

REPORT ON 1984-86 FEDERAL SURVEY OF PCBs IN ATLANTIC COAST BLUEFISH

INTERPRETIVE REPORT

COORDINATED BY

*The
National Oceanic and Atmospher
Administration*

in cooperation with

*The
Food and Drug
Administration*

and

*The
Environmental Protection
Agency*

**MARCH
1987**



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INTRODUCTION

This interpretive report is based on the results of a survey to determine levels of polychlorinated biphenyls (PCBs) in the edible tissues of Atlantic Coast bluefish samples collected from January to November, 1985. A full description of the conduct of the survey, including the basic data obtained during the course of the work and a set of preliminary statistical observations, may be found in: "Report on 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish - Data Report" (NOAA/FDA/EPA, 1986), hereafter called the Data Report. The Data Report also contains background information on the sequence of events that led to the eventual request by Congress in June, 1984, to definitively address growing public concerns that there may be a coast-wide, rather than a localized, problem with PCB-contaminated bluefish. The Congressional action resulted from both public health and socio-economic issues that had developed after the 1976 discovery that certain species of fish, including bluefish, taken from New Jersey and New York waters were contaminated with PCBs. The wide ranging migratory nature of bluefish, which are found along the entire Atlantic Coast of the United States, led to speculation that this highly prized and abundant species could be of special concern from a public health perspective. The National Oceanic and Atmospheric Administration (NOAA), through one of its line agencies, the National Marine Fisheries Service (NMFS), was subsequently funded by Congress to coordinate the survey in cooperation with the Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA) (Figure 1).

The four major objectives of the study, as outlined by Congress, were to:

1. Determine contaminant levels in detail sufficient to support an assessment of public risk.
2. Determine whether the problem is coast-wide, or likely to affect only specific areas.
3. Develop uniform sampling and analysis procedures to allow for the easiest comparison and interpretation of contamination data.
4. Furnish information to state officials to assist them in determining if there is a need for regulation of sport or commercial fishing, and to provide guidelines to ensure that any regulation is rational and equitable.

The results of the survey were presented in the Data Report in reference to the FDA tolerance level of 2 parts per million (ppm) PCBs in fish. This interpretive report is intended to meet the Congressional charge to provide an assessment of any potential health risk for consumers of bluefish and to provide advice and guidance on the issue to State and local authorities.

SURVEY DESCRIPTION

A detailed description of the materials and methods used in the conduct of the survey is provided in the Data Report and is summarized in Appendix I of this report. The survey design called for collection of site-specific, size-stratified samples of bluefish, primarily through dockside purchases, but also through special field collection (e.g., party- or charter-boat, beach seining, hook and line, and research cruises). Historically, 80 to 90% of the bluefish fishery is recreational. It was assumed, however, that commercial and recreational landings come from the same population of fish. This assumption was supported by tagging studies, comparison of length and weight landings statistics, and observations of the proximity of the two fisheries. Advantages of using commercial landings for the survey collections were the stability and availability of the fishery, as well as the fact that commercial landings enter the marketplace and reach a range of seafood consumers.

Twelve seasonal collections (sites) of bluefish were planned in five geographic areas, i.e., New England (MA, RI), New York Bight (CT, NY, NJ), Maryland-Virginia, North Carolina, and Florida (Atlantic Coast) (Figure 2). The rationale used for site selection and timing of sampling was based on a number of considerations that included migratory behavior, distribution and abundance on a seasonal basis, and patterns of commercial and recreational catch and effort. All twelve sites were to be sampled for five-fish composites and five sites in three of the geographic areas (New England, New York Bight, and North Carolina) were to be sampled also for individual fish. Only eleven sites were actually sampled for five-fish composites in conformance with the survey design, however, because bluefish were not available from Florida.

Fish were collected in three size (fork length) categories that represented approximately equal intervals on a logarithmic scale as applied to existing population length-frequency estimates. The length categories to which all fish (composites and individuals) were allocated were: less than or equal to 300 mm (11.8 inches), from 301 to 500 mm (11.8 to 19.7 inches), and greater than 500 mm (19.7 inches). These categories corresponded approximately to whole weight categories of less than 1 lb, 1 to 4 lbs, and greater than 4 lbs, respectively.

The basic survey structure was based on a strategy of posing testable hypotheses relevant to public health concerns about PCB-contaminated bluefish and design a sampling plan that would enable these hypotheses to be tested with specified levels of statistical risk. Under hypothesis testing, the questions posed by Congress may be rephrased as follows: Do PCBs in bluefish exceed the established FDA tolerance of 2 ppm and if so, does this condition exist coast-wide or affect only specific geographic areas? Because bluefish is the principal recreational species in terms of landings (130 to 155 million pounds annually) taken along the Atlantic Coast, quantitative determinations are important from a socio-economic as well as a public health perspective.

A balance was struck among sampling precision, statistical significance, and cost of sample/data collections and analyses. This balance was achieved by estimating and selecting the sample sizes (Tables 1 and 2 of Appendix I) needed to test the hypothesis that the PCB mean per site/fork length category is less than or equal to 2 ppm, with no more than a 5% chance of rejecting this hypothesis when it is true, and at most a 5% chance of failing to reject this hypothesis when the true PCB mean is at least 3 ppm.

In addition to the collection and analysis of samples of bluefish, a literature review was conducted pertinent to the uptake and metabolism of PCBs by bluefish, their prey, and similar fishes. The complete text of the literature review, containing information on the toxicology, kinetics, and metabolism of PCBs in aquatic organisms, with special reference to bluefish, is contained in Appendix II.

STATISTICAL ANALYSIS OF SURVEY DATA

Methodology

The survey data were analyzed by using various statistical procedures to fulfill objectives with respect to several defined hypotheses. The validity of these analyses on the measured PCB values is supported by the distributional form robustness properties of the selected statistical methods and further enhanced by composite mean PCB data and relatively large samples sizes.

To compare sample mean PCB values for the various levels of the survey design factors specified in the hypotheses, an analysis of variance (ANOVA) technique and Duncan's multiple range comparison procedure were used. In addition, the ANOVA was used to partition the total PCB variation among composites into estimates of standard deviations of PCB values and standard errors of mean PCB levels. These variability measures were used to estimate with 95% confidence the maximum percentage of composites that exceeded the FDA tolerance of 2 ppm PCBs, and to provide upper 95% confidence limits on the population mean PCB levels for the survey design factors (sampling sites, fork length classes (strata), etc.).

Other statistical methodologies and practices used were:

1. The Student/Fisher t statistic was used to test hypotheses concerning mean PCB levels exceeding 2 ppm for each site/fork length class.
2. Normal approximations of binomial processes were used to compare site percentages of composite samples exceeding 2 ppm PCBs.
3. Bartlett's test for homogeneity of variances was used to evaluate variability in PCBs among sites and fork length classes.
4. The Smith/Satterthwaite/Wilcoxon procedure was used to test mean PCB comparisons in the presence of heteroscedasticity.
5. Correlation analysis was used on the individual fish PCB data to determine the strength of a linear relationship between PCBs/lipid content and PCBs/fork length.
6. Each hypothesis was tested at the 5% level of significance.

Data from the five-fish composite samples were analyzed separately from those data on individual fish samples. Direct comparisons of composite PCB values to those for individual fish samples were judged inappropriate because of: (1) the averaging effect inherent in compositing; (2) no basis for relating composite and individual PCBs since these results were obtained from different fish samples; and (3) composite samples were randomly selected at all sites while individual samples were collected at but five sites. However, at one site 95 fish collected for individual analysis were also used to randomly form 19 five-fish composite samples and their corresponding PCB values were compared for mean equivalence.

Survey Hypotheses

The results of the statistical analyses of PCBs in composite and individual fish samples that address the fifteen hypotheses outlined in the survey design can be considered to form four separate groups:

1. Group 1 - Hypotheses on PCBs and PCB means

Do PCBs in bluefish exceed 2 ppm?

Does the PCB mean per site exceed 2 ppm?

Does the PCB mean per fork length class exceed 2 ppm?

Does the PCB mean coast-wide exceed 2 ppm?

Do the PCB means differ among sites?

Do the PCB means differ among fork length classes within sites?

Do the PCB means differ among sites within a specified fork length class?

2. Group 2- Hypotheses on PCB variability

Does the PCB variability differ among sites?

Does the PCB variability differ among fork length classes?

3. Group 3 - Hypotheses on PCB correlations

Do PCB levels correlate with lipid content?

Do PCB levels correlate with fork length?

4. Group 4 - Hypotheses on PCB means for sex and spawning period

Does the PCB mean for either sex exceed 2 ppm?

Do the PCB means differ between sexes?

Do the PCB means for pre/post spawning periods exceed 2 ppm?

Do the PCB means differ between pre/post spawning periods?

Results

1. Group 1 - Hypotheses on PCBs and PCB means

These hypotheses were tested by using the analytical results obtained from the composite samples. Tables 1-3 present, respectively, for each fork length class (i.e., ≤ 300 mm = small, 301-500 mm = medium, and > 500 mm = large), site collection data and geographic areas, sample size, mean PCB level, standard deviation, the number and percent of samples that exceeded 2 ppm PCBs, the maximum percent of population composites that exceeded 2 ppm PCBs with 95% confidence, and the upper 95% confidence limit on the population mean PCB level.

a. Do PCBs in bluefish exceed 2 ppm?

For small and medium bluefish, no samples exceeded 2 ppm PCBs at any of the sampling sites, and no statistically significant differences among the site percentages in these classes were noted. However, in the large fork length class, temporal/spatial statistically significant differences in percentages of samples with PCBs exceeding 2 ppm were realized. These differences formed two statistically significant groupings with respect to sampling sites:

- (1) The percentage of samples that exceeded 2 ppm PCBs for ~~New York Bight - Oct/Nov~~ (45.31%) was significantly greater than those for other sampling sites.
- (2) The percentage of samples that exceeded 2 ppm PCBs for North Carolina - March (16.92%), North Carolina - April (23.08%), and New England - August (27.69%) were each significantly greater than those for New England - October (3.08%), New York Bight - May/June (4.62%),

New England - June (4.62%), and New York Bight - August (4.62%). However, no significant differences resulted within these two site groups.

For each fork length class and sampling site, the estimated maximum percentage of population samples that exceeded 2 ppm PCBs was determined using 95% confidence with the following results:

- (1) For small bluefish, the maximum percentage of population samples that exceeded 2 ppm PCBs was virtually zero.
- (2) Although there were no medium bluefish samples that exceeded 2 ppm PCBs, there is 95% confidence that for North Carolina - Jan/Feb and New England - October, at most 9.38% and 18.89% of the population samples exceeded 2 ppm PCBs respectively. For the remaining sites, essentially zero percent of the population samples exceeded 2 ppm PCBs.
- (3) For large bluefish, each sampling site is expected to have had population samples exceeding 2 ppm PCBs. With 95% confidence, the percentage of population samples that exceeded 2 ppm PCBs ranged from a minimum of at most 3.67% in New England - October to a maximum of at most 71.63% in New England - August.

b. Do the PCB means exceed 2 ppm?

The sample PCB means (Table 1-3), categorized by specific survey design factors (sites, fork length classes, and coast-wide), were compared to the FDA tolerance level of 2 ppm for bluefish. The 95% upper confidence limits for small and medium bluefish suggest that the population means for these classes in all sites and coast-wide did not exceed the tolerance level. However, for large bluefish the data indicate the following:

- (1) The sample PCB means for North Carolina - Jan/Feb (2.21 ppm) and New England - August (4.80 ppm) exceeded the tolerance level. For the North Carolina site, however, the mean PCB level was greatly influenced by one five-fish composite PCB value (45.42 ppm). With this value omitted the sample mean PCB level was 1.53 ppm.
- (2) The upper 95% limits on mean PCB levels indicate with 95% confidence that the population PCB means for New York Bight - Oct/Nov, North Carolina - Jan/Feb, and New England - August were at most 2.19, 3.35, and 6.41 ppm, respectively. The upper 95% confidence limits for the remaining sites were all less than the tolerance level. Coast-wide, the population mean PCB level was at most 2.49 ppm with 95% confidence.

c. Do the PCB means differ?

The sample PCB means (Tables 1-3) were compared for each fork length class to assess possible differences due to influences relative to each sampling site with the following results:

- (1) For small bluefish, the sample PCB means did not statistically differ among sites.
- (2) For medium bluefish, the sample PCB means formed two statistically significant site groupings:
 - (a) The sample mean for New England - October (0.94 ppm) significantly exceeded the sample mean for North Carolina - Jan/Feb (0.71 ppm)
 - (b) The sample means for the two sites identified in (a) each significantly exceeded the sample mean for each remaining site.

- (3) For large bluefish, the sample mean for New England - August (4.80 ppm) significantly exceeded the sample mean for each of the other sites. No additional comparisons among sample PCB means were statistically significant.

2. Group 2 - Hypotheses on PCB variability

The survey data were used to assess possible differences in PCB variation among fork length classes and sampling sites. Tables 4 and 5 present for composite and individual fish samples, respectively, sample size, standard deviation, and coefficient of variation (CV) by collection date, geographic areas, and fork length class. The survey data indicated the following:

- a. For small bluefish and for sites expressing variability, the sample variation was statistically homogeneous among sites for both the composite and individual samples. The CV for composite samples ranged from 49.0% to 55.3%. For individual fish samples, the CV ranged from 61.6% to 92.3%.
- b. For medium bluefish and for sites expressing variability:
- (1) The sample variation for the composite samples was statistically homogeneous among sites. The CV ranged from 35.7% to 65.5%
 - (2) For individual fish samples, the sample variation for New York Bight - Oct/Nov was statistically significantly less than that in each of the other sites. The CV ranged from 35.0% to 87.9%.

c. For large bluefish:

- (1) The sample variation for North Carolina - Jan/Feb and New England - August were each significantly greater than the PCB variation for the other sites for composite samples. The CV ranged from 26.3% to 250.2%. For the North Carolina site, however, the sample variation was greatly influenced by one five-fish composite PCB value (45.42 ppm). With this value omitted the standard deviation (0.94 ppm) and the CV (61.6%) were in statistical agreement with all sites except New England - August.
- (2) For individual fish samples, the sample variation for New York Bight - May/June was significantly greater than the other sites. The CV ranged from 45.5% to 225.0%. However, for this site the sample variation was greatly influenced by one PCB sample (23.02 ppm). With this value omitted the standard deviation (0.78 ppm) and the CV (81.9%) were in statistical agreement with all other sites.

3. Group 3 - Hypotheses on PCB correlations

Another objective of the survey was to determine if PCBs correlated with body fat (lipid content) and fork length. Since composite samples provide mean values for PCBs, lipid content, and fork length, only the individual fish samples were used to perform these analyses. Table 6 presents, for the five sites and fork length classes from which individual samples were collected, corresponding samples sizes and correlation coefficients (Pearson r values) for PCBs/lipid content and PCBs/fork length. Statistically significant correlations (5% level) are denoted by an asterisk (*).

Because of relatively large sample sizes, there were several statistically significant correlations between PCBs/lipid content and PCBs/fork length for fork length classes within sites, individual sites and all sites combined. Most of these significant correlations are, however, of questionable practical importance since the amount of linearly related variability accounted for is rather negligible. For example, for PCBs/lipid content and all sites combined, the statistically significant correlation is 0.43. By using the squared correlation coefficient (i.e., the Coefficient of Determination) to evaluate the strength of the relationship, only $(0.43)^2$ or 18.5% of the variability of PCBs is linearly related to the variability in lipid content values. One should note that for a correlation less than or equal to 0.70, less than 50% of the variability in the two variables is linearly related. The survey data failed to sufficiently support useful correlations between PCBs/lipid content and PCBs/fork length.

4. Group 4 - Hypotheses on PCB means for sex and pre/post spawning period

The individual bluefish samples were analyzed for PCB mean comparisons between sexes and between pre/post spawning periods. Table 7 presents, for sex and for each sampling site and fork length class, sample size, mean PCB level, standard deviation, and for all sites combined, the upper 95% confidence limit on the population mean PCB level for each sex/fork length class. Table 8 presents the same information for pre/post spawning periods.

For male or female bluefish, no sample mean PCB level exceeded 2 ppm for fork length classes within sites, for all sites individually, or for all sites combined. There is 95% confidence that the population mean PCB levels for males and females were at most 0.18 and 0.47 ppm for small bluefish, 0.51 and 0.51 ppm for medium bluefish, and 1.52 and 1.73 ppm for large bluefish. To determine if

the sex of bluefish had an effect on PCBs, the PCB means were compared for each sampling site and fork length class. No statistically significant sex comparisons were realized.

For pre/post spawning periods, no sample PCB mean level exceeded 2 ppm for fork length classes within sites, for sites, or for all sites combined. There is 95% confidence for small females in pre-spawning that the population PCB mean level was at most 0.39 ppm. For small females in post-spawning, no upper confidence is available since only one fish was analyzed. There is 95% confidence that the population PCB mean levels for pre/post-spawning was at most 0.59 and 0.58 ppm for medium females and 1.35 and 1.52 ppm for large females. To determine possible spawning period influences on PCBs, the pre/post-spawning period sample PCB means were compared for each sampling site and fork length class. No statistically significant comparisons were realized.

5. Comparison of PCBs obtained from composite versus individual fish samples

A special analysis was performed to determine the efficacy of the economically efficient compositing procedure customarily used to provide an analytical sample for analysis of PCBs in bluefish. At one site, New York Bight - May/June, ninety-five fish collected for individual analysis were also used to randomly form 19 five-fish composite samples and their corresponding PCB values were compared for mean equivalence. Table 9 presents, for composite and individual fish samples and by fork length class, sample sizes, mean PCB levels, standard deviations, and the minimum and maximum PCB values. Within each fork length class, no statistically significant difference was obtained between sample mean PCB levels for composite and individual fish samples.

HUMAN TOXICOLOGY OF PCBs

Polychlorinated biphenyls are a complex mixture of different chlorobiphenyls and their isomers. Isomers of an individual PCB have the same number of chlorine atoms on the biphenyl molecule but the atoms are substituted at different locations. The PCBs that were manufactured by Monsanto in the U.S. are identified by the trade name "Aroclor," and the particular kind of Aroclor is identified by a four-digit number, e.g., Aroclor 1254 or Aroclor 1260. The first two digits refer to the 12 carbon atoms that make up the biphenyl molecule and the second two digits refer to the percentage, by weight, of the chlorine content in the mixture. Under this numbering system, Aroclor 1254 contains 12 carbon atoms and 54% chlorine, while Aroclor 1260 contains 12 carbon atoms and 60% chlorine. The only exception is Aroclor 1016 which also contains 12 carbons and 41% chlorine.

The toxicity of PCBs apparently depends upon the degree of chlorination and the isomeric form. The lower chlorinated biphenyls tend to be more acutely toxic than the higher chlorinated biphenyls. The different patterns of toxicity are associated in part with the longer biological half-life and less susceptibility to metabolic alteration or degradation by the liver and other organs of the higher chlorinated biphenyls. (Matthews and Anderson, 1975a). However, PCB residues in animal and human tissues resulting from environmental exposure resemble PCB mixtures with more than 50% chlorination. This suggests that PCBs with five or fewer chlorine atoms (less than 50% chlorination) are more readily metabolized than the PCBs with higher chlorination (Matthews and Dedrick, 1984). Evaluation of the toxicity of specific PCBs is complicated

because commercial preparations of PCBs may contain impurities such as chlorinated dibenzofurans (Roach and Pomerantz, 1974). Certain isomers of the chlorinated dibenzofurans are appreciably more toxic than are similar amounts of chlorinated biphenyls (Moore et al., 1975).

Human Exposure Data

1. Yusho Incident

When assessing the potential effects of environmental contaminants such as PCBs on humans, it is preferable, though usually not possible, to rely on human rather than animal data. The Yusho incident in Japan provided information about human responses to PCBs. In 1968, Kanechlor 400, a PCB manufactured in Japan, leaked from a heat exchanger into rice oil that was consumed by Japanese families. The toxic manifestation was designated as Yusho disease. Subsequent analysis revealed the presence of PCBs in the blood and tissues of patients who exhibited symptoms of intoxication. Typical clinical findings included chloracne, increased pigmentation of the skin, increased eye discharge, transient visual disturbances, feeling of weakness, numbness in limbs, headaches, and disturbances in liver function. Most of the babies born to mothers with Yusho disease had skin discoloration that slowly regressed as the children grew. Adult patients had protracted clinical disease with a slow regression of symptoms and signs, suggesting a slow metabolism and/or excretion of the PCBs (Kuratsune and Shapiro, 1984).

A review by Kuratsune et al. (1975), reported that the Yusho rice oil was also contaminated with approximately 5 ppm polychlorinated dibenzofurans (PCDFs). The PCDFs produce toxicity which is similar to that elicited by PCBs, but they are orders of magnitude more toxic than PCBs. Kuratsune et al. (1975)

also presented data of Nagayama et al. (1975) showing that PCDFs were present in the liver and adipose tissue of Yusho patients but not in a control group. Nagayama et al. (1975) reported that the ratios of PCBs to PCDFs in Kanechlor 400, in Yusho rice oil and in adipose tissue and liver tissue from a Yusho patient were 50,000 to 1, 200 to 1, 144 to 1, and 4 to 1, respectively. Thus, PCDFs were apparently concentrated more efficiently in liver tissue than were PCBs (PCB/PCDF ratio 4 to 1). If PCDFs were 200 to 500 times more toxic than PCBs, then the contaminated rice oil would be 2 to 3.5 times more toxic than expected from the PCB content alone.

The victims in the Yusho incident were calculated to have consumed an average of 15,000 milligrams (mg) per day of the rice oil contaminated with Kanechlor 400. The rice oil, consumption of which was associated with severe clinical symptoms, was determined to be contaminated with 2,000 to 3,000 ppm PCBs; the average PCB concentration in the oil was 2,500 ppm. By using the average consumption of rice oil and average PCB concentration, the average daily intake of PCBs was estimated to be 37.5 mg/day. The average cumulative dose of PCBs that caused an overt effect in the Japanese victims was reported to be 2,000 mg, but the lowest dose leading to overt symptoms was 500 mg. Based on the average daily intake of 37.5 mg/day, it would take approximately 53 days of exposure for an individual to consume the average cumulative dose of 2,000 mg. Actual periods of exposure undoubtedly varied around this figure. It was estimated that the maximum exposure at Yusho was 100 days. Additional work has indicated that the rice oil contained approximately 1,000 ppm of chlorinated quatrphenyls in addition to PCBs and that the concentration of PCBs in the oil was actually about one half of that originally determined (Kuratsune and Shapiro, 1984).

The rice oil involved in the Yusho incident was contaminated with Kanechlor 400, a Japanese brand of PCBs that contains 48% chlorine (by weight). The 48% chlorine-containing Aroclor 1248, an American product, is very similar to Kanechlor 400. Consequently, the human data derived from the Yusho incident are relevant and significant in assessing health effects resulting from exposure to American-made PCBs.

2. Yu-Cheng Disease

An outbreak of poisoning with acneform eruptions and pigmentation occurred in Taiwan in February 1979. More than 2,000 persons were affected. The source of poisoning was a rice bran oil that was accidentally contaminated with PCBs (Lu and Wu, 1985). PCBs were detected in the oil at concentrations of 4.8 to 205 ppm ($\bar{x} = 52 \pm 39$ ppm). PCB concentrations in blood of the poisoned patients were 0.003 to 1.156 ppm ($\bar{x} = 0.089 \pm 0.007$ ppm). In addition, PCDFs were detected in the rice bran oil and the blood of patients (Chen et al., 1984). In general, the age distribution of patients, the symptoms, the skin pathology, and the way the poisoning occurred were similar to those of the Yusho incident. The disease has been termed Yu-Cheng, which means oil disease.

3. Michigan-Fish Consumption Study

The Michigan Department of Public Health has reported the results of a study of the consequences of human exposure to PCBs from the consumption of freshwater sportsfish caught in different areas of Lake Michigan (Humphrey et al., 1976, Humphrey, 1983). The study included exposed and control subjects from five areas that bordered Lake Michigan. Exposed subjects were individuals who consumed at least 24 to 26 lbs (10.9 to 11.8 kg) of fish per year. Control

subjects were individuals who consumed less than 6 lbs (2.7 kg) of fish per year.

The most frequently recorded quantity of fish consumed by the participants was in the 26 to 27 lbs (11.8 to 12.3 kg) per year range. The highest recorded fish consumption over the two-year period of the study was 180 lbs (81.7 kg) per year and the highest single-season consumption was 260 lbs (118 kg). Mean PCB concentrations in whole lake trout were 19 ppm in 1973 and 23 ppm in 1974. Mean PCB concentrations in Coho salmon were 12 ppm in 1973 and 10 ppm in 1974. The calculated quantity of PCBs ingested by eating Lake Michigan fish averaged 46.5 mg/year and ranged from 14.2 mg/year to 114.3 mg/year. PCB ingestion for each individual was determined by proportioning the reported annual fish consumption by frequency of species eaten and PCB concentrations in cooked fish. The community mean of PCBs in cooked fish was used in instances where cooked fish consumption varied from year to year; the mean annual consumption for each individual during the two-year study was used in each case.

PCBs were found in blood from all of the 182 Michigan study participants, including controls. Concentrations ranged from a low of 0.007 ppm in controls to a high of 0.37 ppm in the exposed group. Although there was a wide range of values for the quantity of fish consumed, there was a statistically significant ($p < 0.001$) positive correlation ($r = 0.51$) between the reported quantity of Lake Michigan fish consumed and the blood PCB concentration. Annual variation in PCBs in blood could not be demonstrated, and the mean PCBs in blood of the control and exposed groups did not appear to change markedly from 1973 to 1974. In addition, abstinence from consumption of Lake Michigan fish for 90 days or more did not significantly change the concentrations of PCBs in blood. PCBs in blood during the abstinence showed variation but no steady decline in PCBs; in

fact, more subjects showed no change or a rise than showed a decline of PCBs in blood over time. These results tend to confirm the expectation based on animal data that higher chlorinated biphenyls are not rapidly depleted from human tissues.

The calculated mean daily dose received by the exposed group was 1.75 micrograms per kilogram body weight per day ($\mu\text{g}/\text{kg}/\text{day}$); doses ranged from 0.09 to 3.9 $\mu\text{g}/\text{kg}/\text{day}$. No symptoms or adverse health effects clearly related to PCB exposure could be identified in the exposed group. This implies that exposure to PCBs from eating quantities of fish at the degree of contamination reported did not cause any observable adverse health effects similar to those observed in the Yusho incident. However, it is possible that effects too subtle for detection are occurring and that long-term adverse health effects may result.

Comparison of PCBs in raw and cooked fish showed that human exposure to PCBs from fish consumption was less than might be expected from the raw fish data because preparation (trimming away fatty tissue) and cooking decreased the amount of PCBs in fish consumed. For example, PCBs in cooked lake trout consumed by the study participants ranged from 1 to 4 ppm; from 0.48 to 5.4 ppm in cooked salmon; and from 0.36 to 2.1 ppm in other cooked fish. These levels were decidedly lower than the PCB concentrations reported in raw whole fish. In contrast to these findings, a cooking study conducted by FDA during the 1984-86 Federal Survey on PCBs in Atlantic Coast Bluefish (NOAA/NMFS, 1986) showed that approximately 26% of the PCBs was removed by cooking (FDA, 1987). The PCB concentration in the cooked fish was essentially unchanged (2.5 to 2.7 ppm) due to the accompanying moisture loss and the consequent decrease in the weight of the cooked fillet. There are enough differences in the design of these two

studies that their results cannot be directly compared. However, it is reasonable to conclude that consumers of cooked fish would receive less PCBs than would be predicted from data on the fish prior to cooking.

4. PCBs in human breast milk

A report describing PCB residues in human breast milk by Wickizer et al. (1981) illustrates the potential long-term presence of such residues in humans who have been exposed. The study, carried out in the State of Michigan, showed that all of the 1,075 breast milk samples collected from 68 of Michigan's 83 counties contained PCBs; concentrations ranged from trace amounts to 5 ppm on a lipid basis. The mean concentration was approximately 1.5 ppm; 50% of the samples had PCB concentrations of 1 but less than 2 ppm, 17% had concentrations of 2 but less than 3 ppm, and 6% had concentrations of 3 ppm or more. The public health significance of PCBs in human breast milk and long-term effects on breast-fed infants is unclear at the present time. Although public health officials and pediatricians have become increasingly concerned about PCBs in human breast milk, authorities have been reluctant to recommend changes in current breast-feeding practices given the known benefits of breast feeding.

5. Recent Studies

Subsequent studies on human exposure to PCBs have provided information that reflects the concerns generated by the Yusho, Yu-Cheng and Michigan exposures. These studies have been on individuals exposed to PCBs either in industrial situations or from general environmental exposure, particularly from food. The effects of particular note are those reported in the offspring of women who have been exposed to PCBs (Ando et al., 1986; Bercovici et al., 1983; Fein et al.,

1984; Jacobsen et al., 1985; Rogan et al., 1986; Taylor et al., 1984; Yakushiji et al., 1984a,b).

Animal Data

Cordle et al. (1982) reported that different animal species vary in their susceptibility to PCBs. Evaluation of susceptibility is complicated by a lack of information on the detailed composition of PCB mixtures used in the studies and the relationship between the composition of a PCB mixture and the toxicity of that mixture. PCBs have been shown to have adverse effects on reproduction, biochemical functions in the liver (Goldstein et al., 1974 and Kimbrough, 1974a) and gastric mucosa (Kimbrough, 1974b), and to cause liver tumors (Kimbrough, 1975). In primates, the skin and the Meibomian glands were affected, in addition to the liver and gastric mucosa (Allen et al., 1974). In rabbits, atrophy of the thymus has been reported (Vos and Beems, 1971). The PCB concentrations that caused these effects depended on the test species and the composition of the mixture.

A dietary level of 20 ppm Aroclor 1254 depressed reproduction in rats, while a level of 500 ppm Aroclor 1260 was needed to reduce reproduction in the same strain of rats (Linder et al., 1974). Dietary levels of 5 ppm Aroclor 1254 had a marked effect on reproduction in mink (Ringer et al., 1972), and limited studies have shown that 2.5 ppm Aroclor 1248 affected reproduction in non-human primates (Allen, 1975).

Numerous biochemical systems are also affected by PCBs. Hepatic porphyria has not been reported in the monkey, mink, or human; but Aroclors 1016, 1242, and 1254 have all produced this condition in rats (Goldstein et al., 1974). Female rats were more sensitive than males, and Aroclor 1016 was less active

than either Aroclor 1242 or 1254 (Goldstein et al., 1974). Other reported biochemical changes in the liver included lipid accumulation, induction of microsomal enzymes (including hydroxylase and demethylase enzymes) and an increase in cytochrome P-448 (Alvares et al., 1973). Mitochondrial function was inhibited when rats were fed extremely high doses of PCBs (1,000 ppm) but no inhibition occurred at 100 ppm (Chesney and Allen, 1974). Histological and ultrastructural changes in the liver reflected the observed biochemical changes, and included hepatomegaly with concomitant increase in smooth endoplasmic reticulum, atypical mitochondria, and the formation of "fingerprints" in the hepatic cytoplasm. These changes were more pronounced at higher doses with Aroclor 1254 and 1260. The effects were less pronounced with Aroclor 1242 and 1016 (Kimbrough, 1975).

Some toxicity data relative to the carcinogenic effect of PCBs in mice and rats are available. In one strain of mice (BALB/cj), neoplastic nodules (hepatomas, hyperplastic nodules) developed after dietary exposure to Aroclor 1254 (Kimbrough and Linder, 1974). In a National Cancer Institute (NCI, 1978) study, Fischer 344 rats were fed Aroclor 1254 in the diet at 25, 50 and 100 ppm levels. It was concluded that under the conditions of the bioassay, Aroclor 1254 was not carcinogenic in Fischer 344 rats; however, it was suggested that the high incidences of hepatocellular proliferative lesions of the gastrointestinal tract in the Aroclor-treated animals might be associated with the administration of the compound.

Barsotti et al. (1976) found the reproductive function of female rhesus monkeys to be very sensitive to PCB intoxication. Aroclor 1248 was administered in the diet at 2.5 or 5.0 ppm levels for six months before the animals were mated to control males. All eight females fed the lower level of PCBs conceived

and five gave birth. Six of the eight females fed the higher level conceived but only one female was able to carry the fetus to term. Similarly, low mating indices and decreased survival of offspring have been reported in rodents (Linder et al., 1974; Keplinger et al., 1971). The dietary levels required to produce this effect are not conclusive at the lower levels. While no effect was observed on reproduction with diets containing 1 to 10 ppm Aroclor 1242 or 1254 in some studies, this range has induced dysfunction in other rodent studies. At 100 ppm Aroclor 1242, reproductive failure ensued (Keplinger et al., 1971). Likewise, Sherman rats exposed to 20 to 500 ppm were adversely affected in regards to reproduction (Linder et al., 1974). Comparison between studies strongly suggests that reproduction in primates is more sensitive to dietary levels of these compounds than that in rodents.

More recent studies have continued to show PCB-induced toxicity in animals such as adverse effects on the immune and reproductive systems, carcinogenic (promoter) activity, and adverse effects on the offspring of mothers exposed to PCBs, particularly in the diet (Becker and McNulty, 1984; Norback and Weltman, 1985; Preston et al., 1985; Schaeffer et al., 1984; Tryphonas et al., 1986). In addition, pharmacodynamics of PCBs, particularly the effect of lactation on the distribution of PCBs between mothers and their offspring have been investigated (Barsotti and Van Miller, 1984, Takagi et al., 1986).

Assessment and Conclusions

Scientists have recently developed statistical extrapolation models to quantitatively estimate risk to humans based on toxicity data from animal studies. These risk assessment methods do not purport to precisely quantify the expected human risk. Rather they attempt to estimate in quantitative terms an upper limit on the risk to humans that can be expected from a given level of

exposure to a toxic substance. This methodology assumes that humans are no more susceptible than members of the animal species for which toxicity data are available. These risks are intended to be worst-case estimates; consequently the actual risk is expected to be less. The upper limits are statistical confidence limits derived from a linear model. The assumption of low-dose linearity in the model may overestimate risk at low dose if the actual risk at low doses is less than would be predicted under this assumption.

Information on relative risks from PCBs, based on the cancer data in animals, was given in the 1979 Federal Register notice on Reduction of Tolerances for PCBs, prepared by FDA (FDA, 1979). At that time, several studies with various endpoints were available that could be used to estimate cancer risks. FDA used the total malignancy data from an NCI bioassay (NCI, 1978) to estimate the upper 99% confidence limit on the lifetime risk of cancer to humans. These data supported a risk that was about three times higher than the risk based on liver carcinomas from Kimbrough (1974a).

Based on the total malignancy data from the NCI bioassay, the upper 99% confidence limit on the lifetime risk for heavy consumers of species of fish known to have high concentrations of PCBs was 7 incidences of cancer per 100,000 people (PCB intake of 0.21 $\mu\text{g}/\text{kg}/\text{day}$). If risk were based on liver carcinomas from the Kimbrough (1974a) study, the upper limit on the lifetime cancer cases would be 2.3 per 100,000 population for a PCB intake of 0.21 $\mu\text{g}/\text{kg}/\text{day}$.

As explained in FDA's 1979 Federal Register notice (FDA, 1979) and supporting documents, the usefulness of this risk assessment for evaluating actual risk to humans from exposure to PCBs is extremely limited. This is due to the difficulties inherent in extrapolating from the high dose to low

dose, difficulties in extrapolating from laboratory animals to humans, and perhaps more importantly, to difficulties in bridging the gaps and uncertainties in the data available for this particular risk assessment. For example, the PCBs that were used in the toxicity studies upon which the risk assessment was based were commercial preparations that were chemically different from the PCB residues found in fish. For these reasons, the risk assessment based on laboratory animal data does not provide a basis for precise quantification of risk to humans from PCB intake from fish. As FDA stated in 1979 (FDA, 1979) when it developed its risk assessment, the agency did not use the risk assessment for precise quantitation of risk but made limited use of it to estimate relative risk for different tolerance levels.

The EPA is about to use the recent study by Norback and Weltman (1985), in which chronic dietary administration of Aroclor 1260 was shown to cause hepatocellular carcinomas in male and female Sprague-Dawley rats, to revise its risk estimates. Based on this study, the carcinogenic potency estimates would be increased over previous EPA estimates.

EPA regards the Norback and Weltman study as a major study because the Sprague-Dawley rat has a low incidence of spontaneous hepatocellular neoplasms, because the study spanned the natural life of the animal, and because concurrent morphologic liver studies showed the sequential progression of liver lesions to hepatocellular carcinomas.

As reported by Cordle et al. (1982), "The scientific data base needed to support a quantitative estimate of risk of human health as a result of exposure to environmental contaminants is nearly always inadequate. There are usually at least four major areas of uncertainty: (1) a lack of adequate information about

the exposures that occur in human populations at environmental levels of the contaminant; (2) a lack of dose-response data in humans to support projections of the effects of likely levels of exposure; (3) a comparable lack of dose-response data, even in laboratory animals, at the very low levels of exposure that commonly occur in the environment; and (4) a lack of understanding of the interactions and influences that environmental variables and characteristics of the exposed population may have on the effects of the contaminant."

Because of uncertainties about potential long-term effects of PCBs, FDA recommended to the people in Michigan that, as a precaution, they limit their fish consumption so as to keep their PCB intakes at 1 $\mu\text{g}/\text{kg}/\text{day}$ or less. This would be 60 $\mu\text{g}/\text{day}$ for the average (60 kg body weight) adult and is consistent with the policy the agency adopted in 1977 when it proposed to reduce the tolerance for PCBs in fish from 5 ppm to 2 ppm. The agency had determined that the allowable daily intake (ADI) of approximately 200 $\mu\text{g}/\text{day}$ estimated directly from the Yusho incident data in 1972 was too high (FDA, 1977). While toxicological data obtained between 1972 and 1977 did not yield a lower ADI, FDA concluded in 1977 that a maximum theoretical exposure of about 45 $\mu\text{g}/\text{day}$ would not be excessive (FDA, 1977).

FDA reviewed data available in 1982 (FDA, 1984) and has continued to monitor the progress and outcomes of a number of studies on the effects of PCBs in laboratory animals and in humans. These studies generated findings which support FDA's original concerns about the potential hazards from exposure to PCBs. While the new data reaffirm the decision to reduce the ADI established in 1972, they still do not conclusively yield a specific permissible level of intake.

ESTIMATION AND EVALUATION OF PCB INTAKE

Results of this survey (NOAA/FDA/EPA, 1986) showed that most PCBs in bluefish resembled Aroclor 1254 or 1260. Because the toxicity endpoints are not definitively related to the degree of chlorination and because regulatory agencies have not assessed the risk of specific Aroclors, the intake evaluation in this report is based on total PCBs.

As discussed in the section on human toxicology of PCBs, a group of recreational fishermen and their families who consumed large amounts of Lake Michigan fish that contained PCBs was studied for several years (Humphrey et al., 1976, Humphrey, 1983). This study provided the best information available on long-term, consistent exposure to PCBs via the diet. The average PCB intake for the study subjects was 1.75 $\mu\text{g}/\text{kg}/\text{day}$. Although no adverse health effects were observed, FDA recommended to the people in the Michigan study that as an additional safety precaution, their PCB intake should not exceed 1 $\mu\text{g}/\text{kg}/\text{day}$.

The FDA tolerance of 2 ppm for PCBs in fish has been cited a number of times in this report as a criterion for evaluating the significance of PCB residues. The tolerance was established to limit PCBs in all fish species in interstate commerce and is sufficient to protect the consumers of these commercial fish. One of the factors that makes the protection adequate is that people who consume commercial fish normally consume a variety of fish from various locations, most of which contain little or no measureable PCBs (FDA, 1979). This argument holds true also for consumers of the bluefish that are sold commercially, historically 10 to 20% of the total catch.

The 1977-78 U.S. Department of Agriculture (USDA) National Food Consumption

Household Survey showed a nationwide bluefish consumption per individual of 0.31 g/day (USDA, 1978). Using an average PCB level of 2 ppm in bluefish, the maximum permitted in fish in interstate commerce, this rate of consumption corresponds to a PCB intake of 0.01 $\mu\text{g}/\text{kg}/\text{day}$ for an individual. This level is 100 times lower than the 1 $\mu\text{g}/\text{kg}/\text{day}$ recommended maximum intake, indicating that the consumer who eats an average amount of commercially caught bluefish would have a PCB intake from bluefish well below the level of concern. That same consumer of bluefish with 2 ppm PCBs would also be able to consume similar amounts of other kinds of fish with PCB residues without exceeding the recommended maximum intake. Other dietary sources of PCB need not be considered here because fish have been shown to be the only food group in which detectable levels of PCB contamination are routinely found (FDA, 1979).

However, as FDA has acknowledged from the time the PCB tolerance was first proposed, use of 2 ppm as the only control criterion might not sufficiently control PCB intake for people who consistently consume PCB-contaminated fish, e.g., recreational fishermen who repeatedly fish in the same waters and consistently eat their catch.

PCB concentrations in bluefish from the Data Report were combined with data on recreational bluefish catch and fishing patterns obtained from the Marine Recreational Fishery Statistics Survey, Atlantic and Gulf Coasts, 1985 (NOAA/NMFS, 1986) to estimate PCB intake for bluefish recreational fishermen and their household members. An exception is North Carolina during January - February, 1985, when no recreational fishing statistics are collected. The latter report contains a detailed description of the methodology employed,

including collection of raw data, procedures for developing expanded estimates of catch, sampling variances, and precision of estimates, as well as the 1985 survey results.

Calculations

The PCB intake ($\mu\text{g}/\text{kg}/\text{day}$) per person shown in Tables 10-20 was estimated for each sampling site in the survey and mode of fishing from the NMFS recreational fishing data by multiplying the amount of bluefish consumed in grams ($\text{g}/\text{day}/\text{person}$) by the average PCB level and dividing by body weight (assuming an average body weight of 60 kg).

The amount of bluefish consumed was estimated by using the weight (WT) of bluefish that were caught by a given mode of fishing at a given sampling site during the collection phase of the bluefish survey. A conversion factor of 50% was used to convert the weight of fish caught to weight of edible portion of fish. To estimate the amount of fish consumed, it was assumed that all fish caught during the average trip for a given site were eaten by all members of the fisherman's household. The average household size estimate for a given area was determined from data obtained from the U.S. Bureau of Census. The values used are as follows: New England, 2.73; New York Bight, 2.77; Maryland/Virginia, 2.80 and North Carolina, 2.78. The formula used to calculate the amount of fish consumed (E) in $\text{g}/\text{day}/\text{person}$ was:

$$E = (\text{WT}) (0.5) / (\text{T})(\text{HS})(365), \text{ where}$$

(WT) (0.5) = amount of edible portion of bluefish caught (g)

T = number of trips to achieve a given catch weight of bluefish

HS = average household size

365 = number of exposure days to amount of bluefish consumed

The average PCB (P) in $\mu\text{g}/\text{kg}$ of body weight was calculated as follows:

$P = \text{PCB}/60$, where

PCB = average PCB concentration ($\mu\text{g}/\text{g}$) in bluefish at a
given sampling site

60 = average body weight of consumer (kg)

The PCB intake (PCBI) in $\mu\text{g}/\text{kg}/\text{day}$ per person was estimated as the product $E \times P$ (e.g., $\text{PCBI} = E \times P$).

The number of trips that a fisherman could make during the specific period at a given location for a given size category and mode of fishing and still have a PCB intake within the $1 \mu\text{g}/\text{kg}/\text{day}$ recommended maximum amount is also given on a sampling-site basis in Tables 10-20. This number was calculated by dividing $1 \mu\text{g}/\text{kg}/\text{day}$ by the PCB intake. Where this value was greater than 60, the value is not shown since it is unreasonable to expect that most fishermen would make more than one trip a day. Data on frequency of participation were obtained from the raw data files of the NMFS 1985 Marine Recreational Fisheries Statistics Survey. It should be noted that all intake calculations are based on a mean catch rate per trip. Intakes will vary as actual catches vary from this mean.

Results

The intake of PCBs for each sampling site, mode of fishing, and size category for which data were available shown in Tables 10-20 is the average intake per person for one fishing trip, assuming that the fish were divided among the members of the household of a given fisherman and consumed over a

period of 365 days. Even if the fish were consumed sooner (e.g., over one or two months), it is reasonable to use the estimate of the effective exposure per day over a one year period, given the storage and clearance patterns of PCBs in the body (Humphrey et al., 1976). Each estimate represents PCB intake for each household member based on one fishing trip. The PCB intakes relating to all survey sites and fishing modes for one trip were less than the recommended maximum intake of 1 $\mu\text{g}/\text{kg}/\text{day}$.

For the year 1985, intake of PCBs per household member, where a given fisherman made several trips a year, would be the sum of the average intakes taken from the appropriate table for all trips made, assuming that all the fish were eaten in one year by the household members. Tables 10-20 show the number of trips that a given fisherman could make at a given site for a given fish size category in 1985, and still have a PCB intake within the recommended limit. The number of trips allowed ranged from two to greater than sixty.

The tables also may be used to estimate how many trips fishermen could make at different sites and for different modes of fishing. The total PCB intake is simply the product of average intake and number of trips taken for each cell of the table, added over the different sites and different modes of fishing. As long as this cumulative intake does not exceed 1 $\mu\text{g}/\text{kg}/\text{day}$, members of the fisherman's household would not appear to be consuming excessive amounts of PCBs, provided that bluefish were their sole source of PCBs.

Caution should be exercised in using these data to predict specific future PCB intakes from bluefish for the identified geographical areas and modes of fishing, since the survey only represents PCBs in bluefish for each location and time of year in 1985. It is highly likely, given the life history of the

bluefish and the chemical characteristics of PCBs, that there will be PCBs in bluefish in subsequent years and that big fish will contain more than small fish. However, it is unlikely, because of the variation in bluefish migratory patterns, that bluefish in one area at a given month in a given year would be in that area at the same time in another year.

RECOMMENDATIONS

A major objective of this interagency study was to provide information on bluefish that could be used by state and local authorities to determine if there is a need for regulation of recreational or commercial fishing, and to provide guidelines to ensure that any regulation is rational and equitable.

As discussed above, the FDA tolerance of 2 ppm which applies to fish in interstate commerce, will adequately control PCB intake from bluefish (and other fish as well) for those individuals who generally consume fish obtained commercially. Accordingly, no additional FDA initiative is recommended at this time with regard to bluefish in interstate commerce.

Many State officials already regulate some contaminants in recreationally caught fish and are familiar with the kinds of regulatory and advisory actions that are effective in controlling excessive intakes of contaminants from recreational fish.

The detailed information in this report will permit authorities to estimate potential PCB intake for various subpopulations who eat recreationally-caught bluefish. For example, in general, there should be no need to regulate consumption of small (300 mm (11.8 inches) or less) and medium (301 to 500 mm

(11.8 to 19.7 inches)) bluefish. The estimated PCB intakes from these fish were low enough that in most cases, anyone catching only small and medium bluefish would not exceed the recommended maximum intake.

However, there may be need for action to control PCB intake for some consumers of the large (greater than 500 mm (19.7 inches)) bluefish. Those who consume large bluefish from only a few trips per year would rarely exceed the maximum PCB intake. For individuals who make more trips, authorities should consider issuing advisories to limit the amount of catch consumed within a single household. When determining what kind of action is appropriate, authorities should consider the mode of fishing as well as the location and the seasonal variations (Tables 10-20).

The number of trips made by an individual was used in this report as a convenient index of PCB intake. However, the tables on intake estimates (Tables 10-20) also contain information on amount of fish caught during specified months. Authorities should evaluate all the available information to determine the most understandable and, therefore, most effective format for presentation to their respective consumer populations. It may be practical, for example, to issue advisories through vessel registration facilities in an area where private and rental boats provide a major portion of the catch of large bluefish. On the other hand, if users of party and charter boats have a high potential for excessive PCB exposure, notices provided through boat operators might be more effective.

Public health authorities in each Atlantic Coast state should assess the PCB intakes estimated from this survey for their areas, paying particular attention to large bluefish. However, because of variation in bluefish migratory patterns

it is necessary to be cautious in predicting that a PCB intake estimated for bluefish collected in 1985 would be repeated in subsequent years. This survey can be used as a model for additional studies or for determining the adequacy of existing data for use in predicting PCB intakes from recreationally-caught bluefish.

LITERATURE CITED

ALLEN, J. R., L. A. CARSTENS, and D. A. BARSOTTI.

1974. Residual Effects of Short-Term, Low-Level Exposure of Non-Human Primates to Polychlorinated Biphenyls. *Toxicol. and Appl. Pharmacol.*, 30:440-451.

ALLEN, J. R.

1975. Response of the Non-Human Primate to Polychlorinated Biphenyl Exposure. *Fed. Proc.*, 34:1675-1679.

ALLEN, J. R.

1976. Unpublished presentation at HEW Toxicology Committee to consider problems of PCB's in mothers' milk.

ALLEN, J. R., and D. A. BARSOTTI.

1976. The Effects of Transplacental and Mammary Movement of PCB's on Infant Rhesus Monkeys. *Toxicology*, 6:331-340.

ANDO, M., H. SAITO, and I. WAKISAKA.

1986. Gas Chromatographic and Mass Spectrometric Analysis of Polychlorinated Biphenyls in Human Placenta and Cord Blood. *Environ. Res.*, 41:14-22.

ALVARES, A. P., ~~D. R.~~ BICKERS, and A. KAPPAS.

1973. Polychlorinated Biphenyls: A New Type of Inducer of Cytochrome P-448 in the Liver. *Proc. of the Natl. Acad. of Sci. U.S.A.*, 70:1321-1325.

BARSOTTI, D.A., and J.P. VAN MILLER.

1984. Accumulation of a Commercial Polychlorinated Biphenyl Mixture (Aroclor 1016) in Adult Rhesus Monkeys and their Nursing Infants. *Toxicology*, 30:31-44.

BARSONI, D. A., R. J. MARLAR, and J. R. ALLEN.

1976. Reproductive Dysfunction in Rhesus Monkeys Exposed to Low Levels of Polychlorinated Biphenyls (Aroclor 1248). *Food Cosmet. Toxicol.*, 14:99-103.

BECKER, G.M., and W.P. MCNULTY.

1984. Gastric Epithelial Cell Proliferation in Monkeys Fed 3,4,3',4'-Tetrachlorobiphenyl. *J. of Pathol.*, 143:267-274.

BERCOVICI, B., M. WASSERMANN, S. CUCOS, M. RON, D. WASSERMANN, and A. PINES.

1983. Serum Levels of Polychlorinated Biphenyls and Some Organochlorine Insecticides in Women with Recent and Former Missed Abortions. *Environ. Res.*, 30:169-174.

CHEN, P.H., M. LUO, C. WONG, and C. CHEN.

1984. Polychlorinated Biphenyls, Dibenzofurans, and Quaterphenyls in the Toxic Rice-Bran Oil and PCBs in the Blood of Patients with PCB Poisoning in Taiwan. *Am. J. of Ind. Med.*, 5:133-145.

CHESNEY, C. F., and J. R. ALLEN.

1974. Oxidative Phosphorylation and Respiration by Liver Mitochondria from Polychlorinated Biphenyl Intoxicated Rats. *Biochem. Pharmacol.*, 23:1577-1582.

CORDLE, F., R. LOCKE, and J. SPRINGER.

1982. Risk Assessment in a Federal Regulatory Agency: An Assessment of Risk Associated with the Human Consumption of Some Species of Fish Contaminated with Polychlorinated Biphenyls (PCBs). *Environ. Health Perspect.*, 45:171-182.

FOOD AND DRUG ADMINISTRATION

1977. Polychlorinated Biphenyls (PCBs). Federal Register, 42:17487-17494.

FOOD AND DRUG ADMINISTRATION

1979. Polychlorinated Biphenyls (PCBs); Reduction of Tolerances. Federal Register, 44:38330-38340.

FOOD AND DRUG ADMINISTRATION

1984. Review of All Submissions on the Health Effects of PCBs in Connection with the Rulemaking to Establish a Tolerance in Fish and Shellfish, May, 1984.

FOOD AND DRUG ADMINISTRATION

1987. Memorandum: Review of FY'86 Cooking Study of Bluefish, February 20, 1987.

FEIN, G., J. JACOBSON, S. JACOBSON, P. SCHWARTZ, and J. DOWLER.

1984. Prenatal Exposure to Polychlorinated Biphenyls: Effects on Birth Size and Gestational Age. *J. of Pediatr.*, 105:315-320.

GOLDSTEIN, J. A., P. HICKMAN, and D. L. JUE.

1974. Experimental Hepatic Porphyria Induced by Polychlorinated Biphenyls. *Toxicol. and Appl. Pharmacol.*, 27:437-448.

HARRIS, C., and W. BRADSHAW.

1984. Alterations in Liver Ultrastructure and Induction of UDP-Glucuronyltransferase in the Rat Following Prenatal Exposure to 3,4,3',4'-Tetrachlorobiphenyl. Arch. Environ. Contam. Toxicol., 13:715-721.

HUMPHREY, H.

1983. Population Studies of PCBs in Michigan Residents. In PCBs: Human and Environmental Hazards, Eds. F. D'Itri and M. Kamrin Butterworth Publishers, pp. 299-310.

HUMPHREY, H. E. B., H. A. PRICE, and M. L. BUDD.

1976. Evaluation of Changes of the Level of Polychlorinated Biphenyls (PCB's) in Human Tissue. Final Report of FDA Contract No. 223-73-2209.

JACOBSEN, S.W., G.G. FEIN, J.L. JACOBSEN, P.R., SCHWARTZ AND J.K. DOWLER

1985. The Effect of Intrauterine PCB Exposure on Visual Recognition Memory. Child Dev. 56:853-860.

KEPLINGER, M.L., O.E. FANCHER, and J.C. CALANDRA.

1971. Toxicologic Studies with Polychlorinated Biphenyls Presented at the Tenth Annual Meeting, Society of Toxicology, March 7-11 1971, Washington, D.C.

KIMBROUGH, R. D.

1974a. "The Toxicity of Polychlorinated Polycyclic Compounds and Related Chemicals." CRC Crit. Rev. Toxicol., 2:445-498.

KIMBROUGH, R. D.

1974b. "The Toxicity of Polychlorinated Polycyclic Compounds and Related Chemicals." CRC Crit. Rev. Toxicol., 2:442.

KIMBROUGH, R. D., and R. E. LINDER.

1974. Induction of Adenofibrosis and Hepatomas of the Liver in BALB/cJ Mice by Polychlorinated Biphenyls (Aroclor 1254). J. Natl. Cancer Inst., 53:547-552.

KIMBROUGH, R. D., R. A. SQUIRE, R. E. LINDER, J. D. STRANDBERG, R. J. MONTALI, and V. W. BURSE.

1975. Induction of Liver Tumors in Sherman Strain Female Rats by Polychlorinated Biphenyl Aroclor 1260. J. Natl. Cancer Inst., 55:1453-1459.

KIMBROUGH, R.

1975. Pathological Findings Associated with Chronic Experimental Exposure to PCB's. Proceedings of the National Conference on Polychlorinated Biphenyls. (EPA-560/6-75-004), 30-34.

KURATSUNE, M., Y. MASUDA, and J. NAGAYAMA.

1975. Some of the Recent Findings Concerning Yusho. Proceedings of the National Conference on Polychlorinated Biphenyls. (EPA-560/6-75-004), 14-29.

KURATSUNE, M., and R. SHAPIRO (Eds.).

1984. PCB Poisoning in Japan and Taiwan, Alan R. Liss, Inc., New York, pp. 1-155.

LEECE, B., M. DENOMME, R. TOWNER, and S. LI.

1985. Polychlorinated Biphenyls: Correlation Between In Vivo and In Vitro Quantitative Structure-Activity Relationships (QSARs). J. Toxicol. Environ. Health, 16:379-388.

LINDER, R. E., T. B. GAINES, and R. D. KIMBROUGH.

1974. The Effect of PCB on Rat Reproduction. Food Cosmet. Toxicol.,
12:63-77.

LU, Y.-C., and Y.-C. WU.

1985. Clinical Findings and Immunological Abnormalities in Yu-Cheng
Patients. Environ. Health Perspect., 59:17-29.

MATTHEWS, H. B., and M. W. ANDERSON.

1975a. Effect of Chlorination on the Distribution and Excretion of
Polychlorinated Biphenyls. Drug Metab. Dispos., 3:371-380.

MATTHEWS, H. B., and M. ANDERSON.

1975b. PCB Chlorination Versus PCB Distribution and Excretion. Proceedings
of the National Conference on Polychlorinated Biphenyls.
(EPA-560/6-75-004), 50-56.

MATTHEWS, H.B., and R.L. DEDRICK.

1984. Pharmacokinetics of PCBs. Ann. Rev. Pharmacol. Toxicol., 24:85-103

MCNULTY, W. P.

1975. Primate Study. Proceedings of the National Conference on
Polychlorinated Biphenyls. (EPA-560/6-75-004) 347-350.

MIEURE, J. P., O. HICKS, R. G. KALEY, and V. W. SAEGER.

1975. Characterization of Polychlorinated Biphenyls. Proceedings of the
National Conference on Polychlorinated Biphenyls. (EPA 560/6-75-004),
84-93.

MOORE, J. A., B. N. GUPTA, and J. G. VOS.

1975. Toxicity of 2,3,7,8-Tetrachlorodibenzofuran - Preliminary Results.
Proceedings of the National Conference of Polychlorinated Biphenyls.
(EPA-560/6-75-004), 77-80.

NAGAYAMA, J., Y. MASUDA, and M. KURATSUNE.

1975. Chlorinated Dibenzofurans in Kanechlors and Rice Oils Used by Patients with Yusho. *Fukuoka Acta Med.*, 66:593-599. (Cited by Kuratsune et al., 1975).

NATIONAL CANCER INSTITUTE

1978. Bioassay of Aroclor 1254 for Possible Carcinogenicity. Publication No. (NIH 78-838), DHEW, Washington, D.C.

NATIONAL OCEANIC and ATMOSPHERIC ADMINISTRATION/FOOD and DRUG ADMINISTRATION/ENVIRONMENTAL PROTECTION AGENCY

1986. Report on 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish - Data Report. NOAA, FDA, and EPA, Washington, D.C. 179 p.
NTIS Accession No. PB86 218070/AS. (Data tapes available from NTIS, Accession No. PB87-142 881).

NATIONAL OCEANIC and ATMOSPHERIC ADMINISTRATION/NATIONAL MARINE FISHERIES SERVICE

1986. Marine Recreational Fisheries Statistics Survey, Atlantic and Gulf Coasts, 1985. *Current Fish. Stat.* No. 8327. NOAA, NMFS, Washington, D.C., 18 p.

NORBACK, D.H., and R.H. WELTMAN.

1985. Polychlorinated Biphenyl Induction of Hepatocellular Carcinoma in the Sprague-Dawley Rat. *Environ. Health Perspect.* 60:97-105.

PRESTON, B.D., E.C. MILLER, and J.A. MILLER.

1985. The Activities of 2,2',5,5' Tetrachlorobiphenyl, its 3,4-Oxide Metabolite, and 2,2',4, 4'-Tetrachlorobiphenyl in Tumor Induction and Promotion Assays. *Carcinog.* 6:451-453.

RINGER, R. K., R. J. AULERICH, and M. ZABIK.

1972. Effect of Dietary Polychlorinated Biphenyls on Growth and Reproduction of Mink. Prepr. Pap. Nat. Meet., Div. Water, Air Waste Chem., Amer. Chem. Soc., 12:149-154.

ROACH, J. A. G., and I. H. POMERANTZ.

1974. "The Finding of Chlorinated Dibenzofurans in Aroclor PCB's of Recent Manufacture," Paper No. 53 presented at 88th Annual Meeting of the Association of Official Analytical Chemists, Washington, DC, Oct. 14-17.

ROGAN, W., B. GLADEN, J. MCKINNEY, N. CARRERAS, P. HARDY, J. THULLEN, J. TINGLESTAD, and M. TULLY.

1986. Neonatal Effects of Transplacental Exposure to PCBs and DDE. J. Pediatr., 109:335-341.

SAVAGE, E. P., J. D. TESSARI, J. W. MALBERG, H. W. WHEELER, and J. R. BAGBY.

1973. Organochlorine Pesticide Residues and Polychlorinated Biphenyls in Human Milk. Colorado--1971-1972. Pestic. Monit. J., 7:1-5.

SCHAEFFER, E., H. GREIM, and W. GOESSNER.

1984. Pathology of Chronic Polychlorinated Biphenyl (PCB) Feeding in Rats. Toxicol. Appl. Pharmacol. 75:278-288.

TAKAGI, Y., S. ABURADA, K. HASHIMOTO, and T. KITaura.

1986. Transfer and Distribution of Accumulated (14C) Polychlorinated Biphenyls from Maternal to Fetal and Suckling Rats. Arch. Environ. Contam. Toxicol. 15:709-715.

TAYLOR, P., C. LAWRENCE, H.-L. HWANG, and A. PAULSON.

1984. Polychlorinated Biphenyls: Influence on Birthweight and Gestation. Public Health Briefs, 74:1153-1154.

TRYPHONAS, L., S. CHARBONNEAU, H. TRYPHONAS, Z. ZAWIDZKA, J. MES, J. WONG, and D. ARNOLD.

1986. Comparative Aspects of Aroclor 1254 Toxicity in Adult Cynomolgus and Rhesus Monkeys: A Pilot Study. Arch. Environ. Contam. Toxicol., 15:159-169.

U.S. DEPARTMENT OF AGRICULTURE

1978. Nationwide Food Consumption Household Survey, Human Nutrition Information Center.

VOS, J. G., and R. B. BEEMS.

1971. Dermal Toxicity Studies of Technical Polychlorinated Biphenyls and Fractions Thereof in Rabbits. Toxicol. Appl. Pharmacol., 19:617-633.

WICKIZER, T.W., L.B. BRILLIANT, R. COPELAND, and R. TILDEN.

1981. Polychlorinated Biphenyl Contamination of Nursing Mother's Milk in Michigan. Am. J. Public Health, 71:132-137.

YAKUSHIJI, T., I. WATANABE, K. KUWABARA, R. TANAKA, T. KASHIMOTO, N. KUNITA, and I. HARA.

1984a. Rate of Decrease and Half-life of Polychlorinated Biphenyls (PCBs) in the Blood of Mothers and Their Children Occupationally Exposed to PCBs. Arch. Environ. Health, 39:368-373.

YAKUSHIJI, T., I. WATANABE, K. KUWABARA, R. TANAKA, T. KASHIMOTO, N. KUNITA, and I. HARA.

1984b. Postnatal Transfer of PCBs from Exposed Mothers to Their Babies: Influence of Breast-feeding. Arch. Environ. Contam. Toxicol., 13:341-345.

FIGURES



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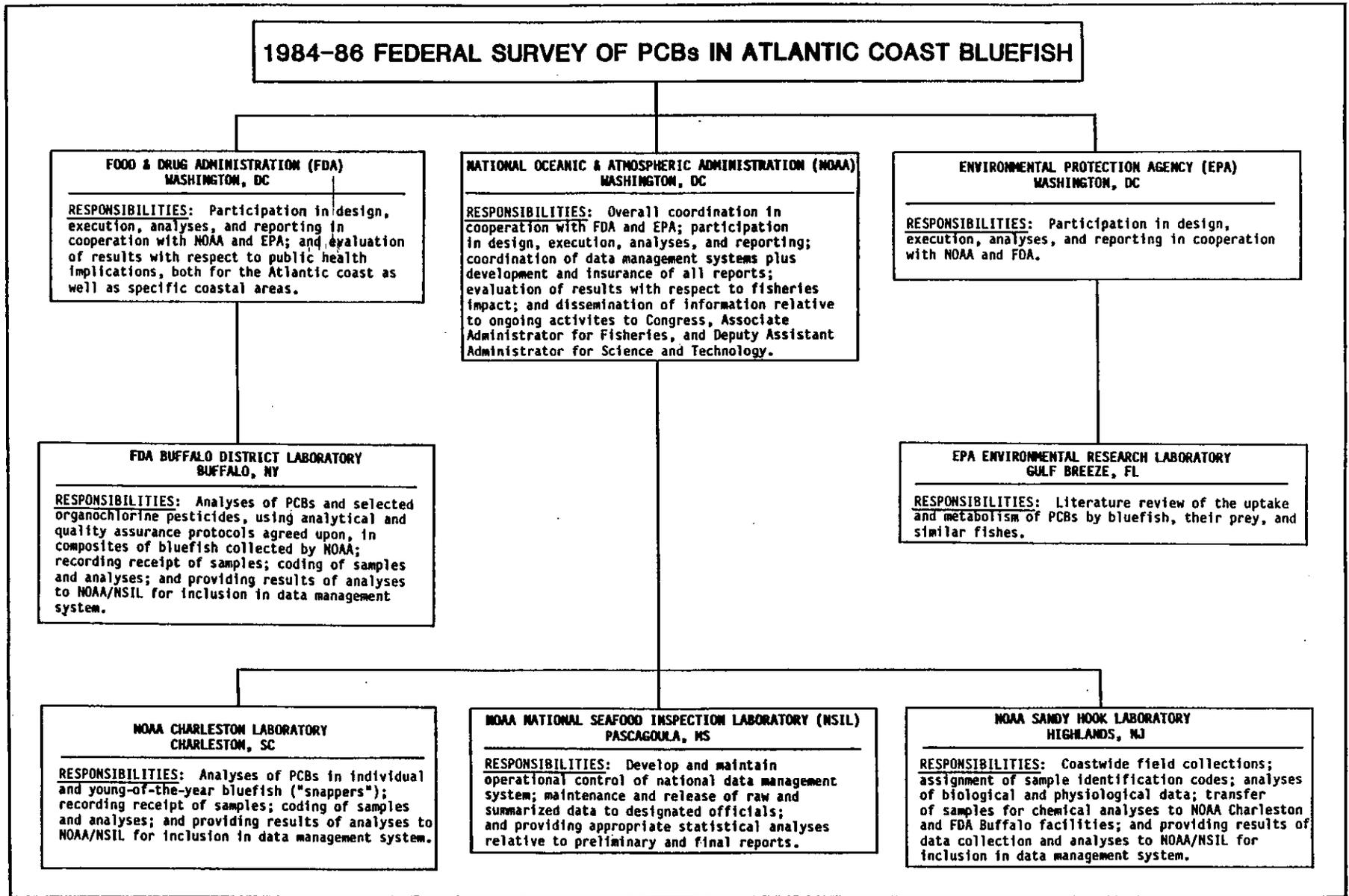


Figure 1. Organizational structure and responsibilities of all participants in the 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish.

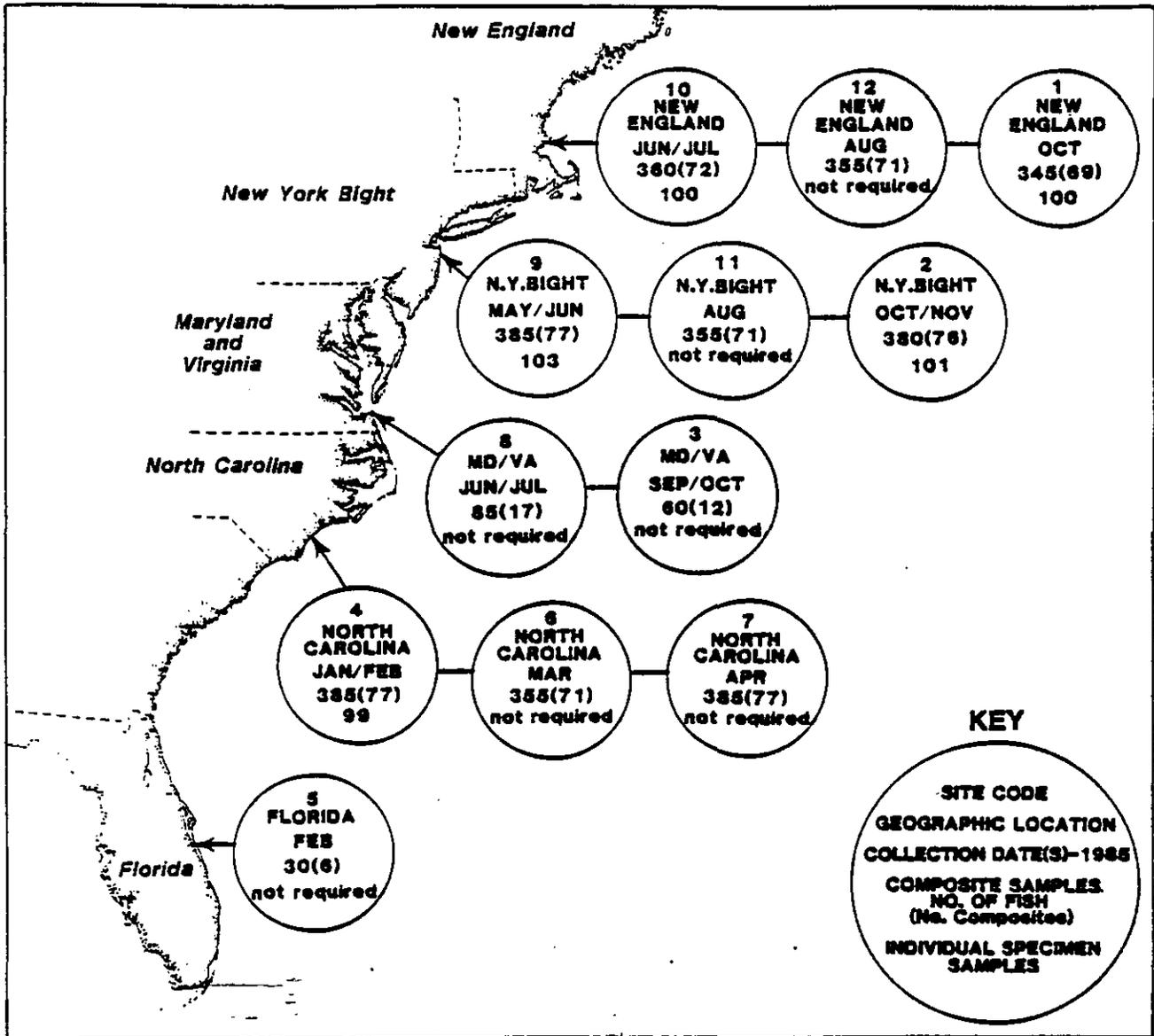


Figure 2. Summary of collections made during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish.

TABLES



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Table 1. Descriptive and inferential statistics on PCB levels, by sampling site, for five-fish composite samples of bluefish with fork lengths ≤ 300 mm, collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. PCB levels in parts per million (ppm), \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), and a dash (-) = no data.

Sampling Site		Number of Samples	PCB Level (ppm)		Samples with PCB Level >2 ppm		Maximum Expected % in Population with PCB Level > 2 ppm with 95% Confidence	Upper 95% Confidence Limit on the Mean PCB Level
Collection Date (1985)	Geographic Area		\bar{x}	s	No.	%		
Jan/Feb	North Carolina	6	0.26	0.14	0	0	0.00	0.38
March	North Carolina	-	-	-	-	-	-	-
April	North Carolina	6	0.25	0.13	0	0	0.00	0.36
May	Maryland/Virginia	7	0.20	0.00	0	0	0.00	0.20
May/June	New York Bight	6	0.25	0.12	0	0	0.00	0.35
June	New England	1	0.20	0.00	0	0	-	-
August	New York Bight	-	-	-	-	-	-	-
August	New England	-	-	-	-	-	-	-
Sep/Oct	Maryland/Virginia	6	0.17	0.08	0	0	0.00	0.23
October	New England	-	-	-	-	-	-	-
Oct/Nov	New York Bight	6	0.17	0.08	0	0	0.00	0.23
Combined		38	0.22	0.10	0	0	0.00	0.25

Table 2. Descriptive and inferential statistics on PCB levels, by sampling site, for five-fish composite samples of bluefish with fork lengths 301-500 mm, collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. PCB levels in parts per million (ppm), \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), and a dash (-) = no data.

Sampling Site		Number of Samples	PCB Level (ppm)		Samples with PCB Level > 2 ppm		Maximum Expected % in Population with PCB Level > 2 ppm with 95% Confidence	Upper 95% Confidence Limit on the Mean PCB Level
Collection Date (1985)	Geographic Area		\bar{x}	s	No.	%		
Jan/Feb	North Carolina	6	0.71	0.43	0	0	9.38	1.06
March	North Carolina	6	0.20	0.00	0	0	0.00	0.20
April	North Carolina	6	0.20	0.00	0	0	0.00	0.20
May	Maryland/Virginia	10	0.20	0.00	0	0	0.00	0.20
May/June	New York Bight	6	0.33	0.20	0	0	0.00	0.49
June	New England	6	0.26	0.14	0	0	0.00	0.37
August	New York Bight	6	0.25	0.13	0	0	0.00	0.36
August	New England	6	0.34	0.22	0	0	0.02	0.51
Sep/Oct	Maryland/Virginia	6	0.20	0.00	0	0	0.00	0.20
October	New England	4	0.94	0.34	0	0	18.89	1.34
Oct/Nov	New York Bight	6	0.20	0.00	0	0	0.00	0.20
Combined		68	0.32	0.18	0	0	0.00	0.44

Table 3. Descriptive and inferential statistics on PCB levels, by sampling site, for five-fish composite samples of bluefish with fork lengths >500 mm, collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. PCB levels in parts per million (ppm), \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), and a dash (-) = no data.

Collection Date (1985)	Geographic Area	Number of Samples	PCB Level (ppm)		Samples with PCB Level >2 ppm		Maximum Expected % in Population with PCB Level > 2 ppm with 95% Confidence	Upper 95% Confidence Limit on the Mean PCB Level
			\bar{x}	s	No.	%		
Jan/Feb	North Carolina	65	2.21	5.52	7	10.77	59.56	3.35
March	North Carolina	65	1.57	0.48	11	16.92	25.62	1.67
April	North Carolina	65	1.80	0.47	15	23.08	41.96	1.90
May	Maryland/Virginia	-	-	-	-	-	-	-
May/June	New York Bight	65	1.06	0.48	3	4.62	5.40	1.16
June	New England	65	1.23	0.46	3	4.62	8.49	1.32
August	New York Bight	65	0.95	0.81	3	4.62	15.53	1.12
August	New England	65	4.80	7.76	18	27.69	71.63	6.41
Sep/Oct	Maryland/Virginia	-	-	-	-	-	-	-
October	New England	65	1.03	0.45	2	3.08	3.67	1.12
Oct/Nov	New York Bight	64	1.99	0.96	29	45.31	57.77	2.19
Combined		584	1.85	0.28	91	15.58	50.88	2.49

Table 4. Variation among PCB levels, by sampling site on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, PCB levels in parts per million (ppm), n = sample size, s = standard deviation (ppm), CV = coefficient of variation (%), and a dash (-) = no data.

Sampling Site		Length Strata (mm fork length)								
Collection Date (1985)	Geographic Area	≤300			301-500			>500		
		n	s	CV	n	s	CV	n	s	CV
Jan/Feb	North Carolina	6	0.14	55.3	6	0.43	60.6	65	5.52	250.2
March	North Carolina	-	-	-	-	-	-	65	0.48	30.4
April	North Carolina	6	0.13	51.6	6	0.00	0.0	65	0.47	26.3
May	Maryland/Virginia	7	0.00	0.0	10	0.00	0.0	-	-	-
May/June	New York Bight	6	0.12	49.0	6	0.20	60.7	65	0.48	44.9
June	New England	1	0.00	0.00	6	0.14	54.1	65	0.46	37.1
August	New York Bight	-	-	-	6	0.13	50.3	65	0.81	85.2
August	New England	-	-	-	6	0.22	65.5	65	7.76	161.7
Sep/Oct	Maryland/Virginia	6	0.08	49.0	6	0.00	0.0	-	-	-
October	New England	-	-	-	4	0.34	35.7	65	0.45	43.9
Oct/Nov	New York Bight	6	0.08	49.0	6	0.00	0.0	64	0.96	48.4
	Combined	38	0.10	47.9	68	0.18	56.4	584	0.28	14.9

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Table 5. Variation among PCB levels by sampling site on a length-stratum basis, for individual samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, PCB levels in parts per million (ppm), n = sample size, s = standard deviation (ppm), CV = coefficient of variation (%), and a dash (-) = no data.

Sampling Site		Length Strata (mm fork length)								
Collection Date (1985)	Geographic Area	<300			301-500			>500		
		n	s	CV	n	s	CV	n	s	CV
Jan/Feb	North Carolina	14	0.23	81.9	30	0.35	87.9	55	0.84	47.7
May/June	New York Bight	15	0.15	78.0	30	0.26	66.6	58	3.00	225.0
June	New England	9	0.19	63.8	37	0.28	70.7	54	0.59	57.3
October	New England	15	0.33	92.3	21	0.35	61.6	64	0.55	45.5
Oct/Nov	New York Bight	15	0.15	61.6	30	0.08	35.0	56	1.16	66.1
	Combined	68	0.22	82.2	148	0.28	72.4	287	1.53	109.9

Table 6. Sample correlations between PCBs/lipid content and PCBs/fork length, by sampling site on a length-stratum basis, for individual bluefish samples collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, n = sample size, Lip = correlation between PCBs and lipid content, Fln = correlation between PCBs and fork length, and an asterisk (*) denotes a statistically significant correlation at the 5% level.

Sampling Site		Length Strata (mm fork length)				
Collection Date (1985)	Geographic Area		≤300	301-500	>500	Combined
Jan/Feb	North Carolina	n	14	30	55	99
		Lip	0.05	0.53*	0.15*	0.71*
		Fln	-0.06	0.41*	0.42*	0.76*
May/June	New York Bight	n	15	30	58	103
		Lip	0.72*	0.41*	0.05	0.16
		Fln	0.05	-0.14	0.15	0.24*
June	New England	n	9	37	54	100
		Lip	0.88*	0.69*	-0.01	0.19
		Fln	-0.31	0.36*	0.15	0.58*
October	New England	n	15	21	64	100
		Lip	0.23	0.39	0.57*	0.67*
		Fln	0.35	0.48*	0.50*	0.67*
Oct/Nov	New York Bight	n	15	30	56	101
		Lip	-0.03	0.25	0.79*	0.88*
		Fln	0.30	-0.03	0.22	0.68*
Combined		n	68	148	287	503
		Lip	0.31*	0.52*	0.26*	0.43*
		Fln	-0.08	0.35*	0.20*	0.44*

Table 7. Descriptive and inferential statistics on PCB levels, by sampling site on a length-stratum and sex basis, for individual bluefish samples collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. PCB levels in parts per million (ppm), lengths in millimeters (mm) fork length, n = sample size, \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), and a dash (-) = no data.

Sampling Site		Length Strata (mm fork length)						
Collection Date (1985)	Geographic Area		≤300		301-500		>500	
			Male	Female	Male	Female	Male	Female
Jan/Feb	North Carolina	n	-	-	2	4	24	31
		\bar{x}	-	-	0.20	0.53	1.94	1.61
		s	-	-	0.00	0.62	0.73	0.89
May/June	New York Bight	n	3	6	6	11	25	33
		\bar{x}	0.13	0.20	0.38	0.37	1.00	1.58
		s	0.12	0.23	0.29	0.25	0.70	3.94
June	New England	n	1	1	11	13	31	22
		\bar{x}	0.20	0.65	0.39	0.44	1.10	0.99
		s	0.00	0.00	0.28	0.33	0.72	0.33
October	New England	n	-	-	8	9	28	29
		\bar{x}	-	-	0.55	0.50	1.35	1.11
		s	-	-	0.44	0.32	0.64	0.41
Oct/Nov	New York Bight	n	-	-	-	-	30	27
		\bar{x}	-	-	-	-	1.66	1.86
		s	-	-	-	-	1.03	1.30
Combined		n	4	7	27	37	138	142
		\bar{x}	0.15	0.27	0.42	0.42	1.40	1.45
		s	0.10	0.27	0.33	0.32	0.84	2.04
Upper 95% Confidence Limit on the Mean			0.18	0.47	0.51	0.51	1.52	1.74

Table 8. Descriptive and inferential statistics on PCB levels, by sampling site on a length-stratum and pre- and post-spawning basis, for individual bluefish samples collected during the 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. PCB levels in parts per million (ppm), lengths in millimeters (mm) fork length, n = sample size, \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), and a dash (-) = no data.

Sampling Site		Length Strata (mm fork length)						
Collection Date (1985)	Geographic Area		≤300		301-500		>500	
			Pre-Spawning	Post-Spawning	Pre-Spawning	Post-Spawning	Pre-Spawning	Post-Spawning
Jan/Feb	North Carolina	n	-	-	4	-	-	31
		\bar{x}	-	-	0.53	-	-	1.61
		s	-	-	0.62	-	-	0.89
May/June	New York Bight	n	6	-	8	3	7	26
		\bar{x}	0.20	-	0.43	0.20	1.23	1.67
		s	0.23	-	0.27	0.00	0.62	4.44
June	New England	n	-	1	8	5	6	16
		\bar{x}	-	0.65	0.45	0.43	0.97	1.01
		s	-	0.00	0.34	0.36	0.34	0.34
October	New England	n	-	-	1	8	1	28
		\bar{x}	-	-	0.20	0.54	1.18	1.11
		s	-	-	0.00	0.32	0.00	0.41
Oct/Nov	New York Bight	n	-	-	-	-	-	27
		\bar{x}	-	-	-	-	-	1.83
		s	-	-	-	-	-	1.29
	Combined	n	6	1	21	16	14	128
		\bar{x}	0.20	0.65	0.45	0.44	1.12	1.49
		s	0.23	0.00	0.36	0.31	0.49	0.19
	Upper 95% Confidence Limit on the Mean		0.39	-	0.59	0.58	1.35	1.52

Table 9. Descriptive statistics, on a length-stratum basis, for PCB analyses of individual and five-fish composite (made up from same fish used for individual analyses) samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, PCB levels in parts per million (ppm), \bar{x} = arithmetic mean (ppm), s = standard deviation (ppm), min = lowest analyzed level (ppm), and max = highest analyzed level (ppm).

Length strata (mm fork length)	Sample Type	Number of Samples	\bar{x}	s	min	max
≤300	Individual	10	0.28	0.15	0.20	0.63
	5-Fish Composite	2	0.21	0.01	0.20	0.22
301-500	Individual	30	0.47	0.21	0.20	1.07
	5-Fish Composite	6	0.40	0.09	0.28	0.53
>500	Individual	55	1.40	3.05	0.20	23.02
	5-Fish Composite	11	1.63	1.87	0.62	7.15

Table 11. SITE 6 (North Carolina, March 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	167,363	-	-
	Catch Weight per Trip (lbs)	0.91	-	-
	Fish Consumed (g/person/day)	0.20	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	29,288	-	-
	Catch Weight per Trip (lbs)	5.31	-	-
	Fish Consumed (g/person/day)	1.19	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0039	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	-	-
>500	No. of Trips	-	1,235,907	377,257
	Catch Weight per Trip (lbs)	-	7.85	5.74
	Fish Consumed (g/person/day)	-	1.75	1.28
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	0.0460	0.0336
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	21	29
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	100.00	83.33

Table 10. **SITE 4 (North Carolina, January-February 1985)** recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	-	-	-
	Catch Weight per Trip (lbs)	-	-	-
	Fish Consumed (g/person/day)	-	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	-	-	-
	Catch Weight per Trip (lbs)	-	-	-
	Fish Consumed (g/person/day)	-	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
>500	No. of Trips	-	-	-
	Catch Weight per Trip (lbs)	-	-	-
	Fish Consumed (g/person/day)	-	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-

NOTE: This table contains no calculations of intake of PCBs since there were no recreational fishing data (ie., catch and trips) collected during this time period.

Table 13. SITE 8 (Maryland/Virginia, May 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	10,329	136,111	31,473
	Catch Weight per Trip (lbs)	0.33	0.18	2.40
	Fish Consumed (g/person/day)	0.07	0.04	0.53
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0002	0.0001	0.0018
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
301-500	No. of Trips	4,976	573,625	43,430
	Catch Weight per Trip (lbs)	2.50	0.69	1.58
	Fish Consumed (g/person/day)	0.55	0.15	0.35
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0018	0.0005	0.0012
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
>500	No. of Trips	6,252	658,948	50,716
	Catch Weight per Trip (lbs)	1.24	2.19	58.49
	Fish Consumed (g/person/day)	0.28	0.49	12.98
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-

Table 12. SITE 7 (North Carolina, April 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	167,363	-	-
	Catch Weight per Trip (lbs)	0.91	-	-
	Fish Consumed (g/person/day)	0.21	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0009	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	-	-
301-500	No. of Trips	29,288	-	-
	Catch Weight per Trip (lbs)	5.31	-	-
	Fish Consumed (g/person/day)	1.19	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0040	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	-	-
>500	No. of Trips	-	1,235,907	377,257
	Catch Weight per Trip (lbs)	-	7.85	5.74
	Fish Consumed (g/person/day)	-	1.75	1.28
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	0.0527	0.0385
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	18	25
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	100.00	83.33

Table 15. **SITE 9 (New York Bight, May-June 1985)** recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	2,357	-	141,129
	Catch Weight per Trip (lbs)	2.41	-	0.45
	Fish Consumed (g/person/day)	0.54	-	0.10
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0023	-	0.0004
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	-	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	-	100.00
301-500	No. of Trips	803,814	130,111	849,909
	Catch Weight per Trip (lbs)	1.80	0.30	1.33
	Fish Consumed (g/person/day)	0.41	0.07	0.30
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0022	0.0004	0.0016
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
>500	No. of Trips	4,714	520,044	103,544
	Catch Weight per Trip (lbs)	23.63	0.65	17.75
	Fish Consumed (g/person/day)	5.30	0.15	3.98
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0940	0.0026	0.0706
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	10	+	14
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	70.73	100.00	91.04

Table 14. **SITE 3 (Maryland/Virginia, September-October 1985)** recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	10,329	136,111	31,473
	Catch Weight per Trip (lbs)	0.39	1.71	0.41
	Fish Consumed (g/person/day)	0.09	0.38	0.09
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0002	0.0011	0.0002
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
301-500	No. of Trips	160,979	2,996,931	1,217,384
	Catch Weight per Trip (lbs)	0.35	0.92	1.26
	Fish Consumed (g/person/day)	0.08	0.21	0.28
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0003	0.0007	0.0009
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
>500	No. of Trips	-	203,873	45,441
	Catch Weight per Trip (lbs)	-	1.15	4.65
	Fish Consumed (g/person/day)	-	0.26	1.04
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-

Table 16. SITE 11 (New York Bight, August 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	2,211,760	-	143,609
	Catch Weight per Trip (lbs)	0.08	-	0.41
	Fish Consumed (g/person/day)	0.02	-	0.09
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	140,203	-	749,205
	Catch Weight per Trip (lbs)	1.13	-	1.29
	Fish Consumed (g/person/day)	0.25	-	0.29
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0011	-	0.0012
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	-	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	-	100.00
>500	No. of Trips	67,088	27,811	251,351
	Catch Weight per Trip (lbs)	5.14	63.27	10.86
	Fish Consumed (g/person/day)	1.15	14.19	2.44
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0183	0.2251	0.0386
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	54	4	25
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	97.06	65.84	88.13

Table 17. SITE 2 (New York Bight, October-November 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	919,522	72,352	355,499
	Catch Weight per Trip (lbs)	0.74	3.57	1.05
	Fish Consumed (g/person/day)	0.17	0.80	0.24
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0005	0.0022	0.0006
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
301-500	No. of Trips	18,109	56,453	528,526
	Catch Weight per Trip (lbs)	2.37	11.67	1.50
	Fish Consumed (g/person/day)	0.53	2.62	0.34
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0018	0.0087	0.0011
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	+	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	100.00	100.00
>500	No. of Trips	669,535	1,201,829	281,357
	Catch Weight per Trip (lbs)	0.84	3.39	13.19
	Fish Consumed (g/person/day)	0.19	0.76	2.96
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0062	0.0252	0.0981
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	+	39	10
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	100.00	94.61	53.50

Table 18. SITE 10 (New England, June 1985) recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
<300	No. of Trips	-	-	-
	Catch Weight per Trip (lbs)	-	-	-
	Fish Consumed (g/person/day)	-	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	-	-	17,452
	Catch Weight per Trip (lbs)	-	-	0.15
	Fish Consumed (g/person/day)	-	-	0.03
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	0.0001
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	100.00
>500	No. of Trips	-	68,761	157,066
	Catch Weight per Trip (lbs)	-	3.61	0.38
	Fish Consumed (g/person/day)	-	0.82	0.09
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	0.0168	0.0018
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	59	+
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	100.00	100.00

Table 19. **SITE 12 (New England, August 1985)** recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	88,456	-	-
	Catch Weight per Trip (lbs)	1.04	-	-
	Fish Consumed (g/person/day)	0.24	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	-	146,457	38,877
	Catch Weight per Trip (lbs)	-	1.74	33.97
	Fish Consumed (g/person/day)	-	0.40	7.73
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	0.0022	0.0432
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	+	23
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	100.00	86.84
>500	No. of Trips	-	-	391,808
	Catch Weight per Trip (lbs)	-	-	10.36
	Fish Consumed (g/person/day)	-	-	2.36
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	0.1886
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	5
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	50.56

Table 20. **SITE 1 (New England, October 1985)** recreational catch, consumption, and intake of PCBs, by mode of fishing on a length-stratum basis, for five-fish composite samples of bluefish collected during 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Lengths in millimeters (mm) fork length, catch weight per trip in pounds (lbs), fish consumed in grams of fish per person per day (g/person/day), and intake of PCBs in micrograms of PCBs per kilogram of mean body weight (60 kg) per day ($\mu\text{g}/\text{kg}/\text{day}$). One pound = 453.6 grams, a dash (-) = no data, and a plus (+) = exceeds maximum feasible number of trips in a two-month period (1 trip/day).

Length Strata (mm fork length)	Recreational Catch, Consumption, and Intake of PCBs Information	Mode of Fishing		
		Shore	Party and Charter Boat	Private and Rental Boat
≤300	No. of Trips	14,208	-	-
	Catch Weight per Trip (lbs)	0.16	-	-
	Fish Consumed (g/person/day)	0.04	-	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	-	-	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	-	-	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	-	-	-
301-500	No. of Trips	6,780	169,955	-
	Catch Weight per Trip (lbs)	9.68	8.61	-
	Fish Consumed (g/person/day)	2.20	1.96	-
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0345	0.0307	-
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	28	32	-
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	75.00	60.00	-
>500	No. of Trips	41,546	484,588	3,961
	Catch Weight per Trip (lbs)	9.79	12.49	104.87
	Fish Consumed (g/person/day)	2.23	2.84	23.86
	Intake of PCBs ($\mu\text{g}/\text{kg}/\text{day}$)	0.0380	0.0486	0.4077
	No. of Trips Equivalent to 1 $\mu\text{g}/\text{kg}/\text{day}$	26	20	2
	% of Consumers Within 1 $\mu\text{g}/\text{kg}/\text{day}$ Range	84.31	85.71	9.30

APPENDIX



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APPENDIX I
SURVEY METHODS AND MATERIALS

Statistical Design

Initially, it was deemed advisable to use stratification by length category within each site to be sampled because of extreme variability of PCB levels in the bluefish population and contaminant assessment within and among stratified length groupings. This decision was based on statistical considerations applied to a 1975-82 combined New Jersey and Massachusetts historical five-fish composite data set (Table 1). These length categories (strata) represent approximately equal intervals on a logarithmic scale as applied to existing population length frequency estimates.

To determine if the mean concentration of PCBs in bluefish exceeded the tolerance limit in each length stratum within each site, the following null hypothesis (H_0) and alternative hypothesis (H_A) were considered:

$$H_0: \mu \leq \mu_0$$

$$H_A: \mu > \mu_0$$

where μ = actual mean PCB level in a predetermined size stratum

μ_0 = 2 ppm FDA tolerance level

For this evaluation, the appropriate formula for calculating the required sample size (n) within each size stratum to test H_0 with specific levels of risk is as follows:

$$n = (\sigma (Z_{\alpha} + Z_{\beta}) / \Delta)^2$$

where: σ = actual standard deviation of the target population
(to be applied to the mean five-fish composite
distribution)

Δ = the preselected increment of interest by which
exceeds μ_0

α = probability of rejecting a true H_0

β = probability of accepting a false H_0 as a function
of Δ

Z_{α}, Z_{β} = specified percentiles of the standard normal
distribution

As an example of this calculation, let $\alpha = 5\%$, $\beta = 5\%$, $\Delta = 0.5$ ppm; and for
length stratum 1 (Table 1) where σ is estimated to be 0.73 ppm:

$$n = (0.73 (1.645 + 1.645) / 0.5)^2$$

$$= 23 \text{ five-fish composites}$$

Table 2 presents the required number of five-fish composites per length stratum
per sample site for various combinations of β and Δ with α fixed at the 5% level.

Acceptable significance and equal weight of concern were given to both the
Producer's Risk (α) and Consumer's Risk (β). Logistic constraints include
limitations on the number of fish that could be collected, transportation of the
estimated 30 to 40 thousand pounds of fish required, and processing capacity by

the participating laboratories within the designated time frame of the survey. The number of five-fish composite samples to be collected and analyzed, the size stratification of these samples, and the individual site allocation of the samples as calculated for $\alpha = \beta = 5\%$ and $\Delta = 1.0$ ppm were determined to be optimal within the purpose, scope, and constraints of the survey (Table 2).

The planned temporal and spatial allocation of sampling for the survey is given in Table 3. As indicated in this table, twelve seasonal collections (sites) were planned in five geographic areas, i.e., New England (MA, RI), New York Bight (CT, NY, NJ), Maryland - Virginia, North Carolina, and Florida (Atlantic Coast). All twelve sites were to be sampled for five-fish, randomly formed composites according to the sampling schedule selected from Table 3, with length stratification and random compositing within each stratum accomplished as outlined in Table 2.

In addition to the five-fish composites indicated above, 100 individual fish were to be collected from each of five sites (underlined in Table 3) and allocated to predetermined fork length categories as follows: 15, 30, and 55 fish in length strata 1, 2, and 3, respectively. These collections of individual fish and the subsequent resulting analyses for PCBs were undertaken primarily to examine cofactors (e.g., length, weight, sex, gonadal development, etc.) which might provide insight into the dynamics of PCB accumulation and distribution. Finally, a minimum of five 25-fish composites of young-of-the-year bluefish ("snappers") were to be collected and analyzed to represent the "pan fishery" which takes place primarily in the estuaries of the New York Bight during summer and early fall.

Bluefish were not available in Florida during the scheduled collection period; hence only 11 of the 12 planned sites were sampled for five-fish

composites. With this exception, the survey sampling design was met.

Collections

Samples were purchased at dockside, iced "in-the-round" (i.e., ungutted) as quickly as possible and transported to NMFS Sandy Hook Laboratory. Upon arrival, fish remained on ice or were refrigerated (not frozen) until examined for basic biological information (usually within 48 hours). Biological data collected were: length (total, fork, and standard); total body weight; sex and stage of maturity (where applicable); and weight of excised gonads. Scales were taken from all specimens and stored dry for subsequent mounting, microscopic analysis, and age and growth determinations. Mature ovaries were excised and fixed for future estimates of fecundity and determinations of spawning mode.

After biological examinations, specimens were fitted to length strata, appropriately coded and labeled, wrapped individually in aluminum foil, packed in appropriate containers, and frozen (-10 to -20°C). Individual fish samples were shipped frozen to NMFS Charleston Laboratory. Samples for five-fish composite analysis were transported frozen to FDA Buffalo Laboratory. Upon arrival, samples were placed in storage at -20 to -30°C to await chemical analysis, after first verifying that the samples were in good condition, i.e., solidly frozen and with packaging intact.

Chemical Analyses

Chemical analyses were conducted by using the protocol described in FDA's Pesticide Analytical Manual, Vol. I (PAM I), (FDA, 1968). The Data Report (NOAA/FDA/EPA, 1986) gave a step-by-step description of the analytical chemistry methodology used during the survey with appropriate references to PAM I, i.e.,

sample preparation, extraction, fat determination, petroleum ether-acetonitrile partitioning, Florisil column chromatography, gas liquid chromatographic determination, quality assurance procedures, and analytical hints.

Appropriate quality assurance was used throughout the survey and was documented in the Data Report. This included identity confirmation analyses, repeat analyses, repetitive analyses of portions of a specially prepared bluefish sample with biologically incurred PCB residues, analyses of bluefish samples fortified with Aroclors and organohalogen pesticides, and method reagent blank analyses.

LITERATURE CITED

FOOD AND DRUG ADMINISTRATION

1968 and Subsequent Revisions. Pesticide Analytical Manual Ed., 1968 and subsequent revisions. Food and Drug Administration, Washington, D.C. 20204

NATIONAL OCEANIC and ATMOSPHERIC ADMINISTRATION/FOOD AND DRUG

ADMINISTRATION/ENVIRONMENTAL PROTECTION AGENCY.

1986. Report on 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish - Data Report. National Oceanic and Atmospheric Administration, Food and Drug Administration, and Environmental Protection Agency, Washington, D.C., 179 p. NTIS Accession No. PB86 218070/AS. (Data tapes available from NTIS, Accession No. PB87-142 881.)

Table 1. Summary statistics for 1975-82 combined New Jersey and Massachusetts historical five-fish composite PCB data set. Length strata are in millimeters (mm) fork length and are based on equal intervals on a logarithmic scale, and PCB levels are expressed in parts per million.

Summary Statistics	Length Strata (mm fork length)		
	1 300 or less	2 301 - 500	3 501 or more
Sample Size (n)	6	14	20
PCB Mean (\bar{x})	0.72	0.68	2.64
PCB Standard Deviation (s)	0.73	0.73	2.44
PCB Range	0.00-1.70	0.27-2.44	0.42-8.28

Table 2. Approximate number of five-fish composites per sampling site per length strata for $\alpha = 5\%$ and various combinations of β and selected values of Δ under the Alternative Hypothesis (H_A). Minimum sample size per category was set at five. Lengths in millimeters (mm) fork length and an asterisk (*) indicates the selected design option for 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish.

Length Strata (mm fork length)	Combinations of β 's and Δ 's with $\alpha = 5\%$								
	$\beta = 5\%$			$\beta = 10\%$			$\beta = 20\%$		
	Δ			Δ			Δ		
	0.5	1.0*	1.5	0.5	1.0	1.5	0.5	1.0	1.5
1 300 or less	23	6	5	19	5	5	14	5	5
2 301-500	23	6	5	19	5	5	14	5	5
3 501 or more	258	65	29	204	51	23	148	37	5
Site Total	304	77	39	242	61	33	176	47	15

Table 3. Planned site allocation of five-fish composites (77/site) and individual fish (100/site) for 1984-86 Federal Survey of PCBs in Atlantic Coast Bluefish. Underlined "Seasons" indicate when sites were to be sampled for individual fish. In addition, at least five 25-fish composites of young-of-the-year ("snappers") were to be collected in the New York Bight during summer and fall.

Geographic Area	Seasons (sites)	No. of Composite Collections	No. of Individual Collections
New England	<u>Early Summer</u> , <u>Late Summer</u> , <u>Fall</u>	3	2
N.Y. Bight	<u>Spring-Early Summer</u> , <u>Late Summer</u> , <u>Fall</u>	3	2
Maryland- Virginia	Spring, Fall	2	0
North Carolina	<u>Early Winter</u> , <u>Late Winter</u> , Spring	3	1
Florida (Atlantic Coast)	Winter	1	0
Survey Total		12 ¹	5 ²

¹ Total composite samples (12 x 77) = 924

² Total individual samples (5 x 100) = 500

APPENDIX



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APPENDIX II
THE TOXICOLOGY, KINETICS AND METABOLISM OF PCBs IN FISHES,
WITH SPECIAL REFERENCE TO BLUEFISH, Pomatomus saltatrix

U.S. Environmental Protection Agency
Gulf Breeze Environmental Research Laboratory
Gulf Breeze, Florida

July, 1986



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EXECUTIVE SUMMARY

THE TOXICOLOGY, KINETICS AND METABOLISM OF PCBs IN FISHES WITH SPECIAL REFERENCE TO BLUEFISH, Pomatomus saltatrix

Polychlorinated biphenyls (PCBs) are ubiquitous contaminants in the global ecosystem. First synthesized in 1881, PCBs were used in industry beginning about 1930 in a variety of closed, open and dissipative uses, including those of plasticizers, heat-exchange fluids, capacitor fluids and carbonless carbon paper. It has been estimated that one million metric tons of PCBs were manufactured between 1930 and 1970.

The PCBs are highly persistent in the environment. Because of their physical-chemical properties, PCBs accumulate in freshwater and marine sediments and become distributed throughout aquatic ecosystems in the water column, on suspended sediments and in the biota. With a decline in the use of PCBs in uncontrolled and dissipative systems, the major source of PCBs to the human population has become the ingestion of seafood from PCB-contaminated waters. In states such as New York, New Jersey and Massachusetts, there have been commercial fisheries closures because of PCB contamination, and a variety of public health advisories have been published warning consumers against the consumption of eels, striped bass, white perch, bluefish and other species from certain waters.

PCBs accumulate in fishes and shellfish as the result of direct water uptake and transport through the food chain. Accumulation has been shown to conform to first-order kinetic models, and body burdens of PCBs in fishes and fisheries products may be estimated based upon equilibrium partitioning of PCB between environmental media and the tissues of fishes. Since the ultimate body burden of

PCB in a fish depends upon the magnitude and frequency of exposure as well as depuration of the compound, body burdens of PCBs in fishes may vary depending upon where the exposure occurs. Fishes in contaminated estuarine and coastal systems may accumulate high body burdens, whereas fishes from less contaminated, oceanic waters may accumulate lower burdens. Sophisticated models aimed at predicting PCB body burdens in fishes have been developed for a number of different aquatic ecosystems.

The greatest amount of data on PCB accumulation, retention and metabolism in fishes is based upon research with striped bass and rainbow trout. PCB data for bluefish are limited to several monitoring and survey studies carried out to ascertain PCB concentrations in commercial, recreational and scientific survey catches of the species. Most such data are available from the states of New York and New Jersey.

Bluefish from New York and New Jersey waters show wide variation in degree of PCB contamination. Concentrations in estuarine waters of the Hudson River, Newark Bay and Raritan Bay vary from below 1.0 part per million to greater than 5 parts per million. Samples taken in Massachusetts have shown PCBs as high as 16 parts per million in the edible flesh. Overall, bluefish from open ocean waters tend to have lower concentrations of PCBs, while samples taken within estuaries have higher levels. PCB contamination varies from year to year and site to site, however, and detecting a clear trend from the sparse data is difficult.

Assuming that the physiology of bluefish and the kinetics of PCBs in bluefish are similar to those of the striped bass and the rainbow trout, predictions of bluefish PCB burdens may be made. Such predictions suggest that, for bluefish feeding upon a PCB-contaminated diet in Atlantic coastal and

estuarine waters, concentrations of from 0.1 to 11.0 parts per million PCBs may be expected. Predicted concentrations would be affected most strongly by the concentrations of PCBs in food organisms, since bluefish spend most of their life cycle in waters with low concentrations of dissolved PCBs.

Like rainbow trout, bluefish probably do not metabolize PCBs to any appreciable extent. Reductions in bluefish PCB body burdens that occur from season to season are due to elimination of parent compound rather than metabolism of the PCB to more polar metabolites. Although the data are sparse, they would suggest that PCB burdens in bluefish will consist of PCB congeners with four or more chlorine substitutions. PCBs with a lesser degree of chlorine substitution will be eliminated. The data from studies with striped bass, rainbow trout and other species suggest that the congeners likely to accumulate in bluefish will be those with a high degree of chlorine substitution in ortho, ortho' positions, and will not be those identified as having a high potential for toxic effects.

More studies are required to determine patterns of PCB contamination in bluefish, and to determine the potential toxicity to man from specific groups of toxic congeners. Such studies are currently underway within a number of state and federal regulatory agencies.

THE TOXICOLOGY, KINETICS AND METABOLISM OF PCBs IN FISHES,
WITH SPECIAL REFERENCE TO BLUEFISH, Pomatomus saltatrix

INTRODUCTION

General Information Concerning Bluefish and "Similar Species"

This report is a summary of the accumulation, metabolism and effects of PCBs in fishes, emphasizing bluefish (Pomatomus saltatrix) and other, similar, marine or freshwater fish species. This report provides information on the problem of PCBs in commercially and recreationally important fish species, as well as a scientific background for discussions related to developing, implementing and enforcing regulations intended to deal with the problems of PCBs in marine fishes, including bluefish. Any discussion of PCB dynamics in bluefish must be qualified, in that the data for PCBs in bluefish are entirely monitoring data describing PCB concentrations in recreational, commercial and scientific survey catches of the species. We know of no experimental data providing information on bioaccumulation or pharmacokinetics of PCBs in bluefish.

The bluefish of the world are described as a single species, Pomatomus saltatrix, the only species in the family Pomatomidae. Bluefish occur in most of the temperate coastal regions of the world, although they have been erroneously reported as occurring in the Eastern Pacific (Briggs, 1960; Grosslein and Azarovitz, 1982). Along the eastern coast of the U.S., bluefish occur in continental shelf waters (Figure 1). Spawning occurs during two distinct periods: (1) during the spring and summer, and (2) in the late fall in waters between the continental slope and the coast (Breder and Rosen, 1966; Kendall and Walford, 1979; Grosslein and Azarovitz, 1982). Spawning occurs in the open sea (Norcross et al., 1974), and juveniles move from the open ocean into coastal waters and estuaries during the mid- to late-summer months (Bigelow and Schroeder, 1953;

Kendall and Walford, 1979). Bluefish populations of the Atlantic coast are highly dependent upon estuaries as nurseries.

Bluefish are migratory, pelagic predators (Grosslein and Azarovitz, 1982), acting as secondary or tertiary carnivores in the food web of coastal and estuarine waters. They feed on a wide variety of fishes and invertebrates (Bigelow and Schroeder, 1953). Stomach content analysis of young-of-the-year bluefish in the Hudson estuary (O'Connor, personal observation) showed that they feed on amphipods, small crabs and small fish, including young-of-the-year striped bass (Morone saxatilis) and white perch (M. americana).

Bluefish are the primary recreational fish species in the waters of New York and New Jersey. About 23,000 metric tons were taken in the New York Bight in 1970 (Deuel, 1973), and bluefish ranked first among recreational marine fishes in the United States (Grosslein and Azarovitz, 1982). The charter-boat fishery for bluefish is a multi-million dollar annual industry in New Jersey and New York. In recent years this industry has suffered substantial losses because of reports of PCB contamination in bluefish, and the subsequent public health advisories concerning the consumption of bluefish (Belton et al., 1983, 1985).

Since there are so few data regarding the dynamics of PCBs in bluefish, the inferences to be drawn in this report will be based upon data from other fish species for which abundant data may be found. We shall rely heavily upon data from experiments with striped bass, a species which, like the bluefish, undertakes migrations in the marine ecosystem, is dependent upon estuaries for development of the young, and which is a secondary or tertiary carnivore in marine and estuarine ecosystems (Bigelow and Schroeder, 1953; O'Connor, 1984a). Like bluefish, striped bass contain large amounts of body lipid; depot fat is stored intramuscularly as well as in mesenteric fat bodies. PCB exposure is

likely to be similar for the two species, especially during the estuarine-dependent, juvenile stages when their geographic distributions overlap and when the two species are part of the same summer estuarine food web.

Additional information to be applied to the question of PCB dynamics in bluefish will be data derived from studies with salmonid fishes; rainbow trout (Salmo gairdneri) and lake trout (Salvelinus namaycush). Both species are predators with a high proportion of body fat, and their responses to PCBs are well known from experimental and field studies. In fact, more is known about the kinetics and metabolism of PCBs in rainbow trout than for any other fish species (Lech and Peterson, 1983).

Data about PCBs in other species will be considered as a secondary source; most PCB studies dealing with marine, freshwater or estuarine fishes other than those listed above have been monitoring or survey studies, or studies providing few data on PCB kinetics and dynamics.

This report follows a format in which the physical, physiological and metabolic processes of PCB accumulation, retention and elimination are addressed individually, along with a discussion of predictive models that are currently available for use in determining PCB accumulation in fishes exposed to PCBs. It is not intended to be exhaustive in detail of PCB accumulation, metabolism or effects; rather, it is intended as an elucidation of those principles that must be accounted for in any program assessing the problem of PCBs in fishes in general and bluefish in particular.

General Facts Regarding Environmental Distribution of PCBs

Polychlorinated biphenyls (PCBs) are substituted derivatives of the biphenyl molecule (Figure 2), in which one or more hydrogen atoms have been replaced by chlorine. PCBs comprise a class of 209 isomers of the chlorobiphenyl molecule,

each having unique chemical and physical characteristics (Hutzinger et al., 1974; Mullin et al., 1984). PCBs were known more than 100 years ago. They were first synthesized by Schmidt and Schultz in 1881 (Subcommittee on Health Effects, DHEW, 1976), and were first prepared commercially in 1929. PCBs were used in industrial operations beginning about 1930 (Hubbard, 1964). Because of their thermal stability, viscosity, low vapor pressure, low solubility, resistance to oxidation, excellent dielectric properties and other characteristics, PCBs rapidly were employed in many industrial applications (Monsanto, 1978). PCBs are or had been produced in a number of industrial countries, although in recent years their production and use has declined precipitously (Nelson, 1972; Monsanto, 1978; Richardson and Waid, 1982).

PCBs are made by the reaction of biphenyl with anhydrous chlorine in the presence of a catalyst (iron filings or ferric chloride). The product of the reaction is a mixture of congeners, with the proportion of chlorination dependent upon the duration of the reaction. The industrial product, formerly marketed in the U.S. by the Monsanto Corporation under the trade name "Aroclor," consisted of different PCB mixtures, each named according to the percent chlorination of the product. Thus, Aroclor 1221 consisted of a mixture of chlorobiphenyls with about 21% chlorine, whereas Aroclor 1254 was a chlorobiphenyl mixture with about 54% chlorine.

PCB use has been widespread since their appearance in industry. Richardson and Waid (1982) classified useage categories as "controlled closed systems," "uncontrolled closed systems," and "dissipative." The many uses of PCBs included dielectric fluids, heat-transfer fluids, hydraulic fluids, plasticizers, sealants, and the manufacture of "kiss-proof" lipstick (Richardson and Waid, 1982; Hutzinger et al., 1974). Between 1930 and 1970 PCB production in the U.S.

was estimated at 500,000 metric tons (Nisbet and Sarofim, 1972; Hutzinger et al., 1974). Worldwide PCB production was estimated to be double the U.S. production (Richardson and Waid, 1982).

Ecological History and Significance of PCBs

Entry of PCBs into the environment occurred as the result of dissipative uses, as well as from controlled and uncontrolled, closed systems (Hutzinger et al., 1974; Nisbet and Sarofim, 1972; Richardson and Waid, 1982). Major routes of entry of PCBs to the environment include vaporization, leaks and disposal of PCB-contaminated fluids, and disposal of PCB-containing products at dumps and in landfills. A flow chart for evaluating various routes of PCB transport in the global ecosystem is given in Figure 3 (from Nisbet and Sarofim, 1972).

The first report of PCBs in the environment appeared in 1966, when compounds causing confounding peaks in chromatograms of DDT in fish samples were identified as polychlorinated biphenyl (Jensen, 1966). Within a short time, the presence of PCBs in all compartments of the global environment was established, and the current distribution of PCBs in the environment may be said to be "ubiquitous" (Risebrough et al., 1968; Koeman and Stasse-Wolthus, 1978; Wasserman et al., 1979). Although some evidence exists for photodegradation and microbial metabolism of some PCB congeners, PCBs will most likely be present and will recycle in the natural environment for many years (National Academy of Sciences [NAS]), 1979).

Problems associated with PCBs in the environment are (1) the PCBs are known to be acutely and chronically toxic to natural populations of animals, and (2) animals used as food by the human population may serve as a vector for the transport of PCBs from the environment to man (Hansen et al., 1971; Nimmo et al., 1971a, 1971b; Hutzinger et al., 1974; Walker, 1976; Mayer et al., 1977; Wasserman

et al., 1979; Belton et al., 1983, 1985). Evaluation of PCB transport in the environment shows that the major route for PCB transport to man is the ingestion of finfish and shellfish caught in PCB-contaminated systems (Nisbet and Sarofim, 1972; Jelinek and Corneiusen, 1976; Swain, 1983; Sloan et al., 1984; Belton et al., 1985). Worldwide use of PCBs coupled with transport and recycling via the atmosphere and surface waters has led to the present situation in which PCBs may be found in virtually any environment, and PCBs may be accumulated in virtually any species of finfish and shellfish used as food (Wasserman et al., 1979; National Academy of Sciences (NAS), 1979; Richardson and Waid, 1982).

Despite laboratory evidence describing PCBs as highly toxic at low concentrations (Hansen et al., 1971; Couch and Nimmo, 1974; Mayer et al., 1977; Califano, 1981), there are few published data showing evidence of ecological effects due to PCBs in natural systems. However, some studies provide evidence that PCB effects in natural systems may be subtle and difficult to isolate from the effects of other environmental contaminants. Mehrle et al. (1982) measured several parameters of skeletal strength in striped bass from estuarine systems on the east coast. They related weakness in vertebral columns to ambient concentrations of PCB in the estuaries; Hudson River bass were found to have the weakest vertebral columns, whereas bass from other systems were significantly stronger. PCBs also have been found to induce mixed-function oxidase activity (MFO; cytochrome P-448/P-450 system) in fishes (Addison et al., 1978, 1979). In complex environments subject to discharges of many different pollutants evidence for increased MFO activity cannot be attributed to PCBs alone. We know of no data demonstrating that PCBs in natural environments are the direct cause of chronic or acute toxicity, and we know of no data showing a relationship between body burden of PCBs and lesions in natural populations of animals.

Lack of evidence demonstrating environmental or ecological impact by PCBs is not proof that PCBs are toxicologically "benign." The tendency for aquatic and terrestrial organisms to accumulate PCBs from environmental media and evidence relating PCB exposure to definable lesions in animal tests (e.g. Lipsky et al., 1978; Klaunig et al., 1979) dictates that the question of PCB environmental impacts continue to be studied in depth. This is necessary so that any adverse impacts that might occur because PCBs are present in natural environments can be identified, characterized and, if possible, eliminated (NAS, 1979).

Toxicological History and Significance of PCBs

The PCBs are listed as animal carcinogens (IARC, 1974, 1978), and as hazardous materials, hazardous waste constituents and priority toxic pollutants by the U.S. EPA (Sittig, 1985). The tissues affected by PCBs are the skin (chloracne), the eyes and the liver. PCBs also cause typical lesions of the thyroid, stomach and lymphoid organs (Klaunig et al., 1979; Sleight, 1983). In many cases the effects of PCBs on animal tissues are indistinguishable from those caused by other chlorinated hydrocarbons such as DDT, dibenzodioxins and dibenzofurans. Certain of the chlorobiphenyls may cause liver tumors in mice and rats after prolonged exposure (IARC, 1974, 1978). Recent data show that the PCBs function more as cancer promoters than as carcinogens (Kolbye and Carr, 1984), and controversy still surrounds the interpretation of the original data used to establish the carcinogenicity of the PCBs (Kimbrough et al., 1975).

Early data on PCB toxicology and pathology were published by Schwartz (1936), who reported skin lesions and systemic poisoning among workers reported to have inhaled PCBs. The skin lesion characteristic of PCBs and other chlorinated hydrocarbons has come to be described as "chloracne." Toxicological and public health interests in PCBs were increased in 1968 with the occurrence of

the "Yusho incident" (Okumura and Katsuki, 1969), in which more than 1600 Japanese ingested rice oil contaminated with 2,000 parts per million (ug/g; ppm) of PCBs (Kanechlor 400) from a heat exchanger. Symptoms of Yusho included chloracne, hyperpigmentation of the skin, eye discharge, weakness, numbness and disturbances in liver function. Subsequent analysis of samples from Yusho suggest that the rice oil was contaminated with high concentrations of dibenzofurans as well as with PCBs (Kuratsune et al., 1976); it would appear that the symptoms of Yusho were the result of exposure to more than a single contaminant, and that PCBs alone were not responsible for the full range of biological and biochemical effects observed in Yusho.

PCB toxicity has been tested in vivo and in vitro using many species, including several phyla of invertebrates and many vertebrates such as fishes, birds, rodents, and non-human primates. Epidemiological data are available on the effects of PCBs on humans in several instances of industrial exposure to PCBs (Wasserman et al., 1979). One of the first indications that PCBs had the potential to cause severe health effects in mammals was the determination that reproductive failure among ranch mink fed Great Lakes fish was due to PCBs in the fish used as food, and that mink were highly susceptible to PCB toxicity (Hartsbrough, 1965; Ringer, 1983). Subsequent studies in primates by Allen and co-workers showed that low concentrations of PCBs caused irregular menstrual cycles, early abortions and stillbirths among Rhesus macaques (Allen et al., 1973, 1974; Allen and Norback, 1976).

Because of the potential for PCBs to cause health effects in humans, the U.S. Food and Drug Administration (FDA) between 1969 and 1971 established temporary tolerance levels for PCBs in food products. Effective April 1, 1981, the FDA Tolerance Limit for PCBs in foods included milk (1.5 ppm on a fat basis),

poultry (3.0 ppm fat basis) animal feed (2.0 ppm), packaging materials (10.0 ppm) and fish and shellfish (2.0 ppm) (Hoeting, 1983). Litigation initiated by the National Fishermen's Association delayed a final ruling on the Tolerance Limit for PCBs in fish until 1984. A 5.0 ppm Temporary Tolerance Limit for total PCBs in fish and shellfish was in force from 1981 through 1984. In 1984 the limit was reduced to the present value of 2.0 ppm.

The concern over PCB levels in fish and shellfish reflects the fact that fish are an important link in the food-chain leading to man, and that the consumption of PCB-contaminated fish is one of the major routes for the transport of PCBs from the environment to the human population (Hutzinger et al., 1974; NAS, 1979; Swain, 1983; Belton et al., 1983, 1985). In recent studies of PCBs in human milk, Schwartz et al. (1983) determined that fish eaters in the Great Lakes region had higher concentrations of PCBs, even among groups consuming only six to 12 fish meals per year. Although recent investigations have shown that the major toxicological effects of PCBs are due to specific, individual congeners (Safe, 1984), present regulations regarding the allowable limits of PCBs in foods such as fish and shellfish are based upon total PCB concentrations (Horn and Skinner, 1985; Belton et al., 1983, 1985).

The Special Problem of PCBs in Fishes and Other Aquatic Organisms

The persistence of PCBs in the environment leads, ultimately, to their transport to and deposition in lakes, rivers, estuaries and oceanic waters. In addition to domestic and industrial waste water disposal serving as local sources for PCBs, atmospheric transport assures that surface waters around the globe will serve as environmental sinks for PCBs and as a source of PCB contamination to all environmental compartments (Nisbet and Sarofim, 1972; Fuller et al., 1976; NAS, 1979; Wasserman et al., 1979). Because PCBs are partially soluble in water, and

because they tend to partition to fine particulate matter, organic matter and lipids, PCBs in aquatic systems are available to aquatic biota through several routes, including bioconcentration from water, accumulation from sedimentary deposits and transport through the food chain (Hamelink et al., 1971; Hutzinger et al., 1974; Branson et al., 1975; Pizza and O'Connor, 1983; Rubinstein et al., 1983, 1984). Once accumulated, PCBs partition to depot lipids where they have a long half-life. Those organisms serving as food sources for other organisms in the aquatic food-web may function effectively as vectors for PCB transport in aquatic systems (Thomann and Connolly, 1984; O'Connor and Pizza, in press, a). Food chain transport appears to be the major source of PCB contamination for many species of fish, including many that are food resources for the human population (Thomann and Connolly, 1984; O'Connor, 1984; Rubinstein et al., 1984; O'Connor and Pizza, in press, a; O'Connor and Huggett, in press).

In highly contaminated aquatic ecosystems, PCBs may accumulate to very high concentrations in sediments and in fishes. In the Great Lakes, for instance, PCB concentrations in many commercial and sport fishes may exceed the 2.0 ppm FDA Tolerance Limit (Cordle et al., 1982; Schwartz, 1983). In some East coast estuaries, such as the Hudson River, Raritan Bay, New York Harbor and New Bedford Harbor, industrial and domestic sources of PCBs have led to the contamination of many fisheries resources such as eels (Anguilla rostrata), striped bass, bluefish and blue crabs (Callinectes sapidus) (Sloan and Armstrong, 1982; Belton et al., 1983, 1985; Weaver, 1984). In several instances public health advisories concerning the consumption of PCB-contaminated fisheries products have been issued. In the New York metropolitan area, as well as in New Bedford, Massachusetts, certain commercial fisheries have been closed or restricted (Belton et al., 1985; Horn and Skinner, 1985). In 1976 Jelinek and Corneliussen

reported that "...the occurrence of PCBs [in the diet] has narrowed to the point where [fish] are now the primary sources of PCBs [to humans]." In certain sections of the country such as the Great Lakes States, metropolitan New York and New Bedford, evaluations of the potential effects of PCBs in the seafood consumed by humans have led to serious concern (Swain, 1983; Belton et al., 1985).

Unlike the problem of PCB contamination in foodstuffs such as eggs, milk and meat, contamination of seafood with PCBs is an ecological problem, rather than a problem of monitoring contaminated sources of animal feeds. In New Bedford, Massachusetts, for example, the discharge of PCBs from industrial sites has led to the contamination of fisheries in and adjacent to New Bedford Harbor. The question of PCB transport from the Harbor system to the fishing grounds adjacent to the Harbor is being addressed; however, it would appear that migration of finfish and shellfish into and out of the Harbor and Buzzards Bay results in PCB contamination of northern lobster (Homarus americanus), winter flounder (Pseudopleuronectes americanus) and other species. This leads to a lack of confidence in the suitability for human consumption of fishes that are caught in the region (Weaver, 1984).

Fishes from the Hudson River, the Hudson estuary, New York Harbor and adjacent oceanic regions are, likewise, contaminated with PCBs (Nadeau and Davis, 1976; Cahn et al., 1977; Spagnoli and Skinner, 1977; Sherwood et al., 1978; Stainken and Rollwagen, 1979; Armstrong and Sloan, 1980, 1982; O'Connor, 1982, 1984a, 1984b; O'Connor et al., 1982; Thomann, 1981; Sloan et al., 1983; Belton et al., 1983, 1985; Brown et al., 1985; Samuelian et al. in review). A summary of PCB concentrations in some important fishery resources was provided in O'Connor et al. (1982) and by O'Connor and Pizza (in press, a). The major source of PCBs to the Upper Hudson River, and a significant contribution of the pollutant to the

estuary, was determined to be an industrial discharge (Bopp, 1979; Bopp et al., 1981, 1984). Although that source has been controlled (Horn et al., 1979), there remain some 200 to 300 metric tons of PCBs still in the process of transport from upstream sites to New York Harbor and adjacent coastal waters (Schroeder and Barnes, 1983). In New York City PCBs are still discharged with domestic wastewater (MacLeod et al., 1981; Mueller et al., 1982). Although PCBs in fishes from the Hudson estuary have declined since elimination of the major upstream source (Sloan and Armstrong, 1982; Sloan et al., 1983; Brown et al., 1985), downstream transport and continued discharge of PCBs to the system from wastewater sources maintain body burdens of PCBs in Hudson River fishes above the FDA 2.0 ppm Tolerance Limit (Horn and Skinner, 1985; Brown et al., 1985).

PCB contamination of fishes and seafood is not confined to systems with large PCB inputs, nor is it restricted to species resident in enclosed or semi-enclosed systems such as rivers, lakes and estuaries. Rather, transport processes, physical partitioning in the environment and food-chain transport of PCBs in the global ecosystem have resulted in measureable concentrations of PCBs in many ecosystems and resources, including the coastal oceans, deep oceans, and remote areas (Risebrough et al., 1968; Richardson and Waid, 1982; GESAMP, 1984; Stegeman et al., 1986).

PCB Accumulation in Fishes

It has long been known that marine organisms, particularly fishes, concentrated certain elements and compounds in their flesh to concentrations greater than those in the environment. The phenomenon is referred to as "bioconcentration" or "bioaccumulation". Such phenomena were described in studies of radionuclides in marine organisms (Lowman, 1971), and in bioaccumulation studies of DDT transport in the Flax Pond ecosystem by Woodwell et al. (1967).

Three different processes may operate when an organism accumulates an environmental contaminant to concentrations greater than those in the ambient environment. The terms describing these processes-bioconcentration, bioaccumulation and biomagnification-were clarified by Brungs and Mount (1978) and Macek et al. (1979) as follows:

Bioconcentration ... the process whereby substances enter aquatic organisms through the gills or other respiratory epithelia directly from water;

Bioaccumulation ... the overall accumulation of a chemical substance from the water, and any other process leading to the accumulation of the substance, including dietary uptake;

Biomagnification ... a process whereby concentrations of accumulated materials increase as these materials pass up the food chain through two or more trophic levels.

In this section of the report we review the current state of knowledge regarding the bioavailability of PCBs in the environment, and the two major mechanisms associated with assimilation of PCBs into the body of fishes from environmental sources; assimilation from water, and assimilation from food.

Bioavailability of PCBs to Fishes

Critical to an understanding of PCB assimilation by fishes is an understanding of the extent to which PCBs in various environmental sinks are "bioavailable"; i.e., exist in a state in which they can enter and be retained by an organism. PCBs that are dissolved in the water may be completely bioavailable. That is, if an organism were to irrigate the gills with water containing dissolved PCBs, or if the organism were to ingest water containing PCBs, they would be assimilated with high efficiency. PCBs that are associated with

sediments, however, are less "bioavailable". Due to their strong tendency to sorb to particulate matter, PCBs associated with either deposited or suspended sediments would, upon breathing or ingestion, be assimilated with a lower degree of efficiency, as determined by partitioning between the particulate matter and the lipids of the organism. PCBs in food items are unavailable to the ingesting organism unless and until the food organism is consumed, at which point a number of factors regarding food conversion efficiency, digestive processes and cross-gut transport phenomena come into play, each with the potential to affect bioavailability of PCBs in food. Although physical processes dictate that some of the PCB in sedimentary deposits may become available through the water column, and that some of the PCB dissolved in water will become adsorbed to sedimentary material, PCBs in organisms tend to remain stable, and can only become available after ingestion.

Availability of PCBs From Water

Although PCBs are only "sparingly soluble" in water (Hutzinger et al., 1974; Haque et al., 1974), PCBs dissolved in the water column may be assumed to be completely available to fishes by the process of equilibrium partitioning (Pavlou and Dexter, 1979; McKim and Heath, 1983). The best measure of the direction and magnitude of equilibrium partitioning for non-polar materials such as PCBs is the octanol-water partition coefficient, a measure of the tendency for the chemical (in this case, PCBs) to dissolve in a non-polar solvent (e.g. n-octanol), as opposed to the highly polar solvent, water (Karickhoff et al., 1979). Octanol-water partition coefficients are often expressed as log values (Log K_{ow}). Although the tissues of fish are not directly equivalent to an organic solvent in their tendency to accumulate PCB from a water solution, octanol-water partition coefficient of organic contaminants such as PCBs and the tendency for fishes to

bioconcentrate such compounds are directly correlated (Hamelink et al., 1971; Neely et al., 1974; Spacie and Hamelink, 1982; Mackay and Hughes, 1984).

The bioconcentration factor (BCF) is a measure of the tendency for an organism to accumulate a substance from the water. The BCF for PCBs has been calculated for many species, and two different estimators of bioconcentration have been proposed. In the first, BCF is expressed as the concentration of PCB attained in the tissues of the fish at equilibrium or steady-state, divided by the concentration of the PCB in exposure water (Hamelink et al., 1971). The second measure of BCF, proposed by Branson (Branson et al., 1975), employed a kinetic definition, i.e., the BCF was stated to be the ratio of the assimilation rate constant for the compound moving into the fish (k_1), divided by the measured elimination rate constant (k_2). Branson's measure was directed primarily toward evaluating the BCF at steady-state, based upon a short-term test (less than 15 days).

Published data for BCF values among fishes exposed to PCBs vary (Spacie and Hamelink, 1982; Mackay, 1982); however, they generally fall into a narrow range, between 1×10^4 to 5×10^5 (NAS, 1979; O'Connor and Pizza, in press, a; Mackay and Hughes, 1984). Values for a number of freshwater and marine species are presented in Table 1.

As originally proposed, the calculation of BCF was used to determine, from water concentration data, what the probable burden of PCBs might be in fishes exposed to a contaminated environment. The original experimental work and evaluation of the technique showed great promise in that the use of the BCF-based calculations provided a reasonably accurate estimate of the actual PCB body burden observed in fishes in the environment (usually within a factor of from 3 to 5; Clayton et al., 1977; Pavlou and Dexter, 1979; Mackay, 1982; Shaw and

Connell, 1984; O'Connor and Pizza, in press, a). However, as observed by Spacie and Hamelink (1982) and Shaw and Connell (1984), such a range of error is unsatisfactory when applied to questions of regulation and environmental impact, in that it is not accurate enough to predict PCB concentrations as being above or below current FDA Tolerance Limits. O'Connor and Pizza (in press, a), using accepted BCF values, calculated probable PCB burdens in a number of fishes from the New York Bight region, including bluefish. They found that in all cases, observed PCB concentrations in fishes from the Bight region were in excess of that calculated by using a BCF of 1×10^4 (Table 2). The probable reasons for such discrepancies are:

- 1) not all the "dissolved" PCB in the aqueous medium is bioavailable;
- 2) fishes do not retain all the PCB assimilated by bioconcentration for a long period of time; and
- 3) fishes accumulate a significant portion of their PCB body burden from sources other than direct uptake from water (Norstrom et al., 1976; Shaw and Connell, 1984; Thomann and Connolly, 1984; O'Connor and Pizza, in press, a).

The subject of bioaccumulation of PCBs from dietary sources will be addressed in a subsequent section of this report.

As noted at the beginning of this report, the PCBs are a family of 209 compounds. Based upon differences in physical and chemical characteristics, each isomer may have different modes of behavior in the environment (Mullin et al., 1984; Oliver and Niimi, 1985). Since different PCBs have different solubilities and octanol-water partition coefficients, it may be expected that different isomers would be bioconcentrated from the environment with different levels of efficiency.

Early studies with different commercial mixtures of PCBs showed that the more highly chlorinated PCBs had a tendency for greater bioconcentration from water (Metcalf et al., 1975; Mayer et al., 1977). Other authors have reasoned that PCBs with greater numbers of chlorines (i.e. up to 6 or 7) should be bioaccumulated in fish to a greater extent, primarily as the result of increased lipophilicity of such molecules (Mackay, 1982; Mackay and Hughes, 1984). As a result of such partitioning, the distribution of PCB congeners in natural populations of fishes may resemble PCB mixtures more similar to the higher chlorinated industrial preparations (e.g., Aroclor 1254; Aroclor 1260) than commercial mixtures containing a mixture of congeners with fewer chlorines. However, the differential accumulation of lower- and higher-chlorinated PCB isomers has been difficult to demonstrate in natural populations of fishes. Karickhoff (1979), Spacie and Hamelink (1982) and Niimi and Oliver (1983) have noted that the congener distributions of PCBs in the bodies of feral fishes are determined more by the kinetics of elimination than by assimilation. The reason for this is that differences in partitioning between water and tissue for different PCB congeners are so small as to be trivial, whereas differences in the structure of PCB congeners with identical K_{ow} 's may be sufficient to lead to measureable differences in rates of elimination or metabolism (Bruggeman et al., 1981; Spacie and Hamelink, 1982; Shaw and Connell, 1984; Oliver and Niimi, 1985).

Availability of PCBs from Food

Some of the earliest research on PCB accumulation in fishes was directed toward defining bioconcentration (Hansen et al., 1971; Hamelink et al., 1971). However, it was well known at the time that PCBs and compounds with similar physical-chemical characteristics were assimilable from the food (Johansson et al., 1972) and were transferred from predator to prey in the food-chain (Isaacs,

1973; Lieb et al., 1974; Krzeminski et al., 1977; Young, 1984).

Whether fishes accumulate PCBs primarily from water or primarily from the food may be academic since the final outcome of the contaminant uptake process is the same, regardless of the source of the contaminant (Pizza, 1983; O'Connor and Pizza, in press, b). However, from the point of view of environmental fate and transport, ecosystem modeling and regulatory decision-making, the distinction is quite important. If, on the one hand, PCBs in fishes derive primarily from the water column, efforts to understand the fate of PCBs in the environment and in fishes can be simplified and directed at straightforward problems of aqueous transport, partitioning and bioconcentration (Mackay and Hughes, 1984; Shaw and Connell, 1984). Models predictive of bioconcentration may be based upon basic environmental parameters such as water concentration, exposure frequency and gill transport (Califano, 1981; Califano et al., 1982; Brown et al., 1982; McKim and Heath, 1983; Mancini, 1983). An aggressive program designed to limit discharge of PCB to surface waters may be implemented as a means for solving the contamination problem (Hetling et al., 1979; Horn and Skinner, 1985). If, on the other hand, PCB transport occurs primarily via the diet, PCB sinks in the sediments and the biota serve as the primary sources for maintaining body burdens in fishes (Rubinstein et al., 1983, 1984; O'Connor and Pizza, in press, a; Connolly and Winfield, 1984), and control of PCB contamination through regulation of waste and wastewater discharges may be much more difficult. More important, if PCBs are accumulated primarily from dietary sources, fishes will retain higher body burdens than might occur from water exposure alone, especially if the species in question feed primarily upon benthic organisms.

Studies of food-chain transport of PCBs in fishes were first conducted in 1973 and 1974 (Metcalf et al., 1975), although the potential for PCBs to be

"magnified" in food chains was noted as early as 1966 and 1968 (Jensen, 1966; Risebrough et al., 1968; Duke et al., 1971). Initial food chain studies (Metcalf et al., 1975) demonstrated both the persistence and transport potential of PCBs in aquatic food chains. In a subsequent food-chain study, Scura and Theilacker (1977) attempted to discern the relative importance of food and water uptake of PCBs; they concluded that there was no evidence of a "food-chain-phenomenon" in a three-tiered laboratory model ecosystem, but that PCB transport was due to equilibrium partitioning. Essentially the same conclusion was reached by Clayton et al. (1977) and Pavlou and Dexter (1979) based upon data from field monitoring studies in Puget Sound.

Food-chain studies in natural and model ecosystems, however, could not provide the degree of resolution needed to ascertain whether dietary PCB transport was an important phenomenon. Since all PCB transport may be assumed to occur due to equilibrium partitioning between source and tissue regardless of the pathway, investigators had to tolerate analytical limitations in the determination of which PCBs derived from which sources.

Beginning in the early 1970's, investigators employed radiotracers in the analysis of PCB transport from food to fishes. Hansen et al. (1976) reported an efficient dietary uptake of the components of Aroclor 1242 in channel catfish (Ictalurus punctatus), as well as differential retention of PCB congeners. Mayer et al. (1977) reported on the magnitude of dietary uptake of PCB by channel catfish. They showed that the dietary accumulation of PCB by catfish increased with the degree of chlorination and that higher chlorinated congeners in Aroclor 1260 accumulated to levels two times that of the congeners in Aroclor 1232.

Mitchell et al. (1977) reported efficient and rapid transport of dietary 14-C PCB in the codfish (Gadus morhua). PCBs were detectable in all tissues of the

codfish within two hours after administration of a single dose. Following long periods of exposure to a PCB-contaminated diet, Sangalang et al. (1981) detected high concentrations of Aroclor 1254 in testes and livers of codfish.

O'Connor (1982) reported widely varying PCB concentrations in larval striped bass taken at different locations in the Hudson estuary. Investigations by Califano (1981) and by Westin et al. (1985) showed that PCBs in early, non-feeding larvae were determined by PCBs passed from the female in the yolk of the egg. Westin et al. (1985) determined that feeding larval stages assimilated PCBs from food with high efficiency, but only if the larvae had low concentrations of PCBs to begin with. Larval body burdens of PCBs increased in proportion to the amount of the contaminant in the food source.

Califano (1981) performed comparative studies of PCB accumulation from food and water in young-of-the-year striped bass by using ¹⁴C-labeled Aroclor 1254. He showed that PCB uptake from food and water was important, but that uptake from food accounted for more than half the body burden accumulated during 48-hour exposures. Based upon experimentally-determined bioaccumulation factors (BAF) for young-of-the-year striped bass, Pizza and O'Connor (1983) estimated that between 55% and 83% of the PCB burden of Hudson River resident striped bass derived from dietary sources.

Pizza (1983 unpublished data) studied PCB accumulation from food in striped bass, spot (Leiostomus xanthurus), white perch and winter flounder (Pseudopleuronectes americana) and determined the following:

- 1) PCBs are accumulated from the food with an efficiency of 85% to 95 %;
- 2) Dietary PCBs accumulate rapidly to high tissue concentrations; and
- 3) The relative contribution of dietary PCBs to body burdens in all these species ranged from about 50% to more than 80% in the environment.

It is important to note, however, that high assimilation efficiency does not always lead to the accumulation of high body burdens. Among species with little body fat (e.g. flounder), PCB accumulations are generally low; the major proportion of the dietary dose may be eliminated during a short period of time (6 to 12 hours) following transport across the wall of the gut (Pizza, 1983; O'Connor and Pizza, in press, b).

Essentially the same conclusions have been reached in studies with freshwater fishes. Following acute dietary exposure of rainbow trout and yellow perch (Perca flavescens) to PCBs, concentrations increased rapidly in all tissues (Guiney and Peterson, 1980). Pizza (1983) calculated the assimilation efficiencies associated with these experiments to be from 80 to 90%. When yellow perch were given a single, oral dose of ^{14}C -labeled 2,5,2',5'-tetrachlorobiphenyl, they retained about 85% of the 800 ng administered; 15% of the total body burden was determined to be in the muscle tissue (Guiney and Peterson, 1980).

Niimi and Oliver (1983) fed rainbow trout mixtures of 80 PCB congeners in a single dose and determined assimilation efficiencies of from 62% to 85%; they detected no trend in assimilation efficiency among the congeners, a fact consistent with the notion that bioaccumulation of PCBs is more dependent upon elimination rate constant than upon congener-specific efficiency of assimilation (O'Connor and Pizza, in press, a).

Among rainbow trout reared on PCB-contaminated diets, Hilton et al. (1983) showed that contaminant accumulation was in direct proportion to dietary exposure, and did not appear to reach a "steady-state". Evaluation of the study by Lieb et al. (1974) in which rainbow trout were fed a PCB-contaminated diet for 32 weeks shows an apparent approach to steady state, at least in terms of

concentration. Lech and Peterson (1983) point out, however, that when growth is factored into experiment, there occurred a continual increase in the total mass of PCB accumulated by the fish. Most important, these studies show that PCBs in rainbow trout have a very long half-life, in excess of 200 days (Niimi and Oliver, 1983). Mayer et al. (1977) exposed coho salmon (Onchorhynchus kisutch), to Aroclor 1254 in the diet for periods up to 250 days. Their data showed that PCB accumulation in coho salmon was in direct proportion to dietary concentrations, and that the approach to "steady-state" required long periods of time (>200 days).

Virtually all attempts to relate PCB accumulation in natural populations of fishes have arrived at the same conclusions: (1) the major source of PCBs to fish may be found in the diet; and (2) the primary determinants of the ultimate burden to be found in a given species of fish are the mass accumulated per dose (meal) and the inherent rate of PCB elimination for the species (Norstrom et al., 1976; Weininger, 1978; Thomann and St. John, 1979; Thomann, 1981; Jensen et al., 1982; Pizza and O'Connor, 1983; O'Connor, 1984a, 1984b; Thomann and Connolly, 1984; O'Connor and Pizza, in press, b). In several instances where authors have concluded that fishes accumulate PCBs directly from water, we have found that insufficient data have been collected with which to evaluate the dietary route of PCB accumulation (e.g. Macek et al., 1979; Brown et al., 1985), or that the criteria applied to a satisfactory prediction of PCB burdens were so broad as to accept predictions \pm 50% or more (Branson et al., 1975; Clayton et al., 1977; Pavlou and Dexter, 1979).

Perhaps the most comprehensive evaluation of PCB transport to fishes from the environment was carried out by Thomann and Connolly (1984). By using data from the study of food-webs in the Lake Michigan ecosystem (see also Weininger,

1978; Thomann, 1981; Connolly and Winfield, 1984), Thomann and Connolly (1984) concluded that PCB transport in the Lake Michigan food-web followed energy flow from smaller organisms in lower trophic levels to the lake trout (Figure 4). Lake trout accumulated as much as 90% of their body burden of PCB from dietary sources.

The only study in which actual dietary doses of PCBs were evaluated simultaneously with body burden was reported by O'Connor (1984a). In that study, striped bass flesh and stomach contents were measured for PCB content, and regression analysis was used to establish the relationship between body burden and daily dose of PCB in the food. O'Connor (1984a) established that striped bass from the New York harbor region ingested a daily ration equivalent to about 5% of body weight per day, and that measurement of the PCB mass in samples of food enabled the calculation of mass of PCB ingested per day per fish (Table 3). Coefficients of determination for the regression of PCB body burden on the daily dose of PCB taken in with the food were 0.67 and 0.65 for bass from samples taken at Weehawken, New Jersey and at Canal Street in Manhattan (O'Connor, 1984a). O'Connor concluded (1984a; p. 157):

"PCB body burdens in...striped bass are maintained by the consumption of a PCB-contaminated diet. The source of PCB to the prey is from both the water and the sediments. Once ingested, the PCB in prey organisms is assimilated into the striped bass with high efficiency... and plateau levels are achieved rapidly...."

Overall, it is apparent from the literature that PCBs in fishes are accumulated from two sources, direct water uptake and food-chain transport. In different environments one or the other of these processes may dominate, depending upon the concentrations of PCB in the water column. In the Upper

Hudson, for example, where dissolved and suspended PCB concentrations in the water may be very high (Schroeder and Barnes, 1983; Sloan et al., 1984; Brown et al., 1985), fishes may accumulate a large proportion of their PCB by direct uptake from water. In environments such as the open ocean where there is little suspended material and PCB concentrations in the water are exceedingly small, the proportion of the body burden deriving from water uptake is reduced. Under such conditions, the primary route for PCB accumulation would be via the food-chain. Such phenomena have been tested experimentally by Rubinstein et al. (1983, 1984) in model ecosystems.

Bluefish

Considering the problem of PCB accumulation in bluefish, it is most likely that the primary source of PCBs is the food chain, and that the processes involved in PCB transport to bluefish in coastal waters are essentially the same as for striped bass in the New York harbor region, and for lake trout in the open waters of Lake Michigan. As noted by O'Connor and Pizza (in press, a) for striped bass, the calculation of PCB concentrations in bluefish from water concentration data results in estimates that are much lower than the values observed (Table 2). If water were the only source of PCB to bluefish in Atlantic coastal waters, one would expect concentrations of PCBs in bluefish to remain at or below 1.0 ppm. However, both bluefish and the food items upon which they prey in the estuary and in the ocean are contaminated with PCBs at concentrations between 1.0 and 20.0 ppm (Belton et al., 1983, 1985).

Assuming the dietary requirements of bluefish to be approximately the same as striped bass (i.e. about 5% of body weight per day), and a food resource contaminated with PCBs at concentrations between 1.0 and 5.0 ppm, application of dietary mass transport models and pharmacokinetics results in the prediction that

bluefish body burdens would range from 2.0 to 10 ppm (wet-weight basis), depending upon the age of the specimen sampled. During winter months, when bluefish move offshore into waters less contaminated with PCB, one would expect lower overall body burdens; however, the approach of body burdens to plateau (or "steady-state") as the result of dietary exposure is so rapid that maximum body burdens could be expected to be reached as soon as the population migrated back to coastal waters and encountered prey contaminated with high concentrations of PCBs (Pizza and O'Connor, 1983).

Pharmacokinetics of PCB Accumulation in Fishes

PCBs are assimilated into fish from water by processes which follow first order kinetics (Branson et al., 1975; McKim and Heath, 1983; Mackay and Hughes, 1984). That is, a constant proportion of the PCBs in the water to which the fish are exposed is transported across the gill surface into the blood and distributed to the tissues. The mechanism for cross-gill transport has not been defined, but is predictable based upon equilibrium partitioning using the concepts of thermodynamic mass-transport (Thomann, 1981). It has been suggested that the transport of PCBs across the gills of fishes is neither a diffusional process nor active transport, but is best described as "ligand-assisted-diffusion," in which large molecules (probably lipoproteins) in the gill tissue sorb or bind the PCBs. Once in contact with the blood on the internal side of the membrane, PCBs sorb to or dissolve in blood lipoproteins, and are transported to the tissues in proportion to the blood supply of each tissue (Califano, 1981).

PCBs in the food of fishes are instantaneously incorporated into the body burden of the fish (i.e. zero-order process). They are not, however, assimilated instantaneously into the various tissues; as with the process of transport from water to the body of the fish, partitioning from the gut to the

tissues follows first-order processes (Bruggeman et al., 1981; Pizza and O'Connor, 1983).

Pizza and O'Connor (1983) defined the kinetics of PCB assimilation from food into striped bass in a series of experiments that involved both single- and multiple dosing of fish with known quantities of PCBs. They found that the dynamics of the PCBs conformed to pharmacokinetic models developed for drugs by Goldstein et al. (1974). Transport of PCBs from the site of absorption (the gut) to the tissues occurred in two phases over a period of 120 hours. The first phase, lasting about 24 hours, showed a rapid loss of PCB from the gut coupled with an increase in the quantity of PCB in the remaining tissues. The loss of PCBs from the gut was equivalent to the rate of assimilation into the remaining tissues, and was defined by

$$\log M = \log M_0 - k_a t/2.30,$$

where M_0 is the quantity at the absorption site at time zero, M is the quantity remaining at time t and k_a is the assimilation rate constant obtained from the slope of the regression of \log unabsorbed dose in the gut over time (Figure 5).

Pizza and O'Connor (1983) noted that elimination of PCBs from striped bass began as soon as PCBs were transported from the gut to the tissues. This suggests that not all the PCBs assimilated remained within the body of the organism, even for a compound with a high degree of persistence. They defined the elimination rate constant k_e for PCBs, as

$$\log X = \log X_0 - k_e t/2.30,$$

where X_0 is the quantity of compound in the body of the fish at time 0, X is the quantity present at time t , and k_e is the elimination rate constant.

In reality, fishes are not exposed to single doses of PCBs in the environment. In contaminated regions fishes are exposed to PCBs at varying

concentrations in the food (O'Connor, 1984a) and in the water (Mancini, 1983). By applying pharmacokinetic principles to multiple doses of PCBs in food, Pizza and O'Connor (1983; O'Connor and Pizza, in press, a) were able to determine the rate at which a long-term PCB burden would accumulate in fishes, and to estimate the burden as it accumulated over time, and as the organism increased in size. Similar approaches have been used by Thomann (1981) and by Thomann and Connolly (1984) in their studies of contaminant accumulation in lake trout from Lake Michigan.

These results show that fishes, once exposed to PCBs in the food, accumulate PCB rapidly and achieve a "plateau" concentration of contaminant quickly. For striped bass in the Hudson, 90% of plateau was reached within 8 doses; assuming fish that feed twice per day, a fish entering a new, contaminated environment will have reached plateau PCB concentrations within 4 to 6 days (O'Connor and Pizza, in press, a).

Pharmacokinetic, or mass-transport, concepts have been applied in several models aimed at predicting PCB concentrations and burdens in fishes from contaminated environments. The most important factors determining the body burden were: (1) the dose of PCB given to the fish per unit time, and (2) the rate constant for elimination of the PCB from the fish. It has also been established that accumulation of PCBs in fishes differs according to the physical-chemical characteristics of individual PCB congeners, and the extent to which individual congeners are metabolized, transformed or eliminated by fishes (Hansen et al., 1976; Shaw and Connell, 1980; Matsuó, 1980; Bruggeman et al., 1981; Califano, 1981; Niimi and Oliver, 1983; Smith et al., in press). Separating the accumulation process into two segments (assimilation and retention), most chlorobiophenyl congeners are assimilated in roughly equal proportions from

environmental media, whereas the elimination process follows kinetics that differ substantially for different congeners (Hutzinger et al, 1972; Bruggeman et al., 1981; Niimi and Oliver, 1983). It has been suggested that for PCBs partitioning from the environment to fishes, whether from water or from food, partition coefficients of PCB congeners are sufficiently alike (10^4 to 10^6) to be unimportant as a determinant of the mass of each congener assimilated. However, congener-specific differences in solubility, lipophilicity, macromolecular binding and physical structure are sufficiently great to lead to measureable differences in the metabolic and transport processes which determine elimination from the body of the fish (Bruggeman et al., 1981; Niimi and Oliver, 1983; Smith et al., in press).

Tissue Disposition and Elimination of PCBs in Fishes

PCBs accumulate in the order from greatest to least concentration as follows: nervous tissue > liver > gonad > muscle > kidney (Mitchell et al., 1977; Guiney and Peterson, 1980; Stein et al., 1984; Califano, 1981; O'Connor and Pizza, in press, b). Pharmacokinetic studies aimed at determining the transport of PCBs from the site of uptake to the tissues are few; O'Connor and Pizza (in press, b; Pizza, 1983) determined that residues of Aroclor 1254 were measureable in all tissues of striped bass within 6 hours after exposure, and that rates of increase of PCB concentration were different among different tissues. For example, in single-dose studies, PCBs in muscle, heart and spleen of striped bass increased during the first 12 to 24 hours after exposure and subsequently declined as the contaminant was either removed from the body or distributed to other tissues. In the liver, however, PCBs continued to increase for 24 to 48 hours before a decline was measureable. O'Connor and Pizza (in press, b) were also able to detect a translocation of ^{14}C -labeled PCB residues from liver

tissue to gall bladder and bile that was related to PCB elimination (see also Melancon and Lech, 1976).

Essentially the same pattern of tissue disposition was observed in multiple exposure studies with striped bass (Table 4), except concentrations in all tissues increased in proportion to the exposure concentration (Pizza, 1983; O'Connor, 1984b). Interestingly, there occurred a rapid loss of a portion of each PCB dose amounting to about 40% to 50% (as mass), whereas between 50% and 60% of the mass of each dietary dose was retained. O'Connor and Pizza (in press, b) speculated that PCB disposition in fishes proceeded in two phases. In the first phase, a fraction of the assimilated PCBs may be described as "labile," subject to the sort of rapid elimination seen in laboratory pharmacokinetic studies (Bruggeman et al., 1981; Pizza and O'Connor, 1983; Lech and Peterson, 1983; McKim and Heath, 1983). A second, "stable" fraction becomes stored in tissues or in depot fat. The stable fraction shows longer elimination half-lives (>200 days) similar to those observed in elimination studies conducted after long-term exposure, or with specimens caught from highly contaminated environments (Hansen et al., 1971; Nisbet and Sarofim, 1972; Metcalf et al., 1975; Mayer et al., 1977; Nimi and Oliver, 1983). O'Connor and Pizza (in press, b) suggested that the proportion of the PCB dose likely to enter the stable compartment is proportional to the body lipid concentration of the species in question. Thus, species such as striped bass, lake trout or blufish, all of which have a high concentration of body lipid, may accumulate PCBs to high concentrations, whereas species with low body fat (e.g. flounder, codfish, etc.) generally show low PCB burdens (Lieb et al., 1974).

Possible routes for PCB elimination from fishes include diffusion across the gill to the water and removal via the hepatic pathway. Loss of PCBs across the

surface of the gill has been demonstrated in both striped bass and in rainbow trout (Califano, 1981; Guiney et al., 1977). For such a phenomenon to occur, however, the organism must be in a medium in which the dissolved PCB in the water is very low, favoring a diffusional exchange from the gill to the water. Given the high lipid solubility of PCBs, it is unlikely that such a pathway will operate for fishes in any situation other than in laboratory exposures where body burdens may be very high. However, the formation through metabolism of any water-soluble PCB metabolites (Melancon and Lech, 1976; Stein et al., 1984) may result in PCB metabolite removal via the gill.

The most likely route for PCB elimination is via the hepatic pathway; i.e. partitioning to liver tissue from the blood, solubilization in bile fluids and excretion with the bile to the intestine (Pizza, 1983; O'Connor and Pizza, (in press, b). In their study of PCB kinetics in individual striped bass tissues, O'Connor and Pizza (in press, b) determined that the k_e for PCBs was essentially the same for all tissues; that is, tissues such as muscle, liver, spleen, etc., released PCBs in constant proportion to the PCB mass in the tissue. Since the liver contained about four times the mass of PCB in other tissues, the greatest mass of PCBs was being removed from liver tissue and being transported into bile for eventual elimination in the feces.

PCB Metabolism in Fishes

Although PCBs are persistent in the environment, their susceptibility to degradation has been well documented. Hutzinger et al. (1974) and Baxter and Sutherland (1984) described the photodegradability of PCBs in the atmosphere, and many workers have demonstrated the potential for microbial populations to either metabolize or transform PCBs (Furukawa and Matsumura, 1976; Tucker et al., 1975; Reichardt et al., 1981; Suflita et al., 1983). Recently, Brown et al. (1984)

provided evidence of PCB degradation by natural populations of aerobic and anaerobic bacteria in the upper Hudson River. Their studies showed that bacteria cause reductive dechlorination of various PCB congeners in anaerobic systems which facilitated later ring-opening and mineralization of PCBs by aerobic microorganisms. Brown et al. (1984) speculated that bacterial metabolism of selected PCB congeners may be one of the major mechanisms, along with selective volatilization, whereby industrial mixtures of PCBs become transformed to consist primarily of higher chlorinated congeners with a high proportion of o,o'-Cl substitutions. Such congeners are generally recognized as the least hazardous of the PCB congeners, and the process of bacterial degradation may, in fact, be a process for PCB detoxification in the environment (Furukawa, 1982).

It has long been known that PCBs were metabolized by mammalian and avian forms. Gage and Holm (1976) demonstrated differential elimination of PCB congeners in the mouse, and Kato et al. (1980) showed metabolism of PCBs in the rat by insertion of hydroxyl groups on the phenyl rings. Kato et al. (1980) determined a relationship between degree of chlorination and chlorine position in mammalian metabolism of PCBs. Preston and Allen (1980) examined the metabolism of 2,2',5,5'-tetrachlorobiphenyl by rat liver microsomes, and showed that about 90% of the metabolites were dihydro- and dihydroxy-PCB derivatives of greater solubility than the parent compound. Chen et al. (1982) showed that degree of chlorination and position of chlorine substitution on the PCB molecule affected their elimination by humans; they concluded that two adjacent, unsubstituted carbons at the meta- and para- positions facilitated PCB metabolism in humans, and that congeners with six or more chlorines were not readily eliminated.

Relatively little is known about the metabolism of PCBs in fishes. This may be due to the fact that PCB metabolism by fishes proceeds at an exceedingly slow

rate. As noted by Lech and Bend (1980):

"Several classes of compounds, including some polychlorinated biphenyls, are metabolized slowly, and their disposition in fish may not be influenced to any great extent by biotransformation."

Our review of the literature has identified few papers dealing with metabolite formation and identification of PCB metabolites in fishes. Like mammals, however, fishes have been shown to possess the hepatic mixed-function oxidase (MFO) system necessary for metabolism of PCBs to a variety of conjugated metabolites (Addison et al., 1978, 1979; Forlin and Lidman, 1981; Forlin et al., 1984). Hutzinger et al. (1972) studied the metabolism of four PCBs in rainbow trout as well as in pigeons and rats. Rats and pigeons produced identifiable hydroxy-PCB metabolites, but no evidence for metabolism was found in the rainbow trout. Hutzinger et al. (1972) found no evidence for reductive dechlorination of PCBs in any of the species studied.

Melancon and Lech (1976) isolated a polar metabolite of 2,2',5,5'-tetrachlorobiphenyl in the bile of rainbow trout exposed to the PCB in water. The metabolite was identified as a glutathione conjugate of 4-hydroxy-2,2',5,5'-tetrachlorobiphenyl (see Hesse et al., 1978; Shimada et al., 1981). Similar results were found for PCBs in English sole (Parophrys vetulus) (Stein et al., 1984); aqueous-soluble radioactivity deriving from apparent metabolism of PCBs was detected in the bile of sole, and was shown to be a glutathione conjugate. However, metabolism of PCB in the English sole proceeded at a very slow rate; more than 98% of the PCB-derived radioactivity recovered by Stein et al. (1984) was in the form of parent PCB compounds.

In general, it may be concluded that although fishes contain the enzyme systems required for the metabolism of PCBs, such metabolism proceeds very

slowly, on the order of 1% of the rates for mammals. Assuming that the same structural and steric phenomena affect PCB metabolism in fishes, we would speculate that some metabolism of the lower-chlorinated PCB congeners (from 2 to 4 chlorines) would occur. However, metabolism of higher chlorinated PCB classes would be virtually zero. In ecological systems where PCB concentrations are substantial, kinetic processes of elimination of parent PCBs would be far more important in the removal of PCBs from the body of fishes than would the process of metabolism (O'Connor and Pizza, in press, a).

Accumulation and Disposition of PCB Congeners in Fish

It has been apparent since the earliest studies of PCB distribution in natural environments that fishes accumulated groups of PCB congeners that were not identical with those found in pollutional sources (Risebrough et al., 1968; Nisbet and Sarofim, 1972; Hutzinger et al., 1974; Nadeau and Davis, 1976). PCB burdens in fishes and shellfish comprise higher-chlorinated congeners (> 4 chlorine molecules) rather than the lower chlorinated congeners more abundant in PCB discharges (Armstrong and Sloan, 1980; Sloan and Armstrong, 1982; O'Connor et al., 1982).

In the Hudson River and Hudson-Raritan estuary, it was found that fish samples taken farther from industrial PCB sources contained a higher proportion of PCBs resembling Aroclor 1254 than Aroclor 1221 or Aroclor 1016, even though the major sources of PCBs to the system were Aroclor 1221 and 1016 (Bopp, 1979; Armstrong and Sloan, 1980, 1982; Brown et al., 1985). Bopp et al. (1981) proposed that the abundance of Aroclor 1242 and 1254 downstream from PCB discharges was related to selective retention of higher chlorinated PCB congeners on sediments subject to bed-load transport; the lower chlorinated congeners present in Aroclor 1016 were gradually lost by transport out of the system in the dissolved form and

by volatilization to the atmosphere. The results of modelling studies by Thomann (1981) and pharmacokinetic studies by Pizza and O'Connor (1983; see also O'Connor, 1984b) would suggest that although fishes may accumulate lower- and higher-chlorinated congeners in roughly equal proportions from the environment, higher rates of elimination for mono-, di-, and trichlorobiphenyls would lead to the presence in fishes of PCB body burdens chromatographically similar to Aroclor 1254, rather than Aroclor 1016. Laboratory studies by other workers substantiate these hypotheses (Bruggeman et al., 1981; Niimi and Oliver, 1983).

Time-series analysis of PCBs in striped bass and other species from the Hudson River (Sloan et al., 1983, 1984; Brown et al., 1985) have demonstrated that as the mass of PCB input to the Lower Hudson was reduced, PCB concentrations in fishes not only declined, but also showed a change in the proportion of lower chlorinated isomers relative to higher chlorinated isomers. Because the downstream sources provide lower-chlorinated congeners to fishes in the New York Harbor region (MacLeod et al., 1981; O'Connor and Pizza, unpublished data), we conclude that reductions in mono-, di-, and trichlorobiphenyls seen in fishes from the Hudson River and estuary are due primarily to selective elimination of these congeners and the retention of congeners with four or more chlorine molecules.

Apart from their value as indicators of selective elimination of PCB congeners, detailed analysis of PCB body burdens in fishes also provides insight into the potential toxic response associated with the consumption of contaminated fish by humans. It has been found that PCB congeners with fewer chlorine molecules are less toxic than congeners with a greater degree of chlorination. Thus, as body burdens of PCBs in fish change due to selective elimination of lower chlorinated congeners, one might expect potential toxicity to increase.

Such a conclusion is not fully warranted, however, since structural factors related to degree and position of chlorine substitution in PCBs play a strong role in defining toxicity (Goldstein et al., 1977). Safe (1984) has discussed such factors and has concluded that PCB congeners that are "approximate isostereomers" of 2,3,7,8-tetrachlorodibenzodioxin (Figure 7) have the greatest potential to exert toxic effects at the metabolic level (as measured by MFO or AHH induction). The rather crude analysis of PCB as "Aroclor 1016" or "Aroclor 1254" provides no information in this regard, and the assessment of potential toxic effects of PCBs based upon Aroclor analysis is probably not warranted.

Recently developed techniques allow the isolation and identification of PCB congeners in environmental samples. Beginning with Ballschmitter and Zell (1980), standard classification of chlorobiphenyl congeners was established. Mullin et al. (1984) reported the synthesis and chromatographic properties of all 209 potential PCB congeners and data on the concentrations and mass of PCB congeners in environmental samples are now emerging from several laboratories (Mullin et al., 1983; Bush et al., 1983; Smith et al., in press; Samuelian et al., manuscript in review).

Smith et al. (in press) analysed samples of sediment, fish and fish food organisms from the Great Lakes for 72 PCB congeners and determined that the most toxic congeners were either absent or present in very low quantities. They concluded from their analysis that "...estimates of toxic exposure based on total PCB values may be unreliable..." due primarily to variation in the partitioning of PCB congeners in the water column-sediment-fauna ecosystem under study. Samuelian et al. (manuscript in review) identified 47 PCB congeners from liver and flesh of Atlantic tomcod (Microgadus tomcod) from the East River, New York, as well as from shrimp (Crangon septemspinosa) used as food by tomcod. Comparison

of PCB congeners in tomcod with those in food organisms showed substantial differences; Crangan contained greater quantities of lower chlorinated PCB congeners (di- and trichlorobiphenyls), whereas tomcod contained no dichlorobiphenyls, and trichlorobiphenyls were present as a minor constituent of the PCB burden. When Samuelian et al. compared the profile of congeners in the fish to a profile of congeners from a mixed standard of Aroclor 1016 and 1254, they concluded that environmental samples of PCB should not be quantified on the basis of Aroclors since body burden profiles differed significantly from Aroclor standards.

From the toxicological perspective, Clarke et al. (1986) applied cluster analysis to PCB congeners identified as having potential biological effects based upon their ability to induce monooxygenase enzymes in mammalian systems. Their technique holds promise as an effective means of evaluating the PCB burden of an environmental fish sample for potential toxicity by isolating those components most likely to influence the health of the consumer.

Evaluation of Data on PCBs in Bluefish

Despite the value of the bluefish fishery and the fact that bluefish are the species most sought by recreational fishermen on the Atlantic coast, there are relatively few data on chemical contamination of the species. A full summary of bluefish PCB data is presented in a recent data report submitted to Congress (Anon., 1986). The New York State Department of Environmental Conservation (1981) reported PCB values for bluefish from a number of sites, including the estuarine portions of the Hudson River near Peekskill, New York Harbor, the Atlantic and Long Island Sound coasts of Long Island and open Atlantic waters. Samples taken in the estuarine system (Peekskill and New York Harbor) had higher PCB concentrations than samples from outside the harbor system (Table 5). However,

one sample taken at Orient Point on the eastern end of Long Island had PCB concentrations of 3.6 ppm, substantially higher than the values measured in bluefish from either Peekskill (3.1 ppm) and in New York Harbor (2.1 ppm). Along the coast of Long Island, PCB concentrations in bluefish ranged from a low of 0.48 ppm in Long Island Sound, to 0.94 ppm at Cold Spring, and 1.15 ppm at a site in the Eastern Sound. In contrast to the usual trend where older specimens contain the higher PCB concentrations, young-of-the-year bluefish from the Peekskill area had PCB concentrations of 3.1 ppm, whereas older specimens taken in the open ocean and in Long Island Sound ranged between 350 and 600 mm total length and had much lower PCB levels (Table 5).

Belton et al. (1983) reported PCB concentrations in bluefish from New Jersey waters of the Hudson River and along the Atlantic Coast. Bluefish from the Hudson River contained 3.44 ppm PCB in 1975 and 1976, while specimens sampled in 1981 had a PCB concentration of 1.78 ppm. Bluefish samples obtained from offshore sites contained from 0.67 to 1.44 ppm total PCBs. In all, the samples analysed by the New Jersey Dept. of Environmental Protection (Belton et al., 1983) confirmed the data reported from NYSDEC; even though sample sizes reported by Belton from the Hudson estuary were small ($n = 4$, $n = 2$ for 1975-76 and 1981, respectively).

As part of a study to determine toxic hazards to recreational urban fishermen, Belton et al. (1985) again sampled bluefish from the Hudson River and Newark Bay region for PCBs. For samples taken in 1982, total PCB concentrations were 3.29 ppm ($\bar{n} = 5$), while in 1983, samples from several sites ranged from 1.51 to 5.44 ppm (n , for the most part, = 1). Belton et al. (1985) concluded that PCB levels in bluefish from the Hudson River to New York Bay were likely to exceed 4.0 ppm, and that PCB levels in bluefish taken from the Newark Bay complex were likely to exceed 2.0 ppm; a public health advisory has been published with regard to the

consumption of Bluefish from New Jersey waters (Figure 8, from Belton et al., 1985).

Hypotheses and Speculations Regarding the Dynamics of PCBs in Bluefish on the Atlantic Coast of North America

Although the data are restricted in quantity and quality, it is possible to propose hypotheses regarding the dynamics of PCBs in bluefish populations of the Western North Atlantic, and to speculate as to sources of PCBs to the bluefish population and the future course of PCB contamination in bluefish. In large part these hypotheses and speculations are based upon data from modeling studies with striped bass and lake trout, and upon pharmacokinetic studies of the behavior of PCBs in striped bass and rainbow trout (Thomann, 1981; Jensen et al., 1982; Pizza and O'Connor, 1983; Thomann and Connolly, 1984; Connolly and Winfield, 1984; O'Connor, 1984a; O'Connor and Pizza, 1985, in press, b). Although these speculations are made with full knowledge that the data are insufficient, the relative constancy of PCB dynamics among fish species studied suggests that the concepts and trends put forth will be accurate, although actual levels of PCB contamination in the bluefish population will be the final determinant of the time-frame involved.

1. Sources of PCB Contamination in Bluefish

PCB contamination is worldwide, mediated by atmospheric transport, surface water flow patterns and the transport through the environment of dissolved and particle-associated PCBs. Due to high concentrations of PCBs in many estuarine systems and transport of PCB-contaminated estuarine water to coastal oceans, PCB concentrations in near-coastal waters will be higher than in waters from more remote ocean areas. Estuarine source of PCBs to coastal waters will influence PCB concentrations in bluefish in two ways: (1) by causing the direct uptake of PCBs

by bluefish exposed to the contaminant dissolved in water; and (2) by causing the contamination of bluefish prey. O'Connor and Pizza (in press, a), in their paper on sources of PCBs in marine fishes showed that, for open ocean waters where dissolved PCB concentrations are low, most of the PCB burden will be accumulated via the food chain. They predicted that for striped bass more than 70% of the PCB burden was the result of dietary uptake. Thomann and Connolly (1984), working with the analogous system of lake trout in Lake Michigan, concluded that more than 90% of the PCB in lake trout derived from dietary uptake.

PCB concentrations in water, sediments and biota generally show a gradient from onshore to offshore sites, with the highest concentrations occurring in estuaries and in near-coastal waters. O'Connor et al. (1982) showed a gradient in PCB concentration among striped bass from New York waters that decreased with distance from New York Harbor. It may be expected, therefore, that PCB concentrations in bluefish will be lower the greater the distance from the coast, and especially in relation to the distance from New York harbor. Conversely, it may be predicted that, as bluefish migrate from shelf waters toward the coast during the spring months, body burdens of PCBs will increase as the fish ingest food more highly contaminated with PCBs.

2. Concentrations of PCBs in Bluefish

We hypothesize that bluefish, like striped bass and lake trout, will derive most of their PCB body burden from the diet. Lacking data on dietary requirements, growth, metabolism and other factors necessary for construction of an accurate model (Thomann and Connolly, 1984; O'Connor and Pizza, in press, a), only crude estimates can be made as to what body burdens may be accumulated. A means for making such an estimate may be derived from the food-chain studies conducted on striped bass in New York Harbor, as well as pharmacokinetic studies

of PCB assimilation. In those studies it was determined first, that the BAF for dietary PCBs in striped bass was about 0.75 (Pizza and O'Connor, 1983), and second, that the relationship between daily dose of PCBs to striped bass and the body burden was equal to about 2 X log of the dose (1.98 and 1.67 for data sets from Weehawken and Canal Street, respectively; O'Connor, 1984a).

Let us assume, then, that a bluefish weighing 1.5 kg (slightly more than 3 lb) resembles a striped bass in that: (1) it has similar PCB kinetics; (2) it has similar rates of metabolism; and (3) it consumes approximately 5% of its body weight per day in food (from O'Connor, 1984a). Under such conditions, a bluefish feeding on a contaminated food resource will reach plateau burdens of PCB after a few days of exposure (Pizza and O'Connor, 1983). The plateau burden, as micrograms of PCB, may be approximated as:

$$\text{Log B} = 2.0 \text{ log D} - 1.0 \quad (\text{from O'Connor, 1984a})$$

where B is the PCB burden and D is the daily dose of PCB. Concentration was estimated as burden divided by fish weight, or B/1500. PCB concentrations in bluefish prey range from less than 0.5 ppm to more than 4.0 ppm total PCBs (NYSDEC, 1981 and Belton et al., 1985). Using the formula above, PCB concentrations in the adult bluefish of 1,500 g weight may be estimated for reasonable PCB doses as follows:

<u>PCB in food (ug/g)</u>	<u>PCB in bluefish (ug/g)</u>
0.5	0.32
1.0	1.27
2.0	5.09
3.0	11.44

These calculated values, ranging from 0.32 to more than 11 ppm PCB in bluefish have a precision of \pm 35%, and may be considered accurate only for

bluefish deriving their PCBs from dietary sources. Interestingly, they cover the full range of PCB concentrations seen in bluefish from the New York-New Jersey metropolitan region. The data are nearly useless, however, in testing the predictive power of the relationships in the literature. Fortunately we have an instance (NYSDEC, 1981) in which Atlantic menhaden and bluefish were collected from New York Harbor within two weeks of one another, making it reasonable to assume that the bluefish in the harbor (average length 590 mm) had been foraging on the menhaden (average length 243 mm). Given a measured PCB concentration in menhaden of 1.34 ppm, and an average weight of bluefish of 2,465 g, we would estimate a concentration from these data of 4.07 ppm total PCB in the bluefish. In fact, the observed range for the bluefish sample was from 0.11 to 5.77 ppm total PCB, with a mean of 2.33 ppm. The calculated value of 4.07 falls within the estimated range of precision of the prediction ($\pm 35\%$) noted by O'Connor (1984a).

Unfortunately we have no real data with which to determine the actual relationship between food organism PCB content and the concentration of PCBs in bluefish. Such data are sorely needed, and plans for their collection should be included in any program designed to obtain further information on PCB contamination of coastal bluefish populations. As shown in the data from O'Connor (1984a), PCB concentrations vary widely at different sites even within a confined environment such as New York Harbor, and the only way to obtain the proper data is to carry out simultaneous sampling of bluefish, bluefish stomach contents and forage fish, all from the same site.

3. Persistence of PCBs in Bluefish

Based upon a wealth of data from striped bass, rainbow trout, lake trout and other species, it is known that PCBs in fish flesh are not permanent; that is, even though assimilated into fish tissues and into depot fat, PCBs may be

removed, gradually, from the body of a fish, as determined through bi-modal elimination kinetics. As seen in the striped bass (O'Connor and Pizza, in press, b) a high proportion (40 to 50%) of the PCBs assimilated from a dietary dose are lost rapidly, whereas the remainder appear to partition to "storage areas" in tissues, tissue lipids and depot fat. These storage areas retain PCBs for a longer period of time, with a half-life for elimination on the order of 100 to 200 days.

For many species of fish from the Hudson River, PCB elimination may proceed at fairly rapid rates once major sources are controlled. Sloan and his co-workers (Armstrong and Sloan, 1980; Sloan and Armstrong, 1982; Sloan et al., 1983; Brown et al., 1985) showed a rapid decline in PCB concentration in Hudson River fish between 1978 and 1984. The calculated half-times for such declines were rapid, far in excess of those estimated for the Hudson system in earlier work by Thomann and St. John (1979). It would appear that the declines observed in PCB concentrations in Hudson River fish have halted, having reached a quasi-steady-state imposed by the presence of PCBs in sediments throughout the system (Sloan et al., 1983, 1984).

4. PCB Congener Distribution in Bluefish

None of the data available to us at this time provide information on the PCB congener distribution in bluefish; all data from NYDEC and NJDEP are available to us only as total PCBs or as Aroclors, with no apportionment among congeners or chlorinated classes. In this regard, one can only speculate that congener distribution in bluefish is similar to that found for other species and in bluefish forage organisms. From the data of Smith et al. (in press) and Samuelian et al. (in review) we would predict the presence of 40 to 50 PCB congeners in bluefish from ocean waters, with the bulk of the congeners representing tetra-,

penta- and hexachlorobiphenyl congeners with a high degree of chlorine substitution in the o, o' positions. Based upon the few environmental data available, it would be most unlikely to find in bluefish high concentrations of PCB congeners known to be particularly hazardous or toxic.

REFERENCES

- Addison, R., M. Zinck and D. Willis. 1978. Induction of hepatic mixed-function oxidase (MFO) enzymes in trout (*Salvelinus fontinalis*) by feeding Aroclor 1254 or methylcholanthrene. *Comp. Biochem. Physiol.* 61(C): 323-325.
- Addison, R., M. Zinck, D. Willis and D. Darrow. 1979. Induction of hepatic mixed-function oxidase in trout by polychlorinated biphenyls and butylated monochlorodiphenyl esters. *Toxicol. Appl. Pharmacol.* 49: 245-248.
- Allen, J., L. Abrahamson and D. Norback. 1973. Biological effects of polychlorinated biphenyls and triphenyls on sub-human primates. *Environ. Res.* 6: 344-354
- Allen, J., D. Norback and I.C. Hsu. 1974. Tissue modifications in monkeys as related to absorption, distribution and excretion of polychlorinated biphenyls. *Arch. Environ. Contam. Toxicol.* 2: 86-94
- Allen, J., and D. Norback. 1976. Pathobiological responses of primates to polychlorinated biphenyl exposure. *Proc. Nat'l. Conf. on Polychlorinated biphenyls.* EPA-560/6-75-004. pp. 43-49.
- Anon. 1986. Report on the 1984-1986 federal survey of PCBs in Atlantic Coast bluefish. NOAA Data Report 179 pp.
- Armstrong, R. and R. Sloan. 1980. Trends in levels of several known chemical contaminants in fish from New York State waters. *Bur. Env. Prot. NY State Dept. Environmental Cons., Albany, New York.* 77 pp.
- Armstrong, R. and R. Sloan. 1982. PCB patterns in Hudson River fish. I. Resident/Freshwater species. *Proc. Hudson River Env. Soc., Hyde Park, New York.*
- Ballschmitter, K., and M. Zell. 1980. Analysis of polychlorinated biphenyls (PCBs) by glass capillary chromatography. Composition of technical Aroclor and Clophen-PCB mixtures. *Fres. Z. Anal. Chem.* 302: 20-31.
- Baxter, R., and d. Sutherland. 1984. Biochemical and photochemical processes in the degradation of chlorinated biphenyls. *Environ. Sci. Technol.* 18: 608-610.
- Belton, T., B. Ruppel, and K. Lockwood. 1983. Polychlorinated biphenyls (Aroclor 1254) in fish tissue throughout the State of New Jersey: A comprehensive survey. *N.J. Dept. of Env. Protection, Trenton, N.J.* 43 pp.
- Belton, T., B. Ruppel, K. Lockwood, S. Shiboski, G. Bukowski, R. Roundy, N. Weinstein, D. Wilson, and H. Whelan. 1985. A study of toxic hazards to urban fishermen and crabbers. *N.J. Dept. of Env. Protection, Trenton, N.J.* 68 pp.

- Bigelow, H., and W. Schroeder. 1953. Fishes of The Gulf of Maine. Fish. Bull. 74: 1-577
- Bopp, R. 1979. The geochemistry of polychlorinated biphenyls in the Hudson River. Ph.D. Diss. Columbia Univ. Lamont-Doherty Geological Observatory. Palisades, New York. 191 pp.
- Bopp, R., H. Simpson, C. Olsen and N. Kostyk. 1981. Polychlorinated biphenyls in sediments of the tidal Hudson River, New York. Environ. Sci. Technol. 15: 210-216.
- Bopp, R., H. Simpson, B. Deck and N. Kostyk. 1984. The persistence of PCB congeners in sediments of the lower Hudson. Northeastern Env. Sci. 3: 180-184.
- Branson, D., G. Blau, H. Alexander and W. Neely. 1975. Bioconcentration of 2,2',4,4'-tetrachlorobiphenyl in rainbow trout as measured by an accelerated test. Trans. Amer. Fish. Soc. 104: 785-792.
- Breder, C., and D. Rosen. 1966. Modes of Reproduction in Fishes. American Museum of Natural History, New York. Natural History Press. 941 pp.
- Briggs, J. 1960. Fishes of world-wide (circumpolar) distribution. Copeia 1960: 171-180.
- Brown, J., R. Wagner, D. Bedard, M. Brennan, J. Carnahan, and R. May. 1984. PCB transformations in upper Hudson sediments. Northeast. Env. Sci. 3: 167-179.
- Brown, M., J. McLaughlin, J. O'Connor and K. Wyman. 1982. A mathematical model of PCB bioaccumulation in plankton. Ecol. Modelling 15: 29-47.
- Brown, M., M. Werner, R. Sloan and K. Simpson. 1985. Recent trends in the distribution of polychlorinated biphenyls in the Hudson River system. Environ. Sci. Technol. 19: 656.
- Bruggeman, W., L. Marton, D. Kooiman and O. Hutzinger. 1981. Accumulation and elimination kinetics of di-, tri-, and tetrachlorobiphenyls by goldfish after dietary and aqueous exposure. Chemosphere 10: 811-832.
- Brungs, W. and D. Mount. 1978. Introduction to a discussion of the use of aquatic toxicity tests for evaluation of the effects of toxic substances. In: J. Cairns, K. Dickson and A. Maki (eds.) Estimating the Hazard of Chemical Substances to Aquatic Life. Philadelphia, ASTM Press. pp. 15-26
- Bush, B., J. Snow and S. Conner. 1983. High resolution gas chromatographic analysis of non-polar chlorinated hydrocarbons in human milk. J. Assoc. Off. Anal. Chem. 66: 248-255.
- Cahn, P., J. Foehrenbach and W. Guggino. 1977. PCB levels in certain organs of some feral fish from New York State. In: F. Vernberg, A. Calabrese, F. Thurberg and W. Vernberg (eds.). Physiological Responses of Marine Biota to Pollutants. New York, Academic Press. pp. 51-61.

- Califano, R.J. 1981. Accumulation and distribution of polychlorinated biphenyls (PCBs) in early life stages of the striped bass, Morone saxatilis. Ph.D. Diss. New York University. 158 pp.
- Califano, R., J. O'Connor and J. Hernandez. 1982. PCB dynamics in Hudson River striped bass. I. Accumulation in early life-history stages. *Aquatic Toxicology* 2: 187-204.
- Chen, P., M. Luo, C. Wong and C. Chen. 1982. Comparative rates of elimination of some individual polychlorinated biphenyls from the blood of PCB-poisoned patients in Taiwan. *Fd. Chem. Toxicol.* 20: 417-425.
- Clarke, J. 1986. Structure-activity relationships in PCBs: Use of principal components analysis to predict inducers of mixed-function-oxidase activity. *Chemosphere* 15: 275-287.
- Clayton, J. S. Pavlou and N. Breitner., 1977. Polychlorinated biphenyls in coastal marine zooplankton: Bioaccumulation by equilibrium partitioning. *Env. Sci. Technol.* 11: 676-681.
- Connolly, J., and R. Winfield. 1984. A user's guide to WASTOX, a framework for modelling the fate of toxic chemicals in aquatic environments. Part 1. Exposure concentration. EPA-600/3-84-077. 126 pp.
- Cordle, F., R. Locke and J. Springer. 1982. Risk assessment in a federal regulatory agency: An assessment of risk associated with the human consumption of some species of fish contaminated with polychlorinated biphenyls. *Env. Health Perspect.* 45: 171-182.
- Couch, J., and D. R. Nimmo. 1974. Ultrastructural studies of shrimp exposed to the pollutant chemical, polychlorinated biphenyl (Aroclor 1254). *Bull. Soc. Pharmacol. Environ. Pathol.* 11: 17-20.
- Defoe, D., G. Veith and R. Carlson. Effects of Aroclor 1248 and 1260 on the fathead minnow (Pimephales promelas). *J. Fish. Res. Bd. Canada* 35: 997-1002.
- Deuel, D. 1973. 1970 Salt water angling survey. Current Fish. Stat. 6200. Nat. Marine Fish. Serv., Washington, D.C.
- Duke, T., J. Lowe and A. Wilson. 1971. A polychlorinated biphenyl (Aroclor 1254) in the water, sediment and biota of Escambia Bay, Florida. *Bull. Env. Contam. Toxicol.* 5: 172-180
- Forlin, L., and U. Lidman. 1981. Effects of Clophen A50 and 3-methylcholanthrene on the hepatic mixed function oxidase system in female rainbow trout Salmo gairdneri. *Comp. Biochem. Physiol.* 70(C): 297-300.
- Forlin, L., T. Andersson, U. Koivusaari and T. Hansson. 1984. Influence of biological and environmental factors on hepatic steroid and xenobiotic metabolism in fish: Interaction with PCB and B-Naphthoflavone. *Mar. Env. Res.* 14: 47-58.

- Fuller, B., J. Gordon and M. Kornreich. 1976. Environmental Assessment of PCBs in the Atmosphere. MTR-7210, Rev. 1. The Mitre Corp. McLean, Va.
- Furukawa, K. 1982. Microbial degradation of polychlorinated biphenyls: . In: A. Chakrabarty (ed.) Biodegradation and Detoxification of Environmental Pollutants. CRC Press, Boca Raton Fl. pp. 33-57.
- Furukawa, K., and F. Matsumura. 1976. Microbial metabolism of polychlorinated biphenyls: Studies on the relative degradability of polychlorinated biphenyls components by Alkiligenes sp. J. Agric. Food Chem. 24: 251-256.
- Gage, J., and S. Holm. 1976. The influence of molecular structure on the retention and excretion of polychlorinated biphenyls by the mouse. Toxicol. Appl. Pharmacol. 36: 555-560.
- GESAMP (Joint Group of Experts on the Scientific Aspects of Marine Pollution) 1982. The Health of the Oceans. UNEP Regional Seas Repts. and Studies No. 16. 108 pp.
- Goldstein, A. L. Aronow and S. Kalman. 1974. Principles of Drug Action: The Basis of Pharmacology. New York. J. Wiley and Sons. 854 pp.
- Goldstein, J., P. Hickman, H. Bergman, J. McKinney and M. Walker. 1977. Separation of pure polychlorinated biphenyl isomers into two types of inducers on the basis of induction of cytochrome P-450 or P-448. Chem. Biol. Interact. 17: 69-87.
- Grosslein, M., and T. Azarovitz. 1982. Fish Distribution. New York Bight Atlas, Monograph 15. Albany, New York. New York Sea Grant Institute. 182 pp.
- Guiney, P., and R. Peterson. 1980. Distribution and elimination of polychlorinated biphenyl after acute dietary exposure in yellow perch and rainbow trout. Arch. Env. Contam. Toxicol. 9:667-674.
- Guiney, P., R. Peterson, M. Melancon and J. Lech. 1977. The distribution and elimination of 2,2',5,5'-(14-C)-tetrachlorobiphenyl in rainbow trout (Salmo gairdneri). Toxicol. Appl. Pharmacol. 39: 329-338.
- Hamelink, J., R. Wybrant and R. Ball. 1971. A proposal: exchange equilibria control the degree chlorinated hydrocarbons are biologically magnified in lentic environments. Trans. Amer. Fish. Soc. 100: 207-214.
- Hansen, D., P. Parrish, J. Lowe, A. Wilson, Jr. and P. Wilson. 1971. Chronic toxicity and uptake and retention of Aroclor 1254 in two estuarine fishes Bull. Env. Contam. Toxicol. 6:113-119.
- Hansen, D., P. Parrish and J. Forester. 1974. Aroclor 1016: Toxicity and uptake by estuarine animals. Env. Res. 7: 363-373.
- Hansen, L., W. Wiekhorst and J. Simon. 1976. Effects of dietary Aroclor 1242 on channel catfish (Ictalurus punctatus) and selective accumulation of PCB components. J. Fish. Res. Bd. Canada 33: 1343-1352.

- Haque, R., D. Schmedding, and V. Freed. 1974. Aqueous solubility, adsorption and vapor behavior of polychlorinated biphenyl Aroclor 1254. *Environ. Sci. Technol.* 8: 139-142.
- Hartsbrough, G. 1965. Great Lakes fish now suspect as mink food. *Amer. Fur Breeder* 38: 25-27.
- Hesse, S., M. Metzger and J. Wolff. 1978. Activation of (14-C)chlorobiphenyls to protein-binding metabolites by rat liver microsomes. *Chem. Biol. Interact.* 20: 355-365.
- Hetling, L., T. Tofflemire, E. Horn, R. Thomas and R. Mt. Pleasant. 1979. The Hudson River PCB problem: Management alternatives. *Ann. N.Y. Acad. Sci.* 320: 630-650.
- Hilton, J., P. Hodson, H. Braun, J. Leatherland and S. Slinger. 1983. Contaminant accumulation and physiological response in rainbow trout (*Salmo gairdneri*) reared on naturally contaminated diets. *Can. J. Fish. Aquat. Sci.* 40: 1987-1994.
- Hoeting, A. 1983. FDA regulation of PCB in food. In: F. D'Itri and M. Kamrin (eds). PCBs: Human and Environmental Hazards. Ann Arbor. Ann Arbor Science Press. pp. 393-408.
- Horn, E., L. Hetling and T. Tofflemire. 1979. The problem of PCBs in the Hudson River system. *Ann. N.Y. Acad. Sci.* 320: 591-609.
- Horn, E., and L. Skinner. 1985. Final Environmental Impact Statement for Policy on Contaminants in fish. Div. Fish and Wildlife, NY State Dept. of Env. Cons., Albany, New York. 150 pp.
- Hubbard, H.L. 1964. Chlorinated biphenyl and related compounds. In: Encyclopedia of Chemical Technology, 2 ed. 5: 289-298.
- Hutzinger, O., D. Nash, S. Safe, A. DeFreitas, R. Norstrom, D. Wildish and V. Zitko. 1972. Polychlorinated biphenyls: Metabolic behavior of pure isomers in pigeons, rats and brook trout. *Science* 178: 312-314.
- Hutzinger, O., S. Safe and V. Zitko. 1974. The Chemistry of PCBs. CRC Press, Cleveland, Ohio. 269 pp.
- International Agency for Research on Cancer (IARC). 1974. IARC Monograph on the carcinogenic risk of chemicals to humans. 7:241.
- International Agency for Research on Cancer (IARC). 1978. IARC Monograph on the carcinogenic risk of chemicals to humans 18:43.
- Interstate Electronics Corporation. (IEC) 1979. Environmental Impact Statement for New York Dredged material ocean disposal site designation. Rept. to U.S. EPA, April 1981. IEC, Anaheim, CA. 148 pp.

- Isaacs, J. 1973. Potential trophic biomasses and trace substance concentrations in unstructured marine foodwebs. *Mar. Biol.* 22: 97-104.
- Jelinek, C. and P. Corneliusen. 1976. Levels of PCBs in the U.S. food supply. *Proc. Nat'l Conf. on PCBs.* EPA-560/6-75-004. pp. 147-154.
- Jensen, A., S. Spigarelli and M. Thommes. 1982. PCB uptake by five species of fish in Lake Michigan and Cayuga Lake, New York. *Can. J. Fish. Aquat. Sci.* 39: 700-709.
- Jensen, S. 1966. Report of a new chemical hazard. *New Sci.* 32: 612
- Johansson, N., A. Larsson and K. Lewander. 1972. Metabolic effects of PCB (polychlorinated biphenyls) on the brown trout (*Salmo trutta*). *Comp. gen. Pharmacol.* 1972: 310-314.
- Karickhoff, S., D. Brown, and T. Scott. 1979. Sorption of hydrophobic pollutants on natural sediments. *Water Res.* 13: 241-248.
- Kato, S., J. McKinney and H. Matthews. 1980. Metabolism of symmetrical hexachlorobiphenyls isomers in the rat. *Toxicol. Appl. Pharmacol.* 53: 389-398.
- Kendall, A., and L. Walford. 1979. Sources and distribution of bluefish, *Pomatomus saltatrix*, larvae and juveniles off the east coast of the United States. *Fish. Bull.* 77: 213-227.
- Kimbrough, R., R. Squire, R. Linder, J. Strandberg, R. Montali and V. Burse. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. *J. Nat'l. Cancer Inst.* 55: 1453-1459.
- Klaunig, J., M. Lipsky, B. Trump and D. Hinton. 1979. Biochemical and ultrastructural changes in teleost liver following subacute exposure to PCB. *J. Environ. Pathol. Toxicol.* 2: 953-963.
- Krzeminski, S., J. Gilbert and J. Ritts. 1977. A pharmacokinetic model for predicting pesticide residues in fish. *Arch. Env. Contam. Toxicol.* 5: 157-166.
- Koeman, J., and M. Stasse-Wolthuis. 1978. Environmental toxicology of chlorinated-hydrocarbon compounds in the marine environment of Europe. *Comm. Eur. Comm.* EUR 5814 en. 137 pp.
- Kolbye, A., and C. Carr. 1984. The evaluation of the carcinogenicity of environmental substances. *Reg. Toxicol. Pharmacol.* 4: 350-354.
- Kuratsune, M., Y. Masuda and J. Nagayama. 1976. Some of the recent findings concerning Yusho. *Proc. National Conf. of Polychlorinated Biphenyls.* EPA-560/6-75-004. pp. 14-29.

- Lech, J., and J. Bend. 1980. Relationship between biotransformation and the toxicity and fate of xenobiotic chemicals in fish. *Env. Health Persp.* 34: 115-131.
- Lech, J., and R. Peterson. 1983. Biotransformation and persistence of polychlorinated biphenyls (PCBs) in fish. In: F. D'Itri and M. Kamrin (eds.). PCBs: Human and Environmental Hazards. Ann Arbor, Ann Arbor Science Press. pp. 187-202.
- Lee, G. and R. Jones. 1977. An assessment of the environmental significance and chemical contaminants present in dredged sediments dumped in the New York Bight. Rept. to U.S. Army Engineers, New York District, NY. 62 pp.
- Lieb, A., D. Bills and O. Sinhuber. 1974. Accumulation of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (Salmo gairdneri). *J. Agr. Food. Chem.* 22: 638-642.
- Lipsky, M., J. Klaunig and D. Hinton. 1978. Comparison of acute response to polychlorinated biphenyl in liver of the rat and channel catfish: A biochemical and morphological study. *J. Toxicol. Environ. Health* 4: 107-121.
- Lowman, F., T. Rice and F. Richards. 1971. Accumulation and redistribution of radionuclides by marine organisms. In: A. Seymour (ed.) Radioactivity in the Marine Environment. Washington, D.C. National Academy Press. pp. 161-199.
- Macek, K., S. Petrocelli and B. Sleight, III. 1979. Considerations in assessing the potential for, and significance of, biomagnification of chemical residues in aquatic food chains. In: L. Markins and R. Kimmerle (eds). Aquatic Toxicology. ASTM STP 667. Philadelphia, Amer. Soc. for Testing Materials. pp. 251-268.
- Mackay, D. 1982. Correlation of bioconcentration factors. *Env. Sci. Technol.* 16: 274-278.
- Mackay, D., and A. Hughes. 1984. Three-parameter equation describing the uptake of organic compounds by fish. *Environ. Sci. Technol.* 18: 439-441.
- MacLeod, W., L. Ramos, A. Friedman, D. Burrows, P. Prohaska, D. Fisher and D. Brown. 1981. Analysis of residual chlorinated hydrocarbons and aromatic hydrocarbons and related compounds in selected sources, sinks and biota of New York Bight. NOAA Tech. Memo. OMPA-6. NOAA/OMPA, Boulder, CO. 128 pp.
- Mancini, J. 1983. A method for calculating effects, on aquatic organisms, of time-varying concentrations. *Water Res.* 17: 1355-1362.
- Matsuo, M. 1980. A thermodynamic interpretation of bioaccumulation of Aroclor 1254 (PCB) in fish. *Chemosphere* 9: 671-675.
- Mauck, W., P. Mehrle and F. Mayer. 1978. Effect of the polychlorinated biphenyl Aroclor 1254 on growth, survival and bone development in brook trout (Salvelinus fontinalis). *J. Fish. Res. Bd. Canada* 35: 1084-1088.

- Mayer, F.L., P. Mehrle, and H.O. Sanders. 1977. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. *Arch. Env. Contam. Toxicol.* 5: 501-511.
- McKim, J., and E. Heath. 1983. Dose determinations for waterborne 2,5,2',5'-(14-C)-tetrachlorobiphenyl in two species of trout (Salmo gairdneri and Salvelinus fontinalis): A mass balance approach. *Toxicol. Appl. Pharmacol.* 68: 177-187.
- Mehrle, P., T. Haines, S. Hamilton, L. Ludke, F. Mayer and M. Ribick. 1982. Relationship between body contaminants and bone development in east-coast striped bass. *Trans. Amer. Fish. Soc.* 111: 231-241.
- Melancon, M., and J. Lech. 1976. Isolation and identification of a polar metabolite of tetrachlorobiphenyl from bile of rainbow trout exposed to 14-C tetrachlorobiphenyl. *Bull. Env. Contam. Toxicol.* 15: 181-187.
- Metcalf, R., J. Sanborn, P. Lu and D. Nye. 1975. Laboratory model ecosystem studies of the degradation and fate of radiolabelled tri-, tetra-, and pentachlorobiphenyl compared with DDE. *Arch. Env. Contam. Toxicol.* 3: 151-
- Mitchell, A., P. Plack and I. Thompson. 1977. Relative concentrations of 14-C DDT and of two polychlorinated biphenyls in the lipids of cod tissue after a single oral dose. *Arch. Env. Contam. Toxicol.* 6: 525-532.
- Monsanto Corp. 1978. Polychlorinated biphenyls (PCBs): A report on uses, environmental health effects and disposal. The Monsanto Corp., St. Louis. Mo. 29 pp.
- Moriarty, F. 1975. Exposures and Residues. In: F. Moriarty (ed.). Organochlorine Insecticides. Persistent Organic Pollutants. New York. Academic Press. pp. 29-72.
- Mueller, J., T. Gerrish and M. Casey. 1982. Contaminant inputs to the Hudson-Raritan estuary. NOAA Tech. Memo. OMPA-21. 135 pp.
- Mullin, M., C. Pochini, S. Safe and L. Safe. 1983. Analysis of PCBs using specific isomer high-resolution capillary gas chromatography. In: F. D'Itri and M. Kamrin (eds). PCBs: Human and Environmental Hazards. Ann Arbor, Ann Arbor Science Press. pp. 165-176.
- Mullin, M., C. Pochini, S. McCrindle, M. Romkes, S. Safe and L. Safe. 1984. High-resolution PCB analysis: Synthesis and chromatographic properties of all 209 PCB congeners. *Environ. Sci. Technol.* 18: 468-476.
- Nadeau, R., and R. Davis. 1976. Polychlorinated biphenyls in the Hudson River (Hudson Falls-Fort Edward, New York State). *Bull. Env. Contam. Toxicol.* 16: 436-444
- National Academy of Sciences (NAS). 1979. Polychlorinated Biphenyls. National Academy of Sciences, Washington, D.C. 182 pp.

- Nau-Ritter, G. 1980. The dynamics of PCB transfers among marine phytoplankton, clay particles and water. MS Thesis, State Univ. of NY at Stony Brook. Marine Sciences Res. Center. 117 pp.
- Neely, W., D. Branson and G. Blau. 1974. Partition coefficients to measure bioconcentration potential of organic chemicals in fish. Environ. Sci. Technol. 8: 1113-1115.
- Nelson, N. 1972. PCBs-Environmental Impact. Env. Res. 5: 249-362
- New York State Department of Environmental Conservation (NYDEC). 1981. Toxic substances in fish and wildlife: 1979 and 1980 Annual Reports. Tech. Rept. 81-1. NYDEC, Div. Fish and Wildlife. Albany, New York. 138 pp.
- Niimi, A., and B. Oliver. 1983. Biological half-lives of polychlorinated biphenyl (PCB) congeners in whole fish and muscle of rainbow trout (Salmo gairdneri). Can. J. Fish. Aquat. Sci. 40: 1388-1394.
- Nimmo, D., P. Wilson, R. Blackman, and A. Wilson, Jr. 1971a. Polychlorinated biphenyls absorbed from sediments by fiddler crabs and pink shrimp. Nature 231: 50-52.
- Nimmo, D., R. Blackman, A. Wilson, Jr. and J. Forester. 1971b. Toxicity and distribution of Aroclor 1254 in the pink shrimp Penaeus duorarum. Mar. Biol. 11: 191-197.
- Nisbet, I., and A. Sarofim. 1972. Rates and routes of transport of PCBs in the environment. Env. Health Perspect. 1: 21-38.
- Norcross, J., S. Richardson, W. Massman and E. Joseph. 1974. Development of young bluefish (Pomatomus saltatrix) and distribution of eggs and young in Virginian coastal waters. Trans. Am. Fish. Soc. 103: 477-497.
- Norstrom, R., A. McKinnon and S. DeFrietas. 1976. A bioenergetics based model for pollutant accumulation by fish. Simulation of PCB and methylmercury residue levels in Ottawa River yellow perch. J. Fish. Res. Bd. Canada 33: 248-267.
- O'Connor, J. 1982. Biological monitoring of PCBs in the Hudson River ecosystem. Rept. to N.Y. State Dept. Env. Conservation, Albany, New York. 202 pp.
- O'Connor, J. 1984a. PCBs: Dietary dose and burdens in striped bass from the Hudson River. Northeast. Env. Sci.: 3:153-159.
- O'Connor, J. 1984b. Polychlorinated biphenyl transport in coastal marine foodwebs. EPA-600/3-84-083. 98 pp.
- O'Connor, J., and R. Huggett. in press. Aquatic Pollution Problems, North Atlantic Coast, Including Chesapeake Bay. Aquatic Toxicology

- O'Connor, J., and J. Pizza. in press, a. Pharmacokinetic model for the accumulation of PCBs in Marine Fish. In: Oceanic Processes in Marine Pollution, Vol. 1. Capuzzo, J., and D. Kester (eds.) Biological Processes and Wastes in the Ocean. Krieger Pub.
- O'Connor, J., and J. Pizza. in press, b. PCB dynamics in Hudson River striped bass. III. Tissue disposition and routes for elimination. Estuaries.
- O'Connor, J., J. Klotz and T. Kneip. 1982. Sources, sinks and distribution of organic contaminants in the New York Bight ecosystem. In: G. Mayer (ed.) Ecological Stress and the New York Bight. Estuarine Research Federation, Charleston, S.C. pp. 631-653.
- Okumura, M., and S. Katsuki. 1969. Clinical observation on Yusho (Chlorobiphenyl poisoning). Fukuoka Acta Med. 60: 440-446.
- Oliver, B., and A. Niimi. 1985. Bioconcentration factors of some halogenated organics for rainbow trout: Limitations in their use for prediction of environmental residues. Environ. Sci. Technol. 19: 842-849.
- Pavlou, S., and R. Dexter. 1979. Distribution of polychlorinated biphenyls (PCBs) in estuarine systems. Testing the concept of equilibrium partitioning. Environ. Sci. Technol. 13:65-71.
- Pequegnat, W., B. James, E. Kennedy, A. Fredericks, R. Fay and F. Hubbard. 1980. Application of the biotal ocean monitor system to a study of the impacts of ocean dumping of dredged material in the New York Bight. TerEco Report to U.S. Army Engineers, NY District. 61 pp.
- Pizza, J. 1983. Pharmacokinetics and distribution of dietary PCBs in Hudson River striped bass, Morone saxatilis. Ph.D. Diss. New York University, New York. 109 pp.
- Pizza, J. and J. O'Connor. 1983. PCB dynamics in Hudson River striped bass. II. Accumulation from dietary sources. Aquatic Toxicol. 3: 313-327.
- Preston, B., and J. Allen. 1980. 2,2',5,5'-tetrachlorobiphenyl: Isolation and identification of metabolites generated by rat liver microsomes. Drug Metab. Disp. 8: 197.
- Reichardt, P., B. Chadwick, M. Cole, B. Robertson and D. Button. 1981. Kinetic study of biodegradation of biphenyl and its monochlorinated analogues by a mixed marine microbial community. Env Sci. Technol. 15: 75-79.
- Richardson, B., and J.S. Waid. 1982. Polychlorinated biphenyls (PCBs): An Australian viewpoint on a global problem. Search 13: 17-25.
- Ringer, R. 1983. Toxicology of PCBs in mink and ferrets. In: F. D'Itri and M. Kamrin (eds). PCBs: Human and Environmental Hazards. Ann Arbor. Ann Arbor Science Press. pp. 227-240.

- Risebrough, R., P. Reichle, S. Herman, D. Peakall and M. Kirven. 1968. Polychlorinated biphenyls in the global ecosystem. *Nature* 220: 1098-1102.
- Rubinstein, N., E. Lores and N. Gregory. 1983. Accumulation of PCBs, mercury and cadmium by Nereis virens, Mercenaria mercenaria and Palaemonetes pugio from contaminated harbor sediments. *Aquatic Toxicology* 3:249-260.
- Rubinstein, N., N. Gregory and W. Gilliam. 1984. Dietary accumulation of PCBs from a contaminated sediment source by a demersal fish species, Leiostomus xanthurus. *Aquatic Toxicol.* 5: 331-342.
- Safe, S. 1984. Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): Biochemistry, toxicology and mechanism of action. *CRC Crit. rev. Toxicol.* 13: 319-393.
- Samuelian, J., M. Moese and J. O'Connor. MS in review. PCB congeners in the tomcod (Microgadus tomcod) from the East River, NY. NYU Inst. Env. Med. Tuxedo, NY.
- Sangalang, G. H. Freeman and R. Crowell. 1981. Testicular abnormalities in Cod (Gadus morhua) fed Aroclor 1254. *Arch. Env. Contam. Toxcol.* 10: 617-626.
- Schroeder, R., and C. Barnes. 1983. Trends in polychlorinated biphenyl concentrations in Hudson River water five years after elimination of point sources. U.S. Geol. Survey Water Res. Invest. Rept. 83-4026. 28 pp.
- Schwartz, L. 1936. Dermatitis from synthetic resins and waxes. *Am. J. Pub. Health.* 26: 586-592.
- Schwartz, P., S. Jacobson, G. Fein, J. Jacobson and H. Price. 1983. Lake Michigan fish consumption as a source of polychlorinated biphenyls in human cord serum, maternal serum and milk. *Am. J. Public Health* 73: 293-296.
- Scura, E., and G. Theilacker. 1977. Transfer of the chlorinated hydrocarbon PCB in a laboratory marine food chain. *Mar. Biol.* 40: 317-325.
- Shaw, G., and D. Connell. 1980. Relationships between steric factors and bioconcentration of polychlorinated biphenyls (PCBs) by the sea mullet (Mugil cephalus Linnaeus). *Chemosphere* 9: 731-743.
- Shaw, G., and D. Connell. 1984. Physicochemical properties controlling polychlorinated biphenyl (PCB) concentrations in aquatic organisms. *Env. Sci. Technol.* 18: 18-23.
- Sherwood, M., A. Mearns, D. Young, B. McCain, R. Murchelano, G. Alexander, T. Heesen and Tsu-Kai. 1978. A comparison of trace contaminants in diseased fish from three areas. Rept. to NOAA/MESA New York Bight Project. Southern California Coastal Water Research Project, El Segundo, CA. 116 pp.

- Shimada, T., Y. Imai and R. Sato. 1981. Covalent binding of polychlorinated biphenyls to proteins by reconstituted monooxygenase system containing cytochrome P-450. *Chem. Biol. Interact.* 38: 29-44.
- Sittig, M. 1985. Handbook of Toxic and Hazardous Chemicals and Carcinogens. Park Ridge, N.J. Noyes Press. 950 pp.
- Sleight, S.D. 1983. Pathologic effect of PCBs in mammals. In: F. D'Itri and M. Kamrin (eds). PCBs: Human and Environmental Hazards. Ann Arbor, Ann Arbor Science Press. pp. 215-226
- Sloan, R., and R. Armstrong. 1982. PCB patterns in Hudson River fish. II. Migrant/Marine species. *Proc. Hudson River Env. Soc.* Hyde Park, N.Y. 53 pp.
- Sloan, R., K. Simpson, R. Schroeder and C. Barnes. 1983. Temporal trends toward stability of Hudson River PCB contamination. *Bull. Env. Contam Toxicol.* 31: 377-385.
- Sloan, R., M. Brown, R. Brandt, and C. Barnes. 1984. Hudson River PCB relationships between resident fish, water and sediment. *Northeast. Env. Sci.* 3:138-152.
- Smith, V., J. Spurr, J. Filkins and J. Jones. In press. Organochlorine contaminants of wintering ducks foraging on Detroit River sediments. *J. Great Lakes Res.* 1985.
- Spacie, A., and J. Hamelink. 1982. Alternative models for describing the bioconcentration of organics in fish. *Environm. Toxicol. Chem.* 1: 309-320.
- Spagnoli, J., and L. Skinner. 1977. PCBs in fish from selected waters of New York State. *Pestic. Mon. Journal.* 11: 69-87.
- Spies, R., J. Felton, and L. Dillard. 1984. Hepatic mixed-function oxidases in California flatfishes are increased in contaminated environments and by oil and PCB ingestion. *Mar. Biol.* 70: 117-127.
- Stainken, D., and J. Rollwagen. 1979. PCB residues in bivalves and sediments of Raritan Bay. *Bull. Env. Contam. Toxicol.* 23: 690-697.
- Stallings, D., and F. Mayer. 1972. Toxicity of PCBs to fish and environmental residues. *Env. Health Perspect.* 1: 159-164.
- Stegeman, J., P. Kloepper-Sams and J. Farington. 1986. Monooxygenase induction and chlorobiphenyls in the deep sea fish Coryphaenoides armatus. *Science* 231: 1287-1289.
- Stein, J., T. Hom and U. Varanasi. 1984. Simultaneous exposure of English sole (Parophrys vetulus) to sediment-associated xenobiotics: Part I- Uptake and disposition of 14-C polychlorinated biphenyls and 3-H Benzo(a)pyrene. *Mar. Env. Res.* 13: 97-119.

- Subcommittee on Health Effects of PCBs and PBBs, U.S. Dept. of Health, Education and Welfare. 1976. Final Report. 193 pp. + Appendices.
- Suflita, J., J. Robinson and J. Tiedje. 1983. Kinetics of microbial dehalogenation of haloaromatic substrates in methanogenic environments. *Appl. Env. Microbiol.* 45: 1466-1473.
- Swain, W.R. 1983. An overview of the scientific basis for concern with polychlorinated biphenyls in the great lakes. In: F. D'Itri and M. Kamrin (eds.) PCBs: Human and Environmental Hazards. Ann Arbor, Ann Arbor Press, pp. 11-48.
- Thomann, R. 1981. Equilibrium model of fate of microcontaminants in diverse aquatic food chains. *Can. J. Fish. Aquatic Sci.* 38: 280-296.
- Thomann, R., and J. St. John. 1979. The fate of PCBs in the hudson River ecosystem. *Ann. N.Y. Acad. Sci.* 320: 610-629.
- Thomann, R and J. Connolly. 1984. Model of PCBs in the Lake Michigan Lake trout food chain. *Environ. Sci. Technol.* 18: 65-71.
- Tucker, E., V. Saeger and O. Hicks. 1975. Activated sludge primary degradation of polychlorinated biphenyls. *Bull. Env. Contam. Toxicol.* 14: 705-712.
- Walker, C.R. 1976. The occurrence of PCB in the National fish and wildlife monitoring program. In: Proceedings of the National Conference on Polychlorinated Biphenyls. EPA-560/6-75-004. pp.161-176.
- Wasserman, M., D. Wasserman, S. Cucos and H. Miller. 1979. World PCBs map: Storage and effects in man and his biologic environment in the 1970's. *Ann. NY Acad. Sci.* 320: 69-124.
- Weaver, G. 1984. PCB contamination in and around New Bedford, Mass. *Env. Sci. Technol.* 18: 22A-27A.
- Weininger, D. 1978. Accumulation of PCBs by lake trout in Lake Michigan. Ph.D. Diss. Univ. Wisconsin-Madison. 232 pp.
- Westin, D., C. Olney and B. Rogers. 1985. Effects of parental and dietary organochlorines on survival and growth of striped bass larvae. *Trans. Am. Fish. Soc.* 114: 125-136.
- Woodwell, G., C. Wurster and P. Isaacsson. 1967. DDT residues in an east coast estuary: A case of biological concentration of a persistent pesticide. *Science* 156: 821-824.
- Young, D. 1984. Methods of evaluating pollutant biomagnification in marine ecosystems. In: H. White (ed.) Concepts in Marine Pollution Measurements. Maryland Sea Grant Press, College Park, pp. 261-278.

Table 1. Bioconcentration of various Aroclors in fishes. Bioconcentration factor calculated as the concentration in the fish divided by the concentration in the water.

Organism	Aroclor Mixture	Exposure Concentration ug/l	Exposure Time (days)	BCF	Reference
Channel Catfish (<u>Ictalurus punctatus</u>)	1248	5.8	77	56,000	Mayer et al., 1977
	1254	2.4	77	61,000	Mayer et al., 1977
Bluegill sunfish (<u>Lepomis macrochirus</u>)	1248	2-10	chronic	26,000 to 71,000	Stallings and Mayer, 1972
	1254	2-10	chronic		
Brook trout (fry) (<u>Salvelinus fontinalis</u>)	1254	6.2	118	46,000	Mauck et al., 1978
Spot (<u>Leiostomus xanthurus</u>)	1254	1.0	56	37,000	Hansen et al., 1971
Pinfish (<u>Lagodon rhomboides</u>)	1016	1.0	56	17,000	Hansen et al., 1974
Rainbow trout (<u>Salmo gairdneri</u>)	2,2',4,4' tetrachloro- biphenyl	1.6	5	29,000	Branson et al., 1975
Fathead minnow (<u>Pimephales promelas</u>)	1248	3.0	250	120,000	DeFoe et al., 1978
	1260	2.1	250	270,000	DeFoe et al., 1978

Table 2. Calculation of estimated PCB body burdens in fishes based upon equilibrium partitioning. The data used are from the New York Bight and adjacent marine waters.

	Minimum value	Maximum value
Water Column PCB Concentration (ng/l) (Note 1)	10	40
Particulate/Dissolved Ratio (Note 2)	0.67	0.67
Dissolved (available) PCB (ng/l)	6.7	27
Bioconcentration Factor (Note 3)	10,000	10,000
Expected Concentration in Fish (ug/g wet weight)	0.07	0.27
Observed Concentrations (ug/g) (Note 4)		
	Striped bass	0.6 - 3.8
	Winter flounder	0.1
	Mackerel	0.5 - 0.7
	Bluefish	0.7 - 3.6
	American eel	0.5 - 0.8
	Tautog	0.6

Note 1. Concentrations from Lee and Jones (1978), IEC (1979), Pequegnat et al. (1980) and MacLeod et al. (1981)

Note 2. Various authors suggest particulate/dissolved ratios from 0.0 to 1.0. The value of 0.67 was arrived at based upon data from Brown et al. (1982), Nau-Ritter (1980) and Pavlou and Dexter (1979).

Note 3. The value of 10,000 was based upon BCF data ranging from 16,000 to 61,000 for various species. Assuming some portion of the BCF was from feeding and water ingestion we concluded 10,000 to be a reasonable BCF approximation.

Note 4. Observed concentration data from O'Connor et al., 1982; NYSDEC, 1981; NJ DEP, 1982.

Table 3. Weight of fish, weight of stomach contents and calculation of PCB dose for a sample of age class 1+ striped bass collected at Canal Street, Manhattan. The ratio of stomach content to weight of fish was used to calculate a daily food ration and an expected rate of ingestion of PCB with the food. The calculated doses were based upon mean ration and mean PCB content of the food. The regression of PCB burden (B; total mass of PCB per fish) on mean daily dose (D; as ug/day) of PCB was $\log B = 1.68 \log D + 0.697$ (r-squared = 0.65).

FISH NUMBER	WEIGHT OF FISH (g)	WEIGHT OF FOOD (g)	RATIO FOOD/FISH WT.	PCB FISH (ug/g dry)	PCB FOOD (ug/g dry)	MEAN DOSE (ug/day)
3	8.6	0.68	0.02	2.3	9.5	2.6
5	8.5	0.96	0.03	1.5	6.0	2.6
6	10.5	0.71	0.02	2.5	7.1	3.2
7	10.9	0.87	0.02	2.8	3.7	3.3
8	12.5	1.35	0.03	2.7	6.3	3.8
9	11.4	1.41	0.03	4.9	7.5	3.5
10	11.9	1.70	0.03	3.4	4.8	3.7
11	12.6	1.45	0.03	4.9	3.2	3.9
12	14.3	1.50	0.02	3.9	9.0	4.4
13	16.7	1.37	0.02	4.7	8.1	5.1
14	21.5	1.06	0.03	2.6	8.1	6.6
15	12.1	2.07	0.02	3.3	6.0	3.7
16	21.3	1.49	0.02	13.1	10.3	6.5
17	19.4	1.44	0.02	5.7	4.7	6.0
18	20.1	1.68	0.03	11.7	5.7	6.2
19	25.9	2.71	0.02	2.6	6.8	8.0
20	23.1	2.13	0.02	2.8	4.1	7.1
21	25.5	2.12	0.03	12.1	8.4	7.8
22	24.0	1.57	0.01	3.7	3.8	7.4
23	26.2	3.33	0.03	5.0	3.5	8.1
24	33.0	2.81	0.02	15.7	7.4	10.1

Table 4. Distribution of ^{14}C -labeled Aroclor 1254 among tissues and organs of young-of the year striped bass measured 48 hours after administration of 1, 2, and 3 doses of PCB in the food. Each dose was 387 ng PCB. Data are presented as the mean of 5 fish (+ standard error of the mean).

Doses Given	Gill	Liver and Gallbladder	Alimentary Tract	Spleen and Heart	Head	Carcass	Epaxial Muscle	Whole Fish

1 (n = 5)								
Percent of retained burden	2.47 (0.38)	5.94 (0.66)	5.35 (0.41)	0.57 (0.08)	28.54 (1.00)	57.14 (1.38)	-	100
ug PCB/g (dry)	0.33 (0.06)	1.51 (0.17)	0.54 (0.06)	0.34 (0.06)	0.41 (0.04)	0.32 (0.03)	0.26 (0.04)	0.37 (0.04)
Percent of cumulative dose	1.92 (0.42)	4.45 (0.43)	4.00 (0.25)	0.42 (0.04)	21.70 (1.82)	46.14 (5.40)	-	76.24 (6.26)

2 (n = 5)								
Percent of retained burden	2.44 (0.18)	6.12 (0.88)	5.64 (0.15)	0.58 (0.11)	30.11 (1.12)	55.11 (1.90)	-	100
ug PCB/g (dry)	0.53 (0.10)	2.98 (0.23)	1.10 (0.11)	0.95 (0.13)	0.69 (0.15)	0.54 (0.09)	0.58 (0.08)	0.63 (0.11)
Percent of cumulative dose	1.61 (0.26)	3.89 (0.33)	3.66 (0.34)	0.36 (0.04)	19.48 (1.40)	36.21 (4.92)	-	65.23 (6.90)

3 (n = 5)								
Percent of retained burden	2.10 (0.22)	6.15 (0.34)	6.48 (1.11)	0.56 (0.04)	27.61 (0.41)	57.09 (1.82)	-	100
ug PCB/g (dry)	0.74 (0.07)	4.47 (0.58)	1.73 (0.16)	0.79 (0.04)	1.01 (0.08)	0.87 (0.07)	0.85 (0.07)	0.98 (0.08)
Percent of cumulative dose	1.25 (0.18)	3.63 (0.31)	3.83 (0.71)	0.34 (0.04)	16.25 (0.82)	33.60 (2.08)	-	58.91 (3.28)

Table 5. PCB concentrations determined in bluefish from the waters of New York, New Jersey and Massachusetts, 1979 through 1983.

Location of sampling	Dates	Number of fish	PCB Concentration
Peekskill, NY (a)	1979	16	3.15
New York Harbor (a)	1978	14	2.33
Fire Island, NY (a)	1978	15	1.03
Cold Spring Hr., NY (a)	1978	15	0.94
Eastern L.I. Sound(a)	1978	2	0.48
	1978	11	1.15
Herod Pt., NY (a)	1978	2	0.49
Orient Pt., NY (a)	1978	15	1.45
	1978	15	3.63
Great South Bay (a)	1979	16	0.68
Hudson River, NJ (b)	1975-76	4	3.44
Hudson River, NJ (b)	1981	2	1.78
Newark Bay, NJ (b)	1976-81	14	1.63
Raritan River, NJ (b)	1976-81	7	0.66
Raritan Bay, NJ (b)	1976-81	10	0.98
Coastal Bays...NJ (b)	1976-81	2	1.50
Delaware Bay (b)	1976-81	3	0.28
NJ Ocean Sites (b)	1976-81	21	0.37 - 0.82
Hudson River, NJ (c)	1982	5	3.29
Hudson River/NY Bay (c)	1983	3	4.03 - 9.61
Newark Bay, NJ (c)	1983	3	2.97
Arhtur Kill, NJ (c)	1983	1	1.51
New Bedford, Mass. (d)	1978-80	11	2.10

(a) Data from NYDEC, 1981; (b) data from Belton et al., 1983; (c) data from Belton et al., 1985; (d) data from Weaver, 1984

Figure 1. The distribution of bluefish on the Atlantic Coast of the U.S. Solid circles indicate the yield from trawl catches performed by the National Marine Fisheries Service. Lightly hatched area shows the general spawning area during the summer months, and the strongly hatched area shows areas of concentrated summer spawning. From Grosslein and Azarovitz, 1982.

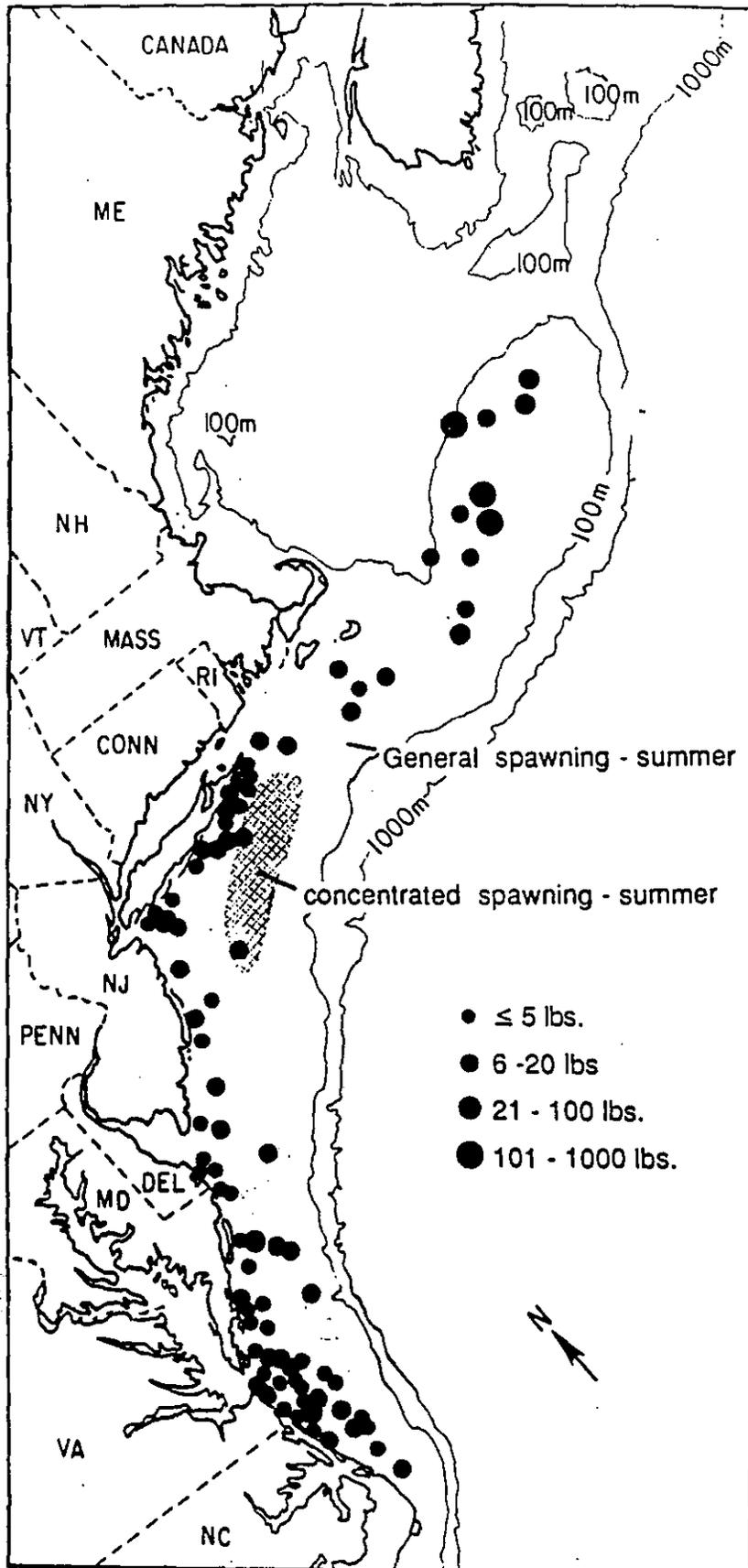


Figure 2. The structure of the biphenyl molecule, with ortho-, meta- and para- positions labelled for the primary and secondary rings.

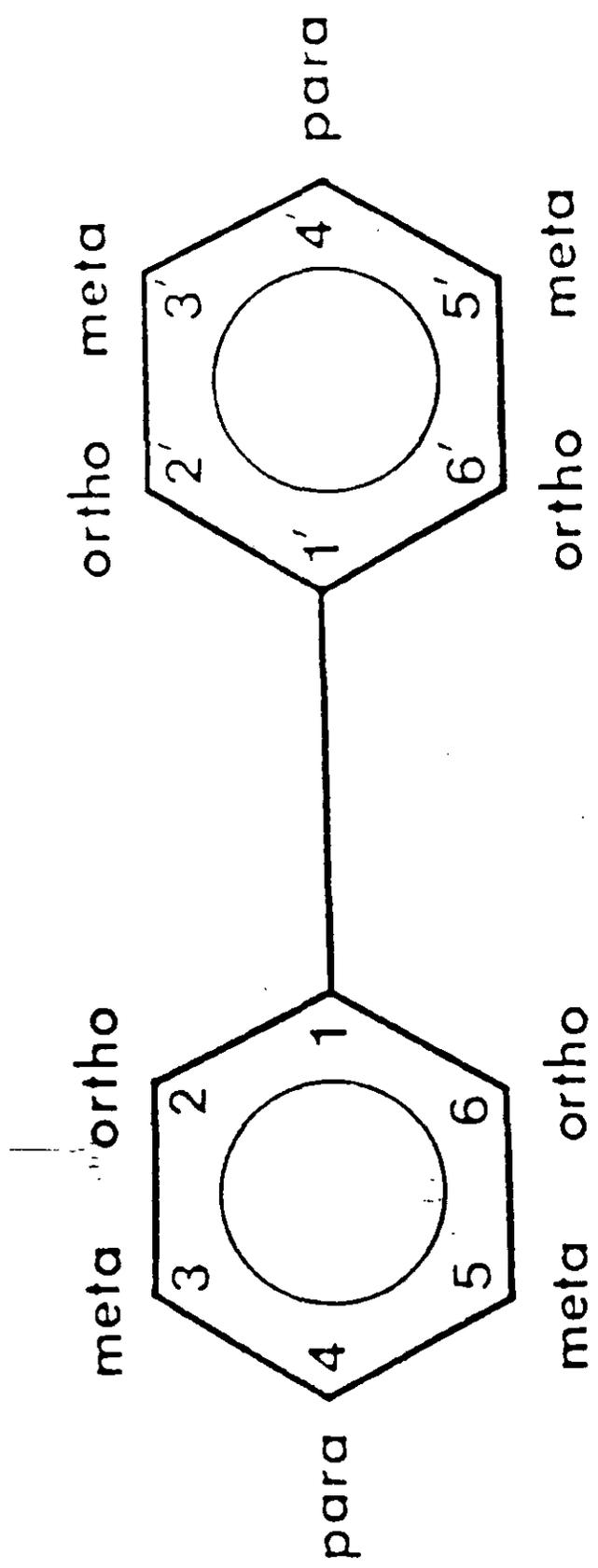


Figure 3. Schematic diagram for transport pathways for PCBs in the environment, with pathways from various manufacturing and applications processes to environmental media labelled. Note that the primary receptor for PCBs from all processes is water (W), whereas the least common transport end point is destruction (D). All transport pathways leading to air (A) have the potential for PCB transport to the water via surface runoff, wet fallout and dry fallout. Diagram from Nisbet and Sarofim, 1972.

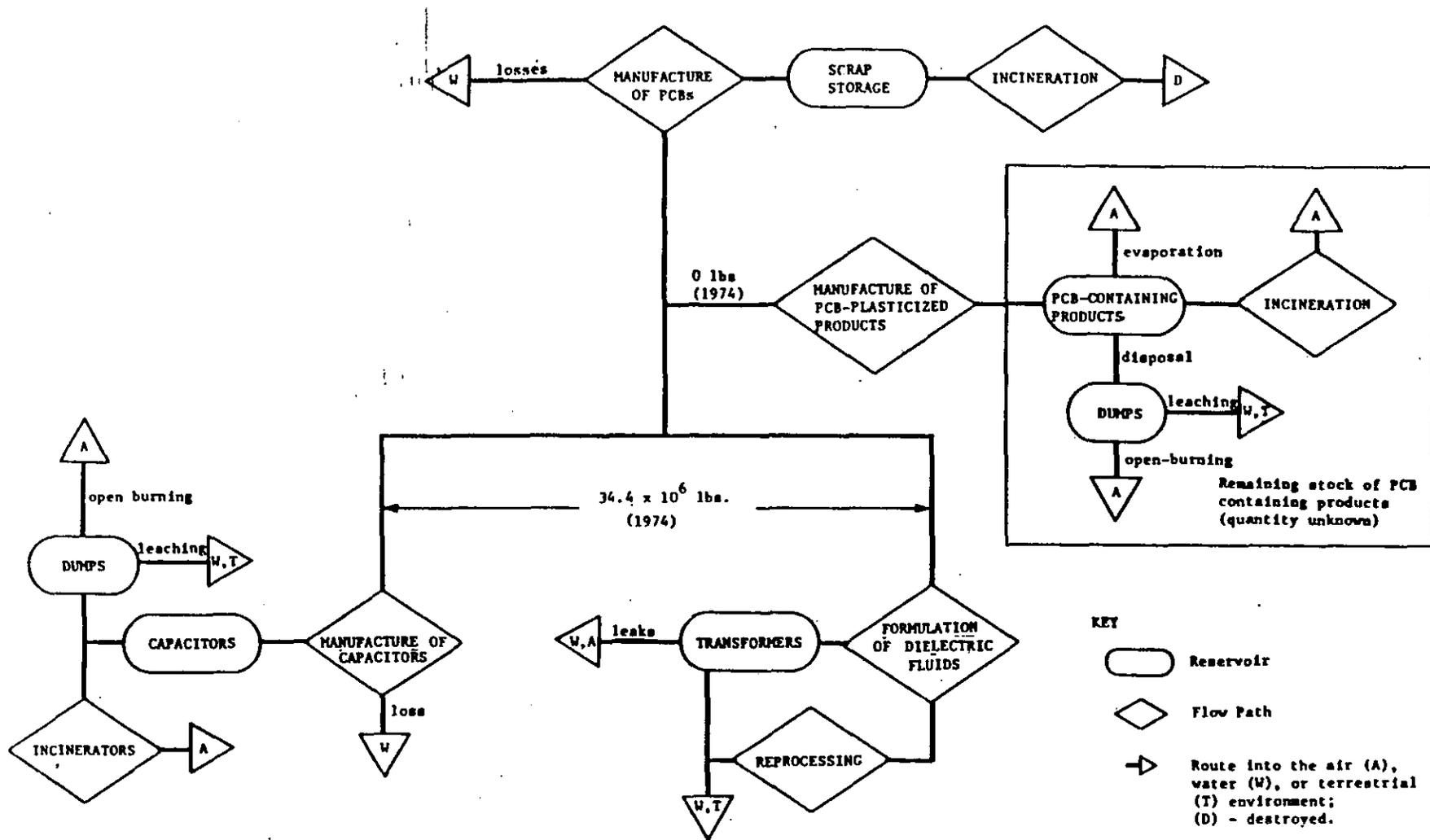


Figure 4. Schematic diagram of the transport of PCBs in a typical food chain showing the relationship of water uptake, assimilation from the food and the effects of metabolism. The schematic was prepared in conjunction with a second portion of the model (below the dotted line) describing transport of PCBs in the physical compartments of the ecosystem. From Thomann, 1981.

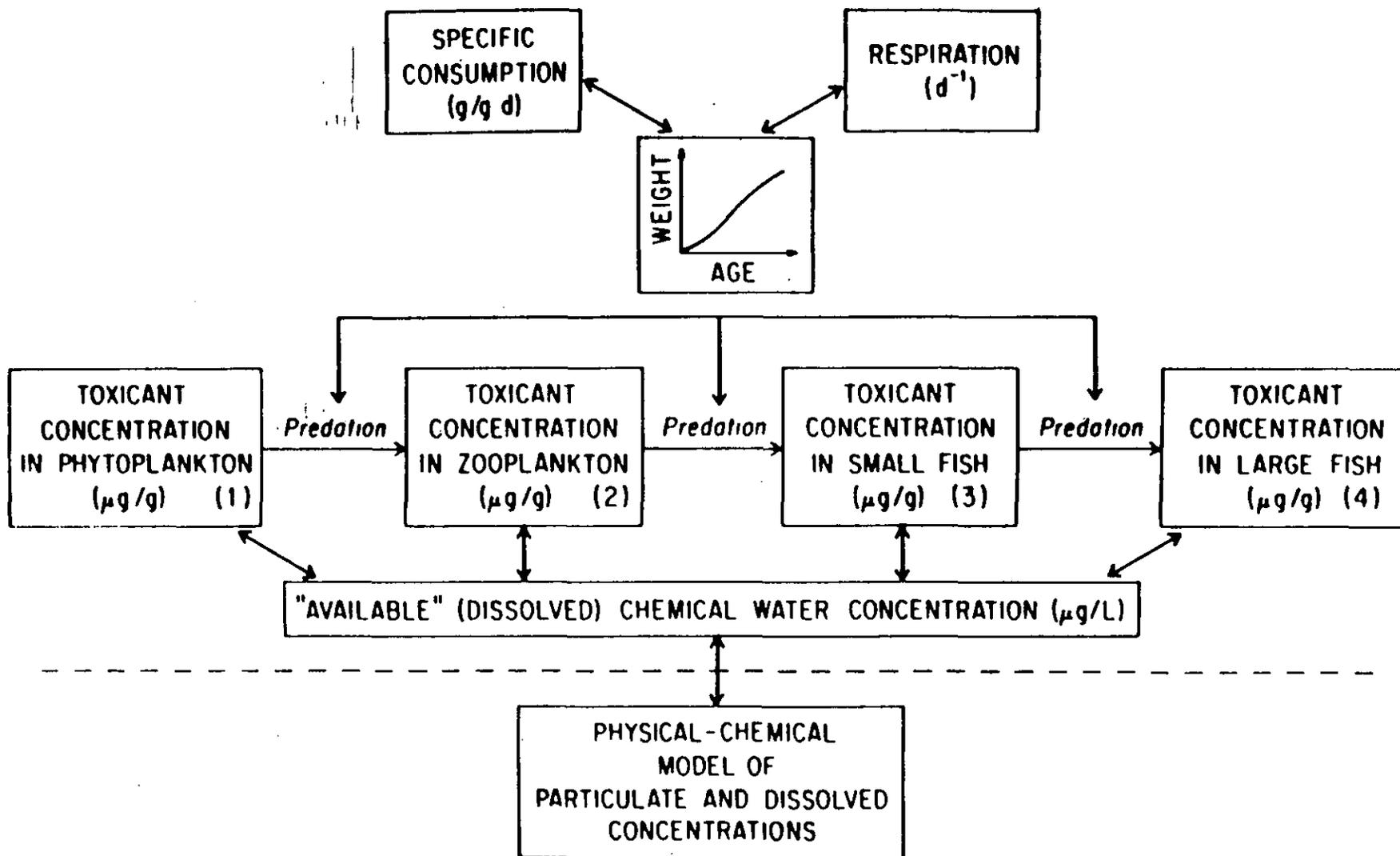


Figure 5. Removal of PCBs (Aroclor 1254) from the gut of striped bass dosed with radiolabelled compound and sampled at intervals for 5 days. PCBs are recorded as the percentage of the dose administered to the fish at time zero. Although more than 90% of the dose had been lost from the gut within 24 hours, the whole body samples showed that the majority of the dose had been distributed from the gut to the tissues. From Pizza and O'Connor, 1983.

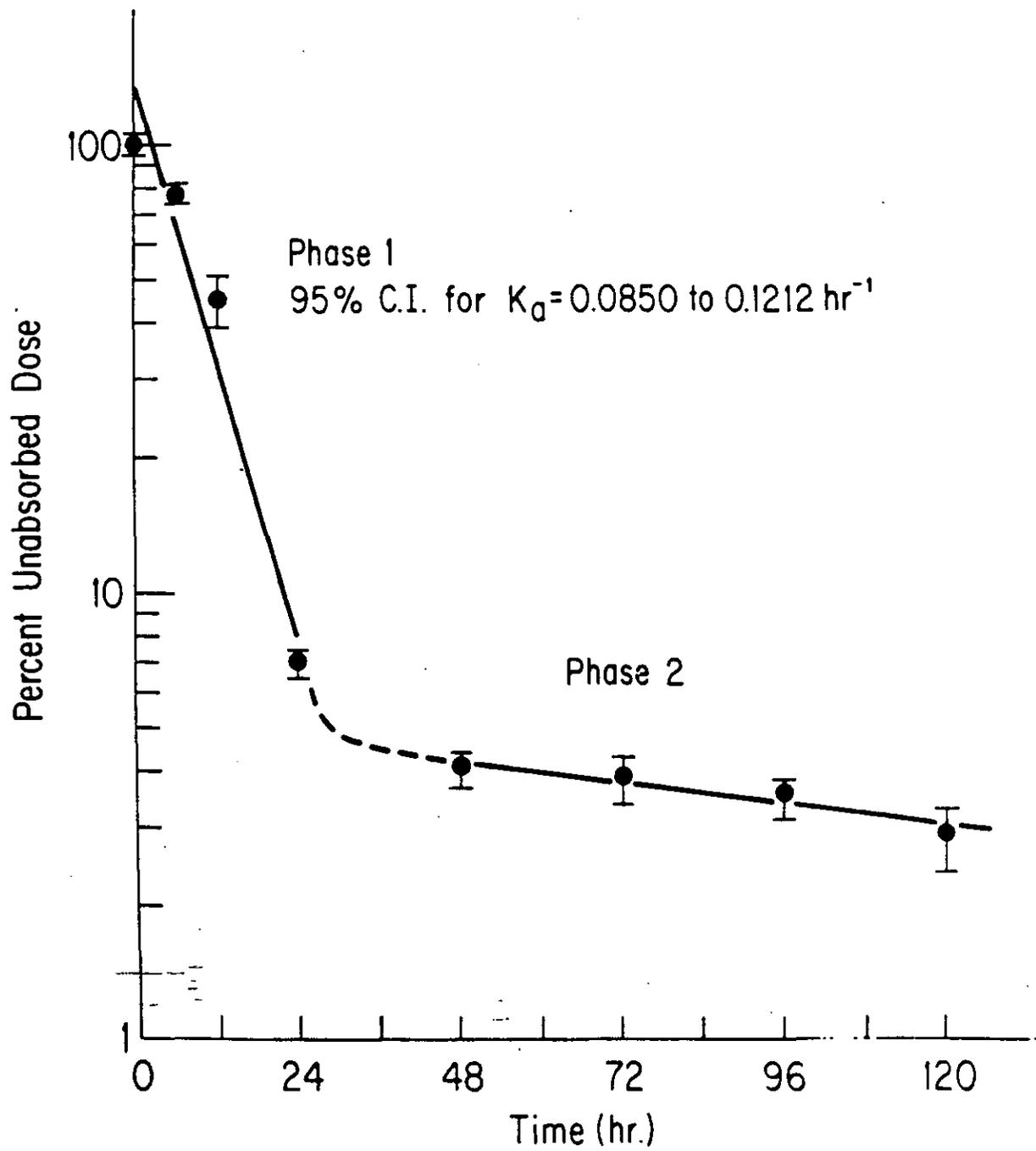
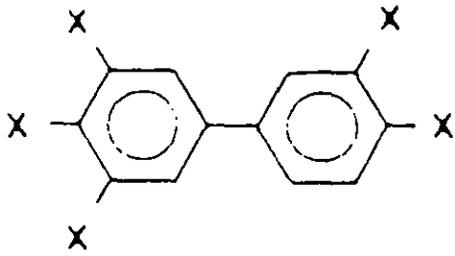
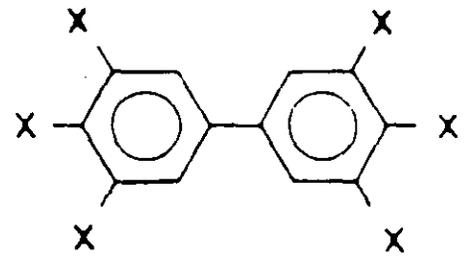


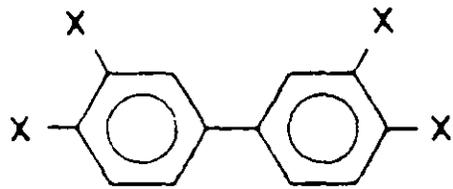
Figure 6. Approximate isostereomes of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as halogenated biphenyls, halogenated azobenzenes and halogenated dibenzofurans. In all cases the molecular size, shape and planarity are sufficiently similar to TCDD to lead to the conclusion that the compounds should have similar biochemical effects. From Safe, 1984.



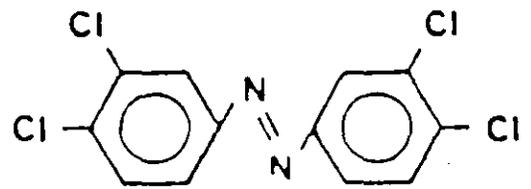
3,3',4,4',5 - Pentahalobiphenyl



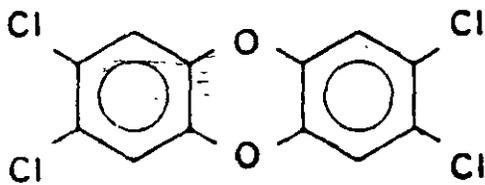
3,3',4,4',5,5'- Hexahalobiphenyl



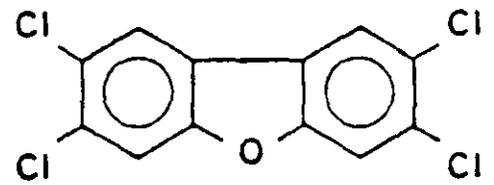
3,3',4,4' - Tetrahalobiphenyl



3,3',4,4' - Tetrachloroazobenzene

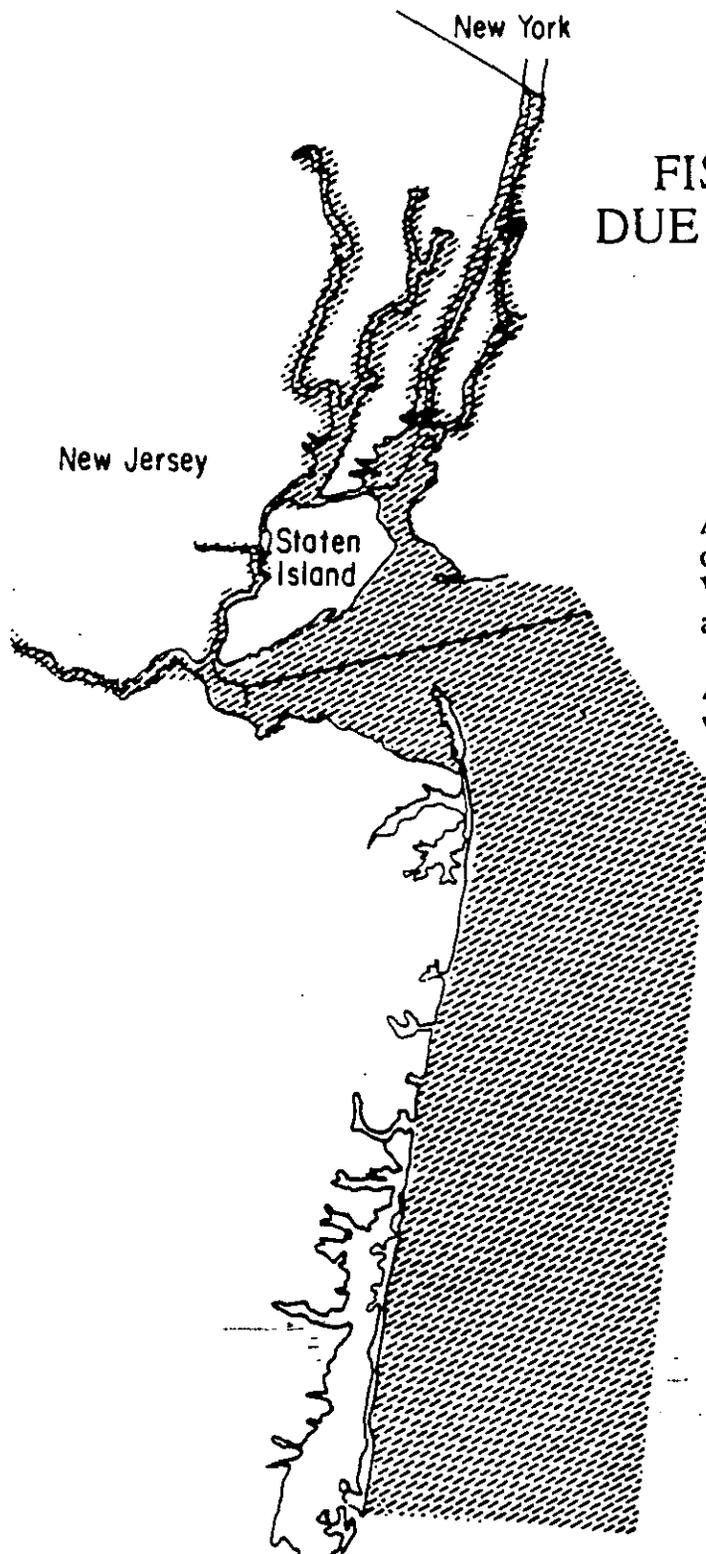


2,3,7,8 - Tetrachlorodibenzo-p-dioxin



2,3,7,8 - Tetrachlorodibenzofuran

Figure 7. Fishing advisory areas in the vicinity of Metropolitan New York and New Jersey. The advisory from the State of New Jersey warns against consuming fish from coastal marine waters due to their high PCB concentrations. In 1986 New York State banned all possession (recreational and commercial) of striped bass in all marine waters of the state due to high PCB concentrations. Figure from Belton et al., 1983.



FISHING ADVISORY AREA DUE TO PCB's IN FISH TISSUE

ADVISORY AREA

Advisory in effect to limit consumption of STRIPED BASS, BLUEFISH, WHITE PERCH, WHITE CATFISH, and AMERICAN EEL.

Advisory area includes the following waterways and tributaries:

- Hudson River
- Upper New York Bay
- Newark Bay
- Tidal Passaic River
- Tidal Hackensack River
- Arthur Kill
- Kill Van Kull
- Tidal Raritan River
- Raritan Bay
- Sandy Hook Bay
- Lower New York Bay

STRIPED BASS and BLUEFISH advisory includes Offshore Waters for Northern Coastal Area.

AMERICAN EEL advisory includes all waterways statewide.