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## Eggshell Thickness and Reproduction in American Kestrels Exposed to Chronic Dietary Lead

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**Abstract.** American kestrels (*Falco sparverius*) were randomly paired and fed 0, 10, or 50 ppm metallic lead in their diet from November 1979–May 1980. Lead levels were elevated in bones and livers of birds receiving the treated diets, particularly the 50 ppm treatment group. Differential deposition of lead was noted between males and females, with the highest levels in the females. No adverse effects were evident with respect to survival, egg laying, or initiation of incubation in any treatment group nor was fertility or eggshell thickness affected. Little or no lead was transferred to the egg contents and although lead was present in the shell, the levels were too variable for this to be considered a useful measure of exposure.

Lead is a wide-spread contaminant that enters the environment through a variety of sources. The primary source is from the combustion of lead-containing fuels such as coal, fuel oil, and gasoline (Corrin and Natusch 1977) but less common sources such as lead shot also pose a hazard to wildlife (Bellrose 1959; McConnel 1967). Birds of prey may ingest lead in the form of shot from dead or crippled game animals (Locke *et al.* 1969; Benson *et al.* 1974; Jacobson *et al.* 1977) or biologically-incorporated lead from lead-poisoned waterfowl (29.3 ppm, wet weight, Stendell 1980), roadside small mammals (2.6–34.8 ppm, dry weight, Getz *et al.* 1977a; Goldsmith and Scanlon 1977), and invertebrates (682 ppm, dry weight, Williamson and Evans 1972).

Little is known about the sublethal effects of lead ingestion on birds of prey but Grandjean (1976) re-

ported a correlation between shell thickness and eggshell lead levels in European kestrels (*Falco tinnunculus*) and suggested that lead may cause shell thinning in raptors. Edens *et al.* (1976) found lead acetate incorporated in the diet caused a depression in egg production in coturnix quail (*Coturnix coturnix*) at 1 ppm but inhibition of growth, reduced hatchability, and soft-shelled eggs occurred only at 1,000 ppm. Stone and Soares (1974) also found that 1,000 ppm lead in the diet reduced egg production and caused soft-shelled eggs in coturnix quail. Morgan *et al.* (1975) found 500 ppm of dietary lead acetate inhibited growth and produced anemia. In a field study with barn swallows (*Hirundo rustica*) associated with a heavily-travelled highway, reproductive success was not affected although lead levels were elevated and delta-aminolevulinic acid dehydratase activity depressed 30 to 34 percent (Grue *et al.*, in press).

The exposure of birds of prey to lead may be increasing. Peregrine falcons (*Falco peregrinus*) are being released in eastern United States cities where they prey on starlings (*Sturnus vulgaris*) and pigeons (*Columba livia*), both of which exhibited elevated tissue lead levels in urban environments (Getz *et al.* 1977b). Bald eagles (*Haliaeetus leucocephalus*) also are exposed to lead in the dead or crippled waterfowl they consume, sometime receiving lethal levels (Kaiser *et al.* 1980). Since American kestrels frequent roadsides and many urban areas where elevated lead levels occur, an evaluation of the effects of ingested lead on shell thickness and reproduction was designed. The resulting data should also be useful in assessing the hazards posed by ingested lead to other species of raptors, particularly eagles and peregrines.



## Materials and Methods

Ninety-six 1 to 6-year-old American kestrels (48 male, 48 female) from the captive colony maintained at the Patuxent Wildlife Research Center, Laurel, MD, were randomly paired and assigned on 7 November 1979 to outdoor pens (14.5 m × 3 m × 1.8 m) containing a nest box, 2 covered perches, 2 hanging perches, a covered feeding platform, and a water bowl (Porter and Wiemeyer 1970). Birds were fed 100 g of commercial bird of prey diet/pair/day. Sixteen pairs containing equal numbers of each age class were assigned to each of 3 treatment groups: 0 ppm Pb (control), 10 ppm Pb, or 50 ppm Pb. Diets were prepared in a Hobart vertical cutter mixer and contained the appropriate amount of metallic lead powder (0, 10, or 50 ppm; Chemical Abstract Service Registry No. 7439-92-1) and were supplemented with Vionate® (1%), calcium phosphate (0.5%), and nf 180 (0.0055% N-[5-nitro-2-furfurylidene]-3-amino-2-oxazolidone). Uneaten food was removed daily and consumption monitored. Birds were maintained on these diets until completion of their clutch (5–7 months), at which time they were killed and selected tissues (tibia, humerus, liver) were removed and stored frozen until residue analysis.

Birds were weighed when the experiment started and monthly thereafter. Nest boxes were filled with 15–20 cm of peat moss and monitored until egg laying commenced. Once laying started, boxes were checked each morning (0900 hr) and afternoon (1600 hr) and eggs sequentially numbered. Five to seven days after the last egg was laid, eggs were removed and stored at 5°C. Length and breadth were measured with a dial caliper (0.01 mm), then each egg was opened by scoring the shell around the equator. Fertility was determined on the basis of apparent embryonic development. Egg contents were stored frozen in acid and solvent-rinsed glass jars with teflon lid liners. Shells were stored in egg cartons and air-dried a minimum of 30 days before shell weight (0.0001 g) and thickness (0.01 mm) were determined. Ratcliffe indices were then determined (Ratcliffe 1967).

Lead residue analyses were done by Hazleton Raltech, Inc., according to the Association of Official Analytical Chemists (1980) Methods 25.068–25.073 and 25.082–25.088. Lower limit of reportable residues was 0.5 micrograms. All data herein are reported as ppm, dry weight, except for diets, which were formulated and analyzed on a wet weight basis. The shell and contents of the third egg laid from each clutch (except for one 2-egg clutch where the second egg laid was used) were analyzed for lead as were all of the shells from five randomly selected clutches (five egg clutches only) from the 50 ppm treatment group. The livers from all the birds plus the tibias and humeri from 16 randomly selected females (0 ppm = 5; 10 ppm = 6; 50 ppm = 5) were also analyzed. No bones from males were analyzed to reduce sample number and because it was felt that bone lead would not be biologically significant in males whereas in females the deposition of medullary bone and the importance of calcium metabolism during shell formation necessitated an analysis of lead levels in bones of females. Feed samples collected during the study period were pooled into three samples per treatment and analyzed for lead content.

The pooled feed samples were subsampled to provide a single sample for nutrient analysis by Hazleton Raltech, Inc. Analyses according to the Association of Official Analytical Chemists (1980) were as follows: protein, Method 2.057; moisture, Method 16.233; fat, Method 7.058; ash, Method 14.006; crude fiber, Method 7.066; vitamin A, Method 43.008; vitamin D, Methods 43.195 and 43.209. Other analyzed nutrients included carbohydrates (Watt and Merrill 1963; p. 164), calories (Watt and Merrill

**Table 1.** Mean body weights for male and female American kestrels fed 0, 10, or 50 ppm metallic lead in their diet, November 1979–April 1980 (n = 6)

Treatment	Sex	Weight (g) ± standard deviation
0	♂	120.9 ± 6.5
	♀	138.2 ± 9.0
10	♂	120.6 ± 7.8
	♀	137.2 ± 8.9
50	♂	118.7 ± 8.5
	♀	143.9 ± 7.3

1963; p. 159–160), and total vitamin C (Roe and Kuether 1943). Trace elements, including calcium and phosphorus, were determined by the method of Dahlquist and Knoll (1978) after sample preparation according to Method 3.005 of the Association of Official Analytical Chemists (1980).

Data concerning clutch size, interval between eggs, date of first egg laid, and fertility were statistically compared by one-way ANOVA. Fertility data were arc-sine transformed prior to analysis to provide a more normal distribution. Clutch size and fertility were also compared by  $\chi^2$ . Data on shell thickness and Ratcliffe indices were averaged by clutch, then treatment means compared by ANOVA. Nested ANOVA (Winer 1962) was used to determine effects of treatment and egg order on shell thickness and Ratcliffe indices. Residue data were not normally distributed, requiring log transformation of the data prior to statistical testing. One-way ANOVA was then used to evaluate treatment effects. Nested ANOVA also was used to examine the possible effects of egg order on eggshell lead levels. Where significant treatment effects were detected, Tukey's or Bonferroni (Neter and Wasserman 1974) multiple comparison procedures were used to separate means. All tests of significance were done at the 0.05 level.

## Results

Food consumption was similar between treatment groups. Body weights were also similar. Mean weights (Table 1) for males and females for the period November 1979–April 1980 were not significantly different between treatments. Reproductive performance between treatments was virtually identical. Of the 16 pairs per treatment that started the study, one pair from each was lost before egg laying and all pairs but one 10 ppm treatment level pair laid and initiated incubation. Average date of initiation of egg laying, mean number of days between eggs, average clutch size, and percent of eggs fertile were not significantly different among treatments (Table 2). Shell thickness was 0.202 mm and mean Ratcliffe indices varied no more than 1.5% between treatments (Table 3), none of the differences being significant; egg order did not have a significant effect on these values.

All of the egg contents analyzed for lead were

**Table 2.** Mean clutch parameters of American kestrels fed 0, 10, or 50 ppm metallic lead in their diet, November 1979–May 1980

Treatment	n	Clutch initiated (Date $\pm$ S.D.)	Interval between eggs (Days $\pm$ S.D.)	Clutch size (Eggs $\pm$ S.D.)	Fertility (% $\pm$ S.D.)
0	15	April 11 $\pm$ 10.7	1.91 $\pm$ 0.28	4.6 $\pm$ 0.83	65.5 $\pm$ 24.7
10	14	April 8 $\pm$ 9.5	1.90 $\pm$ 0.15	4.9 $\pm$ 0.53	66.6 $\pm$ 27.8
50	15	April 10 $\pm$ 9.4	1.86 $\pm$ 0.21	4.7 $\pm$ 0.59	74.1 $\pm$ 25.4

**Table 3.** Mean shell thickness and Ratcliffe indices for clutches of American kestrel eggs from females fed 0, 10, or 50 ppm metallic lead in their diet

Treatment	n	Shell thickness (mm $\pm$ S.D.)	Ratcliffe index ( $\pm$ S.D.)
0	15	0.202 $\pm$ 0.014	1.057 $\pm$ 0.054
10	14	0.202 $\pm$ 0.013	1.073 $\pm$ 0.045
50	15	0.202 $\pm$ 0.015	1.063 $\pm$ 0.066

**Table 4.** Mean lead levels (ppm, dry weight) in eggshells of clutches from American kestrels fed 0, 10, or 50 ppm metallic lead in their diet

Treatment	n	Shell lead (ppm $\pm$ S.D.)
0	15	1.2 $\pm$ 1.7
10	14	1.7 $\pm$ 2.7
50	15	0.6 $\pm$ 0.3

below the detection limit of 0.5 micrograms. Mean eggshell lead (Table 4) was as high as 1.7 ppm, but the variation was such that no treatment effects were detected. In those clutches in which all shells were analyzed, no effect of egg order was found. Significant differences among treatment levels were found in liver lead (Table 5). Males from the 50 ppm treatment level had three times more lead than in the livers of control males whereas the 50 ppm treatment level females had 7 times more lead than the control females. Lead levels in the livers of the 10 ppm treatment level were elevated but not significantly. Additionally, the liver lead levels in the 50 ppm treatment level females were significantly higher than in the males.

Significant treatment effects were evident in the tibia and humerus lead levels (Table 6). Females from the 50 ppm treatment levels accumulated 3.8 times more lead in their humeri and 19 times more lead in their tibia than control females. Although the 10 ppm treatment level females exhibited a trend towards elevated bone lead, the difference was not significant. The tibia tended to accumulate more lead (Table 6); levels were 4.6 times greater than humerus levels in 50 ppm treatment level females.

Actual lead levels found in the diets are shown in Table 7. Wet weight levels were within 10 percent

**Table 5.** Mean lead levels (ppm, dry weight) in livers of male and female American kestrels fed 0, 10, or 50 ppm metallic lead in their diet

Treatment	n	Males (ppm $\pm$ S.D.)		Females (ppm $\pm$ S.D.)	
		n	(ppm $\pm$ S.D.)	n	(ppm $\pm$ S.D.)
0	15	0.43 $\pm$ 0.09A*	15	0.33 $\pm$ 0.05A	
10	15	0.67 $\pm$ 0.76A	15	0.76 $\pm$ 0.65A	
50	16	1.3 $\pm$ 1.00B	14	2.4 $\pm$ 1.40C	

\* Means which do not have a letter in common were significantly different ( $p < 0.05$ )

**Table 6.** Mean lead levels (ppm, dry weight) in humeri and tibiae of American kestrel females fed 0, 10, or 50 ppm metallic lead in their diet

Treatment	n	Humeri (ppm $\pm$ S.D.)		Tibiae (ppm $\pm$ S.D.)	
		n	(ppm $\pm$ S.D.)	n	(ppm $\pm$ S.D.)
0	5	3.5 $\pm$ 2.4A*	3.2 $\pm$ 2.4A		
10	6	4.4 $\pm$ 1.6A	9.1 $\pm$ 6.0A		
50	5	13.5 $\pm$ 5.2B	62.0 $\pm$ 25.0B		

\* Within a column, means which do not have a letter in common were significantly different ( $p < 0.05$ )

of projected levels. Nutrient content (Table 8) and trace elements (Table 9) are provided as background information. The calcium/phosphorus ratio was 1.6/1.0.

## Discussion

No detectable reproductive effects associated with the lead ingested by the American kestrels in this study were found. Food consumption and body weights were similar between treatment groups throughout the study. Significant lead exposure had occurred since Franson *et al.* (1983) found that the same birds as in this study exhibited a significant depression in delta-aminolevulinic acid dehydratase (ALAD) activity of 61% in the 10 ppm treatment group and 80% in the 50 ppm treatment group when sacrificed. As in the measurements reported here, no other adverse effects were evident. Dosing mourning doves (*Zenaidura macroura*) and ringed turtle doves (*Streptopelia risoria*) with lead shot resulted in a 87-90% inhibition of ALAD (Kendall and

**Table 7.** Mean lead levels (ppm, wet weight and dry weight) in diets fed to American kestrels, November 1979–May 1980

Treatment	n	Lead, wet weight (ppm ± S.D.)	Lead, dry weight (ppm ± S.D.)
0	3	0.11 ± 0.03	0.23 ± 0.04
10	3	9.7 ± 0.12	19.80 ± 0.66
50	3	54.0 ± 0.25	104.40 ± 5.60

**Table 8.** Nutritional analysis of the diet given American kestrels, November 1979–June 1980

Protein (%)	19.1
Moisture (%)	56.7
Fat (%)	9.4
Ash (%)	5.4
Crude fiber (%)	0.6
Carbohydrates (%)	8.8
Calories (per 100 g)	196.0
Vitamin A (IU/100 g)	560.0
Vitamin C (mg/100 g)	>1.5
Vitamin D (IU/100 g)	<18.

Scanlon 1982b; Kendall *et al.*, 1982). The lead powder seemingly mimicked lead shot exposure and biologically-incorporated lead relatively well. Bone and liver lead levels were higher than in American kestrels dosed for 60 days with lead shot or homogenized, lead-poisoned ducks (29.3 ppm lead, wet weight) (Stendell 1980) but egg contents were below those reported in American kestrels by Lincer and McDuffie (1974). Their level of sensitivity for lead was lower than in this study. Damron and Wilson (1975) found lead acetate and lead shot to be more toxic to bobwhites (*Colinus virginianus*) than lead powder. Liver and bone lead levels were lower than reported in doves dosed with lead shot (Kendall and Scanlon 1982b; Kendall *et al.* 1982), suggesting a lower rate of uptake of lead powder in kestrels compared to lead shot in doves. However, under the experimental conditions reported here, it is felt that a realistic exposure level was obtained.

No detectable shell thinning occurred. The correlation between lead in the shells and shell thickness in European kestrels reported by Grandjean (1976) could not be confirmed in American kestrels. Lincer and McDuffie (1974) also failed to find any relationship between lead levels and shell thickness in American kestrels. The sample utilized by Grandjean (1976) was a biased one because it represented unsuccessful eggs rather than a random sample from the population; the organochlorine residues reported also were biased by discarding the embryos prior to analysis. The correlations reported were weak ( $R^2 = 31\text{--}35\%$ ), not surprising

**Table 9.** Trace element analysis (mg/100 g) of the diet given American kestrels, November 1979–June 1980

Calcium	1470.0
Phosphorus	900.4
Magnesium	53.4
Sodium	231.6
Aluminum	3.66
Barium	0.93
Iron	13.6
Strontium	3.0
Boron	0.14
Copper	0.46
Zinc	8.0
Manganese	3.0
Chromium	0.054

considering the natural variation in eggshell thickness and lead residues and the variation in measuring these factors. No correlations between shell lead and shell thickness existed in this study and shells from the 50 ppm treatment level were as thick as those from control birds. The evidence strongly suggests that lead does not contribute significantly to eggshell thinning and the correlation between shell thickness and lead reported by Grandjean (1976) was spurious.

Liver lead levels approximated those reported as background levels in other species. The 10 ppm treatment level birds had liver lead levels similar to those reported by Maedgen *et al.* (1982) in royal terns (*Thalasseus maximus*) and Sandwich terns (*T. sandvicensis*) and exceeded those of meadowlarks (*Sturnella neglecta* and *S. Magna*) associated with an interstate highway in Kansas (Udevitz *et al.* 1980). The higher liver lead levels in the 50 ppm treatment group were lower than those reported in the livers of laughing gulls (*Larus atricilla*) from Texas (Munoz *et al.* 1976) or pigeons from urban environments (Hutton and Goodman 1980; Johnson *et al.* 1982), more resembling the rural songbirds examined by Getz *et al.* (1977b) and the rural pigeons examined by Johnson *et al.* 1982 or Kendall and Scanlon 1982a. In contrast to these studies, bone lead levels in these kestrels were low or intermediate. These tissue lead levels probably reflect the relatively short exposure time (5–7 months) but do indicate the test was a valid one for chronic exposure. None of the earlier referenced field studies found any adverse effects associated with the lead exposure in the species they examined.

Although bone lead levels were not compared between sexes, Finley *et al.* (1976) and Hutton and Goodman (1980) found differences between males and females that they associated with differential calcium absorption during eggshell formation.

Hutton and Goodman (1980) speculated that intestinal lead absorption might also be greater at this time. This study appears to confirm this hypothesis, since female liver lead levels were higher than the comparable males, and the tibia (a major storage site of medullary bone) was considerably higher in lead content than the humerus (a minor medullary bone storage site). Differential absorption and deposition of lead was evidently occurring. However, little of this lead was transferred to the eggshell or egg contents and there was no apparent effect on eggshell formation.

The kind of chronic, sublethal lead exposure this study attempted to duplicate is undoubtedly common. Cases of lead shot poisoning in raptors exist and lead poisoning is an important source of mortality in bald eagles (Pattee *et al.* 1981); nine of 168 dead bald eagles (5.4%) examined 1975–1977 died of lead poisoning (Kaiser *et al.* 1980). Excluding outright mortality, the results of the present study suggest that the typical chronic lead exposure most raptors receive is unlikely to cause reproductive problems through the initiation of incubation nor is lead a major cause of shell thinning under normal conditions. Adverse effects associated with hatching, growth and survival of young, or long-term exposure were not investigated. Effects of lead, such as shell thinning, might occur if the diet was nutritionally inadequate but would probably be minor when compared to the effects of poor nutrition.

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## References

- Association of Official Analytical Chemists*: Official methods of analysis of the Association of Official Analytical Chemists. 13 ed. Washington, DC (1980).
- Bellrose, F. C.: Lead poisoning as a mortality factor in waterfowl populations. *Illinois Nat. Hist. Serv. Bull.* **27**, 235 (1959).
- Benson, W. W., B. Pharoah, and P. Miller: Lead poisoning in a bird of prey. *Bull. Environ. Contam. Toxicol.* **11**, 105 (1974).
- Corrin, M. L., and D. F. S. Natusch: Physical and chemical characteristics of environmental lead. In W. R. Boggess and B. G. Wixson (eds.): *Lead in the environment*. p. 7. Washington, DC: National Science Foundation (1977).
- Dahlquist, R. L., and J. W. Knoll: Inductively coupled plasma-atomic emission spectrometry analysis of biological materials and soils for major trace and ultra-trace elements. *Appl. Spectros.* **32**, 1 (1978).
- Damron, B. L., and H. R. Wilson: Lead toxicity of bobwhite quail. *Bull. Environ. Contam. Toxicol.* **14**, 489 (1975).
- Edens, F. W., E. Benton, S. J. Bursian, and G. W. Morgan: Effect of dietary lead on reproductive performance in Japanese quail, *Coturnix coturnix japonica*. *Toxicol. Appl. Pharmacol.* **38**, 307 (1976).
- Finley, M. T., M. P. Dieter, and L. N. Locke: Lead in tissues of mallard ducks dosed with two types of lead shot. *Bull. Environ. Contam. Toxicol.* **16**, 261 (1976).
- Franson, J. C., L. Sileo, O. H. Pattee, and J. F. Moore: Effects of chronic dietary lead in American kestrels (*Falco sparverius*). *J. Wildl. Dis.* **19**, 110 (1983).
- Getz, L. L., L. Verner, and M. Prather: Lead concentrations in small mammals living near highways. *Environ. Pollut.* **13**, 151 (1977a).
- Getz, L. L., L. B. Best, and M. Prather: Lead in urban and rural song birds. *Environ. Pollut.* **12**, 236 (1977b).
- Goldsmith, C. D., Jr., and P. F. Scanlon: Lead levels in small mammals and selected invertebrates associated with highways of different traffic densities. *Bull. Environ. Contam. Toxicol.* **17**, 311 (1977).
- Grandjean, P.: Possible effect of lead on egg-shell thickness in kestrels 1874–1974. *Bull. Environ. Contam. Toxicol.* **16**, 101 (1976).
- Grue, C. E., D. J. Hoffman, and T. J. O'Shea: Lead exposure and reproduction in highway-nesting barn swallows. *Condor* (in press).
- Hutton, M., and G. T. Goodman: Metal contamination of feral pigeons *Columba livia* from the London area: Part 1—Tissue accumulation of lead, cadmium, and zinc. *Environ. Pollut. Ser. A* **22**, 207 (1980).
- Jacobson, E., J. W. Carpenter, and M. Novilla: Suspected lead toxicosis in a bald eagle. *J. Am. Vet. Med. Assoc.* **171**, 952 (1977).
- Johnson, M. S., H. Pluck, M. Hutton, and G. Moore: Accumulation and renal effects of lead in urban populations of feral pigeons, *Columba livia*. *Arch. Environ. Contam. Toxicol.* **11**, 761 (1982).
- 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. W. Swineford: Organochlorine pesticides, PCB, and PBB residues and necropsy data for bald eagles from 29 states—1975–77. *Pestic. Monit. J.* **14**, 145 (1980).
- Kendall, R. J., and P. F. Scanlon: Tissue lead concentrations and blood characteristics of rock doves from an urban setting in Virginia. *Arch. Environ. Contam. Toxicol.* **11**, 265 (1982a).
- : Tissue lead concentrations and blood characteristics of mourning doves from southwestern Virginia. *Arch. Environ. Contam. Toxicol.* **11**, 269 (1982b).
- Kendall, R. J., P. F. Scanlon, and R. T. DiGiulio: Toxicology of ingested lead shot in ringed turtle doves. *Arch. Environ. Contam. Toxicol.* **11**, 259 (1982).
- Lincer, J. L., and B. McDuffie: Heavy metal residues in the eggs of wild American kestrels (*Falco sparverius* Linn.). *Bull. Environ. Contam. Toxicol.* **12**, 227 (1974).
- Locke, L. N., G. E. Bagley, D. N. Frickie, and L. T. Young: Lead poisoning and aspergillosis in an Andean condor. *J. Am. Vet. Med. Assoc.* **155**, 1052 (1969).
- Maedgen, J. L., C. S. Hacker, G. D. Schroder, and F. W. Weir: Bioaccumulation of lead and cadmium in the royal tern and sandwich tern. *Arch. Environ. Contam. Toxicol.* **11**, 99 (1982).

- McConnell, C. A.: Experimental lead poisoning of bobwhite quail and mourning doves. *Proc. S. E. Assoc. Fish and Game Comm.* **21**, 208 (1967).
- Morgan, G. W., F. W. Edens, P. Thaxton, and C. R. Parkhurst: Toxicity of dietary lead in Japanese quail. *Poultry Sci.* **54**, 1636 (1975).
- Munoz, R. V., Jr., C. S. Hacker, and T. F. Gesell: Environmentally acquired lead in the laughing gull, *Larus atricilla*. *J. Wildl. Dis.* **12**, 139 (1976).
- Neter, J., and W. Wasserman: Applied linear statistical methods. Homewood: Richard D. Irwin (1974).
- Pattee, O. H., S. N. Wiemeyer, B. M. Mulhern, L. Sileo, and J. W. Carpenter: Experimental lead-shot poisoning in bald eagles. *J. Wildl. Manage.* **45**, 806 (1981).
- Porter, R. D., and S. N. Wiemeyer: Propagation of captive American kestrels. *J. Wildl. Manage.* **34**, 594 (1970).
- Ratcliffe, D. A.: Decrease in eggshell weight in certain birds of prey. *Nature* **215**, 208 (1967).
- Roe, J. H., and C. A. Kuether: The determination of ascorbic acid in whole blood and urine through the 2,4-dinitrophenylhydrazine derivative of dehydroascorbic acid. *J. Biol. Chem.* **147**, 399 (1943).
- Stendell, R. C.: Dietary exposure of kestrels to lead. *J. Wildl. Manage.* **44**, 527 (1980).
- Stone, C., and J. H. Soares, Jr.: Studies on the metabolism of lead in Japanese quail. *Poultry Sci.* **53**, 1982 (1974).
- Udevitz, M. S., C. A. Howard, R. J. Robel, and B. Curnutte, Jr.: Lead contamination in insects and birds near an interstate highway. *Kansas Environ. Entomol.* **9**, 35 (1980).
- Watt, B. K., and A. L. Merrill: Composition of foods. Agriculture handbook No. 8. Washington, DC: U.S. Government Printing Office (1963).
- Williamson, P., and P. R. Evans: Lead: Levels in roadside invertebrates and small mammals. *Bull. Environ. Contam. Toxicol.* **8**, 280 (1972).
- Winer, B. J.: Statistical principles in experimental design. New York: McGraw-Hill (1962).

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