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

A Report to

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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, PBCs 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 survay 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 synthsized 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 chlorbiphenyls 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-Wolthius, 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 recyle 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 Corneiussen, 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 relatonship 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 pom 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 organsisms 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 suface 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 ^{14}C labelled 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 and 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-labelled 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 occured 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 acuumulate 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 pprocess). 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

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

where M_o is the quantitiy 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

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

where X_o 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 pleateau 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; Matsuo, 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 determinants 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-labelled 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; Niimi 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 chromatigraphically 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. Since 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 iso-stereomers" of 2,3,7,8-tetrachlorodibenzodioxin (Figure 6) 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 sustantial differences; Crangon 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 (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 blufish from the Hudson River to New York Bay were likely to exceed 4.0 ppm, and that PCB levels in blufish 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 7, 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 blufish 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:

$$\text{Log } B = 2.0 \log D - 1.0$$ (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:

PCB in food (ug/g)	PCB in bluefish (ug/g)
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.

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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 (Ictalurus punctatus)	1248	5.8	77	56,000	Mayer et al., 1977
	1254	2.4	77	61,000	Mayer et al., 1977
Bluegill sunfish (Lepomis macrochirus)	1248	2-10	chronic	26,000 to 71,000	Stallings and Mayer, 1972
	1254	2-10	chronic		
Brook trout (fry) (Salvelinus fontinalis)	1254	6.2	118	46,000	Mauck et al., 1978
Spot (Leiostomus xanthurus)	1254	1.0	56	37,000	Hansen et al., 1971
Pinfish (Lagodon rhomboides)	1016	1.0	56	17,000	Hansen et al., 1974
Rainbow trout (Salmo gairdneri)	2,2',4,4' tetrachloro-biphenyl	1.6	5	29,000	Branson et al., 1975
Fathead minnow (Pimephales promelas)	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-labelled 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).

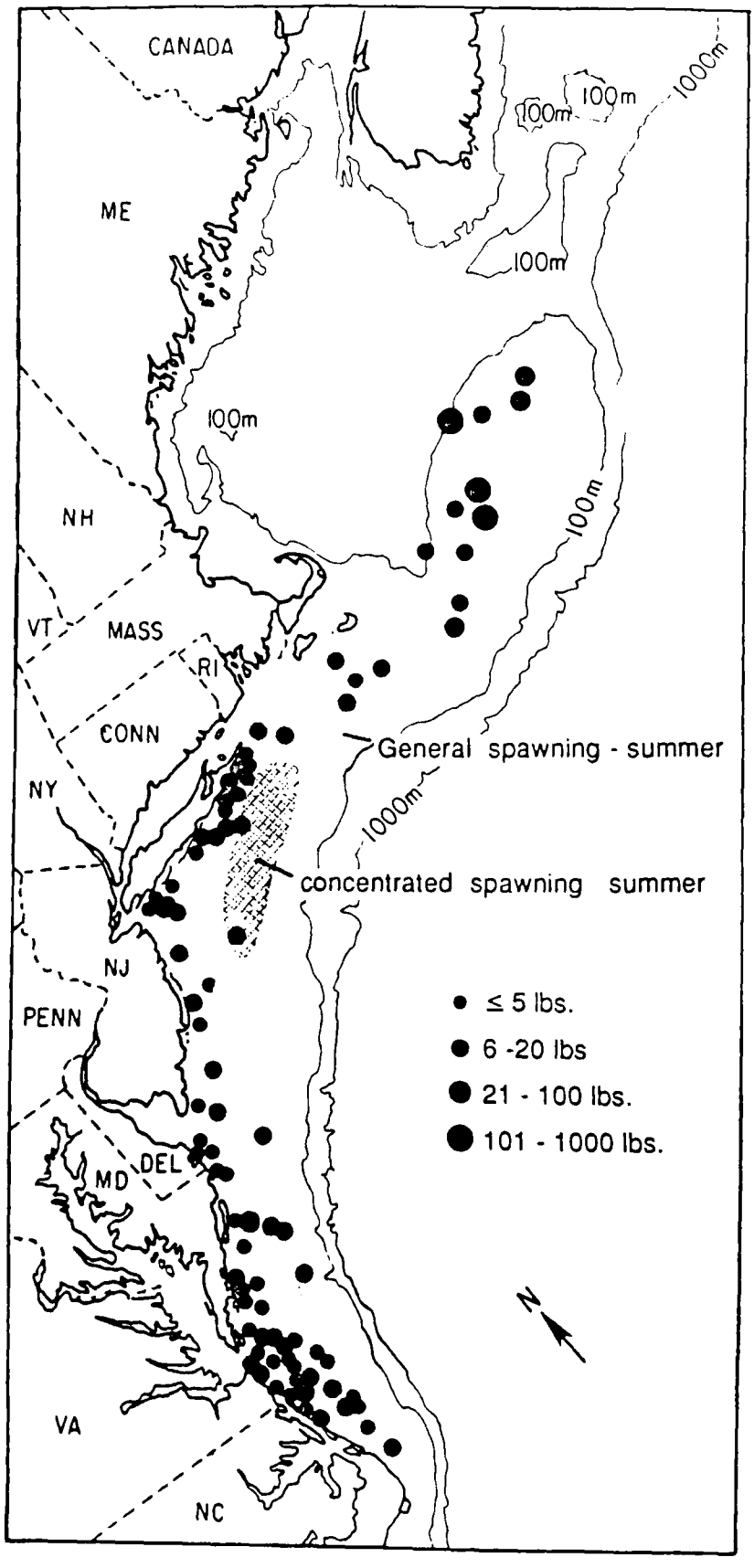
Doses Given	Gill	Liver and Gallbladder	Alimentary Tract	Spleen and Heart	Head	Carcass	Epaxial Muscle	Whole Fish
1 (n = 5)								
Percent of retained burden	2.47 (0.38)	5.94 (0.66)	5.35 (0.41)	0.57 (0.08)	28.54 (1.00)	57.14 (1.38)	-	100
ug PCB/g (dry)	0.33 (0.06)	1.51 (0.17)	0.54 (0.06)	0.34 (0.06)	0.41 (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)	0.53 (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.


CANADA
ME
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1000m
General spawning - summer
concentrated spawning summer
≤ 5 lbs.
6 -20 lbs
21 - 100 lbs.
101 - 1000 lbs.
N

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

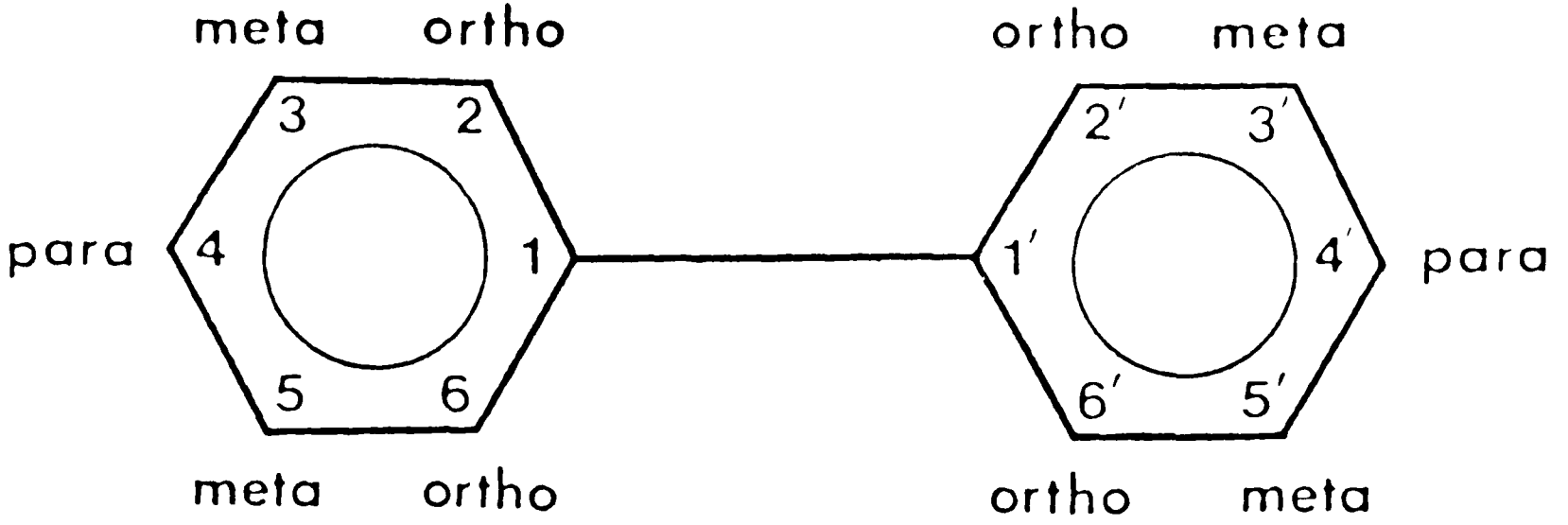
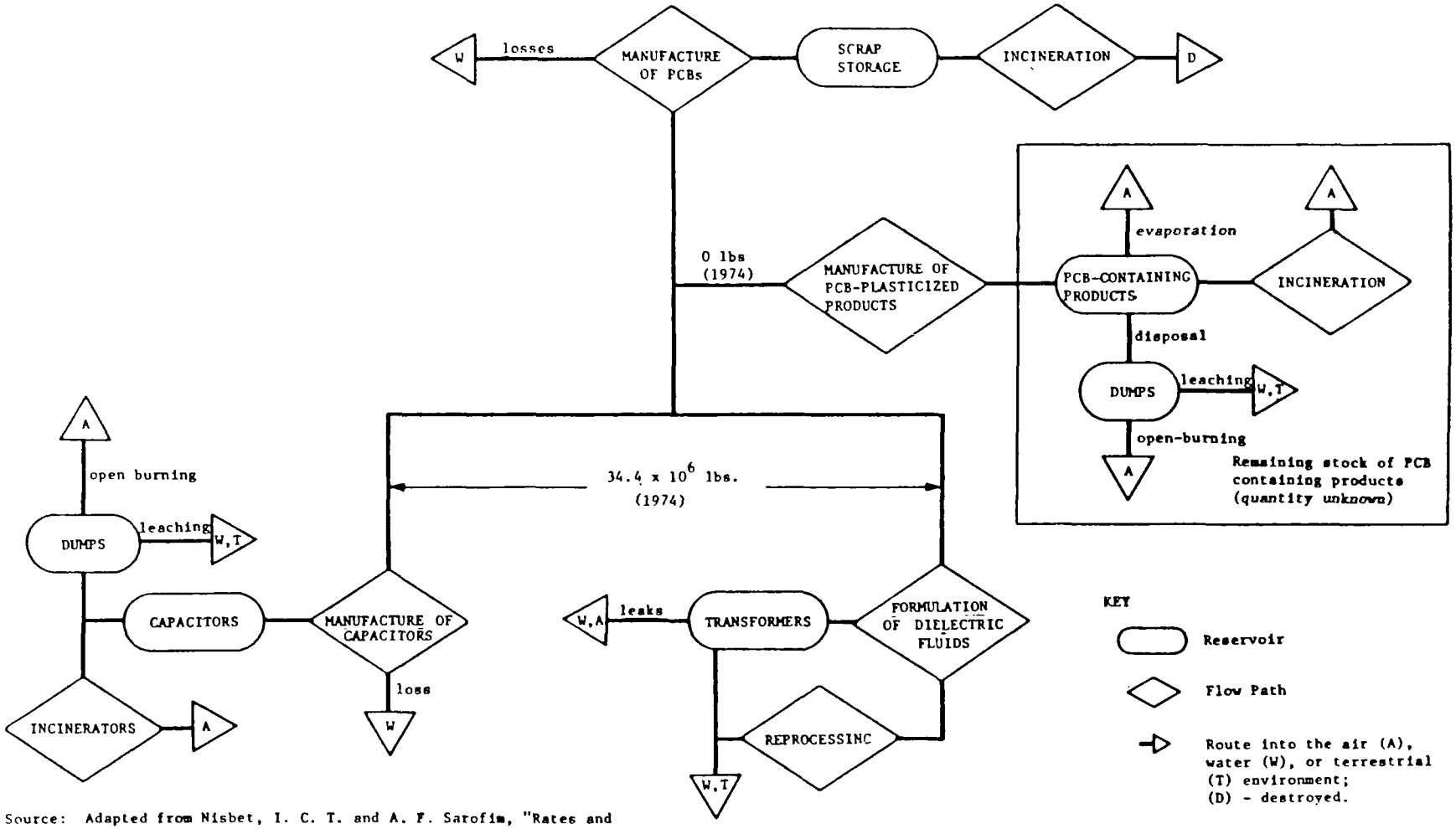


FIGURE 2. The structure of biphenyl.

Figure 3. Schematic diagram of 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.



Source: Adapted from Nisbet, I. C. T. and A. F. Sarofim, "Rates and Routes of Transport of PCBs in the Environment," Environmental Health Perspectives Exp. 1, 21-38, 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 (blow the dotted line) describing transport of PCBs in the physical compartments of the ecosystem. From Thomann, 1981.

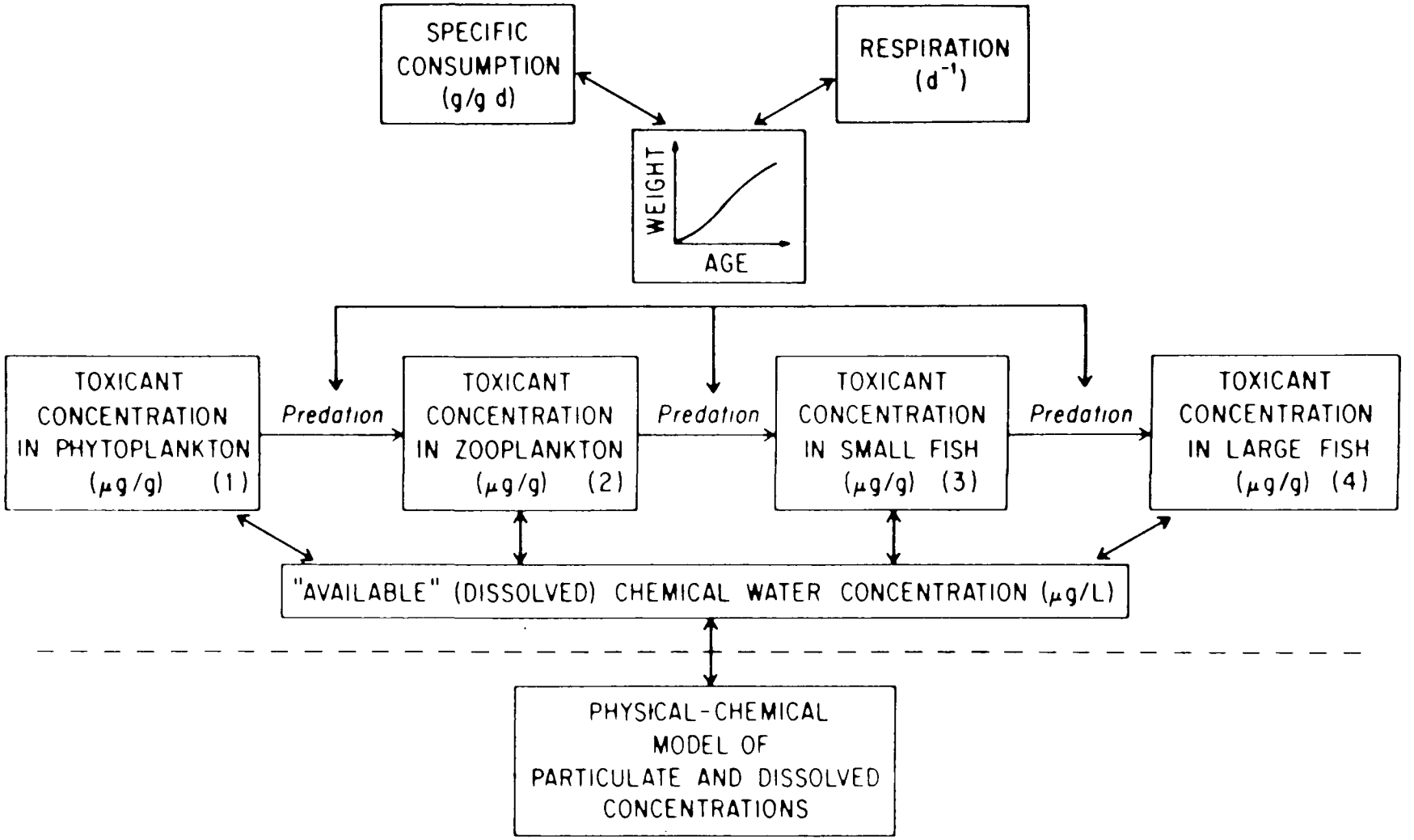

SPECIFIC CONSUMPTION (g/g d)
RESPIRATION (d^{-1})
WEIGHT
AGE
TOXICANT CONCENTRATION IN PHYTOPLANKTON (μg/g) (1)
Predation
TOXICANT CONCENTRATION IN ZOOPLANKTON (μg/g) (2)
Predation
TOXICANT CONCENTRATION IN SMALL FISH (μg/g) (3)
Predation
TOXICANT CONCENTRATION IN LARGE FISH (μg/g) (4)
"AVAILABLE" (DISSOLVED) CHEMICAL WATER CONCENTRATION (μg/L)
PHYSICAL-CHEMICAL MODEL OF PARTICULATE AND DISSOLVED CONCENTRATIONS

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.

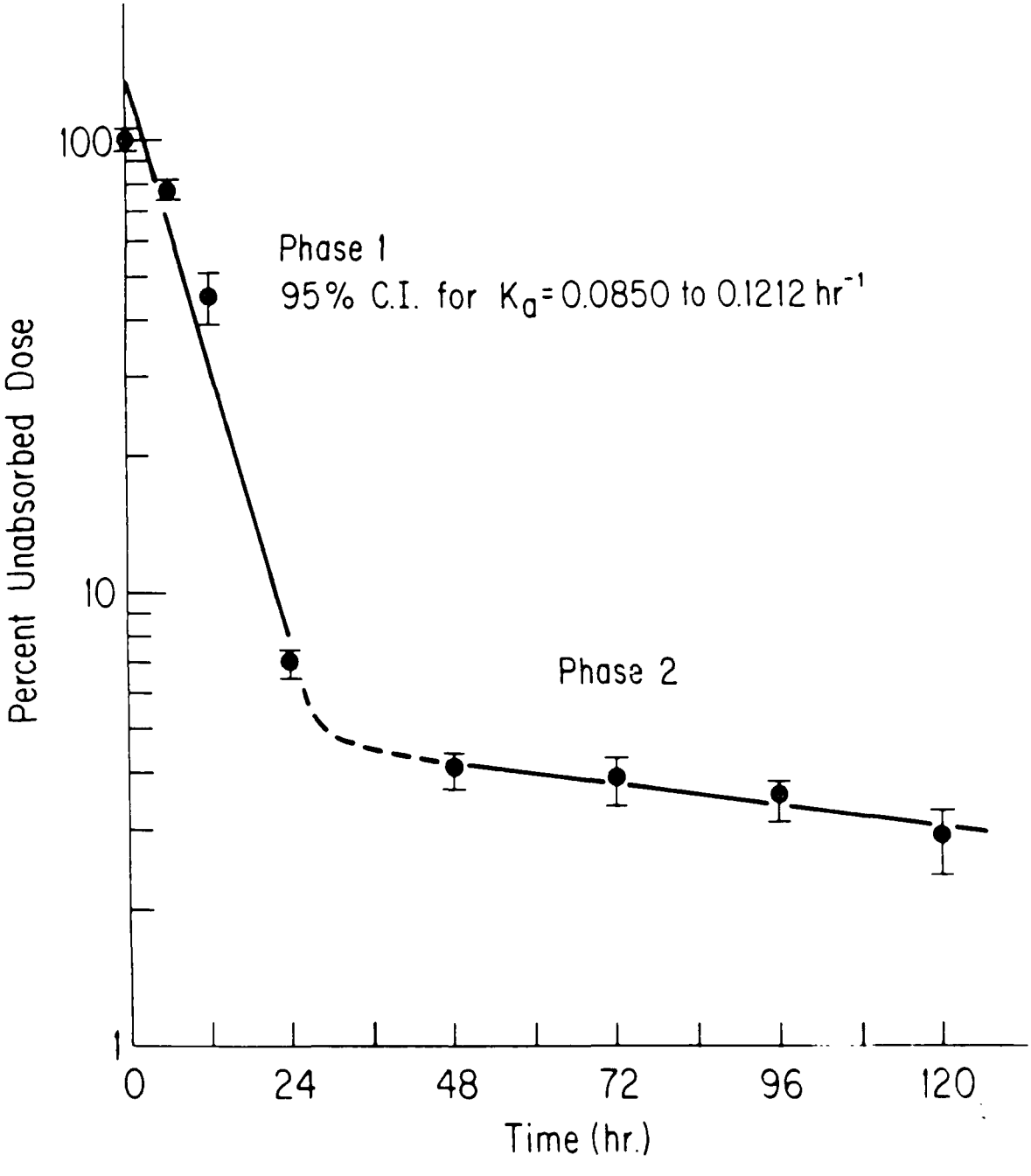
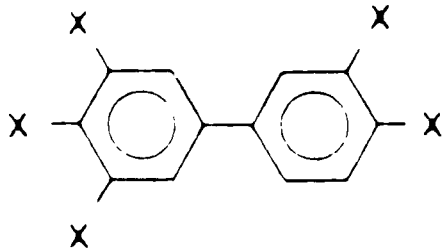
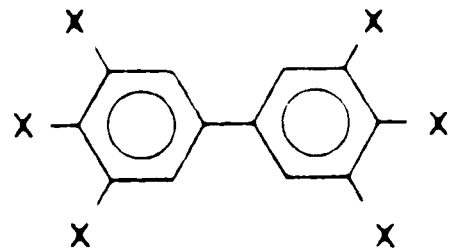

Percent Unabsorbed Dose
100
10
1
Phase 1
95% C.I. for K_a = 0.0850 to 0.1212 hr^{-1}
Phase 2
0
24
48
72
96
120
Time (hr.)

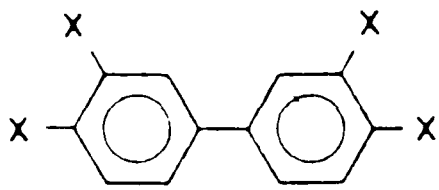
Figure 6. Approximate isostereomers of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) as halogenated biphenyls, halogenated azobenzenes and halogenated dibenzofurans. In all cases the molecular size, shape and planarity are sufficiently similar to TCDD to lead to the conclusion that the compounds should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



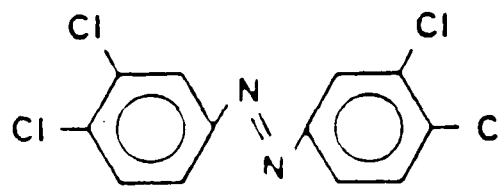
3,3',4,4',5 - Pentahalobiphenyl



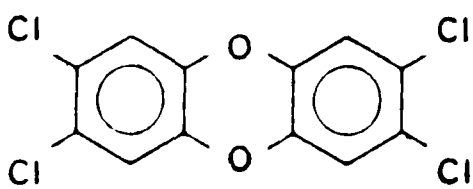
3,3',4,4',5,5' - Hexahalobiphenyl



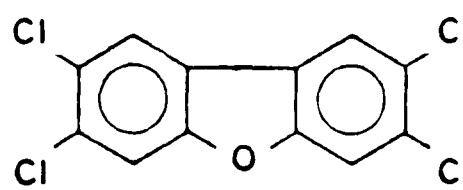
3,3',4,4' - Tetrahalobiphenyl



3,3',4,4' - Tetrachloroazobenzene

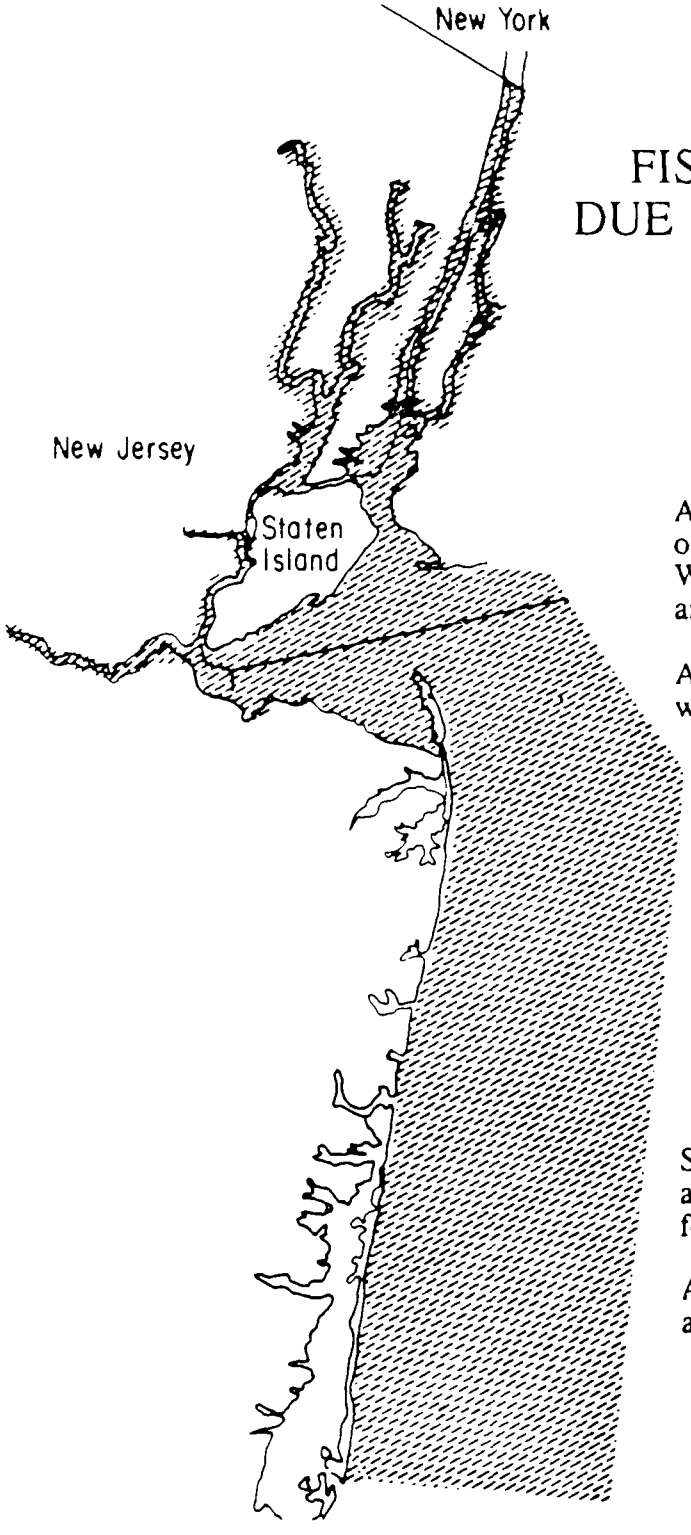


2,3,7,8 - Tetrachlorodibenzo-p-dioxin



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 Costal Area.

AMERICAN EEL advisory includes all waterways statewide.