0768

Arch. Environ. Contam. Toxicol. 12, 235–240 (1983)

Archives of Environmental Contamination and Toxicology
Springer-Verlag 1983

8/20/82

Breeding Success of Barn Owls (*Tyto alba*) Fed Low Levels of DDE and Dieldrin

Vivian M. Mendenhall,[1] Erwin E. Klaas,[2] and M. Anne R. McLane

U.S. Department of the Interior, Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708

Abstract. The relative importance of two organochlorine pesticides in the recent reproductive failure of raptors was investigated. Captive barn owls were fed 3.0 ppm DDE and 0.5 ppm dieldrin; doses were given separately and in combination for two years. Breeding success was followed from the laying of eggs through natural incubation and rearing of young. DDE was associated with significant eggshell thinning, egg breakage, embryo mortality, and reduced production per pair. Dieldrin alone was associated with slight but significant eggshell thinning, but not with reduction of breeding success. Ecological implications of the results are discussed: it is suggested that DDE had a much more severe effect on reproduction in wild raptors than dieldrin, which contributed to their decline primarily through adult mortality.

Many species of raptors have been reduced over wide areas in association with contamination by organochlorine insecticides (Newton 1979; Ratcliffe 1980). The birds are beginning to increase in some regions following restrictions on the use of pesticides (Lockie *et al.* 1969; Spitzer *et al.* 1978; Ratcliffe 1980). Due to the persistence of the compounds, however, as well as their continued introduction into the environment, some raptors are probably still ingesting organochlorines at levels that can reduce productivity (Henny *et al.* 1977; Porter *et al.* 1978; Wiemeyer *et al.* 1978; Oehme 1980; Pruett-Jones *et al.* 1981).

Effects of organochlorines on the breeding success of raptors have been established through a combination of laboratory and field studies. They include eggshell thinning and egg breakage (McLane and Hall 1972; Lincer 1975; Newton and Bogan 1978), embryo mortality (Porter and Wiemeyer 1969; Conrad 1978; Newton and Bogan 1978), and death of young (Porter and Wiemeyer 1969). Effects of two or more toxicants on breeding of birds are poorly known. Several toxic compounds are commonly present at once in tissues of wild birds, and interactions between them may alter tissue storage and mortality from those observed in studies of single contaminants (Lincer 1972; Ludke 1974). DDE and PCBs (polychlorobiphenyls) appeared to interact in reducing breeding success of American kestrels (*Falco sparverius*) (Lincer 1972) and mallards (*Anas platyrhynchos*) (Risebrough and Anderson 1975). DDT and dieldrin together lowered reproductive success of American kestrels (Porter and Wiemeyer 1969). None of these studies included statistical evaluation of interactions between compounds in their effects on breeding.

In this study, the effects of dietary DDE and dieldrin were assessed, singly and in combination, on the breeding cycle of the barn owl (*Tyto alba*) in an effort to obtain information on the relative importance of these contaminants in the decline of wild raptor populations.

Materials and Methods

Owls were obtained from the captive colony at the Patuxent Wildlife Research Center. Nineteen pairs were experienced breeders (had hatched young previously), and were 1.5 to 6 years old; these pairs were kept together. Others were inexperienced birds 5 to 10 months old, and were paired randomly. All pairs were randomly assigned to 3.3m × 16.3m × 1.7m high outdoor pens, supplied with a water bowl and nest box. Eight to 11 inexperienced pairs and 4 to 5 experienced pairs were randomly assigned to each of four treatment groups.

[1] Present address: 15 Thiesfield St., Fig Tree Pocket, Brisbane 4069, Queensland, Australia

[2] Present address: Iowa Cooperative Wildlife Research Unit, Iowa State University, Ames, Iowa

9347

Groups were fed diets containing 3.0 ppm DDE wet weight, 0.5 ppm dieldrin, both compounds, or no pesticides (control group). Toxicants were mixed in Nebraska Brand Bird-of-Prey Diet, a fortified chopped horsemeat ration. DDE and dieldrin were dissolved in a minimum of acetone and then in propylene glycol, which was mixed with the horsemeat at a rate of 1% propylene glycol solution. Control diets contained only acetone and propylene glycol. Samples from 10 batches of control diet and 10 batches of DDE + dieldrin diet were frozen and were analyzed for DDE, dieldrin, and background contaminants.

The experiment began on 21 November 1974 and lasted for two years. When young were hatched in a pen, the diet was changed to whole dead undosed white mice or rats, which had previously been found necessary for successful rearing. When the last young were removed from the pen (at independence or death), the breeding pair was returned to the experimental diet. Rodents were fed for at least one period to 11 control pairs, 8 DDE pairs, 12 dieldrin pairs, and 6 DDE + dieldrin pairs. Adults that died or escaped during the first half of the experiment were replaced with inexperienced birds in November 1975. Inexperienced pairs that hatched young in 1975 were reclassified as experienced in the second year. At the termination of the experiment, 45 adults were sacrificed and their carcasses were analyzed for lipid and pesticide content. Three birds that died during the experiment were also analyzed, including liver, brain, and the remaining carcass. Residue data from sacrificed birds were tested by 3-way analysis of variance (ANOVA) for effect on each toxicant of feeding that compound, presence of the other compound, or sex. Residues were transformed as logs to stabilize variances, and means are geometric means.

Breeding data were recorded daily through fledging of the young, including number and date of eggs laid, broken (cracked or crushed), deserted, and hatched, and number of young fledged. The third egg of each clutch was removed at laying for measurement and residue analysis; breeding success data are based on the remaining eggs. Broken eggs and those that failed to hatch were removed. Addled eggs were examined for embryonation and stage of development. Fledged young were removed from the pen to permit rearing of subsequent broods. Data from all clutches in a year were pooled for each pen. All breeding success data were analyzed by 3-way ANOVA for effects of DDE or dieldrin treatment. Experience was included as the third factor to control for any effect it might have on the response of birds to treatments. Experience did not affect breeding success, so results are presented with data pooled across experience classes. Experience interacted with treatment effects in a few cases. Data were analyzed separately for 1975 and 1976 to preserve independence of samples. Proportions (eggs broken, infertile, deserted, and failing to hatch, and young dying) were transformed as $\sin^{-1}\sqrt{x}$, and proportional data for each pen were weighted according to size of the sample to promote homogeneity of variance.

Third eggs were measured and the contents were analyzed for lipid and pesticide content. Effects of dose on egg weight and lipid content were analyzed by 2-way ANOVA, with factors being DDE and dieldrin; lipid proportion was transformed as $\sin^{-1}\sqrt{x}$. Effects of treatment and year of laying on DDE and dieldrin in eggs were analyzed by 3-way ANOVA; residue data were transformed as logs, and means are geometric means. The eggshell was air-dried and its thickness (including shell membranes) was measured at the equator with a Starrett micrometer. Effects of pesticides on eggshell thickness were analyzed by linear regression of thickness on the log of residue, using 3rd eggs from all clutches. Significance of slopes and r's was tested by t test (Zar 1974). The difference between slopes for DDE and DDE + dieldrin treatments was tested after apportioning the control group randomly between the two regressions to preserve independence of the samples.

Chemical analyses were performed at Raltech Scientific Services, Inc., Madison, WI. Carcass samples were prepared by removal of beak, manus, feet, skin, and gastrointestinal tract, and were ground in a Hobart A0200 meat grinder. Egg samples were air-dried at room temperature for three days. A 10-g aliquot of each carcass, and whole eggs, were mixed with anhydrous sodium sulfate, air-dried for 2 to 5 days, and extracted for eight hours in a Soxhlet apparatus with 50% ethyl ether in petroleum ether. The solution was concentrated on a steam bath and the volume adjusted with petroleum ether. For cleanup, a 2 to 5 ml aliquot of the extract was placed on a Florisil[8] column and eluted with 150 ml of 3 to 5% ethyl ether in petroleum ether followed by 350 ml of 15% ethyl ether in petroleum ether. The solutions were concentrated on a steam bath and made to 25 to 50 ml with petroleum ether (hexane in 1975). An aliquot of 10 μl or less was analyzed by electron-capture gas chromatography by using the following columns: for 1975 eggs, 11% OV-17 + QF-1 or 1.5% SP-2250/1.95% SP-2401, and for PCBs, 5% DC-200; for 1976 eggs and carcasses, 1.5% OV-17 + 1.95% QF-1, or 5% OV-210; and for diet samples, 1.5% SP-2250/1.95% SP-2401. PCBs in eggs and carcasses were quantified on the basis of Aroclor[8] 1254 and in diet on the basis of Aroclor[8] 1016. The lower limit of sensitivity was 0.05 ppm except that for PCBs it was 0.1 ppm. All residues are expressed as ppm wet weight, and are accurate to two significant digits. For lipid determination, an aliquot of the Soxhlet extract was evaporated to dryness on a steam bath, desiccated, and weighed.

Results

Diets: Feed into which both DDE and dieldrin had been mixed contained a mean of 2.83 ppm DDE (standard error 0.10), 0.58 ppm dieldrin (SE 0.028), and 0.25 ppm PCBs (SE 0.090). No residues were detected in control diets. None of the diet residues differed between the first and second years of the experiment.

Effects of Pesticides on Adult Owls: DDE and dieldrin were significantly higher in carcasses of birds that were fed these compounds than in birds not receiving them (Table 1). There were no differences in carcass storage of either toxicant due to presence of the other ($p > 0.10$). Sex affected dieldrin residue, but a large 3-way interaction between all factors made this difficult to interpret. The mean PCB residue was 8.0 ppm.

Three DDE-fed birds (all female), 3 dieldrin-fed birds (2 females and 1 male), 2 DDE + dieldrin birds (female and male), and no control birds died during the experiment. The proportion of dosed birds that died was not significantly higher than among controls (χ^2, $p > 0.05$). Residues were analyzed only in the three dieldrin birds: two had low carcass lipid, and their brain residues (5.0 and 15 ppm) were within the lethal range (Stickel *et al.* 1969). Two DDE females died when they were unable to lay a

Table 1. Residues in carcasses of adult barn owls fed DDE, both toxicants, or control diet for two years

		Carcass residues, ppm wet weight			
		DDE		Dieldrin	
Dose group	Statistic	Male	Female	Male	Female
Control	GM[a]	0.80	0.71	0.33	0.15
	N	2	2	2	2
	SD[b]	0.0078	0.091	0.25	0.12
DDE[c]	GM	112.	78.	0.23	0.30
	N	9	6	9	6
	SD	0.056	0.34	0.14	0.14
Dieldrin[cd]	GM	1.2	1.1	9.6	9.2
	N	12	7	12	7
	SD	0.18	0.16	0.17	0.12
DDE + Dieldrin[cd]	GM	102.	79.	12.	7.8
	N	6	5	6	5
	SD	0.10	0.16	0.13	0.20

[a] Geometric mean
[b] Standard deviation (in logs)
[c] DDE and dieldrin were each higher in birds dosed with that compound ($p < 0.001$)
[d] Dieldrin residues differed with sex ($0.05 > p > 0.01$); 3-way interaction between all 3 factors ($0.05 > p > 0.01$)

virtually shell-less egg. Cause of death in the other birds was not determined.

Effects of Pesticides on Eggs: Egg weight averaged 26.67 g, and lipid content 5.36%. There was no significant change in egg characteristics with treatment in either year. Mean residues of toxicants were higher in eggs of pairs receiving dosed diets than in those of undosed ones (Table 2). There was no effect of either toxicant on deposition of the other ($p > 0.10$). Residues in eggs increased significantly from 1975 to 1976, suggesting that adults continued to accumulate organochlorines for at least 14 months. PCBs in eggs averaged 1.6 ppm in 1975 and 2.1 ppm in 1976.

Eggshells were thinned severely by DDE and slightly by dieldrin (Table 2). Both effects were significant, and they were additive (Figures 1 and 2). Thinning was greater in 1976, which corresponds with the higher egg residues in that year.

Breeding Success: Laying was reduced in dosed groups in 1975 only (Table 3). All pairs were inexperienced that failed to lay in the first year. Interaction between treatment effects and experience, as well as uncontrolled variables that differed between experience groups, make it difficult to interpret the tendency not to lay. Production by birds that laid eggs was significantly influenced by DDE in both years (Table 3). Egg breakage increased greatly with DDE. There was significant interaction between DDE and experience factors in 1975 ($0.05 > p > 0.01$), but it was small and did not interfere with interpretation. More eggs were laid by females in DDE-dosed groups, because eggs that broke before completion of a clutch were often replaced, and because females that lost a clutch renested more often (up to four clutches per year in DDE and DDE + dieldrin groups, up to 3 for control and dieldrin). More embryos died in DDE and DDE + dieldrin groups, and this was significant in 1976. Hatching success was lower with DDE in both years. Mortality of young was not significantly affected by treatment, and therefore fledging success followed the same trends as hatching success.

Mean date of first laying (Table 3), infertility, and desertion of the clutch did not differ between treatment groups ($p > 0.10$).

Discussion

Results of the study are consistent with others in which DDE had severe effects on breeding success of birds. Similar doses of DDE caused eggshell thinning of 11 to 22% in various other species (Heath *et al.* 1969; McLane and Hall 1972; Longcore and Samson 1973; Lincer 1975). The barn owl appears to be among the most sensitive tested species. Embryo mortality in the barn owls was significant only in 1976, when DDE residues were highest, and was a minor component of overall breeding failure, due to the overwhelming incidence of breakage. Death of embryos due to DDE has been found in some laboratory studies (Heath *et al.* 1969; Smith *et al.* 1969; Longcore *et al.* 1971) and in two field studies (Conrad 1978; Newton and Bogan 1978), but not in other cases (Jones and Summers 1968; Britton *et al.* 1974). Death of newly-hatched young did not contribute to breeding losses in this study, although it has in others (Porter and Wiemeyer 1969; Longcore *et al.* 1971; Britton *et al.* 1974).

Dieldrin did not reduce breeding success in the barn owls, either alone or through interaction with DDE. The slight eggshell thinning which it induced resembled that in mallards (Davison and Sell 1974), but did not contribute to egg breakage. Dieldrin did not cause embryo mortality; this is consistent with most studies (DeWitt 1956; Graves *et al.* 1969; Wiese *et al.* 1969; Dahlgren and Linder 1974; Shellinberger 1978), although occasional effects have been found (DeWitt 1956; Neill *et al.* 1971). Earlier suggestions that dieldrin may be a primary cause of embryo mortality, alone or when other organo-

Table 2. Residues and shell thickness of eggs of barn owls fed DDE, dieldrin, both toxicants, or control diet, 3rd egg of 1st clutch in each year

Year		Treatment group			
		Control	DDE	Dieldrin	DDE + dieldrin
1975	DDE, ppm[a]	0.25[d](0.058)[e]	12. (0.029)	0.29 (0.067)	12. (0.060)
	Dieldrin, ppm[b]	0.31 (0.061)	0.13 (0.033)	3.6 (0.26)	4.2 (0.041)
	Shell thickness, mm[c]	0.278(0.004)	0.223(0.005)	0.269(0.004)	0.220(0.007)
	N	13	9	8	13
1976	DDE, ppm[a]	0.40 (0.051)	41. (0.025)	0.36 (0.051)	42. (0.060)
	Dieldrin, ppm[b]	0.20 (0.47)	0.23 (0.025)	8.1 (0.036)	9.0 (0.054)
	Shell thickness, mm[c]	0.288(0.004)	0.207 (0.003)	0.272(0.004)	0.206(0.006)
	N	10	13	11	10

[a] DDE was higher in eggs from groups fed DDE, and was higher in 1976 than 1975: $p < 0.001$
[b] Dieldrin was higher in eggs from groups fed dieldrin, and was higher in 1976 than 1975: $p < 0.001$
[c] Differences in shell thickness tested by regression analysis (Figures 1 and 2)
[d] Residues are geometric means
[e] Standard error, in logs

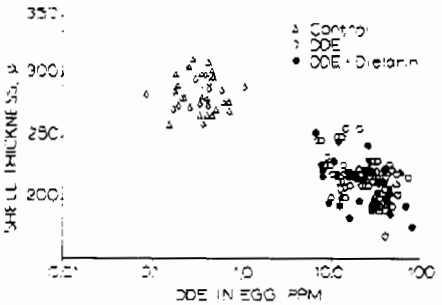

Fig. 1. Shell thickness as a function of DDE in eggs. For DDE, $y = 264 - 33.4 \log x$, $r^2 = 0.79$, $N = 68$: $p < 0.001$ for slope and r. For DDE + dieldrin, $y = 265 - 38.8 \log x$, $r^2 = 0.80$, $N = 61$: $p < 0.001$ for slope and r. Slopes with and without dieldrin are significantly different: $p = 0.04$

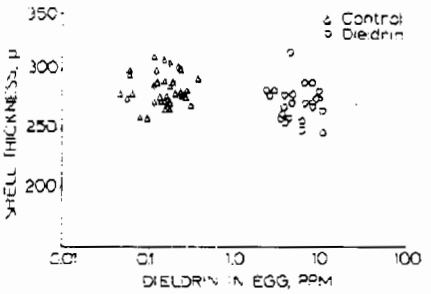

Fig. 2. Shell thickness as a function of dieldrin in egg. $Y = 278 - 6.10 \log x$, $r^2 = 0.10$, $N = 60$: $0.02 > p > 0.01$ for slope and r

chlorines are present (Blus *et al.* 1974; Newton 1979), were not borne out.

Breeding failure caused by DDE in our experiment and others supports the conclusion that DDT contamination has severely reduced productivity of wild raptors. Residues and shell thinning in the barn owls in 1975 were of the same magnitude as in many declining populations of diurnal raptors (Cade *et al.* 1971; Koeman *et al.* 1972; Wiemeyer *et al.* 1972; Gilbertson and Reynolds 1974; Henny *et al.* 1977; Lincer and Clark 1978). Wild owls have generally escaped severe contamination (Gilbertson and Reynolds 1974; Lincer and Clark 1978), although a few may approach it (Klaas *et al.* 1978). Experimental residues in 1976 exceeded almost all records for wild birds.

Failure throughout the breeding cycle of the barn owls was similar in many respects to that in a closely-studied natural population of European sparrowhawks (*Accipiter nisus*) (Newton 1974; Newton and Bogan 1978). Egg breakage accounted for the largest proportion of failure in the sparrowhawks, followed by embryo mortality, and both were associated with DDE; death of young was negligible. Failure to lay and desertion reduced productivity more than in the barn owls.

Dieldrin appears from our results to have been a minor factor in breeding failure of raptors. Residues in the barn owls were higher than in any wild population, yet they were not associated with losses. The compound has been implicated in breeding failure of one species, the golden eagle (*Chrysaetos aquila*) (Lockie *et al.* 1969). In general, however, it appears that DDE rather than dieldrin has been responsible for reduced productivity in wild birds.

Assessing the full effect of pesticides on natural populations requires that we consider not only productivity but also mortality. Mortality due to pesticide poisoning has contributed to raptor declines,

Table 3. Reproductive success of barn owls fed DDE, diel[illegible] both toxicants, or control diet for two years

	1975				1976			
	Control	DDE	[illegible]drin	DDE + dieldrin	Control	DDE	Dieldrin	DDE + dieldrin
Number of breeding pairs[a]	14	13		12	14	14	12	15
Number of pairs laying[b]	13	9		9	13	14	12	14
Mean date of 1st egg	9 Mar.	23 Feb.	[illegible]eb.	25 Feb.	9 Feb.	29 Jan.	8 Feb.	13 Feb.
Mean eggs laid/pair[c]	7.85	14.6	[illegible].75	13.6	10.9	17.3	9.08	15.8
% eggs broken[d,e]	3.0	82.	[illegible].2	47.	5.0	60.	6.0	58.
% embryos dying[f]	21.	25.	[illegible].7	32.	24.	44.	2.8	68.
Mean eggs hatched/pair[g]	3.15	1.11	3.88	2.33	3.69	0.86	3.92	1.00
% nestlings dying	6.6	46.	2.1	0.41	7.6	6.7	10.	6.7
Mean young fledged/pair[g]	2.85	0.67	3.62	2.22	3.08	0.71	3.08	0.78

[a] Excludes pairs in which escape or death interrupted [illegible]eeding
[b] All results listed beneath based on pairs that laid at least one clutch
[c] DDE effect significant: $p < 0.001$ in 1975, $0.01 > p$ [illegible] 0.001 in 1976
[d] Percentages are transformed
[e] DDE effect significant: $p < 0.001$ for both years
[f] DDE effect significant for 1976 only: $p < 0.001$
[g] DDE effect significant: $0.05 > p \geq 0.01$ for 1975, $0.01 > p \geq 0.001$ for 1976

some catastrophic (Prestt and Ratcliffe 1972; Newton 1979). Dieldrin has probably had a much greater impact on survival of wild birds than DDT. It has been assigned primary blame for many raptor deaths (Koeman *et al.* 1969; Mulhern *et al.* 1970; Belisle *et al.* 1972; Prestt and Ratcliffe 1972; Bogan and Mitchell 1973; Kaiser *et al.* 1980). Fewer deaths have been ascribed to DDT, even in North America where it has predominated among contaminants (Mulhern *et al.* 1970; Belisle *et al.* 1972). It appears from laboratory and field results that DDT and dieldrin have exerted their effects on raptor populations largely at opposite ends of the life cycle.

Acknowledgments. We thank Jan Barton, Jane Hildreth, Joe Snyder, Dan Smith, and Sue Brown for technical assistance; Gary Hensler and William Stout for extensive statistical support; and Stanley N. Wiemeyer, Christine Bunck, and Gary Heinz for reviewing the manuscript.

References

Barr, A. J., J. H. Goodnight, J. P. Sall, W. H. Blair, D. M. Chilko, J. T. Helwig, and K. A. Council: SAS users' guide. Raleigh, NC: SAS Institute (1979).

Belisle, A. A., W. L. Reichel, L. N. Locke, T. G. Lamont, B. M. Mulhern, R. M. Prouty, R. B. DeWolf, and E. Cromartie: Residues of organochlorine pesticides, polychlorinated biphenyls, and mercury and autopsy data for bald eagles, 1969 and 1970. Pestic. Monit. J. **6**, 133 (1972).

Blus, L. J., B. S. Neely, A. A. Belisle, and R. M. Prouty: Organochlorine residues in brown pelican eggs: Relation to productive success. Environ. Pollut. **7**, 81 (1974).

Bogan, J. A., and J. Mitchell: Continuing dangers to peregrines from dieldrin. Brit. Birds **66**, 437 (1973).

Britton, W. M., M. B. Palmer, and B. Howarth, Jr.: Fertility, hatchability, and progeny performance of laying hens fed high levels of DDT in the diet. Poultry Sci. **53**, 1053 (1974).

Cade, T. J., J. L. Lincer, C. M. White, D. G. Roseneau, and L. G. Swartz: DDE residues and eggshell thinning in Alaskan falcons and hawks. Science **172**, 955 (1971).

Conrad, B.: Korrelation zwischen Embryonen-Sterblichkeit und DDE-Kontamination beim Sperber (*Accipter nisus*). J. Ornithol. **119**, 109 (1978).

Dahlgren, R. B., and R. L. Linder: Effects of dieldrin in penned pheasants through the third generation. J. Wildl. Manage. **38**, 320 (1974).

Davison, K. L., and J. L. Sell: DDT thins shells of eggs from mallard ducks maintained on *ad libitum* or controlled-feeding regimens. Arch. Environ. Contam. Toxicol. **2**, 222 (1974).

DeWitt, J. B.: Chronic toxicity to quail and pheasants of some chlorinated insecticides. J. Agr. Food Chem. **4**, 863 (1956).

Gilbertson, M., and L. Reynolds: DDE and PCB in Canadian birds, 1969 to 1972. Can. Wildl. Serv., Occas. Pap. 19 (1974).

Graves, J. B., F. L. Bonner, W. F. McKnight, A. B. Watts, and E. A. Epps: Residues in eggs, preening glands, liver, and muscle from feeding dieldrin-contaminated rice bran to hens and its effect on egg production, egg hatch, and chick survival. Bull. Environ. Contam. Toxicol. **4**, 375 (1969).

Heath, R. G., J. W. Spann, and J. F. Kreitzer: Marked DDE impairment of mallard reproduction in controlled studies. Nature, London **224**, 47 (1969).

Henny, C. J., M. A. Byrd, J. A. Jacobs, P. D. McLain, M. R. Todd, and B. F. Halla: Mid-Atlantic coast osprey population: Present numbers, productivity, pollutant contamination, and status. J. Wildl. Manage. **41**, 254 (1977).

Jones, F. J. S., and D. D. B. Summers: Relation between DDT in diets of laying birds and viability of their eggs. Nature, London **217**, 1162 (1968).

Kaiser, T. E., W. L. Reichel, L. N. Locke, E. Cromartie, A. J. Krynitsky, T. G. Lamont, B. M. Mulhern, R. M. Prouty, C. J. Stafford, and D. M. Swineford: Organochlorine pesticides, PCB, and PBB residues, and necropsy data for bald eagles from 29 states—1975–77. Pestic. Monit. J. **13**, 145 (1980).

Klaas, E. E., S. N. Wiemeyer, H. M. Ohlendorf, and D. M. Swineford: Organochlorine residues, eggshell thickness, and nest success in barn owls from the Chesapeake Bay. Estuaries **1**, 46 (1978).

Koeman, J. H., C. F. van Beusekom, and J. J. M. de Goeij: Eggshell and population changes in the sparrowhawk (*Accipter nisus*). TNO-Nieuws **27**, 542 (1972).

Koeman, J. H., J. A. J. Vink, and J. J. M. de Goeij: On the causes of the mortality in predatory birds in the Netherlands in the winter of 1968–1969. Ardea **57**, 67 (1969).

Lincer, J. L.: The effects of organochlorines on the American kestrel (*Falco sparverius* Linn.) Unpubl. Ph.D. dissertation. Cornell Univ. (1972).

———: DDE-induced eggshell thinning in the American kestrel: A comparison of the field situation and laboratory results. J. Appl. Ecol. **12**, 781 (1975).

Lincer, J. L., and R. J. Clark: Organochlorine residues in raptor eggs in the Cayuga Lake basin. NY Fish Game J. **25**, 121 (1978).

Lockie, J. D., D. A. Ratcliffe, and R. Balharry: Breeding success and organochlorine residues in golden eagles in west Scotland. J. Appl. Ecol. **6**, 381 (1969).

Longcore, J. R., and F. B. Samson: Eggshell breakage by incubating black ducks fed DDE. J. Wildl. Manage. **37**, 390 (1973).

Longcore, J. R., F. B. Samson, and T. W. Whittendale, Jr.: DDE thins eggshells and lowers reproductive success of captive black ducks. Bull. Environ. Contam. Toxicol. **6**, 485 (1971).

Ludke, J. L.: Interaction of dieldrin and DDE residues in Japanese quail (*Coturnix coturnix japonica*). Bull. Environ. Contam. Toxicol. **11**, 297 (1974).

McLane, M. A. R., and L. C. Hall: DDE thins screech owl eggshells. Bull. Environ. Contam. Toxicol. **8**, 65 (1972).

Mulhern, B. M., W. L. Reichel, L. N. Locke, T. G. Lamont, A. Belisle, E. Cromartie, G. E. Bagley, and R. M. Prouty: Organochlorine residues and autopsy data from bald eagles, 1966–68. Pestic. Monit. J. **4**, 141 (1970).

Neill, D. D., H. D. Muller, and J. V. Schutze: Pesticide effects on the fecundity of the Gray Partridge. Bull. Environ. Contam. Toxicol. **6**, 546 (1971).

Newton, I.: Changes attributed to pesticides in the nesting success of the sparrowhawk in Britain. J. Appl. Ecol. **11**, 95 (1974).

———: Population ecology of raptors. Vermillion, SD: Buteo Books (1979).

Newton, I., and J. Bogan: The role of different organochlorine compounds in the breeding of British sparrowhawks. J. Appl. Ecol. **15**, 105 (1978).

Oehme, G.: Veränderungen der Eishalendicke beim Seeadler, *Haliaeetus albicilla* (L.), als Parameter der Fremdstoffkontamination und des Reproduktionsvermögens. Wissenschaftliche Zeitschrift der Padagogischen Hochschule "N. K. Krupskaja" (Halle) **18**, 27 (1980).

Porter, R. D., G. R. Craig, D. H. Ellis, J. H. Enderson, and W. G. Hunt: Status of the peregrine falcon in the Rocky Mountains and the southwestern United States, Baja California, and Mexico (south of Texas). In P. P. Schaeffer and S. M. Ehlers (eds.): Current status of peregrine falcon populations in North America, p. 26. Tiburon, CA:National Audubon Society Western Education Center (1978).

Porter, R. D., and S. N. Wiemeyer: Dieldrin and DDT: effects on sparrowhawk eggshells and reproduction. Science **165**, 199 (1969).

———: DDE at low dietary levels kills captive American kestrels. Bull. Environ. Contam. Toxicol. **8**, 193 (1972).

Prestt, I., and D. A. Ratcliffe: Effects of organochlorine insecticides on European birdlife. Proc. Intl. Ornithol. Congr. **15**, 486 (1972).

Pruett-Jones, S. G., C. M. White, and W. B. Emison: Eggshell thinning and organochlorine residues in eggs and prey of peregrine falcons from Victoria, Australia. Emu **80**, Suppl., 281 (1981).

Ratcliffe, D. A.: The peregrine falcon. Vermillion, SD:Buteo Books (1980).

Risebrough, R. W., and D. W. Anderson: Some effects of DDE and PCB on mallards and their eggs. J. Wildl. Manage. **39**, 508 (1975).

Shellenberger, T. E.: A multi-generation toxicity evaluation of *p,p'*-DDT and dieldrin with Japanese quail. I. Effects on growth and reproduction. Drug Chem. Toxicol. **1**, 137 (1978).

Smith, S. I., C. W. Weber, and B. L. Reid: The effect of high levels of dietary DDT on egg production, mortality, fertility, hatchability, and pesticide content of yolks in Japanese quail. Poultry Sci. **48**, 1000 (1969).

Spitzer, P. R., R. W. Risebrough, W. Walker II, R. Hernandez, A. Poole, D. Puleston, and I. C. T. Nisbet: Productivity of ospreys in Connecticut-Long Island increases as DDE residues decline. Science **202**, 333 (1978).

Stickel, W. H., L. F. Stickel, and J. W. Spann: Tissue residues of dieldrin in relation to mortality in birds and mammals. In M. W. Miller and G. C. Berg (eds.): Chemical fallout: Current research on persistent pesticides, p. 174. Springfield, IL:Thomas (1969).

Wiemeyer, S. N., A. A. Belisle, and F. J. Gramlich: Organochlorine residues in potential food items of bald eagles (*Haliaeetus leucocephalus*), 1966 and 1974. Bull. Environ. Contam. Toxicol. **19**, 64 (1978).

Wiemeyer, S. N., B. M. Mulhern, F. J. Ligas, R. J. Hensel, J. E. Mathisen, F. C. Robards, and S. Postupalsky: Residues of organochlorine pesticides, polychlorinated biphenyls, and mercury in bald eagle eggs and changes in shell thickness—1969 and 1970. Pestic. Monit. J. **6**, 50 (1972).

Wiese, I. H., N. C. J. Basson, J. H. Van der Vyver, and J. H. Van der Merwe: Toxicology and dynamics of dieldrin in the crowned guinea fowl, *Numida meleagris* (L). Phytophylactica **1**, 161 (1969).

Zar, J. H.: Biostatistical analysis. Englewood Cliffs, NJ:Prentice Hall (1974).

Manuscript received February 8, 1982; accepted August 20, 1982.