PCB - Effects on Birds

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Polychlorinated biphenyls (PCB) are widely distributed in the environment and, according to many investigators, they seem to be as ubiquitous as DDT (Risebrough et al. 1968; Koeman et al. 1969; Jensen et al. 1969; Prestt 1970; Jensen 1966). Many observations concern high levels of PCBs in the avian fauna, particularly in marine birds which are at the top of a food chain in the sea. Larger residues have been found in predatory birds than in omnivorous or herbivorous species.

There is very little work published on the avian toxicity of the PCBs. No observations concern any acute effects of these compounds in the avian fauna. As regards the sublethal effects, however, the situation may be different and there are indications that the PCBs may be responsible for certain reproduction disturbances.

There are a few works on experimental toxicology. Koeman et al. (1969) fed a diet containing 2000 ppm of Phenochlor DP6, a technical PCB mixture, to Japanese quails, all of which died between day 6 and 55 after dosing had started. Gas chromatographic analysis of the residues in liver and brain of these birds indicated that some of the more lightly chlorinated PCBs present in the original mixture, had been metabolized. Autopsy of the quails demonstrated large amounts of fluid in the pericardial sac. Hydropericard has also been observed in cockerels dosed with 400 ppm of PCB (Arochlor 1242) in their diet (Flick et al. 1965) and in Bengalese finch-

es fed with a diet containing various amounts of Arochlor 54 (Prestt et al. 1970).

In the latter experiment were observed signs of acute poisoning including balance disturbance at perching, paralysis in the legs and trembling with shaking wings. At the autopsy was also observed enlarged kidneys.

Flick et al. (1965) fed Arochlor 1242 to dayold cockerels (200 or 400 ppm in the diet) and reported, besides hydropericard, growth depression, abdominal and subcutaneous edema, hemorrhagic and enlarged kidneys and adrenals, a small spleen, a cream-coloured pale pancreas, decreased hemoglobin values, dermatitis and defeathering. They also discussed the similarity between the symptoms elicited by PCBs and those of the chick edema disease. Certain differences, however, exist and the possibility that the chick edema factor (today known to be a mixture of chlorinated dioxines) might be present in PCB-preparations was not supported by gas chromatographic analyses performed by McCune et al. (1962). McCune et al. (1962) also fed a chlorinated biphenyl to chicks (100 ppm) and observed symptoms similar to those of chick edema disease (hydropericard, subcutaneous and abdominal edema, hemorrhages of internal organs and enteritis).

The toxicity of PCB as compared with other chlorinated hydrocarbons, is somewhat difficult to establish, because, whereas e.g. DDT presents a relatively steep mortality curve, the curve for a PCB is much more gradual. This means that at low dose rates, PCB may have a comparatively greater toxicity than similar amounts of DDT, but at higher dose rates the toxicity of DDT is considerably higher. In the experiments of Prestt et al. (1970) with Bengalese finches, the estimated dose rate for 50 % mortality at 56 days was 254 mg/kg/day. At low doses, PCB was more toxic than DDT; at higher doses PCB was about 10 times less toxic than DDT.

Prestt et al. (1970) also gave some values of the concentrations of PCB in some organs of the birds. One bird, killed after 8 weeks daily feeding with 367 µg of PCB, showed 14 ppm in the brain, 25 ppm in the liver, 574 ppm in fat and 61 ppm as a mean in the rest of the body. 9 % of the ingested amount remained in the body, and was stored mainly in the depot and organ fat. Some of the birds were starved prior to analysis. Starvation resulted in a redistribution of PCB with an increase of the brain concentration.

The population declines of many avian species in Europe and North America has been attributed to the use of chlorinated hydrocarbons. This decline has been traceable not to the death of adults, but to a drastic drop in reproduction. The symptoms observed in connection with this decline have included late breeding, failure to lay eggs, a remarkable thinning of the shells and much breakage of the eggs that are laid, eating of broken eggs by the parents, failure to lay more eggs after earlier clutches have been lost, reduced clutch size and a high mortality of the embryos and among fledglings. The regions of population decline coincide with areas where chlorinated hydrocarbons are widely applied.

The pato-physiological mechanisms behind these symptoms are uncertain, but may to some parts be attributed to the capacity of the chlorinated hydrocarbons to disturb the hormone balance and the function of enzymes.

It has been demonstrated in pigeons, that PCBs, like DDT and DDE, have the capacity to induce steroid hydroxylating liver enzymes to increase their breakdown of estradiol (Risebrogh et al. 1968).

The PCBs are more powerful inducers of these enzymes than DDT or DDE. The breakdown of estradiol in pigeon liver was increased 2.5 times by DDE, 3.5 times by DDT and 5.5 times by PCB, in spite of that only half as much

PCB as DDT or DDE was used (Risebrough et al. 1968).

The late breeding, the delayed egg-laying and the failure to lay again after early loss of eggs could be explained in this way because estradiol is involved in the stimulation and maintenance of the sex organs and breeding behaviour. A delayed laying may be very serious for certain species because this reduces the chances for the young to get optimum of feed and consequently reduces the optimal chance for survival. Concerning this risk it appears that the PCBs are greater threats to birds than DDT.

The failure to lay at all could also be caused by decreased hormone concentrations resulting from hepatic enzyme induction or apparent failure to lay could be caused by early breakage and eating of eggs. A reduced clutch size could also be caused by the breakage of eggs.

Since about 1947, there has been a sharp decrease in the thickness of the egg shell of certain predatory birds whose populations were decreasing (Risebrough 1968 and others). Thin eggshells were found in avian species who accumulated high concentrations of chlorinated hydrocarbons. In uncontaminated populations of these species the production of eggshells was normal. Extremely thin eggshells have been found in the brown pelican in California where the average percentage thinning was 53 % and the extreme was 95 %. In North American peregrines, contaminated with DDT as well as PCBs, a decrease in eggshell thickness of 34 % from the mean has been demonstrated. The reduction in eggshell thickness increases the chances of egg breakage. Water retention, which affects a successful hatching, might also be impaired. The PCBs cause thinning of the eggshells but this thinning seems not to be as effective as that caused by DDT or its metabolites.

The formation of thin egg-shells could be explained by two

fferent mechanisms. The dep tion of calcium in the medullary bone, a storage necessary for the egg-shell production, is under control of estradiol. A decreased concentration of this hormone caused by hepatic enzyme induction as mentioned above, might influence the thickness of the egg-shell. However, it seems unlikely that a reduction of the calcium reserve in the medullary bone, alone could be responsible for the marked thinnings often observed. The birds could compensate this lack by drawing calcium from the skeleton. Furthermore, birds on a very low calcium diet have been found tocease egg-laying rather than laying thin-shelled eggs. An acute hypocalcemia might also result in flight disturbances since the muscle- and the nervous systems would be affected.

Another mechanism therefore seems to play the major role in the thinning of the shells. Carbonic anhydrase is necessary to supply the carbonate ions required for calcium deposition in the shell. Several investigations have shown a lower concentration of carbonic anhydrase in shell glands producing soft-shelled eggs or no eggs than in glands producing normal eggs. Inhibition of this enzyme by DDT and DDE has been demonstrated and the close similarity between these compounds suggests that PCB might have a similar effect. It's interesting to note that no threshold value has been found for the effect of DDE on egg-shell thickness. Very small doses will cause some thinning of the shell.

Eggs from several species of birds have been demonstrated to contain large amounts of PCB (Risebrough 1968; 1970). This might contribute to explain the decreased hatchability. Chick embryo experiments have also shown that injection of small amounts of PCB in the eggs will cause a high mortality. Certain signs also indicate teratogenic effects. (Mc Laughling et al. 1963).

Finally another

be mentioned. It has been demonstrated in flies that various PCBs significantly may increase the toxic effects of dieldrin and DDT (Lichtenstein et al. 1969). No information has been found on such an interaction in the tissues of higher animals. However, the potential risks should not be disregarded in view of the synchronous occurrence of these compounds in many populations.

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