Aroclor FF

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Polychlorinated Biphenyls

Another Long-Life Widespread Chemical in the Environment

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The recent finding that pelagic birds dying on the coasts of Great Britian had polychlorinated biphenyls (PCBs) in their livers in concentrations of several hundred parts per million (Bourne and Mead, 1969) shows that these compounds are present in the ecosystem in large amounts. Thus, it seems worthwhile to summarize and evaluate our current knowledge of these compounds.

Structural and Physical Properties

The picture is complicated by the fact that we are not dealing with a single compound. The basic structure of PCBs is shown in Figure 1. Any of the positions marked with an *x* can be substituted by chlorine. Widmark (1968) has calculated that of the 210 possible combinations 102 are probable. His criteria for these limitations are those compounds containing five to eight chlorine atoms per molecule and the number of chlorine atoms per ring differing by not more than one.

The commercially available Aroclors (Monsanto Company Trademark) are designated by numbers (Monsanto Technical Bulletins). The first two digits represent the molecular type: 12 - chlorinated biphenyls; 25 and 44 - blends of chlorinated biphenyls and chlorinated terphenyls (75% biphenyl and 60% biphenyl, respectively); 54 - chlorinated terphenyls. The last two digits give the weight per cent of chlorine. Thus, Aroclor 1242 is a chlorinated biphenyl containing 42% chlorine. The biphenyls commercially available from Monsanto range from 21% to 68% chlorine. Mass spectrographic studies of Aroclor 1260 (Koeman et al., 1969a) show the presence of 11 isomers: five containing six chlorine atoms, five containing seven chlorine atoms, and one containing eight. Bagley et al. (1970), studying Aroclor 1254, found 18 compounds: one containing three chlorine atoms, four containing four chlorines, four containing five chlorines, five containing six chlorines, and four containing seven chlorines. Thus, the number of compounds present is, fortunately, much smaller than is theoretically possible.

PCBs are chemically inert, are not hydrolyzed by water, and resist alkalies, acids, and corrosive chemicals. They have low volatility, their boiling points ranging from 278 C for Aroclor 1221 to 415 C for Aroclor 1268 (Penning, 1930). All are stable to prolonged heating at 150 C, and the lower Aroclors can be distilled at atmospheric pressure without appreciable decomposition. PCBs are described as insoluble in water and very soluble in hydrocarbon solvents, although no exact figures appear to be available. Nothing is known about the biological decomposition of PCBs, but it is likely that they are more stable than DDT and its metabolites since they lack the ethane component between the aromatic rings, which is the site of action of most of the transformations of DDT. Thus, PCBs have the necessary physical and chemical characteristics for persistence and accumulation up the food chain.

Use of PCBs

Polychlorinated biphenyls were first described in the literature in 1881 (Schmidt and Shultz, 1881), and the successful commercial production was achieved by the Swann Company in 1930. In that year the physical characteristics and commerical possibilities of these materials was described by Penning (1930). PCBs are now manufactured by Monsanto in the United States (Trade name Aroclor), Prodelec in France (Phenochlor), and Bayer in Germany (Colphen). Other manufacturers are located in Japan and the Soviet Union. No figures of the amount of these materials produced annually are available, but to judge from the uses listed in Table I they may be very large. The uses listed in the table are only suggested applications taken from Technical Bulletin 306 of the Monsanto Chemical Company, and again there is no information on the extent to which these recommendations are acted upon by the manufacturers of plastics and resins.

The use of highly chlorinated Aroclors for extending the kill-life of formulations containing chlordane, aldrin, and dieldrin are mentioned under miscellaneous applications of Aroclors. This possibility has been considered in a number of papers. Sullivan and Hornstein (1953), Hornstein and Sullivan (1953), and Tsao et al. (1953) found that the chlorinated terphenyl Aroclor 5460 increased the residual persistence of lindane. Tsao et al. (1953) considered that there was some indication that chlorinated polyphenyl may have a synergistic effect with lindane. Duda (1957)

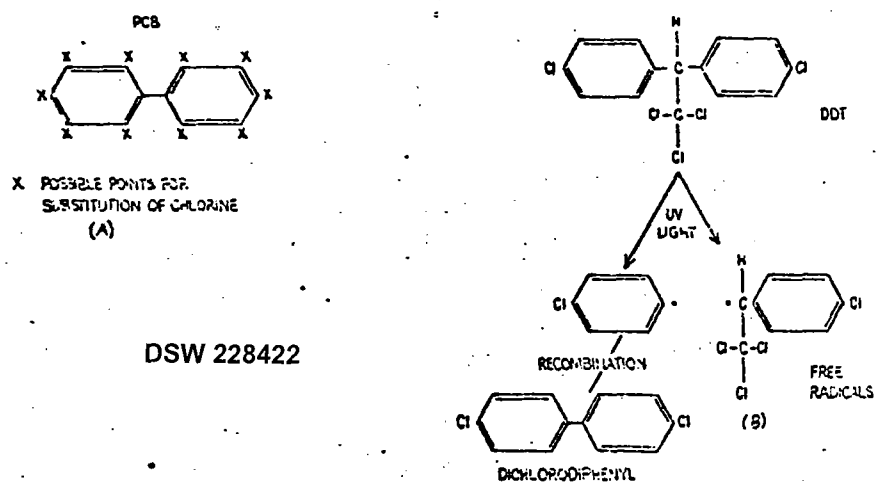

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Fig. 1. Basic structure of PCBs (A) and possible reactions of PCBs (B).

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sprayed elm saplings with a lindane Aroclor 5460 mixture (1:4) and then tested the residual effect on the leaves to infestation with elm leaf beetle (*Galerucella xanthomelaena*). He found that leaves treated with lindane plus Aroclor were protected longer and that the insecticidal effect was more rapid than with lindane alone. Peaks corresponding to Aroclor 5460 have not been detected in the environment. However, under conditions of temperature and flow-rate commonly used for gas chromatographic analysis these compounds would not be detected (Reynolds, 1970).

Nearly 25 years ago it was noted that Aroclor 1242 was one of 175 compounds out of 6000 tested that was effective against mosquito (*Aegyps*) larvae (Deonier et al., 1946). Lichtenstein et al. (1969) tested the effect of adding a variety of biphenyls and terphenyls to DDT and dieldrin with respect to their toxicity to house and fruit flies. It was found that PCBs had very low toxicity to house flies when given alone but PCBs increased the toxicity of dieldrin and DDT, especially the latter. The effectiveness of these compounds decreased as the chlorinated level increased. For example, Aroclor 1221 increased the mortality of fruit flies from 59% to 93%, whereas Aroclor 1268 increased it to only 77%.

Analytical Methods

1) *Identification.* Since various compounds with electron-capturing properties have been tentatively identified in atmospheric samples in the past (Abbott et al., 1966), and some pesticides are capable of hybridizing in the soil to form a new compound (Bartha, 1969), it is important that the presence of PCBs in field samples be proven beyond doubt. Identification by means of a combination of high resolution gas chromatography and mass spectrometry has been carried out by three independent laboratories in Sweden (Widmark, 1967), Holland (Koeman et al., 1969a), and the United States (Bagley et al., 1970). Widmark's report states that all peaks were identified by mass spectrometry (although no data were given), but Koeman and co-workers gave full experimental details, and Bagley et al. (1970) demonstrated that most chemicals in the eagle samples examined were components of Aroclor 1254. Thus, despite a recent statement by the Monsanto Chemical Company (in Risebrough, 1970) that the case for labeling the peaks in question as PCBs was not proven, there is enough evidence to convince an unbiased scientific jury.

2) *Separation.* The chemical techniques preliminary to quantitation of residues in samples containing both PCBs and chlorinated hydrocarbon pesticides fall into two groups: those necessitating the destruction or alteration of one or more of the compounds, and those which do not.

Included in the first group is nitration. Treatment with a 1:1 mixture of sulfuric acid nitric acid at 0 C for 5 min destroys or alters aldrin, p,p'-DDE, p,p'-DDD (TDE), p,p'-DDT, and dieldrin such that they can no longer be detected at the original position on the chromatogram. This procedure leaves unaffected PCB, lindane, and BHC (Jensen and Widmark, 1967). A more rigorous nitration with a 1:1 mixture of sulfuric acid-fuming nitric acid for 15 min at room temperature removes, in addition to DDT and its related products, aldrin, heptachlor, Kelthane, Perthane, Tedion, Telodrin, and Trithion while lindane, heptachlor epoxide, toxaphene, and Strobane are not removed (Erro et al., 1967). Risebrough et al. (1969) reported that this nitration also removed the chromatographic peaks of PCBs. Reynolds (1969) reported that his attempts to nitrate samples were not fully successful in that there appeared to be loss of some of the more volatile PCBs while peaks with longer retention times appeared. Armour and Burke (1969) reported that complex chromatograms resulted after nitration which could not be related to the unreacted DDT-PCB mixture, and nitration was not pursued as a practical means of separating DDT and PCB for further tests.

Saponification with alcoholic NaOH or KOH will dehyrodrochlorinate Perthane, Toxaphene, DDD, and DDT to their respective olefins (Archer and Crosby, 1966; Klein and Watts, 1964). Risebrough et al. (1969) reported that PCB peaks are not removed or displaced but gave no data.

The second, and in some instances more desirable, group of analytical techniques allows the special separation of many chlorinated hydrocarbon pesticides from PCBs. Reynolds (1969, 1970) reported on an activated Florisil column technique which separated heptachlor, aldrin, DDE, and PCB with the first elution (60 ml n-hexane) from lindane; heptachlor epoxide, DDD, and DDT with the second elution (40 ml 50% ethyl ether in hexane). Armour and Burke (1970) developed a method utilizing a silicic acid-Celite column eluting aldrin and PCB with the first fraction (250 ml petroleum ether), and lindane, heptachlor, heptachlor epoxide, dieldrin, endrin, p,p'-DDE, o,p'-DDT, p,p'-DDT, and p,p'-DDD with the second fraction (200 ml acetonitrile hexane methylene chloride—1:19:30). Koeman et al. (1969a), using an activated Florisil column, eluted the apolar compounds including DDE and PCB with hexane, and then dieldrin and endrin with 10% diethyl ether in hexane. Mulhern (1968) reported on a method which utilized silica gel-coated thin-layer plates

TABLE I

Some uses of polychlorinated biphenyls taken from Monsanto Technical Bulletin 0 PL-225

Material with which Aroclor is combined	Aroclor used with at %	Use
Polyvinyl chloride	Aroclor 1248, 1254 & 1260 (7-8%)	Secondary plasticizer to improve flame retardance and chemical resistance.
Nitrocellulose lacquers	Aroclor 1262 (7%)	Co-plasticizer to enhance resistance.
Polyvinyl acetate	Aroclor 1221, 1232, 1242 (11%)	Improve quick tack and fiber-tear properties
Ethylene vinyl acetate	Aroclor 1254 (31%)	Pressure sensitive adhesive
Epoxy resins	Aroclor 1221 & 1248 (20%)	Increase chemical and moisture resistance and adhesive qualities.
Polyester resins	Aroclor 1260 (10-15%) Aroclor 1268 (10-20%)	Effective and economical fire retardant. Increases strength of fiberglass reinforced polyester resins.
Polystyrene	Aroclor 1221 (2%)	Plasticizer.
Chlorinated rubber	Aroclor 1254 (5-10%)	Enhances resistance, flame retardance, and improves electrical insulating properties.
Styrene butadiene co-polymer	Aroclor 1254 (3%)	Improves chemical resistance.
Neoprene	Aroclor 1268 (10%) Aroclor 1268 (1.5%)	Fire retardant Injection moldings
Crepe Rubber	Aroclor 1262 (5-50%)	Plasticizer in paint compositions.
Varnish	Aroclor 1268 (25% of oil)	Improves water and alkali resistance
Wax	Aroclor 1242 (5%)	Maintains oil flame resistance

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and a hexane/ethyl ether (98.2) solvent system. The plates were developed, sprayed with a silver nitrate solution, and exposed to UV light. The plates were then divided into five horizontal sections. Dieldrin, endrin, γ-BHC, heptachlor epoxide, p,p'-DDD, p,p'-DDT, o,p'-DDT, and p,p'-DDE (in that order) were found in the first four fractions, while most of the interfering compounds found in wildlife samples were found in the fifth zone. Bagley et al. (1970) used this method but mentioned that zones three and four contained practically all unknown components as well as p,p'-DDT (zone three) and p,p'-DDE (zone four). Armour and Burke (1969) used precoated (aluminum oxide) sheets and n-heptane and 2% acetone/n-heptane for solvent systems. PCBs (Aroclors 1254 and 1260) and DDE were not separated, but p,p'-DDT and p,p'-DDD were completely separated from PCB by both solvent systems. Another possible means of separating interfering substances is to use a series of differing polarity columns in the gas chromatograph at the time of determination. This is less time consuming and may be useful when operating conditions can be selected such that PCB peaks are absent in the region where sought pesticides emerge (Simmons and Tatton, 1967).

3) *Quantitation.* Koeman et al. (1969a) semiquantitatively measured the residues in Japanese quail fed phenochlor DP6 by using one of the peaks in a phenochlor DP6 mixture as a standard. Risebrough (1969) quantitated relative levels of PCBs by assuming that each PCB compound produced the same peak height with the electron capture detector as the same amount by weight of p,p'-DDE. After summing the heights of the individual peaks, the total was multiplied by a factor derived from measurements of standard solutions with electron capture and microcoulometric detectors. Jensen et al. (1969) reported PCB amounts as the sum of all PCB components and based the estimate on a combination of mass spectrometry and microcoulometric and electron capture detection. Even with this elaborate approach, these investigators suggest that the method is still rough and may be correct only within a factor of 2. Anderson et al. (1969) devised a method for obtaining a crude estimate of PCB residues as Aroclor 1254 from chromatograms where separation had not been attempted. On an empirical basis, it was found that Aroclor 1254 could be quantitated by considering peak 10 as p,p'-DDT and multiplying that value by 10. Since this peak had originally been quantitated as p,p'-DDT, and in fact many of the original samples contained little or no p,p'DDT, it was possible to estimate relative PCB values. Anderson and his co-workers also saponified samples to remove interfering p,p'-DDT and p,p'-TDE and then quantitated PCBs as Aroclor 1254 by relating sample peaks 9 and 10 to the corresponding peaks of an Aroclor 1254 standard. Reynolds (1970) employed a method similar to Koeman et al. (1969a) but based his quantitation on an average of two or more peaks. In addition, his results were reported as Aroclor 1254 or 1260 depending on the overall pattern of the chromatographic peak profile. It is clear that we are still relatively unsophisticated in our PCB quantitation methodology and will continue to estimate only relative amounts of PCBs in field samples until we synthesize the individual PCB components commonly found in the ecosystem and are able to speak in terms of these individual peaks as we do for most pesticide residues. However, it has been kindly pointed out (Risebrough, pers. comm.) that, with reference to biological significance, the correct order of magnitude and accurate relative amounts of PCB give the essential information. This is because biological effects, such as enzyme induction, are related to degree of chlorination, and the existing methods give some indication of this activity. Therefore, that information would be lost if stress were placed only upon quantitating individual peaks.

4) *Magnitude of Error.* Ever since PCB peaks were recognized for what they are, residue chemists and other researchers interested in pesticide residues have asked what magnitude of error is likely to result from ignoring the presence of PCBs. Only recently have studies either directly or indirectly resulted in an estimate of this error. Anderson et al. (1969) quantitated p,p'-DDE, p,p'-DDD, and p,p'-DDT in five egg samples before and after saponification. There was no appreciable change in the p,p'-DDE, but apparent p,p'-DDD was reduced by approximately 58% and p,p'-DDT by 90%. Reynolds (1970) looked at a large number of samples before and after his PCB-Florisil separation and found that the actual p,p'-DDD residue (relative to the apparent residue) represented from 0 to 7% in California gull (*Larus occidentalis*) fat, 0% in cormorant (*Phalacrocorax auritus*) eggs, 89% in 10 pooled mallard (*Anas platyrhynchos*) duck eggs, and from 0 to 104% in great blue heron (*Ardea cinerea*) eggs. Respective values for p,p'-DDT were 0 to 53%, 13 to 41%, 100%, and 18 to 102%. Actual residue levels of heptachlor epoxide generally represented a majority of the apparent values. Before and after values for p,p'-DDE and dieldrin were not significantly different. Obviously, the magnitude of error may vary depending on the trophic level sampled and certainly with the area from which a sample is collected (Risebrough et al., 1968, Jensen et al., 1969). Since the publishing of adequate separation techniques, there is no reason why there should be any error in pesticide quantitation contributed by PCBs.

Toxicology

Despite some recent studies, the toxicology of PCBs remains rather poorly known as compared to that of the chlorinated hydrocarbon pesticides. For example, no definite work has been done to establish LD50 values for the various formulations of PCBs.

1) *Acute, single dose experiments.* Tucker and Crabtree (1970) found that a single dose of 100 mg/kg Aroclor 1254 (stomach tubed in oil) was fatal to two out of three rats, while 500 mg/kg was not fatal to three rats. Aroclor 1268 killed one rat out of three at 500 mg/kg, 1000 mg/kg, 2000 mg/kg, and 4000 mg/kg. Smyth (1931) found that a 4 g/kg (degree of chlorination not stated) was nontoxic to guinea pigs and rabbits, but this appears to be due to the fact that material which was given as a paste passed through the intestine unabsorbed. Tucker and Crabtree (1970) found that Aroclor 1242, 1254, 1260, and 1268 at a dose of 2000 mg/kg was not fatal to mallard ducks (four groups of three birds each).

2) *Acute, feeding experiments.* Monsanto Bulletin 306 states that 100 ppm diet had no effect on rats, although no details of the studies were given. The experiments of Bennett et al. (1938) in which 0.05 g/rat of 65% chlorine biphenyl was given orally every other day led to 50% mortality. If one assumes a body weight of 200 g, then the alternate day dose is roughly 250 mg/kg, which can be compared to oral LD50 for DDT of 113 mg/kg (Frear, 1968). Miller (1944) found that two oral doses of 69 mg of 42% chlorine biphenyl a week apart were fatal to guinea pigs. At an estimated body weight of 400 g, this gives a dose of 170 mg/kg. Tucker and Crabtree (1970) fed rats diets containing 10 and 1000 ppm Aroclor 1254. One rat out of six on the low dose died; this mortality was considered to be due to other causes. Four out of four of the high group died within 53 days and the calculated intake was 1330-1520 mg/kg. The

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food intake of the 1000 ppm group was only 79% of the control group.

Presst et al. (1970) have examined the toxicity of Aroclor 1254 to Bengalese finches (*Lonchura striata*). This is a difficult species for which to calculate the dietary intake. Due to their dependence upon unshelled food, it is only possible to present the PCB-laden food for a few hours a day (Jefferies, 1967). Loss by evaporation and loss by spillage must be allowed for, and increase of weight by defecation must be kept to a minimum. Thus, the calculated dietary intake is subject to more error than is usually the case. Presst et al. (1970) also measured the concentration of PCBs in the liver; they found that the range was large (i.e., 70-697 ppm in birds that died compared to 3-634 ppm in those that survived). These authors conclude that Aroclor 1254 has only 1/13 the toxicity of DDT, although the different shape of the mortality curves--steep with DDT, gradual with PCB—makes comparison difficult. Jefferies and Walker (1966) found good correlation between calculated dietary intake and liver concentrations for pp'-DDT in the Bengalese finch. However, other workers (Dale et al., 1963; Stickel et al., 1966; Stickel and Stickel, 1969) have considered that levels in the brain are a more reliable index of toxic levels than those in the liver or whole carcass.

De Vos and Koeman (1970) fed Phenoclor DP6, Clophen A60, and Aroclor 1260 to chickens at a dosage of 400 ppm. Mortality was complete (20/20) for those birds on Phenoclor in 12-58 days and in 13-29 days for Clophen. The mortality for Aroclor was only 3/20 for a 60-day period. This differential effect is unexplained and is surprising in view of the fact that all three formulations contain 60% chlorine. These workers measured the residue levels in the liver, and, for some birds, in the brain, for chickens dying during the experiment. Although there was considerable variation, most of the brain levels were between 210 and 420 ppm, which can be compared to 50-80 ppm for DDT (Stickel et al., 1966). On the basis of this work, PCB is 1/4-1/5 as toxic as DDT. Little difference was noted between the three different PCB formulations, although the numbers of determinations involved was rather small. If this finding is borne out by subsequent work, it would suggest that differential absorption from the gut or differential penetration of the blood brain barrier is involved. The liver values were more variable. There was a considerable number in the 200-400 ppm range, but there were several values in excess of 2000 ppm. This finding is in agreement with the findings of Dale, Stickel, and co-workers that the brain levels are the best indication of acute toxic levels.

McCune et al. (1962) found no mortality with chickens fed 100 or 200 ppm Aroclor 1242 in their diet for a 4-week period. On diets of 400 ppm and 800 ppm, the mortalities over a 4-week period were, respectively, 50% and 90%. During the first 3 weeks, the mortality figures were 10% and 50%, respectively. Flick et al. (1965), using the same material at 400 ppm in the diet, had three birds out of 24 die in a 3-week period. Koeman et al. (1969a) found that a diet containing 2000 ppm Phenoclor DP6 caused complete mortality with Japanese quail (5/5) in 5-13 days and rats (8/8) in 1-56 days.

Schoettger (unpublished) found that the 96-hr TLm for Aroclor 1221 using cutthroat trout was 1.2 mg/l and for Aroclor 1260 was 6.9 mg/l. In general, they found that the toxicity of Aroclors was inversely proportional to their percentage chlorination and directly proportional to their solubilities. These figures suggest that PCBs are two to three orders of magnitude less toxic to fish than DDT. Work by Lichtenstein et al. (1969) on house and fruit flies found that PCBs were 40-300 times less toxic than DDT, the toxicity decreasing as the chlorine content increases. Thus, it appears that the lower vertebrates and invertebrates are much less susceptible than mammals to direct toxicity from PCBs.

3) *Sublethal effects.* As with the chlorinated hydrocarbon pesticides, the most important effects are long-range sublethal effects. The pathologic changes in various organs are summarized in Table 2. The table shows some interesting differences between mammals and birds. The most striking finds in mammals are alterations to the liver, whereas fluid in the pericardial sac, kidney damage, and reduced spleen was found in birds.

McLaughlin et al. (1963) found that 25 mg Aroclor 1242 injected into the yolk sac of chicken eggs caused complete mortality, whereas 10 mg caused 95% failure and teratogenetic effects were noted among the young that hatched (beak deformity, edema, and growth retardant).

Induction of hepatic hydroxylating enzymes has been demonstrated in the pigeon (Risebrough et al., 1968), rat (Street et al., 1969), and American kestrel (*Falco sparverius*), (Lincer and Peakall, 1970). Street and co-workers studied the effects of a diet of 50 ppm and 100 ppm on sleeping time induced by a standard dose of hexobarbital, in vitro rates of aniline hydroxylation and demethylation of p-nitroanisole, and the rate of excretion of dieldrin. These workers studied 10 compounds ranging in chlorine content from 21% to 68% and found that all the effects increased with increasing chlorine content. For example, 50 ppm of Aroclor 1221 reduced hexobarbital sleeping time by 11%, whereas for Aroclor 1248 and 1268 the figures were, respectively, 35% and 48%. Thus, the sublethal effects have direct correlation with chlorine content, while the lethal effects appear to be inversely correlated (Tucker and Crabtree, in press). Lincer and Peakall (1970) noted an increase of the in vitro rate of metabolism of estradiol in kestrels fed 0.5 and 5 ppm Aroclor 1254 in their diet and also demonstrated increased levels of cytoplasmic RNA with the higher dietary level using a cytophotometric technique.

Tucker (unpublished) found that a single oral dose of 500 mg/kg Aroclor 1254 caused regular egg laying of Japanese quail (*Coturnix coturnix*) to turn first to scattered egg production and then to stop completely for a week. The scattered eggs had shells 9% thinner than normal, but thickness returned to control values when regular laying was resumed. Mallard ducks dosed with 1000 mg/kg of Aroclor laid one or no eggs before stopping for 1-2 weeks. The few eggs laid after the single large dose of Aroclor had shells 18% thinner, and again eggshell thickness was normal after resumption of regular laying. The period before egg laying has been noted to be increased in the ring dove (Peakall, unpublished). It is possible that the mechanism involved is increased rate of metabolism of circulating estradiol in the liver (Peakall, 1970). Anderson et al. (1969) had suggestive evidence that PCBs affected eggshell thickness, although to a less extent than DDE. Preliminary results with ring doves support this conclusion (Peakall, unpublished).

Levels of PCBs Found in Nature

Roburn (1965), comparing total chlorine (by concentration cell techniques) with results calculated from gas chromatography, found that some unknown chlorine compounds were present in several tissue samples and eggs of wild birds in Great Britain. The first identification of these materials was by Jensen and it is stated (Anonymous, 1966) that residues in feathers collected in Sweden go back to 1944. PCB residues have been reported in wildlife from Canada (Holden and Marsden, 1967; Anderson et al., 1969; Reynolds,

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TABLE 2. Pathologic changes induced by PCBs

Treatment	Animal	Liver	Kidney	Pericardium and Peritoneum	Other Observable Changes	References
Single oral dose of 69 mg (42% cl)	Guinea Pig Rat Rabbit	Small fat droplets through lobules, slight to moderate central atrophy, focal necrosis noted in a few animals.	Essentially normal	No noteworthy changes	Adrenals, spleen, and pancreas showed no noteworthy changes.	Miller (1944)
300 mg daily for 6 days (65% Cl)	Rat	Cells swollen, hyaline granules present, most died within few days.				Bennett et al. (1936)
50 mg daily for up to 6 months 65% Cl)	Rat	Enlarged (33% weight increase), large number of hyaline globules in cytoplasm. Several died during experiment.				Bennett
25, 50, & 100 ppm in diet for 15 days (21-68% Cl Aroclors)	Rat	Increase in weight, effect increasing with increasing chlorine content. Aroclor 1232-16%, 1242-12%, 1254-14%, 1268-24% at 50 ppm.				Street et al. (In press)
100 ppm in diet 200 ppm in diet 400 ppm in diet 800 ppm in diet (Aroclor 1242)	Chicken	No effect No effect Enlarged and mottled Damaged	Damaged	Slight Hydropericardium Hydropericardium Hydropericardium, hydroperitoneum, Enlarged.		McCune et al. (1952)
200 & 400 ppm in diet for 3 weeks (42%, Aroclor)	Chicken	No changes noted	Paleness at 200 ppm, extensive hemorrhage, and enlargement at 400 ppm.	Increased fluid in pericardial sac at the higher concentration.	Paleness of pancreas, enlargement of adrenal and small spleen at low concentrations. At higher concentrations pale cream-colored pancreas, adrenals hemorrhagic.	Flick et al. (1965)
Various doses (54% Cl, Aroclor)	Bengalese Finch	No weight changes	Weight was 32.4% of brain weight for controls and 53.5% for those dying from PCB poisoning.	Slight weight increase, a few showed liquid in pericardial sac.		Presst et al. (In press)
400 ppm in diet for 60 days (60% Cl*)	Chicken	Centrolobular necrosis (compd 1 and 2). Liver weight increased from 2.76 g/100 g to 4.21 g/100 g (compd. 3). Fatty degeneration.	Tubular dilatation, (compd. 1 and 2) Rare with compd. 3.	Hydropericardium common with compds. 1 and 2. Rare with compd. 3.	Increased porphyrin, spleen small with reduction of red pulp and atrophy of white pulp (compd. 1 and 2). Spleen decreased from 0.145 g/100 g to 0.136 g/100g, compd. 3).	Vos and Koeman (In press)

*Phenoclor DP 5 (compd. 1), Clophen A50 (compd. 2) and Aroclor 1260 (compd. 3) were used. Differential effects noted under compd. numbers. All chickens died on compd 1 and 2 within 50 days, only 15% mortality on compd 3.

1970), Germany (Fiuczynski and Wendland, 1968; Koeman et al., 1967), Great Britain, (Holmes et al., 1967; Presst and Jefferies, 1969; Presst et al., 1970; Holden and Marsden, 1967), Netherlands (Koeman et al., 1967; 1969), Sweden (Anonymous, 1966; Jensen et al., 1969), and the United States (Anderson et al., 1969; Risebrough et al., 1968; Risebrough, 1969)..

Biological Magnification

No detailed studies, such as those for DDD at Clear Lake (Hunt and Bischeff, 1960) and DDT and its metabolites in Lake Michigan (Hickey et al., 1966), have yet been made for PCBs.

The most detailed studies currently available are those of Jensen et al. (1969). The figures (mean, range of values, and sample size) given in the table below are taken from their paper.

PCBs (ppm in extractable fat)

	Baltic		Stockholm Archipelago	
Mussel	4.3 (1.9-8.6)	(40)	5.2 (3.4-7.0)	(15)
Herring	6.8 (0.5-23)	(18)	5.1 (3.3-8.5)	(4)
Seal	34 (16-44)	(3)	30 (16-56)	(3)
Guillemot eggs	250 (140-350)	(9)	—	
White-tailed Eagle				
Pectoral muscle	—		14,000 (3400-17,000)	(4)
Brain	—		910 (490-1500)	(3)
Eggs	—		540 (250-800)	(5)
Heron	—		9400	(1)

The levels in three species of fish in Clear Lake in 1968 were 0.03-0.005 ppm (wet weight) compared to 0.098 ppm in the breast muscle of a western grebe (Risebrough et al., 1969). Anderson et al. (1969) found that in most fish extracts the levels of PCBs were less than 0.1 ppm, whereas the levels in the eggs of cormorants (*Phalacrocorax auritus*) were 5-9 ppm. Presst, Jefferies, and Moore (unpublished) found that the livers of fish-eating birds in the British Isles ranged up to a maximum of 900 ppm (wet weight), bird feeders up to 70 ppm, mammal eaters to 50 ppm, and insectivores to 1 ppm. Unfortunately, no average values or prey items were included.

The physical properties of PCB and the available residue data clearly indicate that these materials are capable of biological magnification up the food chain.

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Ratio of DDT to PCB

Risebrough et al. (1968) and Risebrough (1970) have examined the ratio of total DDT, i.e., DDT and its metabolites to PCB. He has found that the ratio DDT/PCB was 1-2 in San Francisco Bay and 5-10 for seabirds in the Pacific; in the Gulf of California, a region relatively remote from contamination, the ratio was 9-10. Vermeer (quoted in Reynolds, 1970) in western Canada found a DDE/PCB ratio of 7 for California gull tissues and 13 for great blue heron eggs. In the Baltic the ratio was 1-2 (Jensen et al., 1969), although along the west coast of Sweden the ratio was as low as 0.15. In grebes in the British Isles the ratio was 0.4-0.8 (Presst and Jefferies, 1969). For sea-bird eggs, Presst et al. (unpublished) found ratios of 0.06 to 0.5.

The overall impression is that the amount of PCB in tissues tends to parallel that of DDE, at least in local ecosystems, and the DDE/PCB ratio is lowest near industrial areas suggesting that PCB is not carried quite so readily to remote areas. Nevertheless, the variation of the ratio is small enough to suggest that the routes of dispersal are similar. Since the evidence points to aerial fallout as the route of dispersal of the chlorinated hydrocarbon pesticides (Risebrough et al., 1968; Frost, 1969), it is likely that this is also the main route for PCBs. The pathways by which PCBs escape into the ecosystem are poorly known, although the possibilities have been recently discussed at some length (Risebrough, 1970; Reynolds, 1970). Since a large number of plastics and resins may contain PCBs (Table 1), the most likely route is combustion of these materials. This supposition remains to be tested. The possibility that PCBs could be derived from DDT should also be considered. The possibility that this conversion occurs in tissue is most unlikely for two reasons. First, it has never been detected despite the detailed work on the metabolism of DDT. Second, the only mechanism likely to give rise to a biphenyl is via free radicals and this is unlikely to occur in tissue. However, in the atmosphere under the influence of UV light, such a breakdown is more probable. A possible reaction is shown in Figure 1. Tautomeric shift could lead to a variety of isomers of dichlorophenyl, but it is difficult to envision the formation of more highly chlorinated biphenyls by this route. Since PCBs extracted from biological material match well with higher Aroclors (i.e., 1254), it seems unlikely that PCBs found in nature could be derived from other materials.

Significance of Current Levels

In view of the similarity of PCBs to DDT and its metabolites, the addition of PCB residues to the environment is roughly equivalent to an increase of DDE residues. However, since a synergistic effect of PCBs on DDT has been demonstrated in insects (Tsao et al., 1953), this possibility should not be overlooked in higher organisms. The enzyme induction effects of PCBs have been well documented, and carbonic anhydrase inhibition is likely. The effect of PCBs on photosynthesis is a critical experiment that has not been done. Wurster's (1968) experiments with phytoplankton should be repeated with PCBs.

The most critical area for research on PCBs is to discover the major source(s) of escape into the environment. Legislative control of a material escaping as a side effect of its use may have a different set of problems than the control of pesticides which are broadcast as a function of their normal use. The evidence suggests that it is important to find the leak and stop it.

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