DDE at Low Dietary Levels Kills Captive American Kestrels

by RICHARD D. PORTER and STANLEY N. WIEMEYER
Patuxent Wildlife Research Center
Laurel, Md.

Introduction

Until low dietary levels of DDE [1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene] recently were shown to cause varying degrees of reproductive impairment in several avian species (1, 2), the effect of this chemical on birds was not fully realized (3). The few published accounts of its toxicity suggest that only high dietary levels of DDE cause avian mortality (4-6), but in these experiments DDE was administered in amounts many times greater than those to which wild birds are normally exposed. In its ability to kill, DDE is considered to be less toxic to cowbirds (Molothrus ater) (4), but more toxic to domestic pigeons (Columba livia) (5), than DDT [1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane] or DDD [1,1-dichloro-2,2-bis(p-chlorophenyl)ethane]. Because it is more stable than DDT or DDD (7) and is lost from the animal body more slowly, it accumulates in the tissues of birds in higher concentrations. In the pigeon, for example, its half-life is 250 days, compared to 28 days for DDT and 24 days for DDD (5).

Procedures

Data discussed in this paper are by-products of a larger experiment on the effects of DDE on eggshell quality, utilizing 12 pairs of kestrels which were placed on a diet (8) containing 2.8 p.p.m. p,p'-DDE on a wet weight basis (10 p.p.m., dry weight basis) on 30 March 1968 (2). Additionally, we have included data from one of two young males produced in 1968 by these 12 pairs of birds. All birds were retained on dosage until they either died or were sacrificed (see Footnotes 2 and 3, Table 1). Since birds of several age groups were utilized in this experiment, the residues of birds that died were compared with the residues of those that were sacrificed. The birds' tissues (Table 1) were analyzed for residues of p,p'-DDT, p,p'-DDD, p,p'-DDE and dieldrin [95 percent HEOD, which is 1,2,3,4,10,10-hexachloro-6, 7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo, exo-5, 8-dimethanonaphthalene] by the WARF Institute, Inc.; their digestive tracts, beaks, feathers, and distal segments of legs were discarded. The entire sample of each brain and each liver were analyzed separately. Samples were oven dried at 40° C for 96-120 h, ground with sodium sulphate, extracted with ethyl ether

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for 4 h on a Goldfish extractor. Cleanup and preliminary separation were by two elutions through a florisil column (5 percent ethyl ether in petroleum ether and 15 percent ethyl ether in petroleum ether). Analysis was by electron capture gas chromatography (Barber Colman Pesticide Analyzer Model 5360) using a 4 ft X 4 mm glass column with 5 percent DC 200 Cromoport XXX (60/80 mesh). Temperatures were: column 195° C, injector 230° C, detector 235° C. Nitrogen flow was such that p,p'-DDT retention was about 8-10 min. In some samples, DDD peaks were partially blocked by large amounts of DDE, hence values of DDD were measured from the shoulder of the DDE peak. Analyses of skins and remainders followed the same procedure, except ground samples were extracted with a mixture of ethyl and petroleum ether (70: 170 ml) for 8 h on a soxhlet: column temperature of Cromoport XXX (60/70 mesh) was 190° C; injector temperature was 240° C; 25 g subsample of each remainder was analyzed. The percent recovery of DDE in "spiked" samples of kestrel skins and carcasses was 97.5 and 105.5, respectively. Polychlorinated biphenyls (PCB's) were identified by peak matching and confirmed by dehydrochlorination with KOH.

Results

After 14 months on dosage, a 5-year-old male kestrel died in tremors on 31 May 1969, with 212.5 p.p.m. DDE (wet weight) in its brain (Table 1). Before dying it lost about 30 percent of its pre-treatment weight. A second bird, a yearling male, that had hatched from eggs of DDE-dosed parents and had been retained on dosage after hatching, was found dead on 4 July 1969, after 16 months on dosage; it contained 301.1 p.p.m. DDE (wet weight) in its brain (Table 1). At death it weighed 35 percent less than at 1 week after fledging. Brains and livers of these two males contained much more DDE than did those of sacrificed males that had been on dosage for an equivalent period (Table 1, Footnote 3) of time, even though the one bird that died, for which data are available, contained a somewhat lower body burden (cumulative micrograms of DDE for all tissues) than did the sacrificed birds (Table 1). DDE residues in their brains, in terms of DDT equivalents (4), were within the range of residues considered to be lethal, although that of the 5-year-old male fell below the lower observed limits (250 p.p.m., wet weight) in cowbirds (4). This indicates that the lower lethal limits of DDE residues in the brains of kestrels may be somewhat below that in cowbirds. Less than 1.7 p.p.m. (wet weight) DDT, DDD, and dieldrin combined (<0.8 percent of the total residues) were found in the brains of the two birds that died. PCB occurred in comparatively small amounts.

Both kestrels that died had grossly reduced pectoral muscles and badly depleted fat reserves, and though coronary fat was not visible, some visceral and subcutaneous fat was still present. DDE-killed cowbirds usually retain some fat in all fat tracts and typically lose about one-third of their body weight due to wasting

TABLE 1

DDE residues in tissues of dosed male kestrels that died compared with those in tissues of dosed male kestrels that were sacrificed[1]

Residue basis of DDE	Males that died[2] 5-year-old	Yearling	Sacrificed adult males[3] Mean	Range
		Brains		
Micrograms	558.0	656.0	36.8	10.9 - 65.1
p.p.m. wet wt.	212.5	301.1	14.9	4.47 - 26.6
p.p.m. dry wt.	984.1	1438.6	65.8	19.6 - 118.2
p.p.m. lipid wt.	3321.4	5007.6	232.9	70.8 - 436.9
		Livers[4]		
Micrograms	244.0	433.3	52.0	27.9 - 67.7
p.p.m. wet wt.	128.7	252.9	24.0	12.9 - 32.1
p.p.m. dry wt.	507.3	877.1	88.1	40.4 - 128.2
p.p.m. lipid wt.	13555.6	18839.1	1193.3	497.8 - 2227.6
		Carcasses		
Micrograms	3388.8	984.6	4053.3	2391.5 - 6162.0
p.p.m. wet wt.	78.3	24.6	70.0	44.7 - 93.1
p.p.m. dry wt.	287.2	85.7	204.2	128.3 - 248.9
p.p.m. lipid wt.	19790.0	1128.2	1914.8	739.2 - 3494.6
		Skins[5]		
Micrograms	1354.1	-	3789.3	2458.3 - 6453.0
p.p.m. dry wt.	407.9	-	601.9	381.2 - 893.4
p.p.m. lipid wt.	6327.7	-	1800.9	653.9 - 3392.8
		Whole bodies[6]		
Micrograms	5544.9	-	7931.4	6383.0 - 9832.3
p.p.m. dry wt.	343.0	-	291.7	260.3 - 337.9
p.p.m. lipid wt.	9710.9	-	1782.1	697.2 - 3068.0

[1] Dietary dosage: 2.8 p.p.m. DDE, wet weight (10 p.p.m., dry weight) beginning 30 March 1968 for adults. Yearling hatched from a dosed female's egg on 30 May 1968, and continued on dosage of parents until death.

[2] 5-year-old died 31 May 1969; yearling died 4 July 1969.

[3] Group contained four 5-year-olds, three 4-year-olds, and four 3-year-olds. Birds sacrificed in 1969: 2 April (3), 5 May (3), 16 July (1), and 17 July (4); average live weight at date of sacrifice was 16.0 percent (range 1.1 - 29.1) lower than the pretreatment weights.

[4] Portions of the livers of six birds were taken for liver enzyme studies; to determine total micrograms in these livers we used an assumed liver weight of 2.17±SE 0.11 g (average weight of the complete livers, removed from remaining males).

[5] The skin of the 5-year-old male that died contained 178.8 p.p.m. (wet weight); since skins of the remaining birds were badly dehydrated prior to weighing, p.p.m. wet weight is not given. The yearling skin was not saved.

[6] This is only a close approximation of the total body burden, since residues were not determined for the discarded body parts.

of the breast muscles (4). The sacrificed kestrels, by contrast, lost smaller proportions of their weight (Table 1, Footnote 3), were in better flesh, and had more fat in all fat tracts (including coronary fat) than did those that died of DDE-poisoning. Neither of the birds that died, when autopsied, displayed characteristics which would suggest that they died of causes other than DDE-poisoning.

With a reduction of body fat at time of death, the toxicant in the remaining fat of the two birds that died apparently became more highly concentrated, yielding concentrations in some tissues in the range of 13,000 to 19,000 p.p.m. (lipid weight basis) (Table 1). This suggests that it may be unwise to report residues solely on the basis of lipid weight. Lipid weight residues of DDT+metabolites (mostly DDE) equalling or exceeding those found in our kestrels (Table 1) have been reported in pectoral muscles of wild white-tailed eagles (Haliaeetus albicilla) that were found dead in Sweden (9).

Discussion

In recent years, several species of raptorial and fish-eating birds have undergone severe reductions in breeding populations and/or productivity which have been attributed to the chlorinated hydrocarbons (2, 10-14). These declines are characterized by reproductive failure, followed by desertion of nesting sites and ultimately by disappearance of adult birds (11). However, the rate of decline in some peregrine falcon (Falco peregrinus) (11, 12) and osprey (Pandion haliaetus) (12) populations is considered to be more rapid than that estimated to be the normal adult mortality rate (12), the increased mortality has been attributed to organochlorine poisoning (11, 12). Mortality resulting from organochlorine poisoning must also be included among the causes of death of bald eagles (Haliaeetus leucocephalus) (15, 17). Carcass tissues from a few recently sampled bald eagles contained DDE in concentrations equalling or exceeding those in our kestrels (Table 1) (15). In addition, the brain of a bald eagle found sick in the field contained 385 p.p.m. DDE (wet weight) (17), which is within the lethal range for DDE (4); however, the cause of its death is uncertain since its brain also contained 230 p.p.m. of PCB's, 6 p.p.m. of DDD, 2.2 p.p.m. of dieldrin, and 0.4 p.p.m. of heptachlor epoxide (17). Some peregrine falcons from a North American arctic population, which is apparently undergoing incipient reproductive abnormalities and reduction in number (13), carry DDE residues in some of their tissues in concentrations (16, 18) approaching or equalling those (Table 1) of our captive kestrels. DDE residues in concentrations which appear to exceed those in our kestrels are present in brain and fat (19) tissues of brown pelicans (Pelecanus occidentalis) which were collected from rapidly deteriorating California populations (14).

The tissues of a few peregrine prey species (shore birds) that were collected on the peregrines' nesting grounds in the Arctic contain concentrations of DDE (16, 20) approaching those in the diet of our captive kestrels; and the tissues in some potential peregrine prey species (21) in the United States and Great Britain, and in some potential prey species of several other species of raptorial and fish-eating birds (22), contain equal or greater concentrations of DDE than those in the diet of our captive kestrels.

Our two DDE-killed kestrels died at a time when these birds suffer a seasonal weight loss and depletion of fat reserves caused by stress involved with reproduction and molt. With loss of fat, stored toxicant is released into the blood for redeposition at other sites (23). When it is present in the brain in sufficient quantities it causes death (4, 24). Some raptorial and fish-eating birds are subjected to weight losses of considerable magnitude during their nesting activities and/or during their long distance migrations; extended periods of weight loss may be fatal for those birds whose tissues contain lethal amounts of DDE.

The propensity for small dietary doses of this chemical to accumulate in the tissues of kestrels, and this demonstration of its capability to concentrate in their brains in lethal amounts during a period of seasonal weight loss, suggest that direct mortality due to DDE poisoning should be included among the mortality factors that may have contributed to the recent declines in populations of several raptorial and piscivorous birds.

Summary

Two of 14 male American kestrels died after 14 and 16 months on a diet containing 2.8 p.p.m., wet weight, p,p'-DDE. The brains of the two birds contained DDE residues of 213 and 301 p.p.m. compared with 14.9 p.p.m. (range, 4.47-26.6 p.p.m.) (wet weights) for 11 of the adult males which were sacrificed after 12 to 16 months on dosage. Autopsies of the two birds compared with autopsies of the sacrificed birds, revealed other characteristics typical of DDE poisoning. Neither bird, when autopsied, displayed characteristics which would suggest that they died of causes other than DDE poisoning.

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19. We have compared the skins of our kestrels on a lipid weight basis with the fat samples of pelicans on a wet weight basis as given by Keith, Woods, and Hunt (14). The fat of pelicans contained DDE residues (14) on a wet weight basis of a magnitude equal to the lipid weight basis of our kestrel skins. Since lipid weight values are always higher than wet weight values for the same sample (see Table 1), the residues of DDE in pelican fat on a lipid weight basis would be higher than those in our kestrels on a similar basis. We, therefore, consider these residues to be comparable.
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