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EPIDEMIOLOGIC INVESTIGATION OF LEAD POISONING IN TRUMPETER AND TUNDRA SWANS IN WASHINGTON STATE, USA, 2000–2002

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ABSTRACT: An observational study was conducted to determine the proportionate mortality of wild trumpeter (*Cygnus buccinator*) and tundra (*Cygnus columbianus columbianus*) swans that died during the winters of 2000–02 in northwestern Washington State, USA. Among 400 swans necropsied, 81% were lead poisoned (302/365 trumpeter swans; 20/35 tundra swans). Mortality started in mid-November and peaked from late December through mid-February; swan mortality that was not associated with lead poisoning was uniformly lower throughout the winter months. Lead poisoning was 24 times more likely to be the cause of death in swans found in Whatcom County compared to swans found in other locations in northwestern Washington State (95% CI: 12.7, 47.0). Mortality attributable to lead poisoning was twice as likely in adults as in juveniles (95% CI: 1.0, 4.2). Aspergillosis was documented in 62 trumpeter and two tundra swans, including 37 swans in which mortality was caused by lead poisoning. Males were twice as likely as females to have aspergillosis (95% CI: 1.1, 3.8). Traumatic injuries were documented in 37 trumpeter and seven tundra swans, including seven trumpeter swans with concurrent lead poisoning. Dead swans found outside Whatcom County were four times more likely to have traumatic injuries compared to those found in Whatcom County (95% CI: 1.6, 10.0). Overall, lead-poisoned swans were significantly less likely to have concurrent aspergillosis or traumatic injuries. There was no apparent association between grit ingestion (total mass or mass categorized by size) and lead poisoning or number of lead shot. Not surprisingly, lead-poisoned swans were more likely to have one or more lead shot compared to swans that died from other causes (OR 294; 95% CI: 92, 1,005); lead-poisoned swans were also more likely to have one or more nontoxic shot compared to swans that were not poisoned (OR 63; 95% CI: 19, 318). The source(s) of shot are unknown but likely are in or near Whatcom County, Washington.

Key words: Aspergillosis, *Cygnus buccinator*, *Cygnus columbianus columbianus*, lead poisoning, powerline collisions, trumpeter swan, tundra swan, waterfowl.

INTRODUCTION

Lead poisoning in waterfowl historically has been a problem in wetland areas where spent lead shot and other nontoxic shot is ingested during feeding (Blus, 1994) or while ingesting grit to aid in grinding coarse food in the ventriculus (Mateo and Guitart, 2000). Ingested lead shot is eroded by ventricular grinding, becomes solubilized, and is absorbed by the gastrointestinal system with systemic distribution to soft tissues and bones (Clemens et al., 1975). Trumpeter swans (*Cygnus buccinator*) and tundra swans (*Cygnus columbianus columbianus*) in North America may be more susceptible to lead poisoning than other waterfowl because of their specific feeding habits or increased lead shot burden or availability on their selected breeding or wintering grounds (Gillette, 1991; Blus, 1994; Rocke et al., 1997). Lead poisoning in trumpeter

(Blus et al., 1989) and both trumpeter and tundra (Lagerquist et al., 1994) swans was recorded previously in Washington State. An increase above normal swan mortality was noted in western Washington State in the winter preceding ($n=71$) and the year after the study ($n=186$); however, the number of lead-poisoned swans was not reported (Smith, 2004). Lead-poisoning mortality was documented in 87 of 186 trumpeter swans in British Columbia (Wilson et al., 1998) and 53 of 116 trumpeter swans from refuges in Minnesota that had not been used for hunting in over 20 yr (Degernes and Frank, 1991).

Gross pathology in lead-poisoned waterfowl can include emaciation, green-stained vent, impactions of the proventriculus or esophagus, bile-distended gall bladder, green-stained kaolin lining of the ventriculus, pale flabby heart, cephalic edema, and/or lead shot or fragments in the ventriculus (Beyer et al., 1998). Birds that die acutely may not have gross pathologic changes.

Lead shot was banned for waterfowl hunting in the United States in 1991 and in southwestern British Columbia in 1990 (Wilson et al., 1998) because of documented deleterious effects on waterfowl, raptors, and other wildlife (Scheuhammer and Norris, 1996). Lead shot is still legal for upland game hunting in most areas of the United States and Canada, as well as for trap shooting and training of hunting dogs. During the winters of 2000–02, hundreds of trumpeter and tundra swans died in an outbreak in northwestern Washington State. The objectives of this epidemiologic observational study were to identify causes and associated risk factors for mortality of wild swans in Washington State.

MATERIALS AND METHODS

During the winters of 2000–02, 400 sick, injured, and dead swans were collected in northwestern Washington (47°56′N, 122°02′W and 48°59′N, 122°15′W). Debilitated swans found alive were euthanatized in the field. All swans were tagged with date and location of carcass recovery, whether it was found dead or euthanatized in the field, and whether or not any tissues were removed prior to necropsy (i.e., liver sample or ventriculus). Observations related to traumatic injuries, such as rope entanglement, observation of powerline collision, or radiographic documentation of gunshot injury, were recorded on the tag when applicable. Birds were frozen and stored for necropsy; necropsies were completed in May following each winter season. Weeks were numbered chronologically starting with 12 November 2000 and 11 November 2001.

Necropsies were conducted at Western Washington University's Hannegan Environmental Center in Bellingham, Washington, USA. Body weight was recorded except for cases in which birds were scavenged or in which tissue samples had been previously removed. Age classes were determined based on plumage and markings on feet and bills (Mitchell, 1994). Both trumpeter and tundra swans were classified as juvenile (<1 yr of age) or adult (≥1 yr of age); subadult swans (1–3 yr of age) were included with adults.

The presence and location of traumatic injuries, green staining around vent, extent and location of scavenging, and whether or not the carcass had been previously opened were recorded, and liver, kidneys, heart, lungs, trachea (including tracheal loops embedded within the keel), syrinx, air sacs, gonads, esophagus, proventriculus, ventriculus, small and large intestines, and cloaca were examined. Liver samples (approximately 2–3 g) and contents of the proventriculus and ventriculus were collected and frozen for later examination. Samples were not collected for histopathology because of limited funding and potential difficulty with interpretation of histologic lesions caused by post-mortem autolysis and freezing.

Heavy metal analyses were done at the Analytical Service Laboratory at North Carolina State University, Raleigh, North Carolina, USA. Liver samples were desiccated, processed using a dry ashing protocol, and analyzed for lead, zinc, and copper concentration (mg/kg [ppm] dry weight [d/w]) using ion-coupled plasma (ICP) atomic emission spectrometry (Optima 2000 DV, Perkin-Elmer, Wellesley, Massachusetts, USA) (Gorsuch, 1970; Mader et al., 1996). Lead and zinc analyses were done both years, but copper analyses were done only for samples from 2000–01. Percentage moisture was calculated for 100 trumpeter swan livers collected from intact birds at necropsy (2001–02 samples

only). Standards used to calibrate the ICP emission spectrometer were prepared in the same acid matrix as the liver samples using ICP-grade commercial standard stock solutions. An estimate of individual sample variance was obtained from analysis of replicate samples ($n=20$) that spanned the range in observed metal concentrations. Instrument calibration was confirmed using NIST 1572 orchard leaves. The method limit of detection (MLD) for lead was 1.0 mg/kg d/w for 2000–01 samples and 2.0 mg/kg d/w for 2001–02 samples (a smaller sample was used the second year, doubling the dilution factor). All liver lead concentrations ≤MLD were assigned a value equal to half of the MLD for lead (0.5 or 1.0 mg/kg d/w for 2000–01 and 2001–02 samples, respectively). All zinc and copper MLD were well below the minimum liver concentrations measured in our samples (both zinc and copper MLD were ≤2 mg/kg d/w for both years).

Grit and shot were separated from vegetative matter and were air-dried. Magnetic nontoxic shot (steel, tungsten iron, tungsten polymer) was separated from grit samples using a magnet. The remaining grit and shot contents were systematically scanned using a stereomicroscope (4× magnification, 50, 60 Hz; Olympus Optical Co., Tokyo, Japan) to visualize and remove lead shot and lead fishing sinkers. After lead shot was collected, grit samples were radiographed and examined again by stereomicroscopy to retrieve previously undetected shot. Lead shot was identified by its characteristic dull, blue-gray pitted surface that is easily scratched to reveal a shiny metal appearance. All nonmagnetic shot was visually examined using a stereomicroscope to confirm that the shot was lead and not some other type of nonmagnetic, nontoxic shot (tungsten matrix, tin, bismuth, or Hevi® shot). Nontoxic shot types were not differentiated in this study, and all nontoxic and lead shot were counted. Grit was separated into four size categories using 4-mm, 2-mm, and 1-mm nested sieves and weighed by size (≤1 mm, >1–2 mm, >2–4 mm, and >4 mm; USA Standard Testing Sieve, Newark Wire Cloth Company, Newark, New Jersey, USA). Grit weight data were collected only in the second year of the study. Shot and grit data were excluded for analysis in birds in which scavenging damaged the proventriculus or ventriculus or in which one or both organs were removed prior to necropsy.

Three criteria were used to define lead poisoning. Clinical lead poisoning was diagnosed in birds with liver lead levels ≥20 mg/kg d/w (Trainer and Hunt, 1965), and subclinical lead poisoning was diagnosed in birds with liver lead levels 8–19.9 mg/kg d/w (Locke and Thomas, 1996). An alternative criterion was used when liver tissue was unavailable. In this case, swans with ≥10 lead shot and gross lesions consistent with lead poisoning such as proventricular impactions (Beyer et al., 1998) were classified as lead poisoned.

Birds with extensive fungal respiratory lesions involving lungs, air sacs, and/or trachea were clinically diagnosed with aspergillosis. Cytologic examination of respiratory lesions in several swans confirmed *Aspergillus* sp. based on the presence of characteristic septate hyphae and conidiophores (Campbell, 1995); cultures were not done to identify species.

Traumatic injuries were diagnosed based upon gross pathology and information recorded by biologists at the time of carcass recovery. Mild traumatic injuries or injuries inconsistent with mortality were not included. No attempt was made to differentiate the primary cause of death when more than one cause was possible, such as concurrent lead poisoning and aspergillosis or lead poisoning and powerline collision.

Descriptive statistics were used to develop tables that included heavy metal analysis, body weight, grit weight, and swan frequency distributions by disease status. Wilcoxon Rank Sum tests were used to compare liver lead concentrations and body weights between lead-poisoned and non–lead-poisoned swans categorized by sex, age, and species, and grit weights between species (Sokal and Rohlf, 1995). Similarly, comparisons for categorical data were obtained using logistic regression to estimate odds ratios and 95% confidence intervals. A Fisher exact test was used if sample size in a cell fell below five birds.

Logistic regression models were developed for the three primary causes of mortality: lead poisoning, aspergillosis, and traumatic injuries. Swans with clinical and subclinical lead poisoning were combined with swans classified as lead poisoned using the alternative criterion. Lead-poisoned birds were coded as the index group, and non–lead-poisoned birds were coded as the referent group. Aspergillosis (present or absent) and traumatic injuries (present or absent) also were coded as dichotomous variables. Bivariate logistic regression was used to determine which main effect variables to include in preliminary models using an a priori cutoff point set at $P<0.20$ for level of significance. Interaction terms were tested one at a time by adding them to the full model with main effect variables, using a chi square test with one degree of freedom to calculate the P-value. Confounders were

arbitrarily identified using a change in the estimate criterion (10%) for the odds ratio for the reduced model compared to the full model.

Logistic regression was used to investigate associations between proventricular impactions and lead poisoning and between shot counts and lead-poisoning status. Correlation coefficients were calculated for relationships between liver lead concentration and the number of lead shot, between the number of lead and nontoxic shot, and between the number of lead shot and grit mass (total and by size category). All data analyses were done using commercially available software (SAS 9.1.3, SAS Institute, Cary, North Carolina, USA).

RESULTS

In this observational study, trumpeter and tundra swans comprised 91% ($n=365$) and 9% ($n=35$) of 400 dead swans collected in Washington State during the winters of 2000–02 (Table 1). During this same two-year period, live trumpeter swans comprised 64% and 72% of wild swan midwinter (January) population estimates (D. Kraege, unpubl. data). The wild swan population estimates in Whatcom, Skagit, and Snohomish counties in Washington State for 2000–01 and 2001–02 were 3,605 and 4,206 trumpeter swans and 2,024 and 1,634 tundra swans, respectively. Age class data were not available from midwinter surveys.

The proportionate mortality caused by lead poisoning in this two-year study was 83% (302/365) of trumpeter swans and 57% (20/35) of tundra swans necropsied (Table 1). Liver lead concentrations among lead-poisoned swans were significantly higher in all age and sex categories of both species compared to non–lead-poisoned swans. Body weights were significantly lower in all lead-poisoned trumpeter swans (except juvenile males) compared to non–lead-poisoned trumpeter swans (Table 1). Similar trends were observed among lead-poisoned tundra swans, but the differences were not statistically significant.

Liver lead concentrations of 20 mg/kg d/w and 8 mg/kg d/w are equivalent to approximately 5.7 mg/kg wet weight (w/w) and 2.3 mg/kg w/w, respectively, based on 71.5% (±2.2% SD) average moisture content measured in 100 trumpeter swan livers in this study (all samples were collected from intact carcasses). The percentage moisture content for seven tundra swan livers was comparable, at 72.1% (±1.3% SD). Liver lead concentrations ranged from <2 mg/kg d/w to 489 mg/kg d/w (Fig. 1). Among lead-poisoned swans, tundra swans had significantly higher liver lead levels than trumpeter swans, probably related to eight of 20 lead-poisoned tundra swans (including six juvenile swans) with liver lead levels >100 mg/kg d/w. Subclinical liver lead concentrations (8–19 mg/kg d/w) were observed in 2.5% ($n=10$) of all necropsied swans, and clinical liver lead concentrations (20–489 mg/kg d/w) were observed in 76% ($n=303$) of all necropsied swans. Liver lead concentration inconsistent with lead poisoning was observed in 18.5% ($n=75$) of necropsied swans. Liver samples were missing for 3% ($n=12$) of swans; of these, nine swans were classified as lead poisoned using the alternate criterion. All liver zinc (Zdziarski et al., 1994) and copper (Kobayashi et al., 1991) levels were below toxic levels reported for waterfowl (data not shown). Percentage recovery of lead, zinc, and copper using NIST 1572 orchard leaves was 91, 93, and 95%, respectively. Individual sample uncertainty (expressed as percentage coefficient of variation) ranged from 3% to 16% for samples having >15 mg/kg lead or 3% to 40% for samples having >75 mg/kg copper or zinc.

Nearly 60% of documented lead-poisoning cases were observed in the second year of the study (Table 2). Non–lead-poisoned swan numbers were comparable over the two-year study. Dead swans found in Whatcom County comprised 82% of all necropsied swans ($n=327$), compared to 18% found in all other counties combined ($n=72$). Among lead-

TABLE 1. Liver lead concentration and body weights for lead-poisoned and non–lead-poisoned trumpeter and tundra swans, categorized by age and sex. Swans were part of a group of 400 wild trumpeter and tundra swans necropsied in Washington State during 2000–02. Data are expressed as mean ±SD with frequency in parentheses.[a]

		Lead poisoned[b]			Non–lead poisoned[c]			
		n	Liver lead (mg/kg d/w)	Body weight[d] (kg)	*n*	Liver lead (mg/kg d/w)	Body weight[d](kg)	Total
Trumpeter swans		302			62			365
Adult	Male	129	55.67±25.55 (126)[e]	8.5±1.2 (103)[e]	20	1.74±2.05 (18)	10.7±1.8 (13)	149
	Female	126	59.12±31.92 (124)[e]	7.3±1.2 (91)[e]	15	0.90±0.50 (15)	9.5±1.5 (12)	141
Juvenile	Male	28	42.49±22.77 (28)[e]	7.5±1.0 (18)	14	0.76±0.55 (13)	8.1±1.7 (9)	42
	Female	12	51.85±29.66 (12)[e]	6.2±0.4 (6)[e]	11	0.83±0.80 (11)	9.1±1.8 (7)	23
Tundra swans		20			15			35
Adult	Male	5	75.16±34.39 (5)[e]	5.4±0.6 (5)	3	1.00±0.87 (3)	7.5 (2)	8
	Female	6	81.82±39.81 (5)[e]	4.1±0.5 (5)	7	0.57±0.19 (7)	6.1±2.1 (6)	14
Juvenile	Male	5	146.64±68.18 (5)[e]	4.9±0.6 (4)	2	0.50 (2)	5.1 (2)	7
	Female	4	203.46±197.23 (4)[e]	4.7±1.4 (3)	2	2.09 (2)	5.0 (2)	6

[a] Rows and columns may not add up because of missing values for sex or liver lead concentration.

[b] Lead-poisoned swans had liver lead levels ≥8 mg/kg d/w.

[c] Non–lead-poisoned swans had liver lead levels <8 mg/kg d/w. Causes of death included aspergillosis, traumatic injuries (powerline collisions, gunshot injuries, rope entanglement, and unknown trauma), and unknown causes.

[d] Body weights were not included for swans that were scavenged or those missing tissue samples collected prior to necropsy.

[e] Significantly higher (liver lead) or lower (body weight) values for lead-poisoned versus non–lead-poisoned swans at $P \leq 0.05$.

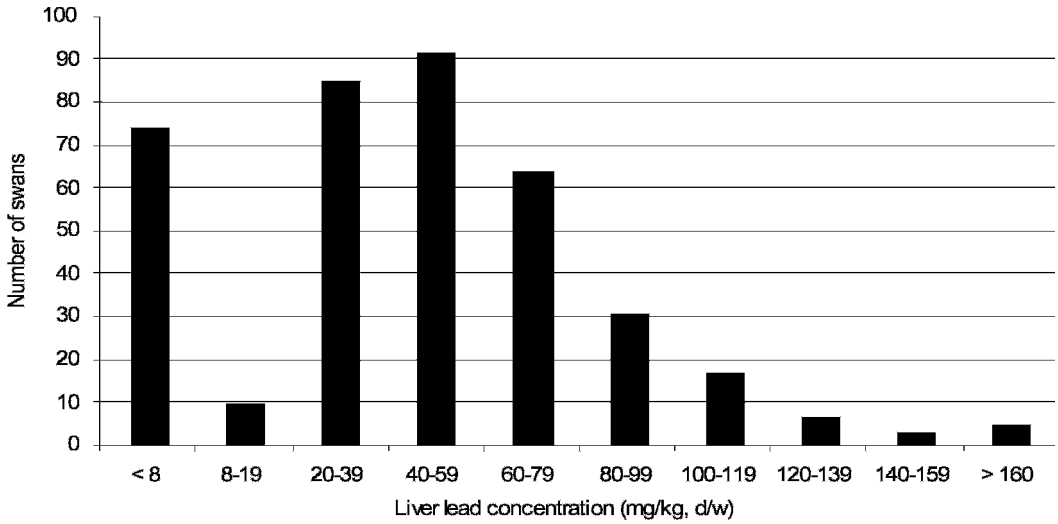

FIGURE 1. Liver lead concentration in 355 trumpeter and 33 tundra swans necropsied in Washington State, 2000–02. Swans were categorized as non–lead poisoned if liver lead concentration <8 mg/kg d/w, subclinically lead poisoned if liver lead concentration 8–19.9 mg/kg d/w, and clinically lead poisoned if liver lead concentration ≥20 mg/kg d/w. Five swans in the highest liver lead concentration had liver lead concentrations between 178 and 489 mg/kg d/w. Liver samples were missing from 12 swans and not included in this graph, including nine swans categorized as lead poisoned based on alternative criteria (≥10 lead shot and gross lesions consistent with lead poisoning).

poisoned swans in this study, 94% of trumpeter and 80% of tundra swans were found in Whatcom County. By contrast, 35% of trumpeter and 27% of tundra swans that were non–lead-poisoned were found in Whatcom County.

Weekly wild swan mortality documented in the winters of 2000–02 in Washing-

TABLE 2. Lead-poisoned and non–lead-poisoned trumpeter and tundra swans, categorized by year and location. Swans were part of a group of 400 wild trumpeter and tundra swans necropsied in Washington State during 2000–02.[a]

		Lead poisoned[b]	Non–lead poisoned[c]	Total
Trumpeter swans		302	62	365
Year	2000–01	124	30	155
	2001–02	178	32	210
Location[d]	Whatcom	284	22	307
	Other	17	40	57
Tundra swans		20	15	35
Year	2000–01	6	10	17
	2001–02	14	4	18
Location[d]	Whatcom	16	4	20
	Other	4	10	15

[a] Rows and columns may not add up because of missing values for location or liver lead concentration.

[b] Lead-poisoned swans had liver lead levels ≥8 mg/kg d/w.

[c] Non–lead-poisoned swans had liver lead levels <8 mg/kg d/w. Causes of death included aspergillosis, traumatic injuries (powerline collisions, gunshot injuries, rope entanglement, and unknown causes), and unknown causes.

[d] Other locations included Jefferson (n = 1), San Juan (n = 2), Skagit (n = 61), and Snohomish (n = 8) counties in northwest Washington State. Location data missing for one lead-poisoned trumpeter swan.

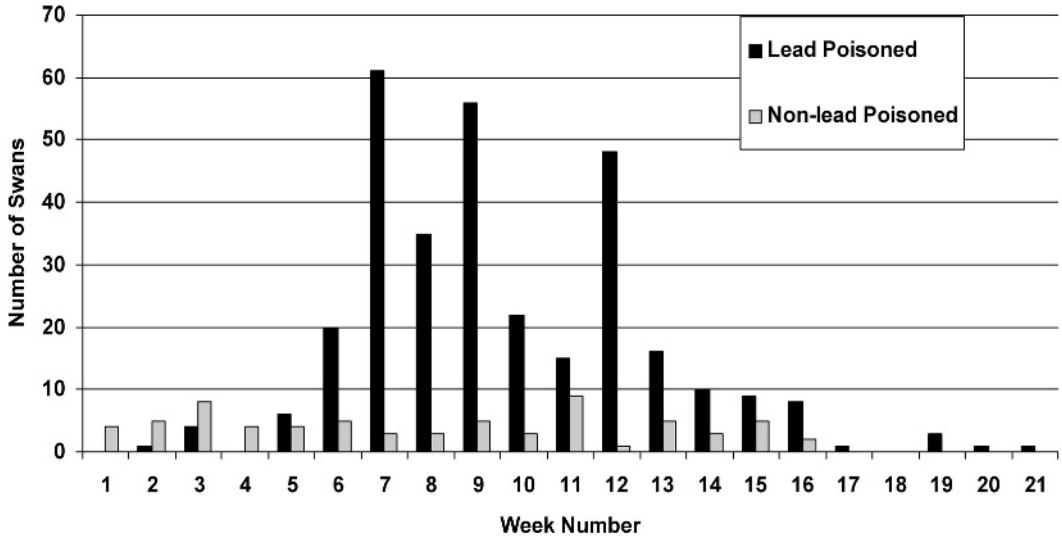

FIGURE 2. Weekly mortality due to lead-poisoning or non–lead-poisoning causes for 386 trumpeter and tundra swans necropsied in Washington State, 2000–02. Week numbers started 12 November (2000) or 11 November (2001). Swans were categorized as lead poisoned if liver lead levels ≥8 mg/kg d/w (n=309) and non–lead poisoned if liver lead levels <8 mg/kg d/w (n=68). Nine swans categorized as lead poisoned based on alternative criteria (≥10 lead shot and gross lesions consistent with lead poisoning) were included with the lead-poisoned swans. Causes of mortality for non–lead-poisoned swans included aspergillosis, trauma, and other or unknown causes. Figure excludes 10 swans with missing collection dates, two swans with undetermined lead-poisoning status, and two non–lead-poisoned swans found during weeks 26 and 28 (May 2001, May 2002).

ton State resembled a point source epidemic curve with the highest mortality from approximately late December (Week 6) through mid-February (Week 13; Fig. 2). In this two-year study, average (±SD) weekly mortality (based on 21 weeks) for lead-poisoning and non–lead-poisoning causes was 15.1±19.1 and 3.3±2.6 birds, respectively.

Based on modeling the association between lead poisoning and species, age, location, and year, no interaction terms were significantly different in the models investigated, and age and location were retained in the final model. Adults were 2.1 times (95% CI: 1.0, 4.2) more likely as juveniles to be lead poisoned. Swans found dead in Whatcom County compared to those recovered from other locations were 24.4 times (95% CI: 12.7, 47.0) more likely to be lead poisoned than non–lead poisoned.

Proventricular impactions were observed in 63 trumpeter and five tundra swans. Only one bird with a proventricular impaction was not lead poisoned, but it had peritonitis. Swans with impacted proventriculus typically had plant fiber (grasses and/or other unidentified plant material or field corn) mixed with grit, and ingesta extended into the oral cavity in many impacted swans. There were no associations between proventricular impactions and liver lead concentration, number of lead shot found in the ventriculus, or total grit mass within the ventriculus.

Aspergillosis was diagnosed in 62 trumpeter and two tundra swans, including 32 trumpeter swans with clinical lead poisoning and five trumpeter swans with subclinical lead poisoning (Table 3). Five trumpeter swans had nearly complete occlusion of the tracheal loops within the keel. The distribution of aspergillosis cases by year and location paralleled the distri-

TABLE 3. Aspergillosis and traumatic injuries at necropsy categorized by age and sex. Swans were part of a group of 400 wild trumpeter and tundra swans necropsied in Washington State during 2000–02.

		Aspergillosis diagnosis[a]			Trauma diagnosis[b]		
		Lead poisoned[c]	Non–lead poisoned[c,d]	Total	Lead poisoned[c]	Non–lead poisoned[c]	Total
Adult	Female	9	4	13	4	13	17
	Male	20	10	30	2	11	13
Juvenile	Female	3	4	7	0	6	6
	Male	5	7	12	1	7	8
Year	2000–01	12	12	24	5	19	24
	2001–02	25	15	40	2	18	20
Location	Whatcom	35	15	50	5	8	13
	Other[e]	2	12	14	2	29	31

[a] Trumpeter ($n = 62$) and tundra ($n = 2$) swans diagnosed with aspergillosis were combined because of the small number of tundra swans.

[b] Trumpeter ($n = 37$) and tundra ($n = 7$) swans diagnosed with traumatic injuries were combined because of the small number of tundra swans. Traumatic injuries included powerline collisions, gunshot injuries, rope entanglement, and unknown trauma.

[c] Lead-poisoned swans had liver lead levels ≥8 mg/kg d/w, and non–lead-poisoned swans had liver lead levels <8 mg/kg d/w.

[d] Sex was missing for one adult and one juvenile non–lead-poisoned trumpeter swan that had aspergillosis.

[e] Other locations included Skagit, Snohomish, and San Juan counties in northwest Washington State.

bution of total swans by year and location (Tables 2 and 3). Non–lead-poisoned swans were 4.5 times (95% CI: 2.5, 8.3) more likely to have aspergillosis compared to lead-poisoned swans, and male swans were 2.1 times (95% CI: 1.1, 3.8) more likely to be diagnosed with aspergillosis compared to female swans.

Traumatic injuries were documented in 11% of swans (Table 3). Powerline collisions accounted for 70% of traumatic injuries, including 18 of 24, three of four, two of two, and eight of 13 swans from Skagit, Snohomish, San Juan, and Whatcom counties, respectively. Four of seven trumpeter swans with concurrent lead poisoning and traumatic injuries had evidence of powerline collisions. Seventy percent of swans with traumatic injuries were found in counties other than Whatcom County. After accounting for age as a potential confounder, non–lead-poisoned swans were 25 times (95% CI: 8.3, 50) more likely to have traumatic injuries compared to lead-poisoned swans. In addition, swans with traumatic injuries were 1.7 times (95% CI: 1.6, 10) more likely to be found in other counties compared to injured swans found in Whatcom County.

One trumpeter swan died of peritonitis and proventricular impaction (etiology unknown). No cause of death was determined for seven trumpeter and six tundra swans.

Lead shot comprised 75% (8,181/10,975) of total shot collected in 2000–02. Of the 333 swans with documented shot counts, non–lead-poisoned swans had significantly fewer shot of either type compared to lead-poisoned swans. Among non–lead-poisoned swans, 9% (6/68) had lead shot and 4% (3/68) had nontoxic shot, compared to 97% (256/265) and 74% (197/265) of lead-poisoned swans with lead shot and nontoxic shot, respectively (Fig. 3). Three lead-poisoned trumpeters had one or two lead fishing sinkers. The median number of lead shot and nontoxic shot found in swan ventriculi was 11 (95% CI: 0, 91) and two (95% CI: 0, 35), respectively. The highest recorded numbers of lead and nontoxic shot counts were 384 and 309, respectively, found in an adult female trumpeter swan; another 123 swans ingested ≥25 shot (Fig. 3; lead and

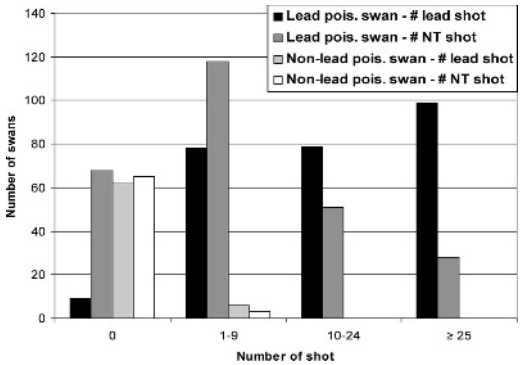

FIGURE 3. Number of lead or nontoxic (NT) shot found in the ventriculus of 256 lead-poisoned and 77 non–lead-poisoned swans. Birds were part of a group of 400 wild trumpeter and tundra swans necropsied in Washington State, 2000–02. Nontoxic shot included steel, tungsten-matrix, and tungsten-polymer. Shot types were not differentiated. Lead-poisoned swans had liver lead levels ≥8 mg/kg d/w, or those swans identified as lead poisoned using the alternative criteria (≥10 lead shot and gross lesions consistent with lead poisoning). Non–lead-poisoned swans had liver lead levels <8 mg/kg d/w. Data excluded 67 swans in which the gastrointestinal tract was scavenged or those in which the proventriculus or ventriculus was removed prior to necropsy.

nontoxic shot combined; all were lead-poisoned swans). The documented lead and nontoxic shot were correlated with each other (correlation coefficient 0.75) but showed lower correlation with liver lead concentrations (correlation coefficient 0.43 and 0.18, respectively, for lead and nontoxic shot). The odds of a lead-poisoned swan having at least one lead shot were 294 times higher than the odds of a non–lead-poisoned swan having zero lead shot (95% CI: 92.0, 1005). The odds of a lead-poisoned swan having at least one nontoxic shot were 63 times higher than the odds of a non–lead-poisoned swan having zero nontoxic shot (95% CI: 19.3, 318). It is possible that not all shot was found, or that some shot was already ground up in the ventriculus or had passed through the gastrointestinal tract. To account for possible misclassification of shot counts, we reclassified 50% of lead-poisoned and non–lead-poisoned swans in which no shot were found as actually having at least one lead or nontoxic shot. Even after adjusting these shot counts, the odds of a lead-poisoned swan having at least one lead shot were still 51 times higher than the odds of a non–lead-poisoned swan having zero lead shot (95% CI: 16.5, 207). Also, the odds of a lead-poisoned swan having at least one nontoxic shot were 6 times higher than the odds of a non–lead-poisoned swan having zero nontoxic shot (95% CI: 3.2, 11.7).

More total grit was found in trumpeter than tundra swans, as well as in the >1–2 mm and >2–4 mm grit categories (Table 4). Total grit mass ranged from 3.5 to 724 g (the swan with 724 g had a proventricular impaction). Within each species, males had more total grit than females ($P \leq 0.05$; data not shown). There were no differences in grit mass (total or by size category) between lead-poisoned and non–lead-poisoned swans (species combined for comparisons due to small tundra swan sample size). There was no association between grit (total mass or by size category) and either lead-poisoning status or number of lead shot.

TABLE 4. Grit mass for four size categories (mm) for 158 trumpeter and 15 tundra swans. Birds were part of a group of 228 wild trumpeter and tundra swans necropsied in Washington State, 2001–02.[a,b]

	≤1 mm (g)	>1–2 mm (g)	>2–4 mm (g)	>4 mm (g)	Total (g)
Trumpeter swan	10.2 (2.8, 86.2)	17.7 (5.8, 116)[c]	3.6 (0.6, 30.1)[c]	0.4 (0, 4.4)	34.3 (11.6, 217)[c]
Tundra swan	9.7 (2.4, 65.0)	8.4 (1.6, 117)	1.3 (0.3, 40.3)	0.2 (0, 13.6)	19.2 (4.2, 223)

[a] Data expressed as median with 95% confidence intervals.

[b] Grit data were collected in 2001–02 only. Data do not include 55 swans in which the gastrointestinal tract was scavenged or those in which the proventriculus or ventriculus was removed prior to necropsy.

[c] Trumpeter swans had significantly more grit than tundra swans at $P \leq 0.05$.

DISCUSSION

The documented number of trumpeter and tundra swans (81%; 322/400 birds necropsied) that died of lead poisoning in northwestern Washington State in the winters of 2000–01 and 2001–02 was unexpected, particularly since lead had been banned for waterfowl hunting in the US for a decade. Elsewhere the prevalence of lead exposure in waterfowl has declined following implementation of nontoxic shot for waterfowl hunting (Samuel and Bowers, 2000).

All heavy metal analyses were conducted on a dry weight basis to minimize variability associated with moisture content of desiccated tissues as occurred in partially scavenged swans. We designated clinical lead poisoning in birds with liver lead concentration ≥20 mg/kg d/w (equivalent to approximately 5.7 mg/kg, w/w) and subclinical lead poisoning in birds with liver lead concentration 8–19.9 mg/kg d/w (8.0 mg/kg d/w is equivalent to approximately 2.3 mg/kg w/w in this study). Both cutoff values are consistent with other reports (Locke and Thomas, 1996). We chose to combine subclinical and clinical lead-poisoned swans for logistic regression analyses, but it is possible that some birds with subclinical liver lead concentrations or swans classified as lead poisoned using the alternative criteria died of causes other than lead poisoning. Since these two groups of swans accounted for less than 5% of necropsied swans, excluding them from data analyses or reclassifying them as non–lead-poisoned did not change our results appreciably. Furthermore, when we ran the logistic analysis with these two groups excluded from the data, the final model remained the same, and the odds ratios for location and age increased by 3% and 8%, respectively.

Trumpeter swan mortality (91% of documented swan mortality) was disproportionately high compared to tundra swan mortality, considering that live trumpeter swans comprised approximately two-thirds of the wild swan population in northwestern Washington State during the same time period. It is possible that midwinter counts may have over- or underestimated the true population of trumpeter and tundra swans, as swans frequently fly across the border between Washington and British Columbia (Wilson et al., 2004). It is also possible that a higher proportion of trumpeter than tundra swan carcasses were found because of either their larger size or proximity to areas commonly searched for carcasses.

No differences between proportions of lead-poisoned male and female swans were observed in our study, similar to other reported studies (Degernes et al., 1989; Wilson et al., 1998). The percentage of adult (87%) versus juvenile lead-poisoned trumpeter swans documented in this study was comparable to 71–82% adult lead-poisoned trumpeter swans reported elsewhere (Degernes et al., 1989; Wilson et al., 1998).

The increased prevalence of lead poisoning during late December to mid-February (Weeks 6–12) suggests that swans developed lead poisoning after migration to wintering grounds in November. Although swan-specific information is unavailable to predict onset of clinical signs following exposure to lead shot, experimentally induced lead poisoning in Canada geese resulted in mortality in 39–72 days after 10 number-four lead shot were force-fed (Cook and Trainer, 1966). The same study reported mortality in <10 days after 25 number-four lead shot were force-fed. The decline in lead-poisoning prevalence after mid-February may have been related to most swans moving to more southerly areas, presumably away from lead shot sources (Wilson et al., 2004). Once a swan develops clinical signs of lead poisoning, it is less able to fly and move long distances (Degernes et al., 1989). Thus, the large numbers of lead-poisoned swans collected in Whatcom County suggest that the source(s) of lead shot were in this geographical area.

A weak relationship between lead shot numbers and liver lead levels was shown in a retrospective study (Beyer et al., 1998). However, in our study, liver lead concentrations were poorly correlated with lead shot numbers (correlation coefficient 0.43). We evaluated shot data but did not include these data in our models because of potential bias associated with the number of shot found at the time of necropsy. The number of lead shot in the gastrointestinal tract is influenced by the rates of shot retention and shot erosion. Lead shot retention is highest in birds that ingest a large number of shot in a short period of time (Cook and Trainer, 1966), probably because of acute lead toxicosis and paresis of the gastrointestinal tract (Hunter and Wobeser, 1980). The rate of expulsion of shot from the gastrointestinal tract may be influenced by shot size (smaller steel shot lost faster than larger shot in mallards fed a low-fiber diet), dietary factors (increased retention in mallards fed a high-fiber diet), and grit composition (increased retention in mallards deprived of grit) (Longcore et al., 1974; Clemons et al., 1975). Rate of lead erosion is highest soon after ingestion, with up to 20% loss in the first 4 days (Clemons et al., 1975).

Because this was an observational study, the number of shot found was probably lower than the true level of exposure. It was not surprising to observe that swans with at least one lead shot had much higher odds of being lead poisoned than swans without lead shot. What was surprising was that lead-poisoned swans were more likely to have ingested nontoxic shot than non–lead-poisoned swans. Even after sensitivity testing was done to account for the possibility of undercounting lead or nontoxic shot, the odds of a lead-poisoned swan having at least one lead or nontoxic shot were much greater than that for non–lead-poisoned swans that had zero shot. These results suggest that swans that ingest shot (of any kind) may be predisposed to lead poisoning. It is not known whether swans ingest shot while seeking grit or during feeding. However, it is likely that some birds selectively or opportunistically ingest shot, as evidenced by one trumpeter swan with 693 lead and nontoxic shot and another 123 swans that ingested ≥25 total shot (lead and nontoxic shot combined). Although we did not analyze food items found in the proventriculus and ventriculus, it is possible that swans were foraging for food that was similar in size or shape to shot. Another group found that steel shot ingestion in mallard ducks decreased slightly with increased grit supplementation and was not related to rice or sorghum grain type, suggesting that shot ingestion was more related to grit-seeking behavior than grain feeding (Mateo and Guitart, 2000).

There were 17,155 trumpeter swans counted in the Pacific Coast population in the 2000 census, with an average annual population increase of 1.6% from 1995 to 2000 (Conant et al., 2002). The 32% increase in documented swan mortality in the second year of the study was higher than one would expect simply due to slightly higher swan numbers counted in northwestern Washington State in the second year. It is possible that greater effort was expended to locate and retrieve sick and dead swans in 2001–02, or that environmental and agricultural conditions were different between the two years, resulting in different foraging behavior and/or more favorable conditions for locating sick and dead swans. The rainy winter of 2001–02 could have flooded some fields subsequently used by swans that became a source of lead shot (Wilson et al., 2004).

Weight loss is a common observation with lead-poisoned birds (Lagerquist et al., 1994), so it was not surprising that lead-poisoned trumpeter swans were low-weight compared to non–lead-poisoned swans. Although similar trends were observed for lead-poisoned tundra swans, a larger sample size may have resulted in statistically significant differences in body weights between groups.

We were unable to show an association between grit ingestion (total mass and mass by size) and either lead poisoning or number of lead shot. A study investigating ventricular contents of hunter-harvested mallards also found no difference in the amount of ingested grit in ducks with or without lead shot (Havera and Anderson, 1999). Similarly, both lead-poisoned and non–lead-poisoned swans in our study had comparable total grit mass. Also, both groups had more than 80% of grit (by mass) consisting of particles <2.0 mm diameter. Comparable grit measurements were reported in non–lead-poisoned mute swans (*Cygnus olor*), which averaged 31.4 ± 1.4 g (SE), with approximately 75% of grit smaller than 1.18 mm (Franson et al., 2001).

Birds with compromised or immature immune function may have a higher likelihood of developing aspergillosis infections (O'Meara and Witter, 1971), so it was not unexpected to find that nearly half the swans with aspergillosis were also lead poisoned (Locke et al., 1969). Surprisingly, lead poisoning was not associated with concurrent aspergillosis; in fact, lead-poisoned swans were one-fifth as likely as non–lead-poisoned swans to have aspergillosis. Similar results were observed in another, albeit much smaller, study involving lead-poisoned trumpeter swans with aspergillosis (Degernes and Frank, 1991). It is possible that most birds with lead poisoning were healthy at the time of lead exposure and died too quickly from lead poisoning for secondary opportunistic diseases such as aspergillosis to develop. The higher proportion of male versus female swans infected with aspergillosis was an unexpected finding. Adult swans form lifetime pair bonds, and bonded pairs stay together year round including during the winter season off the breeding territories (Mitchell, 1994). Winter feeding strategies for swans of different sexes are similar, and one would expect equal exposure to *Aspergillus* spores during feeding (Anderson, 1994).

Elevated and subclinical liver lead levels have been reported in mute swans that died of powerline collisions (O'Halloran et al., 1989). In our study only 1% of all swans had concurrent lead poisoning and traumatic injuries due to powerline collisions. Lead-induced encephalopathy and peripheral neuropathy can impair a bird's ability to fly (Hunter and Wobeser, 1980; Degernes et al., 1989). It is possible that once a lead-poisoned swan is clinically affected, it is less likely to move far from its roost site, thereby decreasing its chances of colliding with powerlines. The higher prevalence of powerline collisions in Skagit and Snohomish counties suggest that proximity to more urban areas north of Seattle may be more relevant to powerline collisions than underlying lead poisoning in these swans. Another possibility is that the detection rate of powerline casualties was higher in other counties because of more people reporting downed swans near roads.

Total swan mortality very likely was underreported, as it was impossible to locate every dead swan before it had been scavenged, and many dead swans could have died in inaccessible locations or have been missed in thick vegetation near night roost sites (Wobeser and Wobeser, 1992). No controlled studies were done in this observational study to determine if lead-poisoned swans were more or less likely to be found than non–lead-poisoned swans, or if there was a difference in recovering trumpeter versus tundra swans. The amount of effort expended to retrieve dead and dying swans may have varied by county or season, so it is possible that Whatcom County sources were overrepresented, or that a lower proportion of swans that died very early or very late in the winter season were found. Also, swans that died from powerline collisions were probably more likely to be found because of the proximity of powerlines to roads. It is also not known if the proportions of swans that died of different causes are representative of the true proportions of

swan mortality causes. A limitation of this observational study was the lack of data from live swans, including swans that may have had subclinical lead poisoning, mild aspergillosis, and/or other diseases.

In summary, we documented that lead poisoning accounted for the majority of known trumpeter and tundra swan mortalities in Washington State during the winters of 2000–02 and that ingested lead shot was responsible for nearly all cases. Birds that died of aspergillosis or traumatic injuries were significantly less likely to be concurrently lead poisoned. Whether swans ingested old shot deposited prior to the ban on lead shot for waterfowl hunting, or new shot via legal or illegal sources, is unknown. Past lead-poisoning mortality outbreaks have been associated with wetlands that have not been used for hunting in over 20 years (Gillette, 1991), so it is possible that swans have newly discovered old sources of lead shot. It is also possible that changes in agricultural practices may be related to increased availability and exposure to lead shot. There was a very strong association between lead poisoning and birds found in Whatcom County, so it is likely that the source(s) of lead shot are in or near that area. Ongoing, multiagency efforts are underway to locate the sources of shot and prevent further mortality in these birds.

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