ENVIRONMENTAL RISK AND HAZARD ASSESSMENTS FOR VARIOUS ISOMERS OF POLYCHLORINATED BIPHENYLS (MONOCHLOROBIPHENYL THROUGH HEXACHLOROBIPHENYL AND DECACHLOROBIPHENYL)

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I. Executive Summary

Potential environmental risk is expected to occur to aquatic organisms if polychlorinated biphenyl (PCB) isomers are released into the aquatic environment at concentrations of 100 ug/L. Expected risks include: (1) reduction in the reproductive success of fish through direct toxicity to eggs and embryos which are fish early life stages; (2) direct lethality and sublethal effects to juvenile and adult fish; (3) effects to aquatic invertebrates which are food sources for fish; and (4) possible but not demonstrated reductions in the growth of phytoplankton, i.e., minute floating plants, usually algae, which produce the basic food for the aquatic ecosystem. Risks to wild mammals and birds could not be determined because of a lack of toxicological information for PCB isomers and lack of an appropriate exposure assessment for terresrial organisms.

Environmental risk for all effects generally increase with an increase in the degree of chlorination of the biphenyl (Figure 1). Environmental risk to aquatic organisms from monochlorobiphenyl isomers are predicted to occur at 10 to 30% of chemical plant sites expected to inadvertently produce PCB isomers as impurities; while risk from hexachlorobiphenyl isomers are expected to occur at 80-90% of plant sites. This increase in risk is due to an increase in the toxicity of PCBs with greater numbers of chlorine atoms. An increase in chlorination of PCBs has been related to increases in toxicity for (1) chronic toxicity to rainbow trout (Figure 3) for monochlorobiphenyl through hexachlorobiphenyl isomers, (2) acute (lethal) toxicity to rainbow trout (Figure 2) for monochlorobiphenyl through tetrachlorobiphenyl isomers, and (3) acute toxicity to aquatic invertebrates (Figure 4) for monochlorobiphenyl through tetrachlorobiphenyl isomers. Acute toxicity of pentachlorobiphenyl and hexachlorobiphenyl isomers to aquatic invertebrates is less than would be predicted (Figure 4) because of the greater water insolubility of these isomers.

Environmental risk to aquatic organisms is shown to increase to as many as 40% of additional chemical plant sites in the US under low streamflow conditions. Risk was estimated for two streamflow conditions for rivers in the US: average streamflow and low streamflow (Table 1). The increase in risk under low streamflow conditions can be attributed to lower stream dilution factors during low streamflow which result in higher predicted environmental concentrations.

The environmental risk of monochlorobiphenyl and dichlorobiphenyl isomers is predicted to increase if discounting factors are incorporated into the exposure assessment. A discounting factor of 50 for monochlorobiphenyl isomers is expected to increase potential risk to an additional 40% of plant sites over risks associated with no discounting factor. A discounting factor of 5 for dichlorobiphenyl isomers is expected to increase risk to aquatic organisms at about 10% additional plant sites. A discounting factor means that monitored concentrations of a PCB isomer will be divided by that factor, e.g., 50 or 5, and then recorded. For example, an effluent concentration for a monochlorobiphenyl of 5 mg/L would be reported as 100 ug/L of isomer. The increase in risk associated with the use of discounting factors can be entirely attributed to higher surface water concentrations used in the exposure assessment.

The environmental concerns for PCB isomers generated by tnis risk assessment are similar to the environmental concerns historically expressed for the commercial mixtures of PCBs (Aroclors). Environmental concerns held in common are: (1) impairment of the reproductive success in fish, especially, to the early life stages of fish; (2) direct adverse effects to juvenile and adult fish; and (3) reduced survival and growth of aquatic invertebrates and aquatic plants. Environmental concerns attributed to PCB commercial mixtures but not yet demonstrated for PCB isomers include: (1) correlation of high body burdens of

PCBs in female fish with failure of eggs to hatch; (2) impaired bone development and testical abnormalities in juvenile fish; (3) contamination of economically important food resources, e.g., closure of fisheries; and (4) impairment of reproductive success in some wild mammals (e.g., mink) and birds.

This environmental risk assessment is a qualitative risk assessment and is relatively conservative, i.e., designed to err on the side of environmental protection. The basis of this risk assessment was the application of information derived from a hazard assessment to situations identified by an exposure assessment. Concentration-effect curves for each group of aquatic organisms, e.g., early life stages of fish, were synthesized for each class of PCB isomer, e.g., monochlorobiphenyls, dichlorobiphenyls, etc., and were compared to hypothetical surface water concentrations downstream from organic chemical plants. Whenever an effective concentration was equal to or exceeded a surface water concentration, a potential risk was noted and the type and degree of risk was determined (Tables 1 and 2). No attempt was made to quantify the impact to a particular population at a particular site over time. Therefore, this risk assessment can be characterized as more qualitative than quantitative.

This assessment can also be characterized as relatively conservative because (1) toxicity information for the most sensitive species and the most sensitive life stage for a species was used whenever possible; and (2) it was assumed that all chemical plants discharged process wastewater containing 100 ug/L of PCBs and that there was no loss due to sorption, transformation, or degradation. However, the risk assessment could have been more conservative. No assessment factor was used to predict a "safe" concentration of PCB isomer from the toxicity information. An assessment factor is defined as a number by which an effect concentration, e.g., EC50 or NOEC, is adjusted (by division) to arrive at a "safe" environmental

concentration. In addition, information for the most sensitive species could not always be used. For example, in some data sets rainbow trout was the most sensitive fish species tested, however, data from five other fish species were also used in the risk assessment.

This risk assessment is as comprehensive as available information for the toxicity of PCB isomers would permit. Concentration-effect curves were synthesized from all available information: which ranged from chronic no-observed-effect concentrations for the most sensitive life stage to acute concentrations which caused 100% lethality in a test population. For example, potential environment risk to fish populations to tetrachlorobiphenyl isomers with no chlorines in the o,o'- positions on the biphenyl (Figure 1 and Table 1) was characterized in terms of effects to fish early life stages (E), sublethal effects to juvenile fish (SL), reductions in the growth of juvenile fish (G), and lethality to juvenile fish (L).

All 18 hypothetical exposure situations (i.e., streamwater concentrations for the 10th through the 90th percentiles of chemical plants under two streamwater condition: low and average streamflow, Table 1) were evaluated for potential risk from each class of PCB isoomer. In all, one hundred forty four situations were evaluated for four groups of aquatic organisms. Including evaluation of discounting factors, a total of 612 situations were evaluated for potential environmental risks.

This risk assessment did not address the additional risk of PCB isomers from bioconcentration, food chain transport, and food chain concentration (or biomagnification). For example, a female fish will bioconcentrate PCBs and subsequently pass these residues to her eggs which could result in inviable eggs and embyros. The reason for this deficiency in the risk assessment was a lack of toxicological information on the PCB isomers with regard to this type of reproductive inhibition in fish, birds, and wild mammals.

II. Environmental Risk Assessment of PCB Isomers to Aquatic Organisms

A. Basis of Risk Assessment

Risk assessment is the application of information derived from a hazard assessment to situations identified by an exposure assessment. The hazard assessment for various PCB isomers can be found in Sections III through VI. The exposure assessment was derived from an exposure assessment for incidentally produced PCBs (Versar 1983). Table H-1 from Versar (1983) (see Table 1) was used to obtain hypothetical surface water concentrations downstream from organic chemical plants in the US expected to inadvertently produce PCB isomers as impurities. Versar estimated 19 hypothetical concentrations: 9 under average streamflow conditions and 9 under low streamflow conditions. Versar estimated the 10th through the 90th percentiles (9 percentiles) for each streamflow condition (Table 1). For this risk assessment, the 18 hypothetical streamwater concentrations were rank-ordered from highest to lowest: 66 to 0.00037 ug/L, respectively. Versar assumed that all plants discharged process wastewater containing 100 ug/L of PCBs, that there was instantaneous mixing, and that there was no loss due to degradation, transformation, or sorption. For this risk assessment, eight risk assessments were performed: one each for monochloro-, dichloro-, trichloro-, tetrachloro(with 1-4 chlorines at the o,o'-positions)-, tetrachloro(with no chlorines at the o,o'-positions)-, pentachloro-, hexachlorobiphenyl isomers, and decachlorobiphenyl (Table 1). It was assumed that all plants discharged 100 ug/L of one class of PCB isomers, e.g., monochlorobiphenyl isomers. Each estimated streamwater concentration was compared to the concentration-effect curve for each class of PCB isomers, and if a risk was predicted (i.e., if an effective concentration was equal to or greater than a streamwater concentration), then the type of risk and its degree were indicated in Table 1. An empty cell in the matrix under "Class of PCB" in Table 1 indicates that the streamwater

concentration was less than any available effective concentration in the hazard assessment and thus no risk.

B. Risk Assessment for Fish

1. Monochlorobiphenyls

a. Discharge of 100 ug/L of monochlorobiphenyl isomers will result in risk to fish populations under only low streamwater conditions at 30 percent of plants with a low streamwater dilution factor of 18.9 or less (Table 1). At a concentration of 66 ug/L, juvenile and adult fish are predicted to have only sublethal effects, e.g., reduction in growth, reduced food consumption, disorientation. No lethality of juveniles and adult fish is expected. The early life stages of fish, i.e., embryos and sac fry, are expected to be affected by the monochlorobiphenyl isomers at all concentrations equal to and higher than 5.3 ug/L. Effects expected to occur are reduced growth and survival of embryos and sac fry. The percentage of fish affected cannot be estimated, but at 5.3 ug/L the percentage will approach 0%.

b. Discharge of monochlorobiphenyl isomers with a discounting factor of 50 will increase the risk to fish populations 50 times. A discount of 50 means that monitored concentrations of monochlorobiphenyls will be divided by 50 and then reported. Actual concentrations would be 50 times higher than PCB surface water concentration estimated in Table 1. Under these conditions, acute lethality of juvenile and adult fish would occur at 30% of plants at low streamflow. Lethality of 50% or higher, would occur at 20% of these plants. Sublethal effects and subchronic mortality would occur at 40% of plants at low streamflow and 10% of plants at average streamflow. Effects to fish early life stages would occur at 70% of plants at low flow and 40% of plants at average streamflow.

2. Dichlorobiphenyls

a. Discharge of dichlorobiphenyls isomers at 100 ug/L will potentially affect fish populations at 20% of plants at average streamflow conditions and 50% of plants at low streamflow (Table 1). Fish early-life stages will be at risk at all the above plants. Effects will be near zero at 0.47 ug/L and increase in severity at plants with smaller stream dilution factors (i.e., as you proceed up Table 1). Juvenile and adult fish will suffer sublethal effects at only 20% of plants under low streamflow. The probability of subchronic and acute lethality is low for dichlorobiphenyls released at 100 ug/L.

b. If a discounting factor of 5 is used for discharge of dichlorobiphenyl isomers, potential risk to fish will extend to 10% more plants and acute lethality will probably occur. Effects to fish early-life stages will occur at 30% of plants during average streamflow and 60% of plants during low flow. Sublethal effects to juveniles and adults will occur at 30% of plants only at low flow and lethality approaching approximately 40% will occur at 10% of plants.

3. Trichlorobiphenyls

Trichlorobiphenyl isomers discharged at 100 ug/L have a potential to affect the early-life stages of fish at 40% of plants during average streamflow and 60% plants at low flow (Table 1). Sublethal and subchronic (30d) lethality will occur only during low streamflow conditions at 30% of plants. Subchronic lethality will occur only at 10% of these plants at low flow and will be much lower than 50%.

4. Tetrachlorobiphenyl Isomers with 1-4 Chlorines at the o,o'-Positions of the Biphenyl

Tetrachlorobiphenyl isomers with 1 to 4 chlorine atoms at the o,o'-positions of the biphenyl (Figure 1) have the potential to affect fish populations at 70% of plants during low streamwater flows and at 40% of plants during average flow. The early-life stages (i.e., embryos and sac fry) of fish will exhibit reduced growth and increased mortality at all of the above plant sites. Juvenile and adult fish will exhibit sublethal effects at only 10% of plant sites during average streamflow, but during low streamflow 40% of plants discharging 100 ug/L could affect juveniles and adults (Table 1). Subchronic lethality will occur only at low streamflow at 20% of plant sites; these isomers have the potential of killing over 50% of the fish populations at 10% of plant sites (Table 1).

5. Tetrachlorobiphenyl Isomers with No Chlorines at the o,o'- Positions of the Biphenyl

Tetrachlorobiphenyl isomers with no chlorines at the o,o'- positions of the biphenyl appear to present significantly greater risk to fish populations than tetrachlorobiphenyl isomers with chlorines at the o,o'-positions. They also have the potential of affecting more plant sites (20% more sites at average streamflow). The early life stages of fish will be affected at 70% of plant sites at average streamflow and 80% of plant sites at low flow (Table 1). Sublethal effects to juvenile and adult fish will (1) begin to occur at 40% of plants during average streamflow, (2) reduce the growth of juveniles by 30% at 30% of plant sites, (3) reduce growth further by 60% at 20% of plants during average flows, and (4) could kill 25% of the fish populations at 10% of plant sites during average flow, but during low flows 40-50% of sites could be affected (Table 1).

6. Pentachlorobiphenyls

Pentachlorobiphenyl isomers were predicted to be slightly more chronically toxic than the tetrachlorobiphenyl isomers in the hazard assessment (Section III. E) and, therefore, will present slightly more risk to resident fish populations than the tetrachlorobiphenyls given the same discharge conditions at 100 ug/L concentration. Table 1 reflects this increase in risk by affecting about 10% more plant sites than the tetrachlorobiphenyls). The subchronic and chronic effects of the pentachlorobiphenyls are less quantified than they were for the tetrachlorobiphenyl isomers, because much more experimentation was available for the tetrachlorobiphenyls and only chronic NOECs could be predicted for pentachlorobiphenyls in the hazard assessment. The type of effects could not be quantified but it was assumed that effects observed for the tetrachlorbiphenyl isomers will also occur with the pentachlorobiphenyl isomers.

7. Hexachlorobiphenyls

Hexachlorobiphenyl isomers were predicted to be more chronically toxic than the pentachlorobiphenyls in the hazard assessment (Section III.F) and, therefore, are assumed to present more risk to fish populations given the same exposure conditions. Table 1 indicates this increase in toxicity, and, therefore, risk.

The hexachlorobiphenyls are expected to affect the early life stages of fish at 80% of the plant sites during average streamflow and over 90% of sites at low flow (Table 1). These effects will increase in severity at sites with smaller stream dilution factors. This fact is demonstrated at the 20th percentile entry. At this surface water concentration (16 ug/L), 100% of the embryo and sac fry fish are expected to be killed. Broyles and Noveck (1979b) observed 100% mortality within 79 days

of lake trout and chinook salmon sac fry after only an 8 ug/L exposure for 15 days (Section III.F.2.c).

8. Decachlorobiphenyl

Decachlorobiphenyl is expected to be similar to or less toxic than the hexachlorobiphenyl isomers (Section III.G), and, therefore, under the same exposure conditions, are expected to present similar or smaller risks to fish populations. Table 1 reflects the assumption that the potential risks for decachlorobiphenyl are similar to those for the hexachlorobiphenyl isomers. No toxicological information is available indicating how much less toxic decachlorobiphenyl is to fish than the hexachlorobiphenyl isomers.

C. Risk Assessment for Aquatic Invertebrates

1. Discharges of 100 ug/L

Discharges of 100 ug/L of the various PCB isomers will probably result in risks to aqutic invertebrates. These risks are similar to risks predicted for juvenile and adult fish. Risks to aquatic invertebrates can best be defined by taking Table 1 and eliminating risks to the early life stages of fish (i.e., eliminate Es' from Table 1; see Table 2). The rationale for using the risk assessment for juvenile fish for aquatic invertebrates is based upon the similar acute toxicity between fish and aquatic invertebrates (Section IV.A) and a lack of chronic toxicity information for aquatic invertebrates (Section IV.B).

There are three major differences between the risk assessment for fish (Table 1) and the risk assessment for aquatic invertebrates (Table 2): (1) risk from acute exposure is expected to occur to aquatic invertebrates (acute risk was not predicted for fish), (2) chronic sublethal effects for aquatic

invertebrates cannot be quantified as precisely as was done for fish, and (3) risk to aquatic invertebrates from the pentachlorobiphenyl isomers, hexachlorobiphenyl isomers, and decachlorobiphenyl may not occur at the lower streamwater concentrations.

a. Risk from acute exposure is expected to occur to aquatic invertebrates from trichlorobiphenyl and tetrachlorobiphenyl isomers at 10% and 20% of plant sites, respectively, during low streamflow (Table 2). During low streamflow, a 100 ug/L discharge of trichlorobiphenyl isomers will kill 50% of the aquatic invertebrates in the receiving stream; discharge of tetrachlorobiphenyl isomers will kill over 50% of the invertebrate population. Acute lethality will also occur at 20% of plants during low streamflow if tetrachlorobiphenyl isomers are released (Table 2). In the risk assessment for fish, lethality was expected to occur only from subchronic exposures.

b. Sublethal effects for aquatic invertebrates cannot be identified or quantified because no chronic studies have been done. Sublethal effects will probably include reductions in weight, fertility, brood size, growth rate, and survival of offspring. It was assumed that the chronic NOECs for aquatic invertebrates will be similar to the subchronic NOECs for juvenile fish (Section IV.B).

c. Risks (lethality) predicted to occur in Table 2 for pentachlorobiphenyl and hexachlorobiphenyl isomers, and decachlorobiphenyl may not occur at some of the lower streamwater concentrations (1.8 to 0.56 ug/l). The acute toxicity of pentachlorobiphenyl and hexachlorobiphenyl isomers was shown (Section IV.A) to decrease relative to acute toxicity of the tetrachlorobiphenyls. It is possible that the chronic toxicity for these isomers could also be less, however, under chronic exposures much more time is available to take up and accumulate an effective dose than under acute exposure conditions.

2. Effect of Discounting Factors

a. Monochlorobiphenyls

Using a discounting factor of 50 for discharge of monochlorobiphnyl isomers to receiving streams, will result in (1) acute lethality to aquatic invertebrates at 30% of plants during low streamflow, and (2) sublethal effects at 40% of plants during low streamflow, and 10% of plants during average flow. Acute lethality will be much greater than 50% at the 10 percentile (low flow), greater than 50% at the 20 percentile (low flow), and less than 50% at the 30 percentile (low flow, Table 2). The net effect of the discounting factor will be to (1) introduce acute risk to 30% of plant sites, (2) increase risk of sublethal effects from 10% of plants to more than 40% of plant sites at low flow, and (3) introduce potential risk from sublethal effects to 10% of plant sites during average flow.

b. Dichlorobiphenyls

Use of a discounting factor of 5 for the discharge of dichlorobiphenyl isomers to receiving streams will result in acute lethality to aquatic invertebrates at 20% of plant sites during low streamflow: lethality of greater than 50% will occur at 10% of sites and lethality of 50% or less will occur at another 10% of sites. Sublethal effects will occur at 30% of sites during low streamflow. The result of using a discounting factor of 5 will be to (1) introduce acute lethality and (2) increase the occurrence of sublethal effects from 20% to 30% of plant sites.

D. Risk Assessment for Algae

A risk assessment for algae cannot be done at this time. The only toxicity information available is for marine phytoplankton communities (Section V.A) and the hypothetical exposure assessment is for freshwater riverine ecosysems (Versar 1983).

E. Risk Assessment for Protozoa

1. Discharges of 100 ug/L

The risk of various PCB isomers to protozoa appears to be low. The NOECs of 13 PCB isomers ranging from monochlorobiphenyls through hexachlorobiphenyls were about 100 ug/L or greater. In the exposure assessment, the highest surface water concentration estimated was 66 ug/L in streams with the smallest dilution factor at low streamflow. This worst case exposure condition is lower than the NOECs estimated for protozoa (Section V.B)

2. Effect of Discounting Factors

a. Monochlorobiphenyls

Use of a discounting factor of 50 will introduce risk to protozoa at 20% of plant sites during low streamflow conditions. At 20% of sites, protozan growth could be reduced 50% in 43h (Table 6); at 10% of sites, growth could be reduced more than 50%.

b. Dichlorobiphenyls

Use of a discounting factor of 5 for the dichlorobiphenyl isomers could introduce risk to protozoa at 10% of plant sites during low streamflow. At these sites growth could be reduced about 10% to 50% in 43h (Table 6).

III. Toxicity of Various PCB Isomers To Fish

A. Monochlorobiphenyl Isomers

1. The subchronic no-observed-effect concentration (NOEC) for monochlorobiphenyl isomers using data for the most toxic isomer to the most sensitive fish species tested is

estimated to be 50. - 80. ug/L to juvenile fish after about a 30-day exposure.

a. The most toxic isomer of monochlorobiphenyl is 2-chlorobiphenyl. Dill et al. (1982) tested all three monochlorobiphenyl isomers to three species of freshwater fish (Table 3). The 2-chlorobiphenyl was the most toxic to all species.

b. The most sensitive species to 2-chlorobiphenyl was rainbow trout (Table 3).

c. The only NOEC for the monochlorobiphenyl isomers is derived from a 32-d toxicity test for fathead minnows to 2-chlorobiphenyl (Dill et al. 1982). The 96-h LC50 for rainbow trout (540 ug/L) is about seven times lower than the 96-h LC50 for fathead minnows (4000 ug/L, Table 3). Therefore, the NOEC for fathead minnows was divided by seven to estimate a NOEC for rainbow trout (i.e., 380. - 550. ug/L divided by 7 equals 50. - 80. ug/L).

2. The NOEC for the early life stages (embryo-sac fry) of fish is estimated to be 2. - 3. ug/L.

a. Broyles and Noveck (1979a) reported that several investigators (Schimmel et al. 1974, Nebeker et al. 1974, and a personal communication from Mac M.J., W.H. Berlin, and D.V. Rottiers, Great lakes Fisheries Laboratory, Fish and Wildlife Service, U.S. Department of Interior, Ann Arbor, MI. 48105) "observed that fish of early developmental stages are more sensitive to PCBs". Mac et al. indicated that the "highest number of mortalities occurred before and up to yolk absorption; fewer mortalities were observed thereafter" as reported by Broyles and Noveck (1979a).

b. Sac fry fish appear to be about 25 times more sensitive to PCBs than juveniles or adults. Schimmel et al.

(1974) found that Aroclor 1254 was 32 times more toxic to sac fry than to juveniles of the sheepshead minnow (Cyprinodon variegatus). Nebeker et al. (1974) found that Aroclor 1242 was 20 times more toxic to newly hatched fry of fathead minnows than to 3-mo old fish.

c. Toxicity data for Aroclors were used to supplement data for individual PCB isomers because it was known that PCBs are more toxic to fry fish than juvenile fish, and the only studies which measured the relative sensitivities of these two life stages used Aroclors. These data for Aroclors are the best estimates available for the relative sensitivities of fry and juvenile fish and will be used for all PCB isomers classes until data for isomers becomes available.

d. The NOEC for embryo-sac fry fish was obtained by dividing the NOEC for the most sensitive fish (see Section III. A.1 above) by 25.

3. The acute (96-h) LC50 to juvenile rainbow trout for the monochlorobiphenyl isomers is about 780 ug/L (Figure 2).

B. Dichlorobiphenyl Isomers

1. The 30-day NOEC for the dichlorobiphenyl isomers to juvenile fish is estimated to be 12. ug/L.

a. No data were available for the dichlorobiphenyl isomers (Table 3). Therefore, the NOEC was estimated through statistical regression analysis of the relationship between PCB chlorine number and available NOECs for rainbow trout (Figure 3).

b. Figure 3 shows that as PCB chlorine number increases, the bioconcentration potential of a PCB, as indicated by it's octanol-water partition coefficient (Kow), also increases. Figure 3 also shows that the toxicity of PCB isomers

increases, as indicated by decreasing NOECs, with increasing chlorine number.

c. It is assumed that chronic toxicity is directly related to a chemical's Kow, if the log Kow is less than about 6 or 7 and if the chemical is a non-reactive non-electrolyte organic chemical. Hermens (1982) has shown that 16-d EC50s for Daphnia magna reproduction (a chronic toxicity endpoint) are linerally related to log Kow for a variety of organic chemicals. Konemann (1981) has shown a strong relationship between 14-d LC50s for guppies and log Kow (up to a log Kow of 6) for a group of non-reactive, non-electrolyte organic chemicals.

2. The NOEC for the dichlorobiphenyl isomers to the early life stages (embryo-sac fry) of fish is estimated to be about 0.5 ug/L.

a. The rationale is the same as presented above in Section III.A.1.a through d.

3. The acute (96-h) LC50 to juvenile rainbow trout for the dichlorobiphenyl isomers is estimated to be about 420 ug/L (Figure 2).

C. Trichlorobiphenyl Isomers

1. The 30-day NOEC for the trichlorobiphenyl isomers to juvenile fish is estimated to be about 2.1 ug/L.

a. A chronic NOEC was not available for the trichlorobiphenyl isomers (Table 3). Therefore, a NOEC was estimated through regression analysis of the relationship between toxicity and PCB chlorine number (Figure 3).

b. The rationale for using Figure 3 is the same as presented in Sections III.B.1.b and c.

2. The NOEC for the trichlorobiphenyl isomers to the early life stages (embryo-sac fry) of fish is estimated to be about 0.1 ug/L.

a. The rationale is the same as presented above in Sections III.A.1.a through d.

3. The acute (96-h) LC50 to juvenile rainbow trout for the trichlorobiphenyl isomers is estimated to be about 220 ug/L (Figure 2).

D. Tetrachlorobiphenyl Isomers

1. The 30-day NOEC for the non o,o'-chlorine (Cl) substituted (Figure 1) tetrachlorobiphenyl (TCB) isomers (5 isomers out of 42 possible isomers) to juvenile fish is less than 0.1 ug/L.

a. Stalling et al. (1979) have demonstrated that the NOEC for rainbow trout exposed to 3,3',4,4'-tetrachlorobiphenyl (TCB) for 50 d was less than 0.1 ug/L which was the lowest exposure concentration tested (Table 3).

b. Stalling et al. (1979) reported that studies by Goldstein et al. (1977) and Poland and Glover (1977) concluded that PCB isomers lacking o,o'-chlorine (Cl) substitution and having four or more Cl atoms, may account for a significant amount of the toxicity of PCB mixtures.

c. Stalling et al. (1979) determined the toxicities of four groups of PCBs: (1) 3,3',4,4'-tetrachlorobiphenyl (a O o,o'-Cl substituted PCB), (2) a mixture of 1 o,o'-Cl PCBs, (3) a mixture of 1 and 2 o,o'-Cl PCBs, and (4) a mixture of 2 - 4 o,o'-Cl PCBs. The 3,3',4,4'-TCB was more toxic to rainbow trout than any of the 1 thru 4 o,o'-Cl mixtures.

d. Bruggeman et al. (1981), Shaw and Connell (1980b), Goldstein et al. (1977), Poland and Glover (1977), and Stalling et al. (1979) have all discussed the importance of the ortho-ortho chlorine (o,o'-Cl) substitution pattern within a class of PCB isomers and its effect upon bioconcentration potential or toxicity. In general, it is suggested that the more chlorines substituted in the ortho-ortho positions on the biphenyl, the lower the bioconcentration potential and the toxicologic activity. Ortho substitution of Cl forces the biphenyl out of a common plane. In summary, the toxicity of PCBs appears to increase with the number of chlorines (Figure 3), and, within a class of PCB isomers, may decrease with greater ortho-ortho Cl substitution.

2. The 42-day NOEC for the 1 thru 4 o,o'-Cl substituted TCB isomers (37 isomers out of 42 possible isomers) to juvenile fish is about 1.5 ug/L.

a. Branson et al. (1975) have demonstrated a NOEC for rainbow trout exposed to 2,2',4,4'-TCB (a 2 o,o'-Cl substituted TCB; one Cl on each side of the biphenyl bond; Figure 1) for 42 d was 1.5 ug/L (Table 3). Branson et al. also showed a no-observed-lethal concentration (NOLC) of greater than 14 ug/L. These data are supported by the data reported by Dill et al. (1982) who exposed fathead minnows to 2,2',4,4'-TCB for 30 d. Dill et al. reported an LC50 of 29 ug/L and a NOEC of less than 14 ug/L (Table 3).

b. These data suggest that a 0 o,o'-Cl substituted TCB is more than 15 times more toxic to juvenile and adult fish than a 2 o,o'-Cl substituted TCB.

3. The NOEC for the 0 o,o'-Cl substituted TCB isomers to the early life stages (embryo-sac fry) of fish is estimated to be less than 0.004 ug/L.

a. The rationale is the same as presented above in Sections III.A.1.a through d.

4. The NOEC for the 1 thru 4 o,o'-Cl substituted TCB isomers to embryo-sac fry fish is estimated to be about 0.06 ug/L.

a. The rationale is the same as presented in Sections III.A.1.a through d.

5. The acute (96-h) LC50 to juvenile rainbow trout for the 1-4-0,0'-Cl substituted tetrachlorobiphenyl isomers is estimated to be about 120 ug/L (Figure 2, Table 3).

a. The acute (96-h) LC50 is for 2,2'2,4'-tetrachlorobiphenyl (Table 3).

6. The acute (96-h) LC50 to juvenile rainbow trout for the non 0,0'-Cl substituted TCB isomers could be as low as 10 ug/l.

a. It is probable that the acute toxicity of non 0,0'-Cl substituted TCB isomers to juvenile fish is greater than the acute toxicity of the three 4 0,0'-Cl substitute isomers because the chronic toxicity of the non 0,0'-Cl substituted isomers was greater than the 1-4 0,0'-Cl substituted isomers by about 15 times.

b. The acute (96-h) LC50 for the 1-4 0,0'-Cl substituted isomers was devided by 15 to estimate an acute (96-h) LC50 for the non 0,0'-Cl substituted isomers.

E. Pentachlorobiphenyl Isomers

1. The 30-day NOEC for the pentachlorobiphenyl isomers to juvenile fish is estimated to be 0.07 ug/L.

a. No data were available for the pentachlorobiphenyl isomers (Table 3). Therefore, the NOEC was estimated through regression analysis of the relationship between toxicity and PCB chlorine number (Figure 3).

b. The rationale for using Figure 3 is the same as presented above in Sections III.B.1.b and c.

2. The NOEC for the pentachlorobiphenyl isomers to the early life stages (embryo-sac fry) of fish is estimated to be about 0.003 ug/L.

a. The rationale is the same as presented in Sections III.A.1.a through d.

3. The acute (96-h) LC50 to juvenile rainbow trout for the pentachlorobiphenyl isomers is estimated to be similar to or less than the LC50 for tetrachlorobiphenyl isomers (about 120 ug/L).

a. The acute toxicity (96-h EC50s) of PCB isomers to aquatic invertebrates decreases with the higher chlorinated isomers (pentachlorobiphenyls and hexachlorobiphenyls). This decrease in toxicity is probably associated with the decreasing water solubility of these higher chlorinated PCBs. A more detailed rationale is in Section IV.A.

b. Since no toxicological information is available which indicates how much less acutely toxic the pentachlorobiphenyl isomers are to fish than the tetrachlorobiphenyls, acute toxicity was assumed to be similar.

F. Hexachlorobiphenyl Isomers

1. The 30-day NOEC for the hexachlorobiphenyl isomers to juvenile fish is estimated to be 0.01 ug/L.

a. No measured NOECs are available for the hexachlorobiphenyl isomers (Table 3). Therefore, the NOEC was estimated through regression analysis of the relationship between toxicity and PCB chlorine number in Figure 3.

b. The rationale for using Figure 3 is the same as presented above in Sections III.B.1.b and c.

2. The NOEC for hexachlorobiphenyl isomers to the early life stages (embryo-sac fry) of fish is estimated to be less than 0.001 ug/L.

a. The rationale is the same as presented above in Sections III.A.1.a through c.

b. In addition, Shimmel et al. (1974) exposed fry, juvenile, and adult sheepshead minnows to 0.1 ug/L Aroclor 1254 and demonstrated that fry were about 30 times more sensitive to PCBs than older fish. These data suggest a NOEC of 0.003 ug/L (i.e., 0.1 ug/L divided by 30) and support the estimated NOEC for sac fry fish of about 0.001 ug/L hexachlorobiphenyl.

c. The 2,2',4,4',5,5'-hexachlorobiphenyl (HCB) isomer (a 2 o,o'-Cl substituted isomer, one Cl on each side of the biphenyl bond) has been shown to be very toxic to sac fry of lake trout and chinook salmon (Table 3). Broyles and Noveck (1979b) exposed lake trout sac fry to 8 ug/L 2,2',4,4',5,5'-HCB for 15 d; all fish died within 79 d. All chinook salmon sac fry died within 31 d.

3. The acute (96-h) LC50 to juvenile rainbow trout for the hexachlorobiphenyl isomers is estimated to be similar to or less than the 96-h LC50 for tetrachlorobiphenyl isomers (about 120 ug/L).

a. The rationale is the same as for pentachlorobiphenyls (Section III.E.3).

G. Decachlorobiphenyl

1. The 30-day NOEC for decachlorobiphenyl to juvenile fish may be similar to or less than the hexachlorobiphenyl isomers. Since the chronic toxicity of decachlorobiphenyl to juvenile fish is not known, it will be assumed that toxicity will be similar to the hexachlorobiphenyl isomers: about 0.01 ug/L.

a. No data were available for decachlorobiphenyl (Table 3) and a NOEC was estimated through graphic interpolation of Figure 3. Estimating a NOEC for decachlorobiphenyl from available data or other PCB isomers will have a high degree of uncertainty due to decachlorobiphenyl's greater water insolubility relative to PCB isomers with six or less chlorines.

b. Konemann (1981) has shown a deviation from a linear quantative structure-activity relationship between log Kow and subchronic toxicity (14-d LC50) to fish with chemicals whose log Kow's are greater than six. For such chemicals effective concentrations should be larger than expected from the struture-activity relationship, and, thus, less toxic. However, the National Research Council (NCR 1979) reported that the degree of bioaccummulation of decacholorobiphenyl in aquatic organisms is greater than for 2,2',4,4',5,5'-hexachlorobiphenyl. Factors of 97,000 in fish and 930,000 in snails were measured in the terrestrial aquatic model ecosystem of Dr. R. Metcalf. Factors for hexachlorobiphenyl were 42,000 in fish and 100,000 in snails, If the bioaccummulation potential of decachlorobiphenyl and the hexachlorobiphenyl isomers are similar, their toxicity to fish could also be similar.

c. The simplist assumption is that the structure-activity relationship becomes asymptotic. Sugiura et al. (1978)

have suggested that bioconcentration factors become asymptotic for chemicals whose log Kow's are greater than six.

2. The NOEC for decachlorobiphenyl to embryo-sac fry fish may be similar to or less than the estimated NOEC for hexachlorobiphenyl: about 0.001 ug/L.

a. The rationale for using the value for hexachlorobiphenyl is presented above in Sections III.G.1.a through c.

3. The acute (96-h) LC50 to juvenile rainbow trout for decachlorobiphenyl will be similar to or less than the LC50 estimated for tetrachlorobiphenyl isomers (about 120 ug/L).

a. The rationale for using the value for tetrachlorobiphenyls is presented above in Section III.E.3.

IV. Toxicity of Various PCB Isomers to Aquatic Invertebrates

Various PCB isomers are acutely toxic to aquatic invertebrates and toxicity increases with an increase in the number of chlorines on the biphenyl up through tetrachlorobiphenyl. Acute toxicity ranges from 700 to 30 ug/L. Chronic toxicity of PCB isomers to aquatic invertebrates is not available, however, aquatic invertebrates are expected to have lower chronic NOECs than those measured for juvenile fish.

A. Acute Toxicity

1. Various PCB isomers are acutely toxic to aquatic invertebrates. Toxicity increases with chlorination and reaches a maximum with the tetrachlorobiphenyls at about 30 ug/L.

a. Static acute (48-h or 96-h) toxicity values (EC50s) have been measured for one or more isomers of every PCB class from monochlorobiphenyl through hexachlorobiphenyl (Table 4).

b. The EC50 values of the monochlorobiphenyl isomers through the tetrachlorobiphenyl isomers are linerally related (Figure 4) and this relationship can be defined through statistical regression analysis: log EC50 (ug/L) = 3.04 - 0.411 chlorine no. (R^2 = 0.92, N = 7). These data indicate that the average EC50 value for the isomers of each class of PCB from one to four chlorines are:

CLASS	VALUE (ug/L)
monochlorobiphenyls	500.
dichlorobiphenyls	110.
trichlorobiphenyls	70.
tetrachlorobiphenyls	30.

2. Acute toxicity of higher chlorinated PCBs (five or more chlorines) is reduced due to their water insolubility.

a. Isomers of higher chlorinated PCBs, i.e., pentachlorobiphenyls, hexachlorobiphenyls, and decachlorobiphenyl are less acutely toxic than tetrachlorobiphenyl isomers (Table 4, Figure 4). These higher chlorinated isomers are becoming relatively more water insoluble which results in reduced uptake rates into aquatic organisms. Thus, acute exposure to low water concentrations does not permit enough PCB to be taken up to kill 50% of a test population. Konemann (1981) and Sugiura et al. (1978) have observed reduced acute toxicity and bioconcentration, respectively, with decreasing water solubility of organic chemicals whose log Kow is greater than six.

B. Chronic Toxicity

1. Chronic toxicity NOECs are not available for

individual PCB isomers, however, NOECs for aquatic invertebrates are expected to be equal to or less than the subchronic NOECs measured and estimated for juvenile fish (Table 5).

a. Aquatic invertebrates have been shown to be more sensitive (i.e., lower EC50 values) to various PCB isomers than fish during acute exposures (Mayer et al. 1977; and Tables 3 and 4) or just as sensitive as the most sensitive fish species tested (Dill et al. 1982, and Tables 3 and 4).

b. It is assumed that trends between aquatic invertebrates and fish with regard to acute toxicity of PCB isomers will also be observed with chronic toxicity information when it becomes available.

c. A NOEC of Aroclor 1254 (i.e., "a safe level of A-1254") for an aquatic invertebrate was suggested as being below 1 ug/L by Nebeker and Puglisi (1974). They calculated a 3-wk LC50 of 0.45 ug/L of Aroclor 1254 with respect to reproductive impairment. This NOEC suggested for Aroclor 1254 is consistent with NOECs for PCB isomers to juvenile fish (Table 5).

V. Toxicity of Various PCB Isomers to Algae and Protozoa

A. Algae

The PCB isomers may be just as toxic to algae as they are to juvenile fish.

1. Only 2,4'-dichlorobiphenyl (DCB) has been tested. Moore and Harriss (1972) predicted a NOEC of less than 7 ug/L after a 24h *in situ* exposure of 2,4'-DCB to a natural marine phytoplankton community (Table 6). This NOEC is similar to the 30-day (subchronic) NOEC estimated for the DCB isomers to juvenile fish (12 ug/L, Section III.B.1, Figure 3).

B. Protozoa

The NOECs of PCB isomers (monochlorobiphenyl through hexachlorobiphenyl) to protozoa appear to be about 100 ug/L or greater.

1. Dive et al. (1976) measured the effect of 13 PCB isomers (Table 6) on the growth of the ciliated protozoa (Colpidium campylum). In general, Dive et al. concluded that toxicity decreased as the number of chlorines increased.

2. Dive et al. also argued that their results with isomers were consistent with results from Aroclor mixtures using several protozoan species. With one execption, all NOECs for Aroclor mixtures were 100 ug/L or greater.

VI. Toxicity of PCB Isomers to Wild Mammals and Birds

Toxicity studies of PCB isomers to wild mammals and birds are not available.

VII. Environmental Concerns of Polychlorinated Biphnyl (PCB) Commercial Mixtures

Ambient concentrations and food chain transport of PCBs may impair the reproductive potential of commercial fisheries and wild mammals, e.g., mink. PCB residues are also strongly correlated with reductions in natural populations of marine mammals and may be correlated with declines in river otter populations. High PCB residues have been found in various birds, especially gulls and carnivorous birds, but no resulting effects have been firmly demonstrated.

A. Commercial Fisheries

PCBs may, even at low concentrations, contribute to a

reduction in populations of sport and economically important fish.

1. PCBs may affect the reproductive success in fish.

a. High body burdens (120 ug/g wet weight in the ovaries) in wild fish (Baltic flounder) been correlated with failure of eggs to hatch. (Von Westernhagen et al. 1981).

b. Experimental data have demonstrated that PCBs can reduce spawning, hatching, and survival of many species of fish. Of the species tested, one (brook trout) is important to recreational fisheries. Bengtsson 1980; Mauck et al. 1978; DeFoe et al. 1978).

c. Seelye and Mac (1981) exposed fry hatched from eggs from Lake Michigan lake trout and fry hatched from hatchery lake trout to 50 ng/L Aroclor 1254 for 50 days. The observed mortality was site-specific. Only the Lake Michigan fish, both the exposed fry and the unexposed control fry, showed size-specific mortality, i.e., the smaller fry died first. The Lake Michigan fish had large residues of PCBs prior to testing; the hatchery reared lake trout had relatively small PCB residues. Although several factors could cause size-specific mortality, Seelye and Mac (1981) suggested that the cause was the large PCB residues.

d. Early life stages (embryos and sac fry) of fish appear to be about 25 times more sensitive to PCBs than juvenile and adult fish (Schimmel et al. 1974, Nebeker et al. 1974).

e. Predicted no-observed-effect concentrations (NOEC) for the early life stages of fish can be found in Table 5 for each class of PCB isomers and these NOECs are similar to toxicity data for PCB commercial mixtures.

f. Many fish species spawn in shallow near-shore areas, and eggs and sac fry rest on the top layer of sediment where PCBs tend to accumulate in the aquatic environment. The early life stages of fish have the potential to receive high exposure of PCBs.

2. PCBs may have direct adverse effects on juvenile and adult fish.

a. Experimental laboratory data have demonstrated that both growth and survival of many fish species are reduced at very low exposure concentrations (0.4 and 4.0 ug/L, respectively).

b. Mauck et al. (1978) exposed brook trout fry to Aroclor 1254 for 118 days post-hatch. Growth was reduced at 1.5 ug/L while survival was reduced at 3.1 ug/L.

c. DeFoe et al. (1978) performed a 240-day life cycle test with fathead minnows and Aroclors 1248 and 1260. The lowest effect concentrations were 0.4 ug/L Aroclor 1248 for reduced growth and 4.0 ug/L Aroclor 1260 for reduced survival.

d. Toxicity of PCB Aroclors and PCB isomers to fish appears to be related to the number of chlorines attached to the biphenyl ring (Figure 1). The greater the number of chlorines (up through six chlorines), the greater the toxicity (Table 3, Figure 3). This toxicity appears to increase an order-of-magnitude with each added chlorine.

e. Subchronic NOECs for juvenile fish can be found in Table 5 for each class of PCB isomers.

f. PCB isomers and mixtures that have no o,o'-Cl substitution appear to be more toxic than PCB isomers with 1 to 4 chlorines substituted in the o,o' positions on the biphenyl

(Figure 1). See information for tetrachlorobiphenyl in Section III and Table 3).

g. Impaired bone development and abnormalities in testes have been observed in two species of fish (brook trout and Atlantic cod) fed or exposed to low levels of PCBs (0.4 ug/L). (Mauck et al. 1978; Sangalang et al. 1981).

3. PCBs can affect the survival of aquatic invertebrates and aquatic plants which are food sources for the fish.

a. PCBs can have lethal and sublethal effects on environmentally important freshwater invertebrates.

(1) Experimental laboratory data have shown that PCBs are toxic to many aquatic invertebrates (Daphnia magna; juvenile scuds, Gammarus pseudolimnaeus; and midges, Tanytarsus [Paratanytarsus] disimilis) in the low ug/L range. (Nebeker and Puglisi 1974).

(2) Experimental data have demonstrated that very low concentrations of PCBs can result in reproductive impairment of aquatic invertebrates (Daphnia magna reproduction was impaired by 16% with 0.48 ug Arclor-1254/L and Gammarus Pseudolimnaeus reproduction was impaired by 50% with 5.1 ug Aroclor- 1248/L). (Nebeker and Puglisi 1974).

b. PCBs affect productivity of phytoplankton and the composition of phytoplankton communities.

(1) PCBs (1 ug/L) decrease the photosynthetic rate of different species of algae. (O'Connors and Mahanty 1979, Kricher and Bayer 1977).

(2) PCBs are differentially toxic to different species of algae at or below 1 ug/L. (O'Connors et al. 1978, and Glooschenko and Glooschenko 1975).

B. Contamination of Food Resources

1. PCBs can be concentrated and transferred in freshwater and marine phytoplankton, invertebrates, fish and mammals; and can result in indirect human exposure by consumption of economically important food resources, the closure of fisheries, and economic losses as a result of this contamination.

a. Numerous sources have demonstrated and reviewed the propensity of PCBs to bioconcentrate in aquatic organisms (32,000X-270,000X) and to be transferred upward in the food web. (Keil et al. 1971; Biggs et al. 1980; Anonymous 1980; Shaw and Connell 1980a,b; Bleavins et al. 1980; Thomann 1978; Weininger 1978; Peterson and Guiney 1979; US EPA 1977, 1980).

b. Residue data collected in the environment demonstrate that PCBs are ubiquitous in aquatic organisms and can be bioaccumulated as much as 15000X over short periods of time (14 days) from contaminated sites based on water concentrations. (Skea et al. 1979, Anonymous 1980, Swain 1980, Davis et al. 1981, Risebrough et al. 1968).

c. Polychlorinated biphenyls can also be bioconcentrated in fish and aquatic invertebrates through contaminated diets and diets alone may contribute a significant proportion to the total body residue of PCBs. (Pizza and O'Connor 1983, Spigarelli et al. 1983, Rubinstein et al. 1983).

d. Residue data collected from commercial and sport fisheries demonstrate that significant quantities of PCBs may be transferred to humans through consumption of fish. (Swain 1982, Schmitt et al. 1981, Zimmerman 1982).

C. Mammals and Birds

1. PCBs may impair reproductive success in some wild mammals (e.g., mink) and birds.

a. Clinical signs (e.g., reproductive failure, death of female breeder mink, impaired growth of kits, excessive early mortality of kits, reduced birth weights of kits, reduced litter sizes, emaciation, anoroxia, and blood stools), pathology (e.g., liver enlargement, hemorrhagic ulcers, degeneration of the liver and kidneys, and fatty infiltration of the liver), and mortality patterns are very similar for mink fed (fish) diets containing PCBs and for mink fed PCBs. (Aulerich et al. 1977).

b. Dietary exposure to mink to PCBs (5 mg/kg) causes reduction in number of kits and reduced survival of kits. (Bleavins et al. 1980, Aulerich et al. 1977, Jensen et al. 1977).

c. PCBs accelerate destruction and alter biosynthesis of normal body steroids (estradiol, testosterone, rosrene-3,17-dione, pregnenolone, progesterone, and androstenedione) in birds and wild mammals. Hormonal alteration can affect mammals that exhibit delayed implantation and mating behavior in birds. (Risebrough et al. 1968, Lincer and Peakall 1970, Nowicki and Norman 1972, Freeman and Sangalang 1977, Reijnders 1980).

d. PCBs are correlated with reductions in natural populations of marine mammals, such as, harbor seals, ringed seals, and California sea lions. (Helle et al. 1976; Reijnders 1980).

e. PCB residues higher than those found in reproductively impaired mink have been found in river otters in the lower Columbia River Valley in Oregon. River otter harvests

by trapping have declined in this area, but have risen in other parts of Oregon where PCB residues are negligible or undetectable (Henny et al. 1981).

f. Experimental laboratory studies have demonstrated that PCBs in the diet (10 mg/kg) of birds (i.e., ring doves and ring-neck pheasants) caused early embryonic mortality and reduced egg production. (Peakall et al. 1972; Dahlgren and Linder 1971).

g. High PCB residues have been found in a number of avian species, especially in the Great Lakes area. Some birds (e.g., gulls) have shown population declines, but no cause and effect relationship has been determined. (Heinz et al. Unpublished Manuscript, Gilbertson and Hale 1974).

Risk is predicted to occur if a hypothetical surface water concentration exceeds any point of the dose-response curves available for individual PCB isomers. Eighteen hypothetical situations from Table H-1 (Versar 1983) were assessed and characterized for potential effects to fish populations. L = lethality to juvenile and adult fish (the subscript indicates percent of a population expected to be affected by a particular effect). E = effects to the early life stages of fish, i.e., embryos and sac fry. SL = indicates sublethal effects to juvenile and adult fish. G = reduction in growth and is a particular sublethal effect.

Table H-1. Estimated PCB Surface Water Concentrations Downstream of Organic Chemical Plants

Percentile[a] Average	Percentile[a] Low	Stream dilution factors[b] Average streamflow	Stream dilution factors[b] Low streamflow	PCB surface water concentration[c] (ug/l) Average streamflow	PCB surface water concentration[c] (ug/l) Low streamflow
	10		1.52		66
	20		6.36		16
	30		18.9		5.3
	40		56.1		1.8
10		58.2		1.7	
	50		179		0.56
20		214		0.47	
	60		476		0.21
30		522		0.19	
40		1,130		0.088	
	70		1,780		0.056
50		2,620		0.038	

	Class of PCB						
			1 to 0.1	0.1 to 0.01			
1	2	3	4	4	5	6	10
SL	SL	L	$L_{>50}$	L	L	L	L
E	SL	SL	L	L	L	E_{100}	E_{100}
E	E	SL	SL	L	L	L	L
	E	E	SL	L	L	L	L
	E	E	SL	L_{25}	L	L	L
	E	E	E	SL	L	L	L
	E	E	E	G_{60}	SL	SL	SL
		E	E	G	SL	SL	SL
		E	E	G_{30}	SL	SL	SL
		E	E	SL	SL	SL	SL
			E	E	E	SL	SL
				E	E	SL	SL

60		5,630		0.018					
	80		6,270		0.016				
70		15,300		0.0065					
	90		40,000		0.0025				
80		63,700		0.0016					
90		270,000		0.00037					

				E	E	SL	SL
				E	E	SL	SL
				E	E	E	E
					E	E	E
						E	E

[a]Percentile refers to the percent of plants with stream dilution factors less than or equal to the stated value. For example, 20 percent of the 348 plants upon which this frequency distribution is based have stream dilution factors of 214 or less at average streamflow and 6.36 or less at low streamflow.

[b]All data obtained from EPA's IFD/GAGE File (see Section H.2. for details).

[c]Assuming that all plants discharge process wastewater containing 100 ug/l of PCBs, the PCB surface water concentrations were calculated by dividing 100 ug/l by the respective dilution factor.

aquatic invertebrates. Risk is predicted to occur if a [illegible] concentration exceeded any point of the dose-response curves available for individual PCB isomers. Eighteen hypothetical situations from Table H-1 (Versar 1983) were assessed and characterized for potential effects to populations of aquatic invertebrates. L = lethality to aquatic invertebrates (the subscript indicates percent of a population expected to be affected by a particular effect). SL = indicates sublethal effects to aquatic invertebrates.

Table H-1. Estimated PCB Surface Water Concentrations Downstream of Organic Chemical Plants

Percentile[a] Average	Percentile[a] Low	Stream dilution factors[b] Average streamflow	Stream dilution factors[b] Low streamflow	PCB surface water concentration[c] (ug/l) Average streamflow	PCB surface water concentration[c] (ug/l) Low streamflow
	10		1.52		66
	20		6.36		16
	30		10.9		5.8
	40		56.1		1.8
10		58.2		1.7	
	50		179		0.56
20		214		0.47	
	60		476		0.21
30		522		0.19	
40		1,130		0.088	
	70		1,780		0.056
50		2,620		0.038	

Class of PCB

1	2	3	4	5	6	7
SL	SL	L_{50}	$L_{>50}$	L	L	L	L
	SL	SL	$L_{<50}$	L	L	L	L
		SL	SL	L	L	L	L
			SL	L	L	L	L
			SL	L	L	L	L
				SL	L	L	L
				SL	SL	SL	SL
				SL	SL	SL	SL
				SL	SL	SL	SL
				SL	SL	SL	SL
						SL	SL
						SL	SL

60		5,630		0.018					
	80		6,270		0.016				
70		15,300		0.0065					
	90		40,000		0.0025				
80		63,700		0.0016					
90		270,000		0.00037					

								SL	SL
								SL	SL

[a]Percentile refers to the percent of plants with stream dilution factors less than or equal to the stated value. For example, 20 percent of the 348 plants upon which this frequency distribution is based have stream dilution factors of 214 or less at average streamflow and 6.36 or less at low streamflow.

[b]All data obtained from EPA's IFD/GAGE File (see Section H.2. for details).

[c]Assuming that all plants discharge process wastewater containing 100 ug/l of PCBs, the PCB surface water concentrations were calculated by dividing 100 ug/l by the respective dilution factor.

Table 3. Toxicity of Various Isomers of Polychlorinated Biphenyls (PCBs) to Fish

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
			MONOCHLOROBIPHENYLS		
2-	Bluegill (Lepomis macrochirus)	S,N,96h	LC50	1100.	Dill et al. 1982
2-	Fathead Minnow (Pimephales promelas)	S,N,96h	LC50	4000.	Dill et al. 1982
2-	Fathead Minnow (Pimephales promelas)	FT,M,32d	LC50 NOEC	820. 380.-550.	Dill et al. 1982
2-	Rainbow Trout (Salmo gairdneri)	S,N,96h	LC50	540.	Dill et al. 1982
2-	Sheepshead Minnow (Cyprinodon variegatus)	S,N,96h	LC50	4100.	Dill et al. 1982
3-	Bluegill (Lepomis macrochirus)	S,N,96h	LC50	2400.	Dill et al. 1982
3-	Fathead Minnow (Pimephales promelas)	S,N,96h	LC50	7800.	Dill et al. 1982
3-	Rainbow Trout (Salmo gairdneri)	S,N,96h	LC50	1000.	Dill et al. 1982
4-	Bluegill (Lepomis macrochirus)	S,N,96h	LC50	1300.	Dill et al. 1982

Table 3. (cont.)

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
4-	Rainbow Trout (Salmo gairdneri)	S,N,96h	LC50	900.	Dill et al. 1982
4-	Sheepshead Minnow (Cyprinodon variegatus	S,N,96h	LC50	680.	Dill et al. 1982
		DICHLOROBIPHENYLS			
		No data are available.			
		TRICHLOROBIPHENYLS			
2,3',4-	Guppy (Poecilia reticulata)	S,R,N,14d	LC50	100.	Konemann 1981
2,3,4-				350.	
2,4,5-				180.	
2,4',5-				180.	
2,3',5-				> 290.	
2,3,6-				150.	
2,4,6-				400.	
2,2',5				100.	

Table 3. (cont.)

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
			TETRACHLOROBIPHENYLS		
3,3',4,4'-	Rainbow Trout (Salmo gairdneri)	FT,M,50d EXP, 28d ELIM	NOEC	< 0.1	Stalling et al. 1979
			Growth (50d):		
			29% reduction (wwt)	0.1	
			61% reduction (wwt)	0.4	
			Lethality:		
			25% on d78	1.4	
			Food consumption reduced.		
			Disorientation		
2,2',4,4'-	Rainbow Trout (Salmo gairdneri)	FT,M,42d	NOEC	1.5	Branson et al. 1975
			Feeding: less vigorous		
			Growth: 1 in 5 lost wt.		
			Liver:		
			28% reduction (wwt)		
			NOLC	> 14.	

Table 3. (cont.)

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
2,2'4,4'-	Fathead Minnow (Pimephales promelas)	FT,M,30d	LC50 NOEC Major effects: Lethality, Melanization, and Disorientation. Other effects: Positive buoyance, Loss of appetite, Bulging eyes, and Hemorrhaging.	29. < 14.	Dill et al. 1982
2,2',4,4'-	Rainbow Trout (Salmo gairdneri)	S,N,96h	LC50	120.	Dill et al. 1982
2,2',4,4'-	Bluegill (Lepomis macrochirus)	S,N,96h	LC50	120.	Dill et al. 1982
2,3,4,5-	Guppy (Poecilia reticulata)	S,R,N,14d	LC50	> 340.	Konemann 1981
2,2',6,6'-				> 340.	

PENTACHLOROBIPHENYLS

No data are available.

Table 3.(cont.)

	Species	Method*	Effect	Value (ug/L)	Reference
		HEXACHLOROBIPHENYLS			
2,2',4,4',5,5'-	Lake Trout (Salvelinuse namaycush)	S,R,M, Sac fry, 15d EXP (8 ug/L), 64d ELIM	Lethality: 100% by 79d		Broyles and Noveck 1979b
2,2',4,4',5,5'-	Chinook Salmon (Oncorhynchus tshawytscha)	S,R,M, Sac fry, 15d EXP (8 ug/L), 16d ELIM	Lethality: 100% by 31d		Broyles and Noveck 1979b
		DECACHLOROBIPHENYL			
	No data are available.				

* S = static, N = nominal concentrations used to estimate effect value, FT = flow through, M = measured concentrations, EXP = exposure period, ELIM = elimination period, R = renewal of test solution, NOEC = no-observed-effect concentration, NOLC = no-observed-lethality concentration, and wwt = wet or fresh weight.

Table 4. Toxicity of Various Isomers of Polychlorinated Biphenyls (PCBs) to Aquatic Invertebrates.

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
		MONOCHLOROBIPHENYLS			
2-	Daphnids (Daphnia magna)	S,N,48h	EC50	700.	Dill et al. (1982)
3-	Daphnids (Daphnia magna)	S,N,48h	EC50	430.	Dill et al. (1982)
4-	Daphnids (Daphnia magna)	S,N,48h	EC50	420.	Dill et al. (1982)
		DICHLOROBIPHENYLS			
2,3-	Oyster (Crossostrea virginica)	FT,N,65d	NOEC Weight. Lipid content. Average. mortality.	>0.06	Vreeland (1974)
2,4'-	Scud (Gammarus pseudolimnaeus)	S,N,96h	EC50	120.	Mayer et al. (1977
4,4'	Scud (Gammarus pseudolimnaeus	S,N,96h	EC50	100	Mayer et al. (1977
		TRICHLOROBIPHENYLS			
2,3,4'	Scub, amphipod. (Gammarus pseudolimnaeus)	S,N,96h	EC50	70	Mayer et al. (1977

Table 4. (Cont.)

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
2',3,4-	Oyster (Crassostrea Virginica)	FT,N,65d	NOEC Weight Lipid content. Average mortality.	>0.06	Vreeland (1974)
		TETRACHLOROBIPHENYLS			
2,2',4,4'-	Daphnids (Daphnia magna)	S,N,48h	EC50	30	Dill et al. (1982)
2,2',5,5'- 2,2',3,5'-	Oyster (Crassostrea virginca)	FT,N,65d	NOEC Weight. Lipid content. Average mortality.	>0.06	Vreeland (1974)
		PENTACHLOROBIPHENYLs			
2,2',4,5,5'-	Scud, amphipod. (Gammarus pseudolimnaeus)	S,N,96h	EC50	210	Mayer et al. (1977)
2,2',3,4,5'-	Oyster (Crassostrea virginica)	FT,N,65d	NOEC Weight. Lipid content. Average mortality.	>0.06	Vreeland (1974)

Table 4. (Cont.)

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
		HEXACHLOROBIPHENYLS			
2,2',4,4',6,6'-	Scud, amphipod. (Gammarus pseudolimnaeus)	S,N,96h	EC50	150	Mayer et al. (1977)
2,2',4,4',5,5'-	Oyster (Crassostrea virginica)	FT,N,65d	NOEC Weight. Lipid content. Average mortality.	>0.06	Vreeland (1974)

*S = static, N = nominal concentrations used to estimate effect value, FT = flow through, NOEC = no-obser effect concentration.

Table 5. The no-observed-effect concentrations (NOEC) of the monochlorobiphenyl through hexachlorobiphenyl isomers and decachlorobiphenyl determined for juvenile-adult fish and early life stages (i.e., embryos and sac fry) of fish.

	Fish NOEC (ug/L)	
Chlorine Number	Juvenile-Adult	Embryo-Sac Fry
1	50. - 80.	2. - 3.
2	12.	0.5
3	2.1	0.1
4	< 0.1 - 1.5	< 0.004 - 0.06
5	0.07	0.003
6	0.01	0.001
10	0.01	0.001

Table 6. Toxicity of Various Isomers of Polychlorinated Biphenyls (PCBs) to Algae and Protozoa.

Isomer	Species	Method*	Effect	Value (ug/L)	Reference
			ALGAE		
2,4'-	Natural marine phytoplankton community; NE Gulf of Mexico, FL.	In situ, N,24h	^{14}C uptake		Moore and Harriss (1972)
			EC50	30.	
			NOEC	<7.	
			PROTOZOA		
			MONOCHLOROBIPHENYLS		
2-	Ciliated protozoa (Colpidium campylum	S,N,43h	Growth (%):		Dive et al. (1976)
			LC100	<10000.	
			EC50	>1000.	
			NOEC	>100.	
			DICHLOROBIPHENYLS		
2,3-			LC100	<1000.	
			NOEC	>100.	
2,5-			LC100	<1000.	
			NOEC	>100.	
4,4'-			NOEC	>10000.	
			TRICHLOROBIPHENYLS		
2,2',5-			LC100	<1000.	
			NOEC	100.	

Table 6 (Cont.)

somer	Species	Method*	Effect	Value (ug/L)	Reference
,4,6-	Ciliated protozoa (Colpidium campylum	S,N,43h	Growth (%):		Dive et al. (1976)
			LC100	<1000.	
			NOEC	>100.	
,4,5-			LC100	<1000.	
			NOEC	>100.	
,3',5-			LC100	<10000.	
			EC90	1000.	
			NOEC	>100.	
			TETRACHLOROBIPHENYLS		
,2',5,5'-			NOEC	100.	
			PENTACHLOROBIPHENYLS		
,2',4,5,5'			NOEC	10000.	
,2'3',4,5-			NOEC	>10000.	
,3',4,4',5-			NOEC	>10000.	
			HEXACHLOROBIPHENYLS		
,2',3,3',4',5'-			NOEC	1000.	

S = static, N = nominal concentrations used to estimate effect value, NOEC = no-observed-effect oncentration, EC50 = median effective concentration, LC100 = concentration which killed 100% of the test rganisms, and EC90 = concentration which reduced growth of test population 90% below control population.

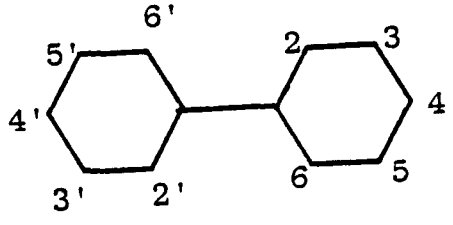


Figure 1. A polychlorinated biphenyl (PCB) is a family of compounds which consists of biphenyl that has been chlorinated at 10 possible sites. For example, monochlorobiphenyl has been chlorinated at a single site and decachlorobiphenyl has been chlorinated at all 10 sites. A o,o'-Cl substituted PCB refers to the chlorination of the 2,2',6, and 6' sites which are the ortho-ortho prime sites on the biphenyl.

Figure 2. Relationships between chlorine number of a PCB, n-octanol/water pertition coefficient (Kow), and acute (96h) median lethal concentration (LC50) for rainbow trout. The relationship for Kow and chlorine number was modified from Wasik et al. (1982, Fig. 3, p. 10). The LC50s for the monochloro-biphenyl isomers for tetrachlorobiphenyl are from Table 3. The relationship between 96-h LC50 and chlorine number of a PCB is defined by the regression equation: log LC50 (ug/L) = 3.16 - 0.27 Chlorine no. (R^2 = 0.92; N = 4) and probably becomes asymptotic around log Kow 6 - 7. Rationale is provided in Sections III.G.1.b. and c.

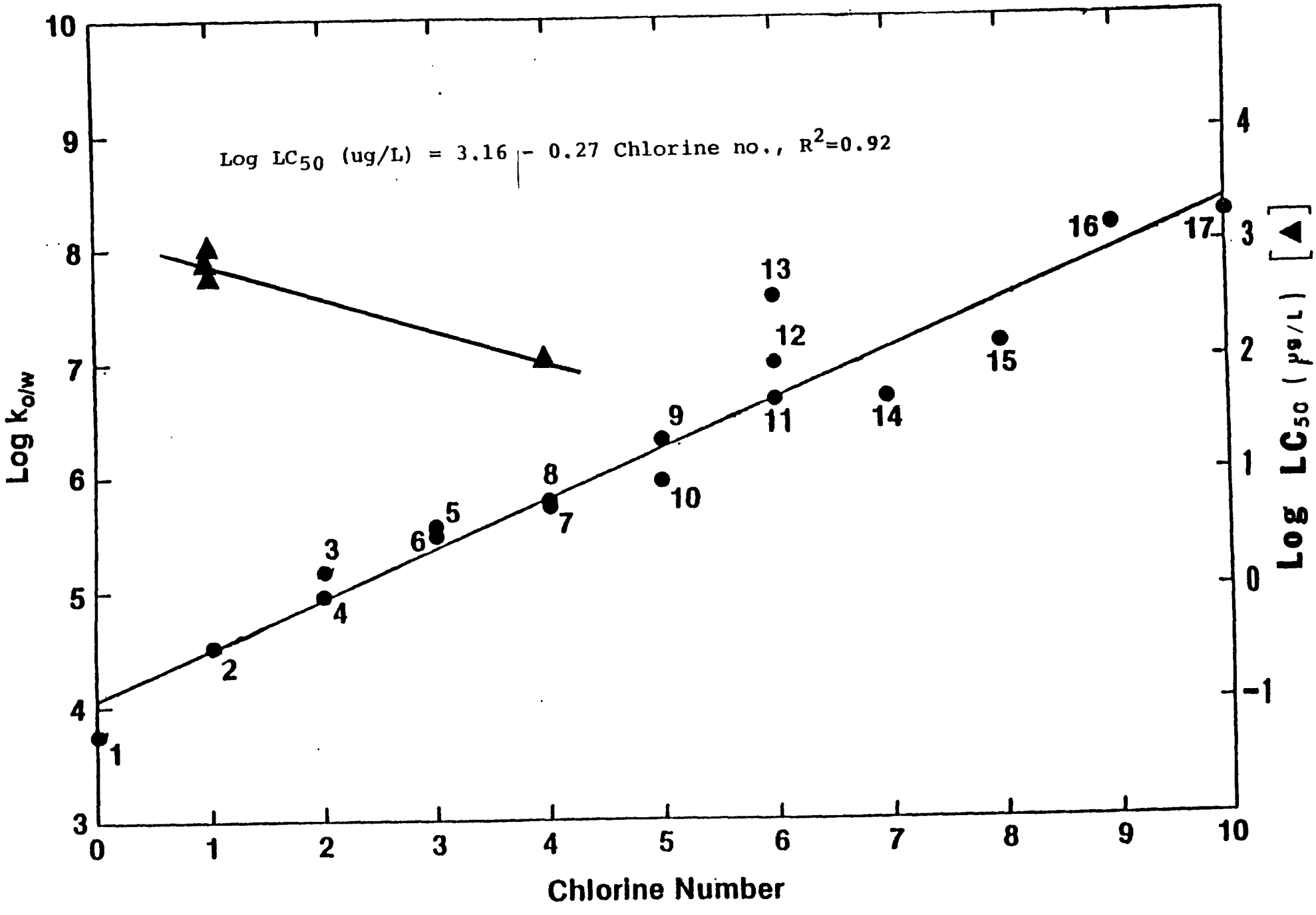

Log LC_{50} (ug/L) = 3.16 - 0.27 Chlorine no., R^2=0.92
Log $k_{o/w}$
Log LC_{50} (μg/L)
Chlorine Number
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17

Figure 3. Relationships between chlorine number of a PCB, n-octanol/water partition coefficient (Kow), and no-observed-effect concentration (NOEC) for rainbow trout. The relationship for Kow and chlorine number was modified from Wasik et al. (1982, Fig. 3, p. 10). The NOECs for monochlorobiphenyl are from Section III.A.1; the NOECs for tetrachlorobiphenyl are from Section III.D.1. and Table 3. The relationship bewteen NOEC and chlorine number of a PCB is defined by the regression equation: log NOEC (ug/L) = 2.53 -0.74 chlorine no. (R^2 = 0.87) and probably becomes asymptotic around log Kow 6 - 7. Rationale is provided in Sections III.G.1.b. and c.

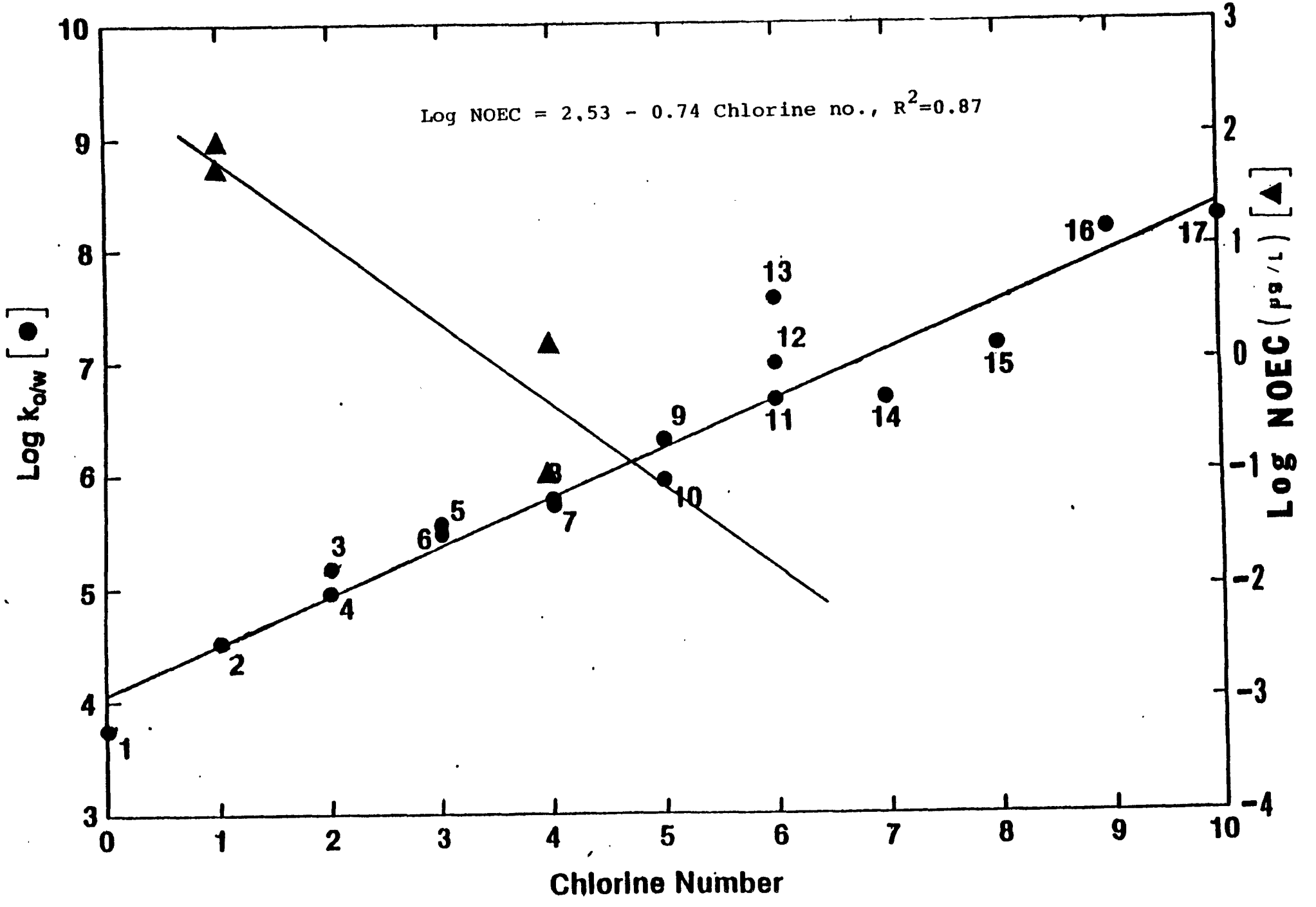
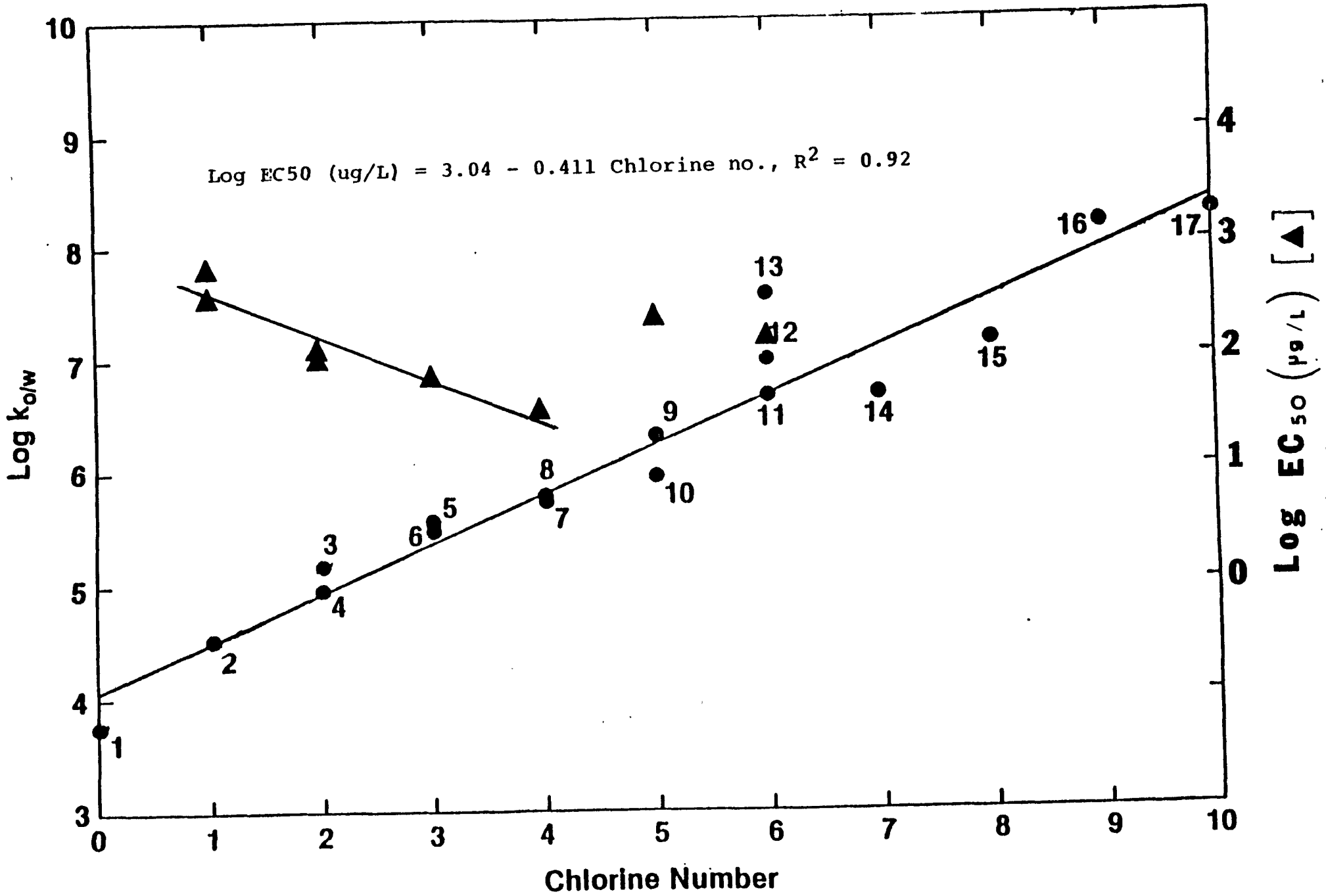

Log NOEC = 2.53 - 0.74 Chlorine no., R^2=0.87
Log $k_{o/w}$ [●]
Log NOEC (μg/L) [▲]
Chlorine Number
0
1
2
3
4
5
6
7
8
9
10
3
4
5
6
7
8
9
10
-4
-3
-2
-1
0
1
2
11
12
13
14
15
16
17

Figure 4. Relationship between chlorine number of a PCB, n-octanol/water partition coefficient (Kow), and acute (96h) median lethal concentration (EC50) for the aquatic invertebrates: Gammarus pseudolimnaeus (a scud or amphipod) and Daphnia magna. The relationship for Kow and chlorine number was modified from Wasik et al. (1982, Fig. 3, p. 10). The EC50s for the PCB isomers are from Table 4. The relationship between 96-h EC50 and chlorine number for PCB isomers from monochlorobiphenyl to tetrachlorobiphenyl (for log Kow's less than 6) is defined by the linear regression equation: log EC50 (ug/L) = 3.04 - 0.411 Chlorine no. (R^2 = 0.92, N = 7). The EC50s for the pentachlorobiphenyl and hexachlorobiphenyl isomers are greater than would be predicted by the regression equation (i.e., not as toxic as tetrachlorobiphenyls). Pentachlorobiphenyl and hexachlorobiphenyl isomers are apparently becoming to water insoluble (log Kow's of about 6 or greater) to cause acute toxicity at concentrations lower than about 30 ug/L, which is the EC50 for tetrachlorobiphenyl.


Log EC50 (ug/L) = 3.04 - 0.411 Chlorine no., R^2 = 0.92
Log $k_{o/w}$
Log EC_{50} (µg/L) [▲]
Chlorine Number
0
1
2
3
4
5
6
7
8
9
10
3
4
5
6
7
8
9
10
0
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VIII. REFERENCES

Anonymous. 1980. The invisible menance: contaminants in the Great Lakes. Madison, Wisconsin: the University of Wisconsin Sea Grant Program. WIS-SG-80-133.

Aulerich RJ, Ringer RK. 1977. Current status of PCB toxicity to mink, and effect on their reproduction. Arch Environm Contam Toxicol 6:279-292.

Bengtsson B-E. 1980. Long-term effects of PCB (Clophen A50) on growth, reproduction and swimming performance in the minnow, Phoxinus phoxinus. Water Research 14:681-687.

Biggs DC, Powers CD, Rowland RG, O'Connors HB, Wurster CF. 1980. Uptake of polychlorinated biphenyls by natural phytoplankton assemblages: field and laboratory determination of 14C-PCB particle-water index of sorption. Environ Pollut Ser A 22:101-110.

Bleavins MR, Aulerich RJ, Ringer RK. 1980. Polchlorinated biphenyls (Aroclors 1016 and 1242): effects on survival and reproduction in mink and ferrets. Arch Environ Contam Toxicol 9:627-635.

Branson DR, Blau GE, Alexander HC, Neely WB. 1975. Bioconcentration of 2,2',4,4'-tetrachlorobiphenyl in rainbow trout as measured by an accelerated test. Amer Fish Soc 104:785-792.

Broyles RH, Noveck MI. 1979a. Uptake and distribution of 2,5,2',5'-tetrachlorobiphenyl in developing lake trout. Toxicol Appl Pharmacol 50:291-298.

Broyles RH, Noveck MI. 1979b. Uptake and distribution of 2,4,5,2',4',5'-hexachlorobiphenyl in fry of lake trout and chinook salmon and its effects on viability. Toxicol Appl Pharmacol 50:299-308.

Bruggeman WA, Martron LBJM, Kooiman D, Hutzinger O. 1981. Accumulation and elimination kinetics of di-, tri- and tetrachlorobiphenyls by goldfish after dietary and aqueous exposure. Chemosphere 10:811-832.

Dahlgren RB, Linder RL. 1971. Effects of polychlorinated biphenyls on pheasant reproduction, behavior, and survival. J Wildl Mngmt 35:315-319.

Davis TS,Pyle JL, Skillings JH, Danielson ND. 1981. Uptake of polychlorobiphenyls present in trace amounts from dried municipal sewage sludge through an old field ecosystem. Bull Environ Contam Toxicol 27:689-694.

DeFoe DL, Veith GD, Carlson RW. 1978. Effects of Aroclor 1248 and 1260 on the fathead minnow (Pimephales promelas). J Fish Res Board Can 35:997-1002.

Dill DC, Mayes MA, Mendoza CG, Boggs GU, Emmitte JA. 1982. Comparison of the toxicities of biphenyl, monochlorobiphenyl, and 2,2',4,4'-tetrachlorobiphenyl to fish and daphnids. In: Aquatic toxicology and hazard assessment: fifth conference. Pearson JG, Foster RB, Bishop WE, eds. Philadelphia, PA: American Society for Testing and Materials. ASTM STP 766.

Dive D, Erb F, Leclerc H, Priem M, Colein M. 1976. Toxicity and bioacumulation of polychlorinated bihphenyl isomers in the ciliated protozoa Colpidium campylum (Stokes). Eur J Toxicol 9:105-111. (in French; English trans.)

Freeman HC, Sangalang GB. 1977. A study of the effects of methyl mercury, cadmium, arsenic, selenium, and a PCB (Aroclor 1254) on adrenal and testicular steroidogenesis in vitro, by the gray seal Halichoerus grypus Arch Environm Contam Toxicol 5:369-383.

Gilbertson M, Hale R. 1974. Characteristics of the breeding failure of a colony of Herring Gulls on Lake Ontario. Canadian Field Naturalist 88:356-358.

Glooschenko V, Glooschenko W. 1975. Effect of polychlorinated biphenyl compounds on growth of Great Lakes phytoplankton. Can J Bot 53:653-659.

Goldstein JA, Hickman P, Bergman H, McKinney J, Walker MP. 1977. Separation of pure polychlorinated bipheny isomers into two types of inducers on the basis of induction of cytochrome P-450 or P-448. Chem Biol Interact 17:69-87.

Heinz GH, Katsma DE, Swineford DM. Unpublished manuscript. Significance of high PCB residues in birds from the Sheboygan River, Wisconsin, 1976-80. Laurel, Maryland: Patuxent Wildlife Res Center, US Fish and Wildlife Service.

Helle E, Olsson M, Jensen S. 1976. PCB levels correlated with pathological changes in seal uteri. Ambio 5:261-263.

Henny CJ, Blus LJ, Gregory SV, Stafford CJ. 1981. PCBs and organochlorine pesticides in wild mink and river otters from Oregon, In: Worldwide Furbearer Conference Proceedings. Chapman JA, Pursely D, eds. Frostburg, Maryland: Worldwide Furbearer Conference, Inc.

Hermens J. 1982. QSAR's in aquatic toxicology studies. Fourth European Symposium on Chemical Structure - Biological Activity Quantitive Approaches, Bath, England, September 6 - 9, 1982. Abstract.

Jensen S., Kihlstrom JE, Olsson M, Lundberg C, Orberg J. 1977. Effects of PCB and DDT on mink (Mustela vision during the reproductive season. Ambio 6(4):239.

Keil JE, Priester LE, Sandifer SH. 1971. Polychlorinated biphenyl (Aroclor 1242): effects of uptake on growth, nucleic acids and chlorophyll of a marine diatom. Bull Environm Contam Toxicol 6:156-159.

Konemann H. 1981. Quantitative structure-activity relationships in fish toxicity studies. Part 1: relationships for 50 industrial pollutants. Toxicology 19:209-221.

Kricher JC, Bayer CL. 1977. Depression of primary productivity of Aroclor 1232 in an interspecific lentic algal assembledge. Bull Environ Contam Toxicol 18:14-17.

Lincer JL, Peakall DB. 1973. PCB pharmacodynamics in the ring-dove and early gas chromatographic peak diminution, Environ Pollut 4:59-68.

Mauck WL, Mehrle PM, Mayer FL. 1978. Effects of the polychlorinated biphenyl Aroclor 1254 on growth, survival, and bone development to brook trout (Salvelinus fontinalis). J Fish Res Board Can 35:1084-1088.

Mayer FL, Mehrle PM, Sanders HO. 1977. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. Arch Environm Contam Toxicol 5: 501-511.

Moore Jr SA, Harriss RC. 1972. Effects of polychlorinated biphenyl on marine phytoplankton communities. Nature 240:356-358.

National Research Council. 1979. Polychlorinated biphenyls. Washington, DC: National Academy of Sciences.

Nebecker AV, Puglisi FA. 1974. Effect of polychlorinated biphenyls (PCBs) on survival and reproduction of Daphnia, Gammarus, and Tanytarsus. Trans Amer Fish Soc 103:722-728.

Nebecker AV, Puglisi FA, DeFoe DL. 1974. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. Trans Amer Fish Soc 103:562-568.

Nowicki HG, Norman AW. 1972. Enhanced hepatic metabolism of testosterone, 4-androstene-3,17-dione, and estradiol-17B in chickens pretreated with DDT or PCB. Steroids 19:85-99.

O'Conners AJ, Mahanty HK. 1979. Growth responses of unicellular algae to polychlorinated biphenyls: new evidence for photosynthetic inhibition. Mauri Ora 7:3-17.

O'Conners HB Jr, Wurster CF, Powers DC, Biggs DC, Rowland RG. 1978. Polychlorinated biphenyls may alter marine trophic pathways by reducing phytoplankton size and production. Science 201:733-739.

Peakall DB, Lincer JL, Bloom SE. 1972. Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves. Environ Health Perspectives 1:103-104.

Peterson RE, Guiney PD. 1971. Disposition of polychlorinated biphenyls in fish. IN: Pesticides and xenobiotic metabolism in aquatic organisms. American Chemical Society. ACS Symp Ser 99:21-36.

Pizza JC, O'Connor JM. 1983. PCB dynamics in Hudson River striped bass. II. Accumulation from dietary sources. Aquatic Toxicology 3:313-327.

Poland A, Glover E. 1977. Chlorinated biphenyl induction of aryl hydrocarbon hydroxylase activity: a study of the structure-activity relationship. Mol Pharmacol 13:924-938.

Reijnders PJH. 1980. Organochlorine and heavy metal residues in harbour seals from the Wadden Sea and their possible effects on reproduction. Netherlands J Sea Res 14(1):30-65.

Risebrough RW, Rieche P, Peakall DB, Herman SG, Kirven MN. 1968. Polychlorinated biphenyls in the global ecosystem. Nature 220:1098-1101.

Rubinstein NI, Lores E, Gregory NR. 1983. Accumulation of PCBs, mercury and cadmium by Nereis virens, Mercenaria mercenaria and Palaemonetes pugio from contaminated harbor sediments. Aquatic Toxicology 3:249-260.

Sangalang GB, Freeman HC, Crowell R. 1981. Testicular abnormalities in cod (Gadus morhua) fed Arochlor 1254. Arch Environ Contam Toxicol 10:617-626.

Schimmel SC, Hansen DJ, J Forester. 1974. Effects of Aroclor 1254 on laboratory-reared embryos and fry of sheepshead minnows (Cyprinodon variegatus). Trans Amer Fish Soc 103:582-586.

Schmitt CJ, Ludke JL, Walsh DF. 1981. Organochlorine residues in fish: national pesticide monitoring program, 1970-74. Pest Monit J 14:136-206.

Seelye JG, Mac MJ. 1981. Size-specific mortality in fry of lake trout (Salvelinus namaycush) from Lake Michigan. Bull Environ Contam Toxicol 27:376-379.

Shaw GR, Connell DW. 1980a. Polychlorinated biphenyls in the Brisbane River Estuary, Australia. Mar Poll Bull 11:356-358.

Shaw GR, Connell DW. 1980b. Relationships between steric factors and bioconcentration of polychlorinated biphenyls (PCB's) by the sea mullet (Mugil cephalus Linnaeus). Chemosphere 9:731-743.

Skea JC, Simonin HA, Dean HJ, Colquhoun JR, Spagnoli JJ, Veith GD. 1979. Bioaccumulation of Aroclor 1016 in Hudson River Fish. Bull Environm Contam Toxicol 22:332-336.

Spigarelli SA, Thommes MM, Prepejchal W. 1983. Thermal and metabolic factors affecting PCB uptake by adult brown trout. Environ Sci Technol 17:88-94.

Stalling DL, Huckins JN, Petty JD, Johnson JL, Sanders HO. 1979. An expanded approach to the study and measurement of PCBs and selected planar halogenated aromatic environmental pollutants. Ann NY Acad Sci 320:48-59.

Sugiura K, Ito N, Matsumoto N, Mihara Y, Murata K, Tsukakoshi Y, Goto M. 1978. Accumulation of polychlorinated biphenyls and polybromonated biphenyls in fish: limitations of correlation between partition coefficients and accumulation factors. Chemosphere 9:731-736.

Swain WR. 1980. An ecosystem approach to the toxicology of residue forming xenobiotic organic substances in the Great Lakes. A manuscript invited by the Environmental Studies Board of the National Research Council, National Academy of Science. Grosse Ile, Michigan: Large Lakes Research Station, US Environmental Protection Agency.

Swain WR. 1982. What is the scientific basis for the present concern in the Great Lakes? Presented before the International Symposium on PCBs in the Great Lakes. March 15-17, 1982 at Michigan State University, East Lansing, Michigan.

Thomann RV. 1978. A size dependent model of hazardous substances in aquatic food chain. Duluth, Minnesota: Environmental Research Laboratory, US Environmental Protection Agency. EPA-600/3-78-036. Available from NTIS, Springfield, VA. PB 281-009.

United States Environmental Protection Agency (USEPA). 1977. Toxic Pollutant Effluent Standards: Standards for polychlorinated biphenyls (PCBs); final decision. Federal Register 42:6532-6555.

United States Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for polychlorinated biphenyls. Washington, DC: Office of Water Regulations and Standards, USEPA. EPA 440/5-80-068. Available from NTIS, Springfield, VA. PB 81-117798.

Versar Inc. 1983. Exposure assessment for incidentally produced polychlorinated biphenyls (PCBs), Vol II. Preliminary draft report. Task 21. Washington, DC: Exposure Evaluation Division, Office of Toxic Substances, US Environmental Protection Agency. Contract 68-01-6271.

Von Westerhagen H, Rosenthal H, Dethlefsen V, Ernst W, Harms U, Hansen P-D. 1981. Bioaccumulating substances and reproductive success in baltic flounder Platichthys flesus. Aquat Toxicol 1:85-99.

Vreeland V. 1974. Uptake of chlorobiphenyls by oysters. Environ Pollut 6:135-140.

Wasik SP, Tewari YB, Miller MM, Martire DE, Ghodbane S. 1982. Final report on project "Water solubility and octanol/water partition coefficient of polychlorinated biphenyls and other selected substances - Task 1C." Draft final report. National Bureau of Standards. Washington, DC: Office of Pesticides and Toxic Substances, U.S. Environmental Protection Agency. Interagency Agreement EPA-80-D-X0958.

Weininger D. 1978. Accumulation of PCBs by lake trout in Lake Michigan. Ph.D. dissertation, Madison, WI: Univ of Wisconsin.

Zimmerman N. 1982. Polychlorinated biphenyls in Great Lakes fish. Lansing, Michigan: Toxic Substance Control Commission, 815 Washington Square Building 48909.