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LEAD POISONING AND OTHER CAUSES OF MORTALITY IN TRUMPETER (*CYGNUS BUCCINATOR*) AND TUNDRA (*C. COLUMBIANUS*) SWANS IN WESTERN WASHINGTON

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ABSTRACT: Lead poisoning and other causes of mortality of 115 trumpeter (*Cygnus buccinator*) and 21 tundra (*C. columbianus*) swans from northwestern Washington (USA) from 1986 to 1992 are reported. Necropsies were performed on all 136 swans, liver lead analysis conducted on 110, and differentiation between lead and steel shot pellets recovered from gizzards in 97 swans. Shot pellets were detected in 44 (32%) of 136 gizzards. Lead shot was recovered from 32 (33%) of 97 gizzards and steel shot from 16 (16%). Mean intensity of lead shot in gizzards was nearly five times greater than steel shot. Thirty-nine (35%) of 110 livers had lead concentrations diagnostic of lead poisoning (>6 ppm, wet weight). Mean (±SE) weight for 61 non-lead poisoned trumpeter swans was 9.8 (±0.30) kg, significantly heavier ($P < 0.05$) than 30 lead poisoned trumpeters ($\bar{x}$ = 6.8 ± 0.23 kg). There was no significant difference ($P > 0.05$) in weights between lead poisoned ($n = 9$) and non-lead poisoned ($n = 12$) tundra swans. Lead poisoning was the primary cause of death, accounting for 29% of the mortalities. Other causes of mortality identified were aspergillosis (17%), illegally shot (11%), and other traumatic factors (12%). The cause of death for 43 swans was not determined. Lead poisoning from the ingestion of lead shot continues to be a principal cause of mortality in swans overwintering in northwestern Washington.

Key words: Lead poisoning, swans, *Cygnus buccinator*, *Cygnus columbianus*, Washington, mortality.

INTRODUCTION

Lead poisoning caused by the ingestion of lead shot from shot shells has affected every major species of waterfowl in North America (Sanderson and Bellrose, 1986). Although the use of nontoxic steel shot in place of lead shot for waterfowl hunting has been required in many areas of the United States since 1976 (DeStefano et al., 1991), and the mandatory use of steel shot for all waterfowl hunting in the United States began in 1991, the problem of lead poisoning in waterfowl continues to persist in some areas.

Some species are more susceptible to lead poisoning than others, primarily due to differences in feeding habits (Friend, 1987). Waterfowl with specialized food habits, particularly piscivorous birds, seldom are affected, while puddle ducks, diving ducks, geese and swans which commonly feed in shallow lake bottoms are affected more often. Swans are particularly vulnerable to lead poisoning because of their habit of feeding in lake bottoms for tubers and roots, ingesting lead pellets in the process (Langelier et al., 1990). Lead poisoning in swans occurs primarily from November through March when the birds are on their wintering grounds or in areas used during spring migrations (Friend, 1987).

Lead poisoning in trumpeter (*Cygnus buccinator*) and tundra (*C. columbianus*) swans has been reported in western Washington (USA) (Kendall and Driver, 1982; Blus et al., 1989) and other regions of North America (Trainer and Hunt, 1965; Blus et al., 1989; Langelier et al., 1990). However, these reports occurred prior to the implementing of steel shot zones, include data from a single year, or include data from relatively few swans. Our objective was to determine the cause of mortality of 136 swans collected in northwestern Washington from 1985 to 1992, with emphasis placed on the recovery of lead and steel shot from gizzards, and on liver lead con-

centrations. Swan body weights and other causes of mortality are summarized.

MATERIALS AND METHODS

One hundred and fifteen trumpeter (63 males, 51 females; 71 adults, 43 juveniles; one age and sex unrecorded) and 21 tundra (13 males, 8 females; 10 adults, 10 juveniles; one age unrecorded) swans were collected by personnel of the Washington Department of Wildlife over a 7-yr period, 1986 to 1992, from northwestern Washington, including Island, San Juan, Skagit and Whatcom Counties (48°00′ to 49°00′N, 120°50′ to 123°15′W). All swans were found dead or debilitated; debilitated birds died shortly after capture or were euthanized with pentobarbital sodium (Anthony Products Company, Arcadia, California, USA). The carcasses were frozen before being transported annually to Washington State University for examination.

Swans were thawed at 21 C prior to necropsy. At necropsy, each bird was weighed and the sex determined. Gizzard and liver were removed, and air sacs and other organs were examined grossly for pathological abnormalities. Gizzards were cut longitudinally and the contents washed into a 500 ml beaker. Plant material was removed from the sediment by adding tap water to the beaker, allowing it to settle for approximately 5 sec, decanting two-thirds of the supernatant and adding more water. This procedure was repeated several times until the supernatant was clear. The sediment and water to cover the contents were placed into a 23 cm diameter aluminum pie plate and examined for shot pellets under a magnifying lens. A magnet was used to distinguish lead from steel shot. In 1986 (n = 24) and 1987 (n = 15) no distinction was made between lead and steel shot recovered from gizzards; therefore only data from 1988 to 1992 (n = 97) were included in statistical analysis concerning recovery of shot from gizzards.

Livers were analyzed for lead concentrations at the University of Idaho Veterinary Toxicology Laboratory, Moscow, Idaho (USA). Liver lead concentrations (wet weight) were determined with a Perkin Elmer P-40 atomic absorption spectrophotometer (Perkin-Elmer Analytical Instruments, Norwalk, Connecticut, USA) set at 220.4 nm wavelength, with the methods described by Martin et al. (1987). The lower limit of detection was 0.20 parts per million (ppm) wet-weight. Liver lead concentrations >6 ppm on a wet-weight basis were considered diagnostic of lead poisoning (Friend, 1987).

One way analysis of variance tests (Ott, 1984) were used to compare weights (juveniles and adults combined) of lead poisoned (liver lead concentration >6 ppm) trumpeter and tundra swans with conspecifics not diagnosed with lead poisoning to identify significant differences ($P < 0.05$). The number of swans with ≥1 pellet in gizzard divided by number of swan gizzards examined (prevalence), the minimum and maximum number of pellets recovered from gizzards (range), and mean number of pellets per infected swan (mean intensity) of lead and steel shot in gizzards were determined for each year; liver lead concentrations were analyzed yearly, except in 1986 when liver lead analysis was not conducted. A tentative diagnosis of aspergillosis was based on finding extensive fungal growth in the lungs and air sacs upon gross examination at necropsy. Diagnosis of other non-lead poisoned mortalities was determined by necropsy examinations and field collection reports.

RESULTS

Lead shot was recovered from the gizzard of 32 (33%) of 97 swans (Table 1), including 25 (31%) of 81 trumpeter and 7 (44%) of 16 tundra swans. Steel shot was detected in 16 (16%) of 97 gizzards, including 11 (14%) of trumpeter and 5 (31%) of tundra swans. Including data from 1986 and 1987, shot pellets were recovered from the gizzard of 44 (32%) of 136 swans. Mean intensity of lead shot in gizzards was nearly five times greater than steel shot (Table 1). Of the 110 swan livers analyzed for lead concentration, 39 (35%) contained levels considered diagnostic of lead poisoning (>6 ppm wet weight) (Table 1), with a mean of 21 ppm. Thirty (33%) of 91 trumpeter and 9 (47%) of 19 tundra swan livers contained lead concentrations >6 ppm. Of these birds with elevated liver lead concentrations, 17 (57%) of the trumpeters and 4 (44%) of the tundras were adults. The maximum lead concentration in liver was 51 ppm.

No significant difference ($P > 0.05$) was observed in body weight between sexes for both trumpeter and tundra swans. Mean (±SE) weight of male trumpeter swans was 8.7 (±0.3) kg (n = 63), females 8.6 (±0.3) kg (n = 51). Mean (±SE) weight of male tundra swans was 5.9 (±0.5) kg (n = 13), females 4.9 (±0.7) kg (n = 8). Trumpeter swans that did not have lead poisoning (n = 61) were significantly heavier ($P < 0.05$)

TABLE 1. Prevalence and intensity of naturally ingested lead and steel shot in gizzards, and liver lead concentrations of trumpeter and tundra swans from western Washington, 1986 to 1992.

Year	Number	Lead shot	Steel shot	Liver lead levels (ppm, wet wt.)
1986	24	21 (0–40) 10.8[a,b]	NA[c]	NA
1987	15	53 (0–36) 11.8[b]	NA	8 (0.1–38) 47[d]
1988	16	31 (0–34) 11.6	31 (0–3) 2.0	10 (0.2–45) 47[e]
1989	14	64 (0–40) 10.0	21 (0–6) 3.7	18 (0.2–51) 57
1990	21	14 (0–18) 6.7	0 (0) 0	1 (0.3–15) 10
1991	27	22 (0–59) 17.7	15 (0–9) 3.3	5 (0.6–26) 22
1992	19	47 (0–23) 5.6	21 (0–5) 2.3	11 (0.1–32) 50[f]
Total	136	33 (0–59) 10.3[g]	17 (0–9) 2.3	9 (0.1–51) 35[h]

[a] Percent prevalence (range) mean intensity.
[b] Lead and steel shot not differentiated, therefore data not included in statistical analysis.
[c] Not analyzed.
[d] Mean (range) percentage of swans with liver lead level >6 ppm.
[e] Based on 15 swans.
[f] Based on 18 swans.
[g] Based on 97 swans.
[h] Based on 110 swans.

than those with lead poisoning (n = 30), with means (±SE) of 9.8 (±0.30) kg and 6.8 (±0.23) kg, respectively. No significant difference ($P > 0.05$) in body weight was observed between lead poisoned and non-lead poisoned tundra swans.

Lead poisoning was the primary cause of mortality, accounting for 29% of swan deaths (Table 2). Excluding data from 1986 when liver lead analysis was not conducted, mortality due to lead poisoning accounted for 35% of all mortalities. Aspergillosis (17%), birds illegally shot (11%) and other trauma factors (12%) contributed to swan morality. The cause of death of 43 (31%) of the swans was not determined.

TABLE 2. Summary of the primary mortality factors of trumpeter and tundra swans from western Washington, 1986 to 1992.

Diagnosis	Trumpeter (n = 115)	Tundra (n = 21)	Combined (n = 136)
Lead poisoning[a]	31 (27)[b]	9 (43)	39 (29)
Aspergillosis	21 (18)	2 (10)	23 (17)
Shot illegally	10 (9)	5 (23)	15 (11)
Other trauma	13 (11)	3 (14)	16 (12)
Unknown	40 (35)	2 (10)	43 (31)

[a] Liver lead levels >6 ppm (wet weight).
[b] Number affected (percent).

DISCUSSION

Trumpeter and tundra swans arrive in northwestern Washington in November from their breeding grounds in Alaska, and remain until March (Davison, unpubl.). This wintering population of swans numbers approximately 1,950 birds, with the largest concentrations occurring in the fresh water lakes and salt water marshes in Skagit County. Because of its close proximity to populated urban developments, and the attraction of large numbers of migrating waterfowl, the area is used heavily by waterfowl hunters. In 1986, 169,987 lead pellets/ha were present in the upper 10 cm of bottom sediments (Davison, unpubl.). The use of lead shot for waterfowl hunting in Skagit county has been prohibited since 1986, and from all of Washington beginning in 1991.

Based on our 7 yr of data, mortality of swans caused by lead poisoning has not measurably decreased in this region. Although steel shot has been required for waterfowl hunting in the area since 1986, there continues to be a greater number of led shot than steel shot recovered from gizzards, and a high percentage of birds with elevated liver lead concentrations. From 1988 to 1992, the prevalence of lead

shot in gizzards was equal to or greater than that of steel shot, and the intensity of lead shot was more than five times greater than steel shot in some years (Table 1). The relatively low prevalence of shot and low liver lead concentration reported in 1990 is unexplained. In 1992, the last year of our study, 9 of 18 swans had liver lead concentrations considered toxic. We believe this is due in part to years of lead shot accumulation prior to establishment of steel shot requirements, and noncompliance among hunters of steel shot regulations. In their study of lead poisoning in Canada geese (*Branta canadensis*) in an area prohibiting use of led shot for nearly 10 yr, DeStefano et al. (1991) attributed the predominance of lead shot to >20 yr of hunting from permanent blinds, the higher decay rate of steel, and the illegal use of lead shot.

Of the swans with liver lead concentrations >6 ppm, 90% had lead shot in the gizzard, compared with 35% when all swans were considered (1988 to 1992). The absence of lead shot in the gizzard does not rule out lead shot induced lead poisoning (Hirai et al., 1990), and ingestion of large numbers of lead shot is not needed to produce lead toxicity. Six of 10 black ducks (*Anas rubripes*) experimentally inturbated with one number four lead shot died within 6 days post-intubation (Pain and Rattner, 1988). Other factors influencing the degree of lead absorption and toxicity include the species of bird, physiologic condition, diet, number of pellets ingested, climate, and stress (Pain and Rattner, 1988; Rocke and Samuel, 1991). We did not investigate other possible sources of lead contamination, although there have been no reports of lead toxicity in waterfowl in the region from sources other than lead shot.

Kendall and Driver (1982) reported lead poisoning of three (one tundra, two trumpeter) swans from Skagit County, with liver lead concentrations ranging from approximately 15 to 22 ppm, wet weight. In a study of swan mortality in seven western states from 1976 to 1987, Washington had the highest incidence of lead poisoning mortality (Blus et al., 1989). Blus et al. (1989) found that 8 of 12 swans from northwestern Washington died from lead poisoning, with liver lead concentrations ranging from 8 to 37 ppm, wet weight, and a mean of 19 ppm.

That trumpeter swans with liver lead concentrations >6 ppm weighed significantly less than those with liver lead concentrations <6 ppm was not unexpected, since muscle atrophy is a typical clinical sign of lead poisoning due to the extended duration of illness in most birds. Tundra swans with liver lead concentrations >6 ppm also weighed less than those below this level, although the difference was not significant.

We believe the swans acquired a significant number of lead shot and subsequent lead poisoning after arriving to northwestern Washington. It is highly unlikely a bird afflicted with lead poisoning could endure the rigorous physiological demands of migration. Most swans arrive in November and remain in the area for 3 to 4 mo. From our data, 94% of the mortality attributed to lead poisoning occurred from December to March, ≥4 wk after the swans arrive. Lead poisoning mortality can occur in several days to several weeks following ingestion of lead pellets (Pain and Rattner, 1988), and is dependent upon the influencing factors mentioned above.

Aspergillosis was the second most frequent cause of mortality in our study, with most cases occurring in 1986. Infection usually results from the inhalation of *Aspergillus* sp. spores, often present in moldy feeds and rotting agricultural waste (Locke, 1987). It is possible that many or all of these birds in 1986 became infected from the same food source. Other cases of aspergillosis mortality occurred in low frequency and uniformly throughout the following years of this study. The "other trauma" category in Table 2 included mortality from hitting powerlines and

fences. Fractures and internal hemorrhage were detected in these birds at necropsy. The cause of death was not identified for many birds. In these cases, most of the swans appeared to be in good body condition, had adequate amounts of subcutaneous fat, low liver lead levels and contained no lesions characteristic of aspergillosis. Because the swans had been frozen prior to necropsy, isolation of bacterial and viral organisms was not attempted, and histological examination was not done.

Despite the ban on lead shot in northwestern Washington since 1986, mortality of swans from lead poisoning continues to occur as a result of ingestion of lead pellets. Until previously deposited lead shot settles into the bottom sediments beyond the reach of feeding swans, birds will continue to die from lead poisoning. More aggressive enforcement of steel shot regulations may help reduce or eliminate the illegal use of lead shot. Ideally, full compliance among hunters of steel shot regulations and continued settling of lead shot deeper into bottom sediments should eventually result in reduced lead poisoning mortality of swans from ingestion of lead shot.

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