LEAD CONCENTRATIONS IN LIVER AND KIDNEYS OF SNOW GEESE DURING AN AVIAN CHOLERA EPIZOOTIC IN CALIFORNIA

Author: Gordus, Andrew G.

Source: Journal of Wildlife Diseases, 29(4) : 582-586

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-29.4.582

Journal of Wildlife Diseases, 29(4), 1993, pp. 582–586

LEAD CONCENTRATIONS IN LIVER AND KIDNEYS OF SNOW GEESE DURING AN AVIAN CHOLERA EPIZOOTIC IN CALIFORNIA

Andrew G. Gordus

Department of Wildlife, Humboldt State University, Arcata, California 95521, USA; Present address: H. T. Harvey and Associates, Ecological Consultants, 423 West Fallbrook Street, Suite 207, Fresno, California 93711, USA

ABSTRACT: During an avian cholera epornitic, between December 1982 and January 1983, 58 dead, 23 sick, and 106 hunter-killed lesser snow geese (*Chen caerulescens caerulescens*) were collected at Delevan National Wildlife Refuge, Colusa County, California, USA. Fifty-one of the dead and sick geese were infected with *Pasteurella multocida*. Lead concentrations in the livers ranged from <1 to 253 parts per million (ppm) (dry weight). Lead concentrations in the kidneys ranged from <1 ppm to 547 ppm (dry weight). Snow geese with >30 ppm lead, considered diagnostic of acute lead poisoning, had significantly ($P < 0.05$) lower heart weights and a smaller band of heart fat, compared to geese with tissue lead concentrations of <30 ppm. Tissue lead concentrations in geese dying from avian cholera generally were lower than concentrations in hunter-killed geese, but the differences were not significant for either kidney ($P = 0.08$) or liver ($P = 0.30$) tissue.

Key words: Snow geese, *Chen caerulescens caerulescens*, avian cholera, *Pasteurella multocida*, lead poisoning, atomic absorption spectrophotometry, California.

INTRODUCTION

Avian cholera and lead poisoning both cause heavy non-hunting mortality among North American waterfowl (Friend, 1981). Avian cholera, caused by the bacterium *Pasteurella multocida*, has been observed annually since 1945 among wild waterfowl wintering in California (Titche, 1979; Botzler, 1991). Lesser snow geese (*Chen caerulescens caerulescens*) have suffered major losses to avian cholera (Vaught et al., 1967; Titche, 1979; Wobeser et al., 1979).

Ingestion of lead by waterfowl causes decreased physiological function, resulting in anemia and paralysis (Anonymous, 1986b). Ingested lead shot causes decreased antibody production in mallards (*Anas platyrhynchos*) (Trust et al., 1990) and decreases circulating leukocytes and spleen plaque-forming cells (Rocke and Samuel, 1991). Among laboratory rodents, lead has decreased resistance to disease (Hemphill et al., 1971; Cook et al., 1975) and decreased immunological function (Koller et al., 1976).

If lead serves as a predisposing factor to *Pasteurella multocida* infection, I predicted that birds dying from avian cholera would have higher concentrations of lead in their tissues than hunter-killed birds. The purpose of this study was to determine if concentrations of lead in tissues of snow geese dying from *P. multocida* were higher than those in the tissues of hunter-killed snow geese.

MATERIALS AND METHODS

This study was conducted during an avian cholera epornitic on Delevan National Wildlife Refuge (NWR), Colusa County, California (USA) (39°21′N, 122°11′W). Habitat, avian cholera history and waterfowl lead poisoning history of this 2,280 ha refuge have been described previously (Gordus, 1985; Anonymous, 1986b).

Dead and sick lesser snow geese were collected on the refuge during an avian cholera epornitic from 23 December 1982 to 31 January 1983. Hunter-killed geese were collected at the refuge hunter-check station 26 December 1982 to 23 January 1983. Because the refuge is so small (2,280 ha), all birds were considered to be taken at the same site. The heart, liver, and kidneys from all birds were frozen. Heart blood from each bird was cultured on brain heart infusion (BHI) agar (Difco Company, Detroit, Michigan, USA) for the presence of *Pasteurella multocida*. The bacteria were identified by the methods of MacFaddin (1980). Representative isolates were confirmed at the U.S. Fish and Wildlife Service National Wildlife Health Center (Madison, Wisconsin, USA). Gizzard contents and lining were subjected to a fluoroscope for ingested shot by refuge personnel. Heart and liver, similarly trimmed, were weighed, and the greatest width of fat in the coronary groove was measured.

For each bird, 2 g (wet weight) samples from the right and left lobe of the liver, as well as all kidney tissue, were placed separately into clean, preweighed 30 ml test tubes. Samples were dried for 24 hr at 100 C, reweighed, and homogenized in 5 ml of 15 N reagent grade nitric acid (Horwitz, 1975) and adjusted to 25 ml aliquots with deionized water in volumetric flasks. A flame atomic absorption spectrophotometer (Model 103, Perkin-Elmer, Spectroscopy Division, Ridgefield, Connecticut, USA) was used to determine lead concentrations in the tissues. The power to the lamp was pulsed at 283.3 nm/second. Standard lead solutions were prepared as described by Horwitz (1975). Lead concentrations were expressed as ppm lead/dry tissue weight. I considered a value >30 ppm lead in the kidney or liver as diagnostic of acute lead poisoning.

The tissue lead concentration data did not fulfill the requirements for a parametric test; therefore, differences in lead concentrations in the birds were analyzed with a Wilcoxon's Rank Sum Test, using the Biomedical Computer Program P Series (BMDP) (Dixon, 1981). The null hypothesis was that there were no significant differences in the distribution of tissue lead concentrations in birds dying from avian cholera compared to apparently healthy hunter-killed birds. This test also was used to evaluate differences in heart weight, heart fat width, and liver weight between lead poisoned and non-lead poisoned geese.

RESULTS

Tissues were collected from 187 lesser snow geese: 58 were found dead, 23 were found sick, and 106 were hunter-killed. Forty-six of the 81 dead or sick geese had avian cholera, five had both avian cholera and lead poisoning, six had lead poisoning only, 15 were gunshot victims, five were gunshot victims and had lead poisoning, and four died from undetermined causes. *Pasteurella multocida* was not isolated from any of the 106 hunter-killed geese; the tissue lead concentrations of these birds were used for comparison to the tissue lead concentrations of 51 geese infected with *P. multocida*.

Based on a fluoroscopic examination, 18 (10%) of the 187 gizzards collected contained lead shot. This included one bird that had ingested a .22 caliber bullet, and another goose with both one lead and one steel shot present. The maximum number of lead shot in any one gizzard was 10. One goose had a single lead shot in the gizzard with corresponding kidney and liver lead concentrations at 16 ppm and 24 ppm, respectively.

Liver lead concentrations from 51 snow geese with *P. multocida* infection ranged from <1 ppm to 135 ppm; the mean ± SE was 11 ± 4 ppm. Kidney lead residues from 51 snow geese with *P. multocida* infection ranged from <1 ppm to 181 ppm (13 ± 5 ppm). Liver lead concentrations in 106 hunter-killed snow geese, from which *P. multocida* was not isolated, ranged from <1 ppm to 253 ppm (20 ± 4 ppm). Kidneys from two birds were lost. Lead residues from the remaining 104 hunter-killed snow geese kidneys ranged from <1 ppm to 547 ppm (31 ± 8 ppm). Lead concentrations of >30 ppm occurred in the liver of 31 (17%) of 187, and in the kidneys of 36 (20%) of 185 (two kidneys were lost) snow geese collected. Five (10%) of the 51 geese infected with *P. multocida* and 20 (19%) of the 106 hunter-killed geese had tissue lead concentrations of >30 ppm.

The six geese that died from lead poisoning and the five gunshot victims with lead poisoning had mean kidney (range, 41 to 254 ppm) and liver (61 to 205 ppm) lead concentrations of 100 ± 23 and 117 ± 13 ppm, respectively.

Based on Wilcoxon's Rank Sum Test, the kidney and liver lead concentration distributions were not significantly different ($P = 0.24$, $P = 0.43$, respectively) between four juveniles and 47 adults within each sex class for snow geese with avian cholera. Kidney and liver lead concentration distributions were not significantly different ($P = 0.06$ to $P = 0.75$) between 18 juveniles and 88 adults within each sex for hunter-killed geese. Due to the small juvenile sample size and the lack of significance between the age classes, adults and juveniles were pooled and a comparison was analyzed between the sexes. There was no significant difference between 20 female and 31 male geese with avian cholera (kidney,

$P = 0.22$; liver, $P = 0.64$); and between 51 female and 55 male hunter-killed geese (kidney, $P = 0.85$; liver, $P = 0.20$). Based on Wilcoxon's Rank Sum Test, the kidney and liver lead concentration distributions were not significantly different ($P = 0.08$ and $P = 0.30$, respectively) between 51 snow geese dying from avian cholera and 106 apparently healthy hunter-killed snow geese.

Of the 187 total snow geese collected, 136 were not infected with *P. multocida*. These geese were used to compare lead poisoned and non-lead poisoned geese for liver and heart weights, and heart fat width. There was no significant difference ($P > 0.20$) in tissue weight and heart fat width between eight immature and 58 adult female geese not infected with *P. multocida*. Adult and immature female tissue measurements were pooled. Tissue measurements were tested for differences between 16 female geese with >30 ppm lead in liver or kidney tissue and 50 female geese with ≤30 ppm lead in both tissues. Female geese with >30 ppm lead had significantly lower liver weights ($P = 0.0002$), heart weights ($P = 0.028$), and heart fat widths ($P = 0.0002$) than female snow geese with ≤30 ppm tissue lead.

Six immature male and nine adult male snow geese with >30 ppm lead did not have significantly different ($P > 0.05$) tissue measurements. Seven immature male and 48 adult male geese with ≤30 ppm lead did not have significantly different liver weights ($P = 0.76$) or heart fat widths ($P = 0.68$); but immatures did have significantly lower ($P = 0.03$) heart weights. These male geese were not infected with *P. multocida*. Due to the small sample size of immature males, all males were pooled, and tested for differences in tissue measurements between 15 male geese with >30 ppm lead and 55 male geese with ≤30 ppm lead. Male snow geese with >30 ppm lead had significantly lower heart weights ($P = 0.024$) and heart fat widths ($P < 0.0001$); but no significant difference ($P = 0.58$) in liver weights.

DISCUSSION

There is no uniform standard tissue concentration for diagnosing lead poisoning in waterfowl. I used >30 ppm (dry weight), based on the following information. Longcore et al. (1974) reported 6 to 20 ppm lead (wet weight) in liver and kidney tissue as diagnostic of acute lead poisoning. Windingstad and Hinds (1987) considered ≥6 ppm (wet weight) lead in liver as diagnostic of plumbism, when accompanied by pathology. Adrian and Stevens (1979) found that using wet tissues for lead analysis created as much as 75% error due to the inconsistency of tissue moisture. White and Stendell (1977) reported >20 ppm lead (dry weight) in wing bones as diagnostic for acute and chronic lead exposure. Zwank et al. (1985) reported using >20.5 ppm liver (dry weight) as acute lead toxicosis.

Szymczak and Adrian (1978) found the lowest lead concentration in liver tissue to be 28 ppm (dry weight) and in kidney tissue to be 39 ppm (dry weight) in Canada geese (*Branta canadensis*) that died from lead poisoning. For comparison they determined tissue lead concentrations in normal captive-reared 10-wk-old Canada goose goslings. The control goslings had a mean tissue lead concentration of 5 to 6 ppm (dry weight); the highest kidney and liver tissue lead concentrations were 11 ppm and 20 ppm (dry weight), respectively. Consequently, they considered >30 ppm (dry weight) as diagnostic of acute lead poisoning. Similarly, I considered >30 ppm (dry weight) lead in the liver or kidney as diagnostic of lead poisoning in lesser snow geese. If 20 ppm (dry weight) were the diagnostic concentration, only three additional geese, including the one goose with a single pellet in the gizzard with corresponding increased tissue lead concentrations, would have been identified as lead poisoned.

There was no significant difference in tissue lead concentration distribution between snow geese dying from avian cholera, and hunter-killed birds without avian

cholera. There was a higher frequency (19%) of lead poisoning in hunter-killed birds than in birds dying from avian cholera (10%), supporting Bellrose's (1951) suggestion that ingested lead increases a mallard's (*Anas platyrhynchos*) vulnerability to hunting. He further reported that few lead poisoned waterfowl are found dead during the hunting season. Dieter and Finley (1979) reported that sublethal concentrations of lead decreased the physiological function of all organ systems, especially the nervous and gastro-intestinal systems. Starvation and paralysis due to lead ingestion (Hunter and Wobeser, 1980) may substantiate Bellrose's (1951) suggestion that ingested lead decreases a bird's mobility. Therefore, sublethal lead poisoning may increase the vulnerability of snow geese to hunting more than it does to avian cholera during the hunting season.

Friend (1976) reported that 10% of the snow geese infected with *P. multocida* also had lead poisoning, suggesting lead may have been a contributing factor to the avian cholera epornitic. This was similar to the findings among the snow geese at Delevan NWR.

Maximum lead concentrations for snow geese in this study appeared to be similar to lead concentrations in geese that died from lead poisoning at other sites (Szymczak and Adrian, 1978; Zwank et al., 1985; Windingstad and Hinds, 1987). Prevalence of ingested lead shot from all these previously cited goose lead poisoning epornitics ranged from 65% to 81%; prevalence of ingested steel shot ranged from 0 to 41%.

The U.S. Fish and Wildlife Service final minimum criteria for establishing nontoxic shot zones is the occurrence of ≥ 1 ingested shot in $\geq 5\%$ of the waterfowl gizzards collected, and ≥ 2 ppm lead (wet weight) in $\geq 5\%$ of the livers examined (Anonymous, 1986a). Based on this study, monitoring only ingested shot may underestimate the prevalence of lead toxicosis in waterfowl populations. Liver and kidney lead analysis would be a better indicator of a bird that has ingested, absorbed, distributed, and metabolized lead. Also, establishing a concentration of between 20 and 30 ppm lead (dry weight) as constituting lead poisoning might be more scientifically valid.

Some hunters were not willing to donate the viscera from their geese because they consume the heart and liver. Lead has been suggested to bioaccumulate in the tissues of raptors fed biologically incorporated lead (Benson et al., 1974; Stendell, 1980; Custer et al., 1984). Very little lead accumulates in the breast muscle; rather, most lead assimilates into the liver, kidney, and bone tissues (Longcore et al., 1974; Szymczak and Adrian, 1978). Hunters should be warned about the possible health risks associated with eating organs of wild waterfowl.

ACKNOWLEDGMENTS

This research was supported by a National Science Foundation Graduate Fellowship administered through the Humboldt State University Foundation. I thank R. G. Botzler, P. F. Springer, R. T. Golightly, and R. D. Gilchrist for reviewing previous manuscripts of this study; E. J. Collins, C. T. Osugi, and the Sacramento National Wildlife Refuge Complex personnel for their cooperation; and California Fish and Game personnel J. H. Snowden, R. Scoonover, T. Phillips and their seasonal aides for assistance at the Delevan NWR hunter check station. I appreciate the cooperation of the hunters at Delevan NWR for allowing me to collect tissues from their geese during the study; and R. K. Stroud of the USFWS for confirming the identification of the bacteriological samples. *Editor's note:* Dr. D. Forrester served as the acting editor in the review of this manuscript.

LITERATURE CITED

ADRIAN, W. J., AND M. L. STEVENS. 1979. Wet versus dry weights for heavy metal toxicity determinations in duck liver. Journal of Wildlife Diseases 15: 125–126.

ANONYMOUS. 1986a. The selected action and alternatives considered for implementation. Section II. *In* Final supplemental environmental impact statement on the use of lead shot for hunting migratory birds in the United States. U.S. Fish and Wildlife Service. Office of Migratory Bird Management, Washington, D.C., pp. 1–32.

———. 1986b. Description of the affected environment. Section III. *In* Final supplemental environmental impact statement on the use of lead

shot for hunting migratory birds in the United States. U.S. Fish and Wildlife Service. Office of Migratory Bird Management, Washington, D.C., pp. 1–91.

BELLROSE, F. C. 1951. Effects of ingested lead shot upon waterfowl populations. Transactions of the North American Wildlife Conference 16: 125–133.

BENSON, W. W., B. PHARAOH, AND P. MILLER. 1974. Lead poisoning in a bird of prey. Bulletin of Environmental Contamination and Toxicology 11: 105–108.

BOTZLER, R. G. 1991. Epizootiology of avian cholera in wildfowl. Journal of Wildlife Diseases 27: 367–395.

COOK, J. A., E. O. HOFFMAN, AND N. R. DILUZIO. 1975. Influence of lead and cadmium on the susceptibility of rats to bacterial challenge. Proceedings of the Society for Experimental Biology and Medicine 150: 741–747.

CUSTER, T. W., J. C. FRANSON, AND O. H. PATTE. 1984. Tissue lead distribution and hematologic effects in American kestrels (*Falco sparverius*) fed biologically incorporated lead. The Journal of Wildlife Management 20: 39–43.

DIETER, M. P., AND M. T. FINLEY. 1979. Delta amino levulinic acid dehydratase enzyme activity in blood, brain and liver of lead dosed ducks. Environmental Research 19: 127–135.

DIXON, W. S. 1981. BMDP statistical software. University of California Press, Berkeley, California, 726 pp.

FRIEND, M. 1976. Disease problems and needs. *In* Transactions of the Second International Waterfowl Symposium, Ducks Unlimited, St. Louis, Missouri, pp. 2–7.

———. 1981. Waterfowl management and waterfowl disease, independent or cause and effect relationships. Transactions of the North American Wildlife and Natural Resources Conference 46: 94–103.

GORDUS, A. G. 1985. Lead concentrations in liver and kidneys of snow geese during an avian cholera epizootic at Delevan National Wildlife Refuge, California. M.S. Thesis. Humboldt State University, Arcata, California, 58 pp.

HEMPHILL, F. E., M. L. KAEBERLE, AND W. B. BUCK. 1971. Lead suppression of mouse resistance to *Salmonella typhimurium*. Science 172: 1031–1032.

HORWITZ, W. 1975. Official methods of analysis of the Association of Official Analytical Chemists, 12th ed. Association of Official Analytical Chemists, Washington, D.C., 1,094 pp.

HUNTER, B., AND G. WOBESER. 1980. Encephalopathy and peripheral neuropathy in lead poisoned mallard ducks. Avian Diseases 24: 169–178.

KOLLER, L. D., J. H. EXON, AND J. G. ROAN. 1976. Humoral antibody response in mice after single dose exposure to lead or cadmium. Proceedings of the Society for Experimental Biology and Medicine 151: 339–342.

LONGCORE, J. R., L. N. LOCKE, G. E. BAGLEY, AND R. ANDREWS. 1974. Significance of lead residues in mallard tissues. U.S. Fish and Wildlife Service, Special Scientific Report. Wildlife Number 182, U.S. Fish and Wildlife Service, Washington, D.C., 18 pp.

MACFADDIN, J. F. 1980. Biochemical tests for identification of medical bacteria. Waverly Press, Inc., Baltimore, Maryland, 527 pp.

ROCKE, T. E., AND M. D. SAMUEL. 1991. Effects of lead shot ingestion on selected cells of the mallard immune system. Journal of Wildlife Diseases 27: 1–9.

STENDELL, R. C. 1980. Dietary exposure of kestrels to lead. Journal of Wildlife Diseases 44: 527–530.

SZYMCZAK, M. R., AND W. J. ADRIAN. 1978. Lead poisoning in Canada geese in southeast Colorado. The Journal of Wildlife Management 42: 299–306.

TITCHE, A. R. 1979. Avian cholera in California. California Wildlife Management Branch. Wildlife Management Administration Report Number 79-2. California Department Fish and Game, Sacramento, California, 49 pp.

TRUST, K. A., M. W. MILLER, J. K. RINGELMAN, AND I. M. ORME. 1990. Effects of ingested lead on antibody production in mallards (*Anas platyrhynchos*). Journal of Wildlife Diseases 26: 316–322.

VAUGHT, R. W., H. C. MCDOUGLE, AND H. H. BURGESS. 1967. Fowl cholera in waterfowl at Squaw Creek National Wildlife Refuge, Missouri. The Journal of Wildlife Management 31: 248–253.

WHITE, D. H., AND R. C. STENDELL. 1977. Waterfowl exposure to lead and steel shot in selected hunting areas. The Journal of Wildlife Management 41: 469–475.

WINDINGSTAD, R. M., AND L. S. HINDS, III. 1987. Lead poisoning in Canada geese on Plum Island, Massachusetts. Journal of Wildlife Diseases 23: 438–442.

WOBESER, G. A., D. B. HUNTER, B. WRIGHT, D. J. NIEMAN, AND R. ISBISTER. 1979. Avian cholera in waterfowl in Saskatchewan, spring 1977. Journal of Wildlife Diseases 15: 19–24.

ZWANK, P. J., V. L. WRIGHT, P. M. SHEALY, AND J. D. NEWSOM. 1985. Lead toxicosis in waterfowl on two major wintering areas in Louisiana. Wildlife Society Bulletin 13: 17–26.

Received for publication 14 August 1992.