive data are available on PCBs in water, sediments, and biota in the Great Lakes (Appendix C, pp. 31-54; 93, 360-368). Estimates of the degree of bio-accumulation are hampered by the difficulty of measuring concentrations of PCBs in water in the low parts per trillion characteristic of the open water of the lakes. The best set of data obtained by modern sampling techniques (use of polyurethane foam plugs for extraction from 20 liter samples -- see ref. 56) is from Lake Superior, where the mean PCB level at Duluth was estimated at 0.8 parts per trillion (363). Corresponding mean levels in biota were estimated as 0.1 ppm in zooplankton, 0.25 ppm in sculpins, and 0.3-3.3 ppm in several other fish species (363).

One of the fish species with the highest residues in this survey was the lake trout (Salvelinus namaycush), with mean residues of 0.7 to 1.9 ppm at various sampling stations in western Lake Superior (363). Another survey of lake trout in western and northern Lake Superior reported residues in the range 2.7-13.8 ppm (mean 7.0 ppm) in the whole fish, and 7.6-62.8 ppm (mean 25.6 ppm) in the fat (extracted oil) (367). Canadian data (360) fall into the same ranges, with the lake trout showing generally the highest residue levels (Table IV.2. 16). These data indicate bio-accumulation factors in fish in the range 3×10^5 to 9×10^6 (3×10^7 when expressed on a fat basis).

In Lake Michigan the average concentration of PCBs in water has not been measured precisely but appears to be substantially less than 10 ppt (362, 368): levels measured in the intake water of municipalities along the Michigan shore were all, with one exception, below 10 ppt (368), and even the concentrations reported in major tributary rivers to the lake have been only in the range 10-65 ppt (Appendix C, p. 46). PCB levels measured in fish in Lake Michigan have been consistently in the range 2-20 ppm, wet weight (16-200 ppm, lipid weight) (361, 368; Appendix C, pp. 40-45). These figures indicate bio-accumulation factors ranging up to at least 2×10^6 in whole fish (2×10^7 in lipids).

In Lake Ontario concentrations of PCBs in water in the range 35-56 ppt were reported in 1972 (Appendix C, pp. 31-32), but the method of extraction and analysis was not stated and the reportedly high levels in water are only partly reflected in high levels in

Table IV.2.16 (from ref. 360)

Polychlorinated biphenyls in commercial fish and fishery products, (area 3)

Location	Major species	Landings[a] (pounds)	PCB (mean ppm)
Lake Superior (A)	chub	306,000	0.96 (8)
	lake herring	1,611,000	1.17 (10)
	lake trout	195,000	2.02 (37)
	whitefish	328,000	0.68 (11)
	smelt	788,000	0.35 (6)
	yellow perch	67,000	0.31 (5)
		3,295,000	
Lake Huron (B)	chub	671,000	2.09 (17)
	whitefish	950,000	0.95 (8)
	carp	56,000	1.55 (4)
	suckers	176,000	1.33 (12)
	yellow pickerel	269,000	0.61 (9)
	sheepshead (drum)	29,000	0.75 (2)
	coho salmon	noncommercial	5.11 (6)
		2,151,000	
Lake Erie	alewife	332,000	1.22 (14)
	rock bass	49,000	0.27 (2)
	carp	41,000	1.27 (7)
	yellow perch	12,190,000	0.19 (10)
	smelt	15,760,000	0.42 (21)
	yellow pickerel	234,000	1.16 (9)
	white bass	2,346,000	1.26 (28)
	catfish	88,000	2.04 (8)
	bullhead	109,000	0.26 (4)
	sheepshead (drum)	354,000	0.74 (15)
	coho salmon	noncommercial	3.14 (8)
		31,503,000	
Lake Ontario (D)	bullhead	248,000	0.73 (12)
	yellow perch	699,000	1.23 (10)
	smelt	103,000	4.16 (17)
	white perch	290,000	1.84 (22)
	sunfish	203,000	0.74 (6)
	carp	395,000	1.69 (10)
	rock bass	42,000	1.76 (18)
	eel	222,000	17.14 (49)
	coho salmon	noncommercial	4.97 (3)
		2,205,000	

[a]Landings less than 25,000 pounds per species are not included.

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biota. Average PCB levels in fish are similar or slightly higher than those in Lake Michigan, in the range 4-30 ppm (360). Taking the water data at face value, these indicate bio-accumulation factors in the range 10^5 -10^6, somewhat lower than those in the same fish in Lakes Michigan and Superior.

Data on eggs of fish-eating birds from the Great Lakes indicate further bio-magnification of PCB residues by the birds. Table IV.2.17 summarizes PCB residues in eggs of Herring Gulls (Larus argentatus) from the Great Lakes (366). The geographical pattern of contamination generally parallels that found in the fish (Table IV.2.17). Taking Lake Superior as an example, the mean PCB level of 60 ppm in gull eggs (roughly 1000 ppm on a lipid basis) is much greater than that of 0.3-7 ppm in fish (5-40 ppm in lipids) (363). This suggests a degree of bio-magnification by the birds much greater than anything reported in the laboratory (Section IV.2.5).

The California Current. Young et al. (12, 369-370) have presented extensive data on PCB residues in the marine environment off southern California. Residues in inshore areas are variable because of localized sources, and hence are difficult to use to derive field estimates of bio-accumulation. However, the maximum water concentration measured at the surface above one sewage outfall was 4 ppt (370, p. 332); corresponding tissue levels in fish, crabs, and mussels ranged up to 4.3, 3.8 and 1.7 ppm respectively, indicating bio-accumulation factors of the order of 10^6.

Young et al. also presented measurements of the background concentrations of PCBs in the California Current which flows into

Table IV.2.17 (from ref. 366)

Organochlorine residue levels[a] in Herring Gull eggs in the Great Lakes[b]

	n	DDE	DDD	DDT	Dieldrin	Heptachlor Epoxide	Mirex	Hexachloro-benzene	PCB[c]	Hg
Lake Ontario	39	22.6 (8.8-35.1)	0.09 (trace-0.83)	0.09 (0.02-1.04)	0.37 (0.08-1.08)	0.12 (0.01-0.36)	5.06 (1.95-18.6)	0.19 (0.01-0.72)	142 (73.8-261)	0.51 (0.29-1.47)
Lake Erie	42	7.04 (3.8-14.3)	0.08 (trace-0.24)	0.04 (0.01-0.15)	0.30 (0.10-0.69)	0.14 (0.04-0.28)	0.31 (0.14-2.19)	0.11 (0.06-0.31)	65.8 (41.2-110)	0.22 (0.11-0.35)
Lake Huron	40	13.8 (5.4-41.9)	0.10 (trace-0.38)	0.08 (0.01-0.32)	0.41 (0.13-0.87)	0.12 (0.04-0.26)	0.56 (0.06-6.92)	0.14 (0.05-0.42)	51.5 (15.4-118)	0.23 (0.11-0.50
Lake Superior	39	18.6 (8.6-47.1)	0.15 (trace-0.4)	0.12 (0.02-0.58)	0.39 (0.13-1.35)	0.14 (0.07-0.38)	0.66 (0.2-5.17)	0.11 (0.02-0.33)	60 (33.4-148)	0.39 (0.16-0.63)
Lake Michigan	10	31.8 (15.8-145)	0.01 (0.01-0.07)	0.13 (0.07-0.39)	0.48 (0.3-0.92)	0.16 (0.11-0.60)	<0.01	0.04 (0.02-0.14)	91.3 (55.1-395)	n.d.

[a]Median Values and ranges (in brackets) in parts per million, wet weight.
[b]Eggs were collected from two colonies in each lake in both 1974 and 1975 except from Lake Michigan where they were from a single colony in 1975.
[c]Polychlorinated biphenyl values are based on a 1:1 mixture of Aroclor 1260:1254.
n.d.Not detected.

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the area from the northwest: these fell into the range 0.27-0.49 ppt with a mean of 0.40 ppt (369, p. 29). Away from the influence of local sources of PCBs, mean residues in mussels and dover sole were in the ranges 14-26 ppb and 40-50 ppb respectively. Referred to the reported background level in the water these indicate bio-accumulation factors of the order of 5×10^4 and 10^5 respectively. However, Risebrough et al. (56, pp. 12-20) have presented evidence that the PCB levels reported in water by Young et al. are at least 10 times too high; if so, these estimates of bio-accumulation factors would be at least 10 times too low.

Reported levels in birds and mammals in the California current and the outer islands are much higher than those reported in mussels and fish: 0.15-2 ppm in whole bodies of various fish-eating birds (371); 10 ppm in the whole bodies (200 ppm in lipids) in plankton-eating birds (225, 371); 20-400 ppm in the egg-lipids of fish-eating birds (372, 373); 12-145 ppm in the fat and 0.5-9.7 ppm in the livers of sea-lions (374). These figures indicate a substantial degree of bio-magnification by the vertebrates and an overall storage ratio (ppm in fat/ppm in ocean water) exceeding 10^9 in some cases.

Despite the variability in residue levels near to major sources of PCBs, the Southern California studies provide useful information about differential bio-accumulation of PCB components. At one of the largest sources, a sewage effluent at Palos Verdes, about 66% of the PCBs discharged during 1974-75 consisted of components characteristic of Aroclor 1242 (tetra- and tri-CBs): the remainder

consisted of components characteristic of Aroclor 1254 (penta- and hexa-CBs) (369: Table 1). The corresponding ratio in the tissues of fish (dover sole) sampled at the same place was reversed (67% Aroclor 1254) (370: Table 3). This suggests that in these circumstances penta- and hexa-CBs were accumulated roughly 4 times more efficiently by fish than tetra- and tri-CBs.

The North Atlantic Ocean. Several authors have published measurements of the order of 1 ppt or higher for concentrations of

PCBs in seawater in the open Atlantic Ocean (52, 53, 375, 376), but the problems of sampling without shipboard contamination are very severe and Risebrough et al. (56) have given evidence that all the figures are much too high. Certainly 1 ppt is an upper limit for levels to be expected in ocean water.*

Discounting data from locally polluted inshore waters, PCB residues reported from biota samples from the open Atlantic Ocean include the following:

	PCB residues (ppm)		
Organism	Whole body or muscle	Lipid	Ref.
Zooplankton	0.01 - 0.12	0.1 - 9.8	378
"	0.1 - 0.45	0.3 - 260	379
Fish	0.002 - 2.1	0.4 - 31	376
"	0.008 - 0.1	0.2 - 3	380
"	0.07 - 2.65		360
Deep-sea fish		4 - 12.5	56
"	0.001 - 0.17	0.04 - 38	376
Oceanic birds	0.2 - 21	0.25 - 74	387
Seabird eggs (Iceland)	0.4 - 27	4 - 320	380
Seals		2 - 75	386

Despite the wide variability (and some problems in sampling plankton -- see ref. 56) these data indicate that PCB residues in plankton and fish often reach 0.1 ppm in the whole body and 1 or even 10 ppm in lipids. Even accepting the 1 ppt levels reported in seawater, these figures indicate bio-accumulation factors of the order of at least 10^5 (10^6 - 10^7 in lipids). Plankton-eating and fish-eating birds and mammals often have residues an order of magnitude higher

* The first report of levels up to 150 ppt (375) has been questioned (377) and is inconsistent even with subsequent reports of around 1 ppt, which are themselves questionable.

still. Levels were highest of all in predators (up to 88 ppm in muscle and 535 ppm in fat in Glaucous Gulls (Larus hyperboreus) at Bear Island -- ref. 385).

The data cited above show that in natural environments animals generally bio-accumulate and bio-magnify PCBs by factors substantially higher than those usually measured in laboratory experiments. Despite the difficulties in measuring PCBs in water at levels near or below 1 ppt, there is now evidence from several areas that aquatic invertebrates and fish can bio-accumulate them by factors as high as several million fold from ambient water, and that fish-eating birds and mammals can further bio-magnify these concentrations by factors of 10-100. Reasons for this enhanced magnification in natural environments have been discussed by Nisbet (336). One likely reason is that PCB residues in many organisms increase with age (see Figure IV.2.18 for an example involving lake trout). Hence long-lived wild animals have an opportunity to accumulate PCBs to much higher levels than individuals exposed experimentally for limited periods in the laboratory.* Other reasons proposed for this field effect include selective exposure in a patchily contaminated environment, and selective predation on contaminated prey (336). Whatever the explanation for the phenomenon, the bio-accumulation factors observed in the field are those that are relevant to assessment of exposure to organisms that may eat the contaminated fish.

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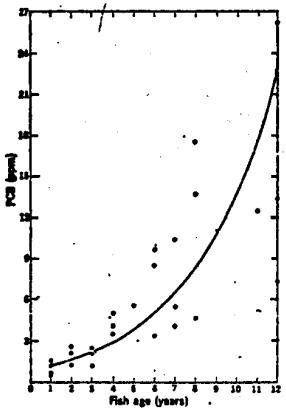
* See also ref. 405 for an example involving seals, whose body burdens were still increasing after 4 years in captivity (see p. 326 above).



The concentration of PCB's in Cayuga Lake trout as a function of age.

Figure IV.2.18 (from ref. 381)

IV.3. Presence in the Aquatic Environment

A review of PCB levels in the environment is appended to this document as Appendix C and only a brief summary is included here. PCBs are widely distributed in the environment of North America. Data gathered from nationwide monitoring activities by the U.S. Geological Survey (48) show mean concentrations of PCBs in the range 0.01 to 0.05 µg/l (10-50 ppt) in unfiltered water samples and from 0.25 - 218 ppb in bottom sediments. The highest levels were found in the Southeast (South Atlantic Slope and Eastern Gulf of Mexico drainage basins) and the Northeast (North Atlantic Slope and St. Lawrence River drainage basins). (See Appendix C, pp. 3-15).

In the National Pesticide Monitoring Program, PCBs have been detected in fish at all of the 100 monitoring stations, although the percentage of composites with detectable residues fell from 100% to 51% between 1969 and 1973 (Appendix C, p. 27). In the same period the number of composite samples of fish with residues greater than 5 ppm fell from 61% to 40% (ibid.). Generally, the higher concentrations were found in certain river systems with industrial activity, including the Allegheny, Kanasha, Cumberland, Tennessee, Ohio, Mississippi and Missouri Rivers (93). High residues have also been found in fish in the Great Lakes, in the Hudson and other northeastern rivers, and in a number of estuaries and inshore waters (Appendix C, pp. 31-87; 2, 93, 359-364, 367-370, etc.). PCB residues in fish detected in monitoring programs include chlorobiphenyls characteristic of Aroclors 1242 and 1248 (tri- and tetra-CBs) as well as those

characteristic of Aroclors 1254 and 1260 (penta-, hexa-, and hepta-CBs (93, 370, 367, 382). For example, in the crosscheck samples analyzed for the National Pesticide Monitoring Program, 1970-73, 26 of 131 samples listed contained components characteristic of Aroclors 1232 or 1242, and 70 had components characteristic of Aroclor 1248; all had components characteristic of Aroclors 1254 and 1260 (382). In lake trout samples in Lake Superior, more than half the PCB residue components were listed as matching Aroclors 1242 and 1248 (367). Similarly, numerous tetrachlorobiphenyls and at least one trichlorobiphenyl component were identified in lake trout and coho salmon from Lake Michigan (95). Thus tri- and tetra-CBs as well as higher CBs are widely stored in fish in the United States.

PCBs are also found very widely and often at high levels in fish-eating birds (1, 2, 56, 93, 225, 364-366, 371-373) and fish-eating mammals (374, 383-386). Levels are particularly high in the fat (blubber) of marine mammals such as seals, dolphins and polar bears (383-386; see Table IV.3.1).

PCBs are widely distributed in human tissues, reflecting general intake in the diet (351-356).

Table IV.3.1 (from ref. 383)

TOTAL DDT PLUS PCBs IN BLUBBER OF HARBOR SEALS COLLECTED IN 1971

LOCATION	NO. OF SEALS	MONTH COLLECTED	AGE, YEARS	TOTAL DDT & PCBs (mg/kg, wet wt.) MEAN	INDIVIDUAL VALUES
Washington: Puget Sound	2	June-August	1	862.7	459.4; 1,620.0
California: San Miguel Island	5	June	9-30+	610.7	380.7; 318.5; 425.5; 518.3; 2,350.0
Oregon: Columbia River	3	May	2-4	62.5	27.7; 80.4; 109.9
Alaska: Pribilof Islands	3	August	(a.)	11.3	6.8; 7.1; 27.8

a. canine teeth lost

IV,4. Effects on Biota and Natural Ecosystems

Effects of PCBs on organisms and ecosystems fall into two categories: (a) effects observed in the laboratory under controlled conditions of exposure to PCBs; (b) effects observed in natural systems and associated with PCB exposure by epidemiological or equivalent methods.

Effects of type (a) are relatively easy to extrapolate to natural ecosystems: if an effect is observed in the laboratory when test organisms are exposed to a certain level of PCBs, it is reasonable to assume that similar effects will take place in the field if the same or comparable species are exposed to the same level of PCBs. It is often likely that effects will occur at lower levels in the field, for the following reasons:

(i) natural systems involve many species and it is likely that some will be more sensitive to PCBs than the species chosen for testing;

(ii) under natural conditions organisms are exposed intermittently to environmental stresses (temperature, pH, oxygen depletion, food shortage, predation, etc.) which generally make them more vulnerable to the effects of an additional toxic stress;

(iii) natural systems are exposed to other pollutants and it is known that additive and and synergistic toxic effects may occur.

Thus it is reasonable to expect adverse effects to occur in the field at lower exposure levels than in the laboratory, but it is difficult to predict the lowest hazardous levels without actual field studies.

Effects of type (b), however, which depend on field studies, are often difficult to interpret or to establish with reasonable certainty. When an adverse effect is observed in the field, it is often associated with exposure to more than one pollutant: it is difficult to establish whether it is caused by PCBs, by another pollutant, or by both acting together. Furthermore, PCBs are patchily distributed in the environment, so without extensive measurements of concentrations in water it is difficult to establish the exact degree of exposure of the affected organisms.

Accordingly to judge the effects of PCBs on natural systems it is necessary to weigh laboratory and field evidence together. ✓

IV.4.1. Effects Observed in the Laboratory

The following effects of PCBs have been observed in controlled experiments at exposure levels which occur locally in the field:

(i) Change in species composition of phytoplankton communities (Section III.3). Effects on the growth and rate of cell division of sensitive species of phytoplankton have been noted at PCB concentrations as low as 0.1 ppb (84, 86) and large effects on the species composition of mixed cultures took place at this concentration (92). The effects of lower concentrations have not been studied. The changes involve replacement of sensitive diatoms by resistant green

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algae and are expected to have substantial effects on consumers and hence on food-webs (83). Synergistic effects with DDE are also involved (90).

(ii) Change in species composition of marine animal communities. Effects on species composition and biological diversity of communities of estuarine animals developing in the laboratory from planktonic larvae in flowing seawater were noted at PCB concentrations as low as 0.1 ppb, the lowest concentration tested (ref. 109; Section III.4.2).

The results reported in these experiments (refs. 88, 92, 109) are of particular importance because they demonstrate that PCBs affect mixed communities of plants and animals at concentrations much lower than those required to show effects on single species populations.

(iii) Effects on aquatic invertebrates (Section III.4). Reproduction and growth is impaired in aquatic invertebrates at concentrations lower than those required to cause mortality. The waterflea Daphnia magna and the midge Tanytarsus dissimilis are both affected at levels between 0.4 and 0.5 ppb (93: Table III.4.1). Synergistic effects with DDT are also involved (94).

(iv) Sublethal effects on fish (Section III.5). Although there is little evidence that PCBs affect survival of fish at concentrations below 1 ppb, adverse effects on reproduction have been noted at concentrations as low as 0.1 - 0.4 ppb, corresponding to residues of 5 ppm in eggs (113, 114, 115). Effects were also noted

on thyroid function in fish exposed to a dietary level of 0.48 ppm PCBs (which corresponds to roughly 0.4 ppm whole-body residue) (refs. 73, 95; Figure III.5.2).

(v) Effects on reproduction in birds (Section III.6.4). Marked effects on reproductive success (especially embryonic mortality) have been noted in chickens at dietary levels as low as 8-10 ppm of PCBs, corresponding to residues of only 1-2 ppm in whole eggs. It is doubtful whether the chicken is a good model for wild birds, since other species have proved less sensitive in laboratory tests. However, second generation effects and synergism with DDE have been noted in other species at dietary levels of 10 ppm (161, 164).

(vi) Hepatic porphyria in birds (Section IV.6.3). PCBs induced ALA-synthetase and caused porphyria in quail at doses as low as 1 mg/kg/day and probably at 0.1 mg/kg/day. These effects were associated with residues as low as 1 ppm in the liver (145: Figure III.6.1).

(vii) Effects on mink (Section III.9.4). Mortality and total reproductive failure took place in mink fed a diet containing only 0.64 ppm of PCB residues, the lowest dose tested (201). In another experiment at a higher dose level, there was evidence of synergistic effects with DDT and dieldrin (200: Table III.7.5). The mink is the only fish-eating mammal whose sensitivity to PCBs has been tested.

All these effects have been noted at exposure levels which occur quite frequently in natural environments. Concentrations of 0.1 ppb are reported not infrequently from polluted fresh waters

(48; Appendix C), and concentrations as high as 0.4 ppb are reported occasionally. Fish throughout the United States have tissue residues as high as those associated with thyroid malfunctions (Appendix C; 93). Fish-eating birds often are exposed to fish with PCB residues as high as 8-10 ppm (Section IV.3); many, perhaps most, fish-eating birds in the United States have residues in their eggs as high as the 1-2 ppm that affect hatchability in chickens (Section IV.3.; 1, 2, 56, 225, 364-366, 371-373). Many fish-eating birds, even from remote parts of the Atlantic Ocean, have PCB residues in the liver as high as 1 ppm (140, 387, 388). Fish-eating mammals in the Great Lakes and many other areas of the United States will be exposed to diets containing 0.64 ppm of PCBs (Section IV.3; 93). Thus it is reasonable to suppose that at least some of the adverse effects reported in the laboratory are taking place in natural environments.

IV.4.2. Effects Observed in the Field.

Several adverse effects on wild animal populations have been reported which have been associated reasonably plausibly with exposure to PCBs.

(i) Effects on reproduction in salmon. Swedish investigators have reported a statistically significant association between hatching failure in eggs of Atlantic salmon and PCB residues. Concentrations in the range 0.4-1.9 ppm, wet weight (7.7-34 ppm, lipid weight) were associated with mortalities between 16 and 100 percent (118). This report was published only as a preliminary note and complete details are not available.

(ii) Reproductive failure in other fish. Several species of fish in Lake Michigan have not been reproducing effectively in recent years (409-410). Despite annual stocking of lake trout since 1965, for example, no wild-raised young have been collected (409). Eggs of Atlantic salmon and northern pike have shown very low hatchability in the laboratory (410). These reproductive failures have been associated circumstantially with residues of PCBs and other chemicals, but no clear link has yet been established.

(iii) Fin rot syndrome in marine fish. "Fin rot syndrome" has been induced in the laboratory in spot exposed to 3-5 µg/l of Aroclor 1254 (106). The syndrome appears identical to that observed in wild fish (croakers and spot) which died under conditions of warm weather and oxygen depletion in Escambia Bay, Florida (106). Although the deaths of the wild fish were attributed originally to lack of oxygen in the water (99), the levels of PCBs in the dying fish (around 10 ppm) were similar to those in fish experimentally poisoned in the laboratory (99, 111). This suggests that PCBs may have played at least a contributory role in the incidents.

In independent observations, McDermott et al. (370) have reported that fin erosion has recently become prevalent in dover sole off Palos Verdes and Orange County, California. Diseased sole have higher PCB residues than healthy fish (median concentration 2 ppm, wet weight, versus 1 ppm), but the difference was not quite statistically significant ($P = 0.1$). The syndrome was associated with concentrations of PCBs in water not greater than 4 ppt (370, p. 32).

(iv) Deaths of fish-eating birds. Koeman (142) reported that a number of Cormorants (Phalacrocorax carbo) found dead in the Netherlands in 1970 contained high residues of PCBs (mean 190 ppm in brain, 320 ppm in liver, wet weight). These concentrations were similar to those in Cormorants experimentally poisoned with PCBs (76-180 ppm in brain, 210-290 ppm in liver) and Koeman attributed the deaths of the wild birds primarily to PCB poisoning (142). However, the corresponding levels of PCBs in the fish and water were not known.

In November 1969, more than 50,000 seabirds, mostly murres (Uria aalge), were found dead in the Irish Sea. The most consistent findings at post mortem were emaciation, liver damage, and high residues of PCBs in the liver (2-880 ppm, wet weight) (389). Birds collected in apparent good health at the same time and place had body burdens similar to those of the dead ~~birds, but~~ in the healthy birds the PCBs were stored in the fat, whereas in the dead birds the PCBs were largely in the liver. It therefore seems likely that food shortage triggered the incident, but since the liver residues of PCBs in some birds were well into the lethal range (142) it seems likely that some of the birds actually died from PCB poisoning. This is an important example of the interaction of environmental and toxic stresses. Residues in fish in the same area were in the range 0.01-2.6 ppm (389).

Glaucous Gulls (Larus hyperboreus) which had been feeding on the eggs of seabirds at Bear Island in the Arctic Ocean had high levels of PCBs in the liver (mean 24 ppm). One individual found

dying with failure of coordination of the limbs had 311 ppm PCBs in the liver (388) and is therefore likely to have succumbed to PCB poisoning (140, 142). The example is important because the PCBs were apparently acquired through the natural food chains of the North Atlantic and Arctic Oceans (387, 388).

Two Bald Eagles (Haliaetus leucocephalus) have been reported dying with high residues of PCBs in the brain. One found dead in Michigan in 1970 had 235 ppm of PCBs and 385 ppm of DDE in the brain (390). Another found dead in Michigan in 1972 had 190 ppm of PCBs and 55 ppm of DDE in the brain and 1200 ppm of PCBs in the whole body: the immediate cause of death was drowning, as it was seen to fall off a perch into the water (391). In each case the residues of ~~both~~ PCBs in the brain were in the lethal range (142, 391) and in the first case residues of DDE were also: it seems likely that the birds died from the combined effects of PCBs and DDE poisoning. The first bird was from the Lake Michigan area (Grand Traverse county); the second bird was probably from southeastern Michigan. The Bald Eagle is an endangered species and is subject to the special provisions of the Endangered Species Act of 1973.

(v) Effects on reproduction in fish-eating birds. Herring Gulls are experiencing low reproductive success at colonies in Lake Ontario, but appear to be breeding normally at colonies in the other Great Lakes, including Lake Michigan (366, 392, 393). The breeding failure is characterized by early embryonic mortality (393) and is associated with high levels of PCBs in the eggs (Table IV.2.17).

Recent studies have shown that the embryonic mortality is associated with porphyria and edema (subcutaneous, pericardial, and abdominal), and that these conditions are statistically associated with high PCB levels (406). In addition, deformities of the feet, toes, and eyes have been recorded in chicks of Herring Gulls and three other fish-eating species in Lake Ontario (407).

In studies of other species of fish-eating birds, reproductive failure and eggshell-thinning have been associated primarily with residues of DDE (56, 364, 372, 373, 394) and no other study has suggested an independent effect of PCBs. However, only a few species have been studied to date.

(vi) Premature births in sea-lions. Premature pupping in sea-lions has been noted on San Miguel and San Nicholas Islands, California, since 1968. Most or all of the premature pups die (374). Residues of PCBs were much higher in the females giving birth to premature pups than in those of females giving birth at full term

(means 112 ppm and 17.1 ppm PCBs in blubber, respectively). However, residues of DDT compounds, primarily DDE, were also elevated in the females giving birth prematurely (means 824 vs. 103 ppm in blubber). Fish in this area have residues generally in the range 0.01-2 ppm PCBs, ranging up to 4-6 ppm in the vicinity of sewage outfalls (370).

In all cases summarized above, the association between PCB levels and adverse effects in wild animals is somewhat circumstantial: this is inevitable in an epidemiological type of investigation. However, in each case the suspected adverse effect can be matched with adverse effects noted in laboratory animals with comparable residue levels. In several of the cases the adverse effects were also associated with high levels of DDE, and additive or synergistic effects seem likely. The interaction of environmental and toxic stresses is also apparent in several cases. Together these examples seem sufficient to show that current levels of environmental contamination with PCBs are having adverse effects on natural ecosystems, but the ambient concentrations at which they do so has not been established.

IV.5. Potential Effects in the Human Population.

As detailed in Section III, several different toxic effects have been observed in experimental animals exposed to PCBs in the diet at levels as low as 0.5-2.5 parts per million. Surveys by the U.S. Food and Drug Administration suggest that the average level of PCBs in the human diet in the United States is now about 2.2 parts per billion (Section IV.2.7 above). There is thus a factor of 200-1000 between the average level in the human diet and levels known

to cause effects in experimental animals.

In addition, residues of PCBs are sporadically distributed in the food supply and some individuals ingest much more than the average: persons with dietary preference for fresh-water fish and breast-fed infants have been identified as high-risk groups (2, 331).

It is not yet possible to use epidemiological studies to estimate safe levels of intake, for the following reasons:

(i) Epidemiological studies in the general population are not being conducted, and in any case would be difficult or impossible because of the widespread exposure of the general public.

(ii) The Yusho incident, although providing some dose-response information, is confused by the high level of PCDFs in the ingested PCBs.

(iii) Occupational experience in the past has provided no quantitative dose-response information and in any case may be confused by effects of PCDFs in older formulations.

(iv) Current studies of occupationally exposed workers are still in progress and in any case will provide information only about adults.

V. Criteria Formulation

The criterion level for PCB's is 0.001 ug/l PCB's. It should be noted that if all PCB entry into the environment were to stop immediately, the environmental burden would in some waters still exceed the ambient water quality criterion. While EPA is attempting to prevent the continuing discharge of PCB's, or at least to minimize the continuing discharge of PCB's into the aquatic environment, the responsiblity for protecting man from contaminated interstate commercial food products is with the Food and Drug Administration of the Department of Health, Education, and Welfare.

The chronic effects of PCB's in man may occur at extremely low concentrations. Although it becomes virtually impossible to state with confidence that any PCB concentration above zero provides an ample margin of safety for man, the PCB criterion number is believed to provide protection for the aquatic environment.

The criterion for PCB's is derived with consideration of the bioaccumulation potential rather than solely upon the more traditional use of an application or safety factor applied to an acute toxic value. The reasons for this approach are the environmental persistence and bioaccumulation potential of PCB's as outlined below:

The persistence of PCB's in the environment is such that they present long-term environmental hazards.

The laboratory determination of the bioaccumulation potential for PCB's in general has been shown to be around 274,000 times the PCB level in the test water. There have been reports that certain animals captured from the aquatic environment have apparently bioaccumulated PCB's in their flesh to levels of between 3 and 10 million times that of the water concentration in which the animals were captured. It is important to note, however, that the level of PCB contamination in the food of the captured aquatic life, as well as the concentrations of PCB's in which these organisms resided for the predominance of their life periods, are unknown. Thus, the actual bioaccumulation potential of PCB's in the aquatic environment is uncertain. Therefore, the bioaccumulation potential demonstrated in the laboratory of 274,000 is the number which is used in determining a criterion level.

The demonstrated physiological effects of PCB's on fish and consumers of fish and wildlife, which include reproductive failures in a broad phylogenetic group of aquatic and terrestrial animals, liver enlargement, enzyme induction and skin lesions, require the consideration of parameters beyond the usual acute toxicity and safety factors.

Data which support a one part per trillion (0.001 ug/l) criterion are:

0.1 ug/l will cause population shifts in phytoplankton. It is important to note that at this level of the food web bioaccumulation does not play a role in the adverse effect. However, this concentration is extremely low and does have significant ecosystem consequences since shifts in population at this level will have an impact on the food for top predators.

0.4 to 0.5 ug/l lowers the reproductive potential of some invertebrates.

0.1 ug/l will lower the species diversity index of some invertebrates.

Bioaccumulation has been demonstrated in the laboratory at concentrations of around 274,000 times the water level.

PCB contaminated fish will cause detrimental effects on consumers of fish.

At 0.64 ppm (milligrams per kilogram in the food), total reproductive failure was observed in mink.

At 3.57 ppm (milligrams per kilogram in the food), mink died.

At 2.5 ppm (milligrams per kilogram in the food), reproductive dysfunctions occurred in the rhesus monkey.

At 3 ppm (milligrams per kilogram in the food), some mortality was observed in the rhesus monkey.

In rats, 5 ppm (milligrams per kilogram in the food) resulted in the induction of enzymes in the liver.

At levels of between 40 and 300 ppm PCB's in chicken feed, deaths of chicken occurred. Reproductive failures occurred at between 8 and 10 ppm PCB's in the food.

Ulceration in the stomachs of dogs occurred after long-term feeding at 1 ppm PCB in feed.

Based upon the highest bioaccumulation factors demonstrated in the laboratory, a water criterion of no more than 0.001 ug/l (1 part per trillion) should provide protection to the aquatic ecosystem, and should provide a margin of safety to the consumers of aquatic life.

Adverse effects in man that are due to PCB or polychlorinated dibenzofurans (PCDF) contamination have included: chloroacene, swelling of the eyes, and liver involvement. There was no quantification of the levels of PCB's ingested by those individuals affected by PCB's. The carcinogenic threat due to PCB's has not been conclusively demonstrated, although based upon interpretation

of data, some researchers have concluded that PCB's have resulted in cancer in the mouse and thus they represent a threat to man.

Based upon the proven bioaccumulation potential of 274,000 times the ambient water concentration in controlled conditions, the level of 0.001 ug/l PCB's should afford protection for consumers whose sole diet consists of aquatic organisms contaminated at the worst or maximal level predicted by the laboratory data.