EPA - 560/7-76-001

Appendix C

REVIEW OF PCB LEVELS IN THE ENVIRONMENT

JANUARY 1976

OFFICE OF TOXIC SUBSTANCES
ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

NOTE: Appendices A and B are copyrighted materials and therefore have been omitted.

MONS 063402

EPA 560/7-76-003

REVIEW OF PCB LEVELS IN THE ENVIRONMENT

Prepared by

Office of Toxic Substances
Environmental Protection Agency
Washington, D.C. 20460

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1.0 Introduction

Polychlorinated biphenyls (PCB's) were first manufactured commercially in 1929. They are currently manufactured in the United States, Great Britain, France, Germany, USSR, Spain, Italy and Czechoslovakia. The sole producer of PCB's in the United States is the Monsanto Industrial Chemicals Company, where products are marketed under the trade name "Aroclor". Various Aroclor products that have been marketed include Aroclor 1221, 1232, 1242, 1248, 1254, 1260, 1262 and 1268. The latter two digits designate the percent chlorine in each formulation. Aroclor 1016 is a new product which has 41% chlorine. Since their introduction, PCB's have been used in a variety of commercial and industrial products such as transformers, capacitors, paints, inks, paper, plastics, adhesives, sealants and hydraulic fluids. Through this widening product usage and their resistance to degradation, PCB's have been identified in the environment since 1966 with increasing frequency.

On September 1, 1971 an interdepartmental task force was established in the United States to review the data linking PCB's with adverse health and environmental effects and "to coordinate a governmentwide investigation into PCB contamination of food and other products". The task force concluded in its report of May 1972 that PCB's were highly persistent, could bioaccumulate to relatively high levels in fish and could have serious adverse effects on human health. It recommended the discontinuance of all PCB uses except in closed electrical systems. Since the 1972 Task Force Report, environmental sampling and laboratory studies have indicated that PCB's were a more serious and continuing environmental and health threat than had been originally realized. These concerns were highlighted this past November at the National Conference on Polychlorinated Biphenyls held in Chicago, Illinois.

This study reviews the current PCB data base to assess the PCB levels in the environment on a national level; the full spectrum of PCB levels reported in man and the environment were of interest. Data were obtained from a number of national monitoring programs, the literature and many unpublished reports. The data examined was inclusive to December 1, 1975.

It should be stressed at the outset, that due to the complexity and difficulty of PCB identification and measurement, that levels reported are not really comparable between different investigators. This aspect could not be compensated for or identified in the data presented. In addition, some of the Aroclor identifications reported may only have been best estimates of the pattern observed and may not have really identified the true Aroclor discharged or the quantitative level of the polychlorinated biphenyl found in the sample. However, the levels reported do give an indication of the contamination levels in different media for a given locality.

Recognizing the faults of combining different data bases, the sheer mass of data supports the conclusion that there is widespread contamination of the environment by PCB's. There are regional variations, but effects are consistent across all media (e.g., water, sediment, fish, birds), generally showing greater concentrations of PCB's in highly developed areas and in areas of industrial activity. Over the years examined, the situation has shown no improvement nationally.

This report is divided into five major sections:

PCB's in the environment

Behavior in the environment

Occurrence in food

Bioaccumulation in man

Environmental trends

Wherever possible, the level of data reported has been greatly reduced over that originally reported, especially for the older studies, however every effort has been made to indicate the locality and the level of contamination originally reported. More recent studies are presented in greater detail. Trend assessments, cross media comparisons and conclusions are drawn where justified.

2.0 PCB Levels in the Environment

2.1 Data from National Monitoring Programs

2.1.1 Water

PCB monitoring in water is an activity of the overall National Pesticides Monitoring Program operated by the U.S. Geological Survey. Analysis of the PCB data in EPA's STORET water quality file has provided little information, although scattered whole water measurements have been taken by a number of states over the years. Those states reporting nonzero readings are few in relation to the number of states showing zero concentrations.

At the direction of various local, state and federal agencies, the USGS collects and analyzes water data, producing a data file of uniform integrity. Current data from this file[1] covering the period October 1972 to 1975 provides the best available national data and the basis for the following analyses.

PCB levels, gathered by the USGS during the period January 1971 to June 1972 were reported by Crump-Wiesner, Feltz and Yates[2]. While the data suffers from the lack of representative sampling within states and multiple samples from the same locations, they concluded that significant concentrations of PCBs were widespread in the water resources of the nation. Their published summary of PCB residue data for surface and ground waters has been updated with the 1972-1975 data and is presented in Table 2.1-1.

The preponderance of zero readings in whole water and the low levels of scattered non-zero readings could be masking widespread PCB contamination in the nation's waters. Zero readings are due to the low solubility of PCBs and the analytical procedure commonly used which limits detection to 0.1 ppb.

There is a disparity between whole water and bottom deposit measurements. In almost all cases in which samples of both whole water and bottom sediment were taken simultaneously and the latter reading was non-zero, the whole water concentration was measured at zero ppb This occurred even when the concentration in bottom sediment was as high as 4000 ppb[6]. More recent water studies[3] to the ppt level have consistently shown measureable PCB levels.

Table 2.1-1
Summary of PCB Residue Data for Surface and Ground Waters, January 1971-(partial) 1975

State	Year	No. of Samples	Occurences	Concentration (ppb)	Median Concentration (ppb)
Alaska	71	3	0	——	——
	73	-	-	——	——
Alabama	73	4	0	——	——
	74	2	0	——	——
Arizona	71	8	0	——	——
	72	8	0	——	——
	73	38	0	——	——
	74	14	0	——	——
	75	4	0	——	——
Arkansas	71	32	0	——	——
	72	8	0	——	——
	73	28	1	0.2	——
	74	22	0	——	——
	75	1	0	——	——
California	71	161	2	0.1, 0.1	——
	72	27	0	——	——
	73	110	0	——	——
	74	99	2	0.1, 0.1	——
	75	42	0	——	——
Colorado	71	32	0	——	——
	72	3	0	——	——
	73	6	0	——	——
	74	9	0	——	——
Connecticut	71	13	6	0.1-0.2	0.1
	72	16	6	0.1	0.1
	73	45	1	0.1	——
Delaware	NO ACTIVITY				
D.C.	NO ACTIVITY				
Florida	72	16	0	——	——
	73	106	4	0.1-1.0	0.1
	74	60	0	——	——
	75	22	0	——	——

Table 2.1-1 (cont.)

State	Year	No. of Samples	Occurences	Concentration (ppb)	Median Concentration (ppb)
Georgia	73	1	0	——	——
	74	29	0	——	——
Hawaii	71	5	0	——	——
	72	1	0	——	——
	73	1	0	——	——
	74	1	0	——	——
Idaho	72	1	0	——	——
	73	1	0	——	——
	74	6	0	——	——
Illinois	74	1	0	——	——
	75	1	0	——	——
Indiana	74	1	0	——	——
Iowa	71	24	0	——	——
	72	1	0	——	——
	73	27	1	0.1	——
	74	2	0	——	——
Kansas	71	10	0	——	——
	72	4	0	——	——
	73	24	1	0.2	——
	74	3	0	——	——
Kentucky	71	7	0	——	——
Louisiana	71	9	0	——	——
	73	195	0	——	——
	74	147	0	——	——
	75	22	2	0.1-0.2	——
Maine	71	2	0	——	——
	72	1	0	——	——
	73	6	0	——	——
	74	1	0	——	——
Maryland	71	6	1	0.1	——
Massachusetts	71	5	1	0.2	——
	73	5	1	0.1	——
	74	6	1	0.1	——
	75	5	1	0.1	——

Table 2.1-1 (cont.)

State	Year	No. of Samples	Occurences	Concentration (ppb)	Median Concentration (ppb)
Michigan	71	2	0	——	——
	72	2	1	0.1	——
	73	6	0	——	——
	74	23	0	——	——
	75	18	0	——	——
Minnesota	71	3	2	0.1-0.3	——
	73	1	0	——	——
	74	1	0	——	——
Mississippi	71	8	0	——	——
	73	1	0	——	——
	74	52	0	——	——
	75	60	0	——	——
Missouri	71	21	0	——	——
	72	7	0	——	——
	73	19	0	——	——
Montana	71	47	0	——	——
	72	9	0	——	——
	73	32	0	——	——
	74	8	0	——	——
Nebraska	71	44	0	——	——
	72	3	0	——	——
	73	24	0	——	——
	74	17	0	——	——
	75	2	0	——	——
Nevada	72	4	0	——	——
	73	6	0	——	——
	74	5	0	——	——
New Hampshire	73	3	0	——	——
	74	3	0	——	——
New Jersey	71	11	3	0.1	0.1
	72	5	1	<0.5	——
	73	29	2	<0.1,<0.1	——
	74	29	0	——	——
New Mexico	71	36	0	——	——
	72	10	0	——	——
	73	5	0	——	——
	74	5	0	——	——

Table 2.1-1 (cont.)

State	Year	No. of Samples	Occurences	Concentration (ppb)	Median Concentration (ppb)
New York	71	325	52	0.1-4.0	0.3
	73	32	1	0.1	—
	74	16	0	—	—
North Carolina	72	3	0	—	—
	73	1	0	—	—
	74	1	0	—	—
North Dakota	71	40	0	—	—
	72	3	0	—	—
	73	4	0	—	—
	74	3	0	—	—
Ohio	72	14	2	0.1-M.2	—
	73	1	0	—	—
	74	3	0	—	—
Oklahoma	71	19	0	—	—
	72	5	0	—	—
	73	77	2	0.1-2.9	—
	74	37	5	0.1-0.2	0.1
	75	11	2	2.0-3.0	—
Oregon	71	13	0	—	—
	72	2	0	—	—
	73	5	0	—	—
	74	9	0	—	—
	75	1	0	—	—
Pennsylvania	71	2	0	—	—
	73	1	0	—	—
	74	25	0	—	—
	75	20	0	—	—
Rhode Islan	73	3	0	—	—
South Carolina	72	2	0	—	—
	73	2	0	—	—
	74	12	0	—	—
South Dakota	71	18	0	—	—
	72	1	0	—	—
	73	5	0	—	—
	74	1	0	—	—
Tennessee	73	1	0	—	—
	74	2	0	—	—

Table 2.1-1 (cont.)

State	Year	No. of Samples	Occurences	Concentration (ppb)	Median Concentration (ppb)
Texas	71	660	12	0.1-3.0	0.4
	72	82	1	0.1	———
	73	385	6	0.1-0.6	0.3
	74	297	4	0.2-0.7	0.3
Utah	72	1	0	———	———
	73	8	0	———	———
	74	16	0	———	———
Vermont	73	1	0	———	———
Virginia	71	4	1	0.1	———
	72	12	0	———	———
	73	5	0	———	———
	74	1	0	———	———
Washington	71	25	0	———	———
	72	1	0	———	———
	73	6	0	———	———
	74	6	1	0.1	———
West Virginia	71	4	0	———	———
	74	1	0	———	———
Wisconsin	71	3	0	———	———
	72	1	0	———	———
	73	12	0	———	———
	74	30	0	———	———
	75	2	0	———	———
Wyoming	71	18	0	———	———
	72	1	0	———	———
	73	6	0	———	———
	74	3	0	———	———
Puerto Rico	73	34	0	———	———
	74	63	1	0.8	———

E - estimated.

SOURCE: This table is an extension of Table 2 in Crump-Wiesner, J.S., H.R. Feltz and M.L. Yates, J. Research USGS, 1, 603-607; (1972) incorporating the newer data of PCB Data Base, October 1972 to (partial) 1975, The U.S. Geological Survey, Quality of Water Branch, Reston, VA.

2.1.2 Sediment

Nationwide sampling of bottom sediments have shown PCB concentrations to have widespread occurrence. Data from Crump-Wiesner, et. al.,[2] January 1971-June 1972 are summarized in Table 2.1-2. The October 1972 to 1975 USGS data are summarized in Table 2.1-3. Table 2.1-3 permits distinguishing between those states where the non-zero PCB readings were from one or a limited number of stations with multiple readings and those states where the non-zero readings come from a number of stations within the state. The map in Figure 2.1-1 indicates the distribution of PCB contamination from 1972-1974 based on the figures in the tables.

States which monitored sediment concentrations to any appreciable extent detected the presence of PCB's in concentrations often higher than 40 ppb. A greater proportion of states are reporting PCB's in bottom sediment each year, as displayed in Table 2.1-4.

Although the sediment data is meaningful, some caution is necessary in interpretation: measurements may be the result of past contamination, not present discharges; mean readings and ranges are somewhat inadequate to summarize the data - geographic locations are essential since readings show patterns only in particular water basins.

Table 2.1-2
Summary of PCB Residue Data for Bottom Sediments
January 1971 - June 1972

State	No. of Samples	Occurrences	Concentration (ppb)	Median Concentration (ppb)
Alaska	3	0	———	———
Arkansas	23	4	20-2,400	60
California	13	3	20-190	85
Connecticut	1	1	40	———
Hawaii	4	0	———	———
Georgia	12	10	10-1,300	300
Maryland	11	5	10-1,200	30
Mississippi	8	2	50;170	———
New Jersey	12	10	8-250	20
Oregon	4	2	15;140	———
Pennsylvania	16	11	10-50	20
South Carolina	11	8	30-200	50
Texas	293	23	7.9-290	80
Virginia	10	8	5-80	40
Washington	10	0	———	———
West Virginia	2	1	10	———

SOURCE: Crump-Wiesner, J.S., H.R. Feltz and M.L. Yates, J. Research USGS, 1, 603-607, (1972).

Table 2.1-3
Summary of PCB Residue Data for Bottom Sediments
Stations measuring/#Stations with at least one non zero reading
Range of non-zero readings (mean), in ppb

STATE	1972	1973	1974	(partial) 1975
Alabama	——	1/0	2/0	——
Alaska	——	1/0	——	——
Arizona	1/0	1/0	1/0	——
Arkansas	6/0	6/0	15/4 <2-29 (12.8)	——
California	1/0	15/0	18/2 <2-65 (33.5)	——
Colorado	1/0	1/0	1/0	——
Connecticut	16/8 <5-2800 (127.5)	16/11 <5-40 (14.1)	40/31 <2-350 (71.5)	——
Delaware		NO ACTIVITY		
Florida	27/7 <5-400 (93.6)	64/34 <2-600 (35.8)	68/16 <2-530 (43.3)	20/2 <140-430 (28
Georgia	——	1/0	9/7 <1-51 (13.6)	——
Hawaii	1/0	1/0	——	——
Idaho	1/0	1/0	1/0	——
Illinois	——	1/1 (20)	——	1/0
Indiana	——	——	4/2 <9-61 (35)	——
Iowa	——	2/0	——	——
Kansas	1/0	1/0	2/0	——
Kentucky		NO ACTIVITY		
Louisiana	——	3/0	18/0	4/1 (5)
Maine	1/0	1/0	——	——
Maryland		NO ACTIVITY		
Massachusetts		NO ACTIVITY		
Michigan	1/1 (<2)		13/8 <4-60 (17.6)	15/4 <10-58 (29.5)
Minnesota	——	1/0	1/0	——
Mississippi	——	1/0	50/13 <3-56 (16.4)	——
Missouri		NO ACTIVITY		
Montana	4/0	6/0	4/0	——
Nebraska	1/0	3/0	1/0	——
Nevada	3/0	3/0	3/0	——
New Hampshire		NO ACTIVITY		
New Jersey	2/2 <5,<10	20/18 <3-4000 (230.2)*	15/7 <1-800 (172.2)	——
New Mexico	3/0	4/0	1/0	——

Table 2.1-3 (cont.)

STATE	1972	1973	1974	1975
New York	——	17/5	17/14	——
		<2-1300 (519)*	<2-450 (95.8)	
North Carolina	——	1/0	——	——
North Dakota	2/0	2/0	——	——
Ohio	——	1/0	——	——
Oklahoma	1/0	3/0	1/0	——
Oregon	1/0	1/0	2/0	——
Pennsylvania	9/7	5/3	23/16	3/3
	<5-E100 (50.7)	<5-20 (11.7)	<6-700 (105.6)	<42-240 (144)
Rhode Island	NO ACTIVITY			
South Carolina	2/0	2/0	5/1 (6)	——
South Dakota	1/0	1/0	——	——
Tennessee	——	1/0	1/0	——
Texas	41/3	58/8	58/17	——
	<4-120 (61.7)	<3-45 (17.6)	<1-153 (43.5)	——
Utah	——	3/0	1/0	——
Vermont	NO ACTIVITY			
Virginia	NO ACTIVITY			
Washington	1/0	3/0	2/0	——
West Virginia	NO ACTIVITY			
Wisconsin	——	9/1 (10)	21/0	2/0
Wyoming	——	3/0	1/0	——
Puerto Rico	——	6/0	16/5	——
			<10-640 (242)	

E.- estimated

*mean figure does not include anomalous values

SOURCE: PCB Data Base, October 1972 to (partial) 1975, U.S. Geological Survey, Quality of Water Branch, Reston, VA.

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Table 2.1-4
States Reporting PCB's in Bottom Deposits

1971-72	1973	1974	1975 (partial)
29/15	39/8	32/14	6/4
(51.72%)	(20.51%)	(43.75%)	(66.67%)

states measuring PCB's in bottom sediments/# with at least one non-zero readir

SOURCE: PCB Data Base, October 1972 to (partial) 1975, U.S. Geological Survey, Quality of Water Branch, Reston, VA.

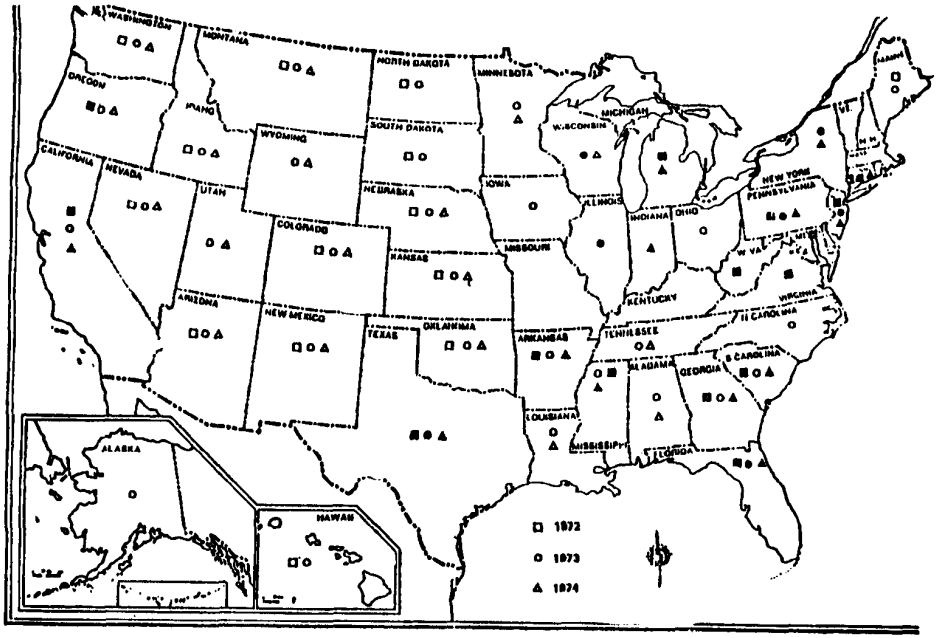

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FIGURE 2.1-1 STATES MONITORING PCB'S IN BOTTOM SEDIMENTS 1972-1974
● DARKENED SYMBOL REPRESENTS AT LEAST ONE DETECTION

2.1.3 Soils

Two national soils monitoring programs are conducted by EPA as part of its Pesticides Monitoring Program covering both cropland and urban soils. In 1973, the only year in which PCB's were analyzed in cropland soils, virtually no occurrences were detected[1].

The urban soils program is a small one, consisting of 5 different urban locations each year. In 1973, the only year for which PCB data in cropland soil are available, virtually no occurrences were detected. The sampling design recovers one analytical sample per square mile in the city proper and one per twenty square miles in the suburbs. The results of the program for the years 1971-1973 are summarized in Table 2.1-5 and Figure 2.1-2[1].

Among each year's set of 5 cites, 3 (60%) had at least one positive PCB site, cities and sites having been randomly selected. Over the three years an average of 2.68% of the sites in these cities (roughly corresponding to 2.68% of the total area) showed PCB concentrations in ranges from 0.9-11.94 ppm. The anomalous reading of 11.94 was found in a residential lawn in Gadsden, Alabama. Seventeen of the twenty two positive readings were below 1 ppm.

Table 2.1-5
National Urban Soil Monitoring Program
PCB Detections 1971 - 1973

	Total No. of Sites	No. of Positive Detections	Percent Positive Detections	Range of Positive Detections (ppm)	Arithmetic Mean (ppm)	PCB Type(s)
1971						
Baltimore, MD.	156	6	3.9	0.09 - 0.74	0.02	1260 1-lawn, 5 waste
Gadsden, AL.	55	1	1.8	11.94	0.21	Not identified, residential lawn
Hartford, CT.	48	ND	-			
Macon, GA.	43	ND	-			
Newport News, VA.	78	1	1.3	3.30	0.04	1254 lawn
1972						
Des Moines, IA	82	3	3.7	0.34 - 0.94	0.03	Not identified, 2-lawn 1-waste
Lake Charles, LA.	70	1	1.4	1.31	0.02	1254
Fitchburg, MA	35	ND	-			
Pittsburgh, PA.	189	1	0.5	1.01	0.01	Not identified, lawn
Reading, PA.	49	ND	-			
1973						
Evansville, IN.	82	ND	-			
Greenville, SC	86	3	3.5	0.13 - 1.59	0.02	1254 3-lawn
Pittsfield, MA.	45	ND	-			
Tacoma, WA.	95	6	6.3	0.18 - 0.63	0.03	1-1254, Others not identified, 2 lawn, 4 waste
Washington, D.C.	116	2	1.7	0.32 - 0.80	<0.01	1-1254, Others not identified, 1 lawn, 1 waste

SOURCE: Carey, Ann, 1975, EPA National Soil Monitoring Program, unpublished.

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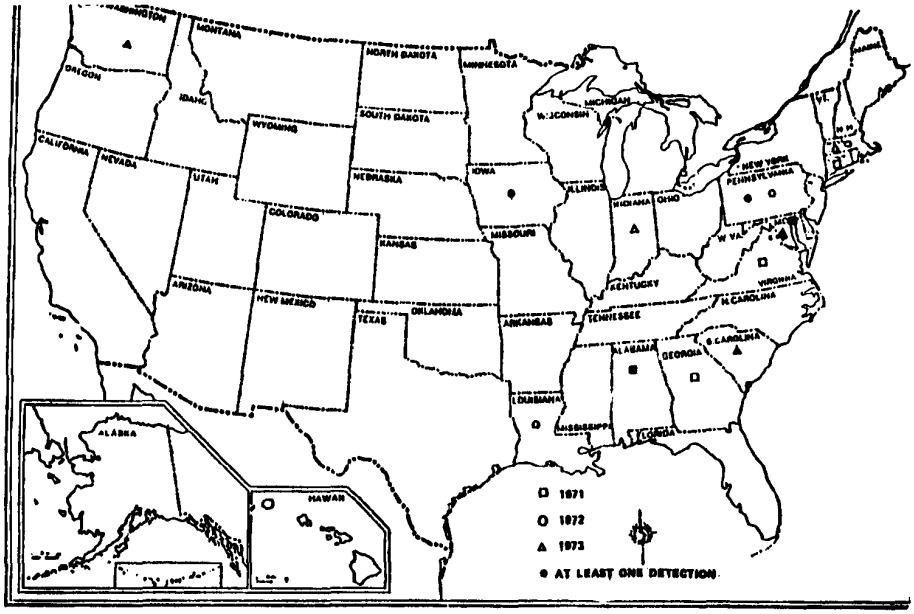

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2.1.4 Air

Polychlorinated biphenyls have not been measured in air on a nationwide basis.

2.1.5 Fish

Fish have long been a popular sampling medium for PCB levels primarily due to the minimal difficulties of sampling and analysis relative to other media. Data on residue levels in fish, however, present problems of interpretation: the eating habits, lipid content, age, sex and size affect PCB levels; residue levels in fish, like those in sediment indicate past, not necessarily current, exposure; residue levels in fish are slow to reflect changes in levels of contamination of the water; distinguishing Aroclor 1242 from 1016 is difficult.

The U.S. Fish and Wildlife Service of the Department of the Interior provides the best national overview with the National Fish and Wildlife Monitoring Program. One of the elements of this program is the annual sampling of freshwater fish at 100 stations (as of 1970). Beginning with the 1969 collections, whole fish samples were analyzed for PCB's.

The most recent data from this program are the unpublished results of the 1973 collection. While many species of fish are sampled throughout the country, a reasonable summary of the data is provided in Table 2.1-6, which concentrates on the four most commonly sampled species of fish, largemouth bass, channel catfish, carp and yellow perch.

The great number of stations reporting "no detection" for those four species in 1973 is significant. Generally lower levels were reported for other species in 1973 also. Furthermore, when sampling data is totalled, ignoring both species and geographic location, as in Table 2.1-7, marked declines in both the proportion of composites with some PCB residues and the proportion with residues above the maximum recommended by Fish and Wildlife of 0.5 ppm are exhibited. While there is a downward trend in the number of stations reporting significant residue levels, those which have reported high concentrations in the past continue to do so. Walker[9] notes that PCB residues have been found in forty to sixty percent of the samples in residue concentrations exceeding 0.5 ppm and that the geographic dispersion shows that the higher concentrations appear to be associated with certain river systems having industrial activity: thirty-one of thirty-five stations in the Mississippi river system reported residues in excess of 0.5 ppm in the 1970-73 sampling program; all stations in the Great Lakes drainage area reported concentrations exceeding 0.5 ppm. Several factors related to interpretation of this data must be recognized: the PCB residues prior to 1973 are reported as "estimated", while those for 1973 are the result of the analysis for three Aroclors (1242, 1254 and 1260) totalled; a trend cannot be inferred from the data of one year, regardless of how dramatic it is, although such data may signal a turning point; analytic protocols for sample analysis, especially determination of specific Aroclors, are suspect and the results of cross check analyses show that some laboratories used for the analysis typically expressed PCB levels of only 5 to 20 percent of those discovered by more sophisticated analytic protocols of the Fish Pesticide Research Laboratory[9].

Table 2.1-6
PCB Residues in Selected Species of Fish
(averages of composite samples, expressed in ppm, wet weight, whole fish)

State	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	197
AL	Alabama R.						
	Chrysler	Largemouth bass	<0.10	3.70	1.35	6.5	N.D.
		Channel catfish				1.5	
	Tombigbee R.						
	McIntosh	Carp		0.86	0.17		N.D.
		Largemouth bass		1.30	0.93	**	N.D.
		Channel catfish				**	
AZ	Colorado R.						
	Imperial Reservoir	Largemouth bass	0.40	0.11		N.D.	N.D.
		Carp	0.25	0.13	0.03	0.01	N.D.
		Channel catfish	0.64	0.17	0.17		
	Colorado R.						
	L. Havasu	Carp		0.17	0.05	N.D.	N.D.
		Largemouth bass		0.14		N.D.	N.D.
		Channel catfish		0.21	0.14		
	Colorado R.						
	L. Powell	Carp		0.09	0.06	<0.10	N.D.
		Largemouth bass		0.09	0.10	0.16	N.D.
	Gila R.						
	San Carlos Res.	Carp		0.04	0.08	**	N.D.
		Largemouth bass		0.08	0.03	**	N.D.
		Channel catfish			0.21	**	
AR	Arkansas R.						
	Pine Bluff	Carp	1.69	1.25	0.52	**	N.D.
		Channel catfish				N.D.	
CA	Sacramento R.						
	Sacramento	Carp	0.10	2.62	1.04	4.35	2.5
		Largemouth bass	<0.10	0.73	0.28	3.7	
	San Joaquin R.						
	Los Banos	Carp	0.10	3.74	0.12	**	N.D.
		Channel catfish	0.10	1.72	0.21		N.D.
				0.14	0.06		
	Klamath R.						
	Hornbrook	Yellow perch		0.24	0.12		0.1C
		Largemouth bass		0.09			
CO	Rio Grande						
	Alamosa	Carp		0.26	0.06	N.D.	
	Arkansas R.						
	John Martin Res.	Carp		0.09	0.05	0.05	N.D.
		Channel catfish		0.15	0.11		N.D.

Table 2.1-6 (cont.)

State	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	1973[b]
CT	Connecticut R.						
	Windsor Locks	Yellow perch	3.40	2.45	17.1	6.6	5.6
FL	St. John's R.						
	Welaka	Channel catfish	0.14	0.18	0.07	N.D.	N.D.
		Largemouth bass	0.31	0.37	0.28	N.D.	N.D.
	St. Lucie Canal						
	Indiantown	Channel catfish	1.25	0.26	0.09	N.D.	N.D.
		Largemouth bass	0.56	0.62	0.08	N.D.	N.D.
	Suwanee R.						
	Old Town	Largemouth bass		0.31	0.5	0.35	0.3
	Apalachicola R.						
	Jim Woodruff Dam	Channel catfish	0.69	0.67	0.62		N.D.
		Largemouth bass	<0.10	0.53	0.12	2.5	N.D.
		Carp	<0.10			N.D.	
GA	Savannah R.						
	Savannah	Largemouth bass	1.18	0.70	0.14	0.64	0.5
		Channel catfish				2.30	
		Carp	0.58	2.92	0.12		3.2
	Altamaha R.						
	Doctortown	Largemouth bass		0.79	0.27	1.20	N.D.
)	Bear R.						
	Preston	Carp		0.89	0.82	1.9	0.8
		Yellow perch		1.29	0.42	1.0	2.35
	Salmon R.						
	Riggins	Carp		0.14	0.06		
	Snake R.						
	Lewiston	Carp		0.29	0.65	0.11	N.D.
IL	Ohio R.						
	Metropolis	Carp		2.01	3.16	7.1	1.4
		Channel catfish		3.78	6.07	5.7	5.45
	Illinois R.						
	Beardstown	Carp	11.3	0.82	0.64	2.1	1.45
IN	Wabash R.						
	New Harmony	Carp		3.10	1.13	1.6	2.1
		Channel catfish		1.07	3.93	3.3	1.27
IA	Des Moines R.						
	Keosauqua	Carp	0.54	0.22	0.16	1.33	1.00
		Channel catfish		0.40	0.86	1.60	0.90
		Largemouth bass				N.D.	
	Mississippi R.						
	Guttenberg	Carp		0.30	1.21	1.0	1.2
		Largemouth bass	1.41	1.79	1.11	1.25	0.96

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Table 2.1-6 (cont.)

[ ]	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	1973[b]
KS	Kansas R.						
	Bonner Springs	Carp		0.55	1.28	1.30	1.2
		Channel catfish			0.77		
LA	Mississippi R.						
	Luling	Carp	0.46	0.86	0.28	4.5	
		Channel catfish	0.66	0.58	1.95	6.6	
	Red R.						
	Alexandria	Channel catfish				N.D.	
		Carp					0.80
ME	Penobscot R.						
	Old Town	Yellow perch	0.31	0.33	0.30	0.36	0.3
	Kennebec R.						
	Hinckley	Yellow perch		0.23	0.15	0.20	N.D.
MD	Susquehanna R.						
	Conowingo	Carp	0.69	2.6	0.69	1.9	0.60
		Channel catfish	1.21	1.77	0.94	4.5	1.2
		Yellow perch	1.31	2.39	0.95	1.9	
	Potomac R.						
	Little Falls	Carp	1.04	0.63	0.81	1.1	
		Channel catfish					0.5
		Largemouth bass	1.04				N.D.
MA	Merrimac R.						
	Lowell	Yellow perch		6.12	21.2	9.5	12.0
MI	L. Huron						
	Bay Port	Carp		2.76	5.50		4.30
		Channel catfish		3.80	1.53		6.6
		Yellow perch		3.33	2.83	0.49	4.25
MN	Red R. (north)						
	Noyes	Channel catfish		0.68	2.29	0.17	N.D.
MS	Yazoo R.						
	Redwood	Carp		1.95	0.68	N.D.	N.D.
		Channel catfish			1.63		N.D.
MO	Mississippi R.						
	Cape Girardeau	Carp		5.26	1.89	2.42	1.9
		Largemouth bass		3.51	3.20		1.0
	Missouri R.						
	Hermann	Carp		1.29	7.6	2.8	4.6
		Channel catfish		0.21		2.00	

Table 2.1-6 (cont.)

State	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	1973[b]
MT	Big Horn R.						
	Hardin	Carp		0.42	0.05	N.D.	N.D.
		Channel catfish		0.55			N.D.
	Yellowstone R.						
	Sidney	Channel catfish		0.14			0.20
		Carp				0.10	N.D.
NE	Missouri R.						
	Nebraska City	Carp		1.38	0.53	0.67	0.30
		Channel catfish				0.50	
	North Platte R.						
	L. McConaughy	Carp		0.24	0.06	N.D.	N.D.
		Channel catfish		0.21	0.09	N.D.	N.D.
	South Platte R.						
	Brule	Carp		0.08	0.22	1.15	0.50
	Platte R.						
	Louisville	Carp		0.71	0.23	1.50	0.33
		Channel catfish		0.62	0.43	4.30	1.70
NV	Colorado R.						
	L. Mead	Carp		0.13	1.68	N.D.	0.20
		Largemouth bass		0.09	0.09	0.26	N.D.
		Channel catfish			0.13	0.76	0.20
	Truckee R.						
	Fernley	Carp	0.54	0.51	1.21	1.8	0.83
		Largemouth bass	<0.10	0.73	0.42	2.1	1.00
NJ	Raritan R.						
	Highland Park	Largemouth bass				4.9	
		Carp					2.1
NM	Rio Grande						
	Elephant Butte Res	Channel catfish		0.35	0.52	0.92	N.D.
		Largemouth bass		0.65	0.30	1.54	N.D.
NY	Hudson R.						
	Poughkeepsie	Largemouth bass	4.82	9.37	34.5	13.0	3.2
	St. Lawrence R.						
	Massena	Yellow perch		1.45	2.12	3.15	1.1
	L. Ontario						
	Port Ontario	Yellow perch		2.48	7.34	6.2	5.2
NC	Roanoke R.						
	Roanoke Rapids	Largemouth bass	0.10	0.89	0.37	1.40	N.D.
		Carp				0.63	N.D.
		Channel catfish					N.D.
	Cape Fear R.						
	Elizabethtown	Largemouth bass		3.33	0.81	3.90	N.D.

Table 2.1-6 (cont.)

State	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	197
ND	Missouri R.						
	Garrison Dam	Carp	0.10	0.08		N.D.	N.D
OH	Ohio R.						
	Marietta	Carp		4.82	5.20	2.5	11.
		Channel catfish		11.85	14.5	9.75	24.
		Largemouth bass	8.07		22.65		
	Ohio R.						
	Cincinnati	Carp		24.5	25.8	19.0	25.
		Channel catfish			27.3	30.0	21.
OK	Verdigris R.						
	Oologah	Carp		0.61	0.8	**	N.D.
		Largemouth bass		0.40	0.09	**	N.D.
	Canadian R.						
	Eufaula	Carp		0.81	0.31		N.D.
		Largemouth bass		0.14			N.D.
		Channel catfish			0.36		
	White R.						
	DeValls Bluff	Carp		1.77	0.73	1.5	N.D.
		Channel catfish		1.82	2.32		N.D.
	Red R.						
	Lake Texoma	Carp		0.25	0.26	N.D.	N.D.
		Largemouth bass		0.21	0.13	N.D.	N.D.
	Arkansas R.						
	Keystone Res.	Carp	0.24	0.45	0.29	0.5	N.D.
		Largemouth bass	0.66	1.25		**	N.D.
		Channel catfish			1.28		
OR	Willamette R.						
	Oregon City	Carp		1.25			
		Channel catfish				0.44	
	Columbia R.						
	Bonneville	Carp			0.19	<0.1	N.D.
	Rogue R.						
	Gold Ray Dam	Carp		2.60		1.4	5.4
		Largemouth bass		0.87			
PA	L. Erie						
	Erie	Yellow perch		2.40	1.11	1.65	0.75
	Allegheny R.						
	Natrona	Carp		4.59	5.52	4.0	1.75
SC	Pee Dee R.						
	Dongola	Largemouth bass		0.62		3.70	0.5
	Cooper R.						
	Summerton	Carp		1.04	0.35	N.D.	1.5
		Largemouth bass	<0.10	0.45	0.18	3.20	3.3

Table 2.1-6 (cont.)

State	Waterway/ Station Location	Species	1969[a]	1970[a]	1971[a]	1972[a]	1973[b]
SD	James R.						
	Olivet	Carp		0.20	0.65	N.D.	N.D.
		Channel catfish			0.20	1.20	<0.10
TN	Cumberland R.						
	Clarksville	Carp	0.89	1.67	0.69	16.0	4.3
		Largemouth bass	3.15	2.92	1.19	5.0	5.8
	Tennessee R.						
	Savannah	Carp		1.46	0.33	4.65	1.5
		Channel catfish		4.32	3.18		9.8
		Largemouth bass		1.20	0.63	11.0	3.1
	Mississippi R.						
	Memphis	Carp		3.26	1.22	10.0	1.5
		Channel catfish				5.4	
TX	Pecos R.						
	Red Bluff Lake	Channel catfish		1.07	0.19	0.60	N.D.
		Largemouth bass		0.06			
	Brazos R.						
	Richmond	Channel catfish		1.17		0.5	
	Colorado R.						
	Wharton	Channel catfish		0.32	0.38	1.1	N.D.
		Carp					N.D.
	Nueces R.						
	Mathis	Channel catfish		0.03		N.D.	
		Largemouth bass		0.05			N.D.
	Rio Grande						
	Brownsville	Channel catfish		0.40	3.62	7.70	3.46
UT	Utah Lake						
	Provo	Carp	0.29	0.18	0.10	**	0.26
	Green R.						
	Vernal	Carp	0.83	0.09	0.6	**	N.D.
		Channel catfish		0.10	0.22	**	N.D.
VT	L. Champlain						
	Burlington	Yellow perch		0.78	0.74	1.20	1.21
VA	James R.						
	Richmond	Channel catfish		0.79	0.44		1.5
		Largemouth		0.34	0.33		1.5
WA	Snake R.						
	Ice Harbor	Channel catfish		1.00	0.62	1.3	0.5
		Carp			0.19		
	Yakima R.						
	Granger	Carp		1.24	0.22	1.70	N.D.
		Largemouth bass		0.63			N.D.
	Columbua R.						
	Pasco	Carp		0.17	0.19	1.4	N.D.

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Table 2.1-6 (cont.)

State	Waterway/ Station Location	Species	1969	1970	1971	1972	197
WV	Kanawha R. Winfield	Carp	0.31	1.25	0.61	12.0	3.8
WI	L. Michigan Sheboygan	Yellow perch		7.42	11.45	9.3	7.8
	Wisconsin R. Woodman	Carp		1.15	4.44		3.6
		Channel catfish		0.17	1.11		

** - PCB-like compound present
N.D. - Not Detectable

a. estimated PCB levels
b. total of Aroclors 1242, 1254 and 1260

SOURCES:
1969 Data - Henderson, C., A. Inglis and W.L. Johnson, Pesticides Monitoring Journal, 5, 1-11 (1971).

1970-1973 Data - National Fish and Wildlife Monitoring Program, Results of Freshwater Fish Monitoring 1970-1973, U.S. Fish and Wildlife Service, U.S. Department of Interior, Washington, DC (unpublished).

Table 2.1-7
Summary of PCB Residues Detected in Fish from 1969-1973
National Pesticide Monitoring Program

		Number of Composites				
		With Residues		With Residues Above 5 ppm		
Year	Total	No.	%	No.	%	Range (ppm)
1969	147	147	100	89	61	<0.10-14.8
1970	393	390	99	239	61	0.03-24.8
1971	587	581	99	284	48	0.02-41.0
1972	401	311	78	215	54	<0.05-30.0
1973	400	202	51	160	40	<0.01-25.0

SOURCES:

1969 - Henderson, C., A. Inglis and W.L. Johnson, Pesticides Monitoring Journal, 5, 1-11 (1971).

1970-1973 Data - National Fish and Wildlife Monitoring Program, Results of Freshwater Fish Monitoring 1970-1973, U.S. Fish and Wildlife Service, U.S. Department of Interior, Washington, DC (unpublished).

2.1.6 Birds

Two of the three elements of the National Fish and Wildlife Monitoring Program mentioned in the previous section are: mallards and black ducks; starlings.

PCB's have been found in all of the starling samples taken in 1970, 1972 and 1974. The level of residues appears to be declining over this period with national arithmetic means of 0.65 ppm, 0.43 ppm and 0.12 ppm for the three years respectively[9].

Results of sampling of mallards in 1970 and 1972[9] are shown by flyway on Figure 2.1-3. Residues are highest in the Atlantic flyways, with average concentrations more than double that of any other flyway and more than 10 times greater in some cases. Samples of black ducks were also taken in the Atlantic flyways in 1969 and 1972 and the mean PCB concentration was constant at 1.36 ppm.

In the 1969 mallard study[9] not only did mallards from the Atlantic flyway show the greatest average PCB concentration, but with the exception of Maryland all of the states in the Atlantic flyway had averages exceeding 0.5 ppm with the highest residues from New Hampshire southward through Delaware. In the Mississippi flyway, residue levels were highest in states bordering the Great Lakes, generally diminishing as sampling moved southward.

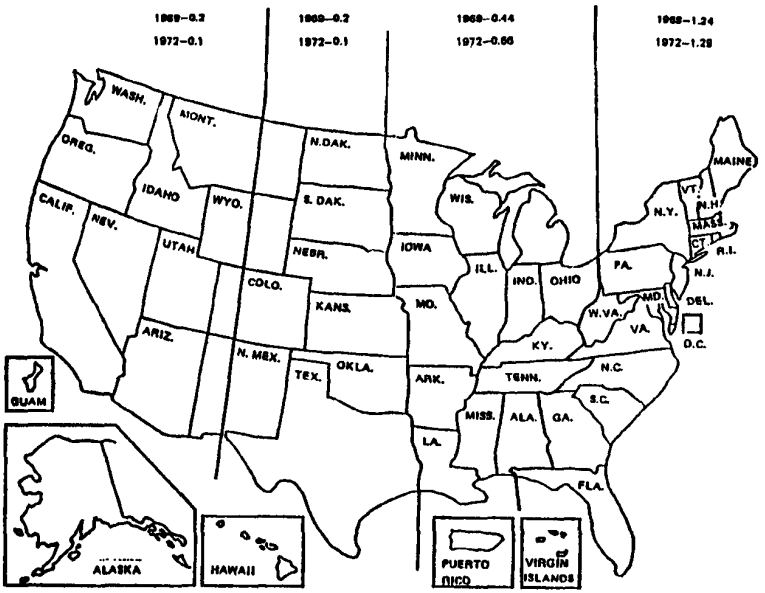

FIGURE 2.1-3 PCB's IN MALLARDS (AVERAGES, WINGS, PPM, WET WEIGHT BASIS)

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REFERENCES, Section 2.1

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2. Crump-Wiesner, J.S., H.R. Feltz and M.L. Yates, J. Research USGS, 1, 603-607 (1972).

3. Haile, C.L., G.D. Veith, G.F. Lee and W.C. Boyle, Chlorinated Hydrocarbons in the Lake Ontario Ecosystem (IFYGL 1975), unpublished EPA report, EPA-660/3-75-022.

4. Heath, R.G. and S.A. Hill, Pesticides Monitoring Journal, 7, 153-163 (March 1974).

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6. Martin, W.E. and P.R. Nickerson, Pesticides Monitoring Journal, 6, 33-40 (June 1972).

7. National Fish and Wildlife Monitoring Program, Results of Freshwater Fish Monitoring 1970-1973, U.S. Fish and Wildlife Service, U.S. Department of the Interior, Washington, DC (unpublished)..

8. PCB Data Base, October 1972 to (partial) 1975, U.S. Geological Survey, Quality of Water Branch, Reston, VA.

9. Walker, Charles R., The Occurence of PCB's in the National Fish and Wildlife Monitoring Program, presented at the National Conference on Polychlorinated Biphenyls, Chicago, Illinois, November 19-21, 1975

2.2 Data from Localized Monitoring Efforts - Lakes

Figure 2.2-1 is provided in order to display the relationship of the different lakes in the Great Lakes region as discussed in 2.2.1 - 2.2.5.

2.2.1 Lake Ontario

Lake Ontario is one of the lesser studied Great Lakes for PCB's. A thorough study of chlorinated hydrocarbons in the Lake Ontario ecosystem was performed in 1972 by Haile, Veith, Lee and Boyle.[6] Samples of various media were taken from 19 different locations in Lake Ontario during the summer of 1972, as indicated in Figure 2.2-2, and significant concentrations of PCBs were reported in all media. Findings for water, sediment and net plankton are summarized in Table 2.2-1. Other than the south shore areas off Oswego (77 ppt) and the north of the Niagara River (97 ppt), the PCB content of the Lake Ontario water appears relatively uniform in the range of 35 to 56 ppt, with an average of 43 ppt. PCB concentrations in sediments around the south shore areas off the mouths of the Welland Canal and Niagara River and off Oswego averaged more than twice the concentrations found at the four other sites (means of 184 ppb vs. 72 ppb). This led the authors to conclude that the Niagara and Oswego Rivers may be important sources of PCB associated with settleable particulates. Plankton samples were not taken at the south shore sites; lake wide levels averaged 7.2 ppm.

Fish samples selected in the same study found mean PCB concentrations on a whole fish basis in smelt (2.65 ppm) similar to those reported in Lake Michigan smelt. Mean PCB levels in alewives (2.35 ppm) were lower than values reported for Lake Michigan alewives (4.6 ppm). Bottom feeding slimy sculpin, which are less migratory than the latter two species, exhibited greater station to station variability in PCB levels, with a lake wide average of 4.63 ppm. Average concentrations in Cladophora were 515 ppb and for benthic fauna were 471 ppb. Samples were not taken consistently across media at stations and the results suffer accordingly, but the authors do draw one of the few estimates of accumulation factors for chlorinated hydrocarbons. They estimate relative concentrations as 1; 2500; 10,000; 150,000; and 300,000 for water, sediment, benthos, net plankton and fish, respectively.

Kaiser[10] shows PCB levels in two 1973 fish samples from northern Lake Ontario as 5.99 ppm (total PCB, fat, northern longnose gar) and 2.38 ppm (total postanal fins, northern pike), magnitudes considered comparable to those of other fishes from Lake Ontario.

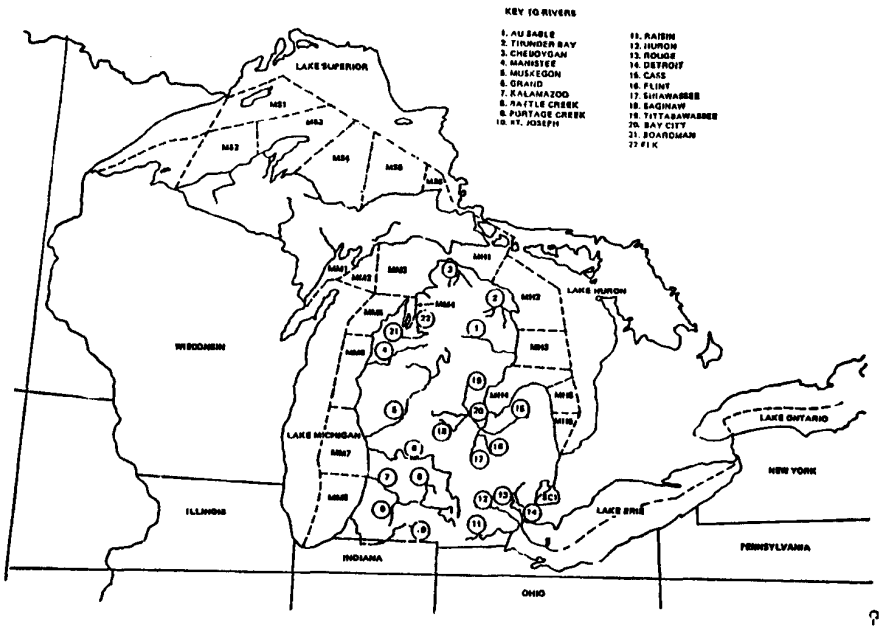

FIGURE 3.2-1 THE GREAT LAKES (AND RIVERS IN MICHIGAN)

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Table 2.2-1

PCB Concentrations, Lake Ontario Ecosystem
Summer 1972

Sample Location (map reference)	Water (ppt)	Sediment (ppb dry)	Net Plankton (ppm dry)
1	49		
8	35		3.4
10			
12			10.6
13		245	
30	97	155	
36	44	80	
45	45	43	
46			7.6
60		79	3.6
75	40	84	
90	56		
91	77		11.8
96		158	
			6.0

SOURCE: Haile, C.L., G.D. Veith, G.F. Lee and W.C. Boyle, Chlorinated Hydrocarbons in the Lake Ontario Ecosystem (IFYGL), EPA Report. EPA 660/3-75-022 (1975).

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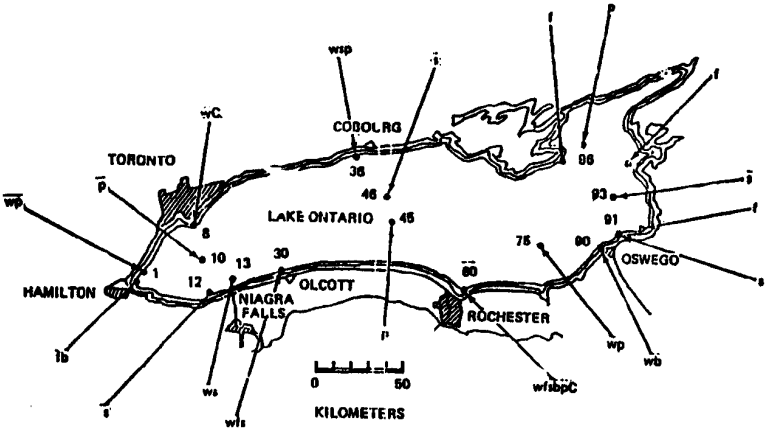

FIGURE 2.2-2 LAKE ONTARIO SAMPLING SITES FOR CHLORINATED HYDROCARBON ANALYSIS. NOTATIONS ARE: f-FISH, w-WATER, s-SEDIMENT, p-NET PLANKTON, C-CLADOPHORA, b-BENTHOS. STATION NUMBERS ARE IFYGL STATION IDENTIFIERS.

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2.2.2 Lake Erie

Several studies of PCB concentrations in fish samples from Lake Erie are summarized in Table 2.2-2. Levels are generally lower than those reported from Lake Michigan and Lake Superior, but are inconclusive other than demonstrating the presence of PCB's in the lake water. Water measurements in tributaries to Lake Erie[3] including the Rouge river, which is tributary to the Detroit River are in Table 2.2-3, the Raisin and Rouge Rivers show relatively high levels of concentration when contrasted to the concentration levels in fish in the preceding table.

Table 2.2-2

Mean PCB Levels in Fish, Lake Erie
(ppm, whole fish)

Species	1970[a]	1972[b]	1973[c]
Carp	2.0		9.3
Catfish	4.4		
Drum	1.1		0.9
Yellow Perch	0.8	0.25	Trace
Salmon	2.1		
Walleye			0.5
White Bass	2.1	1.02	2.5
Smallmouth Bass		0.71	

SOURCES: [a] Carr, R.L., C.E. Finsterwalder and M.J. Schibi, Pesticides Monitoring Journal, 6, 23-26 (1972).

[b] Kelso, J.R.M. and R. Frank, Transactions American Fisheries Society, 103, 577-581 (1974).

[c] 1973 Great Lakes Environmental Contaminant Survey - Data Summary and Analysis, Bureau of Consumer Protection, Michigan (1973).

Table 2.2-3

PCB Concentrations in Michigan Tributaries
to Lake Erie in 1971-72 and June 1973 in
ppb as Aroclor 1254

River	Mean PCB Concentration	Range (1971-72)	1973
Raisin	0.210	<0.068-0.500	<0.010
Huron	0.012	<0.010-0.039	<0.010
Rouge	0.470	0.058-1.600	0.460*
Detroit	0.020	<0.010-0.053	<0.010

* Aroclor 1242

SOURCE: Hesse, J.L., Status Report on Polychlorobiphenyls in Michigan Waters, Michigan Water Resources Commission, June 1973.

2.2.3 Lake Superior

The results of several available fish studies are presented in Table 2.2-4 for different species of fish. Lake trout are in the highest trophic level within the lake and biological accumulation should result in this species possessing some of the highest concentrations of PCB's.

The study by Veith and Glass[19], summarized in the table, drew samples from three areas in the western portion of the lake. There was variability in the levels of PCB's in fish caught in different areas, but no conclusions are drawn in the study. Samples from the Apostle Island region (Wisconsin) had higher mean concentrations for all species than those from the other two regions (Minnesota). Parejko and Johnston[14] note that the biphenyls containing lower percent concentrations of chlorine predominated in the sample. The latter study involved tests for correlation between factors of age, sex, geographical location and concentration level; surprisingly, no significant correlations were discovered.

The geographic distribution of lake trout samples from the 1972-1974 Great Lakes Environmental Contaminant Survey[5] is detailed in Table 2.2-5.. As regards recent screening of fish against the FDA guideline, Kleinert[12] reports that of 64 fish samples collected in the nearshore waters during 1974, none exceeded the 5 ppm guideline and only two exceeded a level of 1 ppm.

Table 2.2-4

Mean PCB Levels in Fish, Lake Superior
(ppm, whole fish)

Species	1971[c]	1972[a]	1973	1974[e]
Burbot		0.7	1.4[a]	
Chubs		0.9	1.6[b]	
Herring		2.0	1.8[b]	
Lake Trout	7.08	0.8	1.8[a] 2.64[d]	1.55
Long-Nose Sucker			0.9[a]	
Smelt		0.3	0.4[a]	
Whitefish			0.3[b]	

SOURCES:

[a] Veith, G.D. and G.E. Glass, PCBs and DDT in Fish from Western Lake Superior, U.S. EPA National Water Quality Lab., Duluth, Minnesota (1974).

[b] 1973 Great Lakes Environmental Contaminant Survey - Data Summary and Analysis, Bureau of Consumer Protection, Michigan (1973).

[c] Parejko, R. and R. Johnston, Uptake of Toxic Water Pollutants, (PCB) by Lake Trout. Project Completion Report, U.S. Dept. of the Interior, Office of Water Resources Research, Contract 14-01-0001-3522 (1973).

[d] Willford, W.A., Contaminants in Upper Great Lakes Fishes, Presented at Great Lakes Fishery Commission, Upper Great Lakes Committee Meetings, Milwaukee, Wisconsin, March 1975.

[e] Hesse, J.L., PCB Situation in Great Lake Fish, Report to Michigan Water Resources Commission, April 17, 1975.

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Table 2.2-5
Mean PCB Levels in Lake Trout
from Lake Superior (ppm)

Lean Variety		<17"	17-20"	20-25"	>25"
MS 1**	1972	—	—	—	—
	1973	2.4 (4)*	2.9 (1)	5.6 (3)	4.8 (4)
	1974	—	—	—	—
MS 2	1972	—	—	—	—
	1973	0.5 (3)	1.8 (3)	3.4 (3)	5.2 (2)
	1974	—	—	1.2 (3)	5.2 (1)
MS 3	1972	—	—	2.2 (6)	2.7 (3)
	1973	1.6 (12)	1.9 (7)	4.5 (8)	4.8 (11)
	1974	0.6 (3)	0.1 (2)	0.4 (9)	2.0 (12)
MS 4	1972	—	—	4.6 (3)	5.7 (8)
	1973	—	1.0 (6)	2.0 (6)	4.7 (9)
	1974	0.5 (2)	0.5 (2)	0.7 (4)	1.4 (3)
MS 5	1972	—	—	3.3 (6)	2.1 (3)
	1973	—	1.2 (3)	1.2 (3)	1.9 (3)
	1974	—	1.8 (2)	1.4 (4)	1.5 (6)
MS 6	1972	—	—	1.4 (3)	—
	1973	0.9 (3)	1.0 (3)	1.5 (3)	1.3 (3)
	1974	2.2 (8)	0.4 (2)	2.8 (7)	2.6 (5)
Fat or Siscowet Variety					
MS 1	1974	0.5 (3)	3.6 (2)	1.9 (4)	3.8 (3)
MS 2	1974	2.5 (5)	6.2 (10)	11.3 (6)	3.3 (2)
MS 3	1974	4.4 (6)	12.6 (2)	7.4 (7)	6.8 (7)
MS 4	1974	12.7 (2)	5.4 (8)	—	6.1 (6)

*Concentrations in parts per million (ppm) in fillet; wet weight. Number of samples in parentheses ( ).

**Refers to geographic area in Figure 2.2-1.

SOURCE: Heese, J.L., Contaminants in Great Lakes Fish, Staff Report, Michigan Water Resources Commission, Department of Natural Resources, (1975).

2.2.4 Lake Michigan

Lake Michigan is the most heavily studied of the Great Lakes. It has been established that the lake contains the highest concentrations of agricultural pesticides due, in part, to the large useage in the watershed relative to the flushing period for the lake and to the relatively low biomass density. There were seizures of Lake Michigan fish by the FDA during 1975. Samples from a shipment of Lake Michigan Chinook and Coho Salmon in May 1975 were found to exceed the 5 ppm guideline.[2] PCB levels in these fish ranged from 7.6-10.9 ppm. Hesse[7] reports a February 1975 seizure of large, surplus salmon exceeding the FDA guideline. In May 1975[2] a seizure of chubs resulted in analysis which showed that concentrations did not exceed the guideline. Such activities, however, have dramatized the hazards to both the lake and its fishing industry. In June, 1975, the Secretary of the State of Wisconsin Department of Natural Resources, based on evidence indicating that most large trout and salmon contain PCB's at levels exceeding the 5 ppm tolerance level, concluded that most fish over 24 inches in length appear to have excessive levels.

To provide a broad historical perspective, data from several studies of Lake Michigan fish are assembled in Table 2.2-6. The question of trend in the levels of fish contamination has been addressed by both Hesse[8] and Willford[21], referring specifically to the data of Table 2.2-7. Both conclude that the situation has generally remained static since 1972. Statistical analyses show no significant differences in the levels of the three species over the three year period 1972-1974. Data from the Great Lakes Environmental Contaminant Survey for this period for lake trout is detailed, by sampling area, in Table 2.2-8. The sample sizes are generally small and, while not allowing for rigorous statistical analysis, document the presence of a widespread PCB problem in lake trout of larger sizes and an intensification of the problem in southern Lake Michigan. The general conclusions that larger fish such as brown, lake and rainbow trout and chinook and coho salmon contain PCB residues at levels exceeding the FDA guideline, that concentrations increase with the percentage of fat and the size of the fish, and that levels found in the southern portion of the lake exceed those in the northern portion are supported by various studies[2].

The results of some earlier (1971-72) fish sampling undertaken by the Illinois EPA[3] found PCB levels in the edible portion of five species (yellow perch, chub, carp, coho salmon and alewife) generally below the 5 ppm tolerance limit. This is in apparent contradiction to the later studies discussed in the preceding paragraph - especially since the Illinois samples came from the southern portion of the lake. Data from this study is included in Table 2.2-6. There are, of course, many variables which impact on the interpretation of data from fish sampling lipid content, fish size and age, season of capture, location, etc. Thus, mean readings may not be truly representative although this statistic is commonly used to provide broad summarization of an abundance of data.

Measurements taken on Lake Michigan open waters and sediment tend to show results which are consistent with reported sediment data from other sections of the country (see section 2.1.3), and somewhat higher readings for concentrations in whole water. The Illinois EPA has found tributary sediments with higher concentrations than those from open waters (1971): averages of 23.06 ppb as Aroclor 1242 and 14.66 ppb Aroclor 1254 for the former contrasted with 95.77 ppb Aroclor 1242 and 32.27 ppb Aroclor 1254 for the latter[5]. Whole water concentrations in tributaries analyzed as Aroclor 1242 or 1254 ranged from 0.1 to 4.0 ppb during the period 1971-1974[2]. PCB concentrations measured by the Illinois State Board of Health in tributaries to Lake Michigan during 1973-1974 ranged from 0.01 to 0.09 ppb.

The Milwaukee River, a Wisconsin tributary to Lake Michigan was sampled in August 1969 for PCB's[20]; concentrations of Aroclors 1260 and 1242 were in the ranges trace to 0.26 ppb and trace to 2.07 ppb, respectively. The high Aroclor 1242 reading was validated by a repeat measurement (of 2.80 ppb in February 1970) and attributed to combined sewer outfalls and contaminated industrial cooling waters. Estimates of PCB concentrations in sewage treatment plant effluents in March, 1970, ranged from 0.04 ppb to 0.25 ppb, but the effluent from a chemical plant had a concentration of 2.50 ppb. This led the authors to conclude that PCB's were discharged to natural waters through municipal and industrial wastes and that PCB's in such large ecosystems as Lake Michigan have resulted in part, through water transport from metropolitan areas. This certainly is true; unfortunately no follow-up studies were performed to estimate the effect of both controls and increased attention to the PCB problem.

With the exception of a contamination problem in the Kalamazoo River, most PCB measurements in Lake Michigan tributaries within the state of Michigan are at levels in whole water of less than 0.1 ppb. Data showing levels identified in 1971-1972 are in Table 2.2-9. The Kalamazoo River was the subject of studies in both 1971 and 1972[6]. Concentration levels were higher downstream from Battle Creek, and some sources of contamination were identified.

Fifty four fish samples were collected from 15 stream sections on the Kalamazoo River in July 1971[6]. Measurable concentrations were found in nearly all fish samples tested with concentrations ranging from less than 0.01 to 109.9 ppm in edible portions of fish on a wet weight basis. Those fish from the north and south branches and the main stream of the river down to Battle Creek had low concentrations ranging from 0.01 to 0.33 ppm. Downstream from the Battle Creek wastewater treatment plant the levels in fish increased ranging from 0.82 to 18.75 ppm and remained high downstream with levels below Kalamazoo ranging from 1.35 to 109.9 ppm[6].

Settleable solids were only sampled at eight stations in 1971 with levels identified ranging from 0.01 to 0.422 ppm[6]. The levels in the mainstream were 10 to 20 times higher than those in the north and south branches and gradually increased downstream. The sample from the Battle Creek River had the highest concentration in the watershed indicating that the tributary contributes significantly

to the total problems in the mainstream.

Since the Kalamazoo River is the most polluted input to Lake Michigan further sampling to identify possible sources of PCB loss to the environment was conducted in the vicinity of the City of Kalamazoo in 1972[15]. PCB concentrations from the discharge of several industries and a wastewater treatment plant were below the limit of detectability. However, Portage Creek, which receives effluents from several industries including two paper mills, had 0.47 ppb of Aroclor 1242. Settleable solids that were collected showed concentrations ranging from 0.23 to 2.63 ppm at eight of the ten stations. The two stations that did not have significant concentrations were upstream from the paper mill ponds. Sediment core samples taken from the Bryant paper mill ponds had concentrations up to 368.7 ppm as deep as six to eight inches.[5]

Further data collected in the spring of 1973 on settleable solids are presented in Table 2.2-10 demonstrating considerable sensitivity of measurements to sampling intervals. The levels detected in 1975 were also generally higher for the Kalamazoo River than were detected in 1971.

Table 2.2-6
Mean PCB Levels in Lake Michigan Fish
(ppm, whole fish)

Species	1971[b]	1972[d]	1973[f]	1974[e]
Alewife	2.84[a], 4.6	2.4		
Brown Trout	7.3			
Carp	2.04[a], 4.2	3.9		
Chub	2.85[a], 6.0	7.0, 2.83[a]	4.48	
Chinook Salmon	11.4	12.4	6.4	
Coho Salmon	1.68[a], 11.5	11.2, 10.93[e]	4.9, 12.17[e]	10.45
Lake Trout	13.53[c], 15.5	7.4, 12.86[e]	18.93[e]	22.91
Perch	5.8	0.4		
Rainbow Trout	9.3			
Red Sucker	3.0			
Smelt	2.7		1.6	
White Sucker	3.9			
Whitefish	3.0	0.7	0.9	
Menominee		1.1	1.0	
Suckers		1.0	0.45	
Steelhead		6.0		
Bloater Chubs		5.66[e]	5.24[e]	5.57
Yellow Perch	0.22[a]	0.3[a]	0.4	

SOURCES:

[a]U.S. EPA, Pesticide Monitoring Programs: Lake Michigan and Tributaries in Illinois, EPA 600/3-74-002.

[b]Veith, G.D., Chlorinated Hydrocarbons in Fish from Lake Michigan, University of Wisconsin, Madison, Wisconsin, unpublished report on EPA Project 16020 PBE, (1973).

[c]U.S. EPA, An Evaluation of DDT and Dieldrin in Lake Michigan, EPA-R3-72-003, (1972).

[d]Hesse, J.L., Status Report on Polychlorobiphenyls in Michigan Waters, Michigan Water Resources Commission, (June 1973).

[e]Bremer, K.E., State of Concerns of the Lake Michigan Toxic Substances Committee Related to Polychlorinated Biphenyls (draft) U.S.EPA, (1975).

[f]1973 Great Lakes Environmental Contaminant Survey-Data Summary and Analysis, Bureau of Consumer Protection, Michigan, (1973).

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Table 2.7-7
Concentrations of PCBs in fall collections of Lake Michigan Bloaters and Lake Trout off Saugatuck, Michigan, and Coho Salmon from between Ludington and Frankfort, Michigan

Species and Year	Number of Fish	Average Length (mm)	Total PCBs[b] (ppm)
Bloaters			
1972	120[c]	255	5.66 (0.95)
1973	160[c]	250	5.24 (0.37)
1974	110[c]	257	5.37 (0.31)
Coho salmon			
1972	10	693	10.93 (2.12)
1973	29	620	12.17 (0.77)
1974	30	665	10.45 (0.92)
Lake trout			
1972	9	648	12.86 (4.75)
1973	30	602	18.93 (2.08)
1974	30	616	22.91 (3.73)

[a]Analysis performed by Great Lakes Fishery Laboratory.

[b]Concentrations in whole fish, wet weight with 95% confidence interval in parentheses.

[c]Composite samples, 10 fish per sample.

SOURCE: Bremer, K.E., State of Concerns of the Lake Michigan Toxic Substances Committee Related to Polychlorinated Biphenyls (draft) U.S. EPA, (1975).

Table 2.2-8
Mean PCB Levels in Lake Trout
from Lake Michigan (ppm)

		<17"	17-20"	20-25"	>25"
MM 1**	1972	—	—	—	—
	1973	2.2 (1)*	3.6 (2)	—	—
	1974	0.7 (1)	1.6 (2)	—	—
MM 2	1972	—	—	—	—
	1973	—	—	—	—
	1974	—	—	—	—
MM 3	1972	—	—	—	11.1 (3)
	1973	2.6 (3)	2.4 (3)	—	10.6 (3)
	1974	—	—	—	—
MM 4	1972	—	—	—	6.5 (3)
	1973	2.6 (2)	1.8 (1)	—	8.5 (3)
	1974	1.0 (3)	3.1 (3)	—	—
MM 5	1972	—	—	—	—
	1973	—	—	5.8 (1)	7.3 (2)
	1974	—	—	7.5 (6)	9.6 (6)
MM 6	1972	2.4 (1)	—	—	4.1 (1)
	1973	—	6.4 (1)	7.1 (2)	11.0 (3)
	1974	1.1 (9)	2.5 (16)	5.6 (11)	9.6 (18)
MM 7	1972	2.7 (2)	—	6.8 (1)	7.3 (3)
	1973	—	—	—	11.2 (3)
	1974	2.4 (9)	—	8.9 (8)	11.9 (2)
MM 8	1972	—	5.8 (2)	6.4 (1)	12.4 (3)
	1973	5.1 (6)	8.2 (3)	10.1 (4)	12.6 (4)
	1974	—	—	—	—

*Concentrations in parts per million (ppm) in fillet; wet weight.
Number of samples in parentheses ( ).

**Refer to geographic area in Figure 2.2-1.

SOURCE: Hesse, J.L., Contaminants in Great Lakes Fish, Staff Report, Michigan Water Resources Commission, Dept. of Natural Resources (1975).

Table 2.2-9
Mean PCB Concentrations in Michigan Tributaries to the Lake Michigan Basin, 1971-72 and 1973
(ppb as Aroclor 1254)

River	1972-72 Average	1972-72 Range	1973
St. Joseph	0.013	<0.010-0.039	<0.010
Kalamazoo	0.065	0.019-0.097	0.040
Grand	0.041	0.011-0.080	0.033
Muskegon	0.010	<0.010-0.037	<0.010
Manistee	0.014	<0.010-0.039	0.037
Boardman	0.017	<0.010-0.044	0.018
Elk	0.012	<0.010-0.039	<0.010

SOURCE: Hesse, J.L., Status Report on Polychlorobiphenyls in Michigan Waters, Michigan Water Resources Commission, (June 1973).

Table 1.2-10

Concentrations of PCB's (polychlorinated biphenyls) in the settleable solids collected from the mouths of the Grand, Kalamazoo and St. Joseph Rivers; Spring 1973, for comparison of variable sampling periods. Concentrations in ppm on an oven-dry basis.

River	Sample Type[a]	Sample Dates 3/21	3/23	3/30	4/2	4/4	4/6	4/9	4/11	4/13	4/20	4/27	4/30	5/2	5/4	5/11	Means
Grand	3X	3.24			1.64	0.95	1.0	<0.1	0.93	1.58			0.83	3.5	0.53		1.43
	Weekly		1.25	0.94			1.1			1.0	0.55	0.74			0.80	0.77	0.89
	Biweekly			0.85						0.73		0.50				0.60	0.67
	Monthly									1.41						0.50	0.96
																Mean =	0.99
Kalamazoo	3X	6.7	27.5		2.08	<0.1	2.52	1.35	3.20	<0.1			3.11	3.50	3.75		4.72
	Weekly		6.83	3.0			3.54			3.77	3.09	2.98			2.88	4.87	3.87
	Biweekly			3.77						2.34		2.84				2.50	2.86
	Monthly									2.11						3.14	2.63
																Mean =	3.52
St. Joseph	3X												0.63	0.82			0.73
	Weekly								2.13	0.68		0.95		0.83	1.21		1.16
	Biweekly									0.85		0.75			0.95		1.24
	Monthly											0.67			0.83		0.75
																Mean =	0.97

[a]3X = Sample collected Mon, Wed. and Friday
[b]Concentration based upon a 1:1 ratio of Aroclor 1242 and 1251.
All other concentrations calculated as Aroclor 1242.

SOURCE: Monitoring for Polychlorinated Biphenyls in the Aquatic Environment, Michigan Water Resources Commission, (May 1973)

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2.2.5 Lake Huron

Levels of PCB's in fish samples from Lake Huron are detailed in Table 2.2-11 from areas as indicated in Figure 2.2-1. The Saginaw Bay area, designated as MH-4 on the map, has been the object of detailed analysis, and significant concentrations have been detected. Water measurements in tributaries to Lake Huron, including the Saginaw Bay area, are in Table 2.2-12. Concentrations in fish taken from the Saginaw River in 1971 were as high as 165 ppm as shown in Table 2.2-13. The waste water treatment plant on the Saginaw River is the Bay City facility. During 1971, this plant had an average effluent concentration of 120 ppb with a high of 340 ppb.

After control measures were initiated by some industrial sources in October 1972, there was a sharp drop in PCB levels at the mouth of the river. Levels measured in 1972 ranged from <0.020 to 0.640 ppb and in 1973 from <0.100 to 0.200 ppb. This is one of the noteworthy examples of controls effecting a marked change in PCB contamination[9].

Table 2.2-11
Mean PCB Levels, Whole Fish
in Lake Huron (ppm)

Species	1973
Brown Trout	3.6 (MH-2)*
Catfish	4.9 (MH-4)
Menominee	0.4 (MH-1)
Yellow Perch	0.2 (MH-1)
Salmon	8.4 (MH-1,3)
Walleye	1.1 (MH-1)
Smelt	0.6 (MH-3)
Whitefish	0.5 (MH-1)

*Refer to geographic area in Figure 2.2-1

SOURCE: 1973 Great Lakes Environmental Contaminant Survey - Data Summary and Analysis, Bureau of Consumer Protection, Michigan (1973).

Table 2.2-12
Mean PCB Concentrations in Michigan Tributaries
to Lake Huron in 1971-72 and 1973
(ppb as Aroclor 1254)

River	1971-72 Average	1971-72 Range	1973
Saginaw	1.100	0.450-2.900	0.210[b]
Cass[a]	0.014	<0.010-0.048	<0.010
Flint[a]	0.078	0.010-0.150	<0.010
Shiawassee[a]	0.029	<0.010-0.073	<0.010
Tittabawassee[a]	0.140	<0.022-0.230	<0.010
Au Sable	<0.010	<0.010-0.010	0.013
Thunder Bay	0.023	<0.010-0.037	<0.010
Cheboygan	0.032	<0.010-0.053	<0.010

[a]Tributaries to Saginaw River

[b]Aroclor 1242

SOURCE: Hesse, J.L., Status Report on Polychlorobiphenyls in Michigan Waters, Michigan Water Resources Commission, (June 1973).

Table 2.2-13
Concentrations of polychlorinated biphenyls (PCB's)
in Fish from Saginaw River, December 7, 1971
(ppm; wet weight)

Species	Location	Length	Percent fat	PCB's based upon 1242 standard
Perch	Karn Weadock Discharge Channel	9.0	1.3	16.3
Carp	" " " "	21.0	0.9	8.8
Catfish	" " " "	8.0	7.0	37.1
Catfish	" " " "	7.5	4.9	47.9
Carp	U.S. Coast Guard Station	23.0	—	48.0
Carp	" " " "	24.0	10.5	20.4
Carp	" " " "	20.0	6.5	15.5
Carp	" " " "	24.0	5.9	45.8
Carp	" " " "	21.0	4.6	30.2
Pike	" " " "	27.0	1.6	16.6
Pike	" " " "	23.0	1.1	6.9
Gizzard Shad	" " " "	14.0	17.5	165.3
Gizzard Shad*	" " " "	6.0	15.3	32.5
Gizzard Shad*	" " " "	4.5	15.0	161.9
Gizzard Shad*	" " " "	5.0	17.0	77.0
Gizzard Shad*	Zilwaukee Bridge	4.5	15.7	24.1
Gizzard Shad*	" "	5.0	9.6	52.0
Gizzard Shad*	" "	5.0	8.7	10.4

*Concentration based upon analysis of whole fish. All others based upon edible portion only.

SOURCE: Hesse, J.L., Status Report on Polychlorobiphenyls in Michigan Waters, Michigan Water Resources Commission, (June 1973).

2.2.6 Cayuga Lake

In October 1970 lake trout were collected from Cayuga Lake in Ithaca, New York. Since these fish are marked each year when they are stocked in the lake their ages were accurately known. Table 2.2-14 shows the residues of PCB's identified along with the age, length, weight and sex of each fish. The peak heights of individual PCB isomers did not vary with the age of the fish indicating that there is no selective metabolism or storage of specific PCB isomers as the fish matures. However, the concentration of total PCB's does increase progressively with maturity. The variation in PCB concentrations among individual 11 or 12 year old fish may be due to differences among foraging, metabolic and excretary capabilities of the older fish.[1]

Table 2.2-14
Residues of PCB's in Cayuga Lake Trout as a function of maturity; j, juvenile.

Age (years)	Sex	Length (cm)	Weight (g)	PCB (ppm)
1	J			0.6
1	J			1.6
1	J			0.5
1	J			1.2
2	J	27.7	181	2.0
2	J	28.7	226	1.3
2	J	33.5	407	2.5
3	J	44.5	815	2.2
3	J	44.5	725	2.4
3	J	41.1	770	1.2
4	J	53.8	1310	3.5
4	J	50.3	1160	4.1
4	J	55.1	1359	5.1
5	M	61.0	2030	5.7
6	M	63.5	2440	3.4
6	M	66.4	2850	9.7
6	F	68.3	2310	8.6
7	M	63.5	2260	4.0
7	M	68.9	3300	5.5
7		59.7	1990	10.5
8	F	75.2	3390	17.5
8	M	71.6	2805	13.4
8	F	69.0	3300	4.5
9	F	71.2	3390	30.4
11	M	80.3	4200	12.4
12	M	71.6	2535	13.4
12	M	75.5	3120	26.2
12	F	70.6	3440	7.4

SOURCE: Sache, C.A., F.W. Sarum, W.D. Youngs and D.J. Lisk, Science, 177, 1191-1192, (1972).

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2.3 Data from Localized Monitoring Efforts - Rivers

2.3.1 Iowa - Mississippi River

Monitoring Iowa rivers for pesticides concentrations over a period of years indicated that chlorinated hydrocarbon insecticides used in row crop agriculture were being carried into the rivers by soil erosion. Since fish are excellent biological compositors, measurement of trace elements in their tissues can give a picture of the relative pollution of a river. Therefore a study of the pesticide levels in the eggs of some fish from locations in Iowa was undertaken in the spring of 1971.[1]

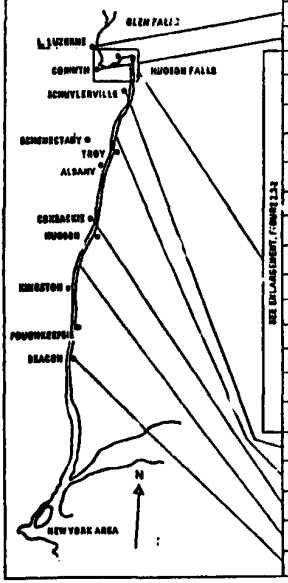
Two of the sites had measurable amounts of polychlorinated biphenyls. The PCB's detected match Aroclor 1254. The concentrations identified in the roe removed from the fish from these sites on the Mississippi were as follows:

Site	Species	Date Collected	Length of Fish	PCB Concentration in 15 grams of roe (ppm)
Near Northern border	Channel Catfish	06-04-71	23.4	1.5
	Largemouth bass	05-20-71	16.4	3.2
	Walleye	04-16-71	27.5	4.2
Near Southern border	Channel Catfish	04-16-71	24.0	0.9
	Largemouth bass	04-15-71	17.5	2.9
	Walleye	04-13-71	24.3	5.5

The levels of PCB's found in the same species from the two locations are approximately the same indicating that the major source of these industrial chemicals is upstream from both of these sites.

2.3.2 New York - Hudson River

The Hudson River in New York State provides an interesting ecosystem in which to study PCB's, primarily because it is the receiving water for effluents from two plants of the General Electric Co., at Hudson Falls and Fort Edward. These plants use PCB's in the production of transformers and capacitors. Summaries of extensive sampling during 1975 of the Hudson River are presented in Figures 2.3-1 and 2.3-2. The GE plants appear to be the major contributors to the PCB residue levels in the river. Although trace levels of PCB's are evident above the GE plant outfall location, levels in water and sediment at Station 1 located at the outfall of the Fort Edward plant were as high as any levels measured in the country. In the immediate area downstream from this outfall, PCB concentrations in water exceed the equilibrium solubility level by more than a factor of 10. Major effects on the levels in sediment are noted more than 35 miles downstream from the outfalls. The data indicate elevated concentrations in aquatic life far from the outfalls; fish samples approximately 40 and 66 miles downstream from the outfalls showed PCB levels in excess of 5 ppm while the same species showed only traces of PCB's directly upstream from the outfalls. Since sediment samples above the plants also show some concentration there does appear to be some PCB source upstream of the plants.



MILE POINT	DESCRIPTION	ESTIMATED PCB'S: WATER (PPM)	SEDIMENT (PPM DRY)	SMALL MOUTH* BASS (PPM)	WALLEYE* (PPM)	WHITE* SUCKER (PPM)	YELLOW* PERCH (PPM)	SNAILS (PPM)	WHITE* PERCH (PPM)
223.8	AT L. LUZERNE		6						
215.8	ABOVE CORINTH	<0.1	2.4	TRACE		TRACE	TRACE		
214.0	BELOW CORINTH		1.0						
208.9	ABOVE SHERMAN ISLAND DAM		4.8 C						
206.7	ABOVE GLEN FALLS LANDFILL		0.83	TRACE	TRACE	TRACE	TRACE		
204.6	BELOW GLEN FALLS LANDFILL		15.8						
195.9	AT PORTLAND CEMENT CO.		<0.30						
	STATION 6	<1.	6.0				17.0	1.0	
187.3	ABOVE BAKER FALLS DAM		4.8						
186.3	BELOW BAKER FALLS DAM		116. C						
	STATION 1	2000	5700					46	
	STATION 2	2.5	540.0						
	STATION 3	3.1	2000.0						
	STATION 4	<1	6.6					27.	
194.2	AT FT. EDWARD		147.9						
186.4	ABOVE THOMPSON ISLAND DAM		3707.2 C	122.5**	38.4**	41.0			
182.6	ABOVE SCHUYLERVILLE		70.5						
157.8	ABOVE WATERFORD		19	45.3	32.4	37.0			
124.9	NEAR CONSACKIE	<0.5		13.75**			4.10		10.7
	ABOVE KINGSTON						6.28		7.78
60.8	NEAR BEACON	<0.							

SEE ENLARGEMENT, FIGURE 2.3-2

C - MAXIMUM VALUE IN A CORE SAMPLE
* - VALUES ARE WEIGHTED AVERAGES OF SAMPLES OF FISH FROM 3 SIZE GROUPS (EXCEPT AS NOTED) TAKEN AT VARIOUS TIMES IN 1975
** - ONLY ONE FISH SAMPLED

SOURCES:
STATIONS 0-4: RADEAN, R.J. AND R.P. DAVIS, INVESTIGATION OF POLYCHLORINATED BIPHENYLS IN THE HUDSON RIVER, 1975, EPA REGION II
OTHER FISH DATA: NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION, DIVISION OF FISH AND WILDLIFE, 1975, ANALYSES OF PCB'S IN FISH FLESH
OTHER WATER DATA: NEW YORK STATE DEPARTMENT OF ENVIRONMENTAL CONSERVATION, DIVISION OF PURE WATERS, PCB MONITORING IN THE UPPER HUDSON RIVER BASIN, OCTOBER 1975

FIGURE 2.3-1 MAP OF HUDSON RIVER, NEW YORK, SHOWING DEVELOPED AREAS AND SAMPLING STATIONS WITH PCB LEVELS

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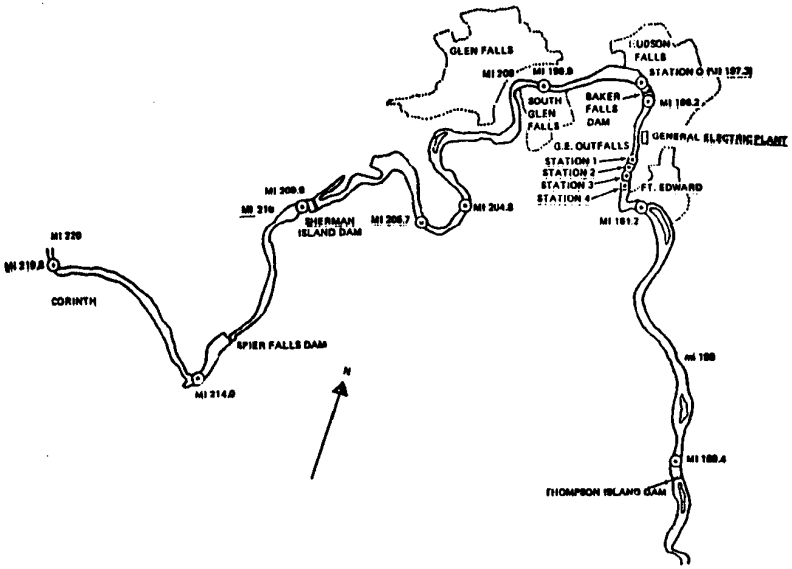

FIGURE 2.3-2 DETAILED MAP OF A PORTION OF THE HUDSON RIVER

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2.3.3 Maryland - Chester River

This study was conducted in the Upper Chesapeake Bay in 1971-1972;[1] the study area is shown in Figure 2.3-3 . The Bay and its tributaries receive chlorinated hydrocarbons as a result of industrial, agricultural and waste disposal activities occurring along the shorelines of these waterways and within the total watershed.

Sediment samples were collected approximately quarterly except for the last set which was taken early in order to collect data shortly after tropical storm "Agnes". The PCB's identified were almost exclusively Aroclor 1242. The first readings, taken in November 1971 ranged from 0-21 ppb with an average of 83 ppb. By April 1972 the average had climbed to 110 ppb with a range from 0-300 ppb. By June the readings taken ranged from 0-150 ppb with the average reduced to 53 ppb. After "Agnes" in July the average had again increased to 96 ppb with readings ranging from 0-180 ppb.

The explanation given for the variations is that during the early spring, massive amounts of suspended sediment from the Susquehanna River entered the Chester River and were carried upstream in the flow of water along the river bottom. The peak sediment loads from the Susquehanna generally come at the end of February, decreasing abruptly after the end of March due to a reduced flow rate of the river. An equilibrium net transport develops in the Chester River in April. In May, fine sediments were transported slowly into the Bay. However, over the year there is a movement of sediment along the river bottom into the river from the Bay. Thus, the upper Chesapeake Bay, i.e., the Susquehanna River is the major source of suspended sediments in the lower tidal portion of the Chester River.

The average values and ranges identified in biota of the Chester River taking into account variability due to seasonal fluctuations, distributional differences resulting from sample location, and species as well as individual uptake differences were as follows:

	Average (ppb)	Range (ppb)
Oysters	55	16-250
Soft shelled clams	58	13-180
Fish	18.5	2-570
Crabs	20	0.4-51

Based on data collected June 6, 1972 from the nine stations along the main river course it was determined that concentrations of PCB's decreased as a function of distance up river from its mouth at Love Point in its upstream direction at the rate of 4.20 ppb/Nautical mile.

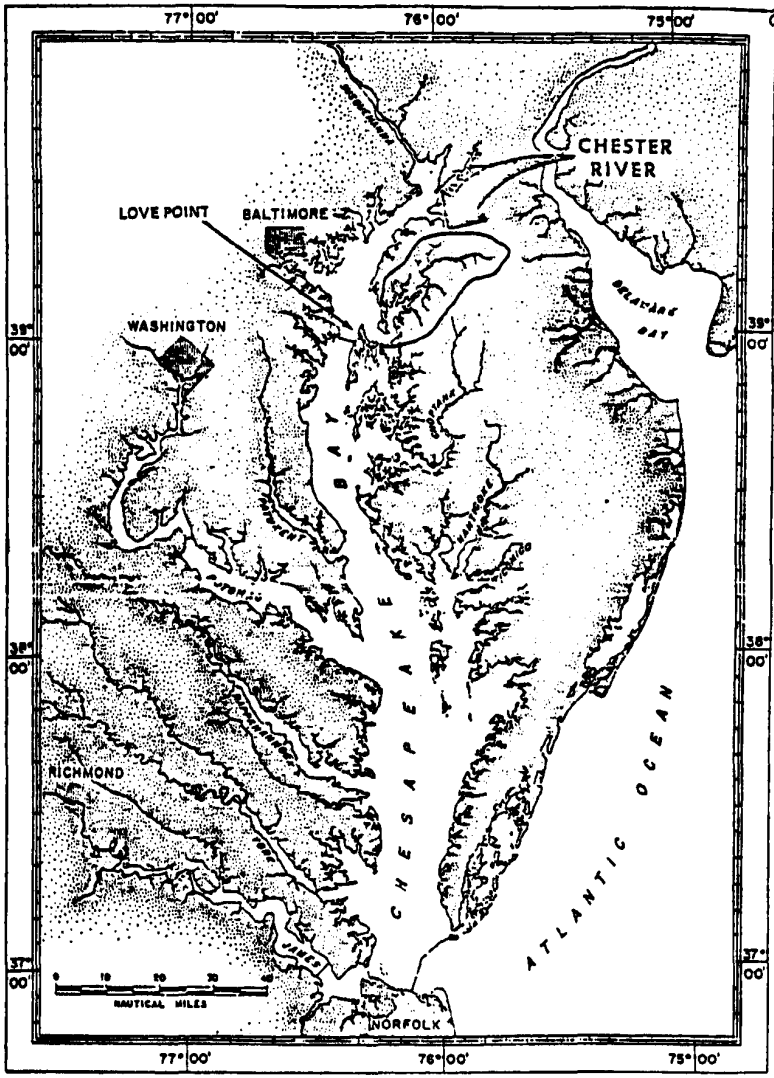

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FIGURE 2.3-3 CHART OF CHESAPEAKE BAY SHOWING MAJOR TRIBUTARIES AND LOCATION OF THE CHESTER RIVER.

2.3.4 Connecticut

In October 1974 the State of Connecticut intensively sampled bottom sediment in rivers and lakes thoughout the state. The results are shown in Figure 2.3-4, based on a computer mapping provided by the Connecticut District Office of the U.S. Geological Survey.[6] The data show PCB contamination to be both widespread and at high levels throughout the state, particularly around the highly developed areas of Hartford, New Haven, Stanford-Greenwich and all along the Housatonic River flowing from Massachusetts. The state had sampled mostly whole water for PCB's prior to 1974, with no appreciable residue levels discovered, but now has switched to a yearly bed material determination. The readings for the baseline period of October 1974 are to be updated yearly.

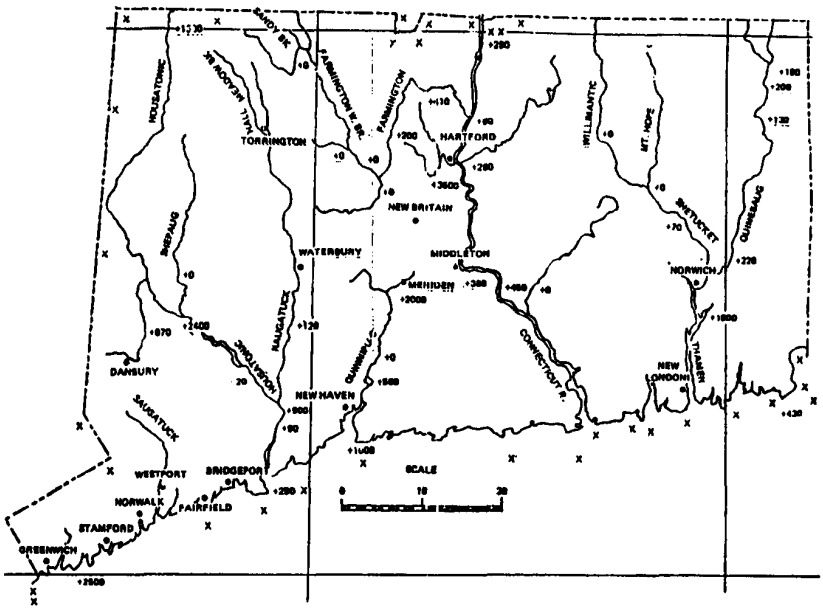

FIGURE 2.3-4 PCB CONCENTRATIONS IN BOTTOM DEPOSITS STATE OF CONNECTICUT, OCTOBER 1974 (ppb X 10)

C-63

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REFERENCES, SECTION 2.3

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6. Thomas, Chester E., Assistant District Chief, Connecticut District, U.S. Geological Survey, Water Resources Division, personal communication, July, 1975.

2.4. Data from Localized Monitoring Efforts - Marine Environment

2.4.1 Atlantic Ocean

In addition to the many PCB studies that have been conducted within the United States, a few studies have been carried out investigating the air, water and fish off the east coast of the United States. These studies, conducted between 1971 and 1974 covering Providence, Rhode Island; Grand Banks; Vineyard Sound; Georges Bank and Bermuda show PCB's to be extensively distributed over the Atlantic Ocean. The general sampling area is shown in Figure 2.4-1.

One of the few air studies that has been published was carried out by Harvey and Steinhauer[11] in 1973. Table 2.4-1 shows the sampling locations and Aroclor 1254 concentrations identified. The highest concentrations were in Vineyard Sound which is approximately 100-150 miles from the Boston, Hartford, New York, New Jersey complex. Over 1000 miles away at Grand Banks the concentrations were 100 times less than in Vineyard Sound. Figure 2.4-2 shows the relationship of atmospheric PCB levels with distance from these industrial areas. The seaward decrease appears to be exponential.

Later studies on PCB's in surface and subsurface waters were done by Harvey[12]. Although the concentration range across the northern North Atlantic is broad, from 1-150 ppt, the average was about 35 ppt in surface waters and 10 ppt at 200 meters depth. Looking at the lower latitudes it appears that the surface waters of the Sargasso Sea have slightly lower concentrations than other parts of the North Atlantic, averaging 27 ppt. The widespread PCB distribution implicates the atmosphere as a predominant mode of transport in the Atlantic with variations due to the seaslicks, localized rainfall or discharges from ships.

During the period of February-June 1973, PCB concentrations in the marine atmosphere of the Bermuda - Sargasso Sea and Providence, Rhode Island area were measured by Bidleman and Olney[1]. It is interesting to note that most of the PCB was trapped on the polyurethane foam collection surface suggesting that PCB's are in the atmosphere mainly as vapors rather than adsorbed onto particulate matter, or that they volatilize from the trapped particles collected on the glass fiber prefilter. Table 2.4-1 gives the locations and levels identified as Aroclor 1242 or 1248.

Sargasso Sea surface water was also measured by Bidleman and Olney[1] in 1973. From Table 2.4-2 it can be seen that the concentrations were higher in the surface layer than in the subsurface samples.

Follow-up by Harvey[12], et. al., indicated that PCB concentrations in North Atlantic surface waters declined forty fold from 1972 to 1974 presumably following the cessation of certain industrial uses of those compounds. However, Longhurst and Radford dispute the decrease stating that either the analytical methods were inaccurate or Harvey's original estimate of the total amount of dissolved PCB's in the upper 200 m was incorrect. Harvey and Steinhauer have agreed[17]

that their extrapolation may well have been improper and planned more sampling for 1975.

In 1971 a seafood monitoring program[18] was conducted on the East Coast of the United States. It is difficult to make interspecies comparisons but as can be seen in Table 2.4-3 the highest Aroclor 1260 level was in flounder from Jacksonville, Florida.

Table 2.4-1
PCB Concentrations Over the Western North Atlantic

Station	Sample volume (m^3)	ng/m^3
Bermuda	560	0.5=
	480	0.4=
	820	0.16=
	500	0.15=
	1070	0.59=
	1320	0.30=
	918	0.65=
	1950	0.62=
	1740	0.55=
	732	0.52=
	1300	0.61=
	860	0.21=
33°20'N, 65°14'W	300	1.6=
34°39'N, 66°15'W	267	0.79=
38°48'N, 69°14'W	222	0.72=
40°32'N, 70°20'W	196	0.83=
Georges Bank	105	1.4=
(41°40'N, 67°30'W)	675	0.82=
	660	0.58=
	655	0.61=
	640	0.80=
	650	1.60=
Vinuyard Sound	105	3.9=
(41°20'N, 70°50'W)	224	5.3=
Grand Banks	780	0.05=
(45°16'N, 52°08'W)	960	0.07=
	840	0.10=
	940	0.16=
	540	0.05=
Providence, R.I.	392	4.0=
	1071	2.1=
	744	5.8=
	76	9.4=

=Calculated as Aroclor 1254
=Calculated as Aroclor 1242 or 1248

SOURCE: Harvey, R.G. and W.G. Steinhauer, Atmospheric Environment, Nature, 252, 387-388 (November 29, 1974).

Table 2.4-2
PCB's Measured in Sargasso Sea Area, 1973

Location	Sample*	ppt**
29°56'N, 64°40'W	SM	11.2
	SS	3.6
30°45'N, 66°55'W	SM	4.9
	SS	<0.9
30°34'N, 66°59'W	SM	8.3
	SS	1.0
28°53'N, 65°07'W	SM	42.6 , 19.3
	SS	<0.9 , <0.9
29°56'N, 63°00'W	SM	3.8
	SS	<0.9
30°00'N, 64°30'W	SM	5.6
	SS	1.6
31°34'N, 63°49'W	SM	5.0
	SS	1.8
31°38'N, 63°57'W	SM	8.4
	SS	0.9

*SM - sample is surface microlayer
SS - sample is subsurface water

**PCB calculated as Aroclor 1260

SOURCE: Bidleman, T.F., and C.E. Olney, Science, 184, 516-518 (Feb. 8, 1974).

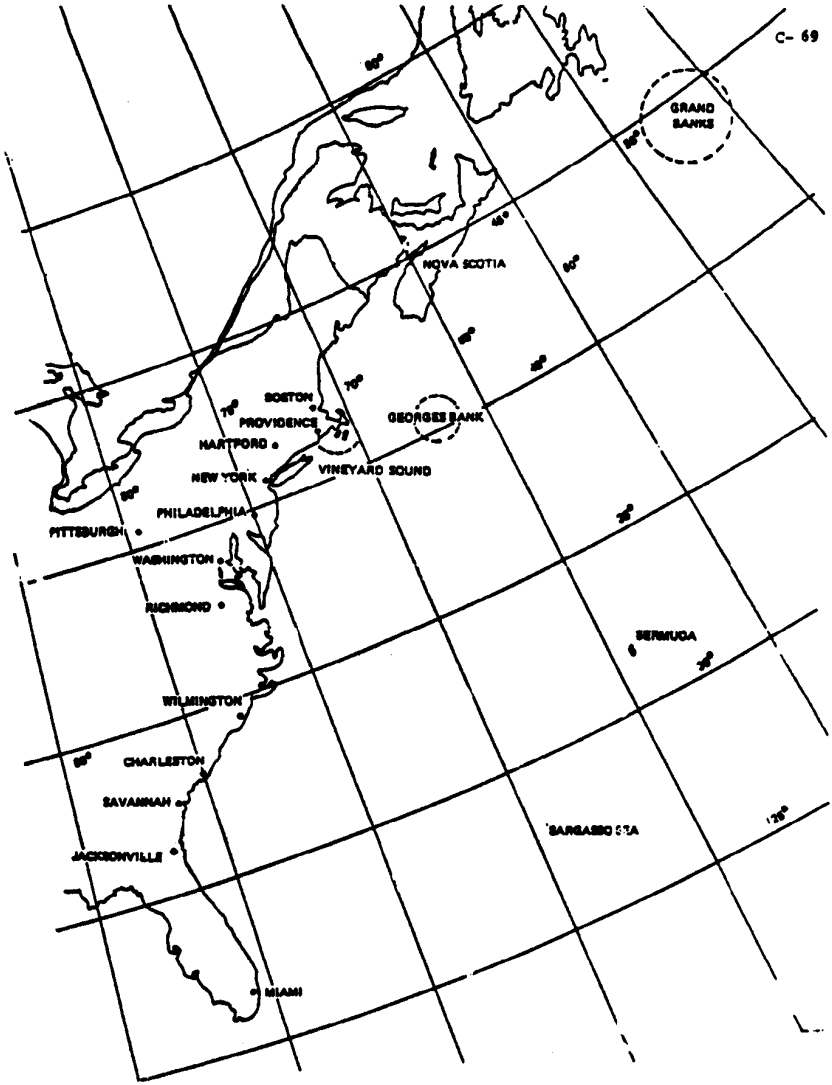

FIGURE 2.4-1 ATLANTIC OCEAN SAMPLING SITES

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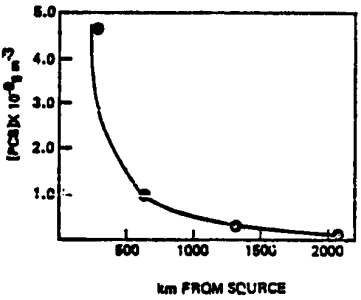


SOURCE:HARVEY, G.R. AND W.G. STEINHAUER, ATMOSPHERIC ENVIRONMENT 8, 777–782, (1974).

FIGURE 2.4-2 RELATIONSHIP OF ATMOSPHERIC PCB LEVELS WITH DISTANCE FROM INDUSTRIAL AREAS

Table 2.4-3
PCB Residues in Seafood

Species	Sample Quantity	Aroclor 1260 (ppm)
	JACKSONVILLE, FLA.	
Blue crabs	15	0.08
Shrimp	1 kg	0.60
Flounder	3	0.63
Weak fish	5	0.44
Drum	5	0.17
	SAVANNAH, GA.	
Blue crabs	16	—
Shrimp	0.5 kg	—
Pilot shrimp	0.5 kg	—
Oysters	15	—
Razorback clams	28	—
Squid	5	—
Flounder	4	—
	CHARLESTON, S.C.	
Blue crabs	10	—
Shrimp	1 kg	0.02
Oysters	>15	—
Squid	>15	—
Croaker	1	—
	MOREHEAD CITY, N.C.	
Blue crabs	15	—
Shrimp	1 kg	—
Oysters	24	0.02
Clams	20	—
Scallops	12	—
Flounder	3	—
Spanish mackerel	2	—
Weak fish	2	—
Blue fish	2	—
Fish meal	0.5 kg	0.11
	CHESAPEAKE BAY	
Blue crabs	25	0.02
Weak fish	3	0.49
Striped sea bass	3	0.02
Spot	5	0.10
Herring	10	—
	DELAWARE BAY	
Blue crabs	15	0.13
Flounder	2	0.07
Weak fish	5	0.23
Striped bass	2	0.07
Mullet	5	0.21
Croaker	1	0.50

SOURCE: Markin, G.P., J.C. Hawthorne, J.L. Collins and J.H. Ford Pesticides Monitoring Journal, 7, 139-143, (March 1974).

2.4.2 Bay of Fundy

As early as 1969, PCB residues were identified in harbour porpoises from the Bay of Fundy, located in the northeast section of Maine[1]. Zitko, et. al.[1b], identified Aroclor 1254 in a number of different fish ranging from 0.07 ppm in the muscle of basking shark and 0.21 ppm in the muscle of sea raven to 3.55 ppm in Herring Oil and 218 ppm in the liver of white shark. The species studied and the levels measured are listed in Table 2.4-4.

Table 2.4-4
PCB and Chlorinated Hydrocarbon Pesticides in Aquatic Animals from the Bay of Fundy - Gulf of Maine Area.

Species	Tissue	Aroclor 1254, ppm
Herring	whole fish	0.34
Mackerel	muscle	0.35
Plaice	muscle	0.38
White hake	muscle	0.44
Ocean perch	muscle	0.32
Cod	muscle	0.55
Sea raven	muscle	0.21
	viscera	0.73
Basking shark	muscle	0.07
	liver	1.07
White shark	muscle	0.77
	liver	218
Bluefin tuna	muscle	1.54
Herring oil		3.55
Fishmeal		0.54
Double-crested cormorant	eggs	43.5
		17.2
	muscle	3.38
	liver	2.13
	subcutaneous fat	38
	abdominal fat	52
Herring gull	eggs	12.6
		5.54
	muscle liver	5.06
		6.50
	subcutaneous fat	75

SOURCE: Zitko, V., O. Hutzinger and P.M.K. Choi, Environmental Health Perspectives, 47-50, (April 1972).

2.4.3 Gulf of Mexico - Caribbean Sea

Runoff from approximately two-thirds of the United States and one-half of Mexico enters the Gulf of Mexico and is swept generally westward to be trapped in the Western Gulf for as much as 100 years. Therefore a buildup of man-made toxic chemicals is possible in the Western Gulf especially since there is a heavy concentration of chemical manufacturers in Louisiana and Texas.[7]

The earliest samples collected in this area were brown Pelicans collected in 1969 from the Atlantic and Gulf Coast of Florida and in 1970 from Florida Bay. The highest levels, 1.0-7.5 ppm came from the Atlantic Coast while those from the Gulf Coast ranged up to 4.2 ppm and those from the Keys in the Bay only had concentrations up to 2.5 ppm. The birds represented a wide range of ages and both sexes, but the immature and adult birds always had higher residues than the young.[1]

In 1971 a seafood monitoring program[10] for the insecticide mirex was set up. Three of the stations were located in the Gulf of Mexico and identified Aroclor 1260 as indicated in Table 2.4-5. Due to the different kinds of species involved a comparison of levels is difficult to prepare. However, it is clear that the levels in samples from Mobile Bay, Alabama ranging from 0.01 to 0.40 ppm were higher than those from the Mississippi Sound where the highest level was only 0.20 ppm.

Fish and other marine organisms were collected in the Gulf of Mexico and the Caribbean Sea in May and October 1971 as shown in Figure 2.4-3. While PCB's were detected in nearly all the samples analyzed the levels were generally low. The samples from coastal waters generally had higher levels than samples from open waters. This same observation holds true for sea plankton. Of the six samples containing PCB's above 100 ppb wet weight four of them were near Coastal areas.[9]

Groupers were also collected[8] since they tend to spend their post larval life in one locality and should therefore yield data more representative of a particular area. Figure 2.4-3 shows the sampling locations. The levels are generally low although the highest levels do come from the western Gulf. Note also that at Anton Lizardo the levels in the groupers increase with increasing size as can be expected since the fat content of the fish increases with increasing size and age of the specimen. Table 2.4-6 presents some of the levels identified in 1971.

Giam et. al.[6] again took measurements from stations in the Gulf of Mexico in 1973-1974 as indicated in Figure 2.4-3 and Table 2.4-7. The concentrations in biota are lower than they were in the 1971 survey with an average according to Giam of about 20 ppb reduced from 60 ppb. It must be noted, however, that these stations were all close to the southern coast of the U.S. with no samples collected in the Western Gulf.

Table 2.4-6
Biota and Plankton from the Gulf of Mexico - 1971

Station	Biota	Weight (lbs.)	Standard Length (mm)	PCB's (ppb)
1	Flounder*			32
2	Flounder*			36
3	Flounder*			59
4	Flounder*			34
	Plankton†			678
5	Crustacean*			151
	Crustacean*			22
6	Shrimp*			167
7	Rock Shrimp*			6
	Squid*			40
8	King Mackerel muscle*			34
	Plankton†			<3
	Tuna muscle*			58
9	Tuna muscle*			36
10	Plankton†			100
11	Plankton†			30
12	Plankton†			<3
13	Plankton†			1055
14	Plankton†			44
15	Plankton†			42
16	Plankton†			191
27	Grouper‖	3.1	398	110
		2.9	378	32
		1.9	306	14
		1.9	287	14
		0.7	222	12
28	Grouper‖	4.9	414	33
		4.8	416	12
		2.8	338	10
29	Grouper‖	10.0	514	7
		3.7	403	7
		2.9	354	6
30	Grouper‖	6.0	495	5
		4.8	459	3
		1.6	315	3
		3.0	385	14
		2.4	339	6
31	Grouper‖	3.3	352	81
32	Grouper‖	18.0	681	220

*Giam, C.S., A.R. Hanks, R.L. Richardson, W.M. Sackett and M.K. Wong, Pesticides Monitoring Journal, 6, 139-143, (Dec. 1972).

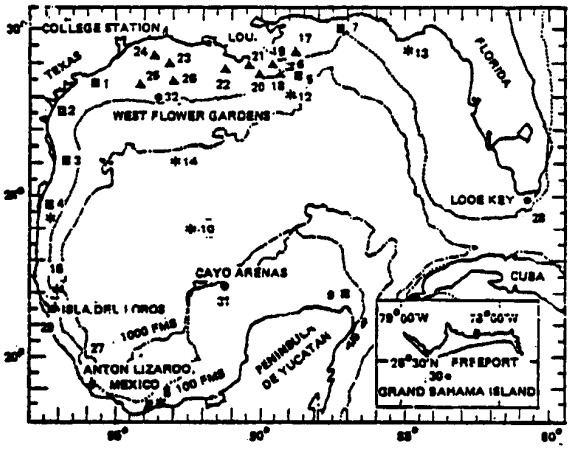
†Giam, C.S., M.K. Wong, A.R. Hanks, W.M. Sackett and R.L. Richardson, Bulletin of Environmental Contamination and Toxicology, 9, 376-382, (1973).

‖Giam, C.S., R.L. Richardson, D. Taylor and M.K. Wong, Bulletin of Environmental Contamination and Toxicology, 11, 189-192, (1974).

Table 2.4-5
PCB Residues in Seafood

Species	Sample Quantity	Aroclor 1260 (ppm)
	MISSISSIPPI SOUND	
Crabs	15	0.07
Shrimp	1 kg	——
Squid	1 kg	0.03
Flounder	3	0.03
Speckled sea trout	3	0.20
Spanish mackerel	3	0.06
Hook fish	5	0.12
Atlantic whiting	5	0.10
Fish oil	1 liter	0.02
Fish meal	1 kg	0.02
Ground fish (unprocessed)	1 kg	0.04
	MOBILE BAY, ALA.	
Blue crabs	15	0.12
Shrimp	1.3 kg	0.01
Oysters	20+	0.03
Squid	10	0.04
Flounder	3	0.11
Speckled sea trout	1	0.40
Spanish mackerel	2	0.18
Hook fish	3	0.37
Atlantic whiting	5	0.11
Croaker	3	0.33
Red snapper	2	0.14
Mullet	5	0.11
Anchovy	>20	0.08
Shad	>10	0.09
	TAMPA BAY, FLA.	
Crabs	10	0.03
Hook fish	5	0.83
Mullet	3	0.31

SOURCE: Markin, G.P., J.C. Hawthorne, J.L. Collins, and J.H. Ford, Pesticides Monitoring Journal, 7, 139-143, (March 1974).



■ BIOTA 1971

● GROUPERS 1971

▲ BIOTA, WATER, SEDIMENT, 1973–1974

✱ PLANKTON 1971

FIGURE 2.4-3 GULF OF MEXICO SAMPLING SITES

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Table 2.4-7
Concentrations of PCBs in Selected Samples
from the Gulf of Mexico, 1973-74

Station	Biota (ppb)	Water (ppt)	Sediment (ppb)
7	20	4.1	<0.2
18	11	2.1	35.0
19	22	1.7	33.0
20	23	—	<0.2
21	37	1.7	<0.2
22	14	2.1	—
23	6	2.4	—
24	11	1.3	—
25	94	0.8	<0.2
26	68	2.7	—

SOURCE: Giam, C.S., J.S. Chan, J.P. Kakareka and G.S. Neff, Trace Analyses of Phthalates and Chlorinated Hydrocarbons in Gulf of Mexico Samples.

2.4.4 California

According to Hom and Risebrough[14] PCBs have become a significant component of the marine food webs of southern California. They have been associated with a high incidence of premature births among sea lions and eggshell thinning with consequent reproductive failure in fish-eating birds.

One study collected different levels of sediment from the Santa Barbara Basin.[14] The dated and analyzed sediments indicated that the deposition of PCB's began about 1945 probably as a result of the rapid increase in PCB use as electrical insulating fluids and paint additives during World War II. Through 1967 there was no indication of a leveling off in the rate of PCB deposition. Levels in the 1940-1945 layer were 31 ppb increasing to 49 ppb in the 1947-1952 layer, 66 ppb in the 1955-1960 layer and 103 ppb in the 1962-1967 layer.[14]

Risebrough[23] has suggested that the observation of PCB's in the sea indicate that they are dispersed by wind currents. Table 2.4-8 shows the distribution of PCB residues in several collections of marine fish collected from the Coastal waters of southern California in late 1965 and early 1966 and in marine birds collected in late 1966. Note that the levels in the birds 6.08-109 ppm were higher than those in fish, N.D.-1.2 ppm. Petrels and shearwaters breed on remote islands spending their entire lives at sea. They do not dive for fish but feed primarily upon organisms obtained at or near the surface where aerial fallout could be expected to retain temporarily the water-insoluble chlorinated hydrocarbons components. Two Peregrine Falcons showed PCB levels ranging from 1.5 ppm, net weight, in the brain of an immature bird to 1,980 ppm lipid weight in the carcass of an adult bird.

Analyses of Western gull eggs[22] show that eggs from San Francisco Bay, with levels from 24-950 ppm contained more PCB's than eggs from the Farallon Islands 27 miles west of the Golden Gate Bridge with levels from 12-1010 ppm. These were both higher than eggs from Baja California where the levels were 1.2-471 ppm.

Concern over reproductive failure of the Double Crested Cormorant led to a study in 1969 of Cormorant eggs from three locations in southern California. The levels identified ranged from 12-1,100 ppm in the yolk lipids.[19] A similar concern also led to a study of Brown Pelicans on Anacapa Island in May 1969. Fat samples from six adult birds and one immature bird ranged from 77 to 366 mg/gm.[13]

Early data collected by Munson[19] in 1970 from San Diego and Orange Counties showed levels in aquatic biota ranging from <2.0 to 8.8 ppm lipid weight (N.D. - 1.0 wet weight) in San Diego and 0.62 - 38 ppm lipid weight (0.008 to 1.4 wet weight) from Orange County. However, the difference in residue levels may be due to a different type of uptake mechanism since the samples from Orange County were invertebrates while those from San Diego were fish.

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In addition to the studies of Double Crested Cormorants and Brown Pelicans, Faber et. al,[9] studied Common Egrets and Great Blue Herons. The PCB levels ranged up to 15 ppm in the brain and 93 ppm in the livers of adult egrets from the Audubon Canyon Ranch. Fish which the birds might feed on from Bolinas Lagoon showed levels from 0.072-0.079 ppm.

In order to assess the potential contamination from chlorinated hydrocarbons the U.S. Geological Survey initiated a study in February 1972 of the San Francisco Bay. Bottom material was collected from 26 streams that discharge into San Francisco Bay. The results of the analyses are given in Figure 2.4-4 illustrating the widespread distribution of PCB's in the San Francisco Bay area. The readings from Steven Creek of 180 ppb and from Alamitos Creek, of 610 ppb were higher than anticipated since neither area had any apparent industrial or commercial development. Despite the extreme range up to 1400 ppb there was no significant difference between the average residue of streams discharging into the Bay south of San Francisco and those discharging into the Bay north of San Francisco.[16]

Studies of Waste Water Treatment Plants in the Southern California area will be presented in Section 2.5.3.4.

Table 2.4-8
Polychlorinated Biphenyl (PCB) Residues
in Marine Fish and Marine Birds, 1965-1966

Species Locality,	PCB
Northern Anchovy	
Terminal Island	1.0
Shiner Perch	
San Francisco Bay	0.4-1.2
English Sole	
San Francisco Bay	0.05-0.11
Monterey	0.04
Jack Mackerel	
Channel Islands	0.02
Hake	
Puget Sound	0.16
Channel Islands	0.12
Bluefin Tuna	
Body muscle	0.04
Liver	0.04
Yellowfin Tuna	
Liver	0.04
Skipjack Tuna	
Liver	0.1
Cassin's Auklet	0.16
Ancient Murrelet	0.15
Fulmar	0.08
Fulmar	0.34
Red Phalarope	0.10
Rhinoceros Auklet	0.36
Slender-billed Shearwater	2.1
Sooty Shearwater	1.2
Sooty Shearwater	0.9
Peregrine Falcon	
Breast muscle, second year	
Female, migrant from Artic	22
Breast muscle, immature	
California	10.5
Breast muscle, adult	
female, California	109

SOURCE: Risebrough, R.W., Chlorinated Hydrocarbons in Marine Ecosystems.

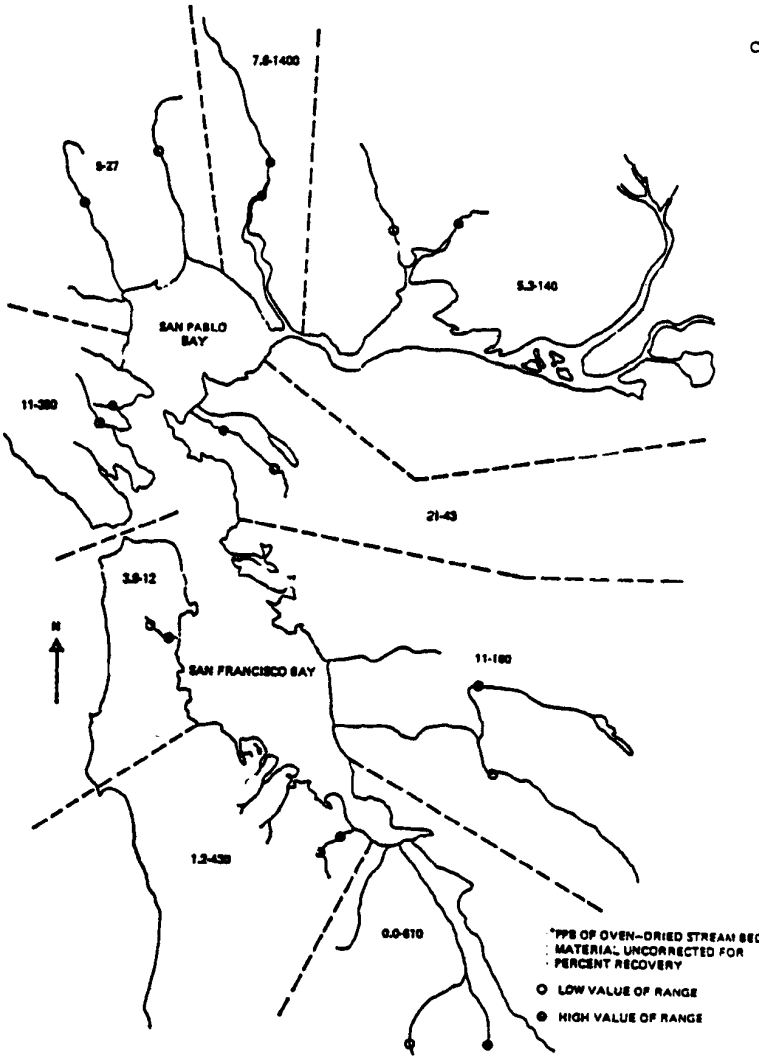


FIGURE 2.4-4 PCB'S IN SAN FRANCISCO BAY AREA STREAM BEDS

2.4.5 Escambia Bay, Florida

The first detection of Aroclor 1254 residues in Escambia Bay was in oysters in April 1969.[3] Later sampling also showed residues in water, sediment, fish, blue crabs and shrimp as indicated in Table 2.4-9. One source was apparently an industrial outfall which had had accidental leakage of a heat exchange fluid. Fish, shrimp and crabs contained higher concentrations than oysters but are also more mobile and therefore not as useful as monitors for a particular area.

Figure 2.4-5 identifies the sampling locations (stations 1-7). Less than 0.1 ppb occurred in the water at station 2 but it was not detected in lower bay water. Leaching from sediments is presumably the cause of the continued presence of Aroclor 1254 in the river water. Sediment samples taken near the outfall reached 486 ppm in August 1969.[3]

Pink shrimp collected at the same time from the bay were found to contain whole body residues of Aroclor 1254 as high as 14 ppm.[20] Residues in seven composite samples of at least five shrimp ranged from 0.6 to 120.0 ppm. Fiddler crabs collected in April 1970 from the lower Escambia River and Upper Escambia Bay had individual whole body residues of 0.45 to 1.5 ppm.

The largest accumulations were found in the sediments with the maximum residue of 61 ppm observed in the River at the outfall from the industry. The maximum in the Bay of 30 ppm was found near the mouth of the river. Although Aroclor was not detected in sediments collected above the plant, soil samples from the bank near the mouth of the river downstream from the source 6.5 km had 1.4 to 1.7 ppm. Subsequent samplings from three stations in the bay showed little change in the chemical even after nine months. Table 2.4-9 shows the levels identified for these sediment collections as stations 8-20.[20]

The amount of PCB in sediment samples appeared to decrease after the initial February 1970 survey.[21] The decrease is especially noticeable in December 1970 and October 1971. In general, the residues identified in 1971 were about one-tenth the 1970 values. Cores taken in a 1972 survey generally indicated less PCB than in 1971. Table 2.4-9 identifies the readings taken for these three years as stations 21-23.

Later surveys of biota from the estuary showed levels to remain relatively high. Table 2.4-10 compares residues in the same species or those occupying similar trophic levels captured on the same day in Escambia and East Bays; the East Bay site being about 35 kilometers from the original source of the material. Although sand seatrout and Atlantic cutlassfish could be expected to have the highest residues since they are predators the highest levels were actually found in silversides whose diet consists mainly of plankton. Note also that the concentrations in species from Escambia Bay were 5 to 10 times greater than those found in East Bay but that Aroclor 1254 was found even in species captured distant from the original source of PCB.[21]

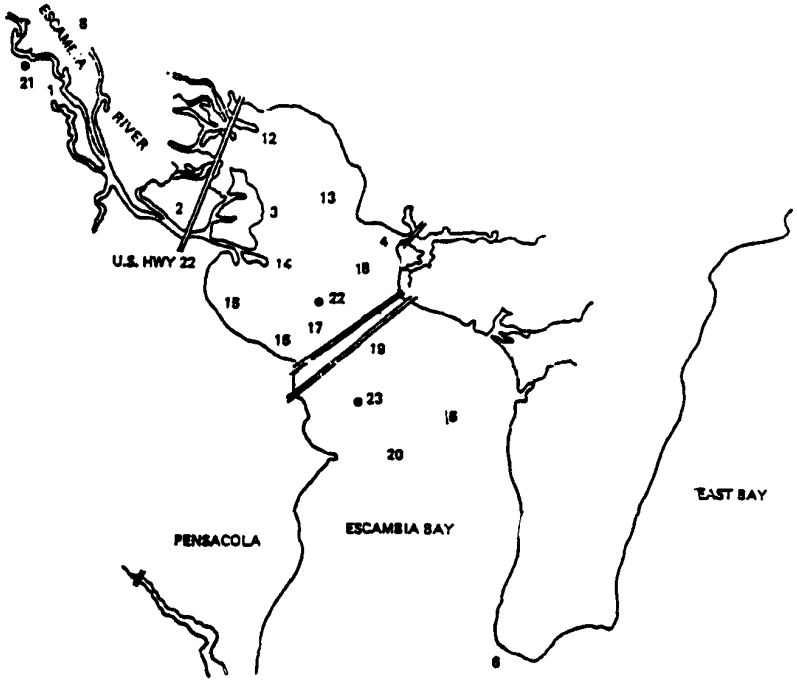
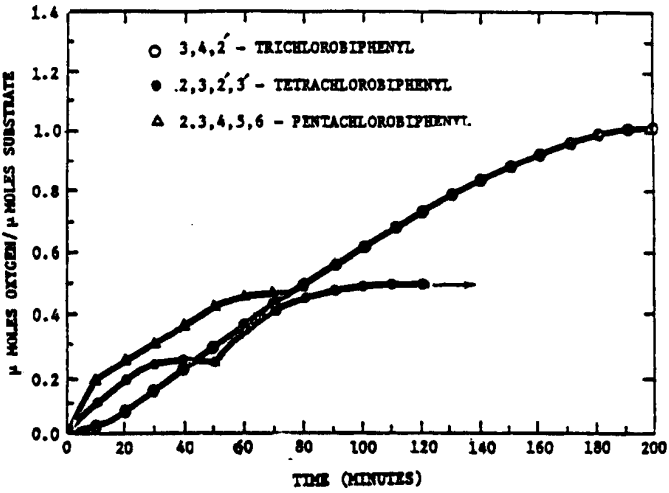


FIGURE 2.4-5 ESCAMBIA BAY SAMPLING STATIONS

Table 2.4-9
Residues of Aroclor 1254 in Samples from Escambia Bay and River

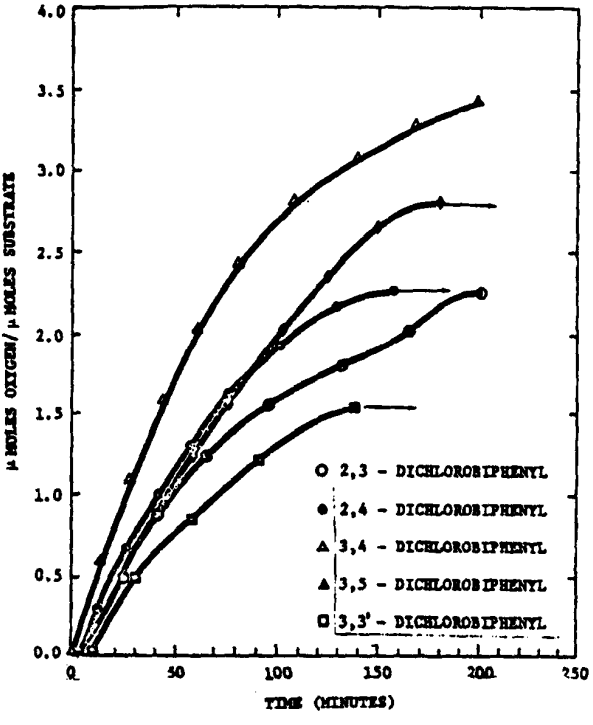
Location	Sample	Residues of Aroclor 1254 (ppm) 1969	1970	1971	1972
3	Speckled trout	20.0			
3	Flounder liver	184.0			
4	Menhaden	5.7			
4	Menhaden	11.0			
4	Menhaden	12.0			
4	Flounder liver	76.0			
4	Flounder muscle	4.5			
4	Flounder gills	19.0			
4	Croaker	12.0			
4	Pinfish	10.0			
3	Shrimp	2.5			
3	Blue crab	7.0			
3	Blue crab	6.3			
4	Shrimp	1.5			
4	Blue crab	1.0			
1	Sediment	486			
2	Sediment	<.03			
6	Sediment	1.7			
8	Sediment		N.D.		
9	Sediment		61.0		
10	Sediment		5.7		
11	Sediment		4.1		
12	Sediment		1.9		
13	Sediment		1.8		
14	Sediment		30.0		
15	Sediment		4.2		
16	Sediment		3.3		
17	Sediment		4.9		
18	Sediment		0.6		
19	Sediment		2.5		
20	Sediment		1.4		
21	Sediment 0-2 in.		78.0	8.1	0.97
	Sediment 2-4 in.		30.0	0.12	5.8
	Sediment 4-6 in.		6.1	N.D.	——
	Sediment 6-8 in.		0.4	N.D.	——
22	Sediment 0-2 in.		10.0	0.91	0.14
	Sediment 2-4 in.		11.0	N.D.	N.D.
	Sediment 4-6 in.		15.0	N.D.	N.D.
	Sediment 6-8 in.		20.0	N.D.	——
	Sediment 8-10 in.		18.0	N.D.	——
	Sediment 10-12 in.		1.2	N.D.	——
23	Sediment 0-2 in.		0.19	0.19	——
	Sediment 2-4 in.		0.08	N.D.	——
	Sediment 4-6 in.		0.02	N.D.	——
	Sediment 6-8 in.		N.D.	N.D.	——

SOURCE: Nimmo, D.R., D.J. Hansen, J.A. Couch, N.R. Cooley, P.R. Parrish and J.I. Lowe, Toxicity of Aroclor 1254 and its Physiological Activity in Several Estuarine Organisms, (unpublished).



Oxidation of tri, tetra, and penta chlorobiphenyls by Achromobacter pCB.

Figure E.3 (from ref. 300)



Oxidation of dichlorobiphenyls by Achromobacter pCB.

Figure E.2 (from ref. 300)

Table E.1 PERCENTAGE LOSS OF PCBs AFTER A GIVEN PERIOD OF TIME (from ref. 82)

Compound	% Degradation/Days	
	Nocardia spp.	Pseudomonas spp.
2,4'-Dichlorobiphenyl	70/7	60/73
2,4'-Dichlorobiphenyl + biphenyl	100/7	100/73
4,4'-Dichlorobiphenyl	nil/121	50/15
4,4'-Dichlorobiphenyl + biphenyl	nil/121	50/10
2,3-Dichlorobiphenyl	67/8	—
2,3-Dichlorobiphenyl + biphenyl	64/8	—
3,4-Dichlorobiphenyl	80/8	—
3,4-Dichlorobiphenyl + biphenyl	100/8	—
2,3,2'-Trichlorobiphenyl	50/7	—
2,3,2'-Trichlorobiphenyl + biphenyl	95/7	—
2,3,4'-Trichlorobiphenyl	94/7	—
2,3,4'-Trichlorobiphenyl + biphenyl	100/7	—
2,5,4'-Trichlorobiphenyl	nil/73	15/73
2,5,4'-Trichlorobiphenyl + biphenyl	60/73	60/73
3,4,3'-Trichlorobiphenyl	76/12	—
3,4,3'-Trichlorobiphenyl + biphenyl	70/12	—
2,4,6-Trichlorobiphenyl	nil/12	nil/84
2,4,6-Trichlorobiphenyl + biphenyl	nil/12	nil/84
2,4,2',4'-Tetrachlorobiphenyl	nil/9	—
2,4,2',4'-Tetrachlorobiphenyl + biphenyl	nil/9	—
2,4,6,2'-Tetrachlorobiphenyl	nil/9	—
2,4,6,2'-Tetrachlorobiphenyl + biphenyl	nil/9	—
2,3,4,5,2',3'-Hexachlorobiphenyl + 2,3,2'- and 2,3',4'-trichlorobiphenyl	nil	—
2,3,4,5,2',3'-Hexachlorobiphenyl + 2,3,2'- and 2,3',4'-trichlorobiphenyl +biphenyl	50/11	—

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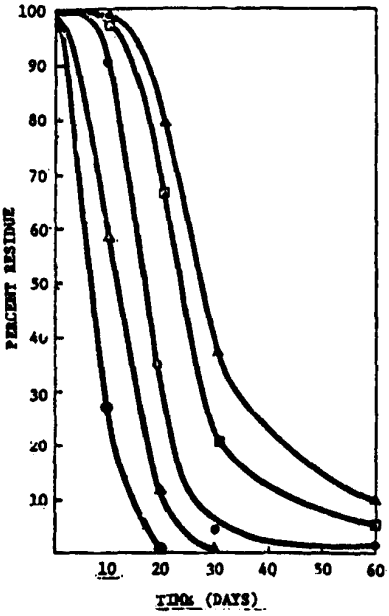
Table E.2
(from ref. 82)

PERCENTAGE DEGRADATION

	Aroclor 1242		Aroclor 1016	
Time (days)	Nocardia spp.	Pseudomonas spp.	Nocardia spp.	Pseudomonas spp.
52	88	76	96	91
100	95	85	>98	>96

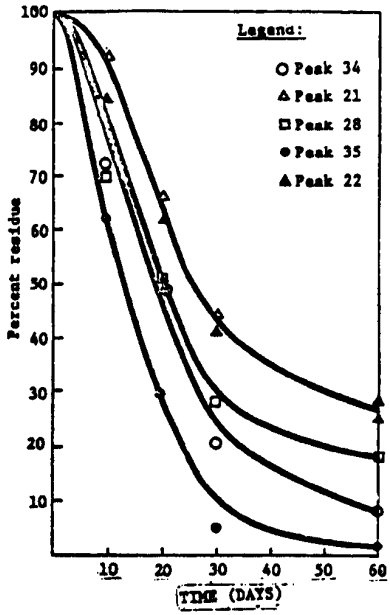


Legend:

- ○ Peak 13 (2 Cl)
- △ Peaks 15 and 18 (3 Cl)
- □ Peak 10 (3 Cl)
- ● Peaks 11 and 17 (3 Cl)
- ▲ Peaks 9 and 12 (3 Cl)

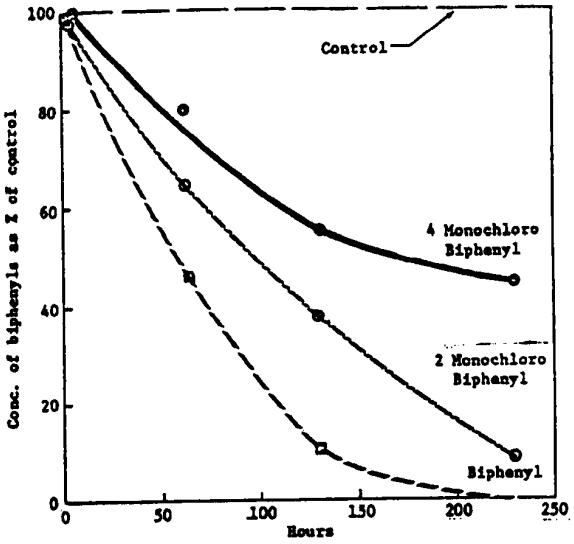
Degradation of typical di- and tri-chlorobiphenyl components of Aroclor 1242 following exposure to Nocardia spp.

Figure E.4 (from ref. 82)



Degradation of typical tetra-chlorobiphenyl components of Aroclor 1242 following exposure to Nocardia spp.

Figure E.5 (from ref. 82)



Concentrations of biphenyl, 2-chlorobiphenyl and 4-chlorobiphenyl in the growth medium after bacterial degradation during three incubation periods.

Figure E.6 (from ref. 76)

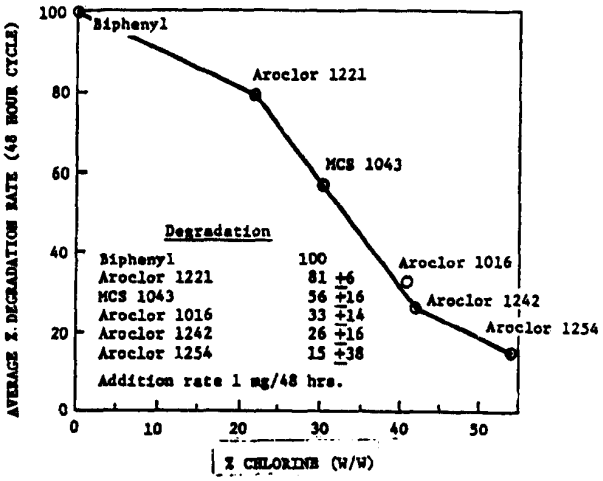
The slower degradation of more highly chlorinated PCBs is illustrated by the observation of Vaight as reported in Wong and Kaiser (76) that Aroclor 1242 concentration decreased at a faster rate than Aroclor 1260 in lake water, but no data were given. Tucker et al. (303) found an inverse relationship between degradation rate and chlorine content in commercial PCBs (see Figure E.7) using supernatant samples from a semi-continuous activated sludge technique*.

Effluent from a secondary sewage treatment laboratory system contained relatively small amounts of the seeded Aroclor 1254, with a considerable PCB accumulation in the sludge (304). By sampling only the supernatant, Tucker et al. (303) may have mistaken PCB removal to sludge through fat dissolution, adsorption on particle surfaces, and microbial ingestion, for degradation.

Other investigations using naturally occurring microorganisms have produced results indicating no metabolism of chlorinated biphenyls. Fries, as reported in Hutzinger et al. (4), found no preferential degradation of Aroclor 1254 components in silage after several months of normal fermentation.

In an experiment using 0.1 ppm Aroclor 1260 added to natural river and marine waters, Oloffs et al. (41) could detect no metabolites;

* This procedure consisted of mixing activated sludge cultures from a sewage treatment plant with synthetic sewage and allowed to acclimate for several weeks before adding polychlorinated biphenyl mixtures. Aeration was interrupted for sampling and at the end of a cycle when the sludge was allowed to settle before withdrawing supernatant. After replacement of the fluid with tap water, the cycle was reinitiated by adding synthetic sewage and PCBs.



Semi-continuous activated sludge primary biodegradation rates of commercial PCBs as a function of the weight percent chloride.

Figure 3.7 (from ref. 303)

but parent compounds were recovered in decreased concentration. Two mechanisms were postulated for Aroclor 1260 loss from water: codistillation and evaporation. Although 0.1 ppm Aroclor 1260 evinced a slight enhancement of bacterial growth, metabolic breakdown probably would be very slow at the natural temperatures maintained (7 and 16°C), one possible explanation for the lack of detectable metabolites.

Cultures isolated on biphenyl media can provide both induced bacteria and greater numbers of the adapted organisms. Wong and Kaiser (76) found that only 1% of the bacteria from nutrient agar could grow on 0.01% Aroclor 1221 enriched agar. Baxter et al. (82) suggested that degradation patterns of Aroclor 1242 could be explained in terms of adaptation of Nocardia sp. grown on biphenyl. Thus, little further adaptation was necessary to degrade mono- and diCBs, but for more highly chlorinated components, increasing adaptation may be required (see Figures E.4 and E.5).

Soil microorganism metabolism of PCBs appears to depend on soil type (4, 305). No indication of any metabolism was found in soil alone or soil enriched with cattle manure in which single chlorobiphenyls (4,4'-di; 2,5,2',5'-, 2,6,2',6'-, 3,4,3',4'-, and 3,5,3',5'-tetra; 2,4,5,2',5'-penta; and 2,4,5,2',4',5'-, and 2,4,6,2',4',6'-hexaCB) were incubated under flooded conditions in the laboratory for one month (306). Under the same conditions, p,p'-DDT degrades almost completely; therefore, the degradation pathways for these compounds are not the same.

E.2. Metabolism by Invertebrates

Metcalf et al. (45) studied the metabolism of three representative chlorobiphenyls in a model ecosystem which included several invertebrate animals. Their findings in the salt-marsh caterpillar (Estimmene acrea) are shown in Table E.3. Although 76% of the tri-CB was metabolized and excreted as polar degradation products, only 4.6% and 2.1% of the tetra- and penta-CBs were so dagraded. Similar results were obtained with a mosquito larva (Culex pipiens) and a snail (Physa) (Table E.4). Small amounts of non-polar metabolites were found also, especially in the case of the tri-CB: much of this non-polar material was retained in the tissues of the organisms and not excreted (Tables E.3 and E.4).

E.3. Metabolism by Fish

Table E.4 also gives the results of Metcalf et al.'s study of metabolism of the same three representative chlorobiphenyls in mosquito fish (Gambusia). As in the case of the invertebrates, a substantial fraction of the tri-CB was metabolized, but only insignificant fractions of the tetra- and penta-CBs. Metcalf et al. (45) define a "biodegradability index": the ratio of polar to non-polar metabolites. This index ranged from 0.17 to 0.60 for the tri-CB, indicating substantial metabolism to polar products, but was less than 0.082 for all organisms for the tetra- and penta-CBs, indicating negligible biodegradation (Table E.5).

Table E.3 (from ref. 45) *Metabolism of ^{14}C radiolabeled compounds by salt marsh caterpillar*[a]

	Body	Feces
A. 2,5,2'-trichlorobiphenyl total ^{14}C (%)	8.66	91.34
Unknown I (R_f 0.53[a])	0.64	–
trichlorobiphenyl (R_f 0.43)	5.84	8.91
Unknown II (R_f 0.31)	0.27	0.37
Unknown III (R_f 0.13)	0.05	0.12
Unknown IV (R_f 0.05)	0.10	4.67
Unknown V (R_f 0.02)	0.11	0.92
Polar (R_f 0.0)	1.65	76.35
B. 2,5,2',5'-tetrachlorobiphenyl total ^{14}C (%)	78.68	21.32
tetrachlorobiphenyl (R_f 0.50[a])	75.60	15.08
Unknown I (R_f 0.41)	0.99	1.36
Unknown II (R_f 0.05)	0.13	–
Unknown III (R_f 0.03)	trace	0.20
Polar (R_f 0.0)	1.96	4.64
C. 2,5,2',4',5'-pentachlorobiphenyl total ^{14}C (%)	75.86	24.14
pentachlorobiphenyl (R_f 0.53[a])	74.00	20.70
Unknown I (R_f 0.46)	0.74	0.74
Unknown II (R_f 0.39)	0.62	0.56
Unknown III (R_f 0.03)	0.08	0.08
Polar (R_f 0.0)	0.42	2.06
D. 2,2-*bis*-(*p*-chlorophenyl)-1,1-dichloroethylene (DDE) total ^{14}C (%)	80.59	19.41
DDE (R_f 0.49[a])	76.88	19.37
Polar (R_f 0.0)	3.71	0.04

[a]TLC with hexane (Skellysolve B, bp 60-68°C).

Table E.4 (ref. 45) *Distribution of chlorinated biphenyls and their degradation products in the model ecosystem*

	Chlorinated biphenyl equivalents (ppm)				
	H_2O	*Oedogonium* (alga)	*Physa* (snail)	*Culex* (mosquito)	*Gambusia* (fish)
I. 2,5,2'-trichloro-biphenyl total ^{14}C	0.03845	23.2155	31.2015	2.7030	3.2055
Unknown I (R_f 0.66[a])	0.00015	15.9575	18.9720	1.1995	0.2085
trichlorobiphenyl (R_f 0.56)	0.00020	1.4630	1.1590	0.1630	1.2800
Unknown II (R_f 0.23)	0.00005	0.0520	0.6480	+	0.1595
Unknown III (R_f 0.10)	–	–	0.9735	–	–
Unknown IV (R_f 0.06)	0.00055	–	0.3460	–	–
Unknown V (R_f 0.04)	0.00040	–	0.2205	–	–
Unknown VI (R_f 0.03)	0.00040	0.0685	0.4410	–	–
Polar (R_f 0.0)	0.02265	0.5185	3.9315	0.4795	0.9985
Unextractable	0.01405	5.1560	4.3100	0.8610	0.5590
II. 2,5,2',5'-tetrachloro biphenyl total ^{14}C	0.02065	23.6845	53.7465	14.5335	15.5685
tetrachlorobiphenyl (R_f 0.48[a])	0.00120	21.5975	47.3275	12.6745	14.2360
Unknown I (R_f 0.23)	0.00005	0.3220	0.7560	0.1070	0.0890
Unknown II (R_f 0.04)	0.00155	0.1030	0.4360	–	–
Polar (R_f 0.0)	0.01225	0.3275	3.9850	0.9670	0.8545
Unextractable	0.00560	1.3345	1.2420	0.7850	0.3900
III. 2,5,2',4',5'-penta-chlorobiphenyl total ^{14}C	0.04340	62.4660	633.0165	181.4565	127.3945
pentachloro-biphenyl (R_f 0.55[a])	0.00985	53.8440	587.3545	170.8480	119.7060
Unknown I (R_f 0.46)	–	0.6850	8.6210	2.4070	2.5380
Unknown II (R_f 0.39)	0.00020	0.5080	2.2490	1.3195	0.5810
Unknown III (R_f 0.21)	0.00015	0.1425	1.9365	1.0520	0.3285
Unknown IV (R_f 0.04)	0.00030	–	0.5000	–	–
Unknown V (R_f 0.02)	0.00385	0.2570	7.4965	–	0.7450
Polar (R_f 0.0)	0.02055	1.6265	16.5550	2.6745	2.3610
Unextractable	0.00850	5.4330	8.3040	3.1555	1.4350

[a]TLC with hexane (Skellysolve B, bp 60-68°C).

Table E.5
(ref. 45)

Ecological magnification (E.M.) and Biodegradability index (B.I.) of PCB's and DDE compared with water solubility and partition coefficient

Chemical	H_2O solubility (ppb)	Partition coefficient	Ecological magnification (E.M.)				Biodegradability index (B.I.)			
			Algo	Snail	Mosquito	Fish	Algo	Snail	Mosquito	Fish
tri-Cl-PCB	16	7,803	7,315	5,795	815	6,400	0.30	0.17	0.35	0.60
tetra-Cl-PCB	16	8,126	17,997	39,439	10,562	11,863	0.015	0.082	0.076	0.060
penta-Cl-PCB	19	16,037	5,464	59,629	17,345	12,152	0.029	0.027	0.0134	0.019
DDE	1.3	18,893	11,251	36,342	59,390	12,037	0.069	0.049	0.033	0.050

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Sanborn et al. (307) obtained similar results with the green sunfish (Lepomis cyanellus) which was able to metabolize about 80% of the tri-CB, but almost none of the tetra- or penta-CB.

By contrast, Hutzinger et al. (308) were unable to detect any metabolites formed by brook trout dosed with representative mono-, di-, tetra- and penta-CBs. There was little, if any, metabolism of a penta-CB by the dogfish (309).

E.4. Metabolism by Birds

Hutzinger et al. (308) reported that pigeons were able to metabolize mono-, di- and tetra-CBs, but not a hexa-CB. The metabolites identified were mono-hydroxy -CBs; unlike rats, pigeons did not produce detectable quantities of di-hydroxy-CBs. De Freitas and Norstrom (27) reported a relation between ease of "metabolism" in pigeons and chlorine substitution patterns, but their measurements were based on differential retention and did not in fact distinguish between metabolism and excretion.

McKinney et al. (29, 310) have recently studied the metabolism of five symmetrical isomers of hexa-CB in chickens, with some novel and important results. In addition to the hydroxylated metabolites identified by others as the major metabolites in birds and mammals, they found evidence of isomerization (rearrangement of chlorine substitutions), dechlorination, and formation of CDFs. The metabolites of 2,4,5,2',4',5'-hexaCB included a penta-CB, a mono-hydroxy derivative, together with the 3,6-quinol and 3,6-quinone derivatives of the

hexa-CB. The metabolites of 2,4,6,2',4',6'-hexaCB included a rearrangement product (2,4,6,2',3',4'-hexaCB), a penta-CB, a hexa-CDF and a penta-CDF. The other three hexaCBs studied (2,3,6,2',3',6'-; 2,3,4,2',3',4'-; and 3,4,5,3',4',5'-) showed little or no metabolism except in the first case, in which a mono-hydroxy derivative was formed. The formation of the two CDFs by metabolism of chlorobiphenyls appears to be well established by gas chromatography--mass spectrometric techniques (310).

The hexa-CDF was the most abundant chlorinated compound present in the faces of the chicks, indicating that it was formed in significant quantities. The authors (210) suggest that it is formed via ortho-hydroxylation in association with chlorine migration, and hence suggest that it may have a 1,2,3,7,8,9 substitution pattern. This newly-discovered phenomenon is of potentially great toxicological significance.

E.5. Metabolism in Mammals

Metabolism of chlorobiphenyls has been extensively studied in mammals. A concise summary of recent work on metabolism in the rat has been published by Goto et al. (311: Figure E.9); as shown in this section, metabolism in the other mammalian species studied to date appears to be generally similar.

The liver appears to be the main site of metabolism of PCBs. The hypothesis that PCBs are metabolically degraded by the liver is supported by studies showing that the biotransformation of PCBs is

markedly retarded when livers are damaged by administration of carbon tetrachloride. Grant et al. (182) found that residues of Aroclor 1254 in rat tissues were higher in rats simultaneously treated with carbon tetrachloride than in rats dosed only with Aroclor 1254. The residue pattern in fat of animals treated with carbon tetrachloride was similar to that of standard Aroclor 1254, indicating little metabolism.

Hydroxylation appears to be the main metabolic route for chlorobiphenyls (4, 28, 308-314). Generally, dechlorination does not seem to take place. Hutzinger et al. (4) did find traces of substances in rat faces whose mass spectra indicated loss of a chlorine atom. The spectra indicated a trichlorinated metabolite of 3,4,3',4'-tetraCB and a heptachlorinated metabolite of 2,3,4,5,2',3',4',5'-octaCB. Hutzinger et al. have also identified a dechlorinated metabolite of 2,4,5,2',4',5'-hexaCB in rabbits (315).

Hutzinger et al.(4) studied metabolic behavior of 11 chlorobiphenyls in rats after intraperitoneal injection. Extracts of urine and feces were analyzed by thin layer chromatography and mass spectrometry. The identities of many metabolites were confirmed by NMR spectroscopy and synthesis. The results of this study are summarized in Table E.6. They did not find metabolites for hexa and octachlorobiphenyls. Decachlorobiphenyl was not soluble enough in oil for injection into rats.

Work has been done on metabolism of individual chlorobiphenyls.

POLAR METABOLITES OF CHLOROBIPHENYLS FOUND IN DIFFERENT ANIMAL SPECIES

Table E.6 (from ref. 4)

Chlorobiphenyl Administered			Animal Species Used	Metabolite Found		
				Monohydroxy* Compound	Dihydroxy* Compound	
No.	Name	Formula		10^6 - m/e Chlorobiphenylol+	10^6 - m/e Chlorobiphenylol+2H	Other Derivative
I	4-Chlorobiphenyl		rat	+	+	-
			pigeon	+	-	-
			trout	-	-	-
			rabbit	+	-	-
II	4,4'-Dichlorobiphenyl		rat	+	+	-
			pigeon	+	-	-
			trout	-	-	-
III	2,2'-Dichlorobiphenyl		rat	+	+	-
IV	3,4'-Dichlorobiphenyl		rat	+	-	-
V	2,2',4,4'-Tetrachlorobiphenyl		rat	+	-	-
VI	2,2',3,3'-Tetrachlorobiphenyl		rat	+	-	-
			pigeon	+	-	-
			trout	-	-	-
			rabbit	+	-	Dihydrodihydroxytetrachlorobiphenyl
VII	3,3',4,4'-Tetrachlorobiphenyl		rat	-	-	-
VIII	2,2',4,4',5,5'-Hexachlorobiphenyl		rat	-	-	-
			pigeon	-	-	-
			trout	-	-	-
			rabbit	+	-	Hydroxypentachlorobiphenyl hydroxypentahexachlorobiphenyl
IX	2,2',4,4',5,5'-Hexachlorobiphenyl		rat	-	-	-
X	2,2',3,3',5,5'-Hexachlorobiphenyl		rat	-	-	-
XI	2,2',3,3',4,4',5,5'-Octachlorobiphenyl		rat	-	-	-

a Including conjugates.
** Detected by deuteriation procedure.

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Monochlorobiphenyls. Hutzinger et al. (308) found that 4-chlorobiphenyl in rat urine metabolized to mono- and dihydroxychlorobiphenyl. Very little starting material could be recovered from the feces. No starting material could be recovered from the urine. These observations indicate extensive metabolic degradation.

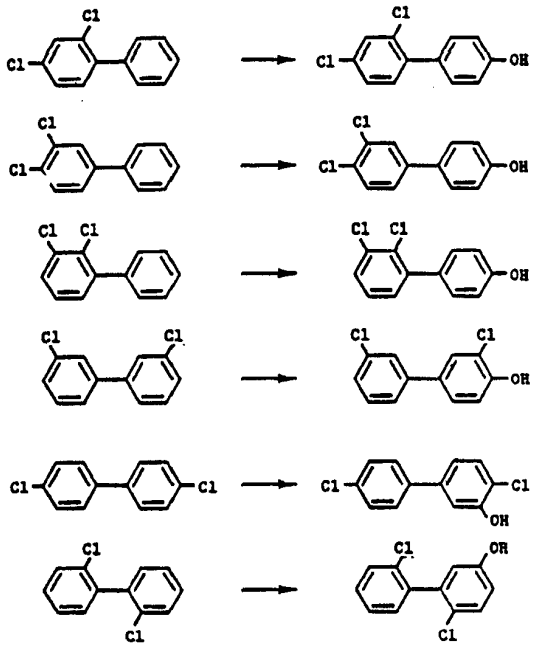
Safe et al. (316) in studies of male Wistar rats, employed NMR spectroscopy to establish the hydroxylation of 4-chlorobiphenyl at the 4' position.

Di- and Trichlorobiphenyls. Grab et al. (317) found that mono and dihydroxylated derivatives were the major urinary metabolites of 2,4'-dichlorobiphenyl and 2,2',5-trichlorobiphenyl in the rhesus monkey. They found similar metabolites of these two compounds to be produced by rat liver microsomes (318).

Hutzinger et al. (308) reported that large quantities of unchanged 4,4'-dichlorobiphenyl could be extracted from rat feces, but only its monohydroxy metabolites could be found in rat urine.

Hutzinger et al. (4) present a number of monohydroxy derivatives observed as metabolic products from specific CBs in rats (Figure E.9). They also observed dihydroxy metabolites of 2,2'- and 4,4'-diCBs.

Safe et al. (316) used NMR spectroscopy to elucidate the structure of the major hydroxylated metabolite of 4,4'-dichlorophenyl fed to rats. They found that hydroxylation occurred at the 3 position to produce 4,4'-dichloro-3-hydroxybiphenyl. A similar result was obtained in goats and cows (324).



Monohydroxy-derivatives observed as metabolic products from dichlorobiphenyls in rats.

Figure E.8 (from ref. 4)

Tetrachlorobiphenyls. Yoshimura and Yamamoto (313) studied the metabolic fate of 3,4,3',4'-tetrachlorobiphenyl in rats. They found 64% of the dose excreted unchanged in the feces. They felt that most of this unchanged material had not been absorbed from the gastrointestinal tract. At least three metabolites were also excreted in the feces. The major metabolite, a mono-hydroxy derivative, excreted during 14 days accounted for only about 3.3% of the dose administered.

The metabolic fate of 2,4,3',4'-tetraCB was also investigated. At least four metabolites of phenolic nature were excreted exclusively into the feces along with a large amount of 2,4,3',4'-tetraCB. The major metabolite was identified as 5-hydroxy-2,4,3',4'-tetraCB (312).

Yoshimura et al. (314) found that the metabolic rate for 2,4,3',4'-tetraCB seemed different from that of 3,4,3',4'-tetraCB despite the fact that all tetraCB metabolites they found in rat feces were monohydroxylated derivatives.

Gardner et al. (319) used infrared and mass spectra to identify the urinary metabolites of 2,5,2',5'-tetraCB in rabbits. Three hydroxylated metabolites were excreted. They were:

3-hydroxy-2,5,2',5'-tetraCB
4-hydroxy-2,5,2',5'-tetraCB
trans-3,4-dihydro-3,4-dihydroxy-2,5,2',5'-tetraCB

The first two compounds are phenols, but the third is a dihydrodiol with the vicinal hydroxy groups in the trans conformation.

Van Miller et al. (320) studied the metabolic fate of the same tetra-CB in rats and found that it was rapidly metabolized to one or more hydroxylated metabolites and excreted. The principal metabolite identified was 3-hydroxy-2,5,2',5'-tetraCB. Little of the material was stored in the fat.

Hsu et al. (321) found distinct differences in the metabolism of the same tetra-CB in infant rhesus monkeys. The 3-hydroxy derivative was not found: one of two metabolites was unidentified; the other was identified as the trans-3,4-dihydroxy derivative. Hsu et al. suggest that the mechanisms of metabolism in rats and monkeys must differ (see below).

Pentachlorobiphenyls. Berlin et al. (322) treated mice with 2,4,5,2',5'-pentaCB and found that 80% was excreted within 19 days, mostly in the form of a mono-hydroxy metabolite.

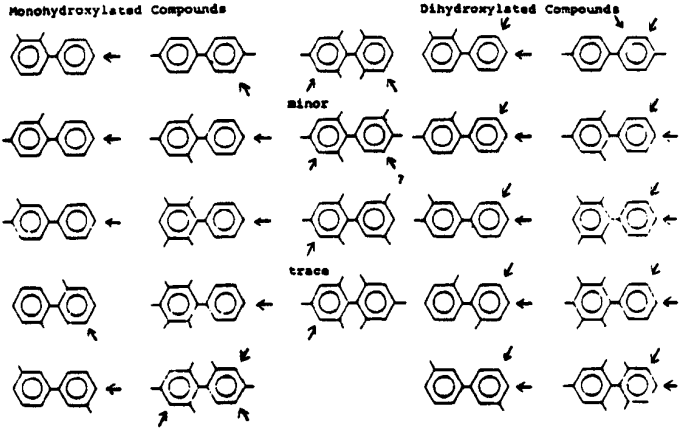
Hexachlorobiphenyls. Metabolic dechlorination of 2,4,5,2',4',5'-hexachlorobiphenyl was found by Hutzinger et al. (315). Rabbits were fed a diet containing 1% 2,4,5,2',5',5'-hexachlorobiphenyl for seven days. The total concentration of chlorobiphenyl administered was 1 g per kg. Urine, collected during the entire feeding period, was collected, extracted and analysed by a high resolution mass spectrometric method involving photoplate detection. Two major metabolites and a minor metabolite were detected. The first major metabolite was formed from the hexachlorobiphenyl by hydroxylation and the second by hydroxylation with concomitant dechlorination. Formation of the minor metabolites also involved dechlorination.

Jensen and Sundström (323) also found a mono-hydroxy derivative of this hexa-CB, probably the 3-hydroxy derivative, in the faces of rats. This is an important finding because 2,4,5,2',5',5'-hexaCB has no adjacent unsubstituted carbon atoms, so that its hydroxylation must take place directly (323).

Summary of metabolism in the rat. Goto et al. (311) have summarized their own work and that of others on the metabolism of chlorobiphenyls (as shown in Figure E.9). These results show that hydroxylation usually takes place at the 3- and 4- positions, rarely at the 2-position. With a few exceptions, hydroxylation usually occurs at one of two adjacent unsubstituted positions.

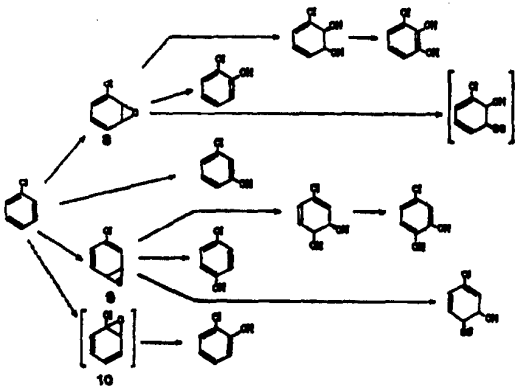
E.6. The Mechanisms of Metabolism

Gardner et al. (319) gave evidence that the principal mechanism of metabolism of chlorobiphenyls was via the formation of an arene oxide (epoxide) intermediate. This has been confirmed for 4-chlorobiphenyl by Safe et al. (325). They used material labelled with deuterium at the 4'-position to show that metabolism in rabbits proceeded via the 3',4'-epoxide, followed by rearrangement to form the 4'-hydroxy- (or to a smaller extent the 3'-hydroxy) derivative. The general importance of arene oxides as intermediates in the metabolism of aromatic compounds has been reviewed by Jerina and Daly (326). One of their examples, the metabolism of chlorobenzene, is shown in Figure E.10. Arene oxides appear to be formed directly by microsomal enzymatic oxidation and then to be rearranged (the "NIH" shift") to



Hydroxylations of polychlorinated biphenyls in the rat
(Main positions are indicated by arrows)

Figure E.9 (from ref. 311)



Mammalian metabolism of chlorobenzene (*50*) to phenols, dihydrodiols, catechols, and glutathione conjugates. Oxides are proposed as intermediates in the formation of 2- and 4-chlorophenol, while the formation of 3-chlorophenol is proposed to occur directly. The structure of the glutathione conjugate derived from 8 is a tentative formulation. Evidence for formation of the oxide 10 has not been obtained. On isomerization, it would be expected to yield 2-chlorophenol.

Figure E.10 (from ref. 326)

form phenols, or to be hydrated to form dihydrodiols (326). In the case of chlorobiphenyls, the data in Figure E.9 suggest that arene oxides tend to form preferentially in the 3,4-position where possible.

The formation of arene oxides requires the presence of adjacent unsubstituted carbon atoms and is therefore more favored for the less chlorinated mixtures. Isomers without adjacent unsubstituted positions can be hydroxylated directly (323), although more slowly. Safe et al. (325) showed that the formation of the dihydroxy derivative proceeded by hydroxylation of the monohydroxy derivative, rather than directly from the arene oxide. Other pathways of metabolism for hexachlorobiphenyls without adjacent unsubstituted positions are shown by the work of McKinney et al. in chickens (29, 310).

E.7. Formation of CDFs by Metabolism

In addition to the demonstration by McKinney et al. (29, 310) that penta- and hexa-CDFs can be produced by metabolism in chickens, Curley et al. (327) gave evidence that tetra-CDFs may be formed by metabolism in rats. They isolated a substance from the urine of rats exposed to Aroclor 1254 in the diet with a molecular weight of 304 and an isotopic cluster of 4 chlorine atoms. This matches tetra-CDF but was not positively identified as such. They found evidence of small quantities of the same material in the Aroclor mixture fed to the rats, but the material was much more prominent in the urine of the rats (327).

E.8. Toxicity of Metabolites

Metabolism of PCBs has often been regarded as detoxification, and indeed ultimately is so when the metabolites are conjugated and excreted. However, some of the primary metabolites may be more toxicologically significant than the parent materials. The CDFs are potentially much more toxic than the parent chlorobiphenyls, even if their production is only a minor metabolic pathway (2). Yamamoto and Yoshimura (312) reported that the acute toxicity of the primary metabolite of 2,4,3',4'-tetraCB is 5 times higher than that of the parent compound.

The formation of arene oxides as intermediates in metabolism is of even greater significance. Arene oxides are chemically active compounds which "react

> readily with a variety of nucleophiles, including such cellular macromolecules as DNA, RNA, and protein. Thus, arene oxides have become prime candidates for the 'bioactivated intermediates' responsible for the binding of aromatic compounds to biopolymers within the cell. Since the toxic, carcinogenic, and mutagenic effects of aromatic compounds often correlate with the extent of this binding, arene oxides are now strongly implicated as causative agents in producing these effects." (326).

Thus, for example, the K-region epoxides of polycyclic aromatic hydrocarbons are believed to be the active primary mutagenic and carcinogenic agents (279, 326). The same is likely to be true for PCBs (325, 319, 310, 29, 328). Indeed, Allen and Norback have shown that PCBs are strongly associated with protein and nucleic acid fractions of cells, and that 20% of the metabolites of a tetra-CB are bound to microsomal protein and RNA in a non-extractable form (69). In the words of Matthews and Anderson (328):

"Thus, we are given a dilemma. Those PCBs which can be metabolized and excreted may be metabolized via a carcinogenic intermediate and those PCBs which are not readily metabolized have an extremely long biological half-life. Several researchers have provided evidence that the PCBs may be carcinogenic (ref. 14-16). On the other hand, Vos et al. (ref. 17) have shown the very slowly metabolized hexachlorobiphenyl used in this study to be nonegenic, to cause liver damage, and to induce hepatic porphyria. It is not yet known if it is the parent PCBs or their metabolites which account for the primate reproductive failures described by Allen (ref. 18). The only way that we are going to establish which of the PCBs or their metabolites are carcinogenic or are going to cause any of the other toxicological problems and at what levels of exposure the problems are likely to arise is through systematic pharmacokinetic studies which will allow us to extrapolate the results of chronic low dose environmental exposures from laboratory animals to man. Until such data is available it is my opinion that every effort should be made to avoid environmental contamination by any type of PCBs" (328).

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16. ABSTRACT

This document summarizes the physical/chemical properties, toxicological information and environmental fate and effects of PCBs, with emphasis on its aquatic behavior. Form these data criteria are developed for the protection of aquatic life.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Criteria Toxicity Aquatic animals Aquatic biology Human ecology Safety factor	Toxic Pollutant Effleunt Standards Federal Water Pollution Control Act	

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Table 2.4-10
Comparison of Concentrations of Aroclor 1254 Found in Species Collected in Escambia and East Bays

Species	Escambia Bay (ppm)	East Bay (ppm)
Spartina	N.D.	N.D.
Zostera	N.D.	N.D.
Olive Nerite	0.49	N.D.
Mangia	N.D.	N.D.
Penaeid Shrimp	0.98	Trace
Blue Crabs	6.90	0.46
Bay Anchovy	3.00	0.68
Catfish	3.80	0.58
Tidewater Silversides	10.00	0.95
Silver Perch	4.50	0.48
Sand Seatrout	1.50	----
Spotted Seatrout	----	0.12
Spot	1.80	Trace
Atlantic Croaker	1.60	Trace
Hogchoker	1.30	N.D.
Atlantic Cutlassfish	2.90	0.72

SOURCE: Nimmo, D.R., D.J. Hansen, J.A. Couch, N.R. Cooley, P.R. Parrish and J.I. Lowe, Toxicity of Aroclor 1254 and its Physiological Activity in Several Estuarine Organisms, (unpublished).

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2.5 Data from Localized Monitoring Efforts Industrial Plants, Products, Sewage Treatment Facilities and Landfills

2.5.1 Industrial Plants

.5.1.1 Monsanto Co., Sauget, Illinois

The only PCB production facility in the United States is the Monsanto plant at Sauget, Illinois which has produced PCB mixtures ranging from 20 to 68 percent chlorine. Polychlorinated terphenyls have been produced at this facility, but production was suspended in 1971.

Soil contamination studies[7] were initiated in February 1976. The sampling locations around the Monsanto facility are identified in Figure 2.5-1. The levels of Aroclor 1242, Aroclor 1260 and decachlorobiphenyl found at these sampling sites are listed in Table 2.5-1. The distribution of all PCBs analyzed appears to be higher near the plant site and generally decreasing with distance from the site. There is some evidence that higher concentrations are present in the soils located to the southeast which corresponds with the predominant wind direction in this area.

A typical chromatogram of a soil sample obtained near the plant is shown in Figure 2.5-2 along with the reference chromatograms of Aroclor 1242 and 1260 run under the same instrument conditions. Using these reference spectra, this sample contains 11 ppm Aroclor 1242, 9.3 ppm Aroclor 1260 and 1.0 ppm decachlorobiphenyl.

All PCB measurements were made using a Varian Model 2760 electron capture gas chromatograph with a 1.8m glass column operated at 200°C. The column had a 3mm Id and was packed with 1.5/1.95% OV-17/WF-1 on chrom W-HP, 80/100 mesh support. The flow rate was 68 ml/min with an inlet pressure of N_2 at 38psig.

Table 2.5-1
PCB Levels in Soils, ppm, Monsanto

Sample Station	Aroclor 1260	Aroclor 1242	Decachlorobiphenyl	Total PCB
1	0.05	<0.01	0.097	0.147
2	0.12	<0.01	0.61	0.73
3	1.4	<0.01	0.90	2.3
4	0.31	0.68	0.38	1.37
5	0.20	<0.01	0.27	0.47
6	1.3	0.82	1.6	3.72
7	2.9	3.0	1.3	7.2
8	9.6	6.1	2.4	18.1
9	0.65	<0.01	0.12	7.7
10	0.28	0.45	0.081	0.811
11	0.10	<0.01	0.049	0.145
12	0.26	<0.01	0.081	0.341
13	9.6	10.0	1.1	20.7
14	0.03	<0.01	0.40	0.43
15	0.39	0.46	0.12	0.97

SOURCE: Unpublished Report, Contract 68-01-2978, USEPA, Office of Toxic Substances; July 1975

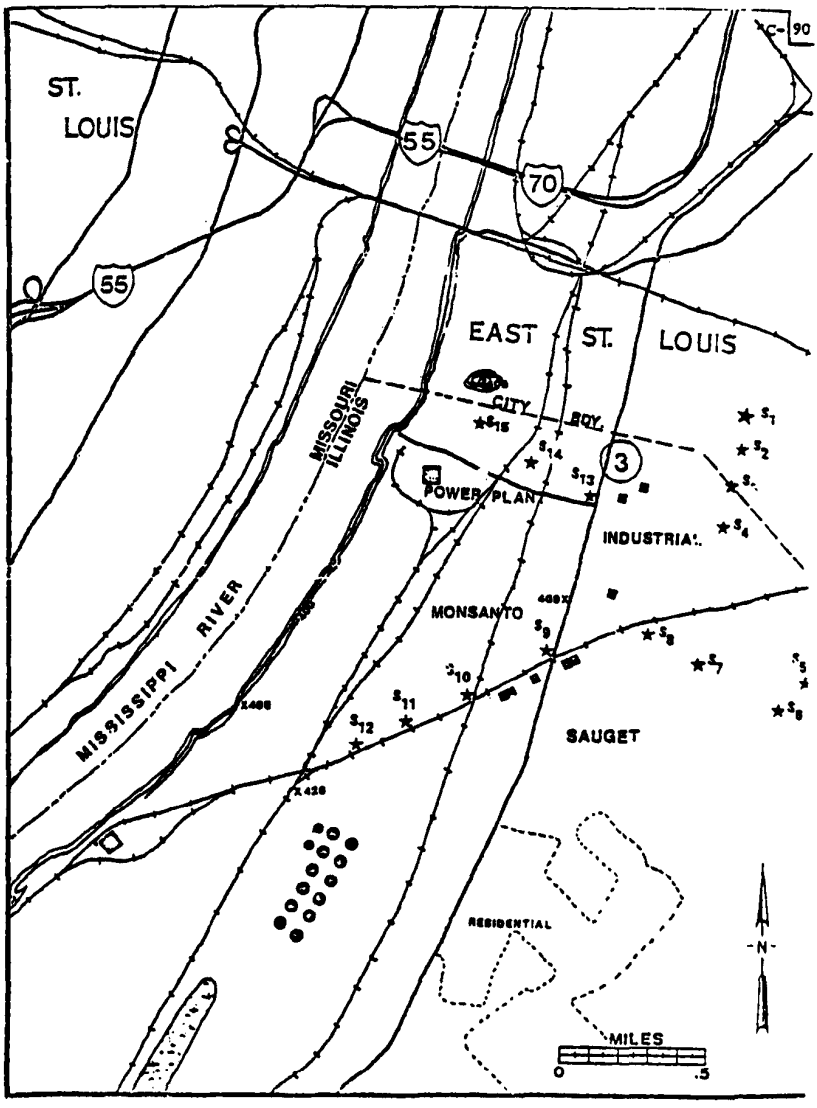

FIGURE 2.5-1 SAMPLING LOCATIONS, MONSANTO

MONS 063495

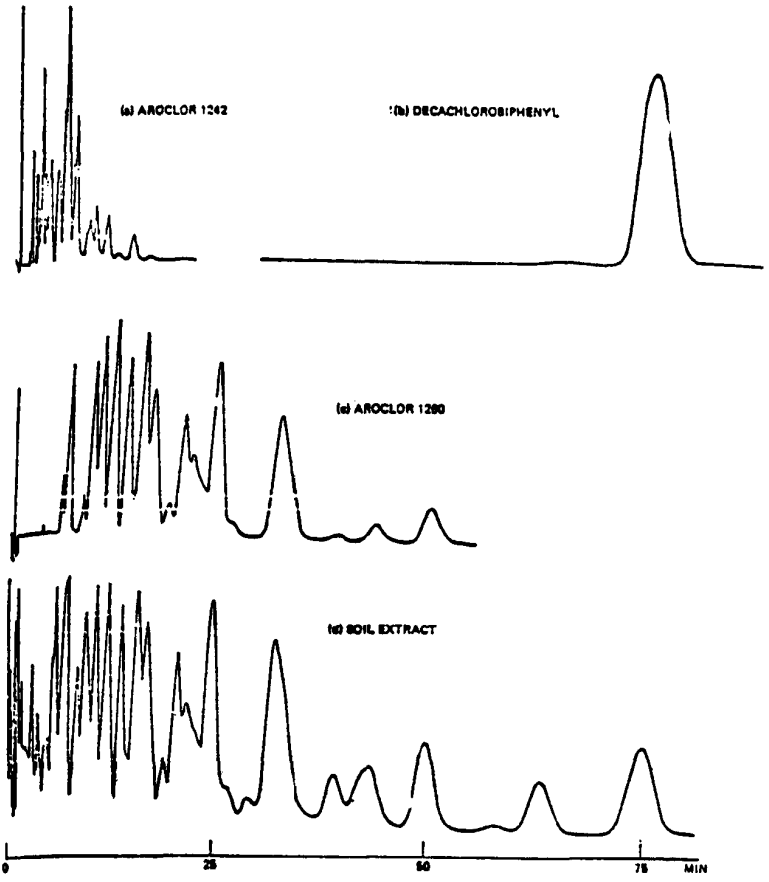

FIGURE 2.5-2 TYPICAL CHROMATOGRAMS OF STANDARD AROCLORS, DECACHLOROBIPHENYL AND A SOIL SAMPLE TAKEN IN THE VICINITY OF MONSANTO CO., SAUGET, ILLINOIS

2.5.1.2 Yates Manufacturing Co., Chicago, Illinois

The Yates Manufacturing Co. is an investment casting wax manufacturer. Prior to 1972, Aroclors 5460, 6090 and 5442, mixtures that contain both PCBs and polychlorinated terphenyls, were used. Subsequent to 1972, this company has been purchasing decachlorobiphenyl from foreign sources.

Soil contamination studies[7] were initiated in February 1976. The sampling locations around the Yates facility used to study soil contamination are identified in Figure 2.5-3. The concentration levels of Aroclor 1242, Aroclor 1260 and decachlorobiphenyl found at these sampling sites are listed in Table 2.5-2.

Table 2.5-2
PCB Levels in Soils, ppm, Yates Manufacturing

Sample Location	Aroclor 1260	Decachlorobiphenyl	Total PCB
1	0.22	<0.001	0.22
2	0.24	0.053	0.293
3	0.81	0.033	0.113
4	0.34	0.19	0.53
5	0.51	0.58	1.09
6	0.40	1.2	1.6
7	0.26	0.51	0.77
8	1.5	0.75	2.25
9	0.57	0.040	0.61
10	0.67	0.020	0.69
11	1.6	3.6	5.2
12	0.29	0.001	0.291
13	0.56	0.034	0.594
14	1.8	<0.001	1.8

SOURCE: Unpublished Report, Contract 68-01-2978, USEPA, Office of Toxic Substances; July 1975

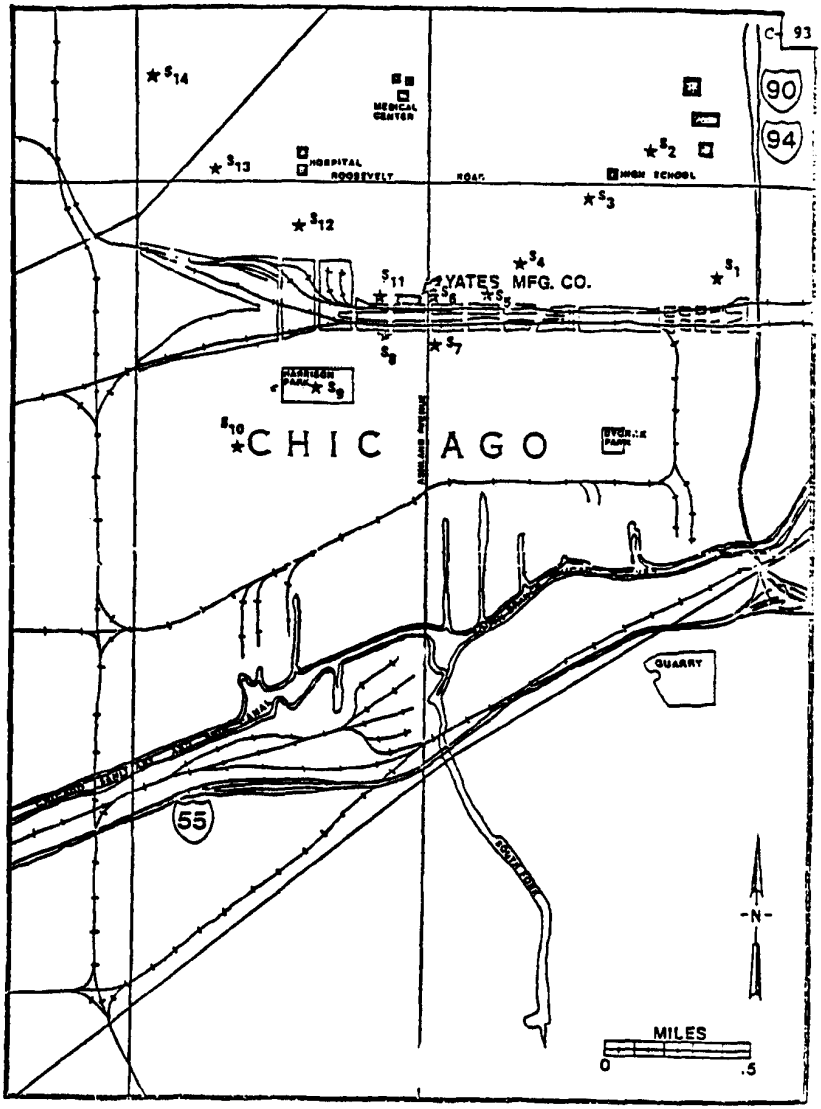

FIGURE 2.5.3 SAMPLING LOCATIONS, YATES MANUFACTURING

2.5.1.3 Valcast Corp., Troy, Michigan

Valcast Corp. is an investment casting facility located in a small industrial park in suburban Detroit, Michigan. PCBs are a constituent of the wax mold compound used to fashion intricate shapes which are to be cast.

Soil contamination studies[7] were initiated in February 1976. The sampling locations around the Valcast facility are identified in Figure 2.5-4. The concentration levels of Aroclor 1260 and 1242 found at these sampling sites are listed in Table 2.5-3.

Figure 2.5-5 is a typical chromatogram of a soil sample extract taken from this location. Aroclor 1260 was present in many of the soil samples taken from the Valcast area, but decachlorobiphenyl was absent. The one observed concentration of 18 ppm of Aroclor 1242 appears to be anomalously high, however, replicate analysis yielded values of 16 ppm and 19 ppm, respectively. Aroclor 1242 was not detected in any other soil samples from this area.

All PCB measurements were made using a Varian Model 2760 electron capture gas chromatograph with a 1.8m glass column operated at 200°C. The column had a 3mm Id and was packed with 1.5/1.95% OV-17/WF-1 on chrom W-HP, 80/100 mesh support. The flow rate was 68 ml/min with an inlet pressure of N_2 at 38psig.

A small drainage ditch that passes adjacent to the north boundary of the Valcast facility was sampled. This ditch serves to remove storm-water runoff in the vicinity and receives discharged cooling water from Valcast and other local small industries. Analysis of the Valcast cooling water at the point of discharge and of water in the drainage ditch failed to detect PCB levels greater than the detection limit of 0.1 ppb Two bottom sediment samples taken from this drainage ditch, however, had concentrations as follows:

Aroclor 1242	2.3 ppm	9.4 ppm
Aroclor 1260	6.7 ppm	8.9 ppm
Decachlorobiphenyl	0.09 ppm	0.11 ppm.

Table 2.5-3

PCB Levels in Soils, ppm, Valcast

Sample Station	Aroclor 1260	Aroclor 1242
1	< 0.01	
2	< 0.01	18.0
3	0.04	
4	0.06	
5	0.05	
6	0.04	
7	0.14	
8	0.07	
9	< 0.01	
10	0.12	
11	< 0.01	
12	0.12	
13	0.03	
14	< 0.01	
15	0.03	
16	0.02	

SOURCE: Unpublished Report, Contract 68-01-2978, USEPA, Office of Toxic Substances; July 1975

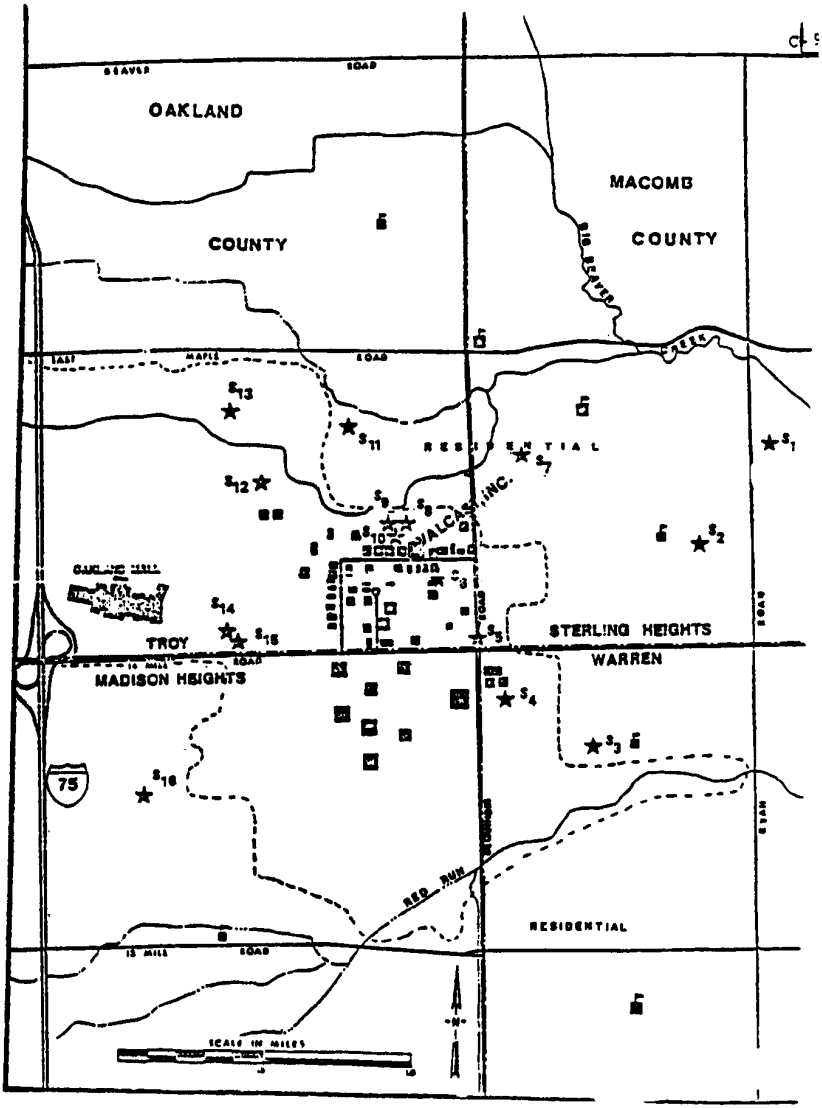

FIGURE 2.5-4 SAMPLING LOCATIONS, VALCAST

MONS 063501

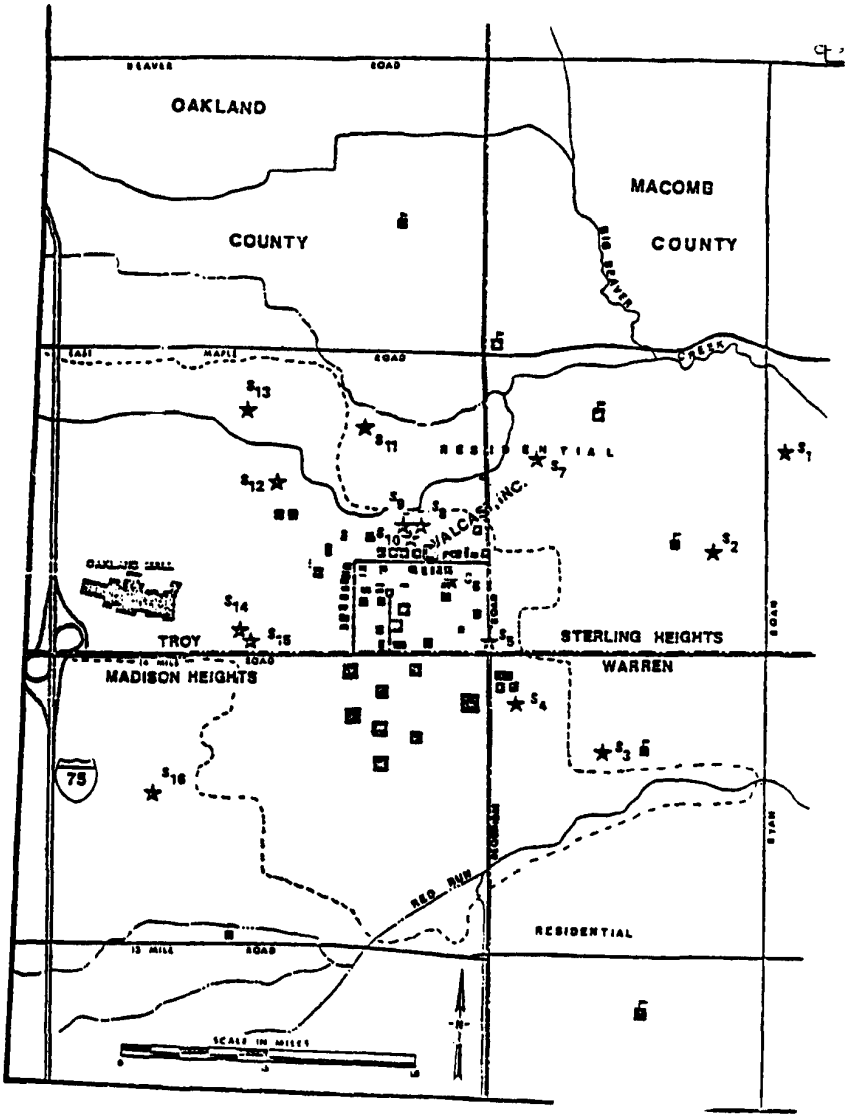

FIGURE 2.5-4 SAMPLING LOCATIONS, VALCAST

MONS 063502

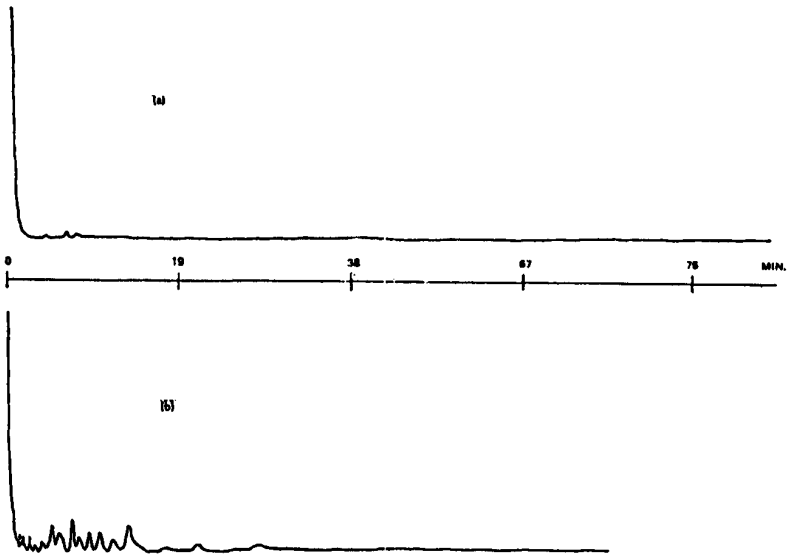

FIGURE 2.8-5 TYPICAL CHROMATOGRAMS OF SOIL SAMPLES TAKEN IN THE VICINITY OF VALCAST CORP., TROY, MICHIGAN – (a) 0.01 ppm, (b) 0.014 ppm ARUCLOR 1260

MONS 063503

2.5.1.4 Genaral Electric, Hudson Falls - Ft. Edward, N.Y.

The General Electric Plants at Hudson Falls and Ft. Edward use large quantities of PCB's for filling capacitors and transformers. Both plants have chemical waste treatment facilities, but, significant PCB and oil/grease levels are discharged by the waste stream. The NPDES Permit Application lists:

Outfall Location	Daily Average Concentration ppm		Average Daily Loading (lbs.)		Maximum Daily Loading (lbs.)	
	Oil/Grease	PCB	Oil/Grease	PCB	Oil/Grease	PCB
Hudson Falls	13.7	.5	239.8	10.0	250.9	17.6
Hudson Falls	2.1	---	4.9	---	5.25	---
Ft. Edward	8.9	5.0	38.27	20.0	44.5	30.0

In the summer of 1974, EPA Region II initiated a sampling program in the general vicinity of the General Electric plants.[1] The five sampling stations established for this program are shown in Figure 2.5-6. Station 0 is upstream from the plants and serves as the control station; station 1 is located at the junction of the outfall stream from the Ft. Edward facility and the Hudson River, station 2 is located about 0.25 miles downstream from the outfall junction, station 3 is about 0.5 miles downstream from station 1, and station 4 is located about 0.75 miles downstream from station 1.

Samples collected included sediment, water, fish and snails; Arclors identified included 1016, 1254, 1248 and 1242. The levels found at each sampling location are listed in Table 2.5-4.

PCB's identified as Aroclor 1016 were conclusively identified in the water samples at detectable concentrations at all sampling locations except Station 0 (control) and station 4, the furthest downstream.

At all stations the sediments contained higher concentrations of Aroclor 1016 than the water column resulting from adsorption of PCB's on suspended or already settled materials.

The biological samples collected at station 0 contained PCB's characteristic of Aroclor 1254 and Aroclor 1248. Distinctly different from the samples at station 0 were the samples collected in the vicinity of or below the General Electric discharge. Fish data from these areas suggests that Aroclor 1242, Aroclor 1016, or a mixture of these two formulations are present in the Hudson. There are no distinguishing features which can reliably determine whether the mixture is Aroclor 1016 or 1242 at this time.

Table 2.5-4
Environmental PCB Levels from the General Electric,
Hudson Falls - Ft. Edward N.Y. Area

Media	Water (ppb)	Sediment (ppm)	Fish (ppm)		
Aroclor	1016	1016	1254	1248	1242
Station					
0	<1.0	6.9	4.0	13.0	
1	2800	6700			45[a]
2	2.2	540			
3	3.0	2980			350[b]
4	<1.0	6.6			78[c] 27[a]

[a]Snails (composite)

[b]Rock Bass

[c]Shiner Minnows

SOURCE: Nadeau R.J., and R.P. Davis, Investigation of Polychlorinated Biphenyls in The Hudson River, USEPA Region II.

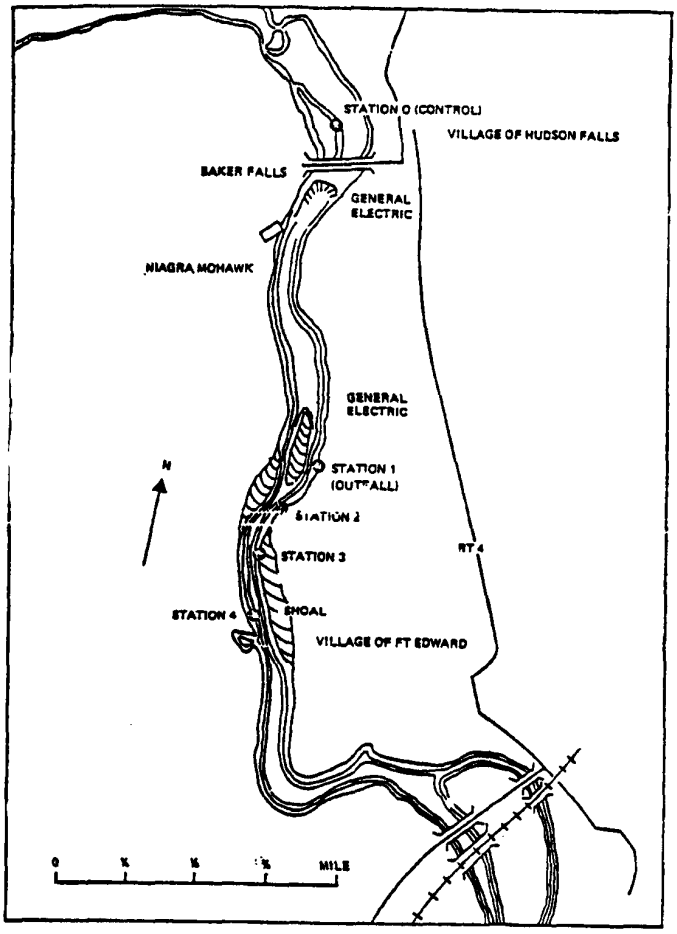

FIGURE 2.5-6 MAP OF HUDSON FALLS-FT. EDWARD, NEW YORK. NOTE LOCATION OF SAMPLING STATIONS RELATIVE TO GENERAL ELECTRIC FACILITIES.

2.5.2 Sewage Treatment Facilities

2.5.2.1 Illinois

An early study[9] of discharges into Lake Michigan was conducted by the state of Illinois in 1970-1972 from locations as shown in Figure 2.5-7. Only two counties in Illinois border on the lake and all sewage treatment plant effluents and tributary stream sources enter the lake from Lake County. In most cases, sewage treatment plant effluents had higher concentrations of PCB's than the tributary streams.

PCB's were not measured in 1970; however, in 1971, 37 sediment samples were collected from tributary streams and ravines in Lake County and at stations offshore from Lake and Cook counties and analyzed for PCB's. The lake samples were collected 40 to 80 yards offshore from seven North Shore Sanitary District sewage treatment plants and at stations approximately one to three miles offshore. Aroclor 1242 in tributary sediments ranged up to 553 ppb in an unnamed channel in Waukegan and Aroclor 1254 ranged from 1.54 to 232.00 ppb in Pettibone Creek in North Chicago. Samples from open water sediments had Aroclor 1242 concentrations ranging up to 106.07 ppb. The highest level was found 40-80 yards offshore from the North Shore Sanitary District sewage treatment plant at North Chicago. Aroclor 1254 concentrations ranged from 2.48 to 46.92 ppb. Selected values are given in Table 2.5-5.

Water samples were collected in both 1971 and 1972 at the tributary streams and sewage treatment plants. Aroclor 1242 and 1254 ranged from 14.00-1810.0 ppt and 192.0-388.0 ppt in 1971 respectively and up to 653.0 ppt for Aroclor 1242 and 61.0 to 841.0 ppt for Aroclor 1254 in 1972 in tributary streams. However, in samples from sewage treatment plants Aroclor 1242 concentrations ranged from 268.0-4020.0 ppt in 1971 and up to 21.0 ppt in 1972. Aroclor 1254 concentrations ranged from 139.0-368.0 ppt in 1971 and from 97.0 to 178.0 ppt in 1972. The highest readings were from the North Shore Sanitary District at Waukegan in 1971. Table 2.5-6 presents the levels identified in water samples from Illinois Streams and Sewage Plants Tributary to Lake Michigan.

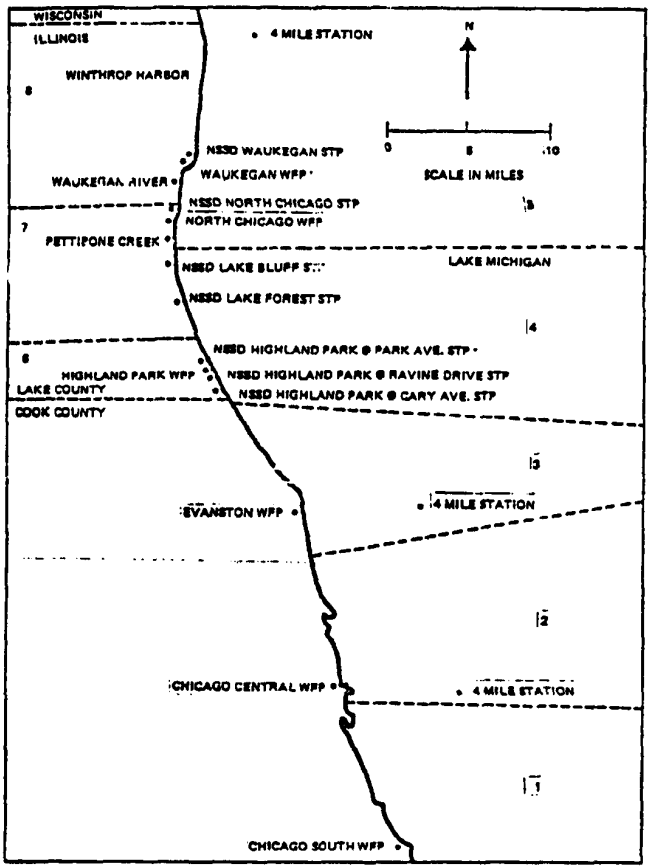

FIGURE 2.5-7 LOCATIONS OF WATER AND SEDIMENT SAMPLING STATIONS

Table 2.5-5
Polychlorinated Biphenyls in Sediments from Lake Michigan
and Tributary streams in Illinois 1971
(ppb) dry weight basis

Location		Aroclor 1242	Aroclor 1254
<1 to 3 miles offshores			
Cook County	1	N.D.-18.55	2.48-17.25
	2	17.23-83.35	9.38-46.92
	3	3.58-13.65	3.15-12.42
Lake County	4	7.43-19.25	5.26-17.45
	5	4.98-46.11	8.36-34.52
Highland Park	STP	11.11	12.42
Lake Forest	STP	10.51	7.02
Lake Bluff	STP	44.36	14.45
North Chicago	STP	106.07	26.54
Waukegan	STP	17.32	11.97
10-50 yards upstream from Lake			
Lake County	6	N.D.-4.32	1.54-17.90
	7	1.31-173.40	2.54-232.00
	8	1.77-553.00	2.56-131.00

SOURCE: USEPA, Pesticide Monitoring Programs: Lake Michigan and Tributaries in Illinois, EPA 600/3-74-002

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Table 2.5-6
Polychlorinated Biphenyls in Water Samples from
Illinois Streams and Sewage Plants Tributary to Lake Michigan
1971 - 1972, (ppt)

Location	Aroclor 1242	Aroclor 1254
1971		
Waukegan River	1810.0	388.0
Pettibone Creek	140.0-187.0	192.0-194.0
Waukegan STP	601.0-4020.0	139.0-568.0
North Chicago STP	268.0-1070.0	153.0-260.0
1972		
Waukegan River	57.0-120.0	61.0-136.0
Pettibone Creek	N.D.-653.0	107.0-841.0
Waukegan STP	N.D.-17.0	97.0-139.0
North Chicago STP	N.D.-21.0	100.0-178.0

SOURCE: USEPA, Pesticide Monitoring Programs: Lake Michigan and Tributaries in Illinois, EPA 600/3-74-002

2.5.2.2 Michigan

Samples collected in 1971 and 1972 from municipal waste water treatment plant effluents throughout Michigan[4] indicated these plants as a major source of PCB's with an average concentration of 2.55 ppb for 60 effluents sampled. Only seven of the effluents exceeded 1 ppb but one, the Bay City treatment plant on the Saginaw River, had an average effluent concentration of 120 ppb with a high of 340 ppb.

Samples collected from 58 waste water treatment plants in 1973 averaged 0.52 ppb. The Bay City PCB discharge had been greatly reduced as a result of control measures in industries served by the waste water treatment plant.

Sewage Sludge from 57 of the 58 plants tested in 1973 showed Bay City to have the highest concentration of PCB's in the sludge being removed by the treatment process averaging 352 ppm compared to a state wide average of 15.6 ppm.

The range of values for all waste water treatment plants sampled in 1971-1973 are given in Table 2.5-7.

Table 2.5-7
PCB Levels from Waste Water Treatment Plants, Michigan

City	Effluent (1971-72) (ppb) Aroclor 1254	Effluent (1973) (ppb) Aroclor 1242	Effluent (1973) (ppb) Aroclor 1254	Sludge (1973) (ppm) Aroclor 1242	Sludge (1973) (ppm) Aroclor 1254
Adrian	0.41-14.00				
Albion	0.44		0.34		1.5
Ann Arbor	<0.10-0.14		0.25		1.1
Battle Creek	0.16-0.92		<0.10		2.8
Bay City	5.70-340.00	3.20		352.0	
Benton Harbor St. Joseph	0.31-0.99		0.20		13.8
Brighton	0.38				<0.1
Charlotte	0.61		0.34		6.8
Constantine	0.85		0.46		2.1
Detroit	0.88-3.00	2.15	32.1		
Dexter	<0.10		0.23		3.2
E. Lansing	0.35-0.69		0.18		4.6
Escanaba	0.29				5.9
Esserville	0.21-0.28		0.10		3.9
Flint	<0.10-1.30		<0.10		6.3
Flushing	0.52				
Gladstone	0.19	0.44			4.1
Grand Haven	<0.50		<0.10		4.1
Grand Rapids	0.37-0.68	1.05			11.8
Holland	0.42-0.79	<0.10			0.8
Houghton-Hancock	<0.10	0.18			5.5
Iron Mountain-Kingsford	0.55-1.20	0.69			9.5
Ironwood	0.16		0.29		5.2
Jackson	<0.10	<0.10			3.0
Kalamazoo	0.19-1.30	1.12	23.3		
L'Anse	<0.10		0.31		4.4
Lansing	0.13-0.23	0.22			5.3
Manistique	<0.20	0.63			1.5
Marquette	0.35	0.29			2.8
Marshall	<0.10	0.15			3.9
Menominee	0.35	0.57			4.2
Midland	0.13-0.40	<0.10			2.9
Milford	<0.10		2.20		
Monroe	0.33-0.60	<0.10			3.3
Mt. Clemens	1.40-10.00		2.90	175.0	
Mt. Pleasant	<0.10	0.29			6.5
Muskegon	0.28	0.83			12.7
Muskegon Heights	0.37	0.18			11.0
Niles	0.68	0.48			7.8
Norway	0.40	<0.10			<0.1

Table 2.5-7 (cont.)

City	Effluent (1971-72) (ppb) Aroclor 1254	Effluent (1973) (ppb) Aroclor 1242	Effluent (1973) (ppb) Aroclor 1254	Sludge (1973) (ppm) Aroclor 1242	Sludge (1973) (ppm) Aroclor 1254
Ontonagon	<0.10				
Owosso	<0.10	0.12			2.0
Parchment	<0.10				
Pontiac (Auburn Rd)	<0.10-0.61		0.60		
Pontiac (E. Blvd)	0.15-1.30		0.54		
Portage	1.90				
Port Huron	0.28-0.52		1.80		
Saginaw	0.74-3.80		0.77		5.0
St. Ignace	<0.20	0.31			1.5
South Haven	<0.10				
Swartz Creek	<0.10				
Three Rivers	<0.30	<0.10			4.1
Trenton	0.14-1.10	<0.10			<0.1
Warren	0.10-0.16	<0.10			<0.1
Wayne County (Wyandotte)	0.17-0.64	0.40		2.01	
Wyoming	0.44-0.55	0.22			
Ypsilanti	0.21-0.22		0.31		2.0
Ypsilanti Twp #1	<0.10-0.12	<0.10			<0.1
Ypsilanti Twp #2	0.16-0.19	<0.10			<0.1
Cadillac		0.53			<0.1
Howell					15.0
Sault Ste. Marie		0.73			2.4
Traverse City		0.10			1.8

SOURCE: Monitoring for Polychlorinated Biphenyls in the Aquatic Environment, Michigan Water Resources Commission, May 1973

2.5.2.3 Wisconsin

Samples from sewage treatment facilities in Wisconsin were collected in March 1970. The PCB concentrations identified in the effluents ranged from 0.04 to 0.25 ppb and are presented in Table 2.5-8.[1]

In 1971 11 municipal sewage treatment plants in eleven southeastern Wisconsin cities were sampled as shown in Figure 2.5-8. Table 2.5-9 shows the results of this study and indicates that six of the eleven sewage plants had effluents ranging from 0.1 to 0.5 ppb of Aroclor 1254 while two sites were greater than 1.0 ppb for the same Aroclor. However Portage had 42 ppb of Aroclor 1248 in the effluent with 5.2 ppm in the digester sludge.

It is interesting to note that even though Port Washington is not as highly industrialized as Grafton the effluents from both cities contained approximately the same concentrations of PCB's ranging from 0.12 to 0.23 ppb.

Since the concentrations for Cedarburg were so high a special 24 hour study was conducted. The concentrations in raw sewage began to increase at the beginning of the working day from 0.54 ppb to a maximum of 3.1 ppb at 4:00 p.m. Table 2.5-10 presents the readings taken. The concentration in the final effluent appears to begin increasing from 0.33 ppb at midnight to a maximum of 0.77 ppb at 2 p.m. The concentration of PCB's in the effluent is approximately 30 percent of that in the influent. The concentration of PCB's in the sludges is approximately 1,000 times higher than in the fluid wastes. These data demonstrate that the time of sampling waste effluents is of importance in mass transport estimates.[1]

Table 2.5-8
Concentrations of PCBs in Outfalls into the
Milwaukee River on March 26, 1970

Location	Aroclor	PCB Concentration (ppb)
West Bend STP effluent	1254	0.25
Fredonia STP effluent	1254	0.12
Tributary at Fredonia	1260	0.04
Saukville STP effluent	1260	0.13
Chemical plant effluent, Saukville	1242	2.50
Grafton STP effluent	1254	0.04

SOURCE: Veith, G.D., and G.F. Lee, Water Research, 5, 1107-1115, (1971)

Table 2.5-9
PCB Concentrations in the Effluents from 11 Southeastern
Wisconsin Sewage Treatment Plants, 1971

City	PCB Concentrations (ppb)	Estimated Mass Transport (in lb/day)
Beaver Dam	0.05	0.0002
Port Washington	0.12-0.22	0.0027
Grafton	0.07-0.23	0.0008-0.0015
Cedarburg	0.28-1.1	0.0027-0.018
Racine	0.60-0.83	0.142
Burlington	0.08-0.14	0.0017
Lake Geneva	2.2-2.8	0.018
Walworth	0.17-0.34	
Beloit	3.89-11.75	0.0038-0.0052
Ft. Atkinson	1.24-2.48	
Portage	32-42	0.0015

SOURCE: Dube, D.J. Polychlorinated Biphenyls in Effluents from Sewage Treatment Plants in Southeastern Wisconsin

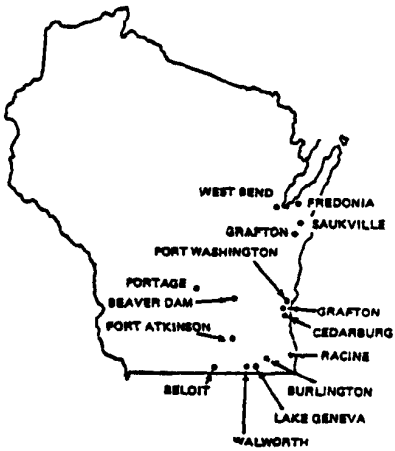

FIGURE 2.5-6 WISCONSIN CITIES WHERE MUNICIPAL SEWAGE TREATMENT PLANTS WERE SAMPLED IN 1971

Table 2.5-10
A 24-hour Survey of PCB's in the Cederburg Treatment Plant on April 15, 1971

Time	Flow (in gal/min)	PCB Concentrations (in ppb)[1] In the Influent	In the Effluent	In the Primary Trickling Filter
0:00	1480	0.30	0.33	----
1:00	1360	----	----	0.19
2:00	1300	0.20	0.10	----
4:00	1250	0.13	0.35	----
6:00	1200	----	0.16	----
8:00	2000	0.54	0.39	----
10:00	2100	0.40	0.50	----
12:00	1900	1.7	0.23	0.69
14:00	1900	1.5	0.77	----
16:00	1850	3.1	0.70	----
17:00	1900	----	----	0.25
20:00	1950	0.30	0.22	----
21:00	1800	----	----	0.14
22:00	1650	0.36	0.34	----

[1]The Chromatograms of the samples most closely resembled the chromatogram of Aroclor 1254.

SOURCE: Dube, D.J. Polychlorinated Biphenyls in Effluents from Sewage Treatment Plants in Southeastern Wisconsin

2.5.2.4 Ohio

Very little information is available from the Ohio River. The values reported are given in Table 2.5-11.

Table 2.5-11
PCB Levels from Sewage Treatment Plants, Ohio, 1971

Collection Site	Aroclor Detected	Concentration (ppb)
Dayton	1254	17
Hamilton	1248	10
Middleton	----	N.D.

SOURCE: Polychlorinated Biphenyls and the Environment, Interdepartmental Task Force on PCB's, May 1972

2.5.2.5 California

In 1970 over a billion gallons of waste water entered the sea each day from urban sewage systems in California. Since two kilograms of PCB output per day are equivalent to one ton per year, outfalls of 9 sewage treatment plants discharging waste to the sea in California were sampled in an effort to identify potential PCB sources. The samples were all taken from sewage outfalls at sites closest to the points of entry into the sea as shown in Figure 2.5-9. The highest output was reported in Los Angeles County which is one of the most industrialized areas in the state. Table 2.5-12 shows the PCB levels identified in the samples collected.[5]

The only other available data were collected in early 1975 on sludge as part of the California Compliance Monitoring Survey for Chlorinated Hydrocarbons.[6] The results obtained are listed in Table 2.5-13.

Only one plant was sampled in both studies, the Hyperion Plant in Los Angeles. The 1970 sludge samples had 78.5 and 98.1 ppb concentrations while the 1975 readings ranged from 1,000-1,400 ppb.

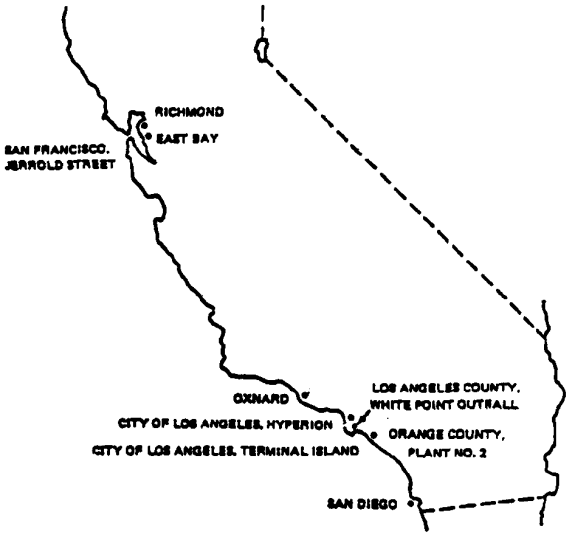

FIGURE 2.5-9 LOCATIONS OF TREATMENT PLANTS SAMPLED, CALIFORNIA

Table 2.5-13
PCB Levels in Sludge, California, 1975

Location	Description	Polychlorinated Biphenyls (ppm) Aroclor 1254	Aroclor 1260	Aroclor 1242
Orange County Sewage Treatment Plant	Fountain Valley Digester Sludge	——	——	38-40
Hyperion Sewage Treatment Plant	Los Angeles, Digester Sludge	1.0-1.4	——	——
Fort Ord Sewage Treatment Plant	Secondary Digester Sludge	0.2-0.4	——	——
Treasure Isle Sewage Treatment Plant	Sludge	0.4-0.6	——	——
Travis Air Force Base, Sewage Treatment Plant	Digester Sludge	0.6-0.8	——	——
Mather Air Force Base	Sacramento Primary Digester Sludge	0.4-0.6	——	——
McClellan Air Force Base	Secondary Sludge	——	4.5-5.5	——
Beale Air Force Base	Primary Digester	0.3-0.5		

SOURCE: Unpublished Data, USEPA National Field Investigation Center, August 1975

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2.6 Data from Localized Monitoring Efforts - Cities

2.6.1 Jacksonville, Florida

Samples of ambient air, water, soils and bottom sediments were collected in the vicinity of Jacksonville, Florida, on September 10, October 6-7 and November 20-21, 1975, for PCB analysis.[1]

Figure 1 illustrates the sampling locations in the Jacksonville area. The outlined area on Figure 2.6-1 circumscribes a municipal sewage sludge disposal site which has been used for the past several years by the City of Jacksonville.

AIR DATA

Air samples were collected at the fire station off Fort Caroline Road using a high-volume sampler equipped with polyurethane foam as the collection medium. Measured PCB concentrations are reported in Table 2.6-1 for the 24-hour period of November 20-21, 1975. The levels measured throughout this period ranged from 4 to 9 ng/m^3, with the lowest levels being observed during the early morning period. Chromatograms indicated the presence of the lower substituted PCB components of Aroclors 1221 and 1016. Quantitation of the PCB levels was performed after perchlorination to decachlorobiphenyl.

WATER, BOTTOM SEDIMENT AND SLUDGE DATA

The analytical data for water, bottom sediment and sludge are summarized in Table 2.6-2 for the sampling points shown on Figure 2.6-1. PCB content of bottom sediments range from 10 to 572 ppb, surface water concentrations ranged from the analytical detection limit of 20 ppt to 285 ppt, expressed as decachlorobiphenyl. The highest sediment concentration (572 ppb) was observed near the cooling water discharge of the Jacksonville Electric Authority Southside Generating Station. This may be related to use of PCB-containing materials at the generating station. Intermediate concentrations were observed in sections of the St. John's River which is surrounded by urban and industrial development. Drainage creeks from the municipal sewage sludge landfill area had bottom sediments of lower concentration. Inspection of gas chromatographic elution patterns indicates the presence of Aroclor 1260 in these sediment samples.

A concentration level of 720 ppb was measured for fresh sludge taken from the Monterey sewage treatment plant. Two-year-old sludge, taken from the landfill area, had a concentration level of 119 ppb.

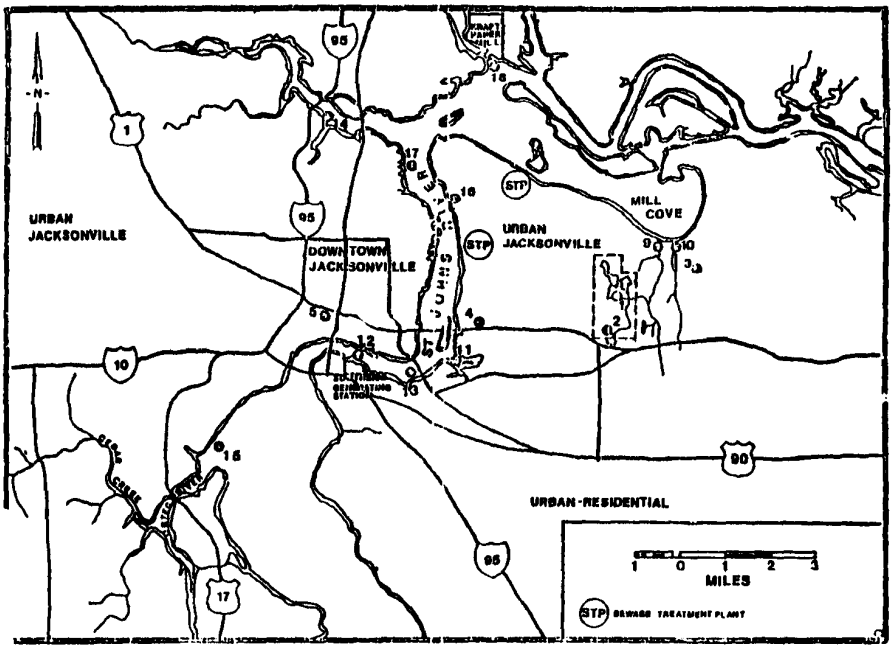

FIGURE 2.6.1 SITE MAP OF JACKSONVILLE, FLORIDA, VICINITY SHOWING SAMPLE LOCATIONS

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Table 2.6-1
PCB in Ambient Air in the Vicinity of Jacksonville, Florida

Date	Period	Concentration (ng/m^3)*
11/20/75	1200-1600	9
11/20/75	1200-1800	8
11/20/75	1800-2400	9
11/20/75	2000-2400	7
11/21/75	2400-0600	4
11/21/75	0600-1200	6

* Reported as decachlorobiphenyl.

Table 2.6-2
PCB Analysis of Environmental Samples
in the Jacksonville, Florida, Area.

Sample Site	Sample Type	Site Description	Concentration (ppb)*
1	Sludge	Monterey sewage treatment plant	720
2	Soil	SW section of sludge landfill; about two years old	119
3	Soil	Air collection site	68
9	Sediment	Unnamed Stream into Mill Cove	10
10	Sediment	Ginhouse Creek	21
11	Sediment	Arlington River Mouth	74
11	Water	Arlington River Mouth	0.260
12	Sediment	Generating Station Discharge	572
13	Sediment	St. John's River	142
14	Sediment	Trout River	167
14	Water	Trout River	0.020
16	Water	Jacksonville College Pier	0.285
17	Water	St. Joseph's River	0.103
18	Water	Broward River Mouth	0.020

*Reported as decachlorobiphenyl.

SOURCE: Unpublished Report. Contract 68-01-2978, Office of Toxic Substances, EPA, December 1975.

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1. Unpublished Report. Contract 68-01-2978, Office of Toxic Substances, EPA, December 1975.

3.0 Behavior of PCB's in the Environment

In general, PCB environmental movement is dependent on stability, solubility and volatility. These properties will control their transport via air, soil, water and/or sediment depending on the partition coefficient in each phase.

General discussions on the thermal, photochemical and chemical properties of PCB's have recently been published by Hutzinger, Safe and Zitko[3]. Various transport mechanisms have received considerable attention[1,4]. Current air data indicates that the predominant movement of PCB's in the atmosphere is not attached to particulates as is the case with DDT, but rather as the free unbound molecular species.

Some of the complexities of the behavior of PCB's in the environment include the following characteristics which are discussed in subsequent sections: (a) the complexity of the composition of different Aroclor products, (b) the difficulty in definition of water solubility and adsorption properties due to the complexity of the mixtures, and (c) the variability of the volatilization process depending on the starting mixture and point of release. By coupling these areas, the focus will be on those PCB properties that influence environmental sample collection, control their distribution in environmental samples and present potential difficulties in environmental sampling.

3.1 Composition of Aroclor Products

PCB's have been manufactured in the United States by the Monsanto Industrial Chemicals Company, and marketed under the trademark Aroclor with a numerical designation[1]. Table 3-1 provides an overview of the major Aroclors produced with their associated chlorine content. These Aroclor products are mixtures of different isomers. A typical analysis of the currently produced products and the associated chlorobiphenyl composition is shown in Table 3-2. In addition, the number of potential isomers making up the chlorobiphenyl component are also indicated. From this chemical analysis, Aroclor 1221 can be considered primarily as a monochlorobiphenyl and is the only Aroclor with a significant biphenyl concentration; Aroclor 1016 and 1242 are primarily trichlorobiphenyls, with the 1016 having a lower penta and hexa content than the 1242 product; Aroclor 1254 is primarily a pentachlorobiphenyl product. The composition of the different isomers could be quite different for each product.

3.2 Water Solubility of PCB's

The water solubility of various Aroclor products and some chlorobiphenyl isomers has been measured by different investigators. A limited set of reported values are shown in Table 3-3. The solubility for all three monochlorobiphenyl isomers is listed and range from 1190-5900 ppb. The solubilities for all of the higher chlorobiphenyl isomers have not been measured and only representative compounds are listed to

show the range of values possible and the component solubilities for Aroclor 1221; for the dichlorobiphenyls, the 2,4-, 2,2'-, 2,4'-, 4,4'-dichloro isomer is listed out of a possible 12 isomers and range in solubility from 80-1880 ppb; for the trichlorobiphenyls, only the 2,2',5-trichloro is listed out of a possible 24 isomers; for the tetrachlorobiphenyls only the 2,2',5,5'-tetrachloro is listed out of a possible 42 isomers. These values indicate that PCB's have low solubilities and the solubility decreases with the number of chlorine atoms present in the isomer, i.e., the lower isomers containing chlorine show the highest solubility. Factors that contribute to the variability of the results include adsorption to particulate matter or wall surfaces and the slow equilibration process.

Studies by Haque et. al.,[9] show that equilibration of Aroclor 1254 in water required approximately two months to reach complete equilibrium, with the major portion being achieved in a week. These studies also included various types of adsorbent surfaces ranging from sand to highly organic soil samples. The adsorption increased strongly with the organic content of the adsorbent studied. Starting with an original concentration of Aroclor 1254 at 56 ppb, the addition of 100 grams of sand or silica gel had very little effect on the final PCB concentration. However, by adding a high organic content soil, the final PCB concentration was reduced by approximately 85%.

Studies by Eichelberger[1] on the behavior of PCB's in river water over a 16 week period indicated that the levels of recovery of 10 ppb spiked Aroclor 1242 samples were 87, 76, 73, 60, 53 and 43 percent for zero time, 1, 2, 4, 8 and 16 weeks respectively. Losses were attributed to irreversible adsorption on either the walls of the sample container or the silt contained in the sample.

3.3 Interactions of PCB's with Soils

The behavior of PCB's in soil is a complex process dependent on a number of factors which include surface properties and composition. Soil composition can be identified by characterizing the percentage of sand, silt, clay and organic carbon. Depending on the surface properties, soils can act as an effective barrier to PCB migration in landfill sites and as an effective sink to PCB's deposited via aerial fallout. Once the PCB's are adsorbed, factors that would influence PCB movement are leaching when water is added to the soil and re-evaporation under normal atmospheric conditions.

In a series of experiments directed at understanding the leaching properties for various soils that could be encountered at different landfill sites, different types of soils were studied[10]. These experiments percolated water through a column packed with soil coated with Aroclor 1016 and monitored the effluent water. Breakthrough of the PCB's was found to be related to the clay content of the soil. Soils with the higher clay content retained the PCB's. The isomer distribution in the

effluent reflected the different water solubility and adsorption properties characteristic of the Aroclor 1016 isomers. The less chlorinated isomers were more readily leached. Results from these experiments are shown in Figure 3-1, where an Aroclor 1016 and 1221 standard solution is compared with the leached mixture. A .32mm id x 50m glass capillary column coated with OV-101 was used for these separations. The flow rate was 2 ml/min helium and the column temperature was programmed from 150° to 230°C at 2°C/min. The resulting isomer distribution is significantly changed from the starting Aroclor 1016 and has a closer resemblance to the isomer pattern of Aroclor 1221. In the worst case, less than 0.05% of the total Aroclor 1016 available was leached under conditions equivalent to forty years of annual rainfall.

Adsorption and evaporation studies[5] with Aroclor 1254 on different soils show that due to the few adsorption sites in sand, sand surfaces adsorb relatively small amounts when compared to the other types of soils. Consequently the vaporization loss from a sand surface will be significantly higher than soil surfaces where it is more tightly bound. The less chlorinated isomers show a greater loss than those isomers of high chlorine content.

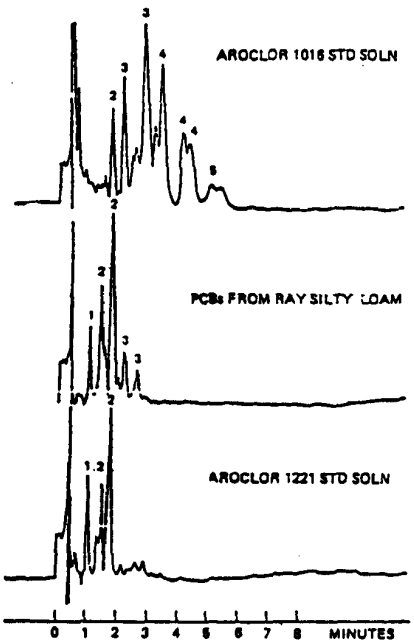
3.4 Evaporation of PCB's from Water

The transfer of PCB's from water to the air environment may be significantly faster than expected when considering that these compounds have a high molecular weight, low solubilities and low vapor pressures[5,7]. On examination, these compounds exhibit very high activity coefficients in aqueous solution resulting in high equilibrium vapor partial pressures and consequently high evaporation rates. The rates are relatively insensitive to temperature. For different Aroclor products, the evaporation process is complex depending on the chlorobiphenyl isomers present, leading to different isomer concentrations in each phase with time. The calculated half-lives for Aroclor 1242 and 1254 for a water depth of one meter are 12.1 and 10.3 hours respectively. Using the half-life values, if a monitoring station is located one hour downstream from a source of Aroclor 1242, by the time the water reaches the station, approximately one twentieth of the initial levels may have been evaporated. Where the water body is turbulent, the evaporation rate will be increased significantly.

3.5 Environmental Sampling Guidelines

Based on these limited studies, the following guidelines and precautions are suggested:

(1) laboratory measurements of field samples should be made as soon as possible;
(2) concentration levels of field samples measured in the laboratory will be lower than actual concentrations;
(3) concentration levels in water may be much lower in some areas depending on the type of suspended particulate matter;
(4) samples collected at plant outfalls will be non-equilibrium samples;



SOURCE: MIEURE, J.P., O. HICKS, R.G. KALEY AND V.W. SAEGER, "CHARACTERIZATION OF POLYCHLORINATED BIPHENYLS," NATIONAL CONFERENCE ON POLYCHLORINATED BIPHENYLS, CHICAGO, ILL., NOVEMBER 19-21, 1975

FIGURE 3-1

(5) sampling methods should be employed which reduce the problem of irreversible adsorption on container walls;
(6) soil samples will have an enriched high chlorobiphenyl isomer concentration;
(7) leached water and air samples will have an enriched lower chlorobiphenyl isomer concentration;
(8) true identification of the specific Aroclor released into the environment becomes more difficult with time.

Table 3-1
AROCLOR PRODUCTS
Produced by Monsanto

Currently in Production	Discontinued	Percent Chlorine
1221		21
	1232	32
1016		41
1242		42
	1248	48
1254		54
	1260	60
	1262	62
	1268	68

Table 3-2
COMPOSITION OF SOME AROCLORS

Chlorobiphenyl Composition	Number of Isomers	Chlorobiphenyl Percent Distribution of some Aroclors			
		1221	1016	1242	1254
$C_{12}H_{10}$		11	<0.1	<0.1	<0.1
$C_{12}H_9Cl$	3	51	1	1	<0.1
$C_{12}H_8Cl_2$	12	32	20	16	<0.5
$C_{12}H_7Cl_3$	24	4	57	49	1
$C_{12}H_6Cl_4$	42	2	21	25	21
$C_{12}H_5Cl_5$	46	<0.5	1	8	48
$C_{12}H_4Cl_6$	42	ND	<0.1	1	23
$C_{12}H_3Cl_7$	24	ND	ND	<0.1	6
$C_{12}H_2Cl_8$	12	ND	ND	ND	ND

ND = None detected, <0.1%

Table 3-3
SOLUBILITY OF AROCLORS AND CHLOROBIPHENYLS IN WATER

Aroclor Products	Solubility (ppb)	Media	References
1016	225-250	distilled water	10
1221	200	distilled water	8
1242	200	distilled water	9
1254	300-3000	fresh water	12
1254	300-1500	salt water	12
1254	50	distilled water	9
1254	56	distilled water	3
1254	40	distilled water	8
Chlorobiphenyl Compounds			
2-chlorobiphenyl*	5900		5
3-chlorobiphenyl	3500		5
4-chlorobiphenyl*	1190		5
2,4-dichlorobiphenyl	1400		5
2,4-dichlorobiphenyl	637		2
2,2'-dichlorobiphenyl*	1500		5
2,4'-dichlorobiphenyl*	1880		5
4,4'-dichlorobiphenyl*	80		5
2,2',5-trichlorobiphenyl	248		2
2,2',5,5'-tetrachlorobiphenyl	46		11
2,2',5,5'-tetrachlorobiphenyl	26		2

*These are the major chlorobiphenyl compounds in Aroclor 1221; the other major constituent is biphenyl which has a solubility in water of 4600 ppb.

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12. Zitko, V., Bull. Environ. Contam. Toxicol. 5, 279 (1970).

4 Occurrence of PCB's in Food

Some of the earliest findings of PCB's in foods occurred during the fall of 1969 in coho salmon and milk from West Virignia which was traced back to the misuse of a transformer fluid containing PCB's for defoliant spraying adjacent to dairy pasturage. In 1971 PCB's were found in poultry from North Carolina. Contaminated fish meal was implicated as the causative agent. The contamination was traced back to a leak in heat exchange equipment using PCB's. The fish meal samples examined contained from 14 to 30 ppm of PCB's. Followup sampling of eggs showed that 71 of 224 eggs contained residues in excess of 0.5 ppm ranging from 0.6 to 4.2 ppm.[1]

The Food and Drug Administration conducts a comprehensive food surveillance program yearly to determine pesticide residues, PCB's, heavy metals and other contaminants in the dietary intake of consumers in the United States and to target emerging problems and trends. The FDA has analyzed all raw agricultural commodities sampled under the pesticide surveillance program for PCB's since 1969. PCB's have been encountered most frequently in fish, both freshwater (catfish, chub, and smelt) and saltwater (porgies, sea trout, bonita and sardines), with trace levels in shellfish. Table 4-1 presents the results of this program from July 1970 to September 1971.[1]

As a result of these findings FDA proposed certain regulations for PCB concentrations in foods in 1972. These temporary tolerances are:

Commodity	PCB Concentration (ppm)
Milk (fat basis)	2.5
Dairy Products (fat basis)	2.5
Poultry (fat basis)	5.0
Eggs	0.5
Finished Animal Feed	0.2
Animal Feed Components	2.0
Fish (edible portion)	5.0
Infant and Junior Foods	0.2

Of these commodities, fish is the only food primarily contaminated by the environment (waterways). The other commodities were contaminated by industrial and agricultural uses.

In FY 73 and 74 Comprehensive Fish Surveys were carried out. While the data are valuable in showing which species and which areas are apt to be of concern, the diversity of the fish sources and reasons for collecting them make it difficult to determine if there have been any trends. In the FY 73 program no PCB's were detected in 70% of the samples; 3% contained over 1 ppm while only 0.5% had over 5 ppm of PCB's. Those over 1 ppm were generally fresh water fish or those apt to be near the shore. Carp were the only fish over 5 ppm with a high of 20.5 ppm. The FY 74 survey, which was not carried to completion, showed no PCB's in over 80% of the samples analyzed and no samples contained more than 2 ppm.[2]

The total diet studies for FY 70 and FY 71 showed composite samples containing PCB residues up to 0.36 ppm. The positive readings were found in meat, fish, poultry, dairy and the grain and cereal composites. The 0.36 ppm value was found to be caused by migration of PCB's from the grayboard container and dividers to packaged shredded wheat.[3]

The FY 73 study included thirty market basket samples from representative areas of the United States consisting of the total 14-day diet of a 15-20 year old male in the region of collection including about 117 individual food items.[4]

These 117 food items are separated after any necessary preparation into twelve food group composites for analysis:

I	Dairy Products
II	Meat, Fish and Poultry
III	Grain and Cereal Products
IV	Potatoes
V	Leafy Vegetables
VI	Legume Vegetables
VII	Root Vegetables
VIII	Garden Fruits
IX	Fruits
X	Oils, Fats and Shortening
XI	Sugar and Adjuncts
XII	Beverages (including drinking water)

Most of the PCB levels identified in 1973 were trace amounts resulting in a daily intake of only 1 µg/day. The most frequent occurrences were in the meat-fish-poultry and grain-cereal products groups with 33% and 17% positive analyses. It has been suggested that environmental contamination may be the source in the first group and lingering recycled paper contamination in the second group.

Of 30 composites examined for each commodity group PCB residues were only determined in five of the groups as follows:

Group	Frequency
Dairy Products	3
Meat, Fish and Poultry	10
Grain and Cereal Products	5
Potatoes	1
Oils, Fats and Shortening	1

The range for all 20 positives was trace to 0.073 ppm. In fact 19 of the 20 readings were only trace amounts with the 0.073 ppm composite appearing in the Grain and Cereal Products group.[1]

Data for 1974 show that there were positive readings in only two food groups: sugar and adjuncts; and meat, fish and poultry. While only 3% of the samples in the first group were positive, 43% of those in the meat, fish and poultry group were positive. The fish components

of these samples were usually the source of the contamination. Preliminary information indicated that the range of levels measured was trace to 0.05 ppm. Data for the first half of 1975 have found 40% of the meat, fish and poultry group to show positive readings for PCB's with no positives in other food group.

Using the preceding information the FDA has estimated the average daily intake from all twelve food group composites and the average daily intake from the meat fish and poultry food class as presented in Table 4-2. For those levels which were reported as trace the assumption was made to average them at 1/2 the quantitative lower level of detection; i.e., 0.025 ppm. It can be seen that there has been a decrease in the estimated total intake. This intake should level out and continue at the 1975 level as long as fish remain almost the sole source of PCB's and the entry of PCB's into waterways is not decreased.[4]

Table 4-1
PCB Residues in Selected Food Commodities
July 1970 - September 1971

	Number of samples examined	Number of samples positive	Percent positive		PCB levels, (ppm) high	Average (ppm)
Fish	670	363	54	T	35.29	1.87
Cheese	1344	91	6	T	1.0	.25
Milk	941	69	7	T	27.8	2.27
Shell eggs	550	161	29	T	3.74	.55
Fish by-products	—	13	—	T	5.0	1.17
Total (excluding fish by-product)	3505	684	19			1.14

SOURCE: Kolbye, A.D., Jr., Environmental Health Perspectives, 85-88, April 1972.

Table 4-2
Estimates of Daily PCB Intakes
Total Diet Study

Fiscal Year	Average Daily Intake of PCB's 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

SOURCE: Jelinek, D.F. and P.E. Corneliussen, National Conference on Polychlorinated Biphenyls, November 1975.

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5.0 Exposure and Biological Accumulation of PCB's in Man

5.1 National Monitoring Programs

The National Human Monitoring Program for Pesticides is conducted by the Office of Pesticide Programs, EPA. Small samples of adipose tissue from postmortem examinations and from specimens submitted for pathological examination during therapeutic surgery are collected and analyzed for the presence of chlorinated hydrocarbon insecticides. Analysis for PCB's was begun in 1968. Summaries of the data from 1971 are reported in Table 5.1-1 and from 1972 to 1974 in Table 5.1-2.

Note that although classes are different in the two sets of results, the proportion of samples showing traces of PCB's is increasing: 50.7%, 1971; 73.99%, 1972; 75.49%, 1973; 90.93%, 1974. The increase between 1973 and 1974 is dramatic. However, the percentage of tissues which contained quantifiable (greater than 1.0 ppm) of PCBs appears to be remaining relatively constant (31.1%, 38.53%, 35.08%, 40.30%, 1971-74).

5.2 Localized Studies

There have been several localized studies showing the presence of PCB's in both human plasma and milk. A 1968 study in Charleston County, South Carolina[3], reported that of 612 plasma samples collected from healthy volunteers, 45% showed some PCB residue. Levels ranged to a maximum of 29 ppb with a mean plasma residue of 2.12 ppb. This study also tested for the significance of several factors on PCB levels, concluding only that residues were more frequent and higher in whites and urban residents. A follow-up study conducted in 1972[1] in the Charleston area sampled plasma and scalp hair specimens from 37 refuse burners and 54 controls. PCB residues were not detected in hair samples, but 81% of the refuse burners (compared with only 11% of the controls) had detectable PCB residues in plasma. Median levels for those with detectable amounts in plasma were 2.6 ppb for the refuse workers and 3.7 ppb for the controls, with maximums of 14.1 ppb and 20.2 respectively.

A 1971-72 study[4] of 40 human milk samples in Colorado discovered PCB residues in 20% of the samples in ranges from 40 to 100 ppb. A 1971 study of 47 lactating women in Texas[2] reported an absence of PCB residues both in milk from all subjects and in serum from 28 subjects. A minor study of blood samples from nine cachectic patients and 15 healthy nonpatients from Missouri and surrounding states[5] reported detectable PCB residues in all patients (mean 48 ppb, range 10-100 ppb) and none detectable in the nonpatients. No conclusions were drawn.

Table 5.1-1
PCB Concentrations in Human Adipose Tissue, 1971

Sample Size	Absent Number	%	Trace to Below 1 ppm Number	%	1-2 ppm Number	%	2 ppm Number	%
637	314	49.3	125	19.6	165	25.9	33	5.2

SOURCE: Yobs, Anne R., Environmental Health Perspectives, 1, 79-81 April 1972.

Table 5.1-2
PCB Concentrations in Human Adipose Tissue, 1972-1974

	Sample Size	Absent Number	%	Below 1 ppm Number	%	1-3 ppm Number	%	3 ppm Number	%
1972	4102	1067	26.01	634	15.46	2079	50.68	322	7.85
1973	1277	313	24.51	513	40.17	378	29.60	70	5.48
1974	1047	95	9.07	530	50.62	371	35.43	51	4.87

SOURCE: Kutz, F.W., Project Officer, National Human Monitoring Program for Pesticides, Office of Pesticides Programs, U.S. EPA, Washington, DC.

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6.0 Environmental Trends

There are few data bases or environmental studies which provide an adequate basis for sound analytic determinations of national trends in environmental levels of PCB's. Such a data base would be a result of a system of studies involving consistent sampling across media, i.e., statistical samples taken at the same locations at representative time periods, analyzed according to standard protocols, taken over a period of time long enough to establish a trend. Some studies do show consistent sampling within specific media, e.g., the Great Lakes Environmental Contaminant Survey, but the literature is full of snapshot studies with neither predecessor nor successor, and baseline studies which have not been followed by any further work. Thus, whether due to funding, modification of priorities or personnel changes, long-term consistency and coordination have not been achieved. As a result, the conclusions drawn in this section represent qualitative judgements drawn from the large mass of generally uncoordinated PCB studies analyzed.

Even recognizing all the faults of the available data base, the sheer mass of data supports the conclusion that there is widespread contamination of the environment by PCB's. There are regional variations, but effects are consistent across all media (e.g., water, sediment, fish, birds), generally showing greater concentrations of PCB's in highly developed areas and in areas of industrial activity. Over the past five years of increasing recognition of and attention to the problems of PCB's, the situation has shown no improvement nationally. Conscious efforts have, however, resulted in substantial improvement in some localized instances.

PCB contamination of the nation's waterways was termed widespread by the USGS over the period 1971-1972; more recent data from the USGS up to 1975 has shown no reduction in either levels or in geographical dispersion. Although sampling stations in the USGS data base are not expected to be representative of the U.S. and the data suffers from multiple observations at some stations and spot readings at others, over the period 1971-1975 states which had reported PCB's in sediment continued to do so while six of the 23 states which had shown no contaminated samples in 1973 did report some contamination in 1974. Sediment concentrations are imperfect indicators of current contamination levels and whole water measurements would be better. However, the currently practiced analytical protocol for water measurements, which limits detectability to 0.1 ppb, leads to a preponderance of zero readings in whole water which mask the very real problem of PCB contamination in the nation's waters. In almost all cases in which samples of both whole water and bottom sediment were taken simultaneously and the sediment had a non-zero PCB level, concentration in the water was reported as zero ppb. This occurred even when the concentration in bottom sediment was as high as 4000 parts per billion! Were water measurements to be taken at the parts per trillion level as some recent studies have done, whole water readings would certainly show PCB contamination, to an extent similar to that shown by sediment readings.

The results of the U.S. Fish and Wildlife Service's fish monitoring activities show that stations which have reported high concentrations in the past continue to do so, and that the higher concentrations are associated with river systems having significant industrial activity. Such results are consistent with those for water and sediment from the USGS water quality file. The fish data, however, shows a decline in both the proportion of composites with some PCB residues and the proportion with residues in excess of 0.5 ppm in the 1970-73 sampling program. Levels of detection are generally lower in 1973 than in previous years. While there are cautionary notes (see section 2.1.5), some improvement in PCB contamination appears evident, although mostly at those stations with prior low contamination levels.

Water is recognized as being a major sink and transport mechanism for PCB's and, therefore, residue levels in water and fish are significant. Yet other media confirm the stability and breadth of contamination over the nation. Data on human adipose tissue show a steady climb over the years 1971-74 in the percent showing traces of PCB's. Soil monitoring shows that sixty percent, i.e., 3 of 5 cities sampled each year since the National Soils Monitoring Program started have had at least one positive PCB sample. Bird monitoring conducted as part of the U.S. Bureau of Fish and Wildlife's portion of the National Pesticides Monitoring Program shows residue levels in black ducks and mallards increasing between 1969 and 1972 in the Atlantic and Mississippi flyways and slightly decreasing in the Central and Pacific flyways. PCB residues in starlings for 1970, 1972 and 1974 appear to be decreasing, yet PCB residues have been found in <u>all</u> samples each year, underscoring the ubiquitous nature of PCB's throughout the country.

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15. SUPPLEMENTARY NOTES

16. ABSTRACT

Review of the current PCB data base to assess the PCB levels in the environment on a national level; the full spectrum of PCB levels reported in man and the environment were of interest. Data were obtained from a number of national monitoring programs, the literature and many unpublished reports up to December 1, 1975.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Polychlorinated Biphenyls (PCB's) Water Great Lakes Behavior Sediment Human Soil Marine Air Industrial Plants Fish Sewage Treatment Plants Birds Food		

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APPENDIX D

REVIEW OF CARCINOGENIC AND CO-CARCINOGENIC EFFECTS OF PCBs

D.1. The Experiment of Kimbrough et al. (266) on Female Sherman Rats.

This experiment followed a sequence of studies of the effects of PCBs on rats involving exposures lasting up to one year (179, 187, 189, 208). The results of these studies have been summarized in Sections III.9.1 and III.10.8 above, and were further summarized by Kimbrough (5) as follows:

> Dietary exposure of rats to the PCB's produces enlargement of the liver cells in some adult rats at a dietary level of 5 ppm (0.4 mg/kg/day) (ref. 3). Liver weights were increased in 21-day-old F_1 male weanlings when the dams were fed as little as 1 ppm of Aroclor 1254 (ref. 3). At a dietary level of 20 ppm or above of Aroclor 1254 or 1260, which is equivalent to a dietary intake of 1.6 mg/kg/body weight per day, for an extended period of time, the hepatocytes in many rats had foamy cytoplasm consistent with lipid accumulation. In addition, hyperchromatic nuclei and binucleation was observed in many liver cells. Inclusions were present in the cytoplasm of the liver cells. These inclusions on electron microscopic examination were consistent with concentrically arranged membranes surrounding lipid vacuoles. Other ultrastructural changes observed at this and higher dietary levels in rats consisted of an increase in smooth endoplasmic reticulum and atypical mytochondria. These ultrastructural changes have also been described in primates (ref. 4) and are known to occur with a number of other xenobiotics. Similar observations were made in a study with smaller numbers of animals where the rats were fed Aroclor 1242 and Aroclor 1016 (ref. 6).
>
> A brown pigment representing ceroid pigment and uroporphyrin was observed in Kupffer cells and macrophages. Particularly at higher dietary levels, livers

showed pink fluorescence under UV lights.

In addition, exposure of rats and mice to PCB's for 6 months or longer produced grayish white lesions in the liver. On microscopic examination, these lesions consisted of proliferation of glandular epithelial cells that formed ducts and were surrounded by very pronounced fibrosis (refs. 6,7). The ducts often contained cellular debris. This lesion, called adenofibrosis, was first described by Edwards and White (ref. 6) in rats fed the carcinogen butter yellow (P-dimethylaminoazobenzene).

When rats were exposed for 6 months to a dietary intake of 500 ppm Aroclor 1254 and then sacrificed first at monthly and then at bimonthly intervals, the adenofibrosis persisted through a 10-month observation period. Usually adenofibrosis in rodents occurs concomitantly with hepatocellular carcinomas and/or neoplastic nodules (hyperplastic nodules). This was true for the Balb/cJ mouse (ref. 7).

[Refs. 3-7 in this passage are the same as refs. 179, 267, 208, 268 and 189 respectively in this document.]

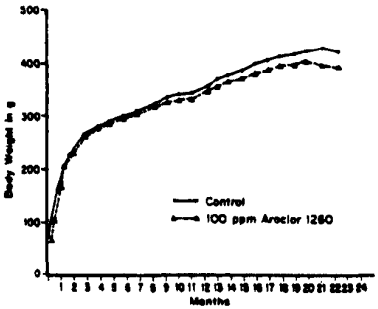
In this experiment (266) 400 weanling Sherman strain COBS female rats 21-26 days old were divided at random into two groups: 200 were maintained as controls and 200 were fed ad libitum a diet containing a nominal concentration of 100 ppm Aroclor 1260 (Lot No. AK-3, which contains 0.8 ppm of PCDFs, according to ref. 32). Measured concentrations of PCBs ranged from 70-107 ppm in the treated diet and less than 0.1 ppm in the control diet. Aflatoxins were not detectable in either diet. Based on measured food consumption, intake of PCBs declined from 11.6 mg/kg/day during the first week of exposure to 6.1 mg/kg/day at 3 months of exposure and to 4.3 mg/kg/day at 20 months (see Text-Figure 2 below). Exposure was continued

until the animals were 21.5 months old; after six weeks on an uncontaminated diet (i.e., at the age of 23 months) all surviving animals were killed and autopsied. Although the treated rats grew significantly more slowly than the controls (Text-Figure 1 below), survival was good in both groups (173/200 controls, 179/200 treated).

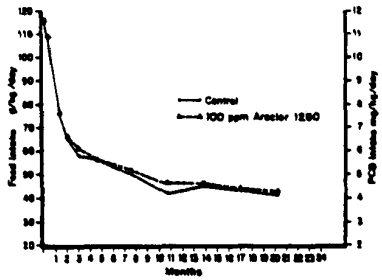
The pathological findings were reported and discussed by the authors as follows:

Pathologic Findings

Control rats (173) and experimental animals (184) were examined grossly and microscopically. The experimental group included 5 rats that were killed 1–2 months before the final kill. The remaining animals



TEXT-FIGURE 1.—Average body weight of control rats and those consuming Aroclor 1260.



TEXT-FIGURE 2.—Food intake of control and experimental rats and consumption of Aroclor 1260 by experimental rats.

were not included because of improper tissue fixation or because they died early in the experiment.

A consistent difference in the appearance of the livers was observed between the experimental and control groups. Almost all (170/184) livers of the experimental animals had from a few to multiple elevated tan nodules on the surfaces; additional nodules were usually seen on sectioning. These nodules varied from 0.1 to several cm in diameter, and in some rats replaced almost the entire liver. In contrast, the liver of only one control showed gross abnormalities and was markedly enlarged, nodular, tan, and firm. In addition, a variety of tumors of other organs was observed in both the experimental and the control groups. The incidence and type of tumors are given in table 1.

Histologic examination showed that 26 experimental animals and 1 control with enlarged, nodular livers had hepatocellular carcinomas. The additional 144 experimental rats with gross liver nodularity had hepatocellular nodules characteristic of neoplastic nodules (synonym, "hyperplastic nodules" (9)). A recent workshop sponsored by The National Cancer Institute[9] recommended the term "neoplastic nodules" for these lesions as a more accurate indication of their biologic significance. No nodules were in control animals.

The hepatocellular carcinomas were well-differentiated trabecular types (figs. 1–3), except for three in the experimental animals which had a glandular, papillary pattern (fig. 4). The trabecular tumors showed severe disruption of normal liver architecture and were usually easily recognized at low magnification. Liver plates, two or more cells thick in some areas, were arranged in haphazard linear, branching, or pseudoglandular patterns. Different patterns were usually present in the same tumor. Blunt-ended plates, sinusoidal ectasia, and congestion were frequent. The hepatocytes varied from a normal appearance to enlarged, acidophilic, or diffusely basophilic cells with large, hyperchromatic nuclei and prominent nucleoli. The cytoplasm often contained eosinophilic inclusions within vacuoles. Mitotic figures were sometimes present. Foci of coagulative necrosis were occasionally observed in cancerous areas, but there

TABLE 1.—*Incidence and type of liver lesions and tumors of other organs examined histologically*

Organ or tissue	Type of lesion	Incidence	
		Controls	Experimental
Liver	Hepatocellular carcinoma	1/173	28/184
	Neoplastic nodules	0/173	144/184
	Foci or areas of cytoplasmic alteration	28/173	182/184
Thyroid gland	Parafollicular cell tumor	37/160	18/166
Adrenal gland	Pheochromocytoma	1/173	1/187
Pituitary gland	Chromophobe adenoma	41/163	28/139
	Carcinoma	0/163	1/139
Uterus	Endometrial polyp	18/149	28/163
	Adenocarcinoma	0/149	2/163
	Sarcoma of endometrial stroma	2/149	7/163
Urinary bladder	Transitional cell papilloma	1/167	0/163
Mammary gland	Fibroadenoma	17/173[a]	13/184[a]
	Adenocarcinoma	0/173[a]	1/184[a]
Salivary gland	Fibrosarcoma	1/173[a]	0/184[a]
Lung	Adenoma	2/173	2/184
Adipose tissue	Lipoma	0/173[a]	2/184[a]
Brain	Glioma	0/173	2/184
Ovary	Granulosa theca cell tumor	8/149	0/163
	Papillary adenoma	1/149	2/163
Hematopoietic system	Granulocytic leukemia	1/173	0/184
	Lymphoma	0/173	2/184
Kidney	Hemangioma	0/173	1/184
Thymus	Thymoma	1/173[a]	0/184[a]
Parathyroid gland	Adenoma	0/173	2/184
Skin	Fibroma	0/173[a]	1/184[a]

[a] Incidence based on gross detection with microscopic confirmation.

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was no fibrosis or other evidence of chronic degenerative changes. Periodic acid-Schiff (PAS) without diastase stained carcinomas less intensely than uninvolved liver, which suggested a decrease in glycogen. Most carcinomas were more basophilic than the normal liver with azure eosin stain. No definite intravascular invasion or metastases were found.

Neoplastic nodules were generally spherical and well demarcated, and occupied areas equal to those of several liver lobules (fig. 5). The cells in these nodules were generally enlarged, and the cytoplasm was either ground-glass-appearing, diffusely basophilic, or clear. Enlarged hyperchromatic nuclei, double nuclei, and mitotic figures were often present. The cytoplasm frequently contained inclusions similar to those in the carcinomas, except they were larger and appeared as whorled, concentric lamellae. In previous studies (*3*) these formations were shown by electron microscopy to represent aggregates of smooth endoplasmic reticulum. The normal liver architecture was absent within nodules, and cells were in sheets or irregular plates. Portal areas and central veins were absent and sinusoids were dilated in some areas. At the periphery of the nodules, the surrounding liver plates were tangentially arranged and narrowed, due to compression (figs. 6, 7). Nodules varied in PAS positivity, but always differed from surrounding liver. Most nodules were more eosinophilic, and a few were more basophilic than the normal liver with azure eosin stain.

In 182 treated and 28 control animals, there were also foci or areas of hepatocytes with altered cytoplasm (figs. 8–10). In controls, these were mostly collections of cells with water-clear cytoplasm. In treated animals, most affected cells were enlarged and had eosinophilic, ground-glass-appearing cytoplasm or were diffusely basophilic and smaller than normal cells. In basophilic areas, sinusoids were dilated and liver plates somewhat tortuous. In general, the cells in these areas were like those in neoplastic nodules, but there were no architectural alterations, and plates of involved liver cells merged with the surrounding liver.

No unusual features were noted in tumors of other organs, and there were no apparent differences in incidence between experimental and control animals. Parafollicular thyroid tumors varied from small circumscribed nodules of pale, oval-to-spindle cells to masses that obliterated the gland and invaded the capsule. They were similar to those described by Boorman et al. (*10*). Other frequent tumors included pituitary chromophobe adenomas, mammary fibroadenomas, and endometrial polyps. Endometrial stromal sarcomas were also present in 10 animals.

The only bladder tumor observed was in a control animal. Therefore, the occurrence of the bladder tumor in the previous experiment was apparently unrelated to the ingestion of Aroclor 1260.

A few rats in the experimental group also showed areas of adenofibrosis (synonym, "cholangiofibrosis") of the liver, a lesion described previously following the administration of Aroclor 1260 (*9*).

DISCUSSION

The livers of treated animals showed neoplastic lesions in 170 of the 184 examined, and in only 1 of 175 controls. Although only 26 of the lesions in treated rats were clearly carcinomas according to traditional histologic criteria, neoplastic nodules are part of the spectrum of response to hepatocarcinogens and must be included in the evaluation of tumorigenesis.

In the past, these liver lesions have been interpreted in various ways, and different names have been used to report them: nodular hyperplasia, hyperplastic nodules (*11*), hepatomas (*12, 13*), and hepatic nodules (*14*). Similar ambiguity exists in the classification of human liver lesions of this type (*15*).

Several studies with known carcinogens have demonstrated the development of liver nodules, indistinguishable from the neoplastic nodules in this study, before the appearance of carcinoma (*11, 16–18*). In a recent review (*9*), the biology and significance of nodules and their relationship to hepatocellular carcinoma were thoroughly discussed. In our study, Aroclor 1260 induced a spectrum of nodules and cancers in treated animals as outlined by the Rat Liver Tumor Workshop.[9]

[9] Rat Liver Tumor Workshop at Silver Spring, Md., Dec. 11–13, 1974; sponsored by Carcinogenesis, Division of Cancer Cause and Prevention, National Cancer Institute.

Comments. This was a well-designed and well-conducted study, deviating from recommended protocols in only one respect: its limitation to female rats. (In earlier studies by the same authors, males appeared as sensitive or more sensitive than females to 100 ppm Aroclor 1260, as measured by the degree of increase in liver weight -- Tables III.9.2 and III.9.3). Two other limitations may be noted: (a) the rats were not exposed prior to weaning, although the authors'

own studies (Table III.9.3) had indicated that rats exposed to PCBs during gestation and nursing show early liver changes at much lower dietary levels; (b) the rats were killed at 23 months, relatively early in comparison to the 26-30 month expected life span. Each of these design features of the experiment, although common practice in carcinogenesis bioassays, is likely to have reduced its sensitivity in demonstrating carcinogenic effects; the early termination is likely to have arrested tumors at a relatively early stage of development.

Survival of the rats was very good; the only gross indication of toxic effects was the reduced growth rate, although Aroclor 1260 is known to have other physiological and biochemical effects in rats at 100 ppm. The liver tumors and nodules were well described, well illustrated, and related to current understanding of liver pathology in rats (269). Indeed, several of the liver slides from this study were examined by a panel of expert pathologists at the workshop where the terminology used in the paper was developed (269). The only unusual aspect of the study was the finding of one hepatocellular carcinoma in an untreated rat.

This study thus provides good evidence for the hepatocarcinogenicity of Aroclor 1260 in female rats. Because only one rather high dose was used, and because the rats were exposed for less than 21 months, however, the results provide little quantitative information on dose-response relationships.

Among other tumors listed in the authors' Table 1, the only type showing a statistically significant change in incidence is the

parafollicular cell tumor of the thyroid gland, whose incidence was reduced from 37/160 to 18/166 ($P < 0.01$). It is not clear whether this reduction should be attributed to chance occurrence among the 17 organs examined, or whether it should be related to the known effects of PCBs on thyroid function (Section III.12.3).

D-2 Industrial Bio-Test Experiment with Charles River Rats.

This experiment was described in an unpublished report dated November 1971 (207) and a brief summary of results was presented at the National Conference on Polychlorinated Biphenyls in November 1975 (68). One thousand Charles River strain albino rats were divided into 10 treatment groups. 100 rats (50 male and 50 female) served as common controls; 100 rats (50 male and 50 female) were included in each of nine exposed groups, fed diets containing 1, 10 and 100 ppm of Aroclors 1242, 1254 and 1260 respectively. Dosage started when the animals were about 4-6 weeks old (mean weights 133-136 g) and continued for 24 months. However, 5 males and 5 females were sacrificed from each treatment group for interim studies after 3, 6 and 12 months, so that each treatment/sex subgroup contained only 35 animals at the start of the second year. Mortality was high, especially in the animals fed 100 ppm (Table D1), so that only 6-21 animals from each treatment/sex subgroup survived to the terminal sacrifice at 24 months (i.e., when the animals were about 25 months old). The original report gives summaries of body weights, food consumption, clinical chemistry, organ weights, and histopathological

diagnoses for each of the 20 subgroups. As in the experiment of Kimbrough et al., the principal effects were noted in the liver and in tumor incidence: these are summarized from the original report (207) in Tables D1 and D2.

Table D1 summarizes data on survival, liver weight, and tumor incidence. Liver weights were significantly increased in the animals fed 100 ppm (except the males fed Aroclor 1242); the effect was largest with Aroclor 1254. The incidence of tumors was similar in all groups, except for chromophobe adenomas of the pituitary, which were not found in controls but were found in 8 of the 9 treated groups and in 12 of the 18 subgroups. The number of surviving animals was too small to compare individual groups with controls, but if all the treated animals are considered together the increased incidence approaches statistical significance. Almost all of these pituitary tumors were found in the animals killed at 24 months, but 3 were found in animals dying at 20-23 months.

Table D2 summarizes the histopathologic findings reported in the original report (207) for the livers of control animals and those treated at 100 ppm. These diagnoses were summarized by the original pathologist as follows:

> Significant liver injury was found in animals fed 100 ppm Aroclor 1260 (T-III). The compound associated lesions consisted of vacuolar change, focal hypertrophy and focal hyperplasia. In addition, there were lesions of inflammation, necrosis, fibrosis and minor degeneration in this group which, although seen in the controls and lower test groups, were more severe and frequent in the T-III group.

Table D1. Industrial Bio-Test Rat Experiment: Basic Results.

Group	Sex	No. at 12 mo.	Survival to month 18	21	24	Liver % of body wt, 24 mo.	Animals with tumors at 20-24 months: Abdomen & Mammary	Pituitary	Others	TOTAL
CONTROL	M	35	25	19	10	3.47	1	0	1	2
	F	35	26	19	16	3.05	6	0	2	7
Aroclor 1242										
1 ppm	M	35	25	18	10	2.93	2	0	1	3
	F	35	28	24	10	3.00	10	2	2	11
10 ppm	M	35	25	20	12	3.18	2	1	1	3
	F	35	29	24	19	3.19	7	2	2	10
100 ppm	M	35	23	16	6	3.95	0	0	1	1
	F	35	27	19	10	4.82**	1	1	2	4
Aroclor 1254										
1 ppm	M	35	22	14	5	2.99	0	1	1	2
	F	35	29	24	21	3.10	11	1	2	11
10 ppm	M	35	26	18	8	3.21	0	0	2	2
	F	35	24	20	13	3.84*	6	2	2	9
100 ppm	M	35	24	17	8	4.75**	2	0	0	2
	F	35	22	18	12	8.62**	10	0	2	12
Aroclor 1260										
1 ppm	M	35	22	17	9	3.40	0	1	0	1
	F	35	27	21	16	3.35	5	3	1	6
10 ppm	M	35	20	12	6	2.87*	0	0	0	0
	F	35	26	21	18	3.19	5	3	3	8
100 ppm	M	35	22	16	6	4.67**	0	1	2	3
	F	35	23	16	12	4.42**	4	1	3	8

* $P < 0.05$, ** $P < 0.01$, significant difference from controls.

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Table D2. Liver pathology reported in Industrial Bio-Test Rat Experiment: Controls and animals fed 100 ppm PCBs.

	Controls		Aroclor 1242 100 ppm		Aroclor 1254 100 ppm		Aroclor 1260 100 ppm	
	M	F	M	F	M	F	M	F
REPORT OF NOVEMBER 1971 (ref. 207)								
Animals surviving 24 months	10	16	6	10	8	12	6	12
Animals with diagnoses reported at "final sacrifice":	11	14	6	14	11	15	10	15
Focal lymphoid infiltration	-	3	-	-	1	-	-	-
Focal degeneration	1	-	1	3	2	-	1	-
Necrosis	-	-	-	-	-	-	-	2
Vacuolar change (fatty)	-	-	3	11	3	10	2	3
Hypertrophy[a]	-	-	2	5	3	6	2	5
Hyperplasia[b]	2	2	0	3	3	3	-	6
Nodular hyperplasia	-	-	-	1	-	1	-	-
Hepatoma	-	-	-	-	-	1	-	-
Miscellaneous[c]	-	1	-	2	1	-	4	2
REPORT OF NOVEMBER 1975 (ref. 68)								
Animals with diagnoses reported at "24 month sacrifice":	-		20		27		27	
Nodular hyperplasia	-		8		13		7	
Hepatoma	-		2		4		5	
Cholangiohepatoma	-		1		2		2	

[a] Includes focal and centrolobular hypertrophy. [b] Includes ductile and focal hyperplasia. [c] Focal hepatitis 2; focal cholangiohepatitis 1; portal fibrosis 2, congestion 3, telangiectasis 1, chronic inflammation 1.

The vacuolar change was not seen in the control animals but was observed occasionally in livers from T-I (1 ppm) and T-II (10 ppm) animals. It was frequent in the T-III animals. This lesion is morphologically indicative of fatty degeneration. Formalin-frozen sections of livers from representative animals which displayed vacuolar changes were stained with Oil Red O to reveal the presence of fat. The vacuolar lesion in the cytoplasm of these cells was positively identified as fat.

The hypertrophic change found in the liver was focal and often limited to the central lobular area where groups of cells were swollen to two or three times their normal size with clear pink homogenous cytoplasm. The hyperplasia was associated with the same cells and appeared to be an extension of the hypertrophic lesion. The hyperplastic cells were also usually hypertrophic. The most severe examples of hyperplasia appeared as nodular growths with limited compression of the surrounding normal hepatic tissue.

The minor lesions of degeneration, hepatitis, ductule cell proliferation, necrosis and focal lymphoid infiltration seen in the control animals and the T-I and T-II groups are lesions of spontaneous disease and are not related to the Aroclor 1254. This level of liver disease is not unusual in rats of this age. (207, pp. 67-68.)

In 1975 the liver slides were re-examined by the original pathologist (416). Revised diagnoses are tabulated in Table D2A and summarized in the second part of Table D. Although the 1975 report states that "in most instances, the spectrum of treatment-related histopathological findings in the liver from this re-evaluation did not differ significantly from that previously reported in our original report dated November 12, 1971" (416, p. 2), the diagnoses were in fact markedly different, including 11 hepatomas, 5 cholangio-hepatomas, and 28 examples of nodular hyperplasia in the animals treated with 100 ppm.

Table D2A. Liver pathology reported in Industrial Bio-Test rat experiment: 1975 re-diagnosis (ref. 416).

Dose rate (ppm):	Control	Aroclor 1242			Aroclor 1254			Aroclor 1260		
		1	10	100	1	10	100	1	10	100
No. of animals	23	31	30	20	31	26	27	25	23	27
Vacuolar change	1	7	8	9	8	10	13	5	6	10
Focal hypertrophy	0	2	3	8	3	5	13	3	10	11
Nodular hyperplasia	1	0	2	8	0	3	13	0	9	7
Hepatoma	0	0	0	2	0	0	4	0	0	5
Ductular hyperplasia	5	3	3	3	6	3	4	6	5	14
Cholangio-hepatoma	0	0	0	1	0	0	2	0	0	2
Hepatocellular necrosis	1	1	1	0	3	1	2	4	2	6

Nodular hyperplasia was also reported in 14 animals treated with 10 ppm and in one control. The tumors were described as follows:

> "Those lesions classified as hepatomas or cholangiohepatomas were larger nodules which showed evidence of confluence or some variation in cell size, shape, staining, or a ductular or adenomatous pattern of growth. In the absence of metastasis, invasiveness, severe basophilia, mitoses, or other evidence of anaplasia, these are benign tumors rather than malignant tumors (carcinomas)." (416, p. 3)

There are substantial discrepancies between the total number of animals diagnosed and the numbers listed as surviving to 24 months in the original report (Table D2).

Comment. Although this experiment was continued for a full 24-month period and provides a useful comparison between the three Aroclors, the experimental groups were too small to provide a sensitive test for carcinogenicity. In particular, the use of only a single control group, reduced to 70 animals by interim sacrifices and to 26 by mortality in the second year, made the entire experiment very insensitive. The cause of the high mortality was not stated. The drastic changes in diagnoses between 1971 and 1975 were not explained and the tumors were inadequately described.

The 1975 report indicates that PCBs at 100 ppm in the diet induced a variety of proliferative lesions, including tumors and hyperplastic nodules, in the livers of exposed rats, and that these effects were induced at roughly similar frequency by each of the three Aroclors. No further conclusions can be drawn without a complete report and independent diagnoses of the lesions in the livers and pituitaries.

D.3 Apparent Conflict Between Results of Kimbrough and Industrial Bio-Test Experiments.

In presenting the revised results of the Industrial Bio-Test experiment, Calandra (68) suggested that they conflicted substantially with those of Kimbrough et al. Specifically, he emphasized that no hepatocellular carcinomas could be diagnosed in the Industrial Bio-Test experiment, that two consultants (in addition to the original pathologist) had reviewed the slides in 1975, and that one of these consultants had also reviewed slides from the Kimbrough, et al. experiment and "does not agree with the reported findings". However, this consultant also disagreed with the Industrial Bio-Test pathologists, reporting no tumors or even hyperplastic nodules in that experiment (417). In his review of the slides from the Kimbrough et al. experiment, this consultant diagnosed among 184 treated animals 136 hyperplastic nodules and 43 "hyperplastic nodules with atypical structures" (Table D2B). A review of this report suggests that the dispute is primarily over terminology, in particular over the severity of the criteria required for a diagnosis of neoplasia. This is an area in which the Agency has considerable expertise and has previously accepted the criteria and diagnoses of the principal pathologist* in the Kimbrough et al. study (270). Recognizing the terminological dispute, there does not seem to be substantial qualitative conflict between these two experiments. Even without the supporting evidence cited below, the two experiments together would justify a finding that Aroclor mixtures induce neoplastic transformations in rat livers.

* Dr. R. A. Squire, Head of the Tumor Pathology Section of the National Cancer Institute.

Table D2B (from ref. 417)

Differences of liver lesions in control and experimental rats treated with Aroclor 1260

	Experimentals	Controls
Vacuolization	155	13
Focal granular alteration	100	31
Single granular alteration	169	45
Single cell necrosis	125	17
Group cell necrosis	36	2
Cell enlargement	175	17
Ductal proliferation	52	3
Cholangiomatous lesion	7	-
Stern cell proliferation	121	87
Hyperplastic nodules:		
without atypia	136	9
with atypia	43	1

D.4 52-week Study by Ito et al. in Rats.

Ito et al. (190) reported a study involving 290 male Wistar rats. The sizes of the experimental groups were not precisely reported but it appears that 20 rats were used as a common control and 30 were included in each of 9 experimental groups, dosed with 100, 500, and 1000 ppm of Kanechlors 300, 400 and 500. Exposure started at 8 weeks of age and the animals were killed after 28 to 52 weeks. The results of the study are summarized in Tables D3 and D4. There were marked increases in liver weight in all treated groups and apparently high mortality in the groups fed 500 to 1000 ppm. Histopathologic changes included bile duct proliferation, cholangiofibrosis and nodular hyperplasia (Table D4). The nodular hyperplasia is not described in detail but is said to have been similar to that induced by other carcinogens. The cholangiofibrosis was apparently similar to that reported by Kimbrough et al. (191, 266): its possible relationship to carcinogenesis is discussed above.

Comment. This study was conducted for far too short a period to serve as an adequate carcinogenicity test: other defects included the small size of the control groups, the high doses used, high mortality, and inadequate description of the lesions. However, it suggests that all three Kanechlors tested induce pre-neoplastic lesions in rats exposed to high doses.

D.5 The Study of Kimura and Baba on Donryu Rats.

Kimura and Baba (271) conducted a short-term experiment in rats of the Donryu strain. Starting at 10 weeks of age, 10 males and

Tables D3 and D4 (from ref. 190)

N. ITO, ET AL.

Changes in Body and Liver Weights of Male Wistar Rats Given Polychlorinated Biphenyls (PCB)

PCB in diet (ppm)	Experimental period (weeks)	Effective No. of rats	Body weight (g) Initial	Body weight (g) Final	Liver weight (g)	Liver weight %, of body wt.
Kanechlor-500 (1000)	49.3	13	126.4±7.6*	288.2±65.7	22.2±3.3	8.0±1.1
(500)	52.0	16	122.5±6.4	378.4±38.2	19.5±2.5	5.2±0.6
(100)	52.0	25	124.8±6.2	449.6±72.7	17.3±2.1	3.7±0.6
Kanechlor-400 (1000)	40.0	10	163.0±7.8	199.6±42.8	16.7±3.4	8.4±2.5
(500)	27.8	8	188.0±6.6	209.5±43.6	12.8±2.0	6.2±1.1
(100)	39.6	16	175.0±18.8	401.8±121.9	14.4±3.4	3.6±0.7
Kanechlor-300 (1000)	32.0	15	128.3±9.0	420.8±96.5	18.4±3.0	4.3±0.4
(500)	52.0	19	133.4±7.2	457.5±80.7	18.8±3.3	4.1±0.4
(100)	52.0	22	123.2±8.3	508.3±83.4	14.8±2.1	2.9±0.2
Control	52.0	18	130.6±4.6	533.3±89.6	13.0±1.9	2.4±0.2

* Values are mean±SD

Histopathological Findings in the Liver of Male Wistar Rats Given Polychlorinated Biphenyls (PCB)

PCB in diet (ppm)	Histopathology of the liver: Oval cell proliferation	Bile duct proliferation	Fatty changes	Cell hypertrophy	Fibrosis	Cholangiofibrosis (%)	Nodular hyperplasia (%)	Amyloidosis (%)
Kanechlor-500 (1000)	+	+	−	++	++	4/13 (30.8)	5/13 (38.5)	0/13 −
(500)	++	+	+	++	+	5/16 (31.3)	3/16 (18.8)	0/16 −
(100)	++	++	±	++	−	0/25 −	3/25 (12.0)	0/25 −
Kanechlor-400 (1000)	+	+	±	++	±	2/10 (20.0)	5/10 (50.0)	0/10 −
(500)	+	+	−	+	−	0/8 −	0/8 −	0/8 −
(100)	++	++	−	+	−	0/16 −	2/16 (12.5)	2/16 (12.5)
Kanechlor-300 (1000)	−	+	+	+	−	2/15 (13.3)	0/15 (6.7)	0/15 −
(500)	−	++	+	+	−	0/19 −	0/19 −	0/19 −
(100)	−	++	+	++	−	1/22 (4.5)	0/22 −	0/22 −
Control	−	++	+	++	−	0/18 −	0/18 −	0/18 −

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10 females were exposed to Kanechlor-400 at dietary levels varying from 38.5 to 616 ppm (for dosage regime see foot of Table D5); 5 males and 5 females served as controls. The treated animals lost weight, and suffered from lung abscesses, pneumonia, spleen atrophy, and intracranial abscesses, which the authors suggest may have been due to a treatment-related loss in resistance to infection. "Multiple adenomatous nodules", which the authors interpreted as a "benign neoplastic lesion...still short of malignancy" were found in the treated females (Table D5).

Comments. In view of the small number of animals used, the high dosage, the animals' poor condition, and the early termination of the experiment, this study can be regarded as no more than a preliminary screening test. However, it did provide evidence of early neoplastic changes, but these were not described and cannot be evaluated.

D.6 The Experiment of Kimbrough and Linder on BALB/cJ Mice.

Kimbrough and Linder (191) reported an experiment in which two groups of 50 BALB/cJ male mice were fed 300 ppm of Aroclor 1254 in the diet starting at the age of 5-6 weeks. One group was exposed for 11 months; the other was fed for six months, followed by five months on untreated diet; two groups of 50 male mice served as controls. Mortality was high early in the experiment due to fighting, but there was still excess mortality in the treated groups after the mice were recaged (34/80 versus 19/77 in the two control groups). The results are summarized in Table D6. The treated mice showed gross enlargement of the liver, including 11 hepatomas and adenofibrosis in all 22 animals

Table D5 (from ref. 271)

N. T. KIMURA AND T. BABA

Pathological Findings Induced by Long-term Oral Administration of Kanechlor-400 in Rats

Rat No.	Sex	Total exp. period (days)	Approx. amount of KC-400 ingested (mg)	Body weight (g)		Liver			Other pathological findings*
				Initial	Final	Ratio to body wt. (%)	Fatty degeneration	Multiple adenomatous nodules	
1	♂	139	450	199	308	4.1	+	−	SA, Pn, LA
2	♂	256	900	192	273	5.4	+	−	
3	♂	323	1200	201	192	6.8	+	−	SA, Pn, FDAd
4	♂	339	1300	204	199	6.8	+	−	Pn, D, FDAd
5	♂	347	1300	200	328	6.2	+	−	Pn, LA
6	♂	353	1300	230	210	5.4	+	−	SA, Pn, IA
7	♂	355	1300	201	296	5.2	+	−	Pn
8	♂	357	1300	185	226	6.3	+	−	Pn, LA, FDAd
9	♂	373	1300	234	325	6.5	+	−	LA
10	♂	530	1800	228	297	5.8	+	−	LA
11	♀	244	700	158	157	5.9	−	−	LA
12	♀	357	1100	161	184	6.2	+	−	Pn, D
13	♀	388	1100	154	177	7.1	+	−	LA, D
14	♀	396	1100	165	123	7.8	+	−	Pn, LA, D
15	♀	402	1200	202	212	6.7	+	+	LA, D
16	♀	461	1500	168	148	8.4	+	+	LA, D, IA, FDAd
17	♀	477	1500	178	165	7.8	+	+	LA, D, IA, SA,
18	♀	482	1500	225	146	6.5	+	+	LA, IA, SA
19	♀	504	1500	191	167	6.5	+	+	LA, D, FN
20	♀	560	1500	209	253	6.4	+	+	D, AAd
21	♂	235		257	471	2.5	−	−	FDAd
22	♂	332		140	432	3.1	−	−	Pn, FDAd
23	♂	538		190	426	2.7	−	−	FDAd
24	♂	538		182	323	2.5	−	−	
25	♂	538		256	456	2.7	−	−	FDAd
26	♀	331		192	198	4.2	+	−	Pn
27	♀	448		192	495	2.6	−	−	Pn
28	♀	538		202	292	2.9	−	−	
29	♀	538		201	320	3.0	−	−	
30	♀	538		199	314	2.6	−	−	

* SA = splenic atrophy, Pn = pneumonia, LA = lung abscess, FDAd = fatty degeneration of the adrenal cortex, D = depilation, IA = intracranial abscess, FN = extensive fat necrosis in the abdomen, AAd = adenoma of the adrenal cortex.

Schedule of feeding:

Concn. of Kanechlor-400 (ppm)	38.5	77	154	308	616	462	——	462	——	462	
Period of feeding (days)	28	57	21	21	56	39	28	90	28	82	Total 400

A pause (——) in PCB feeding was inserted similarly in experimental and control groups, during which and after termination of diet feeding, the rats had free access to the conventional diet (CE-2).

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- *Body weights, liver weights, and hepatoma incidence in surviving mice fed Aroclor 1254 in the diet and in controls fed plain chow*

Dietary level (ppm)	Dosage level (mg/kg/day)	Time exposed to experimental diet	Total mice	Total survivors	Final body weight of survivors: arithmetic mean (g)	Liver weight: arithmetic mean (g)	Liver weight expressed as percentage of body weight	Hepatoma incidence[a] in survivors
0	0.....	0.....	50	34	38.7	1.77	5.8	0
0	0.....	0.....	50	24	39.9	1.78	5.8	0
200	10.0.....	11 months.....	50	22	33.5†	2.69†	28.5†	3(0.1–0.4 cm Ø) 3(0.5 cm Ø) 4(1.5 cm Ø)
200	Not checked....	6 months and 5 months' recovery.	50	34	31.6	2.27†	7.5†	1(0.3 cm Ø)

[a]Determined when mice were 23 months old. Ø=diameter.
†P<0.05. A t-test was used for the statistical evaluation.

Table D6 (from ref. 191)

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fed for 11 months. The pathological changes were illustrated (191, Figures 1-5) and described and discussed as follows:

> Microscopic examination of the livers of the 22 survivors in the group receiving the experimental diet for 11 months (fig. 1) showed markedly enlarged liver cells. The nuclei of the hepatocytes were enlarged, hyperchromatic, and atypical. The cytoplasm was either smooth or vacuolated. Macrophages and Kupffer's cells contained a brown pigment. Slight, diffuse interstitial fibrosis and numerous, large extracellular hyaline bodies were also seen. Some livers had extensive areas of coagulation necrosis and fibrosis (fig. 2). A general pleomorphism was noted in a number of livers. Degeneration, necrosis, and atrophy of individual liver cells were also observed.
>
> A few nuclei showed karyorrhexis. Some smaller cells and proliferation of the focal nodular type were also noted. The irregular outer surface of the liver with areas of retraction indicated that proliferation, atrophy, and regression had occurred. Occasional calcareous deposits ≈5-6μ in diameter were present. In each of the livers, several areas were seen where the parenchyma was replaced by glandular formations of proliferated epithelial cells which formed ducts and often produced mucus. These epithelial cells were surrounded by either sparse (fig. 3) or abundant connective tissue.
>
> In addition to the above findings for all 22 livers of mice treated for 11 months (table 1), 10 hepatomas were found (fig. 4); of these, 3 measured 0.1-0.4 cm in diameter, 3 measured 0.5 cm, and 4 measured 1-1.5 cm. The tumors developed in a total of 9 mice. On gross inspection, they appeared as tan nodules. On microscopic examination, they consisted of well-differentiated hepatocytes that were relatively uniform and usually smaller than the surrounding liver cells. The tumors were well circumscribed and surrounded by compressed hepatic parenchyma or strands of fibrous tissue. The larger tumors contained areas where the sinusoids were dilated and filled with a pink-staining amorphous material. On gross inspection of the organs at autopsy, metastases were not seen. However, detailed screening for metastases including serial sectioning of lungs was not undertaken.
>
> Most livers of the 24 mice surviving 6 months of dietary exposure to PCB's followed by a 5-month recovery had enlarged cells; often these liver cells had enlarged, atypical, hyperchromatic nuclei, although the livers at autopsy weighed only slightly more than those of the controls. Necrosis of individual liver cells was observed and some nuclei showed karyorrhexis.

The macrophages and Kupffer's cells usually contained a brown pigment. Many liver cells, particularly those in the periphery of the lobules, had vacuolated cytoplasm. The livers of about two-thirds of these mice showed slight-to-moderate diffuse, interstitial fibrosis. The outer surface of the livers still showed small indentations but not as pronounced as those in the group receiving PCB's for 11 months. Numerous extracellular hyaline bodies were observed in some livers. In addition, many calcareous deposits, light in the center and darker in the periphery, were also seen (fig. 5). They were more frequent in this group than in the one fed PCB's for 11 months. A few livers showed bile duct proliferation. Only 1 liver contained a small hepatoma measuring 0.3 cm in diameter. The liver cells comprising the hepatoma were well-differentiated hepatocytes.

Except for the subcutaneous abscess formations in some mice and 1 sweat gland adenoma, no other findings of note were made in the mice of any groups.

DISCUSSION

Two significant findings were made in the present study: adenofibrosis of the liver and hepatomas. Thus adenofibrosis can also be induced in mice and does not occur solely in rats where it has been described previously (*1, 3*). A similar lesion may occur in hamsters (*6*). In the mouse lesion, the epithelial component was much better differentiated and less pleomorphic than in the rat lesion (*1*). The pattern of the lesions and the interrelationship with fibrosis were the same. Ducts and cysts formed in areas of adenofibrosis in both the mouse and rat livers. Whether adenofibrosis is a precursor of a malignant lesion or occurs concomitantly with malignant lesions of the liver is still debated. In an excellent review, Stewart and Snell (*6*) concluded that the lesion showed no convincing evidence of a precancerous lesion. Reuber (*8*) postulated that cholangiocarcinomas would develop from adenofibrosis in rats as well as hamsters.

The mouse strain used in this study only rarely develops hepatomas spontaneously. Peters et al. (*5*) observed 6 hepatomas in a group of 2000 BALB/cCr mice, a subline from the Andervont subline at the National Cancer Institute; one would expect incidences similar to those observed in the BALB/cJ subline. Madison et al. (*6*) found 13 (0.62%) hepatomas in 2088 BALB/cCr mice 18 months of age. In the present study some of the controls, 1 of 24 mice fed PCB's for 6 months, and 9 of 22 mice fed PCB's for 11 months developed hepatomas.

Although the hepatomas were well differentiated, according to Andervont and Dunn (9), they represent potentially malignant lesions, a certain percentage of which can metastasize and be transplanted. From the appearance of the lesion, it is impossible to predict which tumor will be malignant. The size of the liver tumors cannot be used as a criterion. Quite possibly the tumors would have been larger and would have occurred more often had the mice been allowed to complete their lifespans.

Comments. This experiment deviated from accepted protocols for carcinogenesis bioassays primarily in its very early termination, and in being limited to males. The high mortality and the consequent small number of treated animals surviving to 11 months makes it further insensitive as more than a preliminary screening test for carcinogenicity. However, there was a significant incidence of liver tumors which, as the authors point out, are rare in control mice of this strain and are at least potentially malignant. These tumors were well described and well illustrated. Thus, despite its limitations, this experiment gives convincing evidence that Aroclor 1254 induces very early neoplastic changes in the mouse liver.

D7. The Experiment of Ito et al. with PCBs and BHC in dd Mice.

Ito et al. (192) reported an extensive series of experiments in which groups of 12-30 male dd mice were exposed to various levels of PCBs (Kanechlors KC-300, KC-400, KC-500 at 100-500 ppm in the diet) alone or in combination with one of three isomers of BHC (hexachlorocyclohexane). Two control groups contained only 6 and 20 male mice. Treatment started at the age of 8 weeks and the animals were killed and examined after 24 or 32 weeks.

Results of the experiment are summarized in Tables D7 and D8. Liver weights were markedly increased in all groups fed PCBs at all dose levels, but were only slightly increased in the groups fed isomers of BHC alone. KC-500 induced nodular hyperplasia and hepatocellular carcinoma in 12/12 mice within 32 weeks at the highest dose rate (500 ppm), but did not do so at lower dose levels; nor did KC-400 or KC-300 even at 500 ppm (Table D7). KC-500 at 250 ppm in the diet also markedly increased the carcinogenic effects of BHC (Table D8). Whereas BHC alone did not induce nodular hyperplasia or carcinomas within 24 weeks, except in the group fed α-BHC at 250 ppm, the groups fed α-BHC and β-BHC together with KC-500 developed both kinds of lesions within 24 weeks, even at a dietary level of 50 or 100 ppm BHC (Table D8). The hyperplastic nodules and carcinomas induced by PCBs were well illustrated (199, Figures 4-6) and were described as follows:

> *Histopathologic findings:* The histologic changes and incidence of nodular hyperplasias and hepatocellular carcinomas in the livers of animals in the various groups are given in table 4.
>
> Oval cell infiltration and bile-duct proliferations were rare in all groups. But hypertrophic changes of liver parenchymal cells in centrolobular areas were clear in groups that received α-BHC or β-BHC plus PCBs or α-BHC alone. Among the 90 mice in the group given 250 ppm α-BHC, nodular hyperplasia was found in 23 (76.7%) and hepatocellular carcinoma in 8 mice (26.7%). No hyperplastic nodules or hepatocellular carcinomas were seen in any other group fed α-BHC or β-BHC only. However, some mice in the groups that received 100 or 50 ppm α-BHC or 250 or 100 ppm β-BHC with PCBs developed nodular hyperplasias and well-differentiated hepatocellular carcinomas (fig. 6). The incidence of hepatocellular carcinoma in mice given 250 ppm α-BHC increased when the diet was supplemented with 250 ppm PCBs-3. No neoplastic changes were seen in groups fed α-BHC. The histologic patterns of nodular hyperplasias and hepatocellular carcinomas induced in mice by α-BHC or β-BHC were not affected by supplementation of the diet with PCBs. The histopathology of neoplasms induced by α-BHC or β-BHC was essentially the same as of that induced by PCBs in series I.

Table D7 (from ref. 199)

Histopathologic findings in the livers of male dd mice treated with technical polychlorinated biphenyls (PCBs) for 38 weeks

PCBs* in diet (ppm)	Histopathology of liver				Liver nodules (percent)	
	Oval cells	Bile-duct proliferation	Cellular hypertrophy	Amyloid degeneration (percent)	Nodular hyperplasia	Hepatocellular carcinoma
PCBs-5 (500)	+	+	++	0/12 —	7/12 (58.3)	5/12 (41.7)
(250)	±	±	+	2/12 (16.7)	0/12 —	0/12 —
(100)	−	−	±	3/12 (25.0)	0/12 —	0/12 —
PCBs-4 (500)	+	+	++	0/12 —	0/12 —	0/12 —
(250)	±	±	+	3/12 (25.0)	0/12 —	0/12 —
(100)	−	−	±	10/12 (83.3)	0/12 —	0/12 —
PCBs-3 (500)	−	−	±	1/12 (8.3)	0/12 —	0/12 —
(250)	−	−	±	4/12 (33.3)	0/12 —	0/12 —
(100)	−	−	−	10/12 (83.3)	0/12 —	0/12 —
Controls	−	−	−	0/6 —	0/6 —	0/6 —

*PCBs-5, Kanechlor 500; PCBs-4, Kanechlor 400; PCBs-3, Kanechlor 300.

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Histologically, the neoplasms induced in mouse liver by PCBs were nodular hyperplasia and well-differentiated hepatocellular carcinomas. They were similar to those described previously, produced on administration of the α-isomer of BHC (*14–16*), and to those induced by other chemical carcinogens (*17–25*).

Comment. Because of the very short periods of treatment and the small number of mice involved, especially in the control groups, this would be unacceptable as a routine carcinogenesis bioassay. No negative conclusions can be drawn from the failure of KC-400 or KC-300 to induce hyperplastic nodules or carcinomas within 32 weeks in these small groups of mice. However, their induction by KC-500 within 32 weeks is a striking positive finding. Survival of the mice was very good and the lesions were well illustrated.

The primary purpose of the experiment was to investigate synergistic effects of PCBs and BHC. The induction of hyperplastic nodules and carcinomas within 24 weeks by joint feeding of PCBs and BHC, at levels below those which had no effects in 32 weeks when fed independently, is a striking case of synergism. Because of the short duration of the experiment, this effect should probably be regarded as a case in which each chemical accelerated the action of the other. Lifetime studies at lower dietary levels would be required to demonstrate whether the life total incidence of carcinomas is increased by joint exposure.

D.8 Other Experiments on Co-Carcinogenesis in vivo.

Two other experiments have been conducted to investigate possible co-carcinogenic effects of PCBs in vivo. In an experiment with mice, PCBs (KC-400) fed in the diet did not appear to accelerate the development of cervical carcinoma induced by methylcholanthrene (272). After 4 weeks of topical application of methylcholanthrene, dysplastic changes in the cervix did not progress to invasive carcinoma in animals fed the control diet or PCBs, although they did so in animals fed DDT (272).

In another study in rats (273) feeding with PCBs (KC-500 at 500 ppm in the diet) for 24 weeks appeared to delay or inhibit the induction of nodular hyperplasia and hepatocellular carcinoma by three other carcinogens, 3'-methyl-4-dimethylaminoazobenzene (3'-Me-DAB), N-2-fluorenylacetamide (2-FAA), and diethylnitrosamine (DEN). The results of this study are summarized in Table D9. The authors speculate that the inhibitory effects may have been attributable to metabolism of primary carcinogens by PCB-stimulated microsomal enzymes, as discussed in the next section.

D.9 Activation and De-activation of Primary and Secondary Carcinogens by PCB-Stimulated Microsomal Enzymes.

In a related experiment, Reynolds et al. (274) have shown that pretreatment with PCBs (150-300 μmoles/kg for 7 days) greatly increases the susceptibility of rat livers to acute injury by vinyl-chloride monomer (VCM). Twenty-four hours after a 6-hour exposure to

Table D9 (ref. 273)

Histopathologic changes of the liver in male rats treated with PCB and chemical carcinogens for 84 weeks

Group [a]	Effective No. of rats [b]	Histopathology of liver						
		Fatty changes	Cell hypertrophy	Oval cell infiltration	Bile duct proliferation	Cholangiofibrosis [c]	Nodular hyperplasia [c]	Cancer [c]
1. 3'-Me-DAB	22	+	±	+	+	1 (4.5)	22 (95.7)	15 (68.2)
2. 2-FAA	13	±	±	++	+÷	0	13 (100.0)	7 (53.8)
3. DEN	13	+	±	+	+	0	13 (100.0)	12 (92.3)
4. PCB	19	++	++	±	+	0	0	0
5. PCB+3'-Me-DAB	23	+	+	±	±	0	0	0
6. PCB+2-FAA	17	+	±	±	±	1 (5.9)	1 (5.9)	0
7. PCB+DEN	9	++	+	±	±	1 (11.1)	0	0
8. 3'-Me-DAB+DEN	13	+	±	++	−−	4 (30.8)	13 (100.0)	12 (92.3)
9. 2-FAA+DEN	11	+	±	++	++	0	11 (100.0)	9 (81.8)
10. PCB+3'-Me-DAB+DEN	13	+	±	+	+	3 (23.1)	4 (30.8)	1 (7.7)
11. PCB+2-FAA+DEN	12	+	±	+	+	2 (14.3)	3 (25.0)	0
12. Control	10	−	−	−	−	0	0	0

[a] See footnote a, table 1.

[b] See footnote r, table 1.

[c] Number (and percent) of rats with cholangiofibrosis, nodular hyperplasia, or cancer of liver.

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5% VCM, there was extensive vacuolization of the panlobular parenchyma in livers of PCB-treated rats, but not in livers of controls similarly exposed to VCM. VCM also caused extensive necrosis in PCB-treated rat livers, and a marked increase in serum alanine α-ketoglutarate transaminase (AKT) activity, a measure of general hepatic injury (Table D10). Phenobarbital (PBT) had similar effects (274, Table D10). Reynolds et al. related this injury to the oxidation of VCM to a biologically active metabolite by the mixed function oxidase system (MFOS) stimulated by PCBs and PBT. They showed that Aroclor 1254 and PBT have especially large effects in stimulating several components of the MFOS, especially cytochrome P-450, NADPH cytochrome c-reductase, and aryl hydrocarbon hydroxylase (AHH, as determined by benzpyrene and 3,4-benzpyrene hydroxylase activities) (Figure D1). Correspondingly, Aroclor 1254 and PBT promote especially severe injury by VCM and large releases of AKT into the serum (Table D10); serum AKT is closely correlated with cytochrome P-450 content (Figure D2). By contrast the toxicity of 1,1-dichloroethylene (DCE, or vinylidene chloride) is sharply reduced by pretreatment with PCBs and PBT (Table D10 and Figure D1), indicating that DCE is metabolized to a less toxic compound. Figure D3 shows schematically how the MFOS acts to oxidize foreign chemicals, and demonstrates the key role of cytochrome P-450.

VCM is a known human liver carcinogen and its liver toxicity appears related to its carcinogenicity (275). Like most carcinogens, VCM is also a mutagen (276), but both its carcinogenicity and mutagenicity appear to depend on metabolism in the liver to an active

Table D10 (ref. 274)

Effect of MFOS Inducers on Serum Enzyme Response Following Vinyl Chloride and Dichloroethylene

Pretreatment	Serum AKT activity*	
	Vinyl chloride†	Dichloroethylene‡
Control (water)	0.32 ± 0.03	20.88 ± 3.98
PBT§	4.86 ± 2.69	11.88 ± 4.34
1254‖	5.55 ± 1.14	0.46 ± 0.01
HCB§	1.09 ± 0.30	26.02 ± 7.86
3-MC§	0.29 ± 0.08	13.94 ± 3.17
SNL§	0.28 ± 0.01	13.09 ± 1.47
PCN§	0.28 ± 0.01	11.19 ± 3.82

* Alanine ketoglutarate transaminase activity (in milligrams of pyruvate per milliliter per hour) measured according to the method of Murphy and Malley[illegible]; the value from unexposed control animals was 0.38 ± 0.08 mg pyruvate/ml/hr.

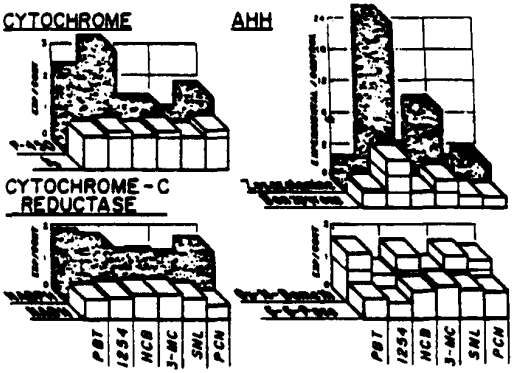
† Measured 24 hours after onset of a 6-hour exposure to 50,000 ppm.

‡ Measured 6 hours after onset of a 4-hour exposure to 200 ppm.

§ Seven-day pretreatment with 400 μmoles/kg/day.

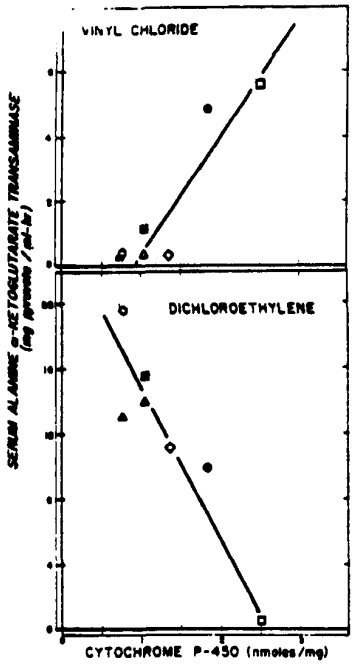
‖ Seven-day pretreatment with 300 μmoles/kg/day.

Figure D1 (ref. 274)



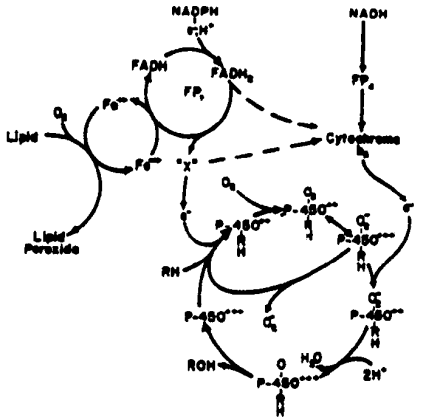
Comparison of the effects of the MFOS inducers. PBT, 1254, HCB, 3-MC, SNL, and PCN, on specific components of the microsomal multienzyme complex. All activities are on a milligram protein basis. The ordinate is expressed as the ratio of experimental to control values. Thus a value of one represents no change from control values. For the most part, each value is the average of the determinations from microsomes of at least 6 animals. Complete details of the assay conditions are described in Nebert *et al.*[274]

Figure D2 (ref. 274)



-Correlations between microsomal cytochrome P-450 content at the beginning of exposure and the serum enzyme response 24 hours after exposure to vinyl chloride and 8 hours after exposure to dichloroethylene in groups of animals given different MFOS inducers: controls, *open circles*; PBT, *solid circles*; 1254, *open squares*; HCB, *solid squares*; 3-MC, *open triangles*; 9VL, *solid triangles*; PCN, *open diamonds*. Correlations between increased SAKT following VCM and cytochrome P-450 ($r = 0.91$) and between decreased SAKT following DCE and increased cytochrome P-450 ($r = 0.95$) are significant at the 1% level

Figure D3 (ref. 274)



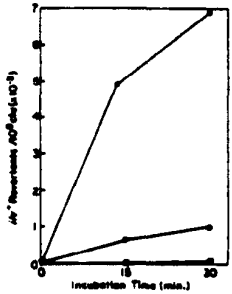
-Schematization of liver multimolecular mixed function oxidase (MFOS) system (adapted from: Estabrook, Concepts in Biochemical Pharmacology, 1971; Gillette *et al*., Ann Rev Pharmacol 12:57, 1972; and Coon *et al*., Drug Metab Disp 1:92, 1973) Electrons are transferred to the central hemoprotein (cytochrome P-450) through either the NADPH or NADH cytochrome P-450 reductase arms. For the monooxygenation process, Estabrook, Gillette, and colleagues have defined a sequence of reactions. First, the substrate (RH) combines with the oxidized form of cytochrome P-450. Second, this substrate–P-450 complex is reduced via a one electron transfer from the NADPH arm to form a reduced substrate P-450 complex. Third, the reduced complex then joins with molecular oxygen and in this triplet the oxygen is considered "active," possibly as a superoxide operates with an electron remaining between the O_2 and the heme *Fe*. A second electron is then transferred in from either NADH or NADPH via cytochrome b_5, reducing the triplet, which decomposes to product, H_2O, and oxidized P-450. Coon *et al*.[29] have suggested additional possible roles for superoxide in hydroxylation reactions, including transfer of either electron or substrate attack with oxygen insertion.

metabolite, probably chloroethylene oxide (277). The mutagenicity of VCM has been demonstrated only in the presence of liver microsomal preparations, including those stimulated by PBT and PCBs (276-278).

This is only one of numerous instances in which PCBs have been shown to activate mutagens and carcinogens by stimulating the MFOS. It is known, for example, that polycyclic aromatic hydrocarbons (PAH) require metabolic activation via the formation of K-region epoxides and cis-dihydrodiols before they become mutagenically active in bacterial and mammalian cell lines (279). The AHH enzyme system responsible for the initial epoxidation of PAH is stimulated extremely efficiently by PCBs (274, Figure D1), and 18 different PAHs have been shown to be mutagenically active using PCB-stimulated MFOS-activation (276). Indeed, it has become standard practice to use Aroclor 1254 as a MFOS-stimulator in bacterial mutagenesis bioassays to screen suspected carcinogens (276, 280).

Chemical carcinogens may be classified as secondary or primary carcinogens according to whether or not they require metabolic activation before they become active carcinogens (and mutagens) (281). VCM, as stated above, appears to be a secondary carcinogen (277). Dimethylnitrosamine (DMN) also appears to be a secondary carcinogen and its activation to a mutagen appears to depend on oxidative demethylation by the cytochrome P-450 dependent MFOS (282). Both this process and the mutagenic activity of DMN in the presence of liver microsomes are enormously enhanced by exposure to single doses of Aroclor 1254 (281, 282) (Figure D4). By contrast N-methyl-N'-nitro-N-nitroso-guanidine

Figure D4 (ref. 282)



Frequency of DMN Bu^+ revertants of *B. subtilis* 168 *Hv*. The complete mixture contained 200 m*M* DMN, 10 m*M* $MgCl_2$, 50 m*M* sx-isocitrate-Na_2, 5 m*M* NADP, 0.2 mg isocitrate dehydrogenase, 15 mg microsomal protein and 3–7 × 10^8 bacterial cfu in 2.5 ml 0.1 *M* phosphate buffer at pH 7.4 and was incubated at 37°. In control mixtures (■—■) either microsomes or the NADPH generating system were omitted. The system with normal microsomes (○—○) contained 0.7 nmole P-450/mg microsomal protein, that with PCB induced microsomes (●—●) 2.5 nmole P-450/mg microsomal protein. The activity of aminopyrine demethylation expressed as nmole HCOH formed/mg protein/min was 10.2 and 25.4 for normal and PCB induced microsomes, respectively.

(MNNG) is a primary carcinogen and its mutagenic activity is reduced by pretreatment with PCBs (281).

These findings are of profound importance in evaluating the environmental significance of PCBs, because PCB mixtures, including both those with high and low percentages of chlorine (Aroclors 1260 and 1242/1016 respectively), have been found to stimulate MFOS at extremely low doses (219, 243). The significance of these findings has been enunciated by Popper et al. (281, pp. 728-729):

> The ability of isolated microsomes to modify the biological activity of mutagens confirms the suggestion that the microsomal biotransformation system plays a key role in chemical mutagenesis. Although there is probably a relationship between carcinogenesis and mutagenesis, there are still differences which have not been explained. The observation that induced microsomes inactivate the mutagenic activity of primary carcinogens or activate that of secondary ones to a greater extent than normal ones suggests that the status of microsomal metabolism affects the response of an individual to carcinogen exposure. If we are permitted to extrapolate from mutagenesis to carcinogenesis, we might understand some old observations. Differences in the status of the biotransformation system at the time of exposure may explain why only a limited percentage of exposed individuals develop cancer. Furthermore, nutritional disturbances, particularly protein (14) or riboflavin (15) deficiency, which reduce microsomal biotransformation, may explain the geographic prevalence of certain cancers such as those of the liver (16), because malnourished populations should be more susceptible to primary carcinogens. By contrast, in the well-nourished technologic societies, the biotransformation system induced by chemical pollutants, drugs and pesticides may prove to be the greater hazard.

APPENDIX E: METABOLISM AND BIODEGRADATION

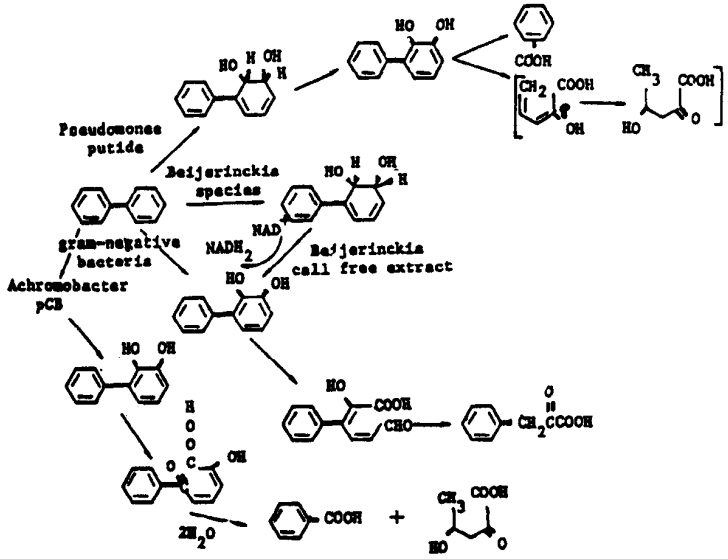
E.1. Metabolism by Bacteria

The ability of bacteria to degrade chlorinated biphenyls is dependent upon the degree of chlorination and chlorine substitution position as well as the stereo-chemistry and electronic structure of the chlorobiphenyl.

In laboratory investigations using single chlorobiphenyls and commercial mixtures, there is general agreement that lower chlorinated compounds can be degraded by microorganisms (see Figures E.2 and E.3; Tables E.1 and E.2), but there is little evidence for significant metabolism of tetra- and higher chlorobiphenyls. The mono- and dichlorinated biphenyls are readily degraded (82), as are chlorinated biphenyls with one unsubstituted ring (300).

The products resulting from microbial degradation of PCBs have been elucidated by several investigators. The products and intermediates obtained on incubating unchlorinated biphenyl with a number of microorganisms are shown in Figure E.1. If there are chlorines substituted, they appear to remain at their original point of attachment. For example, 4-chloro-biphenyl would degrade to 4-chlorobenzoic acid.

Ahmad and Focht (300) showed the preferential degradation of the unsubstituted ring of dichlorinated biphenyls using Achromobacter isolated from sewage effluent and grown on 4-chlorobiphenyl as the sole carbon source. Of the dichlorobiphenyls tested (2-3-; 2,4-;



Metabolic conversion of biphenyl by several microorganisms.
Figure E.1 (from refs. 4, 300, 301, 302)

3,4-; 3,5-; and 3,3'-dichlorobiphenyl), only 3,3' DCB has both rings substituted. Figure E.2 shows the brief lag associated with oxidation of 3,3'-DCB while all other DCBs were oxidized without a lag. The tri-CB, tetra-CB and penta-CB were oxidized slightly with the penta-CB containing an unsubstituted ring oxidized more than the tetra-CB with two substituted rings (see Figure E.3). Manometric data were used to determine the extent of oxidation. Supernatant solutions indicated no chloride, further suggesting that chlorination renders the ring resistant to degradation and associated chlorine release.

Another experiment using bacteria (Nocardia spp. and Pseudomonas spp.) grown on biphenyl supports the proposition that low chlorinated biphenyls are readily degraded (82). The components of Aroclor 1242 tend to fall into three groups: (1) mono- or di-CBs which are degraded in less than 10 days; (2) tri-CBs which are degraded rapidly after an induction lag, then are degraded more slowly as their concentration drops to 10% of the original value; and (3) tetra-CBs which are degraded most slowly (see Figures E.4 and E.5). However, there were definite differences in degradation of the same compound alone or in combination (see Table E.1). For example, 4,4'-diCB, as a component of Aroclor 1242, was degraded almost completely in 20 days when exposed to Nocardia spp. (see Figure E.4), yet 4,4'-diCB showed no detectable change after 121 days when incubated alone with Nocardia spp. (see Table E.1). Mutual solubilization may be one mechanism involved, as supported by the degradation of hexaCB in combination with two triCBs and biphenyl, but not when the biphenyl

was eliminated (see Table E.1). Co-metabolism has also been suggested to account for the microbial transformation of many pesticides in nature, compounds that do not supply the responsible populations with energy, carbon, nitrogen, or phosphorous, and may be another mechanism associated with the action of bacteria on PCBs. A more complete answer to the process of degradation is needed, as 4,4'-diCB could not be induced to degrade when mixed with 3,4,2'- and 2,3,2'-triCBs (82).

The evidence appears to support adaptation of the organisms. Thus, mono- and diCBs can probably be degraded with little further adaptation of the biphenyl-degrading bacteria (82). Increasing chlorination may require increasing induction time and be dependent upon the availability of precursors. The PCB degradation times probably reflect these adaptation periods (see Table E.1).

Woog and Kaiser (76) found that the ability of bacteria (Achromobacter sp. and Pseudomonas sp.), grown on 0.1% Aroclor 1242, to degrade the chlorinated biphenyl compounds in Aroclor 1242 mixture decreased with increasing chlorination. Specifically, the rates of degradation of biphenyl, 2-chlorobiphenyl and 4-chlorobiphenyl decreased in that order (see Figure E.6). Further, gas chromatography and mass spectrometry showed all metabolites to be aliphatic and aromatic compounds containing no chlorine. No phenols, alcohols or other oxidized derivatives of the PCBs were detected, indicating a rapid reduction of the expected intermediate hydroxylated metabolites to hydrocarbons.