BioOne COMPLETE

EFFECTS OF LEAD SHOT INGESTION ON δ-AMINOLEVULINIC ACID DEHYDRATASE ACTIVITY, HEMOGLOBIN CONCENTRATION, AND SERUM CHEMISTRY IN BALD EAGLES

Authors: HOFFMAN, DAVID J., PATTEE, OLIVER H., WIEMEYER, STANLEY N., and MULHERN, BERNARD

Source: Journal of Wildlife Diseases, 17(3) : 423-431

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-17.3.423

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

EFFECTS OF LEAD SHOT INGESTION ON δ-AMINOLEVULINIC ACID DEHYDRATASE ACTIVITY, HEMOGLOBIN CONCENTRATION, AND SERUM CHEMISTRY IN BALD EAGLES

DAVID J. HOFFMAN, OLIVER H. PATTEE, STANLEY N. WIEMEYER and BERNARD MULHERN, U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20811, USA.

Abstract: Lead shot ingestion by bald eagles *(Haliaeetus leucocephalus)* is considered to be widespread and has been implicated in the death of eagles in nature. It was recently demonstrated under experimental conditions that ingestion of as few as 10 lead shot resulted in death within 12 to 20 days. In the present study hematological responses to lead toxicity including red blood cell ALAD activity, hemoglobin concentration and 23 different blood serum chemistries were examined in five captive bald eagles that were unsuitable for rehabilitation and release. Eagles were dosed by force-feeding with 10 lead shot; they were redosed if regurgitation occurred. Red blood cell ALAD activity was inhibited by nearly 80% within 24 hours when mean blood lead concentration had increased to 0.8 parts per million (ppm). By the end of 1 week there was a significant decrease (20-25%) in hematocrit and hemoglobin, and the mean blood lead concentration was over 3 ppm. Within as little as 1-2 weeks after dosing, significant elevations in serum creatinine and serum alanine aminotransferase occurred, as well as a significant decrease in the ratio of serum aspartic aminotransferase to serum alanine aminotransferase. The mean blood lead concentration was over 5 ppm by the end of 2 weeks. These changes in serum chemistry may be indicative of kidney and liver alterations.

INTRODUCTION

There is increasing concern that ingestion of lead shot may pose a substantial threat to bald eagles *(Haliaeetus leucocephalus)*. Since eagles frequently prey upon weak, moribund or dead animals, they have a high risk of exposure to hunter-crippled game or lead shot-poisoned waterfowl. Documentation of lead shot poisoning in waterfowl is quite extensive.[6,7,37] Lead shot poisoning also has been reported in ring-necked pheasants *(Phasianus colchicus)*,[24] bobwhite quail *(Colinus virginianus)*,[17] and mourning doves *(Zenaidura macroura)*.[26] Eagles have been reported to be attracted to waterfowl concentration areas including national and state refuges. Dunstan[16] found that 50-60% of the bald eagle castings collected from two midwestern refuges contained lead shot. Lead shot poisoning in bald eagles has been accompanied by elevated tissue lead levels and occasionally by shot in the digestive tract.[24,25,29] Nine of 168 dead or dying bald eagles examined in 1975-77 appeared to have died from lead poisoning and had liver lead levels of 23-38 parts per million (ppm); lead shot was found in the digestive system of three of these.[25]

ALAD (δ-aminolevulinic acid dehydratase) is an essential enzyme in the biosynthetic pathway of heme synthesis and is required to maintain hemoglobin content in erythrocytes. Inhibition of red blood cell ALAD has become accepted as a standard bioassay to detect acute and chronic lead exposure in humans,[22] in other mammals,[28] and in birds.[13,30] Anemia is a primary sign of lead toxicity in mammals[36] and has been

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

detected after lead poisoning in avian species.[11,27]

Pattee *et al.*[31] reported tissue levels of lead, as well as histopathological lesions, in captive bald eagles following acute experimental poisoning of captive bald eagles by lead shot ingestion. The number of lead shot ingested, the total milligrams of lead shot eroded and the number of days following ingestion until death were reported by Pattee *et al.*[31] They showed the days until death ranged from 10 to over 100; the median was 20 days. Retention time for shot ranged from 0.5 to 48 days. At least one shot was found in the stomach of each bird at death. Lead levels in birds at death averaged 16.6 ppm in the liver and 6.0 ppm in the kidney. Renal, cardiovascular, and liver lesions were found upon histopathological examination; renal lesions were the most notable.

The present investigation was conducted as a companion study to the above and reports the toxic effects of lead on red blood cell ALAD activity and hemoglobin concentration, as well as the effects on blood serum chemistries in the same eagles.

MATERIALS AND METHODS

Six bald eagles were selected from birds held at the Patuxent Wildlife Research Center at Laurel, Maryland. All of these eagles were unsuitable for rehabilitation and release, or for captive breeding since most had sustained permanent wing damage. The eagles were otherwise healthy and in good condition. Birds were placed individually in wire mesh cages (3 m × 3 m × 1.9 m) elevated over a concrete slab. Each pen was provided with a log for perching and a large pan of water. Birds generally were acclimated to the pens for 1 week or more before treatment and were maintained on a fish diet.

Treatments were replicated in time using no more than two birds at any one time. In this manner 5 of the 6 eagles served first as untreated controls at one time or another and then served as treated controls afterwards to permit a total of 5 controls and 5 treated birds. The 5 that received lead shot were examined by necropsy at death and the 6th that was not dosed was examined at necropsy after euthanization as a control bird, to provide tissues for the histopathological and the lead analysis portion of this study which has been reported by Pattee *et al.*[31] Each bird was weighed, examined, and radiographed before dosing to inspect for any preexisting shot. Treatment consisted of dosing with 10 #4 lead shot which were preweighed and sorted to insure that all shot in a dose were within 0.5 mg of each other. Dosing was conducted by inserting the shot down the throats of small smelt (95-120 mm total length) and force-feeding the smelt to the eagles. The cement slab under each pen was searched daily for regurgitated shot. If all shot were regurgitated, the eagle was redosed within 48 h. Birds also were radiographed weekly or sooner if most of the shot were regurgitated. This was done to confirm the presence or absence of shot prior to additional dosing. If all shot were regurgitated, the eagle was redosed within 48 h.

Blood samples were obtained from the brachial veins of birds before initial dosing (a 2 cc ammonium heparinized sample and a 5 cc unheparinized sample) and at 1, 3, 7, and 14 days following dosing (2 cc ammonium heparinized sample). A 5 cc sample was collected after birds appeared to be weakened from exposure to lead, appearing lethargic with decreased appetite. The timing of this sample for individual birds was 7, 7, 8, 93 and 121 days after dosing. Heparinized blood was used for the determination of hematocrit, hemoglobin concentration, red blood cell ALAD activity and lead concentration. We measured hematocrit in duplicate by the microhematocrit method using an International micro-capillary centrifuge at 7,500 rpm for 5 min. Hemoglobin was

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

determined in duplicate by the cyanmethemoglobin method. Red blood cell ALAD (EC 4.2.1.24) activity was determined with 0.1 ml aliquots in duplicate;[10] unit activity was defined as an increase in absorbance at 555 nm of 0.100 with a 1.0 cm light path/ml erythrocytes/hour at 38 C.

The remaining heparinized blood, about 1.5 cc, was frozen and analyzed for lead by dry ashing of the sample followed by dissolving the ash in acid. The lead in this solution was subsequently quantified by flame atomic absorption spectrophotometry with a Perkin Elmer model 703-AAS equipped with a deuterium arc background corrector, an AS-50 autosampler, and a PRS-10 printer. The lower limit of quantification for lead residues was 0.1 ppm.[31] Unheparinized blood samples were placed in tubes without additives and permitted to stand at room temperature for about 30 min to insure adequate clotting, centrifuged at 3000 rpm for 10 min and the serum removed. Serum samples were frozen at -80 C and then transported to a commercial laboratory (Vet Path, Bethesda, Maryland) at a later date for determination of 23 serum chemistry values on a computer process-controlled "autochemist" (Autochem AB, Broma, Sweden). These serum chemistries were: glucose, creatinine, blood urea nitrogen, uric acid, total bilirubin, direct bilirubin, total protein, albumin, globulin, total lipids, cholesterol, triglycerides, calcium, phosphorous, sodium, potassium, chloride, iron, alkaline phosphatase, alanine aminotransferase (ALT), aspartate aminotransferase (AST), δ - glutamyl transpeptidase, and lactic dehydrogenase. The methodologies for all serum chemistry determinations were described by Gee *et al.*[18] Hematocrit, hemoglobin, and red blood cell ALAD activity were compared by one way analysis of variance and Duncan's multiple range test. Serum chemistries between dosed and control birds were compared by Student's t test.

RESULTS

The red blood cell ALAD activity was depressed by nearly 80% compared with predose values or with the undosed controls analyzed concurrently (Table 1). Depression of ALAD activity occurred within 24 h of lead shot ingestion; and the blood lead concentration rose from less than 0.1 ppm to a mean concentration of 0.8 ppm. Blood lead levels continued to rise following treatment. By the end of 1 week there was a significant decrease of 20-25% in both hematocrit and hemoglobin concentration. These effects became more apparent in dosed eagles that survived for longer than 2 weeks. The number of days from initial dosing until death were 10, 12, 20, 125, and 133. The specific blood chemistries that were affected following lead dosage (Table 2) included a small but highly significant elevation ($P<0.01$) of 13% in serum glucose and a significant elevation ($P<0.05$) of nearly 40% in serum creatinine. An increased albumin to globulin ratio was aparent and approached the significant level but was not significant ($0.10>P>0.05$). Serum ALT activity was significantly elevated by nearly 50%; and the ratio of serum AST to ALT decreased by about 30% ($P<0.05$).

DISCUSSION

Ingestion of 10 #4 lead shot by bald eagles resulted in an elevated mean blood lead concentration to 0.8 ppm within 24 h and of over 3 ppm within one week. Equally rapid increments in blood lead concentration have been reported in ducks and geese following lead shot ingestion. Finley *et. al.*[17] reported a blood lead concentration of over 1 ppm within 1 day of ingestion of one #4 lead shot in mallards *(Anas platyrhynchos)* and a concentration of greater than 2 ppm within 1 week. Cook and Trainer[12] reported a blood lead concentration of over 4 ppm within 3 days of ingestion of five #4 lead shot in Canada geese *(Branta*

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

TABLE 1. Erythrocyte ALAD, blood lead, hematocrit and hemoglobin values in bald eagles.

Parameter (Units)	Treatment (N=5)	Number of Days after Treatment 0[a]	1	3	7	14[b]
ALAD	Controls	188.3 (12.5)[c]	185.5 (13.7)	191.3 (17.2)	198.2 (13.1)	208 (23.0)
(Units)	Lead-dosed	186.0 (13.9)	38.0[d] (12.9)	53.8[d] (14.5)	41.6[d] (14.5)	49.7[d] (24.8)
Lead. Conc.[e]	Controls	ND	ND	ND	ND	ND
(ppm)	Lead-dosed	ND	0.8 (0.6)	2.8 (1.3)	3.4 (1.4)	5.4 (4.3)
Hematocrit	Controls	45.0 (1.6)	44.5 (1.7)	45.0 (2.1)	43.0 (1.6)	44.0 (2.7)
(%)	Lead-dosed	43.0 (1.3)	41.0 (1.2)	40.0 (2.0)	34.0[d] (2.2)	32.0[d] (4.2)
Hemoglobin	Controls	15.3 (0.6)	15.0 (0.5)	14.6 (0.8)	14.5 (0.5)	14.3 (1.2)
(g/dl)	Lead-dosed	14.5 (0.5)	13.5 (0.3)	13.4 (1.1)	11.0[d] (0.8)	10.7[d] (1.2)

[a] Predose

[b] Two of five eagles died by this time, at 10 and 12 days after treatment.

[c] Mean (standard error)

[d] Differed significantly from the predose value and from the untreated controls by one-way analysis of variance ($P<0.01$) and Duncan's multiple range test ($P<0.05$).

[e] Lead was not detected (ND) in untreated eagles. The limit of quantification was 0.1 ppm.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

TABLE 2. Blood serum chemistries affected by lead shot ingestion in bald eagles.

Parameter (units)	Controls (N=5)	Lead-dosed (N=5)	Laboratory Method
Glucose (g%)	315.5 (4.0)[a]	356.0 (9.6)[b]	Oxidase peroxidase method
Creatinine (mg%)	0.8 (0.09)	1.1 (0.1)[c]	Kinetic Jaffe reaction
Albumin/Globulin ratio	0.57 (0.04)	0.63 (0.01)[d]	BCG method
Alanine aminotransferase (IU/l)	68.3 (7.9)	100.8 (15.6)[c]	Modified Reitman-Frankel Technique
Aspartate aminotransferase (IU/l)	247.1 (36.1)	255.0 (41.3)	Modified Reitman-Frankel Technique
Aspartatate aminotransferase/ Alanine aminotransferase ratio	3.62 (0.18)	2.53 (0.33)[c]	Modified Reitman-Frankel Technique

[a]Mean (standard error). Samples were collected as indicated in the Materials and Methods section.
[b]t-test, $P<0.01$
[c]t-test, $p<0.05$
[d]t-test, not significant but approached the significant level, $0.05<P<0.10$.

canadensis). With blood lead concentrations in this range, an inhibition of nearly 80% in the red blood cell ALAD activity was not surprising. Significant negative correlations have been reported between ALAD activities and lead concentration in ducks where only 0.2 ppm lead in the blood resulted in greater than 50% inhibition of ALAD activity.[13,17] Retention of lead shot for at least 24 h resulted in abnormal ALAD activity for 4 weeks.[13,17] The sensitivity of this enzyme to lead exposure has been demonstrated in other species of birds including pigeons *(Columba livia),*[30] canvasback ducks *(Aythya valisineria),*[11] quail *(Coturnix coturnix),*[34] wood thrushes *(Hylocichla mustelina)* (Hoffman, unpubl. data) and barn swallows *(Hirundo rustica)* (Hoffman, unpubl. data). It is of some interest that in spite of species diversity, healthy adult birds examined so far have shown ALAD activities within a relatively narrow range whereas the activity range in different species of adult mammals is rather dissimilar.[1] This class difference may be because avian erythrocytes are nucleated and have a generally higher metabolic activity than mammalian erythrocytes. The degree of ALAD activity in mammals has been correlated with the percentage of reticulocytes in the circulation, as well as the plasma zinc content, which are species dependent.[1] Inhibition of ALAD activity in birds may have more severe effects than in mammals since the more metabolically active nucleated red blood cells of birds require porphyrin synthesis not only for hemoglobin production, but also for production of respiratory heme containing enzymes.[2,8] Lead toxicity also has been linked to inhibition of this enzyme in the liver and brain of both birds and mammals, and therefore probably affects critical processes, such as synthesis of protoporphyrins for incorporation into cytochromes which are needed to support detoxication in the liver.[9,13,20]

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

Blood lead concentrations of greater than 0.8 ppm in humans have been generally associated with anemia,[19] and a decrease in hemoglobin concentration of about 10% or greater is considered supportive of clinical diagnosis of lead poisoning.[5] Lead ingestion of 8 mg/kg as lead nitrate in the diet of mallards resulted in up to a 66% decrease in the erythrocyte count after 6 days with a corresponding decrease in hemoglobin concentration.[11] Young Japanese quail *(Coturnix coturnix)* receiving 500 ppm lead acetate in the diet developed significant anemia with decreased hemoglobin within several weeks.[27] These findings are in agreement with the anemia we have reported in eagles.

Most of the alterations of serum chemistries we reported following lead ingestion were not great, but nevertheless represented significant differences. Serum creatinine has been considered useful in the diagnosis of mammalian renal diseases since with kidney dysfunction creatinine is retained in the blood.[4] Although the avian kidney normally secretes small amounts of creatinine compared with the mammalian kidney, it has been shown to be highly capable of secreting creatinine when the creatinine level is elevated by intravenous infusion of creatinine.[1] This would suggest that the significant elevation in creatinine in the eagles might have been representative of the renal lesions reported by Pattee *et al.*[31] Alterations in the albumin/globulin ratio may be indicative of liver or kidney damage in mammals.[4] The chief application of ALT (GPT) determination in mammals has been in the diagnosis of hepatocellular destruction.[21] With acute injury to the liver in mammals, ALT (GPT) values usually are as high or higher than AST (GOT).[38] Dieter and Wiemeyer[15] reported elevated plasma AST and ALT values in bald eagles following an acute dosage of dieldrin, known to be hepatotoxic. Tissue ALT activities are nearly five times higher in the kidney of untreated ducks than in the liver,[32] suggesting that elevated ALT values might reflect some renal damage as well as liver damage in birds. Rozman *et al.*[32] reported that lead shot poisoning in ducks produced effects that were similar to what we reported in eagles; these included a significant elevation in ALT but not in AST. Sileo *et al.*[33] reported significantly elevated AST and myocardial necrosis in geese without a significant elevation in ALT after lead shot ingestion. At death histopathological lesions in the eagles were more apparent than alterations in serum chemistries and consisted of mainly renal tubular degeneration (nephrosis) with some glomerulonephritis; cardiovascular lesions and hepatic necrosis were also apparent.[31] Alterations in serum chemistries probably are dependent on the time of serum sampling between dosing and death. Serum was collected when birds first appeared weakened, appearing lethargic with decreased appetite; this was usually at a time greater than half way between the time of dosing and death. In retrospect, it is possible that subsequent sampling in some instances at a time closer to death but before the eagles were in a moribund state, might have revealed more severe biochemical lesions.

Acknowledgements

The authors would like to thank J.W. Carpenter for the radiography and assistance in procuring serum samples. We acknowledge the able assistance of K. Babcock, R. Edwards, R. Epperson, H. Hankin, K. McGarigal, and M. Weber for handling and care of the birds. We thank L.J. Thomas for her excellent technical assistance and L. Sileo and E.H. Dustman who reviewed this manuscript.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

LITERATURE CITED

1. ABDULLA, M., S. SVENSSON, B. HAEGER-ARONSEN, A. MATHUR and K. WALLENIUS. 1978. Effect of age and diet on delta-aminolevulinic acid dehydratase in red blood cells. Enzyme 23: 170-175.
2. ALLEN, R.L. 1971. *Physiology and Biochemistry of the Domestic Fowl.* pp. 873-881. Academic Press, New York.
3. ANNINO, J.S. 1964. *Clinical Chemistry: Principles and Procedures.* pp. 173-178. Little, Brown and Co., Boston.
4. AUSTIC, R.E. and R.K. COLE. 1974. Specificity of the renal transport inpairment in chickens having hyperuricemia and articular gout. Proc. Soc. Exp. Biol. Med. 146: 931-935.
5. BELISLES, R.P. 1975. *Toxicology and The Basic Science of Poisons.* L.J. Casarett and J. Doull, eds. pp. 477-481. Macmillan Publishing Co., Inc., New York.
6. BELLROSE, F.C. 1959. Lead poisoning as a mortality factor in waterfowl populations. Illinois Nat. Hist. Serv. Bull. 27: 235-288.
7. BELLROSE, F.C. 1964. Spent shot and waterfowl poisoning. In: *Waterfowl Tomorrow,* J. Linduska, ed. pp. 479-485, U.S. Govt. Printing Office, Washington, D.C.
8. BRACE, K. and P.D. ALTLAND. 1956. Life span of the duck and chicken erythrocyte as determined with C^{11}. Proc. Soc. Exp. Biol. Med. 92: 615-617.
9. BUCHET, J.T., H. ROELS, G. HUBERMONT and R. LAUWERYS. 1976. Effect of lead on some parameters of the heme biosynthetic pathway in rat tissues *in vivo.* Toxicology 6: 21-34.
10. BURCH, H.B. and A.L. SIEGEL. 1971. Improved method for measurement of delta-aminolevulinic acid dehydratase activity of human erythrocytes. Clin. Chem. 17: 1038-1041.
11. COBURN, D.R., D.W. METZLER and R. TREICHLER. 1951. A study of absorption and retention of lead in wild waterfowl in relation to clinical evidence of lead poisoning. J. Wildl. Manage. 15: 186-192.
12. COOK, R.S. and D.O. TRAINER. 1966. Experimental lead poisoning of Canada geese. J. Wildl. Manage. 30: 1-8.
13. DIETER, M.P. and M.T. FINLEY. 1979. δ-aminolevulinic acid dehydratase enzyme activity in blood, brain, and liver of lead-dosed ducks. Environ. Res. 19: 127-135.
14. ——, M.C. PERRY and B.M. MULHERN. 1976. Lead and PCB's in canvasback ducks: relationship between enzyme levels and residues in blood. Arch. Environ. Contam. Toxicol. 5: 1-13.
15. —— and S.N. WIEMEYER. 1978. Six different plasma enzymes in bald eagles *(Haliaeetus leucocephalus)* and their usefulness in pathological diagnosis. Comp. Biochem. Physiol. 61C: 153-155.
16. DUNSTAN, T.C. 1974. Our Eagles' Future. Proceedings of the Bald Eagle Days, pp. 62-67, Eagle Valley Environmentalists, Apple River, Illinois.
17. FINLEY, M.T., M.P. DIETER and L.N. LOCKE. 1976. δ-aminolevulinic acid dehydratase: Inhibition in ducks dosed with lead shot. Environ. Res. 12: 243-249.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

18. GEE, G.F., J.W. CARPENTER and G.L. HENSLER. 1981. Species differences in hematological values of captive cranes, geese, raptors, and quail. J. Wildl. Manage. 45 (2): (in press).

19. GOYER, R.A. and P. MUSHAK. 1977. *Advances in Modern Toxicology,* vol. 2: *Toxicology of Trace Elements.* R.A. Goyer and M.A. Mehlman, eds. pp. 41-71. Hemisphere Publishing Corp., John Wiley and Sons, New York.

20. HAMMOND, P.B. 1973. The relationship between inhibition of δ-aminolevulinic acid dehydratase by lead and lead mobilization by ethylene diamine tetracetate (EDTA). Toxicol. Appl. Pharmacol. 26: 466-475.

21. HENRY, J.B. 1979. *Clinical Diagnosis and Management by Laboratory Methods.* pp. 361-362. W.B. Saunders and Co., Philadelphia.

22. HERNBERG, S., J. NIKKANEN, G. MELLIN and H. LILIUS. 1970. Delta-aminolevulinic acid dehydratase as a measure of lead exposure. Arch. Environ. Health 21: 140-145.

23. HUNTER, B.F. and M. ROSEN. 1965. Occurrence of lead poisoning in a wild pheasant *(Phasianus colchicus).* California Fish and Game 51: 207.

24. JACOBSON, E., J.W. CARPENTER and M. NOVILLA. 1977. Suspected lead toxicosis in a bald eagle. J. Am. vet. med. Ass. 171: 952-954.

25. KAISER, T.E., W.L. REICHEL, L.N. LOCKE, E. CROMARTIE, A.J. KRYNITSKY, T.G. LAMONT, B.M. MULHERN, R.M. PROUTY, G.J. STAFFORD and D.M. SWINEFORD. 1980. Organochlorine pesticide, PCB, and PBB residues and necropsy data for bald eagles from 29 states, 1975-77. Pestic. Monit. J. 13: 145-149.

26. LOCKE, L.N. and G.E. BAGLEY. 1967. Lead poisoning in a sample of Maryland mourning doves. J. Wildl. Manage. 31: 515-518.

27. MORGAN, G.W., F.W. EDENS, P. THAXTON and C.R. PARKHURST. 1975. Toxicity of dietary lead in Japanese quail. Poultry Sci. 54: 1636-1642.

28. MOUW, D., K. KALITIS, M. ANVER, J. SCHWARTZ, A. CONSTON, R. HARTUNG, B. COHEN and D. RINGLER. 1975. Lead: Possible toxicity in urban vs rural rats. Arch. Environ. Health 30: 276-280.

29. MULHERN, B.M., W.L. REICHEL, L.N. LOCKE, T.G. LAMONT, A. BELISLE, E. CROMARTIE, G.E. BAGLEY and R.M. PROUTY. 1970. Organochlorine residues and autopsy data from bald eagles. Pestic. Monit. J. 4: 141-144.

30. OHI, G., H. SEKI, K. AKIYAMA and H. YAGYU. 1974. The pigeon, a sensor of lead pollution. Bull. Environ. Contam. Toxicol. 12: 92-98.

31. PATTEE, O.H., S.N. WIEMEYER, B.M. MULHERN, L. SILEO and J.W. CARPENTER. 1981. Experimental lead shot poisoning in bald eagles. J. Wildl. Manage. 45 (3): (in press).

32. ROZMAN, R.S., L.N. LOCKE and S.F. McCLURE. 1974. Enzyme changes in mallard ducks fed iron or lead shot. Avian Dis. 18: 435-445.

33. SILEO, L., R.N. JONES and R.C. HATCH. 1973. The effects of ingested lead shot on the electrocardiogram of Canada geese. Avian Dis. 17: 308-313.

34. STONE, C.L., M.R.S. FOX, A.L. JONES and K.R. MAHAFFEY. 1977. δ-aminolevulinic acid dehydratase — a sensitive indicator of lead exposure in Japanese quail. Poultry Sci. 56: 174-181.

35. STOUT, I.J. and G.W. CORNWALL. 1976. Nonhunting mortality of fledged North American waterfowl. J. Wildl. Manage. 40: 681-693.

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use

36. WALDRON, H.A. 1966. The anemia of lead poisoning: A review. Br. J. Ind. Med. 23: 83-100.

37. WESTEMEIER, R.L. 1966. Apparent lead poisoning in a wild bobwhite. Wilson Bull. 78: 471-478.

38. WOLF, P.L., D. WILLIAMS and E. VON DER MUEHLL. 1973. *Practical Clinical Enzymology: Techniques and Interpretations and Biochemical Profiling*. pp. 215-226. John Wiley and Sons, Inc., New York.

Received for publication 11 November 1980

Downloaded From: https://bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://bioone.org/terms-of-use