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Incidence of lead poisoning in Bald Eagles and lead shot in waterfowl gizzards from British Columbia, 1988–91

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Abstract

From 1988 to 1991, 294 sick, injured, or dead Bald Eagles *Haliaeetus leucocephalus* from British Columbia were examined for lead exposure. Lead exposure was determined by evaluating clinical signs, radiographs, pathology, δ-aminolevulinic acid dehydratase (ALA-d) activity ratios in blood, and lead levels in kidney, liver, and bone. We present here the bone and kidney lead levels and ALA-d ratios in blood for 65 eagles analyzed to date. The results indicate that 37% (24/65) of eagles tested exhibited significant lead exposure, with 14% (9/65) classified as lead poisoned and 23% (15/65) classified as subclinically exposed. The greatest numbers of lead-poisoned and lead-exposed birds were received during the months of January to March, when eagles feed heavily on wintering waterfowl. Radiographic analysis of a sample of 246 waterfowl gizzards collected from hunters during the 1988–89 hunting season in southwestern British Columbia showed the frequency of lead shot was 13.5% in Canada Goose *Branta canadensis*, 8.3% in Lesser Snow Goose *Anser caerulescens caerulescens*, 20.7% in Mallard *Anas platyrhynchos*, 7.1% in American Wigeon *Anas americana*, and 26.8% in Northern Pintail *Anas acuta*. A ban on the use of lead shot for waterfowl hunting in southwestern British Columbia was implemented in the 1990 hunting season. Although the majority of both lead-poisoned and subclinically exposed Bald Eagles came from the nontoxic shot zones, substantial numbers (i.e., 4/9 poisoned birds, 4/15 subclinically exposed birds) came from outside those zones.

Introduction

Lead poisoning of waterfowl as a result of ingestion of lead shot has been well studied (Bellrose 1959; Longcore et al. 1974; Mudge 1983; Sanderson and Bellrose 1986). The importance of lead poisoning as a cause of death for birds of prey has received less attention, although at least 119 Bald Eagles *Haliaeetus leucocephalus* have been reported to have died of lead poisoning since 1963 in the United States (U.S. Fish and Wildlife Service 1986). In British Columbia, the first published report of lead poisoning in Bald Eagles was of birds that died in 1989 (Langelier et al. 1991a), although the first documented poisoning occurred in 1986 (Langelier et al. 1991b).

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As a result of the above information and because of concerns over the use of lead shot in Canada, a formal project was initiated in 1989 to determine causes of mortality in Bald Eagles from British Columbia. In this paper, preliminary results for bone and kidney lead levels and blood δ-aminolevulinic acid dehydratase (ALA-d) ratios for 65 birds examined between 1988 and 1991 are reported. In addition, the incidence of lead shot in the gizzards of waterfowl collected from hunters in British Columbia during the 1988–89 hunting season is reported.

Methods

This study was part of an overall investigation into the health status of Bald Eagles in British Columbia. A request for sick, injured, and deceased Bald Eagles was published in local periodicals and circulated to government and non-government wildlife agencies. Birds were subsequently received from the B.C. Ministry of Environment, Canadian Wildlife Service, wildlife rehabilitators, and members of the public. Blood samples were taken by wildlife rehabilitators and veterinarians as soon as possible after sick and injured birds were presented for treatment. Control blood samples for the ALA-d assay were taken from seven nonreleasable Bald Eagles that had lived in rehabilitation centres for longer than six months with no known history of lead shot ingestion. Postmortem examinations of Bald Eagle carcasses were performed at the Island Veterinary Hospital, Nanaimo, B.C. Sex was determined by hallux claw and bill depth measurement (Bortolotti 1984) or on postmortem examination. Age was determined from moulting sequence (McCollough 1989).

Tissue samples were stored in chemically cleaned (acetone/hexane) glass jars and frozen; heparinized blood samples were stored in nitric acid-rinsed cryovials and frozen in liquid nitrogen. Samples were shipped on dry ice to the National Wildlife Research Centre in Hull, Quebec, for analysis. Lead levels in bone were determined by flame atomic absorption spectrophotometry (AAS) and in kidney by graphite furnace AAS; they are expressed on a dry-weight basis in this paper. ALA-d activity was determined as described in Scheuhammer (1987, 1989). Normal ALA-d activity can vary among species and individuals; therefore, the results are presented as the ratio of activated:non-activated enzyme activity to reduce variability.

Waterfowl gizzards collected from hunters in the Lower Mainland and on Vancouver Island near Ladysmith from October 1988 through January 1989 were packaged and frozen until examination for the presence of lead shot. Each gizzard was opened, and the contents were washed into a Buchner funnel. The excess water was drawn off, and the contents and the filter paper were air dried and packaged individually in plastic bags. The bags were then radiographed, and the presence or absence of lead shot was recorded.

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Results

Tissue lead levels and ALA-d activity ratios

Of the 65 eagles tested to date, nine (14%) were classified as lead poisoned (Table 1) based on ALA-d ratios greater than or equal to 5 (indicating a blood lead level above 80 μg/100 mL) and/or lead levels in kidney greater than or equal to 20 mg/kg dry weight (based on data in Pattee et al. 1981). Mean ALA-d ratios for the poisoned birds were 16.7 ± 9.4, whereas mean lead levels were 34 ± 18 mg/kg dry weight in kidney and 7.3 ± 2.2 mg/kg dry weight in bone.

Fifteen eagles (23%) were classified as subclinically lead exposed based on ALA-d ratios greater than or equal to 2 and less than 5 and/or lead levels in kidney greater than or equal to 2.0 mg/kg dry weight and less than 20 mg/kg dry weight and/or detectable levels in bone (Table 2). Mean ALA-d ratios for this group were 2.8 ± 0.7, whereas mean tissue lead levels (including only those results greater than the limit of detection) were 2.8 ± 0.9 mg/kg dry weight in kidney and 1.9 ± 1.4 mg/kg dry weight in bone.

Table 3 shows the data for 41 eagles tested that were considered to be free from lead exposure. Lead levels were below detection limits in both kidney and bone for all such birds analyzed (N = 14 and 13, respectively), whereas the mean ALA-d activity ratio was 1.2 ± 0.2 (N = 27). The mean ALA-d ratio was 1.2 ± 0.1 for seven eagles that had resided for more than six months in captivity and therefore were not exposed to lead shot during at least that time.

Geographical distribution of lead poisoning

Figure 1 shows the collection sites for 65 eagles tested for tissue lead and/or ALA-d. Of the nine birds classified as lead poisoned, eight came from coastal areas and one from the interior of British Columbia near Kamloops. Five of the nine poisoned birds were collected from the areas currently zoned for use of nontoxic shot for waterfowl hunting. Of the four birds from outside the zoned areas, one was from the Kamloops area and the other three came from the northeast coast of Vancouver Island.

Of the 15 birds classified as subclinically lead exposed, 13 were picked up from coastal areas, whereas two were from the interior. Most (11/15) were found in the areas currently zoned for nontoxic shot.

Seasonal distribution of lead poisoning

The majority of eagles received for the study were collected during the months of January to April (Fig. 2). The incidence of lead exposure and lead poisoning was highest during the months of February and March.

Lead shot in waterfowl gizzards

The overall incidence of lead shot in waterfowl gizzards from the Lower Mainland was 16.6% (Table 4). The highest proportions were in Northern Pintail (26.9%), Mallards (23.7%), and Lesser Snow Geese (10.3%) collected on Westham Island.

Discussion

A higher percentage of eagles was classified as lead poisoned based on tissue analysis and/or ALA-d ratios (14%) compared with the percentage exhibiting clinical signs of lead toxicosis (8.6%) (Table 5). Some of the birds that were diagnosed as having died from other causes, especially inanition, may prove also to have been lead poisoned once tissue analysis for lead is completed for the entire collection of eagles.

The incidence of acute lead poisoning reported in Table 5 should be relatively unbiased, because the probability of finding birds that are sick or dead as a result of lead exposure should be no different from that of finding eagles that have died of most causes. The incidence of subclinical lead exposure may be somewhat biased because of the possible role of lead-induced neurological impairment as a precursor to other causes of death. Sublethal exposure to lead and the associated neurotoxic effects leading to incoordination and effects on other critical functions, such as vision, hearing, and behaviour, could increase the chance of death from such causes as electrocution, vehicle collision, and inanition.

Waterfowl are an important winter food source for Bald Eagles on the Pacific coast (Stalmaster et al. 1985), particularly ducks that have been killed or crippled by hunters (Griffen et al. 1982). Recent studies show that crippling losses of ducks by lead shot can be as high as 39% (Nieman et al. 1987), and that large numbers (average 30%) of healthy ducks carry lead pellets embedded in their muscle (U.S. Fish and Wildlife Service 1986). Eagles may also be exposed to lead while consuming the gizzards of waterfowl that were killed or debilitated by ingestion of lead shot.

In 1990, the United States implemented a complete ban on the use of lead shot for waterfowl hunting. In Canada, lead shot is banned only in "hot spots" designated by evidence of significant waterfowl exposure to lead shot. In British Columbia, lead shot was banned in 1990 for waterfowl hunting in four wildlife management areas (Fig. 1), based on the data in Table 4 and a high incidence of chronic lead poisoning of Trumpeter Swans *Cygnus buccinator* on Vancouver Island (Langelier et al. 1989). Banning of lead shot should substantially reduce the number of sick and crippled ducks carrying both ingested and embedded lead shot in the zoned areas. Inspection of 108 gizzards collected from three duck species during the winter of 1990–91 (the first lead shot-free season) from the Fraser River delta showed that steel shot was present in amounts approximately equal to lead shot (J. Baldwin, pers. commun.). Thus, a reduction in the secondary exposure of scavenging eagles and other birds of prey is expected in those areas.

Eagles may still be exposed to lead from feeding on migrant ducks carrying embedded shot received outside the designated nontoxic shot zones. Most of the ducks that migrate through or winter in southern British Columbia come from the B.C. interior, Alberta, Alaska, Yukon, and Northwest Territories (McKelvey and Smith 1990). The Yukon and Northwest Territories are sparsely populated and waterfowl hunting pressure is low, whereas only steel shot is permitted in Alaska. Therefore, only in the B.C. interior and Alberta is there enough waterfowl hunting pressure with lead shot to produce a significant reservoir of ducks with embedded shot. Those ducks may become eagle prey if they are subsequently crippled (by steel shot) or fall sick during winter.

However, our data show that 4/9 poisoned Bald Eagles came from outside the nontoxic shot zones. Those eagles were presumably exposed to lead shot by eating ducks that were debilitated as a result of local waterfowl hunting, particularly in northeastern Vancouver Island. Further waterfowl gizzard surveys for lead shot should be undertaken in areas such as Port Hardy and the Cariboo region. The role of lead shot in eagle mortality should also be considered in other areas of Canada, such as New Brunswick, where waterfowl consumption by Bald Eagles has been reported (Wright 1953).

In conclusion, lead poisoning is an important cause of death for Bald Eagles in British Columbia. Monitoring of eagles for lead exposure should be continued in order to assess the effectiveness of regulatory measures to restrict use of lead shot.

Figure 1
Geographical distribution of lead exposure in Bald Eagles collected from 1988 to 1990 in British Columbia

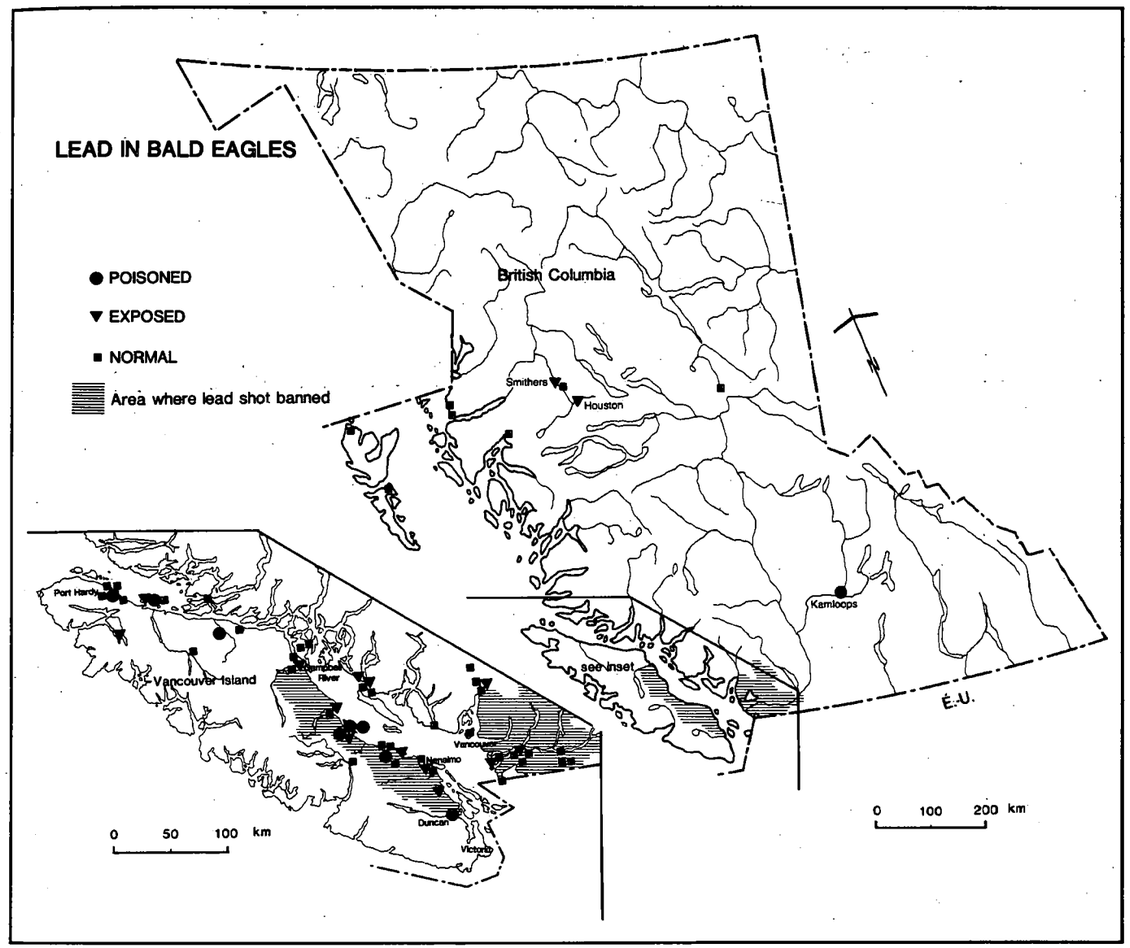

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Figure 2
Seasonal distribution of lead poisoning in Bald Eagles collected from 1988 to 1991 in British Columbia

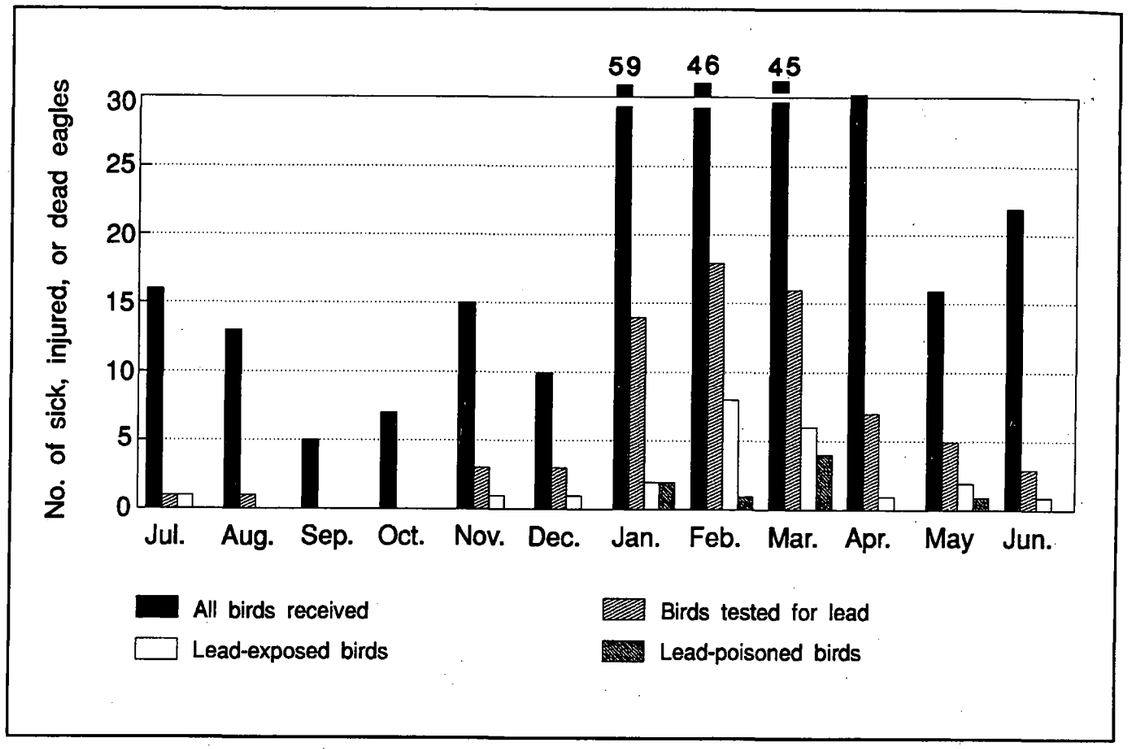

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Table 1
Data on the nine Bald Eagles that showed evidence of lead poisoning

Date	Age	Sex	Location	Lead in kidney (mg/kg)	Lead in bone (mg/kg)	ALA-d ratio	Initial clinical diagnosis
Apr. 89	Adult	F	Denman Is.			25	Inanition
Dec. 89	1 year	F	Bowser			25	Lead toxicosis
Jan. 90	Adult	F	Eve R.			20	Lead toxicosis
Feb. 90	Adult	M	Duncan	8.8	ND	6.7	Gunshot
Mar. 90	1 year	F	Port Hardy	48	6.9		Inanition
Mar. 90	Adult	M	Coombs	49	10		Undetermined
Mar. 90	2 years	M	Sointula	21	5.1		Lead toxicosis
Mar. 90	Adult	M	Kamloops	42	7.0		Undetermined
May 90	Adult	F	Hornby Is.			6.6	Interspecific aggression
Mean ± SD				34 ± 18	7.3 ± 2.2	16.7 ± 9.4	

ND = nondetectable

Table 2
Data on the 15 Bald Eagles that showed evidence of subclinical lead exposure

Date	Age	Sex	Location	Lead in kidney (mg/kg)	Lead in bone (mg/kg)	ALA-d ratio	Initial clinical diagnosis
Jul. 89	1 year	M	Houston			2.8	Nutritional deficiency
Nov. 89	Adult	F	Surrey	ND	1.4		Intraspecific aggression
Dec. 89	Adult	F	Squamish	ND	0.8		Undetermined trauma
Dec. 89	1 year	M	Buckley Bay			3.9	Lead toxicosis/water soaked
Feb. 90	Adult	M	Powell R.	2.6	ND	2.3	Undetermined trauma
Feb. 90	Adult	M	Sointula	3.8	ND		Vehicle collision
Feb. 90	Adult	F	Nanaimo	ND	1.2		Electrocution
Feb. 90	Adult	M	Richmond			2.9	Pesticide toxicosis
Feb. 90	4 years	M	Campbell R.			2.6	Oiled
Feb. 90	1 year	F	Powell R.	ND	4.3	1.7	Undetermined trauma
Feb. 90	4 years	M	Comox	2.0	ND		Power line collision
Mar. 90	3 years	M	Ladysmith	ND	2.7		Undetermined trauma
Mar. 90	3 years	M	Port Alice	ND	0.8		Myositis
Apr. 90	Adult	F	Nanoose Bay			3.2	Undetermined trauma
May 90	Adult	F	Smithers			2.6	Intraspecific aggression
Mean ± SD				2.8 ± 0.9*	1.9 ± 1.4*	2.8 ± 0.7	

ND = nondetectable
* indicates mean calculated from detectable results only

Table 3
Data on the 41 Bald Eagles found to be free from lead exposure

Date	Age	Sex	Location	Lead in kidney (mg/kg)	Lead in bone (mg/kg)	ALA-d ratio
May 88	Adult	F	Port Hardy	ND	ND	
Jun. 88	2 years	M	Summit Lake	ND	ND	
Jun. 89	Adult	F	Port Hardy		ND	
Aug. 89	1 year	M	Queen Charlotte Is.			1.1
Nov. 89	1 year	F	Port Alberni			1.8
Nov. 89	1 year	F	Unknown			1.2
Dec. 89	Adult	M	Powell R.	ND	ND	
Dec. 89	1 year	F	Sandspit			1.1
Jan. 90	1 year	F	Comox	ND	ND	1.1
Jan. 90	4 years	M	Squamish			1.0
Jan. 90	2 years	M	Minstrel Is.	ND	ND	
Jan. 90	Adult	M	Qualicum			1.1
Jan. 90	Adult	F	Surrey			1.2
Jan. 90	1 year	M	Kitimat			1.1
Jan. 90	Adult	M	Point Roberts			1.3
Jan. 90	Adult	M	Richmond			1.7
Jan. 90	Adult	F	Powell R.			1.3
Feb. 90	2 years	M	Sointula			1.5
Feb. 90	1 year	F	Port Hardy			1.4
Feb. 90	1 year	F	Kelsey Bay	ND	ND	
Feb. 90	2 years	M	Surrey	ND	ND	
Feb. 90	2 years	F	Mission			1.2
Feb. 90	1 year	M	Surrey			1.3
Feb. 90	Adult	M	Qualicum			1.1
Feb. 90	Adult	F	Chain Is.			1.1
Feb. 90	Adult	M	Sechelt			1.0
Feb. 90	Adult	F	Quadra Is.	ND	ND	
Feb. 90	1 year	M	Upper Squamish	ND	ND	
Mar. 90	1 year	F	Englishman R.	ND	ND	
Mar. 90	1 year	M	Nanaimo	ND		
Mar. 90	1 year	F	Smithers	ND	ND	
Mar. 90	1 year	M	Nanaimo	ND	ND	
Mar. 90	Adult	F	Abbotsford			1.0
Mar. 90	3 years	F	Woss	ND		
Apr. 90	Adult	F	Campbell R.			1.4
Apr. 90	1 year	F	Campbell R.			1.0
Apr. 90	Adult	M	Port Hardy			1.0
Apr. 90	Unknown	F	Bowen Is.			1.1
Apr. 90	Unknown	M	Prince Rupert			1.0
Apr. 90	Adult	M	Prince Rupert			1.1
Jun. 90	Adult	M	Abbotsford			1.0
Mean ± SD						1.2 ± 0.2

ND = nondetectable

Table 4
Summary of the presence of lead shot or lead fragments in gizzard contents of waterfowl collected in the Lower Mainland and Vancouver Island in 1988–89

	Canada Goose		Lesser Snow Goose		Mallard		American Wigeon		Northern Pintail		Total		
Location	N	Pb	N	Pb	N	Pb	N	Pb	N	Pb	N	Pb	%
Unknown	4	—	4	—	7	2	5	1	9	—	29	3	10.3
Abbotsford	—	—	2	—	—	—	—	—	—	—	2	—	0.0
Boundary Bay	8	1	—	—	6	1	10	3	—	—	24	5	20.8
Fraser R.	—	—	7	3	—	—	—	—	—	—	7	3	42.9
Ladysmith	—	—	—	—	18	1	—	—	—	—	18	1	5.6
Pitt R.	—	—	14	2	2	1	—	—	—	—	16	3	18.8
Westham Is.	16	1	10	—	59	14	26	7	39	4	151	26	17.3
Total	28	2	37	5	92	19	41	11	48	4	246	41	16.7
%	7.1		13.5		20.7		26.8		8.3		16.7		

Pb = presence of lead shot or lead fragments

Table 5
Diagnosed cause of death or illness in all eagles examined to date (N = 267) and in eagles tested for lead exposure (N = 65)

Diagnosis	All eagles (%)	Eagles tested for lead (%)
Flight collision	3.0	7.7
Power line collision	2.2	3.1
Vehicle collision	10.1	4.6
Undetermined trauma	15.7	21.5
Total trauma	31.0	36.9
Lead	8.6	13.9
Pesticide	3.7	6.2
Mercury	0.4	0
Undetermined toxicosis	0.8	0
Total toxicosis	13.9	20.1
Electrocution	14.6	7.7
Fall from nest	6.4	0
Gunshot	6.0	1.5
Intraspecific aggression	4.9	4.6
Water soaked	5.2	3.1
Oiled	0.8	3.1
Inanition	3.0	7.7
Trapped	3.0	0
Infectious disease	4.1	4.6
Undetermined cause	4.9	1.5
Other causes	1.5	9.2

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