

Effects of 15 Common Environmental Pollutants on Eggshell Thickness in Mallards and Coturnix

by

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Introduction

Eggshell thinning has been documented in several species of fish-eating and raptorial birds in recent years (ANDERSON et al. 1969, FYFE et al. 1969, HICKEY and ANDERSON 1968, KEITH et al. 1970). High residue levels of DDE, the principal metabolite of DDT, were found in the affected birds and their eggs. Most researchers believe that DDE is the causative agent in the shell-thinning phenomenon (ANDERSON et al. 1969, CADE et al. 1971, FYFE et al. 1969, HICKEY and ANDERSON 1968, KEITH et al. 1970, PEAKALL 1970 b). However, laboratory studies with birds given DDE have not shown shell thinning of the magnitude (as much as 50%) seen in some species of wild birds (RISEBROUGH et al. 1971). For example, studies with penned ducks (HEATH et al. 1969, LONGCORE et al. 1971) have shown that DDE can cause eggshell thinning of 13 to 23%. The lower degree of thinning in ducks may be due to species differences, or to the possibility that the responses of this and other species under laboratory conditions may not be representative of those of fish-eating and raptorial birds in the wild. However, it is also possible that additional chemicals are partly responsible for the greater shell thinning and reproductive failure seen in some wild birds. Long and extensive use of DDT has led to the ubiquitous occurrence of the stable metabolite DDE in the environment (HICKEY 1969), but other chemicals, such as mercury, lead, and polychlorinated biphenyls, have also been shown to be widespread environmental pollutants (KNAPP 1970, LAZRUS et al. 1970, PEAKALL and LINCER 1970). This study was conducted to investigate the capacity of some of these compounds to cause eggshell thinning. Fifteen common pesticides and environmental pollutants, including DDE, were tested by a rapid, short-term screening procedure in two common species of laboratory birds.

Procedure

Eighty-four unmated female coturnix quail (*Coturnix coturnix japonica*) were randomly distributed into 14 groups of 6 each and placed in individual indoor cages on a regimen of 14 hours of light and 10 hours of dark. Eighty female mallards (*Anas platyrhynchos*) in their first reproductive season were randomly distributed into 16 groups of 5 birds each and placed in outdoor cages. Eggs were collected from all birds for 6 days to obtain an average pre-treatment eggshell thickness. Each group was orally administered a single dose of one of the chemicals, by gelatin capsule through glass tubing to the level of the proventriculus. So that maximum shell-thinning potential for each chemical would be expressed, dosages were chosen to be high but

not lethal. Eggs laid after treatment were collected and measured for 6 days, except for eggs from the DDE-treated mallards, which were collected for 18 days, and those from the Aroclor-treated mallards, which were collected for 10 days. Eggs (including membranes) were measured at the equator to the nearest 5 microns with a micrometer.

Results and Discussion

Table 1 lists the chemicals and dosages given, and shows the 12 treatments that had no apparent effect on eggshell thickness. Figures 1, 2, and 3 show the results with the 14 treatments that caused eggshell thinning in coturnix or mallards. The graphs show that single oral doses of several chemicals and pesticides caused temporary eggshell thinning in coturnix and mallards. But the only prolonged eggshell thinning observed was that caused by p,p'-DDE in mallards. Much of the eggshell thinning observed in coturnix was probably caused by low food consumption in the treated birds. Food consumption was greatly reduced during the first few days after treatment in coturnix that showed eggshell thinning (Figure 1), but not in the coturnix which did not exhibit eggshell thinning (Table 1). Figure 1 shows that untreated birds, when fasted for 36 hours, laid thin-shelled eggs for a few days during and after the fast. This pattern was quite similar to that shown by the chemically treated birds.

Mallard food consumption was not measured because of inclement weather and outdoor pens, but lower food consumption after treatment probably had some transitory shell effect, as in the coturnix. Mallards that have been fasted for a few days in some of our other studies have laid eggs with thinner shells during and shortly after the fast.

The timing of the eggshell thinning in mallards given DDE differed from that seen with the other compounds tested (Figure 3). These birds were still laying thin-shelled eggs at 6 days post-treatment, so eggs were collected for an additional 12 days. Even by this time, there was no appreciable recovery to normal shell thickness. Not only did thinning last longer, but maximum thinning also occurred more quickly (within about 20 hours after treatment). PEAKALL (1970 a) also found thinning in the first eggs laid after ring doves (*Streptopelia risoria*) were injected with DDE. The fact that birds treated with a single dose of DDE lay thin-shelled eggs in less than 24 hours suggests that eggshell thinning is not associated with enzyme induction. In this amount of time, liver microsomal enzymes probably could not be induced at levels sufficient to cause the thinning observed.

Except for the pattern shown by DDE, the shell thinning produced by the compounds tested appears to be associated with reduced food consumption caused by sublethal intoxication and could be termed toxic thinning. In contrast, the shell-thinning response to DDE appears to occur in the absence of any other clinical sign of intoxication. Thus it appears that DDE, the major degradation product of DDT in the environment, can be a very quick-acting eggshell-thinning agent with long-term effects. This suggests that birds exposed to DDE on the winter grounds or during migration north could lay thin-shelled eggs, even though the food supply where they nested was not significantly contaminated.

TABLE 1

Chemicals tested for eggshell-thinning effects

Compound	Single Oral Dose (mg/kg)	
	Coturnix	Mallards
Aroclor 1254 ^{a/} (PCB)	500	1000
Ceresan M ^{b/}	500	500
2,4-D acid	250	1500
p,p'-DDE	125 ^{c/}	500
		1000
		2000
o,p'-DDT	125 ^{c/}	
p,p'-DDT	125 ^{c/}	
DDT	125 ^{c/}	
Dieldrin	10 ^{c/}	60 ^{c/}
Chlordecone		25 ^{c/}
Heptachlor		1000
Parathion	2.5	1 ^{c/}
Carbaryl	1000	1000 ^{c/}
Sodium arsenite		100
Tetraethyllead	6	6 ^{c/}
Toxaphene	10 ^{c/}	

^{a/}Trade name of Monsanto for polychlorinated biphenyl containing 54% chlorine. Reference to trade names does not imply endorsement of commercial products by the Federal Government.

^{b/}Trade name of DuPont for N-(ethylmercuri)-p-toluene sulfonanilide.

^{c/}Caused no appreciable eggshell thinning (Reduction between pre- and post-treatment thickness, < 5 microns).

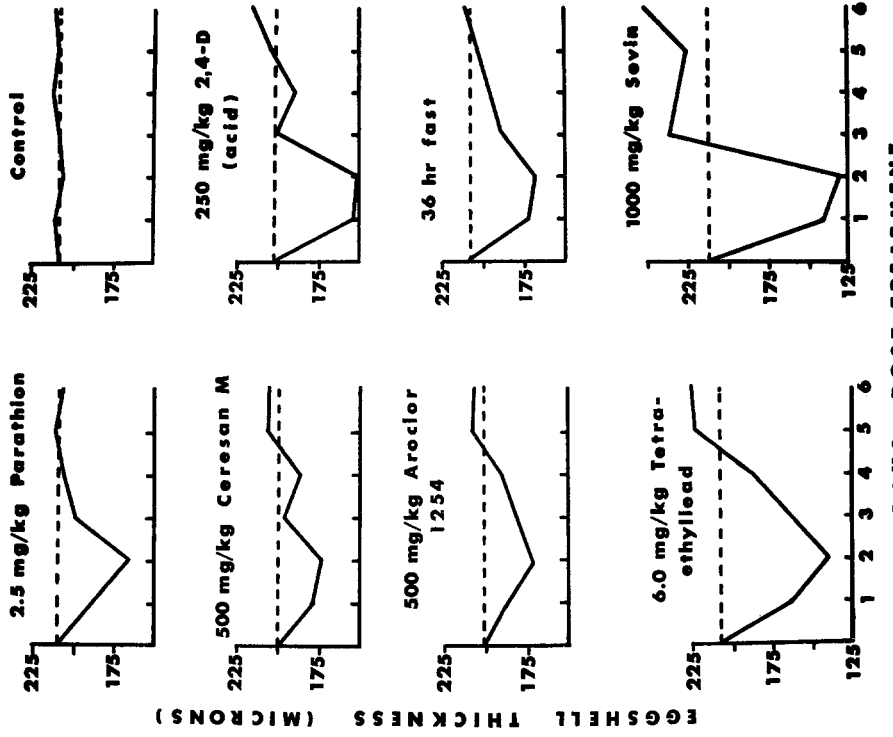


Figure 1. Treatments causing short-term eggshell thinning in coturnix (six birds per treatment). Dotted lines represent average 6-day pre-treatment eggshell thickness. ■ Eggs collected for days 7 through 10 averaged greater than normal thickness.

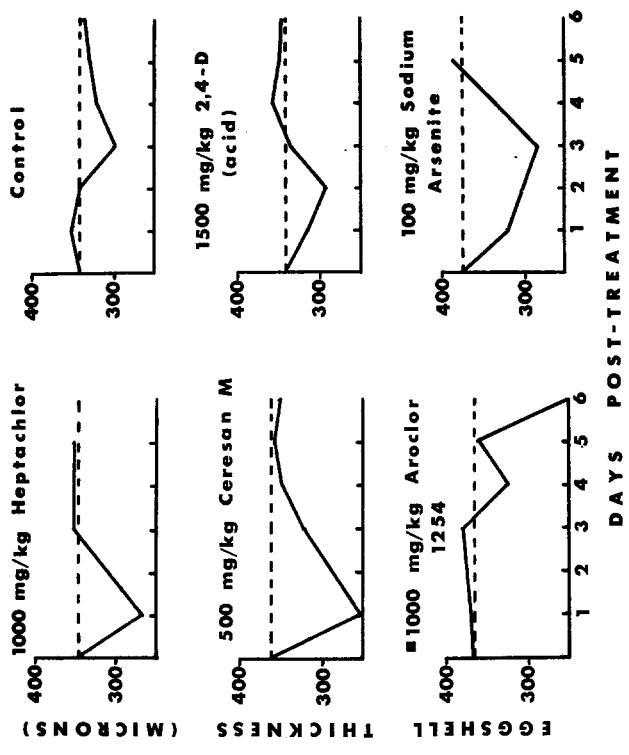


Figure 2. Treatments causing short-term eggshell thinning in mallards (five birds per treatment). Dotted lines represent average 6-day pre-treatment eggshell thickness. ■ Eggs collected for days 7 through 10 averaged greater than normal thickness.

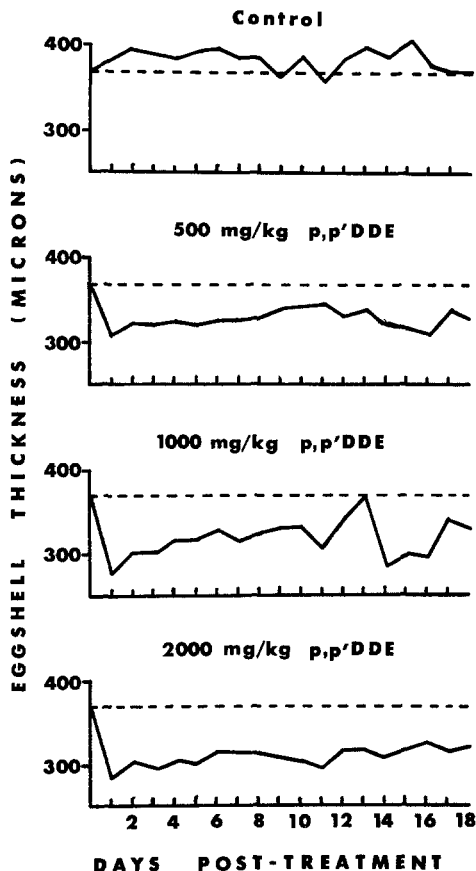


Figure 3 Effects of p,p'-DDE on mallard eggshell thickness (five birds per treatment). Dotted lines represent average 6-day pre-treatment eggshell thickness.

References

- ANDERSON, D. W., J. J. HICKEY, R. W. RISEBROUGH, E. F. HUGHES, and R. E. CHRISTENSEN: *Can. Field Natur.* **83**, 91 (1969).
- CADE, T. J., J. L. LINCER, C. M. WHITE, D. G. ROSENEAU, and L. G. SWARTZ: *Science* **172**, 955 (1971).
- FYFE, R. W., J. CAMPBELL, B. HAYSON, and K. HODSON: *Can. Field Natur.* **83**, 191 (1969).
- HEATH, R. G., J. W. SPANN, and J. F. KREITZER: *Nature* **224**, 47 (1969).
- HICKEY, J. J.: *Atlantic Natur.* **24**, 86 (1969).
- HICKEY, J. J., and D. W. ANDERSON: *Science* **162**, 271 (1968).
- KEITH, J. O., L. A. WOODS, JR., and E. G. HUNT: *Trans. 35th North Amer. Wildl. Natur. Resources Conf.*, p 56 (1970).
- KNAPP, C. E.: *Environ. Sci. Technol.* **4**, 890 (1970).
- LAZRUS, A. L., E. LORANGE, and J. P. LODGE, JR.: *Environ. Sci. Technol.* **4**, 55 (1970).
- LONGCORE, J. R., F. B. SAMSON, and T. W. WHITTENDALE, JR.: *Bull. Env. Cont. and Toxicol.* **6**, 485 (1971).
- PEAKALL, D. B.: *Science* **168**, 592 (1970 a).
- PEAKALL, D. B.: *Sci. Amer.* **222**, 73 (1970 b).
- PEAKALL, D. B., and J. L. LINCER: *BioScience* **20**, 958 (1970).
- RISEBROUGH, R. W., F. C. SIBLEY, and M. N. KIRVEN: *Amer. Birds* **25**, 8 (1971).