Rough Draft

POLYCHLORINATED BIPHENYLS

HEALTH EFFECTS AND RECOMMENDATIONS

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III. SUMMARY AND RECOMMENDATIONS

Polychlorinated biphenyls (PCBs) are very persistent chemicals which are extremely widespread throughout the environment. They are present in varying amounts in food and drinking water. Polychlorinated biphenyls are also highly toxic agents which have been shown to cause debilitating effects in numerous animal species, including man.

Animal studies have revealed that PCBs can adversely affect numerous organ systems including the liver,[25] kidney,[23] adrenal gland,[28] spleen,[27] and skin.[20,23] Other studies have shown that it may cause chromosomal abberrations[31] as well as suppress the immune response system.[20,23,27] Polychlorinated biphenyls have been recently reported as the causative factor in the development of liver cancer in rat experiments.[38] Other very recent experiments have indicated that as little as 2.5 ppm of a PCB (Aroclor 1242) in the diet of monkeys has caused loss of hair on the face and neck, as well as the development of acne and other skin lesions. Furthermore, the reproductive capabilities of PCB-fed females were markedly affected so as to influence the development of a greater number of both resorbed and significantly undersized infants.[38]

Despite the numerous research articles pointing out the toxicity of PCBs for animal species and their possible implications for human beings, the best knowledge of how PCBs affect man comes from the "Yusho" incident where over 2,000 Japanese people actually became intoxicated by consuming food contaminated with PCBs. Many of the toxic effects which the afflicted Japanese people had were similar to results reported in animal studies. For example, there were numerous lesions of the skin, facial swelling, neurological disorders and teratogenic effects.[42]

Even though evidence is considerable that PCB exposure may be harmful to human life, the environmental levels of PCBs in Illinois still remain quite high. For example, two tributaries of Lake Michigan have levels of Aroclor 1254 consistently exceeding 100 parts per trillion (ppt) while the present EPA recommended water quality criteria is 1 ppt.[16,38] Of greater concern is the fact that the citizens of Chicago draw their drinking water from Lake Michigan which has PCB levels, in certain areas of the Lake,

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of from 4 - 10 ppt.[38] Also, aquatic animals and fish accumulate PCBs to levels more than 100 times higher than those in the surrounding water. Hence, edible fish from Lake Michigan with PCB levels exceeding the FDA tolerance limit of 5 ppm are not uncommon.[23] Such biological magnification occurs because, although the food is digested, metabolized and excreted, the PCBs are retained and concentrated (magnified) in the lipid tissue of the organism.[17] Storage of PCBs in lipid areas causes considerable concern since nerve tissue is usually insulated by various lipid layers.

Much of the theoretical human problem with respect to PCBs is that man usually resides at the top of various food chains. Once PCBs get into the food chain, biological magnification seems to occur with the concentration becoming magnified by a factor of 10-100 at each step.[23]

PCBs have been manufactured in the United States since 1929 by only the Monsanto Industrial Chemicals Company. It has been speculated that approximately 400,000 tons of PCBs have been produced between 1948 and 1973. The more highly chlorinated PCB isomers are highly resistant to microbial and physical chemical action in the environment.[2,20,23]

Despite the all too apparent environmental problems associated with PCBs, they have remarkable properties which have prompted their use in industry in numerous products such as hydraulic fluids, plasticizers, adhesives (asphalt and concrete), printing products (carbonless carbon paper), and paper coating. They are still utilized as filling agents on impregnants in electrical transformers and capacitors. Since 1971, Monsanto voluntarily restricted its sales to only "closed cycle" systems in transformers and capacitors; this undoubtedly has been a highly significant factor in restricting the accessibility of PCBs to the environment. However, PCBs were already widespread and difficult to degrade and even the cessation of their sale has little bearing on the amount already present.[2,4]

What does the future hold for PCBs as an environmental problem? The action by Monsanto to restrict PCB sales to closed systems was a major step in preventing the immediate release of PCBs to the environment. However, Monsanto is still producing about 20,000 tons of PCBs each year. This material must be properly disposed of when it is to be discarded. There are 6 major incinerators in the United States to

handle discarded PCB waste. If waste products are sent to these incinerators, needless environmental pollution could be avoided. Unfortunately, many companies discarding their old electrical units (containing PCBs) are still discharging many pounds of PCBs into the atmosphere and waterways each day. Of course, it costs money to transport waste products hundreds of miles to regional incinerators and therein lies the major problem.

As a result of the current state of our knowledge concerning the environmental and human health effects of PCBs, the Environmental Health Resource Center supports the following policy and research recommendations:

POLICY RECOMMENDATIONS

1. The only method of PCB destruction should be through properly heated and timed incinerators.
2. Due to degradation characteristics, only PCBs up to Aroclor 1248 should be used.
3. Proposed tolerances for PCBs should not be made without regard for the properties of the different Aroclor isomers.
4. The food packaging industry should either cease using recycled paper which contains PCBs, or make use of an impermeable barrier on the inner surface of the packages, thus restricting the transfer of PCBs into food.

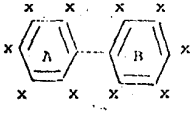
RESEARCH RECOMMENDATIONS

1. Research on the possibility of replacing PCBs with a substitute chemical in closed electrical systems and capacitors.
2. Further research for the purpose of setting standards with regard to toxicity and carcinogenicity of PCBs in humans.
3. Determine if the lack of a functional glucuronic enzyme pathway predisposes individuals to unusual accumulation of PCBs.
4. Screening of substances given to neonatals which inhibit the glucuronidation process.
5. Determination of the relative toxicity of different isomers with respect to standard setting.
6. Determination of the rate of PCB absorption through the lungs.
7. Development of appropriate animal simulation models which would lead to the most accurate extrapolative predictions for man.

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IV. CHEMICAL STRUCTURE

Polychlorinated biphenyls (PCBs) encompass a very complex heterogenous group of chemicals which have been used and manufactured in Europe, the Soviet Union and the United States.[1] In the United States, they are produced by the Monsanto Chemical Company and sold under the trade name AROCLOR.[2] The different Aroclor isomers are distinguished by using a four digit numbering system. The first two digits, 12, specify polychlorinated biphenyl and the last two digits specify the approximate percentage of chlorine in the mixture (e.g., Aroclor 1254).[2] Polychlorinated biphenyls are one member of a class of organic compounds composed of two, six carbon ring structures, with differing numbers of chlorine attachments.[3] This structure can be seen in Figure 1.



(x indicates the possible chlorine positions)

Number of Chlorine Atoms on Ring B

Number of Chlorine Atoms on Ring A	0	1	2	3	4	5
0	1	3	6	6	3	1
1		6	18	18	9	3
2			21	36	18	6
3				21	18	6
4					6	3
5						1

210 possible different isomer arrangements

Figure 1. Structure of PCB and the Number of Possible Isomers Resulting From Different Chlorine Atom Attachments.

Polychlorinated biphenyls were first identified in environmental samples in 1966. They were established as interference peaks on gas liquid

chromatographic analysis of DDT, and found to couple with DDT when analyzing the chlorinated pesticide residue.[3]

V. INDUSTRIAL USES

Polychlorinated biphenyls are available in liquid, resin and solid forms.[2,3] Table 1 denotes how each form can be used. Polychlorinated biphenyls have very broad industrial uses because of the following properties: non-flammability, high chemical stability, low vapor pressure, inert decomposition products, high dielectric strength and low dissipation factor.[12]

TABLE 1

WHERE PCBs WERE FOUND AND IN WHAT FORM PRIOR TO 1972

RESIN	LIQUID	SOLIDS
Protective coating	Dielectrics	Impregnate carbon resistors
Plasticizers and extenders	Hydraulic fluids	Sealers
Sealers in: waterproofing compounds and putty	Thermostats	Impregnating agents for electrical apparatus
Asphaltic materials	Cutting oils	
Printing inks	Extreme pressure lubricants	
Synthetic adhesives	Grinding fluids	
	Heat transfer media	

Since 1970 when the first substantial data concerning PCBs in the environment were recorded, Monsanto's use and production has been reduced; where it was an imminent health hazard, it was removed. From 1960-1970 Monsanto's peak production was 42,527 short tons of PCBs. By the end of 1972, it was down to 20,236 short tons. As of 1972 PCBs have only been used by Monsanto on closed systems such as dielectric fluid systems, capacitors and resistors.[2,4]

At one time fluorocarbons were used in capacitors, but it was found that they have low dielectric constants and also have decomposition products that are toxic. In 1969, 96% of United States transformers used mineral

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oil. This lasted until it was found that PCBs prevented fire and explosion hazards. The major value of PCB liquids with 4 or more substituted chlorines per molecule is the non-flammability of the PCB itself, as well as its decomposition products. They can be used as fluids at temperatures up to 700° F, without the danger of explosions or fire. In many areas electrical codes required the use of askarels (PCB capacitor liquids, commonly referred to as askarels, are mixtures of chlorinated biphenyls and chlorinated benzenes). The use of askarels is more expensive (1.3 X the cost of mineral oil); however, fire underwriters will not accept use of mineral oil. When fluorocarbons were tested in the U.S. their volatility and high cost quickly eliminated them from consideration for continued use.[12]

/Polychlorinated biphenyls are also used as lubricants in hydraulic fluids. Due to the fact that suitable replacments have been found, the degree to which PCBs are now being used is unknown. Tricresyl phosphate, for example, is chemically stable as an additive in lubricants and although toxic, it is more biodegradable than the PCBs.[12]

With rare exceptions, after compiling the properties needed in order to make capacitors, transformers, hydraulics, gas turbines and vacuum pumps work efficiently and safely, PCBs are by far the best chemical fluid available.

VI. WHERE PCBs ARE FOUND AND THEIR MODE OF TRANSPORT

Prior to 1970, 60% of sales were for closed systems (electrical and heat transfer uses); 25% for plasticizer applications; 10% for hydraulic fluids and lubricants; less than 5% for miscellaneous applications such as surface coating, adhesives, printing inks and pesticide extenders. Exports by Monsanto had averaged 13% of domestic sales for 1963-1970. Imports are thought to be small, comprised primarily of plasticizers in resins and adhesives, transformer oils and capacitor fluid in electrical equipment. Exports to Canada are equivalent to 7% of United States sales.[2] As of 1971, approximately 90% of sales were for closed electrical systems, and by 1972, supposedly 100% of sales were for closed electrical systems. However, it was found that an independent Chicago based chemical company is selling

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1 oz. jars of Aroclors (1248-1260) to science departments of area schools. They are being used as sealants for coverslips on microscope slides. The company, McCrone Research Institute, 2820 South Michigan Avenue, purchased large amounts prior to Monsanto's 1971 decision.[5] Consequently, they still have supplies and are selling them independently.

In the summer of 1971, Monsanto Chemical Company stopped producing Aroclor 1260. They also reduced production of the higher numbered PCBs that are considered more persistent in the environment (i.e., 5, 6, 7 chlorines/biphenyl). The more highly chlorinated isomeric forms of Aroclors (1254 and up) comprised 30% of sales prior to 1971; this fell to 22% by 1972.[2]

VII. ROUTES INTO THE ENVIRONMENT

Data on the amount of PCBs lost into the environment are limited. Theoretically, possible routes that have been considered are the following:[2]

1. Leaks from sealed transformers and heat exchangers.
2. Leaks of PCB containing fluids from hydraulic systems which are only partially sealed.
3. Spills and losses in the manufacturing of PCBs or PCB containing fluids.
4. Vaporization or leaching from PCB containing formulations.
5. Disposal of waste PCBs or PCB containing fluids.

Any of these possible routes of PCB movements into the environment could eventually end up in the food chain. Examples are the Japanese rice oil contamination resulting from heat exchanger leaks[6] and the Escambia Bay area of Florida where leakage of hydraulic fluid from an air compressor contaminated chicken feed on the Holly Farms.[7]

The input into soil by use of Aroclors as pesticide extenders is believed to have been small. Based on purchase reports, it has been estimated that less than 10 tons/year are dumped into soil. Direct discharge into oceans by dumping of hydraulic fluids and lubricants from ships has been considered infinitesimal, but may have been locally significant.[2]

An important factor to consider is which Aroclor isomer is being discharged. This can be determined easily from sales and chemical records.

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Most PCBs released into the atmosphere have been Aroclor 1248 and 1260, vaporized from burning dumps. Discharge into waters are primarily isomeric forms of Aroclors that are used in hydraulic fluids and lubricants, including mixtures of Aroclor 1242 and 1260. The residual in dumps will have a large fraction of the Aroclor 1242.[2]

VIII. RATE OF LOSS INTO THE ENVIRONMENT

The following figures represent orders of magnitude estimates of rates of loss. These estimates are yearly figures, most closely resembling losses incurred during 1970, which was the peak year of PCB production.

Transformers are fairly permanent installations, and therefore, only 10% of sales of transformer fluid is to replace oil that was removed. Ninety percent is for new units. The useful life of capacitors is usually under a decade. It has been estimated that the rate at which capacitors are discarded is equal to 1/2 the rate of production. A similar figure is assumed for the scrapping of heat exchangers. It is estimated that the rate of evaporation of plasticizers amounts to 10 to 20% of sales. The rate of disposal of plasticizers into dumps is assumed to equal the residual 80 to 90% of sales since numerous plastic objects have short useful lives. Hydraulic fluids and lubricants are rarely re-used; thus, it is assumed that a major fraction of these fluids used for this purpose, together with those going into miscellaneous applications, were scrapped at rates about equal to those of corresponding sales.[2]

On the basis of these gross approximations, it seems that only about 20% of the 1970 sales in North America, about 7×10^3 tons, represented a net increase in the amount of PCBs in service (in transformers, heat exchangers and capacitors). The remainder is considered to have been discharged into the environment; $1\text{-}2 \times 10^3$ tons by evaporation of plasticizers; $4\text{-}5 \times 10^3$ tons by leaks and disposal of hydraulic fluid and lubricants plus small quantities by disposal of heat transfer and transformer oils; and 22×10^3 by disposal in incinerators, dumps and sanitary landfull. Of the 22×10^3 quantity, it has been estimated that 10 to 20% (3×10^3 tons) were destroyed by burning, and 2% ($.4 \times 10^3$ tons) were

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vaporized, mainly by open burning of wire scraps, automobile components and material in dumps.[2]

IX. RATES OF TRANSPORT WITHIN THE ENVIRONMENT

Transport mechanisms take complex pathways within natural biogeochemical cycles. For example, vaporized PCBs may be absorbed on particulates, transported with the prevailing wind and deposited on land or water by particle sedimentation or rain-out. Polychlorinated biphenyls in streams may be absorbed by the waterborne particulates on benthos. Absorbed PCBs may diffuse into bottom sediments, redissolve in the water stream or be entrained into sediment eroded from bottom surface.[7,8] This whole process is further complicated by the assimilation, transport and degradation of PCBs by the biota.[9]

A major part of the PCBs discarded into dumps is in closed containers or plastic resins. This rate of loss will be low until the confining material is degraded and the PCBs released, usually diffusing slowly thru surrounding soil.[2] Polychlorinated biphenyls from discarded non-carbon paper will rapidly be diffused into the environment. However, because much of this paper is recycled, the actual rate of diffusion into the environment will often be retarded. PCB-impregnated paper is often used in the packaging of various common food products. This is now recognized as another avenue of food contamination.[10,11] Theoretically, the rates of transport may be calculated from knowledge of the physico-chemical properties of PCBs and pertinent data on atmospheric conditions, particulate transport, hydraulic dispersion, bottom sediment transport and biological degradation rates. Presently, data are too incomplete and the interactions are too complex to attempt formulation of but the crudest of transport models.[2]

To avoid needless environmental pollution with PCBs and to stop the discarding of them in dumps, rivers and soil, the Monsanto Chemical Company built an incinerator at their plant in St. Louis, Missouri. It runs on 30 million BTU's per hour, and can handle 10 million pounds per year of PCBs and other discardable chemicals. It has been shown that this 10 million pound capacity only requires 5% of use time for repairs and cleaning.[4]

There are 6 other such incinerators in the United States. They are

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located in Pittsfield, Massachusetts; Model City, New York; Wilmington, Delaware; Gloucester City, New Jersey; Baton Rouge, Louisiana; and Houston, Texas.[4] These incinerators, if timed and heated properly, can burn PCBs and not let any toxic pollutants escape into the environment. Effluent standards for PCBs are regulated by the Federal EPA's Toxic Pollutant Effluent Standards for both fresh and salt water.[13,14]

X. PCB LEVELS IN ILLINOIS

A. Levels in Lake Michigan Fish Species

The Illinois EPA initiated a program in 1970 to obtain pesticide residue levels in several species of Lake Michigan fish. In 1971 and 1972, testing included PCBs.

A total or 209 fish were analyzed over the 2 year period, including samples of yellow perch, chubs, carp, coho salmon, alewife and brown trout. Difficulties in obtaining all species in 1972 limited collections to the yellow perch and chubs. Fish were collected from commercial fishermen and the Illinois Conservation Department in the Chicago and Waukegan regions of Lake Michigan. Table 2 shows that levels found in the edible portions of species of fish with the highest fat content (chubs and alewives) also had the highest PCB levels.[15]

Recent EPA tests have found that most Lake Michigan trout and salmon longer than 12 inches exceed the 5 ppm tolerance limit set by the U.S.F.D.A.[37] Also, on May 30, 1975, the FDA seized nearly 125,000 cans of Lake Michigan coho salmon because the PCB levels in the fish ranged from 7.6 to 10.9 ppm.[38] The FDA also seized 6,480 pounds of frozen Lake Michigan coho and chinook salmon from the Point Adams Packing Company because PCB levels ranged from 8.2 to 9.2 ppm.[37] A possible explanation for the relatively low levels of PCBs in coho salmon, as reported by the EPA in Table 2, and the high concentrations recently reported by the FDA, may be that the 1971 and 1972 EPA results reported levels for specific isomers while the FDA reported the value for all PCB isomers in each fish.

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TABLE 2

POLYCHLORINATED BIPHENYLS FOUND IN EDIBLE PORTIONS OF SPECIES OF LAKE MICHIGAN FISH (1971, 1972)[15]
Values (ppm on a wet weight basis) for approximately 10 fish were used for each measurement.

SPECIES	SIZE RANGE IN INCHES	SAMPLE LOCATION	AROCLOR (MEAN VALUES) 1242	1254
Yellow Perch (1971)	7.0 - 12.0	Chicago	.1	0.3
(1972)	8.0 - 9.5	Waukegan	N.A.[a]	0.3
Chub (1971)	9.0 - 12.0	Waukegan	.3	2.6
(1972)	8.0 - 10.0	Waukegan	N.A.[a]	2.9
Carp (1971)	17.0 - 27.5	Chicago	1.0	1.5
Coho Salmon (1971)	18.0 - 23.5	Chicago	1.0	1.3
Alewife (1971)	6.0 - 7.0	Chicago	N.D.[b]	3.0

[a] N.A. - Not analyzed for
[b] N.D. - Not detected

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B. Levels in the Sediment of Lake Michigan and Some of Its Tributary Streams[15]

The results indicated that levels of Aroclor 1254 and 1242 found in Lake Michigan sediments in 1971 ranged between 2.48 and 46.92 ppb and ND (not detectable) to 106.07 ppb, respectively (Table 3 and Fig. 2). In tributary sediments, the levels for Aroclor 1242 ranged from ND to 553.00 ppb with the maximum values found at an unnamed channel in Waukegan (Table 4 and Fig. 2). Aroclor 1254 ranged from 1.54 to 232.0 ppb with the maximum level found at Pettibone Creek in North Chicago (Table 4).

C. Levels in the Water of Streams and Sewage Effluent Entering Lake Michigan During 1971 and 1972

The results indicated that Aroclor 1242 levels were markedly reduced in 1972 compared to 1971 in both the stream and sewage discharge water. Aroclor 1254 levels also decreased from 1971 to 1972 in sewage discharge, but stream discharge levels increased slightly. Also, in most cases, sewage treatment plant effluents were shown to discharge higher concentrations of PCBs than the tributary streams in 1971, but in 1972, PCB levels were higher in the tributary streams. Tables 5 and 6 also show levels of PCBs in comparison to DDT in Illinois streams and sewage plants tributary to Lake Michigan for the years 1971 and 1972 (Fig. 3).

Highest concentrations of PCBs in sediments were obtained in tributary streams and ravines, indicating possible contaminations by residential spraying and industrial discharge. Highest levels were obtained in industrial areas in North Chicago and Waukegan. In sediments, PCB levels were generally higher than pesticide (DDT, heptachlor expoxide, dieldrin, methoxychlor, phtalates and lindane) levels.[15]

It is also important to point out the extreme variability in PCB levels (Table 6). In 1971, for example, water from the Waukegan River showed over 1,800 ppm PCB on one day, but less than a month before, samples revealed no detectable levels.

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TABLE 3

DDT AND PCBs FOUND IN LAKE MICHIGAN SEDIMENTS IN ILLINOIS (1971)
Values are expressed in parts per billion (ppb) based on dry weight.[15]

SAMPLE LOCATION	TOTAL DDT	AROCLOR 1242	AROCLOR 1254
1 to 3 Miles Offshore			
*NSSD Park Ave. STP (Highland Park)	4.93	11.11	12.42
NSSD Lake Forest STP (Lake Forest)	2.25	10.51	7.02
NSSD Lake Bluff STP (Lake Bluff)	2.99	44.36	14.45
NSSD North Chicago STP (North Chicago)	2.55	106.07	26.54
NSSD Waukegan STP (Waukegan)	1.37	17.32	11.97
Lake Bed Station 60	16.08	4.46	8.63
Lake Bed Station 55	10.75	3.58	3.15
Lake Bed Station 52	7.02	8.48	3.33
Lake Bed Station 49	10.01	13.65	12.42
Lake Bed Station 46	25.20	49.33	38.65
Lake Bed Station 43	26.46	83.35	46.92
Lake Bed Station 40	9.85	17.23	9.38
Lake Bed Station 37	10.18	30.82	33.50
Lake Bed Station 34	3.99	1.24	2.48
Lake Bed Station 31	3.55	ND	3.87
Lake Bed Station 28	6.76	18.55	17.25
Lake Bed Station 24	5.03	2.48	5.83
Lake Bed Station 21	3.16	8.60	6.64
Lake Bed Station 18	1.36	10.53	5.56
Lake Bed Station 15	1.63	7.43	5.26
Lake Bed Station 12	1.34	19.25	17.45
Lake Bed Station 8	1.90	24.76	16.30
Lake Bed Station 5	2.23	46.11	34.52
Lake Bed Station 2	1.02	4.98	8.36
(Av.)	(6.73)	(23.66)	(14.66)

*NSSD - North Shore Sanitary District

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TABLE 4

DDT AND PCBs FOUND IN SEDIMENTS OF SEVERAL STREAMS AND RAVINES TRIBUTARY TO LAKE MICHIGAN (1971)
Values expressed in ppb.[15]

SAMPLE LOCATION	TOTAL DDT	AROCLOR 1242	AROCLOR 1254
10 to 50 Yards Upstream From Lake			
Bull Creek (Zion)	0.42	13.00	9.10
Dead River (Zion)	0.79	12.30	6.29
Unnamed channel (Waukegan)	13.79	553.00	83.04
Waukegan River (Waukegan)	10.17	374.00	131.00
Pettibone Creek (North Chicago)	134.96	173.40	232.00
Stone Gate Lane Ravine (Lake Forest)	79.35	2.48	8.74
Berry Hall School Ravine (Lake Forest)	52.98	6.34	12.34
Cary Avenue Ravine (Highland Park)	123.41	3.00	17.90
Ravine Drive Ravine (Highland Park)	84.70	ND	6.38
Park Ave Ravine (Highland Park)	201.19	4.32	1.54
Barat Ravine (Lake Forest)	5.29	4.37	2.91
Ravine Park Ravine (Lake Bluff)	40.46	1.31	2.54
Kellogg Creek (Zion)	1.48	1.77	2.56
(Av.)	(57.61)	(95.77)	(32.27)

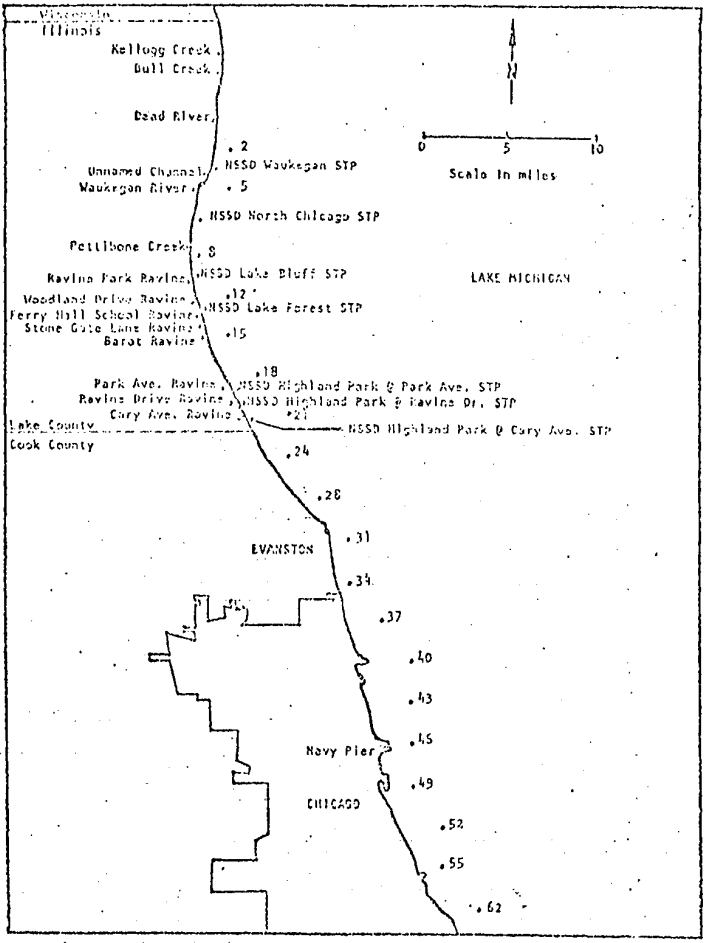


Figure 2: Locations of sediment sampling stations along Lake Michigan in Illinois [15].

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TABLE 5

DDT AND PCBs FOUND IN WATER SAMPLES FROM
ILLINOIS STREAMS TRIBUTARY TO LAKE MICHIGAN (1971-72)
Values are expressed in parts per trillion (ppt).[15]

SAMPLE LOCATION	DATE COLLECTED		TOTAL DDT	AROCLOR 1242	AROCLOR 1254
Waukegan River	8/18/71		23.7	1810.0	388.0
Waukegan River	7/29/71		15.0	ND	ND
Pettibone Creek	8/26/71		18.7	187.0	194.0
Pettibone Creek	7/29/71		20.4	140.0	192.0
		(Av.)	(19.5)	(534.2)	(193.2)
Waukegan River	5/24/72		58.1	62.0	114.0
Waukegan River	5/23/72		59.4	120.0	136.0
Waukegan River	5/25/72		39.8	57.0	61.0
Waukegan River	6/29/72		44.8	84.0	119.0
Pettibone Creek	5/24/72		57.6	ND	111.0
Pettibone Creek	5/25/72		39.8	ND	107.0
Pettibone Creek	6/29/72		56.3	653.0	841.0
			(51.0)	(125.1)	(213.0)

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TABLE 6

DDT AND PCBs FOUND IN WATER SAMPLES FROM
SEVERAL ILLINOIS SEWAGE PLANTS TRIBUTARY TO LAKE MICHIGAN (1971-72)
Values are expressed in parts per trillion (ppt).[15]

SAMPLE LOCATION	DATE COLLECTED		TOTAL DDT	AROCLOR 1242	AROCLOR 1254
NSSD Waukegan STP	9/17/71		37.7	601.0	139.0
NSSD Waukegan STP	8/04/71		27.3	2105.0	472.0
NSSD Waukegan STP	7/22/71		20.0	4020.0	568.0
NSSD North Chicago STP	7/23/71		4.5	1070.0	250.0
NSSD North Chicago STP	8/04/71		20.6	266.0	153.0
NSSD North Chicago STP	8/25/71		14.9	534.0	217.0
		(Av.)	(20.0)	(1433.0)	(295.6)
NSSD Waukegan STP	5/16/72		94.1	ND	139.0
NSSD Waukegan STP	5/19/72		101.8	ND	105.0
NSSD Waukegan STP	5/23/72		82.2	ND	97.0
NSSD Waukegan STP	5/22/72		259.0	ND	118.0
NSSD Waukegan STP	6/17/72		30.7	17.0	111.0
NSSD North Chicago STP	5/22/72		208.3	ND	104.0
NSSD North Chicago STP	5/23/72		157.1	ND	100.0
NSSD North Chicago STP	5/16/72		133.1	ND	132.0
NSSD North Chicago STP	5/19/72		134.1	ND	161.0
NSSD North Chicago STP	6/17/72		21.4	21.0	170.0
		(Av.)	(122.^)	(3.8)	(124.0)

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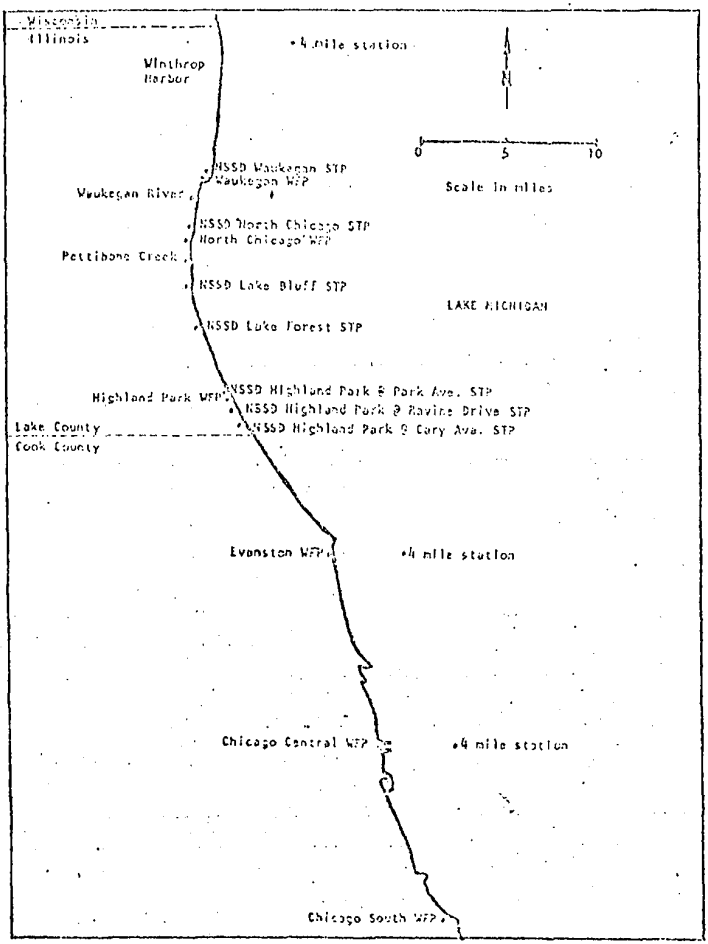


Figure 3: Location of water sampling stations, used in 1971 and 1972 for determination of PCB levels in Lake Michigan[15].

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XI. HEALTH EFFECTS OF PCBs: ANIMAL STUDIES

A. Tissue Storage, Metabolism and Excretion

Experimental exposure of rats to dietary PCBs have revealed that the different isomeric forms are retained to various degrees (as tissue residues) in most body fluids and tissue, with the highest value in adipose (fat) tissue. The adipose tissue often contained more than ten times the concentration of the tissue with the second highest PCB concentration (liver, usually) and more than 100 times the levels in other tissues including the blood, heart, kidney and brain.[16,17,18] Why different isomeric forms are retained to various degrees is still not resolved. A possible explanation includes the differential absorption through the digestive tract of the different isomers.[16]

The turnover rate of PCBs in tissue will vary with the degree of chlorination. The higher isomers are usually retained the longest in mammals and other vertebrates. It is still uncertain whether PCBs are stored in tissue until a specific point when a steady state is reached and the concentration does not increase further. The given level at which this equilibrium may be reached in adipose tissue could vary with the amount fed to the animals and the type of compound.[13,16,19]

The urinary excretion of biphenyl and 4-chlorobiphenyl has been studied in rabbits. Biphenylglucosiduronic acid and 4-hydroxybiphenyl were isolated from the urine. The rabbits fed the 4-chlorobiphenyl excreted 4-(p-chlorobiphenyl)-phenol and 4-chlorobiphenyl glucosiduronide. Twice as much 4-chlorobiphenyl as biphenyl was excreted as the glucosiduronic acid derivative. It was suggested that other low chlorinated biphenyls are excreted in a similar manner.[20]

Of dogs injected with 2,4,4'-trichloro-2'-hydroxydiphenyl ether, nearly 100% of the material is recovered as the glucuronide or sulfate in urine and feces over a 5-day period. Humans similarly exposed, excrete 65% in urine and 20% in feces after intravenous injection. It is excreted either as free compound or as a glucuronide.[19] Other evidence suggests that some chlorinated biphenyls are hydroxylated

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by species such as the rat and pigeon. No evidence of reductive dechlorination was observed in either the trout, rat, or pigeon.[21]

B. Toxic Effects on Animals

There is no consistent relationship between toxicity and the degree of chlorination which is valid for different species and different routes of exposure. For example, in rats, acute oral toxicity decreases with increasing chlorine content, while in rabbits, acute dermal toxicity appears to be highest for aroclors of intermediate chlorine content. Also, as will be seen later, most of the data on mammals concerning reproductive effects, demonstrate that toxicity decreases with increasing degrees of chlorination.[22]

1. Effects on the liver and spleen

The most significant effect of PCBs to the liver for birds and a variety of mammals (e.g., rats, rabbits, dogs) included weight increase, fatty degeneration and necrosis. Increased liver weights are probably caused by the proliferation of smooth endoplasmic reticulum. Along with such structural changes, there are increased activites of some drug metabolizing enzymes such as nitroreductase and aromatic hydroxylase.[19,23]

In a study testing the effect of PCBs on pentobarbital metabolism and alteration of sleeping time in rats, it was found that Aroclors 1254 and 1260 reduced sleeping time. Such results suggested an accelerating of pentobarbital metabolism caused by liver enzyme induction.[24]

In rats exposed to Aroclor 1242, vitamin A storage in the liver was reduced by as much as 50%, although no toxic or deficiency symptoms were seen. If only marginal amounts of vitamin A were in the diets of some rats, it has been theorized that avitaminosis could result.[25] This suggests that PCBs may alter lipid metabolism and/or affect preferential absorption from the gastrointestinal tract.

Histopathological effects in pheasants fed 210 mg of PCBs daily were observed, including loss of appetite, degeneration of liver cord cells and depletion of lymphatic nodules in the spleen.[26]

2. Effects on adrenal gland

Rats receiving 200 ppm Aroclor 1221 in their drinking water for 6 weeks, showed morphological alterations in the zone fasciculata of the adrenal gland, as well as increased levels of corticosterone. Such results have been interpreted as possible evidence of the need for a higher level of glucosteroids in defense against the stressor action of Aroclor 1221.[27]

3. Effects of PCBs on the reproductive system (not including primates)

Rats were exposed to Aroclors 1242, 1254, and 1260 in the diet at levels of 1, 10, and 100 ppm. It was found that Aroclor 1242 did not affect the first generation, but mating indices were decreased in the second generation at 100 ppm. With Aroclor 1254, the number of young delivered and the number surviving to weaning were decreased in both the second and third litters. Aroclor 1260 was found to increase the number of stillbirths at 100 ppm. No effects were seen at concentrations of 1 and 10 ppm.[22]

Studies with chickens indicated decreased egg production at 100 ppm with Aroclors 1242 and 1254, but not with Aroclor 1260. Decreased egg shell thickness occured at 10 and 100 ppm of Aroclor 1242 but only at 100 ppm with Aroclor 1254; however, such effects were not seen with Aroclor 1260 even at 100 ppm. Pheasants have also been reported to have their reproductive capacities decreased by PCBs with reduced numbers of eggs laid and reduced developmental success of those that did hatch.[22]

Biochemical studies have indicated that PCBs are capable of inducing the microsomal hydroxylating activity in the liver so as to affect the hydroxylation of both progesterone and testosterone.[28] Such biochemical findings may provide the basis for understanding how PCBs interfere with reproductive processes. Such information may shed light on the possible effects of PCBs on the depression of secondary sexual characteristics.[29]

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4. Effects on chromosomes

The effects of Aroclor 1254 (10 ppm in the diet) on dove embryo chromosomes were examined. The experiments studied the largest 8 chromosome pairs occurring in metaphase cells of allantoic sac and limb bud origin with 365 metaphase cells examined per embryo. Parameters observed were aneuploidy, polyploidy and breakage rearrangement. The results, as summarized in Table 7, provide evidence that PCBs may act as clastogenic agents.[30]

TABLE 7

THE CHROMOSOME ABERRATION RATE IN DOVE EMBRYOS TREATED WITH PCBs

	Rate	Range	Total # Sampled
Control	0.8%	0 - 2%	6
PCB Treated	1.8%	0 - 9.4%	17

5. Synergism

In one study where male rats were orally dosed with carbon tetrachloride (CCl_4) used in conjunction with Aroclor 1254, the Aroclor potentiated the toxicity of CCl_4.[17] However, when pheasants were fed PCBs along with dieldren[26] or DDE,[31] only additive and not synergistic interactions were found. (See High Risk Segments of the Population)

6. Immunosuppression

A reduction in lymphoid tissue and the presence of amyloid or amyloid-like material in the liver of PCB exposed chicks has been reported. (Amyloid formation has been found to be stimulated by certain immunosuppressive

drugs).[32] Also, lymphopenia has been reported in rabbits,[33] while in guinea pigs,[34] PCBs decreased the number of antibody-forming cells after stimulation of the humoral lymphoid system with tetanous toxoid. Such immunosuppressive actions by PCBs may help to explain the increase in susceptibility of PCB exposed fish to fungal disease,[35] PCB exposed ducks to viral hepatitis[36] and the onset of liver cancer in PCB exposed rats.[37] (See section on Carcinogenicity)

7. Carcinogenicity

In a study to be published in the Journal of the National Cancer Institute, Kimbrough et al has found that dietary Aroclor 1260 has a hepatocarcinogenic effect in female Sherman strain rats.[37]

8. Primate studies

Recent studies have indicated that very low levels of PCB exposure are toxic to primates. In one experiment, rhesus monkeys which ate food containing 25 ppm of Aroclor 1248 for 2 months developed facial swelling, loss of hair and acne lesions within a month. One monkey died from PCB intoxication 2 months after going off the experimental diet.[56] Another experiment utilized 2 groups of rhesus monkeys which ate food containing 5 and 2.5 ppm of Aroclor 1248. Within 2 months both groups had lost hair from the face and neck and their skin was of "a sandpaper-like texture". Both groups developed acne, although the 2.5 ppm groups developed the lesions later. In 6 months, both groups reached a steady state concentration of PCBs in adipose tissue. Test monkeys were then mated with monkeys that had not been fed PCBs. Six of the 8 female monkeys which had eaten food containing 5 ppm of PCBs became pregnant, in contrast to all 12 females in the control group. In the test group, 4 of the 6 pregnant females aborted or resorbed the fetus, 1 gave birth to a stillborn and 1 to a very undersized infant. In the 2.5 ppm group, all 8 females in the test group conceived and of these, 3 resorbed and 5 gave birth to undersized infants.[37]

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XII. HUMAN EXPOSURE VIA FOOD, AIR, AND WATER

A. Industrial Exposure

Several cases of human toxicity to PCBs in an industrial setting have been reported.[38,39] Exposed workers developed small dermal cysts and comedos (blackheads). These were usually found on the face and ears, although such sores have been reported on numerous other parts of the body. Dermal sores generally persisted for several months after removal from the source and in some instances, lasted four years.[40] Systemic effects have also occurred in several cases. These effects include nausea, lassitude, anorexia,[40] digestive disturbances, impotence and hematuria.[22]

The American Conference of Governmental Industrial Hygienists has set a TLV of 1 mg/m^3 for occupational exposure to vapors of Aroclor 1242, with lower values for higher Aroclors. Based on an 8-hour exposure, this would result in the inhalation of approximately 5 mg/day. Presently, it is not known how much of the inhaled PCBs humans absorb through the lungs.[22]

B. Case History of PCB Poisoning - Japan

The largest known case of PCB contamination has occurred in Japan, with documented toxic affects in over 2,000 Japanese exposed to rice oil contamination. Kaneuri Rice Oil was polluted with Kanechlor 400, which is equivalent to Aroclor 1248. This contamination incident, which was finally traced to a rice oil shipment in February, 1968, was called "Yusho" and occurred in western Japan in the area of Fukuoka prefecture. Following a latency period of 7 months, the first affected people showed the development of dermal cysts predominantly on the face and ears, but also on most other parts of the body. In severely intoxicated individuals, nausea, lassitude, anorexia, impotence and hematuria were observed. It is apparent that the severity of symptoms are directly related to the amount of PCBs ingested.[42]

It is known that the concentration of Kanechlor 400 in the rice oil was 2,000-3,000 ppm and the average quantity of rice oil consumed by patients in a first epidemiological study (325 cases) was 800 ml.[41] Consequently, the average dose of PCB ingested by affected individuals was approximately 2 g. The smallest dose ingested by a patient was estimated to be 0.5 g.[42]

The children of 13 women (9 of whom consumed between 0.3-2.6 liters of rice oil during pregnancy) were studied for possible teratogenic effects. Eleven children were born alive while 2 were stillborn.[41,42] The infants had a characteristic grayish, dark brown skin pigmentation; 5 had dark nails and gingivae (the gums; the tissues which surround the necks of the teeth and cover the alveolar processes of the maxilla and mandible) and 9 had increased eye discharge. A detailed study of 4 infants revealed abnormalities which included enlarged frontal sagittal suture. Faces were edematous, and there was an abnormal protrusion of eyeballs.[43,44] As a result of such symptoms, it has been generally concluded that placental transfer of PCBs may occur affecting the child.

The growth of school children afflicted with Yusho has also been studied. Both height and weight gain of boys with Yusho illness were significantly less than a control group while the girls of Yusho did not differ significantly from the control group.[41]

C. Consumption Via Food and Drink

Based on market basket samples of PCB levels in food, an adult is estimated to ingest approximately 5-10 µg PCB/day. This figure was based, in part, on an assumed level of 0.1 ppm in 3% of the food. Such a calculation may lead to an inaccurate representation of the total PCB intake of various consumer groups. It is known that human fish consumption and PCB levels in fish vary considerably.[22] Although the FDA tolerance for PCB residues in fish is 5 ppm,[11] it is not uncommon to find PCB levels between 10-20 ppm in fish found in the Great Lakes, especially Lake Michigan.[45] Thus, individuals who habitually eat large quantities of fish, especially from such areas as the Great Lakes,

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will have much higher intakes than the general population. For example, an individual who eats 50 g of fish per day (1.75 oz./day) containing 2 ppm of PCBs will ingest 100 µg/day of PCBs. For a person following the recommended "weight watcher's diet", the levels of PCBs in the diet may be even significantly higher. The "weight watcher diet" suggests 5 fish meals/week, which is approximately 93 g (3.5 oz.) of fish consumed per day. Assuming a contamination of 2 ppm, the "faithful weight watcher" would consume about 200 µg/day of PCB just from fish alone.

In addition to high levels of PCBs in fish, considerable concentrations may also be found in human milk. For example, samples of human milk from two cities in California contained average PCB levels of 60 ppb while average levels in human milk in Sweden and Germany were 16 ppb and 100 ppb, respectively. Based on a daily milk intake of 150 µg/Kg, breast fed infants in California would ingest about 9 µg/Kg/day of PCB (at least 3 X more than a recommended reasonable dose).[22,47]

The only report of measurement of ambient PCB levels ranged from 1-50 ng/m^3. This would result in the inhalation of less than 1 µg/day by an adult.[22] The intake from drinking PCB-contaminated water, assuming 10 ppt as the concentration, would result in about 2-5 µg/day by an adult. Lake Michigan levels are usually less than 10 ppt.[37] A range of 1-3 µg/Kg/day (70-210 µg/day for an adult) has been suggested as a "reasonable" level for an Acceptable Daily Intake.[22] One µg/Kg/day has been reported as being 100 times less than the lowest "no-effect level" reported in animal studies.[34]

XIII. PCB LEVELS IN TISSUE OF HUMANS IN THE UNITED STATES

Adipose samples were collected from tissues withdrawn for therapeutic surgery or from post-mortem exams. Positive samples were obtained from 18 states, one of which was Illinois. The other 17 states include eastern, western and southern states, thus indicating that PCBs are widespread,[48] It was reported that 165 or 637 (25.9%) samples of human adipose tissue contained 1-2 ppm. Thirty-three of the samples (5.2%) contained greater than 2 ppm; 125 (19.6%) contained between a trace and 1.0 ppm; the remaining 314 (49.3%) were negative.

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A study done on PCB concentration in the plasma of refuse workers found that 32 out of 37 (81%) of the refuse workers had detectable levels as compared to only 11% (6 out of 54) of the controls. Median PCB concentrations for those with detectable amounts were similar in the two groups; refuse workers - 2.6 ppb, controls - 3.7 ppb. The higher frequency of measurable plasma PCB levels in refuse workers is thought to reflect their increased PCB exposure from incinerated materials.[49]

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XIV. HIGH RISK SEGMENTS OF THE POPULATION

Embryos, fetuses, and neonatals (2 to 3 months old) often lack the liver microsomal enzyme systems that oxidize various natural and foreign chemicals (including those of a biphenolic nature). The activation of such enzyme systems often facilitate the detoxification and excretion of such substances. Usually by the age of 2 to 3 months, adult levels of the enzyme systems are achieved.[50] Unfortunately, this "developmental immaturity" in the unborn and the very young may predispose them to the toxic effects of certain substances since they may be unable to detoxify and excrete them as quickly as necessary. Clinical experience has shown that infants may respond differently to doses of drugs which are easily tolerated by older children and adults. Presumably, this is because older children and adults have fully functioning enzyme detoxification systems while the neonatal lacks such development.[51]

One such microsomal enzyme system includes the glucuronidation pathway which functions in the detoxification and excretion of variety of chemicals including alcohols and phenols.[50] Inefficient glucuronidative mechanisms in the very young have broad significance since the absence of such mechanisms prevents the prompt elimination of toxic substances from the body. Since some young children have been shown to consume more PCBs per unit of body weight than adults (since PCBs have appreciable concentrations in human milk),[47] the lack of appropriate detoxification enzyme systems compounds the PCB problem for the very young.

A tragic example of toxicity arising from the inability to form glucuronides has been reported. The drug chloramphenicol, which is known to be metabolized in humans by the action of the glucuronic pathway, caused the death of more than 30 premature babies who had been treated with the antibiotic for infectious diseases.[57] Theoretically, the premature babies were not able to conjugate the drug with glucuronic acid and thus the drug could only be slowly excreted and so tended to accumulate in the body to toxic proportions eventually leading to death.

An additional problem encountered by many neonatals is that approximately five percent of the mothers of normal infants secrete milk

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which inhibits the activity of glucuronyl transferase (and thus the glucuronidation process) by more than 20% via the action of a steroid present in the breast milk. Inhibition of this glucuronyl transferase has been reported for up to 49 days after birth. Clinically, these children have been reported to develop unusually severe neonatal jaundice. This condition develops because glucuronide formation, which assists in the elimination of bilirubin (breakdown product of hemoglobin), is partially inhibited. Cow's milk does not contain sufficient amounts of this steroid to affect a noticeable inhibition of the glucuronide-conjugation process.[52] Consequently, about 5% of the breast-fed neonatals would be expected to have their ability to excrete PCBs impaired. Because the benefits of breast feeding have been widely discussed in newspapers, on television and in popular magazine articles since the late 1960's in the U.S., it is expected that any problems associated with the inhibition of the glucuronide process by the steroid is more widespread than in previous decades. Administration of the antibiotic novabiocin has also been associated with unconjugated hyperbilirubinemia in infants.[53] Novabiocin is a noncompetitive inhibitor of glucuronyl transferase activity in vitro.[54] Thus, children receiving concomitant exposure of novabiocin and PCBs would be expected to have their ability to detoxify and excrete PCBs impaired.

After the neonatal period, a broad range of conditions is associated with the improper or incomplete development of the glucuronide conjugation system. The usual physiological problem associated with these conditions is the inadequate detoxification of bilirubin. This spectrum extends from the frequently occurring "mild" condition known as Gilbert's syndrome, to the very rare, but severe and often fatal Crigler and Najjar syndrome.[55]

Since the clinical effects associated with these syndromes are considered to be caused by metabolic disturbances of the glucuronide conjugation scheme, it is expected that PCB elimination in these individuals would be impeded. Although Gilbert's syndrome has been mentioned as being "encountered relatively often" in a clinical sense, no estimates of the gene frequency of such a trait has been reported.

Concomitant exposure to PCBs and other drug-like chemicals may enhance the toxic effects of PCBs. For example, rats and monkeys given

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β-diethylaminoethyl-2,2-diphenylpentanoate (SKF525A, a non-specific inhibitor for many of the microsomal enzyme activities, especially hepatic microsomal enzymes) during the initial 24 hours of exposure to PCB, succumbed rapidly as compared to the control group.[56] Of possible significance is the fact that SKF525A inhibits the proper functioning of the glucuronidation pathway.[57]

Individuals with liver infections may also be at high risk with respect to PCBs. For example, depression of glucuronide synthesis has been observed in humans with infectious hepatitis.[57]

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