& PCB

HEALTH RISKS POSED TO CONSUMERS OF FISH CONTAMINATED WITH PCBs FROM LAKE MICHIGAN

By I. C. T. Nisbet

Clement Associates, Inc.
1010 Wisconsin Avenue, N.W.
Washington, D.C. 20007

Prepared for:

U.S. Environmental Protection Agency
Region V
Chicago, Illinois

February 1981

MONS 012877

I. INTRODUCTION

This report presents estimates of the likely magnitude of risks to human health resulting from consumption of fish contaminated with polychlorinated biphenyls (PCBs), caught by sports fishermen in Lake Michigan. It includes discussion of likely additional risks to consumers of fish caught near a local source of PCBs at Waukegan, Illinois. This report is based primarily on an earlier study by Crump and Masterman (ref 1, attached hereto as Appendix A), conducted in 1979 for the U. S. Congress Office of Technology Assessment. However, this report extends the analysis by Crump and Masterman in several ways and includes a fuller discussion of the range of uncertainty in the estimates. This report does not include background information on the chemistry, environmental behavior, or toxicity of PCBs: the reader is referred to extensive accounts in references 1 - 3.

The analysis in this report progresses in three stages, as follows:

1. Estimation of the number of persons consuming substantial quantities of sports fish from Lake Michigan.
2. Estimation of the quantities of PCBs ingested by these persons.
3. Estimation of health risks resulting from this exposure.

None of these quantities has been measured directly (except (2) for a limited number of people), so these estimates are derived from available data, using various assumptions which are stated as explicitly as possible.

MONS 012878

Each of these assumptions introduces uncertainty into the final estimates of risks. As shown below, the largest uncertainties result from the use of toxicological studies in animals to estimate likely risks to humans. Because of these uncertainties, no attempt is made to make precise estimates at any stage in the analysis. In the present state of scientific knowledge, even the best-documented estimates of health risks resulting from chronic exposure to toxic chemicals are likely to be accurate only to within an order of magnitude (i.e., within a factor of about 10).

II. NUMBER OF CONSUMERS OF FISH FROM LAKE MICHIGAN

According to data cited in a report by Humphrey (ref. 4), about 381,000 licensed fishermen reside in the 18 counties bordering Lake Michigan. In 1974 these fishermen caught about 14,000,000 lb of fish in Lake Michigan, including about 4,000,000 lb each of Lake Trout and Chinook Salmon, about 3,000,000 lb of Coho Salmon, and about 2,500,000 lb of Rainbow and Steelhead Trout.

It is difficult to estimate how many people eat these fish, because fish consumption varies greatly between individuals. Assuming that each fisherman provides fish that are eaten by 2-3 people, the total number of people who eat Lake Michigan fish would be about 1 million, and their average consumption would be about 14 lb (as raw fish) annually. At the other extreme, heavy consumers of Lake Michigan fish eat between 24 and

MONS 012879

180 lb/year, with an average of about 47 lb/year (as dressed fish *) (Table 13 in ref. 4.) Assuming that about one half of each fish consists of edible tissues, this would correspond to an average of about 94 lb/year of raw fish. Hence the total catch of 14 million lb would suffice for about 150,000 heavy consumers.

Thus, two estimates of the number of consumers of Lake Michigan fish are possible: about 1,000,000 consumers (eating an average of 14 lb/year), or about 150,000 heavy consumers (eating an average of 94 lb/year). In practice, the difference between these estimates is of little consequence for risk estimation. As shown below, estimates of health risks depend primarily on the total quantity of PCBs ingested by the population, and not on their distribution among the population.

III. QUANTITIES OF PCBs INGESTED

Humphrey (ref. 4) measured concentrations of PCBs in portions of cooked fish eaten by heavy consumers of Lake Michigan fish included in his study. Average PCB concentrations in the cooked portions fell in the range 2.22 - 4.02 parts per million (ppm) for Lake Trout, 2.20 - 2.78 ppm for Salmon, and 0.59 - 1.43 ppm for other fish (Table 12 in ref. 4). These measurements were much lower than those in raw fish (Table 1 in ref. 4), indicating substantial losses during preparation and cooking.

* Ref. 4 did not state explicitly whether the figures presented for fish consumption refer to raw fish or to edible portions. However, the statements on p. 10 strongly imply the latter. Consumption of 47 lb of raw fish would be only about twice the average annual fish consumption per capita in the U. S. and would not qualify as heavy consumption.

MONS 012880

Humphrey used these direct measurements of PCB concentrations in cooked fish to estimate the average annual intake of PCBs by 91 heavy consumers as 46.5 mg/year (range 14-114 mg/year). This corresponds to an average daily intake of 130 ug/day,* or 1.7 ug/kg body weight/day (Table 14 in ref. 4). It can also be expressed as an average dietary concentration of 0.087 ppm, assuming an average daily consumption of about 1.5 kg of food per day.

For a consumer eating 14 lb of Lake Michigan fish per year, the intake of PCBs can be estimated as about 14/94 times the above estimate for heavy consumers. This leads to an estimated annual intake of 7 mg/year, an average daily intake of 19 ug/day or 0.25 ug/kg bodyweight/day, or an average dietary concentration of 0.013 ppm. These figures are estimates of the average intake of approximately 1 million potentially exposed persons (see Section II).

IV. ESTIMATES OF POTENTIAL CARCINOGENIC RISKS TO EXPOSED PERSONS

Studies of the carcinogenic (cancer-causing) effects of PCBs are reviewed and summarized in refs. 1-3 and 5. In brief summary:

1. Two studies have reported excess frequency of cancer in groups of humans highly exposed to PCBs, but these studies are not conclusive because the persons involved were also exposed to other toxic chemicals.
2. Several studies have shown that various commercial mixtures of PCBs induce cancer in rats and mice following prolonged administration.

*This quantity was erroneously tabulated as 0.130 ug/day in ref. 4, Table 12.

MONS 012881

3. PCBs increase the activity of several enzymes in the liver that can metabolize chemicals into active carcinogens and mutagens. Under appropriate circumstances, PCBs can increase or decrease the effects of other carcinogens.

These results together provide strong evidence that PCBs are likely to increase the risk of developing cancer among persons exposed to them, either directly or indirectly (by augmenting the effects of other cancer-causing agents). However, the studies in humans are insufficient to serve as the basis for estimating the magnatude of the risks. Accordingly, it is necessary to use the results of controlled experiments in laboratory animals as the basis for such estimates.

The scientific basis for the use of animal experiments to estimate risks to humans is discussed at length in pp.47 - 79 of ref. 1 (see Appendix) and in Chapter II (pp. 19 - 59) of ref. 5. The process involves two stages: (i) extrapolation of effects observed in animals at high doses to predict risks to animals at doses similar to those to which humans are exposed; (ii) prediction of low dose effects in humans from estimated low-dose effects on animals. Stage (i) involves the use of one or more mathematical models of the dose-response curve; stage (ii) involves the use of one or more scaling factors to extrapolate from animal species to humans. For stage (i), the "multi-stage" model of the dose-response curve is generally preferred, because it is based on a well-supported biological theory of the initiation and development of cancer, and because it often gives a good fit to experimental data (see Appendix A, pp. 68 - 79). Other models generally give comparable results for moderate degrees of extrapolation, but usually yield much lower estimates of risk if attempts are made to extrapolate by factors of more than 1,000 - 10,000 in dose (see Appendix A, p 76). For stage (ii), several different

MONS 012882

scaling factors are available, and it is not clear which should be used in any specific case (see below).

In the case of PCBs, only 3 experiments have been conducted for the full lifetime of the experimental animal species, and hence are suitable for estimating lifetime risks to exposed humans. The results of these experiments are summarized in refs. 1 and 2 (see Appendix A, pp 24-30), and only brief summaries are included here.

1. In an experiment reported by Kimbrough et al (ref. 6), 200 female Sherman rats were exposed for 21 months to Aroclor 1260 at a concentration of 100 ppm in the diet; 200 rats served as controls. On sacrifice at the age of 23 months, 26 of the 184 exposed rats had hepatocellular carcinomas and 144/184 had hepatic neoplastic nodules. The frequency of these pathological conditions on the controls was 1/173 and 0/173, respectively.

2. In an experiment conducted by the National Cancer Institute (ref. 7), groups of 24 Fischer 344 rats of each sex were exposed to Aroclor 1254 at one of three concentrations, 25, 50, or 100 ppm, in the diet for two years; 24 rats of each sex served as controls. Exposed rats showed a higher incidence of hepatocellular carcinomas and hepatocellular adenomas (an alternative name for "neoplastic nodules") than control rats, as shown in Table 1 below.

MONS 012883

TABLE 1. INCIDENCE OF LIVER TUMORS IN RATS EXPOSED TO AROCLOR 1254 (ref. 7)

Dose (ppm)	Hepatocellular Carcinomas		Hepatocellular Adenomas	
	Male	Female	Male	Female
0	0/24	0/23	0/24	0/23
25	0/24	0/24	0/24	0/24
50	1/24	0/22	0/24	1/22
100	2/24	0/24	1/24	2/24

MONS 012884

Although the increases were not statistically significant, the small group sizes made the experiment insensitive. Crump and Masterman (Appendix A, pp. 26-27) showed that the results in the males were consistent with those in the females in the Kimbrough experiment.

3. In an experiment conducted at Industrial Bio-Test Laboratories, groups of 50 male and 50 female Charles River rats were exposed to Aroclors 1242, 1254, or 1260 at one of three dietary concentrations, 1, 10, or 100 ppm, for two years; 50 rats of each sex served as controls. Although the survival of the rats was poor, rats exposed to each of the three PCB mixtures showed a significant increase in incidence of liver tumors and of hepatic nodular hyperplasia, as shown in Table 2 below. Although this experiment has not yet been fully reported, the results in Table 2 are useful for three purposes:

TABLE 2. INCIDENCE OF LIVER TUMORS AND NODULES IN RATS EXPOSED TO AROCLORS 1242, 1254 and 1260 (from ref. 1, Table D2A)

MIXTURE	DOSE (ppm)	"HEPATOMAS" OR "CHOLANGIOHEPATOMAS"	NODULAR HYPERPLASIA
-	0	0/23	1/23
Aroclor 1242	1	0/31	0/31
"	10	0/30	2/30
"	100	3/20	8/20
Aroclor 1254	1	0/31	0/31
"	10	0/26	3/26
"	100	6/27	13/27
Aroclor 1260	1	0/25	0/25
"	10	0/23	9/23
"	100	7/27	7/27

MONS 012886

the results in Table 2 are useful for three purposes:

(i) the results with Aroclor 1260 are quantitatively consistent with the results of the Kimbrough experiment;

(ii) they show that Aroclors 1242 and 1254, as well as Aroclor 1260, induce liver tumors in rats;

(iii) they show that Aroclors 1242 and 1254 produced similar responses in rats to those produced by Aroclor 1260 in the Kimbrough experiment (15% and 22% incidence of liver tumors, respectively, versus 14% in the Kimbrough experiment).

Since Aroclor 1248 is intermediate between (and overlaps substantially with) Aroclors 1242 and 1254 in composition (ref. 1), these results justify the use of the Kimbrough data obtained with Aroclor 1260 to predict the likely response of humans to a PCB mixture resembling Aroclor 1248.

In summary, the results of the Kimbrough study form the most satisfactory basis for quantitative extrapolation, but the results of the other two studies are consistent with it and provide evidence that it can be applied to Aroclor 1248.

Crump and Masterman (Appendix A, pp. 31-35) fitted the data from the Kimbrough study to the multi-stage model. At low doses the multi-stage model reduces to a simple linear form:

Excess probability of developing a tumor (following lifetime exposure to dietary concentration d) = $q_1 d$,

where d is the dietary concentration in ppm and q_1 is a constant to be determined from the data. Crump and Masterman calculated that $q_1 = 0.0015$ from the Kimbrough data on hepatocellular carcinomas, and $q_1 = 0.016$ from

the Kimbrough data on neoplastic nodules.* Since neoplastic nodules are usually considered to be stages in the development of liver tumors, either of these figures could be considered as estimates of tumor risk, but the latter would apply only to tumors that develop at the very end of life.

For a rat consuming food containing 0.087 ppm PCBs (the concentration appropriate to heavy human consumers of Lake Michigan fish), the lifetime risk of developing hepatocellular carcinoma is then estimated to be $0.087 \times 0.0015 = 1.3 \times 10^{-4}$, or about 1 in 8,000 (see Appendix A, p. 13). The lifetime risk of developing neoplastic nodules is about 1.4×10^{-3}, or 1 in 700.

If it could be assumed that humans and rats are equally sensitive to PCBs when the chemicals are included in the diet at the same concentration, these would be estimates of the likely excess risk of developing cancer among high consumers of fish, exposed to PCBs in the diet at an average concentration of 0.087 ppm for a lifetime. Although this is one possible way to scale doses between rats and humans, it is not the only plausible way. Three other assumptions that are frequently made are the following:

(i) Humans and rats are equally sensible to PCBs when exposed at the same dose rate (expressed in mg/kg bodyweight/day). This leads to an estimate of excess risk of 5×10^{-5} (1 in 20,000) for hepatocellular carcinomas (Appendix A, p. 37).

(ii) Humans and rats are equally sensitive to PCBs when they ingest the same total dose (expressed in mg/kg bodyweight) in the course of their

*Crump and Masterman also calculated upper statistical confidence limits on q1 as 0.0020 and 0.018 respectively. These upper confidence limits reflect statistical uncertainty resulting from the use of limited numbers of animals and are in this sense more "conservative" than the figures used in the text.

MONS 012888

lifetimes. This leads to an estimate of excess risk of 1.8×10^{-3} (1 in 550) for hepatocellular carcinomas.

(iii) Humans and rats are equally sensitive to PCBs when exposed to the same dose rate expressed in units of mg/body surface area/day. This leads to an estimate of excess risk of 4×10^{-4} (1 in 2,500) for hepatocellular carcinomas (ref. 5, p. 794, applied to dosage of 130 ug/day).

None of the above estimates, however, takes account of an important property of PCBs: their propensity to be retained for very long periods in the human body, with correspondingly high concentrations in body fluids and sensitive organs resulting from relatively small doses. Ref. 1, pp. 327 - 329, summarized evidence that PCBs are stored at least 20 times more efficiently in humans than in rats exposed to the same dietary concentrations. Ref. 4, Table 10, showed that PCB levels in human blood did not decline measurably even after 9 months of sharply reduced intakes. In contrast, PCB levels in the blood of rats declined markedly within 2 - 4 months after cessation of dosage (ref. 8).

When rats were fed a diet containing Aroclor 1254 at a concentration of 100 ppm, the concentration in the blood became constant after 2 months at about 0.40 ppm (ref. 8, Table 2). In contrast, the heavy consumers of fish in the Humphrey study, who were eating a diet containing about 0.087 ppm PCBs, had blood concentrations of PCBs about 0.058 ppm higher than controls who did not eat fish (ref. 4, Tables 8,9 and.14). In both rats and humans the ratios between PCB concentration in diet and blood were essentially constant over a range of doses. Hence it can be calculated that the heavy fish consumers studied by Humphrey had blood concentrations

MONS 012889

equal to those expected in rats exposed to a dietary concentration of 100 X 0.058 ÷ 0.40 = 14 ppm. In other words, humans retain PCBs in their blood 160 times (14 ÷ 0.087) more efficiently than rats. If it is assumed that humans and rats are equally at risk when their blood concentrations are the same, then the average excess risk of the heavy fish consumers would be 0.022 (1 in 50)for hepatocellular carcinoma.

A final assumption that could be made is that humans and rats are equally at risk when the concentration of PCBs in their livers are the same. (This is probably the most plausible assumption of all, because the liver is the most sensitive organ in the rats.) Surveys in Japan have suggested that the average ratio between PCB concentration in body fat and liver is about 15 (ref. 1). Hence the estimated PCB concentration of 4 ppm in the fat of theheavy fish consumers (Appendix A, p. 21) would correspond to an average concentration of about 0.3 ppm in their livers. Although this estimate is obtained indirectly, it is likely to be a reasonable estimate for the purposes of an approximate calculation of risks.

When rats were fed a diet containing Aroclor 1254 at a concentration of 2 ppm, the average concentration of PCBs in their livers became constant after 6 months at about 1.1 ppm (ref. 8, Table 3). This study indicated that the liver concentration was roughly proportional to the dietary concentration. Hence it can be calculated that the heavy fish consumers studied by Humphrey had liver concentrations of PCBs equal to those expected in rats exposed to a dietary concentration of about 2 X 0.3 ÷ 1.1 = 0.55 ppm. If it is assumed that humans and rats are equally at risk when their liver concentrations are the same, then the average excess risk of the heavy fish consumers would be 8.2×10^{-4} (1 in 1,200) for hepatocellular carcinoma.

MONS 012890

These five estimates of the lifetime risks of cancer likely to be suffered by lifetime consumers of Lake Michigan fish range from 1 in 50 to 1 in 20,000. In my judgment, the most reasonable estimates are those derived from comparisons of concentrations of PCBs in blood and liver (1 in 50 and 1 in 1,200, respectively), since these involve comparisons of the degree of exposure of target tissues, rather than comparisons of the quantities ingested into the gut. Hence I conclude that the most likely range of risks accruing to heavy consumers of Lake Michigan fish is between 1 in 100 and 1 in 1000. These estimates fall into the range where they are not strongly dependent on the mathematical model used to extrapolate from high doses to low doses (Appendix A, p. 76). Hence similar estimates could be obtained from other models in these circumstances of relatively high exposure.

However, there are two ways in which these estimates could substantially underestimate risks. First, they are based on data on hepatocellular carcinomas alone. In the Kimbrough experiment, there was a much larger incidence of neoplastic nodules, so that estimates of risk based upon these tumors would be about 10 times higher than those based upon hepatocellular carcinomas alone. Since neoplastic nodules appear to be early stages in the development of liver cancer in rats, they are sometimes used as the basis for estimating human risks. This procedure is not followed in this report, but it should be recognized that total risks may thereby be underestimated by a factor of 10.

Second, the experimental data were based on studies of rats that were exposed to PCBs only after weaning. Hence estimates of risk derived therefrom are not applicable directly to breast-fed human infants, who are exposed more intensely to PCBs via breast milk than at any time later in

life. Crump and Masterman attempted to calculate the likely magnitude of the risks accruing to breast-fed infants whose mothers consume fish from Lake Michigan. They estimated that the risk to such infants would lie between 1.3 and 4.2 times that for individuals exposed after weaning only, depending on the assumptions made about the mechanism of action and duration of exposure (Appendix A, Table 15).

For the above reasons, it is my scientific judgment that the range of risks specified above (between 1/100 & 1/1000) is more likely to be an underestimate than an overestimate of the total risks accruing to the population of heavy consumers of Lake Michigan fish. Assuming that this population includes 150,000 people (see section II), the total number of excess cancers likely to result in a lifetime would be in the range 150-1,500, or more. If the larger population of 1,000,000 persons discussed in Section II is considered, the risk to the average individual would be lower by a factor of about 7, but the total population would be larger by the same factor. Hence the overall effect on the population would be the same, but it would be spread over alarger number of people.

In either case, the average number of excess cancers expected in the entire exposed population would be of the order of 2 - 20 per year. The smaller figure could not be detected by any reasonable type of epidemiological study. The larger figure could be detected if the cancers were of a distinctive type, but the effects are unlikely to be manifested for another 20 years or more. Hence, in the present state of scientific knowledge, calculations of the type presented in this report are the only way to estimate the likely magnitude of the health risks.

V. OTHER TYPES OF HEALTH RISK POSED BY PCBs

In addition to increasing the risk of cancer, PCBs are known to have a number of other adverse effects on health in humans and experimental animals (refs. 1 - 3). One effect of particular concern is disruption of reproduction. Female rhesus monkeys exposed to PCBs at a concentration of 2.5 ppm in the diet for 6 months displayed an increased frequency of infertility and spontaneous abortions. Within two months after birth, the infant monkeys that were born displayed signs of PCB poisoning; three of six infants died within 6 months, and the remainder showed learning impairments (refs. 1 -3). These observations are particularly significant because the monkeys were exposed to Aroclor 1248, the PCB mixture most similar to that found in fish in Lake Michigan. The dose level that caused these severe effects on rhesus monkeys was within a factor of 30 of the average dose ingested by heavy consumers of Lake Michigan fish when compared on the basis of dietary concentration, and within a factor of 50 when compared on the basis of dose per unit of body surface area per day. Furthermore, the concentration of PCBs in the milk of the nursing rhesus monkeys was very close to that measured and calculated to occur in the milk of highly exposed human mothers (Appendix A, pp. 18-21, 39-41). Thus, at least for the more highly exposed consumers of Lake Michigan fish, there is no "margin of safety" at all. Although there are no established techniques for calculating the exact magnitude of reproductive risks due to ingestation of toxic chemicals, these individuals and their children are clearly at high risk.

Several other adverse effects of PCBs on mammals have been reported at dose levels equivalent to dietary concentrations between 0.64 and 5 ppm.

These effects include reproductive failure and death in mink, induction of stomach nodules in dogs, and induction of liver enzymes in rats (ref.2, p. 356). The "margins of safety" for these effects for heavy consumers of Lake Michigan fish are in the range 7.5 - 60 (when compared on a dietary concentration basis). Again, at least the higher consumers of fish are likely to be at high risk.

VI. POSSIBLE ADDITIONAL RISKS TO CONSUMERS OF FISH CAUGHT NEAR WAUKEGAN, ILLINOIS

Special concern has been expressed about possible additional health risks that may be posed to consumers of fish caught near Waukegan, Illinois. This concern has arisen because of high concentrations of PCBs in fish caught in Waukegan harbor (ref. 9). Unfortunately, the sampling of fish in the Waukegan area has not been sufficiently extensive to permit firm estimates of the likely magnitude of exposure of consumers to PCBs.

A survey of fishermen in the Waukegan area (ref. 10) suggests that about 16,000 fish are caught annually by fishermen based at Waukegan (i.e, fishing in the harbor or from boats launched there). Of these fish, about 58% are Coho Salmon, 11% are Chinook Salmon, 13% are various species of trout, and 17% are Yellow Perch. The catch of Coho Salmon is about 1.6% of the total annual catch in Lake Michigan, but as a whole the area accounts for slightly less than 1 percent of the total annual catch of fish in the lake (ref. 4, Table 2). Unfortunately, there appear to be no direct measurements of PCB levels in trout, salmon, or yellow perch from the immediate area of Waukegan. PCB concentrations in fish of other species collected in Waukegan harbor were in the range 1.7 - 39 ppm, with an overall average of 19 ppm (ref. 9). Hence consumers of fish caught inside the harbor are likely to be

MONS 012894

exposed to PCBs at levels roughly twice as high as those for Lake Michigan as a whole (ref. 4, pp 4 - 5). It is not known whether this would apply to consumers of fish caught outside the harbor.

These incomplete data suggest that at least some consumers of fish caught in the Waukegan area are likely to be exposed to larger quantities of PCBs than consumers of fish from other parts of Lake Michigan. On the basis of the total catch, the number of consumers involved may be in the range 13,000 (heavy consumption) - 80,000 (average consumption).

VII. CONCLUSIONS

1. About 14 million pounds of sports fish are caught annually in Lake Michigan. The number of people who eat significant quantities of these fish probably lies between 150,000 (heavy consumers) and 1 million (general consumers).

2. Direct measurements have shown that heavy consumers of fish from Like Michigan ingest between 14 and 114 mg of PCBs annually (mean 46 mg). This exposure is reflected by high concentrations of PCBs in their blood.

3. PCBs are carcinogenic in experimental animals and are very likely to pose excess cancer risks in humans exposed to them.

4. Several calculations are presented of the likely magnitude of the cancer risks posed by PCBs to heavy consumers of Lake Michigan fish. The calculations vary over a wide range, primarily because of uncertainty about the way in which risks should be extrapolated from data on experimental animals to predict risks to humans. The most likely range of risks to a typical heavy consumer is between 1 in 100 and 1 in 1000 (excess risk

of developing cancer after lifetime exposure).

5. PCBs also cause a number of other adverse effects in mammals exposed to them. The most serious of these effects is disruption of reproduction. For heavy consumers of Lake Michigan fish, there is little or no "margin of safety" for these effects. Although there are no established techniques for calculating how many persons may be affected, heavy consumers of Lake Michigan fish are at high risk.

6. There is insufficient information to estimate risks to individuals who eat fish caught near a local source of PCB contamination at Waukegan, Illinois.

REFERENCES

1. CRUMP, K.S., and MASTERMAN, M.D. 1979. Assessment of carcinogenic risks from PCBs in food. Unpublished report to U.S. Congress Office of Technology Assessment, April 1979.

2. NISBET, I.C.T. 1976. Criteria Document: PCBs. Unpublished report to U.S. Environmental Protection Agency, April 1976.

3. NATIONAL RESEARCH COUNCIL. 1979. Polychlorinated Biphenyls. Washington, D.C., National Academy of Sciences.

4. HUMPHREY, H.E.B. 1977. Evaluation of changes of the level of polychlorinated biphenyls (PCB) in human tissue. Final report from Michigan Dept. of Public Health to U.S. Food and Drug Administration.

5. NATIONAL RESEARCH COUNCIL. 1977. Drinking Water and Health. Washington, D.C., National Academy of Sciences.

6. KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J., and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polyclorinated biphenyl, Aroclor 1260. J. Nat. Cancer Inst. 55: 1453 - 1459.

7. NATIONAL CANCER INSTITUTE. 1978. Bioassay of Aroclor 1254 for possible carcinogenicity. NCI Technical Report Series Carcinogenesis No. 38.

8. GRANT, D.L., MOODIE, C.A., and Phillips, W.E.J. 1974. Toxicodynamics of Aroclor 1254 in the male rat. Environ. Physiol. Biochem. 4: 214 - 225.

9. U.S. ENVIRONMENTAL PROTECTION AGENCY. 1979. The analysis for polychlorinated biphenyls in fish collected in Waukegan harbor (1978, 1979). Unpublished reports, U.S. Environmental Protection Agency, Chicago, Illinois.

10. ANON. (1980) Illinois creel census survey, May 15 - November 15, 1979. Unpublished MS.