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Polychlorinated Biphenyls: Their Effects on Penned Pheasants*

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Introduction

Polychlorinated biphenyls (PCBs) have been useful industrial products since the beginning of their commercial production in 1929. The first report of PCBs occurrence in wildlife was made from the work of Sören Jensen, a Swedish Chemist, in the *New Scientist* in 1966 (1). Since then PCBs have been reported to be widespread in the world's ecosystem, building up in food chains as has been reported for organochlorine insecticides. The chemical structure of PCBs and their action on organisms, although less toxic, are similar to that of DDT. The contamination of human milk, poultry feed, and all classes of wild vertebrates, has created a concern for the need to determine possible harmful effects of PCBs.

Because of the economic importance of the pheasant (*Phasianus colchicus*), it was chosen as the experimental animal using PCBs in this study. The objectives were (1) to determine the patterns of absorption, storage, and excretion for PCBs, and (2) the effects of PCBs on reproduction, behavior, and survival. Since many published reports indicated that the chromatograms of PCBs in wildlife most nearly resemble those of Aroclor 1254, this was the PCB product chosen for study.

The use of brand names in this paper does not indicate an endorsement of any product.

Materials and Methods

Aroclor 1254 (supplied by the Monsanto Co.) was mixed (w/w) in a 1:9 dilution with corn oil and weighed into No. 00 size gelatin capsules to within ±2.5 percent of the required weight. Capsules were administered into the esophagus using a glass tube. Capsules containing only corn oil were administered to control birds.

Analyses for residues were made by Dr. Yvonne A. Greichus, Experiment Station Biochemistry Department, South Dakota State University, and the Bureau of Sport Fisheries and Wildlife laboratory at the Denver Wildlife Research Center. Analytical procedures used at the South Dakota State University laboratory are described by Dahlgren et al. (2) and procedures of the Denver Laboratory by Dahlgren et al. (3).

Hens for breeding experiments were purchased in the winters of 1970 and 1971 (30 and 34 respectively) from the South Dakota Pheasant Co. of Canton, and cocks were raised from birds previously obtained from the Canton source. All birds used for breeding were about 1 year of age.

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Table 1. Reproductive statistics from control pheasants and pheasants given PCBs 1970–71.

Treatment group[a]	No. eggs per hen per day	No. eggs set in incubator	Fertile eggs		Fertile eggs hatched		Fertile eggs pipped but not hatched	
			No.	Percent	No.	Percent	No.	Percent
1970								
0–0[b]	0.621	277	101	36	74	73	6	6
0–12.5	0.459	220	128	58	87	68	29	23
0–50	0.292	136	50	37	32	64	8	16
25–0	0.335	168	91	54	73	80	9	10
25–12.5	0.362	179	71	40	50	70	18	25
25–50	0.198	98	63	64	41	65	10	16
1971								
0–0	0.337	168	82	49	65	79	6	7
0–12.5	0.436	199	128	64	64	50	20	16
0–50	0.328	148	96	65	51	53	4	4
25–0	0.465	233	170	73	116	68	8	5
25–12.5	0.385	288	153	53	109	71	15	10
25–50	0.228	95	42	44	10	24	5	12

[a] Each treatment group had five hens, except that the 25–12.5 mg group had nine hens in 1971.
[b] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

which hens had received 50 mg PCBs. In 1970, these differences tested by chi-square were not significant ($P>0.05$), while in 1971 hatchability was reduced ($P<0.01$) among hens given PCBs. Significant differences ($P<0.05$) were found in the number of eggs that were pipped but not hatched for hen groups in both 1970 and 1971. Apparently, the administration of PCBs to the hen adversely affected the viability of the embryo at hatching. McLaughlin et al. (6) injected both 10 and 25 mg of Aroclor 1242 into chicken eggs and found only 0.5 percent hatchability with growth retardation, edema, and beak deformities in embryos.

Eggshell thickness was measured only in 1970, and no significant differences ($P>0.05$) in eggshell thickness were found using analysis of variance (5). Eggshells from hatched eggs (without membranes) laid by control hens averaged 0.26±0.02 mm standard deviation, and those from all hen groups receiving PCBs averaged 0.23±0.02 mm. Eggshells from unhatched eggs averaged 0.32±0.02 mm for control hens, and 0.31±0.02 mm for hens receiving PCBs. Dahlgren and Linder (7) found the eggshell thickness of pheasants to be unaffected by weekly administration of capsules containing 6 mg dieldrin.

Heath et al. (8) fed 25 ppm of Aroclor 1254 to mallards (*Anas platyrhynchos*) through two breeding seasons. They also fed bobwhite quail (*Colinus virginianus*) 50 ppm PCBs and at a joint level of 25 ppm PCBs and 15 ppm DDE for one reproductive season. They found no effects on egg production, cracked eggs, eggshell thickness, embryonation, embryos alive at 3 weeks, normality of hatchlings, and normal hatchlings alive at 14 days.

Scott et al. (9), who fed 0, 0.5, 1.0, 10.0, and 20.0 ppm Aroclor 1248 for 8 weeks to chickens in full egg production, found no reduction in egg production on the lowest levels of PCBs after 8 weeks; however, they noted a 10 percent reduction was associated with 10 ppm, and a 13 percent reduction was associated with 20 ppm. They reported that 10 ppm Aroclor 1248 reduced hatchability of chicken eggs by 8 percent after 4 weeks and 44 percent after 8 weeks. The 20 ppm level nearly eliminated all hatchability. Most embryos died at 21 days of development; many after pipping. They also found that eggshell strength was not affected when as much as 20 ppm was fed

Effects on Behavior

Seventy-one 11-week old hens given one 210-mg

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Table 3. Effects of PCBs on hand capture of penned pheasants, 1970–71. (Numbers represent birds caught in the first half of all birds caught; numbers in parentheses represent one-half the number of that category in the pen. Chi-square was used to compare numbers actually caught with half of the numbers in each category.)

Dates of capture	No. of hatches caught	No. of pens	Parents receiving PCB			
			Both	Hens only	Cocks only	Neither
1970						
July 22	1–8	14	16(10)**	8(8.5)	7(10.5)	11[a](10.5)
July 29	1–9	15	19(11.5)**	7(10)	9(12)	6(13.5)**
Aug. 13	1–10	18	11(12.5)	12(12.5)	12(13.5)	8(13)*
Aug. 19	1–12	20	21(15.5)*	17(14)	18(17.5)	19(18)
Sept. 1	1–14	22	25(18)*	19(15.5)	22(20)	14(19.5)
Oct. 15	1–15	23	20(16)	14(16)	25(17.5)*	9(14)
Nov. 21	1–15	13	14(11.5)	14(12.5)	8(10.5)	12(11.5)
Dec. 5	1–15	13	11(11)	14(11.5)	12(10.5)	6(10)**
All 1970 catches combined			137(106)**	105(100.5)	110(112)	85(110)**
Ratio, 1970[b]			1.55	1.91	1.98	2.59
1971						
July 21	1–7	17	27(19.5)*	23(24.5)	25(21.5)	12(14.5)
Sept. 1–3	1–15	28	75(70.5)	41(37)	39(36.5)	21(22)
Sept. 21–23	1–15	28	71(70.5)	38(36)	37(36)	24(21.5)
Oct. 7–11	1–15	28	33(37)	33(35.5)	27(35)	19(20.5)
Oct. 19–22	1–15	28	34(35.5)	23(33.5)*	35(34.5)	17(21)
Nov. 2–5	1–15	28	30(30.5)	29(32.5)	23(28.5)	19(18)
All 1971 catches combined			270(264.5)	187(199)	187(192)	112(117.5)
Ratio, 1971			1.96	2.13	2.05	2.10
All 1970–71 catches			407(370.5)**	292(299.5)	300(304)	197(227.5)**
Ratio, 1970–71			1.82	2.05	2.03	2.31

[a] Totals of row numbers may exceed one-half total birds because odd numbers of birds in pens were rounded higher.
[b] Birds in pen/birds caught in first half.
* ($P<0.05$).
** ($P<0.01$).

Table 4. Weights and survival for the first 6 weeks of offspring from control pheasants and pheasants given PCBs, 1970–71.

Treatment group	Average weight at 6 weeks (g)		No. of chicks to brooder		No. of chicks alive after 6 weeks		Percent survival		
	1970	1971	1970	1971	1970	1971	1970	1971	Both years
0–0[a]	396	429	73	65	49	50	67	77	72
0–12.5	378	435	84	64	49	45	58	70	64
0–50	303	425	29	50	3	37	10	74	51
25–0	389	428	72	115	50	91	69	79	75
25–12.5	403	458	50	109	30	72	60	66	64
25–50	344	373	40	10	9	6	22	60	30

[a] The first number is the weekly PCB level in mg given to cocks; the second, that for hens.

PCBs via the egg depressed the weight of offspring, McLaughlin et al. (6) mentioned growth retardation of embryos as an effect of PCBs injected into the yolk sac of chicken eggs. We fed no pheasant chicks PCBs, but Flick et al. (10) found that 1-day-old chicks fed Aroclor 1242 at 200 and 400 ppm had depressed growth by the second week of feeding and that the growth depression was related to the level fed. Vos and Koeman (13) fed PCBs to 1-day-old cockerels and found body-weight depression from 400 ppm Aroclor 1260. Platonow and Funnell (14) found that 1-day-old cockerels fed 250 ppm Aroclor 1254 had depressed body weights between the sixth and ninth week of their feeding trial; this depression was associated with reduced feed consumption. Rehfeld (15) also found depressed weight gains in 1-day old chicks fed sublethal levels (10–50 ppm) of Aroclor 1254; chicks fed 30 and 50 ppm for 2.5 weeks and then fed a clean diet recovered from the growth depression, while chicks fed 40 and 50 ppm for 5 weeks and then fed a clean diet for 8 weeks did not show a recovery from the growth depression.

Body weights and Mortality in Birds Given PCBs

Adult hens in both the 1970 and 1971 breeding experiments were unaffected in body weight by administration of as much as 50 mg PCBs in single capsules weekly. It was characteristic, however, of birds that died on PCB treatment to stop eating and die within several days.

Hens 11-weeks old given a 210-mg capsule of PCBs in the evening ate very little the following day and appeared weak by the end of that day (3). After receiving the second capsule 24 hours subsequent to the first, birds sat with feathers fluffed; some tremoring was noticed; and no food was consumed. Birds of both sexes 6–9 months of age given 100 mg or 50 mg PCBs every 3.5 days for 5 weeks continued to eat, as did 11-week-old hens repeatedly given either 10 or 20 mg daily. Scott et al. (9) found no effect on feed consumption when laying chickens were fed up to 20 ppm Aroclor 1248, and no mortality that could be attributed to treatment. Prestt et al. (16) found no effect on weight of Bengalese finches (*Lonchura striata*) when they were fed up to 400 ppm Aroclor 1254 for 56 days.

Birds 11-weeks old given daily capsules containing 210 mg PCBs died within 1.3 and 5.9 days; the 16 designated to be analyzed upon death lived longer than the other birds in the experiment, from 2.2–5.9 days, averaging 3.8±1.0 day standard deviation (3). They lost from 15–37 percent of their initial weight before death. All control birds lived. Correlations of the initial weight, days to death, and percentage weight loss were obtained for 53 birds in this experiment. Initial weight was correlated with days to death ($r=0.699$, $P<0.01$); heaviest birds lived longest. The birds which were heaviest initially lost the greatest percentage of their weight before death ($r=0.589$, $P<0.01$). Birds that lived the longest lost the greatest percentage of their weight before death ($r=0.744$, $P<0.01$). Time of death of the 11 birds that were not given PCBs, but were starved, ranged from 2.3 days to 8.6 days, averaging 3.9±1.8 days. They lost from 27–51 percent of their weight by the time of death (3).

Of the birds given 50 or 100 mg every 3.5 days for 5 weeks, 4 of 22 died in the 50-mg group and 7 of 22 died in the 100-mg group. In addition, two birds in the 100-mg group were so weak they were near death at the conclusion of the experiment.

Mortality of birds given 10 mg began 30.0 days after the first capsule was given; the ninth bird died after 179.3 days. The other seven birds died between 50.3 and 60.6 days after initial treatment. The tenth bird of this group, still alive after 8 months of treatment, was sacrificed (3).

In the 20-mg group, the first bird died 39.6 days and the last bird 54.1 days after capsules were first given. The average number of days to death was 46.1±5.3 days (3).

Mortality was light in breeding experiments in 1970 and 1971. In 1970, only 2 hens died from among the 30 hens and 10 cocks under study: both hens had received 50 mg PCBs weekly (5). In 1971, among 34 hens and 14 cocks in the study, three hens died, two that had received 12.5 mg and one that had received 50 mg weekly.

Tucker and Crabtree (17) reported that 2000 mg/kg of either Aroclor 1242, 1254, 1260, or 1268 given to mallards caused no mortality or symptoms. Prestt et al. (16) estimated that 254 mg/kg/day given to Bengalese finches would give 50 percent mortality at 56 days. Heath et al. (8) reported that Aroclor 1254 had an LC_{50} of 1090

week, highest during the second week, and declined thereafter. The average excretion per hen via the egg was calculated to be 4.2 mg PCBs (2). Excretion of PCBs via the egg could be higher than excretion through the feces when hens are in full egg production. A single egg laid between 1 and 2 weeks after a hen was administered a single capsule containing 50 mg PCBs was shown to contain 1.5 mg of PCBs. This egg was one of two laid that week by one hen.

Heath et al. (8) reported that two mallard eggs from the second reproductive season, when birds had been on 25 ppm Aroclor 1254, had 56 ppm and 33 ppm PCBs, wet weight. Peakall (18) reported that ring dove (*Streptopelia risoria*) eggs taken from birds given 10 ppm Aroclor 1254 in the diet averaged 4.81±1.08 ppm standard error. The actual amounts in mg of PCBs in ring dove (18), mallard (8), and pheasant eggs in the present study would be far below the 10 mg injected by McLaughlin et al. (6) into chicken eggs. The 10 mg resulted in poor hatchability, edema, and beak deformation.

Scott et al. (9) found that PCBs deposited in chicken eggs were less than 0.5 ppm after 8 weeks with 0.5 and 1.0 ppm Aroclor 1248 in the diet. They found levels of over 3 ppm after 8 weeks with 10 ppm in the diet and levels of about 6–7 ppm in eggs of hens on 20 ppm. These values are much lower than those found in the present study from 3–4 weeks after administration of a single capsule of 50 mg Aroclor 1254. Their results, showing a drastic reduction in hatchability associated with 7 ppm or less residue in eggs, are not comparable to our findings with pheasants. Differences with the experimental animal or the Aroclor product used may have resulted in the gross differences in findings between our studies. Heath et al. (8) reported a nearly four-fold difference between bobwhite and Japanese quail in the LC_{50}; thus species differences may be important.

Residues in eggs of wild birds have been determined by several authors from diverse collection points. Anderson et al. (19) found PCBs in all egg pools of cormorants (*Phalacrocorax auritus*) and pelicans (*Pelecanus erythrorhynchos*); cormorants were shown to have an estimated 8 ppm in eggs and pelicans 0.6 ppm. Jensen et al. (20) reported 48 ppm in the egg of a heron (*Ardea cinerea*) collected in Sweden, and 8–21 ppm from 9 guillemot (*Uria aalge*) eggs from the Baltic Sea. Risebrough et al. (21) reported that an egg of a peregrine falcon (*Falco peregrinus*) contained 10 ppm; 8 black petrel (*Loomelania melania*) eggs had an average of 1 ppm; 5 eggs of a barn owl (*Tyto alba*) had <1 ppm; and a golden eagle egg (*Aquila chrysaetos*) had <1 ppm. Dustman et al. (22) reported median measurements of egg residues of bald eagles to be 1.65 ppm for Alaska and 9.7 for those from all other states. They further reported median egg levels of 15.9 ppm for the osprey (*Pandion haliaetus*) of Connecticut and 2.5 ppm for those in Maryland; 5–6 ppm in brown pelican (*P. occidentalis*) eggs from different areas; and 5 ppm in eggs of royal terns (*Thalasseus maximus*).

Prestt et al. (16) found residues ranging from 0–80 ppm in 363 eggs from 28 species of both land and water birds collected in Britain. Most of these had less than 10 ppm, but those which had higher levels included a single egg of the great crested grebe (*Podiceps cristatus*), 40 ppm; one egg among 101 of the heron that had 80 ppm, while the arithmetic mean was 5 ppm; one egg of a moorhen (*Gallinula chloropus*) with 15 ppm, among 13 samples that averaged 2.4 ppm; and a single egg of a great skua (*Sterocorarius skua*) that had 25 ppm.

Particularly in view of findings that about 5 ppm was associated with nearly complete negation of hatchability in the chicken (9), the above findings in some wild birds are alarming. Apparently, though, as earlier pointed out, there must be considerable differences among species in (1) the rate at which they deposit PCBs in the egg, and (2) what a particular ppm range may mean in associated deleterious effects.

Residue Levels in Tissues

We analyzed tissues from seven different groups of birds: (1) 16 birds given 210 mg PCBs daily that were designated for analysis upon death, (2) five additional birds on 210 mg daily that died, (3) nine birds that were killed at intervals for matching with the 16 designated to die, (4) pooled samples of birds killed 12 hours and 24 hours after receiving a single capsule containing 210 mg PCBs, (5) pooled samples of four birds dying and from four birds surviving capsules con

of 3–634 ppm in livers of survivors and 70–697 ppm in livers of Bengalese finches dying during treatment with Aroclor 1254, and that liver level was correlated with the amount received ($P<0.01$).

Prestt et al. (16) stated that the liver:brain residues should be compared and that brain ppm/liver ppm X 100/1 was three times higher in Bengalese finches that died during their experiment than in birds killed at the end. Data in the present study showed a complete overlap in brain ppm/liver ppm X 100/1; birds that died during testing had a range in ratios of 5.4 to 112.8, while matching birds sacrificed had 10.0 to 37.0.

Relatively little sampling of brain tissue for PCB residues has been done in wild birds. Dustman et al. (22) reported that a sick bald eagle had 230 ppm PCBs in its brain, and that PCBs may have contributed to its death. Jensen et al. (20) reported that three white-tailed eagles (*Haliaeetus albicilla*) had a brain residue range of 29–70 ppm PCBs, averaging 47 ppm; muscle residues ranged from 150–240 ppm, averaging 190 ppm. Risebrough et al. (21) found 0.04, 1.5, 21, and 34.6 ppm PCB in the brains of four peregrine falcons. Prestt et al. (16) reported liver residues ranging from 0 to around 900 ppm from a wide variety of British birds; arithmetic averages ranged from 0.5 ppm in the buzzard (*Buteo buteo*) to 98 ppm in the heron. The tissue levels reported above were below those at which mortality occurred in the present study, except that the muscle level for the white-tailed eagle was higher than those in this study associated with death in the pheasant from PCBs. However, these levels from wild birds do constitute substantial percentages of the brain and liver levels in the present study where death occurred from PCBs.

Histopathologic Effects

Dahlgren et al. (3) found that PCBs decreased weights of heart and spleen at all treatment levels ($P<0.01$). PCB treatment increased weights of kidney and liver in birds given 10- and 20-mg doses ($P<0.01$), but no effect was seen in the 210-mg group. Starved birds had smaller hearts ($P<0.05$) and livers ($P<0.01$) than controls.

Splenic atrophy, as described by Flick et al. (10) and Vos and Koeman (13) was found in all pheasants given 20 mg daily and in the 10-mg birds except for one that survived 8 months and was sacrificed (3). Splenic atrophy was characterized by almost complete absence of lymphatic nodules and an increase in the relative abundance of red pulp. In one of the birds given 20 mg, loci of necrosis were found in lymphatic nodules.

Prestt et al. (16), using the Bengalese finch, reported that kidneys were larger in birds that died from PCBs than in controls. Flick et al. (10), using chickens, mentioned both enlarged adrenals and kidneys from PCB treatment. In our study, kidneys were larger in pheasants given 10 or 20 mg daily, but neither kidney nor adrenal enlargement was visually detected during necropsy. McCune et al. (23), using Aroclor 1242 with chickens, mentioned both enlarged livers and kidneys in birds given PCBs. Platonow and Funnell (14) and Rehfeld (15), using 1-day-old chicks, reported enlarged livers with dietary intake of Aroclor 1254. Grant et al. (24) reported enlarged livers and decreased spleen size over a period of time in the rat. Flick et al. (10) and Vos and Koeman (13) reported small spleens in their studies. Although hydropericardium was found in varying degrees by many of the authors cited, it was found only rarely in the present study. Vos and Koeman (13) found that Phenoclor DP 6 and Clophen A60 caused much more liver necrosis and hydropericardium than Aroclor 1260; this was probably due to contaminants (25).

PCBs in Combination with Dieldrin

Mortality among pheasants of both sexes 6–9 months old given dieldrin, PCBs, or a combination of the two, varied with the level of chemical administered (Table 7). Among the 22 pheasants on each level, 3 died with 4 mg dieldrin per capsule, and 6 died with 8 mg dieldrin. The same proportions held true for PCBs, since 4 died with 50 mg PCBs per capsule, and 9 died with 100 mg PCBs. When 50 mg PCBs and 4 mg dieldrin were administered together, a total of 9 birds died. None of 11 control birds died during this period of time. These data suggest that effects of PCBs and dieldrin together are additive, not synergistic. Heath et al. (8) found that the joint toxicity of Aroclor 1254 and DDE given to Japanese quail was additive, and found no evidence of synergism in their joint effect.

kidneys and livers were also useful characters in attempting to diagnose PCB toxicosis.

PCBs and dieldrin were not, when combined, synergistic in their joint toxicity to pheasants.

Livers from wild pheasants collected in Pennsylvania, Indiana, and South Dakota, did not exceed 2 ppm PCBs, indicating relatively low-level contamination.

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