POLYCHLORINATED BIPHENYLS

CRITERIA:

Aquatic Life - 0.001 ug/l in freshwater;
0.01 ug/l in marine water - p7 1016 vs p6 other

INTRODUCTION:

Polychlorinated biphenyls (PCB's) are a class of compounds produced by the chlorination of biphenyls and are known commercially as Aroclors(R). The degree of chlorination determines their chemical properties and generally their composition can be identified by the numerical nomenclature, e.g. Aroclor 1242, Aroclor 1254, etc. The first two digits represent the [redacted] and the last two digits the average weight percent of chlorine (NTIS, 1972).

Identification of PCB's in the presence of organochlorine pesticides such as DDT and DDE has been extreemely difficult in the past because of their similar chromatographic characteristics (Risebrough et al., 1968). In PCB analysis today, however, the interference of organochlorine hydrocarbons is overcome by sequential column chromatography on Florsil and silica gel (Armour and Burke, 1970; FDA, 1971). Gas-liquid chromatography with highly sensitive and selective detectors has been employed successfully in the sub-micron range (Nebecker and Puglisi, 1971).

PCB compounds are only slightly soluble in water; highly soluble in fats, oils and nonpolar liquids, and highly resistant to both heat and biological degradation (NTIC, 1972). Typically, the specific gravity, boiling point, and melting point of PCB's increase with chlorine content as exemplified in the following table of vaporization rates.

% Chlorine	Vaporization Rate at 100° C ($g/cm^2/hr$
21	.00174
32	.00084
42	.000338
48	.000152
54	.000053
60	.000013
62	.000009

PCB's are relatively non-flammable, have superior cooling, insulating, and dielectric properties and principally are used in the electrical industry. In 1970, the sale of PCB-containing products as additives to paints, plasticizers, pesticides and the encapsulating of carbonless carbon paper was discontinued by the producer. The sale of PCB compounds for heat transfer fluids was discontinued in 1971. Accordingly, the only remaining use of PCB compounds is for capacitors and transformers (NTIS, 1972).

As a consequence of past wide usage of PCB's over a long period, they are ubiquitous and have been found to have adverse environmental effects. Even a total cessation of the manufacture and use in PCB's would not be expected to result in disappearance of the material, and extinction from the environment is expected to take many years.

RATIONALE

Exposure to PCB is known to cause skin lesions (Schwartz and Peck, 1943) and to increase liver enzyme activity that may have a secondary effect on reproductive processes (Risebrough, 1968; Street et al., 1968; Wasserman et al., 1970). It is

not clear whether the effects are due to the PCB's or their contaminants, the chlorinated dibenzofurans, that are highly toxic (Bauer et al., 1961; Schulz, 1968; Varrett, 1970). While chlorinated dibenzofurans are a by-product of PCB production, it is not known whether they are also produced by the degradation of PCB's (NAS, 1974).

An epidemiological study on severe poisoning by rice oil contaminated with PCB's in 1967 indicated that about 0.5 grams ingested over a period of approximately one month was sufficient to cause "Yusho" disease. Many of those affected showed no signs of relief after about three years (Kuratsume et al., 1969).

It is estimated on the basis of 194 samples that 41 to 45 percent of the general United States population may have PCB levels of 1.0 mg/kg or higher (net weight) in adipose tissue (Price and Welch, 1972). It appears, therefore, that PCB may accumulate in the human body. On this basis, it can be calculated that a daily intake of 0.02 mg would require about 70 years to be toxic. Applying a safety factor of 10 would permit a daily intake of 0.002 mg, and assuming a two-liter per day intake, suggests a permissable concentration in water to be 0.001 mg/l (NAS, 1974).

As a consequence of widespread pollution, appreciable PCB residues exist in fish. Analyses of 40 fish from the 1970 National Pesticide Monitoring Program indicated only one to contain less than 1 ug/g PCB with the ten highest residue levels ranging from 19 ug/g to 213 ug/g whole body weight (NAS, 1974). Studies of the Milwaukee River revealed ambient water concentrations of 2.0 to 2.8 ug/l and residue levels in fish as high as 405 ug/g (Veith and Lee, 1971). Open water Lake Michigan concentration levels have been reported to be less than

0.01 ug/l with mean residue levels in large coho salmon of about 15 ug/g (Veith, 1973). By contrast, residues in ocean fish generally have been below 1 ug/g (NAS, 1974). Based on Lake Michigan data, which indicate that at a level of 0.01 ug/l, the fish tissue concentration exceeds a level found to be non-hazardous to man, a criterion of 0.001 ug/l in freshwater appears warranted.

Polychlorinated Biphenyls have been found to be toxic to aquatic organisms and to exhibit a wide variation in toxicity as a function of the organism and the chlorine content of the PCB. Midge reproduction was reduced at 0.45 ug/l of Aroclor 1254 and reproduction of Daphnia magna was reduced at 1.3 ug/l (Nebeker and Puglisi, 1974). The highest concentration of Aroclor 1248 having no chronic effect on the fathead minnow was about 0.3 ug/l (Brungs, 1974).

Observed PCB bio-concentrations in aquatic organisms range over six orders of magnitude. Bluegills exposed to Aroclors 1248 and 1254, exhibited a bioaccumulation factor of 7.1×10^4 (Stalling and Mayer, 1972). The bioaccumulation factor for gizzard shad in the Saginaw River (Michigan) varied between 0.6×10^5 and 1.5×10^5 for Aroclor 1254 (Michigan Water Res. Comm., 1973). Fathead minnows in water containing 0.3 ug/l of Aroclor 1245 concentrated it 3×10^5 times (Brungs, 1974). Applying Lake Michigan coho salmon residue data to Lake Michigan ambient water data taken at the same time and location suggests a bioaccumulation factor of 1.5×10^6.

The Food and Drug Administration guideline for protecting the health of human consumers of fish is 5 ug/g in tissue residue of the fish. A residue level of 2 ug/g in fish consumed by commercial ranch mink has been shown to prevent survival of offspring (Ringer et al., 1972). Reproduction was almost totally eliminated in ranch mink fed a beef diet containing 0.64 ug/g of

Aroclor 1254 (Platonow and Kalstad, 1973). This suggests that a mink-food tissue level of not more than 0.5 ug/g would be required to protect the wildlife consumer.

Surveys of Escambia Bay (Florida) during the period September 1969 to December 1971 have produced data on the pathways and effects of PCB's in the estuarine and marine environments. Through the major PCB source, accidental leakage from a PCB manufacturing plant has been terminated, residues continue to appear in aquatic organisms of the bay (Duke, 1974).

The sediment reservoir of Aroclor 1254 is thought to be a continuing source of this PCB to biota of Escambia Bay. After periodic sampling and analysis of both filtered and unfiltered water from homogeneous surface to bottom water columns, little recoverable Aroclor 1254 was found in the filtered samples. These observations suggest that most of the chemical was attached to suspended particles.

The initial survey of Escambia Bay biota revealed fish, shrimp, and crabs with levels as high as 12 ug/g. Higher levels were detected in higher trophic levels than shrimp, which could implicate a chain transfer from sediment to large animals. Median PCB concentrations in whole fish of eight species from Long Island Sound obtained in 1970 were reported to be in the order of 1 ug/g, as were comparable concentrations off the coast of Southern California.(Hays and Risebrough, 1972; Risebrough, 1969).

Few data existed prior to 1969-1971 on the effects of PCB's on marine organisms. From the Escambia Bay data, which include flow-through bioassays with residue analyses where possible, the following conclusions were reached: (1) all of the Aroclors tested are acutely toxic for certain estuarine organisms;

(2) bioassays lasting longer than 96 hours demonstrated that acute toxicity tests underestimate toxicities of some Aroclors and that Aroclor 1254 is toxic to commercial shrimp at less than 1 ug/l; (3) fish, particularly sheephead minnows, are extremely sensitive to Aroclor 1254 with 0.1 ug/l being lethal to fry; and, (4) acute toxicity of Aroclor 1016 to estuarine organisms is similar to the toxicity of other Aroclors but appears less toxic to fish in long-term exposures than does Aroclor 1254 (Duke, 1974).

Oysters were sensitive to Aroclor 1260 with growth diminished by 44 percent in 10 ug/l and by 52 percent in 100 ug/l. Approximately 10 percent of the pink shrimp died in 100 ug/l, but no apparent effects on pinfish were noted at 100 ug/l.

Aroclor 1254 had no apparent effect on juvenile pinfish at 100 ug/l in 48-hour flow-through tests, but killed 100 percent of the pink shrimp. At 100 ug/l of Aroclor 1254 for 96 hours, shell growth was inhibited but decreased only 41 percent at levels of 10 ug/l.

The toxicity of Aroclor 1248 and 1242 to shrimp and pinfish was similar to that of Aroclor 1254. Aroclor 1242 was toxic to oysters at 100 ug/l. Killfish exposed to 25 mg/l of Aroclor 1221 suffered an 85 percent mortality.

In 96-hour bioassays, Aroclor 1016 was toxic to an estimated 50 percent of the oysters, brown shrimp, and grass shrimp at 10 ug/l; it was toxic to 18 percent of the pinfish at 100 ug/l.

Young oysters exposed to Aroclor 1254 in flowing sea water for 24 weeks experienced reduction in growth rates at 4.0 ug/l, but apparently were not affected by 1.0 ug/l. Oysters accumulated as much as 101,000 times the test-water concentration of 1.0 ug/l. General tissue alterations in the vesicular

around the diverticula of the hepatopancreas were noted in the oysters exposed to 5.0 ug/l. No significant mortality was observed in oysters exposed continuously to 0.01 ug/l of Aroclor 1254 for 56 weeks (Duke, 1974).

Blue crabs apparently were not affected by 20 days' exposure to 4.0 ug/l of Aroclor 1254. Pink shrimp exposed under similar conditions experienced a 72 percent mortality. In subsequent flow-through bioassays, 51 percent of the juvenile shrimp were killed by Aroclor 1254 in 15 days and 50percent of the adult shrimp were killed at 3.0 ug/l in 35 days. From pathological examinations of the exposed pink shrimp, it appears that Aroclor 1254 facilitates or enhances the expression of latent viral infections.

Aroclor 1254 was lethal to grass shrimp at 4.0 ug/l in 16 days, to amphipods at 10 ug/l in 30 days, and to juvenile spot at 5.0 ug/l after 20 to 45 days. Sheephead minnows were the most sensitive estuarine organisms to Aroclor 1254 with 0.3 ug/l being lethal to the fry within 2 weeks.

Aroclor 1016 in two different 42-day flow-through bioassays caused significant mortalities of pinfish at 32 ug/l and 21 ug/l. Pathological examination of those exposed to 32 ug/l revealed several liver and pancreatic alterations. Sheephead minnows in 28-day Aroclor 1016 flow-through bioassays were not affected by concentrations of 10 ug/l or less, but died at 32 and 100 ug/l.

The bioaccumulation factors for the different flow-through bioassays are summarized below.

Aroclor	Organism	Time	Accumulation Facts (as a multiplier of test water concentrations)
1254	Oyster	30 days	1.01×10^5
1254	Blue Crab	20 days	4×10^3
1254	Grass Shrimp	7 days	3.2×10^3 to 11×10^3
1254	Spot	1 -28 days	37×10^3
1254	Pinfish	35 days	21.8×10^3
1016	Pinfish	42 days	11×10^3 to 24×10^3
1016	Sheephead Minnow		2.5×10^3 to 8.1×10^3

Evidence is accumulating that PCB's do not contribute to shell thinning of bird eggs (NAS, 1974). Dietary PCB produced no shell thinning in eggs of Mallard ducks (Heath et al., 1972). PCB's may increase susceptibility to infectous agents such as viral diseases (Friend and Trainer, 1970), and increase the activity of liver enzymes that degrade steroids, including sex hormones (Risebrough et al., 1968; Street et al., 1968). Laboratory studies have indicated that PCB with its derivatives or metabolites, causes embryonic death of birds (Voss and Koeman, 1970).

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