Interim Document

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POLYCHLORINATED BIPHENYLS

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CHEMICAL AND PHYSICAL PROPERTIES/DEFINITION

Polychlorinated biphenyls (PCB's) is a class of compounds produced by the chlorination of biphenyls, of which there are 210 possible different combinations. Each commercial PCB compound is a combination of homologs of finite proportions of chlorinated biphenyl isomers ranging from one chlorine atom per biphenyl group in the case of monochlorobiphenyls, to groups containing ten chlorine atoms per biphenyl group i.e., decachlorobiphenyls.

Data from laboratory bio-degradation studies indicate that, while higher polychlorinated biphenyl groups e.g., those containing three or more chlorine atoms, are more persistent and tend to accumulate in the environment, mono- and dichlprobiphenyls, are rapidly bio-degraded.

For purposes of this document, therefore, polychlorinated biphenyls means that group of PCB's having more than two chlorine atoms per biphenyl group.

PCB's manufactured in the United States are registered under the trade name Aroclor of which there are only four still being

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produced for commercial applications. They are Aroclor 1221, Aroclor 1016, 1242, and Aroclor 1254. These PCB's are marketed by the average weight percentage of chlorine in the Aroclor. The last two digits of the product number indicate the average percent by weight of chlorine, except in the case of Aroclor 1016 which has a chlorine weight content of forty-one percent (1).

The specific gravity, boiling point, and melting point of PCB's increases with chlorine content, while the water solubility varies inversely with chlorine. Because of their non-polar chemical characteristics, PCB's are generally referred to as insoluble (2). However, the reported low water solubility of PCB's cannot be used to predict the behavior of these materials in the aquatic environment, for non-ionic surface active agents have been found to affect the behavior of PCB's by increasing their water solubility (3, 4). The potential presence of surface active agents from municipal and industrial sources, as well as naturally occurring organic constituents, increases the probability of increased solubility of PCB's in water and in bottom sediments.

Identification of PCB's in the presence of organochlorine pesticides such as DDT and DDE has been extremely difficult in

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the past due to their similar chromatographic characteristics (5, 6). Interference by organochlorine hydrocarbons is overcome in current PCB analysis by first separating the PCB's and pesticides using salicytic acid column cleanup and then making chromatographic determinations (7, 8). Gas-liquid chromatography with highly sensitive and selective detectors has been employed successfully in the sub-micron range (9, 7).

TOXICOLOGICAL ASPECTS

Summarized below is the latest PCB toxicological information on aquatic organisms, birds, mammals, and humans. Acute data on aquatic species are also presented in tabular form. See Table I.

FRESHWATER AQUATIC LIFE

INVERTEBRATES

The 96-hr LC50 for scud, Garmarus pseudolimnaeus, under continuous-flow exposure to Aroclors 1242 and 1248 was reported to be 73 ug/l and 20 ug/l respectively (11). Reproduction and survival of young were not discernably affected at 2.8 ug/l and 2.2 ug/l.

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With Aroclors 1221, 1232, 1242, 1248, 1254, 1260, 1262, and 1268, Aroclor 1248 was found to be the most toxic to the water flea, Daphnia magna, with an LC50 of 25 ug/l during intial static 3-wk LC50 bioassays. However, under continuous flow conditions Aroclor 1254 was the most toxic with a 3-wk LC50 of 1.3 ug/l (11).

A sevenday bioassay with Aroclor 1242 resulted in a LC50 of 30 ug/l for crayfish. In the same test, the use of Aroclor 1254 produced an LC50 of 80 ug/l.

FRESHWATER FISH

Due to the low solubility of PCB's, tests to obtain 96-hr LC50 data do not adequately reflect their toxicities to fish. The toxicity of PCB's to fish is inversely related to their chlorine content (7, 10). For cutthroat trout, Salmo clarki, the 96-hr LC50 for Aroclors 1221-1268 ranged from 1,170 ug/l to 50,000 ug/l (10, 13). The acute oral toxicity of Aroclors 1242, 1248, 1254, and 1260 was greater than 1,500 mg/kg in rainbow trout Salmo gairdneri.

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Intermittent-flow bioassays with the bluegill, Lepomis macrochirus, exposed to Aroclors 1242, 1248 and 1254 resulted in 15-day LC50 values of 54 ug/l, 76 ug/l, and 204 ug/l respectively (10). Under similar conditions channel catfish, Ictalurus punctatus, exhibited LC50 values of 107 ug/l, 127 ug/l, and 741 ug/l. The toxicity of Aroclor 1248 to bluegills and channel catfish increased two-fold (a factor of two reduction in numerical value) when bioassays were run at temperatures of 27°C rather than 20°C (7, 10).

Newly-hatched fathead minnows, Pimephales promelas, exhibited 96-hr LC50 toxic concentrations of 15 ug/l and 7.7 ug/l during continuous-flow bioassays with Aroclors 1242 and 1254. Three month old fathead minnows had a 96-hr LC50 of 300 ug/l when exposed to Aroclor 1242. In chronic tests to determine the effects of Aroclors 1242 and 1254 on fathead minnow reproduction, all fish exposed to greater than 8.3 ug/l of each Aroclor died (11, 14).

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MARINE AQUATIC LIFE

INVERTEBRATES

Oysters exhibited a 96-hr LC50 of 10 ug/l during exposure to Aroclor 1016. They were sensitive to Aroclor 1260, with growth diminished 44 percent at 10 ug/l, and 52 percent at 100 ug/l. After exposure to 100 ug/l of Aroclor 1254 for 96 hours, shell growth of oysters was totally inhibited and exposure to 10 ug/l resulted in a decrease in growth of 41 percent compared to that of exposed controls. No significant mortality was observed in oysters exposed continuously to 0.01 ug/l of Aroclor 1254 for 56 weeks (15).

Grass shrimp, Palaemonetes kadiakensis, were very sensitive to Aroclor 1254 with a 7-day LC50 of 3 ug/l (10, 12). In bioassays lasting 96 hours, 10 ug/l of Aroclor 1016 caused a 50 percent mortality in brown shrimp, Penaeus aztecus. After 20 days exposure to 5 ug/l of Aroclor 1254 in flowing unfiltered water, pink shrimp, Penaeus duorarum, experienced 72 percent mortality. In subsequent flow-through bioassays, 51 percent of the juvenile shrimp were killed by 0.94 ug/l of Aroclor 1254 in 15 days and 50

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percent of the adult shrimp were killed by 3.5 ug/l in 35 days (10, 15).

Blue crabs, Callinectes sapidus, were not noticeably affected after 20 days exposure to 5 ug/l of Aroclor 1254 (15, 16).

The amphipod, Gammarus oceanicus, was exposed to concentrations of Aroclor to 10,000 ug/l for 30 days. Aroclor 1254 was lethal at concentrations as low as 10 ug/l (15, 17).

MARINE FISH

Exposure of pinfish to 100 ug/l of Aroclor 1016 during 96-hour bioassays resulted in a 18 percent mortality. Pinfish were not noticeably affected during 48-hour flow-through tests by 100 ug/l of Aroclors 1242, 1248, and 1254. However in two separate bioassays lasting 14 and 35 days, 5 ug/l of Aroclor 1254 was found to be lethal to juvenile pinfish (15). Pinfish and spot, Leiostomus xanthurus, died when exposed for 14-45 days to 5 ug/l of Aroclor 1254, but spot appeared unaffected by exposure to 1 ug/l for periods up to 56 days (10, 18).

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Of all the estuarine organisms tested, sheephead minnow Cyprinodon variegatus, were the most sensitive to Aroclor 1254 (15, 19). Newly hatched fry were killed at 0.3 ug/l of Aroclor 1254 during 3-wk intermittent-flow bioassays, while 0.1 ug/l had no apparent effect.

BIRDS

Most of the data concerning the effects of PCB's on avian species dealswith long-term feeding studies and resulting chronic and reproductive effects. Some have been carried through to death.

PCB's have been shown to be potent inducers of liver enzymes in test animals. When fed to laboratory animals in small doses they stimulate the liver to produce enzymes which act on other body substrates. An overabundance of PCB's at certain times could cause serious disruptions of physiological functions such as reproduction (20).

Dietary PCB levels of 100 ppm fed for two months to Japanese quail and laboratory rats resulted in enlarged livers and sharply reduced the vitamin D content of the liver of these animals (20,

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21). From a chronic feeding study with pigeons, Columba fasciata, and bob-white quail, Colinus virginianus, at 500 and 250 ppm respectively, significant increases in liver weights as well as increased liver microsomal protein and cytochrome levels were noted with PCB's of both 42 and 54 percent chlorine content (22).

Reduced resistance to certain pathogenic organisms was reported in ducks. Ten-day-old ducklings were fed PCB's of 54 percent chlorine content at dietary levels of 25, 50, and 100 ppm and no apparent clinical symptoms of poisoning were noted. However, when they were exposed five days later to the hepatitis virus, these ducklings suffered a higher incidence of mortality than did birds that had not been pre-exposed to PCB's (20, 23).

In a toxicity study lasting 56 days, Bengalese finches were fed a PCB diet with the average daily dosage ranging between 6 and 171 mg/kg of body weight. At the 6 mg/kg level 90 percent survival was observed, while 60 percent survival was observed at 101 mg/kg (24).

Much of the information related to eggshell thinning as a result of chlorinated hydrocarbons has come from field studies,

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which have attempted to correlate PCB residues levels in parents, eggs, and fledglings with reproductive failure. Such studies are difficult to interpret in a definitive manner since DDT and its metabolites, as well as other chlorinated hydrocarbons such as dieldrin, are often found in significant quantities (5). Laboratory studies aimed at elucidating the "cause and effect" relationship between PCB residues and reproductive failure have not been conclusive.

Mallard ducks, Anas platyrhynchos, fed a 25 ppm diet of 54 percent chlorine PCB through two breeding seasons, or for five weeks at dietary levels up to 500 ppm, produced eggs with shell thickness equivalent to control groups. However PCB's of 42 percent chlorine content at dietary concentrations of 10 and 100 ppm caused chickens to produced fewer eggs, with less hatching frequency, and with thinner shells than controls. When the dietary level was decreased to 1 ppm, no such effects were observed. The same effects were noted at 100 ppm of 54 percent chlorine PCB, while 10 ppm was a "no effect" dosage. Surprisingly, dietary levels up to 100 ppm of 60 percent chlorine content PCB produced none of the effects on reproduction that were observed with the lighter homologs (25).

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Two possible mechanisms by which PCB's and other chlorinated hydrocarbons affect eggshell thickness have been advanced and are reviewed in detail elsewhere (5, 26, 27). The one most generally subscribed to is that of induction of hepatic oxidase enzyme systems which alter sex hormone metabolism, thus resulting in rapid hormone degradation and excretion. In the case of avian species, hormone level and excretion apparently exert a direct depleting influence on the calcium reserves which are required for eggshell formation. The second possible mechanism is that of direct inhibitory action by the chlorinated hydrocarbons on the enzyme carbonic anhydrase which is critical to carbonate ion metabolism and the subsequent formation of calcium carbonate, the principal constituent of eggshells (27).

MAMMALS

Single oral dose LD50's for various PCB's have been reported in the range of 4,000 to 10,000 mg/kg of body weight for the rat, Rattus norvegicus (25). An extensive chronic study on rats, using PCB's of 54 and 60 percent chlorine content, with dietary levels ranging between 20 and 1,000 ppm, documented a variety of histopathological effects after 8 months of exposure (29). Degenerative liver changes observed in both male and female test

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animals at all dosages of both PCB's included hypertrophy, hyperchromatic nuclei, pleomorophism, cytoplasm vacuoles, and porphyria, all characteristic of chlorinated hydrocarbon intoxication. In some cases, adenofibrosis was observed. All of the mortalities that occurred during exposure to the PCB containing 60 percent chlorine were female. In nearly all gross pathological examinations, males demonstrated enlarged livers while females did not. Similar increased liver weight and increased liver lipid content were observed in another study employing male rats (30).

Experimental PCB intoxication of a primate, the rhesus monkey, Macacus rhesus, showed bone marrow hypoplasia, increased hepatic microsomal enzyme activity, liver hypertrophy, cytoplasmic lipid droplets, and a proliferation of endoplasmic reticulum. Infants exposed to 35 mg/kg/day gained less weight than did controls, but in general displayed a greater tolerance to PCB's than did adults (31).

PCB experiments with boars showed hepatic mixed oxidase enzyme induction as expressed by altered sex hormone excretions, with either a single 100 mg/kg dose or doses of 10 mg/kg/day for 10 days. No effects in hormone metabolism were observed with a

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single oral dose of 10 mg/kg or multiple doses of 1 mg/kg/day for 10 days (32).

A study of the effects of PCB's containing 21 and 54 percent chlorine on female rabbits and their reproductive capabilities showed no effect in mating, pregnancy, or weight gain of adults at levels of 1 and 10 mg/kg/day. Vitamin A content and 3 enzyme levels in the liver were assayed in both adults and fetuses. Vitamin A content was found to vary in mothers, but only the 21 percent chlorine PCB affected fetal livers by increasing Vitamin A content. Of the 3 enzymes, only carboxyesterase showed significant change by increasing in the mothers. No alterations in enzyme activity were detected in either fetuses or placental tissue (30). Although placental transport of PCB's does occur, no changes in fetal livers were detected by the measured parameters. In a preliminary report, some embryotoxicity was observed with oral PCB dosages of 50 mg/kg administered to pregnant female rabbits (33.

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HUMANS

In addition to data being provided by continuing studies of the various sub-acute effects of PCB's on laboratory animals, some information is available on the sub-acute or chronic effects of PCB's on humans. In October of 1968, an epidemic of a skin disease affecting over 1,000 people was reported in Japan (20, 34). The cause was soon traced to ingestion of rice oil contaminated with 2 to 3 g/l of a 48 percent chlorine content PCB. The disease is commonly known as "Yusho" or oil disease. The most notable symptoms of intoxication were increased pigmentation of skin and nails, acne-like eruptions, increased eye discharge, increased perspiration in the palms, and weakness. Other symptoms reported included swelling of the limbs, gastrointestinal disturbance, and signs of neurological disorders (34).

No fatalities directly attributable to PCB ingestion were reported. An epidemiological study found no significant difference in the severity of intoxication between the sexes. The same study showed a significant difference in clinical severity by age, with the 13 to 29 year-old group being the most sensitive. Of the 11 babies born to affected mothers, 2 were

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stillborn, 9 had dark-brown stained skin, and increased eye discharge was noticeable in most. Growth rates of affected children, as measured by both height and weight gains, were monitored and compared with unaffected classmates. A significant decrease in growth rate was detected in the males after poisoning, while no definite change was found in the females.

More than 3 years after the first symptoms of intoxication appeared, most of the patients were still affected by chronic symptoms of chlorinated hydrocarbon poisoning. Of the 159 patients examined approximately one year after initial symptoms appeared, only 50 percent showed clinical evidence of improvement, approximately 36 percent maintained stationary levels, while 12 percent had, in fact, worsened. The obvious implication is that human detoxification from chronic PCB intoxication is an extremely slow process (34).

The apparent human health threat from chronic ingestion of PCB's prompted the United States Food and Drug Administration to issue proposed limitations on the levels of PCB's in foods, animal feeds, and food packaging material (35). On July 6, 1973, final regulations were issued (36), which established temporary

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tolerances for PCB residues arising from unavoidable contamination. These tolerances are:

1. 2.5 parts per million in milk (fat basis).

2. 2.5 parts per million in manufactured dairy products (fat basis).

3. 5 parts per million in poultry (fat basis).

4. 0.5 parts per million in eggs.

5. 0.2 parts per million in finished animal feed for food-producing animals (except the following finished animal feeds: feed concentrates, feed supplements, and feed premixes).

6. 2 parts per million in animal feed components of animal origin, including fishmeal and other by-products of marine origin and in finished animal feed concentrates, supplements, and premixes intended for food-producing animals.

7. 5 parts per million in fish and shellfish (edible portion). The edible portion of fish excludes head, scales, viscera, and inedible bones.

8. 0.2 parts per million in infant and junior foods.

9. 10 parts per million in paper food-packaging material intended for or used with human food, finished animal feed and any components intended for animal feeds. The tolerance shall not apply to paper food-packaging material separated from the food therein by a functional barrier which is impermeable to migration of PCB's.

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DEGRADATION AND ENVIRONMENTAL TRANSPORT

When discussing persistent environmental contaminants such as PCB's, degradation, transport, and fate are of critical concern. The relative importance of these parameters becomes obvious from the observation that ambient concentrations of PCB's in our lakes, streams, and oceans are, in most cases, below the concentrations found toxic to aquatic species and higher organisms.

Both biomagnification and degradability are important factors in determining when contaminants such as PCB's pose a threat to the health and welfare of aquatic species and their predators. They often dictate which species or trophic level will be the ultimate receptor of the material in toxic amounts.

Physical transport processes such as vaporization, solubilization, and particulate absorption often dictatewhat part of the environment will be affected by the material. Chemical transformations such as oxidation, photo-oxidation, and hydrolysis can significantly affect the lifetime and form of the material in theenvironment.

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Degradation

The fact that Aroclors are mixtures of isomers and homologs, rather than a single uniform molecular structure, suggests examination of the degradation properties of each Aroclor. See Table II for homolog composition. The relative persistence of each Aroclor varies with the synthesizing homologs, as has also been shown in the case of their physical, chemical and toxicological properties.

Although there is a paucity of research on the degradability of the individual PCB homologs, data from the few studies completed and from numerous monitoring programs indicate that the higher chlorinated biphenyls are more persistent and as a result are more prevalent in the environment (1, 37, 38, 39, 40, 41). The PCB components obtained in water samples most nearly resemble Aroclors 1242, 1254 and 1260 (40, 42).

Investigations of the degradation of biphenyl, monochlorobiphenyl, and dichlorobiphenyl by two species of Achromobacter from sewage sludge established unequivocally that they are rapidly degraded by these microorganisms (43). Subsequent investigations were made on the biodegradation by

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Achromobacter of other isomers of PCB's containing 2 to 5 chlorine atoms. It was concluded that increasing chlorine substitution rendered the biphenyl molecule more resistant to microbial attack (37).

Commercial PCB mixtures, such as Aroclor 1221 which contains predominately mono- and dichlorobiphenyls, were shown to be readily degraded under experimental conditions (44). See Table II and Figure I. On the other hand, significantly less degradation was noted in the case of Aroclors whose major components are biphenyl groups having more than two chlorine atoms each.

Aroclor 1221, which is comprised predominately of biphenyl groups with two or less chlorine atoms, was degraded 81 percent in 48 hours by activated sludge microorganisms. Aroclors 1016 and 1042, the major components of which are tri- and tetrachlorobiphenyls, were degraded 33 and 26 percent respectively. Aroclor 1254, which is a mixture of predominately tetra-, penta-, and hexachlorobiphenyls was degraded only 15 percent.

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PHYSICAL TRANSPORT

There are few in situ data on the rate of physical transport of PCB's in the environment. Some projected studies of the extent to which these processes might occur are available, but these are usually from data obtained under controlled laboratory conditions. When extrapolating these data to actual environmental conditions, where a multitude of uncontrolled variables exist, they can only be used in a qualitative manner.

Transport to the atmosphere occurs either by direct vaporization or by co-vaporization with water (34, 45). Adsorption of PCBs on suspended solids, soil clays, and organic matter reduces their concentrations in water and also significantly slows the vaporization process. A PCB containing 54 percent chlorine has a vapor pressure nearly equivalent to DDE, and by analogy would be expected to have a soil half-life of approximately 5 years due to vaporization processes (34).

Depending on the chlorine content and the distribution of homologs within each batch, PCB solubilities have been reported to range from 0.025 mg/l to 5 mg/l (34, 46). When acute and chronic toxicity data are considered along with bioaccumulation,

these solubilities are sufficient to produce damage to the aquatic environment. Environmental factors such as temperature, dissolved solids, turbidity and naturally occurring organic matter all act to alter observed laboratory solubilities.

Biological Transport

Biological transport of PCB's became evident initially through numerous findings of residues in nearly all segments of the biota (25). Laboratory studies have since demonstrated bioaccumulation of PCB's by a variety of aquatic plants and animals directly from water and sediments. Feeding studies with mammals and birds have verified the bioaccumulative nature of these chlorinated hydrocarbons in higher animals through oral ingestion. Recognition of the accumulative nature and chronic toxicity of PCB's caused the Food and Drug Administration to issue the tolerances on PCB residues in food sources listed in this document.

Bioaccumulation of PCBs has been observed in various species representing most levels in the aquatic food web. A variety of aquatic macroinvertebrates exposed to radiolabeled PCB's of 54 percent chlorine content at concentrations of 1 to 2 ug/l

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exhibited magnification of 3,000 to 27,000 times depending on species and duration of exposure (50). The data are tabulated and presented in Table III. Oysters held for 8 weeks in water containing 0.01 ug/l of Aroclor 1254 exhibited a biomagnification of 160,000 (52). Fathead minnows, Pimephales promelas, held for 8 months in water containing 4.6 ug/l of Aroclor 1254 exhibited a biomagnification factor of 225,000 (11). Concentration factors from additional studies are summarized in Table IV.

Evidence of direct transport from sediments to the biota was obtained when two filter feeding organisms, the fiddler crab, Uca pugilator and pink shrimp, Penoeus duorarum, were exposed to PCB contaminated sediments under clean flowing water for 30 days. Both species bioaccumulated PCB's directly from the sediments and in proportion to concentration in the substrate (51). Maximum body concentrations were 80 and 240 ppm (wet weight) for the crab and shrimp. These occurred with sediments contaminated to a level of 61 ppm (dry weight basis).

The rate of uptake of PCB's by aquatic organisms is varied, and is maximal in 14 to 50 days for these species tested (17, 25, 51). Detoxification and excretion of body residue is rather slow. In the spot, Leiosomus xanthurus with whole body residues

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of 7 ppm, concentrations in the tissuedecreased to 7.2 ppm after 84 days in clean water (17). After 4 weeks in clean water, the blue crab, Callinectes sapidus, still retained 50 percent of accumulated PCB's (15).

Although PCB's are generally distributed in all body tissue and organs of fish, birds, and mammals, their high lipid solubility is expressed by a preferential accumulation in body fat tissue and the liver (23, 31, 32). Rapid accumulation and slow detoxification of orally ingested PCB's are evident in the Yusho disease epidemic in which, 3 years after intoxication, nearly all patients still suffered chronic symptoms of chlorinated hydrocarbon intoxication (34).

Adsorption on suspended clay particles can remove PCB's from solution, while organic matter, such as the humic acids, can have the opposite effect of solubilization (3, 34, 46). Under bioassay conditions, a PCB of 54 percent chlorine content was found to have very little adsorption affinity and remained in solution, while a 21 percent chlorine PCB adsorbed rapidly, showing a decrease in concentration of from 10 to 6 mg/l in about 5 hours. The primary site of adsorption was presumed to be the glass of the bioassay test chambers. PCB vapors were detected in

the environmental chamber atmosphere. Presumably, co-vaporization was facilitated by aeration within the bioassay tanks (47).

Chemical Transport

One of the properties of PCB's which led to their wide variety of uses was their chemical inertness. Although generally regarded as quite stable, limited photochemical breakdown has been observed with some PCB homologs (5, 8). In one observation, ultraviolet irradiation resulted in dechlorination to hydroxyl derivatives, which are presumably further degraded by microbial action (49)

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CRITERIA FORMULATION

Criteria

The PCB criteria set forth below are established at two levels, acute and chronic.

The acute criteria are based upon short-term bioassays, of 4 days duration, carried through until death of 50 percent of the test organisms. They are established at levels that should protect aquatic organisms from short-term exposure to PCB's.

Chronic criteria are normally based upon bioassays conducted over longer periods, preferably several life cycles, and established at levels that will sustain physiological functions of aquatic organisms, i.e. reproduction, growth rate, etc. However, the PCB chronic criteria are based on bioaccumulation of PCB's by aquatic organisms and are established at levels that should protect human consumers of aquatic organisms from the ingestion of PCB's in excess of FDA tolerances.

Acute Criteria

10 ug/l for freshwater and marine aquatic life

Chronic Criteria

0.01 ug/l for freshwater and marine aquatic life

FORMULATION OF THE CRITERIA

Acute Toxicity

Freshwater Invertebrates (Acute Toxicity)

Aroclors 1242 and 1248 induced LC50 toxicities of 73 ug/l and 20 ug/l in the scud under 96-hr continuous-flow conditions(11).

Freshwater Fish (Acute Toxicity)

Fathead minnows at 3-month-old when exposed to Aroclor 1242 exhibited a 96-hr LC50 of 300 ug/l, while Aroclors 1242 and 1248 induced 96-hr LC50 toxicities of 15 and 7.7 ug/l in newly hatched fathead minnows. Hence from the above, the acute toxicity levels of PCB's to fathead minnows are shown to range from 300 ug/l down to 7.7 ug/l under static and continuous-flow conditions(11,14).

THE ABOVE FRESHWATER INVERTEBRATE AND FISH DATA THEREFORE SUGGEST A FRESHWATER ACUTE TOXICITY LEVEL OF NOT MORE THAN 10 ug/l.

Marine Invertebrates (Acute Toxicity)

Aroclor 1016 induced a 96-hr LC50 of 10 ug/l in the oyster and the brown shrimp(10,15).

Marine Fish (Acute Toxicity)

Aroclor 1016 at 100 ug/l induced an 18 percent mortality in pinfish during 96-hr static bioassays(15).

THE ABOVE MARINE INVERTEBRATE AND FISH DATA THEREFORE SUGGEST A MARINE ACUTE TOXICITY LEVEL OF NOT MORE THAN 10 ug/l.

CHRONIC TOXICITY

Freshwater Aquatic Life (Chronic Toxicity)

Based on a possible accumulation factor in freshwater organisms of 225,000 as exhibited by the fathead minnow, a maximum PCB ambient water concentration of 0.02 ug/l would be possible without exposing humans consumers to levels in excess of the PCB tolerance of 5 ~~ug/l~~ ppm in edible portions of freshwater fish and shellfish(11).

$$\frac{0.02 \times 2.25 \times 10^5}{10^9} = \frac{X}{10^6}$$

$$\frac{2 \times 10^{-2} \times 2.25 \times 10^6 \times 1 \times 10^6}{1 \times 10^9} = X = 4.5$$

A CHRONIC PCB LEVEL OF 0.01 ug/l IN FRESHWATER SHOULD PROVIDE AN ADDITIONAL 2:1 SAFETY FACTOR, AND IS THEREFORE RECOMMENDED FOR THE PROTECTION OF HUMAN CONSUMERS OF FRESHWATER AQUATIC LIFE.

Based on bioconcentration factor of 35,000 (Marine) and a safety factor of 2

Marine Aquatic Life (Chronic Toxicity)

Based on a possible accumulation factor in marine organisms of 160,000 as exhibited by the oyster, a maximum ambient water concentration of 0.03 ug/l would be possible without exposing human consumers to levels in excess of the FDA PCB tolerance level of 5 ~~ug/l~~ ppm in edible portions of marine fish and shellfish(52).

$$\frac{0.03 \times 1.6 \times 10^5}{1 \times 10^9} = \frac{X}{10^6}$$

$$\frac{3 \times 10^{-2} \times 1.6 \times 10^5 \times 1 \times 10^6}{1 \times 10^9} = 4.8 \text{ ppm}$$

A CHRONIC PCB LEVEL OF 0.01 ug/l IN MARINE WATERS WOULD PROVIDE AT LEAST AS MUCH SAFETY AS THE FRESHWATER CRITERION AND WOULD BE IN CONCERT WITH IT; IT IS THEREFORE RECOMMENDED FOR THE PROTECTION OF HUMAN CONSUMERS OF MARINE AQUATIC LIFE.

Based on a bioaquification factor of 160,000. (Eastern Oyster) and a safety factor of 3

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TABLE I

Acute Toxicity of PCB's

PCB Type	Organism	Bioassay Type	Exposure (Days)	LC50 (ppb)	Reference
Aroclor 1221	Cutthroat Trout	Static	4	1170	10
	Bluegill	Static	4	230	54
	Channel Catfish	Static	4	3340	54
Aroclor 1016	Bluegill	Static	4	650	54
	Channel Catfish	Static	4	750	54
	Oysters	Flow-through	4	10	19
Aroclor 1242	Rainbow Trout	Intermittent-Flow	5	109	53
	Rainbow Trout	Intermittent-Flow	10	39	53
	Cutthroat Trout	Static	4	5430	10
	Bluegill	Static	4	240	55
	Bluegill	Intermittent-Flow	5	154	53
	Bluegill	Intermittent-Flow	15	54	55
	Channel Catfish	Static	4	130	55
Aroclor 1242	Crayfish	Static	7	30	10
	Scud	Continuous-Flow	4	10	10
	Scud	Flow-through	4	73	11
	Damselfly	Continuous-Flow	4	400	10

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TABLE I (Continued)

PCB Type	Organism	Bioassay Type	Exposure (Days)	LC50 (ppb)	Reference
	Fathead Minnow	Continuous-Flow	4	15	11
Aroclor 1248	Rainbow Trout	Intermittent-Flow	5	51	53
	Cutthroat Trout	Static	4	5750	10
	Bluegill	Intermittent-Flow	5	307	53
	Bluegill	Intermittent-Flow	10	160	53
	Bluegill	Intermittent-Flow	20	10	53
	Bluegill	Static	4	270	10
Aroclor 1254	Fathead minnow	Continuous-Flow	4	17.7	11
	Glass Shrimp	Continuous-Flow	7	3	10
	Rainbow Trout	Intermittent-Flow	5	156	53
	Rainbow Trout	Intermittent-Flow	10	8	53
	Crayfish	Static	4	100	10
	Scud	Static	4	2400	10
	Bluegill	Intermittent-Flow	10	443	53
	Bluegill	Intermittent-Flow	20	135	53
Aroclor 1254	Dragonfly	Static	7	1000	10
	Damselfly	Continuous-Flow	4	200	10

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Table IV

Test Species	Exposure Time	Exposure Concentration (ug/l)	Concentration Factor	% Chlorine	Comments	Reference
Eastern Oyster	8 weeks	0.01	160,000	54	Whole body residues	52
Pink Shrimp	22 days	2.5	12,400	54	Whole body residues	44
Blue Crab	4 weeks	5	4,600	54	Whole body residues	25
Finfish	2-15 weeks	5	2,800-21,800	54	Whole body residues	52
Spot	4-8 weeks	1	17,000-27,000	54	Whole body residues	52
Spot	3-6 weeks	5	9,200-30,400	54	Whole body residues	52
Bluegill Sunfish	11 weeks	14.1	52,000	48	Whole body residues	54
Fathead Minnow	32 weeks	4.6	225,00	54	Whole body residues	44
Channel Catfish	15 weeks	13.3	72,300	48	Whole body residues	53
Channel Catfish	15 weeks	4.1	76,100	54	Whole body residues	53
Bluegill	15 weeks	4.9	63,700	48	Whole body residues	53
Bluegill	15 weeks	6.8	12,800	54	Whole body residues	53
Glass Shrimp	4 days	1.3	12,300	54	Whole body residues	50
Stonefly	4 days	2.8	2,500	54	Whole body residues	50

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TYPICAL % COMPOSITION OF POLYCHLORINATED BIPHENYL PRODUCTS*

HOMOLOG # Cl/BIPHENYL	AROCLOR 1221	AROCLOR 1016	AROCLOR 1242	AROCLOR 1254
0	11	<0.1	<0.1	<0.1
1	51	1	1	<0.1
2	32	20	16	<0.5
3	4	57	49	1
4	2	21	25	21
5	<0.5	1	8	48
6	ND	<0.1	1	23
7	ND	ND	<0.1	6
8	ND	ND	ND	ND

*PER CENT (W/W) BY GC/MASS USING AREA CORRECTION FACTORS BY HOMOLOG RESPONSE

NONE DETECTED, <0.01% = ND

TABLE II

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Semi-Continuous Activated Sludge Degradation of Polychlorinated Biphenyls

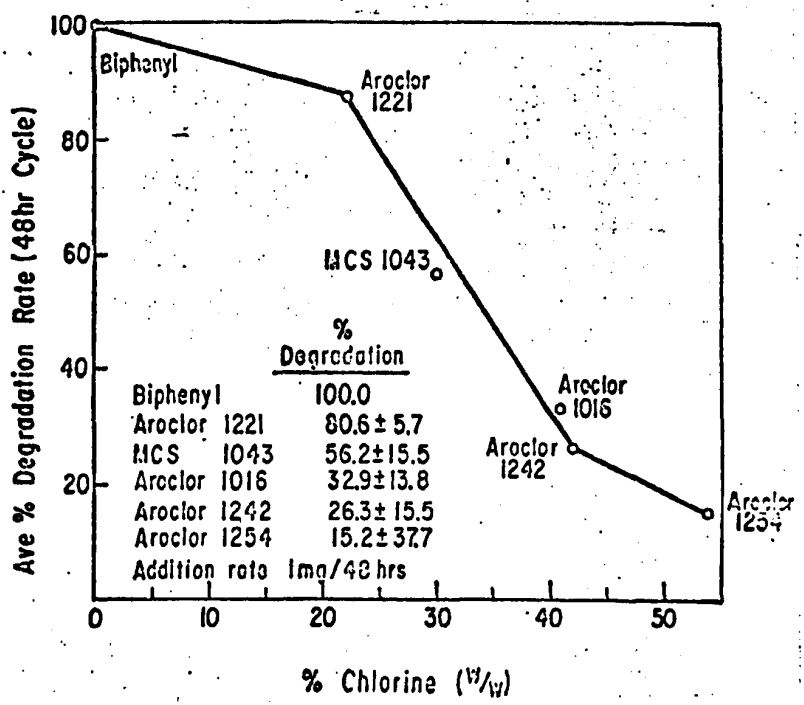

Figure 1 (Ref. 1)

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TABLE III

Biological magnification of ^{36}Cl-labeled Aroclor® 1254 by aquatic invertebrates (Ref 52)

Organism	Organisms per sample 1/	Water concentration ppb x̄ ± SE 2/	Organism concentration (4 day exposure) ppm x̄ ± SE	Magnification factor 3/ 1-day	4-day	7-day	14-day	21-day
Daphnid Daphnia magna	60	1.1 ± 0.2	52 ± 2.0	24,700	47,000	-	-	-
Phantom midge Chaoborus punctipennis	5	1.3 ± 0.1	30 ± 1.6	22,000	23,000	23,800	24,800	-
Scud Gammarus pseudolimnaeus	6	1.6 ± 0.1	39 ± 3.0	17,000	24,000	26,000	27,500	27,000
Mosquito larvae Culex tarsalis	10	1.5 ± 0.3	27 ± 2.0	12,600	18,000	20,000	-	-
Grass shrimp Palaemonetes kadiakensis	3	1.3 ± 0.1	16 ± 2.6	10,300	12,300	13,700	14,200	16,600
Stonefly Pteronarcys dorsata	3	2.8 ± 0.8	7.0 ± 0.30	2,100	2,500	2,800	2,900	2,800
Dobsonfly Corydalus cornutus	3	1.1 ± 0.1	5.1 ± 0.23	1,400	4,600	5,700	6,600	6,800
Crayfish Orconectes nais	2	1.2 ± 0.1	0.2 ± 0.20	570	1,700	3,400	4,500	5,100

1/ Samples were taken in triplicate.

2/ Samples were taken in triplicate and expressed as mean value ± standard error (P=.05).

3/ Concentration in organism/concentration in water.

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